The Role of a Wildlife Reservoir in the Epidemiology of Bovine Tuberculosis

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ABSTRACT

The objective of this project was to study the epidemiology of bovine tuberculosis in the presence of a wildlife reservoir species. Cross-sectional and longitudinal studies of possum populations with endemic bovine tuberculosis infection were analysed. The results were used to develop a computer simulation model of the dynamics of bovine tuberculosis infection in possum populations. A case-control study of breakdowns to tuberculosis infection in cattle herds in the Central North Island of New Zealand was conducted to identify risk factors other than exposure to tuberculosis in local possum populations.

The cross-sectional study was based on data gathered some years earlier in the Hauhungaroa Ranges from a number of traplines with a total length of 60km, hence it provided information about the epidemiology of possum tuberculosis on a large geographical scale with varying environmental conditions. The results from the study showed that disease occurrence was clustered in space with local prevalence reaching up to 20% while the overall prevalence was about 1.2%.

The longitudinal study was conducted using an area of 21 hectare of mixed pasture and bush on a sheep/beef farm. The study showed that incidence and prevalence of tuberculosis infection in possum populations has distinct spatial and temporal patterns. Environmental conditions were a major factor in determining the temporal pattern. Spatial and temporal analysis of the occurrence of different strains of *Mycobacterium bovis* allowed inferences to be made about the importance of particular transmission paths. Survival of possums depended on environmental conditions and tuberculosis disease status. Adverse weather conditions increased mortality and the incidence of clinical disease in possums. On average clinically tuberculous possums survived for about 2 to 3 months from the onset of clinical disease.

The simulation model uses a Monte-Carlo modelling approach and incorporates geographical features. Biological mechanisms which are considered important for population and infection dynamics were implemented in the model. These include mating, density-dependent and -independent mortality, pseudo-vertical transmission, transmission through spatial or temporal proximity, and transmission during mating contact. Each possum's movements and behaviour are simulated on a day-by-day basis. Simulations are conducted using a geography and possum population based on data from the longitudinal field study. For preliminary validation, model output was compared with field data from the longitudinal study. Sensitivity analyses and some initial simulation experiments were conducted to identify areas in the model structure which require the collection of additional field data. Use of the model for simulation of a possum population occupying a 400ha area in the Central North Island of New Zealand is demonstrated.

The case-control study of breakdowns to tuberculosis infection in cattle herds showed that in the Waikato area of New Zealand exposure to tuberculosis infection in local possum

populations was not the dominant cause of breakdowns when the study was conducted in 1989/90, at a time when tuberculous possums were first discovered in the region. Farmers who had breakdowns tended to follow cattle purchase and management practices which traditionally have been considered to put farms at risk of introducing tuberculosis. The results of the study indicate that there was a lack understanding among farmers about the epidemiology of tuberculosis.

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CHAPTER 1

INTRODUCTION

For a brief period after the advent of such advances in infectious disease control as antibiotics and modern vaccines, it was possible for people to envision a future with total control over most infectious human and animal diseases. This view changed dramatically with the advent of a growing range of apparently new diseases such as acquired immunodeficiency disease (AIDS) and the re-emergence of 'old' diseases such as human and bovine tuberculosis. It became recognized that traditional purely laboratory-based scientific approaches have difficulty in providing adequate solutions for complex disease problems. In keeping with a similar development in physics, this positivistic and reductionistic epistemology has had to be replaced by a more comprehensive scientific method which allows for uncertainty and offers a more holistic view of the nature of problems (van Gigch 1991).

With regard to tuberculosis infection, after a period in which the disease was seen to have declined to negligible proportions, quite pessimistic visions of the future have been drawn more recently both for human disease in most countries and for animal disease in countries where wildlife reservoirs exists. In the case of human tuberculosis, an alarming increase in tuberculosis incidence in humans has been seen in the United States (Glassroth 1992) and elsewhere. Discussing the rise of drug-resistance in *Mycobacterium tuberculosis,* Bloom points out that we may be working our way back to a frightening future (Bloom 1992). Recently, reports have come from the United States about deaths in HIV-infected prisoners caused by multi-drug resistant strains of *Mycobacterium tuberculosis*, which would pose a very major health hazard if they become established in the wider community. Stanford *et al* (1991) suggest that we are facing one of the greatest public-health disasters since the bubonic plague.

Although bovine tuberculosis does not represent a significant human health hazard now in the way it did in the nineteenth century, and the disease in animals has responded very favourably to classical test and slaughter control methods in the absence of a wildlife reservoir, such reservoirs have emerged in various countries over recent decades, and have undermined control efforts under these circumstances. Although various wild and feral species can become infected with *Mycobacterium bovis* (Allen 1991), the most important reservoir hosts so far discovered are the European badger (*Meles meles*), the Australian brushtail possum (*Trichosurus vulpecula* Kerr) and various species of deer.

In New Zealand, Australian brushtail possums had been introduced to the country on multiple occasions between about 1840 and 1940, and wild populations grew and expanded from initial colonization sites to the point where they occur in most of the country and total numbers have been estimated at about 70 million. In 1967 the first tuberculous possum was found in an area of the West Coast of the South Island where it had proved difficult to eradicate bovine tuberculosis using methods which had been successful in other parts of the country. Over the following 25 years it has gradually become clear that possums are a major reservoir species for *Mycobacterium bovis* infection in New Zealand, although not in their native Australia. In the presence of this source of infection eradication of bovine tuberculosis from the cattle population seems unlikely to succeed. Traditional methods of disease control

such as test and slaughter of cattle and culling of possums have not proved adequate to achieve effective control of tuberculosis in either the cattle or the possums.

It was concluded from an examination of the situation that it was essential to better define the epidemiology of the disease in possums in New Zealand. Using an epidemiological approach, it would become possible to adopt a structured systems approach towards identifying and implementing effective control of tuberculosis infection in domestic cattle populations which are exposed to tuberculous possums. This study forms the first stage of a long term research program to achieve these goals.

CHAPTER 2

BACKGROUND TO THE STUDY

Since the start of the tuberculosis eradication campaign in 1945, tuberculosis incidence in New Zealand cattle has been successfully reduced from about 8.6% to 0.11% in dairy cattle and from 0.8 % to 0.29% in beef cattle, using figures derived for the testing year 1990/91 (Hennessy 1986, O'Hara 1992). In 1991 2.7% of herds in the national population (n=47000) were classified as infected or under movement control restrictions. However, despite continuing "test and slaughter" efforts, in 18 areas (in 1991) of the country reactor incidence remained significantly higher than in the rest of the country. These 'endemic areas' are now considered to be continuously infected with tuberculosis because of the existence of a reservoir of infection in possums and possibly in other wild animals. Some have expressed the view that eradication of the disease in cattle may be impossible (O'Hara 1992). After the first possum with tuberculosis infection was discovered in 1967, it took a few years before the first surveys for non-bovine sources of tuberculosis infection were conducted. It was then found that in most 'endemic' areas tuberculosis was present in the possum population. Introduction of extensive possum control operations resulted in a prolonged reduction in cattle reactor rates (Hennessy 1986).

An experiment to determine the risk from a tuberculosis - infected possum population to cattle grazing adjacent areas was conducted on the West Coast of the South Island in 1970/71. Twenty nine tuberculosis-free calves were introduced into an area where a tuberculosis prevalence of 12% had been found previously in the possum population. After 6 months 26 animals were tuberculin positive, and lesions were found at slaughter in 16 (Davidson 1976).

In a summary of experiences with *Mycobacterium bovis* infection in cattle on the West Coast Stockdale (1975) states that the likelihood of recurrence of cattle tuberculosis on individual farms appeared to be linked to the prevalence of tuberculosis in possums. Properties with a persistent problem seemed to have in common a topography and vegetation which made it difficult for possum control to be successful. Tuberculosis "break downs" in cattle herds usually coincided with the presence of only one tuberculous possum, which was found during subsequent investigations on the property. Often, possum tuberculosis was found in local clusters and it was then possible to demonstrate that only animals which had grazed the associated areas were infected. Other potential wildlife reservoirs of tuberculosis infection including deer, pigs, ferrets and hedgehogs were considered to be a minor risk of infection for cattle. Stockdale concluded that there was ample circumstantial evidence that *Mycobacterium bovis* infection cycling in possum populations was the major factor contributing to persistent tuberculosis problems in cattle in New Zealand.

In the Wairarapa the first tuberculous possum was detected in 1969. From then until 1981 tuberculous possums were found on 114 farms all over the Masterton district. Most of these farms, and very often their neighbouring properties, had a persistent tuberculosis problem in cattle (Shortridge 1981).

A number of reports have found that in problem areas cattle reactor rates decreased significantly over the years following a reduction in the possum population density as a result

of a control operation (Hennessy 1986). After recognizing that the disease situation did not respond to normal testing procedures a 3 monthly whole herd testing scheme was introduced in Buller County. This resulted in a reduction of reactor incidence from 12.5% of 4306 cattle tested in January 1970 to an average incidence of 4.97% (s.d. 1.02) in April 1972. Extensive aerial poisoning operations to kill possums were conducted in June-July 1972 and were followed by ground control work over the following years. Cattle reactor incidence dropped to an average of 1.36% (s.d. 0.46) over the period from January 1973 to July 1975 (Stockdale 1975). In the Wairarapa, cattle tuberculosis incidence averaged 2 - 2.3% per year over four years before possum population control was implemented. After two years reactor incidence reached a minimum of 0.3 and started to increase steadily over the following 4 years to about 0.7 (Hennessy 1986). Recurrence of tuberculosis in cattle has been observed in several areas of the country a number of years after initial reduction following possum control operations (Batcheler and Cowan 1988).

In the Central North Island between 1975 and 1987 the size of the area with endemic tuberculosis infection in possums was increasing by a multiplication factor of 1.104 every year. Assuming that spatial spread follows a model of logistic growth, tuberculosis infection would be present in all North Island possum populations by the year 2024 and in all South Island populations by 2031 (Batcheler and Cowan 1988).

Following recognition of tuberculosis-infected possums as a significant source of persistent infection in cattle herds in the early seventies it became the policy of the Ministry of Agriculture and Fisheries to conduct possum population control operations in areas where there was a persistent cattle tuberculosis problem and tuberculous possums were found on or near the property. In the early eighties it was concluded that despite the huge financial investment incurred in reducing possum population density, total eradication of tuberculosis in the major areas with endemic tuberculosis infection in possums, was neither technically feasible nor cost-effective. The modified objective therefore became to achieve a break-even point between the costs of reactor compensation and possum population control (Anon. 1984). In 1985 the objectives of the tuberculosis control scheme were redefined and included the cost-effective prevention of spread of tuberculosis infection from endemic areas (Anon. 1985). According to the Chief Veterinary Officer's annual reports for the period it was decided in 1986 in consultation with ecologists to establish low possum density buffers of about 3-5 km width on the edge of a major endemic area in Central North Island to contain the spread of tuberculosis infection in possum populations (Anon. 1986, Livingstone 1988). This policy was revised in 1989 when tuberculous possums were found outside the buffer zone. The current objective is to restrict the spread of tuberculous possums from the major endemic areas and to reduce cattle tuberculosis incidence by localised cost-effective possum population control within these areas. Based on a results from a deterministic simulation model, management plans for eradication of tuberculosis from possum populations were developed for a number of small- to medium-sized endemic areas (Anon. 1989).

At the end of the tuberculosis testing year 1991/92 there were 20 areas in New Zealand where tuberculosis infection was present in possum populations. In 1991/92 about 83% of cattle reactors to the tuberculin test and herds under movement control restrictions were found in these areas (TB endemic and tuberculosis investigation areas; O'Hara 1993). The predicted cost of the New Zealand bovine tuberculosis control programme for the financial year 1992/93 was NZ$ 21.82 million. Of this sum a total of NZ$ 5.1 million was to be spent on possum population control (Anon. 1992).

CHAPTER 3

REVIEW OF THE LITERATURE

In this review special emphasis will be given to the epidemiology of bovine tuberculosis in species of potential importance as disease reservoirs. A section on human tuberculosis is included to take advantage of the wealth of historical information available on the epidemiology of tuberculosis in humans.

EPIDEMIOLOGY OF TUBERCULOSIS IN HUMANS

Tuberculosis infection occurs throughout the world. A recent report by the World Health Organization (Anon. 1992) stated that about a third of the world population is harbouring the pathogen. Tuberculosis in man is caused primarily by *Mycobacterium tuberculosis*, but *Mycobacterium bovis* remains a contributor to the total disease, even though it is now much less important than it was in the nineteenth century before control of transmission from cattle. The World Health Organization reports that in humans there are about 20 million active cases of tuberculosis in the world, who probably infect 50 to 100 million people (mainly children) annually. It is estimated that about 3 million die due to the disease every year, at least 80% in developing countries (Stead and Dutt 1988). As species identification is not carried out routinely, it is difficult to estimate the present contribution of *Mycobacterium bovis* to total tuberculosis morbidity and mortality in humans (Pritchard 1988).

Since the beginning of this century the epidemiology of human tuberculosis has been studied extensively. This review looks at parallels between mycobacterial infection in humans and animals. Rich (1951) has reviewed reports of the occurrence of tuberculosis caused by *Mycobacterium bovis* in humans. He writes that after Koch's insistence that this bacillus would not be pathogenic in man, it took a number of years before it was generally accepted that *Mycobacterium bovis* can cause all of the forms of tuberculosis which the human-type bacillus is able to produce. Milk turned out to be the most important source of *Mycobacterium bovis* infection for humans. Hence, the incidence of infection in any area where the bacillus was prevalent in cattle was determined by the extent to which raw milk and milk products were consumed, which in turn was determined by local habits. Rich quotes a number of studies from the beginning of this century where up to 60% of cases with bone and joint tuberculosis and 90% of cases with tuberculous cervical adenitis in Scottish children were found to be caused by infection with *Mycobacterium bovis*. At that time human tuberculosis of bovine origin was proportionately more common in the British Isles than in any other industrialised country in the world. It was estimated that around the beginning of this century at least 2000 children died annually from bovine tuberculosis in Great Britain, because powerful dairy interests had prevented the introduction of legislation requiring the pasteurization of milk sold to the public. Rich writes that infection and progressive disease caused by *Mycobacterium bovis* is more likely to occur in children than in adults. He attributes this to a combination of factors. These include, that children are more susceptible, that they are more likely to be exposed and that infection through the alimentary tract (which is the main portal of entry for the bovine bacillus) is easier to achieve. Rich extensively discusses the relative importance of the alimentary and respiratory tract as portals of entry with particular reference to *Mycobacterium tuberculosis*

infection in humans. He concludes that the respiratory tract is the most common portal of entry, and that to achieve infection via the alimentary tract far greater quantities of bacteria are needed. Rich writes that intrauterine infection occurs infrequently. He also mentions that infection can be induced by dropping tubercle bacilli into the conjunctival sac, which has led to cervical adenitis and generalized tuberculosis in the absence of a macroscopic local conjunctival lesion. Rich believes that there is no greater susceptibility in humans towards infection with *Mycobacterium tuberculosis* than there is to *Mycobacterium bovis*.

Yates and Grange (1988) suggest that despite virtual eradication of bovine tuberculosis infection in cattle in the region, about 1% of bacteriologically proven tuberculosis cases in South-East England are still caused by bovine tubercle bacilli. In the period 1954-1957 in areas in Germany with high cattle reactor rates, up to 25% of tuberculosis cases in humans were caused by *Mycobacterium bovis*. By comparison only 2.7% of human tuberculosis cases in an area with low cattle tuberculosis incidence were related to infection with *Mycobacterium bovis* (Schliesser 1982).

Blood and Radostits (1989) write that complete eradication of bovine tuberculosis has not been achieved in any country of the world, but in many a state of virtual eradication with minor recrudescences can be claimed. The complex epidemiology of this organism, which has one of the broadest host ranges of all pathogens, complicates control efforts (Grange and Collins 1987).

The transmission dynamics of *Mycobacterium bovis* infection changed considerably from prehistoric times to the present. This was mainly associated with the rise of civilization. There are some reports which consider the possibility that after the domestication of cattle a mutation occurred in the bovine bacillus to create the human type (Lancaster 1990, Wadsworth 1984). Due to its chronicity, tuberculosis would have been able to perpetuate itself in small communities. But, it has not been detected in many modern day isolated communities, which leads Lancaster to the conclusion that it can be thought of as a common or even true human disease only after the development of agricultural societies. There is some evidence from prehistoric skeletons that it existed in simple early human societies, even though it was uncommon among such groups (Cohen 1989, Clark *et al* 1987).

Stead and Bates (1980) describe *Mycobacterium tuberculosis* as having the potential to cause epidemics when introduced into any susceptible populations. They write that early skin-testing data shows that most people in Europe eventually became infected, but only some of these developed disease. One possible explanation of this is that there are variations in natural resistance to tuberculosis, which over several generations resulted in the elimination of the relatively more susceptible population, although a simpler explanation would be that not all individuals were exposed to sufficient risk factors to precipitate the conversion of their subclinical infection into clinical disease.

First reports on the current tuberculosis pandemic are from 16th century England. Stead and Bates estimate that the disease requires about 300 years to complete its course in one geographic area. There is some data which suggests that the present pandemic began in 16th century England, reaching a peak in 1750 during the early beginnings of the industrial revolution. During this period of urbanization the life-style of people changed significantly and as a result the risk of person-to-person spread was considerably higher. From England it spread all over the world, reaching high incidence in western Europe in the early 1800s, in the late 1800s in North America and still has not reached its peak in some developing countries. Stead and Bates suggest that the downward slope of tuberculosis mortality was only little affected by tuberculin testing, BCG vaccination and early chemotherapeutic efforts until in 1952 isoniazid was introduced as a chemotherapeutic agent.

Mercer (1990) reviewed changes in tuberculosis incidence and mortality since the 18th century in England. He writes that the disease had become endemic in the towns of England by the 18th century and 'consumption', as the disease in humans was called, contributed about 15-20% of deaths in the London Bills of Mortality. Sharing of airspaces with infected individuals along with under-nutrition were likely to have affected the risk of clinical disease establishing from initial contact, while other living conditions affected the risk of mortality on re-activation of initially dormant disease later in life. During both centuries people in towns were likely to have contracted primary infection at an early age. Its destructiveness has to be seen in the context of the other epidemic diseases to which everyone was exposed in overcrowded living conditions. Mercer writes that suppression of tuberculin reaction has been found during scarlet fever, glandular fever, measles infection and probably smallpox. He points out that the actual physical disruption produced by the respiratory complications of such infections was probably even more important. During the second half of the 19th century there was a decline in mortality, but the population continued to have 100% contact with the tubercle bacillus. Mercer suggests that this decline could have been an indirect benefit of vaccination against smallpox, since this disease probably re-activated latent infection or weakened the resistance of the survivors. He does not believe that any immuno-genetic changes can account for the decline in tuberculosis death rate, particularly as the trends for non-respiratory forms of tuberculosis did not fall much in the second half of the 19th century. Mercer thinks that changes in transmission were rather unlikely to account for the decline in mortality from tuberculosis, as during this time the population in towns was increasing most rapidly but there were only slight improvements in general living conditions. The first countries to industrialise were also the first to experience the downturn in tuberculosis mortality. But even in the 1940s tuberculosis case-fatality was about 50%. Mercer notes that in addition to preventive measures used against tuberculosis such as vaccination and chemotherapy, changes in family size (initially among the middle classes), and in housing conditions among the less well-off, could have been involved with a reduction of transmission rates and severity of air-borne infectious disease.

Stead and Bates (1980) reviewed factors of importance in the epidemiology of *Mycobacterium tuberculosis* infection in humans. They consider industrialization and urbanization resulting in crowded living conditions as more important than any other single factor. Low socio-economic status appears to be associated with increased tuberculosis mortality. Poor nutrition is probably less important, as are racial differences. Stead and Bates emphasize that individual infection is more likely, given a sustained and intimate contact with infectious persons resulting in the probability of multiple exposures. Since infection with *Mycobacterium bovis* has become a rarity in cattle and humans, infection through inhalation is considered the most important transmission path. Size of the inhaled particle is more important than the quantity of organisms. Stead and Bates quote a landmark study by Riley and colleagues (1962) which demonstrated that infectious particles having the aerodynamic properties of droplet nuclei were capable of passing through the duct system from a hospital ward occupied by sputum smear-positive patients and infecting guinea pigs kept on the roof. Droplet nuclei are so small that they can stay constantly airborne and can rapidly disperse throughout an enclosed atmosphere. These particles are not filtered by simple gauze masks. It was shown that in guinea pigs and mice almost all tubercle bacilli that were inhaled as single organisms, reached the lung alveoli and produced a tubercle. Survival of aerosolised bacilli is brief, with only about 1 percent surviving for several hours. It was therefore concluded that effective prevention of transmission has to be targeted at these aerosolised particles. In a recent official statement the American Thoracic Society (Anon. 1990) points out that because of the extraordinary significance of airborne droplet nuclei for successful transmission of *Mycobacterium tuberculosis* any of the other methods which were once thought to be important for preventing infection such as disposing of clothes and bedding, sterilizing fomites, using caps and gowns and gauze or paper masks, boiling dishes, and washing walls are unnecessary because they have no bearing on airborne transmission.

Schliesser (1985) reviewed the epidemiology of bovine tuberculosis in humans. He emphasizes the importance of aerogenous as well as alimentary transmission from cattle to humans. Traditionally infection was attributed mainly to the consumption of milk from tuberculous cattle. Schliesser quotes work by Schmiedel (1970) which suggests that due to its high fat content which increases absorption into the lymph system, milk represents an ideal medium for alimentary transmission. Schliesser notes the accepted fact that pasteurization kills pathogenic bacteria in milk if the correct procedures are followed. In the absence of pasteurization, bacteria contained in milk products can remain infectious for extended periods of time. This can be up to 100 days in butter and 322 days in certain types of cheese. Schliesser states that meat from tuberculous animals may constitute a significant risk of infection if available for consumption. *Mycobacterium bovis* has even been isolated from mince meat. Schliesser quotes Jensen (1937) who considered the risk of aerogenous infection as being higher in a shed with tuberculous cattle than in a hospital with tuberculosis cases. Infection by direct contact would be a risk factor for certain occupations such as farmers, abattoir workers and veterinarians.

In Australia human tuberculosis caused by *Mycobacterium bovis* is now rare. About 10 cases are recorded every year. Most of them show pulmonary lesions and are usually older people, but in earlier times a number of younger abattoir workers probably did become infected by aerosol transmission from cattle (Patel and Streeton 1990). In New Zealand the annual incidence of tuberculosis infection (including *Mycobacterium tuberculosis* and *Mycobacterium bovis*) averaged 4.2 cases per 100,000 population during 1985-90. The risk of infection was higher in Maori and Pacific Islanders compared with people of European ethnic background (Stehr-Green 1992). During 1988 1.8% of a total of 228 *Mycobacterium* isolates from human tuberculosis cases were typed as *Mycobacterium bovis*. All 4 cases were between 41 and 60 years of age. In 3 cases it was possible to isolate *Mycobacterium bovis* from sputum, in all cases from pleural fluid and in 1 of the cases from a urine sample. One case each was reported from Hamilton and Wellington Area Health Board and 2 cases were identified in the Palmerston North area (Anon. 1989). During 1989 a total of 15 cases with specimens yielding *Mycobacterium bovis* isolates were reported by the New Zealand Communicable Disease Centre (Anon. 1989, Anon. 1990). Amongst these were three infants, 8 people older than 41 years of age, and 4 of intermediate age. 13 of the 15 individuals were males.

Concurrently with the recent epidemic of the human immunodeficiency virus (HIV) a resurgence of tuberculosis has been reported from many areas of the world where HIV infection is widespread. One example for which data are available is New York City (Brudney *et al* 1991). The authors discuss the possible reasons for this unexpected development. They found that the disease is a growing problem in impoverished populations, particularly in homeless people living in mass shelters. In this group non-compliance with the treatment regimen was found to be particularly common, further augmenting the disease problem. It was noted that despite the current belief that most tuberculosis cases in HIV patients are related to reactivation of old tuberculosis infection, there is also a substantial amount of primary tuberculosis. This is becoming more likely as there are more inadequately treated individuals with active tuberculosis crowded together with highly susceptible HIV-infected homeless people. In New Zealand an outbreak of tuberculosis in a ward of 30 psychogeriatric patients was reported, which was probably caused by a single undiagnosed fatal index case (Taylor *et al* 1991). The incident probably resulted in three deaths and the infection of 43.3% of the patients accommodated in the closed geriatric ward. Non-compliance with treatment instructions is also likely to result in the appearance of drug-resistant strains of *Mycobacterium tuberculosis.* This has recently been reported from New York prisons, where 13 HIV-infected inmates died due to tuberculosis treatment failure (Purvis 1991).

The occurrence of AIDS and tuberculosis has been termed 'the cursed duet' (Chretien 1990). Tuberculosis is considered the only AIDS-related opportunist disease that can infect healthy members of the community. Tuberculosis infection may hasten the onset of AIDS in otherwise asymptomatic HIV-infected individuals, possibly by several years (Festenstein and Grange 1991). Contrary to the general belief that tuberculosis follows immunosuppression

caused by HIV infection, it has been suggested that immune suppressant conditions such as tuberculosis, malnutrition and others may have preceded HIV infection and facilitated its transmission (Packard and Epstein 1991). The recent developments in human tuberculosis point to the continued importance of this disease, which was once considered to be nearly conquered (Rich 1951).

EPIDEMIOLOGY IN DOMESTIC ANIMALS

Cattle

The first confirmed description of *Mycobacterium bovis* infection in cattle is based on a report by Columella in the year 40 A.D.. In the 17th and 18th century in Germany "Perlsucht" (a term referring to the grape-like lesions) was considered a symptom of syphilis, which resulted in strict procedures for the disposal of the affected animals. When this misconception was corrected, the control measures were removed. It was considered possible to eat meat from tuberculous animals and there were then no obstacles in the way of the spread of the disease. It was not until Robert Koch identified *Mycobacterium bovis* that the relationship between "Perlsucht" and lung tuberculosis in cattle was established (Schliesser (1982).

Based on the tuberculin test, the first disease control programme which was successfully implemented was Bang's eradication scheme in Denmark in 1892. In Germany such a programme based on tuberculin testing every animal was not considered economically feasible. Ostertag's eradication scheme which relied on the detection of animals with open lesions was introduced in Germany in 1912 on a voluntary basis. In 1939 the scheme was discontinued, because it was found that reactor rates were higher in herds which took part in the programme. In 1952 a compulsory tuberculosis control scheme was introduced in West Germany which was based on identification of infected cattle using the tuberculin test. The number of tuberculosis-free herds increased from 9.9% in 1952 to 99.7% in 1961. Schliesser (1982) discussed the major factors associated with maintaining reactor rates in cattle at a low level. Major problem areas included the specificity of the tuberculin test and the risk of reintroduction into tuberculosis free herds. The latter, Schliesser attributes mainly to international trade of animals and their products and to transmission from *Mycobacterium bovis* infected humans. He also points out the differences between the epidemiology of tuberculosis in humans and domestic animals.

During the last 100 years in industrialized countries human living conditions have improved and infection rates have decreased despite the absence of a disease eradication programme. On the other hand, during the same period of time changes in domestic animal husbandry occurred (such as increased herd sizes, higher stocking densities and greater numbers of animal movements) which facilitated transmission and spread of infectious diseases. In Germany by 1975 such good progress had been made that it was possible to reduce herd testing to once every three years. Since then, the number of infected herds has steadily decreased, but the number of reactors within infected herds has increased (Schliesser 1985).

Weber *et al* (1988) report that the proportion of reactors in infected herds increased with the time elapsed since the last tuberculin test.

In intensive production systems bovine tuberculosis is more common in dairy cattle than in beef cattle. This is related to the fact that dairy cattle are usually kept in sheds in close contact with one another whereas beef cattle are usually kept on pasture. The different age structure of the two major cattle production systems may also be of importance. Prevalence increases with age and dairy cattle are usually kept longer than beef cattle (Schliesser 1985). Francis (1958) quotes Villemin (1868) who observed higher prevalences of tuberculosis in those cattle which were kept indoors. In extensive cattle production systems in North and South America, in Asia and Africa tuberculosis usually is less prevalent. But in some situations (Africa, Australia) high incidences have been reported in range cattle. This may be related to aggregation of animals around watering points. Shortridge (1981) analysed the patterns of tuberculosis infection in a problem area in New Zealand. He considered congregation of animals around water ponds and dams as conducive to the spread of infection within herds.

Recent studies in Northern Ireland suggest that within- and between-herd transmission through aerosolised secretions may be of continued importance in the epidemiology of bovine tuberculosis. It was found that in cattle infected with *Mycobacterium bovis,* lung lesions and nasal excretion occurred frequently and could be diagnosed from 2 months after the last clear tuberculin test (McIlroy *et al* 1986, Neill *et al* 1988). Dunnet *et al* (1986) also mention in their report that analysis of breakdown data from Great Britain shows that tuberculosis is rarely transmitted to neighbouring herds. They conclude that the presence of non-reacting excreters is more likely to affect within- herd transmission. Dunnet *et al* mention the importance of the introduction of infection through purchases of tuberculous cattle. Especially in the early 1970s imported Irish cattle were related to a significant number of breakdowns in Great Britain. In Ireland in areas with a high incidence, transmission between cattle is considered the principal means of spread. In some areas other transmission paths are also thought to be of relevance (O'Connor and O'Malley 1989). Downey (1990) outlined the main reasons for failure of the Irish tuberculosis eradication programme in containing spread of infection. He mentions that one of the factors involved was residual infection within herds which had not been detected by tuberculin testing. A major reason for failure was spread from infected cattle to other cattle by movement and contact, especially under stressful conditions.

There have been few attempts to model the epidemiology of bovine tuberculosis infection within cattle herds. As part of the Australian Brucellosis and Tuberculosis Eradication Campaign (BTEC) a deterministic simulation of the interaction of bovine tuberculosis in range cattle was developed to evaluate the effect of different disease control strategies in northern Australia (Stoneham and Johnston 1987). In these areas in many large herds it had not been possible to eliminate infection despite immense testing and culling pressure. Herds appeared to clear up, but sometimes the disease re-emerged at even higher incidence than the original level. The results of several simulation model runs suggest that the disease can be maintained at low

levels in small matriarchal breeder groups (9 to 15 head) and is spread by wandering infected males. Disease build-up is generally slow except in situations where cattle congregate in very large numbers. Efficient mustering to ensure that all groups are represented in the yards for testing would probably be more important than test efficiency. It was recommended that when an infected female was detected all members of its social group should be culled to reduce the risk of undiscovered residual infection. The model was used to estimate the management pressure which had to be exerted for particular geographic locations to achieve tuberculosis eradication, depending on cattle density and water availability.

Livingstone (1985) developed a computer simulation model of tuberculosis in a cattle herd. He tested the hypothesis that tuberculosis can persist due to factors such as incomplete mustering, presence of anergic infectious animals in the herd and infrequent testing. Modelling results were compared with actual cattle TB testing information from the West Coast of the South Island, New Zealand. Progression of the disease after the addition of one infected animal was significantly slower in extensively farmed herds compared with those intensively farmed. It was concluded that infection cannot be maintained, given the current testing and culling pressure in this area, without an external source of infection.

Farmed Deer

In 1978, tuberculosis infection was reported from farmed red deer in New Zealand (Beatson 1985). It is now considered the most important bacterial disease in farmed deer in New Zealand and in the United Kingdom (de Lisle and Havill 1985).

It has been suggested that deer kept under farm conditions may be more susceptible to *Mycobacterium bovis* infection than are cattle. Given the circumstances with regard to behavioural and environmental factors, extensive lesions can develop rapidly which results in increased probability of spread within a herd (Clifton-Hadley *et al* 1991). As is the case with cattle, the pathogenesis of the disease is mainly dependent on the size of the infecting dose and the susceptibility of the host. The latter depending on variables such as genetic constitution, previous exposure to *Mycobacterium spp.*, nutrition, social status in the herd, handling stress and sex hormone levels (de Lisle *et al* 1985).

Recognizing the threat of tuberculosis to the deer farming industry, New Zealand, Denmark and Great Britain have all embarked on tuberculosis control or eradication programmes. They are mainly based on tuberculin skin testing and subsequent slaughter of reactors. Clifton-Hadley and Wilesmith (1991) write that due to the presence of endemic infection in wildlife in New Zealand and in the United Kingdom the eradication of tuberculosis in farmed deer may be difficult to achieve. Tuberculous captive deer have been implicated in New Zealand as a source of infection for possums which are an important wildlife reservoir for bovine tuberculosis (Livingstone 1988).

Other Domestic Animals

Prevalence of tuberculosis infection in small ruminants is believed to be linked to the disease frequency in other hosts such as cattle. In industrialized countries detection of tuberculous lesions in small ruminants is less common when disease levels within the cattle population decreases. In extensive animal husbandry systems transmission probabilities are low resulting in low overall prevalences (Schliesser 1985). In some instances prevalences of up to 5% have been observed in sheep flocks in New Zealand (Davidson *et al* 1981).

Disease levels in pigs also usually reflect the incidence in local cattle populations. Myers and Steele (1969) report that in 1921 in the U.S. 12% of hogs slaughtered under federal inspection were found to have tuberculous lesions. They note that in the mid west of the U.S.A. it was possible to trace 96% of swine carcass condemnations for tuberculosis to feeding of unsterilized skim milk or other dairy products or to keeping them together with cattle. Prevalence in pigs is thought to increase with age. The principal route of infection in the pig is the digestive tract, by consumption of milk or milk products, kitchen and abattoir scraps, and excreta from tuberculous cattle (Acha and Szyfres 1989). Transmission between pigs is considered epidemiologically insignificant, as lesions usually remain localized and pigs are slaughtered at an early age. High disease levels in cattle can result in prevalences of up to 20% in local pigs (Blood and Radostits 1989).

Lepper and Corner (1983) quote work by Snider and Cohen who found tuberculous lesions in four dogs and 24 cats out of 61 contacts on farms with *Mycobacterium bovis* infection. The authors concluded that on premises with tuberculosis infection surveillance of these species is recommended. Yet, it is unlikely that domestic dogs and cats represent a epidemiologically significant factor in the dynamics of tuberculosis infection.

EPIDEMIOLOGY IN WILDLIFE

Tuberculosis has been known as a serious clinical disease in wild mammals in captivity for more than a century (Thoen and Himes 1981). These authors write that it is widely distributed in wild mammal populations in the United States, where outbreaks caused by infection with *Mycobacterium bovis* have been reported mainly from zoos, game parks and primate colonies. Schliesser (1985) notes that in European countries sporadic incidents of bovine tuberculosis in wild mammals were mainly reported before eradication of cattle tuberculosis was achieved. Evidence from various countries in the world shows that given specific epidemiological circumstances significant levels of tuberculosis infection can be found in wild species such as buffalo, goats, pigs, deer, badgers and brush-tailed possums (Lepper *et al* 1983). The risk which these reservoirs of infection constitute for infection in domestic animals and man is difficult to estimate.

Badger

Badgers are an important reservoir of reinfection for cattle in the United Kingdom and Ireland (Zuckerman 1980, O'Connor and O'Malley 1989). The disease is considered to be endemic

throughout badger populations in both countries (Wilesmith 1991, Morris and Pfeiffer 1990). In Great Britain, local surveys of badger populations around areas with recently infected cattle herds revealed tuberculosis prevalences of up to 50% in localized areas (Wilesmith 1991). In the Republic of Ireland, during the period 1980-1989 17.4% of 3915 badgers examined showed evidence of tuberculosis infection. Infection was present in every county of the country (Dolan 1990). In a local post-mortem survey in an area with a continuing TB problem in the cattle population 48% of 30 badgers showed tuberculous lesions (McAleer 1990). Wilesmith (1991) reviewed the epidemiology of bovine tuberculosis infection in badgers based on the results from a longitudinal study in Gloucestershire, in the southwest of England. Most badgers get infected via the respiratory route. Submandibular abscesses are often found as the first clinical sign of infection. Transmission through bite wounds from territorial fights could account for a higher prevalence in males. Mother to cub transmission could be a major epidemiological factor. It is suggested that adult females are responsible for maintenance of infection within a population. A large proportion of excreting (ie infectious) tuberculous badgers survived for more than 12 months. Mortality induced by tuberculosis does not seem to have an effect on population density. Single badgers with advanced tuberculous lesions shedding large numbers of bacteria in their urine are thought to be the primary source of infection in cattle. Hence, the probability of transmission of infection from badgers to cattle is low and probably occurs mainly as a sporadic incident. The risk increases with increasing badger density (Wilesmith 1991).

In contrast to the findings from the above field study, Mahmood *et al* (1987) challenged badgers with bovine tubercle bacilli in an experimental study and found that intradermal inoculation was almost always successful and intratracheal challenge always failed. The badgers did show immune responses which enabled them to hold the disease in check for up to 22 months. They begin to shed organisms only in the late stages of the disease. Disease is found in local "pockets" of infection, which do not necessarily equate with individual social groups. They may involve a number of contiguous groups (Dunnet *et al* 1986).

Benham and Broom (1989) studied the interactions between badgers and cattle on pasture. They found that badgers were approached by cattle of all ages. Badgers preferred not to use pastures occupied by cattle and avoided approaching closer than 10-15m from cattle. They concluded that tuberculosis transmission would be more likely through badger products such as urine or faeces than direct contact. Other researchers found that badgers did not always avoid cattle (Kruuk 1989). Kruuk also found that badgers need short-grazed pastures to feed efficiently, which could bring them into contact with cattle. Badgers defend their territories ferociously. They form social groups which use the same territory and setts. Female offspring tend to stay with their mother's group. Young males stay for extended periods of time within their mother's social group. They eventually may emigrate into a vacancy in a neighbouring group. This tendency to stay close to their mother's home range could explain why infection has not spread evenly through areas with endemic infection (Cheeseman *et al*

1988). Stuart and Wilesmith (1988) consider that the social structure of the population, their subterranean existence, social grooming and group sleeping are all important factors for maintenance of infection in a badger population.

An attempt has been made to develop a simulation model of the population dynamics of the badger and the epidemiology of bovine tuberculosis (Anderson and Trewhella 1985). A simple mathematical model was developed which was mainly aimed at identifying areas where current knowledge was inadequate. It was recognized at that time that the understanding about the epidemiology of the disease was only limited. The modelling results suggest that average prevalence is positively correlated with badger density. The disease should be able to persist endemically at low badger population densities due to factors such as frequent pseudo-vertical transmission and long survival periods of infectious animals. Anderson and Trewhella (1985) suggest that a high risk of cattle TB breakdowns is probably associated with high tuberculosis prevalence in infected badgers, high cattle herd density on pastures close to good badger habitat and farm management practices such as allowing badgers access to cattle sheds, salt licks and water troughs. The authors predict that very substantial reductions in badger abundance are necessary to induce marked changes in prevalence within the badger population. However, moderate reductions in badger density would significantly reduce, but not eliminate the risk of cattle herd infections.

Brush-Tailed Possum

Between the beginning of the nineteenth century and the start of this century the Australian common brushtailed possum (*Trichosurus vulpecula* Kerr) has been introduced and liberated at numerous locations in New Zealand (Pracy 1962). The susceptibility of possums to *Mycobacterium bovis* infection was first experimentally demonstrated in 1948 by Bolliger and Bolliger (1948). In 1967 the first possum with tuberculous lesions to be identified in New Zealand was found by a trapper on a farm with a persistent tuberculosis problem. In this and later cases it was reported that in all tissues examined, large numbers of *Mycobacterium bovis* organisms were present and the animals were discharging organisms through open sinuses or the respiratory system (Ekdahl 1970).

As a result of these findings, in 1970/71 the New Zealand Ministry of Agriculture and Fisheries conducted an epidemiological survey of possum tuberculosis in the Buller and Inangahua Counties. The study had a duration of one year. Possum kill operations were done at monthly intervals. 4.9% of 5908 possums captured showed tuberculous lesions on post mortem examination. Monthly prevalence ranged from 2.5% in August to 9.35% in December. In mature possums there was a trend towards higher prevalences in summer than in the winter months, although the difference was not statistically significant. Cattle tuberculosis incidence rates declined from the beginning to the end of the survey. 65% (N=100) of tuberculous possums had lung lesions and 52.6% (N=81) had lesions in the axillary and pre-scapular lymph nodes. It was 2.3 times as likely that infection would be found in a possum population if infected cattle were grazing in the same area, although the difference was not statistically

significant ($p > 0.1$). A "spring rise" in cattle tuberculosis incidence was attributed to the fact that on the New Zealand West Coast it is common practice to graze herds in bush areas during winter months (Cook 1975).

In 1973/74 a one year study of *Mycobacterium bovis* infection in possums involving two-day trapping surveys at two-monthly intervals was conducted in the Hohonu Range, New Zealand, in a cooperative project between the New Zealand Forest Research Institute, Christchurch and the Ministry of Agriculture and Fisheries. An overall tuberculosis prevalence of 7.7% in a total of 1486 possums was found. Both tuberculosis prevalence and relative density of possums were highest on pasture and decreased from the bush/pasture margin to the remote forest. Tuberculous possums were clustered in small foci of 2-5 animals. It was suggested that indirect transmission through sequential or simultaneous den sharing might be a important infection route. Seasonal variation in prevalence was observed, with highest levels in autumn and winter along the pasture margin and in spring within the forest. It was suggested that possums were at higher risk of infection during summer and autumn months due to increased foraging and social activities and that the rise in detectable prevalence in winter reflects the length of the incubation period. It was found that infection was more likely in immature male possums than in immature female possums. The body condition of the possums appeared to be worse in infected than in uninfected possums. 29.5% of tuberculous animals showed lesions in the axillary lymph nodes and 55.4% in the lung (Cook undated, Coleman 1988).

In an experimental study on the course and pathology of the disease in possums it was possible to demonstrate initiation of infection by subcutaneous and intranasal inoculation and transmission through direct and close contact, both by aerosol and by mother - joey contact (pseudo-vertical). Disease developed rapidly to a fatal stage and lesions were typically widely disseminated. However, it was realized that the disease process might have been accelerated by reduced body defenses in captive possums and that the infection dose was very high (O'Hara *et al* 1976).

In 1981 the pathology and histopathology of *Mycobacterium bovis* infection in possums was reviewed by Julian (Julian 1981). Distribution of lesions was summarised, including data on 327 tuberculous possums necropsied in three different areas. In 62% (N=203) of the cases, superficial lymph nodes and in 61.8% (N=202) the lungs were involved in the disease process. The pathological appearance of affected somatic lymph nodes is typically a soft fluctuating abscess with a diameter of up to 4 cm containing semi-liquid lime green pus, which on occasion may form open sinuses to the exterior. In visceral organs and lymph nodes white to yellow nodules of up to 2 cm in diameter are most commonly seen. These lesions may be multilobular with a more caseous centre than is seen in typical somatic lesions. Miliary white lesions occur in lungs, liver, spleen or kidneys. In lungs, generalised grey-white consolidation of lobes or part of a lobe has been observed. Histopathologically, lesions consist typically of granulomatous tissue, with no distinctive fibrous capsule and with varying amounts of

amorphous eosinophilic debris centrally. Lymphocytes, plasma cells, macrophages, giant cells and many neutrophils in great concentration around the caseous material were observed in the reaction process. Large numbers of acid-fast organisms were present. Typical tubercles (including epithelioid cells, Langhans' giant cells and capsule formation) were rarely seen.

Results of a preliminary analysis of the data from the possum tuberculosis survey in the Hauhungaroa Ranges have been published previously (Hennessy 1986), but are analyzed more fully in this thesis. A mean prevalence of 1.4% was reported. There was no association between relative density estimates (derived from trapping and faecal pellet count data) and tuberculosis prevalence in possums. No statistically significant association between infection and sex, breeding ability and condition was found. Sexually mature animals were more likely to be infected than immature animals ($p < 0.05$). A correlation coefficient of 0.9 was observed between cattle tuberculosis incidence (averaged estimates for 11 zones) and possum tuberculosis prevalence. It was concluded that level of infection in cattle represents a good indicator for level of infection in adjacent possum populations. Hence, in areas with endemic tuberculosis infection in possums direct inference from presence of infection in cattle to presence of tuberculous possums would be possible.

Based on current knowledge of possum ecology and the epidemiology of tuberculosis in possum populations a deterministic simulation model was developed to aid the understanding of the disease problem, evaluate different options for control strategies and identify future directions of research. The model suggested that disease in possums has to be aggregated locally at high prevalence levels to persist at the relatively low overall prevalence which has been observed in possum population surveys all over the country. It showed that assuming immigration is insignificant, eradication of tuberculosis infection within a possum population is theoretically possible if 16% of the possum population is removed every year. The model predicts that control of tuberculosis in endemic areas is most likely to be successful using repeated single possum population control operations or an initial single control operation, followed by sustained cropping or vaccination. In the light of the high cost involved in intensive possum control operations it was suggested that separation of cattle from margins of high-density possum habitats, or ground control operations targeted at clusters of disease aggregation may be more cost-effective (Barlow 1989).

Feral Buffalo and Bison

Bovine tuberculosis is known to be endemic in feral swamp buffalo populations of the Australian Northern Territory. Hein and Tomasovic (1981) found a prevalence of 0.017 in 11322 buffalo examined during routine post-mortem examination at 2 abattoirs during 1979. This was a significant reduction compared with a prevalence of 0.16, which was reported by Letts (1964) in 1964. This reduction in prevalence was explained by Hein and Tomasovic (1981) as due to selective harvesting of mature animals for meat, resulting in removal of many tuberculous animals from the population. But as prevalence was highest along the relatively narrow coastal strip of land, which was mainly sampled during the earlier survey, differences in

habitat may provide another explanation for the decrease in prevalence. The authors suggest that lower population density of feral buffalo may affect transmission probability. The large proportion of cases with sole or predominant involvement of the thoracic organs suggests that as in cattle the respiratory route is the most important transmission path in feral buffalo. McCool and Newton-Tabrett (1979) indicate that 97% of buffalo tuberculosis in northern Australia is contracted via the respiratory route. They considered this finding rather surprising in view of the work by Tulloch (1978) on buffalo behaviour which showed that there was ample opportunity for both alimentary and respiratory infection. In fact, Tulloch was reported to have estimated buffaloes were spending on average 4 hours per day in intimate contact in communal wallows. McCool and Newton-Tabrett write that this extended period of close contact is not seen in northern Australian cattle when resting around watering points, only during the few minutes when they drink from crowded water troughs. They concluded with respiratory transmission being the main method of spread, sources of mycobacteria in the environment are probably unimportant. Hence, control of tuberculosis in feral buffalo populations should be possible through standard tuberculosis control measures.

Freeland and Boulton (in preparation) developed a model of the epidemiology of bovine tuberculosis in swamp buffalo in the Northern Territory of Australia. They included the effects of social groups into their model. Modelling results suggested that the mode of group size regulation has a major effect on prevalence levels. The importance of matriarchal breeder groups was stressed for maintenance of infection. Spread of disease would depend on the mode of group size regulation (emigration of matriarchal family clans). Small group sizes which resulted from migration of clans were likely to result in low prevalences. Infected males could potentially spread the disease during emigration or when visiting females from other social groups during breeding season.

Woodford (1982) studied the occurrence of tuberculosis in wild Cape buffalo in Ruwenzori National Park, Uganda. He found tuberculous lesions in 10% of 52 buffaloes from a random sample and 38% of 64 animals which were selected based on being in poor condition. *Mycobacterium bovis* was identified in 12 of 14 cases. Most cases appeared to be infected by respiratory transmission and no lesions were seen which could be ascribed to alimentary infection. The author explains this finding with the close herding habits of wild buffaloes and their propensity for wallowing in tight groups in small mud holes which facilitates droplet transmission. Woodford concludes that bovine tuberculosis could cause an annual mortality of about 1% in this particular area.

In 1961 Choquette *et al* (1961) reported that they found tuberculous lesions in 50% of 436 bison from Wood Buffalo National Park, Canada, which had reacted to the tuberculin test. They quote other work by Fuller who wrote that tuberculous lesions had been found in 39% of 1508 bison slaughtered between 1952 and 1956. The same authors refer to a report by Hadwen who during the period between 1923 and 1939 found tuberculous lesions in 53.4% of 12,005 bison slaughtered at Wainwright. Choquette *et al* examined another 500 bison from Elk

Island National Park, which were not tuberculin tested and did not show any lesions on necropsy. They conclude that in Wood Buffalo National Park current tuberculosis control based on tuberculin testing and slaughter of reactors would be at best a disease reduction program. More recently concern has been expressed again about the levels of tuberculosis infection in bison in Canada. Between 1983 and 1985 during a survey in and around Wood Buffalo National Park, Canada, 21% of 72 bison found dead showed tuberculous lesions on post-mortem. The results suggest that infection occurred primarily via the respiratory route. It was concluded that the disease was endemic within the population and that therefore there was a growing risk of spread to uninfected bison and cattle (Tessaro *et al* 1990).

Wild Deer

Infection with *Mycobacterium bovis* has been reported from a number of free-ranging deer species, as pointed out in a recent review by Clifton-Hadley and Wilesmith (1991). These authors and others report that there have been some incidents where infected wild deer were suspected of introducing infection into captive deer populations (Mackintosh and Beatson 1985). Mackintosh and Beatson write that in New Zealand a high proportion of wild deer captured or shot was found to be infected with *Mycobacterium bovis*. It has been suggested that in New Zealand where deer had been live-captured in many parts of the country and then traded for breeding purposes, infected animals probably provided a means for introducing tuberculosis infection into areas which previously had been free of tuberculosis.

Other Wild Animals

Wild pigs have been found to be infected with *Mycobacterium bovis* at significant levels in a number of countries. Letts (1964) confirmed *Mycobacterium bovis* infection in 54% of 149 tuberculosis-like lesions from a total of 260 wild pigs in Australia's Northern Territory which were autopsied. He associated this relatively high prevalence with the pigs living in close association with swamp buffalo. At the end of each dry season hundreds of old buffalo die, thereby providing food and a potential source of infection with *Mycobacterium bovis* taking into account the high incidence of tuberculosis in wild buffalo in the Northern Territory. Corner *et al* examined 751 wild pigs in Australia's Northern Territory and found a infection prevalence of about 19%. No pulmonary lesions were found. Corner *et al* concluded that the wild pig is probably an end host for *Mycobacterium bovis* and not a significant source of infection for cattle. Recently a survey was conducted in New Zealand, where 251 wild pigs were post-mortemed and 31% were found to have tuberculous lesions (Wakelin and Churchman 1991). The authors suggested that the disease possibly had spread between pigs by aerogenous transmission because 33% of infected pigs had either lung or bronchial lymph node lesions. This finding suggests a possible difference in the epidemiology of *Mycobacterium bovis* in Central Otago, New Zealand, because in general the importance of infection in pigs through the digestive tract is emphasized in the literature.

Other Species

Feral goats were found with tuberculosis prevalences of up to 31% within individual groups in areas with endemic tuberculosis in New Zealand (Sanson 1988). The epidemiological significance of bovine tuberculosis in goats is generally considered as minimal. In most cases it is related to the presence of a reservoir of infection in another species, such as the brushtail possum in New Zealand.

Woodford (1982) found that bovine tuberculosis infection was endemic in the wart hog population of Ruwenzori National Park, Uganda. He concluded that the disease must have been introduced with domestic cattle.

Infection in wild carnivorous species has to be expected in areas with endemic tuberculosis in important infection reservoir species such as for example the brush-tail possum, cattle and deer in New Zealand. Allen (1991) reviewed the occurrence of bovine tuberculosis in feral species other than cattle, possums and deer in New Zealand. He suggests that most of the few cases with bovine tuberculosis in feral cats and ferrets which were found in New Zealand were related to infection in at least one of the major reservoir species mentioned above. Cats and ferrets are unlikely to contribute significantly to the maintenance and spread of bovine tuberculosis infection.