Relationships between Diseases of Deer and Those of Other Animals

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Abstract

Epidemiological concepts are first discussed: these include the determinants of infection, such as agent, host and environmental factors, patterns of disease occurrence, and sources and transmission of infection. Relationships between diseases of deer and of other animals are then considered in 2 main situations: in the wild and on farms.

Wild deer populations tend to be in equilibrium with the environment and diseases are a natural means of population control. Examples of diseases affecting both wild deer and other wild animals are given and their epidemiology discussed. The introduction by man of new species of animals and new diseases can alter the *status quo*. When wild deer are brought onto farms they are subjected to stress and are potentially more susceptible to disease. In addition, farmed deer are often kept in contact with other domestic animals, thereby being exposed to new infectious agents which can result in the emergence of new disease problems.

Keywords: deer, epidemiology, disease, zoonoses

Introduction

Rather than presenting an exhaustive catalogue of aetiological agents or clinical diseases known to affect both deer and other animals, we have approached the subject by discussing the epidemiological principles involved in the interchange of diseases between deer and other animals, giving examples. These epidemiological factors include the determinants of infection and disease, patterns of disease occurrence, and transmission of infection. Of necessity this paper is very general because of the numerous species of deer, their wide distribution, and the variety of ecosystems of which they are part. However, the 2 main categories of deer, wild and farmed, are considered separately in a more detailed discussion of some important disease interactions.

Infection and Disease

For infection to take place there must be a susceptible host, 1 or more suitable infectious agents and a conducive environment thus:

susceptible host + infective agent + conducive environment = INFECTION

However, not all infections cause clinical disease; in deer or any other animal clinical disease syndrome results from a complex interaction between the 3 factors outlined below:

Host factors
sex
age
stress
nutritional status
genetic resistance
acquired resistance
concurrent disease

Agent factors
virulence
pathogenicity
host specificity
infective dose
ability to multiply
resistance to environmental insults

Environmental factors
climate
food availability
population size and density
seasonal factors
ecological relationships

An individual animal is not of great importance epidemiologically; thus we must study the patterns and frequency of occurrence of infections and the severity of diseases in groups or populations of animals to gauge the true significance of any disease.

Disease occurrence

The interaction of the 3 factors (host, agent, and environment) determines what proportion of a population of animals will be affected by a certain disease and the pattern of disease occurrence, i.e. whether endemic, sporadic, or epidemic:

- An *endemic disease* is one normally always present in a particular group of animals at a relatively steady level of occurrence, e.g. lameness due to

Brucella suis type 4 infection in many reindeer and caribou herds (Dieterich 1980).

• *Sporadic diseases* are those which occur rarely and at irregular intervals. These may be due to occasional clinical cases of what normally are inapparent infections such as ataxia in wapiti caused by *Elaphostrongylus cervi* (Watson 1981). Alternatively, a sporadic disease may be due to occasional interspecies transmission, and many of the disease interactions with other animals fall into this category: 2 examples are sylvatic plague in deer caused by *Yersinia pestis*, and a case of rabies in a deer.

• A *disease epidemic* is defined as the occurrence of a disease clearly in excess of its normal frequency. Epidemics either have a common source or are propagated by direct or indirect transmission within a group of animals. Most of the serious disease interactions are propagated epidemics, e.g. the outbreak of foot and mouth disease in white-tailed deer in North America in the 1920s (McDiarmid 1975), and epizootic haemorrhagic disease outbreaks in white-tailed deer (Hoff and Trainer 1978).

It must be noted that a disease which is endemic in 1 species or population of deer may be sporadic or epidemic in another, and this depends largely on the complex interaction of host, agent, and environmental factors. Alterations in these determinants, such as changes in population size or density, food availability, or the introduction of new infectious agents or species of animal into the ecosystem, can affect these interactions.

Transmission of infection

Infection can be transmitted by a number of means including: *(i)* direct contact between animals; *(ii)* indirect contact due to contamination of the environment (food, water, air, and inanimate objects) by secretions, excretions, aerosols, or droplet nuclei; *(iii)* invertebrate vectors such as ticks, lice, fleas, mosquitoes, etc. The source of infection is, directly or indirectly, another infected animal. Most infective agents are not strictly host specific, but not all hosts fulfil the same epidemiological role, i.e. for any given infective agent there are usually some animals that act as natural reservoir hosts, some as inefficient or short-term carriers, and others as accidental or dead-end hosts.

A *reservoir host* is one that is capable of perpetuating the infection in its ecosystem and this may include intermediate hosts and arthropod vectors. Often there is more than 1 reservoir host for a particular infectious agent in different ecosystems, e.g. bats, foxes, skunks, and wolves can all act as reservoir hosts for rabies in different countries. Reservoir hosts, sometimes called maintenance hosts, are often the species least clinically affected by a particular agent, yet they shed or provide a source of large numbers of infective organisms. In contrast an accidental host species is often severely affected. In this respect bluetongue infection is usually inapparent in adult cattle, which are regarded as possible reservoir host, but can cause severe disease and high morbidity in white-tailed deer and sheep, which are regarded as short-term or accidental hosts (Jones *et al* 1981).

Short-term carriers may be able to propagate an epidemic when conditions are satisfactory, such as when the population density is high. For example, cattle act as short-term carriers for *Leptospira pomona* for which the pig is regarded as the reservoir host in New Zealand (Blackmore 1981). Recently, a propagating epidemic of *L. pomona* occurred in a group of 70 farmed red deer calves in the North Island of N.Z. (Fairley *et al* 1984), but it is considered likely that deer act in a similar way to cattle and serve as relatively short-term carriers of *L. pomona* and that epidemics of this type will die out when most animals in the group have become infected. The duration of the carrier state in deer is unlikely to be sufficiently long for infection to become endemic in farm deer populations.

Under extensive range conditions or in the wild state deer may be much less efficient at propagating an outbreak, and may then act as *dead-end hosts*. Such dead-end hosts are usually unimportant epidemiologically, either because the animals die and the infections die with them, or they fail to shed sufficient organisms to transmit infection to other animals. It has been suggested that in Scotland this is the case with deer infected with louping-ill virus; the deer do not appear to produce a high enough virus titre to infect the tick vector *Ixodes ricinus* and are therefore dead-end hosts, while sheep appear to be the reservoir host (Reid *et al* 1982). It must be noted that the environment can also act as a reservoir of infection, as in the case of anthrax and clostridial organisms and for parasites with direct life cycles.

In their natural state, deer tend to be in equilibrium with their environment and serious epidemics usually occur only when there has been a change to their ecosystem, such as the introduction of new animals, a new disease, a significant rise in population size or density, a change in food availability, or abnormal climatic conditions. Farmed deer, on the other hand, are kept under somewhat unnatural conditions where their environment and their interactions with other animals are largely controlled by man. These 2 extreme situations of wild and farmed deer will therefore be considered separately.

Wild Deer

Deer in the wild are usually regarded as being relatively free from clinical disease, although they can be subclinically infected with a variety of lice, ticks, dipterid larvae, nematodes, cestodes, trematodes, protozoans, bacteria, and viruses. Many of these infectious agents are not host specific and there are natural disease relationships between deer and other animals sharing their range; for example, the reindeer, caribou, moose, black-tailed deer, wild sheep, and goats of Alaska can all act as intermediate hosts for the encysted stages of *Echinococcus* and some of the taeniid tapeworms, the final hosts of which are foxes, wolves, coyotes, and dogs. Deer are also reservoir hosts of *Fasciola magna*, while bison are regarded as accidental or dead-end hosts (Barrett and Dau 1981). Bison, muskox, and reindeer all carry small lungworm burdens (*Dictyocaulus viviparus*), strains of which may be transmitted between these species. Some of the gastrointestinal nematodes may also be shared by wild deer, cattle, sheep, and goats.

Some infections of deer which do produce clinical disease are shared with other species of animals. Necrobacillosis caused by *Fusobacterium necrophorum* commonly affects free-living deer, sheep, and cattle. Foot abscess is the most common manifestation of necrobacillosis, but it can cause abscessing of any wound and often spreads to internal organs (Morton 1981). A common cause of chronic arthritis in reindeer is *Brucella suis* type 4. This organism, which also affects the reproductive performance of reindeer, infects other species of wildlife, especially predators (Morton 1985). Of the vector-borne diseases, anaplasmosis and Q-fever are usually asymptomatic, while bluetongue and epizootic haemorrhagic disease (EHD) can cause severe clinical disease (Hoff and Trainer 1978).

The above are examples of infections transmitted between free-living deer and other wildlife. In addition, because of man's continual encroachment into wilderness areas deer in the wild are often exposed to diseases of domestic animals and vice versa, especially where they are grazing on shared ranges or forest margins. This is apparent from a serological survey of deer in the central North Island of N.Z. where Daniel (1966) demonstrated that approximately 1% had positive titres to *Brucella abortus* and *Leptospira pomona*. The seropositive animals had all been shot very close to the forest edges where adjoining farms had experienced outbreaks of brucellosis and leptospirosis in the cattle herds. Domestic livestock are regarded as the reservoirs of *B. abortus* and *L. pomona* in N.Z. The deer which had seroconverted in that area could be regarded as accidental hosts, because these infections had not become endemic in that deer population. However, it has been shown that under conditions of high population density deer – deer and deer – cattle transmission of *Brucella abortus* can occur, and brucellosis is highly prevalent in some elk herds in western Wyoming (Thorne *et al* 1979).

Wild deer are also susceptible to bovine tuberculosis (Tb), caused by *Mycobacterium bovis*. In parts of N.Z. populations of possums (*Trichosurus vulpecula*) have acquired Tb from feeding on pasture contaminated by infected domestic cattle (Julian 1981); the disease has become endemic in possums in these areas and has subsequently been transmitted back to cattle, causing breakdowns in the cattle Tb eradication scheme. A similar situation is seen in the U.K. where tuberculous badgers (*Meles meles*) have reinfected cattle (Muirhead *et al* 1974; Little *et al* 1982). Recently a high proportion of deer shot or live-captured in these areas in N.Z. have had Tb, presumably acquired from infected possums (Fig. 1). So far there is no evidence that infected wild deer have transmitted Tb to domestic cattle, although there is a risk of this occurring. Areas with the highest incidence of tuberculous possums also have the highest incidence of tuberculous deer and cattle.

Thus, in the wild or under range conditions deer are a potential source of infection for domestic livestock, if they are sharing the same environment. However, it should be remembered that infectious agents may be carried some distance by wind, water, or mechanically from their source. Examples of these are vector-borne diseases, agents such as foot and mouth disease (FMD) virus, leptospirosis, and the resistant spores of anthrax and clostridial bacteria.

Farmed Deer

For present purposes we define farmed deer as those kept under similar conditions to domestic livestock such as cattle and sheep. In N.Z. farmed deer are kept behind high fences on land ranging from extensive high-country tussock to intensively stocked lowland pasture, and usually fairly close to other domestic animals. Disease interactions between farmed deer and other animals fall into 4 broad categories:

(i) diseases brought onto the farm with deer captured from the wild;
(ii) diseases of farmed deer affecting other domestic livestock;
(iii) diseases of livestock affecting farmed deer;
(iv) diseases of wildlife present on farms affecting farmed deer.

The list of relevant conditions is not exhaustive, but a few examples are given for each category.

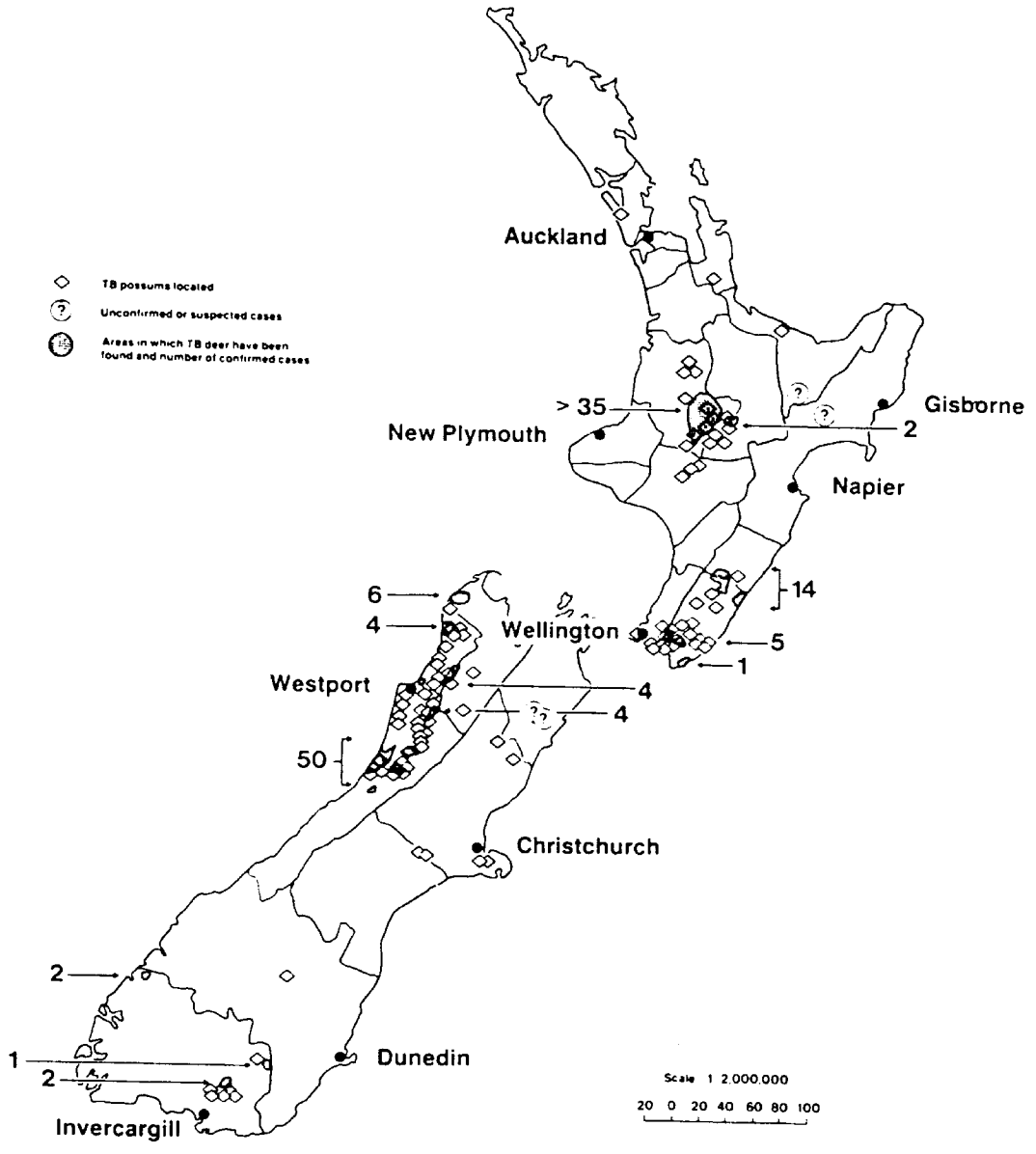

Fig. 1: Areas where Tb-infected possums and wild deer have been identified. (K. Corrin *pers. comm.*)

• *Diseases brought onto the farm with wild deer:* Foundation stock for most deer farms in N.Z. and Australia have been live-captured wild deer descended from animals introduced over 50 years ago from Europe, North America, and Asia. Fortunately it appears that when introduced they had no serious pathogens and only low burdens of parasites. However, as discussed in the previous section, some of the wild deer in this country have subsequently contracted Tb and in the last 10 years over 100 wild deer have been found with this disease (K. C. Corrin *pers. comm.*). Infected deer brought onto farms have caused outbreaks of Tb in farmed deer and they pose a threat to other domestic animals.

In North America, where there are viral diseases such as EHD and bluetongue, protozoal conditions such as anaplasmosis and trypanosomiasis, and parasitic conditions such as parelaphostrongylosis, the live capture and domestication of wild deer may introduce new disease problems into traditional livestock areas.

• *Diseases of farmed deer affecting other domestic livestock:* In N.Z. deer are usually farmed close to traditional livestock. As well sheep and cattle are often brought onto deer farms for pasture management thereby exposing them to deer infections such as lungworm and Tb. So far, however, there have been no confirmed cases of domestic livestock or man

contracting Tb from farmed deer, nor have any serious problems with lungworm in cattle been traced back to deer.

If an exotic disease such as FMD were introduced into N.Z., farmed deer would have to be regarded as possible reservoirs of infection, although some workers (Forman and Gibbs 1974) suggest that red and fallow deer are not good disseminators of FMD virus and are therefore unlikely to be significant reservoirs of infection.

• *Livestock diseases affecting farmed deer:* Farmed deer are likely to come into direct contact with livestock or graze on pasture previously grazed by sheep and cattle and are therefore exposed to infectious agents associated with these animals. Because of their recent "domestication" farmed deer are subjected to many stresses including capture, transport, handling, social stress at high stock densities, climatic stress due to lack of shelter, and nutritional stress due to poor quality or inadequate quantity of feed. These stresses make farmed deer more susceptible to diseases such as lungworm and malignant catarrhal fever (MCF). The strain of *D. viviparus* which usually affects deer may well be deer-adapted, but cattle strains can cause patent infections in deer (Corrigall *et al* 1982). Sheep may be silent hosts for a virus which is responsible for causing MCF in deer, analogous to the wildebeest-associated herpes virus that causes bovine malignant catarrh in cattle in East Africa. However, until the MCF agent is isolated from sheep or deer this cannot be proven.

Farmed deer may also be exposed to *Leptospira* sp., *Brucella abortus, Mycobacterium paratuberculosis, Campylobacter* sp., *Toxoplasma, Trichophyton* sp., *Fusobacteria* sp., *Corynebacteria* sp., *Dermatophilus congolensis* etc. which are commonly carried by domestic livestock. It remains to be seen how important these agents will become in farmed deer. Of the above *L. hardjo* is endemic in cattle populations while *L. pomona* is maintained by pig populations and causes sporadic outbreaks of disease in cattle in N.Z. and both serovars have been isolated from deer in the North Island (D. K. Blackmore *pers. comm.*). *L. hardjo* appears to produce no clinical disease in deer, while *L. pomona* can cause death in young deer. *Fusobacteria* and *Corynebacteria* can cause severe foot abscesses in deer, and *Dermatophilus* infections of velvet can considerably reduce its quality.

• *Diseases of wildlife transmitted to farmed deer:* Another disease of deer very much related to stress is yersiniosis caused by *Yersinia pseudotuberculosis*. This occurs predominantly in young deer in winter and takes the form of an acute gastroenteritis causing a watery smelly diarrhoea usually resulting in death in 24–48 hours if untreated. Reservoirs of infection include rabbits, hares, rodents, feral cats, and some birds (Mair 1973; Obwolo 1976; Mackintosh and Henderson 1984). It seems likely that farmed deer are more exposed to *Yersinia* than are wild deer and this may be due to the following factors: population density of lagomorphs is high on farm land; deer graze closely to the ground on improved pasture; rodent populations in N.Z. are greatest near human habitation, so hay and concentrates fed to deer may be contaminated by rodents.

Farmed deer may also contract bovine Tb from infected possums and avian Tb from infected birds. In North America and Europe, diseases such as tularaemia, leptospirosis, and rabies may be transmitted to farmed deer from wildlife, and caribou may act as reservoirs of warbles for reindeer herds.

Deer and Zoonoses

Man, like other animals, is susceptible to many of the diseases which deer may carry, e.g. anthrax, tuberculosis, leptospirosis, brucellosis, yersiniosis, tularaemia, rabies, and Q-fever (Choquette 1970; Emmons *et al* 1976; Enright *et al* 1969; Mair 1973). Hunters, forestry workers, and trampers may be exposed to wild deer carrying some of these infections. In the livestock industry, farmers, transport operators, veterinarians, meat workers, and meat inspectors have a relatively high occupational risk from the diseases of sheep and cattle, such as brucellosis, leptospirosis, Tb, and Q-fever (Blackmore 1983). Now that deer are farmed intensively and slaughtered under similar conditions to sheep and cattle, these workers may be exposed to similar occupational risks from the diseases of deer.

Conclusion

With man's activities continually encroaching on the wild deer environment, and the ever present possibility of the introduction of new disease agents and new species of animals into these areas we may see the emergence of new disease problems in wild deer in future.

Capture of wild deer and their introduction to farms has inherent dangers and a lesson can be learnt from the New Zealand experience with Tb. It is important that all live-captured wild deer be kept in isolation before being released onto farms, so that they can be tested for any potentially significant disease.

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