RABIES IN THE SERENGETI: THE ROLE OF DOMESTIC DOGS AND WILDLIFE IN MAINTENANCE OF DISEASE

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Introduction

Although dogs account for over 90 percent of human rabies cases (WHO 1992), relatively little is known about the epidemiology of canine rabies. Much of our understanding of rabies epidemiology comes from empirical and theoretical studies, not of dogs, but of wildlife populations in Europe and North America. Here, in any given geographic area, antigenically and genetically distinct strains of rabies (virus "biotypes") are maintained by a single principal host species (Carey 1985; Blancou 1988; Smith 1989; Rupprecht et al 1991; Wandeler 1991; Blancou et al. 1991).

The increasing incidence of dog and wildlife rabies in Africa (King 1993) is causing concern, not only for public health but also for the conservation of some endangered canids (Macdonald 1993). In the Serengeti, recent rabies outbreaks have seriously affected the small population of African wild dogs (*Lycaon pictus*) (Gascoyne et al. 1993; Alexander et al 1993) and control of the disease has become a component of their conservation strategy. However, in order to control rabies effectively, we need to identify which animals are reservoirs and by what mechanisms the disease is maintained in reservoir populations.

Reservoir hosts in the Serengeti

In the first part of this study we evaluate the roles of domestic dogs and wildlife as reservoirs of rabies in the Serengeti, using five criteria: 1) reservoir host populations should show evidence of persistent infection, 2) cases should occur in the reservoir host in the absence of cases in other species, but the converse should not occur, 3) outbreaks in other species should follow cases in the reservoir host population, 4) control of rabies in the

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reservoir host should result in elimination of disease in all other species, and 5) the virus isolate that is characteristic of the reservoir species should be found in all other species.

1. Evidence for maintenance in dog populations. Historical records show that from 1958 to 1977, rabies was apparently absent from the Serengeti (Rweyemamu et al. 1973; Magembe 1985). In Serengeti District (SD) (an agropastoralist area to the west of the Serengeti National Park), dog cases, or human cases derived from dogs, were reported every year for which records were available (1977-1984; 1986-1995). In contrast, rabies was reported only sporadically in pastoralist areas to the east of the park (Loliondo Game Control Area, LGCA and Ngorongoro Conservation Area, NCA).

Evidence for maintenance in wildlife populations. Since the early 1980s, rabies has only rarely been recorded in Serengeti's wildlife, despite intensive monitoring of carnivore populations. The disease has been confirmed in two wild carnivore species - African wild dogs in 1990 (Gascoyne et al. 1993) and bat-eared foxes (Otocyon megalotis) in 1987 and 1988 (Maas 1993), and in 1994 and 1995. The African wild dog population is undoubtedly too small and dispersed to maintain rabies. While recurrent cases have occurred in bat-eared foxes, the short duration of epidemics (5 and 7 weeks in 1987 and 1988 respectively) (Maas 1993) together with an absence of reported cases between 1988 and 1994 suggests that this species is also unlikely to be a reservoir host in Serengeti.

2. Independent maintenance of infection. Since rabies is maintained in domestic dog populations of SD but occurs only sporadically in wildlife, rabies in dogs can occur in the absence of cases in wildlife, but wildlife rabies does not occur in the absence of dog rabies.

3. Temporal sequence of epidemics. With so few rabies cases in Serengeti, we draw on data from Zimbabwe to investigate the temporal relationship between dog and wildlife cases. Case incidence data from 1950-1991 (Foggin 1988; Bingham 1993) together with results of a cross-correlation analysis indicate that cases in dogs preceded outbreaks in jackals with a lag of one year (r=0.523, p<0.05). During this period, therefore, rabies epidemics in jackals appear to have been driven by infection in dogs and not vice versa.

4. Rabies control. In the late 1950s, dog rabies control measures (dog vaccination, movement restriction and culling) resulted in the apparent elimination of disease from the Serengeti District between 1958 and 1977 (Rweyemamu et al. 1973; Magembe 1985).

5. Virus isolates. Three virus isolates from Serengeti (a domestic dog, an African wild dog and a cow) were found to be antigenically and genetically indistinguishable with characteristics consistent with southern African canid-associated virus. These results support findings of other studies, which show that three African wild dog isolates from the Masai Mara National Reserve, Kenya (part of the Serengeti ecosystem) were identical both to the Serengeti African wild dog isolate and to four domestic dog isolates from the Masai Mara (Alexander et al. 1994).
Drawing together these data, we argue that domestic dogs, not wildlife, are the likely reservoir of rabies in the Serengeti. It is clear, however, that surveillance measures need to be improved before definite conclusions can be reached. In particular, the observation that bat-eared foxes can maintain rabies in parts of South Africa (Thomson and Meredith 1993) emphasises the need for further investigation of the role of this species in the Serengeti.

**Mechanisms of disease maintenance**

In the second part of the study, we explore possible mechanisms of maintenance in dog populations in Serengeti. Although rabies is among the best studied microparasite infections, explanations for disease maintenance remain problematic. Several mechanisms have been proposed to account for persistence, for example, reintroduction of disease (Voigt et al. 1985; Artois et al. 1991), inapparent reservoirs (Carey 1985), prolonged or variable incubation periods (Bacon 1985; Aubert et al. 1991) and carrier animals (Crandall 1991). Although many of these can be invoked for dog rabies, few studies have evaluated the quantitative significance of different possible mechanisms of disease maintenance in dog populations.

In theory, microparasite infections are more likely to be maintained at higher host densities. In this study, we provide evidence for a threshold density for rabies persistence in Serengeti, with infection appearing to be maintained in populations exceeding 5 dogs/km² (SD), but not in pastoralist populations (LGCA and NCA) of less than 1 dog/km². However, this threshold is several orders of magnitude lower than has been suggested from other studies. For example, dog rabies in Guayquil, Ecuador occurred continuously where dog densities exceeded 600 dogs/km², but only sporadically in lower density areas (Beran and Frith 1988). These observations led us to investigate the role of atypical rabies in possible mechanisms of disease maintenance in the Serengeti.

**Rabies Serology**

Results of serological analyses using a liquid-phase blocking ELISA (Esterhuysen et al. 1995) have shown that a proportion of healthy, unvaccinated Serengeti dogs have detectable serum levels of rabies antibody. Several lines of evidence support the view that rabies seropositivity detected by this test reflects genuine, and recent exposure to rabies virus.

a) Antibody titres in Serengeti seropositives were higher than in any individual from rabies-free islands (89 dogs from Mauritius and 99 dogs from UK).

b) Seropositive dogs were significantly more likely to be found in villages where rabies had been confirmed or reported than in villages where it had not (odds ratios with 95 percent confidence limits as follows: for villages where rabies confirmed OR=3.96 (1.88-8.24), p<0.001, n=900; for villages where rabies reported OR=5.02 (2.41-10.66), p<0.001, n=900).
c) Individual case history data. i) Seroconversion was detected in a dog which had been bitten by a suspect rabid dog 2-4 weeks earlier and which remained healthy for at least four months; ii) Four of 12 dogs bitten by a suspect rabid dog one to two weeks earlier were seropositive. Two dogs remained healthy and seropositive for at least two years, with fluctuating antibody levels. One of the 8 bitten dogs that was seronegative died of confirmed rabies and three died of suspect rabies one to two months later.

In a follow-up study, only one of 32 seropositives subsequently died of suspect rabies and this was an animal showing neurological signs at the time of sample collection. Nine seropositives remained alive and healthy for at least two years. Case histories indicated that none of the seropositive dogs had recovered from neurological disease. Rabies virus was not isolated from saliva samples collected from six seropositive dogs. Of the atypical rabies infections that have been described (recovery, carrier states, latency, aborted rabies, reactivation of infection; Fekadu 1991), an interpretation of "aborted" rabies is the most consistent with our observations.

**Mathematical models**

While we cannot explain rabies seropositivity in terms of rabies pathogenesis or immunity, we can use mathematical models to explore three different interpretations of seropositivity - long incubators, carriers and immune animals. Each has been invoked as a possible mechanism for rabies maintenance (for example Bingham *et al.* 1994; Chaparro and Esterhuysen 1993 for long incubators; Andral 1964; Fekadu 1991 for carrier animals; Coyne *et al.* 1989 for immune animals). In this study, infection parameters derived from Foggin (1988) and dog demographic data from Serengeti were incorporated into a deterministic model of classic dog rabies, which generated regular cycles of disease incidence. The model was relatively insensitive to the inclusion of 5 percent of dogs as long incubators (with an incubation period of 20 weeks rather than 4 weeks), or 10 percent dogs as immune animals. In contrast inclusion of only 0.1 percent carrier dogs had a marked impact on the dynamics, stabilising fluctuations in disease incidence and lowering the probability of extinction in troughs between epidemics. While it was beyond the scope of this study to carry out extensive surveys for carrier animals, studies in Ethiopia and Nigeria have demonstrated salivary virus excretion in 0.46 percent (5/1083) and 0.27 percent (4/1500) of healthy dogs respectively (Fekadu 1972; Aghomo *et al.* 1989).

**Summary**

Drawing together data from recent field studies and historical records, we present evidence to support the view that domestic dogs are the likely reservoir of rabies in the Serengeti. Rabies is maintained in dog populations of Serengeti District where densities exceed 5 dogs/km² but not in pastoralist dog populations of less than 1 dog/km² suggesting not only that there is a threshold density for rabies maintenance, but also that disease control measures should be directed towards higher-density dog populations. Rabies seropositivity occurs in a proportion of healthy, unvaccinated Serengeti dogs. While an interpretation of rabies seropositivity in terms of rabies pathogenesis has not been possible, mathematical
models show that maintenance of rabies in Serengeti dog populations is more likely if seropositives are infectious carriers, rather than long incubators or immune animals.

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