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Rabies in Zimbabwe: reservoir dogs and the implications for disease control

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Using detailed field study observations of the side-striped jackal (*Canis adustus*) and a simple stochastic model of the transmission dynamics of the virus and host demography, we discuss the epidemiology of rabies virus infection in the jackal population of Zimbabwe. Of the two jackal species in Zimbabwe, the other being the black-backed jackal (*Canis mesomelas*), the bulk of notified rabies cases are in side-striped jackals. Specifically, we show that the side-striped jackal population itself does not seem able to support rabies infection endemically, i.e. without frequent reintroduction from outside sources of infection. We argue that this is probably because the overall average jackal population density is too low to maintain the chain of infection. This study suggests that the disease is regularly introduced to jackals by rabid dogs from populations associated with human settlements. Given the rapidly rising dog population in Zimbabwe, estimates are derived of the future incidence of jackal rabies based on different dog-vaccination scenarios.

Keywords: rabies; jackals; mathematical model

1. INTRODUCTION

Rabies is a communicable disease capable of infecting all mammals. The viral pathogen concentrates in cerebral and nervous tissues, and upon succumbing to infection the host often exhibits marked behavioural changes. In humans, the clinical symptoms of rabies, and the virtual certainty of death following the emergence of these symptoms, have made this disease one of the most feared in the world.

Historically, in Europe, domestic dogs have been the main vector of the disease to man, although in recent years the red fox (*Vulpes vulpes*) has acted as the primary disease reservoir (Blancou *et al.* 1991). The population biology of foxes and the dynamics of fox rabies in Europe have been topics of extensive study, and ultimately this has led to effective disease eradication and containment strategies which are generally based on oral vaccination of foxes (Anderson *et al.* 1981; Smith & Harris 1991; White *et al.* 1995). Risk of exposure to humans has been considerably reduced by the availability of effective vaccines for domesticated dogs and cats.

Rabies is also present in the diverse mammalian populations of Africa (Swanepoel *et al.* 1993), which is home to some of the world's most endangered canid species (Gascoyne *et al.* 1993*a*; Sillero-Zubiri *et al.* 1996). However, at present, the complex epidemiology of rabies in Africa is not so well-understood as that in Europe. Serious outbreaks of rabies infection could jeopardize conservation programmes aimed at preserving species which are currently on the threshold of extinction, so it is important that a better understanding is reached of the transmission of rabies in this context (Gascoyne *et al.* 1993*b*; Macdonald 1993). Furthermore, a greater prevalence of rabies infection would have an impact on the agricultural sector of many African countries, as stock animals are currently lost each year owing to rabies infection acquired from itinerant rabid canids (Bingham 1995*a*).

Using a combination of detailed field-observation data and simple mathematical models this paper analyses the particular form of rabies epidemics in Zimbabwe. The large population of domestic dogs appears to act as the primary self-sustaining reservoir of the rabies virus. It is possible that other species of animal can then acquire infection through interaction with the dog population. Typically, this results in temporally sporadic and spatially localized outbreaks of rabies in other species, commonly jackals in some parts of southern Africa. Jackals, which our calculations suggest are unable to support rabies endemically (i.e. without frequent reintroduction from outside sources of infection), might then in their turn infect other wild and domestic animals, with welfare, conservation and economic consequences before the disease dies out among them. Using the mathematical model, it is possible to explore the potential variation that uncertainty in some of the ecological parameters can induce.

Our findings support the conclusion of recent studies on rabies infection in the dog populations of the Serengeti region of Tanzania (Cleaveland 1995; Cleaveland & Dye 1995) where it was shown that dogs are the most likely reservoir of the disease. Furthermore, our model predicts an increasing dog population in Zimbabwe will lead to an increasing incidence of jackal rabies, and we outline the effect of different dog-vaccination strategies on the incidence of the disease in jackals.

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2. FIELD STUDY OBSERVATIONS

Ecological parameters (*per capita* birth rate, *per capita* death rate, carrying capacity and contact rate) were derived from a year-long study of side-striped jackals (*Canis adustus*) on 150 km^2 of commercial farmland centred 40 km south-west of Harare, Zimbabwe. Unless otherwise stated, all further discussion of jackals in this paper pertains to the side-striped jackal. The site consists of *Brachystegia*-dominated savannah woodland ('miombo'), and low-lying, seasonally flooded, natural grassland. Both habitat types occupy approximately 40% of the total area and are used by grazing animals. Arable land, comprising planted grass leys, ploughed and fallow fields, and tobacco and maize crops, occupies the remaining 20%. The study site is typical of commercial farmland around Harare.

The main ecological study techniques were trapping and radio-tracking. Jackals were trapped using humanely modified leg-hold traps (Victor Soft-catch size $1\frac{1}{2}$, Woodstream Corporation Inc., Lititz, Pennsylvania, USA), baited with putrid meat. Once caught, jackals were fitted with radio-collars operating at a frequency of 173 MHz (Biotrack, Wareham, Dorset, UK). A total of ten males and nine females were trapped and intensively radio-tracked from a four-wheel drive vehicle using a Mariner Radar M57 receiver, and a hand-held directional four-element Yagi aerial. A radio-fix was taken every 10 min and the jackals' positions were located on a map of the study site overlaid by a grid. Each jackal was tracked for 8 h a night for three nights, and all night (12 h) for one night, after which we started a four-night tracking session on the next jackal. A total of four nights was sufficient to identify at least 80% of the area currently in use, by the minimum convex polygon method (Stickel 1954). All-night tracking was done to get complete pictures of total nightly distance travelled. On every fix, data were collected on the proximity of neighbouring radio-collared jackals to the focal jackal. In total, we radio-tracked jackals for over 1700 h and took over 10 000 radio-fixes (R. P. D. Atkinson and D. W. Macdonald, unpublished data).

To obtain estimates of the demography of the study population, records of all jackals seen were kept while radio-tracking, whether they were radio-collared or not. The age and group size of the jackals, and whether they were seen in or out of home ranges of known jackals, were recorded for this purpose.

(a) *Birth rate*

The estimated birth rate is 5.4 pups per pair, born between August and January (Skinner & Smithers 1990) with most appearing between August and October (J. Bingham, personal observation). Females can breed as yearlings and onwards (J. Bingham, personal observation).

(b) *Life expectancy*

Pup mortality is likely to be high, as free-ranging pups were very rarely seen (once in three years), and never caught in traps. It is likely that only two pups per litter survive past six months. By estimating age by tooth-wear of captured and dead jackals, we assume that territory-holding adults rarely live beyond six years, and that the average lifespan of such animals is three to four years. The main cause of death on the study site was accidental snaring in traps where edible species were the intended target. For those animals that survive beyond six months, but which fail to secure a territory, we estimate life expectancy to be 1–2 years.

(c) *Home range size and movements*

The territories of males and females do not differ in size, nor is there a seasonal difference in territory size. We estimated territory size using 200 m × 200 m grid cells. Over a year the mean territory area used by jackals was 1224 ± 92.3 hectares (range 1056–1672 ha.). Over a period of four nights each jackal covered a mean of 33% (range 23–41%) of the mean yearly territory. Over one night jackals covered a mean of 14% of the mean yearly and 48% of the mean four-night territory.

Over a 12-hour-night the average minimum distance moved by each jackal was 10.3 ± 2.4 km (range 3.4–31.7 km). This is the sum of straight line distances between positions of the jackal at successive fixes. The actual distance travelled will be somewhat greater than this figure suggests. Jackals travel approximately 1.4 ± 0.4 km h^{-1}, but can run at approximately 20 km h^{-1} (R. P. D. Atkinson, personal observation).

(d) *Population density*

The jackal population size and the structure of the study area were estimated using intensive trapping, radio-tracking and night-time sightings. The population of resident, territory-holding adults was estimated to be 20–30 100 km^{-2}. The total jackal population of the study area was estimated at 60–90 jackals 100 km^{-2} during the breeding season, giving a peak density of between 80 and 120 jackals 100 km^{-2} when helpers and itinerants are included.

3. A MODEL FOR JACKAL RABIES

Kingdon (1997) suggests the side-striped jackals occupy a similar ecological niche to red foxes in Europe, an observation supported by our data and Atkinson's (1996), and like foxes they are susceptible to infection with the rabies virus. Figure 1 shows the annual incidence of reported cases of jackal rabies in Zimbabwe (Foggin 1988; Bingham 1992; Bingham 1995).

Jackals live in family groups that exist within territorial boundaries and juveniles disperse annually to establish new territories or take over the gaps left in the social hierarchy by adult mortality. These are all features shared with European foxes (Macdonald 1980). The most striking difference, however, appears to be in the overall population density of jackals; European foxes have been observed at densities between 0.1 and 5 km^{-2}, whereas jackals typically appear to exist at a density of around 1 km^{-2} (J. Bingham, personal observation).

Given these ecological similarities we consider it appropriate to employ a modification of a model which has been used to account for rabies infection in foxes. The model was introduced by Anderson *et al.* (1981) and can account for the 3–5-year cycles seen in the incidence of rabies infection in European foxes. Furthermore, it was used to assess the impact of a variety of different intervention strategies

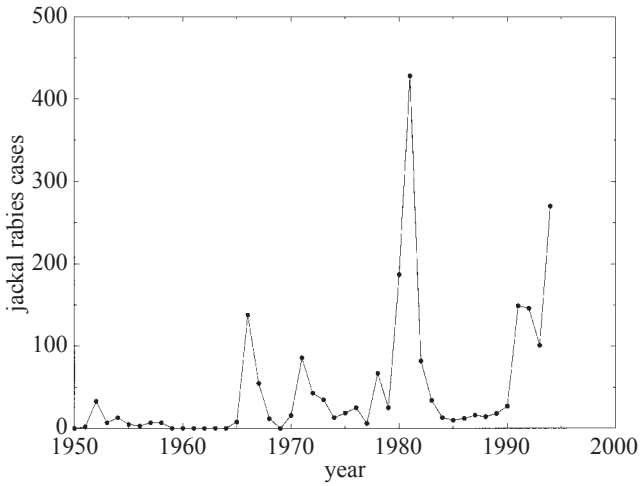

Figure 1. Annual incidence of jackal rabies in Zimbabwe, 1950–1994.

against the disease, such as culling and vaccination. We adopt this model, with the appropriate modifications, to discuss the population biology of the rabies virus in the jackal population of Zimbabwe. A more complete discussion of a variety of different approaches to the modelling of rabies in wildlife populations is given by Bacon (1985). Of particular relevance is the work of Ball (1985) who studied fox rabies epidemics with a spatially explicit stochastic model; this is an approach that has potential for significant future development, although in this paper we use a simple stochastic-coupled differential equation model.

The jackal population is divided into three classes: Susceptibles (S), those not exposed to rabies infection; Exposed (E), those infected individuals who are incubating the virus and are not capable of transmitting the infection; Infectious (I), those individuals who have moved from the Exposed class and are now capable of transmitting the infection. For simplicity, and given that we do not have the appropriate data, we do not consider the possibility that there can be asymptomatic carriers or jackals with any other more complex epidemiological status. The total number of jackals, N, at any given time is equal to $S + E + I$. We assume that there is a density-independent birth rate, a, and an intrinsic uninfected jackal death rate, b. Jackals also die because of density-dependent pressures which, in the absence of any fatal infectious disease, regulate the population density to some quasi-stationary state determined by habitat quality. Thus, the Susceptible jackal class obeys the following equation:

$$\mathrm{d}S/\mathrm{d}t = \alpha S - bS - \gamma SN - \beta SI. \quad (1)$$

The third term represents the density-dependent death rate and the fourth term represents the depletion of the Susceptible class as they mix with the Infectives and enter the Exposed class. Once in the Exposed class we assume that an individual remains there for a mean incubation period, σ^{-1}. This gives

$$\mathrm{d}E/\mathrm{d}t = \beta SI - bE - \gamma EN - \sigma E. \quad (2)$$

Once in the infective class we assume that the life expectancy of the animal is extremely short and given by α^{-1}. Thus, the population of the Infectious class is given by

$$\mathrm{d}I/\mathrm{d}t = \sigma E - bI - \gamma IN - \alpha I. \quad (3)$$

By summing these three equations the rate of change of the total jackal population, N, is given by

$$\mathrm{d}N/\mathrm{d}t = aS - bN - \gamma N^2 - \alpha I. \quad (4)$$

In the absence of rabies infection ($\alpha = 0$, $S = N$), equation (4) reduces to

$$\mathrm{d}N/\mathrm{d}t = rN(1 - N/K), \quad (5)$$

with the carrying capacity of the environment $K = r/\gamma$. Equation (5) is the well-known logistic equation for population growth and it is not unreasonable to assume that this describes jackal population density in the absence of infection.

(a) *Parameter estimation*

The model described in the previous paragraph is the simplest possible representation of the most important ecological factors in the jackal rabies system, and aims to describe the temporal evolution of the infection in the jackal population as a whole within the borders of Zimbabwe. As yet we cannot make statements about the spatial distribution of jackal rabies cases or the rates of spread of the infection into largely susceptible areas. These are important issues, but at present we do not have the data available to undertake a detailed analysis of the spatial dynamics of disease spread and persistence.

The terrain of Zimbabwe is diverse, but the land can be divided into three broad categories; wildlife–forest land, communal land and commercial farmland. The black-backed jackal (*C. mesomelas*) predominates in the wildlife–forest areas, particularly in the south-west and central areas of the country, whereas the side-striped jackal (*C. adustus*) is most commonly found on commercial farmland in the north and east (Foggin 1988). Very little is known about the difference between jackal behaviour in areas of differing land-use, and we assume that conclusions drawn from our field observations made in an area of commercial farmland are broadly applicable to jackals throughout the country. Specifically, this means that the parameters we estimate from the field observations we assume can be applied to jackals of both species across the country as a whole. Clearly, this simplification is one we must impose in the absence of more detailed field data. However, the only scientific study of black-backed and side-striped jackals living in sympatry suggests strongly that there are only small interspecific differences in group size, feeding ecology, territory size and ranging behaviour (A. Loveridge, personal communication).

Estimates for the birth rate, death rate and carrying capacity come from observations of the jackal population in the study area, as described in §2. The estimates for the viral incubation period (around 20 days) and the life expectancy of a rabid jackal (around 5 days) are taken from the results of rabies inoculation experiments on captive jackals (Foggin 1988; J. Bingham, personal observations). At a jackal density of around $1\,\mathrm{km}^{-2}$, β^{-1} is the average time between jackal contacts. From the radio-tracking data we find that β^{-1} is approximately equal to seven days, clearly a figure estimated from monitoring interactions between a focal radio-tracked jackal and

Table 1. *Parameters used in the epidemiological model of jackal rabies.*

per capita birth rate	a	1.0 year^{-1}
per capita death rate	b	0.4 year^{-1}
net population growth rate	$r=a-b$	0.6 year^{-1}
jackal carrying capacity	K	1.0 km^{-2}
average viral incubation period $^{-1}$	σ	18 year^{-1}
average life-expectancy of a rabid jackal^{-1}	α	73 year^{-1}
contact rate	β	52 km^2 year^{-1}

neighbouring radio-collared jackals. We also assume that such a contact rate is maintained when rabies is present in the population and that the virus is transmitted when an infectious jackal encounters a susceptible one. This suggests that our estimate of the contact rate is a maximum, and that in reality contacts occur somewhat less frequently than this. In the case of European foxes (Anderson *et al.* 1981), where the population is maintained at a higher density than the study-area jackals, estimates of β^{-1} are found to be approximately five days. The parameters we use for the model are shown in table 1.

(b) *Implementation of the model*

Given the correlation between land-use and jackal prevalence in Zimbabwe we estimate that the total population is of the order of 400 000 individuals. With this relatively small number we expect stochastic effects to be important, so we use a Monte-Carlo implementation of the model. This technique has often been used to model the spread of disease in small or severely fluctuating populations and it explicitly takes into account the fact that the jackal population is composed of N individual units (Olsen *et al.* 1988; Anderson & May 1991; Grenfell 1992). Also, we make a further modification in line with field observations relating to the seasonality of the birth rate. Jackal cubs are usually born between August and January, so we double the *per capita* birth rate but apply it for only half the year, thus preserving the overall average annual birth rate. The additional implication of this simple assumption is that for those months during which there is no cub birth there will be no density-dependent death either.

4. DYNAMICS OF RABIES IN JACKALS

Using the mathematical model and parameters derived from field observation we can investigate the dynamics of rabies infection in the jackal population. The first question we can ask is whether the jackal population is capable of supporting rabies virus infection endemically. Experience with the fox population in Europe (Anderson *et al.* 1981) suggests that there will be a minimum critical density of jackals needed to support the infection.

From our model, equations (1)–(4), it is possible to define the basic reproductive rate, R_0, i.e. the average number of secondary infections produced by an individual Infective jackal,

$$R_0 = \frac{\sigma\beta K}{(\sigma + a)(\alpha + a)}. \quad (6)$$

Because $\sigma \gg a$ and $\alpha \gg a$ this gives the approximation

$$R_0 \simeq \beta K/\alpha. \quad (7)$$

For the disease to be endemic we need $R_0 > 1$, or equivalently

$$K > \alpha/\beta. \quad (8)$$

Using the parameters from our field observations we calculate that the minimum density required to maintain infection is around 1.4 jackals km^{-2}. This suggests that the population density is too low to support endemic rabies infection and, in turn, implies that the jackals cannot act as a wildlife reservoir of rabies. It is probable that they are being subjected to continual, but sporadic, infection from some external source. When this occurs, spatially separated and temporally incoherent epidemics are triggered which, when aggregated over the country as a whole, could give the impression that the jackals are capable of maintaining rabies infection, a similar conclusion to that reached by Cumming (1982).

The observed jackal density of around 1 km^{-2} is quite close to the threshold for disease maintainance. Consequently, we expect that whenever rabies is introduced into a jackal community the infection can persist for some time, thus placing agricultural stock animals at risk of infection. Also, it is entirely feasible that in other circumstances jackals can exist at densities of up to 2 km^{-2}, at which they could maintain the disease and possibly initiate front-like rabies epidemics, as have been observed around Harare between 1979 and 1982 (Foggin 1988) and again between 1990 and 1995 (Bingham 1995*a*).

This initial result suggests that to observe the pattern of jackal rabies seen in figure 1, either the contact rate (determined by β^{-1}) among jackals is more frequent than we estimated from the field data or, alternatively, that frequent reintroduction of rabies into the jackal population (from a source external to, and independent of, the population) is required. Using the mathematical model, it is straightforward to investigate both possibilities.

(a) *Variation of the jackal contact rate*

In our model of rabies infection in jackals the most difficult parameter to estimate is, without doubt, the jackal contact rate β. Our estimate that β^{-1} is approximately equal to seven days was obtained from radio-tracking data. Here we perform several different numerical simulations using the model, assuming that contacts between jackals occur more frequently, to see if these have any effect on the persistence of rabies. We ran 40 simulations, each lasting 100 years, for each contact rate shown in figure 2. We calculated the percentage of years for which there were jackal rabies fatalities. There are two separate plots superimposed in this figure. The first includes a non-seasonal birth rate, whereas the second, more realistically, assumes that, as we discussed in § 3b, birth only takes places during six months of the year.

For the non-seasonal birth calculations there is a window of contact rates with β^{-1} between three days and four-and-a-half days which allows persistence of rabies. More frequent contact results in rabies spreading through the jackal population so fast that susceptibles cannot be produced quickly enough to maintain the epidemic. For

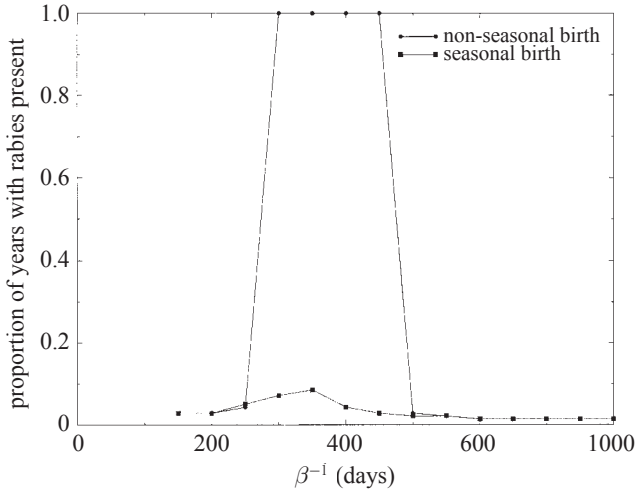

Figure 2. Plot showing the calculated percentage of years in which jackal rabies fatalities occur as a function of reciprocal contact rate β^{-1}. The solid circles are the calculation for a non-seasonal birth rate. The solid squares are the calculation for a seasonal birth rate. A host population of jackals approximately 400 000 in size was challenged with 20 Exposed and 20 Infective jackals. Each point is the mean of 40 simulations after the elimination of transients; the standard deviations are negligible.

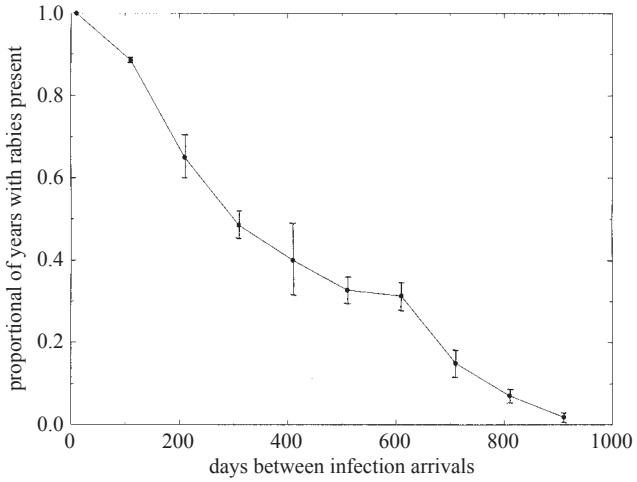

Figure 3. Plot showing the calculated proportion of years in which jackal rabies fatalities occur as a function of contact with an external rabies reservoir. The *x*-axis represents the mean time-interval (in days) between immigrations of infection into the jackal population. The same population parameters were used as in figure 3. The error bars represent one standard deviation.

low contact rates, jackals make insufficient contacts to ensure disease persistence.

The second, more realistic, calculation with seasonal births indicates that persistence of rabies is not possible at any time, although persistence is enhanced for contact rates, β^{-1} around three-and-a-half days.

The radio-tracking data suggest that jackal contacts are infrequent (at least every five days), and we conclude that it is unlikely that, given the jackal density observed in the study area, rabies can persist in such a jackal population without frequent reintroduction from an external source.

Fade-out of the disease in the seasonal-birth stochastic calculations appears to be connected to the relatively small size of the jackal population. Equivalent calculations in a deterministic framework, using an adaptive step-size Runge–Kutta routine to solve equations (1)–(4) (Press *et al.* 1994), suggest that persistence is possible for jackal contact rates more frequent than every four days in the seasonal-birth case. However, as in the stochastic calculation, rabies does not persist in the deterministic calculation for the ecological parameters set out in table 1.

Neither our field data, nor the mathematical model, can yet address the more complex issue of how jackal contact rates may depend on jackal population density, so our calculations are confined to investigating persistence as a function of contact rate at a fixed population density.

(b) *Increasing the immigration rate of rabies from an external reservoir*

Assuming that our estimate of the jackal contact rate listed in table 1 is representative of the overall jackal contact rate, then the only other way that rabies can persist in the jackal population is if the immigration of infection into the jackal population from an external disease reservoir takes place sufficiently frequently. Without it, after an interval of time, jackal rabies would eventually disappear. Rabies must therefore arrive in the jackal community sufficiently frequently to suggest an almost continuous presence of rabies in the population.

To investigate this more systematically we have plotted in figure 3 the proportion of years for which rabies is present in the jackal population as a function of the mean time-interval, τ, between immigration of infection into the population. Thus, the immigration process is modelled as a Poisson process with a mean of $f = 1/\tau$ immigrations per day. As before, we maintained the jackal density at $1\,\mathrm{km}^{-2}$ and all other parameters as in table 1.

This calculation suggests that as the immigration rate of infection, f, is increased (i.e. the time-interval between immigrations is decreased), we move from a phase where extinction is commonplace to another phase, at high immigration rates, where chance disappearance of rabies in jackals completely ceases. At this point there is a practically constant presence of rabies in the jackal population.

Given that in Zimbabwe rabies epidemics in dogs are sometimes correlated with subsequent rabies epidemics in jackals (Cleaveland & Dye 1995) we believe that of the two explanations presented above, in § 4a,b, it is more likely that the continued presence of rabies in jackals stems from increasing contact with the growing dog population and the associated degree of rabies circulation within these dogs. In § 5, we suggest that infected domestic dogs are the most likely source of rabies transmission to jackals, and then we go on to investigate how the incidence of jackal rabies may be influenced by the prevalence of rabies in these dogs as their population increases over time.

5. DOGS AS A RESERVOIR FOR RABIES INFECTION

The importance of domestic dogs in the transmission and maintenance of rabies virus infection in southern Africa cannot be overstated (Perry 1993; Kitala *et al.* 1993; Butler 1995; Cleaveland & Dye 1995; Cleaveland

1995; De Balogh *et al.* 1995; Madzima 1995). The density of dogs often found in the urban areas and communal lands are thought to be sufficient to maintain rabies infection. The role of dog populations in Africa as a rabies reservoir has been studied in detail by Cleaveland & Dye (1995).

In Zimbabwe, as in other countries in the sub-region, most human exposure to rabies occurs through bites from infected dogs. To reduce the threat to humans, veterinary authorities in Zimbabwe have, since 1950, maintained an active programme of dog vaccination which, along with other control measures, has often been able to reduce the numbers of susceptible dogs and curb rabies outbreaks (Foggin 1988).

We are particularly interested in the propensity for rabies cases in dogs to initiate secondary epidemics in jackals. Our belief is that while jackal populations are themselves unable to support infection endemically, they are constantly exposed to infection when they interact with the dogs along the communal land–commercial farmland boundaries. Contact between a rabid dog and a jackal can trigger a localized epidemic in the jackal population which largely inhabits the commercial farmland. This infection transfer process can occur continually along the communal land–commercial farmland boundaries and is, we believe, the primary route by which rabies enters the jackal population. Also, we might expect that the greater the incidence of rabies occurring in dogs, the greater the corresponding increase in jackal rabies. In the next paragraph we set out estimates of the rate of input of rabies into the jackal population from the dogs, and incorporate this into the mathematical model described above, to investigate how it affects the incidence of rabies cases in the jackal population of Zimbabwe. We do not aim to provide an ecologically realistic picture of rabies in the dog population; rather, we generate estimates of the rate of input of infection into the jackal population based on the incidence of dog rabies and the rate of contact between dogs and jackals.

Over the past 40 years rabies infection has been continually present in the dog population of Zimbabwe. Figure 4 shows the reported annual incidence of dog rabies (Foggin 1988; Bingham 1992; Madzima 1995). At present, we have no information on case-reporting accuracy, but we believe that these figures give a fairly accurate picture because dogs live in proximity to humans and, in any community, the appearance of rabies is always a cause for concern. It is therefore reasonable to assume that there will be fairly uniform reporting biases between communities. Also, the disease-incidence figures do reflect those times when dog-vaccination programmes were working particularly well or particularly poorly, suggesting that rates of under-reporting are fairly consistent.

The dog population of the country has increased dramatically over recent years. No regular, accurate census figures are available, but in 1954 an authoritative source put the dog population at around 195 000 (Adamson 1954), and in 1986, Brooks (1990) estimated the dog population to be 1.3 million.

Assuming a constant *per capita* growth rate, the total dog population of Zimbabwe is shown in figure 5*a*. An active national programme of dog rabies vaccination has reduced the overall number of susceptible dogs in circulation, and the total number of dog vaccinations per year is shown in figure 5*b* (Madzima 1995). The estimated number of susceptible dogs since 1950 is plotted in figure 5*c*.

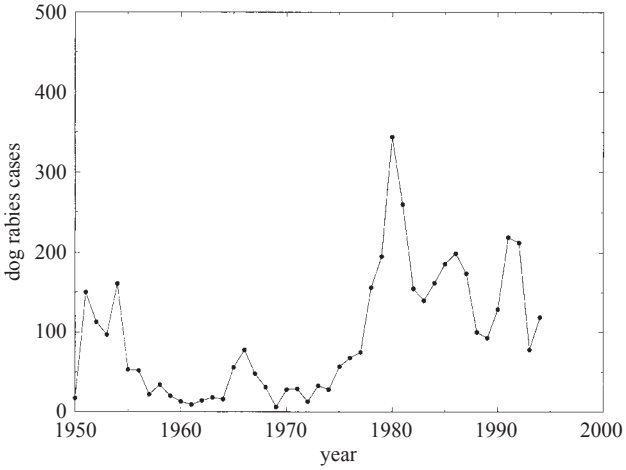

Figure 4. Annual incidence of dog rabies in Zimbabwe.

Because we wish to derive estimates of the future incidence of jackal rabies, we need a reliable measure of the incidence of dog rabies. This will allow us to calculate the numbers of rabid dogs present as the dog population rises in the years ahead. From the data in figure 4 and figure 5*c*, we estimate that at any one time approximately 0.001% of susceptible dogs in Zimbabwe are infected with rabies. This figure is in line with that obtained from the rabies model of Cleaveland & Dye (1995), from which they found an average of approximately 0.003% of dogs are infected at any one time. By using our approximate value for the prevalence of dog rabies we can calculate how many dog rabies cases have occurred in the past and, given a continuing dog population growth rate with a 38% vaccination coverage, the number of expected dog rabies cases up to the year 2020. Figure 6 shows the estimated incidence of dogs rabies, with the actual number of dog rabies cases (1950–1993) superimposed. We believe that as a broad measure of the incidence of dog rabies our approximation works quite well over the period that dog rabies data exist and can, therefore, act as a reasonable predictor for the next 20 years.

6. DOG–JACKAL INTERACTIONS

Given that we have an estimation of the incidence of rabies in the dog population, the next step is to estimate how often the dogs and jackals interact, and hence obtain an estimate of the frequency of dog-to-jackal rabies transmission events.

The jackals and unvaccinated dogs interact along the boundaries of the communal lands and the commercial farmlands. It is estimated that approximately 21% of the total land area of Zimbabwe is available for dog–jackal interaction (Foggin 1988), so we scale the relevant populations accordingly to get an idea of the actual numbers of animals involved in this border region. Little is known about dog–jackal interactions so, in the absence of hard data, we make the assumption that in the border regions jackals contact dogs as often as they encounter other jackals, i.e. approximately once per week. From a knowledge of the dog and jackal populations in the border

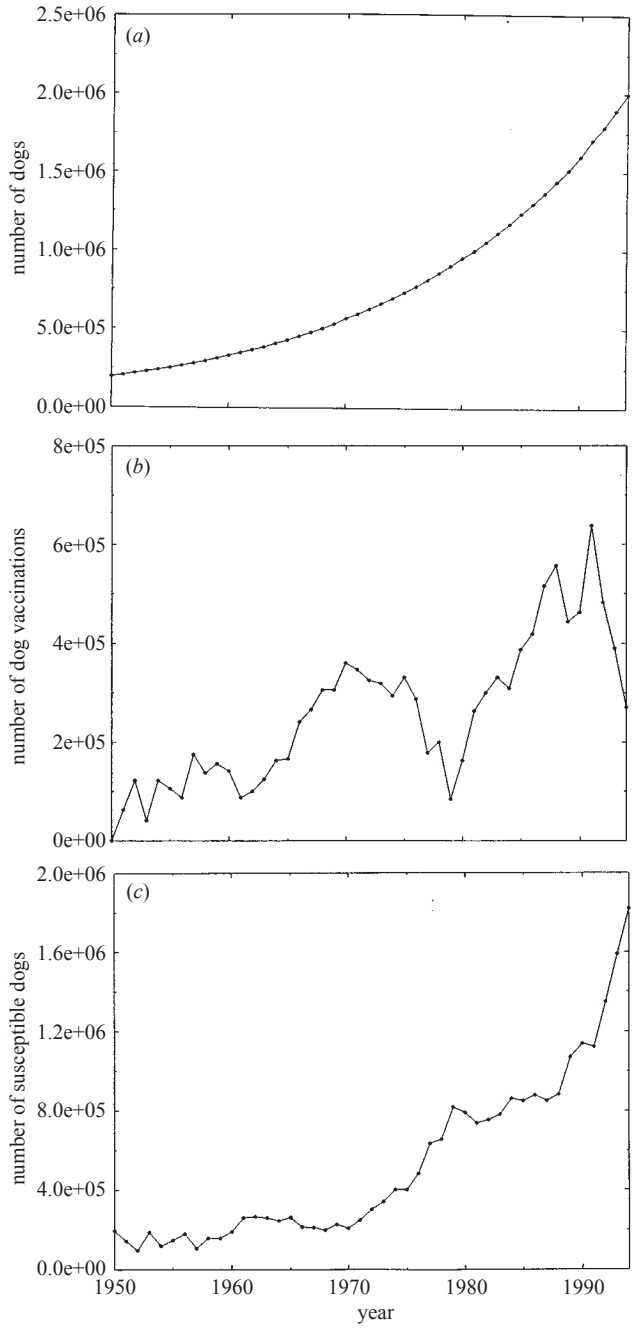

Figure 5 (*a*) Estimated total dog population of Zimbabwe. (*b*) Number of dog vaccinations against rabies carried out each year by veterinary authorities. (*c*) Estimated number of susceptible dogs in the population.

regions, the expected proportion of dogs that are rabid and the frequency of dog–jackal contacts, a straightforward calculation gives us the number of days between individual transmission events of rabies from dogs to jackals in Zimbabwe. For example, in 1960 we calculate that approximately every 19 days a jackal somewhere in the entire population would become infected with rabies. By contrast, in 1980 the rate of transmission of infection was around every three days because the dog population was higher and vaccination coverage was running at around 10%.

Using our estimates of the rate of input of infection into the jackal population, we can make a simple modification to our mathematical model to allow for this effect. We perform the calculation in two ways. First, we calculate the incidence of jackal rabies using the historical recorded incidence of dog rabies, as shown in figure 4, as an input to the mathematical model. The result of this calculation is shown in figure 7*a*. From this it is clear that the incidence of rabies in jackals is strongly driven by the incidence of rabies in dogs. Indeed, the calculated incidence of jackal rabies strongly resembles the actual pattern of disease incidence. Because this calculation uses historical data, it is not possible to extrapolate this calculation into the future, but it does suggest that the observed pattern of rabies epidemics in jackals is driven by the incidence of rabies in dogs.

Second, we repeat the above calculation, but instead of using historical data we use the estimated incidence of dog rabies shown in figure 6. The result of this calculation in figure 7*b* indicates that coupling the jackal population to the estimated level of dog rabies still gives a reasonable picture of the overall incidence of jackal rabies. As it is based on an averaging technique it cannot reflect the true fluctuations of disease, but nevertheless it is able to track the overall average prevalence of disease in jackals. Given that this calculational approach works well for past jackal rabies incidence, in § 7 we show how the future incidence of jackal rabies can be calculated assuming that the dog population continues to grow at its present rate.

These calculations demonstrate that the overall incidence of rabies in jackals tracks the incidence of disease in dogs, suggesting that dogs act as a reservoir of rabies. The calculation of jackal rabies done by using the actual incidence of dog rabies, see figure 7*a*, strongly suggests that the overall level of rabies in dogs correlates with the number of jackals with the disease. If at any time this source of infection is removed (i.e. interactions between rabid dogs and jackals cease completely), then the incidence of rabies in jackals could decline to zero very quickly. Interestingly, Cleaveland & Dye (1995) reached a similar conclusion from an analysis of the raw data from Zimbabwe, and they suggested that there is a one-year time lag between the jackal and dog disease incidences.

7. ESTIMATES OF FUTURE INCIDENCE OF JACKAL RABIES

Using our mathematical framework, it is straightforward to calculate future incidences of jackal rabies based on different vaccination coverage rates. In figure 6, we gave an estimate of the past incidence of dog rabies. Assuming that the dog population continues to grow at the rate shown in figure 5*a*, and assuming a 0.001% prevalence of rabies in dogs, we can calculate the future incidence of rabies in jackals, maintaining the assumption that the jackals interact with dogs no more frequently than they do with other jackals.

For 1994, the last year for which we have data, around 15% of dogs were vaccinated against rabies. If this level of vaccination coverage is maintained into the future, given the current rate of increase in the dog population, the likely incidence of jackal rabies up to the year 2020 will be similar to that shown in figure 8*a*. The projected increase in jackal rabies is extremely large, suggesting

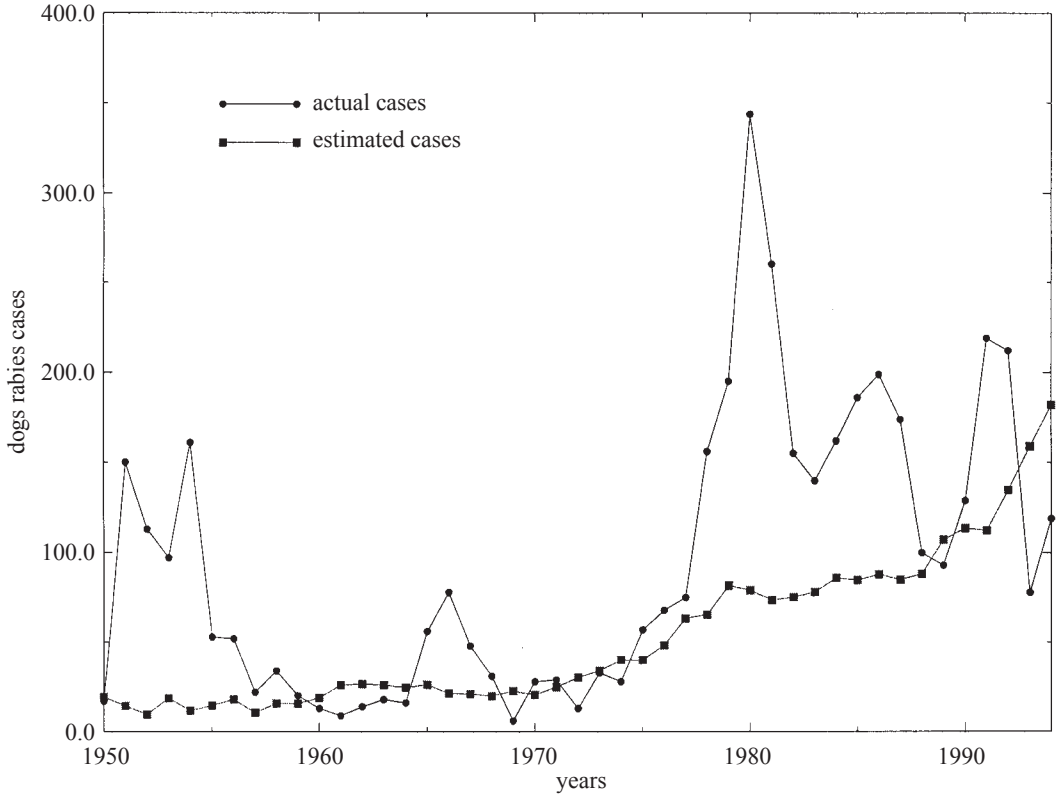

Figure 6. Estimated incidence of dog rabies based on an assumption about the average prevalence of dog rabies. Also shown are the actual recorded cases of dog rabies from figure 5.

that there will be a significant increase in potential threats to livestock and humans.

Through the 1990s as a whole, the average dog-vaccination coverage has been around 25%. If this coverage can be maintained in future, reversing the decline of the past couple of years, the resulting future incidence of jackal rabies will be approximately that shown in figure 8*b*.

In figure 8*c* we repeat the calculations of the previous paragraph, but now assume that 50% of susceptible dogs are vaccinated. Clearly, the future incidence of jackal rabies is considerably reduced, indicating that if resources dedicated to dog vaccination can be increased to broadly double that of the average 1990s level, the incidence of rabies in Zimbabwe would be considerably reduced.

In reality these rough estimates are probably on the high side, as the jackal rabies dynamics are strongly driven by the assumed future exponential increase in the dog population. Density-dependent pressures are likely to restrict dog population growth to below full exponential growth, thus reducing the numbers of rabid dogs and hence reducing the incidence of rabies in jackals. Also, these calculations suggest that because the actual incidence of dog rabies is a good indicator of the prevalence of rabies in jackals, the control of rabies in dogs will have an immediate impact on the level of jackal rabies. Ideally we would like to have a precise estimate for the critical vaccination proportion of dogs in Zimbabwe that would result in the eradication of rabies from the dog population. This could then be used as a target in dog vaccination programmes. Unfortunately, such estimates are highly variable and difficult to guess given the lack of a detailed picture of dog ecology and community structure in Zimbabwe. Also, we have, at present, little understanding of the complexities introduced when considering a spatially distributed host population, as the dogs and jackals clearly are. Related work on measles virus infection has recently shown (Ferguson 1996) that consideration of spatial heterogeneity can lead to models with a rich spectrum of dynamical behaviour, and these necessarily lead to revised estimates for critical community sizes and critical vaccination proportions.

Over the past 40 years the dog population in Zimbabwe has, on average, increased at broadly an exponential rate, mirroring the human population expansion, whereas the numbers of dogs vaccinated each year has grown, at best, linearly. This ever-widening gap between total dog numbers and those vaccinated must be addressed if rabies is not to become a serious threat to public health and, in turn, a threat to the diverse and sometimes vulnerable mammal populations of Africa (Perry 1992). As we do not have a comprehensive model of rabies in the dog population itself, we do not consider more sophisticated intervention strategies here. Increased understanding of dog ecology and rabies epidemiology will be essential to combat rabies in Zimbabwe. The focus of our interest in this study, however, is disease in the jackal population.

8. DISCUSSION

Using a combination of field observations and a simple mathematical model of the population biology of rabies infection in jackals in Zimbabwe, we have shown that it is unlikely that jackals act as a reservoir of this disease.

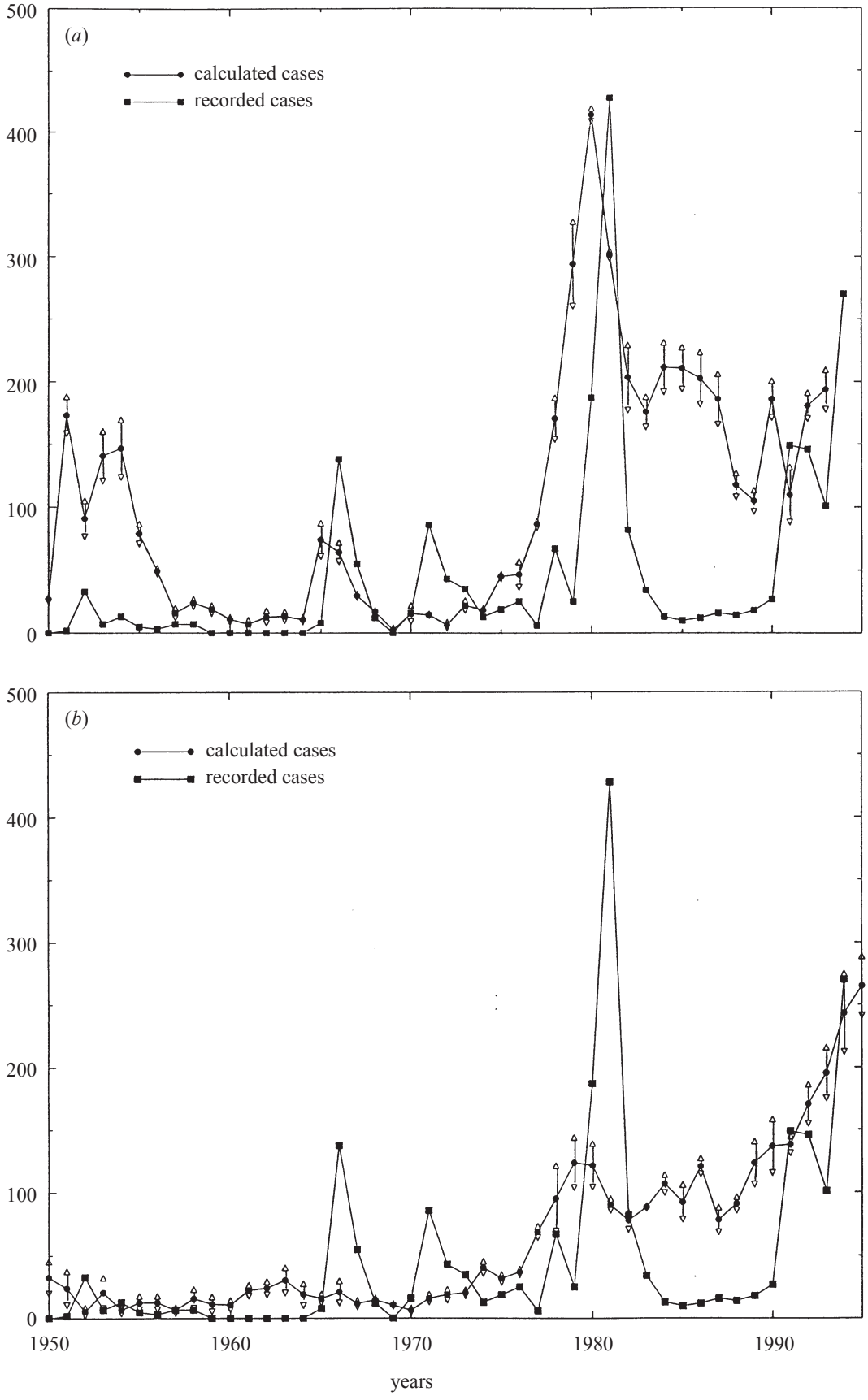

Figure 7. (*a*) Incidence of jackal rabies, 1950–1994, calculated using the mathematical model. The actual recorded incidence of dog rabies was used in this calculation. The recorded cases of jackal rabies (figure 1) are also shown for comparison. (*b*) Incidence of jackal rabies, 1950–1994, calculated using the mathematical model. The estimated incidence of dog rabies was used in this calculation. The recorded cases of jackal rabies (figure 1) are also shown for comparison.

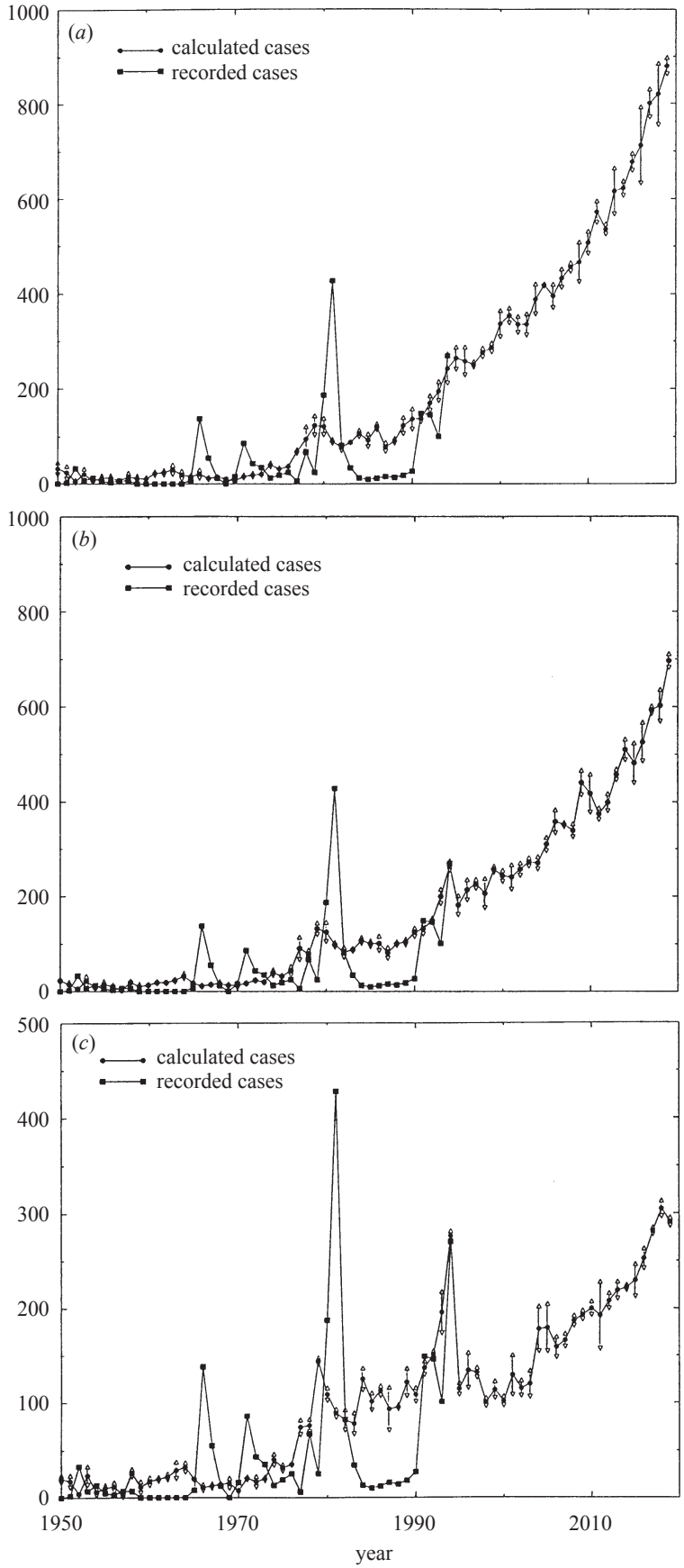

Figure 8. (*a*) Calculated incidence of future jackal rabies up to the year 2020, based on maintaining the current vaccination programme of around 15% of dogs each year after 1994. Also shown are the recorded jackal rabies cases. (*b*) Calculated incidence of future jackal rabies up to the year 2020, based on maintaining the average 1990s vaccination programme of around 25% of dogs each year after 1994. Also shown are the recorded jackal rabies cases. (*c*) Calculated incidence of future jackal rabies up to the year 2020, based on a considerably expanded vaccination programme where 50% of dogs are vaccinated against rabies each year after 1994. Also shown are the recorded jackal rabies cases.

Specifically, this means that if there ceased to be any interaction between rabid dogs and susceptible jackals then we might soon see the elimination of rabies from the jackal population. Our model, although simple in structure, has been furnished with parameters obtained from detailed field observations of jackal populations, and the population density of jackals appears, on average, to be too low to permit long-term persistence. As noted by other workers (Cleaveland 1995; Cleaveland & Dye 1995), the comparatively dense dog populations on the communal lands are the most likely source of rabies infection.

Using historical data on the prevalence of rabies in dogs and an estimate of the frequency of dog–jackal contacts, we have shown calculations of the total number of expected jackal rabies cases per year which demonstrate how rabies infection in jackals is strongly forced by the incidence of the disease in dogs. Furthermore, we were able to show likely incidences of the disease in jackals for the next 20 years for different levels of vaccination coverage.

Our results also have implications for proposed rabies control policies in Zimbabwe. Clearly, the dog populations must be the object of most attention. Programmes aimed at increasing control and vaccination of dogs would have the most significant impact on reducing rabies in Zimbabwe. Culling of jackals would have less impact, as the population density is, on the whole, too low to sustain endemic infection in the absence of injection of infection from external sources. Jackal culling will, at best, and only on a local scale, only serve to reduce the risk of rabies infection being passed to agricultural-stock animals and cannot act as an effective means of rabies control (Aubert 1994). It is possible that the jackals act as a buffer to protect stock animals on the large commercial farms from incursion by itinerant rabid dogs from the communal areas. Furthermore, it might be possible to protect jackals living along the communal land–commercial farmland boundaries by implementing a European-style oral vaccination campaign. This is likely to be cost-effective, as it is restricted to a sub-section of the entire jackal population, acting as a cordon sanitaire preventing rabies reaching agricultural-stock animals on commercial land. Studies on the efficacy of oral rabies vaccines for jackals in Zimbabwe have been undertaken (Bingham *et al.* 1995*b*).

The mathematical model we have used is a simple and effective framework within which we have been able to discuss the epidemiology of rabies virus infection in jackals. However, we have not been able to discuss the effect of a spatially distributed host population in a manner similar to that of Murray *et al.* (1986) in their studies of European fox rabies, nor have we included any estimate of the effect of asymptomatic infection. Future work will probably be able to type more accurately the rabies viruses extracted from dogs and jackals, although at present they are thought to be identical. Also, little is known about the relative susceptibilities of dogs and jackals to the virus. Improved demographic and ecological data concerning dogs and jackals (both side-striped and black-backed) will improve our undertanding of rabies in Zimbabwe. More generally, it has been shown recently that mass-action-based epidemiological models can have limitations when applied to sporadic outbreaks of disease in populations well below that needed to maintain the chain of infection (Rhodes & Anderson 1996). This is precisely the epidemiological scenario that arises with rabies in jackals, and improved models of spatially and temporally limited disease outbreaks in largely susceptible populations are needed.

Given the high public health profile of rabies infection, initial studies that have been targeted at gaining a better understanding of the population biology of dogs on communal lands and the impact of rabies on these populations should be continued (Butler 1995). More resources must be added to the national programme of dog vaccination, and steps must also be taken to curtail the rapidly expanding dog population.

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REFERENCES

Adamson, J. S. 1954 Ecology of rabies in Southern Rhodesia. *Bull. World Health Org.* **10**, 753–759.

Anderson, R. M. & May, R. M. 1991 *Infectious diseases of humans, dynamics and control*. Oxford University Press.

Anderson, R. M., Jackson, H. C., May, R. M. & Smith, A. D. M. 1981 Population dynamics of fox rabies in Europe. *Nature* **289**, 765–771.

Atkinson, R. P. D. 1996 The side-striped jackal. In *The complete book of southern African mammals*, pp. 231–247. Cape Town: Struik Publishers (Pty) Ltd.

Aubert, M. 1994 Control of rabies in foxes: what are the appropriate measures? *Vet. Rec.* **134**, 55–59.

Bacon, P. J. 1985 (ed.) *Population dynamics of rabies in wildlife*. London: Academic Press.

Ball, F. G. 1985 *Population dynamics of rabies in wildlife* (ed. P. J. Bacon), chap. 8, pp. 197–222. London: Academic Press.

Bingham, J. 1992 Rabies in Zimbabwe. In *Proceedings of the international conference on the epidemiology, control and prevention of rabies: eastern and southern Africa* (ed. A. King). Lyon: Editions Fondation Marcel Merieux.

Bingham, J. 1995 Rabies in Zimbabwe. In *Proceedings of the third international conference of the southern and eastern African rabies group* (ed. J. Bingham, G. C. Bishop & A. A. King). Lyon: Editions Fondation Marcel Merieux.

Bingham, J. & Foggin, C. M. 1993 Jackal rabies in Zimbabwe. *Onderstep. J. Vet. Res.* **60**, 365–366.

Bingham, J., Kappeler, A., Hill, F. W. G., King, A. A., Perry, B. D. & Foggin, C. M. 1995 Efficacy of SAD (Berne) rabies vaccine given by the oral route in 2 species of jackal (*Canis mesomelas* and *Canis adustus*). *J. Wildl. Dis.* **31**, 416–419.

Blancou, J., Aubert, M. F. A. & Artois, M. 1991 Fox rabies. In *The natural history of rabies*, 2nd edn (ed. G. M. Baer). Boca Raton: CRC Press.

Brooks, R. 1990 Survey of the dog population of Zimbabwe and its level of rabies vaccination. *Vet. Rec.* **127**, 592–596.

Butler, J. R. A. 1995 A survey of communal land dogs in Zimbabwe with reference to improving rabies vaccination coverage. In *Proceedings of the third international conference of the southern and eastern African rabies group* (ed. J. Bingham, G. C. Bishop & A. A. King), pp. 81–94. Lyon: Editions Fondation Marcel Merieux.

Cleaveland, S. C. 1995 Rabies in the Serengeti: the role of domestic dogs and wildlife in maintenance of disease. In *Proceedings of the third international conference of the southern and eastern African rabies group* (ed. J. Bingham, G. C. Bishop & A. A. King), pp. 112–118. Lyon: Editions Fondation Marcel Merieux.

Cleaveland, S. C. & Dye, C. 1995 Maintenance of a microparasite infecting several host species: rabies in the Serengeti. *Parasitology* **111**, S33–S47.

Cumming, D. H. M. 1982 A case history of the spread of rabies in an African country. *South African J. Sci.* **78**, 443–447.

De Balogh, K. K. I. M., Wandeler, A. I. & Meslin, F. X. 1993 A dog ecology study in an urban and semi-rural area of Zambia. *Onderstep. J. Vet. Res.* **60**, 437–443.

Ferguson, N., May, R. M. & Anderson, R. M. 1998 *Spatial ecology: the role of space in population dynamics and interspecific interactions* (ed. D. Tilman & P. Kareiva). Princeton Monographs in Population Biology. (In the press.)

Foggin, C. M. 1988 Rabies and rabies-related viruses in Zimbabwe: historical, virological and ecological aspects. PhD thesis, University of Zimbabwe, Harare.

Gascoyne, S. C., Laurenson, M. K., Lelo, S. & Borner, M. 1993*a* Rabies in African wild dogs (*Lycaon pictus*) in the Serengeti region. *J. Wildl. Dis.* **29**, 396–402.

Gascoyne, S. C., King, A. A., Laurenson, M. K., Borner, M., Schildger, B. & Barrat, J. 1993*b* Aspects of rabies infection and control in the conservation of the African wild dog (*Lycaon pictus*) in the Serengeti region, Tanzania. *Onderstep. J. Vet. Res.* **60**, 415–420.

Grenfell, B. T. 1992 Chance and chaos in measles dynamics. *J. R. Stat. Soc.* B **54**, 383–398.

Kingdon, J. 1997 *The Kingdon field guide to African mammals.* London: Academic Press.

Kitala, P. M., McDermott, J. J., Kyule, M. N. & Cathuma, J. M. 1993 Features of dog ecology relevant to rabies spread in Machakos district, Kenya. *Onderstep. J. Vet. Res.* **60**, 445–449.

MacDonald, D. W. 1980 *Rabies and wildlife. A biologist's perspective.* Oxford University Press.

MacDonald, D. W. 1993 Rabies and wildlife: a conservation problem? *Onderstep. J. Vet. Res.* **60**, 351–355.

Madzima, W. N. 1995 Rabies control in dogs in Zimbabwe. In *Proceedings of the third international conference of the southern and eastern African rabies group* (ed. J. Bingham, G. C. Bishop & A. A. King), pp. 134–138. Lyon: Editions Fondation Marcel Merieux.

Murray, J. D., Stanley, E. A. & Brown, D. L. 1986 On the spatial spread of rabies among foxes. *Proc. R. Soc. Lond.* B **229**, 111–150.

Olsen, L. F., Truty, G. L. & Schaffer, W. M. 1988 Oscillations and chaos in epidemics: a nonlinear dynamic study of six childhood diseases in Copenhagen, Denmark. *Theor. Popul. Biol.* **33**, 344–370.

Perry, B. D. 1992 The epidemiology of rabies and its control in eastern and southern Africa. In *Proceedings of the first international conference of the southern and eastern African rabies group* (ed. A. A. King), pp. 107–121. Lyon: Editions Fondation Marcel Merieux.

Perry, B. D. 1993 Dog ecology in eastern and southern Africa: implications for rabies control. *Onderstep. J. Vet. Res.* **60**, 429–436.

Press, W. H., Teukolsky, S. A., Vetterling, W. T. & Flannery, B. P. 1994 *Numerical recipes*, 2nd edn. Cambridge University Press.

Rhodes, C. J. & Anderson, R. M. 1996 Power laws governing epidemics in isolated populations. *Nature* **381**, 600–602.

Sillero-Zubiri, C., King, A. A. & MacDonald, D. W. 1996 Rabies and mortality of Ethiopian wolves (*Canis simensis*). *J. Wildl. Dis.* **32**, 180–186.

Skinner, J. D. & Smithers, R. H. N. 1990 *Mammals of the Southern African sub-region*, 2nd edn. University of Pretoria Press.

Smith, G. C. & Harris, S. 1991 Rabies in urban foxes (*Vulpes vulpes*) in Britain: the use of a spatial stochastic simulation model to examine the pattern of spread and evaluate the efficacy of different control regimes. *Phil. Trans. R. Soc. Lond.* B **334**, 459–479.

Stickel, L. F. 1954 A comparison of certain methods of measuring ranges of small mammals. *J. Mamm.* **35**, 1–15.

Swanepoel, R., Barnard, B. J. H., Meredith, C. D., Bishop, G. C., Bruckner, G. K., Foggin, C. M. & Hubschle, O. J. B. 1993 Rabies in southern Africa. *Onderstep. J. Vet. Res.* **60**, 325–346.

White, P. C. L., Harris, S. & Smith, G, C. 1995 Fox contact behaviour and rabies spread: a model for the estimation of contact probabilities between urban foxes at different population densities and its implications for disease control in Britain. *J. Appl. Ecol.* **32**, 693–706.