Theoretical Population Biology **60**, 265–279 (2001)
doi:10.1006/tpbi.2000.1494, available online at http://www.idealibrary.com on IDEAL®

A Mixed Ectoparasite–Microparasite Model for Bat-Transmitted Rabies

E. Massad, F. A. B. Coutinho, M. N. Burattini, P. C. Sallum, and L. F. Lopez

School of Medicine, The University of Sao Paulo, Av. Dr. Arnaldo 455, SP 01246-903, Brazil

Received September 21, 1999

This paper considers the transmission of rabies to domestic livestock by vampire bats. Vampire bats act as ectoparasites on cattle both by ingesting a small amount of blood every night and by prolonging bleeding by the action of anticoagulant substances in their saliva. In addition to this parasitic action bats may also transmit rabies, inflicting important losses on affected herds by the inevitable mortality due to the infection. We modeled this complex interaction and we also demonstrate that bat control measures are more effective in reducing rabies prevalence and mortality by rabies than cattle vaccination.

Key Words: **vampire bats; rabies; control; modeling.**

1. INTRODUCTION

Populations of vampire bats in Latin America have increased sharply in areas where Europe-type livestock have been introduced. These livestock production practices have created a more favorable and more readily obtainable food supply for the vampire than that afforded by wildlife alone (Brass, 1994). Some population studies of vampire bats in affected regions have demonstrated that vampire populations are almost twice as large in cattle-raising ecosystems as in natural ecosystems (Delpietro *et al.*, 1992). Therefore, both the net growth rates and the carrying capacities of vampire bat populations are strongly influenced by the presence of domestic cattle.

A single vampire may be responsible for as much as 5.75 gallons of blood loss annually (Goodwin and Greenhall, 1961). Cattle are, therefore, debilitated by this continuous bleeding, both by the amount of ingested blood and by that which continues to bleed from open wounds, as may be the case when bats revisit animals each night. The impact is increased in herds that are subject to physical stress associated with poor management practices, like improper diet, parasitism, or infection, a situation typical of tropical, bat-infested areas. In these poorly managed, parasite-infected herds, a 100% reduction in the number of daily vampire bites resulted in a 16% increase in milk production, as demonstrated by a study by Thompson *et al.* (1977).

In addition to the direct effects of vampire bats on livestock in the course of their blood meals, the bats have the potential to spread a variety of diseases (Constantine, 1988). Of major concern is the periodic threat of rabies, a deadly infection transmitted while the bats drink blood. So important are bats in rabies transmission that they are referred to as the best adapted of all mammalian hosts to rabies virus (Beran, 1981). Examples of other bat-transmitted infections include brucellosis, salmonellosis, and trypanosomiasis, in addition to the widespread, and sometimes fatal, myiasis (Constantine, 1988).

Vampire bats are so efficient in transmitting the rabies virus to cattle that they wreak great havoc on the populations of their preferred hosts (Beran, 1981). As a consequence, rabies of vampire bat origin has been considered a major obstacle to expansion of the livestock industry in Latin America (WHO, 1957, 1966, 1973, 1984; Greenhall, 1970). According to Steele (1973), control of vampire bat rabies is probably the biggest challenge facing animal production authorities.

This double role of vampire bats as ectoparasites and vectors of infections of such an importance as rabies

0040-5809/01 $35.00

makes this interaction between bats and cattle a rather peculiar one, and makes possible an interesting theoretical analysis, namely, a system which combines the features of host–ectoparasite and host–microparasite systems in a single model. The aim of this paper is to study the dynamics of rabies and the consequent implications for its control taking into account the peculiarities mentioned above.

2. THE NATURAL HISTORY OF VAMPIRE AND CATTLE INTERACTIONS

Vampire bats act both as ectoparasites upon livestock and as vectors of infections of major economic and health importance. Therefore, the system for describing this complex interaction between vampire bats and cattle should include the specific features of a host–ectoparasite and those of a host–parasite in the same dynamical model. However, before we present our model, some words on the natural history of the interactions between bats and cattle populations, which will guide the model assumptions, are necessary.

We begin by assuming the fact that bat populations are strongly influenced by the presence of cattle in the same environment. This is expressed mathematically by adding specific terms into the total carrying capacity of bats that describes this positive influence of cattle on bats.

The negative influence of bats upon cattle populations is assumed by including a term that limits the net growth rate of the cattle and that takes into account the presence of bats in their ecosystem. Furthermore, an additional mortality rate is considered, which is due to the continuous bleeding, both by ingestion and by the effect of an anticoagulant substance present in the vampire saliva and secondary infections transmitted (Constantine, 1988). This last term is an explicit function of the biting rate of the vampire bats on cattle.

As related to the rabies infection, its natural history considered in our model takes into account the following facts:

1. Bats acquire rabies infection almost exclusively from other bats, although a very small probability of acquiring the infection from infected cattle is considered in our model. This assumption of bat-to-bat predominant infection is based on the fact that rabies is not a blood-borne infection but rather a saliva-borne one (Johnson, 1982). In addition, the rabies viraemia is usually negligible and very short-lived (Johnson, 1982). Also, it is a well-known fact that inside the bat colony, bats lick and bite each other very frequently (Fenton, 1992).

2. Once infected, a vampire enters into a latent status, which lasts from 2 to 4 weeks (Brass, 1994) and during this phase they are already transmitters of the infection but no behavioral change is observed in the infected animals.

3. After this latency period, infected bats develop the clinical phase, which lasts for a week or so (Brass, 1994), and during which major behavioral changes are observed. The flight orientation of rabid bats is lost and the animals become very aggressive, biting literally any object or prey around, including other bats (Brass, 1994).

4. Bitten cattle are revisited almost every night. A single bat is able to feed practically every night and normally does not survive for more than two consecutive nights without a blood meal (Fenton, 1992). The cattle may acquire the rabies infection through the infected saliva from vampire bats and goes, after a very short incubation period, straight to the clinical phase. This phase is typically characterized by the paralytic form of the disease and the affected animal dies inexorably after a period that lasts from 1 to 4 weeks (Brass, 1994).

5. Control of rabies infection is possible by cattle vaccination, which is considered to be very effective in protecting the animals from infection but which lasts for no more than 1 year (Arellano-Sota, 1988). Alternatively, bat control is possible by methods designed primarily to selectively kill vampire bats while in the process of attacking cattle or of licking each other inside the colony. These methods include poisons (such as strychnine and arsenic) and various anticoagulants either topically applied to the open wounds or to the back of captured vampire bats.

3. THE MODEL

The model assumes a cattle host population, with bats acting both as ectoparasites of cattle and as biological vectors transmitting rabies within and between the two populations. These are divided into six categories, describing the number of bats (indexed as $\cdot_b$) and cattle (indexed as $\cdot_c$) subpopulations that are susceptible (X_b and X_c), infected but without noticeable disease (H_b) also called "latent," clinically ill (Y_b and Y_c), and vaccinated (Z_c). Total populations of bats and cattle are labeled N_b and N_c, respectively.

Figure 1 illustrates the model compartments and the flow between them.

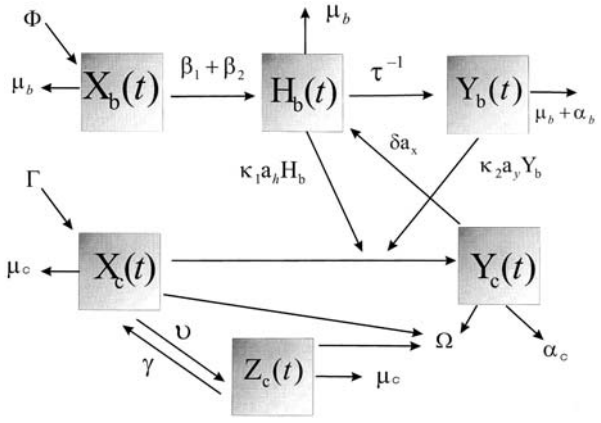

FIG. 1. Model compartments and the flow between them. Input and output symbols are $\Phi = r_b N_b(1 - N_b/(K_b + \sigma_1 N_c))$, $\Gamma = (r_c/(1+\sigma_2 N_b))$, $N_c(1 - N_c/K_c)$, and $\Omega = \varepsilon(a_x X_b + a_h H_b + a_y Y_b)$.

The model is described by the following set of delay differential equations,

$$\frac{d}{dt}X_b(t) = -\beta_1 X_b \frac{H_b}{N_b} - \beta_2 X_b \frac{Y_b}{N_b} - \delta a_x X_b \frac{Y_c}{N_c} + r_b N_b \left(1 - \frac{N_b}{K_b + \sigma_1 N_c}\right) - \mu_b X_b$$

$$\frac{d}{dt}H_b(t) = \beta_1 X_b \frac{H_b}{N_b} + \beta_2 X_b \frac{Y_b}{N_b} + \delta a_x X_b \frac{Y_c}{N_c} - \beta_1 X_b(t-\tau)\, e^{-\mu_b \tau} \frac{H_b(t-\tau)}{N_b(t-\tau)} - \beta_2 X_b(t-\tau)\, e^{-\mu_b \tau} \frac{Y_b(t-\tau)}{N_b(t-\tau)} - \delta a_x X_b(t-\tau)\, e^{-\mu_b \tau} \frac{Y_c(t-\tau)}{N_c(t-\tau)} - \mu_b H_b$$

$$\frac{d}{dt}Y_b(t) = \beta_1 X_b(t-\tau)\, e^{-\mu_b \tau} \frac{H_b(t-\tau)}{N_b(t-\tau)} + \beta_2 X_b(t-\tau)\, e^{-\mu_b \tau} \frac{Y_b(t-\tau)}{N_b(t-\tau)} + \delta a_x X_b(t-\tau)\, e^{-\mu_b \tau} \frac{Y_c(t-\tau)}{N_c(t-\tau)} - (\mu_b + \alpha_b) Y_b$$

$$\frac{d}{dt}X_c(t) = -\nu X_c - (\kappa_1 + \varepsilon)\, a_y Y_b \frac{X_c}{N_c} - (\kappa_2 + \varepsilon)\, a_h H_b \frac{X_c}{N_c} - \mu_c X_c - \varepsilon a_x X_b \frac{X_c}{N_c} + \frac{r_c}{1+\sigma_2 N_b} N_c \left(1 - \frac{N_c}{K_c}\right) + \gamma Z_c$$

$$\frac{d}{dt}Y_c(t) = \kappa_1 a_y Y_b \frac{X_c}{N_c} + \kappa_2 a_h H_b \frac{X_c}{N_c} - \alpha_c Y_c - \varepsilon(a_x X_b + a_h H_b + a_y Y_b) \frac{Y_c}{N_c}$$

$$\frac{d}{dt}Z_c(t) = \nu X_c - \gamma Z_c - \mu_c Z_c - \varepsilon(a_x X_b + a_h H_b + a_y Y_b) \frac{Z_c}{N_c}, \tag{1}$$

where the definition, the biological meaning, and value of each of the parameters used are described in Table I.

Let us describe briefly a few features of the model.

The first thing to note is that we choose to consider density dependence among cattle only on births, rather than on deaths. This is due to the assumption that this is a herd raised under strictly controlled conditions, and whose births are controlled by the farmer and the deaths are determined by harvesting or diseases other than rabies. The amount of available food will be, therefore, the factor determining the net growth rate of this population.

Bats, on the other hand, although assumed as wild species (which they are indeed), are also assumed to have their growth rate checked by density dependence on births.

TABLE I

Definition, Biological Meaning, and Values of the Parameters Used in the Numerical Simulations

Parameter	Biological meaning	Value
β_1	Contact rate between susceptible and latent bats	4 year^{-1}
β_2	Contact rate between susceptible and rabid bats	8 year^{-1}
δ	Probability of bats acquiring rabies from rabid cattle	0.0001
a_x	Rabid bats biting rate	200 year^{-1}
μ_b	Bats natural mortality rate	0.083 year^{-1}
r_b	Birth rate of bats	2 year^{-1}
K_b	Bats carrying capacity	1000
σ_1	Influence of cattle on bats carrying capacity	0.01
τ	Rate of transference from latent to rabid bats	0.083 year^{-1}
α_b	Bats addition mortality rate due to rabies	12 year^{-1}
v	Cattle vaccination rate	variable
κ_1	Fraction of rabid bats' bites that result in an infection	0.7
ε	Fraction of biten cattle that die from bleeding	0.001
a_y	Rabid bats biting rate	300 year^{-1}
κ_2	Fraction of latent bats' bites that result in an infection	0.4
a_h	Latent bats biting rate	200 year^{-1}
μ_c	Cattle harvesting + natural mortality rate	0.067 year^{-1}
r_c	Cattle birth rate	10 year^{-1}
σ_2	Influence of bats on cattle population growth rates	0.001
K_c	Carrying capacity of cattle	10000
γ	Rate of immunity loss	1 year^{-1}
α_c	Total mortality rate of rabid cattle	60 year^{-1}

This is due to the fact that bats are assumed to be dependent on cattle as their main source of food, which when restricted induces a diminishment in the reproductive output (Mayr, 1970; Roughgarden, 1979). As mentioned in the Introduction, this seems to be the case since it was observed that, in South America, the bat population greatly increased after the introduction of European livestock. In addition, bats, by living in family groups in which individuals are genetically related to each other, are able to adjust their reproductive rates proportionally to the ratio N_b/K_b (Lomnicki, 1988, p. 12). In Appendix B, we derive the main formulas considering density dependence on both births and deaths for a general case.

Rabies, like a sexually transmitted disease, is acquired by direct contact between a susceptible individual and an infected one and, therefore, does not obey the mass-action law of transmission. We assumed that the number of contacts per unit of time is independent of the population size in a sufficiently large population. Let then $\varphi_1 X_b$ be the number of direct contacts that susceptible bats have with other bats. A fraction of those contacts H_b/N_b is with latent bats and the probability of getting the disease is ξ_1 so that the rate of change due to those contacts is $\varphi_1\xi_1 X_b(H_b/N_b)$. Similarly, the fraction of contacts with infected bats is Y_b/N_b and the probability of getting the disease is ξ_2. Therefore, the contribution of those contacts to the rate of change is $\varphi_1\xi_2 X_b(Y_b/N_b)$. Similarly, the latent (infected) bats make direct contacts with other bats with a rate $\varphi_2 H_b(\varphi_3 Y_b)$ and this results in rates of change $\varphi_2 H_b\xi_3(X_b/N_b)(\varphi_3 Y_b\xi_4(X_b/N_b))$. Collecting all those terms, and calling $\beta_1 = \varphi_1\xi_1 + \varphi_2\xi_3$ and $\beta_2 = \varphi_1\xi_2 + \varphi_3\xi_4$, we have that susceptible bats acquire rabies mainly through direct contact with other infected bats ($\beta_1 X_b(H_b/N_b)$ and $\beta_2 X_b(Y_b/N_b)$) although a small fraction, δ, of bats which feed on infected cattle are assumed to acquire infection ($\delta a_x X_b(Y_c/N_c)$). The latter assumption is a theoretical possibility and is considered here for completeness only. Another important assumption of the model refers to the change in the bats' behavior when their infection with rabies, H_b, after a latent period, τ, became clinically noticeable, Y_b. This change implies an increase in both the infectious contact among bats, β_2, and the average biting rate upon cattle, a_y, and results from a noticeably more aggressive behavior of rabid bats. The transition from latent to rabid bats occurs with rates

$$\beta_1 X_b(t-\tau)\, e^{-\mu_b\tau}\frac{H_b(t-\tau)}{N_b(t-\tau)} + \beta_2 X_b(t-\tau)\, e^{-\mu_b\tau}\frac{Y_b(t-\tau)}{N_b(t-\tau)} + \delta a_x X_b(t-\tau)\, e^{-\mu_b\tau}\frac{Y_c(t-\tau)}{N_c(t-\tau)}.$$

Susceptible cattle are removed from this condition either by vaccination with a 100% effective vaccine, with a rate ν, or through a fraction, κ_i, of the biting rates, a_y and a_h, from rabid and latent bats ($\kappa_1 a_y Y_b(X_c/N_c) + \kappa_2 a_h H_b(X_c/N_c)$), respectively. A fraction of the cattle, ε, is also assumed to die by bleeding from bats biting with rates equal to $\varepsilon(a_x X_b + a_h H_b + a_y Y_b)/N_c$. Healthy cattle are removed from the population by natural mortality or harvesting, at a rate μ_c. Vaccinated cattle lose their immune status after an average period of $1/\gamma$ years. Rabid bats have an additional mortality rate, α_b over their natural mortality rate, and rabid cattle have a total death rate, α_c.

Note that if one adds up the cattle equations then the ε term (deaths from biting and due to secondary infections) does not depend on the numbers of cattle. This is a good approximation when the number of cattle is much larger than the number of bats, because the amount of blood a bat takes is independent of the number of cattle available if cattle are abundant. In this case the number of bitten cattle per unit of time depends only on the number of bats. As a result of bleeding and secondary infections, a fraction of them may die per unit of time. However, when the number of bats approaches the number of cattle this must be modified. In this paper we consider always $N_c \gg N_b$ and therefore shall not modify our expression. When $N_b \to N_c$, the terms will depend on a largely unknown behavior of the bats. However, if one wants to avoid that the bat population drives the cattle population to extinction one should replace the e terms by something like

$$-\varepsilon(a_x X_b + a_h H_b + a_y Y_b)\, f(N_c), \tag{2}$$

where $f(N_c)$ is a function which is 0 when $N_c \to 0$ and increases rapidly to one when N_c increases. The function f is commonly known as a functional response; a standard form, which has the property described here, is the Holling type II response (Pulliam, 1989): $f(x) = x/(x+c)$.

4. THE BASIC REPRODUCTION NUMBER (R_0)

The central parameter related to the intensity of transmission of infections is the so-called basic reproduction number (R_0), defined by Macdonald (who called it the "basic reproduction rate") as the number of secondary infections produced by a single infective in an entirely susceptible population (Macdonald, 1952). In

our case there is a vaccine. So, we are going to calculate the number of secondary infections produced by a single infective among the susceptible, non-vaccinated individuals.

There are several ways of estimating R_0. In what follows, we give an intuitive derivation of R_0. In Appendix A we outline a more formal derivation.

We begin by calculating the numbers $\bar{X}_b$, $\bar{X}_c$, $\bar{Z}_c$ of bats, susceptible cattle, and vaccinated cattle, respectively, in a population free of rabies. They satisfy the system of equations

$$\begin{aligned} -\mu_b \bar{N}_b + r_b \bar{N}_b \left(1 - \frac{\bar{N}_b}{K_b + \sigma_1(\bar{X}_c + \bar{Z}_c)}\right) &= 0 \\ -\nu \bar{X}_c - \varepsilon a_x \bar{N}_b \frac{X_c}{N_c} - \mu_c \bar{X}_c + \gamma \bar{Z}_c + \frac{r_c}{1+\sigma_2 \bar{N}_b} \bar{N}_c \left(1 - \frac{\bar{N}_c}{K_c}\right) &= 0 \\ \nu \bar{X}_c - \gamma \bar{Z}_c - \varepsilon a_x \bar{N}_b \frac{Z_c}{N_c} - \mu_c \bar{Z}_c &= 0, \end{aligned} \tag{3}$$

where $\bar{N}_c = \bar{X}_c + \bar{Z}_c$.

An infected bat can transmit rabies in two ways: either it can directly infect other bats or it can infect cattle which in turn can transmit to bats. In a completely susceptible population an infected bat will, during its latent phase, infect

$$\beta_1 \int_0^\tau \exp(-\mu_b x)\, dx = \frac{\beta_1(1 - e^{-\mu_b \tau})}{\mu_b} \tag{4}$$

other bats.

The probability of surviving the latent phase is $e^{-\mu_b \tau}$ and the average lifetime of an ill bat is $1/(\mu_b + \alpha_b)$. So, during the clinically ill phase a bat will on average infect

$$\frac{\beta_2 e^{-\mu_b \tau}}{\mu_b - \alpha_b} \tag{5}$$

other bats.

Following a similar reasoning, a bat will infect

$$\left[\kappa_2 a_h \frac{(1 - e^{-\mu_b \tau})}{\mu_b} + \kappa_1 a_y \frac{e^{-\mu_b \tau}}{\mu_b + \alpha_b}\right] \frac{\bar{X}_c}{\bar{N}_c} \tag{6}$$

cows.

In an uninfected population each infected cow will in turn give rise to

$$\frac{m\, \delta a_x}{\alpha_c + \varepsilon a_x m} \tag{7}$$

new infections. In this equation $m = \overline{N_b}/\overline{N_c}$.

Multiplying these last two terms, and adding up the contribution from the different infection routes, yields

$$\begin{aligned} R_0 = {} & \frac{\beta_1(1 - e^{-\mu_b \tau})}{\mu_b} + \frac{\beta_2 e^{-\mu_b \tau}}{\mu_b + \alpha_b} + \frac{\delta \kappa_1 a_x a_y m e^{-\mu_b \tau} \dfrac{\bar{X}_c}{\bar{N}_c}}{(\mu_b + \alpha_b)(\alpha_c + (\varepsilon a_x m))} \\ & + \frac{\delta \kappa_2 a_x a_h m (1 - e^{-\mu_b \tau}) \dfrac{\bar{X}_c}{\bar{N}_c}}{\mu_b (\alpha_c + (\varepsilon a_x m))}. \end{aligned} \tag{8}$$

The biological interpretation of R_0 has a clear meaning. Given a population without the disease one can ask if the introduction of a small number of infective individuals would trigger an epidemic, which eventually could converge to a steady state with the disease. The answer is, as usual, that if $R_0 > 1$, a small number of infective individuals will trigger an epidemic, which will settle to an endemic level different from zero. This will be formally shown in Appendix A.

It can also be seen from Eq. (8) that even if we vaccinate the total population of cattle ($\bar{X}_c \to 0$) rabies may still persist in the environment provided that the sum of the first two terms is greater than one.

If the two first terms of Eq. (8) sum to a number less than one and R_0 is greater than one, then the disease could be controlled by keeping the ratio between bat and cattle m below a certain threshold, m_{thre}, given by

$$m_{thre} = \frac{\alpha_c \left(1 - \dfrac{\beta_1(1 - e^{-\mu_b \tau})}{\mu_b} - \dfrac{\beta_2 e^{-\mu_b \tau}}{\mu_b + \alpha_b}\right)}{\Delta - \varepsilon a_x \left(1 - \dfrac{\beta_1(1 - e^{-\mu_b \tau})}{\mu_b} - \dfrac{\beta_2 e^{-\mu_b \tau}}{\mu_b + \alpha_b}\right)}, \tag{9}$$

where

$$\Delta = \frac{\Delta \kappa_1 a_x a_y e^{-\mu_b \tau} \dfrac{\bar{X}_c}{\bar{N}_c}}{(\mu_b + \alpha_b)} + \frac{\Delta \kappa_2 a_x a_h (1 - e^{-\mu_b \tau}) \dfrac{\bar{X}_c}{\bar{N}_c}}{\mu_b}. \tag{10}$$

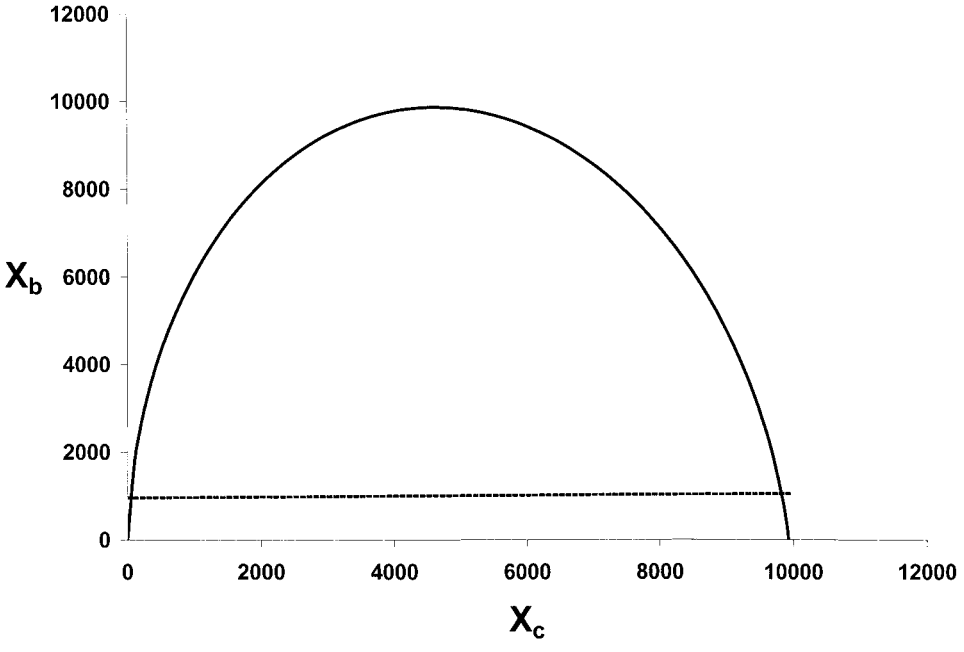

FIG. 2. Equilibrium isoclines in the absence of rabies. The two points at which the isoclines cross each other are the possible equilibrium but only the rightmost is stable.

Note that if the term

$$\left[1-\frac{\beta_1(1-e^{-\mu_b\tau})}{\mu_b}-\frac{\beta_2 e^{-\mu_b\tau}}{\mu_b+\alpha_b}\right]$$

is negative (which is indeed the case for the set of parameters we used), then R_0(bat-to-bat) > 1, which implies that rabies can be maintained in the bat population alone. On the other hand, if R_0(bat-to-bat) were smaller than one , then the terms

$$\frac{\delta\kappa_1 a_x a_y e^{-\mu_b\tau} m\,\frac{\bar{X}_c}{\bar{N}_c}}{(\mu_b+\alpha_b)(\alpha_c+(\varepsilon a_x m))}+\frac{\delta\kappa_2 a_x a_h(1-e^{-\mu_b\tau})\,m\,\frac{\bar{X}_c}{\bar{N}_c}}{\mu_b(\alpha_c+(\varepsilon a_x m))} \qquad (11)$$

should be great enough so that the total R_0 is greater than one. In this case, the threshold m_{thre} for infection, very similar to the one proposed by Macdonald (1952) for vector-borne infections, would be positive.

5. MODEL DYNAMICS

5.1. Equilibrium Analysis

Let us first analyze the model's behavior in the absence of disease. In the absence of disease the equilibrium points of Eqs. (1) can be deduced from Eqs. (3). Summing the two last equations of system (3) results in the following couple of isoclines

$$N_b=\frac{r_b-\mu_b}{r_b}K_b+\sigma_1\frac{r_b-\mu_b}{r_b}N_c$$

$$N_b=\frac{\left(-(\mu_c\sigma_2 N_c+\varepsilon a_x)+\sqrt{(\mu_c\sigma_2 N_c+\varepsilon a_x)^2+4\varepsilon a_x\sigma_2\left(r_c N_c\left(1-\frac{N_c}{K_c}\right)-\mu_c N_c\right)}\right)}{2\varepsilon a_x\sigma_2} \qquad (12)$$

graphically represented by Fig. 2.

As can be seen from Fig. 2 there are two equilibrium points but only the one on the right is stable as can be shown by linearizing Eqs. (1) in the absence of disease around those points. There is another stable equilibrium in which bats eradicate the cattle population. This is due to the choice of the functional response, and again, this has to do with the fact that with the functional response chosen the description at low cattle densities is not valid.

5.2. Model Simulations

In order to analyze the dynamics of the model we numerically simulated the complete system of six

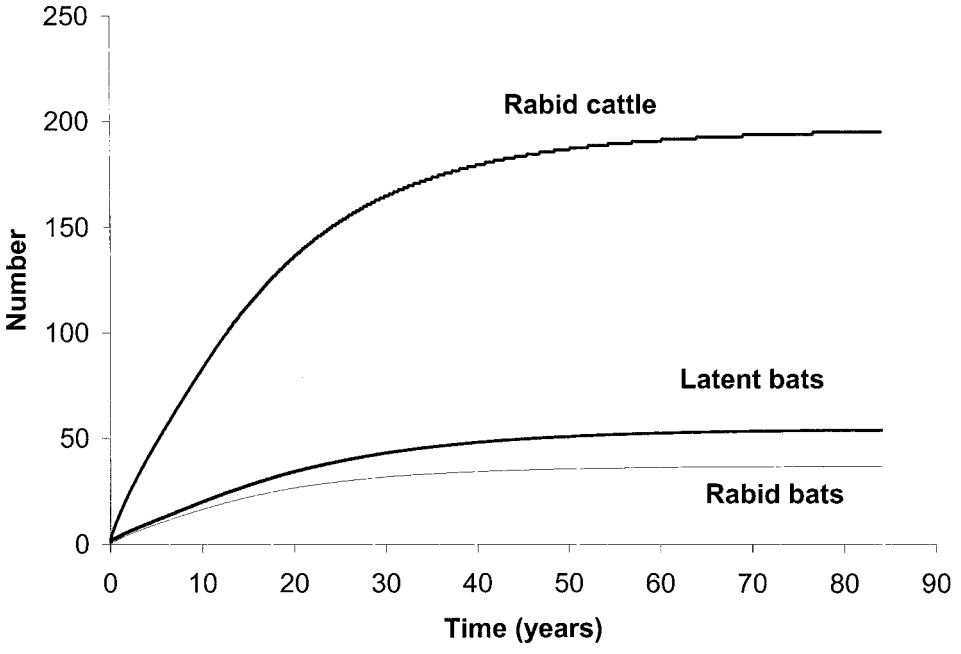

FIG. 3. Temporal dynamics of the model with the parameters as in Table I.

equations (1). The values of the parameters can be seen in Table I and the temporal dynamics of rabid animals are shown in Fig. 3.

As can be seen from Fig. 3, the disease evolves very slowly, as in other endemic diseases with low values of R_0, reaching the endemic equilibrium after several decades.

The equilibrium numbers of individuals in each of the model's compartments in the absence of control measures can be seen in Table II.

The results of the model simulations are in accordance with the current epidemiological beliefs on the disease.

We also calculated the numerical values of each of the components of the basic reproduction number, applying the same parameters used for the numerical simulation of the model. The results are shown in Table III.

TABLE II

Equilibrium Numbers of Individuals in Each of the Model's Compartments in the Absence of Control Measures

Variable	Symbol	Value
Susceptible bats	X_b^*	665
Latent bats	H_b^*	54
Rabid bats	Y_b^*	37
Susceptible cattle	X_c^*	6919
Rabid cattle	Y_c^*	195
Vaccinate cattle	Z_c^*	0
Total bat population	N_b^*	756
Total cattle population	N_c^*	7114

5.3. Sensitivity Analysis

In this subsection we investigate how sensitive the equilibrium values are to variation of the parameters. We do this by calculating changes in the equilibrium values

TABLE III

Numerical Values of Each of the Components of the Basic Reproduction Number

Component	Meaning	Value
$\frac{\beta_1(1-e^{-\mu_b\tau})}{\mu_b}$	Intraspecific bats–bats infective contact	0.482
$\frac{\beta_2 e^{-\mu_b\tau}}{\mu_b+\alpha_b}$	Intraspecific bats–bats infective contact	0.655
$\frac{\delta\kappa_1 a_x a_y m e^{-\mu_b\tau}\frac{\bar{X}_c}{\bar{N}_c}}{(\mu_b+\alpha_b)(\mu_c+\alpha_c+(\varepsilon a_x m))}$	Interspecific cattle–bats infective contact	0.001
$\frac{\delta\kappa_2 a_x a_h m(1-e^{-\mu_b\tau})\frac{\bar{X}_c}{\bar{N}_c}}{\mu_b(\mu_c+\alpha_c+(\varepsilon a_x m))}$	Interspecific cattle–bats infective contact	0.001
R_0	Total	1.14

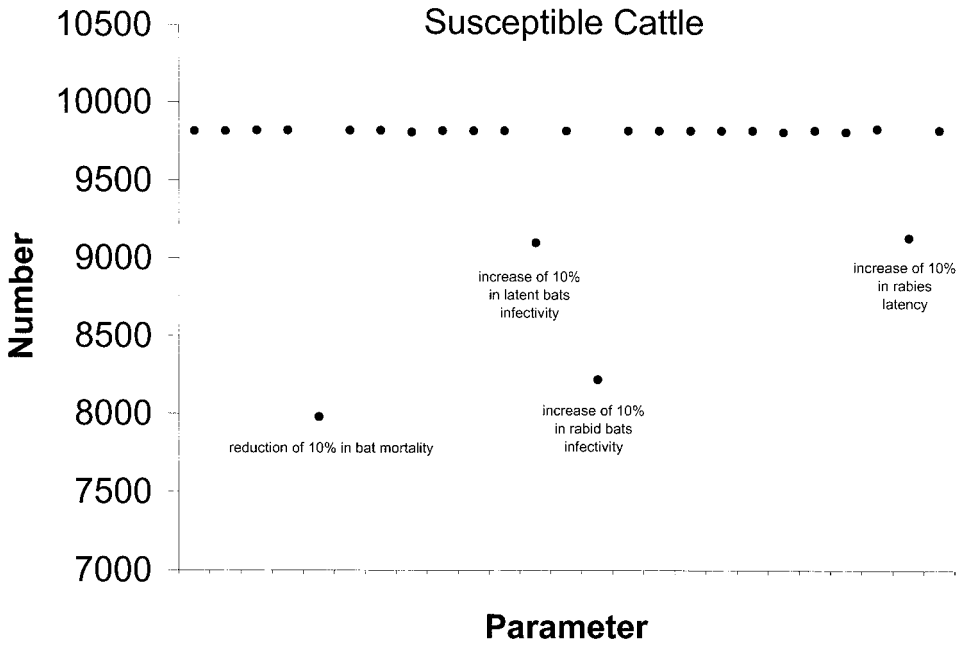

FIG. 4. Result of the sensitivity analysis. The figure shows the absolute number of susceptible cattle as a function of the model parameters. The first point to the left represents the cattle population at equilibrium given by the parameters as in Table I. The remaining points represent 10% positive and 10% negative variations of the parameters in the following order: a_h, α_b, α_a, a_x, a_y, β_1, β_2, κ_1, κ_2, σ_1, σ_2, and τ. Note that the absolute number of susceptible cattle is most sensitive to reduction in the bat mortality due to rabies, to an increase in the transmission of rabies from bat-to-bat, and to the increase in the rabies latency period among bats, that is, α_b, β_1, β_2, and τ, in this order. As mentioned in the text, the cattle population is also marginally sensitive to σ_1 and σ_2, respectively.

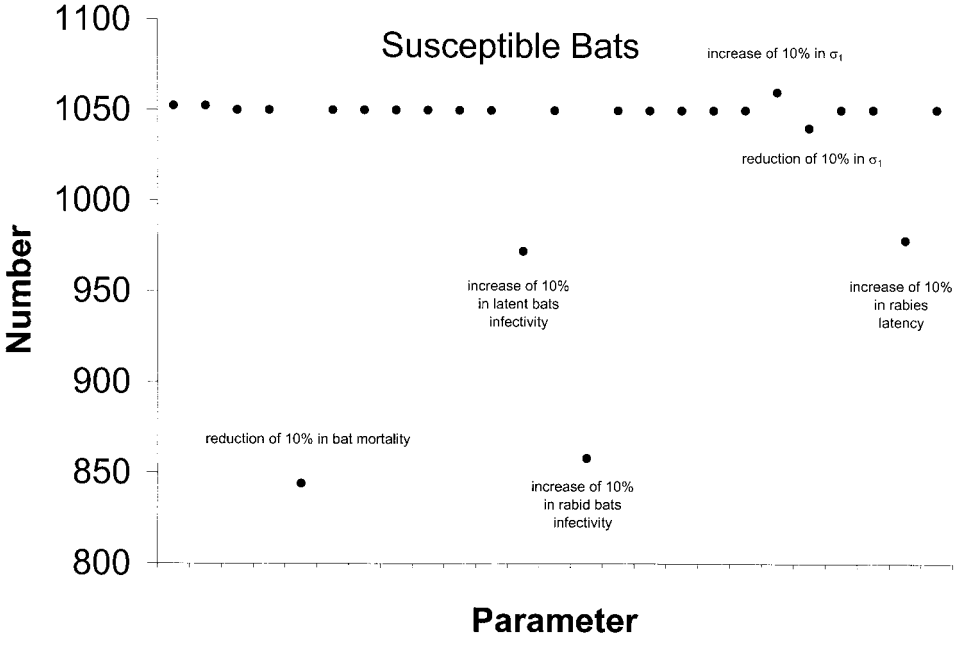

FIG. 5. Result of the sensitivity analysis. The figure shows the absolute number of susceptible bas as a function of the model parameters, as in Fig. 4. Note that, as in Fig. 4, the absolute number of susceptible bats is also most sensitive to reduction in the bat mortality due to rabies, to an increase in the transmission of rabies from bat-to-bat, and to the increase in the rabies latency period among bats, that is, α_b, β_1, β_2, and τ, in this order. Again, as mentioned in the text, the bat population is also sensitive to σ_1 and σ_2, respectively.

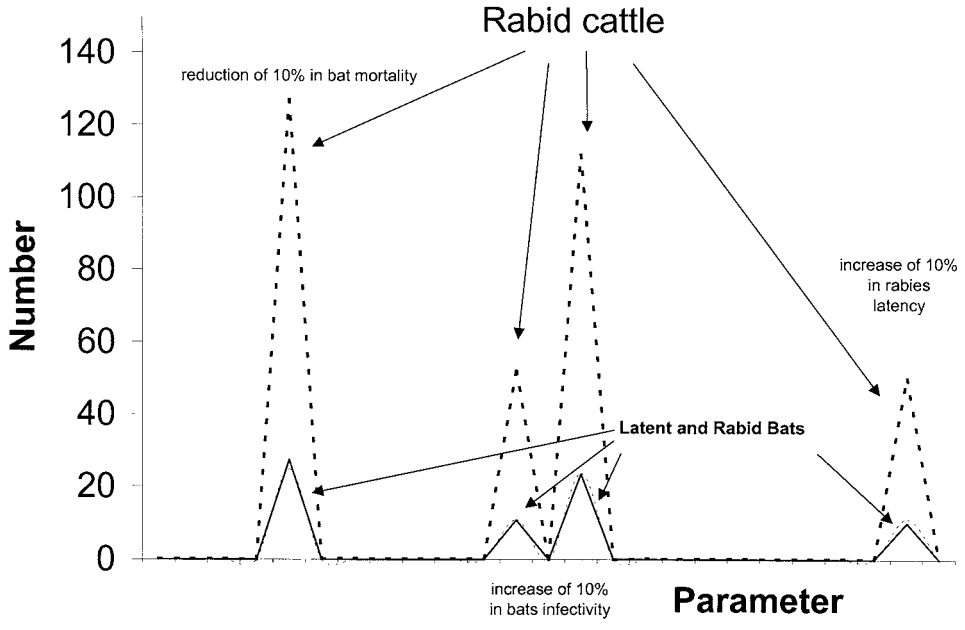

FIG. 6. Result of the sensitivity analysis for both cattle and bat populations. The figure shows the absolute number of rabies cases as a function of the model parameters, as in Fig. 4. This variable is also most sensitive to variations in the parameters α_b, β_1, β_2, and τ, in this order.

when we vary each parameter in turn while keeping the others fixed.

Figures 4 to 6 show the results of the sensitivity analysis, as related to the absolute number of susceptible cattle (Fig. 4), bats (Fig. 5), and rabid animals in both populations (Fig. 6).

As can be seen from the figures, there are four parameters to which all variables are significantly sensitive, namely α_b, β_1, β_2, and τ, in this order. It is noteworthy that all those parameters are primarily related to the intensity of the infection among bats. In addition, bat and cattle populations are also sensitive to σ_1.

Since we choose the parameters that reproduce the known equilibrium values, the result of the sensitivity analysis highlights those parameters which, for practical application of this model, should be carefully studied.

5.4. Analysis of Competing Control Strategies

We considered two types of intervention. The first is an increase in the natural bat mortality due to control measures, like the use of vampiricide gel. This variation is measured as a proportion q of the original value. In the simulations we took the original value of μ_b and, for each simulation , we used $(\mu_b + q\mu_b)$.

The second type of intervention is cattle vaccination. This type of intervention is measured by the proportion p of vaccinated cattle in equilibrium. In order to obtain the relation between the vaccination rate v and the proportion of vaccinated cattle in equilibrium, $p = Z_c/N_c$, we used the last equation of system (1). Making $dZ_c/dt = 0$ and dividing by N_c, we get

$$v = \frac{p\left[\mu_c + \gamma + \dfrac{\varepsilon(a_x X_b + a_h H_b + a_y Y_b)}{N_c}\right]}{(1-p) - \dfrac{Y_c}{N_c}}. \tag{13}$$

In the absence of disease, Eq. (13) reduces to

$$v = \frac{p[\mu_c + \gamma + \varepsilon a_x m]}{(1-p)}, \tag{14}$$

where $m = \overline{N_b}/\overline{N_c}$.

In order to examine the effectiveness of different control strategies, namely bat control and cattle vaccination, we simulated the model for several levels of vaccination coverage as compared to bat control measures. Figures 7 and 8 show the results of the numerical simulation for both possible intervention strategies. In Fig. 7 we see the comparative effect of each intervention expressed as averted deaths of cattle due to control. In Fig. 8 we see the same results expressed in terms of rabies prevalence in the cattle population.

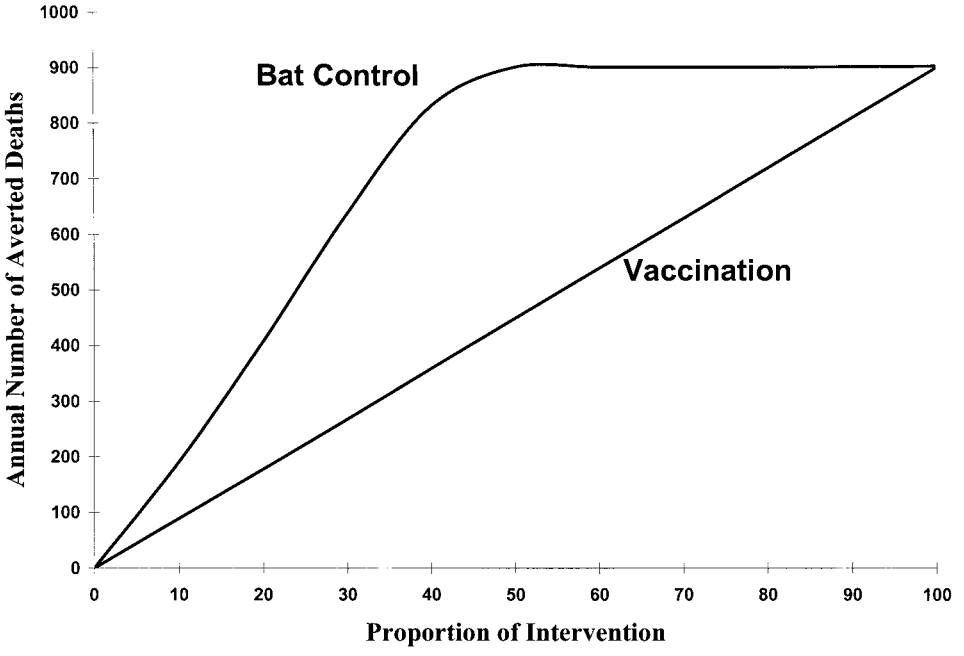

FIG. 7. The comparative effect of each intervention expressed in terms of averted deaths of cattle due to control. We can see that the effect of bat control is superior to cattle vaccination in terms of bringing back the number of cattle heads to pre-control levels.

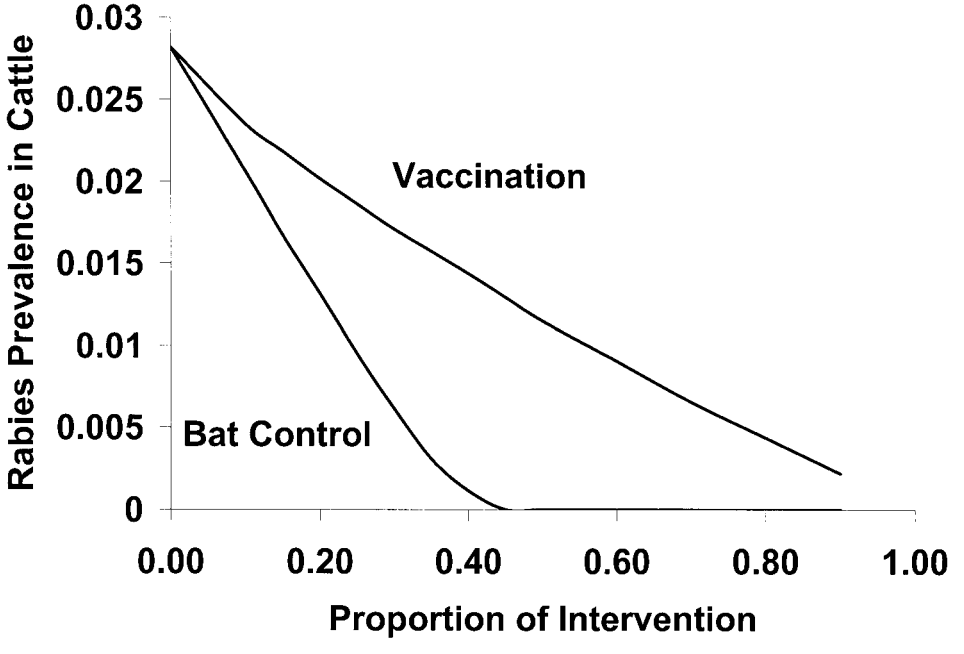

FIG. 8. The same results as in Fig. 6 now expressed in terms of rabies prevalence in the cattle population. Again it is also demonstrated that bat control is a more efficient control strategy than cattle vaccination.

It is noteworthy in Figs. 7 and 8 the striking differences in the effectiveness of the competing strategies with clear advantage for bat control measures. Note that this conclusion reflects only the sensitivity to variations in the parameters chosen. Therefore, one cannot say which method is better since there is no common measure of effort (see, however, the next section). It can also be seen from Fig. 7 and 8 that the response of the system to vaccination is linear, contrasting with the strong non-linear behavior of the response to bat control measures.

Preliminary cost analysis. In this section we present a rather preliminary analysis of the relative costs of each possible intervention.

According to Delpietro *et al.* (1992), each vampire treated with the vampiricide gel kills an average of 10 other bats. The unitary cost of this treatment is estimated in U.S. $ 24.00. Therefore, we can calculate the cost of increasing bat's mortality by 20%, that is, increasing μ_b from the original value of 0.083 to 0.099 year^{-1}. For this increase, under the conditions of the simulations, it would be enough to kill an average of 26 additional bats per year at a cost of U.S. $ 62.40/year. The calculation of the additional number of bats to be killed per year is given by

$$\frac{[\int_0^T 1.2\mu_b N_b'(t)\,dt - \int_0^T \mu_b N_b(t)\,dt]}{T}, \qquad (15)$$

where $N_b(t)$ and $N_b'(t)$ are the total number of bats when the mortality is μ_b and $1.2\mu_b$, respectively, and T is the duration of the intervention.

If we keep this control strategy for a period long enough to reduce cattle rabies prevalence by 50% of its original value, we would avert the death of an average of 408 animals/year. This was calculated according to

$$\frac{[\int_0^T \alpha_c Y_c(t)\,dt - \int_0^T \alpha_c Y_c'(t)\,dt]}{T}, \qquad (16)$$

where $Y_c(t)$ and $Y_c'(t)$ are the number of rabid cattle when the mortality of bats is μ_b and $1.2\mu_b$, respectively, and T is the duration of the intervention.

If we consider an average value of U.S. $ 600.00 per head, we obtain a profit of U.S. $ 3948.40 per dollar spent on killing bats.

On the other hand, vaccinating cattle represents a direct cost of U.S. $ 4.00/head/year. In order to obtain the same reduction in rabies prevalence it would be necessary to vaccinate an average of 535 animals per year, obtained by $\int_0^T vX_c(t)\,dt/T$. This implies, in our simulations, an average cost of U.S. $ 2140.00 per year. This strategy also results in averting the death of an average of 350 animals/year, calculated according to

$$\frac{[\int_0^T \alpha_c Y_c(t)\,dt - \int_0^T \alpha_c Y_c'(t)\,dt]}{T}, \qquad (17)$$

where $Y_c(t)$ and $Y_c'(t)$ are the number of rabid cattle in the absence and presence of vaccination, respectively.

Therefore, in the case of vaccination, the strategy results in an average profit of U.S. $ 98.13 per dollar spent on vaccination.

The above result should be taken with some care as it applies to a simulation where rabies is highly incident.

In conclusion, the above cost analysis supports Delpietro *et al.*'s recommendation of not vaccinating cattle but rather killing the bats. It is not only a more efficient strategy in reducing rabies prevalence but it is also more cost-effective.

6. DISCUSSION

Bat-transmitted rabies is an infection with very peculiar characteristics which make it almost unique among the so-called "vector-borne" infections. If, from the one hand, bats can be considered vectors of the rabies virus , and on the other hand, contrasting with the classical paradigms of vector-borne infections like malaria, the vectors do not predominantly acquire the infection from the host but from other vectors. This epizootic setting was also observed for a few other infections, like the recent discovery of Mead *et al.* (2000) on the transmission of vesicular stomatitis virus, an arbovirus of great economic importance of livestock. Affected animals do not have detectable viraemia but, in spite of this, the susceptible black flies (the vector) acquire the infection from infected flies when co-feeding in the same animal host. This was also observed in some tick-borne viruses (Mead *et al.*, 2000).

The basic reproduction number of our model shares an interesting characteristic with other epidemic models which involve more than two populations, namely, it is a sum of the components of each transmission leg. So, for example, in some of the models of schistosomiasis described so far (Macdonald) 1965; May, 1977; Hyun *et al.*, 1997), the value of R_0 is the result of the sum of the two transmission legs, from man to snail and from snail to man, T_{hs} and T_{sh}, respectively. In our recent leishmaniasis model (Burattini *et al.*, 1999), the value of R_0 is obtained by summing four transmission components, namely, the individual contributions of latent and infected dogs and humans. For these models, even in the

case in which each individual component of R_0 is less than one the infection can prevail provided that their sum is greater than one.

We suspect, although we have not proved it yet, that this phenomenon can be generalized for all infections whose dynamics involve more than two populations.

Worth mentioning the fact that for the set of parameters chosen, there is no positive density of bats as related to cattle, m_{thre}, below which rabies would get eradicated. This is due to the fact that for that set of parameters the contribution of the cattle population for the circulation of rabies virus among the involved species is negligible, as can be concluded from the extremely low values of the third and fourth terms of R_0, as shown in Table III, which reflect the extremely low and short-living viraemia of rabies virus.

Note that none of the control measures proposed (cattle vaccination and killing of bats) can control the disease of $R_0(\text{bat-to-bat}) > 1$. If bat vaccination would be feasible then control, and perhaps eradication, of the disease would be possible. Actually, bat vaccination would be effective for essentially two types of vaccine: a live attenuated virus (which is infective but does not cause rabies) and a recombinant, oral vaccine which could be applied in the same gel currently used with the vampiricide substance. We are carrying out a phase I study of the last type of vaccine in our laboratory.

The analysis of the relative impact of two competing control strategies presented in Section 5.4 was intended as a theoretical exercise of the potential of our model in providing solutions to questions of intervention policies. Its results are, obviously, very much dependent on the value of the set of parameters applied in the numerical simulations. However, the parameters we chose were the ones which resulted in equilibrium densities of each variable that are in accordance with current epidemiological beliefs. We think that our model is consistent with the known reality of bat- transmitted rabies and it was proposed as a theoretical framework upon which practical solutions for the problem of cattle rabies could be tested, as the exercise presented in Section 5.4 demonstrated.

A more accurate cost-effectiveness analysis of possible control strategies could be carried out with the model presented here and we intend to refine this preliminary analysis in a future publication.

APPENDIX A

In this appendix we show that if the basic reproduction number, R_0, given by Eq. (8) is less than one, then rabies cannot invade the bat–cattle population. This is done by calculating the relationship between the parameters that cause instability of the trivial solution of the system of equations (1) that represents the absence of disease in the populations considered.

We begin by linearizing the system (1) around the solution in the absence of disease, namely,

$$\begin{aligned} X_b &= \bar{N}_b + x_b^* \\ H_b &= 0 + h_b^* \\ Y_b &= 0 + y_b^* \\ X_c &= \bar{X}_c + x_c^* \\ Y_c &= 0 + y_c^* \\ Z_c &= \bar{Z}_c + z_c^*, \end{aligned} \tag{18}$$

where $\bar{N}_b$, $\bar{X}_c$, and $\bar{Z}_c$ are solutions of the equations

$$\begin{aligned} -\mu_b \bar{N}_b + r_b \bar{N}_b \left(1 - \frac{\bar{N}_b}{K_b + \sigma_1(\bar{X}_c + \bar{Z}_c)}\right) &= 0 \\ -\nu \bar{X}_c - \varepsilon a_x \bar{N}_b \frac{X_c}{N_c} - \mu_c \bar{X}_c + \gamma \bar{Z}_c + \frac{r_c}{1 + \sigma_2 N_b} \bar{N}_c \left(1 - \frac{\bar{N}_c}{K_c}\right) &= 0 \\ \nu \bar{X}_c - \gamma \bar{Z}_c + \varepsilon a_x \bar{N}_b \frac{Z_c}{N_c} - \mu_c \bar{Z}_c &= 0, \end{aligned} \tag{19}$$

where $\bar{N}_c = \bar{X}_c + \bar{Z}_c$.

Therefore, the linearized model takes the form

$$\begin{aligned} \frac{d}{dt} x_b^* &= -\beta_1 h_b^* - \beta_2 y_b^* - \delta a_x m y_c^* \\ &\quad + r_b n_b^* - r_b \frac{\bar{N}_b}{K_b + \sigma_1 \bar{N}_c} n_b^* + \frac{r_b \sigma_1 \bar{N}_b^2}{(K_b + \sigma_1 \bar{N}_c)^2} n_c^* \\ \frac{d}{dt} h_b^* &= \beta_1 h_b^* + \beta_2 y_b^* + \delta a_x m y_c^* - e^{-\mu_b \tau} \beta_1 h_b^*(t-\tau) \\ &\quad - e^{-\mu_b \tau} \beta_2 y_b^*(t-\tau) \\ &\quad - e^{-\mu_b \tau} \delta a_x m y_c^*(t-\tau) - \mu_b h_b^* \\ \frac{d}{dt} y_b^* &= e^{-\mu_b \tau} \beta_1 h_b^*(t-\tau) + e^{-\mu_b \tau} \beta_2 y_b^*(t-\tau) \\ &\quad + e^{-\mu_b \tau} \delta a_x m y_c^*(t-\tau) - (\alpha_b + \mu_b) y_b^* \end{aligned}$$

$$\begin{aligned}
\frac{d}{dt}x_c^* &= -\nu x_c^* - (\kappa_1+\varepsilon)\,a_y\frac{\bar X_c}{\bar N_c}y_b^* - (\kappa_2+\varepsilon)\,a_h\frac{\bar X_c}{\bar N_c}h_b^* \\
&\quad +\gamma z_c^* - \mu_c x_c^* - \varepsilon a_x\frac{\bar X_c}{\bar N_c}x_b^* \\
&\quad +\frac{1}{1+\sigma_2\bar N_b}\left[\frac{r_c n_b^*}{(1+\sigma_2\bar N_b)}\left(\frac{\bar N_c^2}{K_c}-\sigma_2\bar N_c\right)\right. \\
&\quad \left. +r_c n_b^*\left(1-2\frac{\bar N_c}{K_c}\right)\right] \\
\frac{d}{dt}y_c^* &= \kappa_1 a_y\frac{\bar X_c}{\bar N_c}y_b^* + \kappa_2 a_h\frac{\bar X_c}{\bar N_c}h_b^* - \varepsilon a_x m y_c^* - \alpha_c y_c^* \\
\frac{d}{dt}z_c^* &= \nu x_c^* - (\gamma+\mu_c)\,z_c^* - \varepsilon a_x m z_c^*,
\end{aligned} \tag{20}$$

where m is the ratio between the bat and cattle populations at equilibrium before the introduction of the disease, $n_b^* = x_b^* + h_b^* + y_b^*$ and $n_c^* = x_c^* + y_c^* + z_c^*$.

In the system above the equations for h_b^*, y_b^*, and y_c^* are uncoupled from the others and so we get the following reduced system:

$$\begin{aligned}
\frac{d}{dt}h_b^* &= \beta_1 h_b^* + \beta_2 y_b^* + \delta a_x m y_c^* - e^{-\mu_b\tau}\beta_1 h_b^*(t-\tau) \\
&\quad -e^{-\mu_b\tau}\beta_2 y_b^*(t-\tau) \\
&\quad -e^{-\mu_b\tau}\delta a_x m y_c^*(t-\tau) - \mu_b h_b^* \\
\frac{d}{dt}y_b^* &= e^{-\mu_b\tau}\beta_1 h_b^*(t-\tau) + e^{-\mu_b\tau}\beta_2 y_b^*(t-\tau) \\
&\quad +e^{-\mu_b\tau}\delta a_x m y_c^*(t-\tau) - (\alpha_b+\mu_b)\,y_b^* \\
\frac{d}{dt}y_c^* &= \kappa_1 a_y y_b^*\frac{\bar X_c}{\bar N_c} + \kappa_2 a_h h_b^*\frac{\bar X_c}{\bar N_c} - (\alpha_c+\varepsilon a_x m)\,y_c^*.
\end{aligned} \tag{21}$$

Let us assume, as in Burattini *et al.* (1999), that $h_b^* = h_b e^{\lambda t}$, $y_b^* = y_b e^{\lambda t}$, $y_c^* = y_c e^{\lambda t}$ to get the characteristic equation for λ

$$\begin{vmatrix}
-\lambda+\beta_1(1-e^{(\lambda-\mu_b)\tau})-\mu_b & \beta_2(1-e^{(\lambda-\mu_b)\tau}) & \delta a_x m(1-e^{(\lambda-\mu_b)\tau}) \\
\beta_1 e^{(\lambda-\mu_b)\tau} & -\lambda+\beta_2 e^{(\lambda-\mu_b)\tau} - (\mu_b+\alpha_b) & \delta a_x m e^{(\lambda-\mu_b)\tau} \\
\kappa_2 a_h\dfrac{\bar X_c}{\bar N_c} & \kappa_1 a_y\dfrac{\bar X_c}{\bar N_c} & -\lambda-(\alpha_c+\varepsilon a_x m)
\end{vmatrix} = 0. \tag{22}$$

If all the solutions of λ in the above equation have negative real parts, the system is asymptotically stable. On the other hand, stability is broken when at least one of the roots has a positive real part. This is a standard result demonstrated by El'sgol'ts (1966) for systems with delay and it is also true for systems without delay (see May (1973) for the mathematical basis and Massad *et al.* (1994) for the epidemiological application).

Using the same methods applied by Burattini *et al.* (1999) and by Lopez *et al.* (1999), it can be shown that the first root to cross the imaginary axis is real and this happens when the parameters are such that

$$\begin{aligned}
&\frac{\beta_1(1-e^{-\mu_b\tau})}{\mu_b} + \frac{\beta_2 e^{-\mu_b\tau}}{\mu_b+\alpha_b} + \frac{\delta\kappa_1 a_x a_y m e^{-\mu_b\tau}\dfrac{\bar X_c}{\bar N_c}}{(\mu_b+\alpha_b)(\alpha_c+(\varepsilon a_x m))} \\
&\quad + \frac{\delta\kappa_2 a_x a_h m(1-e^{-\mu_b\tau})\dfrac{\bar X_c}{\bar N_c}}{\mu_b(\alpha_c+(\varepsilon a_x m))} = 1.
\end{aligned} \tag{23}$$

The left-hand term of the above equation is identical to the expression of R_0.

APPENDIX B

In this appendix we outline the main formulae for the case when we consider that density dependence acts in both births and deaths.

Let $\mu_b(N_b)$ be the bats' mortality rate, which is an increasing function with N_b. Let $b_b(N_b)$ be the bats' birth rate, which is a decreasing function of N_b. Similarly, let $\mu_c(N_c)$ and $b_c(N_c)$ be the mortality and birth rates for cattle.

The system of equations (1) now takes the form

$$\begin{aligned}
\frac{d}{dt}X_b(t) &= -\beta_1 X_b\frac{H_b}{N_b} - \beta_2 X_b\frac{Y_b}{N_c} + b_b(N_b)\,N_b \\
&\quad -\mu_b(N_b)\,X_b \\
\frac{d}{dt}H_b(t) &= \beta_1 X_b\frac{H_b}{N_b} + \beta_2 X_b\frac{Y_b}{N_b} + \delta a_x X_b\frac{Y_c}{N_c} \\
&\quad -\beta_1 X_b(t-\tau)\,e^{-\mu_b\tau}\frac{H_b(t-\tau)}{N_b(t-\tau)} \\
&\quad -\beta_2 X_b(t-\tau)\,e^{-\mu_b\tau}\frac{Y_b(t-\tau)}{N_b(t-\tau)} \\
&\quad -\delta a_x X_b(t-\tau)\,e^{-\mu_b\tau}\frac{Y_c(t-\tau)}{N_c(t-\tau)} - \mu_b(N_b)\,H_b
\end{aligned}$$

$$\begin{aligned}
\frac{d}{dt}Y_b(t) &= \beta_1 X_b(t-\tau)\, e^{-\mu_b \tau}\frac{Hob(t-\tau)}{Nob(t-\tau)} \\
&\quad + \beta_2 X_b(t-\tau)\, e^{-\mu_b \tau}\frac{Y_b(t-\tau)}{N_b(t-\tau)} \\
&\quad + \delta a_x X_b(t-\tau)\, e^{-\mu_b \tau}\frac{Y_c(t-\tau)}{N_c(t-\tau)} \\
&\quad - (\mu_b(N_b)+\alpha_b)\, Y_b \\
\frac{d}{dt}X_c(t) &= -\nu X_c - (\kappa_1+\varepsilon)\, a_y Y_b \frac{X_c}{N_c} - (\kappa_2+\varepsilon)\, a_h H_b \frac{X_c}{N_c} \\
&\quad - \mu_c(N_c)\, X_c - \varepsilon a_x X_b \frac{X_c}{N_c} \\
&\quad + b_c(N_c)\, N_c + \gamma Z_c \\
\frac{d}{dt}Y_c(t) &= \kappa_1 a_y Y_b \frac{X_c}{N_c} + \kappa_2 a_h H_b \frac{X_c}{N_c} - \alpha_c Y_c \\
&\quad - \varepsilon(a_x X_b + a_h H_b + a_y Y_b)\frac{Y_c}{N_c} \\
\frac{d}{dt}Z_c(t) &= \nu X_c - \gamma Z_c - \mu_c(N_c)\, Z_c \\
&\quad - \varepsilon(a_x X_b + a_h H_b + a_y Y_b)\frac{Z_c}{N_c}.
\end{aligned} \tag{24}$$

The population in the absence of disease $\overline{N_c}$, $\overline{X_c}$, and $\overline{Z_c}$ now obey

$$\begin{aligned}
-\mu_b(\overline{N_b}) + b_b(\overline{N_b}) &= 0 \\
-\nu\bar{X}_c - \varepsilon a_x \bar{N}_b \frac{X_c}{N_c} - \mu_c(\overline{N_c})\,\bar{X}_c + \gamma\bar{Z}_c + b_c(\overline{N_c})\,\bar{N}_c &= 0 \\
\nu\bar{X}_c - \gamma\bar{Z}_c - \varepsilon a_x \bar{N}_b \frac{Z_c}{N_c} - \mu_c(\overline{N_c})\,\bar{Z}_c &= 0,
\end{aligned} \tag{25}$$

where $\overline{N_c} = \bar{X}_c + \bar{Z}_c$.

The basic reproduction number is now given by

$$\begin{aligned}
R_0 &= \frac{\beta_1(1-e^{-\mu_b\tau})}{\mu_b(\overline{N_b})} + \frac{\beta_2 e^{-\mu_b\tau}}{\mu_b(\overline{N_b})+\alpha_b} \\
&\quad + \frac{\delta\kappa_1 a_x a_y m e^{-\mu_b\tau}\dfrac{\bar{X}_c}{\bar{N}_c}}{(\mu_b(\overline{N_b})+\alpha_b)(\alpha_c+(\varepsilon a_x m))} \\
&\quad + \frac{\delta\kappa_2 a_x a_h m(1-e^{-\mu_b\tau})\dfrac{\bar{X}_c}{\bar{N}_c}}{\mu_b(\overline{N_b})(\alpha_c+(\varepsilon a_x m))}.
\end{aligned} \tag{26}$$

The remaining formulas in Section 5.4 are trivially derived.

ACKNOWLEDGMENTS

We thank the comments and suggestions of the editor and three anonymous reviewers.

REFERENCES

Anderson, R. M., and May, R. M. 1991. "Infectious Diseases of Humans: Dynamics and Control," Oxford Univ. Press, Oxford.

Arellano-Sota, C. 1988. Vampire bat-transmitted rabies in cattle, *Rev. Infect. Dis.* **10**(Suppl. 4), S707.

Beran, G. W. 1981. Rabies and infections by rabies-related viruses, *in* "CRC Handbook Series in Zoonoses" (G. W. Beran, Ed.), Vol. II, CRC Press, Boca Raton, FL.

Brass, D. A. 1994. "Rabies in Bats: Natural History and Public Health Implications," Livia Press, Ridgefield.

Burattini, M. N., Coutinho, F. A. B., Lopez, L. F., and Massad, E. 1999. Modelling the dynamics of leishmaniasis considering human, animal host and vector populations, *J. Biol. Syst.* **6**(4), 337–356.

Constantine, D. G. 1988. Transmission of pathogenic microorganisms by vampire bats, *in* "Natural History of Vampire Bats" (A. M. Greenhall and U. Schmidt, Eds.), pp. 167–189, CRC Press, Boca Raton, FL.

Darling, K. 1997. "Komodo Dragon," Lothrop, Lee & Shepard Books, New York.

Delpietro, H. A., Marchevsky, N., and Simonetti, E. 1992. Relative population densities and predation of the common vampire bats (*Desmodus rotundus*) in natural and cattle-raising areas of north-east Argentina, *Prev. Vet. Med.* **14**, 13–20.

El'sgol'ts, E. L. 1966. "Introduction to the Theory of Differential Equations with Deviating Arguments," Holden–Day, San Francisco.

Fenton, M. B. 1992. "Bats," Facts on File, New York.

Goodwin, G. C., and Greenhal, A. M. 1961. A review of the bats of Trinidad and Tobago, *Bull. Am. Mus. Nat. Hist.* **122**, 187–203.

Greenhall, A. M. 1970. Vampire bat control: A review and proposed programme for Latin America, *in* "Proceedings of the Fourth Vertebrate Pest Conference" (R. H. Diana, Ed.) California Vertebrate Pest Commitee, West Sacramento, CA.

Harinasuta, T., and Bunnag, D. 1990. Liver, lung and intestinal trematodiasis, *in* "Tropical and Geographical Medicine," (K. S. Warren and A. A. F. Mahmoud, Eds.), Chap. 55, MCGraw–Hill, New York.

Johnson, R. T. 1982. "Viral Infections of the Nervous System," Raven Press, New York.

Lomnicki, A. 1988. "Population Ecology of Individuals," Princeton Univ. Press, Princeton, NJ.

Lopez, L. F., Coutinho, F. A. B., Burattini, M. N. and Massad, E. 1999. Modelling the spread of infections when the contact rate among individuals is short ranged: Propagation of epidemic waves, *Math. Comput. Model* **29**(7), 55–69.

Macdonald, G. 1952. The analysis of equilibrium in malaria, *Trop. Dis. Bull.* **49**, 813–828.

Macdonald, G. 1965. The dynamics of helmith infections, with special reference to schistosomes, *Trans. R. Soc. Trop. Med. Hyg.* **59**, 489–506.

Massad, E. 1987. Transmission rates and the evolution of pathogenicity, *Evolution* **41**(5), 1127–1130.

Massad, E., Coutinho, F. A. B., Yang, H. M., Carvalho, H. B., Mesquita, F. and Burattini, M. N. 1994. The basic reproduction ration of HIV among intravenous drug users, *Math. Biosci.* **123**, 227–247.

May, R. M. 1973. "Stability and Complexity in Model Ecosystems," Princeton Univ. Press, Princeton, NJ.

May, R. M. 1977. Togetherness among schistosomes: Its effects on the dynamics of the infection, *Math. Biosci.* **35**, 301–343.

Mayr, E. 1970. "Populations, Species and Evolution," Harvard Univ. Press, Cambridge, MA.

Mead, D. G., Ramberg, F. B., Besselsen, D. G., and Maré, C. J. 2000. Transmission of vesicular stomatitis virus from infected to noninfected black flies co-feeding on nonviremic deer mice, *Science* **287**, 485–487.

Pianka, E. R. 1983. "Evolutionary Ecology," 3rd ed., Harper & Row, New York.

Pulliam, H. R. 1989. Individual behaviour and the procurement of essential resources, *in* "Perspectives in Ecological Theory" (J. Roughgarden, R. M. May, and S. A. Levin, Eds.), Chap. 2, pp. 25–38, Princeton Univ. Press, Princeton, NJ.

Roughgarden, J. 1979. "Theory of Population Genetics and Evolutionary Ecology: An Introduction," Macmillan Co., New York.

Steele, J. H. 1973. The epidemiology and control of rabies, *Scand. J. Infect. Dis.* **5**, 312–316.

Thompson, R. D., Elias, D. J., and Mitchell, G. C. 1977. Effects of bat control on bovine milk production, *J. Wildlife Manage.* **41**(4), 736–739.

Yang, H. M., Coutinho, F. A. B., and Massad, E. 1997. Acquired immunity on a schistosomiasis transmission model—Fitting the data, *J. Theor. Biol.* **188**(4), 495–506.

World Health Organization (WHO) 1957. Expert Committee on Rabies, Third Report, WHO Technical Report No. 121, Geneva.

World Health Organization (WHO) 1966. Expert Committee on Rabies, Fifth Report, WHO Technical Report No. 321, Geneva.

World Health Organization (WHO) 1973. Expert Committee on Rabies, Sixth Report, WHO Technical Report No. 523, Geneva.

World Health Organization (WHO) 1984. Expert Committee on Rabies, Seventh Report, WHO Technical Report No. 709, Geneva.