A Simple Model for the Spatial Spread and Control of Rabies

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A simple mathematical model for the spatial spread of rabies is presented. It models the dynamics of the front of an epizootic wave. We show how the model can be used to estimate the minimum width (in kilometers) of a break, that is, a region in which a control scheme is employed in order to stop the spatial progression of the rabies wave front. A simple expression is derived for the surviving fox population, after the passage of the epizootic, in terms of measurable parameters of the model.

1. Introduction

The rabies epizootic now crossing continental Europe has persisted and spread for more than 40 years. Starting on the edge of the German/Polish border the front of the epizootic has moved westward at an average speed of about 30–60 km a year. Here we discuss a very primitive model based on the assumption that the migration of rabid foxes determines the dynamics of the epizootic front. Quantitative conditions under which the wave propagates are determined and a formula for its speed in terms of relevant parameters is obtained. We use the model to discuss how to determine the width of a barrier which would prevent the epizootic from spreading to a rabies free area.

The reason we present such a simple model for such a complex problem is that it poses relevant questions that more realistic models must also address. There is, of course, in any modelling a trade-off between complexity and the difficulty of estimating many parameters and a simpler one where values can be reasonably assessed. In this paper, we have opted for the latter strategy.

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Although other species are involved, foxes are the main carrier of rabies in the present European epizootic, so we assume that the spread of the epizootic is essentially determined by the ecology of fox populations. If the fox population density is estimated at different times as the rabies epizootic passes by, as in Fig. 1, the wave is seen to consist of two main parts; the front through which the population is rapidly decreasing in magnitude and the much longer tail where there are essentially periodic outbreaks of the disease. The model here mainly addresses the front of the wave. In section 2 we describe the model while in section 3 we describe a control measure in the form of a partial break, and in section 4 we estimate values for the epidemiological parameters. We consider the periodic behaviour of the tail (endemic situation) in section 5 where we discuss some generalizations and other models, such as the model of Anderson et al. (1981), who consider the tail part of the wave (with no spatial effects). In section 6 we consider the implicit assumptions involved, and in section 7 we make some general conclusions.

2. The Model

We use a deterministic approach and divide the fox population into two groups, infective and susceptible foxes; the former consists of rabid foxes and those in the incubation stage. The basic facts (see, for example, the books by Macdonald (1980) and Kaplan (1977)) and assumptions of our model are:
(i) The rabies virus is contained in the saliva of the rabid fox and is normally transmitted by bite. Therefore a contact between a rabid and a susceptible fox is necessary for the transmission of the disease.

(ii) Rabies is invariably fatal in foxes.

(iii) Foxes are territorial and seem to divide the countryside into non-overlapping home ranges which are marked out by scent.

(iv) The rabies virus enters the central nervous system and induces behavioural changes in its host. If the spinal cord is involved it often takes the form of paralysis. However, if it enters the limbic system the foxes become aggressive, lose their sense of direction and territorial behaviour, and wander about in a more or less random way.

These assumptions result in a model similar to one by Noble (1974) for the spread of the Black Death in the 14th century. Our model, like that of Noble, is an extension of the classical Kermack-McKendric (1927) model in epidemiology to include spatial effects. Another such extension is due to Kendall (1965). For a review of mathematical models in epidemiology, see, for example, the book edited by Anderson (1982), the review article by Becker (1979), or the book by Bailey (1975).

In the model, the change with time of the number of infective foxes within a small area (like a territory) is equal to the rate of transitions from the susceptible population minus the mortality rate and the migration from the area. In the short time interval we are interested in (~1 year), the temporal variation in the susceptible population is simply the rate of loss to the infective population. Assuming that the dispersal of the infective population can be reasonably approximated by random walks (from assumption (iv)) our model equations are

\[
\frac{\partial S}{\partial t} = -KIS \\
\frac{\partial I}{\partial t} = D\frac{\partial^2 I}{\partial x^2} + KIS - \mu I. \tag{1}
\]

Here \(S\) and \(I\) are the susceptible and infective population densities respectively and for simplicity only we consider the one-dimensional (x) spatial problem. The term \(KIS\) comes from (i): \(KS\) is the number of new infectives produced per unit time per infective and the constant \(K\) is the transmission coefficient. Note that only a fraction of the infective foxes, that is the rabid ones, can transmit the disease. The term \(-\mu I\) comes from (ii) with the additional assumption that the probability for an infective fox to survive for at least time \(T\) is given by \(e^{-\mu T}\), so that \(1/\mu\) is the life expectancy of an infective fox. The absence of a migration term in the equation for the susceptibles is motivated by (iii): susceptible foxes do occasionally travel considerable distances but they always return home (with the exception of young foxes leaving their home territory in search of territories of their
own, an event briefly considered below). The diffusion term comes from (iv) and represents the random motion of rabid foxes averaged out over the whole infective population: the diffusion coefficient $D$ can be estimated by

$$D = kA$$

where $k$ is the rate at which infective foxes leave their territories which have average area $A$, that is, $1/k$ is the average time until a fox leaves its territory. More accurate estimates of $D$ would be obtained by field observations of net distances travelled by infectives during observation periods.

Several other assumptions in the model are important. A static population of susceptibles is assumed in the sense that deaths are equally balanced by births. This is unrealistic on a long time scale but for the short period of about a year, with which the model is concerned, is perhaps not unrealistic. We are primarily interested in the rate of propagation of the epizootic. We show below that this depends on the susceptible density ahead of the outbreak, where deaths and births balance since the fox population is assumed to be in equilibrium with the carrying capacity of its environment. The extension of the model to include susceptible births is briefly considered in section 5. We also neglect all routes of infection other than binary contacts between rabid and susceptible foxes.

In leaving out a migration term in the equation for susceptibles we are also neglecting the fact that male foxes may leave their territory in the rutting season looking for a mate, and that juvenile foxes leave their home territory in the autumn travelling distances that typically may be 10 times a territory size in search of a new territory. If a fox happened to have contracted rabies around the time of such long-distance movement, it could certainly increase the rate of spreading of the disease into uninfected areas. However, such events represent a very small fraction of the overall life history of the fox, implying the effective rate of diffusion of susceptibles is very small compared to the dynamics incorporated in the simple model equations (1).

We also assume that space is completely homogeneous so that there are no preferred directions to move in. Some of these effects could easily be incorporated in the model. They have been excluded so as to isolate with simplicity what we believe to be some key features.

To obtain a qualitative understanding of the system (1) in terms of the model parameters we introduce the non-dimensional quantities

$$u = I/S_0, \quad v = S/S_0,$$

$$x^* = (KS_0/D)^{1/2} x, \quad t^* = KS_0 t,$$

$$r = \mu/KS_0$$

(3)
where $S_0$ is the initial (maximum) susceptible density. Dropping the asterisks for convenience, the model equations become

$$\frac{\partial u}{\partial t} = \frac{\partial^2 u}{\partial x^2} + u(v - r)$$

$$\frac{\partial v}{\partial t} = -uv$$

(4)

where $u \geq 0$, $0 < v \leq 1$. Note that the qualitative behaviour of the system depends on only one dimensionless parameter, $r$. Its inverse, $R = 1/r$, is equivalent to the basic reproduction rate of the disease in the initial stages of an outbreak of the disease (see section 7 or Dietz 1974, 1976, or Anderson & May, 1982a, b). If $r > 1$ the infection dies out quickly, which is to be expected since this is equivalent to $\mu > KS_0$, that is, the mortality rate is greater than the rate of recruitment of new infectives. We can also formulate this result by saying that, given $K$ and $\mu$, there is a typical critical minimum fox density $S_c$ below which rabies cannot persist, given by

$$S_c = \mu / K = rS_0.$$  (5)

This is important from the point of view of developing ecological strategies to stop the spread of the disease. A similar threshold result appears in the classical Kermack-McKendric model for infectious diseases. As discussed in section 7, Mollison (1983) has shown that this result is common to a large class of epidemiological models which use multiplicative terms to represent interactions between populations.

Some elementary mathematical analysis of the model equations (4) is given in the Appendix. For a detailed analysis of these equations, including a proof for the existence of travelling waves and conditions for the waves to travel at the minimum speed, see Källén (1984). In this paper the qualitative features of the system are obtained in a straightforward manner from a first integral of (4) followed by standard linearization techniques. We find that if $r < 1$ and the initial distribution of susceptibles is uniformly equal to 1 (that is, $S = S_0$ everywhere), then in the one-dimensional case a small localized introduction of rabies evolves into two travelling waves going in opposite directions. Figure 2 gives the shape of the wave moving to the right for $r = 0.5$. We show in the Appendix that the proportion $a$ of susceptible foxes which remain after the infective wave has passed satisfies $0 < a < r < 1$, and is given in terms of $r$ by

$$a - r \log a = 1.$$   (6)

Figure 3 shows this relationship between $a$ and $r$. The waveforms for the two species travel together at the same speed $c$, which is bounded below by

$$c = 2\sqrt{1 - r}.$$  (7a)
or in dimensional terms, by

$$c = 2\left[ D(KS_0 - \mu) \right]^{1/2} = 2\left[ D\mu (1/r - 1) \right]^{1/2}. \quad (7b)$$

The numerical simulation of equations (4) in one or two space dimensions is relatively straightforward in the sense that travelling wave solutions are easily obtained. The problem of economically computing solutions which travel at the correct speed is more difficult, and has been studied in depth by Arcuri (1984).

Fig. 3. The fraction $a (= S/S_0)$ of susceptible foxes which survive an epidemic wave as a function of $r (= \mu/KS_0)$: $a$ and $r$ are related by $a - r \log a = 1$ where $0 < a < r < 1$.

3. Control Measures

Now consider the problem of introducing control measures in a protective break in order to stop the epizootic wave from entering a rabies-free region. From a strictly mathematical point of view this is not possible using the model here because whatever the infective density $I$ is initially, we have $I$ positive everywhere at any later time. This is a side effect of introducing classical diffusion as the mode of dispersal and of considering continuous
density functions. This is traditionally remedied by setting \( I = 0 \) if \( I \) is sufficiently small.

To be more precise we could, for example, proceed as follows. Consider a one-dimensional problem and let \( 0 \leq x \leq L \) be a protective barrier between a rabies free region \( x > L \) and an infected region \( x < 0 \). Then we can say, for example, that in effect \( I = 0 \) in \( x > L \) if

\[
\int_{L}^{\infty} I(x, t) \, dx < 1
\]

that is, there is not a single rabid fox in \( x > L \). We take a more stringent approach and say that \( I = 0 \) if it is smaller than a given small number.

With this convention we can, for a given control scheme in the break, numerically determine the minimum width \( L \) which keeps \( x > L \) free of rabies. We have done this for the following simple (illustrative) control scheme. Assume that initially we have a uniform fox density and that we try to remove all foxes within the barrier \( 0 \leq x \leq L \). Suppose we succeed in reducing the density to 20% of what it was, and that we do this well before the rabies wave has arrived at \( x = 0 \). In this case we find that the width \( L = L(r) \) of the barrier varies with \( r \) as shown in Fig. 4. Here we used the non-dimensional system (4) and took the infective population to be zero if it is less than \( 10^{-4} \) (the result is very insensitive to this choice away from the dashed part of the curve in Fig. 4). Note that no such barrier exists for \( 0 < r < 0.2 \), since under those circumstances the susceptible population in the protective barrier \( (0.2S_0) \) is sufficient to sustain the epizootic (see equation (5)). The length \( L \) in Fig. 4 is given in non-dimensional units. To get the actual length in km we need to know the various constants \( K, D, \)
μ, and $S_0$, and these we discuss below. It is easy to extend this analysis to more realistic control schemes where the equation for susceptible foxes is changed in the barrier region $0 \leq x \leq L$ to correspond to vaccination, shooting, gassing or whatever control method is to be used (see Anderson et al. (1981) for a discussion of control strategies).

4. Estimates for the Epidemiological Parameters

To get quantitative information out of the qualitative behaviour of solutions to the model (1) we need to estimate the various parameters involved.

As the epizootic front passes there is often a high mortality. Since these figures vary quite a lot in the literature, depending on region, we have chosen to use Fig. 1 which is taken from MacDonald (1980). There we find a mortality rate of about 65–80% during the height of the epizootic. Taking the fraction $a$ of surviving foxes to be 0.2 we can use Fig. 3 to estimate $r$ to be 0.5. We choose the upper bound of 80% mortality because of the assumption in the model that births and non-rabid deaths balance. In real life we expect a net birth rate when the fox population has passed below the critical density $S_c$ which from equation (5) is $S_c = rS_0$. Over a longer time period this net birth rate when below the critical density can be expected to settle the fox population on this critical density. With $r = 0.5$ this means that the disease reduces the fox population by about 50%, a frequently cited figure (World Health Organization, 1973, 1978; Wandeler et al., 1974; Bogel et al., 1974; Lloyd, 1976; Bogel & Moegle, 1980; Spittler, 1973). This aspect of fox ecology is discussed briefly in the next section.

To estimate $μ$ recall that $1/μ$ is the life expectancy of an infective fox. An infective fox first goes through an incubation period that can vary from 12 to 110 days, and then a rabid state lasting from 3 to 10 days. A life expectancy of about 35 days gives $μ$ as approximately 10 yr$^{-1}$.

To estimate the diffusion coefficient $D$ we use the expression (2). Territories can vary from 2.5 km$^2$ to 16 km$^2$ depending on the habitat, food availability and fox density. If we assume an average territory to be about 5 km$^2$ and that an infective fox leaves its territory about the time it becomes rabid, that is after about a month, then $k$ is approximately 12 yr$^{-1}$. Thus we get $D$ as approximately 60 km$^2$ yr$^{-1}$.

These estimates are crude and are presented only to indicate an order of magnitude. To use the model in a real life situation, more accurate estimates must be obtained. Putting these values into the right hand side of equation (7b) we get an estimate for the wave-speed of about 50 km per year, which is in good agreement with the empirical data from Europe.
Note that if $S_0$ is not too close to $S_c$, so that $r (= S_c/S_0)$ is not too close to 1, then the wave-speed, given by equations (7), does not vary much with fox density. Again, this is in agreement with empirical observation.

One way of using our model is to make tentative predictions as to how an epizootic might spread if introduced into a region where the initial distribution of (susceptible) foxes is known: a knowledge of this could prove helpful in combating the epizootic. In principle, this should be possible to do by computer using the parameter estimates above. However, our attempts to perform such simulations on a map of Britain were relatively crude (a finite element space discretization with 226 nodes giving 452 coupled ordinary differential equations) due to a lack of accuracy and resolution necessitated by computational restrictions. Taking estimates for the initial distribution of susceptible foxes from MacDonald (1980), together with the speed of propagation given by equations (7) and the results from our simulations as a guide, we have drawn by hand the map shown in Fig. 5 to illustrate how a small population of infective foxes introduced around Southampton might spread throughout Britain.

![Map showing projected spatial spread of rabies in Great Britain](image)

**Fig. 5.** The projected spatial spread of rabies in Great Britain if introduced in the vicinity of Southampton, based on the distribution of foxes given by MacDonald (1980), the formula (7) for the wavespeed $c = 2[D(KS_0 - \mu)]^{1/2}$, and the results of crude numerical simulations of the model equations (1). The map depicts how the wavespeed of the epizootic depends on the (susceptible) fox density, and predicts that the disease would reach Manchester in about 6 years. This projected spreading is, of course, only suggestive, and is based on the analysis for the model parameters given in the text plus the assumption that the critical fox density $S_c$ is around 1 fox/km. If the true value of $S_c$ is lower, the important difference is how far the epizootic front will spread into Scotland and Wales (where the native fox density is lower).
5. Generalizations and Other Models

There are at least two good reasons for keeping a mathematical model simple: there are fewer parameters to estimate and since it is easier to analyse, crucial questions which have to be asked are highlighted. Clearly there is plenty of room for generalisations in our model which might make it more realistic.

If we want to model more than the front we must take into account reproduction in the fox population. We expect this to affect the tail of the front as discussed above. Assuming that $S_0$ is the carrying capacity in a particular rabies-free habitat, we can add a logistic population growth term to the equation for the susceptible foxes to obtain in place of the first of equations (1)

$$\frac{\partial S}{\partial t} = -KS + \beta S(1 - S/S_0)$$  \hspace{1cm} (9)

where $\beta$ is the (linear) birth rate. We assume that the rabid foxes do not reproduce. The corresponding non-dimensional system, equivalent to equation (4), is then

$$\frac{\partial u}{\partial t} = \frac{\partial^2 u}{\partial x^2} + u(v - r)$$

$$\frac{\partial v}{\partial t} = -uv + bv(1 - v)$$  \hspace{1cm} (10)

where $b = \beta/KS_0 = r\beta/\mu$. The travelling epizootic "wave" which results is illustrated in Fig. 6, at least near the front. Note the similarity with Fig. 1. A linear analysis ahead of the wave again gives the lower bound of the speed of propagation as $c = 2\sqrt{1 - r}$. In simulations we find the oscillations damp out so that $v$ approaches $r$ and $u$ approaches $b(1 - r)$ far behind the front. These damped oscillations might persist if the incubation period or

![Fig. 6. The shape of the travelling wave solution when the susceptible foxes have logistic population growth with a net birth rate of 0.5 per year, that is $b = 0.05$ (b is the net birth rate times the life expectancy of an infective fox). Here the model parameter $r = 0.5$.](image-url)
the seasonal variation of the cub births is taken into account. One suggestion from this is that the observed periodic outbreak of rabies in endemic areas could perhaps be a late effect of the "over-kill" of the front. Anderson et al. (1981) have speculated that it is primarily an effect of the incubation period.

Dunbar (1983) has studied the system (10) analytically and has shown the oscillations are damped exponentially in time if \( b \leq b_c \) for some critical value \( b_c \). If \( b > b_c \), no oscillations occur, and \((u, v)\) approaches \((b(1-r), r)\) monotonically. Dunbar also gives the estimate \( b_c < 4(1-r)/r \). Since

\[
b = \frac{\beta}{KS_0} = \frac{r\beta}{\mu} = \frac{\beta}{\mu} S_c
\]

the condition for oscillations \( b \leq b_c \) is equivalent to \( S_0 > aS_c \) (here \( a = \mu/\beta b_c \)), that is, the system (10) will give oscillations if the carrying capacity of the environment \( S_0 \) is sufficiently greater than the minimum fox density required for the persistence of rabies, \( S_c \). As discussed in section 6, Mollison (1983) has shown that a large class of epidemic models which do not include dispersal exhibit a similar threshold for (non-damped) oscillatory behaviour.

There are a few other mathematical models for rabies. Most of these do not include spatial effects or are too complex to easily estimate the parameters involved. One exception is the diffusion-approximation to Kendall's classical model for the spatial spread of infectious disease (Kendall, 1965). His model is, in one dimension for simplicity,

\[
\begin{align*}
\partial I/\partial t &= KIS - \mu \bar{I} \\
\partial S/\partial t &= -KIS
\end{align*}
\]  

(11)

where

\[
\bar{I}(x, t) = \int_{-\infty}^{\infty} k(x, y)I(y, t)\,dy
\]  

(12)

for some kernel function \( k \geq 0 \) with \( \int_{-\infty}^{\infty} k(x, y)\,dy = 1 \) for all \( x \). The basic assumption here is that the infective fox at position \( y \) can infect a susceptible at position \( x \) to an extent determined by the kernel function. Expanding the kernel in a standard way we get the above diffusion approximation, that is

\[
\bar{I}(x, t) \approx I(x, t) + D \partial^2 I(x, t)/\partial x^2.
\]  

(13)

This gives the same number of parameters to estimate as in our model.

Our model is perhaps a little closer to the biological facts of rabies. Rabies is mainly spread by rabid foxes moving into neighbouring ranges, not by
distant transmission of the virus from a more or less stationary rabid fox as would be the biological assumption implicit in equations (11) and (12).

Thieme (1979) has presented a deterministic integral equation model and concludes from it that a reduced fox population ahead of the epizootic will prevent the spread into a rabies-free area. Diekmann (1978) discusses more general thresholds and travelling waves of infection.

Probably the most realistic model to date has been proposed by Anderson et al. (1981). Their model equations are

\[
\begin{align*}
\frac{dS}{dt} &= aS - (b + \gamma N)S - KSR \\
\frac{dI}{dt} &= KSR - \sigma I - (b + \gamma N)I \\
\frac{dR}{dt} &= \sigma I - \mu R - (b + \gamma N)R
\end{align*}
\]  

(14)

where \( S, I \) and \( R \) are the susceptible, incubating and rabid population densities, \( N = S + I + R \) is the total population density, and the various parameters are all positive. To interpret the model parameters it is helpful to consider the evolution of the total fox population. Adding the three equations in (14) gives

\[
\frac{dN}{dt} = aS - (b + \gamma N)N - \mu R.
\]  

(15)

Thus \( a \) is the per capita birth rate of the susceptibles, \( b \) is the per capita death rate due to natural causes, that is \( 1/b \) is the mean fox life expectancy in the absence of resource limitation, and \( \gamma \) is a measure of the severity of density dependent constraints. The parameters \( K \) and \( \mu \) in equations (14) play the same role as the parameters \( K \) and \( \mu \) in equations (1): \( K \) is the disease transmission coefficient and \( \mu \) is the death rate due to rabies. The parameter \( \sigma \) specifies the rate at which the incubating foxes become rabid, that is, \( 1/\sigma \) is the mean duration of the incubating stage.

Anderson et al. (1981) cite as support for their model the agreement between observed and predicted threshold susceptible densities, \( S_0 \), contact rates between infectives and susceptibles, average levels of endemic infection, the 3–5 year cycle of disease prevalence in endemic populations, and the tendency for these cycles to be more pronounced in regions with large carrying capacities. In principle it would not be too difficult to include dispersal effects in this model and to obtain travelling epizootic wavefront solutions such as we have done in this paper.

6. Implicit Assumptions

It is instructive to examine the implicit assumptions common to most simple deterministic epidemic models. An essential ingredient for this dis-
Discussion is the concept of the basic reproduction rate, $C$, of a disease, defined as the number of potentially infectious contacts made by an infected individual. Clearly, if $C$ is less than 1 the disease will die out, since for a constant level of infection to be maintained, each infective, on average, must transmit the disease to one susceptible before dying or recovering, and in order to do this must contact at least one individual in the population.

The following simple argument, given by Mollison (1983), relates $C$ to the various model parameters in equations (1). If $1/\mu$ is the mean infectious period, then an individual makes contacts at an average rate of $\mu C$ while infectious. Assuming the contacts are made randomly with both susceptibles and infectives, the probability of disease transmission is proportional to \( S^r \) where $N = S + I$ is the total population density. Multiplying by the infective density, we have that the overall transmission rate is $\mu CS/N$, which is equivalent to the $KIS$ term in equations (1). This gives $C = KN/\mu$.

The mean number of contacts where the disease is transmitted, termed the effective reproduction rate of the disease, is $R = CS/N = KS/\mu$.

In a simple epidemic (epizootic) model the infectives die or recover with immunity at a rate $\mu$, and the overall rate of transmission is modeled by a multiplicative term, $KIS$. The crucial parameter governing the spread of the disease is $R$. When $R$ is less than one, each infective transmits the disease, on average, to less than one susceptible, and the net infective population decreases. Since $R$ is proportional to the susceptible density, $S$, this implies there is a threshold density, $S_c = \mu/K$, below which the disease will die out. Note this result is the same as equation (5); it is common to all simple epidemic models. In the initial stages of an outbreak, $S = N = S_0$ where $S_0$ is the carrying capacity. Thus, as mentioned in section 2, $C = R = 1/\tau$ holds initially, where $\tau = \mu/KS_0$ is the single free parameter in our non-dimensionalized model.

Needless to say, in a model without susceptible births the infectives eventually die out when $S$ falls below $S_c$. Models which include a susceptible birth term can be expected to give rise to an endemic equilibrium state at $S_c$: if $S > S_\infty$ then the number of infectives increases, and if $S < S_c$ the number of susceptibles increases.

Mollison (1983) shows that the tendency for oscillations about $S_c$ when $S_0 > aS_c$ for some constant $a$ is also a common trait of simple epidemic models. Small amplitude oscillations are insensitive to the detailed form of the susceptible growth term and a quantity called the generation gap $\tau$ of the disease, which is the mean time an individual is contagious. In our model $\tau = 1/\mu$, whereas in the model of Anderson et al. (1981), $\tau = 1/\sigma + 1/\mu$. Mollison shows that in simple epidemic models with no spatial effects the period of oscillation is approximately $2\pi \sqrt{\tau/\rho}$, where $\rho$ is the
net susceptible birth rate at $S_c$. Considering several simple models in addition to the model of Anderson et al. (1981), Mollison (1983) shows that they all predict endemic oscillations of 3 to 5 years when $S_0 > \alpha S_c$. Thus, it is not surprising that our extended model (10) also gives such oscillations. The damping of oscillations could be due to the diffusive dispersal. Were the oscillations of limit cycle type then we would expect travelling periodic waves.

Thus, the qualitative behaviour of simple epidemic models is implicitly built into these models, primarily by the commonly used linear removal and multiplicative transmission terms $\mu I$ and $KIS$. The rates $\mu$ and $K$ are usually taken to be constant for mathematical convenience. A constant death rate assumes an exponential distribution of the duration of the infective state which is usually not justified quantitatively. A constant transmission coefficient $K$ is equivalent to assuming that the basic reproductive rate $C$ is proportional to the total population $N$ and independent of the carrying capacity $S_0$. This seems less plausible for a territorial animal such as the fox: the number of neighbouring dens and hence the contact rate between families will generally increase more slowly than the population density.

The dependence of $C$ on $N$, independent of $S_0$, points out another implicit assumption—that of homogeneous mixing. This implies that initially almost all infectious contacts are with susceptibles (although including a dispersal term reduces this probability). More realistic models may take into account the local mixing and clumping (i.e. family structures) within the fox population. In this case, $R$ may be substantially lower than $C$ because the infectives may be clumped together. This in turn will affect the critical density $S_c$ and the effectiveness of a particular control strategy.

7. Conclusions

The use of models in epidemiology has often proved helpful in a variety of important ways, not least in the control of epidemics. Even when the actual transmission mechanisms of the disease are not completely known, models can elicit questions which need to be answered. Although our model mechanism for the spatial spread of rabies is deliberately simple, it encapsulates some key features. Importantly it is a model for which we can obtain estimates for the relevant parameters. Using these estimates the model predicts a wave-speed of the rabies epizootic of approximately 50 km per year which compares quite well with the observed value from observations in Europe since 1945.
If the possibility of an epizootic break is to be pursued, it is essential to have an indication of the width it will have to be. This width depends on various parameters. For our model control scheme, if \( L \) is the non-dimensional break-width, it is in dimensional terms \( l = (D/KS_0)^{1/2}L \); \( L \) can be obtained from Fig. 4 if \( r = \mu/KS_0 \) is known. If \( r = 0.5 \), the value derived above, then \( KS_0 = \mu/0.5 = 20 \text{ yr}^{-1} \) and \( D \approx 60 \text{ km}^2 \text{ yr}^{-1} \), and from Fig. 4, \( L \approx 8 \). This gives \( l \approx 15 \text{ km} \). More reliable estimates would be obtained if an upper bound on the diffusion coefficient could be obtained by estimating how far a rabid fox can travel per day. This distance varies greatly between different foxes, and our estimate of \( D \approx 60 \text{ km}^2 \text{ yr}^{-1} \) is a rough average value. The result, however, seems to be of the right order of magnitude judged from the experiences in Southern Jutland in Denmark (MacDonald, 1980), where a belt of essentially the above type 20 km wide with an additional 20 km zone of less intensive control, was formed in the mid-sixties. It has been successful.

Because of the high probability that rabies will reach disease-free countries such as England and Sweden, it is essential to have as full an understanding as possible of both fox ecology and the spatial spread of the disease into disease-free areas. We have tried to highlight some essential quantitative questions that have to be answered before claiming to understand the gross dynamics of the rabies epizootic front.

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To study the qualitative features of the model system (4), we look for travelling wave solutions $u(x, t) = f(z)$, $v(x, t) = g(z)$ where $z = x - ct$ and $f$ and $g$ are waveforms travelling to the right at speed $c$. Substituting these relations into (4) gives

$$f'' + cf' + fg - rf = 0$$

$$cg' - fg = 0$$

where primes denote differentiation with respect to $z$, and $f$ and $g$ are subject to the boundary conditions $f(\pm\infty, t) = 0$, $g(\pm\infty, t) = 1$, $g(-\infty, t) = a$ where $a$ is a constant to be found. Substituting the second of (A1) into the first and dividing through by $c$ gives

$$\frac{1}{c}f'' + f' + g' - rg'/g = 0$$

(A2)

which on integrating gives

$$\frac{1}{c}f' + f + g - r \log g = A$$

(A3)

where $A$ is a constant. Using the boundary conditions at $z = \infty$ shows that $A = 1$, while the conditions at $z = -\infty$ give

$$a - r \log a = 1$$

(A4)
which relates the fraction $a$ of susceptibles remaining after the epizootic has passed to the model parameter $r$.

To obtain a lower bound for the wavespeed $c$, we use (A3) and the second of (A1) and consider the phase plane of the system

\[
\begin{align*}
 f' &= c[1 - f - g + r \log g] \\
 g' &= \frac{1}{c}fg
\end{align*}
\]  

which has singular points at $(0, a)$ and $(0, 1)$. Linearizing, we find that $(0, a)$ is a saddlepoint for $0 < r < 1$. The point $(0, 1)$ is a stable node for $c^2 > 4(1 - r)$ but is a stable spiral for $c^2 < 4(1 - r)$. The latter type of singularity implies negative population densities and is thus not allowed. Hence any travelling wave solutions must satisfy

\[ c \geq 2\sqrt{1 - r}. \]