Chapter 8
Spatial Structure: Partial Differential Equations Models

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Abstract This chapter introduces some basic concepts and techniques in modeling spatial spread of diseases involving hosts moving randomly during certain stages of the disease progression. First we derive some reaction diffusion models using the conservation law and Fick's law of diffusion. We then discuss the usefulness of these models in describing disease spread rates and evaluating the effectiveness of some spatially relevant disease control strategies. We illustrate the general theory via two case studies, one about the spread of rabies in continental Europe during the period 1945–1985 and another about spread rates of West Nile virus in North America.

8.1 Introduction

As discussed in [19], spatial structures play an important role in describing the spreading of communicable diseases, not only because the environment is heterogeneous but also because individuals move around in space. Many prevention and control strategies involve spatial aspects such as immigration, vaccination, border control and restriction of individual movements.

In this chapter, we introduce an approach, based on reaction diffusion equations, to describe the spread of communicable diseases in spatially structured populations. We shall also illustrate this approach and demonstrate its applications via two case studies; one is about the spread of rabies in continental Europe during the period 1945–1985 and another is about spread rates of West Nile virus.

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We have no intention here to give a comprehensive introduction to a subject that has been intensively studied, and we refer to [10] that has great influence on the next two sections of this chapter, and [5–7, 15, 18] for relevant literature and detailed discussions.

8.2 Model Derivation

We first consider the case in which a collection of individuals moves randomly in one dimensional space, with an average step length Δx in every time unit Δt. Assume the growth rate (with respect to time) at spatial location x and time t is given by $f(t, x)$ (this term, in most cases, also depends explicitly on the numbers of the individuals) and assume the probability of moving to the left and to the right are both equal, and hence are 0.5.

Let $u(t, x)$ be the number of individuals within the spatial segment $[x, x + \Delta x]$ at the time t. Then

$$u(t + \Delta t, x) - u(t, x) = \frac{1}{2}u(t, x - \Delta x) + \frac{1}{2}u(t, x + \Delta x) - u(t, x) + f(t, x)\Delta t.$$

Using the Taylor series expansions for $u(t, x \pm \Delta x)$ and $u(t + \Delta t, x)$ as follows

$$\begin{aligned} u(t, x \pm \Delta x) &= u(t, x) \pm \tfrac{\partial}{\partial x}u(t, x)\Delta x + \tfrac{1}{2}\tfrac{\partial^2}{\partial x^2}u(t, x,)(\Delta x)^2 + \cdots, \\ u(t + \Delta t, x) &= u(t, x) + \tfrac{\partial}{\partial t}u(t, x)\Delta t + \tfrac{1}{2}\tfrac{\partial^2}{\partial t^2}u(t, x)(\Delta t)^2 + \cdots, \end{aligned}$$

we obtain

$$\frac{\partial}{\partial t}u(t, x)\Delta t + \frac{1}{2}\frac{\partial^2}{\partial t^2}u(t, x)(\Delta t)^2 + \cdots = \frac{1}{2}\frac{\partial^2}{\partial x^2}u(t, x)(\Delta x)^2 + \cdots + f(t, x)\Delta t, \tag{8.1}$$

where $\cdots$ denotes higher order terms. Assume that the temporal and spatial scales are chosen appropriately so that

$$\frac{(\Delta x)^2}{2\Delta t} = D \tag{8.2}$$

remains to be a given constant, called the *diffusion coefficient*. Dividing (8.1) by Δt and then letting $\Delta t \to 0$ and $\Delta x \to 0$, we get

$$\frac{\partial}{\partial t}u(t, x) = D\frac{\partial^2}{\partial x^2}u(t, x) + f(t, x). \tag{8.3}$$

Note the above derivation also suggests how D can be estimated from field data.

The above reaction diffusion equation can also be established through the *conservation equation* based on a certain *balance law* [5]. To describe the

model derivation, we consider the spatial segment $[x, x + \Delta x]$ and note that the change of the total number of individuals in this segment is due to the flow into and out of the interval through its boundary points; and due to the (local) growth process that reproduces the individuals within the segment. In other words, if we denote by $J(t, x)$ the number of individuals crossing at x in the positive direction per unit time, then we have the following balance law:

$$\frac{\partial}{\partial t}u(t,x)\Delta x = J(t,x) - J(t,x+\Delta x) + f(t,x)\Delta x.$$

Again, we use the Taylor series expansion for $J(t, x + \Delta x)$ to obtain that

$$\frac{\partial}{\partial t}u(t,x)\Delta x = -\frac{\partial}{\partial x}J(t,x)\Delta x - \frac{1}{2}\frac{\partial^2}{\partial x^2}J(t,x)(\Delta x)^2 + \cdots + f(t,x)\Delta x.$$

Dividing by Δx and then letting $\Delta x \to 0$ gives

$$\frac{\partial}{\partial t}u(t,x) = -\frac{\partial}{\partial x}J(t,x) + f(t,x). \tag{8.4}$$

It remains to specify $J(t, x)$, the flux of individuals at (t, x). A popular choice of such a flux is based on the so-called *Fick's law*, which states that the flux due to random motion is approximately proportional to the local gradient of the number of individuals. This yields

$$J(t,x) = -D\frac{\partial}{\partial x}u(t,x). \tag{8.5}$$

Combining (8.4) and (8.5) gives the reaction diffusion equation (8.3).

In population ecology, we can translate Fick's law of diffusion into the statement that the individuals move from a region of high concentration to a region of low concentration in search for limited resources. We must, however, use this law with caution when modeling spatial spread of infectious diseases since the individual movement behaviors may be altered during the course of outbreaks of diseases.

To determine the value of $u(t, x)$ in space and for all future time $t \geq 0$, we need to specify the initial distribution of the population $u(0, x)$ (*initial condition*). Also, when the space is bounded, say $x \in (0, L)$, we need to specify the boundary conditions. Typical boundary conditions include the homogeneous *Dirichlet condition*

$$u(t,0) = u(t,L) = 0$$

when the boundary is uninhabitable, or the homogeneous *Neumann condition*

$$\frac{\partial}{\partial x}u(t,0) = \frac{\partial}{\partial x}u(t,L) = 0$$

when there is no flux through boundaries.

8.3 Case Study I: Spatial Spread of Rabies in Continental Europe

We now demonstrate the usefulness of the partial differential equation approach for the study of spatial spread of rabies in continental Europe during the period roughly 1945–1985.

Starting on the edge of the German/Polish border, the front of the epizootic moved westward at an average speed of about 30–60 km a year. The spread of the epizootic was essentially determined by the ecology of the fox population as foxes are the main carrier of the rabies under consideration. If the fox population density is estimated at different times as the rabies epizootic passes, 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.

A model was formulated in [9] in order to describe the front of the wave, its speed and the total number of foxes infected after the front passes, and the connection of the wave speed to the so-called propagation speed of the disease. We shall also use this case study to illustrate how the partial differential equation model can help us in designing some spatial intervention strategies by considering the minimal length of protective zones. Various extensions of this basic model were also proposed to discover the mechanism for the periodic outbreaks and to estimate the periods and amplitudes, and we shall briefly discuss these extensions at the end of this section.

We start with a list of basic facts and some standing assumptions that we will use in our modeling and analysis.

- Foxes are the main carriers of rabies in the rabies epizootic considered.
- The rabies virus is contained in the saliva of the rabid fox and is normally transmitted by bite.
- Rabies is invariably fatal in foxes.
- Foxes are territorial and seem to divide the countryside into non-overlapping home ranges which are marked out by scent.
- The rabies virus enters the central nervous system and induces behavioral changes in its host. If the spinal cord is involved it often causes paralysis. However, if it enters the limbic system the foxes become aggressive, lose their sense of direction and territorial behavior and wander about in a more or less random way.

The last observation is the basis on which a reaction diffusion equation can be used to model the dynamics of rabid foxes. To formulate a deterministic model, at time t and spatial location x, let

$$S(t,x) = \text{the total number of susceptible foxes,}$$
$$I(t,x) = \text{the total number of infective foxes.}$$

Note that for the sake of simplicity in the above definition of $I(t,x)$, we do not distinguish rabid foxes and those in the incubation period, although it should be pointed out that only a fraction of infective foxes, namely, rabid foxes, transmit the disease.

A salient feature of rabies is the rather lengthy incubation period of between 12 and 150 days from the time of an infected bite to the onset of the clinical infectious stage. This feature was taken into account in the ordinary differential equation model [1] and its reaction diffusion analogue was developed in [16].

Ignoring this lengthy incubation period, then the model formulated in [9] in a one-dimensional unbounded domain takes the following form

$$\begin{cases} \frac{\partial}{\partial t}S(t,x) = -KS(t,x)I(t,x), \\ \frac{\partial}{\partial t}I(t,x) = D\frac{\partial^2}{\partial x^2}I(t,x) + KS(t,x)I(t,x) - \mu I(t,x), \end{cases}$$

where

K = the transmission coefficient,
μ^{-1} = life expectancy of an infective fox,
D = diffusion coefficient = A/k,
k = the average time until a fox leaves its territory,
A = the average area of a typical fox's territory.

In [16], other approaches, based on field observations of net distances traveled by infectives during observation periods, were developed for estimating the parameter D.

Rescaling the variables by

$$\begin{aligned} &u(t,x) = I(t,x)/S_0,\ v(t,x) = S(t,x)/S_0, \\ &x^* = (KS_0/D)^{1/2}x,\ t^* = KS_0t, \\ &r = \mu/(KS_0), \end{aligned}$$

where S_0 is the initial susceptible density that is assumed to be uniform in space, and dropping the asterisks for convenience, we obtain the nondimensional system

$$\begin{cases} \frac{\partial}{\partial t}u(t,x) = \frac{\partial^2}{\partial x^2}u(t,x) + u(t,x)(v(t,x) - r), \\ \frac{\partial}{\partial t}v(t,x) = -u(t,x)v(t,x). \end{cases} \tag{8.6}$$

Observe that r^{-1} is in fact the basic reproduction number of the corresponding ODE model. Therefore, if $r > 1$ the infection dies out quickly. Epidemiologically, this is reasonable since $r > 1$ if and only if $\mu > KS_0$. That is, $r > 1$ (or equivalently, the basic reproduction number is less than 1) if the mortality rate is greater than the rate of recruitment of new infectives. In this case, rabies cannot persist.

The above discussion also gives the minimum fox density $S_c := \mu/K$ below which rabies cannot persist. Mathematically, in [8], it was proven that if $r \geq 1$,

$u(0,x) \geq 0$ for all $x \in R$ and u has bounded support, and if $v(0,x) = 1$ for $x \in R$, then $u(t,x) \to 0$ as $t \to \infty$ uniformly on R.

A natural question is what happens if $r < 1$. In what follows, we will illustrate that if the initial distribution of susceptibles is uniformly equal to 1 (that is, $S = S_0$ everywhere), then a small localized introduction of rabies evolves into a traveling wave with a certain wave speed.

A solution of (8.6) is called a *traveling wave* (or *traveling wavefront*) at speed c if

$$u(t,x) = f(z),\ v(t,x) = g(z)$$

where

$$z = x - ct$$

is the *wave variable* and f and g are *waveforms* (or *wave profiles*).

Intuitively speaking, a traveling wave is a solution that moves in space with a constant speed c and without changing shape. In other words, if a fox or an observer moves at the same speed of the wave, the fox will not notice the change of the wave.

Substituting the above special form into system (8.6), we obtain the equations for the waveforms:

$$\begin{cases} f'' + cf' + fg - rf = 0, \\ cg' - fg = 0. \end{cases} \tag{8.7}$$

This is a system of ordinary differential equations, where primes denote differentiation with respect to z.

To solve the above system for the waveforms, we need to specify the asymptotic boundary conditions that are given naturally by

$$f(\pm\infty) = 0,\ g(+\infty) = 1, g(-\infty) = a, \tag{8.8}$$

where a is an important parameter to be found, that tells us the proportion of susceptible foxes that remain after the infective wave has passed, and this number is given by

$$a - r \ln a = 1. \tag{8.9}$$

To obtain the above formula (8.9), we rewrite the system for the waveforms as

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

that gives, after integration, the following

$$\frac{1}{c} f' + f + g - r \ln g = B \tag{8.10}$$

for a constant B that can be found, using the boundary condition at $z = \infty$, as $B = 1$. Therefore, using the boundary condition at $-\infty$, we get $a - r \ln a = 1$.

This is a very useful relation in order to obtain an estimation of r (and hence K). For example, in [14], it is suggested that the mortality rate is about 65–80% during the height of the epizootic. Therefore, if we take the fraction a of surviving foxes to be 0.2, we obtain approximately $r = 0.5$.

It follows also from (8.10) that system (8.7) is equivalent to the following planar system

$$\begin{cases} f' = c[r \ln g - f - g + 1], \\ g' = fg/c. \end{cases} \tag{8.11}$$

The linearization around the stationary point $(0, 1)$ has eigenvalues

$$\lambda_{\pm} = -\frac{1}{2}[c \pm \sqrt{c^2 - 4(1-r)}].$$

Hence, if $c^2 < 4(1-r)$ we have complex eigenvalues and all of the trajectories cannot stay in the positive quadrant near $(0, 1)$.

If $c \geq 2\sqrt{1-r}$, $(0, 1)$ is a stable node and $(0, a)$ is a saddle point with the unstable trajectory entering the positive quadrant that, using some phase-plane analysis (see [8]), converges to $(0, 1)$ as $z \to \infty$. Therefore, in [9] it is shown that if $r < 1$ there exists a traveling wave of system (8.6) subject to boundary condition (8.8) with the speed

$$c \geq c_0 = 2\sqrt{1-r}. \tag{8.12}$$

The traveling wave with the minimal wave speed c_0 is of paramount importance since any initial function $u(0, x)$ of compact support splits up into two traveling waves going in opposite directions with the same speed. More precisely, it was proved in [8] that if $u(0, \cdot)$ has compact support, then for every $\delta > 0$ there exists N so that

$$u(t, x + c_0 t - \ln t/c_0) \leq \delta$$

for every $t > 0$ and for all $x > N$. Therefore, if a fox travels with speed $c(t) = c_0 - (c_0 t)^{-1} \ln t$ towards $+\infty$ (in space) to the right of the support of $u(0, \cdot)$, the infection will never overtake the fox (hence the title "Run for your life, a note on the asymptotic speed of propagation of an epidemic" of the paper [3]). In other words, the asymptotic speed of the infection must be less than $c(t)$. As a consequence, if $u(t, x)$ takes the form of a traveling wave for large t, it must do so for the one with the minimal speed c_0.

A key issue for potential applications of the model is to identify all parameters involved. The parameter r is related to the transmission coefficient K which can hardly be estimated directly. Fortunately, as discussed above, formula (8.9) enables us to calculate r indirectly by considering the mortality as the epizootic front passes. We have $r = 0.5$, hence the disease reduces the fox population by about 50%.

The next parameter is μ. Recall that $1/\mu$ is the life expectancy of an infective fox. An infective fox first goes through an incubation period that

can vary from 12 to 150 days, and then a rabid state lasting from 3 to 10 days. Thus, a life expectancy of about 35 days give μ as approximately $(1/10\,\mathrm{yr})^{-1}$.

The diffusion coefficient D is the one related to the spatial spread and, as shown in [9], this can be calculated by the formula $D = A/k$. Fox territories can range from $2.5\,\mathrm{km}^2$ to $16\mathrm{km}^2$ depending on the habitat, food availability and fox density. If we assume an average territory to be about $5\,\mathrm{km}^2$ and that an infective fox leaves its territory about the time it becomes rabid, that is after about a month, then k is approximately $12\mathrm{yr}^{-1}$. Thus we get D as approximately $60\,\mathrm{km}^2\mathrm{yr}^{-1}$.

Putting this together, we then obtain the minimal wave speed of about 50 km per year, which seems to be in good agreement with the empirical data from Europe.

The diffusion model provides a useful framework to evaluate some spatially related control measures. For example, in [9], some estimate about a protective barrier is given. The mathematical formulation can be stated as follows: Let $0 \le x \le L$ be a protective barrier between a rabies free region $x > L$ and an infected region $x < 0$. How large should $L > 0$ be in order for $I(t, x) < \epsilon$ for all $t \ge 0$ and for all $x > L$, where $\epsilon > 0$ is a parameter, below which the infection is regarded as dying out?

This problem was investigated in [9] via numerical simulations, and the result of course also depends on the susceptible fox density in the protective zone and the parameter r. Reduction of the susceptible fox population in the protective zone can be achieved be shooting, gassing, vaccination, etc. Admittedly, the above model is only an approximation, but such a relatively simple model that captures some basic features of the disease spread requires very fewer parameters to estimate.

In the model considered so far, the natural birth and death are assumed to be balanced. Using a classical logistic model for the growth of susceptible foxes, we can explain the tail part of the wave, and in particular, the oscillatory behavior. Indeed, Anderson et al. [1] speculated that the periodic outbreak is primarily an effect of the incubation period, and Dunbar [4] and Murray et al. [16] obtained some qualitative results that show sustained oscillations if the classical logistic model is used and the carrying capacity of the environment is sufficiently large.

The model also ignores the fact that juvenile foxes leave their home territory in the fall, traveling distances that typically may be 10 times a territory size in search of a new territory. If a fox happens to have contracted rabies around the time of such long-distance movement, it could certainly increase the rate of spread of the disease into uninfected areas. This factor was pointed out in [16], and the impact of the age-dependent diffusion of susceptible foxes was recently considered in [17] by using structured population models.

8.4 Case Study II: Spread Rates of West Nile Virus

Although West Nile virus (WNv) was isolated in the West Nile district of Uganda in 1937, and WNv in the Eastern Hemisphere has been maintained in an enzootic cycle involving culicine mosquitoes (vectors) and birds (reservoirs), WNv activities in North America were not recorded until August of 1999 in the borough of Queens, New York City [2]. In the subsequent five years the epidemic has spread spatially to most of the west coast of North America, as a consequence of the interplay of disease dynamics and bird and mosquito movement. We refer to [21] for a detailed discussion of the ecological and epidemiological aspects of the disease spread and the recent modeling efforts of the WNv transmission dynamics.

Here we present the work [11], where the spread of WNv is investigated by spatially extending the non-spatial dynamical model [20] to include diffusive movements of birds and mosquitoes. The simplified spatial model that is analyzed takes the form:

$$\begin{cases} \frac{\partial I_V}{\partial t} = \alpha_V \beta_R \frac{I_R}{N_R}(A_V - I_V) - d_V I_V + \epsilon \frac{\partial^2 I_V}{\partial x^2}, \\ \frac{\partial I_R}{\partial t} = \alpha_R \beta_R \frac{N_R - I_R}{N_R} I_V - \gamma_R I_R + D \frac{\partial^2 I_I}{\partial x^2}, \end{cases} \tag{8.13}$$

where the parameters and variables are defined below:

d_V: adult female mosquito death rate,
γ_R: bird recovery rate from WNv,
β_R: biting rate of mosquitoes on birds,
α_V, α_R: WNv transmission probability per bite to mosquitoes and birds, respectively,
ϵ, D: diffusion coefficients for mosquitoes and birds respectively,
$I_V(t,x), I_R(t,x)$: numbers of infectious (infective) female mosquitoes and birds at time t and spatial location $x \in R$,
N_R: number of live birds,
A_V: number of adult mosquitoes.

The initial model is much more complicated and system (8.13) is obtained after a sequential procedure of simplification. Indeed, in the work [11] the female mosquito population is divided into larval, susceptible, exposed and infectious classes, and the bird population consists of compartments for susceptible, infectious, removed and dead birds. Under the assumption that the death rate of birds due to WNv can be ignored and the removed birds become immediately susceptible (no temporary immunity arising from WNv), it is shown that N_R remains a constant and the number of removed birds tend to zero. Hence the spatially homogeneous model for infectious birds is given by

$$\frac{dI_R}{dt} = \alpha_R \beta_R \frac{N_R - I_R}{N_R} I_V - \gamma_R I_R. \tag{8.14}$$

Furthermore, if exposed mosquitoes are immediately infective, then the exposed class (of mosquitoes) can be ignored and the dynamical system for the adult and larval mosquitoes is a simple planar linear system, solutions of which approach constants. Therefore, the spatially homogeneous version for the infectious mosquitoes becomes

$$\frac{dI_V}{dt} = \alpha_V \beta_R \frac{I_R}{N_R}(A_V - I_V) - d_V I_V. \tag{8.15}$$

Phase-plane analysis of the spatially homogeneous coupled system (8.14)-(8.15) shows that a nontrivial (endemic) equilibrium (I_V^*, I_R^*) exists if and only if the basic reproduction number $\mathcal{R}_0$ is large than 1, where

$$\mathcal{R}_0 = \sqrt{\frac{\alpha_V \alpha_R \beta_R^2 A_V}{d_V \gamma_R N_R}}.$$

Moreover, this endemic equilibrium, if it exists, is globally asymptotically stable in the positive quadrant.

For the spatially varying model (8.13), the vector field is cooperative, therefore an application of the general result in [12] ensures that there exists a minimal speed of traveling fronts c_0 such that for every $c \geq c_0$, the nonlinear system (8.13) has a nonincreasing traveling wave solution $(I_V(x-ct), I_R(x-ct))$ with speed c so that

$$\lim_{(x-ct)\to-\infty}(I_V, I_R) = (I_V^*, I_R^*), \qquad \lim_{(x-ct)\to\infty}(I_V, I_R) = (0,0).$$

Here a traveling wavefront with speed c for system (8.13) is a solution that has the form $(I_V(x-ct), I_R(x-ct))$ and connects the disease-free and endemic equilibria so that the above boundary conditions are satisfied. Note that the traveling wave solution is then given by

$$\begin{cases} -cI_V' = \epsilon I_V'' + \alpha_V \beta_R \frac{I_R}{N_R}(A_V - I_V) - d_V I_V, \\ -cI_R' = DI_R'' + \alpha_R \beta_R \frac{N_R - I_R}{N_R} I_V - \gamma_R I_R. \end{cases}$$

What makes the minimal wave speed c_0 so important epidemiologically is the following mathematical relation: the minimal wave speed c_0 coincides with the spread rate c^* defined as follows: if the initial values of $(I_V(\cdot,0), I_R(\cdot,0))$ have compact support and are not identical to either equilibrium, then for small $\epsilon > 0$,

$$\begin{aligned} &\lim_{t\to\infty}\left\{\sup_{|x|\geq(c^*+\epsilon)t} ||(I_V(t,x), I_R(t,x))||\right\} = 0, \\ &\lim_{t\to\infty}\left\{\sup_{|x|\leq(c^*-\epsilon)t} ||(I_V(t,x), I_R(t,x)) - (I_V^*, I_R^*)||\right\} = 0. \end{aligned}$$

This relation holds due to the cooperative nature of the vector field. It is also due to this nature that the spread speed c^* is linearly determined: namely,

the spread speed is the same as the number $\tilde{c}$ so that the solution $(\tilde{I}_V, \tilde{I}_R)$ with nontrivial initial values of compact support of the linearized system of (8.13) about the disease endemic equilibrium satisfies, for small ϵ,

$$\begin{aligned}&\lim_{t\to\infty}\left\{\sup_{|x|\geq(\tilde{c}+\epsilon)t}||(\tilde{I}_V(t,x),\tilde{I}_R(t,x))||\right\}=0,\\&\lim_{t\to\infty}\left\{\sup_{|x|\leq(\tilde{c}-\epsilon)t}||(\tilde{I}_V(t,x),\tilde{I}_R(t,x))||\right\}>0.\end{aligned}$$

Consequently, it is shown in [11] that

$$c_0 = c^* = \tilde{c} = \inf_{\lambda>0}\sigma_1(\lambda)$$

where $\sigma_1(\lambda)$ is the largest eigenvalue of the matrix

$$B_\lambda = \lambda\begin{pmatrix}\epsilon & 0\\ 0 & D\end{pmatrix} + \lambda^{-1}\begin{pmatrix}-d_V & \alpha_V\beta_R\frac{A_V}{N_R}\\ \alpha_R\beta_R & -\gamma_R\end{pmatrix}.$$

The characteristic equation of B_λ is given by

$$\begin{aligned}p(\sigma;\lambda,\epsilon) = {}&\sigma^2 - \sigma\lambda^{-1}[\theta + (D+\epsilon)\lambda^2]\\&+\lambda^{-2}[d_V\gamma_R - \alpha_V\alpha_R\beta_R^2\tfrac{A_V}{N_R}] - Dd_V - \epsilon\gamma_R + \epsilon D\lambda^2 = 0.\end{aligned}$$

In the general case $\epsilon > 0$, the larger root $\sigma_1(\lambda,\epsilon)$ can have more than one extremum and hence it is difficult to obtain a result for the minimal spread rate by examining roots of $p(\sigma;\lambda,\epsilon)$. However, the case with $\epsilon = 0$ is sufficiently simple and due to the continuous dependence of eigenvalues on parameters, it is shown in [11] that as $\epsilon \to 0$, the spread speed rate approaches the positive square root of the largest zero of an explicitly defined cubic.

In [11], an example is provided to show how the spread rate varies as a function of the bird diffusion coefficient D, in the range $D \in [0, 14]\, km^2/day$ as estimated in [20]. This example is based on the assumption that $A_V/N_R = 20$ and $\gamma_R = 0.01$/day and using the parameters $d_V = 0.029, \alpha_V = 0.16, \alpha_R = 0.88, \beta_R = 0.3/day$ estimated in [20]. Since WNv has spread across North America in about five years, the observed spread rate is about $1,000\, km/year$. This, together with the aforementioned functional relation between the spread rate and the diffusion rate of birds, shows that a diffusion coefficient of about 5.94 is needed in the model to achieve the observed spread rate.

Needless to say, the reaction-diffusion system (8.13) is a first approximation for the spatial spread of WNv, and it is based on the assumption of random flight of birds and mosquitoes. In reality, as pointed out in [11], flight is influenced by topographical, environmental and other factors. The work in [13] based on a patchy model seems to indicate the spread speed may be different if the movement of birds has preference to direction. Certainly, to incorporate more ecology and epidemiology, models should contain more realistic bird and mosquito movements.

8.5 Remarks

We conclude this chapter with a few remarks. First of all, we note that reaction diffusion equations arise naturally from modeling spatial spread of infectious diseases when a subpopulation moves randomly in space, and use of such a model is appropriate when transmission mechanisms and control measures involve spatial movements.

We must, however, be extremely cautious when modeling spatial spread when individual movement is not so obviously random. Other type of models will be needed, and in particular, the discrete space models considered in [19] seem to be more appropriate. When other factors such as disease age and social structures are considered, model systems could be much more complicated.

We have shown that when the space is large in scale, traveling waves of the reaction diffusion equations are important as they describe the progress of the disease to uninfected regions. The wave speed is obviously important to understand the speed of propagation: in some cases it coincides with the spread rate and can be determined by considering the linearization of the nonlinear system at a disease endemic equilibrium.

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