Mathematical Biosciences 270 (2015) 192–197

Contents lists available at ScienceDirect

Mathematical Biosciences

journal homepage: www.elsevier.com/locate/mbs

Models for the effects of host movement in vector-borne disease systems

Chris Cosner[1]

University of Miami Department of Mathematics Coral Gables, FL 33124, United States

ARTICLE INFO

Article history:
Available online 6 July 2015

Keywords:
spatial epidemiology
vector-borne disease
host movement
Ross-Macdonald models

ABSTRACT

Host and/or vector movement patterns have been shown to have significant effects in both empirical studies and mathematical models of vector-borne diseases. The processes of economic development and globalization seem likely to make host movement even more important in the future. This article is a brief survey of some of the approaches that have been used to study the effects of host movement in analytic mathematical models for vector-borne diseases. It describes the formulation and interpretation of various types of spatial models and describes a few of the conclusions that can be drawn from them. It is not intended to be comprehensive but rather to provide sufficient background material and references to the literature to serve as an entry point into this area of research for interested readers.

1. Introduction

Vector-borne diseases have long been recognized as significant threats to public health. In recent years globalization, development, and global change have altered the spatial distribution and movement patterns of human populations, introduced pathogens to places where they were not previously present, and modified environments in ways that affect vector populations. Some of the effects of those changes are not surprising. Greater connectivity has increased the risk of biological invasions by novel pathogens, and indeed such invasions have occurred. The case of West Nile virus in North America is an important example. Some other effects, or the absence thereof, are more surprising. Although development has caused human populations in some regions to move to urban environments which are inhospitable to vectors, malaria may still persist in places where transmission rates are very low, and there are reasons to think that this effect may be related to human movement patterns (see [9,22,27]). There have been a number of studies focussed on the role of host movement on the transmission of vector-borne diseases; see for example [1,5,18,19,30,36,37,45]. The goal of this paper is to describe some of the modeling approaches that have been used to study spatial effects, especially the effects of host movement, in vector-borne disease systems. It is not intended to be a systematic review, but rather an introduction to the ideas and literature in the area. Although the references are not comprehensive, the interested reader should be able to find much additional literature on spatial models for vector-borne diseases in the works that they cite, or by searching for works that cite them. The underlying local disease dynamics in the spatial models described in this paper will be formulated in terms of Ross-Macdonald models, but the methods for extending local models to include spatial effects can be and have been used with other types of epidemiological models; see for example [2,3]. The notation used in this paper is based on that used in [5].

2. Some basic models

2.1. Nonspatial models

Spatial models in epidemiology are typically constructed by starting with a non-spatial model for disease transmission and extending it or modifying it to incorporate spatial effects. The classical model for the local transmission of vector-borne diseases is the Ross-Macdonald model. A discussion of that model and its history is given in [35]. The version described here is similar to the one derived by [33] but there are many variations and extensions in the literature. A systematic review of vector-borne disease models from 1970 to 2010 is given by [26]. The Ross-Macdonald model was originally formulated for malaria. Related models can be used to study other vector-borne diseases but some modifications may be required; for example, for Dengue fever the presence of distinct serotypes of the pathogen is important and so it may be necessary to keep track of the serotypes of the viruses causing infection, while for Rift Valley fever there is vertical transmission of the disease in some of its vectors in some locations. However, the methods for incorporating spatial effects would be similar for most mosquito-borne diseases. There may be other considerations for other types of disease vectors that require distinct modeling approaches; for example, the movement patterns and life

E-mail address: gcc@math.miami.edu

[1] Research partially supported by NSF Grant DMS 11-18623.

http://dx.doi.org/10.1016/j.mbs.2015.06.015
0025-5564/

cycles of ticks are very different from those of mosquitoes, partly because ticks are transported by their hosts, so separating vector and host movements in models for tick-borne diseases can be somewhat problematic. In what follows we will generally assume that the disease vectors are mosquitoes and the hosts are humans and will sometimes refer to them in those ways. The state variables for the Ross-Macdonald model are the fractions of the host and vector populations that are infectious, denoted by x and y. The model also involves various parameters, as shown:

x fraction of infectious individuals in human population,
y fraction of infectious individuals in mosquito population,
a human feeding rate of mosquitoes (number of bites on humans, per mosquito, per unit time),
b transmission efficiency from infectious mosquitoes to humans,
c transmission efficiency from infectious humans to mosquitoes,
m mortality rate of mosquitoes,
r recovery rate of humans,
n incubation period from the time a mosquito becomes infected until it becomes infectious,
M ratio of mosquitoes to humans.

In terms of those variables and parameters the non-spatial model takes the form

$$\frac{dx}{dt} = Maby(1-x) - rx,$$
$$\frac{dy}{dt} = acx(e^{-mn} - y) - my. \tag{1}$$

The model (1) is highly idealized. It does not allow for variable populations of humans or mosquitoes. Unlike many models for directly transmitted diseases, it only treats two classes of individuals explicitly, namely infectious humans and infectious mosquitoes. However, it does implicitly take into consideration the fact that there is a latency period between the time mosquitoes are infected and the time they become infectious. The distinction between infected and infectious is significant in mosquitoes because their lifespans are so short that many infected individuals may die between the time of being infected and the time they would have become infectious. That is the reason for the term e^{-mn} in the equation for the vectors in (1); it accounts for the loss of infected but not yet infectious individuals. (Since humans are removed from the infectious class by recovery rather than death and the timescale for that is relatively long it is less important to consider the time it takes for humans to progress from infected to infectious.) It can be argued that more complex models which explicitly include latent classes of individuals who are infected but not yet infectious, that is, SEI or SEIR type models, would be more realistic; see [4,11,12,44]. Similarly, since there is heterogeneity in the rates at which different types of individuals are bitten by mosquitoes, it can also be argued that multigroup models would be more realistic; see [10]. In fact, such issues have been considered and more detailed models that take some of these complexities into account have been formulated. The primary focus of this paper is on describing how movement can be modeled and what sorts of effects movement can have, so I will illustrate those by using simple models based on (1) rather than more complex and realistic epidemiological models. To formulate some of the spatial models we will want to work with the total populations of infected individuals rather than the fractions of populations rather than the fractions of populations consisting of infectious individuals. The reason is that some spatial models allow changes in the size of local populations because of the effects of movement. In that case M may not be constant. If we use the variables

H the total human population,
X the number of infected humans,
V the total mosquito population,
Y the number of infected mosquitoes,

we can replace M with V/H. In general, $X = xH$ and $e^{-mn}Y = yV$. Using those relations we can rewrite (1) as

$$\frac{dX}{dt} = \frac{abe^{-mn}}{H}Y(H-X) - rX,$$
$$\frac{dY}{dt} = \frac{ac}{H}X(V-Y) - mY. \tag{2}$$

This formulation assumes that there is no change in the local populations of humans or mosquitoes because of disease or demography (there may be turnover of individuals), but allows for the possibility of population change due to movement from other locations. It is possible to construct models that allow for demography or more complex disease processes (e.g. SEIR disease dynamics) within populations as well as movement. As an example we introduce the variables S_H, E_H, I_H, and R_H denoting populations of susceptible, infected, infectious, and recovered (immune) humans, and S_V, E_V, and I_V denoting populations of susceptible, infected, and infectious mosquitoes. We also need new parameters and terms in the model as follows:

λ_H, λ_V birth rates of humans and mosquitoes, respectively,
$f_H(H)$, $f_V(V)$ natural death rates of humans and mosquitoes,
ν_H, ν_V rates of progression from infected to infectious classes,
α rate of progression from infected to immune,
β rate of loss of immunity,
γ rate of extra mortality due to infection.

The remaining variables and parameters will be the same as in (2). (The parameter m in (2) corresponds to the coefficient of the linear term in the mortality rate $f_V(V)$, while n is inversely proportional to ν_V.) Using these variables and parameters we can formulate a non-spatial model that is similar to those used to develop spatial models in [11]:

$$\frac{dS_H}{dt} = \lambda_H H - f_H(H)S_H - \frac{ab}{H}S_H I_V + rI_H + \beta R_H,$$
$$\frac{dE_H}{dt} = -f_H(H)E_H - \nu_H E_H + \frac{ab}{H}S_H I_V,$$
$$\frac{dI_H}{dt} = \nu_H E_H - f_H(H)I_H - \alpha I_H - \gamma I_H - rI_H,$$
$$\frac{dR_H}{dt} = \alpha I_H - f_H(H)R_H - \beta R_H,$$
$$\frac{dS_V}{dt} = \lambda_V V - f_V(V)S_V - \frac{ac}{H}S_V I_H,$$
$$\frac{dE_V}{dt} = -f_V(V)E_V - \nu_V E_V + \frac{ac}{H}S_V I_V,$$
$$\frac{dI_V}{dt} = \nu_V E_V - f_V(V)I_V. \tag{3}$$

The model (3) can be extended to a spatial model in the same ways as (2), the only difference being that (3) has more components which could have their own distinct movement patterns. Obviously, various other extensions of (2) may be needed to describe vector borne diseases with multiple serotypes (such as Dengue), with nonhuman as well as human hosts (such as West Nile), or with vertical transmission in mosquitoes (such as Rift Valley Fever.) However, the general approaches used in putting space and movement into the models will the same as for (2).

2.2. Spatial modeling

There are two distinct approaches that have been used to incorporate spatial effects into epidemiological models in general and vector-borne disease models in particular. One approach is to view space implicitly and to consider being in a location to be an attribute analogous to belonging to the group associated with that location (if an