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Tick seeking assumptions and their implications for Lyme disease predictions

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ABSTRACT

In vector-borne disease modeling, a key assumption is the host–vector interaction pattern encapsulated in the host seeking rate. Here, a model for Lyme disease dynamics with different host seeking rates is used to investigate how different patterns of tick–host interaction affect the model predictions in the context of tick-borne disease control. Three different host seeking behaviors (the frequency-dependent rate, the density-dependent rate and the Holling type 2 rate) are compared. The comparison of results illustrates not only variable relationships between rodents and tick abundance but also different implications for disease control: (i) for the model with the frequency-dependent rate, reducing rodents is always bad for containing the disease; (ii) for density-dependent or the Holling type 2 rate, reducing or increasing rodent population should be carefully considered, since large host population may facilitate the development of immature ticks, resulting in the immature tick population level so low to sustain the transmission cycle. Furthermore, we distinguish different mechanisms of dilution effects (pathogen reduction with the increasing of the host biodiversity) from different tick–host interaction patterns.

1. Introduction

Prevention and control of tick-borne diseases is important to human health, animal welfare and economics. Tick-borne encephalitis virus causes thousands of human cases of encephalitis in Europe and Asia every year (Mansfield et al., 2009; Nonaka et al., 2010), and Lyme disease remains the world's most frequently recorded vector-borne diseases in the temperate zone. More than 20,000 cases are reported in the United States each year (Kurtenbach et al., 2006; CDC, 2007), and the number of known endemic areas of Lyme disease in Canada is predicted to be acceleratingly increasing with climate change (Ogden et al., 2009). Though a variety of models have been proposed (see, for example, Caraco et al., 1998; Schmidt and Ostfeld, 2001; Ogden et al., 2005; Ostfeld, 2011; Wu et al., 2013), little has been done to describe the relationship between the model structure and predictions for Lyme disease.

In eastern and central North America, Lyme disease is caused by spirochete bacterium *Borrelia burgdorferi* and transmitted by the tick vector *Ixodes scapularis*. The transmission process of Lyme disease as a zoonotic vector-borne disease is largely affected by the interaction between the vector and its hosts. The tick vector has a wide range of hosts, adult ticks always feed on white-tailed deers while immature ticks normally feed on small mammals, with white-footed mouse as the most efficient reservoir. Thus it is naturally proposed that Lyme disease is closely tied to two host species: deers which determine tick numbers, and rodents (particularly white-footed mice) which determine tick infection (Ostfeld, 2011). Two relationships are important in the ecology and epidemiology of this complex ecological and epidemiological system. The first is the relationship between deer and tick abundance. This relationship seems to be variable, sometimes strong and sometimes weak or nonexistent (Ostfeld, 2011). Another is the relationship between the disease risk and host community diversity. Dilution effect, defined when disease frequency decreases with increasing biodiversity, and the opposite-amplification effect have been discussed in the literature (see Ostfeld, 2011; Keesing et al., 2006; Rudolf and Antonovics, 2005; Schmidt and Ostfeld, 2001, 2001; Mitchell et al., 2002; LoGiudice et al., 2003; Ostfeld and Keesing, 2000; Ostfeld and LoGiudice, 2003; Van Buskirk and Ostfeld, 1995; Ogden and Tsao, 2009). In particular, different possible mechanisms responsible for dilution effects were classified in Keesing et al. (2006). These mechanisms include encounter reduction, transmission reduction, susceptible host regulation, infected host mortality, recovery augmentation

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and vector regulation. Three of these mechanisms seem to be popular and important for the tick-borne disease transmission: encounter reduction, host regulation and vector regulation (Ostfeld, 2011). Here, we employ a mathematical dynamical modeling approach to explore how tick–host interaction patterns affect disease transmission, with Lyme disease as a case study.

As noted in Wonham et al. (2006), a model's mathematical structure should be determined by its underlying biological assumptions, and the model-based prediction is strongly influenced by the model structure. For vector-borne disease modeling, a key assumption is the host–vector interaction represented by the transmission term. Epidemiologists increasingly appreciate that the disease-transmission term may greatly affect the predicted disease transmission pattern and disease control strategies (Wonham et al., 2006). In tick-borne interaction modeling, density-dependent (Caraco et al., 1998) and frequency-dependent (Gaff and Gross, 2007) functions have been widely used. Some other more complicated functional responses, such as the one in Ogden et al. (2005), are also used. These complicated responses, as will be shown, can be considered as intermediate scenarios between the density-dependent and frequency-dependent cases and these responses can be expanded as a combination of the density-dependent and frequency-dependent forces. In dynamic modeling, prediction of long-term disease transmission dynamics and evaluation of disease control strategies are closely related to the calculation of a few indices summarizing the collective impact of model parameters and initial conditions. One important predictive index is the basic reproduction number (Hartemink et al., 2008) which, for tick population is the total number of female adult ticks produced by a single female tick during her entire reproduction period; and for Lyme disease is defined as the average number of secondary cases caused by one infectious individual placed in a population consisting entirely of susceptibles. These indices should be evaluated under different assumptions of tick–host interactions. Also important for determining the human risk of exposure to Lyme disease, as emphasized by Ostfeld (2011), is the number of nymphs responsible for the majority of Lyme disease cases, and three temporally varying measures: nymphal infection prevalence (NIP: which is the proportion of nymphs infected with *B. burgdorferi*), the density of infected nymphs (DIN) and the density of nymphs (DON). In this study, we examine how the choice of different transmission terms qualitatively and quantitatively alters the basic reproduction numbers, the time-evolution of nymphs, NIP, DIN and DON, therefore alters predicted disease transmission patterns and control implications. We hope this modeling approach can contribute to addressing the question "why the relationship between deer and tick abundance be so variable" (Ostfeld, 2011). We also show that while an excessively large rodent population size has positive effects on disease control, the conceptual mechanisms underlying various tick–host seeking patterns are different. We clarify this idea in the context of dilution and/or amplification effect. Moreover, in the case of density-dependent and Holling type 2 transmission terms, we observe both the dilution effect and amplification effect of the host community may take place. Thus, we encounter the dilemma with respect to the disease control: to reduce or to increase rodent population. Solving this dilemma relies on and thus calls for accurate formulation of the transmission patterns of the disease under consideration.

2. Effects of host seeking patterns on disease dynamics

2.1. The core model and ecological/epidemiological reproduction numbers

There are many mathematical models developed for tick-borne disease transmission dynamics, such as those in Bolzoni et al. (2012), Tagliapietra et al. (2011), Pugliese and Rosà (2008), Rosà and Pugliese (2007). Here, we adapt the simple model structure of Caraco et al. (1998) to capture some important features of the complex epidemiology of Lyme disease. Namely, we use the system

Table 1
Variables for the model system (1) and (2).

L	The total number of larval ticks
N	The total number of tick nymphs
A	The total number of adult ticks
M_I	The number of infectious rodents
N_I	The number of infectious nymphs
A_I	The number of infectious adult ticks

$$\begin{aligned}\frac{dL(t)}{dt} &= \left(bF_A - \frac{1}{K}A(t)\right)A(t) - (F_L+\mu_L)L(t),\\ \frac{dN(t)}{dt} &= F_L L(t) - (\mu_N + F_N)N(t),\\ \frac{dA(t)}{dt} &= F_N N(t) - \mu_A A(t)\end{aligned} \tag{1}$$

to describe the tick population dynamics, and formulate the system

$$\begin{aligned}\frac{dM_I(t)}{dt} &= \beta_M F_N \frac{M - M_I(t)}{M} N_I(t) - \mu_M M_I(t),\\ \frac{dN_I(t)}{dt} &= \beta_L F_L \frac{M_I(t)}{M} L(t) - (F_N + \mu_N)N_I(t),\\ \frac{dA_I(t)}{dt} &= F_N N_I(t) + \beta_N F_N (N(t) - N_I(t))\frac{M_I(t)}{M} - \mu_A A_I(t)\end{aligned} \tag{2}$$

for the disease transmission dynamics. All of the variables and parameters are presented in Tables 1 and 2. We will also adopt the parameter set from Caraco et al. (1998). Ticks can feed on various vertebrates as hosts (Mannelli et al., 2012). To illustrate our findings, we assume that the immature ticks mainly feed on rodents, while adults feed on deer. We further assume that the total rodent population and the deer population are in their demographic equilibria, fixed at the equilibrium values M and D respectively, but we assume that rodents may change epidemiological status from susceptible to infected through infection by infectious ticks. We should emphasize that the ticks host feeding rates F_L, F_N, F_A are supposed to have various forms (as detailed in Section 2.2 later). This is different from the model in Caraco et al. (1998), where only density-dependent tick seeking rate is used. Since the pathogen is maintained among the immature ticks and their hosts, the last equation of system (2) can be decoupled from the system.

Since one adult tick, with average life span $1/\mu_A$, can produce an average of bF_A larvae per unit time which will survive to the nymphal stage with the probability of $F_L/(F_L+\mu_L)$, and $F_N/(F_N+\mu_N)$ gives the nymph survival probability to adults, an adult tick can reproduce $bF_AF_L/(F_L+\mu_L)F_N/(\mu_N+F_N)/\mu_A$ adults in its life time, we naturally define the (ecological) reproduction number for the tick population as

$$R_{Tick} = bF_A \frac{F_L}{F_L+\mu_L}\frac{F_N}{\mu_N+F_N}\frac{1}{\mu_A}.$$

The tick subsystem (1) has always a tick-free equilibrium (0, 0, 0). If the reproduction number for ticks is greater than unity, then there exists a positive equilibrium, denoted by (L^*, N^*, A^*), with

$$A^* = K\left(bF_A - \frac{\mu_N+F_N}{F_L}\frac{\mu_A}{F_N}(F_L+\mu_L)\right) = KbF_A\left(1-\frac{1}{R_{Tick}}\right),$$