ORIGINAL ARTICLE

Modeling Tick-Borne Disease: A Metapopulation Model

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Abstract Recent increases in reported outbreaks of tick-borne diseases have led
to increased interest in understanding and controlling epidemics involving these
transmission vectors. Mathematical disease models typically assume constant pop-
ulation size and spatial homogeneity. For tick-borne diseases, these assumptions
are not always valid. The disease model presented here incorporates non-constant
population sizes and spatial heterogeneity utilizing a system of differential equa-
tions that may be applied to a variety of spatial patches. We present analytical
results for the one patch version and find parameter restrictions under which the
populations and infected densities reach equilibrium. We then numerically explore
disease dynamics when parameters are allowed to vary spatially and temporally
and consider the effectiveness of various tick-control strategies.

1. Introduction

Tick-borne diseases have had an increasing impact on human health during the
past hundred years (CDC, 1997; McQuiston et al., 1999). Ticks are involved in the
transmission of several human diseases including Lyme disease, Rocky Mountain
spotted fever, human babesiosis, ehrlichiosis, tick-borne relapsing fever, Colorado
tick fever and tick paralysis. The pathogens that cause these diseases include
viruses, bacteria, protozoan and ricksettia. Some tick-borne diseases have been
documented for many years while others have only recently been identified. The
spatial and temporal patterns of outbreaks of these diseases in humans tend to be
erratic.

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In general, hard-bodied (Ixodid) ticks have a 2-year life cycle. After hatching from an egg, ticks have three distinct life stages: larval, nymph and adult, and the number and distribution of blood meals required in each life stage varies between tick species. The preferred host, or hosts, are also species-dependent and can be different for each life stage, adding complexity to the study of tick-borne diseases. Rodents have often been implicated as one of the important hosts for many species of ticks at the nymph stage of life, while deer or other large mammals are preferred at the adult stage (Barbour, 1996). Tick species differ in their levels of competency for spreading different diseases (Des Vignes et al., 1999). To be a competent vector for a disease, the tick must be able to pass the disease transstadially, i.e., from one life stage to the next, or transovarially, i.e., from mother to offspring. The preferred host or hosts vary widely in reservoir competence, the probability that a vector feeding on an infected host will become infected, so the community composition of an area can greatly impact the disease prevalence (Richter et al., 2000; LoGiudice et al., 2003).

The work described here was motivated by an effort to control an outbreak of human monocytic ehrlichiosis (HME) that occurred during the summer of 1993 at a golf-oriented retirement community in Cumberland County, Tennessee (Standaert et al., 1995). HME is a recently-diagnosed tick-borne disease that has very patchy outbreaks both geographically and temporally. First diagnosed in a human in 1987 (Maeda et al., 1987), HME produces symptoms that can range from a mild illness to a severe, life-threatening disease. Treatment with antibiotics is effective when initiated early in the course of infection (University of Rhode Island Tick Research Laboratory, 2003). The rickettsial pathogen that causes HME, *Ehrlichia chaffeensis*, was discovered in 1991 (Anderson et al., 1992). The lone star tick (*Amblyomma americanum*) is suspected to be one of the vectors that transmits HME (Anderson et al., 1993; Gerhardt et al., 1998), and the white-tailed deer (*Odocoileus virginianus*) has been identified as the reservoir host for *E. chaffeensis* (Ewing et al., 1995; Lockhart et al., 1997). White-tailed deer are the preferred blood meal host for all stages of the lone star tick (Lockhart et al., 1995).

During the HME outbreak in 1993 in Cumberland County, Tennessee, eleven cases of symptomatic ehrlichiosis were identified, and more than 10% of the people in the surrounding community exhibited serologic evidence of past infections. This outbreak led to a survey of the tick populations in the area (Marsland, 1997), and tick data have been collected in the area for the past ten years using a variety of methods. In order to alleviate the incidence of HME and possibly other tick-borne diseases among the members of the retirement community, there was an effort to reduce the tick population in this part of Cumberland County. Ivermectin is a systemic acaricide that kills individual ticks feeding on deer as well as reduces the reproductive capabilities of the females that are not killed. Deer were fed acaricide-laced corn, a technique that has been proven to be effective in reducing the lone star tick population (Pound et al., 1996). The application of this control measure is necessarily periodic so as to protect the November deer hunting season. Federal law mandates that the acaricide-laced corn only be made available to deer from spring through the end of summer because the systemic acaricides remain in the deer's system for a month or two after ingestion has stopped. While

complete removal of the deer or ticks would locally eradicate the disease, this is difficult to implement unless an area is isolated from immigration. Additionally, there are potentially unknown biotic community impacts from this action.

Other disease models have been developed for ticks to address a variety of questions. Mount & Haile and other formulated a serious of computer simulations based on age-structured difference equations (Haile and Mount, 1987; Mount and Haile, 1989; Mount et al., 1991, 1993, 1997a,b). These simulations provided insight into the relationship between host density, tick density and the persistence of a tick-borne disease. Another matrix model (Sandberg et al., 1992 Awerbuch and Sandberg, 1995) investigates the seasonally varying population densities of questing ticks. Randolph (1999) suggests linking the parameters of a tick population model to remotely sensed data to estimate the spatial and temporal resolution of risk for tick-borne diseases. Many vector-borne disease models are made continuous in space by extending an aspatial model to a partial differential equation model (Radcliffe and Rass, 1984, 1985; Fitzgibbon et al., 1996). These models have provided understanding for the impacts of spatial diffusion, but the models are more appropriate for mosquitoes than ticks. Recently, Ghosh and Pugliese (2004) have developed a model that focuses on refining the temporal elements of a tick population model. Each of these models offers different insights into the complex dynamics of the tick life history. In contrast to these models, the model discussed here is a metapopulation model. This allows investigation of situations appropriately modeled by discrete spatial patches, while incorporating population dynamics within and between patches using differential equations.

The overall goal of this study is to provide general methods to evaluate strategies for predicting and possibly managing outbreaks of tick-borne diseases in a temporal and spatial context. First, using the data collected in Cumberland County, we develop a model for the dynamics of tick–deer interaction and the associated *E. chaffeensis* rickettsia. We initially consider a single spatial patch and use constant parameter values in our model to provide theoretical criteria for population stability and to determine the tick control measures that would result in elimination of the disease while maintaining the tick population. Numerical simulations of the single patch model were produced and compared to the theoretical results, and then some parameters were allowed to vary in time so that we may more realistically model the tick–deer interactions. Finally, we consider a spatially-explicit version of the model and conduct numerical simulations that explore the effectiveness of various culling strategies, including those that change with time and space. This is preliminary to future efforts to use optimal control strategies to seek locations and times for which the acaricide-laced corn might be most effectively placed.

2. Single patch model

2.1. Model equations

In this section, first we consider a model for a the dynamics of a tick-borne infection in the case of a single host, a single pathogen, and a single life stage. While

Table 1   Variable and parameters used in model.

Name	Description (all rates per month)	Value
β	Growth rate for hosts	0.2
$\hat{\beta}$	Growth rate for ticks	0.75
K	Carrying capacity for hosts per m^2	0.002 (woods), 0.001 (grass)
M	Maximum number of ticks per host	200
b	External death rate of hosts	0.01
$\hat{b}$	External death rate of ticks	0.01 (woods), 0.1 (grass)
A	Transmission rate from hosts to ticks	0.02
$\hat{A}$	Transmission rate from ticks to hosts	0.07
ν	Recovery rate of hosts	0

these assumptions do not apply to every species, the model is appropriate for the lone star tick because this tick has the same preferred host, the white-tailed deer, for all life stages, thus reducing the need to model multiple life stages. In modeling tick-borne disease, we track the host (N) and tick (V) population densities as well as the densities of individuals in each population that are infected with the disease (Y and X, respectively for hosts and ticks). Our model description follows:

$$\frac{dN}{dt} = \beta\left(\frac{K-N}{K}\right)N - bN, \tag{1}$$

$$\frac{dV}{dt} = \hat{\beta}V\left(\frac{MN-V}{MN}\right) - \hat{b}V, \tag{2}$$

$$\frac{dY}{dt} = A\left(\frac{N-Y}{N}\right)X - \beta\frac{NY}{K} - (b+\nu)Y, \tag{3}$$

$$\frac{dX}{dt} = \hat{A}\left(\frac{Y}{N}\right)(V-X) - \hat{\beta}\frac{VX}{MN} - \hat{b}X, \tag{4}$$

where the physical meaning of and numerical estimates for the parameters are given in Table 1. The above model is used to describe the interaction of lone star ticks and their hosts, the white-tailed deer, as their populations become infected with the *E. chaffeensis* rickettsia. For both the deer and tick populations, we assume no within-population structure except for infection status, and thus individuals of different locations, ages, and sizes are equivalent. We further assume that the disease is not spread from tick to tick or host to host. Also, it is not transmitted vertically from one generation to the next in either population. The disease pathogen is assumed to pass from an infected tick to a susceptible host or from an infected host to a susceptible tick only during a blood meal. The model does not allow the ticks to recover from the disease, but the hosts may recover at a given rate (ν) with no lasting immunity. It is assumed that there is no disease-specific mortality for either the hosts or the ticks.

Equation (1) describes the population dynamics of the deer, reflecting logistic growth with carrying capacity (K) and an external death rate (b) which would stem from hunting or removal from the area. It is assumed that the deer are not harmed directly by the ticks. The tick population is described in Eq. (2). Similar to

other ticks, these ticks also go through different life stages, and as they are feeding on the same host in each of their life stages, it can be considered as a whole to simplify the mathematical model, i.e., there is no need to divide the total ticks into different stages. As ticks depend on their hosts for a blood meal the tick population is restricted by a maximum number of ticks per host (M). The tick population also has an external death rate ($\hat{b}$), e.g., desiccation and acaricide impacts. Note that the birth rate of ticks incorporates not only the actual birth rate but also host-finding rate and survival rate as explained in the parameter estimation section below.

The disease model is governed by the Eqs. (3) and (4). The first term in both the Eqs. (3) and (4) represents disease transmission, assumed to be a constant times the fraction of non-infected hosts times the density of infected vectors or the fraction of infected hosts times the density of non-infected vectors, respectively. The second terms on the right-hand side of Eqs. (3) and (4) represent reductions of infected hosts and vectors as a result of competitive interactions within each species.

The time unit of this model is taken as 1 month, and the spatial unit is per m². All rates and results are per m² and per month. In the initial analysis of the model, we assume constant growth, transmission, death and recovery rates, e.g., no seasonality or externally-driven environmental conditions. However, in numerical simulations of the model we allow both temporal- and spatial-dependence for some of these parameters.

2.2. Parameter estimates

The host-finding rates and weather conditions are two major factors which influence the population dynamics of lone-star ticks the most (Davidson et al. (1994a). Host-finding rates depend on host density, vegetation and the movement patterns of hosts and ticks as well as changes or disruptions to the habitat. A particularly wet winter or dry summer can lower the survival rates of the ticks. Additionally, ticks in open grassland areas are much more susceptible to changes in temperature and humidity than ticks protected in a wooded area (Haile and Mount, 1987). Habitat type, humidity, temperature and length of daylight are correlated factors that have been shown to explain variation in questing behavior, i.e., searching for an appropriate host (Haile and Mount, 1987; Schulze et al., 2001). Thus, many model parameters for tick populations are seasonally variable. Initially, we simplify the mathematics by assuming constant (average observed) values for the parameters, and in later numerical simulations we vary key parameters to assess the impact of seasonal variability.

There is no consensus in the literature for any of the parameters of this model, so parameters are estimated by averaging values from geographic regions similar to the Cumberland County area (Table 1 summarizes our assumed values). Deer population densities can vary from 7.5 to 40.0 per km², according to the habitat type (Davidson et al., 1994b; Mount et al., 1993). For this model, we assume 20 deer per km², and thus, we use the parameter estimate $K = 0.002$ deer per m² for a wooded area. We note that a carrying capacity of a strictly grassy area was found to

be approximately half that of the wooded area (Mount et al., 1993; Davidson et al., 1994b). The maximum number of ticks per deer varies according to life stage of the tick, but the average density is estimated to be somewhere between 50 (Lockhart et al., 1996) and 400 (Haile and Mount, 1987) ticks per deer. Again, as an approximate value, $M = 200$ ticks per deer is used.

Many factors have impact on the value of $\hat{\beta}$, the vector (tick) birth rate. The annual reproductive output of ticks depends on temperature with a maximum of 5000 eggs at 23 °C, with an average closer to 1000 eggs per female. Approximately 70% of these eggs survive (Mount et al., 1993), and we assume that half of the population is female. The probability of a tick finding a host is estimated to be 0.03 per week (Haile and Mount, 1987). The off-host survival rates average between 0.7 and 0.95 depending on the habitat type (Haile and Mount, 1987), and here we use 0.85. The birth rate $\hat{\beta}$ is calculated to be the product of all of these factors, i.e., $\hat{\beta} = 0.5 \times 1000/12 \times 0.7 \times 0.03 \times 0.85 = 0.75$.

The externally-imposed death rate on ticks ($\hat{b}$) is a parameter which can be controlled in an effort to eradicate the disease, and is dependent upon control measures employed and habitat type (Mount et al., 1993). In the following sections, we explore values for this parameter that efficiently control the disease. Without external control methods, the mortality rate is estimated to be ten times greater in grassy patches than in wooded areas because the lower humidity and higher temperatures of grassy areas reduce tick survival. Thus, without the control measures, we estimate that in a wooded area $\hat{b} = 0.01$, while in a grassy area $\hat{b} = 0.1$.

The external death rate of deer is allowed to be non-zero under the assumption that the area allows hunting. This removal rate is seasonal and depends on the rate of hunting allowed in that area. The growth rate for hosts, β, was set to an arbitrary value of 0.2. The effects of varying this parameter are explored in a later section. We assume that deer do not recover from the ricksettia, i.e., $v = 0$.

It is perhaps most difficult to accurately estimate the transmission rates A and $\hat{A}$. The probability of infection from a tick–deer encounter has not been explicitly estimated (Paddock and Childs, 2003). Attempts to artificially infect deer using infected ticks or ticks with infected deer provide extremely low infection rates (Ewing et al., 1995). The results indicate that tick to deer infection is much lower than deer to tick. Possible transmission rates are estimated here as $A = 0.02$ and $\hat{A} = 0.07$ (Ewing et al., 1995).

Note that all the model's parameters must be non-negative to make biological sense. From Eq. (1), for the host population to survive we require

$$\beta > b, \tag{5}$$

and from Eq. (2), for the tick population to survive we require

$$\hat{\beta} > \hat{b}. \tag{6}$$

2.3. Equilibrium analysis

To assess the long-term population and disease dynamics, we perform an equilibrium analysis. The details of this equilibrium analysis are provided in the Appendix. The final equilibria, E_3, translate back to the original variables and parameters as

$$N^* = K\left(1 - \frac{b}{\beta}\right),$$

$$V^* = MK\left(1 - \frac{b}{\beta}\right)\left(1 - \frac{\hat{b}}{\hat{\beta}}\right),$$

$$Y^* = \frac{K(b-\beta)(\hat{\beta}^2[\beta + \nu] + A\hat{A}M[\hat{b} - \hat{\beta}])}{\hat{A}\beta(MA[\hat{\beta} - \hat{b}] + \hat{\beta}[\beta + \nu])},$$

$$X^* = \frac{K(b-\beta)(\hat{\beta}^2[\beta + \nu] + A\hat{A}M[\hat{b} - \hat{\beta}])}{A\beta\hat{\beta}(\hat{A} + \hat{\beta})}. \tag{7}$$

The final requirement in the hypothesis of Theorem A.4 in the original parameters is:

$$\hat{b} < \hat{\beta} - \frac{\hat{\beta}^2(\beta + \nu)}{A\hat{A}M}. \tag{8}$$

Note that this requirement does not depend on the host carrying capacity, K. This implies that while the carrying capacity would determine the number of ticks that can be supported in an area, it would not have any effect on the fraction of ticks that are infected. This can also be verified from (7) since the ratio of infected ticks to total ticks is independent of K. The requirement (8) may be interpreted as a type of basic reproductive rate, R_0, for this model. Equation (8) can be rearranged as follows

$$R_0 = A\hat{A}\frac{V^*}{N^*}\frac{1}{\hat{\beta}}\frac{1}{\beta + \nu} > 1. \tag{9}$$

This can now be seen to be a product of the infection rates, A and $\hat{A}$, the equilibrium ratio of ticks to hosts, $\frac{V^*}{N^*}$, the average length of survival for the tick, $\frac{1}{\hat{\beta}}$ and the average length of survival for the host combined with the recovery rate, $\frac{1}{\beta + \nu}$.

2.4. Implications of the equilibrium analysis

The hypothesis given by the equivalent Eqs. (8) and (9) can be used to determine minimal control values to eradicate the disease. Using the wooded parameter estimates in Table 1 and Eq. (8), the external death rate needed to eliminate the disease can be calculated. If $\hat{b} > 0.35$, then the disease will no longer be endemic. Table 2 gives the equilibrium values for various parameter sets. The results

Table 2 Equilibrium analysis.

Parameter values	$\hat{b} = 0.1$, $\beta = 0.2$	$\hat{b} = 0.36$, $\beta = 0.2$	$\hat{b} = 0.1$, $\beta = 0.3$	$\hat{b} = 0.16$, $\beta = 0.3$
N^* (m^{-2})	0.0019	0.0019	0.00193	0.00193
V^* (m^{-2})	0.329	0.198	0.335	0.304
Y^* (m^{-2})	0.00068	0.0	0.00013	0.0
X^* (m^{-2})	0.012	0.0	0.002	0.0

indicate that the disease persists in time if $\hat{b} > 0.35$ and dies not otherwise. Recalling that the choice of values for the host birth rate was somewhat arbitrary, we now consider the impact of raising β from 0.2 to 0.3. From Eq. (7), increasing the deer birth rate decreases the necessary amount of control. Thus, if $\hat{b} > 0.15$, the disease will be eliminated, and the deer population will increase slightly. Again as shown in Table 2, we calculated the equilibrium values for $\hat{b}$ above and below 0.15. From these calculations it is clear that an accurate estimate of the birth rate for the hosts is necessary to determine the appropriate level of control needed to eliminate the disease without decimating the tick population.

2.5. Numerical results

While the long-term goals of this study are to seek optimal temporal and spatial tick control strategies, we initially seek to verify our numerical techniques through comparison with our single-patch results and to observe the change in behavior of the model when time-dependent parameters are allowed. We solved the system of differential Eqs. (1)–(4) using a fourth order Runge-Kutta method. The code is written in C++ and uses the Runge-Kutta code from (Press et al., 1988).

2.5.1. Constant parameters

The first set of simulations involved separate runs for single patches of either wooded or grassy areas using the parameters from Table 1. Additionally, we assume there are initially 0.00025 ticks per m^2 ($V(0) = 0.00025$) and 0.00001 hosts per m^2 ($N(0) = 0.00001$) of which no hosts are infected ($Y(0) = 0$) and 1% of the ticks are infected ($X(0) = 0.01 \times V(0)$). The resulting equilibria are an exact match to those predicted by Eq. (7). The outcome in a single grass patch shows $N^* = 0.00095$, $V^* = 0.164667$, $Y^* = 0.00031437$, $X^* = 0.00493343$. This suggests that without control efforts, in a single grass patch 33% of the hosts would be infected and 3% of the ticks would be infected. Similarly, for the wooded patch, $N^* = 0.00190000$, $V^* = 0.374933$, $Y^* = 0.000775541$, $X^* = 0.0137595$, and we conclude that in this environment 41% of the hosts would be infected and 3.67% of the ticks would be infected. (Lockhart et al., 1996) find that 125 out of 300 (42%) deer test positive for ehrlichiosis while (Dawson et al., 1994) find 546 out of 1269 (43%) test positive. (Anderson et al., 1993) find that the percent of ticks that test positive for ehrlichiosis vary widely from a minimum of 0.1% to a high of 3.57%.

Later simulations will allow for time-dependent parameters, and for comparison it is helpful to consider the time-plots of the host and tick populations and the percent of each infected. Figure 1(a) shows the simulation results for the grassy
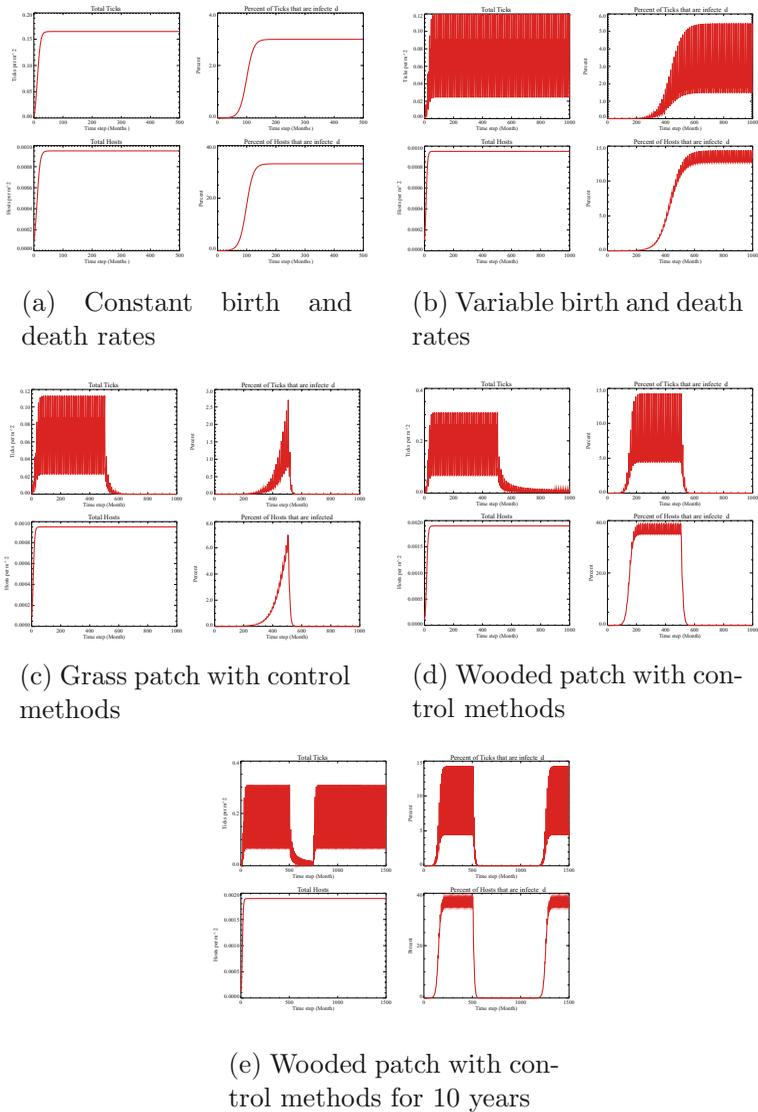
(a) Constant birth and death rates

(b) Variable birth and death rates

(c) Grass patch with control methods

(d) Wooded patch with control methods

(e) Wooded patch with control methods for 10 years

Fig. 1 For each subplot, there are four plots: the *upper left graph* shows the tick density over time, the *lower left graph* shows the host density over time the *upper right graph* shows the percent of ticks that are infected, the *lower right graph* shows the percent of hosts that are infected. (a) Constant birth and death rates in a single grass patch, all values reach a constant equilibrium value; (b) variable birth and death rates in a single grass patch, all values reach a constant cycle based on the variable birth and death rates given in Fig. 2; (c)variable birth and death rates in a single grass patch with control added in the 500th month, both the tick population and the disease are eliminated within 10 years; (d) variable birth and death rates in a single wooded patch with control added in the 500th month, while the disease is eliminated within 10 years, the tick population remains positive although greatly reduced; (e) variable birth and death rates in a single wooded patch with control for 10 years, the disease remains at negligible levels for an additional 20 years after the end of the control effort.

patch, and the results of the wooded patch are qualitatively equivalent. The equilibrium values in the grass patch for total host and tick populations are reached within 50 months, while the percent infected takes approximately 150 months to reach equilibrium. If the initial percent infected for ticks is reduced to 0.00001% ($X(0) = 0.0000001$), the system reaches equilibrium in 220 months. If the same simulation starts with one percent of the hosts infected in addition to the one percent of ticks infected, the percent infected reaches the same equilibrium values in 100 months, about half the time of the previous run. If only one percent of the hosts are infected and no ticks are infected, the same equilibrium values are attained within just over 100 months. Thus, the time to equilibrium depends on a combination of initial conditions and the parameter values for the tick population, but the system consistently achieves the equilibria predicted by the above analysis.

2.5.2. Nonconstant birth and death rates

To include seasonality in the model, the birth and death rates of the ticks are varied according to the plot in Fig. 2 which attempts to reflect the tick life cycle. Because tick off-host survival rates drop considerably as the temperature drops (Haile and Mount, 1987) and because temperature also affects other parts of the tick life history including clutch size, we assume an increase in mortality during the winter months. We assume high mortality in the middle of the summer because adults will die shortly after laying their eggs in early summer (Sonenshine and Mather, 1994) and because high temperatures themselves increase mortality. The two birth peaks reflect the two periods when ticks become active. This variation reduced the average birth rate to 0.35 and raised the average death rate to 0.23 for grass and 0.17 for wooded. The actual parameter values chosen are estimates and may need further refinement.

When the grassy and wooded patch simulations are repeated from the previous section with these new time-dependent birth and death rates, the results show that the system now cycles rather than approaching constant values (see Fig. 1(b) for the grassy patch simulation results). The percent of ticks and hosts that are infected changes markedly from the constant birth and death results. In the grass patch, the percent infected ticks now varies from 1.5 to 5.5%, which averages to the 3.0% in the constant case, but the percent infected hosts dropped from nearly 35% in the constant case to less than 15% in the varying case. The wooded patch has just the opposite effect with the percent infected hosts in the varying case remaining close to the outcome of the constant case while the percent infected ticks jumps from less than 4% to an oscillation between 4 and 14%. The system reaches this periodic yearly fluctuation after approximately 500 months. This requires much longer than the constant birth and death rate simulations because the birth rate is now lower and the death rate is higher on average. These results produce similar fluctuations to those found in field data (Marsland, 1997).

2.5.3. Nonconstant controls

The next simulation provides for nonconstant controls similar to those carried out in a field study by Marsland (1997). As mentioned in the introduction, the feeding of acaricide-laced corn to the host is necessarily periodic, and so we now alter
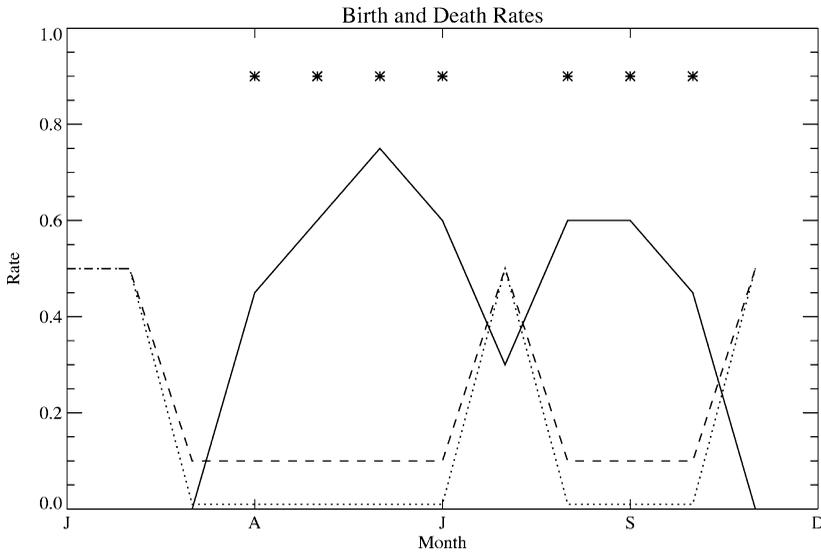
Fig. 2 To obtain more realistic tick density values, the birth and death rates are allowed to vary seasonally. The graph shows the annual cycle used for each of the habitat types. The *solid line* is the birth rate for both the wooded and grass patches. The *dashed line* is the death rate for the grass patch, and the dotted line is the death rate for the wooded patch. The increases in death rate during the winter are caused by the drop in temperatures while the increases in death rate during the summer are related to loss of adults during that time from age specific mortality. The stars mark the months of the year when R_0 is greater than 1.0 for both the wooded and grass patches.

our simulations to allow for a time-dependent control, raising the external death rate of the ticks to on average 0.75 for the months when acaricide-laced corn is available to the deer (from May through August). This systemic acaricide has a decay time as it leaves the hosts' systems at the end of summer that is not included in this model. Rather, it is projected that the death rate returns to the original value 1 month after the feeding is stopped. For each simulation, the system is allowed to come to equilibrium before the control is applied.

When the control is added to the grass patch, the result is a reduction of both the disease and the tick population to virtually zero after less than 10 years of treatments (see Fig. 1(c)). When control is added to the wooded patch, the disease is reduced to immeasurably low levels while the tick population persists (see Fig. 1(d)).

Since the wooded patch allows the reduction of the disease to extremely low levels without eradicating the disease, we ran further simulations to determine the amount of time the control has an effect. If the control is applied for less than 5 years, the disease is not reduced to the near-zero levels and returns within a few years to the pre-treatment level. However, if the control is applied for 10 years, it takes approximately 10 years after control is stopped for the disease levels to again increase. If the control is applied for 20 years, the disease will take approximately 40 years to return (see Fig. 1(e)).

3. Multiple patch model

3.1. The model

The simulations in the previous sections indicated a clear difference in how tick control should be implemented in grassy and wooded areas. The area we considered in Cumberland County (see Fig. 3), as with most areas of interest, contained a combination of grass and wooded areas. Thus, we now expand our model given in the system (1)–(4) of the tick–deer-disease interaction to a spatial model with multiple patches of different types and/or locations.

$$\frac{\mathrm{d}N_i}{\mathrm{d}t} = \beta_i \left(\frac{K_i - N_i}{K_i} \right) N_i - b_i N_i + \sum_j m_{ij} \left(N_j - N_i \right) \tag{10}$$

$$\frac{\mathrm{d}V_i}{\mathrm{d}t} = \hat{\beta}_i V_i \left(\frac{M_i N_i - V_i}{M_i N_i} \right) - \hat{b}_i V_i + \sum_j m_{ij} \left(V_j - V_i \right) \tag{11}$$

$$\frac{\mathrm{d}Y_i}{\mathrm{d}t} = A_i \left(\frac{N_i - Y_i}{N_i} \right) X_i - \beta_i \frac{N_i Y_i}{K_i} - (b_i + v_i)Y_i + \sum_j m_{ij} \left(Y_j - Y_i \right) \tag{12}$$

$$\frac{\mathrm{d}X_i}{\mathrm{d}t} = \hat{A}_i \left(\frac{Y_i}{N_i} \right) (V_i - X_i) - \hat{\beta}_i \frac{V_i X_i}{M_i N_i} - \hat{b}_i X_i + \sum_j m_{ij} \left(X_j - X_i \right) \tag{13}$$

The variables and parameters remain as in Table 1, with each patch i having unique copies of these variables and parameters identified by the subscript i. The migration term m_{ij} allows movements between patches. Given patches i and j, we define m_{ij} to be the percentage of both deer and ticks that migrate from patch i into patch j. For the purposes of this study, we assume that movement between these patches is symmetric, i.e., $m_{ij} = m_{ji}$. The migration rates for the ticks and their hosts are assumed to be equal because although ticks are capable of movement over short distances, the majority of the movement of ticks is assumed to be a result of movement of the host while the tick is attached for a blood meal.

This model allows more realistic spatial components that are comprised of grassy and wooded areas. Recall from our earlier discussion that two parameters differ between these settings: the host carrying capacity and the tick mortality rate. Because of the complexity of this multi-patch model, analytical results such as those provided for the single patch system are not easily obtained, and our analysis of this system relies on numerical results alone.

3.2. Numerical results

In the numerical simulations, we divide space into patches and assign appropriate parameter values based on habitat type to each patch. The movement parameters between patches are chosen based on geographic location of and obstacles between the patches. Simulations are first done using two patches, one each of grass and wooded, and second, using 12 patches. The 12-patch system is developed
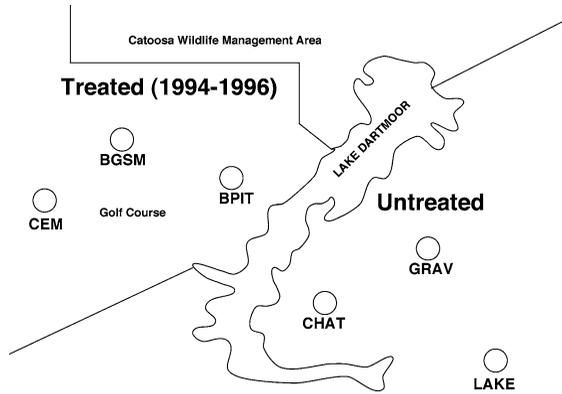
Fig. 3 Map of sites for tick collection. Data were collected in a grass area and a wooded area at each of the sites marked by a circle. Feeders were placed in the treated area to distribute the acaricide laced corn to the deer in the area. Reference: redrawn from Fig. 2.1 of Marsland (1997).

in an attempt to mimic a field experiment with six sites performed in Cumberland County (Marsland, 1997) (Fig. 3). Each of the six sites have a grass and a wooded patch, and these sites are linked according to their geographic relationship (Table 3).

3.2.1. The two-patch model

In the two-patch simulation, constant birth and death rates were assumed in each patch, and the migration rate between the two patches was varied from 0 to 100% to explore the sensitivity of the equilibrium results to this parameter. Equilibrium densities were achieved for each migration rate value. The equilibria for the wooded patch drop from the 0.38 ticks per m^2 in an isolated patch to near 0.25 ticks per m^2 when the migration rate is at 100%. In contrast, the equilibrium density for the grass patch increases from 0.17 to 0.22 ticks per m^2. The percent of ticks and hosts that are infected converge and then cross with just slightly higher percentages infected in the grass patch when migration is greater than 0.3. The majority of the change of equilibria in each patch occurs with fairly low migration rates. The

Table 3 The migration rates for the six data collection sites are determined based upon the geographic distance between the sites as well as the presence of Lake Dartmoor.

	CEM	BGSM	BPIT	CHAT	GRAV	LAKE
CEM	1.0	0.75	0.0	0.05	0.05	0.05
BGSM	0.75	1.0	0.75	0.05	0.05	0.05
BPIT	0.0	0.75	1.0	0.05	0.05	0.05
CHAT	0.05	0.05	0.05	1.0	0.8	0.8
GRAV	0.05	0.05	0.05	0.8	1.0	0.8
LAKE	0.05	0.05	0.05	0.8	0.8	1.0

Note. The migration rates between the grass and wooded patch at each site are assumed to be 100% and the migration rates below are applied to both patches at each site.

equilibria in the patches do not change rapidly when the migration rate is varied at levels greater than 0.5.

Adding seasonal variation in birth and death rates to the two-patch model, gives convergence of equilibria similar to the constant birth and death results. The densities of ticks and hosts are lower in the wooded patch and higher in the grass patch than when each patch is considered in isolation. The amount of change depends on the migration rates. The percent infected for hosts and ticks converges to similar levels, even at very low migration rates. Unlike the constant birth and death results, the difference between percent infected in the individual patches is much larger than noted above. This difference is not significant with migration rates as low as five percent (see Fig. 4(a)). The time-to-equilibrium in the grass patch with varying birth and death rates is much longer than in the wooded patch—500 months compared to 100 months, respectively. When the two are connected with migration, the time-to-equilibrium is nearly identical at approximately 200 months. Raising the migration rate to 50% will result in a slightly higher percent infected ticks and hosts in the grass patch as compared with the wooded patch. The percent infected also has a larger amplitude in the grass patch than the wooded patch as the migration rate is increased.

Now we explore the effect of various tick control strategies in the model. Figure 4(c) and (d) show the results of model runs using two patches, one wooded and one grass. Each patch uses the varying birth and death rates. Control is applied to the wooded patch only starting in the 500th month. Figure 4(c) shows the results of the model run with a migration rate between patches as 0.05. The tick populations are reduced in both patches, more significantly in the wooded patch, and the percent infected in both patches is reduced to virtually zero. Any migration rate higher than 0.05 has similar results. However, when the migration rate is lower, the results are different. Figure 4(d) shows the results when the migration rate is set to 0.01. The total tick densities react in a similar manner to the case with higher migration rates, but the disease is no longer reduced to close to zero infection in either patch. While the percent infection in the wooded patch is quite low, it is still persistent. Figure 4(b) shows the results when the control is applied in the grass patch. Unlike the case of control applied to the wooded patch, the figure shows that the tick populations and the infection are not eliminated as they were when the grass patch is isolated. This result holds true for any positive migration rate.

3.2.2. The 12 patch model

A simulation was developed for a 12 patch system designed to mimic the data collection sites used by Marsland (1997). The objective of the original study was to evaluate if feeding ivermectin treated corn to a free-ranging, unrestricted population of white- tailed deer could reduce the free-living stages of the Lone Star Tick. The treatment area of about 450 ha included a 27 hole golf course and was located between the Catoosa Wildlife Management Area and Lake Dartmoor (see Fig. 3). The untreated area was chosen south of Lake Dartmoor and was similar in size and vegetation to the treated area. Three tick sampling sites of approximately 10 ha each were selected in the treated and untreated areas. At each of the six sites,
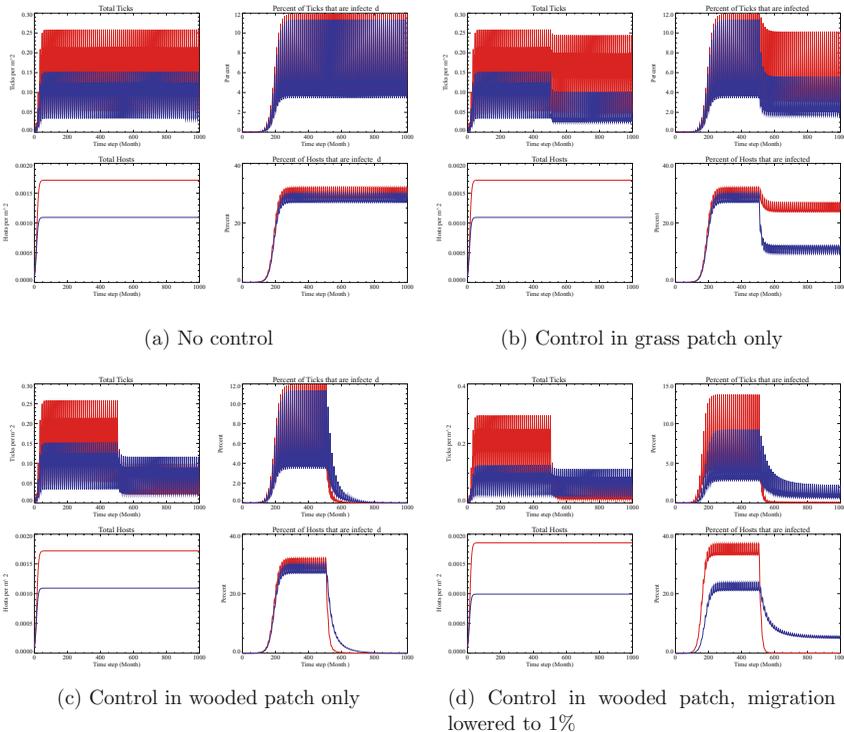
(a) No control
(b) Control in grass patch only

(c) Control in wooded patch only
(d) Control in wooded patch, migration lowered to 1%

Fig. 4 For each scenario, two patches, one grass and one wooded, are connected by migration. Both patches use varying birth and death rates for the ticks. See Fig. 1 for a description of each plot. Additionally, the red line represents a patch with the wooded parameter set, while the blue line represents a patch with the grass parameter set. (a) The grass and wooded patches are connected by a migration rate of 5% with no control, as compared with each patch model result from isolation, the density of ticks and hosts are both modified slightly while the percent infected converge much more dramatically; (b) the grass and wooded patch are connected by a migration rate of 5% with control applied in the grass patch only, the result show that both patches have sustained tick populations with reduced percent infected; (b) the grass and wooded patch are connected by a migration rate of 5% with control applied in the wooded patch only, the result show that both patches have sustained, although substantially reduced, tick populations, but the disease is successfully eliminated; (d) the grass and wooded patch are connected by a migration rate of 1% with control applied in the wooded patch. Again, the tick populations are reduced significantly, and the disease, although substantially reduced, is not effectively eliminated from either patch.

sampling was done in both grass and wooded areas. The study found a consistent decline in average number of ticks found in the treated area with a 61% reduction in adult females, 66% reduction in adult males, 80% reduction in nymphs and 44% reduction in larvae from 1994 to 1996 (Marsland, 1997).

To model this system, a grass patch and a wooded patch was assigned to each of the study sites. Migration rates between patches were estimated based on the distance between the sites and geographic boundaries (see Fig. 3). As with the field study, control in the model is applied only in the patches that represent the treated
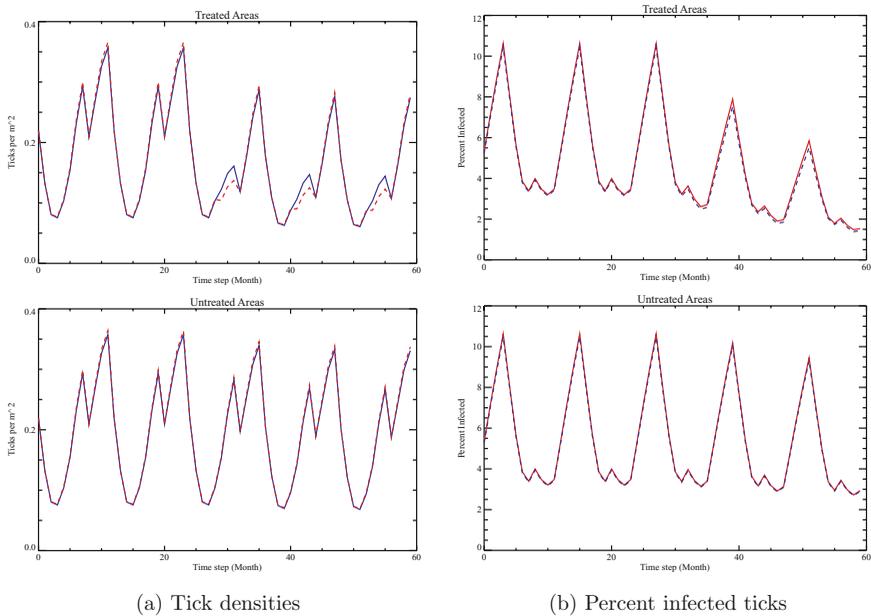
(a) Tick densities   (b) Percent infected ticks

Fig. 5 These two graphs show the results of the model using the 12 patch system developed to mimic the Fairfield Glades experimental design. The model uses varying birth and death rates. Control is applied beginning in the 500th month. For each graph, the *blue line* represents the average of the grass patches while the *dotted red line* represents the average of the wooded patches, and the top graph is the averages for the treated areas while the bottom graph is the average for the untreated areas. The months shown are 2 years prior to and three years after control is applied. The dark bar indicates the months in which control is applied in the treated areas. (a) Tick densities, the treated area experiences an approximately 60% drop in tick density when yearly averages are compared, (b) percent infected ticks, the treated areas experience significant reductions in percent infected in the second and third year of treatment while the untreated areas show very slight reductions during the same time periods.

areas study sites. The time varying parameters from Fig. 2 are used here. The total externally-imposed mortality rate for the ticks as a result of the acaricide application is estimated to be 0.75 (Marsland, 1997). Thus, we change the death rate of the ticks to 0.75 for the period May through August for every year after the system has reached equilibrium. The simulation results are shown in Fig. 5(a) and (b). These plots show only the 2 years prior to the control and the first 3 years with control to mimic the Fairfield Glade observations. The results show that the control has a very small effect on the untreated areas. However, in the treated areas, the total tick density and the percent of ticks infected drop by approximately 60% after three years of control application, approximating the field results. In the treated areas, the tick population is reduced significantly in the first year of treatment, but the population level is maintained in subsequent years. In contrast, the percent of ticks that are infected in the treated areas does not decline until the second year of treatment, and the downward trend continues in subsequent years.

4. Conclusions

Controlling ehrlichiosis or any tick borne disease is a major concern to managers of wildlife refuges, golf courses or other areas where ticks and humans interact. As human activity encroaches into previously undisturbed areas, the potential for outbreaks increases. A better understanding of potential methods of controlling the ticks and the diseases for which ticks are vectors is crucial to public health. In many circumstances it would seem the complete removal of ticks would be a valid control method, but the consequences of such an action on the remaining food chain are unknown. Thus, while ticks may be a pest, it would seem more ecologically sound to attempt to find methods to reduce the threat of disease without completely eliminating the tick populations, which also could be financially infeasible.

To this end, the results of the models described here are quite interesting. With only minor modifications of the parameters used for each patch, the results are quite different. In a single grass patch, it is impossible to remove the disease or even suppress it to low levels without removing the ticks. In a single wooded patch, it is possible to reduce the disease to extremely low levels without completely destroying the tick populations. Varying the number of years that control is applied directly affected the time to return of the disease. The model implies that control needs to be used in an area for longer than 5 years to reduce the disease to extremely low levels. Consequently, one suggestion is that there could be an optimum length of time to apply control to best quell the disease with minimum disruption of the tick populations or with minimum cost of control. A potential improvement to the control model would be to include the decay time for the acaricide in the host. The death function applied to the ticks would then be a function dependent upon the acaricide used, reflecting the appropriate values for the time during application of the acaricide and the fade out time.

Decomposing the system into patches illustrates the importance of the spatial location of control. The model shows that where control is applied can be as important as how long it is applied. At different levels of migration between wooded and grass patches, it is more effective to apply control in the wooded patch than in the grass patch. By carefully studying an area and obtaining better parameter values, a model like this could be used to determine theoretically ideal locations to apply the control measures. To achieve this, more research would be needed to improve the parameter estimates, including improved estimates of the birth and death functions. In further studies, different patches could be defined. For example, some parameter values could vary depending on the type of wooded area. Additionally, increasing the number of patches with appropriate parameters could provide better estimates for ideal locations for control application. To truly understand a large area, many thousands of patches might be necessary. This model could be used to explore the implications of patch size as well as number of patches.

Variation in the assumed migration rates between patches may be appropriate. The assumption that tick migration rates are equal to those for deer may not be accurate. Ticks only feed on deer during certain times of the year, and therefore the migration rates for ticks may need to be seasonally linked to deer movement. Ticks are attached to deer for less than a week for a bloodmeal and ticks move

much shorter distances so, as compared to the hosts, tick movement may be negligible. It would be helpful to know the distance and the speed at which individual ticks travel. Also, including information about seasonal deer movement patterns and the effects of this on the migration rate estimates could enhance model applications to particular regions.

The results of applying this model to specific geographic regions can help determine the expected results of different control programs, with evidence for the suitability of application given by the fact that the results of the model reflected the actual field results for the Fairfield Glades study. While a program may very quickly reduce the tick population, our model indicates that it may require a sustained effort to substantially reduce the percent of the ticks that are infected. By creating a set of patches that reflect an area of interest, one could explore different control options prior to field application to aid in choosing the most effective program.

Acknowledgements

Appendix: Single patch equilibrium analysis

To assess the long-term population and disease dynamics, we perform an equilibrium analysis. The mathematics is simplified by considering an equivalent non-dimensional system with six parameters rather than the original nine:

$$\frac{dn}{d\tau} = (s_0 - n)\, n \tag{A.1}$$

$$\frac{dv}{d\tau} = s_1 v \left(1 - s_2 - \frac{v}{n}\right) \tag{A.2}$$

$$\frac{dy}{d\tau} = s_3 \left(1 - \frac{y}{n}\right) x - s_4 y - yn \tag{A.3}$$

$$\frac{dx}{d\tau} = s_5 \frac{y}{n}\,(v - x) - s_1 s_2 x - s_1 \frac{vx}{n} \tag{A.4}$$

where: $n = \frac{N}{K}$, $v = \frac{V}{MK}$, $y = \frac{Y}{K}$, $x = \frac{X}{KM}$, $\tau = \beta t$, $s_0 = 1 - \frac{b}{\beta}$ $s_1 = \frac{\hat{\beta}}{\beta}$, $s_2 = \frac{\hat{b}}{\hat{\beta}}$, $s_3 = \frac{AM}{\beta}$, $s_4 = \frac{b+v}{\beta}$, $s_5 = \frac{\hat{A}}{\beta}$.

Biologically reasonable assumptions for these parameters include requiring s_1, $s_2, \ldots, s_5$ to be non-negative. From Eq. (5), for the host population to survive, we need $s_0 > 0$, and from Eq. (6) it follows that in order for the tick population to survive, we require $s_1 < 1$. These criteria are used in the following theorems analyzing equilibria for the dimensionless system. Finally, the system is limited to the region P,

$$P = \{(n, v, x, y) | n > 0, v \geq 0, x \geq 0, \text{ and } y \geq 0.\}. \tag{A.5}$$

Theorem A.1. *If $s_0, s_1, s_3, s_5 > 0$, then the region P defined in Eq. (A.5) is invariant.*

Proof: To see that n cannot become negative, note from Eq. (A.1): if $n > s_0 > 0$, then $\frac{dn}{d\tau} < 0$; if $n = s_0$, then $\frac{dn}{d\tau} = 0$; and if $0 < n < s_0$, then $\frac{dn}{d\tau} > 0$. Also, $\frac{dn}{d\tau} \to 0$ as $n \to 0$. Therefore, P is invariant with respect to n.

From Eq. (A.2) we see that as $v \to 0$, $\frac{dv}{d\tau} \to 0$, and if $v = 0$, $\frac{dv}{d\tau} = 0$. Therefore, P is invariant with respect to v.

From Eqs. (A.3) and (A.4) it follows that as $x \to 0$ and $y \to 0$, $\frac{dx}{d\tau} \to 0$ and $\frac{dy}{d\tau} \to 0$. To see this, note that: if $x \to 0$ and $y > 0$, $\frac{dx}{d\tau} = s_5 yv \geq 0$; if $y \to 0$ and $x > 0$, $\frac{dy}{d\tau} = s_3 x \geq 0$; if $x = 0$ and $y > 0$, $\frac{dx}{d\tau} = s_5 yv \geq 0$; if $y = 0$ and $x > 0$, $\frac{dy}{d\tau} = s_3 x \geq 0$; and if $x = 0$ and $y = 0$, $\frac{dx}{d\tau} = 0$ and $\frac{dy}{d\tau} = 0$. Therefore, P is invariant with respect to x and y. Thus, P is invariant.

For the system (A.1)–(A.4) there are three equilibria:

E_1:
$\quad n^* = s_0, \quad x^* = 0,$
$\quad v^* = 0, \quad\ \ y^* = 0\,;$

E_2:
$\quad n^* = s_0, \qquad\qquad x^* = 0,$
$\quad v^* = s_0(1 - s_2), \quad y^* = 0;$

E_3:
$\quad n^* = s_0 \qquad\qquad x^* = \dfrac{s_0(s_3 s_5 - s_0 s_1 - s_1 s_4 - s_2 s_3 s_5)}{s_3(s_1 + s_5)},$

$\quad v^* = s_0(1 - s_2), \quad y^* = \dfrac{s_0(s_3 s_5 - s_0 s_1 - s_1 s_4 - s_2 s_3 s_5)}{s_5(s_3[1 - s_2] + s_4 + s_0)}.$

The following three theorems explore parameter relationships for which each of E_1, E_2 and E_3 becomes a stable equilibrium for the model

Theorem A.2. *If $s_i > 0$ for $i = 0, \ldots, 5$ and $s_2 > 1$, then the equilibrium E_1*

$$n^* = s_0, \ x^* = 0,$$
$$v^* = 0, \ \ y^* = 0;$$

is a locally asymptotically stable equilibrium for the system (A.1)–(A.4).

Proof: To show local stability, the system is linearized about the equilibrium. The Jacobian matrix, J, at general point (n, v, y, x) is

$$
J = \begin{pmatrix}
s_0 - 2n & 0 & 0 & 0 \\
s_1 \frac{v^2}{n^2} & s_1(1 - s_2) - 2s_1 \frac{v}{n} & 0 & 0 \\
\frac{s_3 xy}{n^2} - y & 0 & -s_3 \frac{x}{n} - s_4 - n & s_3 \left(1 - \frac{y}{n}\right) \\
\frac{s_5 y}{n^2}(x - v) + \frac{s_1 vx}{n^2} & s_5 \frac{y}{n} - s_1 \frac{x}{n} & s_5 \frac{v}{n} - s_5 \frac{x}{n} & \frac{-s_5 y}{n} - s_1 s_2 - s_1 \frac{v}{n}
\end{pmatrix}. \quad \text{(A.6)}
$$

When the (n, v, x, y) values from equation E_1 are substituted into J the resulting matrix has eigenvalues

$$
\lambda_1 = -s_0,
$$

$$
\lambda_2 = s_1(1 - s_2),
$$

$$
\lambda_3 = -s_1 s_2,
$$

$$
\lambda_4 = -s_4.
$$

Each of these eigenvalues is negative under the given conditions on s_i, and thus E_1 is locally asymptotically stable.

Theorem A.3. *If $s_i > 0$ for $i = 0, \ldots, 5$, $s_2 < 1$ and*

$$
R = s_1(s_0 + s_4) + s_3 s_5(s_2 - 1) \geq 0 \quad \text{(A.7)}
$$

then the equilibrium E_2

$$
\begin{aligned}
n^* &= s_0, & x^* &= 0, \\
v^* &= s_0(1 - s_2), & y^* &= 0;
\end{aligned}
$$

is locally stable. This equilibrium is locally asymptotically stable if $R > 0$.

Proof: If $R = 0$, then when E_2 is substituted into J (A.6), the eigenvalues from the resulting matrix are

$$
\lambda_1 = -s_0,
$$

$$
\lambda_2 = -s_1(1 - s_2),
$$

$$
\lambda_3 = -s_0 - s_1 - s_4,
$$

$$
\lambda_4 = 0.
$$

We observe that $\lambda_4 = 0$ and the remaining eigenvalues are negative under the conditions on s_i. Thus, by Theorem 4.1 in (Brauer and Nohel, 1969), E_2 is locally stable. If $R > 0$, then when E_2 is substituted into J (A.6), the matrix has eigenvalues

$$
\lambda_1 = -s_0, \quad \text{(A.8)}
$$

$$
\lambda_2 = -s_1(1 - s_2), \quad \text{(A.9)}
$$

$$\lambda_{3,4} = \frac{1}{2}\left\{-s_0 - s_1 - s_4 \pm \sqrt{(s_0 + s_1 + s_4)^2 - 4(s_0s_1 + s_1s_4 + s_3s_5[s_2 - 1])}\right\}$$

$$(A.10)$$

Again, we see that the real parts of all eigenvalues are negative. This holds trivially for the first two eigenvalues from the assumptions. To see that $\lambda_{3,4}$ have negative real parts, let $A = -s_0 - s_1 - s_4$ and $B = 4(s_0s_1 + s_1s_4 + s_3s_5[s_2 - 1])$. Then $A < 0$ and $B > 0$ by assumption. If $A^2 - B < 0$, then $Re(\lambda_{3,4}) = A/2 < 0$. Or, if $A^2 - B > 0$, then since $B > 0$ and $A < 0$, $\sqrt{A^2 - B} < -A$.
Therefore, $A + \sqrt{A^2 - B} < 0$ and $A - \sqrt{A^2 - B} < 0$, and $Re(\lambda_{3,4}) < 0$. Since the real parts of all eigenvalues are negative, E_2 is locally asymptotically stable.

Theorem A.4. *If $s_i > 0$ for $i = 0, \ldots, 5$, $s_2 < 1$ and*

$$R = s_1(s_0 + s_4) + s_3s_5(s_2 - 1) < 0 \qquad (A.11)$$

then equilibrium E_3

$$n^* = s_0 \qquad x^* = \frac{s_0(s_3s_5 - s_0s_1 - s_1s_4 - s_2s_3s_5)}{s_3(s_1 + s_5)},$$

$$v^* = s_0(1 - s_2), \quad y^* = \frac{s_0(s_3s_5 - s_0s_1 - s_1s_4 - s_2s_3s_5)}{s_5(s_3[1 - s_2] + s_4 + s_0)}.$$

is a locally asymptotically stable equilibrium for the system (A.1)–(A.4).

Proof: As before, we substitute E_3 into the Jacobian matrix (A.6) and consider the resulting eigenvalues:

$$\lambda_1 = -s_0, \qquad (A.12)$$

$$\lambda_2 = -s_1(1 - s_2), \qquad (A.13)$$

$$\lambda_{3,4} = \left\{s_3(s_2 - 1)([s_1 + s_5]^2 + 2s_0s_5 + s_3s_5[1 - s_2] + 2s_4s_5) - s_5(s_0 + s_4)^2\right.$$

$$\pm \sqrt{4(s_0 + s_4 + s_3[1 - s_2])^2(s_1 + s_5)^2(s_0s_1 + s_1s_4 + s_3s_5[s_2 - 1])}$$

$$\overline{+ (s_3[s_2 - 1][s_1 + s_5^2 + 2s_0s_5 + s_3s_51 - s_2 + 2s_4s_5] - s_5[s_0 + s_4]^2)^2}\left.\right\}$$

$$\times \left\{(s_0 + s_4 + s_3[1 - s_2])(s_1 + s_5)\right\}^{-1}. \qquad (A.14)$$

The first two eigenvalues are negative from the assumptions on s_i. With additional computations, we show that $Re(\lambda_{3,4}) < 0$. Let

$$A = s_3(s_2 - 1)([s_1 + s_5]^2 + 2s_0s_5 + s_3s_5[1 - s_2] + 2s_4s_5) - s_5(s_0 + s_4)^2,$$

$$B = 4(s_0 + s_4 + s_3[1 - s_2])^2(s_1 + s_5)^2(s_0s_1 + s_1s_4 + s_3s_5[s_2 - 1]),$$

and

$$C = (s_0 + s_4 + s_3[1 - s_2])(s_1 + s_5).$$

Then

$$\lambda_{3,4} = \frac{A \pm \sqrt{A^2 + B}}{C}. \tag{A.15}$$

Since $s_2 < 1$, $C > 0$. Also, $s_3 > 0$, $s_2 < 1$, $([s_1 + s_5]^2 + 2s_0s_5 + s_3s_5[1 - s_2] + 2s_4s_5) > 0$ and $s_5(s_0 + s_4)^2 > 0$, which together imply that $A < 0$. If $A^2 + B < 0$, then $Re(\lambda_{3,4}) = A/C < 0$. Now consider the case where $A^2 + B > 0$. Since $4(s_0 + s_4 + s_3[1 - s_2])^2(s_1 + s_5)^2 > 0$, and $(s_0s_1 + s_1s_4 + s_3s_5[s_2 - 1]) < 0$ by (A.11), therefore, $B < 0$.

Thus, $A^2 + B < A^2$ and $A < 0$ implies that $\sqrt{A^2 + B} < -A$. It follows that $A + \sqrt{A^2 + B} < 0$ and obviously $A - \sqrt{A^2 + B} < 0$. Therefore, $Re(\lambda_{3,4}) < 0$.
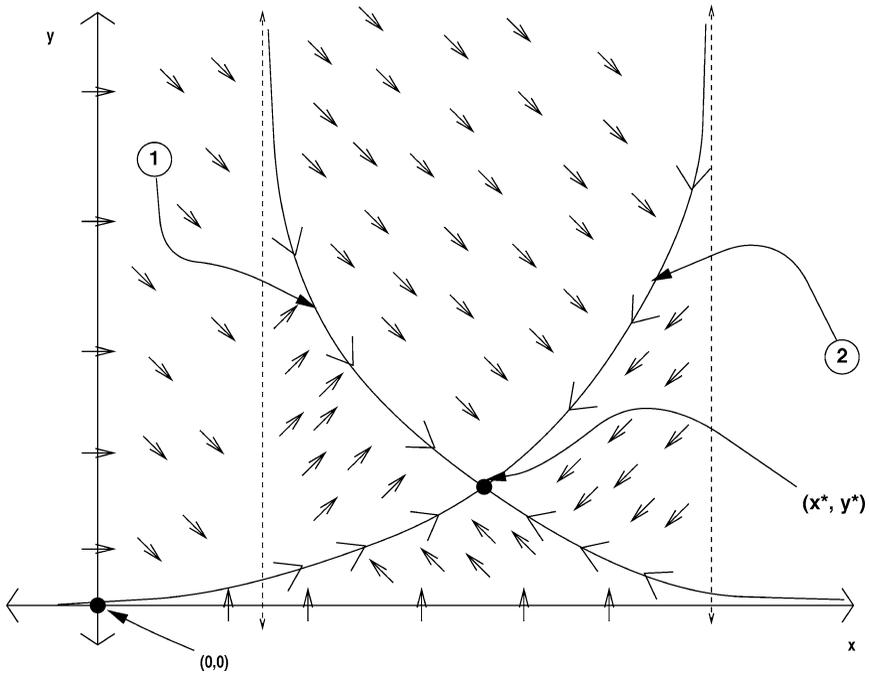So, the real parts of every eigenvalue are negative, and therefore, E_3 is locally asymptotically stable.

Fig. 6 Phase Diagram for $x(\tau)$ and $y(\tau)$ with $s_0 > 0$, $s_2 < 1$ and $\frac{s_0s_3s_5}{s_1s_2s_4 + s_0s_2s_3s_5} > 1$. Equation 1 is $y = \frac{s_3xn}{s_3x - s_4n - n^2}$. Equation 2 is $y = \frac{s_2xn + s_1^2xv}{s_1s_5(v - x)}$

The results of Theorem A.4 are illustrated in Fig. 6, the phase plane diagram for y versus x under the hypotheses of Theorem A.4 Additional numerical results indicate that E_3 is globally stable under the hypotheses of Theorem A.4, but global stability has yet to be proven analytically for the four-dimensional system.

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