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The influence of host competition and predation on tick densities and management implications

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Abstract Host community composition and biodiversity can limit and regulate tick abundance which can have profound impacts on the incidence and severity of tick-borne diseases. Our understanding of the relationship between host community composition and tick abundance is still very limited. Here, we present a novel mathematical model of a stage-structured tick population to study the influence of host behaviour and competition in the presence of heterospecifics and the influence of host predation on tick densities. We examine the influence of specific changes in biodiversity that modify the competition among and the predation on small and large host populations. We find that increasing biodiversity will not always reduce tick populations, but depends on changes in species composition affecting the degree and type competition among hosts, and the host the predation is acting on. With indirect competition, tick densities are not regulated by increasing biodiversity; however, with direct competition, increased biodiversity will regulate tick densities. Generally, we find that biodiversity will regulate tick densities when it affects tick-host encounter rates. We also find that predation on small hosts have a limited influence on reducing tick populations, but when the predation was on large hosts this increased the magnitude of tick population oscillations. Our results have tick-management implications: while controlling large host populations (e.g. deer) and adult ticks will decrease tick densities, measures that directly control the nymph ticks could also be effective.

Keywords Stage-structured model · Biodiversity · Competition · Predation · Tick population management · Tick-borne zoonoses

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Introduction

Most vectors of zoonotic pathogens, diseases normally present in wildlife that can be transmitted to humans, are host generalists (Keesing et al. 2009). Recent studies have suggested that high biodiversity in the host community can prevent the emergence and spread of zoonoses (Ostfeld and Keesing 2000). Hosts form the vector's food source; indeed, the presence or absence of particular hosts will determine vector densities (Sonenshine 2005). However, the vector's hosts will be influenced by the other species present in their ecosystem, which may compete with them for resources or prey on them. As a result, the presence of host competition or predation can potentially regulate the populations of the hosts, and thus regulate vector populations. Our objective is to gain an understanding of how

host biodiversity and the interactions within a complex host community may limit and regulate vector abundance. In this paper, we describe a stage-structured model of vector dynamics. We use the model to examine how vector abundance is influenced by specific changes in biodiversity that affect competition among and predation on the vector's host population.

We focus our study on the disease vector: ticks. Ticks can transmit numerous zoonoses (Gratz 1999; Goodman et al. 2005; Jongejan and Uilenberg 2004), of which Lyme disease is perhaps the most well known. Lyme disease, caused by the spirochete *Borrelia burgdorferi s.l.* and present in North America, Europe and Asia (Barbour and Fish 1993; Ogden et al. 2008; Sperling and Sperling 2009), is a debilitating neurodegenerative disease with over 20,000 new cases reported per year in the USA (Hanincova et al. 2006). However, ticks are also the vectors of many other diseases such as Rocky Mountain fever, Babesiosis, Ehrlichiosis, Tick-Borne Encephalitis (Walker 1998; Labuda and Nuttall 2004). Here, we focus on hard ticks (e.g. *ixodes spp., Dermacentor spp.*), rather than soft ticks (e.g. *Argas spp., Alveonasus spp.*); hard ticks are diurnal and search for hosts in the environment, while soft ticks are nocturnal and search for hosts in their nests or burrows (Oliver 1989; Needham and Teel 1991). Depending on the life-stage, ticks normally feed on either small hosts (e.g. deer mice, *Peromyscus mainculatus*, or chipmunks, *Tamias townsendii*) during the larval and nymphal life- stages, and, large hosts (e.g. deer, *Odocoileus hemionus*) during the adult life-stage. However, ticks are opportunistic feeders that attempt to feed on any animal they encounter, they have been reported on more than 1000 species of mammals, birds and reptiles (Ostfeld and Keesing 2000).

One way in which host biodiversity can potentially have an impact on tick abundance is through tick-host encounter rates. The hosts provide the blood meal required for ticks to molt to their next life-stage and the feeding success and survival of ticks varies between host species (Keesing et al. 2009). Recent studies have begun to provide evidence that disease vectors can elicit host choice and preference. Mosquitos are an example of such a vector, where host preference is evident from patterns in feeding indices which could not be simply explained by random foraging and host availability alone (Lyimo and Ferguson 2009). In ticks, data suggests a preference for opossums and squirrels over mice (Keesing et al. 2009). Likewise, there is evidence for hosts displaying behavioural changes in response to the presence of heterospecifics, hosts of different species, which also impacts on vector-host encounter rate. For instance, deer mice, a host for larval and nymphal stage ticks, have been found to change their foraging behaviour in the presence of other competitors (Davidson and Morris 2001). Similarly deer, a host for adult ticks, changes their behaviour when other large hosts are present (Hobbs et al. 1996; Latham 1999). Tick-host encounters can also increase if hosts show more clumping in the presence of heterospecifics (Keesing et al. 2006). These behaviour modifications are examples of *direct* host competition, whereby hosts modify their behaviour in the presence of heterospecifics. Host biodiversity also effects indirect competition between hosts. We use the term *indirect* competition to refer to competition between hosts that simply effects their relative abundance. Hosts competing indirectly do not modify their encounter rate with ticks based on the composition of the ecosystem, which is in contrast to direct competition. There is mixed evidence for indirect competition. Chipmunks and mice are both small granivorous rodents so are likely to compete with each other for resources; however, some recent empirical evidence puts this into question (Brunner et al. 2013), and yet it is indirect competition that is commonly used in theoretical studies of tick dynamics in multi-host systems.

Changes to ecosystem biodiversity through the introduction of predators can also effect the vector-host system. In particular, high levels of predation can induce population fluctuations in the host (Ostfeld and Keesing 2000; Ostfeld and Holt 2004). Host fluctuations can also be a result of bottom-up and environmental processes such as masting events (Giardina et al. 2000). Both the behavioural responses of hosts to heterospecifics and the fluctuating host populations associated with high predator abundance mean that increasing the abundance of any individual host species may not have a simple additive effect on tick abundance. Indeed, an empirical study by Keesing et al. (Keesing et al. 2009) found removal of hosts from a community could increase tick numbers, and that host identity may be key to understanding this. Here, we address this knowledge gap and study the relationship between tick abundance and host behaviour and abundance in the presence of heterospecifics.

Most previous studies, both empirical and theoretical have focussed on the dynamics of tick-borne pathogens and given less consideration to how host community composition influences tick abundance. These works have examined the transmission dynamics of tick-borne zoonoses using stage-structured models of the different tick life-stages and SIR epidemiological models of the disease (e.g. Caraco et al. 1998; Mwambi 2002; Randolph and Rogers 1997; Rosá et al. 2003; Stanko et al. 2007). Using this approach, studies have gained insights on how transmission dynamics are affected by factors such as climate and seasonality (Ghosh

and Pugliese 2004; Brownstein et al. 2003; Ogden et al. 2006) or metapopulation and spatial dynamics (Caraco et al. 2002; Gaff and Gross 2007). But, while some works have examined multi-host systems (Norman et al. 1999; Schmidt and Ostfeld 2001; Dobson 2004), they were interested in the abundance of pathogens and did not include predation, nor did they distinguish between the different hosts of the tick life-stages (i.e. small hosts vs. large hosts; but see (Ogden et al. 2005) for the influence of only predation, though without host competition).

Recent studies have begun to consider the impact of variation in host-vector encounter rates. Wonham et al. (2006) demonstrated that the choice of transmission term in their west Nile virus models, equivalent to a combined feeding and encounter rate in the tick setting, could both qualitatively and quantitatively alter predictions. Lou and Wu (2014) considered the role of frequency-dependent, density-dependent and Holling type II vector-host contact rates in their model of the tick life-cycle. Frequency-dependent contact is independent of host density and as such tick densities are unaffected by host densities. In contrast, density-dependent contact rates depend linearly on host densities. Density-dependent contact is used in the majority of tick models in the literature, with a fixed transmission probability per contact (here, transmission means the transmission of a blood meal from the host to the vector). Density-dependent contact is a good model when hosts compete indirectly, such that host abundance is the soul outcome of competition. However, none of these previous investigations have taken into account when host behaviour and host-vector encounter rates are modified by the presence of heterospecifics. Depending on the effect of heterospecifics on a particular host species, their presence may increase or decrease the contact rate between the host and vector. We refer to this as an effect of *direct* competition.

Here, we take a phenomenological approach to examining how biodiversity can regulate tick density through ecological processes. Given the variety of hosts that ticks can feed on and the limited number of studies of host behavioural responses to heterospecifics that currently exist in the literature, we chose not to model the host population explicitly, but instead we implicitly assume hosts are either at their demographic equilibrium or fluctuating. By not explicitly modelling host demographic processes, but merely describing the final host density, we have the flexibility to explore consequences of host competition by varying the choice of function describing vector-host contact rates, accounting for host behavioural responses to the other species in the ecosystem without explicitly modelling the complex ecosystem. We can also account for the effects of biodiversity that result in increased or decreased predation on tick hosts by describing host densities by fluctuating functions, but without specifying the detailed biological interactions that give rise to these fluctuations. In the model, we consider two types of small host and two types of large host, which is the minimum needed to model the behavioural effects of competition between small hosts and between large hosts. This approach to studying the effects of biodiversity on tick abundance has the virtue of illuminating how sensitive tick dynamics may be to these features. In the discussion, we return to the role of ticks in the spread of zoonoses, which allows us to more readily relate our findings to those in the literature. We discuss the implications of our findings on the pathogen basic reproduction number and on the nymphal infection prevalence, the two measures of disease risk commonly applied to study the tick-borne zoonosis, lyme disease.

Stage-structured tick model

We develop a stage-structured model of the tick life-cycle based on their ecology and life history, using empirical field data from the previous works (Ogden et al. 2005; Caraco et al. 2002; Gaff and Gross 2007; Perkins et al. 2006; LoGiudice et al. 2003; Giardina et al. 2000). The model accounts for the questing life stages of the tick that are dependent on obtaining a host blood meal: larva (x_1), nymph (x_2) and adult (x_3). Questing ticks are not attached to a host, but are dormant or searching for one to attach to. Once a tick finds and attaches itself to a host, it may take up to 1–3 days to obtain a blood meal, after which it detaches from the host and molts into the next stage (Needham and Teel 1991). As our focus is on hard ticks (e.g. *Ixodes spp.* and *Dermacentor spp*), we model ticks such that they require only a single blood meal to molt to the next stage (Sonenshine 2005). To study the effects of small host biodiversity, we make the assumption that tick larvae and nymphs can parasitise either their most common small host, H_1 (i.e. deer mice or white-footed mice), or an alternative small host, H_2 (e.g. chipmunks and birds). Similarly, we make the assumption that adult ticks can either parasitise their most common host H_3 (i.e. deer) or an alternate large host H_4 (e.g. raccoons, cattle and horses). We make the simplifying assumption that ticks do not explicitly regulate the density of their hosts, while the hosts can regulate the tick numbers. A resent study on white-footed mouse survival supports this assumption (Hersh et al. 2014).

Our assumption allows us to consider the host dynamics independently of the ticks and so we do not explicitly model the hosts. Instead, we assume host density is either a constant or an oscillating function of time. Oscillations describe the temporal effect of predation pressure or environmental conditions on host density.

We describe the tick population with three ordinary differential equations corresponding to the three tick life-stages:

$$\boxed{\text{Larvae}} \quad \frac{dx_1}{dt} = \overbrace{-\mu_1 x_1}^{\text{natural death}} + \overbrace{\beta_1(\sigma_3(p_L)H_3(t)\lambda_{3,3} + \sigma_4(1-p_L)H_4(t)\lambda_{3,4})\frac{x_3}{a_3+x_3}}^{\text{tick larvae produced by adults}} - \overbrace{(\sigma_1(p_s)H_1(t)\lambda_{1,1} + \sigma_2(1-p_s)H_2(t)\lambda_{1,2})\frac{x_1}{a_1+x_1}}^{\text{tick nymphs produced by larvae}}, \tag{1}$$

$$\boxed{\text{Nymphs}} \quad \frac{dx_2}{dt}Z = \overbrace{-\mu_2 x_2}^{\text{natural death}} + \overbrace{\beta_2(\sigma_1(p_s)H_1(t)\lambda_{1,1} + \sigma_2(1-p_s)H_2(t)\lambda_{1,2})\frac{x_1}{a_1+x_1}}^{\text{tick nymphs produced by larvae}} - \overbrace{(\sigma_1(p_s)H_1(t)\lambda_{2,1} + \sigma_2(1-p_s)H_2(t)\lambda_{2,2})\frac{x_2}{a_2+x_2}}^{\text{tick adults produced by nymphs}}, \tag{2}$$

$$\boxed{\text{Adults}} \quad \frac{dx_3}{dt} = \overbrace{-\mu_3 x_3}^{\text{natural death}} + \overbrace{\beta_3(\sigma_1(p_s)H_1(t)\lambda_{2,1} + \sigma_2(1-p_s)H_2(t)\lambda_{2,2})\frac{x_2}{a_2+x_2}}^{\text{tick adults produced by nymphs}} - \overbrace{(\sigma_3(p_L)H_3(t)\lambda_{3,3} + \sigma_4(1-p_L)H_4(t)\lambda_{3,4})\frac{x_3}{a_3+x_3}}^{\text{tick larvae produced by adults}}. \tag{3}$$

The three tick stages undergo natural mortality at rate μ_i. After each blood meal, a new tick stage is produced: new larvae are produced from the eggs laid by adult ticks; new nymphs are produced from larvae, and new adults are produced from nymphs; β_i is the number of stage i ticks produced by a tick in the previous stage. For example, β_1 is the number of new larvae produced per fed and mated adult. Hence, development from one stage to the next gives rise to a loss and production term for each equation. The transition from a larvae to nymph and nymph to adult produce at most a single new stage, while the adult is the only stage that can produce eggs and hence multiple larvae. The final term in Eq. 3 accounts for the fact that adult ticks can only produce one batch of eggs in their lifetime. Once an adult tick has found a blood meal and mated the females will overwinter and lay their eggs the following spring and will no longer be contributing to the numbers of questing adults (Ostfeld and Keesing 2000).

Each tick stage i obtains its blood meal from its preferred or alternate host, H_n, where a host can carry an average of $\lambda_{i,n}$ stage i ticks per time unit. Since each host can carry a maximum number of ticks the production terms saturate with a type II functional response in tick density (Brunner and Ostfeld 2008), where a_i is half the maximum number of stage i ticks per hectare. The full list of parameters and their values are summarised in Table 2.

Implicit modelling of host competition The probability that larvae find and feed on host n is given by $\sigma_n(y)$, where y is the proportion of small (large) hosts that are of type H_n and host n is a small (large) host. Models in the literature often make the simplifying assumption that $\sigma_n(y)$ is a constant (e.g. Norman et al. 1999; Sandberg and Awerbuch 1992). We, however, include the effects of host competition by relaxing this assumption and allowing the feeding probability to depend on the relative abundance (y) of a given host. By varying the relative abundance of either small (large) host and keeping the total number of small (large) hosts fixed, we simulate the effects of competition. We summarise our choices of $\sigma_n(y)$ in Table 1.

When $\sigma_n(y)$ is a constant ($\sigma_n(y) = 1$), the hosts do not affect each others behaviour and only complete for resources, this implies *indirect competition*, such that the difference in abundance between hosts reflect their ability to forage (Wooton 1994). In this case, the ticks' probability of finding and feeding on a host is not affected by

Table 1 Functional forms of $\sigma_n(y)$, where y is the proportion of small (large) hosts that are of type H_n, where host n is a small (large) host. Thus, for $n = 1$, then $y = p_s$, while for $n = 2, 3$ and 4 then y is $1 - p_s$, p_L and $1 - p_L$, respectively

Competition	Probability of finding and feeding on host n	Notes
Indirect	$\sigma_n(y) = 1$	
Direct	$\sigma_n(y) = \dfrac{\phi_n y}{\phi_n y + (1-\phi_n)(1-y)} = y$	$\phi_1 = 1 - \phi_2$ and $\phi_3 = 1 - \phi_4$
(no behavioural difference among hosts, $\phi_n = 0.5$)		
Direct	$\sigma_n(y) = \dfrac{\phi_n y}{\phi_n y + (1-\phi_n)(1-y)}$	$\sigma_1(y) = 1 - \sigma_2(1-y)$ and
(behavioural difference among hosts, $\phi_n \neq 0.5$)		$\sigma_3(y) = 1 - \sigma_4(1-y)$

the relative composition of the host community and the term describing tick-host interaction is simple mass action (density-dependent contact). Finding the host is only conditional on its presence and so if the tick encounters the host it feeds with probability 1.

When we have *direct competition* between hosts, tick-host encounter rates can depend on the relative abundance of the hosts in a nonlinear manner and the probability of finding and feeding on host n is given by

$$\sigma_n(y) = \frac{\phi_n y}{\phi_n y + (1-\phi_n)(1-y)}, \tag{4}$$

where ϕ_n is the probability of encountering host n as opposed to encountering the alternative host of the same type (small/large). Note $\phi_1 = 1 - \phi_2$ and $\phi_3 = 1 - \phi_4$. The parameter ϕ_n reflects behavioural differences of host n in response to heterospecifics. Deer mice have been found to change their foraging behaviour in the presence of other competitors (Davidson and Morris 2001). When more hosts of one type are present, the probability of encountering the other hosts may be much lower or higher than predicted by relative abundance alone, since the behaviour of one host may change in the presence of the more abundant competitor; hence, differences in the hosts' relative abundances would lead to a nonlinear relationship in the ticks' host-finding probability, similar to the relationship proposed by Rosá et al. (2003). Equation 4 describes direct competition, since the hosts' behaviour and ability to forage for resources, and hence contact ticks, are affected by the presence or absence of the other host and competitor. In particular, $\sigma_1 + \sigma_2 = 1$ (similarly $\sigma_3 + \sigma_4 = 1$) so the probability of feeding on host 1 is determined by the probability of feeding on host 2, which is in contrast to indirect competition. When $\phi_n = 0.5$, there is an equal probability of the ticks encountering either small (large) hosts and $\sigma_n(y) = y$. When $\phi_n > 0.5$ ticks have a higher probability of encountering H_n than would be suggested by relative abundance alone; with a value of $\phi_n < 0.5$ ticks have a lower probability of encountering H_n. We denote $p_s = H_1/(H_1 + H_2)$ as the proportion of small hosts that are of type H_1. Similarly, we denote $p_L = H_3/(H_3 + H_4)$, the proportion of large hosts of type H_3. Thus, in Eq. 4, y is chosen to be p_s, $(1 - p_s)$, p_L or $1 - p_L$ for the cases $n = 1, 2, 3$ and 4, respectively.

Implicit modelling of predation or seasonality To include the effects of predation on the tick hosts, we account for the presence of predators through the dynamics of the host term $H_n(t)$. Assuming Lotka—Volterra predator-prey interactions in the ecosystem, we model the host population, $H_n(t)$, as a constant or a periodic function depending on the predator, prey (host) species and the environment we wish to describe. When predation pressure is low, the host population is constant, and when predation pressure is high, the host population is assumed to be oscillating, where the amplitude of the oscillations reflects the intensity of predation. For temporally oscillating host populations, we use a cosine function.

Since we consider small and large hosts separately, when we discuss biodiversity of small hosts, we are referring to a population of two 'species', H_1 and H_2 and so diversity is maximised when $p_s = 0.5$. As p_s moves away from 0.5, in either direction, diversity declines and reaches a minimum at $p_s = 0$ or 1. The same argument applies to large host diversity where p_L is used as a proxy for diversity.

Analytical results

In this section, we consider the case when H_n is constant for all n. We calculate general analytical conditions for tick population persistence and cyclic dynamics. These both have management implications that are further examined in our numerical analysis: persistence criteria can be used to identify conditions where ticks may be eradicated, while

criteria for cyclic behaviour can be used to identify conditions where tick population dynamics are unstable and potentially vulnerable to interventions. To analyse the model, we introduce some simplifying notation. Let the coefficients in front of the second term of equation i be denoted by α_i and the coefficients in front of the third term be denoted by γ_i. Thus (1)–(3) can be rewritten as:

$$\dot{x_1} = -\mu_1 x_1 + \frac{\alpha_1 x_3}{a_3 + x_3} - \frac{\gamma_1 x_1}{a_1 + x_1}, \tag{5}$$

$$\dot{x_2} = -\mu_2 x_2 + \frac{\alpha_2 x_1}{a_1 + x_1} - \frac{\gamma_2 x_2}{a_2 + x_2}, \tag{6}$$

$$\dot{x_3} = -\mu_3 x_3 + \frac{\alpha_3 x_2}{a_2 + x_2} - \frac{\gamma_3 x_3}{a_3 + x_3}\,. \tag{7}$$

We note that

$$\gamma_1 = \alpha_2/\beta_2 \geq \alpha_2 \quad \text{and} \quad \gamma_2 = \alpha_3/\beta_3 \geq \alpha_3, \tag{8}$$

because each larvae produces at most one nymph and each nymph produces at most one adult. Similarly,

$$\gamma_3 = \alpha_1/\beta_1 \leq \alpha_1$$

as each adult female successfully produces of the order of 350 female eggs.

As the model is stage-structured, it can be easily seen that the only axial equilibrium is the trivial (0, 0, 0) equilibrium. To examine the stability of this equilibrium and address the question of population persistence, we consider the equation for the total tick population, $x = x_1 + x_2 + x_3$. Thus,

$$\dot{x} = -\mu_1 x_1 - \mu_2 x_2 - \mu_3 x_3 + (\alpha_2 - \gamma_1)\frac{x_1}{a_1 + x_1} + (\alpha_3 - \gamma_2)\frac{x_2}{a_2 + x_2} + (\alpha_1 - \gamma_3)\frac{x_3}{a_3 + x_3}\,. \tag{9}$$

All of the terms in Eq. 9 are negative except the final term, which saturates for sufficiently large x_3. As the final term is bounded while the first three terms grow linearly with x, for all sufficiently large x, we have $\dot{x} < 0$ and hence the population is bounded and the system is dissipative, in other words the tick population is self-regulated. The necessary condition for persistence is $\alpha_1 > \gamma_3$. This is equivalent to requiring that, on average, adult ticks produce more than one surviving offspring. A sufficient condition for persistence can be obtained from standard stability analysis and applying the Routh-Hurwitz criteria (Murray 1989). Persistence occurs if

$$(\mu_1 a_1 + \gamma_1)(\mu_2 a_2 + \gamma_2)(\mu_3 a_3 + \gamma_3) - \alpha_1\alpha_2\alpha_3 < 0\,. \tag{10}$$

Equation 10 is a local stability condition. Rearranging (10) gives us an expression for R_{tick}, the average number of female adult ticks produced by a single female during her lifetime,

$$R_{\text{tick}} = \frac{\alpha_1}{(\mu_3 a_3 + \gamma_3)}\,\frac{\alpha_2}{(\mu_1 a_1 + \gamma_1)}\,\frac{\alpha_3}{(\mu_2 a_2 + \gamma_2)}\,. \tag{11}$$

The first term in Eq. 11 corresponds to the average number of larvae produced by an adult female over her lifetime and the next two terms correspond to the respective probabilities that a larvae will survive to become a nymph and a nymph will survive to become an adult. Biologically, this is not particularly informative as it involves all of the model parameters and it is difficult to discern the relative importance of any particular process.

By a novel application of compound matrix theory and constructing Lyapunov functions (see Appendix A for details) global stability criteria can be found which establish when population persistence is not possible. These conditions involve less parameters and are therefore biologically more informative. In a similar manner, we can construct criteria for when the system does not exhibit periodic orbits. Table 3 summarises these results.

Criteria A and B describe conditions for the persistence of tick populations. Criteria A can be rearranged to see that it corresponds to the average number of larvae produced by an adult female over her lifetime being less than 1. It can be reexpressed in the original parameters as $\mu_3 > \beta_1[\sigma_3(p_L)H_3\lambda_{3,3} + \sigma_4(1 - p_L)H_4\lambda_{3,4}]/a_3$. From this, reducing numbers of large hosts (H_3 and H_4) can lead to tick eradication; however, the reduction would need to be of the order of a thousand fold reduction in deer density to around 0.0002 deer per hectare. On the other hand, if we consider the alternative criteria B, then of the three inequalities $\mu_1 > \frac{\alpha_1}{a_3}$ is the most difficult inequality to satisfy due to the high larval production by adults (α_1). But, interestingly, if larval mortality is sufficiently high and small host density is low, the three conditions that constitute criteria B can be satisfied and tick eradication is possible. This finding is in line with Loguiudice (LoGiudice et al. 2008) who suggest there are frequent tick extinctions in small habitat fragments where one might expect tick mortality to be high and hosts to be present in low densities.

Criteria C and D give the conditions for when tick population cycles are absent and the dynamics are stable. As with criteria A, criteria C is difficult to satisfy and would require an extreme reduction in the number of large hosts. Criteria D describes more practical conditions for stable tick populations, which is achieved when either small hosts are reduced or by increasing larval and nymph mortality.

In addition to the extinction equilibrium, the model has a coexistence equilibrium. However, it is not possible to derive an explicit analytical expression for this equilibrium; it can be found by numerically solving an implicit equation.

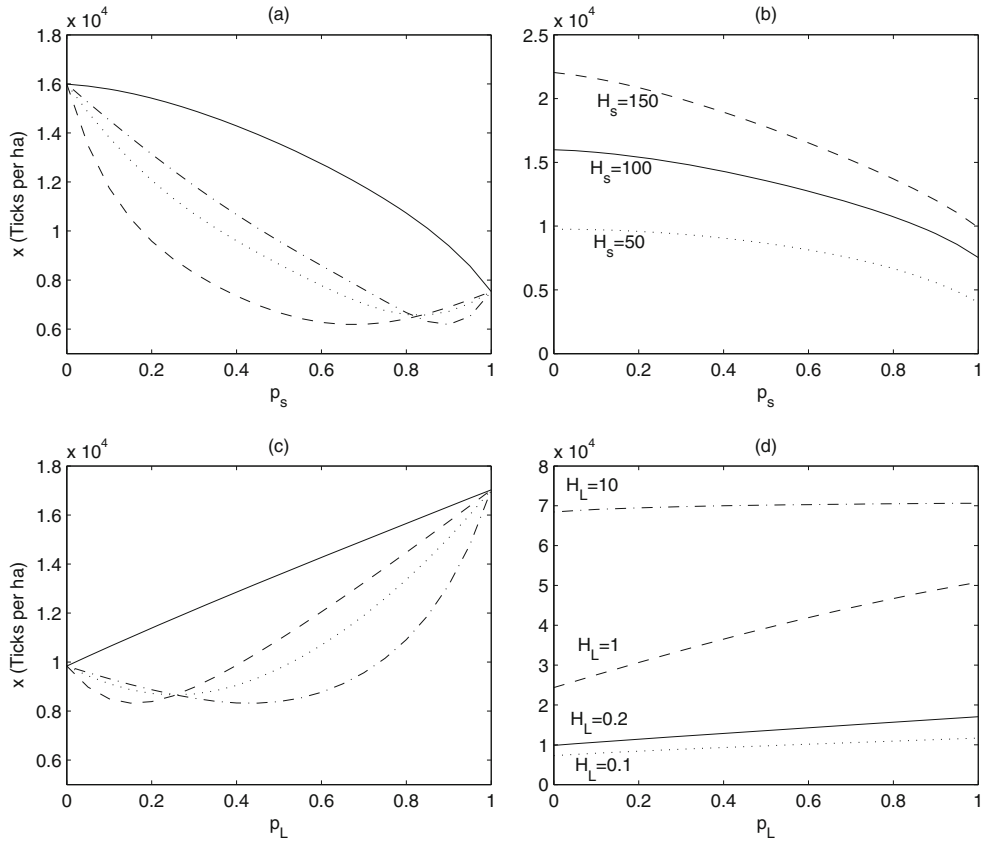

Fig. 1 The effect of direct and indirect host competition. In **a** and **b**, total tick density is plotted as a function of $p_s = H_1/H_s$, the proportion of small hosts that are of type H_1. In **a**, we fix $\sigma_3(y) = \sigma_4(y) = 1$ and vary the functional form of $\sigma_n(y)$, $n = 1, 2$. The *solid line* is indirect competition $\sigma_1(y) = \sigma_2(y) = 1$; the remaining *lines* represent direct competition, where the *dotted line* represents no preference ($\phi_1 = 0.5$), the *dashed line* represents preference for H_1 ($\phi_1 = 0.8$), and the *dash-dot line* represents preference for H_2 ($\phi_1 = 0.2$). In **b**, we fix $\sigma_n(y) = 1$, for each n and vary H_s. In **c** and **d**, total tick density is plotted as a function of $p_L = H_3/H_L$, the proportion of large hosts that are of type H_3. In **c**, we fix $\sigma_1(y) = \sigma_2(y) = 1$ and vary the functional form of $\sigma_n(y)$, $n = 3, 4$, as with **a** the *solid line* is indirect competition; the *dotted line* is $\phi_3 = 0.5$, the *dashed line* is $\phi_3 = 0.8$ and the *dash-dot line* is $\phi_3 = 0.2$. **d** We fix $\sigma_n(y) = 1$ for each n and vary the total large host density, H_L. Unless otherwise stated parameters are a given in Table 2 and $H_s = H_1 + H_2 = 100$, $H_L = H_3 + H_4 = 0.2$, $\sigma_n(y) = 1$ and $p_s = p_L = 0.5$

Thus, in the next section, we numerically examine the coexistence equilibrium and how it is impacted by changes in host competition and predation.

Numerical results

The influence of host competition

Throughout this section, we assume the host population is at equilibrium, such that H_n is a constant. We focus on changes in biodiversity that modify host competition and hence tick-host contact rates, via the function $\sigma_n(y)$, and examine the effects on equilibrium tick densities.

Indirect competition between hosts In Fig. 1a, we explore how p_s affects total tick density. The total density of small hosts (H_S) is fixed, thus $H_1 = p_s H_s$ and $H_2 = (1 - p_s)H_s$. Under indirect competition, $\sigma_n(y) = 1$ for each n, and the equilibrium total tick density is found to depend on p_s in a monotonic fashion, decreasing with higher densities of H_1, ($p_s \to 1$). H_1 is a less suitable host for the nymphal class, but more suitable for the larval class. However, since the parameters in the production terms in the nymph equation are smaller than those in the larval equations, the nymph production determines the rate of total tick production. Consequently, as host H_2 can support the highest number of nymphs (compare λ_{21} and λ_{22} in Table 2), maximising the density of H_2 hosts maximises tick densities and hence we see tick densities in Fig. 1a are maximised when $p_s = 0$ and

Table 2 Description and typical parameter values used in numerical simulations

Parameter	Description (units)	Value
μ_1^*	Natural mortality of larvae (yr^{-1})	1.1
μ_2^*	Natural mortality of nymph (yr^{-1})	0.73
μ_3^*	Natural mortality of adult (yr^{-1})	0.037
β_1^{**}	Number of female eggs laid by an adult which survive to be larvae (larval ticks/ adult tick)	350
β_2^{**}	Number of nymphs produced by a larvae (nymph ticks/ larvae tick)	1
β_3^{**}	Number of adults produced by a nymph (adult ticks/nymph tick)	1
$\lambda_{1,1}^{***}$	Average tick load of larvae on H_1 (ticks/deer mice/yr)	28.95
$\lambda_{1,2}^{***}$	Average tick load of larvae on H_2 (ticks/chipmunk (birds)/yr)	12.57 (3)
$\lambda_{2,1}^{***}$	Average tick load of nymphs on H_1 (ticks/deer mice/yr)	0.68
$\lambda_{2,2}^{***}$	Average tick load of nymphs on H_2 (ticks/chipmunk (bird)/yr)	4.90 (9.67)
$\lambda_{3,3}^{****}$	Average tick load of adults on H_3 (ticks/deer/yr)	201.84
$\lambda_{3,4}^{****}$	Average tick load of adults on H_4 (ticks/racoon/yr)	69.54
a_i^{*****}	Half the maximum number of stage i ticks per hectare	650
H_1^{****}	Number of mice per hectare	0-100
H_2^{****}	Number of chipmunks (birds) per hectare	0-50 (31)
H_3^{****}	Number of deer per hectare	0.075-0.4
H_4^{****}	Number of raccoon per hectare	0.2
p_s	Proportion of small hosts of type H_1	$\frac{H_1}{H_1+H_2} = \frac{H_1}{H_s}$
p_L	Proportion of large hosts of type H_3	$\frac{H_3}{H_3+H_4} = \frac{H_3}{H_L}$

The data refers to *Ixodes scapularis* ticks. Average tick loads $\lambda_{i,n}$ account for successful molting of the tick which is typically around 50 % (LoGiudice et al. 2003). β_1 assumes an average of 1000 eggs produced per engorged adult, of which 70 % survive (Gaff and Gross 2007) of which half are female, giving the 350 adopted in the paper. In the absence of data on alternative large hosts (H_4), we used raccoon data, a common tick host with large tick burdens. We convert ticks per host into ticks produced per host per year by multiplying by the reciprocal of the average duration of the tick stage. We estimated tick mortality assuming that larvae are the most sensitive stage (Ogden et al. 2005; Caraco et al. 2002; Gaff and Gross 2007)

*(Ogden et al. 2005), **(Gaff and Gross 2007), ***(Giardina et al. 2000), ****(LoGiudice et al. 2003), *****(Perkins et al. 2006)

minimised when $p_s = 1$. These results hold for a range of values of H_s (see Fig. 1b). So, we conclude that under indirect host competition maximising biodiversity in small hosts ($p_s = 0.5$) does not reduce tick densities; instead, reducing or completely eliminating the abundance of the host which is more suitable for nymphs (H_2) would most effectively reduce tick densities.

To study indirect competition between the large hosts, we vary p_L, while fixing the total density of large hosts, H_L, thus $H_3 = p_L H_L$ and $H_4 = (1 - p_L)H_L$. The results are similar to those found under indirect competition between small hosts. In Fig. 1c, we see that increasing biodiversity in large hosts ($p_L \to 0.5$) does not decrease tick densities. Tick densities are maximised when there are only large hosts of type H_3 (p_L=1); this is because H_3 hosts can carry the largest burden of adult ticks, and only the adult tick feeds on the large hosts.

Direct competition between small hosts. Under direct competition host finding probability depends on relative host abundance. Here we assume $\sigma_3(y) = \sigma_4(y) = 1$ while $\sigma_1(y)$ and $\sigma_2(y)$ are given in Eq. 4. We varied the probability of ticks encountering H_1 from ϕ_1 =0.5, 0.8 to 0.2. Under direct competition, we find that increasing biodiversity in small hosts, away from the extremes of $p_s = 0$ or 1, can lower tick densities, such that there is a minimum in tick densities at an intermediate value of p_s (Fig. 1a). In accordance with the previously described role of nymphs as a rate limiting life-stage, when $\phi_1 = 0.5$, the minimum tick density lies to the right of $p_s = 0.5$ and so higher proportions of H_1 hosts lead to the lowest tick density, because the H_1 host is the less suitable hosts for the nymphs. However, the minima can shift closer towards $p_s = 0$ or $p_s = 1$, when ticks have a respectively higher probability of encountering host H_1 or H_2 (ϕ_1 equal to 0.8 or 0.2). We can estimate the location of the minima be considering the rate limiting step in tick production. In Eq 5, γ_2 determines the rate nymphs become adults. Taking $\phi_1 = 0.5$, thus $\sigma_1(p_s) = p_s$ and $\sigma_2(1 - p_s) = 1 - p_s$, we have $\gamma_2 = p_s H_1 \lambda_{2,1} + (1 - p_s)H_2\lambda_{2,2}$. Applying $H_1 = H_s p_s$ and $H_2 = H_s(1 - p_s)$, then γ_2 is minimised, with respect to p_s, when $2p_s H_s \lambda_{2,1} - 2(1 - p_s)H_s\lambda_{2,2} = 0$, that is,

when $p_s = \lambda_{2,2}/(\lambda_{2,1} + \lambda_{2,2}) = 0.88$. The minimum of the dotted curve for $\phi_1 = 0.5$ in Fig. 1a is located at $p_s \approx 0.85$, in good agreement with our estimate. Notice that this estimate of the location of the minimum is independent of total small host density and is determined only by average nymph loads on the hosts. So, the identity of the host species and the tick loads they carry is important to determining whether high biodiversity will lower tick densities.

Direct competition between large hosts Here, we assume $\sigma_1(y) = \sigma_2(y) = 1$ while $\sigma_3(y)$ and $\sigma_4(y)$ are given by Eq. 4. Under direct competition between large hosts tick densities are minimised when there is high biodiversity among the large hosts, that is, at intermediate values of p_L (Fig. 1c). When the ticks have no host preference ($\phi_3 = 0.5$), the minimum is located to the left of $p_L = 0.5$, that is majority of large hosts are of type H_4, which is a less suitable host for the adult ticks. When there is a preference for H_3 (e.g. $\phi_3 = 0.8$) or H_4 (e.g. $\phi_3 = 0.2$), the minima shifts to the right or left, respectively. Tick densities are minimised when γ_3, the rate adults produce larvae, is minimised. In an analogous calculation to the small host case, we ask what value of p_L minimises γ_3 in the simple case that $\phi_3 = 0.5$. We find $p_L = \lambda_{3,4}/(\lambda_{3,3} + \lambda_{3,4}) = 0.26$, which is a good approximation to the minimum in Fig. 1c and is determined only by adult tick densities per host. The identity of the large hosts in the ecosystem will be important in determining if high biodiversity will reduce tick densities.

Both the indirect competition results and the direct competition results hold for a range of values for H_L (see Fig. 1d). However, we find that, when large-host densities are very high (H_L=10), changing p_L has little effect on the equilibrium tick density and there is no longer a minimum. In other words, here biodiversity has an effect only when large hosts are at low enough densities, that is, at ecologically relevant levels for deer and other wildlife; while, in the presence of high densities of large hosts (e.g. cattle farms), changing biodiversity (p_L) has a minimal effect on tick numbers. At high values of H_L, the availability of large hosts no longer limits larval production. So, although increasing biodiversity of large hosts lowers larval numbers slightly, larval densities are so high that $\gamma_1 x_1/(a_1 + x_1) \approx \gamma_1$, and we thus see virtually no effect from a change in the biodiversity of large hosts when they are abundant.

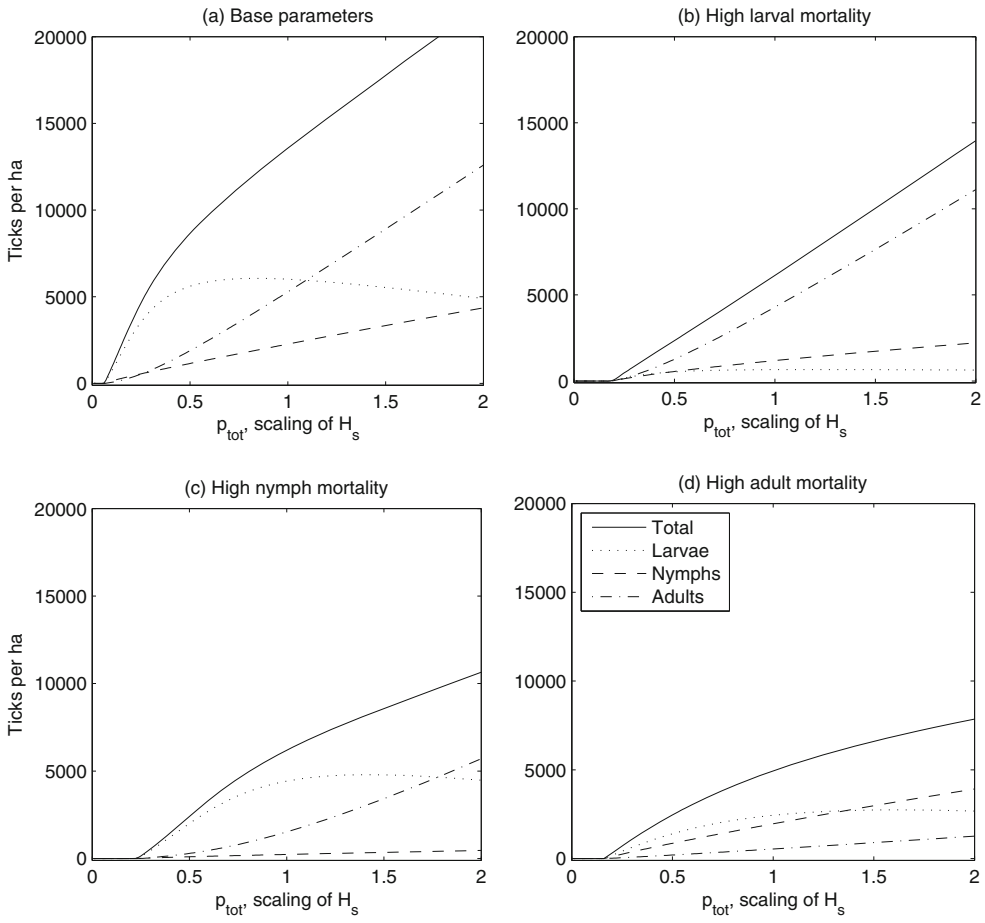

Fig. 2 The effect of varying the total number of small hosts and tick mortality. Total tick density, and its break down into larval, nymph and adult tick densities, is plotted as a function of p_{tot}, the scaling factor multiplying the baseline number of small hosts H_s, e.g. a scaling of $p_{\text{tot}} = 1.5$ implies $H_s = 1.5 \times 100 = 150$. In **a**, the parameters are as given in Table 2. In **b**, larval tick mortality is high, $\mu_1 = 11$, **c** nymph tick morality is high, $\mu_2 = 7.3$ and in **d** adult tick mortality is high, $\mu_3 = 0.37$. In all cases $\sigma_n(y) = 1$ for each n, $p_s = p_L = 0.5$ and $H_3 = 0.1$, $H_4 = 0.1$

Comparing the effects of large and small hosts Next, we aim to compare the differing effects of small and large host density on tick densities. To focus the study, we only consider indirect competition and fix $\sigma_n(y) = 1$ for each n and $p_s = p_L = 0.5$. We vary H_s/H_L, by introducing p_{tot} as a factor to scale this ratio up or down. When $p_{\text{tot}} = 1$ then $H_s/H_L = 500$ and host densities are at their base line values. Initially, we vary H_s/H_L by fixing H_L and varying only H_s. In this case, a scaling factor $p_{\text{tot}} < 1$ reflects a decrease in small hosts, and a scaling factor $p_{\text{tot}} \gg 1$ reflects an increase in the number of small hosts.

Figure 2a illustrates a biphasic pattern in tick densities as a function of the scaling factor p_{tot}. When $p_{\text{tot}} < 1$ tick densities are low and increase rapidly with small additions to the densities of small hosts; while, when the populations of small hosts are higher and $p_{\text{tot}} \gg 1$ a small increase in the density of the small hosts has a smaller effect on tick densities. At low densities of small hosts ($p_{\text{tot}} \ll 1$), the availability of small hosts acts as a rate limiting step in the tick life-cycle. In contrast, when densities of small hosts become large ($p_{\text{tot}} \gg 1$), they are no longer rate limiting, and further increases in small hosts have significantly less effect on the total density of ticks. The breakdown of total tick density into the different life stages reveals that changes in larval densities are the source of the biphasic pattern in total tick density. Larval densities follow a hump shape graph and eventually decrease as p_{tot} increases. The decrease is due to the fact that larval production from adults is a saturating function of adult tick densities. Even though densities of adult ticks are increasing as H_s is increased, there is little change in the rate larval ticks are produced once the production term saturates. The rate at which larvae become nymphs (γ_1) is a linear function of H_s and does not saturate leading to the observed net decrease in larval densities.

The biphasic pattern, we observe when we change small host densities is also present when we change large host densities (results not shown). Much like with small hosts, large hosts can cause a rate limiting step in the tick life-cycle. Larval densities increase with increasing the density of large hosts; however, in this case, because the population of small hosts are fixed, the rate of nymph production saturates and becomes the rate limiting step.

We tested the robustness of our findings to changes in tick mortality. The biphasic pattern in total tick density persisted (Fig. 2b–d). Increasing adult tick mortality (d) led to the largest reduction in total tick densities, followed by nymph mortality (c) leading to the next largest reduction. This is an unsurprising result, as adult ticks are responsible for a very large production of larvae (i.e. 350), while larvae and nymph individuals produce at most one other individual. Increasing adult mortality shortens the duration of the adult life stage available to reproduction.

We also explored the effects of changes to average tick loads per host, $\lambda_{i,n}$. Reducing nymph loads on H_2 led to the biggest reduction in tick densities, while changing larval loads had little effect (Fig. 3a). Generally, changes to nymph

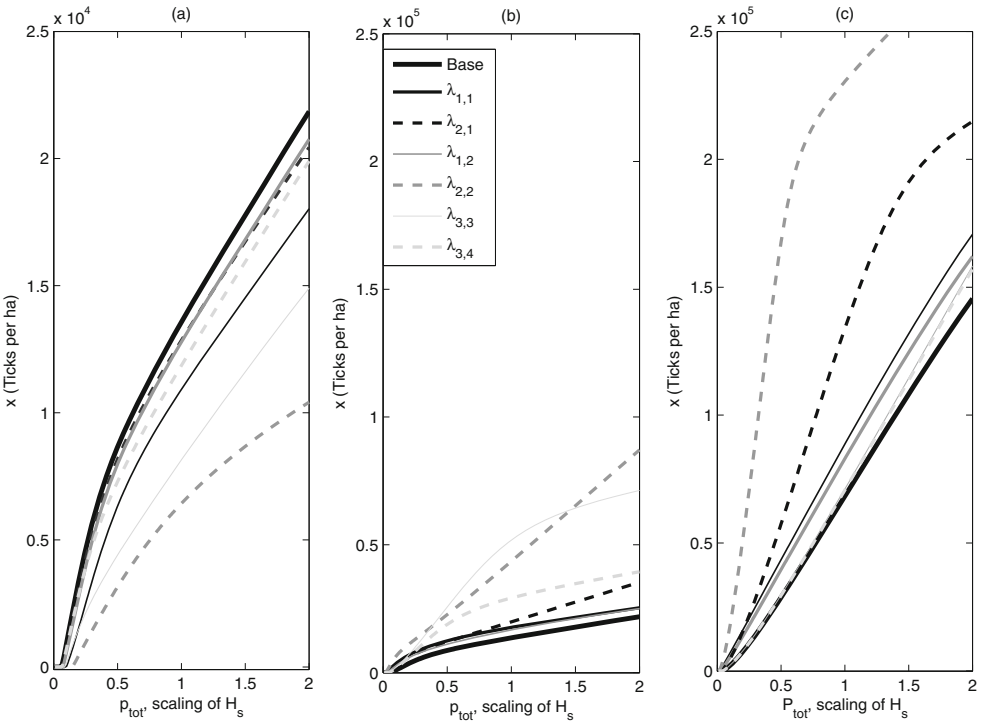

Fig. 3 The effect of varying the total number of small hosts and host tick loads. Total tick density is plotted as a function of p_{tot}, the scaling factor multiplying the baseline number of small hosts $H_s = 100$. In **a** and **b**, large host density is fixed at $H_L = 0.2$ and in **a** $\lambda_{i,n}$ is reduced 90 %, while in **b** $\lambda_{i,n}$ is increased by a factor of 10. Finally, in **c**, we consider large host densities with $H_L = 5$ and $\lambda_{i,n}$ is increased by a factor of 10. Unless otherwise stated the parameters are as given in Table 2 and $\sigma_n(y) = 1$ for each n and $p_s = p_L = 0.5$

tick loads caused the most significant change in tick densities and was most noticeable at high small-host densities (Fig. 3c).

The influence of host oscillations

Changes in biodiversity can also influence predation, we consider this by assuming that predation is at a sufficiently high level that it leads to Lotka-Volterra oscillations in the host populations. We allow periodic temporal cycling in the small or large host densities. Cycling in the small hosts, $H_1(t)$, is given by $p_s H_s(1 + A\cos(2\pi t/10))$, where the average density is $p_s H_s$, and where A is the amplitude, with increasing A analogous to increased predation pressure on $H_1(t)$, and similarly for $H_2(t)$. Cycling in large hosts, $H_3(t)$, is given by $p_L H_L(1 + A\cos(2\pi t/10))$ where the average density is $p_L H_L$, and where A is the amplitude, or predation pressure on $H_3(t)$, likewise for $H_4(t)$. For simplicity, we assume indirect host competition, $\sigma_n(y) = 1$ for each n.

Oscillations in either H_1 or H_3 have little effect on average tick densities (Fig. 4). Predation induced oscillations in small hosts are damped out with essentially no corresponding oscillations in tick densities (Fig. 4a, c). For large hosts, predation induced oscillations lead to corresponding large oscillations in the tick population (Fig. 4b a, d). Upon examining the tick time series, we found that the oscillations in $H_1(t)$ are damped by the differing responses of nymph and larvae to $H_1(t)$ densities. While larvae and nymph oscillations have the same period as the host, they are out of phase with one another (Fig. 5a), their effects essentially cancel one another out resulting in no oscillations in total tick density. Oscillations in adults have extremely small amplitude so do not result in oscillations in total tick density either.

We can understand the relative phases of the larval and nymph oscillations by considering how $H_1(t)$ effects the flow into and out of the larval and nymph classes. When $H_1(t)$ is high, there is a large flow out of the larval class, and the flow out of the larval class oscillates in line with $H_1(t)$. We, thus, see a minimum in larval density when $H_1(t)$ is at its peak and a maximum in larval density when $H_1(t)$ is at a minimum. Nymphs, on the other hand, have a flow in and out of the nymph class that oscillates in line with $H_1(t)$, but the flow in is greater because $H_1(t)$ hosts support more larvae ($\lambda_{1,1} \gg \lambda_{2,1}$). The net effect is that nymph cycles are in phase with $H_1(t)$, while larvae cycles are out of phase. Furthermore, because $\lambda_{1,1} \gg \lambda_{2,1}$, the oscillations in nymph

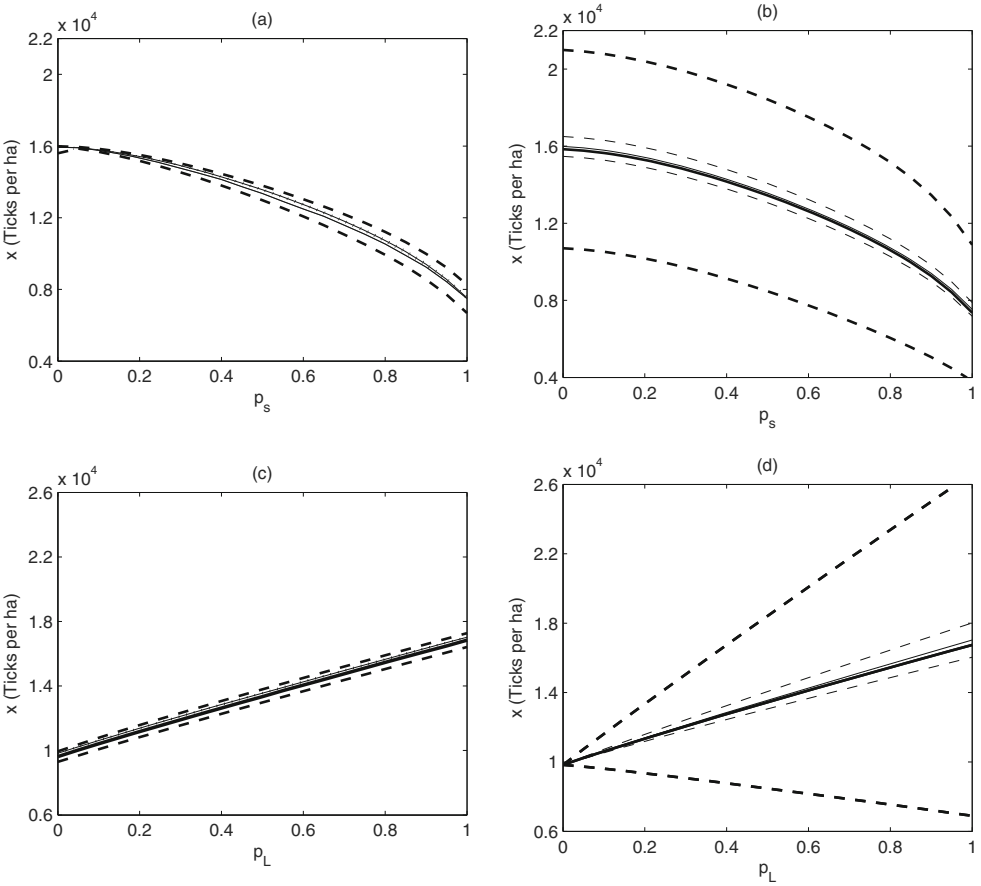

Fig. 4 The effect of host oscillations. The *solid lines* in all the plots correspond to the average tick density over the period of the attractor, and the *dashed lines* correspond to the maximum and minimum tick densities over the period of the attractor. **a** and **b** Illustrate total tick density plotted as a function of p_s. **a** Illustrates the effect of oscillations in $H_1(t)$, while **b** illustrates the effect of oscillations in $H_3(t)$. **c** and **d** Illustrate total tick density plotted as a function of p_L. **c** Illustrates the effect oscillations in $H_1(t)$, while **d** illustrates the effect of oscillations in $H_3(t)$. In all plots, the bold lines corresponds to $A = 1$, a 100 % fluctuation in $H_1(t)$ or $H_3(t)$ around the average, while the lighter *lines* correspond to $A = 0.1$, a 10 % fluctuation about the average. Unless otherwise stated H_n is a constant, $H_s = 100$, $H_L = 0.2$, $p_s = p_L = 0.5$ and $\sigma_n(y) = 1$ for each n

density transitioning into the adult class are small in amplitude and the type II functional response to nymph density further dampens any oscillations in the production of adult ticks resulting in the very low amplitude adult oscillations which are in phase with the nymphs.

The response of tick density to oscillations in $H_3(t)$ gives a quite different picture to that of the small-host oscillation case. While adults ticks continue to exhibit very low amplitude oscillations, the larvae and nymph populations now oscillate in phase (Fig. 5b) resulting in large amplitude oscillations in total tick density. Larvae oscillate in phase with $H_3(t)$ because of the large numbers of larvae produced per adult. Because H_1 and H_2 are held constant in this scenario, the larvae oscillations get transmitted directly into nymph oscillations with only a slight modification in the phase caused by the time spent in the larval life-stage. The amplitude of oscillations in nymph density is damped by the type II functional response, which is damped further by the time the ticks reach the adult stage.

Lotka-Volterra oscillations may occur at different frequencies depending on the predator type and on seasonal fluctuations in resource availability, so we also examine changes in the period of host oscillations (Fig. 6). Increasing the period of host oscillations increased the magnitude of oscillations in total tick density. Results were most sensitive to oscillations in large hosts (Fig. 6c). When the larger hosts undergoes oscillations, the average tick density is lower than is predicted from the scenario on non-oscillating hosts; however, the fluctuations in tick density are large. The two types of small host did not have the same effect on tick density. When $H_1(t)$ oscillates, the average tick density is lower than in the non-oscillating case. When $H_2(t)$ oscillates, the average tick density is the same as in the non-oscillating case. However, tick oscillations were more sensitive to the period of oscillations in H_2 hosts rather than H_1. The sensitivity to H_2 oscillations is due to the sensitivity of total tick densities to the rate that nymphs become adults which is enhanced by increases in H_2 hosts.

All of the results of Section 3 continued to hold when we examined predation on H_2 and H_4 individually, or with simultaneous (synchronous) predation on H_1 and H_2 or on H_3 and H_4. Since synchronous oscillations in small or large host populations occurs only very rarely in natural populations, we also examined the cases when the small or large hosts are cycling asynchronously. In accordance to some works (Holt and Roy 2007), we find that asynchronous predation on small hosts can increase average tick densities, but the effect is very limited (data not shown). Similarly, asynchronous predation on both large hosts may decrease tick densities, but again, the magnitude of the effect is negligible. We have also examined situations where one of the small or large hosts have higher predation levels than the other, as well as cases where both the small and large hosts are being predated on, but the results do not differ from the previous cases (data not shown).

Discussion

Ticks can transmit numerous pathogens and so it is important to understand the factors that regulate and limit tick abundance. Ticks are opportunistic feeders and have been shown to feed on a wide range of hosts, but whether the abundance of ticks is a function of host abundance remains controversial (Keesing et al. 2006). Evidence of altered host behaviour or space use in the presence of heterospecifics is likely to affect tick-host encounter rates, which Keesing et al. (2006) termed *encounter augmentation* and this is one example of a mechanism that could generate complex relationships between tick and host abundance. More generally, the hosts are part of a complex ecosystem and studies have suggested that community composition of hosts are an important factor in determining tick abundance (Giardina et al. 2000). By constructing a tick stage-structured model, we were able to determine how changes in biodiversity that affect the ecological processes of host competition, behaviour and predation influence tick populations. In so doing, we determined the conditions where biodiversity may potentially regulate tick populations. Our results show that, though increasing biodiversity can in many cases have a regulating influence on tick densities, it is in itself too coarse of a measure to predict the magnitude and direction of the regulating effect. Instead, the effect can be better understood and predicted by examining the underlying ecological processes affecting ticks, which, in turn, can be translated into meaningful tick management approaches. Below, we discuss the results and their management implications.

Competition type and encounter augmentation

When we considered aspects of biodiversity that affect host competition, then increasing host biodiversity did not necessarily have a regulating effect on tick densities. In order for high host biodiversity to lower tick densities, the competition among the hosts had to be direct, rather than indirect (Fig. 1). With indirect competition, the presence of another competitor will not affect the hosts' ability to forage and so tick-host encounter rate is independent of the presence of host heterospecifics; while, with direct competition, the presence of a competitor affects the host ability to forage and encounter ticks (e.g. through changes in behaviour). This difference affects tick densities because, with indirect competition, the hosts do not modify their encounter rate with ticks and tick densities are not lowered by increased

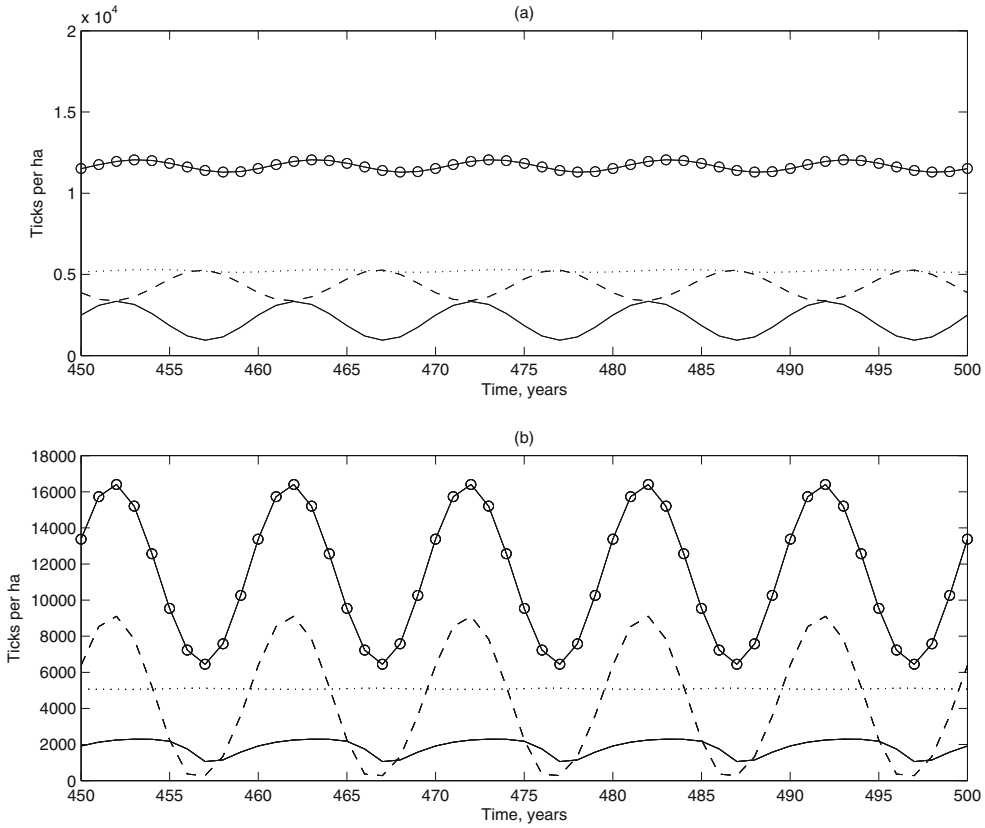

Fig. 5 The effect of host oscillations on larval, nymph and adult tick densities as a function of time. In **a**, the preferred small host density oscillates according to $H_1(t) = 50(1 + \cos(2\pi t/10))$, all other host densities are held constant. In **b**, the preferred large host density oscillates according to $H_3(t) = 0.1(1 + \cos(2\pi t/10))$, all other host densities are held constant. In each plot, the *solid lines* with the *circles* indicates total tick density ($x(t)$) while the *dashed line* indicates larval density($x_1(t)$), the *solid line* is nymph density ($x_2(t)$) and the *dotted line* is the adult density ($x_3(t)$). Unless otherwise stated $H_s = 100$, $H_L = 0.2$, $p_s = p_L = 0.5$ and $\sigma_n(y) = 1$

biodiversity. In fact, an ecosystem, with a single host species supporting low tick loads leads to the lowest tick densities. In contrast, with direct competition, the hosts' encounter rate with ticks is modified in a non-linear manner, and in a diverse host environment, results in decreased total tick densities. These results suggest that the changes in tick-host encounter rates, which can often be associated to direct competition, are one of the fundamental ecological processes that can determine whether increasing biodiversity will regulate tick populations. Indeed, there is ample evidence that the presence of other small hosts greatly affect the behaviour of deer mice and other rodents, so that their encounter rate with ticks would necessarily be also modified (Mitchell et al. 1990; Yunger et al. 2002); similarly, the behaviour of deer also change when other large hosts are present (Hobbs et al. 1996; Latham 1999). As well, recent work has even shown that rodents may also change their behaviour in the presence of large hosts (Munoz and Bonal 2007). The situation of indirect competition would most likely occur when the resources available are plentiful, and there is no need to compete directly (Wooton 1994). High resource availability may occur around spatially heterogeneous agricultural areas where there is often the presence of food subsidies, and also habitat regions that support small hosts (e.g. orchards or corn fields).

Host biodiversity may also play a role in answering the question posed by Ostfeld (Ostfeld RS 2011): "why is the relationship between deer and tick abundance so variable?" Luo and Wu (2014) proposed one answer based on tick seeking assumptions. They found frequency-dependent contact between ticks and hosts, which is expected at high host densities, resulted in no relationship between tick and deer densities, while density-dependent contact, which is expected to occur when host densities are lower, led to a positive relationship between tick and deer densities. We offer a different answer based on the biodiversity of the ecosystem in which the deer reside. If deer modify their behaviour in response to other animals present in the ecosystem, then the composition of the ecosystem can alter the tick-host encounter rate. Looking at Fig. 1c, with direct competition between large hosts, we see that increasing the proportion of deer, p_L could lead to an increase or decrease in tick numbers depending on the relative abundance of the other large hosts in the system and on how those other large hosts affect deer behaviour.

Our analytical results suggest that the importance of any particular host or tick life-stage in regulating tick densities depends on whether they cause a rate limiting step in the tick population cycle. If the host densities are low, or if the

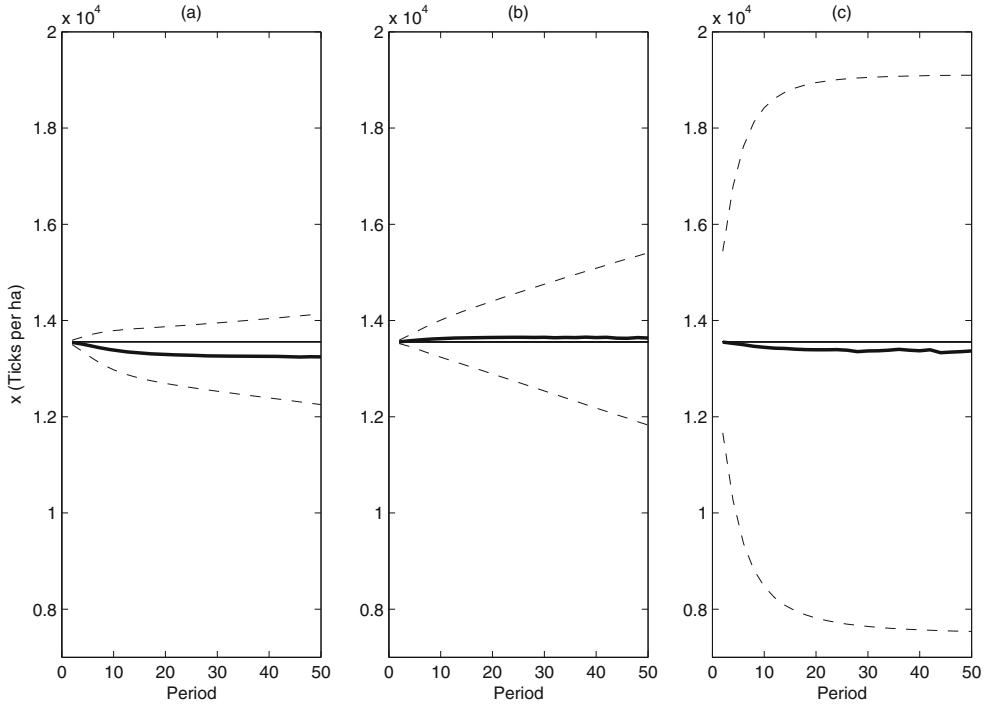

Fig. 6 The effect of host oscillation period. **a** Varying the period of oscillations in H_1, where $H_1(t) = 50(1+\cos(2\pi t/\text{period}))$. The *solid line* indicates the case where is constant $H_1 = 50$. When $H_1(t)$ oscillates, so does the equilibrium total tick density, and the *bold solid line* is the average tick density (averaged over the period of the attractor), the *dashed lines* indicate the maximum and minimum tick densities over the period of the attractor. **b** Varying the period of oscillations in H_2, where $H_2(t) = 50(1+\cos(2\pi t/\text{period}))$, the *lines* are as described in (**a**). **c** Varying the period of oscillations in H_3, where $H_3(t) = 0.1(1+\cos(2\pi t/\text{period}))$, the *lines* are as described in **a**. Unless otherwise stated H_n is a constant, $H_s = 100$, $H_L = 0.2$, $p_s = p_L = 0.5$ and $\sigma_n(y) = 1$

transition rate to one life stage is slower than the transition rates for other stages, then that host or tick life-stage will cause a rate limiting step that effectively regulates the rate of total tick production. Notably, the particular rate limiting influence of nymphs that we found here is based on the parameterisation derived from the North-Eastern *Ixodes scapularis* tick; in other tick species, the rate limiting step may be in another life-stage—highlighting the need to consider the ecological properties of the tick-host system. Our results are consistent with the finding that mused-based interventions had only weak effects on tick abundance (Brisson et al. 2008). Reducing mouse densities is equivalent to reducing p_s, whereby the competitors of the mice replace those mice that are removed (Keesing et al. 2009). Reducing p_s increased tick densities because, in our model, the competitor of the mice (chipmunks or birds) supported higher loads of nymph ticks and thus allowed higher numbers of ticks to transition to adults (see Fig. 1a).

A common tick-management practice targets the adult ticks feeding on large hosts; the practice essentially attracts deer to a device that applies acaricides (i.e. tick-specific pesticides) on the deer as it feeds (Schulze et al. 2009). Applying acaricides is equivalent to reducing $\lambda_{3,3}$ in the model. Figure 3a shows that reducing tick loads on deer had one of the largest impacts on total tick densities. Lowering nymph loads on alternative small hosts such as chipmunks, was the only strategy that would lower tick densities more. Targeting large hosts have been shown to be effective, coupling the practice to a similar approach that targets nymphs and small hosts could increase the reduction of tick densities. One approach may be to cull small hosts, but this is impractical given their ubiquity and high density (Myers et al. 1998). Another promising method would be similar to that employed with deer, where small hosts could be given access to nest bedding that is imbibed with acaricides (Jaenson et al. 1991); in so doing, they would kill the ticks that have attached to the rodents when they return to their nest. Other innovative approaches may involve orally vaccinating small hosts against the tick bites, which has been shown to have some preliminary success (Gomes-Solecki et al. 2006). A side from management practices that modify $\lambda_{i,n}$, climate can also modify $\lambda_{i,n}$. High humidity can cause ticks to quest higher on vegetation which increases their encounter rate with larger hosts and

reduces their encounter rate with smaller hosts such as mice (LoGiudice et al. 2008).

Predation and host population cycles

Changes in biodiversity can also lead of host oscillations, as associated with increasing predation pressure or fluctuations in host resources (Ostfeld and Keesing 2000). Under these scenarios, the average tick densities did not deviate significantly from the cases with no oscillations in host density. Moreover, oscillations in small hosts were damped out in the tick population. However, oscillations in large hosts were transmitted to the ticks to give rise to oscillations in tick densities (see Fig. 5). Therefore, one may expect that sudden changes in large host densities to have a more dramatic impact on tick densities than corresponding changes to the density of small hosts. Lengthening the period of the host oscillations (see Fig. 6) further increased the amplitude of oscillations in tick numbers. These changes in the period of the host oscillations may occur as a result of changes in the type or behaviour of the predators; but, the change in period may also result from changes in the host dynamics themselves, which may be sensitive to resource availabilities (e.g. production of acorns during masting events) or climate variations (Ostfeld et al. 2006). While these fluctuations in resource availability will certainly occur in wildlife situations, they are likely not observed in areas close to human land use, as there may be sufficient food subsidies to support stable populations of hosts. Our results remained insensitive to various combinations of asynchronous predation and predation on small and large hosts.

Implications of Lyme disease

In Appendix B, we provide a simple extension of our model following (Lou and Wu 2014) which allows us to calculate the basic reproduction number $\mathcal{R}_0$ for lyme disease and hence allows us to relate out findings to disease transmission, the focus of many tick studies. We assume that the H_2 hosts are not competent reservoirs for the disease, but the H_1 hosts are. The basic reproduction number is given by

$$\mathcal{R}_0 = \sqrt{\frac{\beta_H \gamma_2'}{\mu_2(a_2 + x_2^*) + \gamma_2'} \frac{\beta_L \alpha_2'}{H_1 \alpha_2 \mu_{H_1}} \left(\mu_2 + \frac{\gamma_2}{a_2 + x_2^*}\right) x_2^*}, \tag{12}$$

(β_H and β_L are the transmission coefficients of the infection to H_1 hosts and larval ticks, respectively. γ_i' (α_i') is the contribution to γ_i (α_i) that comes from feeding on H_1 hosts only. Lastly, x_1^* and x_2^* are the equilibrium tick densities).

Our formulae in Appendix B are very similar to those of Lou and Wu (2014), only our stage-structured model of tick dynamics, and consequently the epidemiological model, differs in two key ways:

(a) Since each host can carry a maximum number of ticks the production terms in Eqs. 1–3 saturate with a type II functional response in tick density compared to a type I response in Lou and Wu (2014).
(b) The inclusion of additional host types and the modification of encounter rate in response to host biodiversity is omitted in Lou and Wu (2014)

These two differences both modify the expression for $\mathcal{R}_0$ calculated in Lou and Wu (2014). In particular, (a) results in $\mathcal{R}_0$ no longer being a simple increasing function of nymph density, instead (12) can increase and then decrease as we increase nymph density. The decrease only happens if γ_2' is small meaning that most of the hosts that nymphs feed on are type H_2 rather than H_1. The decrease in $\mathcal{R}_0$ at high densities of H_2 is a 'dilution effect', whereby ticks feed on hosts that are not a disease reservoir (H_2 hosts), so do not transmit the disease, resulting in the pathogen being diluted and maintained in the environment at a much lower level (e.g. Schmidt and Ostfeld 2001). However, (b) allows γ_2' to also be low when H_2 hosts are less abundant. If the presence of other hosts modify the tick-host encounter rate in such a way that very few ticks feed on H_1 hosts, in other words $\phi_1 \ll 0.5$ then we still have a dilution effect, except the pathogen is diluted because of the effects of direct competition between the hosts, importantly this result means that the existence and strength of a dilution effect is likely to depend on the biodiversity of the ecosystem in a complex way.

Limitations of the results

For our analysis, we made a number of simplifying assumptions. First, in terms of the ecology of ticks, tick-host dynamics are more complex than modelled. While research has suggested that tick-host dynamics can be at equilibrium in wild populations (Wikel 1996; Bull and Burzacott 1993; Lack 1954; Irvine 2006), other studies have found that ticks can affect the behaviour as well as the fitness of domestic hosts (e.g. White et al. 2003; Bock et al. 2004) and wild hosts (McKilligan 1996). Similarly, while the tick stages do usually feed on small or large hosts as we described, it is possible for any stage of a tick to feed on any host, since they are opportunist feeders. The inclusion of these considerations would modify the dynamics of the tick-host system,

Table 3 Analytical criteria for tick eradication (A, B) and the absence of tick cycles (C, D) (see Appendix A for details of the derivations). Note that (A) and (B) are alternative criteria, only one of these needs to be satisfied and similarly for (C) and (D)

	Result	Criteria
(A)	(0,0,0) is globally asymptotically stable.	$\mu_3 > \frac{\alpha_1-\gamma_3}{a_3}$.
(B)	(0,0,0) is globally asymptotically stable.	$\mu_1 > \frac{\alpha_1}{a_3}$, $\mu_2 > \frac{\alpha_2}{a_1}$ and $\mu_3 > \frac{\alpha_3}{a_2}$.
(C)	There exist no invariant closed curves and the omega limit set of any orbit is a single equilibrium.	$\mu_2 + \mu_3 > \frac{\alpha_1-\gamma_3}{a_3}$.
(D)	There exist no invariant closed curves and the omega limit set of any orbit is a single equilibrium.	$\mu_1 + \mu_2 > \frac{\alpha_1}{a_3}$, $\mu_2 + \mu_3 > \frac{\alpha_2}{a_1}$ and $\mu_3 + \mu_1 > \frac{\alpha_3}{a_2}$.

as the ticks would then be causing population fluctuations in the hosts, since they can influence host fitness, and likely also the potential for changes in tick-stage transition rates, since they may feed on small and large hosts.

However, changes in tick loads or transition rates would only affect the location of the rate limiting step and which tick life stage is acting as a bottleneck. The trends of our results are thus robust to changes in parameters and the dynamics we identified should be robust for a wide range of parameter values.

Conclusion

Our paper examined how two ecological processes, competition and predation, that underlie biodiversity can potentially regulate tick populations. While biodiversity can in many cases regulate tick populations, this will depend more on the ecological processes determining the relationship between the ticks and their hosts. Significantly, the ecological processes may often trump the predictions of biodiversity, such that increased biodiversity may have no or the opposite effect than intended. Hence, though biodiversity may be a good initial measure of whether disease emergence may occur, the ecological processes that govern the vector-host dynamics must be examined more closely. The added benefit of examining the ecological processes is that it would lead to specific management implications that can be implemented.

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Appendix A: Global stability of the extinction equilibrium and nonexistence of periodic orbits

Consider a system of differential equations $dx/dt = f(x)$, where $x = (x_1, x_2, x_3) \in \mathbb{R}^3$ and $x(t, x_0)$ is a solution of the equations which satisfies $x(0, x_0) = x_0$. We use a generalisation, to higher dimensions, of a criteria of Bendixson for the non-existence of invariant closed curves such as periodic or homoclinic orbits. The theory was developed by Li and Muldowney (1993, 1996) and shows that oriented infinitesimal line segments, $y(t, y_0)$, evolve as solutions of

$$\frac{dy}{dt} = \frac{\partial f}{\partial x}(x(t, x_0))y \tag{13}$$

and oriented infinitesimal areas, $z(t, z_0)$ evolve as solutions of

$$\frac{dz}{dt} = \frac{\partial f}{\partial x}^{[2]}(x(t, x_0))z \tag{14}$$

where $\frac{\partial f}{\partial x}^{[2]}$ is the second additive compound matrix. For a general matrix A, the corresponding second additive compound matrix is given by $A^{[2]}$ as follows,

$$A = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix},$$

$$A^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix}. \tag{15}$$

Thus, for Eqs. (5)–(7), the second additive compound matrix is given by Eq. (16).

$$\left(\frac{\partial f}{\partial x}\right)^{[2]} = \begin{pmatrix} -\mu_1 - \mu_2 - \frac{\gamma_1 a_1}{(a_1+x_1)^2} - \frac{\gamma_2 a_2}{(a_2+x_2)^2} & 0 & -\frac{\alpha_1 a_3}{(a_3+x_3)^2} \\ -\frac{\alpha_3 a_2}{(a_2+x_2)^2} & -\mu_1 - \mu_3 - \frac{\gamma_1 a_1}{(a_1+x_1)^2} - \frac{\gamma_3 a_3}{(a_3+x_3)^2} & 0 \\ 0 & \frac{\alpha_2 a_1}{(a_1+x_1)^2} & -\mu_2 - \mu_3 - \frac{\gamma_2 a_2}{(a_2+x_2)^2} - \frac{\gamma_3 a_3}{(a_3+x_3)^2} \end{pmatrix}. \quad (16)$$

By Theorem 3.3 of Li and Muldowney (1993) if for each $x_0 \in \mathbb{R}^3_+$ (13) and (14) are uniformly asymptotically stable then all line segments collapse to the origin and we have global stability of (0,0,0) and there exists no invariant closed curves (periodic orbits, homoclinic or heteroclinic cycles) and the orbits converge to a single equilibrium.

Asymptotic stability of (13) and (14) is shown by constructing Lyapunov functions. Using the Lyapunov function $V(x_1, x_2, x_3) = |x_1| + |x_2| + |x_3|$ and together with (13), we have

$$\dot{V}(y) = (1,1,1)\cdot\frac{\partial f}{\partial x} = -\mu_1 + \frac{a_1(\alpha_2-\gamma_1)}{(a_1+x_1)^2} - \mu_2 + \frac{a_2(\alpha_3-\gamma_2)}{(a_2+x_2)^2} - \mu_3 + \frac{a_3(\alpha_1-\gamma_3)}{(a_3+x_3)^2}$$

If $\dot{V}(y) < 0$, we have global stability of the zero solution of (13). Since $\gamma_1 \geq \alpha_2$ and $\gamma_2 \geq \alpha_3$, then a sufficient condition for $\dot{V}(y) < 0$ is $\mu_3 > (\alpha_1 - \gamma_3)/a_3$, condition (A) in Table 3. Showing that $\dot{V}(y) = (1,1,1)\cdot\left(\frac{\partial f}{\partial x}\right)^{[2]} < 0$ guarantees asymptotic stability of (14) and gives condition (C).

Alternatively, using the Lyapunov function $V(x_1, x_2, x_3) = \sup\{|x_1|, |x_2|, |x_3|\}$ gives stronger results (conditions B and D in Table 3).

Appendix B: $\mathcal{R}_0$ and tick-borne disease dynamics

While ticks can feed on a variety of hosts, it is commonly believed that pathogens are associated with a particular host that acts as a disease reservoir that maintains the pathogen in the environment (Randolph 2004). For instance, the spirochete *Borrelia burgdorferi s.l.* is maintained mainly in deer mice: the spirochete is transferred to the tick when it feeds on an infected deer mouse; after which, the infected tick can transfer the disease to a human, causing Lyme disease, or to another deer mouse—thus maintaining the disease in the environment. If the tick feeds on an alternate small or large host that is not a disease reservoir (e.g., pocket mice, rabbits, humans), the pathogen will either be eliminated by the immune system, or lead to the death of the host, or not be transferred to another host, in all cases effectively acting as a dead end that removes the pathogen from the environment.

Larval ticks typically hatch free from infection and can acquire infection through a blood meal with an infected small host, at which point they molt to become infected nymphs. So larval ticks cannot transmit the disease. Infected nymphs can transmit the infection to the hosts they feed upon and the infection remains in the ticks when they molt to the adult stage. Adopting the approach of Lou and Wu (2014) we can extend our model in a simple way to capture the disease dynamics of Lyme disease by describing the disease status of the individuals in our model. The rate of change of infected small H_1 hosts $H_1^I(t)$, infected nymphs $x_2^I(t)$ and infected adult ticks $x_3^I(t)$ are given by Eqs. (17)–(19).

We do not track infected large hosts as they can only transmit the infection to adult ticks which cannot pass the infection onto their offspring, so the large hosts are not acting as a reservoir for the disease the way that the small hosts are. We assume only the H_1 small hosts (e.g. deer mice) are a competent reservoir for the disease and that the H_2 small hosts are not (Ostfeld and Keesing 2000). β_H, β_L and β_N are the transmission coefficients of the infection to H_1 hosts, larval ticks and nymphal ticks, respectively. γ_i' is the contribution to γ_i that comes from feeding on H_1 hosts only similarly for α_i'. For example, $\gamma_2' = \sigma_1(p_s)H_1\lambda_{2,1}$. Assuming the tick population are at equilibrium then, we can study the disease dynamics in isolation replacing $x_1(t)$ and $x_2(t)$ by their equilibrium values x_1^* and x_2^* and noting that the equation for infected adult ticks decouples. Hence, two equations form the epidemiological model, Eqs. (20)–(21).

$$\dot{H}_1^I = -\overbrace{\mu_{H_1} H_1^I}^{\text{death}} + \overbrace{\beta_H \frac{H_1 - H_1^I}{H_1} \frac{\gamma_2' x_2^I}{a_2 + x_2}}^{\substack{\text{infected nymphs transmitting} \\ \text{disease to healthy hosts}}}, \tag{17}$$

$$\dot{x}_2^I = -\overbrace{\mu_2 x_2^I}^{\text{death}} + \overbrace{\beta_L \frac{H_1^I}{H_1} \frac{\alpha_2' x_1}{a_1 + x_1}}^{\substack{\text{larvae feeding on infected hosts} \\ \text{molting to become infected nymph}}} - \overbrace{\frac{\gamma_2' x_2^I}{a_2 + x_2}}^{\substack{\text{infected nymphs molting} \\ \text{to become infected adults}}}, \tag{18}$$

$$\dot{x}_3^I = -\overbrace{\mu_3 x_3^I}^{\text{death}} + \overbrace{\frac{\alpha_3'(x_2 - x_2^I)}{a_2 + x_2}}^{\substack{\text{infected nymphs molting} \\ \text{to become infected adults}}} + \overbrace{\beta_N \frac{H_1^I}{H_1} \frac{\alpha_3'(x_2 - x_2^I)}{a_2 + x_2}}^{\substack{\text{uninfected nymph feeding on infected hosts} \\ \text{and molting to become infected adults}}} - \overbrace{\frac{\gamma_3 x_3^I}{a_3 + x_3}}^{\substack{\text{adults taking final blood meal}}}. \tag{19}$$

$$\dot{H}_1^I = -\mu_{H_1} H_1^I + \beta_H \frac{H_1 - H_1^I}{H_1} \frac{\gamma_2' x_2^I}{a_2 + x_2^*}, \tag{20}$$

$$\dot{x}_2^I = -\mu_2 x_2^I + \beta_L \frac{H_1^I}{H_1} \frac{\alpha_2' x_1^*}{a_1 + x_1^*} - \frac{\gamma_2' x_2^I}{a_2 + x_2^*}. \tag{21}$$

We can calculate the basic reproduction number for the disease using the next generation matrix method (see Van den Driessche and Watmough 2002). The transmission matrix and transition matrix are given by

$$F = \begin{pmatrix} 0 & \frac{\beta_H \gamma_2'}{a_2 + x_2^*} \\ \frac{\beta_L \alpha_2' x_1^*}{H_1(a_1 + x_1^*)} & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \mu_{H_1} & 0 \\ 0 & \mu_2 + \frac{\gamma_2'}{a_2 + x_2^*} \end{pmatrix} \tag{22}$$

respectively. Together these yield the next generation matrix

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\beta_H \gamma_2'}{\mu_2(a_2 + x_2^*) + \gamma_2'} \\ \frac{\beta_L \alpha_2' x_1^*}{H_1(a_1 + x_1^*)\mu_{H_1}} & 0 \end{pmatrix}, \tag{23}$$

the dominant eigenvalue of which gives the basic reproduction number $\mathcal{R}_0$ for the disease.

$$\mathcal{R}_0 = \sqrt{\frac{\beta_H \gamma_2'}{\mu_2(a_2 + x_2^*) + \gamma_2'} \frac{\beta_L \alpha_2' x_1^*}{H_1(a_1 + x_1^*)\mu_{H_1}}} \tag{24}$$

$$= \sqrt{\frac{\beta_H \gamma_2'}{\mu_2(a_2 + x_2^*) + \gamma_2'} \frac{\beta_L \alpha_2'}{H_1 \alpha_2 \mu_{H_1}} \left(\mu_2 + \frac{\gamma_2}{a_2 + x_2^*}\right) x_2^*}$$

The unique endemic equilibrium is

$$H_1^{I*} = H_1 \left(1 - \frac{1}{\mathcal{R}_0^2}\right) \tag{25}$$

$$x_2^{I*} = \beta_L \frac{\alpha_2'}{\alpha_2} x_2^* \left(1 + \frac{\gamma_2 - \gamma_2'}{\gamma_2' + (a_2 + x_2^*)\mu_2}\right) \left(1 - \frac{1}{\mathcal{R}_0^2}\right) \tag{26}$$

Applying Theorem 2.1 from Lou and Jianhong (2014) shows that $\mathcal{R}_0$ determines the global stability of the endemic equilibrium. Specifically, if $\mathcal{R}_0 > 1$, the endemic equilibrium is globally asymptotically stable.

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