



Seasonal Population Dynamics of Ticks, and its Influence on Infection Transmission: a Semi-discrete Approach

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1 In this paper, a simple semi-discrete (ticks' feeding is assumed to occur only during
2 the summers of each year) model for tick population dynamics is presented. Con-
3 ditions for existence, uniqueness, and stability of a positive equilibrium are found;
4 the system is then studied numerically using parameter estimates calibrated for the
5 tick *Ixodes ricinus* in Trentino, Italy, and the sensitivity to parameters is examined.

6 Then, this model is extended to consider a tick-transmitted infection of one
7 species of hosts, while other hosts are incompetent to the infection. Assuming,
8 for simplicity, that the infection is not affecting the total number either of hosts or
9 ticks, a threshold condition for infection persistence is obtained. The dependence
10 of the equilibrium infection prevalence on parameters is studied numerically; in
11 particular, we considered how infection prevalence depends on host densities. This
12 analysis reveals that a 'dilution effect' occurs both for competent and for incompe-
13 tent hosts; this means that, besides a lower threshold for host densities for infection
14 to persist, there exists also an upper threshold: if host densities were higher than the
15 upper threshold, the infection would go to extinction. Numerically, it is found that,
16 for realistic parameter values, the upper threshold is not much higher than observed
17 densities.

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1. INTRODUCTION

21 Tick-borne diseases [for instance, rickettsiosis, Lyme disease, Ehrlichiosis,
22 relapsing fever, TBE (tick-borne encephalitis)] are serious health problem affect-
23 ing humans as well as domestic animals in many parts of the world. These
24 infections are generally transmitted through a bite of an infected tick, and it
25 appears that most of these infections are widely present in some wildlife species;
26 hence, an understanding of tick population dynamics and its interaction with hosts
27 is essential to understand and control such diseases (Hudson *et al.*, 2002).

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Our work is tailored to Lyme disease, which is transmitted, in Europe and North America, by *Ixodes ricinus*. Ixodidae ticks, after hatching from eggs, go through three life stages: larva, nymph, and adult. They pass from one life stage to another by moulting, after a blood meal. In temperate climates, the life cycle is strongly influenced by the seasonal rhythm: simplifying a very complex pattern (Randolph *et al.*, 2002), one can say that larvae and nymphs who feed in the season in which they emerge generally develop into nymphs and adults respectively in the next season. If they do not feed in their first season (summer), most die off but some (especially nymphs) can survive through winter and feed in the following year, developing (into adults) in the same year in which they feed. Adult ticks feed also during winter; they mate, with the male dying shortly after mating and the female remaining longer on the host. Afterwards, the female drops off the host and deposits about 3000 eggs. The hatching of larvae takes several weeks (48–135 days) and they start appearing from the summer onwards.

For most of the infections named above, transmission occurs during blood meals: a tick feeding on an infected host may become infected, and then carry the infection throughout its life, being able to transmit the infection to subsequent hosts. For some infections, especially TBE, infection may also be transmitted directly among ticks feeding close to each other ('co-feeding') (Jones *et al.*, 1987); this route will not be considered in the present paper.

There exist several papers [for instance, Caraco *et al.* (1998), O'Callaghan *et al.* (1998), Norman *et al.* (1999), Rosà *et al.* (2003)] that model tick and infection dynamics as a continuous process in time. However, as already described, tick population dynamics is strongly influenced, in temperate climates, by the seasonal pattern, with tick development from one stage to the next generally requiring one year. Randolph and Rogers (1997) described tick population dynamics under the influence of environmental conditions, while Sandberg and Awerbuch (1992) used a matrix model with month-dependent transition rates; neither of them, however, considered infection transmission.

Here we present a simple model for tick dynamics and infection transmission that takes into account the seasonal cycle, albeit in an extreme way. Tick feeding and infection dynamics is described as a continuous process in each 'summer', while tick development occurs through 'winters'. Therefore, we obtain a semi-discrete model in the variables L_n , N_n , and A_n , the densities of larvae, nymphs, and adults at the beginning of season n . We find the threshold conditions for tick persistence, and for the stability of the endemic equilibrium.

This simple model is then extended to consider infections from ticks to host and vice versa, under the simplifying assumption that infection is not affecting the total number of either ticks or hosts. Here too a threshold condition for infection-free equilibrium is obtained.

The system is then studied numerically, using parameter estimates based on data obtained from the Centre for Alpine Ecology, but considering also, through bifurcation diagrams, how uncertainties in parameters reflect in the qualitative behaviour

1 of the system. In particular, we study the effects of season length, host density,
 2 probability of immediate development, and winter survival probability of larvae on
 3 tick and infection dynamics.

4 2. THE MATHEMATICAL MODEL FOR TICK DYNAMICS

5 As stated above, we consider a very simple model for tick dynamics with two
 6 distinct seasons: ‘summer’ and ‘winter’. Feeding occurs as a continuous process
 7 during summer, while only moulting generally occurs in winter. To be precise,
 8 we assume that larvae and nymphs that feed during one ‘summer’ go through
 9 the moulting stage but arrest their development and emerge (as nymphs or adults,
 10 respectively) in the following ‘summer’. On the other hand, we assume that after
 11 the adult females feed and produce eggs, a proportion of the eggs hatch imme-
 12 diately, so that larvae emerge in the same ‘summer’, while the rest arrest their
 13 development, and larvae emerge in the following ‘summer’. Finally, we assume
 14 that larvae, nymphs, and adults die at the end of the ‘summer’ in which they have
 15 emerged, if they have not succeeded in feeding. Of course, these assumptions are
 16 rather crude with respect of the complex interactions between climatic factors, indi-
 17 vidual fat reserves, and feeding time (Randolph *et al.*, 2002); we believe, however,
 18 that they capture some essential features of ticks’ seasonal rhythm.

19 For the sake of simplicity, host population is assumed to be constant [see Rosà
 20 *et al.* (2003) for other assumptions].

21 These assumptions translate into the following model: in summer n , the vari-
 22 ables $L_n(t)$, $N_n(t)$, and $A_n(t)$ (densities of larvae, nymphs, and adults at time t of
 23 summer n) satisfy the following system of differential equations:

$$\begin{cases} \dot{L}_n(t) = pcg_A A_n(t) - (d_L + g_L)L_n(t), \\ \dot{N}_n(t) = -(d_N + g_N)N_n(t), \\ \dot{A}_n(t) = -(d_A + g_A)A_n(t), \end{cases} \quad (1)$$

24 where d_z ($z = L, N, A$) are the death rates (during the season) of the various
 25 stages, and g_z their feeding rates. Feeding rates will depend on encounter rates β_z
 26 and host densities; since we assumed host densities to be constant, feeding rates
 27 will also be constant: however, when we consider how tick dynamics is affected
 28 by host densities, we will use the explicit dependence of g_z on host densities.
 29

30 The constant p is the probability of immediate development of tick larvae, and c
 31 is the number of larvae produced per feeding adult (considering also their sex ratio);
 32 the latter is assumed to be constant [in contrast to the case for Rosà *et al.* (2003)],
 33 since in the literature density dependence is documented in moulting probabilities
 34 (Randolph and Rogers, 1997) but not in this quantity.

35 To this equation, valid in $(0, T)$ (T is the length of a summer), we associate initial
 36 conditions, depending on the previous year’s variables; to be precise:

$$\begin{cases} L_n(0) = w \int_0^T (1-p) c g_A A_{n-1}(s) ds \\ N_n(0) = m_L(\overline{L_{n-1}}) \int_0^T g_L L_{n-1}(s) ds \\ A_n(0) = m_N(\overline{N_{n-1}}) \int_0^T g_N N_{n-1}(s) ds. \end{cases} \quad (2)$$

Here w is the probability of survival through winter for larvae that have delayed development; $m_Z(\cdot)$ are the moulting rates, assumed to be decreasing functions of the average density (over the season) of that stage [see Randolph and Rogers (1997) for empirical evidence for this assumption]. The average densities are defined as

$$\overline{L_{n-1}} = \frac{1}{T} \int_0^T L_{n-1}(s) ds \quad (3)$$

and analogously for $\overline{N_{n-1}}$.

Solving (1), we find

$$\begin{cases} N_n(t) = N_n(0) e^{-(d_N+g_N)t} \\ A_n(t) = A_n(0) e^{-(d_A+g_A)t} \\ L_n(t) = p c g_A \int_0^t e^{-(d_L+g_L)(t-s)} A_n(s) ds \\ \quad = L_n(0) e^{-(d_L+g_L)t} + p c g_A A_n(0) \frac{e^{-(d_A+g_A)t} - e^{-(d_L+g_L)t}}{d_L + g_L - (d_A + g_A)} \end{cases} \quad (4)$$

under the generic assumption $d_L + g_L \neq d_A + g_A$.

From (4), we obtain

$$\overline{N_{n-1}} = \frac{N_{n-1}(0)}{T} \tau_N \quad \text{and} \quad \overline{L_{n-1}} = \frac{1}{T} (p c f_A \tau_{L_i} A_{n-1}(0) + \tau_{L_d} L_{n-1}(0)), \quad (5)$$

where

$$\tau_N = \int_0^T e^{-(d_N+g_N)s} ds = \frac{1 - e^{-(d_N+g_N)T}}{(d_N + g_N)}$$

represents the average time spent questing by a nymph,

$$\tau_{L_d} = \int_0^T e^{-(d_L+g_L)s} ds = \frac{1 - e^{-(d_L+g_L)T}}{(d_L + g_L)}$$

represents the average time spent questing by a larva that has delayed development,

$$f_A = g_A \int_0^T e^{-(d_A+g_A)s} ds = \frac{g_A}{d_A + g_A} (1 - e^{-(d_A+g_A)T})$$

represents the probability that an adult feeds, and

$$\tau_{L_i} = \frac{g_A}{f_A} \int_0^T e^{-(d_A+g_A)s} \int_s^T e^{-(d_L+g_L)(t-s)} dt ds$$

1 represents the average time spent questing by a larva with immediate development,
2 remembering that this will start at time s of a season.

3 The constants τ_N and τ_{L_d} arise naturally from (4), and happen to have a nice
4 biological interpretation. On the other hand, the constants f_A and τ_{L_i} have been
5 introduced in order to give a consistent biological interpretation of (5). To see that
6 (5) is indeed correct, one can compute

$$\begin{aligned} 7 \quad f_A \tau_{L_i} &= \frac{g_A}{(d_A + g_A)(d_L + g_L)} - \frac{g_A}{d_L + g_L - (d_A + g_A)} \\ 8 \quad &\times \left(\frac{e^{-(d_A + g_A)T}}{d_A + g_A} - \frac{e^{-(d_L + g_L)T}}{d_L + g_L} \right) \\ 9 \quad &= g_A \int_0^T \frac{e^{-(d_A + g_A)t} - e^{-(d_L + g_L)t}}{d_L + g_L - (d_A + g_A)} dt. \end{aligned}$$

10 Note that, for reasonable values of death and feeding rates, and of season lengths,
11 the exponential terms in these expressions are very close to zero, and one has the
12 approximations

$$13 \quad \tau_N \approx \frac{1}{d_N + g_N} \quad \tau_{L_d} \approx \tau_{L_i} \approx \frac{1}{d_L + g_L} \quad f_A \approx \frac{g_A}{d_A + g_A}. \quad (6)$$

14 With this notation, we can then obtain a discrete system for the densities at the
15 beginning of each season: $L_n \equiv L_n(0)$, $N_n \equiv N_n(0)$, and $A_n \equiv A_n(0)$. In fact,
16 from (2), using (4) and (5), we obtain

$$\begin{cases} 17 \quad L_n &= c(1-p)wf_A A_{n-1}, \\ 18 \quad N_n &= g_L(f_A c p \tau_{L_i} A_{n-1} + \tau_{L_d} L_{n-1})m_L \left(\frac{(f_A c p \tau_{L_i} A_{n-1} + \tau_{L_d} L_{n-1})}{T} \right), \\ 19 \quad A_n &= g_N \tau_N N_{n-1} m_N \left(\frac{N_{n-1} \tau_N}{T} \right). \end{cases} \quad (7)$$

20 All the parameters used in this model are presented, together with their biological
21 interpretation and a reference value, in Table 1.

22 Let us now look for a stationary (over n) solution of (7): set $L_n \equiv L^*$, $N_n \equiv N^*$
23 and $A_n \equiv A^*$ in (7). From the first equation, we obtain

$$24 \quad L^* = c(1-p)wf_A A^*. \quad (8)$$

25 Then, using (8) in the second and third equation, we obtain

$$26 \quad N^* = c g_L f_A A^* (p \tau_{L_i} + (1-p)w \tau_{L_d}) m_L \left(\frac{c f_A A^*}{T} (p \tau_{L_i} + (1-p)w \tau_{L_d}) \right) \quad (9)$$

Table 1. Notation used to denote the various parameters included in the model, together with the reference values used. In all parameters time is measured in days, and host densities are per hectare.

Symbol	Description	Value
H_1, H_2	Densities of two types of hosts	30, 0.1
β_1^z	Rates of encounters between questing ticks in stage z ($z = L, N, A$) and host H_1	0.028, 0.0009, 0
β_2^z	Rates of encounters between questing ticks in stage z ($z = L, N, A$) and host H_2	0.05, 0.03, 0.13
σ_i^z	Detachment rate of ticks in stage z ($z = L, N, A$) feeding on hosts H_i ($i = 1, 2$)	0.5
c_i^z	Computed quantity	β_i^z / β_i^z
g_z	Feeding rates in stage z ($z = L, N, A$) given by the relation (18)	0.31, 0.028, 0.013
d_z	Death rates of questing ticks in stage z ($z = L, N, A$)	0.05, 0.03, 0.03
$m_z(x)$	Moulting probability depending on average values x of ticks in stage z	$0.15 e^{-0.008x}$
p	Probability of immediate development of tick larvae	0.8
w	Winter survival probability of larvae that have delayed development	0.1
T	Length of summer	182
c	Average number of eggs per fed adult	1300

and

$$A^* = g_N N^* \tau_N m_N \left(\frac{N^*}{T} \tau_N \right). \quad (10)$$

Substituting (10) in (9), we see that N^* can either be equal to 0, or it must be a solution of the equation $G(N) = 1$ where

$$G(N) = g_L c_1 m_N(c_2 N) m_L \left(\frac{c_1}{T} N m_N(c_2 N) \right) \quad (11)$$

with

$$c_1 = c f_{AGN} \tau_N (p \tau_{L_i} + (1 - p) w \tau_{L_d}) \quad \text{and} \quad c_2 = \frac{\tau_N}{T}.$$

Once we have a solution N^* of $G(N) = 1$, (8) and (10) yield A^* and hence L^* . Thus we have a nontrivial equilibrium, say $E^* = (L^*, N^*, A^*)$.

If we assume

$$\lim_{x \rightarrow \infty} \min\{m_L(x), m_N(x)\} = 0 \quad (12)$$

we then see that a sufficient condition for having a nontrivial equilibrium is $G(0) > 1$, i.e.,

$$g_L c_1 m_N(0) m_L(0) = c g_L f_{AGN} \tau_N (p \tau_{L_i} + (1 - p) w \tau_{L_d}) m_N(0) m_L(0) > 1. \quad (13)$$

Condition (13) can be easily interpreted. In fact, $g_N \tau_N$ represents the probability

1 that a nymph feeds; analogously, $g_L(p\tau_{L_i} + (1-p)w\tau_{L_d})$ represents the probabili-
 2 ty that a larva feeds, averaging over the probability p that it develops immediately,
 3 and that, discounted by overwinter survival probability w , that it delays develop-
 4 ment. Hence, the left hand side of (13) computes the expected number of larvae
 5 produced by a larva, when density-dependent effects do not operate: the probability
 6 of developing into a nymph ($g_L(p\tau_{L_i} + (1-p)w\tau_{L_d})m_L(0)$) times the probability
 7 that the nymph develops into an adult ($g_N\tau_N m_N(0)$) times the expected number of
 8 larvae produced (f_{AC}).

9 Now we are interested in finding conditions that guarantee uniqueness of the
 10 roots of $G(N) = 1$, and hence of the positive equilibrium. If either m_L or m_N
 11 is constant, while the other is decreasing, it is immediate from the expression (11)
 12 that $G(N)$ is a decreasing function; hence, there is at most one root of $G(N) = 1$.

13 Things are different if both m_L and m_N are decreasing functions, because then
 14 $G(N)$ could be increasing. We will consider in detail two possible choice for these
 15 functions:

$$16 \quad m_z(x) = \frac{s_z}{1 + \mu x} \quad \text{or} \quad m_z(x) = s_z e^{-\mu x} \quad (14)$$

17 for $z = L, N$; here $s_z = m_z(0)$ represents the probability of moulting in a tick-free
 18 habitat, while μ measures the strength of density dependence in moulting proba-
 19 bility.

20 First, we compute $G'(N)$ without specifying the functions $m_z(\cdot)$; then we will
 21 insert (14). We have

$$22 \quad G'(N) = \frac{g_L c_1^2}{T} m'_L \left(\frac{c_1}{T} N m_N(c_2 N) \right) [m_N(c_2 N)]^2 + g_L c_1 c_2 m'_N(c_2 N) \\ 23 \quad \times \left[m_L \left(\frac{c_1}{T} N m_N(c_2 N) \right) + \frac{c_1}{T} N m_N(c_2 N) m'_L \left(\frac{c_1}{T} N m_N(c_2 N) \right) \right]. \\ 24 \quad (15)$$

25 The first term in (15) is clearly negative. As for the second term, we see that
 26 $m_L(x) = \frac{s_L}{1 + \mu x}$ satisfies

$$27 \quad m_L(x) + x m'_L(x) > 0 \quad \forall x \geq 0. \quad (16)$$

28 Hence the term in square brackets in (15) is positive, and the whole expression is
 29 negative.

30 On the other hand, if $m_L(x)$ and $m_N(x)$ are exponential functions, the situation
 31 is not this easy, and we need to compute (11) explicitly. Let then $m_L(x) = s_L e^{-\mu x}$
 32 and $m_N(x) = s_N e^{-\mu x}$ (for the sake of simplicity, we chose the same constant μ in
 33 both functions); setting $\mu c_2 N = x$, equation (11) with $G(N) = 1$ reduces to

$$34 \quad f(x) = 0, \quad (17)$$

35 where $f(x) = x(c_3 e^{-x} + 1) - \log(c_4)$, with

$$c_3 = \frac{c_1 s_N}{T c_2} = c f_A g_N (p \tau_{L_i} + (1-p) w \tau_{L_d}) s_N \quad 2$$

$$c_4 = g_L c_1 s_L s_N = g_L c_1 m_L(0) m_N(0). \quad 3$$

From equation (17) it is immediate that to have a positive root we need $\log(c_4) > 0$; this is equivalent to condition (13), which is then, in this case, a necessary and sufficient condition for the existence of positive equilibria. 4
5
6

Still we may have more than one positive root of equation (17), and we look for conditions that guarantee uniqueness. 7
8

We note that $f'(x) = 1 + c_3 e^{-x}(1-x)$, and $f''(x) = c_3 e^{-x}(x-2)$. This implies that the minimum of $f'(x)$ is at $x = 2$; hence $f'(x) \geq f'(2) = 1 - c_3 e^{-2}$. Therefore $f'(2) \geq 0$ (i.e., $c_3 \leq e^2$) $\Rightarrow f'(x) \geq 0 \forall x$. 9
10
11

Thus we see that a sufficient condition for the uniqueness of the positive root of (17) is $c_3 \leq e^2$. This condition is largely satisfied for the parameter values estimated from field data (see below). 12
13
14

The Jacobian matrix corresponding to the system (7) at the equilibrium point (L^*, N^*, A^*) can be written as follows: 15
16

$$M = \begin{pmatrix} 0 & 0 & c(1-p)w f_A \\ m_{21} & 0 & m_{23} \\ 0 & m_{32} & 0 \end{pmatrix}, \quad 17$$

where 18

$$m_{21} = g_L \tau_{L_d} m_L \left(\frac{f_A c p \tau_{L_i} A^* + \tau_{L_d} L^*}{T} \right) \quad 19$$

$$+ g_L (f_A c p \tau_{L_i} A^* + \tau_{L_d} L^*) \frac{\tau_{L_d}}{T} m'_L \left(\frac{f_A c p \tau_{L_i} A^* + \tau_{L_d} L^*}{T} \right) = g_L \tau_{L_d} D \quad 20$$

$$m_{23} = g_L f_A c p \tau_{L_i} \left[m_L \left(\frac{f_A c p \tau_{L_i} A^* + \tau_{L_d} L^*}{T} \right) \quad 21$$

$$+ \frac{f_A c p \tau_{L_i} A^* + \tau_{L_d} L^*}{T} m'_L \left(\frac{f_A c p \tau_{L_i} A^* + \tau_{L_d} L^*}{T} \right) \right] = g_L f_A c p \tau_{L_i} D \quad 22$$

$$m_{32} = g_N \tau_N [m_N (c_2 N^*) + c_2 N^* m'_N (c_2 N^*)] \quad 23$$

and $D = \left[m_L \left(\frac{c_1}{T} N^* m_N (c_2 N^*) \right) + \frac{c_1}{T} N^* m_N (c_2 N^*) m'_L \left(\frac{c_1}{T} N^* m_N (c_2 N^*) \right) \right]$ 24

Here $m'_z(u)$ (where $z = L$ or N) denotes the derivative of the function $m_z(\cdot)$ with respect to its argument, computed in terms of the value u . 25
26

We immediately note that, if $m_z(x) = \frac{s_z}{1+\mu_z x}$, we have that m_{21} , m_{23} , and m_{32} are all positive since then condition (16) holds. 27
28

Now, the characteristic polynomial of M is given by 29

$$P(\lambda) = \lambda^3 - m_{23} m_{32} \lambda - c(1-p)w f_A m_{21} m_{32}. \quad 30$$

1 The necessary and sufficient conditions (Jury conditions) for all roots of $P(\lambda) = 0$
 2 to satisfy $|\lambda| < 1$ are

- 3 (i) $P(1) > 0$;
 4 (ii) $(-1)^3 P(-1) > 0$;
 5 (iii) $|a_3| < 1$ and $|b_3| > |b_1|$, where

6
 7
$$a_3 = c(1-p)w f_A m_{21} m_{32} \quad b_3 = 1 - a_3^2, \quad b_1 = -m_{23} m_{32}.$$

8 It is easy to see that

9
$$P(1) = 1 - G(N^*) - N^* G'(N^*) = -N^* G'(N^*).$$

10 Hence, if $G'(N^*) < 0$ [which will always be true when $m_L(x) = \frac{s_L}{1+\mu_L x}$ and,
 11 more generally, when there exists a unique positive equilibrium], condition (i) will
 12 always hold.

13 Moreover, if the coefficients $m_{21} m_{32}$ and $m_{23} m_{32}$ are both positive, condition
 14 (i) implies (ii) and (iii). Hence, from the previous considerations, we see that, if
 15 $m_z(x) = \frac{s_z}{1+\mu_z x}$, the unique positive equilibrium is always stable when it exists, i.e.,
 16 when (13) is satisfied.

17 On the other hand, if $m_z(x) = s_z e^{-\mu_z x}$, conditions (ii) and (iii) do not follow from
 18 (i), so that it is possible to have a (unique) positive equilibrium which is unstable.
 19 This is later shown numerically.

20 Note finally that condition (i) for the stability of the tick-free equilibrium $E_0 \equiv$
 21 $(0, 0, 0)$ is $g_L c_1 m_N(0) m_L(0) < 1$, i.e., the opposite of (13); since in this case
 22 m_{21} , m_{23} , and m_{32} are all positive, conditions (ii) and (iii) will then be automatically
 23 satisfied.

24 **2.1. Simulations.** The simulations were performed using parameter values con-
 25 sidered to be reasonable for describing *Ixodes ricinus* tick populations in Trentino
 26 [see [CEA Report \(2000\)](#) for background information]. As in [Rosà et al. \(2003\)](#),
 27 the feeding rates g_z are assumed to depend on host densities according to a saturat-
 28 ing function, because of the extended feeding period. To be precise, we used the
 29 relation

30
$$g_z(H_1, H_2) = \frac{\beta_1^z H_1 + \beta_2^z H_2}{1 + c_1^z H_1 + c_2^z H_2} \quad (18)$$

31 where H_1 and H_2 are the densities of two types of hosts (typically H_1 and H_2
 32 represent rodents, especially *Apodemus spp.* and *Chlethrynomis galreolus*, while
 33 H_2 represent ungulates, especially roe deer), β_i^z are the rates of contacts between
 34 hosts i and questing ticks in stage z ($z = L, N, A$), σ_i^z are the detachment rates of
 35 ticks in stage z feeding on hosts H_i , and $c_i^z = \frac{\beta_i^z}{\sigma_i^z}$. This saturating function is found
 36 using a quasi-equilibrium relation in a model that distinguishes between questing
 37 and feeding ticks ([Mwambi et al., 2000](#)).

All parameter values used are shown in Table 1; we briefly sketch here the motivation for the choices. From several experiments and observations, described in Rosà *et al.* (2004), estimates for the encounter rates β_i^z were found, and these are reported in Table 1. The following estimates (measuring time in days, and host densities per hectare) were obtained:

$$\beta_1^L = 0.028, \quad \beta_1^N = 0.0009, \quad \beta_1^A = 0, \quad \beta_2^L = 0.05, \quad \beta_2^N = 0.03, \quad \beta_2^A = 0.13.$$

The duration of a meal is in the range of 2–3 days (Sonenshine, 1991), so that $\sigma_i^z \approx 0.5 \text{ d}^{-1}$. Densities of rodents in the province of Trento range, according to year and location, between 5 and 30 per hectare, while densities of roe deer are generally around 0.1 per hectare. Using the previous estimates with $H_1 = 30$, $H_2 = 0.1$, we get the following values for g_L , g_N , and g_A that will be used as reference values:

$$g_L = 0.31, \quad g_N = 0.028, \quad g_A = 0.013 \quad (\text{d})^{-1}.$$

As for demographic parameters, we use the following values based on some literature data: $c = 1300$ (average number of eggs per fed adult), taking into account a 1 : 1 sex ratio; $d_L = 0.05$, $d_N = d_A = 0.03$ (death rates of questing ticks). As for the moulting probability of fed larvae and nymphs, we choose exponential functions, using 0.15 as a normal moulting probability (Humair *et al.*, 1999); the value of μ is chosen so as to have a reasonable density of nymph population per hectare ($N^* \approx 400$). Finally, the values of $p = 0.8$ and $w = 0.1$ have been chosen somewhat arbitrarily.

For the reference values, system (7) has a stable equilibrium, to which all numerical solutions appear to converge (see an example in Fig. 1) to the values

$$L^* = 147.124, \quad N^* = 376.846, \quad A^* = 20.6533.$$

We have studied rather extensively how the equilibrium values depend on the parameter values. In Fig. 2 we show the dependence on p , the probability of immediate development. It can be seen that p must be larger than 0.22 to have a positive equilibrium, and that the equilibrium densities of nymphs and adult ticks always increase with p [this can be understood from (13), since, for these parameter values, $\tau_{L_i} > w\tau_{L_d}$].

Season length (T) also, as expected, has a positive effect on the equilibrium level of ticks, as shown in Fig. 3. It appears that the dependence is almost linear.

A more complex effect can be seen in the variation of the equilibrium when the feeding rates are changed. In Fig. 4, we show the dependence of N^* on g_N . First of all, it can be seen that, surprisingly, the relation is not monotone. Initially, N^* increases with g_N , but, at values not much higher than the estimated one, it starts decreasing. Second, at a value of $g_N \approx 0.43$, there is a Neimark–Sacker bifurcation, meaning that for g_N greater than that, the equilibrium will be unstable, and there will probably be periodic or quasi-periodic solutions. Remember that, if the

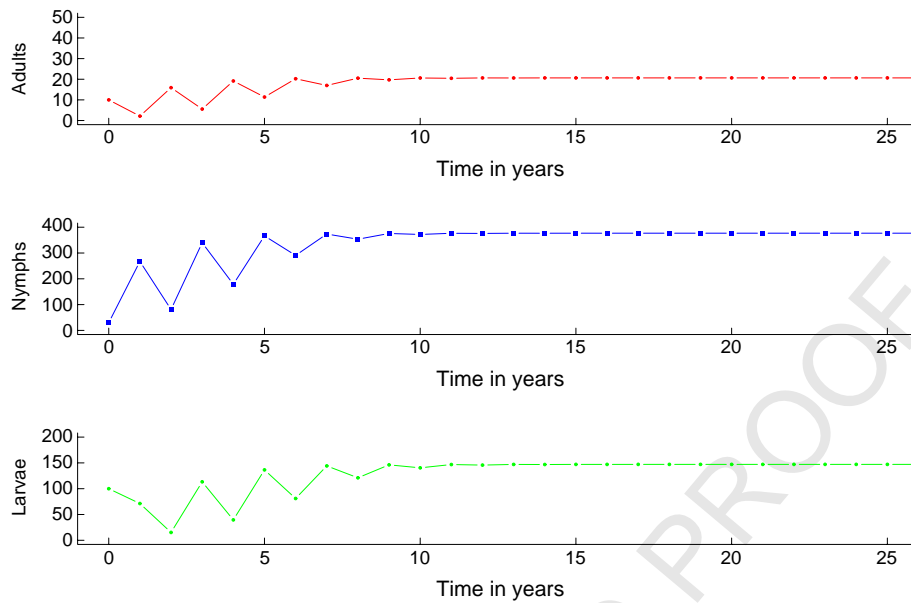


Figure 1. Variation of larvae, nymphs, and adults with time. The parameter values are as in Table 1.

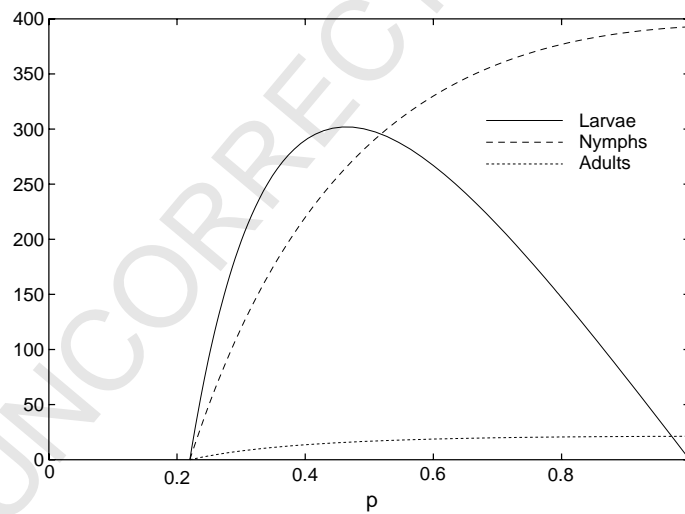


Figure 2. Variation of the equilibrium level of larvae, nymphs, and adults with p . All other parameter values are as in Table 1.

- 1 functional form $m_z(x) = \frac{s_z}{1+\mu x}$ had been used, instability of the equilibrium could
- 2 not occur.
- 3 In Fig. 5 we show the Neimarck–Sacker bifurcation points in the two-dimen-
- 4 sional $(p - g_N)$ plane. It can be seen that high values of either parameter tend to

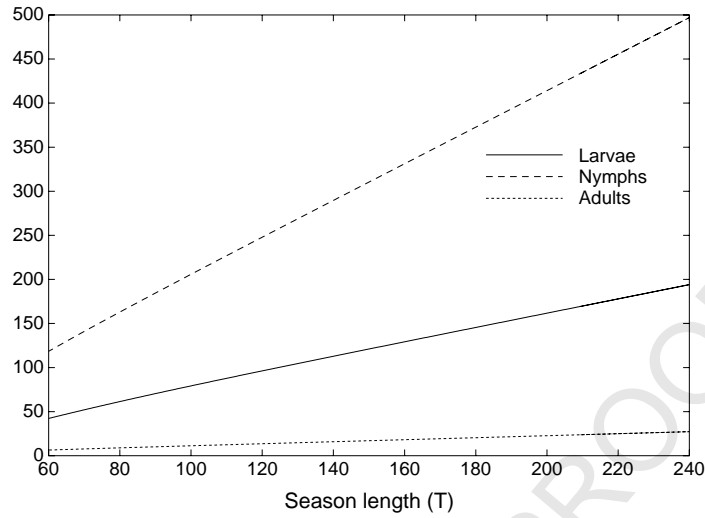


Figure 3. Variation of the equilibrium level of larvae, nymphs, and adults with T . All other parameter values are as in Table 1.

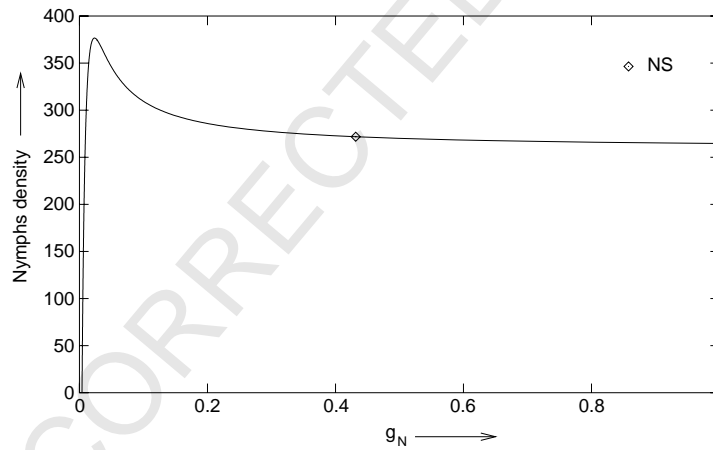


Figure 4. The equilibrium level of the nymphs for varying g_N . All other parameter values are as in Table 1. NS is the value at which Neimarck–Sacker bifurcation occurs.

destabilize the equilibrium; a similar pattern is shown in Fig. 6 for the $(g_A - g_N)$ plane. 1

In Fig. 7, we show a numerical simulation of the system for parameter values beyond the Neimarck–Sacker bifurcation point. It can be seen that the solution approaches a four-year cycle. 2 3 4

The feeding rates depend on host densities through the relations (18). However, changing host densities will affect all feeding rates simultaneously, and so the parameters $f_A, \tau_N, \tau_{L_i}, \tau_{L_d}$ present in $G(N)$. In order to understand this cumulative effect, we computed the equilibrium values for different sets of parameters 5 6 7 8 9

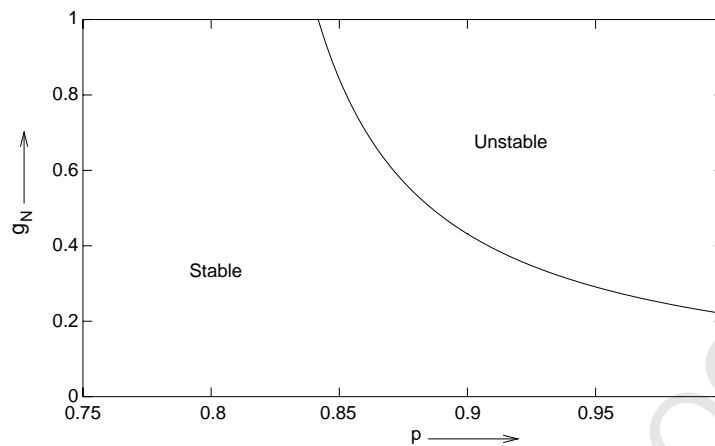


Figure 5. The stability region in the $(p - g_N)$ plane. All other parameter values are as in Table 1.

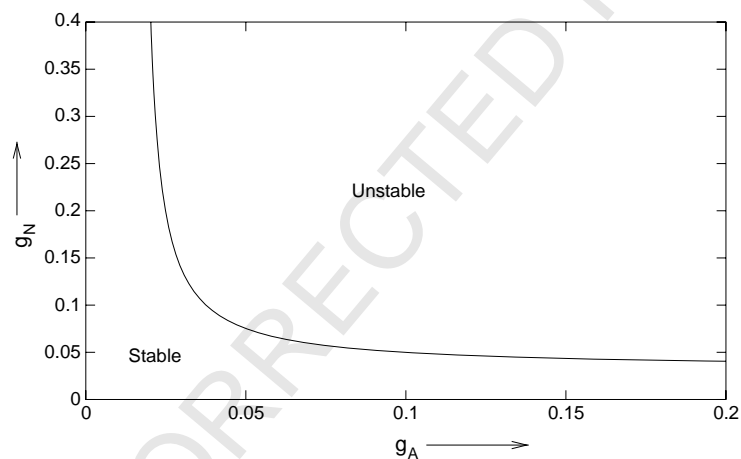


Figure 6. The stability region in the $(g_A - g_N)$ plane. All other parameter values are as in Table 1.

1 obtained by changing H_1 and H_2 . Fig. 8(a) and 8(b) shows the effect of host
 2 densities on the equilibrium level of nymphs; in the upper part H_2 is kept fixed at
 3 0.1, while H_1 varies between 5 and 50 (which encompasses densities usually found
 4 in the region); in the lower part, H_1 is fixed at 30, while H_2 varies between 0.08
 5 and 0.12.

6 3. THE MATHEMATICAL MODEL FOR TICKS WITH INFECTION

7 In this section we integrate the previous model for tick population dynamics with
 8 the dynamics of a tick-borne infection.

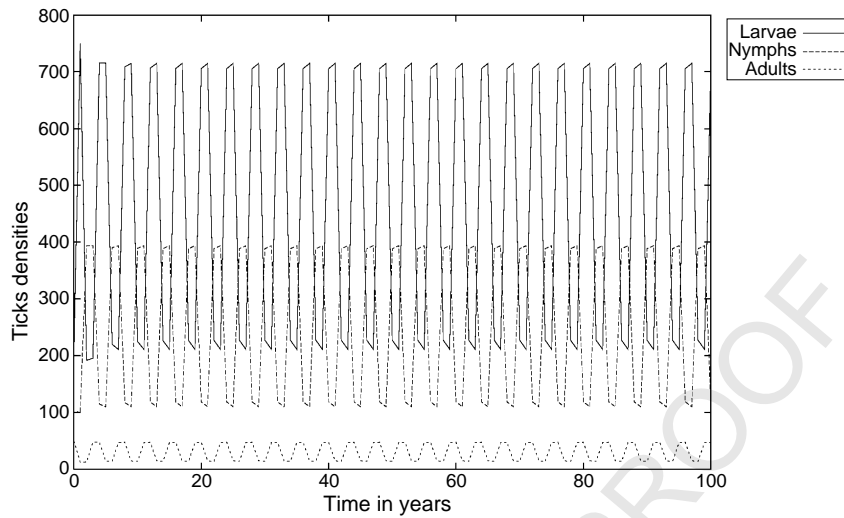


Figure 7. A numerical simulation of system (1) beyond the bifurcation point. The parameter values used are $g^N = 0.2$, $g^A = 0.05$; all other parameter values are as in Table 1.

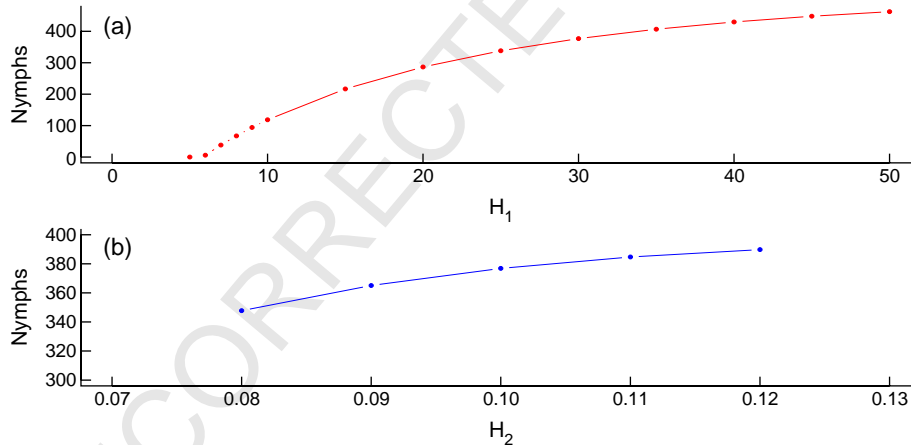


Figure 8. The equilibrium value of nymphs for varying H_1 and H_2 . All other parameter values are as in Table 1.

We assume that infection is transmitted from infected ticks to susceptible hosts, or vice versa from infected hosts to susceptible ticks, while a tick is feeding on a host. A larva feeding on an infected host will become, after moulting, an infected nymph; analogously, a nymph feeding on an infected host will become an infected adult. In both cases, infection is assumed to last forever. On the other hand, we will assume that a host, after a period of infection, will become immune and no longer capable of transmitting the infection, although the evidence is dubious.

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1 For the sake of simplicity, we disregard transmission between co-feeding ticks,
 2 although this may be relevant in certain infections (Rosà *et al.*, 2003). We also
 3 assume that the infection does not affect either tick or host demography; hence,
 4 we can assume that host population is constant (otherwise, it might fluctuate for
 5 reasons other than interactions with ticks), and also that total tick population is
 6 described by the model presented in the previous section.

7 Therefore, the variables of the model will be just the densities of ticks and hosts
 8 in the various infection stages: to be precise, we will consider susceptibles (N^s)
 9 and infective (N^i) nymphs [$N = N^s + N^i$ will satisfy equation (7)]; similarly, we
 10 consider susceptibles (A^s) and infective (A^i) adults ($A = A^s + A^i$). Only hosts
 11 of species 1 are assumed to become infected. These are divided into susceptibles
 12 (H^s), infectives (H^i), and immune (H^r) classes where $H_1 = H^s + H^i + H^r$ is
 13 taken as a constant.

14 We will append a subscript n to denote the values of the variables in the year n .
 15 As in the previous section, we assume that tick feeding occurs only during sum-
 16 mers, of length T . The equations for the tick stages are exactly like (1), except that
 17 we distinguish between susceptible and infected ticks. Using the same assumptions
 18 as in Rosà *et al.* (2003), the following differential equations valid for $t \in (0, T)$
 19 are obtained (T is the length of the summer):

$$\begin{aligned}
 \dot{L}_n &= pcg_A(A_n^i + A_n^s) - (d_L + g_L)L_n, \\
 \dot{N}_n^s &= -(d_N + g_N)N_n^s, \\
 \dot{N}_n^i &= -(d_N + g_N)N_n^i, \\
 \dot{A}_n^s &= -(d_A + g_A)A_n^s, \\
 \dot{A}_n^i &= -(d_A + g_A)A_n^i.
 \end{aligned} \tag{19}$$

21 Hosts can become infected (with probability q^z) if an infective tick feeds on them.
 22 As in the previous section [see also Rosà *et al.* (2003)] we assume that feeding
 23 rates g^z have the form (18) which we rewrite as

$$\begin{aligned}
 g^z(H_1, H_2) &= (\beta_1^z H_1 + \beta_2^z H_2) \psi^z(H_1, H_2) \quad \text{with} \\
 \psi^z(H_1, H_2) &= \frac{1}{1 + c_1^z H_1 + c_2^z H_2}.
 \end{aligned}$$

26 Hence, the rate at which susceptible hosts become infected is

$$q^N \beta_1^N \psi^N(H_1, H_2) N_n^i + q^A \beta_1^A \psi^A(H_1, H_2) A_n^i$$

28 giving rise to the following equations:

$$\begin{aligned}
\dot{H}_n^s &= a_1(H_1)H_1 - b_1H_n^s - q^N\beta_1^N H_n^s\psi^N(H_1, H_2)N_n^i \\
&\quad - q^A\beta_1^A H_n^s\psi^A(H_1, H_2)A_n^i, \\
\dot{H}_n^i &= q^N\beta_1^N H_n^s\psi^N(H_1, H_2)N_n^i + q^A\beta_1^A H_n^s\psi^A(H_1, H_2)A_n^i - (b_1 + \gamma)H_n^i, \\
\dot{H}_n^r &= \gamma H_n^i - b_1H_n^r.
\end{aligned} \tag{20}$$

During winter, hosts are assumed to follow the same equations, except for the infections. Hence in winter n for $t \in (T, 365)$, we have the following equations:

$$\begin{aligned}
\dot{H}_n^s &= a_1(H_1)H_1 - b_1H_n^s, \\
\dot{H}_n^i &= -(b_1 + \gamma)H_n^i, \\
\dot{H}_n^r &= \gamma H_n^i - b_1H_n^r.
\end{aligned} \tag{21}$$

Finally, (19) and (20) need to be complemented with initial conditions. Those for hosts come simply from (21) with the appropriate change in subscripts. Those for ticks are the same as (2), except that we keep track of the fact that larvae (nymphs) that have fed on infected hosts will emerge as infected nymphs (adults):

$$\begin{aligned}
L_{n+1}(0) &= w \int_0^T (1-p)cg_A(A_n^i(s) + A_n^s(s))ds, \\
N_{n+1}^s(0) &= m^L(\overline{L}_n) \left[\int_0^T g_L L_n(s)ds - \int_0^T \beta_1^L H_n^i(s)\psi^L L_n(s)ds \right], \\
N_{n+1}^i(0) &= m^L(\overline{L}_n) \int_0^T \beta_1^L H_n^i(s)\psi^L L_n(s)ds, \\
A_{n+1}^s(0) &= m^N(\overline{N}_n) \left[\int_0^T g_N N_n^s(s)ds - \int_0^T \beta_1^N H_n^i(s)\psi^N N_n^s(s)ds \right], \\
A_{n+1}^i(0) &= m^N(\overline{N}_n) \int_0^T \beta_1^N H_n^i(s)\psi^N N_n^s(s)ds, \\
H_{n+1}^s(0) &= H_n^s(365), \\
H_{n+1}^i(0) &= H_n^i(365), \\
H_{n+1}^r(0) &= H_n^r(365).
\end{aligned} \tag{22}$$

All parameters of the model (19)–(22) are summarized in Table 2.

As already stated, we will consider this model only at stationary population sizes. Namely, we will assume that

$$H_n^s + H_n^i + H_n^r \equiv H_1^*$$

Table 2. Parameters included in the model with infection beyond those already listed in Table 1.

Symbol	Description	Value
b_1	Birth or death rate of host H_1	1/365
γ	Recovery rate of host H_1	0.01
q^z	Probability of becoming infected for a host 1 bitten by an infectious tick in stage z ($z = N, A$)	0.5
ψ^z	Computed quantity	$\frac{1}{1+c_1^z H_1+c_2^z H_2}$
β^N	Computed quantity	$q^N \beta_1^N \psi^N$

1 where $a_1(H_1^*) = b_1$. As for ticks, we will assume

$$\begin{cases}
 N_n^s(t) + N_n^i(t) = N^* e^{-(d_N+g_N)t} \\
 A_n^s(t) + A_n^i(t) = A^* e^{-(d_A+g_A)t} \\
 L_n(t) = L^* e^{-(d_L+g_L)t} + pcg_A A^* \frac{e^{-(d_A+g_A)t} - e^{-(d_L+g_L)t}}{d_L + g_L - (d_A + g_A)} = A^* l(t)
 \end{cases} \quad (23)$$

3 with

$$l(t) = c \left(pg_A \frac{e^{-(d_A+g_A)t} - e^{-(d_L+g_L)t}}{d_L + g_L - (d_A + g_A)} + (1-p)wf_A e^{-(d_L+g_L)t} \right). \quad (24)$$

5 The only unknowns of the model are then $H^s(t)$, $H^i(t)$, $N^i(t)$, and $A^i(t)$. If we
 6 assume that $\beta_1^A = 0$ (i.e., adult ticks do not feed on hosts 1, as is generally true
 7 if they represent rodents), it turns out that all the other variables are independent
 8 of the value of $A^i(t)$, since we assumed that there is no transovarial transmission.
 9 The only variables of interest are then $H^s(t)$, $H^i(t)$, and $N^i(t)$.

10 We start by solving (19) and (20) on $(0, T)$. To simplify the notation, we drop
 11 the subscripts n and we let $b = b_1 = a_1(H_1^*)$ be the birth or the death rate of the
 12 host population, and $\beta^N = q^N \beta_1^N \psi^N$. We easily find

$$13 \quad N^i(t) = N^i(0)e^{-(d_N+g_N)t}.$$

14 We then have a linear equation for H^s :

$$15 \quad \dot{H}^s + [b + \beta^N N^i(0)e^{-(d_N+g_N)t}]H^s = bH_1^*$$

16 which gives

$$17 \quad H^s(t) = H^s(0)F_1(t, N^i(0)) + H_1^*F_2(t, N^i(0)),$$

18 where

$$19 \quad F_1(t, N^i) = e^{-bt} e^{-\frac{\beta^N N^i}{d_N+g_N}[1-e^{-(d_N+g_N)t}]}$$

18

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and

$$F_2(t, N^i) = b \int_0^t e^{-b(t-s) - \frac{\beta^N N^i}{d_N + g_N} [e^{-(d_N + g_N)s} - e^{-(d_N + g_N)t}]} ds.$$

Similarly we can write,

$$H^i(t) = H^i(0)e^{-(b+\gamma)t} + N^i(0)[H^s(0)G_1(t, N^i(0)) + H_1G_2(t, N^i(0))],$$

where

$$G_1(t, N^i) = \beta^N \int_0^t F_1(s, N^i) e^{-(d_N + g_N)s} e^{-(b+\gamma)(t-s)} ds,$$

$$G_2(t, N^i) = \beta^N \int_0^t F_2(s, N^i) e^{-(d_N + g_N)s} e^{-(b+\gamma)(t-s)} ds.$$

Now, using (21) and (22), we obtain a system of difference equations in the variables $N^i[n] \equiv N_n^i(0)$, $H^i[n] \equiv H_n^i(0)$, and $H^s[n] \equiv H_n^s(0)$:

$$\begin{cases} N^i[n+1] = k^L A^* (k^i H^i[n] + H^s[n] N^i[n] R_1(N^i[n]) \\ \quad + H_1^* N^i[n] R_2(N^i[n])) \\ H^i[n+1] = p_a^i H^i[n] + p_w^i N^i[n] (H^s[n] G_1(N^i[n]) + H_1^* G_2(N^i[n])) \\ H^s[n+1] = p_w^s H^s[n] F_1(N^i[n]) + H_1^* (1 - p_w^s (1 - F_2(N^i[n]))) \end{cases} \quad (25)$$

with

$$F_j(N^i) \equiv F_j(T, N^i) \quad \text{and} \quad G_j(N^i) \equiv G_j(T, N^i) \quad \text{for } j = 1, 2,$$

and

$$k^L = m^L (\bar{L}) \psi^L \beta_1^L \quad k^i = \int_0^T e^{-(b+\gamma)t} l(t) dt,$$

$$p_a^i = e^{-(b+\gamma)365} \quad p_w^i = e^{-(b+\gamma)(365-T)} \quad p_w^s = e^{-b(365-T)}$$

$$R_1(N^i) = \int_0^T G_1(t, N^i) l(t) dt, \quad R_2(N^i) = \int_0^T G_2(t, N^i) l(t) dt$$

using, in all cases, the expression (24).

The structure of the system (25) is rather simple, although the functions involved, F_2 , G_1 , G_2 , R_1 , and R_2 , cannot be written in an explicit analytic form, and even the analytic computations that are possible (such as that for k^i) do not help in making the results more transparent.

It must be noted however that, for reasonable values of the recovery rate γ , we have that $p_a^i, p_w^i \approx 0$; hence, the values of $H^i[n]$ are always very close to 0, and system (25) is approximately two-dimensional.

1 **3.1. Equilibria and stability.** System (25) has a disease-free equilibrium at $E_0 =$
 2 $(0, 0, H_1^*)$. The Jacobian at equilibrium is

$$3 \quad J_0 = \begin{pmatrix} k^L A^* H_1^* (R_1(0) + R_2(0)) & k^L A^* k^i & 0 \\ p_w^i H_1^* (G_1(0) + G_2(0)) & p_a^i & 0 \\ p_w^s H_1^* (F_1'(0) + F_2'(0)) & 0 & p_w^s F_1(0) \end{pmatrix}.$$

4 The eigenvalues of J_0 are $\lambda_3 = p_w^s F_1(0) = e^{-365b} < 1$, and the two eigenvalues of

$$5 \quad A = \begin{pmatrix} k^L A^* H_1^* (R_1(0) + R_2(0)) & k^L A^* k^i \\ p_w^i H_1^* (G_1(0) + G_2(0)) & p_a^i \end{pmatrix}.$$

6 Since all components of A are positive, the dominant eigenvalue of A is real and
 7 positive and through some computations that exploit the fact that $p_a^i < 1$, one
 8 easily see that the dominant eigenvalue is larger than 1; hence E_0 is unstable if and
 9 only if

$$10 \quad R_{0,\text{inf}} = k^L A^* H_1^* \left(R_1(0) + R_2(0) + \frac{k^i p_w^i (G_1(0) + G_2(0))}{1 - p_a^i} \right) > 1. \quad (26)$$

11 From

$$12 \quad G_1(0) + G_2(0) = \beta^N \frac{e^{-(b+\gamma)T} - e^{-(d_N+g_N)T}}{d_N + g_N - (b + \gamma)}$$

13 and

$$14 \quad R_1(0) + R_2(0) = \frac{\beta^N}{d_N + g_N - (b + \gamma)} \int_0^T (e^{-(b+\gamma)t} - e^{-(d_N+g_N)t}) l(t) dt$$

15 we obtain, recalling also the definition of β^N and k^L ,

$$16 \quad R_{0,\text{inf}} = A^* H_1^* \frac{m^L(\bar{L}) \beta_1^L \psi^L q^N \beta_1^N \psi^N}{d_N + g_N - (b + \gamma)} \left(\int_0^T (e^{-(b+\gamma)t} - e^{-(d_N+g_N)t}) l(t) dt \right. \\ 17 \quad \left. + \frac{k^i p_w^i (e^{-(b+\gamma)T} - e^{-(d_N+g_N)T})}{1 - p_a^i} \right). \quad (27)$$

18 Expression (27) can be interpreted as the average number of larvae that get infected
 19 starting with a newly infected larva. We can think that a larva infected during a
 20 ‘summer’ will have, in order to transmit the infection, to successfully moult and,
 21 then, as a nymph, bite a susceptible host of type 1 and infect it. That host can then
 22 transmit the infection to other larvae within the same season, or in the next year;
 23 the first term in (27) counts infections occurring within the same season, while
 24 the second term (which will be very small since $p_w^i \approx 0$) counts infections in the

following years. If that host infects other nymphs, these are dead ends for the infection, since, at least in this model, adults bite only incompetent hosts.

In order to interpret (27), we note that, for a nymph emerging at the beginning of a season, the probability density of biting a host of type 1 at time s is

$$\beta_1^N H_1^* \psi^N e^{-(d_N+g_N)s}.$$

To obtain the probability density, for a newly infected larva, of infecting a host of type 1, this quantity has to be multiplied by the probability of successfully moulting ($m^L(\bar{L})$) and that of infecting the host (q^N). The total number of larvae infected in the same season by that newly infected host will be computed by integrating for t from time s to the end of the season T the rate at which it is bitten by larvae: this is

$$e^{-(b+\gamma)(t-s)} \beta_1^L A^* l(t) \psi^L,$$

since $e^{-(b+\gamma)(t-s)}$ is the probability that the host is still alive and infectious at time t , and $A^* l(t)$ is the density of larvae at time t of a season. Overall, the average number of larvae that, starting with a newly infected larva in year n , get infected in year $n + 1$ is

$$m^L(\bar{L}) \int_0^T \beta_1^N H_1^* \psi^N e^{-(d_N+g_N)s} q^N \int_s^T e^{-(b+\gamma)(t-s)} \beta_1^L A^* l(t) \psi^L dt ds. \quad (28)$$

By exchanging the order of integration, (28) can be written as

$$m^L(\bar{L}) \beta_1^N H_1^* \psi^N q^N \beta_1^L A^* \psi^L \int_0^t e^{-(b+\gamma)t} l(t) \int_0^t e^{[b+\gamma-(d_N+g_N)]s} ds dt$$

which, on computing the inner integral, is clearly equal to the first term in (27). The second term can be interpreted analogously.

The rather complex expression (27) can be approximated, by recalling that $p_w^i \approx 0$ and that the same is true for all exponential terms like $e^{-(b+\gamma)T}$, $e^{-(d_N+g_N)T}$...

Then, one obtains

$$\begin{aligned} R_{0,\text{inf}} \approx & \frac{c A^* \beta_1^L H_1^* \psi^L m^L(\bar{L}) q^N \beta_1^N \psi^N}{(d_N + g_N - (b + \gamma))(d_L + g_L + b + \gamma)(d_N + g_N + d_L + g_L)} \\ & \times \left[p g_A \left(\frac{d_N + g_N + d_L + g_L}{b + \gamma + d_A + g_A} - \frac{b + \gamma + d_L + g_L}{d_N + g_N + d_A + g_A} \right) \right. \\ & \left. + w(1 - p) f_A(d_N + g_N - (b + \gamma)) \right]. \quad (29) \end{aligned}$$

Expression (29) is still rather complex, so it is difficult to understand well the effect of parameters. One can see that tick density, A^* , density of hosts 1, H_1^* , probability

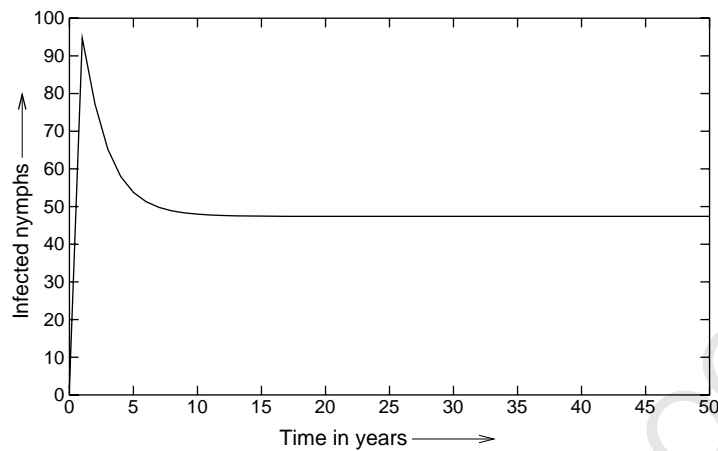


Figure 9. Variation in time of infected nymphs. Parameter values are as in Tables 1 and 2.

1 of host infection, q^N , all have a direct positive effect on infection persistence. On
 2 the other hand, there are indirect negative effects, since an increase in tick den-
 3 sity decreases the term $m^L(\bar{L})$; considering then host densities: if they increase,
 4 generally tick densities will also increase (as seen in the previous section) with the
 5 consequent direct and indirect effects; moreover, increasing host density will also
 6 decrease the term ψ^N , and affect all feeding rates g^z with results difficult to predict.

7 **3.2. Effect of host densities.** Using the reference values shown in Tables 1 and
 8 2 for the parameters, we found that $R_{0,\text{inf}} > 1$ and that the solutions converge to an
 9 endemic equilibrium with infection present (in Fig. 9 we show a simulation).

10 Instead of performing a sensitivity analysis on all parameters, we concentrate on
 11 the effect of host densities on the system, since this has also been the focus of other
 12 theoretical works on tick-borne infections (Norman *et al.*, 1999; Rosà *et al.*, 2003).

13 As discussed before, it is not easy to study analytically the effect of host densi-
 14 ties on the infection threshold, let alone on equilibrium densities. Therefore, we
 15 resorted to a numerical study, whose results are shown in Fig. 10. It turns out
 16 that host densities have a non-monotone effect: there exists a first threshold below
 17 which infection cannot be sustained, but also a second threshold above which infec-
 18 tion is eradicated. In between, solutions converge to an endemic equilibrium, at
 19 least for the parameter values considered.

20 Moreover, it can be seen that this negative effect of host densities on infection
 21 persistence occurs at densities not much larger than those usually estimated in
 22 Trentino: densities of infected nymphs and hosts start decreasing just as the den-
 23 sity of hosts 2 (roe deer) passes beyond the average density (0.1) and go to zero
 24 at a density of 0.4 per hectare; as for hosts 1, infection density starts decreasing
 25 as density goes beyond 50–60 per hectare (against an average density in the range
 26 10–30) and reaches 0 at a density around 110 per hectare.

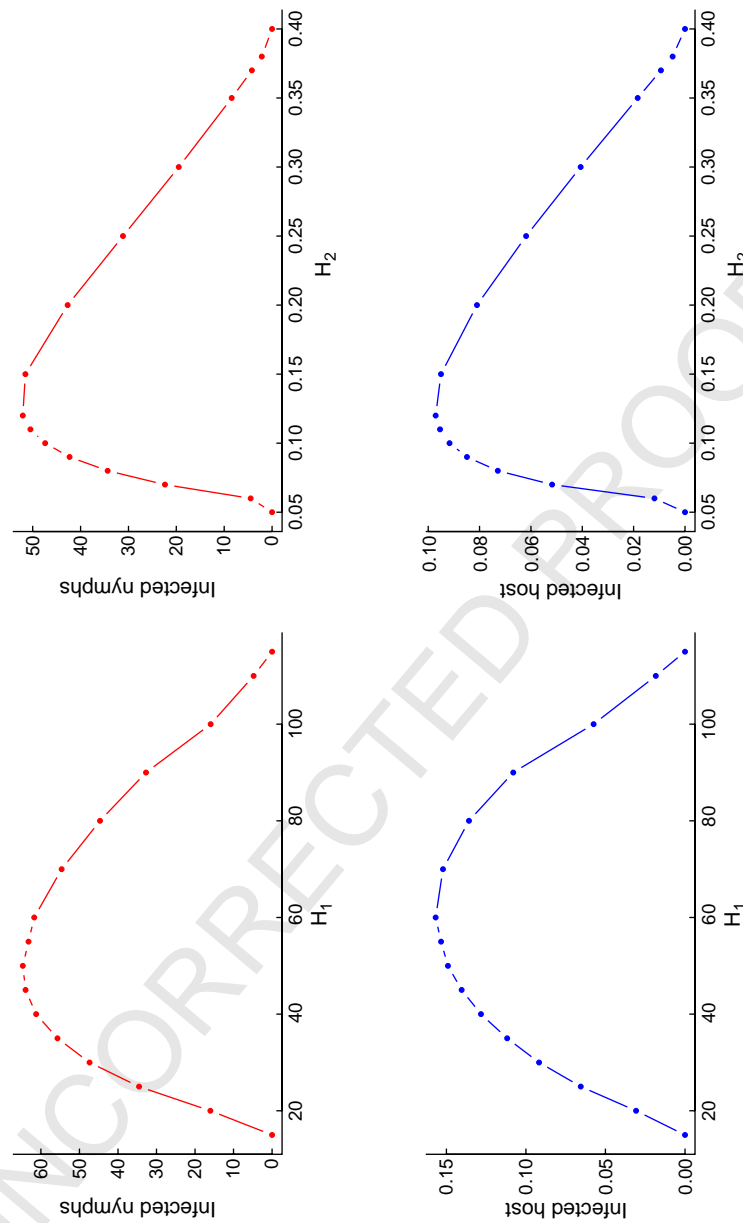


Figure 10. Variation of infected nymphs and hosts with H_1 and H_2 . Other parameter values are as in Tables 1 and 2.

4. DISCUSSION

1

The aim of this paper was to study how the discrete nature of the tick life cycle, especially in temperate climates, could influence the transmission of infections.

2

3

1 This seems especially interesting, since most models on tick-borne infections use
2 continuous time, thus neglecting developmental delays and assuming that newly
3 infected ticks are immediately capable of infecting new hosts.

4 The life cycle we assumed for ticks was particularly simple: larvae and nymphs
5 that feed during a season emerge as nymphs or adults at the beginning of the fol-
6 lowing seasons; on the other hand, out of the eggs produced by fed adults during a
7 season a proportion p of the larvae emerge during the same season, while the rest,
8 $1 - p$, emerge at the beginning of the following season. In reality, ticks' devel-
9 opment periods are strongly influenced by climatic factors (Randolph *et al.*, 2002)
10 so that often the interaction of the development period and the onset of winter is
11 indeed such that ticks will emerge in the new stage in the following spring, but
12 sometimes they will be able to emerge within the same growing season. All tick
13 stages, not only for larvae as assumed in the model, will spread their emergence
14 times among different months and years; moreover, the extent of this spread will
15 vary among years due to climatic fluctuations Gern and Humair (2002) and CEA
16 Report (2000), and will also depend on the time in which the ticks have fed. Our
17 choice of letting all nymphs and adults develop during winter has been made for the
18 sake of simplicity, so that we would be able to assess the effect of this time delay
19 on the dynamics of the infection. There would be no problem in adding many dif-
20 ferent paths within years, and from one year to the next. However, the analysis,
21 which was already rather cumbersome in our case, would probably become more
22 awkward. Finally, we must remark that we found it preferable to assume that the
23 development of at least one stage (in our case, larvae) was spread among years.
24 Otherwise, if $p = 1$, system (7) would decouple into three separate systems for
25 years $n - 2$, $n - 1$, and n .

26 As already discussed, the emergence time is spread over several weeks, and gen-
27 erally differs among stages. This could certainly be added to the present model,
28 yielding more realistic abundance curves of questing ticks, but without strongly
29 affecting the dynamics, we believe.

30 The dynamics of the model for the population of ticks appears to be generally
31 rather simple. If the density-dependent functions (moulting rates) are of compen-
32 satory types, for instance $m_z(x) = \frac{s_z}{1+\mu x}$, there exists a unique equilibrium which is
33 always asymptotically stable. Even when the density dependence is of the Ricker
34 type ($m_z(x) = e^{-\mu x}$), for most realistic parameter values there is a unique equi-
35 librium, and this is generally asymptotically stable, although for high values of
36 the parameter p (the probability of immediate development of larvae) and of the
37 feeding rates (which would occur at extremely high host population densities), a
38 Neimarck–Sacker bifurcation may occur, giving rise to oscillating tick population
39 densities (see Fig. 7). It must be remarked that destabilization of equilibrium tends
40 to occur with high values of p , and hence when delays are shorter (contrary to the
41 general view on the effect of delays) but when each cohort tends to reproduce sep-
42 arately over the generations; in fact, when $p = 1$ even- and odd-year cohorts are
43 completely decoupled.

This model of tick population dynamics provides a natural framework for introducing the dynamics of a tick-transmitted infections. The resulting model consists of three coupled difference equations whose variables are the densities of infected nymphs, of infected hosts, and of susceptible hosts at the beginning of each season. For reasonable parameter values, the density of infected hosts at the beginning of each season is negligible, and the density of susceptible hosts is not very far from their carrying capacity, so that the system can be approximated by a single difference equation. The function relating the density of infected ticks of one year to that of the previous one is, however, rather complex and cannot be written in an explicit form, so the system has been analysed mainly through computer simulation.

The most interesting result we found has been the dependence of the equilibrium density of infected ticks on host densities. A so-called ‘dilution effect’ of non-competent hosts had already been found in the analysis of a continuous-time model (Norman *et al.*, 1999). At low levels, an increase of non-competent hosts causes an increase of tick densities, and hence more possibilities of transmitting the infection; however, increasing further the densities of non-competent hosts may result to most bites of infected ticks being ‘wasted’ on hosts not capable of acquiring and transmitting the infection. This fact has been found in this model too and actually this ‘dilution effect’ causes extinction of the infection at much lower densities than had been found in continuous-time models for similar parameter values (Rosà *et al.*, 2003). In this case, pathogen extinction occurs at densities about 4 times the average densities of Trentino (Fig. 10) against 1000 times higher for the continuous-time model.

Unexpectedly, we found that the ‘dilution effect’ occurs in this model also for competent hosts (left part of Fig. 10); the explanation of this cannot be in bites being ‘wasted’ since these hosts would all contribute to infection transmission. Indeed, the explanation for this dilution effect can be found by looking at the expression (26) for the reproductive ratio. Simplifying the exact value, we may say that R_0 can be obtained by multiplying the probability that a newly infected larva infects (as a nymph) a susceptible host times the average number of larvae that bite that host during its infectious period.

The probability is equal to $m^L(\bar{L})q^N\beta_1^N H_1^* \psi^N / (d_N + g_N)$ which, when hosts of type 1 are very abundant, is approximately $m^L(\bar{L})q^N$, since then each larva will find very quickly a host of type 1.

On the other hand, the average number of larvae that bite a host is equal to $\bar{L}\beta_1^L \psi^L / (b + \gamma)$; when hosts of type 1 are very abundant, ψ^L goes to 0, while \bar{L} tends to a constant, so the number of infected larvae goes to 0, causing the ‘dilution effect’. In other words, when hosts are very abundant, each one will find few larvae around; hence, an infected host will not be able to spread the infection sufficiently. This model prediction depends on the fact that, even when the density of hosts of type 1 becomes infinitely large, the number of ticks remains limited, both because hosts of type 2 are needed for reproduction, and because the feeding rates g^z become at most equal to σ^z . From the graphs shown (Fig. 10), it can be

1 seen that this ‘dilution effect’ occurs at realistic population densities, not only at
2 ‘infinitely large’ densities.

3 Several studies have been performed on the dilution effect that non-competent
4 species may produce; especially for Lyme disease in North America, the value
5 of mammal biodiversity in decreasing infection prevalence of ticks has been sug-
6 gested by LoGiudice *et al.* (2003). As far as we know, nobody had suggested that
7 also a high density of competent hosts might decrease Lyme disease prevalence;
8 since the mice densities theoretically required to cause this decrease are rather high,
9 probably experimental work would be needed to test whether this effect is real.

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16 REFERENCES

- 17 Caraco, T., G. Gardner and B. K. Szymanski (1998). Lyme disease: self regulation and
18 pathogen invasion. *J. Theor. Biol.* **193**, 561–575.
- 19 CEA Report (2000). *Zecche e malattie trasmesse. La situazione in Trentino*. Centro di
20 Ecologia Alpina, Report No. 21, Trento.
- 21 Gern, L. and P. F. Humair (2002). Ecology of *Borrelia burgdorferi sensu lato* in Europe,
22 in *Lyme Borreliosis: Biology, Epidemiology and Control*, S. Gray, O. Kahl, R. S. Lane
23 and G. Stanek (Eds), CABI-Publishing, pp. 47–90.
- 24 Hudson, P. J., A. Rizzoli, B. T. Grenfell, H. Heesterbeek and A. P. Dobson (2002). *Ecology*
25 *of Wildlife Diseases*, Oxford University Press.
- 26 Humair, P. F., O. Rais and L. Gern (1999). Transmission of *Borrelia Ixodes* from *Apodemus*
27 *mice* and *Clethrionomys voles* to *Ixodes ricinus* ticks: differential transmission pattern
28 and overwintering maintenance. *Parasitology* **118**, 38–42.
- 29 Jones, L. D., C. R. Davies, G. M. Steele and P. A. Nuttall (1987). A novel mode of arbovirus
30 transmission involving a nonviraemic host. *Science* **237**, 775–777.
- 31 LoGiudice, K., R. S. Ostfeld, K. A. Schmidt and F. Keesing (2003). The ecology of infec-
32 tious diseases: effects of host diversity and community composition on Lyme disease
33 risk. *Proc. Natl. Acad. Sci. USA* **100**, 567–571.
- 34 Mwambi, H. G., J. Baumgartner and K. P. Haderler (2000). Ticks and tick-borne diseases: a
35 vector–host interaction model for the brown ear tick. *Stat. Meth. Med. Res.* **9**, 279–301.
- 36 Norman, R., R. G. Bowers, M. Begon and P. J. Hudson (1999). Persistence of tick-borne
37 virus in the presence of multiple host species: tick reservoirs and parasite mediated
competition. *J. Theor. Biol.* **200**, 111–118.

- O'Callaghan, C. J, G. F. Medley, T. F. Peter and B. D. Perry (1998). Investigating the epidemiology of heartwater by means of a transmission dynamics model. *Parasitology* **117**, 49–61. 1
- Randolph, S. E., R. M. Green, A. N. Hoodless and M. F. Peacey (2002). An empirical quantitative framework for the seasonal population dynamics of the tick *Ixodes ricinus*. *Int. J. Parasitology* **32**, 979–989. 2
- Randolph, S. E. and D. J. Rogers (1997). A generic population model for the African tick *Rhipicephalus appendiculatus*. *Parasitology* **115**, 265–279. 3
- Rosà, R., A. Pugliese, R. Norman and P. J. Hudson (2003). Basic reproduction number in models for tick-borne infections including non-viraemic transmission, extended feeding and tick aggregation. *J. Theor. Biol.* **224**, 359–376. 4
- Rosà, R., A. P. Rizzoli, A. Pugliese and P. J. Hudson (2004). The role of host biodiversity, density and transmission routes in generating non-linearities in tick borne infections (submitted). 5
- Sandberg, S. and T. E. Awerbuch (1992). A comprehensive multiple matrix model representing the life cycle of the tick that transmits the agent of Lyme disease. *J. Theor. Biol.* **157**, 203–220. 6
- Sonenshine, D. E. (1991). *Biology of Ticks*, Vol. 1, Oxford University Press. 7

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