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Impact of life stage-dependent dispersal on the colonization dynamics of host patches by ticks and tick-borne infectious agents

Sarah Kada^{1*} , Karen D. McCoy² and Thierry Boulinier¹

Abstract

Background: When colonization and gene flow depend on host-mediated dispersal, a key factor affecting vector dispersal potential is the time spent on the host for the blood meal, a characteristic that can vary strongly among life history stages. Using a 2-patch vector-pathogen population model and seabird ticks as biological examples, we explore how vector colonization rates and the spread of infectious agents may be shaped by life stage-dependent dispersal. We contrast hard (Ixodidae) and soft (Argasidae) tick systems, which differ strongly in blood-feeding traits.

Results: We find that vector life history characteristics (i.e. length of blood meal) and demographic constraints (Allee effects) condition the colonization potential of ticks; hard ticks, which take a single, long blood meal per life stage, should have much higher colonization rates than soft ticks, which take repeated short meals. Moreover, this dispersal potential has direct consequences for the spread of vector-borne infectious agents, in particular when transmission is transovarial.

Conclusions: These results have clear implications for predicting the dynamics of vector and disease spread in the context of large-scale environmental change. The findings highlight the need to include life-stage dispersal in models that aim to predict species and disease distributions, and provide testable predictions related to the population genetic structure of vectors and pathogens along expansion fronts.

Keywords: Allee effect, *Borrelia burgdorferi*, Climate change, *Ixodes uriae*, Lyme disease, *Ornithodoros maritimus*, Parasite spread, Range expansion, Vertical transmission

Background

Vector-borne disease emergence depends on the presence or spread of both a suitable vector and the associated infectious agent [1–7]. A growing number of models are used to describe temporal change in disease distributions [8] and to establish predictions on the risk of disease spread under climate change scenarios [9–15]. While climate is a key driver of vector range expansion because of its effects on habitat suitability [16–22], population structure and dispersal processes are also expected to play crucial roles in the speed of species' spread [23]. Parasite dispersal is also an important driver of host-parasite coevolution [24], affecting local adaptation [25, 26] and host and parasite genetic structure (e.g.

Blouin et al. [27]; McCoy et al. [28]). The dynamics of dispersal, its speed and frequency, are particularly relevant for assessing vector expansion and the associated risk of spread of vector-borne infectious agents [12, 27, 29–31], but the key potential role of the dispersal stage is rarely considered in vector-borne disease models. Heterogeneity in dispersal rates among vector subpopulations is known to affect population genetic structure [28, 32, 33] and the geographical distribution of vector-borne infectious agents [33, 34]. For example, vector stage structure is thought to influence the speed and distance of vector colonization in ticks responsible for Lyme borreliosis [22, 35–37]. Dispersal distance in these ectoparasites may vary because of stage-specific host feeding preferences (e.g. nymphs feeding on birds versus small mammals; Wilson et al. [38]; Norte et al. [39]). Likewise, some stages might be more successful than others at (i) establishing a local population; and (ii) contributing to

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the spread of vector-borne infectious agents. For instance, releasing adults capable of breeding right away is considered to be more efficient than releasing non-reproductive juveniles in reintroduction programs [40, 41]. Also, density-dependent factors, notably Allee effects [30, 42, 43], can be related to difficulties for some stages to locate a mate or survive at low population densities [43, 44]. Allee effects are especially significant when studying the dynamics of population range expansion [45–47] because, by definition, the size of a newly established population is low. In addition, the maintenance of the infectious state across vector life stages (i.e. transstadial transmission, Randolph [48]; Hasle [21]) and vertical transmission to offspring (i.e. transmission from an infected adult female vector to its eggs, Rollend et al. [49]) will play critical roles in the emergence of vector-borne infectious agents [21, 48, 50, 51], and may interact with stage-dependent vector dispersal. Considering the impact of stage-dependent dispersal using a clear conceptual framework could therefore be particularly useful in order to gain a better understanding of the sources of eco-epidemiological variability in the context of vector and pathogen population expansions.

The ecological peculiarities of hard and soft ticks, i.e. the two main tick families, offer a unique opportunity to understand how stage-dependent dispersal strategies affect the process of tick colonization and the spread of tick-borne infectious agents. Tick dispersal is typically mediated by tick attachment on a vertebrate host for the blood meal [52, 53], and the two families have evolved contrasting feeding strategies [48, 54–56]. All hard tick life stages tend to have a long attachment duration (several days) on the hosts. Longer blood meal duration is generally associated with higher chances that the host moves to a new location, thereby increasing tick dispersal opportunities [57]. In contrast, soft ticks (particularly nymphs and adults) generally take several very short meals (15 to 30 min), mostly at night when the host is resting [54, 56, 58–60]. Dispersal opportunities for soft ticks may thus be more associated with the larval blood meal, as this meal may last several hours to days [61, 62]. These differences may have an effect on the relative capacities of hard and soft ticks (and their pathogens) to disperse and colonize new habitats [54, 63–65], and can be particularly important to consider in the current context of tick range expansions [6, 12, 66].

Here, we explore how differential stage-dependent dispersal of tick vectors, in interaction with demographic effects, conditions the colonization of suitable habitats and the spread of associated microparasites. We use a 2-patch modeling approach of a vector population structured in three explicit life stages (larva, nymph and adult), where one vector population may colonize the empty patch. We base our model on populations of soft

and hard ticks that differ ecologically in their dispersal stage, and consider seabird-tick systems as case studies. Our hypothesis is that species for which only larval stages disperse, as is the case for soft ticks, will experience a time lag in population growth on a colonized patch because of (i) the time required to reach reproductive maturity, and (ii) potential Allee effects due to low dispersal rates. We expect these differences to affect the probability of successful vector colonization, but also the spread of vector-borne infectious agents among host patches. To explore this second aspect, we develop a Susceptible-Infected (SI, i.e. with no recovery) model of vector-borne infection for the vector population and SIR (Susceptible-Infected-Recovered) model for the vertebrate host, encompassing the relative contribution of stage-dependent vector dispersal. This allows us to evaluate the relative risks of spatial spread for microorganisms that use hard versus soft ticks as vectors.

Why model seabird-tick systems?

Seabirds breed in discrete colonies that are highly structured in space (Furness & Monaghan [67]), providing dense and reliable sources of food and habitat for nest-dwelling ectoparasites like ticks [65]. These systems are particularly suitable for addressing questions related to tick dispersal because they involve a limited number of host species, notably compared to forest systems [12], and all hosts share common life history traits. Although only a limited number of tick species exploit seabirds, these ticks are present in most colonies and include species of the two main tick families, the Ixodidae and the Argasidae [65]. In seabird-tick systems, tick dispersal is passive, conditioned by host movements between breeding colonies during the breeding season [68–70] and the characteristics of the blood meal taken on the host. Dramatic differences exist between hard and soft ticks in terms of the length of the blood meal as outlined above. Finally, seabird colonies and their associated ticks are distributed at broad geographical scales [65, 71, 72], and seabird ticks are vectors of a broad range of viruses and bacteria [65], which make them especially interesting to study in the current context of climate change. Indeed, recent studies have revealed a northward expansion in the distribution of ixodid ticks [6, 66, 73]. Here, we consider two widespread seabird ticks, the hard tick *Ixodes uriae* which is found in the polar areas of both hemispheres, and the soft tick complex *Ornithodoros* (*Carios capensis*) (*sensu lato*) which lives in seabird colonies across the equatorial region [65]. As mean global temperatures increase, we may expect soft and hard tick populations to expand towards higher latitudes, where host populations exist for both species (see Fig. 1).

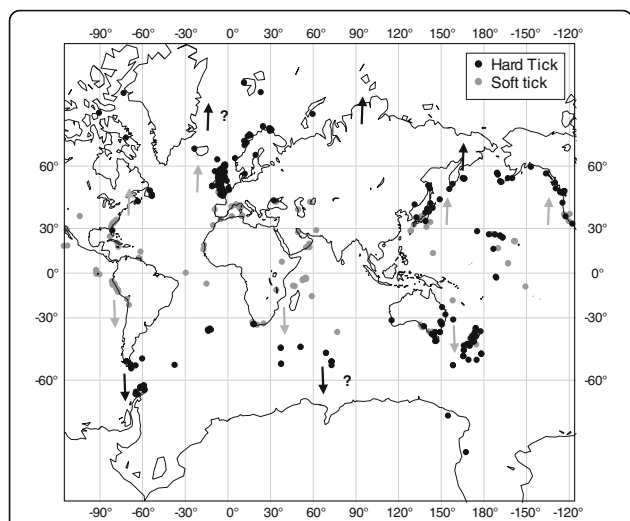


Fig. 1 The known worldwide distribution of hard (black dots) and soft (grey dots) ticks of seabirds. Only ticks from the Ixodes and Ornithodoros (Carios) genera are represented. Arrows are illustrative and represent the potential colonization areas for hard (black arrow) and soft (grey arrow) seabird ticks, respectively. Data from Dietrich et al. [65]

Methods

Population dynamics

In order to explore the potential effect of life-stage-dependent dispersal on vector colonization dynamics, we build a continuous-time model of vector population dynamics, structured in 3 life stages: larvae (L), nymphs (Np) and adults (A), on two patches (i and j). σ_L and σ_N are the maturation rates, i.e. the respective rates at which larvae moult to nymphs and nymphs to adults, provided they completed their blood meal. μ_L , μ_N and μ_A are the respective natural mortality rates of larvae, nymphs and adults. The total population size is $N_v = L + Np + A$, and m^{ij} and m^{ji} represent dispersal rates between the two patches, which can differ among larvae, nymphs and adults (see below). The per capita birth rate of ticks is b_v , and is scaled to a sex ratio of 0.5 and depends on the survival rate of eggs reaching the larval stage. The term $f(N_v^i)$ represents the density-dependent growth of the total vector population in patch i and is dependent on the carrying capacity, K_v (density dependent constraint on tick population size assumed to be caused by the local availability of off-host shelters and the limit of tick density on a given host). We use the following expression:

$$f(N_v^i) = \left(1 - \frac{N_v^i}{K_v}\right).$$

The model is written:

$$\begin{aligned} \frac{dL^i}{dt} &= \underbrace{b_v A^i f(N_v^i)}_{\text{birth}} - \underbrace{(\mu_L + \sigma_L)L^i}_{\text{mortality + maturation}} + \underbrace{m_L^{ji}}_{\text{migration in}} - \underbrace{m_L^{ij}}_{\text{migration out}}, \\ \frac{dNp^i}{dt} &= \underbrace{\sigma_L L^i}_{\text{maturation}} - \underbrace{(\mu_{Np} + \sigma_{Np})Np^i}_{\text{mortality + maturation}} + \underbrace{m_{Np}^{ji}}_{\text{migration in}} - \underbrace{m_{Np}^{ij}}_{\text{migration out}}, \\ \frac{dA^i}{dt} &= \underbrace{\sigma_N Np^i}_{\text{maturation}} - \underbrace{\mu_A A^i}_{\text{mortality}} + \underbrace{m_A^{ji}}_{\text{migration in}} - \underbrace{m_A^{ij}}_{\text{migration out}}. \end{aligned}$$

$N_v^i = \text{Tick population size in patch } i.$

For the sake of simplicity, and because we are mainly interested in the effect of stage-dependent dispersal, we consider all rates as continuous, even though vector activity follows a seasonal pattern under natural conditions [9, 74].

Tick dispersal occurs once the vector population reaches its equilibrium in the source patch. For the soft ticks, we consider that the blood meal is long enough for dispersal to occur only at the larval stage. Our assumption is that the propensity for dispersal is constrained by blood meal duration, therefore nymph and adult soft ticks, which take several short blood meals have no dispersal potential ($m_{Np} = m_A = 0$). For the hard ticks, the blood meal duration lasts several days across all stages. All stages are therefore considered to have the same probability of dispersing, such that we set equivalent dispersal rates: $m_L = m_{Np} = m_A$. Assuming a sex ratio of 0.5, only half of the adults have the opportunity to disperse, as only females take a blood meal after mating off-host with a male (Eveleigh & Threfall [75]). When comparing soft and hard tick dispersal towards an empty patch, we always let the same total number of individuals disperse. We use this approach because even though only larvae can disperse for soft ticks, these larvae can exploit the host throughout the reproductive season, whereas the different life stages of hard ticks tend to have specific, partially non-overlapping periods of host exploitation [76]. Moreover, we consider that this assumption results in a conservative comparison of the life-cycles.

Allee effects on reproduction

Dispersing individuals at the edge of the distributional range can benefit from the low density of conspecifics because of a release from competition (positive density-dependent effect), but this low density may reduce population growth rate notably due to constraints on the probability of locating a mate (negative density-dependent effect, also known as Allee effect). Tick dispersal, which is achieved by host transportation, ensures arrival in a patch where hosts are available, but not necessarily where conspecifics are present at a high density. This can induce an Allee effect and result in a local decrease of the per capita birth rate, which could accordingly affect colonization

success or time to vector establishment. To consider the impact of an Allee effect, the reproductive function was set to be dependent on the number of local adults. This constraint takes the following form:

$$f(N_v^i) = \underbrace{\left(1 - \frac{N_v^i}{K_v}\right)}_{\text{density-dependence}} \underbrace{\left(\frac{A_i}{A_i + a}\right)}_{\text{Allee effect component on reproduction}},$$

where a is the strength of the Allee effect. An increase of a results in a decrease in local reproduction due to an increasing difficulty in locating a mate in the adult stage (see Additional file 1: Figure S1).

Infection dynamics

To explore the potential effect of life-stage dependent dispersal on the spread of vector-borne infectious agents, we then added the epidemiological dynamics of an infectious agent to our vector population model [77]. The vector population was set to follow a susceptible-infected (SI) model (see Fig. 2): larvae (L), nymphs (Np) and adults (A) are either susceptible (S) or infectious (I). We assume vectors do not recover from infection (i.e. no R compartment) and that its vital rates (birth, death and maturation) are unaltered (see Fig. 2). We also introduce infectious disease dynamics for the vertebrate host (SIR), as the infection is transmitted from an infected vector to a susceptible host (H_S^i) and from an infected host (H_I^i) to a susceptible vector. The 2-patch model describing the

epidemiological dynamics in the vector population, for $i = 1, 2$ is thus as follows:

$$\begin{aligned} \frac{dL_S^i}{dt} &= \underbrace{b_v(A_S^i + (1-\theta)A_I^i)f(N_v^i)}_{\text{birth}} - \underbrace{\lambda_v^i L_S^i}_{\text{disease transmission}} - \underbrace{(\mu_L + \sigma_L)L_S^i}_{\text{mortality + maturation}} + \underbrace{M_L^i}_{\text{migration}}, \\ \frac{dL_I^i}{dt} &= \underbrace{\lambda_v^i L_S^i}_{\text{disease transmission}} + \underbrace{b_v \theta (A_I^i)f(N_v^i)}_{\text{vertical transmission}} - \underbrace{(\mu_L + \sigma_L)L_S^i}_{\text{mortality + maturation}} + \underbrace{M_L^i}_{\text{migration}}, \\ \frac{dNp_S^i}{dt} &= \underbrace{-\lambda_v^i Np_S^i}_{\text{disease transmission}} + \underbrace{\sigma_L L_S^i}_{\text{maturation}} - \underbrace{(\mu_N + \sigma_N)Np_S^i}_{\text{mortality + maturation}} + \underbrace{M_N^i}_{\text{migration}}, \\ \frac{dNp_I^i}{dt} &= \underbrace{\lambda_v^i Np_S^i}_{\text{disease transmission}} + \underbrace{\sigma_L L_I^i}_{\text{maturation}} - \underbrace{(\mu_N + \sigma_N)Np_I^i}_{\text{mortality + maturation}} + \underbrace{M_N^i}_{\text{migration}}, \\ \frac{dA_S^i}{dt} &= \underbrace{-\lambda_v^i A_S^i}_{\text{disease transmission}} + \underbrace{\sigma_N Np_S^i}_{\text{maturation}} - \underbrace{\mu_A A_S^i}_{\text{mortality}} + \underbrace{M_A^i}_{\text{migration}}, \\ \frac{dA_I^i}{dt} &= \underbrace{\lambda_v^i A_S^i}_{\text{disease transmission}} + \underbrace{\sigma_N Np_I^i}_{\text{maturation}} - \underbrace{\mu_A A_I^i}_{\text{mortality}} + \underbrace{M_A^i}_{\text{migration}}. \end{aligned}$$

And for the vertebrate host:

$$\begin{aligned} \frac{dH_S^i}{dt} &= \underbrace{b_h N_h^i \left(1 - \frac{N_h^i}{K_h}\right)}_{\text{birth}} - \underbrace{\lambda_h^i H_S^i}_{\text{disease transmission}} - \underbrace{\mu_h H_S^i}_{\text{mortality}}, \\ \frac{dH_I^i}{dt} &= \underbrace{\lambda_h^i H_S^i}_{\text{disease transmission}} - \underbrace{(\mu_h + \gamma_h) H_I^i}_{\text{mortality + recovery}}, \\ \frac{dH_R^i}{dt} &= \underbrace{\gamma_h H_I^i}_{\text{recovery}} + \underbrace{\mu_h H_R^i}_{\text{mortality}}. \end{aligned}$$

The host dynamics follow a classical SIR model, and the host density is determined by the local carrying capacity (K_h). Transmission is frequency-dependent [78] and consequently depends on the proportion of infect hosts (H_I) and vectors (L_I, Np_I, A_I) in the populations. Transmission efficiency from host to vector and from vector to host is considered to be the same ($\beta_{hv} = \beta_{vh}$), and vector competence is equivalent among life stages. Hosts can recover from the infection at rate γ_h . Infections in the vectors (λ_v^i) and the hosts (λ_h^i) are produced at rates:

$$\lambda_v^i = \beta_{hv} c \frac{H_I^i}{N_h^i}, \quad \text{and} \quad \lambda_h^i = \beta_{vh} c \frac{(L_I^i + Np_I^i + A_I^i)}{N_v^i}.$$

Vertical transmission of the infection can occur between an infected adult female and a larva at rate θ . We do not consider direct, non-systemic transmission for this model (i.e. tick to tick transmission through co-feeding). Because survival between molts is high for tick-borne pathogens (e.g. *Borrelia burgdorferi* (s.l.), Bellet-Edimo et al. [79], Mitzel et al. [80]), transmission occurs transstadially, meaning that infected larvae become infected nymphs. As ticks must take one blood meal per life stage, we assume one transmission event can occur per developmental stage. This is translated by a vector-host contact rate, c , set to 1 (e.g. Cobbold et al. [81]). By later varying c (see Additional file 2: Table S1), we

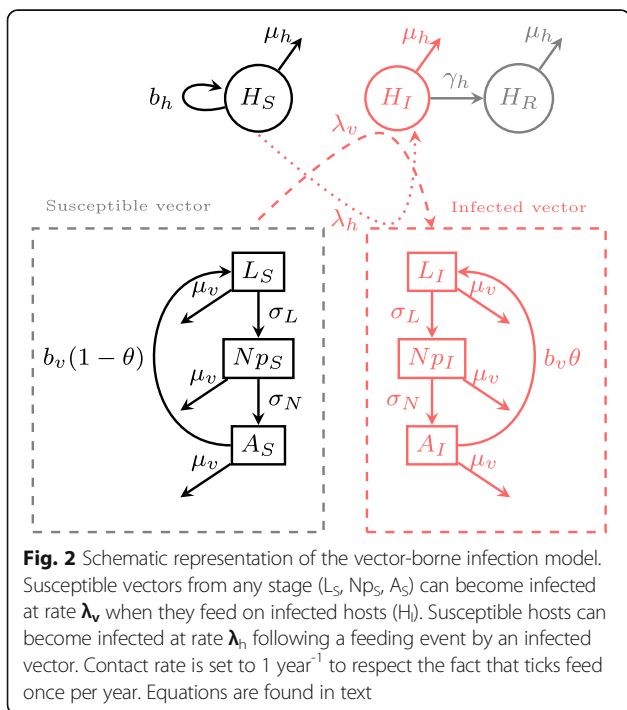


Fig. 2 Schematic representation of the vector-borne infection model. Susceptible vectors from any stage (L_S, Np_S, A_S) can become infected at rate λ_v when they feed on infected hosts (H_I). Susceptible hosts can become infected at rate λ_h following a feeding event by an infected vector. Contact rate is set to 1 year⁻¹ to respect the fact that ticks feed once per year. Equations are found in text

investigate the impact of considering the multiple short blood meals that soft ticks take on the spread of infections. Although repeated blood meals at nymphal and adult stages could affect transmission among hosts within a patch, this will not lead to higher tick dispersal because, as discussed above, these blood meals are extremely short and taken at night.

For the sake of simplicity, the vector and vertebrate host populations are assumed to have reached their respective equilibrium in both patches (see Caraco et al. [35]). Additionally, we consider that the infection has reached its endemic equilibrium in the source patch before dispersal is introduced (in practical terms, $t > 100$ years). We then allow for dispersal between the two patches and investigate the effect of stage-dependent dispersal on the spread of infection. The term for dispersal, similar to that of Moore et al. [82], is as follows,

$$M_Y^i = - \underbrace{\sum_{i=1}^n m^{ij} V_Y^i}_{\text{migration out}} + \underbrace{\sum_{i=1}^n m^{ji} V_Y^i}_{\text{migration in}}$$

In this case, m^{ij} represents the migration rate of the vector (V) from patch i to patch j . A vector can belong to the susceptible or infected class ($Y \in \{S, I\}$). Note that throughout the analyses, soft ticks disperse only at the larval stage whereas hard ticks are able to disperse at any stage. However, only half of the hard tick adults are able to disperse, as only females take a blood meal on the host. For the sake of simplicity, we only consider vector dispersal for the spread of the infectious agent, and not vertebrate host dispersal, because we are specifically interested in the contribution of the vector to the spread of vector-borne diseases.

To study the combined effects of stage-dependent dispersal and vertical transmission efficiency, we introduce a parameter for vertical (or transovarial) transmission, θ , a significant route of transmission for certain vector-borne infections [79, 83]. When $\theta = 0$, there is no vertical transmission and all larvae are born susceptible. If $\theta > 0$, then a proportion of eggs from an infected mother are born infected.

Sensitivity analysis

In order to explore the robustness of results and their sensitivity to particular parameters, we performed a global sensitivity analysis [84]. The Latin Hypercube Sampling (LHS) method, which enables one to explore a wide range of parameter combinations, was performed using R 3.0.2 (*pse* package). Model outputs were obtained using Matlab 2010b for a range of parameter conditions (see Additional file 2: Table S1). The number of simulation runs ($N = 150$) respects the $N > (4/3) K$ ratio for a sufficient level of relevance, N being the number of

bootstrap replicates and K the number of unknown parameters. We let the population reach an equilibrium in the first patch (100 years) before allowing individuals to disperse to the second patch, and let the population and epidemiological models run for respectively 30 and 20 years, using the sampled parameter values. Model outputs were then analyzed using the Partial Rank Correlation Coefficient method (PRCC, R package *pse*, [85]) to determine which parameters had a large influence on colonization dynamics and the spread of vector-borne infections.

Results

Population dynamics and Allee effects

For a broad set of reasonable parameter values (see Table 1), soft tick population growth in the newly colonized patch is much slower compared to the invading population of hard ticks (Fig. 3a, b). This can induce a time lag to establishment of a local population (i.e. time at which a viable number of individuals in the population is reached) of a local population which reflects a strong difference between the two tick types in the shape of population growth in the colonized patch. Soft ticks experience a delay in the local production of offspring, thus in local population growth, because of the required developmental time and associated likelihood for larvae to reach adulthood (Fig. 3b). The population dynamics observed at the early stage of invasion by soft ticks highlights the essential role of immigrants from the source patch (i.e. *propagule rain*, Gotelli [86]) in initiating local reproduction. For the sake of simplicity, our model assumed the same number of dispersing individuals for larvae of soft ticks. Although potentially justified based on the phenological differences in host exploitation between soft and hard ticks (see above), this advantage may be overestimated and soft ticks may experience even lower rates of colonization than suggested in our model.

Including an Allee effect on vector populations in our model created an even greater disparity between hard and soft tick colonization dynamics. When population size is low, the Allee effects (affecting local reproduction) limit the population growth for only a very short period for the hard tick (Fig. 4a), but lasts over a much longer period for the soft tick (Fig. 4b). Here again, this is easily understood because the dispersal of only juvenile tick life stages delays the local buildup of a breeding population. As in the case with no Allee effect, the soft tick population growth exhibits a different shape than the expected logistic curve we find for the hard tick population. We see a clear point of inflexion (I_2) at the beginning of the soft tick population growth curve, after an initial concave period (Fig. 4b). This concavity again reflects the propagule rain in the

Table 1 Parameters used for the population dynamics and epidemiological models

Parameter	Description	Value (year ⁻¹)
Vector		
b_V	Number of eggs per adult female tick surviving to reach larval stage	200
K_V	Carrying capacity for vector	1e3
μ_L	Mortality rate per tick larva	0.95
μ_N	Mortality rate per tick nymph	0.3
μ_A	Mortality rate per adult tick	0.4
σ_L	Maturation rate from larvae to nymph	0.3
σ_N	Maturation rate from nymph to adult	0.3
m_{ij}	Migration rate	variable (from 0.01 to 0.02)
a	Strength of the Allee effect	variable (from 1 to 40)
Vertebrate host		
b_h	Vertebrate host birth rate	1.4 per pair
K_h	Carrying capacity for the vertebrate host	3e2
μ_h	Vertebrate host mortality rate	0.12
Epidemiological parameters		
β_{hv}	Transmission from tick to vertebrate host	0.8
β_{vh}	Transmission from vertebrate host to tick	0.5
γ_h	Average duration of infection	0.33
θ	Vertical transmission from adult tick to eggs	variable (from 0.001 to 0.1)
c	Yearly contact rate (number of yearly bloodmeal)	variable (from 1 to 1.2)

absence of local reproduction. Once a threshold number of mature adults is attained, the population growth then follows a logistic curve.

Infection dynamics

In this model, an epidemiological component is integrated into the previous tick dispersal model. We find that infection prevalence in the infection-free patch

increases faster in the hard tick population than in the soft tick population (Fig. 5). Dispersing nymphs and adults are associated with a greater transmission risk compared to dispersing larvae because of their higher survival rate. Higher prevalence is found when vertical transmission is high (see Fig. 5, dashed line), and increases significantly when hard ticks disperse because the arrival of adult ticks directly translates into a higher number of infected individuals. The repeated blood

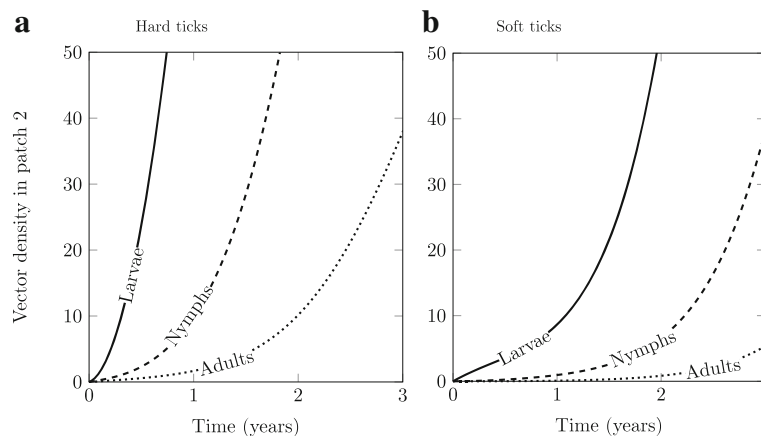


Fig. 3 Density of **a** hard and **b** soft ticks in each life stage in the receiving patch. Default parameter values are found in Table 1. $m_L = m_N = m_A = 0.01 \text{ year}^{-1}$ for hard ticks, and the migration rate for soft ticks is adjusted so that an equivalent number of larval ticks disperse

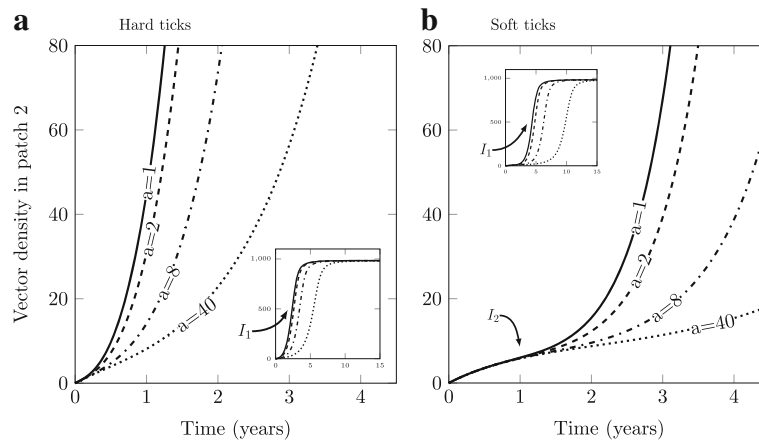


Fig. 4 Strength of the Allee effect and total vector population density in the receiving patch for **a** hard and **b** soft ticks ($a = 1, 2, 8$ and 40 ; higher values indicate a stronger Allee effect). The arrows are for illustrative purposes. They represent the inflexion points for the short (I_2) and long-term (I_1) dynamics. On the right panel (**b**), the inflexion point (I_1), represents the threshold above which the population growth becomes positive. Default parameter values are found in Table 1. $m_L = 0.017 \text{ year}^{-1}$ for the soft and $m_L = m_N = m_A = 0.01 \text{ year}^{-1}$ for hard ticks

meals in the soft tick life-cycle also result in higher infection prevalence due to increased contact rates with the host (Additional file 2: Figure S2), but never equals diffusion rates in hard ticks. Because soft ticks are nidicolous, repeated blood meals may be taken on the same host individual such that the potential increase in

infection prevalence due to this life history trait may be overestimated by a model not accounting for local spatial structure.

Sensitivity analysis

The results of the LHS-PRCC sensitivity analysis indicate that vector birth rate (b_v), carrying capacity (K) and nymphal maturation rate (σ_N) have a strong positive impact on vector population density on patch 2. Nymphal and adult dispersal rates also have a positive relationship with vector population density, while larval dispersal, the strength of the Allee effect and mortality rates of all tick stages negatively interact with it (Fig. 6a). As expected for the epidemiological model, the PRCC index showed a strong positive relationship of transmission rates (systemic, β_{vh} , β_{hv} and vertical, θ transmission), contact rate between hosts and vector (c) on the infection prevalence in patch 2, while the vertebrate host recovery rate (γ_{vh}) negatively influences infection prevalence (Fig. 6b). Dispersal rates do not strongly affect the prevalence of infection, but interestingly we remark that nymphal and adult dispersal rates positively influenced infection prevalence, while larval dispersal negatively influenced it.

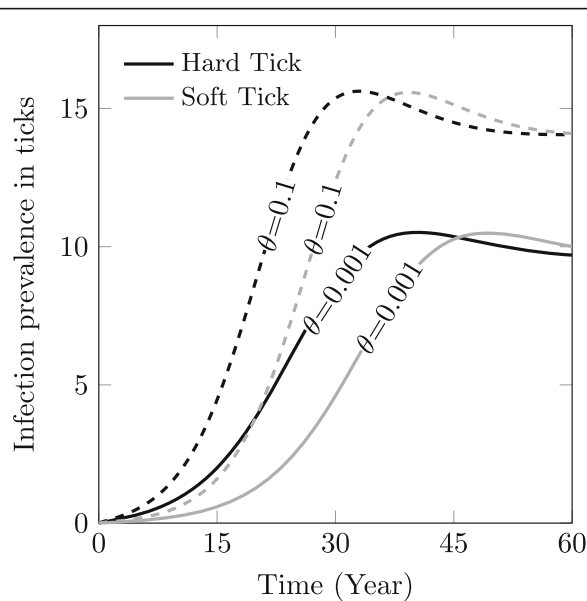


Fig. 5 Infection spread in the new patch through stage-dependent and bidirectional dispersal prevalence for low (solid lines, $\theta = 0.001$) and high vertical (transovarial) transmission rates ($\theta = 0.1$, dashed lines), following arrival of susceptible and infected ticks from patch 1. Default parameters are found in Table 1. Only 1 bloodmeal per stage ($c = 1$) is considered. The polyphagous nature of soft ticks ($c > 1$) partially compensates for the lag in local density, but the infection prevalence in soft ticks, still does not reach that of hard ticks (see Additional file 2: Figure S2, for details)

Discussion

Population dynamics

Parasite dispersal is a key component of host-parasite ecology and coevolution [24]. One of the key challenges in the eco-epidemiology of vector-borne diseases is to understand the dynamics that may explain vector species distributions at a hierarchy of temporal scales. While climate change has an inevitable impact on the establishment of vector species and the spread of vector-borne diseases at different spatial scales [87], the

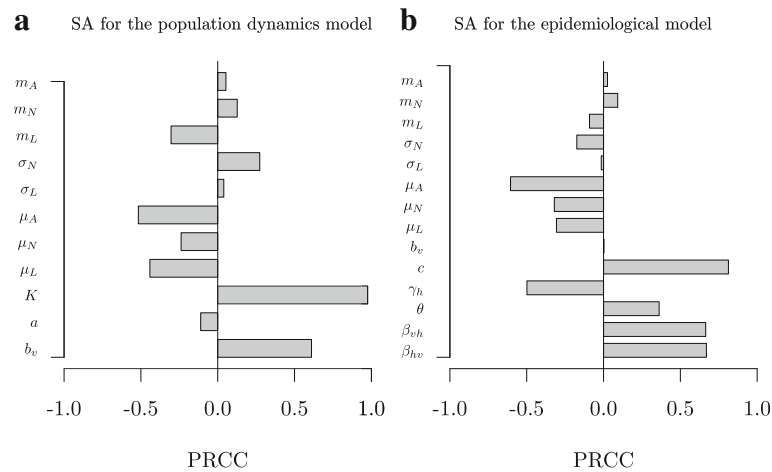


Fig. 6 Results of the sensitivity analysis (SA) for the vector population size in the newly colonized patch after 30 years (**a**) and the prevalence of the infection in this patch after 30 years (**b**), using the PRCC index which indicates the quantitative impact and direction of the relationship between a parameter and the model output. The SA is performed for 150 runs of the LHS matrix. Parameter used for the models are found in Additional file 2: Table S1

processes governing dispersal capacity have been largely overlooked in the case of vector species with complex life-cycles, such as ticks (but see Auld & Tinsley [88]). Vector populations are a striking example of stage-structured populations where life-stage peculiarities may feedback on ecological and epidemiological dynamics [30, 35, 89], and might also impact the genetic structure of vector species and their infectious agents. Our results show that stage-dependent dispersal, in association with maturation time and the survival probabilities of immature stages, is likely to constrain strongly the time-to-establishment of soft tick populations. The differences in population growth and establishment success we observe for hard and soft ticks are produced by the contrasted dispersal opportunities and their demographic effects. The general implications of our results suggest that inclusion of differential dispersal opportunities among life-stages could help predict the expansion of vector species more accurately. Specifically, we may expect strong differences in the speed of colonization for soft and hard ticks at comparable spatial scales. While we did not explicitly allow for dispersal rates to depend on blood meal lengths, it is likely tightly linked to dispersal opportunity. For instance, Heylen & Matthysen [57] suggested that the contrasted feeding duration and detachment timing (day versus night) between two ixodid ticks feeding on the same songbird lead to strong differences in their respective dispersal capacities. Further empirical work is now needed to understand how blood meal length and detachment strategies influence tick dispersal potential.

We also found that density-dependent mechanisms (Allee effects, notably related to the difficulty in finding

mates at low density) are likely to interact with stage-dependent dispersal to strongly affect colonization dynamics. This effect is particularly strong for soft ticks, for which we observed strongly delayed population growth at the early stages of invasion compared to hard ticks. Establishment success typically depends on the local immigration of particular life-stages, notably adults, due to their rapid contribution to local reproduction.

Infection dynamics

Predicting the speed of vector-borne disease spread depends on our ability to understand eco-epidemiological peculiarities of the system. Our model shows that stage-dependent dispersal and particular epidemiological parameters can play critical roles in the spread of vector-borne infections. Given the importance of stage structure for vector-borne disease dynamics in relation to host use and tick stage susceptibility [90], a better understanding such eco-evolutionary processes should result in a better appraisal of the conditions favoring the spread of vector-borne infections (e.g. Pelosse et al. [30]). We showed here, using a simple model, that stage at dispersal and vertical transmission play an important role on the spread of vector-borne infections.

Although our results show that hard tick vectors should spread infectious agents more rapidly than soft ticks, the detailed biological cycle of soft ticks may temper our views regarding their ability to spread vector-borne infections. Soft ticks have many nymphal instars, each typically requiring at least one blood meal, and adult ticks feed numerous times. This repeated contact with the host can potentially increase the transmission of vector-borne infectious agents, and can lead to a

faster increase in infection prevalence (see Additional file 2: Figure S2). However, the spread of infection in soft tick systems and our ability to compare predictions to other vector types will depend on the specific ecology of the tick species and whether or not it feeds on different host individuals at each blood meal. Indeed, the nidicolous lifestyle of many soft ticks may limit the transmission of infectious agents because ticks are restricted to hosts found in the same nest, or burrow. As mentioned in the model description, it is unlikely that the multiple blood meals per stage of soft ticks will increase their dispersal capacity (and thus colonization rate), because these meals are of very short duration (few minutes) and tend to be taken while the host is sedentary. The spatial restriction in the distribution of infectious agents found in examined soft tick species to date is consistent with these assumptions and model predictions. For instance, Arnal et al. [91] screened 19 Yellow-legged gull (*Larus michahellis*) colonies in the Mediterranean Sea for the local circulation of a Meaban-like virus, both by testing for specific antibodies in egg yolks, and by directly detecting viral RNA in the soft tick vector, *Ornithodoros maritimus*. They observed the presence of the tick in many of the colonies, but over the 3-year observation period only found one colony positive for the presence of the virus. They explained this restricted distribution by the high fidelity of gulls to their nesting sites combined with the limited dispersal abilities of the soft tick. A recent study found a similar restricted pattern of presence of the Meaban-like virus in other host species, further supporting the hypothesis of a limited dispersal capacity of soft ticks [92]. In this case, and more generally, we can expect tick dispersal characteristics to affect the timing of tick-borne infection establishment, and thus this trait needs to be incorporated directly into predictive models of disease spread. This should notably be important for species distribution models that may be proposed to predict range shifts in hosts and parasites, and their potential consequences for biodiversity.

Implications for epidemiology and population genetic structure

The results presented here have several implications for the genetic structure of ticks and tick-borne infectious agents. Because of their contrasted stage-dependent feeding behaviors, soft ticks may experience lower colonization success and restricted gene flow among populations. For the infectious agents that rely on these vectors, their genetic diversity and population structure may be affected in cascade depending on their mode of transmission [93]. To our knowledge, no study has yet examined the population genetic structure of soft ticks using hypervariable markers, but high diversity and strong clade structure at conserved genes was found in *Ornithodoros*

sonrai (Argasidae), a soft tick living in rodent burrows in west Africa, suggesting relatively low dispersal rates [94]. Based on our model results, we generally expect higher population genetic structure to be found for soft tick species than for hard ticks and that this structure may contribute to reducing the circulation of the vector-borne infectious agents. Molecular studies of infectious agents shared by both soft and hard ticks would be ideal to test these predictions [95, 96]. In this context, seabird ticks interacting with various flaviviruses and *Borrelia* bacteria (e.g. Lyme disease versus relapsing fever) could represent worthwhile systems to explore [97, 98].

Model limitations

The present model relies on several simplifications: (i) the life-cycles of soft and hard tick vectors (i.e. similar maturation rates and cycle), (ii) assumption that the source population is at equilibrium, (iii) absence of seasonality. We expect these simplifications to either be unimportant, or to result in more extreme differences between the vector groups. In this way, our basic model contributes substantially to a better understanding of how patterns of tick dispersal influence species spread (e.g. Leighton et al. [20]). For simplicity, and to better track the impact of tick movements, our model only accounts for vector movements for the introduction of the vector-borne infections. Host movements influence vector dispersal rates, and can also be the source of local infections when an infected individual disperses to a new patch where a vector population is already present. Migratory birds are, for instance, hypothesized to be responsible for the introduction of a number of such infectious agents (e.g. West Nile virus introduction in the New World; Rappole & Hubálek [99]; Gubler [100]). Recent population genetics studies have allowed us to better track vector movements between populations [28, 101], but require further developments to inform us about the relative contribution of vector *vs* vertebrate host to vector-borne parasite structure. Finally, our model does not take into account the alternative transmission mode of the pathogen via co-feeding, where an infected tick transmits the infectious agent directly to other ticks feeding on the host, without a systemic infection in the host itself. We would expect co-feeding to similarly impact hard and soft tick systems, such that our general results and predictions would not change.

Conclusions

Using our model, we highlight the importance of considering the ecological properties of the vector life-cycle, notably the role of stage-dependent dispersal, for understanding colonization potential and gene flow in the vector and in associated parasites. Although this model is based on seabird-tick systems, it is conceptually valid to be

generalized to other vector-borne systems. As several tick species are currently expanding their geographic distributions in relation to global changes [6], our approach may be especially useful to assess the associated invasion risk of vector-borne pathogens. Other ecological factors, such as sex-biased vector dispersal [102] or host-associated dispersal in multi-host systems [28], would also be worthwhile to consider with this framework to better understand and predict the spatial spread of vector-borne infections.

Additional files

Additional file 1: Figure S1. Density-dependence function (f) with respect to varying strength of the Allee effect (a). Where $K = 600, 200, 100$ and a vary from 0 to 100. High a reduces the population reproductive function and represents notably the difficulty to locate a mate when the population density is low. (PDF 48 kb)

Additional file 2: To fully understand the consequences of the soft tick polyphasic cycle on vector-infection spread, we investigated the impact of an increase in the parameter feeding rate, c . This contact rate is only increased for nymphal and adult stages, as soft ticks take multiple short bloodmeals during those stages, while they only feed once during the larval stage, therefore c is kept equal to 1 at the larval stage. A slight increase in feeding rate leads to a rapid rise in the infection prevalence in soft tick systems, but as the soft ticks nidicolous lifestyle means that they probably feed on the same host or its offspring, soft tick multiple bloodmeal per life stage would certainly not increase vector-borne infection prevalence to the extent induced by direct changes of c . **Figure S2.** Vector-borne infection prevalence for different contact rates (i.e. tick feeding rate). Hard tick feed only once a year ($c = 1$ at all times), while soft can feed multiple times during nymphal and adult stages ($c > 1$ for nymphs and adults, $c = 1$ for larvae), which could potentially increase the opportunity for transmission, if they are to feed on different hosts. Default parameters are found in Table 1, $\theta = 0.001$. **Table S1.** Parameters used for sensitivity analysis. (ZIP 217 kb)

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Availability of data and materials

All data analyzed during this study are included in the article and its additional files.

Authors' contributions

SK, KDM and TB conceived the idea. SK wrote the code, ran the model. SK, KDM and TB analyzed output data and wrote the paper. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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