CRITICAL CONTACT RATE FOR VECTOR– HOST–PATHOGEN OSCILLATION INVOLVING CO-FEEDING AND DIAPAUSE

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We consider the dynamic vector–host–pathogen interaction motivated by tick-borne diseases such as tick-borne encephalitis and Lyme disease. We stratify the vector population in terms of the stage before and after the vector’s contact with hosts when co-feeding transmission may take place, and we also consider the case where vector development may involve two time lags due to normal development and diapause. We derive threshold conditions for disease persistence and for nonlinear oscillations in the vector population and in the diseased vector and host populations. Our objective here is to use a simple mechanistic dynamic model to show that diapause and co-feeding transmission may generate periodic and irregular oscillations even when seasonal variations of the environmental conditions are ignored. These oscillations are not necessary in synchrony with the seasonality of vector development, and hence complicated oscillatory patterns of vector-borne disease dynamics in the field and surveillance observations should be expected.

Keywords: Diapause; Tick Population Dynamics; Tick-Borne Disease; Oscillation.

1. Introduction

We consider the transmission dynamics of tick-borne diseases such as tick-borne encephalitis and Lyme disease. Tick-borne diseases can be caused by infection

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with a variety of pathogens, including rickettsia and other types of bacteria, viruses and protozoa. In Europe and Asia, *Ixodes ricinus* and *Ixodes persulcatus* ticks are primary species transmitting tick-borne encephalitis and Lyme borreliosis, although infections can also be transmitted through ingesting contaminated unpasteurized cow, sheep or goat milk and milk products obtained from infected animals. Ticks can be found primarily in forests, wooded parks, grasslands and low-growing dense bush, they normally have three post-egg stages: larva, nymph and adult.^{1–5}

Mathematical models have been developed to estimate tick–host interaction and pathogen transmission dynamics. Rosà and Pugliese⁶ assessed how changes in host species densities can influence the persistence of tick-borne pathogen. Their work considered different infection routes occurring between hosts and ticks. Dunn *et al.*⁷ performed a global sensitivity analysis to rank the importance of model parameters in terms of their contribution to the observed variations in the basic reproduction number. They concluded that there are three most influential factors: the transmission efficiency from the vertebrate host to ticks, the survival rate from fed larva to feeding nymph, and the fraction of nymphs finding a competent host. Lou *et al.*⁸ proposed a stage-structured periodic model to identify the tick reproduction threshold and the disease spread threshold, and investigated effects of seasonal temperature variation and host community composition on the pattern of Lyme disease spread. Unfortunately, none of these models have incorporated the roles of co-feeding transmission and diapause.

The tick's life cycle is at least one year and normally lasts 2–3 years. This creates a long delay in the population dynamics model of ticks at a particular stage. In addition, adverse environmental conditions or a decline in day length may cause ticks to enter diapause, leading to further delay in seeking, development, or oviposition for a year or longer. Furthermore, a post-egg tick must take a blood meal from an appropriate host to progress from one stage to another. In each progress, a tick successfully quested a host attaches and bites the host, and remains in the host until engorging, and then subsequently developing into the next stage.⁹ During this progress, co-feeding transmission can take place when one susceptible tick may get infection by co-feeding with another infected tick (normally at a later stage) in the same host. Our focus here is to formulate a simple model to describe the roles of diapause and co-feeding transmission in tick population dynamics and tick-borne pathogen spread. A minimal set of compartments in such a model must involve tick populations in two different stages (relevant to the co-feeding) and two different development lags (one is related to the diapause effect). A natural question is whether such a model can exhibit nonlinear oscillations.

2. The Framework and Hypotheses

We consider a system consisting of a bacteria pathogen, a vector population with two stages, and a host population which is considered as a vehicle capable of transmitting the pathogen through both co-feeding and systemic transmission routes. We now list our major assumptions.

Model Assumptions:

- (i) In terms of the bacteria pathogen, the host and vector populations are stratified by their infection status as susceptible (s) and infected (i);
- (ii) We assume, in reference to the co-feeding process (described below), the vector population is divided into two distinguished subgroups, the subgroup of ticks questing for the first (post-egg stage) feeding (B) and the subgroup of ticks questing for the second feeding (A). The feedback from A to B can be the outcome of a sequence of developments and reproduction irrelevant to the co-feeding process. Note also that we have implicitly ignored the immediate feeding and engorging process for a B -stage questing tick to an A -stage questing tick, very much like the normal practice of SIR modelling when the incubation and/or latent period is omitted.
- (iii) Once infected vectors in the A -subgroup co-feed on a host with susceptible vectors in the B -subgroup, co-feeding may take place so that the B -stage susceptible vectors can be infected due to the co-feeding. Note that if the host is already infected, the B -stage susceptible vectors can also be infected through the systemic infection route.
- (iv) A susceptible host can be infected if it is bitten by an infected vector (at either stage) and this infection rate can be described by the mass action;
- (v) In addition to subcategory for being susceptible or infected, a host is further divided into being bitten (infested) or not by some infected A -stage vectors to reflect if co-feeding is possible. So we have H_{s+} , H_{s-} , H_{i+} , H_{i-} , here $+/-$ indicating that the host is/is not infested by A -stage infected ticks for the realization of co-feeding transmission. More precisely, H_{s+} is the subgroup of hosts which are currently susceptible to the infection, currently fed (infested) by infected vectors at the A -stage, and can pass infection to the co-feeding B -stage ticks. In contrast, H_{s-} is the subgroup of hosts which are currently susceptible to the infection, with a small number of (or no) infected vectors at the A -stage infested, but cannot pass infection to the co-feeding B -stage ticks. Throughout this work, co-feeding transmission refers to the transmission to a susceptible B -stage vector from some infected A -stage vectors when they are co-feeding on the same host, while systemic infection refers to a susceptible vector gets infection through bites from an infected host. Whether co-feeding transmission takes place or not depends on a large number of factors including the co-feeding duration and the density and (physical) proximity of A -stage infected ticks to the susceptible B -stage ticks.^{10,11} Here, we made a simplified assumption that there are only two groups of hosts: those in which co-feeding can take place and those co-feeding cannot happen (note that this is different from the assumption whether the host has one or none infested tick). A more realistic model formulation will have to involve the density and spatial distributions of the infected A -stage ticks and their relative spatial locations to the B -stage susceptible ticks within the same host. We remark that H_{i+} is the

Fig. 1. Illustrations show (a) co-feeding and systemic transmissions and (b) delays induced by normal development and diapause. At (a), a B-stage vector can get infection by feeding (i) a host from the subgroup H_{s+} through co-feeding transmission route; (ii) a host from the subgroup H_{i-} through the systemic transmission route; and (iii) a host from the subgroup H_{i+} through both co-feeding and systemic transmission routes. Development from a B-stage vector to a susceptible A-stage host is not marked in the illustration. At (b), a portion of A-stage ticks produces the next generation of B-stage vectors with an enlarged delay τ_2 than the normal development delay τ_1 for ticks without experiencing the diapause.

subgroup of hosts which are already infected and are also currently infested by the A-stage infected vectors, so a susceptible B-stage vector can have the infection risk from both systemic and co-feeding transmission routes if this B-stage vectors co-feed on the same host in the host group H_{i+} (see Fig. 1(a) for an illustration).

- (vi) Normally, it takes a development delay (denoted by τ_1) for a A-stage tick to complete a sequence of developments, reproduction (into eggs) and a further development (molting) to generate some B-stage vectors. Due to diapause,^{12–14} a portion (ϵ) of these A-stage ticks will complete this sequence with a larger development delay τ_2 , with $\tau_2 > \tau_1$ (see Fig. 1(b) for an illustration).

3. The Model

We now introduce model variables relevant to our particular focus on diapause and co-feeding. We will use $T_B(t)$ and $T_A(t)$ to denote the number of ticks questing for the first (post-egg) and second feeding, respectively, and $H(t)$ the total number of hosts involved in the transmission from B-stage to A-stage of ticks. In terms of the status of infection, $T_{As}(t)$ and $T_{Ai}(t)$ represent the numbers of susceptible and infected A-stage ticks at time t , and $T_A(t) = T_{As}(t) + T_{Ai}(t)$ is the total number of A-stage ticks.

The dynamics of $T_B(t)$, following Fig. 1(b), is described by

$$T'_B(t) = \rho_1 b(\rho_2[(1 - \epsilon)T_A(t - \tau_1) + \epsilon T_A(t - \tau_2)]) - C_B H T_B - d_B T_B, \quad (3.1)$$

where ρ_1 is the survival rate from eggs to B-stage, $b(x)$ is the egg production function, ρ_2 is the survival rate from the A-stage to egg-laying stage, $\tau_2 > \tau_1$ are the development delays from A-stage to the B-stage of ticks with or without diapause, and we assume ϵ -portion of A-stage ticks experience the diapause; d_B is the death rate of ticks at B-stage. In the above formulation, it is assumed that each B-stage vector is making C_B -number of effective contacts (attachment followed by feeding) with a host. In our analysis below, the Ricker function is used for the reproduction function, that is,

$$b(x) = r_T x e^{-s_T x}, \quad x \geq 0, \quad (3.2)$$

where r_T is the maximal number of eggs that an egg-laying female can lay per unit time since $b'(0) = r_T$, and s_T measures the strength of density dependence. This assumption reflects the ecological consideration that “the reproduction is linear in x only for small densities, decreases as a consequence of intra specific competition, and then drops significantly at very large densities due to the available resources being utilized by the adults only for their own physiological maintenance”.¹⁵ We note that

$$b(x) \leq b_\infty := b(s_T^{-1}) = r_T (s_T e)^{-1} \quad \text{for all } x \geq 0. \quad (3.3)$$

It is more complicated to describe the dynamics of $T_A(t)$ due to co-feeding and systemic transmissions. First, we use m_B to denote the molting proportion of B-stage ticks after the first feeding, so $m_B C_B T_B(t) H(t)$ is the total amount of inflow into A-stage ticks. Then, A-stage ticks seek hosts again and attach them for feeding, i.e., the corresponding feeding rate can be given by the product $C_A T_A(t) H(t)$, where C_A represents contact rate between hosts and A-stage ticks. This yields

$$T'_A(t) = m_B C_B T_B(t) H(t) - d_A T_A(t) - C_A T_A(t) H(t), \quad (3.4)$$

where d_A is the tick death rate at A-stage. We use the classical logistic model for host population dynamics:

$$H'(t) = r_h H(t) \left(1 - \frac{H(t)}{K_h} \right) - d_h H(t), \quad (3.5)$$

where d_h is the death rate per host, r_h is the intrinsic growth rate and K_h the carrying capacity of hosts. Then, we end up with the following vector-host population dynamics system

$$\begin{cases} T'_B(t) = \rho_1 b(\rho_2 [(1 - \epsilon) T_A(t - \tau_1) + \epsilon T_A(t - \tau_2)]) - C_B H(t) T_B - d_B T_B, \\ T'_A(t) = m_B C_B H(t) T_B - C_A T_A H(t) - d_A T_A, \\ H'(t) = r_h H(t) \left(1 - \frac{H(t)}{K_h} \right) - d_h H. \end{cases} \quad (3.6)$$

To describe the epidemiological dynamics for ticks, we use η_s to denote the proportion at which an A-stage vector newly developed from a successfully molted B-stage vector fed on an infected host becomes infected through the systemic

transmission. Therefore, $\eta_s m_B C_B H_{i-} T_B(t)$ gives the total inflow into $T_{Ai}(t)$ due to the contacts of B-stage vectors fed on H_{i-} hosts. We also make a simplified assumption about the co-feeding transmission that the hosts involved for the vectors transition from B-stage to A-stage have only two categories; those infested with infected A-stage vectors which can transmit the disease to susceptible B-stage vectors and those without or with lower densities of infected A-stage vectors which cannot transmit the disease to susceptible B-stage vectors through the co-feeding transmission route. We assume that an A-stage vector newly developed from a successfully molted B-stage vector fed on a H_{s+} or H_{i+} host can get the infection through the co-feeding transmission route. We use η_c to denote the proportion that a B-stage vector get infection from co-feeding infected A-stage ticks. For instance, the total inflow into $T_{Ai}(t)$ due to $T_B(t)$ contacting with H_{s+} is $\eta_c m_B C_B H_{s+}(t) T_B(t)$. These detailed systemic and co-feeding transmissions are illustrated in Figs. 2(a) and 2(b), thereby the dynamics of $T_{As}(t)$ and $T_{Ai}(t)$ are given by

$$\begin{aligned} T'_{As}(t) &= (1 - \eta_c) m_B C_B H_{s+} T_B + m_B C_B H_{s-} T_B + (1 - \eta_s) m_B C_B H_{i-} T_B \\ &\quad + (1 - \eta_s)(1 - \eta_c) m_B C_B H_{i+} T_B - C_A T_{As} H - d_A T_{As}, \\ T'_{Ai}(t) &= \eta_c m_B C_B H_{s+} T_B + (\eta_s + \eta_c - \eta_s \eta_c) m_B C_B H_{i+} T_B \\ &\quad + \eta_s m_B C_B H_{i-} T_B - C_A T_{Ai} H - d_A T_{Ai}, \end{aligned} \quad (3.7)$$

where $\eta_s + \eta_c - \eta_c \eta_s$ is the proportion that the tick gets systemic infection (η_s) and additional co-feeding infection ($\eta_c(1 - \eta_s)$), or alternatively, $\eta_s + \eta_c - \eta_c \eta_s$ is the proportion that the tick gets co-feeding infection (η_c) and additional systemic infection ($\eta_s(1 - \eta_c)$).

For the host dynamics, as we mentioned above, the classical logistic model is used to model the total host population (Eq. (3.5)). To model H_{s+} , H_{s-} , H_{i+} and H_{i-} in detail, we denote by d_f the rate at which a H_{s+} (or H_{i+}) host becomes H_{s-} (or H_{i-} respectively) due to ticks dropping off the host, and C_A be the contact rate between an A-stage vector and a host (so that after the contact, a host

Fig. 2. Illustrations show co-feeding and systemic transmission when T_B vectors feed on (a) H_{s+} and H_{i-} hosts, and (b) H_{i+} , respectively.

Table 1. Model parameters description.

Parameter	Description
ρ_1	Survival rate of eggs developing into B-stage ticks
ρ_2	Survival rate of A-stage ticks becoming egg-laying adult female
r_T	Maximum number of eggs produced by an egg-laying adult female
s_T	Reproduction reduction of egg-laying adult females
τ_1	Normal time delay of A-stage ticks developing into egg-laying adults
τ_2	Enlarged time delay of A-stage ticks developing into egg-laying adults due to diapause
ϵ	Proportion of ticks experiencing diapause
C_B	Contact rate between hosts and B-stage ticks
d_B	Death rate of B-stage ticks
d_A	Death rate of A-stage questing ticks
m_B	Molting proportion of B-stage after the first feeding
η_c	Proportion that susceptible ticks get infection from co-feeding transmission
η_s	Proportion of susceptible ticks get infection through systemic transmission
C_A	Contact rate between hosts and A-stage ticks, including the proportion of infectious ticks that are free to bound to a host
η_h	Proportion of uninfected hosts become infected when bitten by infected ticks
d_f	Rate at which an A-stage tick leaves H_+ for H_-
d_h	Death rate of hosts
r_h	Intrinsic growth rate of hosts
K_h	Carrying capacity of hosts

in the category “−” moves to the category “+”). Thereby, the host dynamics is described by

$$\begin{aligned}
 H'_{s+}(t) &= -\eta_h C_A H_{s+} T_{Ai} + (1 - \eta_h) C_A H_{s-} T_{Ai} - d_f H_{s+} - d_h H_{s+}, \\
 H'_{s-}(t) &= r_h H \left(1 - \frac{H}{K_h} \right) - C_A H_{s-} T_{Ai} + d_f H_{s+} - d_h H_{s-}, \\
 H'_{i+}(t) &= \eta_h C_A H_{s+} T_{Ai} + \eta_h C_A H_{s-} T_{Ai} + C_A H_{i-} T_{Ai} - d_f H_{i+} - d_h H_{i+}, \\
 H'_{i-}(t) &= -C_A H_{i-} T_{Ai} + d_f H_{i+} - d_h H_{i-},
 \end{aligned} \tag{3.8}$$

where H is the total number of hosts given by

$$H(t) = H_{s+}(t) + H_{s-}(t) + H_{i+}(t) + H_{i-}(t). \tag{3.9}$$

Accordingly, Eqs. (3.1)–(3.8) for $(T_B, T_{As}, T_{Ai}, H_{s+}, H_{s-}, H_{i+}, H_{i-})$ give a closed system of the vector-host dynamics. Detail explanations for model parameters are provided in Table 1.

4. Critical Value for Vector Population Oscillations

We now consider the three-dimension model (3.6) for the vector population dynamics, where

$$\begin{aligned}
 &\rho_1 b(\rho_2[(1 - \epsilon)T_A(t - \tau_1) + \epsilon T_A(t - \tau_2)]) \\
 &= \alpha_0[(1 - \epsilon)T_A(t - \tau_1) + \epsilon T_A(t - \tau_2)]e^{-\alpha_1[(1 - \epsilon)T_A(t - \tau_1) + \epsilon T_A(t - \tau_2)]}
 \end{aligned}$$

with $\alpha_0 = \rho_1 r_T \rho_2$ and $\alpha_1 = s_T \rho_2$. Assume

$$R_h := r_h d_h^{-1} > 1 \quad (4.1)$$

and $H(0) > 0$. Then, we have $H(t) \rightarrow H^*$ as $t \rightarrow \infty$, where

$$H^* := K_h(1 - R_h^{-1}) > 0. \quad (4.2)$$

Then, we consider the following limiting system

$$\begin{cases} T'_B(t) = \rho_1 b(\rho_2 T_A^{\epsilon, t}) - (C_B H^* + d_B) T_B, \\ T'_A(t) = m_B C_B H^* T_B - (d_A + C_A H^*) T_A, \end{cases} \quad (4.3)$$

with

$$T_A^{\epsilon, t} = (1 - \epsilon) T_A(t - \tau_1) + \epsilon T_A(t - \tau_2). \quad (4.4)$$

From the definition of b_∞ in (3.3), we have

$$\limsup_{t \rightarrow \infty} T_B(t) \leq \frac{\rho_1 b_\infty}{C_B H^* + d_B} := T_B^\infty, \quad (4.5)$$

and

$$\limsup_{t \rightarrow \infty} T_A(t) \leq \frac{m_B C_B H^* \rho_1 b_\infty}{(d_A + C_A H^*)(C_B H^* + d_B)} := T_A^\infty. \quad (4.6)$$

Define the phase-space for (4.3) as $X := \mathbb{R} \times C([- \tau_2, 0], \mathbb{R}^2)$ (recall $\tau_2 \geq \tau_1 \geq 0$) and $X^+ := [0, +\infty) \times C([- \tau_2, 0], \mathbb{R}_+^2)$ and assume the initial data $(T_B(0), T_A(\cdot)) \in X^+$ is given, then we obtain a solution of (4.3) for all positive time $t \geq 0$ and a corresponding semiflow on X^+ . The semiflow has a trivial equilibrium since $b(0) = 0$. The other nontrivial equilibrium is given by

$$\begin{cases} \rho_1 b(\rho_2 T_A^*) = (C_B H^* + d_B) T_B^*, \\ m_B C_B H^* T_B^* = (d_A + C_A H^*) T_A^*. \end{cases} \quad (4.7)$$

In particular, we have

$$\rho_1 b(\rho_2 T_A^*) = \frac{(C_B H^* + d_B)(d_A + C_A H^*)}{m_B C_B H^*} T_A^*. \quad (4.8)$$

Therefore, if we assume that the intrinsic birth rate

$$b'(0) = r_T > \frac{(C_B H^* + d_B)(d_A + C_A H^*)}{\rho_1 \rho_2 m_B C_B H^*} \quad (4.9)$$

is satisfied, then (4.8) always has a positive solution $T_A^* > 0$.

Define

$$R_T := \frac{r_T \rho_1 \rho_2 m_B C_B H^*}{(C_B H^* + d_B)(d_A + C_A H^*)}. \quad (4.10)$$

R_T is the basic reproduction number of the tick population: starting from a single tick at the B-stage (larva), the surviving probability during the B-stage to advance to A-stage is $C_B H^*/(C_B H^* + d_B)$ multiplying the successful molting proportion

m_B , the survived and molted questing tick grows into the egg-laying tick after $1/(d_A + C_A H^*)$ with the survival probability ρ_2 , produces a total number r_T of eggs and then grows into ticks at the B-stage with the probability ρ_1 .

In what follows, we assume $R_T > 1$. Therefore, (4.3) has one and only one positive solution $T_A^* > 0$. Using (3.2), we obtain

$$\begin{cases} T_A^* = (s_T \rho_2)^{-1} \ln(R_T), \\ T_B^* = (d_A + C_A H^*)(m_B C_B H^*)^{-1} T_A^*. \end{cases} \quad (4.11)$$

Expression (4.11) has clear ecological interpretations: As $b'(x) = r_T(1 - s_T x)e^{-s_T x} = 0$ if and only if $x^* = s_T^{-1}$, the maximal population size at which the birth rate is no longer increasing is s_T^{-1} so the equilibrium value for T_A^* is this maximum population size multiplied by the intrinsic growth rate $\ln R_T$ scaled appropriately by the survival probability of A-stage ticks to become egg-laying ticks.

Note that $b'(x) > 0$ for $x \in (0, s_T^{-1})$ and $b'(x) < 0$ for $x \in (s_T^{-1}, +\infty)$. Moreover, $b''(x) < 0$ when $x \in (0, 2s_T^{-1})$ and $b''(x) > 0$ when $x > 2s_T^{-1}$.

Note also that under the condition $R_T > 1$, $T_A^* > 0$ is satisfied. If

$$s_T^{-1} \ln(R_T) =: \rho_2 T_A^* < x^* := s_T^{-1} \quad (4.12)$$

holds, then at the equilibrium (T_B^*, T_A^*) the linearized system of (4.3) is given by

$$\begin{cases} T_B'(t) = \rho_1 \rho_2 b'(\rho_2 T_A^*) [(1 - \epsilon) T_A(t - \tau_1) + \epsilon T_A(t - \tau_2)] \\ \quad - (C_B H^* + d_B) T_B(t), \\ T_A'(t) = m_B C_B H^* T_B(t) - (d_A + C_A H^*) T_A(t). \end{cases} \quad (4.13)$$

This is a positive feedback system since $\rho_1 \rho_2 b'(\rho_2 T_A^*) > 0$, so the monotone dynamical theory can be applied. Under the assumption (4.12), we have

$$\rho_1 \rho_2 b'(\rho_2 T_A^*) < \frac{(C_B H^* + d_B)(d_A + C_A H^*)}{m_B C_B H^*}. \quad (4.14)$$

This is important for the following statement.

Theorem 4.1. *Assume $1 < R_T < e$. Then, the equilibrium (T_B^*, T_A^*) of (4.3) is locally asymptotically stable.*

Proof. Since (4.12) holds, (4.13) generates a positive semigroup. Therefore, the stability of (4.13) is equivalent to the stability of the zero solution for the linear ordinary differential equation^{16,17}

$$\begin{cases} T_B'(t) = \rho_1 \rho_2 b'(\rho_2 T_A^*) T_A - (C_B H^* + d_B) T_B, \\ T_A'(t) = m_B C_B H^* T_B - (d_A + C_A H^*) T_A, \end{cases} \quad (4.15)$$

and the stability of the above solution is determined by whether or not

$$(C_B H^* + d_B)(d_A + C_A H^*) > \rho_1 \rho_2 b'(\rho_2 T_A^*) m_B C_B H^*, \quad (4.16)$$

see Corollary 3.2 and the discussion in Ref. 17. In view of (4.14) implied by (4.12), namely $R_T < e$, we complete the proof. $\square$

It should also be noted from (4.5) and (4.6) that solutions of (4.3) eventually enter the rectangle $\Pi := \{(T_B(0), T_A)|_{[-\tau_2, 0]} | 0 \leq T_B(0) \leq T_B^\infty \text{ and } 0 \leq T_A(\theta) \leq T_A^\infty, \theta \in [-\tau_2, 0]\}$. With $1 < R_T < e$ and whenever $0 \leq T_A^{\epsilon, t} \leq T_A^\infty, T_B(t) = T_B^\infty$, we have

$$\begin{aligned} T_B'(t) &\leq \rho_1 b(\rho_2 T_A^\infty) - (C_B H^* + d_B) T_B^\infty \\ &= \rho_1 b(\rho_2 T_A^\infty) - \frac{(C_B H^* + d_B)(d_A + C_A H^*)}{m_B C_B H^*} T_A^\infty \leq 0. \end{aligned} \tag{4.17}$$

Similarly, whenever $0 \leq T_B(t) \leq T_B^\infty, 0 \leq T_A^{\epsilon, t} \leq T_A^\infty$ and $T_A(t) = T_A^\infty$, we have $T_A'(t) \leq 0$. Therefore, the set Π is also positively invariant, and $(0, 0)$ and (T_B^*, T_A^*) are the only equilibria in Π . Consequently, we also have from the monotone dynamical system theory^{16,17} that

Theorem 4.2. *If $1 < R_T < e$, then every nontrivial solution of (4.3) converges to (T_B^*, T_A^*) as $t \rightarrow \infty$.*

Note that $R_T = R_T(C)$ defined in (4.10) is an increasing function of $C > 0$, where $C = C_B H^*$. Therefore, there exists $0 < C_{B,1} < C_{B,2}$ such that $R_T(C_{B,1}) = 1$ and $R_T(C_{B,2}) = e$. Therefore, we have shown that system (4.3) has no nontrivial equilibrium if $C < C_{B,1}$; system (4.3) has a unique positive equilibrium (T_B^*, T_A^*) when $C > C_{B,1}$; and this positive equilibrium is locally asymptotically stable if $C \in (C_{B,1}, C_{B,2})$.

The characteristic equation of (4.3) at the given positive equilibrium (T_B^*, T_A^*) is $\det[\lambda I - J] = 0$ with the matrix J given by

$$J = \begin{pmatrix} -C_B H^* - d_B & J_{12} \\ m_B C_B H^* & -(d_A + C_A H^*) \end{pmatrix}, \tag{4.18}$$

with $J_{12} = \alpha_0[(1 - \epsilon)e^{-\lambda\tau_1} + \epsilon e^{-\lambda\tau_2}]e^{-\alpha_1 T_A^*}(1 - \alpha_1 T_A^*)$. Therefore, the characteristic equation becomes

$$\begin{aligned} &(\lambda + C_B H^* + d_B)(\lambda + d_A + C_A H^*) - \alpha_0 m_B C_B H^*(1 - \alpha_1 T_A^*) \\ &\times [(1 - \epsilon)e^{-\lambda\tau_1} + \epsilon e^{-\lambda\tau_2}]e^{-\alpha_1 T_A^*} = 0. \end{aligned} \tag{4.19}$$

That is,

$$\lambda^2 + A_1(C_B)\lambda + A_2(C_B) + A_3(C_B)e^{-\lambda\tau_1} + A_4(C_B)e^{-\lambda\tau_2} = 0, \tag{4.20}$$

where

$$\begin{aligned} A_1(C_B) &= C_B H^* + d_A + C_A H^* + d_B > 0, \\ A_2(C_B) &= (d_A + C_A H^*)(C_B H^* + d_B) > 0, \\ A_3(C_B) &= -(1 - \epsilon)\alpha_0 m_B C_B H^*(1 - \alpha_1 T_A^*)e^{-\alpha_1 T_A^*}, \\ A_4(C_B) &= -\epsilon\alpha_0 m_B C_B H^*(1 - \alpha_1 T_A^*)e^{-\alpha_1 T_A^*}. \end{aligned}$$

Assume that a pair of purely imaginary solution of the form $\lambda = \pm i\omega$ exists in Eq. (4.20), where ω is a positive real number. Substituting it into (4.20) and separating the real and imaginary parts, we can obtain the following transcendental equations:

$$\begin{cases} f_1(\omega) = -\omega^2 + A_2(C_B) + A_3(C_B) \cos(\omega\tau_1) + A_4(C_B) \cos(\omega\tau_2) = 0, \\ f_2(\omega) = A_1(C_B)\omega - A_3(C_B) \sin(\omega\tau_1) - A_4(C_B) \sin(\omega\tau_2) = 0. \end{cases} \quad (4.21)$$

Moving the terms $A_3(C_B) \cos(\omega\tau_1)$ and $A_3(C_B) \sin(\omega\tau_1)$ into right sides of the equations of (4.21), then taking square on both sides and summing them up, we obtain

$$\begin{aligned} \omega^4 + (A_1^2 - 2A_2 - 2A_4 \cos(\omega\tau_2))\omega^2 - 2A_1A_4 \sin(\omega\tau_2)\omega \\ + A_2^2 - A_3^2 + A_4^2 + 2A_2A_4 \cos(\omega\tau_2) = 0. \end{aligned} \quad (4.22)$$

Equation (4.22) has a positive real root ω^* if the following condition holds

$$(A_2 + A_4)^2 < A_3^2. \quad (4.23)$$

Since $1 - \alpha_1 T_A^* < 0$ is satisfied, Eq. (4.23) can be reduced into $A_2 + A_4 < A_3$ due to $A_3 > 0$ and $A_4 > 0$. Therefore, Eq. (4.22) has a positive real root ω^* if

$$2\epsilon > 1, \quad (4.24)$$

that is, if at least half of the ticks have gone through the diapause process, and if

$$C_B > \frac{(d_A + C_A H^*)d_B}{H^*} (\alpha_0 m_B e^{-1 + \frac{1}{2\epsilon-1}} - d_A - C_A H^*)^{-1}. \quad (4.25)$$

In what follows, we define C_B^* as the minimal contact rate (larger than $C_{B,2}$) at which there is a pair of purely imaginary eigenvalues (denoted by $\pm i\omega^*$). To confirm there is indeed a Hopf bifurcation of periodic solutions bifurcated from the equilibrium, we need to verify the transversal condition. Differentiating (4.20) with respect to C_B and substituting the eigenvalue ω^* and critical value C_B^* , we obtain

$$\begin{aligned} P \left(\frac{d(\operatorname{Re}\lambda)}{dC_B} \right) \Big|_{C_B^*} + Q \left(\frac{d\omega}{dC_B} \right) \Big|_{C_B^*} &= R, \\ -Q \left(\frac{d(\operatorname{Re}\lambda)}{dC_B} \right) \Big|_{C_B^*} + P \left(\frac{d\omega}{dC_B} \right) \Big|_{C_B^*} &= S, \end{aligned} \quad (4.26)$$

where

$$\begin{aligned} P &= A_1(C_B) - \tau_1 A_3(C_B) \cos(\omega\tau_1) - \tau_2 A_4(C_B) \cos(\omega\tau_2), \\ Q &= -2\omega - \tau_1 A_3(C_B) \sin(\omega\tau_1) - \tau_2 A_4(C_B) \sin(\omega\tau_2), \\ R &= -A_2'(C_B) - A_3'(C_B) \cos(\omega\tau_1) - A_4'(C_B) \cos(\omega\tau_2), \\ S &= -A_1'(C_B)\omega + A_3'(C_B) \sin(\omega\tau_1) + A_4'(C_B) \sin(\omega\tau_2). \end{aligned}$$

From these equations, we can have the transversality condition

$$\left(\frac{d(\operatorname{Re}\lambda)}{d\tau_1} \right) \Big|_{C_B^*} := \frac{PR - QS}{P^2 + Q^2} \neq 0. \quad (4.27)$$

Therefore, assuming that the condition (4.25) holds and (4.27) is nonzero, then the positive equilibrium E of model (3.6) is locally asymptotically stable for $C_B \in (C_{B,1}, C_B^*)$ and there undergoes a Hopf bifurcation at $C_B = C_B^*$.

To summarize, we have

Theorem 4.3. *Assume $R_T > e$, $2e > 1$. Then, there exists $C_B^* > C_{B,2}$ so that at C_B^* , the linearization at the nontrivial equilibrium (T_B^*, T_A^*) has a pair of purely imaginary eigenvalues $\pm i\omega^*$ with $\omega^* > 0$. Assuming the transversality condition (4.27) is satisfied, then the model system (3.6) undergoes a Hopf bifurcation.*

In Sec. 6, we will numerically examine an example to calculate the corresponding critical contact rate C_B for such a Hopf bifurcation to take place.

5. Implication for Pathogen Spread

Substituting constant solutions T_B^* and T_A^* into the full epidemic model (3.1)–(3.8), we now consider the following system relevant to infected ticks and hosts:

$$\begin{aligned} T'_{Ai}(t) &= \eta_c m_B C_B T_B^* H_{s+} + (\eta_s + \eta_c - \eta_s \eta_c) m_B C_B T_B^* H_{i+} \\ &\quad + \eta_s m_B C_B T_B^* H_{i-} - C_A T_{Ai} H^* - d_A T_{Ai}, \\ H'_{s+}(t) &= (1 - \eta_h) C_A (H^* - H_{s+} - H_{i+} - H_{i-}) T_{Ai} \\ &\quad - \eta_h C_A H_{s+} T_{Ai} - (d_f + d_h) H_{s+}, \\ H'_{i+}(t) &= \eta_h C_A H_{s+} T_{Ai} + \eta_h C_A (H^* - H_{s+} - H_{i+} - H_{i-}) T_{Ai} \\ &\quad + C_A H_{i-} T_{Ai} - (d_f + d_h) H_{i+}, \\ H'_{i-}(t) &= -C_A H_{i-} T_{Ai} + d_f H_{i+} - d_h H_{i-}. \end{aligned} \quad (5.1)$$

We can define and calculate the basic reproduction ratio of pathogen population of system (5.1) following the idea in Ref. 18. In particular, we linearize system (5.1) at the disease-free equilibrium $(T_{Ai}, H_{s+}, H_{i+}, H_{i-}) = (0, 0, 0, 0)$ to obtain the new infection matrix

$$F = \begin{pmatrix} 0 & \eta_c m_B C_B T_B^* & (\eta_s + \eta_c - \eta_s \eta_c) m_B C_B T_B^* & \eta_s m_B C_B T_B^* \\ (1 - \eta_h) C_A H^* & 0 & 0 & 0 \\ \eta_h C_A H^* & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and the progression matrix

$$V = \begin{pmatrix} C_A H^* + d_A & 0 & 0 & 0 \\ 0 & d_f + d_h & 0 & 0 \\ 0 & 0 & d_f + d_h & 0 \\ 0 & 0 & -d_f & d_h \end{pmatrix}.$$

Thus, the basic reproduction ratio $\mathcal{R}_0$ of pathogen population of system (5.1) is the spectral radius of matrix FV^{-1} , given by

$$\begin{aligned}\mathcal{R}_0 &= \rho(FV^{-1}) \\ &= \sqrt{\frac{C_A H^* m_B C_B T_B^*}{C_A H^* + d_A} \left(\frac{(1 - \eta_h) \eta_c}{d_f + d_h} + \frac{\eta_h (\eta_s + \eta_c - \eta_s \eta_c)}{d_f + d_h} + \frac{\eta_h \eta_s}{d_h} \frac{d_f}{d_f + d_h} \right)}.\end{aligned}\quad (5.2)$$

Furthermore, the value of $\mathcal{R}_0$ defined in Eq. (5.2) can offer as a threshold to characterize the co-existence of pathogen population in the following theorem.

Theorem 5.1. *The following statements are valid:*

- (i) *If $\mathcal{R}_0 < 1$, system (5.1) admits a unique zero equilibrium;*
- (ii) *If $\mathcal{R}_0 > 1$, system (5.1) admits a unique positive equilibrium*
 $E_1 = (T_{Ai}^*, H_{s+}^*, H_{i+}^*, H_{i-}^*).$

Proof. Let

$$x_1 = T_{Ai}(t), \quad x_2 = H_{s+}(t), \quad x_3 = H_{i+}(t), \quad x_4 = H_{i-}(t). \quad (5.3)$$

Assuming all right hands of equations of system (5.1) be zeros, and then adding the second and third equations and third and fourth equations, respectively, lead to

$$x_2 + x_3 = \frac{C_A H^* x_1}{C_A x_1 + d_f + d_h} \quad \text{and} \quad x_3 + x_4 = \frac{\eta_h C_A H^* x_1}{\eta_h C_A x_1 + d_h}. \quad (5.4)$$

Moreover, from the fourth equation of system (5.1), we obtain

$$x_3 = \frac{C_A x_1 + d_h}{d_f} x_4. \quad (5.5)$$

Thus, through substituting Eq. (5.5) into above Eqs. (5.4), we can express the expressions of x_2 , x_3 and x_4 in terms of x_1 as:

$$x_4 = \frac{\eta_h C_A x_1 H^*}{\eta_h C_A x_1 + d_h} \frac{d_f}{C_A x_1 + d_f + d_h}, \quad (5.6)$$

$$x_3 = \frac{\eta_h C_A x_1 H^*}{\eta_h C_A x_1 + d_h} \frac{C_A x_1 + d_h}{C_A x_1 + d_f + d_h}, \quad (5.7)$$

$$x_2 = \frac{C_A x_1 H^*}{C_A x_1 + d_f + d_h} \frac{(1 - \eta_h) d_h}{\eta_h C_A x_1 + d_h}. \quad (5.8)$$

Substituting the above expressions of x_2 , x_3 and x_4 into the right hand of the first equation from system (5.1) and letting this be zero, we can obtain a quadratic equation with respect to nonzero variable x_1 as follows:

$$ax_1^2 + bx_1 + c = 0, \quad (5.9)$$

where

$$\begin{aligned} a &= (C_A H^* + d_A) \eta_h C_A^2, \\ b &= (C_A H^* + d_A) C_A (d_h + \eta_h (d_f + d_h)) - m_B C_B T_B^* C_A^2 H^* (\eta_s + \eta_c - \eta_s \eta_c) \eta_h, \\ c &= (C_A H^* + d_A) d_h (d_f + d_h) - m_B C_B T_B^* C_A H^* ((1 - \eta_h) \eta_c d_h \\ &\quad + \eta_h (\eta_s + \eta_c - \eta_s \eta_c) d_h + \eta_h \eta_s d_f) \\ &= (C_A H^* + d_A) d_h (d_f + d_h) (1 - \mathcal{R}_0^2). \end{aligned}$$

Denoting by λ_1 and λ_2 , the two solutions of the above quadratic equation (5.9), we obtain

$$\lambda_1 + \lambda_2 = \frac{d_h + \eta_h (d_f + d_h)}{\eta_h C_A} \left(\frac{m_B C_B T_B^* C_A H^*}{C_A H^* + d_A} \frac{\eta_h (\eta_s + \eta_c - \eta_s \eta_c)}{d_h + \eta_h (d_f + d_h)} - 1 \right).$$

One can prove that

$$\frac{\eta_h (\eta_s + \eta_c - \eta_s \eta_c)}{d_h + \eta_h (d_f + d_h)} < \frac{(1 - \eta_h) \eta_c}{d_f + d_h} + \frac{\eta_h (\eta_s + \eta_c - \eta_s \eta_c)}{d_f + d_h} + \frac{\eta_h \eta_s}{d_h} \frac{d_f}{d_f + d_h}.$$

Accordingly, we have

$$\lambda_1 + \lambda_2 < \frac{d_h + \eta_h (d_f + d_h)}{\eta_h C_A} (\mathcal{R}_0^2 - 1)$$

and

$$\lambda_1 \lambda_2 = \frac{d_h (d_f + d_h)}{\eta_h C_A^2} (1 - \mathcal{R}_0^2).$$

Obviously, when $\mathcal{R}_0 > 1$, there exists a unique positive solution $x_1^* > 0$ of Eq. (5.9); when $\mathcal{R}_0 < 1$, there is no positive solution of x_1 of Eq. (5.9). When there is a unique positive solution of x_1^* , we can easily find the unique positive solution of x_2 , x_3 and x_4 of system (5.1). This completes the proof. $\square$

We also note that when C_B crosses the first bifurcation value C_B^* , the tick system (3.6) admits a unique periodic solution of $T_B^*(t)$ and $T_A^*(t)$. Substituting $T_B^*(t)$ and $T_A^*(t)$ into the full system, we can have a limiting periodic system and we can then follow the procedure in Refs. 3 and 19 to define a next generation operator and its spectral radius as the basic reproduction number which is close to the formula defined in Eq. (5.2) where C_B is close to C_B^* due to the continuity of the spectral radius.

6. Numerical Illustrations

We use some numerical simulations to illustrate the theoretical stability and bifurcation analysis. This sets a stage for our final discussions about the possible research topics in the future.

We mention that for the purpose of numerical simulations, all parameter values listed below are purely illustrative, though elaborated from the literature.^{6,20,21} We fix $\rho_1 = 0.0252$, $\rho_2 = 0.1$, $r_T = 2000 \text{ (day}^{-1}\text{)}$, $s_T = 0.001$, $\tau_1 = 750 \text{ (day)}$, $\tau_2 = 1100 \text{ (day)}$, $\epsilon = 0.6$, $d_B = 0.0277 \text{ (day}^{-1}\text{)}$, $d_A = 0.0277 \text{ (day}^{-1}\text{)}$, $m_B = 1$, $\eta_c = 0.0004$, $\eta_s = 0.8$, $C_A = 0.001$, $\eta_h = 0.8$, $d_f = 0.2 \text{ (day}^{-1}\text{)}$, $d_h = 0.087 \text{ (day}^{-1}\text{)}$, $r_h = 0.1 \text{ (day}^{-1}\text{)}$, $K_h = 200 \text{ (day}^{-1}\text{)}$.

A stability and bifurcation analysis for even a scalar delay differential equation with two distinct delays is always difficult. In most existing analyses, the two time lags are considered as bifurcation parameters. In the model system, we are considering the biological issues relevant to diapause and co-feeding suggest two other parameters: the parameter ϵ , the portion when the population has to go through a diapause process to gain maturity after time lag τ_2 and the contact rate C_B for the development of ticks to the next stage before co-feeding. In our case, motivated by tick-borne encephalitis and Lyme disease, τ_1 and τ_2 are relatively fixed to approximately 2 and 4-years. Therefore, τ_1 as 750 (days) and τ_2 as 1100 (days) and $\epsilon = 0.6$ so that $2\epsilon > 1$.

When $C_B = 0.7 \times 10^{-4} \text{ (day}^{-1}\text{)}$, there exists a positive equilibrium (485440, 18803, 26) for the model (3.6), but the algebraic equation (4.22) has no positive solution, leading to positive steady state solution of tick population which is locally asymptotically stable. Moreover, we can calculate the basic reproduction ratio of pathogen population as 11.0017 which is greater than 1. Thus, by Theorem 5.1, the epidemic model (5.1) admits a positive steady state (13182, 0.3254, 21.0260, 2.9926) which is locally asymptotically stable as shown in Fig. 3(a).

Based on the theoretical analysis, we can calculate the critical value of the contact rate $C_B^* = 1.195 \times 10^{-4} \text{ (day}^{-1}\text{)}$, this leads to the corresponding positive equilibrium $E(388480, 22477, 26)$ of the model (3.6), and Eq. (4.21) has a unique

Fig. 3. Solutions of $T_A(t)$ for different contact rate (a) $C_B = 0.7 \times 10^{-4}$, (b) $C_B = 1.25 \times 10^{-4}$; (c) $C_B = 2.5 \times 10^{-4}$, and (d) $C_B = 3.3 \times 10^{-4}$. (a) $T_A(t)$ approaches a positive steady state; (b) $T_A(t)$ approaches a periodically oscillatory solution; (c) solution of $T_A(t)$ approaches a dual-peak periodic function when C_B is increased to 2.5×10^{-4} ; (d) solution of $T_A(t)$ increases the complexity of oscillation when C_B is further increased to 3.3×10^{-4} .

(b)

(c)

(d)

Fig. 3. (Continued)

solution $\omega^* = 3.4199 \times 10^{-3}$ corresponding to this C_B^* . Thereby, according to the Hopf Bifurcation Theorem, model (3.6) undergoes a Hopf bifurcation at E and a bifurcated periodic solution exists for C_B slightly larger than C_B^* , as depicted in Fig. 3(b). The period is 2100 (days), close to double the size of τ_2 .

Increasing the contact rate further generates multiple peaks and valleys in both tick population and infections. For instance, when we choose $C_B = 2.5 \times 10^{-4}$,

within the period of a periodic solution, there are dual-peaks in both tick population and infections, where the period is also increased to 2310 days (see Fig. 3(c)). Increasing the contact rate further to 3.3×10^{-4} , we observe more complex oscillations in the infected A-stage ticks while the period is also increased (see Fig. 3(d)).

7. Discussions

In our theoretical analyses and simulations, we have used the contact rate C_B as the bifurcation parameter to examine the impact of diapause on nonlinear oscillations when co-feeding takes place. We have fixed the parameter ϵ , the fraction of ticks going through the diapause process to reach maturity with some delays. It will be a very interesting problem for future study to conduct the bifurcation analysis when we change ϵ , and in particular, to look at the critical diapause fraction (when two delays τ_1 and τ_2 are fixed) when Hopf bifurcation takes place. A good starting point would be to consider the case where the tick population model exhibits convergence to equilibria without diapause, and exhibits convergence to periodic solutions when all populations have to go through the diapause to reach the maturity.

Annual periodic cycles of tick population dynamics have been frequently observed in the fields and theoretically confirmed in the model study²² where a system of ordinary differential equations is used and where periodic coefficients are assumed to represent the seasonal variation of tick ecological activities including questing, feeding, development, reproduction and mortality. These annual cycles have also been established in the work,²³ using a system of delay differential equations with both periodic delays (for development times) and periodic coefficients. When these periodic coefficients and delays are replaced by constants, the model systems converge to equilibrium states. Therefore, the periodicity observed in these studies is caused by the seasonal variations and the period of the periodic solutions is fixed to one year. Here, we show that there is another mechanism for nonlinear oscillations in tick population dynamics, arising from diapause which is caused by environmental conditions to delay the development of ticks from one stage to the next by a year or two. These diapause-caused oscillations are generally not in synchrony with the seasonality, and multiple peaks and valleys can appear within a single period. While the oscillation frequency of the diapause-induced periodic oscillations can be estimated using model parameters, from the bifurcation analysis (at least when the bifurcation value C_B is close to the critical value), the distance between consequent peaks within a single period is hard to estimate. Developing effective methods to estimate this distance remains to be a topics for future research and this will be practically useful for predicting the (temporally) local outbreak.

Our model formulation is motivated by the co-feeding transmission observed in tick-borne diseases, and for the sake of simplicity, we have chosen our focus on the transition of the ticks between the two stages relevant to the co-feeding. As such, we have ignored a few physiologically distinct stages and the model parameters, specially the development delay and the development and survival rates, all involve

distinct stages for which human interventions and environmental conditions have different effects. To fit the model with real surveillance data and to use the model to evaluate the impact of human interventions and environmental variation on controlling tick population growth and tick-borne disease spread, we need to refine the model to incorporate all four different tick development stages and also to use periodic coefficients rather than constant coefficients to reflect the seasonal variation aforementioned. The diapause-induced oscillation and the seasonal variations combined are expected to generate more complicate tick population dynamics oscillations, which in turn will generate complex temporal patterns of tick-borne disease dynamics.

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