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MODELLING MALARIA DYNAMICS IN TEMPERATE REGIONS WITH LONG TERM INCUBATION PERIOD

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The incubation period of malaria can vary depending on the species of parasite or the geographic regions. In particular, in endemic areas of temperate climate, the incubation period of *Plasmodium vivax* shows bimodal distribution of short and long term incubation periods. In this paper, we compare two transmission models for *P. vivax* malaria, where we model the long term incubation period using ordinary differential equations or delay differential equations. We show that, while the qualitative behaviors of the two models are similar, the ODE model overestimates the basic reproduction number and also the level of endemicity, compared to the DDE model. However, when we incorporate seasonality, the interplay of the time delay and the periodicity results that in some situations the DDE model predicts higher prevalence of malaria.

1. Introduction

Malaria is a mosquito-borne infectious disease caused by protozoan parasites of the genus *Plasmodium*. While feeding on humans, infected female mosquitoes inject parasites into the bloodstream, which infect liver cells. Parasites stay in liver cells until they are released back into the bloodstream and ready to be spread to another mosquito. The time between the mosquito bite and the release of the parasites from the liver is the incubation period [1]. The incubation period can vary depending on the species of the parasite or the geographic regions. In particular, the incubation period of *Plasmodium vivax* – the malaria inducing parasite species most
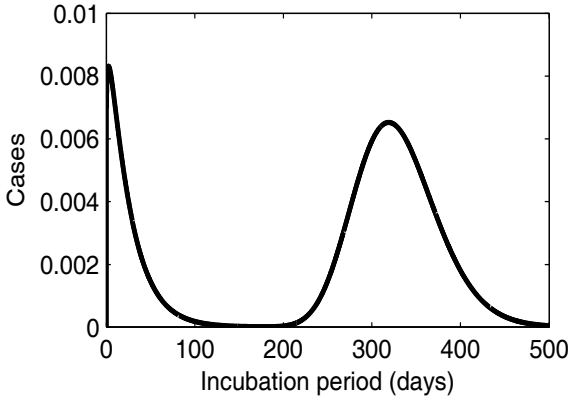
Figure 1. Estimation of the probability density function of *P. vivax* incubation time in Korea [8,15].

prevalent in temperate zones – shows a bimodal distribution, with clearly distinct short term and long term incubation periods.

The classical mathematical models for the dynamics of malaria transmission are based on differential equations[1,2], following the works of Ross and Macdonald. The simplest models describe the incubation period by exponential distribution, thus using ordinary differential equations in which the latent compartment decays exponentially in the absence of inflow from the susceptible compartment. Meanwhile, the delayed Ross-Macdonald model considers that the incubation periods of hosts and vectors have fixed lengths, thus including constant delay terms in the model equations. Most of the previous malaria models adapted either exponential distribution or Dirac–delta distribution[3,4,5,6,7].

The research of Nah *et al.*[7] used two separated compartments for the latent class – distinguishing short and long incubation times – in order to express the distribution of incubation times of *P. vivax* observed in Korea. That model assumes exponential distribution for both the short and the long incubation times, with different mean values. However, based on the empirical estimations of *P. vivax* incubation time in Korea[8,9], it is natural to use discrete delay for the long term incubation period, as a much better approximation of the empirical observation (see Fig. 1) than the exponential distribution assumption. The cumulative distribution functions are depicted in Fig. 2, and one can see that assuming fixed length for the long term incubation period gives a distribution that is much closer to
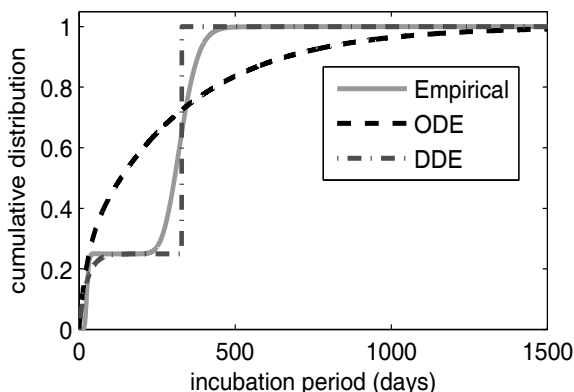
Figure 2. Cumulative distribution functions of incubation time. The Kantorovich distance between the distributions of the incubation time using DDE model and Empirical observation was 31.4, being much smaller than the distance between ODE model and Empirical observation, which is 154.8.

the empirical distribution in the most common probability metrics (such as the Kantorovich metric or the Lévy metric[a]), than the exponentially distributed long term incubation period.

In this paper, we introduce two models for *P. vivax* transmission dynamics where both short and long incubation times are present. In both cases, we separate the exposed individuals into two distinct compartments, depending on the length of their incubation period (short term or long term). In the first model, we assume exponential distribution for the long term incubation period, thus resulting a system of ordinary differential equations (ODE). In the second model, we assume fixed length for the long term incubation period, obtaining a system of delay differential equations (DDE). Our goal is to compare the two models by means of mathematical analysis, to investigate the qualitative and quantitative differences between the two models, and to discuss the implications of these two approaches. Since a contact rate between mosquitoes and humans has strong seasonality in temperate regions where *P. vivax* is endemic, we also study the disease dynamics given by those two models in a periodic environment.

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[a]If F_X and F_Y are the distribution functions of random variables X and Y, the Kantorovich distance is defined by $d_K(X,Y) := \int_{-\infty}^{\infty} |F_X(x) - F_Y(x)| dx$, and the Lévy distance is $d_L(X,Y) := \inf\{\epsilon : F_Y(x-\epsilon) - \epsilon \leq F_X(x) \leq F_Y(x+\epsilon) + \epsilon\}$ [10].
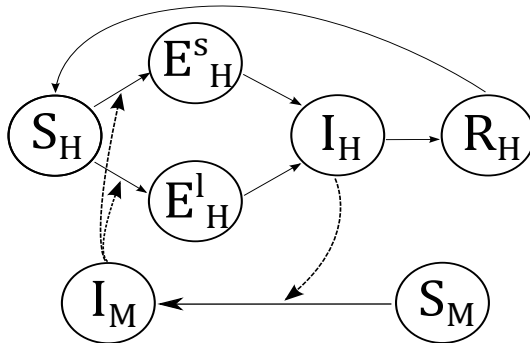
Figure 3.   *P. vivax* transmission model.

2. Model description

To describe the transmission of *P. vivax* malaria, we assume SEIRS disease dynamics for the human and SI for the mosquito population (Fig. 3). Exposed humans are divided into two classes by having short term or long term incubation periods. If a susceptible human (S_H) is successfully infected by a mosquito (I_M), then this individual goes through short incubation period (E_H^s) with probability p, or long incubation period (E_H^l) with probability $1 - p$; then becomes infectious (I_H) after this incubation time and be able to infect susceptible mosquitoes (S_M). Recovered humans are in the class R_H, and return to S_H after their immunity wanes.

The cross-infection between mosquitoes and humans is described by the terms $abI_M \frac{S_H}{H}$ and $acS_M \frac{I_H}{H}$, where a is the per capita biting rate of mosquitoes with b, c transmission efficiency, and H is the human population size. Assuming constant human and mosquito populations $(H$ and $M)$, our system can be rescaled by introducing the new variables

$$s_H = \frac{S_H}{H}, \quad e_H^s = \frac{E_H^s}{H}, \quad e_H^l = \frac{E_H^l}{H}, \quad i_H = \frac{I_H}{H}, \quad r_H = \frac{R_H}{H},$$

$$s_M = \frac{S_M}{M} \quad \text{and} \quad i_M = \frac{I_M}{M}.$$

The cross-infection terms become $abmi_M s_H$ and $acs_M i_H$, where $m = \frac{M}{H}$.

We consider two models: the short term incubation time has an exponential distribution in both models, but the distributions of long term incubation time are different. The first model assumes exponential distribution for long incubation time, as in previous research. In the second model, individuals going through long term incubation time have the same

length of incubation time, i.e. long term incubation time has Dirac–delta distribution. The second model better describes the observed distribution of long term incubation time than the first one (see Fig. 1 and 2), however, the second model is more difficult to analyze mathematically.

2.1. *ODE model with exponentially distributed incubation periods*

First we consider the system

$$
\begin{cases}
\dfrac{ds_H}{dt} = \xi - abms_H i_M + \omega r_H - \xi s_H, \\[2mm]
\dfrac{de_H^s}{dt} = pabms_H i_M - d_s e_H^s - \xi e_H^s, \\[2mm]
\dfrac{de_H^l}{dt} = (1-p)abms_H i_M - d_l e_H^l - \xi e_H^l, \\[2mm]
\dfrac{di_H}{dt} = d_s e_H^s + d_l e_H^l - r i_H - \xi i_H, \\[2mm]
\dfrac{dr_H}{dt} = r i_H - \omega r_H - \xi r_H, \\[2mm]
\dfrac{ds_M}{dt} = \mu - acs_M i_H - \mu s_M, \\[2mm]
\dfrac{di_M}{dt} = acs_M i_H - \mu i_M.
\end{cases}
\tag{1}
$$

For the explanation of the parameters we refer to Table 1. Model (1) is modified from the model of Nah *et al.* [7], and assumes that individuals leave the exposed compartments at constant rate. The feasible domain $D = \{(s_H, e_H^s, e_H^l, i_H, r_H, s_H, i_M) \in \mathbb{R}_+^7 | s_H + e_H^s + e_H^l + i_H + r_H = 1, s_M + i_M = 1\}$ is clearly invariant.

The term *abm* describes the successful contacts infectious mosquitoes have with humans per unit time, $\frac{1}{\mu}$ is the length of the infectious period of mosquitoes. Since $p\frac{d_s}{d_s+\xi} + (1-p)\frac{d_l}{d_l+\xi}$ is the probability that an infected human survives the exposed state and becomes infectious, ac is the number of valid contacts infectious humans have with mosquitoes per unit time and $\frac{1}{r+\xi}$ is the length of the infectious period of a human; we can define the basic reproduction number R_o for the ODE model by

$$
R_o = \sqrt{\frac{a^2 bcm}{(r+\xi)\mu}\left(p\frac{d_s}{d_s+\xi} + (1-p)\frac{d_l}{d_l+\xi}\right)},
\tag{2}
$$

where we adapted the convention of taking the square root as reproduction requires two epidemiological generations. We show that R_o works as a threshold for the existence and stability of equilibria of system (1).

Table 1.   Description of model parameters.

parameter	description
ξ (μ)	human (mosquito) birth/death rate
b (c)	transmission efficiency from infected mosquito (human) to human (mosquito)
a	biting rate of mosquitoes
m	proportion of mosquito population to human population
d_s (d_l)	rate of progression from the short (long) term exposed state to the infectious state
r	recovery rate
ω	rate of loss of immunity
p	probability of exposed humans going through short term incubation periods
τ	length of long-term incubation period

Lemma 2.1. *The disease free equilibrium (DFE) $(1, 0, 0, 0, 0, 1, 0)$ of system (1) always exists. An endemic equilibrium (EE) exists if and only if $R_o > 1$ and it is given by the following relations:*

$$i_H^* = \frac{R_o^2 - 1}{\frac{ac}{\mu} + K_o R_o^2}, \quad e_H^{s*} = \frac{\frac{p}{d_s + \xi}(r + \xi)}{p\frac{d_s}{d_s + \xi} + (1 - p)\frac{d_l}{d_l + \xi}} i_H^*, \quad e_H^{l*} = \frac{1 - p}{d_l + \xi}\frac{d_s + \xi}{p} e_H^{s*},$$

$$r_H^* = \frac{r}{\omega + \xi} i_H^*, \quad i_M^* = \frac{\frac{ac}{\mu} i_H^*}{1 + \frac{ac}{\mu} i_H^*}, \quad s_H^* = 1 - e_H^{s*} - e_H^{l*} - i_H^* - r_H^*$$

and $s_H^* = 1 - i_M^*$, where $K_o = \frac{\frac{p}{d_s + \xi} + \frac{1 - p}{d_l + \xi}}{p\frac{d_s}{d_s + \xi} + (1 - p)\frac{d_l}{d_l + \xi}}(r + \xi) + 1 + \frac{r}{\omega + \xi}.$

Proof. See Appendix A.   □

Theorem 2.1. *The DFE of system (1) is locally asymptotically stable if $R_o < 1$ and is unstable if $R_o > 1$. The EE is locally asymptotically stable whenever exists, i.e. if $R_o > 1$.*

Proof. See Appendix C.   □

2.2. *DDE model with fixed length incubation period*

For the ODE model in section 2.1, exponential distribution was assumed for the long incubation period, with mean $1/d_l$. In this section, we introduce a DDE model assuming every individual has the same length of long incubation period, τ. For the sake of comparison with the ODE model, we

set $\tau = 1/d_l$. The model reads as

$$
\begin{cases}
\dfrac{ds_H}{dt} = \xi - abms_H i_M + \omega r_H - \xi s_H, \\[2mm]
\dfrac{de_H^s}{dt} = pabms_H i_M - d_s e_H^s - \xi e_H^s, \\[2mm]
\dfrac{de_H^l}{dt} = (1-p)abms_H i_M - (1-p)abms_H(t-\tau)i_M(t-\tau)e^{-\xi\tau} - \xi e_H^l, \\[2mm]
\dfrac{di_H}{dt} = d_s e_H^s + (1-p)abms_H(t-\tau)i_M(t-\tau)e^{-\xi\tau} - ri_H - \xi i_H, \\[2mm]
\dfrac{dr_H}{dt} = ri_H - \omega r_H - \xi r_H, \\[2mm]
\dfrac{ds_M}{dt} = \mu - acs_M i_H - \mu s_M, \\[2mm]
\dfrac{di_M}{dt} = acs_M i_H - \mu i_M.
\end{cases}
\tag{3}
$$

To guarantee that solutions remain in the feasible domain, compared to the ODE model here we need the additional condition that the initial functions satisfy $e_H^l(0) \geq (1-p)abm \int_{-\tau}^0 s_H(u)i_M(u)e^{-\xi u}du$.

The basic reproduction number R_d of the DDE model is given by

$$
R_d = \sqrt{ \frac{a^2 bcm}{\mu(r+\xi)} \left((1-p)e^{-\xi\tau} + p\frac{d_s}{d_s+\xi} \right) },
\tag{4}
$$

being defined in the same manner as R_o. Comparing with Eq. (2), the term $(1-p)e^{-\xi\tau} + p\frac{d_s}{d_s+\xi}$ is the only different part, which is the probability that a human will survive the exposed state to become infectious. R_d is a stability threshold of system (3).

Lemma 2.2. *The disease free equilibrium (DFE) $(1,0,0,0,0,1,0)$ of system (3) always exists. An endemic equilibrium (EE) exists if and only if $R_d > 1$ and it is given by the following relations:*

$$
i_H^* = \frac{R_d^2 - 1}{\frac{ac}{\mu} + K_d R_d^2}, \quad
e_H^{s*} = \frac{p\xi}{(1-p)(d_s+\xi)e^{-\xi\tau} + pd_s} \frac{r+\xi}{\xi} i_H^*,
$$

$$
e_H^{l*} = \frac{(1-p)(d_s+\xi)(1-e^{-\xi\tau})}{p\xi} e_H^{s*}, \quad
r_H^* = \frac{r}{\omega+\xi} i_H^*, \quad
i_M^* = \frac{\frac{ac}{\mu} i_H^*}{1 + \frac{ac}{\mu} i_H^*},
$$

$$
s_H^* = 1 - e_H^{s*} - e_H^{l*} - i_H^* - r_H^* \quad and \quad s_M^* = 1 - i_M^*, \quad where
$$

$$
K_d = \frac{\frac{p}{d_s+\xi} + (1-p)\frac{1-e^{-\xi\tau}}{\xi}}{p\frac{d_s}{d_s+\xi} + (1-p)e^{-\xi\tau}}(r+\xi) + 1 + \frac{r}{\omega+\xi}.
$$

Proof. See Appendix B. □

Theorem 2.2. *The DFE of system* (3) *is locally asymptotically stable if* $R_d < 1$ *and is unstable if* $R_d > 1$. *The EE is locally asymptotically stable whenever exists, i.e. if* $R_d > 1$.

Proof. See Appendix D. □

3. Results

3.1. *Comparison of models*

In Section 2, we have shown that for both models there exists a threshold value determining the existence and stability of equilibria. Now we compare these threshold values and also the endemic equilibria of the two models.

Proposition 3.1. *When all parameters are fixed, the basic reproduction number* R_o *of the ODE model is greater than the basic reproduction number* R_d *of the DDE model. Moreover, when* $R_d > 1$, i^*_H *of the ODE model (denoted by* i_o) *is greater than* i^*_H *of the DDE model (denoted by* i_d).

Proof. Comparing (2) and (4), we obtain

$$R_o = \sqrt{\frac{a^2bcm}{(r+\xi)\mu}\left(p\frac{d_s}{d_s+\xi}+(1-p)\frac{1}{1+\xi\tau}\right)}$$

$$> \sqrt{\frac{a^2bcm}{(r+\xi)\mu}\left(p\frac{d_s}{d_s+\xi}+(1-p)\frac{1}{e^{\xi\tau}}\right)} = R_d,$$

because $e^{\xi\tau} > 1 + \xi\tau$. To compare the equilibria, consider

$$i_o - i_d = \frac{(R_o^2-1)\left(\frac{ac}{\mu}+K_dR_d^2\right)-(R_d^2-1)\left(\frac{ac}{\mu}+K_oR_o^2\right)}{\left(\frac{ac}{\mu}+K_oR_o^2\right)\left(\frac{ac}{\mu}+K_dR_d^2\right)}$$

$$= \frac{\frac{ac}{\mu}(R_o^2-R_d^2)+(K_d-K_o)R_o^2R_d^2}{\left(\frac{ac}{\mu}+K_oR_o^2\right)\left(\frac{ac}{\mu}+K_dR_d^2\right)}.$$

Since $R_o > R_d$, it is sufficient to show that $K_d > K_o$. Since $e^{\xi\tau} > 1 + \xi\tau$, $e^{-\xi\tau} < \frac{1}{1+\xi\tau}$. Recall that $\tau = 1/d_l$, hence

$$e^{-\xi\tau} < \frac{d_l}{d_l+\xi}. \tag{5}$$

Table 2.   Baseline parameter values for simulations.

parameter	value	ref.
ξ	0.00004	human life span
μ	0.2 [0.1,0.24]	[11]
b	0.5	[6]
c	0.5	[6]
a	0.3 [0.25,0.5]	[12]
d_s	0.04	[8,9]
d_l	0.003	[8,9]
r	0.07 [0.005,0.5]	[13,12,14]
p	0.25	[15]
ω	1/365	
P	365	
L	365/2	
a_s	0.3	

Thus, $1 - e^{-\xi\tau} > 1 - \frac{1}{1+\xi\tau} = \frac{\xi}{d_l+\xi}$, implying

$$\frac{1 - e^{-\xi\tau}}{\xi} > \frac{1}{d_l + \xi}. \tag{6}$$

By (5) and (6), we find

$$\frac{\frac{p}{d_s+\xi} + (1-p)\frac{1-e^{-\xi\tau}}{\xi}}{p\frac{d_s}{d_s+\xi} + (1-p)e^{-\xi\tau}} > \frac{\frac{p}{d_s+\xi} + \frac{1-p}{d_l+\xi}}{p\frac{d_s}{d_s+\xi} + (1-p)\frac{d_l}{d_l+\xi}}$$

which is equivalent to $K_d > K_o$.   □

In conclusion, the ODE model gives a larger basic reproduction number than the DDE model, because of the higher probability of surviving the incubation period. Fig. 4 and Fig. 5 show numerical solutions. Fig. 4 shows the case $R_o > 1$ and $R_d > 1$, when $i_H(t)$ converges to the endemic equilibrium for both models. Fig. 5 shows a particular case when $R_o > 1$ but $R_d < 1$. Despite that all parameters are the same, here $i_H(t)$ of ODE model converges to the endemic equilibrium, and $i_H(t)$ of DDE model converges to disease free equilibrium, thus the two models provide very different predictions. To investigate the robustness of the basic reproduction number with respect to the long term incubation time, in Fig. 6 we compared

$$\frac{\partial R_o}{\partial \tau} = \frac{-1}{2R_o} \frac{a^2bcm}{\mu(r+\xi)} \frac{(1-p)\xi}{(1+\xi\tau)^2} \quad \text{and} \quad \frac{\partial R_d}{\partial \tau} = \frac{-1}{2R_d} \frac{a^2bcm}{\mu(r+\xi)}(1-p)\xi e^{-\xi\tau}.$$

The magnitude of $\frac{\partial R_o}{\partial \tau}$ is smaller than $\frac{\partial R_d}{\partial \tau}$ when τ is in the reasonable range, so the basic reproduction number is more sensitive to the long term incubation time in the DDE model.
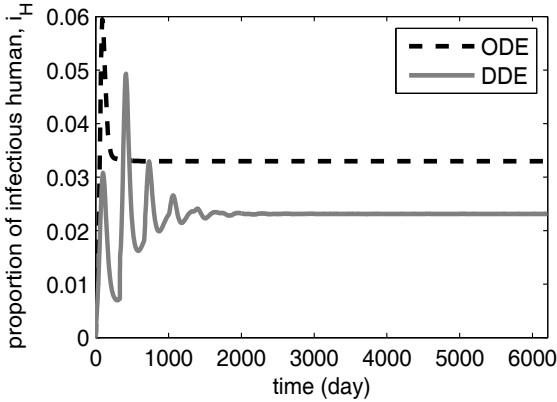
Figure 4. $R_o > 1$ and $R_d > 1$. For both models, $i_H(t)$ converges to endemic equilibrium with $i_o > i_d$. To clearly show the difference of i_o and i_d, we set $\xi = 0.004$ and $m = 10$. Other parameter values are as indicated in Table 2. Initial condition for ODE model is $(s_H, e_H^s, e_H^l, i_H, r_H, s_M, i_M)(0) = (1, 0, 0, 0, 0, 0.09, 0.01)$. For sake of convenience, initial condition of DDE model is set to be $s_H(t) = 1$ and $s_M(t) = 1$ for $t < 0$, introducing infectious mosquito at $t = 0$, $(s_H, e_H^s, e_H^l, i_H, r_H, s_M, i_M)(0) = (1, 0, 0, 0, 0, 0.09, 0.01)$.
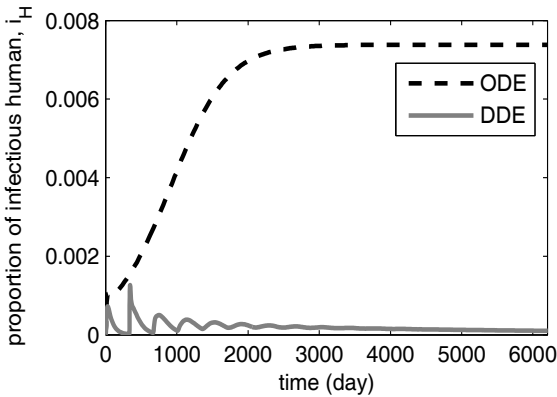
Figure 5. $R_o > 1$ and $R_d < 1$. For the ODE model, $i_H(t)$ converges to EE, while it converges to DFE for the DDE model. To compare with Fig. 4, we used parameter value $m = 1.5$. Other parameter values and initial condition are same as the one in Fig. 4.

3.2. Role of the mosquito population

In Fig. 7, the infectious human component of the endemic equilibrium is plotted for various mosquito populations. As expected from Proposition
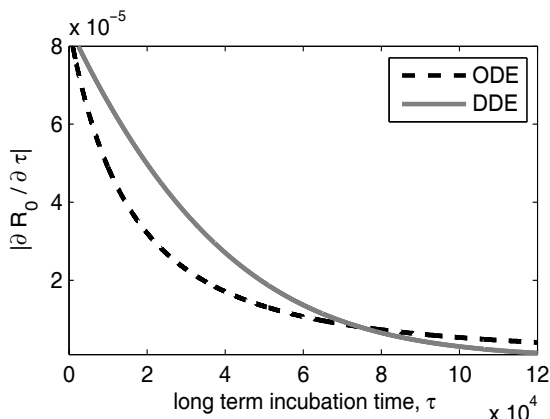
Figure 6. Sensitivity of the basic reproduction number to the long term incubation time. For τ being in reasonable range, $|\frac{\partial R_o}{\partial \tau}| < |\frac{\partial R_d}{\partial \tau}|$. Parameter values are as in Table 2 with $m = 20$.
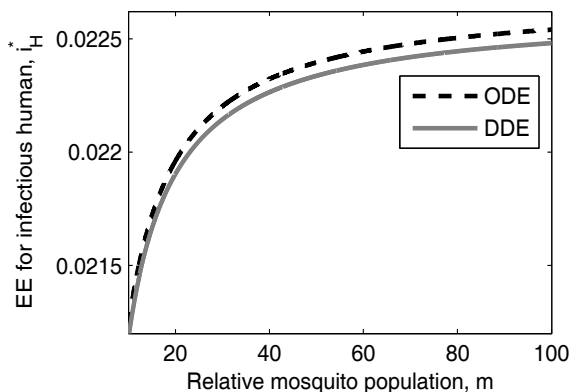

Figure 7. Relation of m and i_H^*. Change of i_H^* is more drastic at smaller mosquito population.

3.1, the infectious human equilibrium for the ODE model is greater than for the DDE model. Moreover, we can see that a small change in the mosquito population affects the level of endemicity more significantly when the mosquito population is relatively small.

3.3. Comparison with seasonality

In temperate regions, mosquito populations show huge seasonal variation, and so the transmission of *P. vivax* malaria is seasonal as well. To account
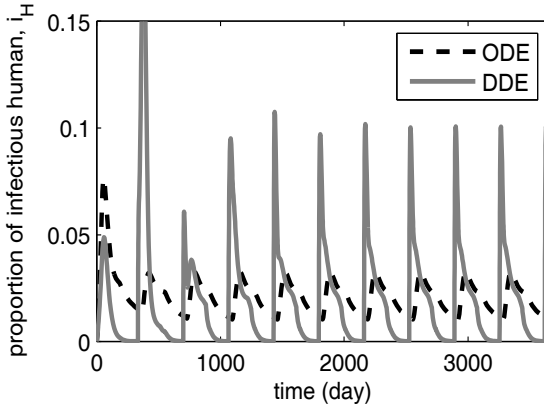
Figure 8. Dynamics of the infectious human population proportion with periodic biting rate. In both models, $i_H(t)$ converges to a periodic attractor. While the DDE model has a higher peak of infection, its annual average is smaller than that of the ODE model. Parameter values are as in Table 2 with $m = 20$. Initial condition is the same as the one used for Fig. 4.

for seasonality in a simplified way, a year is divided into a mosquito season, during which the parasite is transmitted via the mosquitoes, and an off-season, during which no new infection occurs. Let L be the length for a mosquito season and P be the natural period (one year). For the sake of simplicity, we incorporate temporal variation into the biting rate, thus replacing the constant a by

$$a(t) = \begin{cases} a_s & kP \leq t < kP + L, \\ 0 & kP + L \leq t < (k+1)P, \end{cases}$$

where k is an integer and $L \leq P$.

Numerical simulations are shown in Fig. 8, 9, 10 (compare to Fig. 4, 5). With such a periodic biting rate, $i_H(t)$ converges to a periodic attractor, instead of a steady state. Generally, the DDE model shows larger oscillations and predicts higher peaks and lower yearly bottoms of malaria prevalence. In contrast to the non-seasonal case, in some situations the DDE model even has higher annual average of infectious humans (Fig. 9) than its ODE counterpart. In addition, in some cases the DDE model predicts the persistence of the disease even though the infection dies out for the ODE model with the same parameter values (Fig. 10).
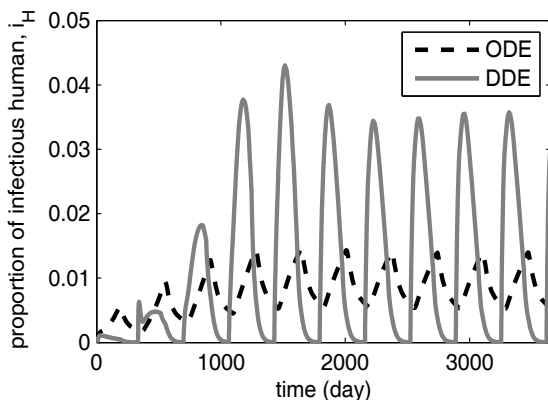
Figure 9. To compare with Fig. 9, we used the parameter value $m = 2$. Other parameter values and initial condition are the same as in Fig. 8. The DDE model has a higher peak of infection, just as in Fig. 9, however, its annual average is also greater than that of the ODE model.
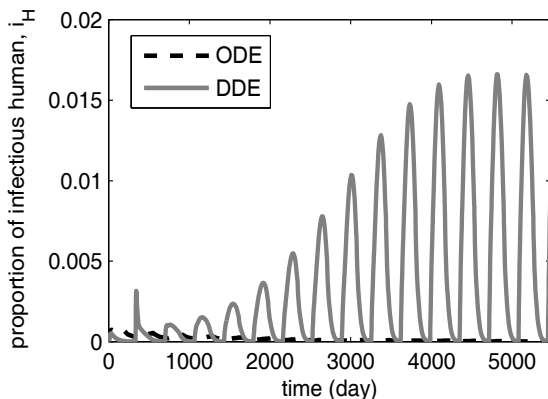

Figure 10. For comparison, we use $m = 1$. Here $i_H(t)$ dies out in the ODE model, however, it converges to a periodic attractor in the DDE model.

4. Conclusion

The exact mechanism governing the development of malaria parasites from dormancy to activation is not known [16]. Motivated by the empirical estimations of the incubation times in Korea, in this work we compared two models having different distribution of long term incubation time,
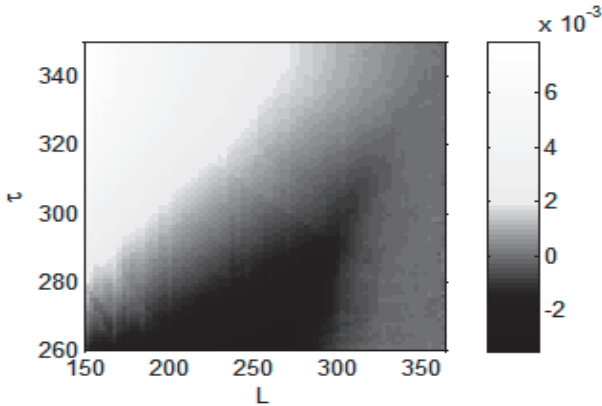
Figure 11. Differences between the yearly average number of infections for the ODE and the DDE model, as a function of length of incubation period τ and mosquito season length L. Black (white) areas represent a parameter region where the DDE (ODE) model predicts higher prevalence of malaria.

resulting an ODE and a DDE system. For both models we identified the basic reproduction number as a threshold value determining the stability of the disease free equilibrium and the existence of the endemic equilibrium. Having all parameters fixed, the exponential distribution assumption for the long term incubation (ODE model) predicts a greater basic reproduction number and higher level of endemicity than the constant length assumption (DDE model). Also, the DDE model generates more oscillatory behavior even without seasonal coefficients. As it was pointed out[17], if the inter-annual cycle of malaria is generated not only by seasonal change, but also by internal mechanisms, the baseline autonomous model should be able to produce oscillations itself. Given that the distribution generated by the constant length assumption approximates much better the empirically estimated distribution, than the exponential distribution assumption, we consider the DDE as a better model for the description of *P. vivax* malaria transmission dynamics, thus previous ODE-based *P. vivax* models are likely overestimate the basic reproduction number.

However, when seasonality is also included in the model, things become more complicated. It is known that periodic delay differential equations can produce unexpected behavior, such as resonances[18], and their dynamics is not completely understood even in the scalar case[19]. In the present situation, the time periodic DDE results larger annual oscillations than the ODE (i.e. higher peaks and lower bottoms); as expected in the case of time

delays. However, due to the complicated interplay of the time delay and periodicity, in some situations the DDE model predicts a higher number of infections throughout a year than the ODE, in contrast with the autonomous case. In other cases, the DDE predicts lower average prevalence (as in the autonomous case), depending on the particular choice of parameters. To illustrate this striking behavior, we prepared Fig. 11, where we plotted these domains on the $L - \tau$ parameter plane (length of mosquito season and long incubation period). The results stress the importance of future work incorporating both delay and seasonality into $P.\ vivax$ models in temperate regions.

Appendix A. Proof of Lemma 2.1

To find equilibria, we set the LHS of system (1) to zero, and obtain

$$0 = pabms_H^* i_M^* - (d_s + \xi)e_H^{s*}, \tag{A.1}$$

$$0 = (1 - p)abms_H^* i_M^* - (d_l + \xi)e_H^{l*}, \tag{A.2}$$

$$0 = d_s e_H^{s*} + d_l e_H^{l*} - (r + \xi)i_H^*, \tag{A.3}$$

$$0 = ri_H^* - \omega r_H^* - \xi r_H^*, \tag{A.4}$$

$$0 = acs_M^* i_H^* - \mu i_M^*. \tag{A.5}$$

If either $i_H^* = 0$ or $i_M^* = 0$, we have a DFE $(s_H^*, e_H^{s*}, e_H^{l*}, i_H^*, r_H^*, s_M^*, i_M^*) = (1, 0, 0, 0, 0, 1, 0)$. Consider the case $i_H^* > 0$ and $i_M^* > 0$. Adding three equations, (A.1) multiplied by $\frac{d_s}{\xi + d_s}$, (A.2) multiplied by $\frac{d_l}{\xi + d_l}$, and (A.3),

$$abm\left(p\frac{d_s}{\xi + d_s} + (1 - p)\frac{d_l}{\xi + d_l}\right)s_H^* i_M^* = (r + \xi)i_H^*. \tag{A.6}$$

By (A.5),

$$acs_M^* i_H^* = \mu i_M^*. \tag{A.7}$$

Multiplying each sides of (A.6) and (A.7), and dividing by $(r + xi)\mu i_H^* i_M^*$,

$$R_0^2 s_H^* s_M^* = 1. \tag{A.8}$$

Multiplying $\frac{ac}{(r+\xi)\mu}$ to (A.6) gives

$$R_o^2 s_H^*(1 - s_M^*) = \frac{ac}{\mu} i_H^*. \tag{A.9}$$

By (A.8) and (A.9), we get

$$s_H^* = \frac{1}{R_o^2} + \frac{ac}{R_o^2 \mu} i_H^*. \tag{A.10}$$

Comparing (A.1) and (A.6), we have

$$e_H^{s*} = abms_H^* i_M^* \frac{p}{d_s + \xi} = \frac{\frac{p}{\xi+d_s}(r+\xi)}{p\frac{d_s}{\xi+d_s} + (1-p)\frac{d_l}{\xi+d_l}} i_H^*. \tag{A.11}$$

Comparing (A.2) and (A.6), we have

$$e_H^{l*} = abms_H^* i_M^* \frac{1-p}{d_l + \xi} = \frac{\frac{1-p}{\xi+d_l}(r+\xi)}{p\frac{d_s}{\xi+d_s} + (1-p)\frac{d_l}{\xi+d_l}} i_H^*. \tag{A.12}$$

By (A.4),

$$r_H^* = \frac{r}{\omega + \xi} i_H^*. \tag{A.13}$$

By (A.11), (A.12) and (A.13),

$$\begin{aligned}
s_H^* &= 1 - e_H^{s*} - e_H^{l*} - i_H^* - r_H^* \\
&= 1 - \frac{\left(\frac{p}{\xi+d_s} + \frac{1-p}{\xi+d_l}\right)(r+\xi)}{p\frac{d_s}{\xi+d_s} + (1-p)\frac{d_l}{\xi+d_l}} i_H^* - i_H^* - \frac{r}{\omega + \xi} i_H^* \\
&= 1 - K_o i_H^*.
\end{aligned} \tag{A.14}$$

By (A.10) and (A.14), we get $i_H^* = \frac{R_o^2 - 1}{\frac{ac}{\mu} + K_o R_o^2}$, which exists when $R_o > 1$.

Appendix B. Proof of Lemma 2.2

We put LHS of the system (3) to be zero and obtain the following equations

$$0 = pabms_H^* i_M^* - (d_s + \xi)e_H^{s*}, \tag{B.1}$$

$$0 = (1-p)abms_H^* i_M^* - (1-p)abms_H^* i_M^* e^{-\xi\tau} - \xi e_H^{l*}, \tag{B.2}$$

$$0 = d_s e_H^{s*} + (1-p)abms_H^* i_M^* e^{-\xi\tau} - (r+\xi)i_H^*, \tag{B.3}$$

$$0 = ri_H^* - (\omega + \xi)r_H^*, \tag{B.4}$$

$$0 = acs_M^* i_H^* - \mu i_M^*. \tag{B.5}$$

If either $i^*_H = 0$ or $i^*_M = 0$, we have a DFE $(s^*_H, e^{s*}_H, e^{l*}_H, i^*_H, r^*_H, s^*_M, i^*_M) = (1, 0, 0, 0, 0, 1, 0)$. Consider the case $i^*_H > 0$ and $i^*_M > 0$. Adding two equations, (B.1) multiplied by $\frac{d_s}{\xi + d_s}$, and (B.3), one has

$$\left((1-p)e^{-\xi\tau} + p\frac{d_s}{\xi + d_s} \right) abms^*_H i^*_M = (r + \xi)i^*_H. \tag{B.6}$$

By (B.5),

$$acs^*_M i^*_H = \mu i^*_M. \tag{B.7}$$

Multiplying both sides of (B.6) and (B.7), and dividing by $(r + \xi)\mu i^*_H i^*_M$,

$$R^2_d s^*_H s^*_M = 1. \tag{B.8}$$

Meanwhile, multiplying $\frac{ac}{(r+\xi)\mu}$ to (B.6),

$$R^2_d s^*_H (1 - s^*_M) = \frac{ac}{\mu} i^*_H. \tag{B.9}$$

By (B.8) and (B.9), we get

$$s^*_H = \frac{1}{R^2_d} + \frac{ac}{R^2_d \mu} i^*_H. \tag{B.10}$$

Comparing (B.1) and (B.6), we have

$$e^{s*}_H = abms^*_H i^*_M \frac{p}{d_s + \xi} = \frac{\frac{p}{\xi + d_s}(r + \xi)}{(1-p)e^{-\xi\tau} + p\frac{d_s}{\xi + d_s}} i^*_H. \tag{B.11}$$

Comparing (B.2) and (B.6), we have

$$e^{l*}_H = abms^*_H i^*_M \frac{(1-p)(1 - e^{-\xi\tau})}{\xi} = \frac{\frac{(1-p)(1-e^{-\xi\tau})}{\xi}(r + \xi)}{(1-p)e^{-\xi\tau} + p\frac{d_s}{\xi + d_s}} i^*_H. \tag{B.12}$$

By (B.4),

$$r^*_H = \frac{r}{\omega + \xi} i^*_H. \tag{B.13}$$

By (B.11), (B.12) and (B.13),

$$\begin{aligned}
s^*_H &= 1 - e^{s*}_H - e^{l*}_H - i^*_H - r^*_H \\
&= 1 - \frac{\frac{p}{\xi + d_s} + (1-p)\frac{1 - e^{-\xi\tau}}{\xi}}{(1-p)e^{-\xi\tau} + p\frac{d_s}{\xi + d_s}}(r + \xi)i^*_H - i^*_H - \frac{r}{\omega + \xi} i^*_H \\
&= 1 - K_d i^*_H.
\end{aligned} \tag{B.14}$$

By (B.10) and (B.14), we get $i^*_H = \frac{R^2_d - 1}{\frac{ac}{\mu} + K_d R^2_d}$, which exists when $R_d > 1$.

Appendix C. Proof of Theorem 2.1

Consider the linearized system of (1) at an equilibrium:

$$
\begin{cases}
\dfrac{de^s_H}{dt} = pabms^*_H i_M - pabmi^*_M(e^s_H + e^l_H + i_H + r_H) - (d_s + \xi)e^s_H, \\[2mm]
\dfrac{de^l_H}{dt} = (1-p)abms^*_H i_M - (1-p)abmi^*_M(e^s_H + e^l_H + i_H + r_H) - (d_l + \xi)e^l_H, \\[2mm]
\dfrac{di_H}{dt} = d_s e^s_H + d_l e^l_H - (r + \xi)i_H, \\[2mm]
\dfrac{dr_H}{dt} = r i_H - (\omega + \xi)r_H, \\[2mm]
\dfrac{di_M}{dt} = acs^*_M i_H - aci^*_H i_M - \mu i_M.
\end{cases}
$$

The characteristic function $F(\lambda)$ is

$$
\begin{vmatrix}
\lambda + d_s + \xi + A & A & A & A & -pabms^*_H \\
B & \lambda + d_l + \xi + B & B & B & -(1-p)abms^*_H \\
-d_s & -d_l & \lambda + r + \xi & 0 & 0 \\
0 & 0 & -r & \lambda + \omega + \xi & 0 \\
0 & 0 & -acs^*_M & 0 & \lambda + \mu + aci^*_H
\end{vmatrix},
$$

where $A = pabmi^*_M$, $B = (1-p)abmi^*_M$. After simplification,

$$
\begin{aligned}
F(\lambda) = \quad & (\lambda + r + \xi)(\lambda + \omega + \xi)(\lambda + \mu + aci^*_H)[(\lambda + d_s + \xi)(\lambda + d_l + \xi) \\
& + abmi^*_M\{p(\lambda + d_s + \xi) + (1-p)(\lambda + d_l + \xi)\}] \\
& + \{-a^2 bcms^*_H s^*_M(\lambda + \omega + \xi) + abmi^*_M(\lambda + \omega + \xi)(\lambda + \mu + aci^*_H) \\
& + rabmi^*_M(\lambda + \mu + aci^*_H)\}\{(1-p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)\} \\
& + abmi^*_M(d_l - d_s)(\lambda + \mu + aci^*_H)(-1 + 2p)(\lambda + \omega + \xi)(\lambda + r + \xi).
\end{aligned}
$$

At the DFE, it reduces to

$$
\begin{aligned}
F(\lambda) = (\lambda + \omega + \xi)\{ & (\lambda + d_s + \xi)(\lambda + d_l + \xi)(\lambda + r + \xi)(\lambda + \mu) \\
& -(1-p)d_l a^2 bcm(\lambda + d_s + \xi) - pd_s a^2 bcm(\lambda + d_l + \xi)\}.
\end{aligned}
$$

Assume that $R_o < 1$. Suppose there exists a root of $F(\lambda) = 0$ with non-negative real part. Then,

$$
\left|\frac{\lambda}{d_s + \xi} + 1\right|\left|\frac{\lambda}{d_l + \xi} + 1\right|\left|\frac{\lambda}{r + \xi} + 1\right|\left|\frac{\lambda}{\mu} + 1\right|
$$

$$
\leq \frac{a^2 bcm}{(r + \xi)\mu}\left((1-p)\frac{d_l}{d_l + \xi}\left|\frac{\lambda}{d_s + \xi} + 1\right| + p\frac{d_s}{d_s + \xi}\left|\frac{\lambda}{d_l + \xi} + 1\right|\right)
$$

$$
\leq \left|\frac{\lambda}{d_s + \xi} + 1\right|\left|\frac{\lambda}{d_l + \xi} + 1\right|R_o^2,
$$

which contradicts to $R_o < 1$. Therefore, the roots of $F(\lambda) = 0$ have negative real part, implying that the DFE is locally asymptotically stable if $R_o < 1$.

Now, assume $R_o > 1$. Note that $F(\lambda) = 0$ has at least one real root. Since $F(\lambda) \to \infty$ for real $\lambda \to \infty$ and

$$F(0) = (\omega + \xi)(d_s + \xi)(d_l + \xi)(r + \xi)\mu(1 - R_o^2) < 0,$$

$F(\lambda) = 0$ has a positive real root. Therefore, the DFE is unstable.

Suppose that the EE is not LAS. Then there exists a characteristic root λ with nonnegative real part. For the EE, $s_H^* s_M^* = 1/R_o^2$ and the characteristic equation can be re-written as

$$\frac{a^2 bcm}{R_o^2}(\lambda + \omega + \xi)\{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)\}$$
$$= (\lambda + r + \xi)(\lambda + \omega + \xi)(\lambda + \mu + aci_H^*)[(\lambda + d_s + \xi)(\lambda + d_l + \xi)$$
$$+ abmi_M^*\{p(\lambda + d_s + \xi) + (1 - p)(\lambda + d_l + \xi)\}] + abmi_M^*(\lambda + \mu + aci_H^*)$$
$$\{(\lambda + \omega + \xi) + r\}\{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)\}.$$

Suppose there exists a root of the equation with nonnegative real part. Dividing by $\{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)\}(r + \xi)\mu(\omega + \xi)$ gives

$$\left|\frac{1}{p\frac{d_s}{d_s+\xi} + (1 - p)\frac{d_l}{d_l+\xi}}\right|\left|\frac{\lambda}{\omega + \xi} + 1\right|$$
$$= \left|\frac{\lambda}{\mu} + 1 + \frac{aci_H^*}{\mu}\right|\left|\left(\frac{\lambda}{r + \xi} + 1\right)\left(\frac{\lambda}{\omega + \xi} + 1\right)\right.$$
$$\times \frac{(\lambda + d_s + \xi)(\lambda + d_l + \xi)}{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)}$$
$$+ abmi_M^*\left(\frac{\lambda}{r + \xi} + 1\right)\left(\frac{\lambda}{\omega + \xi} + 1\right)\frac{p(\lambda + d_s + \xi) + (1 - p)(\lambda + d_l + \xi)}{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)}$$
$$\left. + \frac{abmi_M^*}{r + \xi}\left(\frac{\lambda}{\omega + \xi} + 1\right) + \frac{rabmi_M^*}{(r + \xi)(\omega + \xi)}\right|$$
$$\geq \left|\frac{\lambda}{\omega + \xi} + 1\right|\left|\frac{(\lambda + d_s + \xi)(\lambda + d_l + \xi)}{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)}\right|.$$

This implies

$$p\frac{d_s}{d_s + \xi} + (1 - p)\frac{d_l}{d_l + \xi} \leq \left|\frac{(1 - p)d_l(\lambda + d_s + \xi) + pd_s(\lambda + d_l + \xi)}{(\lambda + d_s + \xi)(\lambda + d_l + \xi)}\right|$$
$$= \left|(1 - p)\frac{d_l}{d_l + \xi}\frac{1}{\frac{\lambda}{d_l+\xi} + 1} + p\frac{d_s}{d_s + \xi}\frac{1}{\frac{\lambda}{d_s+\xi} + 1}\right|$$
$$< (1 - p)\frac{d_l}{d_l + \xi}\frac{1}{\left|\frac{\lambda}{d_s+\xi} + 1\right|} + p\frac{d_s}{d_s + \xi}\frac{1}{\left|\frac{\lambda}{d_l+\xi} + 1\right|},$$

which is a contradiction, as one can check that 0 is not a root of $F(\lambda)$. Hence, the EE is LAS.

Appendix D. Proof of Theorem 2.2

Consider the linearized system of (3) at an equilibrium:

$$
\begin{cases}
\dfrac{de^s_H}{dt} = & -pabmi^*_M(e^s_H(t) + e^l_H(t) + i_H(t) + r_H(t)) + pabms^*_H i_M(t) \\
& - (d_s + \xi)e^s_H(t), \\[2mm]
\dfrac{de^l_H}{dt} = & -(1-p)abmi^*_M(e^s_H(t) + e^l_H(t) + i_H(t) + r_H(t)) + (1-p)abms^*_H i_M(t) \\
& + (1-p)abmi^*_M(e^s_H(t-\tau) + e^l_H(t-\tau) + i_H(t-\tau) + r_H(t-\tau))e^{-\xi\tau} \\
& - (1-p)abms^*_H i_M(t-\tau)e^{-\xi\tau} - \xi e^l_H(t), \\[2mm]
\dfrac{di_H}{dt} = & -(1-p)abmi^*_M(e^s_H(t-\tau) + e^l_H(t-\tau) + i_H(t-\tau) + r_H(t-\tau))e^{-\xi\tau} \\
& + d_s e^s_H(t) + (1-p)abms^*_H i_M(t-\tau)e^{-\xi\tau} - ri_H(t) - \xi i_H(t), \\[2mm]
\dfrac{dr_H}{dt} = & ri_H(t) - \omega r_H(t) - \xi r_H(t), \\[2mm]
\dfrac{di_M}{dt} = & acs^*_M i_H(t) - aci^*_H i_M(t) - \mu i_M(t).
\end{cases}
$$

The characteristic function $F(\lambda)$ is the determinant of the matrix

$$
\lambda I + \begin{pmatrix}
d_s + \xi + A & A & A & A & -pabms^*_H \\
B & \xi + B & B & B & -(1-p)abms^*_H(1 - e^{-\xi\tau - \lambda\tau}) \\
-d_s + C & C & r + \xi + C & C & -(1-p)abms^*_H e^{-\xi\tau - \lambda\tau} \\
0 & 0 & -r & \omega + \xi & 0 \\
0 & 0 & -acs^*_M & 0 & \mu + aci^*_H
\end{pmatrix},
$$

where $A = pabmi^*_M$, $B = (1-p)abmi^*_M(1 - e^{-\xi\tau - \lambda\tau})$, and $C = (1-p)abmi^*_M e^{-\xi\tau - \lambda\tau}$. After simplification,

$$
\begin{aligned}
F(\lambda) = & (\lambda + \mu + aci^*_H)(\lambda + \omega + \xi)(\lambda + \xi + abmi^*_M)(\lambda + d_s + \xi)(\lambda + r + \xi) \\
& -\omega rabmi^*_M\{pd_s + (1-p)e^{-\xi\tau - \lambda\tau}(\lambda + d_s + \xi)\}(\lambda + \mu + aci^*_H) \\
& -a^2 bcms^*_H s^*_M\{pd_s + (1-p)e^{-\xi\tau - \lambda\tau}(\lambda + d_s + \xi)\}(\lambda + \omega + \xi)(\lambda + \xi).
\end{aligned}
$$

At the DFE, it reduces to

$$
\begin{aligned}
F(\lambda) = & (\lambda + \omega + \xi)(\lambda + \xi)\{(\lambda + d_s + \xi)(\lambda + r + \xi)(\lambda + \mu) \\
& -(\lambda + d_s + \xi)(1-p)a^2 bcme^{-\xi\tau - \lambda\tau} - pa^2 bcmd_s\}.
\end{aligned}
$$

Assume that $R_d < 1$. Suppose there exists a root for $F(\lambda) = 0$ with non-negative real part. Then,

$$|(\lambda+d_s+\xi)(\lambda+\mu)(\lambda+r+\xi)| = |(\lambda+d_s+\xi)(1-p)a^2bcme^{-\xi\tau-\lambda\tau}+pa^2bcmd_s|.$$

Dividing both sides by $(d_s + \xi)\mu(r + \xi)$ gives

$$
\left|\frac{\lambda}{d_s + \xi} + 1\right|\left|\frac{\lambda}{\mu} + 1\right|\left|\frac{\lambda}{r+\xi} + 1\right|
$$

$$
= \frac{a^2bcm}{\mu(r + \xi)}\left|\left(\frac{\lambda}{d_s + \xi} + 1\right)(1 - p)e^{-\xi\tau-\lambda\tau} + p\frac{d_s}{d_s + \xi}\right|
$$

$$
\leq \left|\frac{\lambda}{d_s + \xi} + 1\right| R_d^2,
$$

which contradicts to $R_d < 1$. Therefore, every root of $F(\lambda) = 0$ has negative real part, implying the DFE is locally asymptotically stable if $R_d < 1$.

Now assume $R_d > 1$. Note that $F(\lambda) = 0$ has at least one real root. Since $F(\lambda) \to \infty$ for real $\lambda \to \infty$ and

$$F(0) = (d_s + \xi)\mu(r + \xi)(1 - R_d^2) < 0,$$

$F(\lambda) = 0$ has a positive real root. Therefore the DFE is unstable.

Suppose that the EE is not LAS. Then there exists a characteristic root λ with nonnegative real part. For the EE, $s_H^* s_M^* = 1/R_d^2$ holds and the characteristic equation can be re-written as

$$
\mu(r + \xi)\frac{pd_s + (1 - p)e^{-\xi\tau-\lambda\tau}(\lambda + d_s + \xi)}{(1 - p)e^{-\xi\tau} + p\frac{d_s}{d_s+\xi}}(\lambda + \omega + \xi)(\lambda + \xi)
$$

$$
= (\lambda + \mu + aci_H^*)(\lambda + \omega + \xi)(\lambda + \xi + abmi_M^*)(\lambda + d_s + \xi)(\lambda + r + \xi)
$$

$$
- \omega rabmi_M^*\{pd_s + (1 - p)e^{-\xi\tau-\lambda\tau}(\lambda + d_s + \xi)\}(\lambda + \mu + aci_H^*).
$$

Dividing both sides by $\mu(r + \xi)(d_s + \xi)(\omega + \xi)\xi$ gives

$$
\frac{p\frac{d_s}{d_s+\xi} + (1 - p)e^{-\xi\tau-\lambda\tau}(\frac{\lambda}{d_s+\xi} + 1)}{(1 - p)e^{-\xi\tau} + p\frac{d_s}{d_s+\xi}}\left(\frac{\lambda}{\omega + \xi} + 1\right)\left(\frac{\lambda}{\xi} + 1\right)
$$

$$
= \left(\frac{\lambda}{\mu} + 1 + \frac{aci_H^*}{\mu}\right)\left[\left(\frac{\lambda}{\omega + \xi} + 1\right)\left(\frac{\lambda}{\xi} + 1 + \frac{abmi_M^*}{\xi}\right)\left(\frac{\lambda}{d_s + \xi} + 1\right)\right.
$$

$$
\times \left(\frac{\lambda}{r + \xi} + 1\right)
$$

$$
\left. - \frac{\omega}{\omega + \xi}\frac{r}{r + \xi}\frac{abmi_M^*}{\xi}\left\{p\frac{d_s}{d_s + \xi} + (1 - p)e^{-\xi\tau-\lambda\tau}\left(\frac{\lambda}{d_s + \xi} + 1\right)\right\}\right].
$$

Then,

$$\left|\frac{\lambda}{\mu}+1+\frac{aci_H^*}{\mu}\right|\left|\frac{\lambda}{\omega+\xi}+1\right|\left|\frac{\lambda}{\xi}+1+\frac{abmi_M^*}{\xi}\right|\left|\frac{\lambda}{d_s+\xi}+1\right|\left|\frac{\lambda}{r+\xi}+1\right|$$

$$=\left|\frac{p\frac{d_s}{d_s+\xi}+(1-p)e^{-\xi\tau-\lambda\tau}(\frac{\lambda}{d_s+\xi}+1)}{(1-p)e^{-\xi\tau}+p\frac{d_s}{d_s+\xi}}\left(\frac{\lambda}{\omega+\xi}+1\right)\left(\frac{\lambda}{\xi}+1\right)\right.$$

$$\left.+\frac{\omega}{\omega+\xi}\frac{r}{r+\xi}\frac{abmi_M^*}{\xi}\left\{p\frac{d_s}{d_s+\xi}+(1-p)e^{-\xi\tau-\lambda\tau}\left(\frac{\lambda}{d_s+\xi}+1\right)\right\}\right.$$

$$\left.\times\left(\frac{\lambda}{\mu}+1+\frac{aci_H^*}{\mu}\right)\right|$$

$$=\left|\frac{p\frac{d_s}{d_s+\xi}+(1-p)e^{-\xi\tau-\lambda\tau}(\frac{\lambda}{d_s+\xi}+1)}{(1-p)e^{-\xi\tau}+p\frac{d_s}{d_s+\xi}}\right|\left|\left(\frac{\lambda}{\omega+\xi}+1\right)\left(\frac{\lambda}{\xi}+1\right)\right.$$

$$\left.+\frac{\omega}{\omega+\xi}\frac{r}{r+\xi}\frac{abmi_M^*}{\xi}\left\{(1-p)e^{-\xi\tau}+p\frac{d_s}{d_s+\xi}\right\}\left(\frac{\lambda}{\mu}+1+\frac{aci_H^*}{\mu}\right)\right|$$

$$\leq\left|\frac{\lambda}{d_s+\xi}+1\right|\left|\left(\frac{\lambda}{\omega+\xi}+1\right)\left(\frac{\lambda}{\xi}+1\right)\right.$$

$$\left.+\frac{\omega}{\omega+\xi}\frac{r}{r+\xi}\frac{abmi_M^*}{\xi}\left\{(1-p)e^{-\xi\tau}+p\frac{d_s}{d_s+\xi}\right\}\left(\frac{\lambda}{\mu}+1+\frac{aci_H^*}{\mu}\right)\right|$$

$$\leq\left|\frac{\lambda}{d_s+\xi}+1\right|\left(\left|\frac{\lambda}{\omega+\xi}+1\right|\left|\frac{\lambda}{\xi}+1\right|+\frac{abmi_M^*}{\xi}\left|\frac{\lambda}{\mu}+1+\frac{aci_H^*}{\mu}\right|\right)$$

$$<\left|\frac{\lambda}{d_s+\xi}+1\right|\left|\frac{\lambda}{\omega+\xi}+1\right|\left|\frac{\lambda}{\xi}+1\right|\left|\frac{\lambda}{\mu}+1+\frac{aci_H^*}{\mu}\right|\left(1+\frac{abmi_M^*}{\xi}\right)$$

which is a contradiction, hence the EE is LAS.

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