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ORIGINAL ARTICLE

Analysis of a Vector-Bias Model on Malaria Transmission

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Abstract We incorporate a vector-bias term into a malaria-transmission model to account for the greater attractiveness of infectious humans to mosquitoes in terms of differing probabilities that a mosquito arriving at a human at random picks that human depending on whether he is infectious or susceptible. We prove that transcritical bifurcation occurs at the basic reproductive ratio equalling 1 by projecting the flow onto the extended centre manifold. We next study the dynamics of the system when incubation time of malaria parasites in mosquitoes is included, and find that the longer incubation time reduces the prevalence of malaria. Also, we incorporate a random movement of mosquitoes as a diffusion term and a chemically directed movement of mosquitoes to humans expressed in terms of sweat and body odour as a chemotaxis term to study the propagation of infected population to uninfected population. We find that a travelling wave occurs; its speed is calculated numerically and estimated for the lower bound analytically.

Keywords Malaria transmission · Time delay · Travelling waves

1 Introduction

Malaria is a mosquito-borne disease caused by protozoan parasites in the genus *Plasmodium*. It is one of the most common infectious disease, kills over a million people a year, causes public health problems, and dampens the economics (Snow et al. 2005; Guerra et al. 2006). The endemic areas are Africa, Asia, and South America. Five species of *Plasmodium* parasites infect humans: (1) *P. falciparum*, (2) *P. vivax*,

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(3) *P. ovale*, (4) *P. malariae*, and (5) *P. knowlesi*. Clinically, *P. falciparum* is malignant while the others are benign. Their common symptoms include a cyclical fever, headache, shivering, and vomiting, for instance. Infection with *P. falciparum* can lead to other complications such as severe anaemia, acute respiratory distress syndrome, kidney failure, and cerebral malaria.

The life cycle of malaria starts when female *Anopheles* mosquitoes successfully inject sporozoites of the parasites from their salivary gland into human during their blood meal. The sporozoites undergo certain developmental stages in the hepatocytes and finally appear in the human bloodstream in the form of merozoites, some of which are transformed into male and female gametocytes that are ingested by mosquitoes during their blood meal (Doolan et al. 2009). In mosquitoes, after the ingestion, those gametocytes multiply themselves to produce sporozoites that makes their way to the salivary gland. The cycle is completed by the inoculation of the sporozoites into new hosts.

A mathematical model for malaria transmission was first introduced by Ross (1916) and further extended by Macdonald (1952, 1957). Models with acquired immunity were studied later by Dietz et al. (1974), Bailey (1975) and Aron (1988). Because immunity to malaria is not fully acquired and declines with time, without new exposures individuals may lose immune memory and become infected again (Doolan et al. 2009). Hence, we use the SIS model to describe the dynamics of malaria in a human population. For a mosquito population, we use the SI model under the assumption that mosquitoes do not recover from malaria parasites and also the malaria parasites do not harm them. This type of model is based on the Ross–Macdonald model (Macdonald 1957).

Several blood-seeking mosquitoes search for their meal by the use of host odours, breath, and sweat (Costantini et al. 1996; Takken and Knols 1999; Mukabana et al. 2004). Moreover, Lacroix et al. (2005) show that malaria parasites manipulate a host to be more attractive to mosquitoes via those chemical substances. A general framework on the manipulation driven by the parasites in the predator-prey community and expressed via the predator's functional response (such as foraging efficacy) is further studied by Fenton and Rands (2006). A model that takes account of this manipulation in malaria transmission is primarily introduced by Kingsolver (1987). It was later extended to include the incubation time in mosquitoes. Here, we express the model in a different way from previous authors and study the dynamical behaviours of the model in detail. Also, in our work, we include the study of some behavioural factors in mosquitoes such as the incubation time of the parasites, the mosquito's movement, and chemically directed movement to humans.

This article is organised as follows: (1) formulation of the model, (2) stability analysis of the basic model, (3) bifurcation analysis (a proof of transcritical bifurcation), (4) a model incorporating incubation time in mosquitoes, (5) the study of travelling waves, and lastly (6) conclusion and discussion.

2 A Vector-Bias Model

A vector-bias model in malaria was first introduced by Kingsolver (1987). The model is extended from the Ross–Macdonald model to account for the greater attractiveness

of infectious humans to mosquitoes (Macdonald 1952, 1957). Following Kingsolver's work, Hosack et al. (2008) incorporate an extrinsic incubation time in mosquitoes to study the dynamics of the disease in term of a reproduction number. Here, we include the recruitment rate and the natural death rate and define the attractiveness in a different way. In the model, hosts might get repeatedly infected due to not acquiring complete immunity so the population is assumed to be described by the SIS model. Mosquitoes are assumed not to recover from the parasites and the parasites are not harmful to them so the mosquito population can be described by the SI model. The model takes the form:

$$\begin{aligned}
\frac{dS}{dt} &= \mu N - \beta \frac{lS}{pI + lS} V - \mu S + \nu I, \\
\frac{dI}{dt} &= \beta \frac{lS}{pI + lS} V - (\mu + \nu) I, \\
\frac{dU}{dt} &= \eta M - \alpha \frac{pI}{pI + lS} U - \eta U, \\
\frac{dV}{dt} &= \alpha \frac{pI}{pI + lS} U - \eta V,
\end{aligned} \tag{1}$$

where S, I, U, V, N, and M represent the number of susceptible humans, infectious humans, susceptible mosquitoes, infectious mosquitoes, the total size of the human population, and the total size of the mosquito population, respectively, $\beta = bp_h$ and $\alpha = bp_v$. The description of the parameters can be found in Table 1. From the model, we assume that searching for a blood meal is equally likely to arrive at any human in the population, but bites that human with probability p if the human is infectious, l if the human is susceptible. Otherwise, the mosquito arrives at another human at random. Hence, the probability that a mosquito picks the first human and the first human is infectious is (pI/N). The probability that a mosquito picks the first human and the first human is susceptible is (lS/N). Hence, the probability that the first human is infectious under the condition that a mosquito picks him is $pI/(pI + lS)$. The probability that the first human is susceptible under the condition that a mosquito picks him is $lS/(pI + lS)$. Similar arguments hold for all subsequent humans. At $l = p$, the model is without vector-bias. Since infectious individuals are more attractive to the mosquitoes (Lacroix et al. 2005), $p > l$. It can be seen that a primary case in the human population makes infectious contacts with mosquitoes at rate $\alpha p M \frac{1}{lN}$ for an expected time $1/(\mu + \nu)$ and a primary case in the mosquito population makes infectious contacts with humans at rate $\beta \frac{lN}{lN}$ for an expected time $1/\eta$. Hence, the basic reproductive ratio is

$$R_0 = \frac{\beta \alpha p q}{l \eta (\mu + \nu)}.$$

Note that some authors define R_0 to be the square root of this expression.

3 Stability Analysis

We introduce the new variables in term of proportions as follows:

$$s = \frac{S}{N}, \qquad i = \frac{I}{N}, \qquad u = \frac{U}{M}, \qquad v = \frac{V}{M}.$$

Table 1 Lists of parameters for malaria transmission

Parameter	Description	Value	References
β	Transmission rate in humans	bp_h	–
α	Transmission rate in mosquitoes	bp_m	–
N	The total size of human population (per 40000 km^2) (5 persons/km^2)	200000	Estimated
M	The total size of mosquito population	qN	
μ	Birth and death rate	1/70 (year^{-1})	Estimated
b	Biting rate	100–182 (year^{-1})	Gupta et al. (1994)
p_h	Probability of successful infection in humans	0.1	Gupta et al. (1994)
p_m	Probability of successful infection in mosquitoes	0.3–0.4	Drakeley et al. (2006)
ν	Recovery rate	365/180 (year^{-1})	Fillipe et al. (2007)
η	Natural birth and death rate of mosquitoes	365/20 (year^{-1})	Anderson and May (1992)
q	The number of mosquitoes per individual	1–2	Gupta et al. (1994)
p	Probability that a mosquito arrives at human at random and picks that human if he is infectious	0–1	Varying
l	Probability that a mosquito arrives at human at random and picks that human if he is susceptible ($p > l$)	0–1	Varying
D	Diffusion rate of mosquitoes from $D = 1.25 \times 10^{-2}$ (km^2/day)	4.6 (km^2/year)	Maidana and Yang (2008), Lewis et al. (2006)

Since $s + i = 1$, $u + v = 1$, system (1) is reduced to

$$\begin{aligned} \frac{di}{dt} &= \beta q \frac{l(1-i)}{pi + l(1-i)} v - (\mu + \nu) i, \\ \frac{dv}{dt} &= \alpha \frac{pi}{pi + l(1-i)} (1 - v) - \eta v. \end{aligned} \tag{2}$$

It has two steady states which are

1. the disease-free steady state

$$(i, v) = \left(i^0, v^0\right) = (0, 0), \tag{3}$$

2. the disease-present steady state

$$(i, v) = \left(i^*, v^*\right) = \left(i^*, \frac{\alpha p i^*}{\eta(pi + l(1 - i^*)) + \alpha p i^*}\right), \tag{4}$$

where i^* satisfies the equation

$$a_2 i^{*2} + a_1 i^* + a_0 = 0 \tag{5}$$

with

$$a_2 = (\mu+\nu)(p-l)\big[\alpha p + \eta(p-l)\big],$$
$$a_1 = (\mu+\nu)l\big[\alpha p + 2\eta l(p-l)\big] + \beta\alpha q p l,$$
$$a_0 = (\mu+\nu)\eta l^2 - \beta\alpha q p l = l^2\eta(\mu+\nu)(1-R_0).$$

Because $p > l$, a_1 and a_2 are always positive and a_0 is positive when $R_0 < 1$, negative when $R_0 > 1$. Hence, if $R_0 < 1$, there is no positive root of (5), while if $R_0 > 1$ there is one positive and one negative root. If $R_0 > 1$, we define i^* to be the positive root of (5).

The linearised system at the disease-free steady state is given by

$$\begin{aligned} \frac{di}{dt} &= \beta q v - (\mu+\nu)i, \\ \frac{dv}{dt} &= \alpha\frac{p}{l}i - \eta v. \end{aligned} \tag{6}$$

Therefore, the characteristic equation is

$$\lambda^2 + (\mu+\nu+\eta)\lambda + \eta(\mu+\nu)(1-R_0) = 0.$$

Two eigenvalues are given by

$$\lambda_{1,2} = \frac{-(\mu+\nu+\eta) \pm \sqrt{(\mu+\nu+\eta)^2 - 4\eta(\mu+\nu)(1-R_0)}}{2}.$$

Both of them are negative whenever $R_0 < 1$. One is positive and one is negative when $R_0 > 1$. Hence, the disease-free steady state is stable if and only if $R_0 < 1$. The linearised system at the disease-present steady state is given by

$$\begin{aligned} \frac{di}{dt} &= -\left[\frac{\beta q l v^*}{(pi^* + l(1-i^*))} + \frac{\beta q l(p-l)(1-i^*)v^*}{(pi^* + l(1-i^*))^2} + (\mu+\nu)\right]i \\ &\quad + \frac{\beta q l(1-i^*)}{(pi^* + l(1-i^*))}v, \\ \frac{dv}{dt} &= \left[\frac{\alpha p(1-v^*)}{(pi^* + l(1-i^*))} - \frac{\alpha p(p-l)(1-v^*)i^*}{(pi^* + l(1-i^*))^2}\right]i \\ &\quad - \left[\frac{\alpha p i^*}{(pi^* + l(1-i^*))} + \eta\right]v. \end{aligned} \tag{7}$$

The system is unwieldy for finding explicit eigenvalues or stability conditions by the Routh–Hurwitz condition. However, at $R_0 = 1$, at the disease-free steady state, the system has two eigenvalues $\lambda_1 = -(\mu+\nu+\eta)$ and $\lambda_2 = 0$, and hence is non-hyperbolic, so that $R_0 = 1$ may be a bifurcation value. In the next section, we prove that a transcritical bifurcation occurs when $R_0 = 1$ by projecting the flow onto the extended centre manifold. This is one of the important techniques for studying bifurcations of non-linear systems. From the proof, we can consequently conclude that the disease-present steady state is stable if and only if $R_0 > 1$.

4 Bifurcation Analysis

In order to prove that the transcritical bifurcation occurs at $(R_0, (i, v)) = (1, (0, 0))$, we write βq in terms of R_0 and other parameters. So, linearised system (6) is now

$$\begin{aligned} \frac{di}{dt} &= \frac{R_0\eta(\mu+\nu)l}{\alpha p}v - (\mu+\nu)i, \\ \frac{dv}{dt} &= \alpha\frac{p}{l}i - \eta v. \end{aligned} \tag{8}$$

The proof is done by projecting the flow onto the extended centre manifold. It follows the similar steps with Glendinning (1999). The eigenvectors corresponding to the eigenvalues $\lambda_1 = -(\mu+\nu+\eta)$ and $\lambda_2 = 0$ when $R_0 = 1$ are

$$\bar{e}_1 = \begin{bmatrix} -l(\mu+\nu) \\ \alpha p \end{bmatrix}, \quad \text{and} \quad \bar{e}_2 = \begin{bmatrix} l\eta \\ \alpha p \end{bmatrix}, \quad \text{respectively.}$$

The matrix P with its column vector as the eigenvector is

$$P = \begin{bmatrix} -l(\mu+\nu) & l\eta \\ \alpha p & \alpha p \end{bmatrix},$$

so that

$$\begin{bmatrix} -(\mu+\nu) & \frac{l\eta(\mu+\nu)}{\alpha p} \\ \frac{\alpha p}{l} & -\eta \end{bmatrix} P = P \begin{bmatrix} -(\mu+\nu+\eta) & 0 \\ 0 & 0 \end{bmatrix}.$$

By setting

$$\begin{bmatrix} z \\ w \end{bmatrix} = P^{-1}\begin{bmatrix} i \\ v \end{bmatrix},$$

we obtain the linear part of the equation in terms of z and w as follows:

$$\begin{bmatrix} \dot{z} \\ \dot{w} \end{bmatrix} = \begin{bmatrix} -(\mu+\nu+\eta) & 0 \\ 0 & 0 \end{bmatrix}\begin{bmatrix} z \\ w \end{bmatrix}.$$

The inverse matrix of P is

$$P^{-1} = \frac{-1}{\alpha pl(\mu+\nu+\eta)}\begin{bmatrix} \alpha p & -l\eta \\ -\alpha p & -l(\mu+\nu) \end{bmatrix}.$$

Hence,

$$\begin{bmatrix} z \\ w \end{bmatrix} = P^{-1}\begin{bmatrix} i \\ v \end{bmatrix} = \frac{1}{\alpha pl(\mu+\nu+\eta)}\begin{bmatrix} -\alpha pi + l\eta v \\ \alpha pi + l(\mu+\nu)v \end{bmatrix}, \tag{9}$$

and

$$\begin{bmatrix} i \\ v \end{bmatrix} = P\begin{bmatrix} z \\ w \end{bmatrix} = \begin{bmatrix} -l(\mu+\nu)z + l\eta w \\ \alpha pz + \alpha pw \end{bmatrix}. \tag{10}$$

We define a new parameter r that $R_0 = 1 + r$ so that investigating the bifurcation at $R_0 = 1$ is equivalent to investigating it at $r = 0$. Hence, r can be treated as small as we want in the local investigation. By substituting di/dt and dv/dt from the system (2) (where βq is substituted by $R_0(1-p)\eta(\mu+\nu)/(\alpha p)$) and then i and v in terms of z and w into finding dz/dt and dw/dt obtained from (9), consequently, the extended system is

$$\begin{aligned}
\frac{dz}{dt} &= \frac{1}{(\mu+\nu+\eta)}\big\{-(\mu+\nu+\eta)^2 z + \eta(\mu+\nu)\big[\alpha p - (2p-l)(\mu+\nu)\big]z^2 \\
&\quad + \eta\big[p(\mu+\nu)(\eta-\mu-\nu) - \alpha p\eta + \alpha p(\mu+\nu) + 2(p-l)\eta(\mu+\nu)\big]zw \\
&\quad + \eta^2\big[p(\mu+\nu) - \alpha p - (p-l)\eta\big]w^2 - \eta(\mu+\nu)zr - \eta(\mu+\nu)wr\big\}, \\
\frac{dw}{dt} &= \frac{1}{(\mu+\nu+\eta)}\big\{(\mu+\nu)^2\big[p\eta + \alpha p - (p-l)(\mu+\nu)\big]z^2 \\
&\quad + (\mu+\nu)\big[-\alpha p\eta + \alpha p(\mu+\nu) + 2(p-l)\eta(\mu+\nu) - p\eta(\eta-\mu-\nu)\big]zw \\
&\quad - \eta(\mu+\nu)\big[p\eta + \alpha p + (p-l)\eta\big]w^2 + \eta(\mu+\nu)zr + \eta(\mu+\nu)wr\big\}, \\
\frac{dr}{dt} &= 0.
\end{aligned} \tag{11}$$

Note that in finding di/dt and dv/dt in terms of z and w, we make an approximation as follows:

$$\frac{1}{l+(p-l)i} \approx \frac{1}{l}\left(1 - \frac{(p-l)}{l}i + \text{h.o.t.}\right),$$

where $0 < (p-l)/l < 1$, $i < 1$, and i is in terms of z and w in (10). Because r is treated in the similar way with z and w, there is only a linear term in the equation dz/dt. The linear centre manifold is

$$E^c(0) = \big\{(z, w, r) | z = 0\big\}$$

and the linear stable manifold is

$$E^s(0) = \big\{(z, w, r) | w = r = 0\big\}.$$

An approximation of the nonlinear centre manifold is in the following form:

$$z = h(w, r) \quad \text{with } \frac{\partial h}{\partial w}(0,0) = \frac{\partial h}{\partial r}(0,0) = 0$$

and that

$$h(w, r) = aw^2 + bwr + r^2 + \cdots.$$

Thus,

$$\frac{dz}{dt} = \frac{dh}{dw}\frac{dw}{dt} + \frac{dh}{dr}\frac{dr}{dt}.$$

By substituting h, dw/dt, dr/dt in terms of w and r and then comparing the coefficients of w^2, wr, and r^2 with those from the equation dz/dt in (11) after the substitution, we obtain

$$\begin{aligned} a &= \frac{\eta^2}{(\mu+\nu+\eta)^2}\big[p(\mu+\nu)-\big(\alpha p+(p-l)\eta\big)\big], \\ b &= -\frac{\eta(\mu+\nu)}{(\mu+\nu+\eta)^2}, \\ c &= 0. \end{aligned}$$

Therefore, the centre manifold is

$$z = \frac{\eta^2}{(\mu+\nu+\eta)^2}\big[p(\mu+\nu)-\big(\alpha p+(p-l)\eta\big)\big]w^2 - \frac{\eta(\mu+\nu)}{(\mu+\nu+\eta)^2}wr + \text{cubic terms}.$$

We substitute z back into the equation of dw/dt to get the projection of the motion on the centre manifold onto the w axis:

$$\frac{dw}{dt} = \frac{\eta(\mu+\nu)}{(\mu+\nu+\eta)}\big[-\big(p\eta+\alpha p+(p-l)\eta\big)w^2+wr\big] + \text{cubic terms}.$$

Note here that the expression for the centre manifold does not contribute any quadratic terms so the calculation of the centre manifold is not exactly required. On the centre manifold, we have

$$\frac{dw}{dt} = G(w,r)$$

with

$$\begin{aligned} &G(0,0) = G_w(0,0) = G_r(0,0) = 0, \\ &G_{ww} = -\frac{2\eta(\mu+\nu)}{(\mu+\nu+\eta}\big[p\eta+\alpha p+(p-l)\eta\big], \quad G_{wr} = \frac{\eta(\mu+\nu)}{(\mu+\nu+\eta)}, \quad G_{rr} = 0. \end{aligned}$$

Since $p > l$ and all of the parameters are positive, G_{ww} is negative while G_{wr} is positive. By the centre manifold theorem and the transcritical bifurcation theorem, the disease-free steady state is stable when $R_0 < 1$ ($r < 0$) and there is a separate unstable branch (from the disease-present steady state), and when $R_0 > 1$ ($r > 0$) the disease-free steady state becomes unstable while the separating branch becomes stable (Glendinning 1999). Note that when $R_0 = 1$ ($r = 0$), the centre manifold is approximated by

$$\frac{dw}{dt} \approx -\frac{\eta(\mu+\nu)}{(\mu+\nu+\eta)}\big[p\eta+\alpha p+(p-l)\eta\big]$$

so that the disease-free steady state is stable if it is approached from $w > 0$ and unstable when it is approached from $w < 0$. The proof is completed. Hence, we conclude that there is a transcritical bifurcation occurring at the bifurcation value $R_0 = 1$. Therefore, the disease-free steady state is stable if and only if $R_0 < 1$ and the disease-present steady state is stable if and only if $R_0 > 1$.

5 Incubation Time in Mosquitoes

Because mosquitoes have a short lifespan, including the incubation time of malaria parasites in the model might be important in studying dynamic behaviours of the disease. It approximately takes $\tau = 10$ days after blood feeding for the sporozoites to appear in the mosquito salivary gland (Beier 1998) and for those 10 days the mosquito is infected but not infectious, and so it is in a latent state. For simplicity, we omit the incubation time in humans (≈5.5–15 days, a negligible fraction of a human lifetime) to study only incubation time for mosquitoes in the model. We assume that the mosquito that has just bitten the infectious human moves immediately to the latent compartment (W). As long as it still survives, it becomes infectious after duration τ and enters the infectious compartment (V). Hence, at time t, the flux into the latent compartment is $\alpha pi(t)u(t)/[l + (p - l)i(t)]$ and the flux out is $\alpha pi(t-\tau)u(t-\tau)e^{-\eta\tau}/[l + (p - l)i(t-\tau)]$. The equations can be described by the following system:

$$\begin{aligned}
\frac{di}{dt} &= \beta q \frac{l(1-i)}{(p-l)i + l} v - (\mu + \nu)i, \\
\frac{du}{dt} &= \eta - \alpha \frac{pi}{(p-l)i + l} u - \eta u, \\
\frac{dv}{dt} &= \alpha \frac{pi(t-\tau)}{(p-l)i(t-\tau) + l} u(t-\tau)e^{-\eta\tau} - \eta v(t).
\end{aligned} \tag{12}$$

Consequently, the basic reproductive ratio of the system is

$$R_0^{\tau} = \frac{\beta\alpha q p e^{-\eta\tau}}{\eta(\mu + \nu)l}.$$

Note that by including incubation time of malaria parasites in humans (ω), the system leads to the basic reproductive ratio into the following form:

$$R_0^{\omega\tau} = \frac{\beta\alpha q p e^{-\mu\omega} e^{-\eta\tau}}{\eta(\mu + \nu)l}.$$

Since human life span is long comparing with incubation time of the parasites, $e^{-\mu\omega}$ is close to 1. Hence, the incubation time in humans does not much alter the basic reproductive ratio which helps to determine whether the disease spreads. Consequently, we can omit the incubation time in human in the model for simplicity in the analysis.

The system (12) has two steady states, which are

1. The disease-free steady state

$$(i, u, v) = (0, 1, 0).$$

2. The disease-present steady state

$$(i, u, v) = \left(i^*, u^*, v^*\right)$$

with

$$u^* = \frac{\eta[(p-l)i^* + l]}{\eta[(p-l)i^* + l] + \alpha p i^*},$$
$$v^* = \frac{\alpha p i^* e^{-\eta\tau}}{\eta[(p-l)i^* + l] + \alpha p i^*},$$

where i^* satisfies

$$(\mu+\nu)(p-l)\big[\eta(p-l)+\alpha p\big]i^{*2} + \big\{\beta\alpha q p l e^{-\eta\tau} + (\mu+\nu)\big[\alpha p l + 2\eta l(p-l)\big]\big\}i^* + (\mu+\nu)\eta l^2 - \beta\alpha q p l e^{-\eta\tau} = 0.$$

The linearised system of (12) at the disease-free steady state is

$$\begin{aligned} \frac{di}{dt} &= \beta q v - (\mu+\nu)i, \\ \frac{du}{dt} &= -\alpha\frac{p}{l}i - \eta u, \\ \frac{dv}{dt} &= \alpha\frac{p}{l}i(t-\tau)e^{-\eta\tau} - \eta v. \end{aligned} \tag{13}$$

The system can be written in matrix form as follows:

$$\frac{dx}{dt} = J_0 x(t) + J_1 x(t-\tau),$$

where $x(t) = (i(t), u(t), v(t))^T$,

$$J_0 = \begin{bmatrix} -(\mu+\nu) & 0 & \beta q \\ -\alpha\frac{p}{l} & -\eta & 0 \\ 0 & 0 & -\eta \end{bmatrix} \quad \text{and} \quad J_1 = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ \alpha\frac{p}{l}e^{-\eta\tau} & 0 & 0 \end{bmatrix}.$$

By assuming that the solution of the system is $x = e^{-\lambda t}\hat{v}$, we obtain

$$\lambda\hat{v} = \big(J_0 + e^{-\lambda\tau}J_1\big)\hat{v}$$

and the characteristic equation is

$$\big|J_0 + e^{-\lambda\tau}J_1 - \lambda I\big| = 0$$

or

$$(\lambda+\eta)\left[\lambda^2 + (\mu+\nu+\eta)\lambda + \eta(\mu+\nu) - \beta\alpha q\frac{p}{l}pe^{-(\lambda+\eta)\tau}\right].$$

Clearly, one of the eigenvalues is $\lambda_1 = -\eta$, that always give a stable manifold. Hence, the stability of the disease-free steady state depends upon the following function:

$$F(\lambda,\tau) = \lambda^2 + (\mu+\nu+\eta)\lambda + \eta(\mu+\nu) - \beta\alpha q\frac{p}{l}e^{-(\lambda+\eta)\tau}.$$

In order to prove that the disease-free steady state is stable if and only if $R_0^\tau < 1$, we consider the properties of the function F and follows the steps of proof by Ruan et al. (2008) and Wei et al. (2008). The function F is analytic with $F(0,\tau) = \eta(\mu+\nu)(1-R_0^\tau)$ and $F(\lambda,0) = \lambda^2 + (\mu+\nu+\eta)\lambda + \eta(\mu+\nu) - \beta\alpha q\frac{p}{l}$. We separate the proof into three cases according to the basic reproductive ratio:

1. $R_0^\tau < 1$. For all positive λ and τ, $F(0,\tau) > 0$ and $\partial F(\lambda,\tau)/\partial\lambda > 0$ so that F is an increasing function for $\lambda > 0$. Consequently, there is no zero root and no positive root for any positive τ. Next, we show that F has no imaginary roots by assuming that $\pm i\omega$ are imaginary roots of F. So, ω must be a positive root of the following equation:

$$\omega^4 + \left[(\mu+\nu)^2 + \eta^2\right]\omega^2 + (\mu+\nu)^2\eta^2\left(1 - R_0^{\tau 2}\right) = 0.$$

   Obviously, this equation does not have nonnegative real roots. A contradiction is found and hence F does not have purely imaginary roots. Moreover, $F(\lambda,0)$ has two negative real roots which are

$$\lambda_\pm = \frac{-(\mu+\nu+\eta) \pm \sqrt{(\mu+\nu+\eta)^2 - 4\eta(\mu+\nu)(1-R_0^\tau)}}{2}$$

   and $\partial F(\lambda_\pm,0)/\partial\lambda \neq 0$. By the implicit function theorem and the continuity of F, we conclude that $F(\lambda,\tau)$ has no root with positive real parts for positive τ. The disease-free steady state is stable if $R_0^\tau < 1$.
2. $R_0^\tau = 1$. Because $F(0,\tau) = 0$ and $dF(\lambda,\tau)/d\lambda > 0$ for all $\lambda \geq 0$ and $\tau > 0$, zero is a simple root and there is no positive root. In the similar way with 1, we can show that there is no positive root. Hence, the disease-free steady state is degenerate.
3. $R_0^\tau > 1$. Since $F(0,\tau) < 0$ and $\partial F(\lambda,\tau)/\partial\lambda > 0$, for all $\lambda \geq 0$ and $\tau > 0$, F has a positive root. Thus, the disease-free steady state is unstable.

The proof is completed. Therefore, the disease-free steady state is stable if and only if $R_0^\tau < 1$.

The linearised system at the disease-present steady state is

$$\begin{aligned}
\frac{di}{dt} &= \frac{\beta ql(v - i^*v - v^*i)}{[(p-l)i^* + l]} - \frac{\beta ql(p-l)(v^* + i^*v^*)i}{[(p-l)i^* + l]^2]} - (\mu+\nu)i,\\
\frac{du}{dt} &= -\frac{\alpha p(u^*i + i^*u)}{[(p-l)i^* + l]} + \frac{\alpha p(p-l)i^*u^*i}{[(p-l)i^* + l]^2} - \eta u,\\
\frac{dv}{dt} &= \frac{\alpha pe^{-\eta\tau}(u^*i(t-\tau) + i^*u(t-\tau))}{[(p-l)i^* + l]} - \frac{\alpha p(p-l)e^{-\eta\tau}i^*u^*i}{[(p-l)i^* + l]^2} - \eta v.
\end{aligned} \tag{14}$$

The characteristic equation can be derived in the similar way with the linear system at the disease-free steady state and it is of order 3. Here, we study the dynamics of the disease-present steady state numerically.

In Fig. 1(a), the basic reproductive ratio is plotted against the incubation time in mosquitoes. It is decreased when the incubation time is increased. In other words,

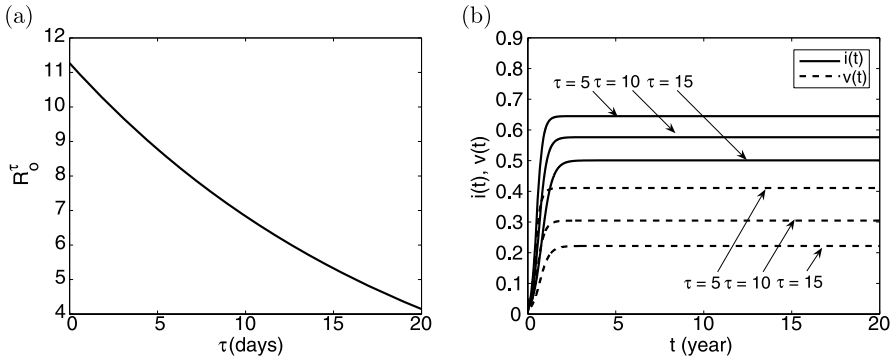

Fig. 1 (**a**) An incubation effect from mosquitoes on the basic reproductive ratio ($p = 0.7, l = 0.6$). (**b**) An incubation effect from mosquitoes on the number of infectious humans and mosquitoes (τ is in days, $p = 0.7, l = 0.6$)

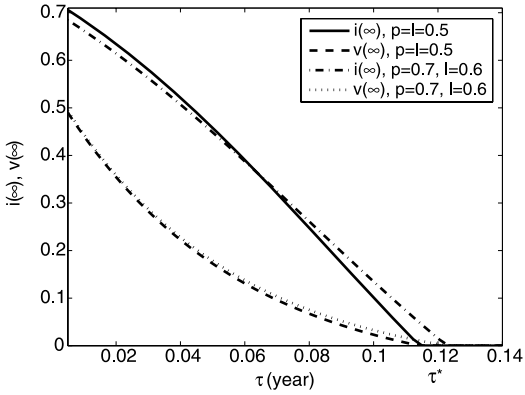

Fig. 2 A plot of asymptotic solutions ($i(\infty), v(\infty)$) against incubation time in mosquitoes is shown. At $\tau = \tau^*$, a transcritical bifurcation between the disease-free and disease-present steady states occurs ($R_0 = 9.4$)

when parasites need longer time to appear in bloodstream, the disease transmission is reduced. In Fig. 1(b), we study the solution of the system (12) at some values of τ. As we can see, the proportions of infectious humans and mosquitoes decrease when the incubation time of the malaria parasites in mosquitoes increases. The solution tends to the disease-present steady state finally.

By varying p and considering v^* which is an increasing function of p,

$$v^* = \frac{\alpha p i^* e^{-\eta\tau}}{\eta[(p-l)i^* + l] + \alpha p i^*},$$

τ is consequently increased whenever p is increased. Figure 2 shows numerical results of asymptotic solutions against incubation time in mosquitoes in case $R_0 = 9.4$, $p = l = 0.5$, where vector-bias does not occur, and $R_0 = 9.4$, $p = 0.7, l = 0.6$, where p is increased and vector-bias occurs, for example. The bifurcation occurs at τ^* where both of the proportions of infectious humans and mosquitoes become zero so that there is interchange of stability between two steady states (when $\tau < \tau^*$, the disease-present steady state is stable while the disease-free steady state is unstable,

and when $\tau > \tau^*$, the former becomes unstable while the latter becomes stable). At $p = l = 0.5$, τ^* is approximately 42 days (a minimum length of incubation time for malaria with $R_0 = 9.4$ dying out), while at $p = 0.7, l = 0.6$, τ^* is approximately 45 days. The former is longer than the lifespan of mosquitoes. Hence, in this case, malaria is endemic no matter how long incubation in mosquitoes takes. Note that this result also depends on other parameters such as the basic reproductive ratio. For example, in case the basic reproductive ratio is quite small, there might be possible range of incubation time in mosquitoes that the disease dies out.

6 The Study of Travelling Waves

In this section, we introduce the diffusion term to represent the mosquito's movement as a random walk. Also, because attractiveness of humans to mosquitoes is expressed in chemical forms such as sweat, body odour, and breath (Skinner et al. 1965; Takken and Knols 1999; Mukabana et al. 2004), we incorporate a chemotaxis term to explain the chemically directed movement to humans (Murray 2002). Referring to (1), the system after incorporating spatial terms becomes

$$\begin{aligned}
\frac{\partial S}{\partial t} &= \mu N - \beta \frac{lS}{pI + lS} V - \mu S + \nu I, \\
\frac{\partial I}{\partial t} &= \beta \frac{lS}{pI + lS} V - (\mu + \nu) I, \\
\frac{\partial U}{\partial t} &= \eta M - \alpha \frac{pI}{pI + lS} U - \eta U + D \frac{\partial^2 U}{\partial x^2} - \psi \frac{\partial}{\partial x} U \frac{\partial I}{\partial x}, \\
\frac{\partial V}{\partial t} &= \alpha \frac{pI}{pI + lS} U - \eta V + D \frac{\partial^2 V}{\partial x^2} - \psi \frac{\partial}{\partial x} V \frac{\partial I}{\partial x},
\end{aligned} \tag{15}$$

where x is a space variable, D is the diffusion coefficient ($D > 0$), and ψ is the chemotaxis coefficient ($\psi > 0$). Previously, in the system (1), compartmental variables are time-dependent but not space-dependent, and both of the total numbers of human and mosquito populations are constant with time. However, in this section, compartmental variables are dependent on time and space. Hence, we express them in terms of densities (numbers/space unit). For example, here, S represents the number of susceptible individuals per km^2. Also, β and α have a slightly different interpretation, as the transmission rates per susceptible per unit density of infectives. The total density of human population (N) is constant with time and space while the total density of mosquito population (M) depends on both time and space variables. Consequently,

$$\begin{aligned}
-\psi_1 \frac{\partial}{\partial x} U \frac{\partial S}{\partial x} - \psi_2 \frac{\partial}{\partial x} U \frac{\partial I}{\partial x} &= -(\psi_2 - \psi_1) \frac{\partial}{\partial x} U \frac{\partial I}{\partial x} \\
&= -\psi \frac{\partial}{\partial x} U \frac{\partial I}{\partial x},
\end{aligned}$$

where $S = N - I$, and $\psi_2 > \psi_1 > 0$ because malaria parasites manipulate hosts to be more attractive to mosquitoes via sweat and body odour, for instance.

We rescale and introduce new variables as follows:

$$s = \frac{S}{N}, \qquad i = \frac{I}{N}, \qquad u = \frac{U}{M^0}, \qquad v = \frac{V}{M^0}, \qquad q = \frac{M^0}{N}, \qquad T = (\mu + \nu)t,$$

where M^0 is the total density of mosquito population at the spatially uniform disease-free steady state. The governed system after rescaling becomes

$$\begin{aligned}
\frac{\partial i}{\partial T} &= \frac{\beta q l(1-i)}{(\mu+\nu)[(p-l)i+l]}v - i, \\
\frac{\partial u}{\partial T} &= \frac{\eta}{\mu+\nu}(u+v) - \frac{\alpha p i}{(\mu+\nu)[(p-l)i+l]}u - \frac{\eta}{\mu+\nu}u \\
&\quad + \frac{D}{(\mu+\nu)}\frac{\partial^2 u}{\partial x^2} - \frac{\psi}{(\mu+\nu)}\frac{\partial}{\partial x}u\frac{\partial i}{\partial x}, \\
\frac{\partial v}{\partial T} &= \frac{\alpha p i}{(\mu+\nu)[(p-l)i+l]}u - \frac{\eta}{\mu+\nu}v + \frac{D}{(\mu+\nu)}\frac{\partial^2 v}{\partial x^2} - \frac{\psi}{(\mu+\nu)}\frac{\partial}{\partial x}v\frac{\partial i}{\partial x}.
\end{aligned} \tag{16}$$

We study the propagation of malaria from the infected population into the uninfected population (infected human population to uninfected mosquito population and infected mosquito population to uninfected human population) by introducing the initial conditions as step functions in one-dimensional domain $[x_1, x_2]$ with malaria initially present at its spatially uniform steady state in $[x_1, \bar{x}]$) and absent in $[\bar{x}, x_2]$, where $\bar{x} = (x_1 + x_2)/2$. The initial conditions are given by

$$\begin{aligned}
i(x,0) &= i^* - i^* H(x-\bar{x}), \\
u(x,0) &= u^* + (1-u^*)H(x-\bar{x}), \\
v(x,0) &= v^* - v^* H(x-\bar{x}),
\end{aligned}$$

where $x_1 \le x \le x_2$, $H(x)$ is a heaviside step function, $u^* = 1 - v^*$, i^* and v^* correspond to (4). The initial conditions are shown in Fig. 3(a).

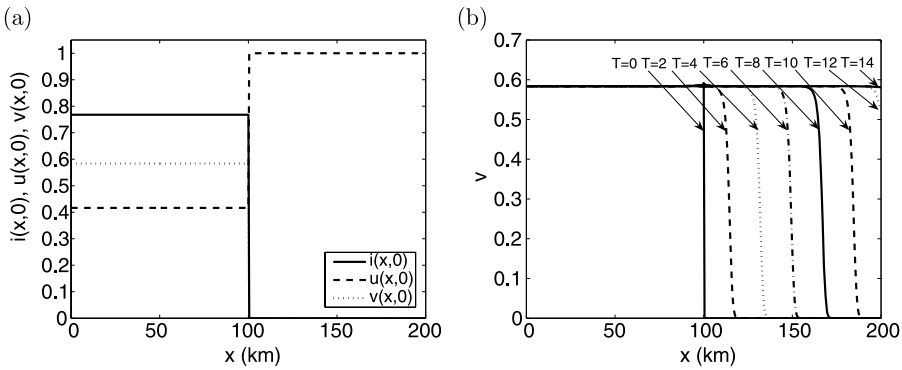

Fig. 3 (**a**) Initial conditions in the one-dimensional domain $[0, 200]$. (**b**) This graph shows the travelling waves of infectious mosquitoes from the endemic area to the disease-free area ($T = (\mu+\nu)t$, $p = 0.7$, $l = 0.4$, $c = 17.9$, $D = 4.6$, $\psi = 2$)

The boundary conditions are assumed as zero fluxes:

$$D\frac{\partial u}{\partial x}(x_1,T)-\psi u(x_1,T)\frac{\partial i}{\partial x}(x_1,T)=0=D\frac{\partial u}{\partial x}(x_2,T)-\psi u(x_2,T)\frac{\partial i}{\partial x}(x_2,T),$$
$$D\frac{\partial v}{\partial x}(x_1,T)-\psi v(x_1,T)\frac{\partial i}{\partial x}(x_1,T)=0=D\frac{\partial v}{\partial x}(x_2,T)-\psi v(x_2,T)\frac{\partial i}{\partial x}(x_2,T),$$

where $T_1 \le T \le T_2$. In Fig. 3(b), we can see that malaria spreads from infected population to uninfected population like travelling waves. The wave speed (c) can be calculated from the graph by the formula

$$c=\frac{\hat{x}_2-\hat{x}_1}{t_2-t_1},$$

where $\hat{x}_2-\hat{x}_1$ is the distance on the space and t_2-t_1 is the time difference corresponding to the wave. When $p=0.7$ and $l=0.4$, the wave speed is 17.9 km/year, for example. Other variables also share the same wave speed with v or V (see Fig. 3). When p is larger, for example, $p=0.8$, the speed of travelling waves becomes bigger, which is 18.9 km/year. These results can be further compared with actual data on malaria disemmination. Hence, it suggests that the more the infectious hosts become attractive to the mosquitoes, the faster the propagation of the disease from the infected population to the uninfected population is.

We further study the wave speed c numerically by considering the relation between p and l. In Fig. 4(a), when p and l increase but the difference between them is fixed at 0.1, or in other words when l/p becomes larger ($l/p \to 1$), the wave speed of malaria propagation decreases. Figure 4(b) shows that wave speed of the disease increases when p increases while l is fixed at 0.4. Both results also suggest that wave speed depends on how different between p and l or how attractive the infectious human to the mosquito comparing with the susceptible human is.

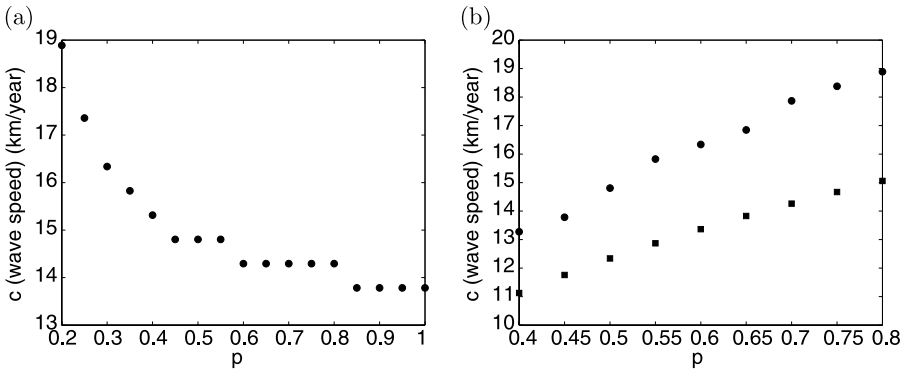

Fig. 4 (**a**) A relation between the probability that a mosquito picks an infectious human (p) and the wave speed (c) when $p-l=0.1$ and p varies. (**b**) A relation between p and c, when l is fixed at 0.4 while p varies, from a numerical simulation comparing with the minimum wave speed

Next, we calculate the lower bound of wavespeed (c_l) by employing the method in Murray (2002). We look for travelling wave solutions by setting

$$i(x,T)=i(z), \qquad u(x,T)=u(z), \qquad v(x,T)=v(z), \qquad z=x-cT. \tag{17}$$

By substituting them into (16), we obtain the ordinary differential equation system

$$\begin{aligned}
&ci'+\frac{\beta ql(1-i)}{(\mu+\nu)[(p-l)i+l]}v-i=0,\\
&\frac{D}{(\mu+\nu)}u''-\frac{\psi}{(\mu+\nu)}(ui')'+cu'+\frac{\eta}{(\mu+\nu)}(u+v)\\
&\quad-\frac{\alpha pi}{(\mu+\nu)[(p-l)i+l]}u-\frac{\eta}{(\mu+\nu)}u=0,\\
&\frac{D}{(\mu+\nu)}v''-\frac{\psi}{(\mu+\nu)}(vi')'+cv'+\frac{\alpha pi}{(\mu+\nu)[(p-l)i+l]}u-\frac{\eta}{(\mu+\nu)}v=0,
\end{aligned} \tag{18}$$

where the prime denotes differentiation with respect to z. Our goal is to find solutions with positive wave speed c and nonnegative i, u and v of the eigenvalue problem such that

$$i(-\infty)=i(\infty)=0, \qquad 0\le u(-\infty)<u(\infty)=1, \qquad v(-\infty)=v(\infty)=0.$$

The conditions on i and v lead to a pulse wave of infection that propagates into the uninfected population. We linearise the system (18) near the leading edge of the wave where $i\to 0$, $u\to 1$, and $v\to 0$ to have

$$\begin{aligned}
&ci'+\frac{\beta q}{(\mu+\nu)}v-i\approx 0,\\
&\frac{D}{(\mu+\nu)}u''-\frac{\psi}{(\mu+\nu)}i''+cu'+\frac{\eta}{(\mu+\nu)}v-\frac{\alpha p}{(\mu+\nu)l}i\approx 0,\\
&\frac{D}{(\mu+\nu)}v''+cv'+\frac{\alpha p}{(\mu+\nu)l}i-\frac{\eta}{(\mu+\nu)}v\approx 0.
\end{aligned} \tag{19}$$

Since the variable u does not appear in the other two equations (decoupled), we can omit the differential equation of u, and hence the dynamics of the system are governed by the differential equations of i and v. By introducing a new variable w as v', the dynamics of the wave solutions of the system (19) can be described by the following first-order ordinary differential equation system

$$\begin{aligned}
\frac{di}{dz}&=\frac{1}{c}i-\frac{\beta q}{c(\mu+\nu)}v,\\
\frac{dv}{dz}&=w,\\
\frac{dw}{dz}&=-\frac{\alpha p}{Dl}i+\frac{\eta}{D}v-\frac{c(\mu+\nu)}{D}w.
\end{aligned} \tag{20}$$

The characteristic equation of the system (20) is

$$\lambda\left(\lambda+\frac{c(\mu+\nu)}{D}\right)\left(\lambda-\frac{1}{c}\right)-\frac{\eta}{D}\left(\lambda-\frac{1}{c}\right)-\frac{\beta\alpha qp}{cDl(\mu+\nu)}=0. \tag{21}$$

In order to find the minimum value of the wave speed (c_l), we differentiate throughout (21) with respect to λ to find λ that minimizes c and the equation is given by

$$3\lambda^2+\left(\frac{2c(\mu+\nu)}{D}-\frac{2}{c}\right)\lambda-\left(\frac{(\mu+\nu)}{D}+\frac{\eta}{D}\right)=0. \tag{22}$$

Because c_l cannot be easily written in an explicit form, we employ a numerical technique such as Newton's method to solve for the solution. By solving (21) and (22) for c, we can find the minimum value of the wave speed of the system (16). The minimum value of the wave speed at some values of p and l is shown in Fig. 4(b) in space units per year.

7 Conclusion and Discussion

Lacroix et al. (2005) suggests that infectious humans may be more attractive to mosquitoes than susceptible humans due to the manipulation of the malaria parasites in them. Previously, Kingsolver (1987) introduce a model that takes account for this manipulation. Here, we introduce the model in a different way from previous authors and analyse it relating to certain factors in mosquitoes to the spread of malaria such as incubation time, random movement, and chemically directed movement to humans.

First, we introduce the model with the dynamics of malaria in humans and mosquitoes described by SIS (susceptible-infectious-susceptible) and SI (susceptible-infectious) compartment models, respectively. The vector-bias term is expressed in terms of different probabilities that a mosquito arriving at a human at random and picks that human depending on whether that person is infectious, or susceptible. A vector-bias model based on SIRS (susceptible-infectious-recovered-susceptible) in humans and SI in mosquitoes on malaria transmission is currently investigated (unpublished)

Second, we prove that the transcritical bifurcation occurs at the basic reproductive ratio equalling 1 ($R_0 = 1$) by projecting the flow onto the extended centre manifold. Consequently, the disease-free steady state is stable if and only if $R_0 < 1$ and the disease-present steady state is stable if and only if $R_0 > 1$. The greater attractiveness of infectious humans to mosquitoes affects malaria transmission that the populations favour high prevalence of the parasites.

Due to short lifespan of mosquitoes, we incorporate the incubation of malaria parasites that might play an important role in studying the dynamics behaviours of the disease. From our study, it suggests that the malaria transmission, the proportion of infectious humans and mosquitoes, decreases when incubation time of malaria parasites in mosquitoes increases. We show that the disease-free steady state is stable if $R_0^\tau < 1$ analytically and the disease-present steady state is stable if $R_0^\tau > 1$ numerically. Also, for our parameter ranges, we show that malaria is endemic no matter how long the incubation time is.

Finally, we include the random movement of mosquitoes in term of a diffusion term and attractiveness of humans to mosquitoes in chemical forms such as sweat and body odour in term of chemotaxis term into the model. We then study the propagation of the infected population to the uninfected population. We show that travelling waves occurs as a pulse wave and the wave speed can be calculated from the numerical results. We also approximate the minimum wave speed analytically and compare it with the numerical result.

All in all, this work should be further studied by approximating the values of p and l from real data from a malaria endemic area, or including control measures into the model.

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