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Review

A personal overview of epidemic models for mosquito-borne infections

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Abstract: In this paper, we present a brief, nonexhaustive overview of the features we consider most relevant in models for mosquito-transmitted infections, comparing them with the corresponding features in models for directly-transmitted infections. In particular, we focus on the basic reproduction number $\mathcal{R}_0$ and its relations with the exponential growth rate, the probability of an outbreak, and the final size of the epidemic. The concept of a threshold for epidemic spread based on the basic reproduction number ($\mathcal{R}_0 > 1$) was first introduced by Ronald Ross for malaria. Later, the study of models for vector-transmitted infections, where the vector naturally follows a seasonal cycle, has led to a definition of $\mathcal{R}_0$ when model parameters are time-periodic. Host heterogeneity in attractiveness to mosquitoes leads to the counter-intuitive result that protective behaviors may increase $\mathcal{R}_0$; we present the assumptions behind this result, and the factors that can instead help decreasing $\mathcal{R}_0$.

Keywords: Ross–Macdonald model; vector-borne diseases; basic reproduction number; final epidemic size; host heterogeneity; epidemic models with periodic coefficients

1. Introduction

Recent rises in cases of several mosquito-borne diseases, such as dengue (especially in Latin America, where the number of yearly reported cases has increased by an order of magnitude [1, 2], and locally transmitted cases are reported also in nonendemic areas [3]), Chikungunya [4], Zika [5], and West Nile [6], without forgetting the persistent toll of deaths caused by malaria in sub-Saharan Africa [7], have increased the interest in the use of mathematical models to understand the spread of vector-borne infections, and the potential effects of strategies that can be adopted to control them.

A first mathematical model for malaria was formulated, although not in the current notation, between 1908 and 1911 by Ronald Ross [8], who derived what we now call the basic reproduction number (or ratio) $\mathcal{R}_0$. Subsequently, George Macdonald [9] extended the model in several directions; among these, he allowed for time delays between the time of infection and the time when an individual (human or mosquito) becomes infectious. It should be noted that the use of delays to model latent

periods in both humans and mosquitoes was first considered by Sharpe and Lotka in [10], although mosquito mortality during this phase was neglected. Moreover, Macdonald credited Armitage [11] with advancing mathematical ideas on delays; see [12] and references therein for a comprehensive historical overview. From their work, a model framework known as the Ross–Macdonald model has emerged and remains the basis for most models studied in recent years.

Thus, our overview starts by clearly specifying the assumptions behind the Ross–Macdonald model. In Section 2, we compute the basic reproduction number $\mathcal{R}_0$ in an epidemic setting, first for the simple case of an ordinary differential equation (ODE) model of SIR (susceptible–infectious–removed) type for hosts and SI (susceptible–infectious) type for vectors, and then allowing for latent periods with arbitrary distributions. In Section 3, we explore whether $\mathcal{R}_0$ has the same relation as in models for directly transmitted diseases with three important quantities: the epidemic growth rate during the initial phase of exponential growth, the final epidemic size in the case of an uncontrolled outbreak, and the probability of a major epidemic outbreak after the introduction of a few infected individuals (hosts or vectors) in a stochastic variant of the model.

Moving in Section 4 to the endemic situation, we introduce an extension of the model for an epidemic outbreak, in order to take into account hosts' births and deaths; for this model, we present a general result on a strong threshold property depending on the value of $\mathcal{R}_0$: When $\mathcal{R}_0 \leq 1$, the epidemic dies out, whereas when $\mathcal{R}_0 > 1$, the solutions converge to a unique endemic stationary state. This is not necessarily true if host mortality due to disease is sufficiently strong, because a backward bifurcation at $\mathcal{R}_0 = 1$ may occur. We also present an extension to the case in which coefficients are periodic.

We conclude our overview in Section 5 by presenting a model with host heterogeneity in attractiveness to mosquitoes, and/or competence in transmitting the infection, as this may give rise to counterintuitive results, differing from what happens in the case of models for directly transmitted infections.

Most (or all) of the results presented here are well-known, and we try to refer each of them to the original source. With our review, we aim at placing them in a coherent structure, stressing the similarities with and differences from the corresponding results in models for directly transmitted infections, and highlighting some issues that, in our opinion, deserve further investigation.

Since hundreds of papers that study mathematical models for vector-borne infections are published every year, we do not try to be exhaustive, and there certainly exist many relevant papers that are not cited here. An important feature of vector-transmitted infections is that epidemic control can be achieved by targeting the vectors. We do not touch on this topic at all, as there exists a recent review centered on dengue [13]. Moreover, we do not consider variation in host attractiveness across epidemiological states (vector bias) nor changes in host preference, feeding frequency, or behavior driven by infection with a pathogen (conditional vector preferences); for these aspects, we refer respectively to [14, 15] and [16], and to the references therein.

As we prioritize generality, we abstract from factors that are typical of specific infections. For instance, it has been shown that an important factor in dengue epidemiology is antibody-dependent enhancement (ADE) [17], so that many models for dengue study the dynamics with multiple strains; for this topic, we refer to [18] and the references therein.

2. Ross–Macdonald model and the reproduction number $\mathcal{R}_0$

2.1. The assumptions and model construction

We start by discussing the implicit assumptions underlying the simplest Ross–Macdonald-type models, which are used to describe vector-borne epidemic outbreaks in nonendemic scenarios:

- No infection-induced mortality, thus leading to an equilibrium demography of humans and mosquitoes.
- The interest is in a short timescale, thus neglecting human births and deaths. The human population size $\bar{H}$ is constant.
- Mosquitoes are at demographic equilibrium, with adult mortality rate μ compensated by recruitment of (female) adults at rate Λ. Thus, the population size of adult female mosquitoes M satisfies $M' = \Lambda - \mu M$, and it is assumed that $M = \Lambda/\mu =: \bar{M}$. Implicitly, it is assumed that the values of $\bar{H}$ and $\bar{M}$ are independent, that is, humans are not a limiting resource for mosquitoes, and their density is regulated by (unmodeled) effects in the aquatic stages, which determine the value of Λ.
- The per capita biting rate of (female adult) mosquitoes on humans is constant, b, and does not depend on the value $\bar{H}$. This means that mosquitoes are always able to quickly find humans when searching for a host. Generally, b is estimated as the inverse of the average length of the gonotrophic cycle, that is, the time required for a mosquito to go from a blood meal to completing egg-laying; see, for instance, [19], or the recent review [20], in which this approach has been criticized.

From these assumptions, one easily arrives at a system of ODEs describing the dynamics of a vector-borne infection. For the sake of simplicity, we start by neglecting latent periods. Moreover, as we think of infections such as dengue or Chikungunya (and not of malaria), we assume that once recovered from infection, a human is permanently immune to further infections (at least from the same strain); thus the host dynamics will be of SIR type, and the corresponding compartments will be denoted by S_H, I_H, and R_H. It is generally agreed that infected mosquitoes remain infectious for their whole (short) life, and thus vector dynamics will be of SI type, and its compartments will be denoted as S_M and I_M.

We assume that, upon being bitten by (respectively biting) an infectious mosquito (human), a susceptible human (mosquito) has a given probability of acquiring the pathogen. Let p_{MH} (p_{HM}) be the probability that a susceptible human (mosquito) gets infected when bitten by (biting) an infectious mosquito (human). Since the total number of bites per unit of time of infectious mosquitoes is bI_H, and the probability of biting a susceptible human is $S_H/\bar{H}$, the rate at which susceptible humans are infected is

$$bI_M \frac{S_H}{\bar{H}} p_{MH}.$$

On the other hand, the rate at which susceptible mosquitoes are infected is given by

$$bS_M \frac{I_H}{\bar{H}} p_{HM}.$$

If the rate at which infected humans recover is γ (with $1/\gamma$ being the average length of the infectious

period), then one obtains (recalling the assumptions on human and mosquito demography) the system

$$\begin{cases} S'_H = -bI_M \dfrac{S_H}{\bar{H}} p_{MH}, \\ I'_H = bI_M \dfrac{S_H}{\bar{H}} p_{MH} - \gamma I_H, \\ S'_M = \Lambda - bS_M \dfrac{I_H}{\bar{H}} p_{HM} - \mu S_M, \\ I'_M = bS_M \dfrac{I_H}{\bar{H}} p_{HM} - \mu I_M. \end{cases} \tag{2.1}$$

The equation for R_H has not been written, as it is not necessary; if needed, one can obtain $R_H = \bar{H} - S_H - I_H$. Similarly, one may note that, if $S_M(0) + I_M(0) = \bar{M} = \Lambda/\mu$, then $S_M(t) + I_M(t) \equiv \bar{M}$ for all $t \geq 0$ so that one can obtain $S_M(t) = \bar{M} - I_M(t)$ and reduce (2.1) to a system in the three variables $(S_H,\ I_H,\ I_M)$.

2.2. *Basic reproduction number* $\mathcal{R}_0$

Linearizing (2.1) around the disease-free equilibrium (DFE) $(\bar{H}, 0, 0)$, one obtains the linear system

$$\begin{cases} I'_H = bI_M p_{MH} - \gamma I_H, \\ I'_M = b\bar{M} \dfrac{I_H}{\bar{H}} p_{HM} - \mu I_M, \end{cases}$$

which can be written as

$$\begin{pmatrix} I_H \\ I_M \end{pmatrix}' = A \begin{pmatrix} I_H \\ I_M \end{pmatrix}, \tag{2.2}$$

with $A = B - D$, where B accounts for *infection*, and D accounts for *transition* [21, 22], and they are respectively defined as

$$B := \begin{pmatrix} 0 & bp_{MH} \\ b\frac{\bar{M}}{\bar{H}} p_{HM} & 0 \end{pmatrix}, \quad \text{and} \quad D := \begin{pmatrix} \gamma & 0 \\ 0 & \mu \end{pmatrix}.$$

It is well-known that the largest eigenvalue of A has negative (positive) real part if and only if $\mathcal{R}_0 < 1$ ($\mathcal{R}_0 > 1$), where $\mathcal{R}_0$ is the spectral radius of $K := BD^{-1}$; see, for instance, [21, Theorem A.1]. In this case, computing $\mathcal{R}_0$ is immediate because

$$K = \begin{pmatrix} 0 & \dfrac{bp_{MH}}{\mu} \\ \dfrac{bp_{HM}}{\gamma} \dfrac{\bar{M}}{\bar{H}} & 0 \end{pmatrix}, \tag{2.3}$$

so that

$$\mathcal{R}_0 = \rho(K) = b\sqrt{\frac{p_{MH}p_{HM}}{\gamma\mu} \frac{\bar{M}}{\bar{H}}}, \tag{2.4}$$

where ρ denotes the spectral radius. Notice that the matrix K can be immediately interpreted as a next-generation matrix (NGM) [23, Section 7.2]. Indeed, K_{12} is the average number of (susceptible)

humans infected by an infected mosquito over her entire lifespan, and K_{21} is the average number of (susceptible) mosquitoes infected by an infected human over her/his whole infectious period [24].

In [8], Ross defined $\mathcal{R}_0$ as the square of the term in (2.4), as he considered generations in terms of human-to-human, or mosquito-to-mosquito, infections. Of course, this is irrelevant if we are interested in the threshold condition $\mathcal{R}_0 < 1$. However, let us mention that, for this system, $\mathcal{R}_0^2$ is actually the type-reproduction number [25], which, in general multitype models, represents the expected number of cases in individuals of a certain type (e.g., humans) caused by one infected individual of the same type in a completely susceptible population, either directly or through chains of infection passing through any sequence of the other types (e.g., mosquitoes).

Two immediate observations arise from Formula (2.4): The first is that the biting rate b is an extremely important parameter, as it is the only parameter that enters linearly in $\mathcal{R}_0$; hence, if b is increased or decreased of a given percentage, the same change occurs in $\mathcal{R}_0$. On the other hand, an increase or decrease in any other parameter (say, mosquito density $\bar{M}$) does not translate into the same change in $\mathcal{R}_0$; indeed, using the Taylor approximation for $\sqrt{1+x}$, one sees that an increase (decrease) of $\bar{M}$ of a small percentage x results in an increase (decrease) of $\mathcal{R}_0$ of approximately $x/2$.

The second observation is that $\mathcal{R}_0^2$ is proportional to the ratio $\bar{M}/\bar{H}$. Consequently, decreasing $\bar{M}$ could be effective for controlling infection outbreaks. For instance, this might be achieved by using larvicides, although their continuous use might lead to the appearance of resistance. We do not go into the details of this issue, as it is out of the scope of this review; we refer the interested reader to [13, 26] for more details in the case of dengue.

In the present context of short-term dynamics with no recruitment of new susceptible humans, one can describe only an epidemic outbreak. What can be proved (see details later) is that, if $\mathcal{R}_0 \leq 1$, the introduction of a small number of infected human hosts will give rise to at most a few new cases; on the other hand, if $\mathcal{R}_0 > 1$, a large outbreak can be expected.

2.3. Allowing for latent periods

We start as in [9], considering a fixed latent period: τ_H for humans, τ_M for mosquitoes. Now, the recruitment rate of infected mosquitoes at time t will be equal to

$$bS_M(t-\tau_M)\frac{I_H(t-\tau_M)}{\bar{H}}p_{HM}e^{-\mu\tau_M}, \tag{2.5}$$

where the last factor in (2.5) accounts for the probability for a mosquito infected at time $t-\tau_M$ to still be alive at time t.

For the infected hosts, the recruitment rate is similar, but simpler, as we assumed no mortality in humans. Hence, one obtains the system of delay-differential equations

$$\begin{cases} S'_H(t) = -bI_M(t)\dfrac{S_H(t)}{\bar{H}}p_{MH}, \\ I'_H(t) = bI_M(t-\tau_H)\dfrac{S_H(t-\tau_H)}{\bar{H}}p_{MH} - \gamma I_H(t), \\ I'_M(t) = b\left(\bar{M} - I_M(t-\tau_M)\right)\dfrac{I_H(t-\tau_M)}{\bar{H}}p_{HM}e^{-\mu\tau_M} - \mu I_M(t). \end{cases} \tag{2.6}$$

To investigate the behavior of the system near the DFE, one can linearize (2.6) and study the exponential solutions of the linearization. Alternatively, without being completely rigorous, one can simply

modify the previous NGM (2.3) into

$$K = \begin{pmatrix} 0 & \frac{bp_{MH}}{\mu} e^{-\mu\tau_M} \\ \frac{bp_{HM}}{\gamma}\frac{\bar{M}}{\bar{H}} & 0 \end{pmatrix}.$$

In this case, one obtains

$$\mathcal{R}_0 = \rho(K) = b\sqrt{\frac{p_{MH}p_{HM}e^{-\mu\tau_M}}{\gamma\mu}\frac{\bar{M}}{\bar{H}}}. \tag{2.7}$$

Note that, with respect to (2.4), the presence of the delay τ_M in (2.7) makes $\mathcal{R}_0$ decrease exponentially as μ increases, which adds to the decreasing effect already caused by the term μ in the denominator. This suggests that increasing adult mortality by the use of adulticides can be effective in controlling an infectious disease, even if this were not to reduce mosquito population density, which is assumed to be determined by density-dependence in the aquatic stages. This conclusion was taken as a mathematical support for the extensive use of dichlorodiphenyltrichloroethane (DDT) between 1950 and 1970 [27], before it was shown to be ineffective in the areas of high malaria incidence and the cause of serious environmental damage.

For the sake of completeness, we add a proof of the fact that $\mathcal{R}_0 < 1$ is the condition for stability of the DFE in a slightly more general context. In fact, the length of the latent period is expected to differ among infected individuals. This suggests that, instead of a fixed delay, one can assume a distributed delay, described by the probability density $f_H(\tau)$ or $f_M(\tau)$ for humans or mosquitoes, respectively. Then, one modifies the equations for I_H and I_M in (2.6) to the following system of integrodifferential equations:

$$\begin{cases} I_H'(t) = bp_{MH}\displaystyle\int_0^{+\infty} I_M(t-\tau)\frac{S_H(t-\tau)}{\bar{H}} f_H(\tau)\,d\tau - \gamma I_H(t), \\ I_M'(t) = bp_{HM}\displaystyle\int_0^{+\infty} (\bar{M} - I_M(t-\tau))\frac{I_H(t-\tau)}{\bar{H}} e^{-\mu\tau} f_M(\tau)\,d\tau - \mu I_M(t). \end{cases}$$

Linearizing this system, one obtains

$$\begin{cases} I_H'(t) = bp_{MH}\displaystyle\int_0^{+\infty} I_M(t-\tau) f_H(\tau)\,d\tau - \gamma I_H(t), \\ I_M'(t) = bp_{HM}\frac{\bar{M}}{\bar{H}}\displaystyle\int_0^{+\infty} I_H(t-\tau)e^{-\mu\tau} f_M(\tau)\,d\tau - \mu I_M(t). \end{cases} \tag{2.8}$$

Then, looking for solutions of (2.8) of the form

$$(I_H(t), I_M(t)) = (ue^{\lambda t}, ve^{\lambda t}), \quad u, v, \lambda \in \mathbb{C}, \quad (u, v) \neq (0, 0),$$

one sees that such a solution is admissible if and only if λ is a root of the characteristic equation

$$(\lambda + \gamma)(\lambda + \mu) = b^2 p_{MH}p_{HM}\frac{\bar{M}}{\bar{H}}\hat{f}_H(\lambda)\hat{f}_M(\lambda + \mu), \tag{2.9}$$

where we denote with $\hat{}$ the Laplace transform. For $\lambda \in \mathbb{R}$, $\lambda > \max\{-\gamma, -\mu\}$, let us consider the function

$$G(\lambda) := \frac{b^2 p_{MH} p_{HM} \frac{\bar{M}}{\bar{H}} \hat{f}_H(\lambda) \hat{f}_M(\lambda + \mu)}{(\lambda + \gamma)(\lambda + \mu)}.$$

Note that G is a decreasing function such that

$$\lim_{\lambda \to \max\{-\gamma,-\mu\}^+} G(\lambda) = +\infty, \qquad \lim_{\lambda \to +\infty} G(\lambda) = 0.$$

Hence, there exists a unique root r of $G(\lambda) = 1$ (which is clearly equivalent to (2.9)) in the interval $(\max\{-\gamma, -\mu\}, +\infty)$. Furthermore, one has $r < 0$ if and only if

$$\mathcal{R}_0 := G(0) = b \sqrt{\frac{p_{MH} p_{HM} \hat{f}_M(\mu)}{\gamma \mu} \frac{\bar{M}}{\bar{H}}} < 1. \tag{2.10}$$

Considering complex roots of (2.9), we note that if $\mathrm{Re}\,(\lambda) > \max\{-\gamma, -\mu\}$, and $\mathrm{Im}\,(\lambda) \neq 0$, then

$$(\mathrm{Re}\,(\lambda) + \gamma)(\mathrm{Re}\,(\lambda) + \mu) = |(\mathrm{Re}\,(\lambda) + \gamma)(\mathrm{Re}\,(\lambda) + \mu)| < |(\lambda + \gamma)(\lambda + \mu)|.$$

Hence, if λ is a root of (2.9) with $\mathrm{Re}\,(\lambda) > \max\{-\gamma, -\mu\}$, one has

$$\begin{aligned}(\mathrm{Re}\,(\lambda) + \gamma)(\mathrm{Re}\,(\lambda) + \mu) < |(\lambda + \gamma)(\lambda + \mu)| &= b^2 p_{MH} p_{HM} \frac{\bar{M}}{\bar{H}} \left| \hat{f}_H(\lambda) \hat{f}_M(\lambda + \mu) \right| \\ &\leq b^2 p_{MH} p_{HM} \frac{\bar{M}}{\bar{H}} \hat{f}_H\left(\mathrm{Re}\,(\lambda)\right) \hat{f}_M\left(\mu + \mathrm{Re}\,(\lambda)\right),\end{aligned}$$

that is, $G(\mathrm{Re}\,(\lambda)) > 1$. This implies $\mathrm{Re}\,(\lambda) < r$.

Note that, in the case of a constant delay τ_M, (2.10) yields (2.7). In fact, one can formally set $f_M(\cdot)$ as a Dirac delta centered at τ_M so that $\hat{f}_M(\mu) = e^{-\mu \tau_M}$. Another special case is when the kernels $f_H(\cdot)$ and $f_M(\cdot)$ are the densities of exponential distributions. In this case, it is well-known that the system reduces to a system of ODEs of SEIR-SEI type (see, e.g., [28]), where 'E' represents the *exposed* class consisting of those individuals who have been infected but are not yet infectious. For instance, if $f_M(\tau) = \nu e^{-\nu \tau}$, then

$$\hat{f}_M(\mu) = \frac{\nu}{\mu + \nu},$$

and (2.10) gives

$$\mathcal{R}_0 = b \sqrt{\frac{p_{MH} p_{HM} \nu}{\gamma \mu (\mu + \nu)} \frac{\bar{M}}{\bar{H}}}.$$

One may observe that if we compare this case to that of a constant delay τ_M, setting $\nu = 1/\tau_M$, so that the average values correspond, then

$$\hat{f}_M(\mu) = \frac{1}{1 + \mu \tau_M} > e^{-\mu \tau_M}, \quad \text{for} \quad \tau_M > 0.$$

Hence, the reduction of $\mathcal{R}_0$ due to delays is lower for an exponentially distributed delay than for a fixed one.

3. The implications of $\mathcal{R}_0$ in models for vector-borne infections

As shown before, computing $\mathcal{R}_0$ is important, as the threshold condition $\mathcal{R}_0 > 1$ is necessary (and sufficient in deterministic models) for an infection outbreak. In models for infections with direct transmission, the value of $\mathcal{R}_0$ also determines: the rate of exponential growth in the initial epidemic phase; the probability, in a stochastic version of the model, that a major outbreak occurs after the introduction of one (or few) infectious individuals; and the *final epidemic size*, that is, the fraction of the population that has been infected during the whole epidemic outbreak.

In this section, we discuss the relations between $\mathcal{R}_0$ and these three quantities in vector-borne infections, drawing (if possible) a parallel with the results for directly transmitted infections.

3.1. The rate of exponential growth

It is well-known that in deterministic models for directly transmitted infections, there is a one-to-one correspondence between the value of $\mathcal{R}_0$ and r, the rate of exponential growth in the initial epidemic phase. Precisely, for the SIR model, one has

$$r = (\mathcal{R}_0 - 1)\gamma = \frac{(\mathcal{R}_0 - 1)}{\text{length of infectious period}}. \tag{3.1}$$

More generally, a simple relation also holds for models with arbitrary distributions of the infectious period, and with variable infectiousness during that period. In fact, following [23], let us denote by $A(\tau)$ the contribution to the force of infection of an individual which was infected τ time units ago. Then, the incidence (i.e., the rate of new infections) at time t, $i(t)$, follows the equation

$$i(t) = \frac{S(t)}{N} \int_0^{+\infty} A(\tau) i(t-\tau)\, d\tau.$$

It is not difficult to see [23] that

$$\mathcal{R}_0 = \int_0^{+\infty} A(\tau)\, d\tau, \tag{3.2}$$

and r solves

$$\hat{A}(r) = 1. \tag{3.3}$$

In particular, in this case, computing r generally requires solving a transcendental equation. Often, the relation between r and $\mathcal{R}_0$ is used in the opposite direction, with r estimated from data and $\mathcal{R}_0$ computed from (3.2). In fact, let us consider the generation time distribution (i.e., the probability density function of secondary cases),

$$a(\tau) := \frac{A(\tau)}{\int_0^{+\infty} A(\tau) d\tau} = \frac{A(\tau)}{\mathcal{R}_0}, \tag{3.4}$$

and let us assume that it is known. Then, from (3.3), one obtains $\mathcal{R}_0 = 1/\hat{a}(r)$ [29]. This formula is also the basis of the usual estimates of $\mathcal{R}_t$, the time-varying estimates of the effective reproduction number [29, 30].

How can these formulae be translated to models for vector-borne infections? Starting from the simplest case of the SIR-SI ODE model (2.1), one finds that the largest eigenvalue r of the matrix A in

(2.2) is

$$r = \sqrt{(\mathcal{R}_0^2 - 1)\mu\gamma + \frac{(\mu+\gamma)^2}{4}} - \frac{\mu+\gamma}{2}. \tag{3.5}$$

If $\mu = \gamma$ (i.e., the length of the human infectious period equals the average mosquito adult lifespan), then (3.5) simplifies to $r = (\mathcal{R}_0 - 1)\gamma$, exactly as (3.1) for cases of direct transmission.

On the other hand, if $\mu \ll \gamma$, expanding (3.5) in powers of μ/γ, one obtains

$$r = \mu(\mathcal{R}_0^2 - 1) + \mu O\left(\frac{\mu}{\gamma}\right) \approx \frac{(\mathcal{R}_0^2 - 1)}{\text{length of the longer infectious period}}.$$

In general, because of the inequality between the geometric and arithmetic means $\sqrt{\mu\gamma} < \frac{\mu+\gamma}{2}$, from (3.5), one obtains

$$r < (\mathcal{R}_0 - 1)\frac{(\mu+\gamma)}{2}.$$

In the more general setting of latent periods with distributed delays, one may look at the generation time from human to human. This will be the sum of the latent period in humans (with distribution f_H), of the time from the start of human infectiousness to the transmission event (with exponential distribution of parameter γ), of the latent period in mosquitoes (with defective distribution $\tilde{f}_M(\tau) = f_M(\tau)e^{-\mu\tau}$ because of the potential mortality during the latent period) and of the time from the start of mosquito infectiousness to the transmission event (with exponential distribution of parameter μ).

This means that $a(\tau)$, as defined in (3.4), will be the convolution of $f_H(\tau)$, of $\gamma e^{-\gamma\tau}$, of $f_M(\tau)e^{-\mu\tau}$, and of $\mu e^{-\mu\tau}$. Hence, from the properties of the Laplace transform, we get

$$\hat{a}(\lambda) = \hat{f}_H(\lambda)\frac{\gamma}{\gamma+\lambda}\frac{\hat{f}_M(\lambda+\mu)}{\hat{f}_M(\mu)}\frac{\mu}{\mu+\lambda}.$$

Substituting this expression into (2.9), with r the leading eigenvalue, one obtains

$$1 = b^2\frac{p_{MH}p_{HM}}{\gamma\mu}\frac{\bar{M}}{\bar{H}}\hat{a}(r)\hat{f}_M(\mu) = \mathcal{R}_0^2\hat{a}(r).$$

Consequently, from estimates of the exponential growth rate r and of the distribution of the generation time from human to human (or mosquito to mosquito), it is possible to obtain an estimate of $\mathcal{R}_0^2$ (the type-reproduction number) using exactly the same formula used to obtain $\mathcal{R}_0$ in the case of a directly transmitted infection.

3.2. Probability of a major outbreak

As is well-known, in stochastic models for directly transmitted infections, a major epidemic (i.e., one in which the number of infected individuals is $O(N)$ as N, the population size, grows to infinity [31, p. 13]) may occur only if $\mathcal{R}_0 > 1$. In this case, the probability that a major outbreak occurs can be computed through an equation involving the generating function of the number of individuals infected by a newly infected person over the whole infectious period.

In the case that the model is a continuous-time Markov chain of SIR-type (i.e., the state of the process is given by the number of individuals in the class S, I, or R, and the only possible transitions

are from S to I through a new infection or from I to R through a recovery), the probability that a major epidemic occurs upon the introduction of an infected individual is $\pi = 1 - 1/\mathcal{R}_0$.

Lloyd et al. [32] (see also [33]) have extended the results to the case of vector-borne epidemics, possibly with many species of hosts. One has to distinguish the case of the introduction of an infected host from when an infected vector is introduced. In the first case, let π_H be the probability that a major outbreak occurs, and let $s_H = 1 - \pi_H$ be the probability of early epidemic extinction. In the second case, these quantities will be denoted as π_M and s_M. Furthermore, one has to split the expression (2.4) of $\mathcal{R}_0^2$ into the product of two terms,

$$\mathcal{R}_0^{HM} = \frac{bp_{HM}}{\gamma}\frac{\bar{M}}{\bar{H}}, \qquad \mathcal{R}_0^{MH} = \frac{bp_{MH}}{\mu},$$

representing the expected number of mosquitoes infected by an infected host, or, respectively, the expected number of hosts infected by an infected mosquito. Notice that

$$\mathcal{R}_0^2 = \mathcal{R}_0^{HM}\mathcal{R}_0^{MH}.$$

Using these quantities, it is not difficult to show that

$$\pi_M = \frac{\mathcal{R}_0^{HM}\mathcal{R}_0^{MH} - 1}{\mathcal{R}_0^{HM}(1 + \mathcal{R}_0^{MH})} = \frac{\mathcal{R}_0^2 - 1}{\mathcal{R}_0^{HM}(1 + \mathcal{R}_0^{MH})}, \qquad \pi_H = \frac{\mathcal{R}_0^{HM}\mathcal{R}_0^{MH} - 1}{\mathcal{R}_0^{MH}(1 + \mathcal{R}_0^{HM})} = \frac{\mathcal{R}_0^2 - 1}{\mathcal{R}_0^{MH}(1 + \mathcal{R}_0^{HM})}.$$

The similarity to the case of a directly transmitted infection becomes clearer considering the probabilities of early extinctions

$$s_M = \frac{1 + \mathcal{R}_0^{HM}}{\mathcal{R}_0^{HM}(1 + \mathcal{R}_0^{MH})}, \quad \text{and} \quad s_H = \frac{1 + \mathcal{R}_0^{MH}}{\mathcal{R}_0^{MH}(1 + \mathcal{R}_0^{HM})},$$

so that

$$s_M s_H = \frac{1}{\mathcal{R}_0^{MH}\mathcal{R}_0^{HM}} = \frac{1}{\mathcal{R}_0^2}.$$

Because, for an epidemic of a directly transmitted infection, the probability of early extinction s, after the introduction of one infected individual, is given by $s = 1/\mathcal{R}_0$ (see, for instance, [34, p. 107, Eq. (3.16)]), we obtain that $s_M s_H = s^2$.

3.3. Final epidemic size

It is well-known that in epidemic models for directly transmitted infections, the value of $\mathcal{R}_0$ also determines the final epidemic size [23]. Indeed, for deterministic models of SIR type with any distribution of the infectious period and variable infectiousness during that, it is known that, if an epidemic outbreak occurs in a totally susceptible population with the introduction of an infinitesimal fraction of infected individuals, the susceptible fraction s_∞ at the epidemic end satisfies the equation

$$\log(s_\infty) + \mathcal{R}_0(1 - s_\infty) = 0, \tag{3.6}$$

and the infected fraction is $z = 1 - s_\infty$.

The final epidemic size for vector-transmitted infection has been studied, as far as we know, only in two recent papers by Brauer [35, 36], and, more recently, by other authors in [37, 38]. In [35], Brauer proved bounds for $S_H^\infty := \lim_{t\to+\infty} S_H(t)$, and in [36], he used a timescale separation to obtain an approximate relation. A similar argument was used in [38] to estimate the final size under the assumption $I'_M(t) \approx 0$, whereas in [37], the authors derived approximate and error formulae under the assumption $I_M(t)/\bar{M} \ll 1$ for $t \geq 0$.

Here, we repeat the arguments of [35] on System (2.1), although a more complex system was considered in that paper. Computing $\dfrac{d}{dt}\log(S_H(t))$ from the first equation of (2.1) and integrating it from 0 to $+\infty$, one obtains

$$\log\left(\frac{S_H^\infty}{S_H(0)}\right) = -\frac{bp_{MH}}{\bar{H}}\int_0^{+\infty} I_M(t)\,dt. \tag{3.7}$$

If one writes the second equation of (2.1) as $I'_H = -S'_H - \gamma I_H$, solves it, and then integrates it from 0 to $+\infty$, one obtains

$$I_H(t) = I_H(0)e^{-\gamma t} - \int_0^t S'_H(s)e^{-\gamma(t-s)}\,ds \Longrightarrow \int_0^{+\infty} I_H(t)\,dt = \frac{I_H(0)}{\gamma} + \frac{S_H(0) - S_H^\infty}{\gamma}. \tag{3.8}$$

Finally, solving the equation for $I_M(t)$, assuming $S_M(\cdot)$ and $I_H(\cdot)$ as known, and integrating it from 0 to $+\infty$, one obtains

$$I_M(t) = I_M(0)e^{-\mu t} + \frac{bp_{HM}}{\bar{H}}\int_0^t S_M(s)I_H(s)e^{-\mu(t-s)}\,ds \Longrightarrow \int_0^{+\infty} I_M(t)\,dt = \frac{I_M(0)}{\mu} + \frac{bp_{HM}}{\mu\bar{H}}\int_0^{+\infty} S_M(s)I_H(s)\,ds. \tag{3.9}$$

Assuming $I_M(0) \approx 0$ and $I_H(0) \approx 0$, one has from (3.8) and (3.9),

$$\begin{aligned}\frac{bp_{HM}S_{M,min}(S_H(0) - S_H^\infty)}{\mu\gamma\bar{H}} &= \frac{bp_{HM}S_{M,min}}{\mu\bar{H}}\int_0^{+\infty} I_H(s)\,ds \\ &\leq \int_0^{+\infty} I_M(t)\,dt \leq \frac{bp_{HM}\bar{M}}{\mu\bar{H}}\int_0^{+\infty} I_H(s)\,ds = \frac{bp_{HM}\bar{M}(S_H(0) - S_H^\infty)}{\mu\gamma\bar{H}},\end{aligned} \tag{3.10}$$

where $S_{M,min} := \min_{t\geq 0} S_M(t)$.

Substituting (3.10) in (3.7), and using $s_\infty := S_H^\infty/\bar{H}$ together with $S_H(0) \approx \bar{H}$, one arrives at

$$\log(s_\infty) + \frac{S_{M,min}}{\bar{M}}\mathcal{R}_0^2(1 - s_\infty) \leq 0 \leq \log(s_\infty) + \mathcal{R}_0^2(1 - s_\infty). \tag{3.11}$$

Brauer [35] also obtained a lower bound for $S_{M,min}$, but that is not very sharp.

Consider instead the right inequality in (3.11). Studying the graph of $F(x) := \log(x) + \mathcal{R}_0^2(1 - x)$, one sees that $s_\infty \geq \bar{x}$, where $F(\bar{x}) = 0$. In practice, in the numerical cases that we considered (see Fig. 1), $s_\infty \approx \bar{x}$; presumably, the fraction of infected mosquitoes generally remains relatively small, so that $S_{M,min}$ does not fall much below $\bar{M}$.

In this sense, we could say that (3.6) is approximately correct for vector-transmitted infections, but with $\mathcal{R}_0^2$ substituted for $\mathcal{R}_0$.

As mentioned above, Brauer established $s_\infty \approx \bar{x}$ using a timescale argument. In this notation, he assumed $bp_{HM} = O(\varepsilon^{-1})$ and $\mu = O(\varepsilon^{-1})$ as $\varepsilon \to 0^+$, with further parameters γ and $bp_{MH}\bar{M}/\bar{H}$ as

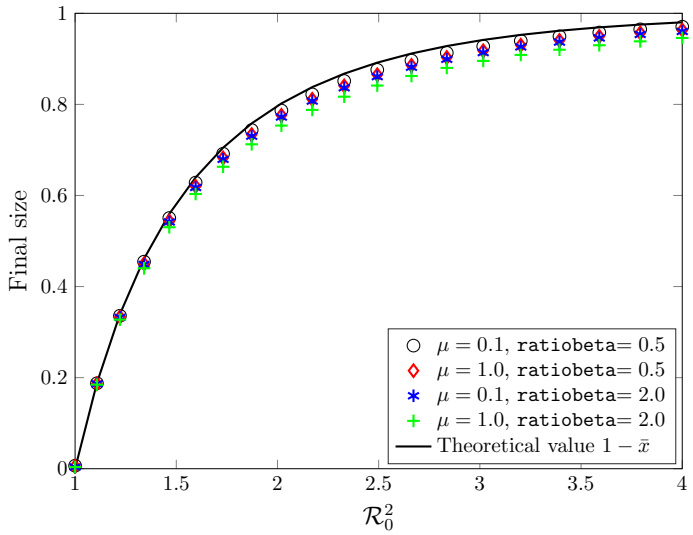


Figure 1. Final epidemic size as a function of $\mathcal{R}_0^2$ for Model (2.1). The line represents $1 - \bar{x}$, where $\bar{x}$ solves $F(\bar{x}) = 0$. The points are obtained from numerical solutions of (2.1) for different values (shown in the legend) of μ and of the ratio $\dfrac{p_{MH}\bar{M}}{p_{HM}\bar{H}}$ (`ratiobeta` in the legend); $\gamma = 1$, $\Lambda = \mu$, and initial conditions $S_H(0) = \bar{H}$, $I_H(0) = 0$, $S_H(0) = \bar{M} - I_M(0)$, and $I_M(0) = 10^{-5}\bar{M}$, and b is adjusted to have the required value of $\mathcal{R}_0^2$.

$O(1)$. On the other hand, most estimates for these parameters for dengue [39], Chikungunya [40], or malaria [41] are of the same order of magnitude. Note, moreover, that the previous estimates on order of magnitudes imply that $\dfrac{p_{MH}\bar{M}}{p_{HM}\bar{H}} = O(\varepsilon)$, whereas in areas where mosquito-borne infections are endemic, it is generally estimated $\bar{M} > \bar{H}$.

4. The endemic model

In order to extend Model (2.1) to study endemicity, one has to complement it with the recruitment and death of humans. We assume that the human population is in demographic equilibrium at level $\bar{H}$, so the rate of new deaths $\mu_H\bar{H}$ is equal to the rate of new births $\Lambda_H = \mu_H\bar{H}$. We thus obtain the system

$$\begin{cases} S'_H = \Lambda_H - bI_M\dfrac{S_H}{\bar{H}}p_{MH} - \mu_H S_H, \\ I'_H = bI_M\dfrac{S_H}{\bar{H}}p_{MH} - (\mu_H + \gamma)I_H, \\ I'_M = b(\bar{M} - I_M)\dfrac{I_H}{\bar{H}}p_{HM} - \mu_M I_M, \end{cases} \tag{4.1}$$

where the equation for S_M is disregarded, because $S_M + I_M \equiv \bar{M}$.

4.1. Existence and stability of equilibria

System (4.1) clearly admits the DFE $E_0 = (\bar{H}, 0, 0)$. A computation, analogous to the one performed in the previous section, shows that E_0 is asymptotically stable (unstable) if $\mathcal{R}_0 < 1$ ($\mathcal{R}_0 > 1$), whereas here,

$$\mathcal{R}_0 = \rho(K) = b\sqrt{\frac{p_{MH}p_{HM}}{(\mu_H+\gamma)\mu_M}\frac{\bar{M}}{\bar{H}}}.$$

The only difference from (2.4) is that the infectious period may end because of the infected host's death (at rate μ_H). This difference is negligible if $\mu_H \ll \gamma$, as for most infections.

Esteva and Vargas [42] considered a model slightly more general than (4.1), as they allowed for host mortality due to infection. They proved that E_0 is globally asymptotically stable if $\mathcal{R}_0 \leq 1$; furthermore, if $\mathcal{R}_0 > 1$, there exists a unique positive equilibrium that is globally asymptotically stable. It must be remarked that, in [42], the authors used M instead of H in the denominator of the first two equations of (4.1). The derivation of the Ross–Macdonald model shows that H naturally occurs in the denominator of both equations. This does not make any difference, as long as host and vector populations are constant, but it does if they vary, as happens if the infection causes additional mortality.

Indeed, Garba, Gumel, and Abu Bakar [43] considered an extension of System (4.1) that allowed for disease-induced mortality (at rate α) in humans and for latent periods in humans and mosquitoes. Neglecting the latent periods (that are not relevant when studying equilibria), their system can be written as

$$\begin{cases} S_H' = \Lambda_H - bI_M\dfrac{S_H}{H}p_{MH} - \mu_H S_H, \\ I_H' = bI_M\dfrac{S_H}{H}p_{MH} - (\mu_H+\gamma+\alpha)I_H, \\ H' = \Lambda_H - \mu_H H - \alpha I_H, \\ I_M' = b(\bar{M}-I_M)\dfrac{I_H}{\bar{H}}p_{HM} - \mu_M I_M. \end{cases} \tag{4.2}$$

In [43], the authors showed that, if there is disease-induced mortality ($\alpha > 0$), System (4.2) exhibits a backward bifurcation at $\mathcal{R}_0 = 1$ under certain conditions on the parameters. This implies that there exists $R_m < 1$ such that, when $\mathcal{R}_0 \in (R_m, 1)$, the DFE is asymptotically stable, but there exist two positive equilibria E_- and E_+ of which E_- is unstable, and E_+ is asymptotically stable. The presence of a backward bifurcation thus makes the elimination of an infectious disease more difficult, as bringing $\mathcal{R}_0$ below 1 may not be enough. It must be added that to show numerical examples of backward bifurcation, the authors in [43–45] had to adjust several parameters away from their reference values, especially increasing disease-induced death rates beyond empirical estimates.

As long as disease-induced mortality is not considered, so that population size of humans and mosquitoes are constant, a very strong threshold property has been established under very general conditions. In [46], the authors considered a model analogous to (2.6), but added constant recruitment and mortality of humans. Allowing for arbitrary distributions of latent periods, they proved that, for $\mathcal{R}_0 \leq 1$, the DFE is globally asymptotically stable, whereas for $\mathcal{R}_0 > 1$, it is unstable, and there exists a unique positive equilibrium that is globally asymptotically stable. The formula for $\mathcal{R}_0$ is similar to (2.10), except that one has to take into account the probability that an infected human dies during the latent period, similarly to the term present for mosquitoes.

This result is similar to what has been proved for directly transmitted infection. In fact, [47] had considered an SEIR model with arbitrary distribution of the infection period and arbitrary infectiousness during the infectious period; they proved that, for $\mathcal{R}_0 \leq 1$, the DFE is globally asymptotically stable, whereas for $\mathcal{R}_0 > 1$, it is unstable, and there exists a unique positive equilibrium that is locally asymptotically stable. [48] completed the analysis by showing that, for $\mathcal{R}_0 > 1$, the positive equilibrium is globally asymptotically stable. In this sense, we may say that transmission through a vector does not change the main properties of the system.

4.2. Seasonality

So far, it has been assumed that all model parameters are constant. However, the population dynamics of mosquitoes are strongly affected by environmental factors. This factor has been considered with two different approaches.

On the one hand, the dependence of mosquito demographic parameters and of the probability of infection transmission (p_{HM} in the current notation) on temperature (and, more rarely, on humidity) has been identified through laboratory studies [49]. A model has been fitted to observed data using observed temperatures [50] and has been extrapolated to the future environmental patterns, predicted from accepted climatic models [51]. There are a number of studies in this direction, which fall outside the scope of this review.

On the other hand, it has been assumed that mosquito population dynamics are periodic (often, for the sake of simplicity, a sinusoidal shape has been used), and the consequences on mosquito-borne infections have been studied [52]. In this direction, the most important theoretical contribution has been the extension of the concept of $\mathcal{R}_0$ to a periodic setting, due to Bacaër and Guernaoui [53] (see also [54]). Building on this, Wang and Zhao [55] gave a definition of $\mathcal{R}_0$ for general epidemic compartmental models with periodic rates, and showed that the disease-free periodic solution (assumed to exist) is asymptotically stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$. They also showed that $\mathcal{R}_0$ can be larger than, equal to, or smaller than the value that would be obtained by considering a system with constant coefficients equal to the time average of the periodic coefficients.

Here, we present the definition of $\mathcal{R}_0$ for the simple case in which Model (4.1) is extended to allow for periodic coefficients. In particular, it seems reasonable to assume that coefficients relative to humans are approximately constant, whereas $\bar{M}(t)$, $\mu(t)$, $b(t)$, and $p_{HM}(t)$ are all ω-periodic functions, depending on environmental conditions. Thus, we consider the system

$$\begin{cases} S'_H(t) = \Lambda_H - b(t)I_M(t)\dfrac{S_H(t)}{\bar{H}}p_{MH} - \mu_H S_H(t), \\ I'_H(t) = b(t)I_M(t)\dfrac{S_H}{\bar{H}}p_{MH} - (\mu_H + \gamma)I_H(t), \\ I'_M(t) = b(t)(\bar{M}(t) - I_M(t))\dfrac{I_H(t)}{\bar{H}}p_{HM}(t) - \mu_M(t)I_M(t). \end{cases}$$

Using the notation of [55], we denote by C_ω the space of ω-periodic continuous functions endowed with the sup-norm, and by C^2_ω its Cartesian product. Then, $\mathcal{R}_0 = \rho(L)$, where the next-infection operator

$L\colon C^2_\omega \to C^2_\omega$ is defined as

$$L\begin{pmatrix}\phi_1\\ \phi_2\end{pmatrix}(t) = \begin{pmatrix} \dfrac{p_{MH}}{\bar{H}} \displaystyle\int_0^{+\infty} e^{-(\mu_H+\gamma)a} b(t-a)\phi_2(t-a)\,da \\ \dfrac{1}{\bar{H}} \displaystyle\int_0^{+\infty} e^{-\int_{t-a}^{t}\mu_M(s)\,ds}\, b(t-a)p_{HM}(t-a)\bar{M}(t-a)\phi_1(t-a)\,da \end{pmatrix}. \tag{4.3}$$

Since L is an infinite-dimensional operator, computing $\mathcal{R}_0$ generally requires approximation methods. See [56] for a review of methods to compute $\mathcal{R}_0$ in the context of periodic epidemic models and an analysis of the cases when one can compute $\mathcal{R}_0$ by considering the corresponding system with constant coefficients equal to the time average of the periodic coefficients. A recent preprint [57] provides a general procedure to approximate $\mathcal{R}_0$ using pseudospectral collocation methods, along the lines of [58, 59] and references therein.

5. Host heterogeneity in host-vector models

There are many sources of heterogeneities in host-vector models. First of all, most mosquitoes are opportunistic feeders; thus, they would feed on several host species. Some of them may be better able to transmit the infection, others less or not at all. Second, even within a host species, some individuals may be more attractive to mosquitoes than others. Finally, considering mosquito-transmitted human infections, some individuals may be less available to mosquito bites (as they use mosquito repellents, covering dresses, or live in air-conditioned settings), whereas others do not use protection measures.

To consider a situation as simple as possible, we follow [60], and consider that the host population H is divided in two groups, H_1 and H_2, that have different probabilities of being bitten by mosquitoes, but for the rest are identical. Furthermore, we allow for the possibility that there are noncompetent secondary hosts, that is, hosts that may be infected by the pathogen under consideration, but cannot transmit the infection to mosquitoes. As an example, for West Nile virus, humans and other mammals, such as horses, can become infected through the bite of an infected mosquito but are considered dead-end hosts, as the virus is mainly transmitted in a cycle involving mosquitoes (vectors) and birds (amplifying hosts) [61]. As for dengue, humans and nonhuman primates are the main reservoir host maintaining the epidemic cycles. However, although some other species, including bats, can be infected, evidence suggests they are likely dead-end hosts, and their role in transmitting the virus to mosquitoes is limited [62]. We let H_1 be the reference (unprotected) population, while the relative probability of biting a host H_2 is $q \in (0, 1)$. As for the other hosts, we assume that their density is W and that the relative (with respect to an unprotected human) probability of biting one of these animals is p_W. We can then let $L = p_W W$ be the "equivalent size", in terms of attractiveness to mosquitoes, of the other hosts. Specifically, letting p the fraction of H_2 hosts ($H_2 = pH$), we assume that mosquito bites are distributed among H_1, H_2, and other hosts as $P_b^{H_1}(p, q)$, $P_b^{H_2}(p, q)$, and $P_b^L(p, q)$, where

$$P_b^{H_1}(p,q) \coloneqq \frac{H_1}{H_1 + qH_2 + L} = \frac{(1-p)H}{c(p,q)H + L}, \qquad P_b^{H_2}(p,q) \coloneqq \frac{qH_2}{H_1 + qH_2 + L} = \frac{pqH}{c(p,q)H + L},$$

and

$$P_b^L(p,q) = \frac{L}{H_1 + qH_2 + L},$$

with $c(p,q) := 1 - p(1-q)$.

Now, we extend System (2.1) to allow for the two groups of hosts, letting S_i and I_i denote the susceptible and infected individuals of group $i = 1, 2$. Under the classical Ross–Macdonald assumptions, as in Model (2.1), we assume that mosquito biting rate b is constant, independent of p, q, H, and L (notice, however, that a different assumption, where biting rate depends on host density, is used in [41]). We then obtain [60]

$$\begin{cases} S_1' = -bp_{MH}I_M\dfrac{S_1}{c(p,q)H+L}, \\ I_1' = bp_{MH}I_M\dfrac{S_1}{c(p,q)H+L} - \gamma I_1, \\ S_2' = -bp_{MH}I_M\dfrac{qS_2}{c(p,q)H+L}, \\ I_2' = bp_{MH}I_M\dfrac{qS_2}{c(p,q)H+L} - \gamma I_2, \\ S_M' = \Lambda - bp_{HM}S_M\dfrac{qI_2+I_1}{c(p,q)H+L} - \mu S_M, \\ I_M' = bp_{HM}S_M\dfrac{qI_2+I_1}{c(p,q)H+L} - \mu I_M. \end{cases} \tag{5.1}$$

5.1. *Effect of host heterogeneity on* $\mathcal{R}_0$

Linearizing (5.1) near the DFE, where $S_1 = (1-p)H$, $S_2 = pH$, and $S_M = \bar{M} = \Lambda/\mu$, we obtain

$$\begin{cases} I_1' = bp_{MH}I_M\dfrac{(1-p)H}{c(p,q)H+L} - \gamma I_1, \\ I_2' = bp_{MH}I_M\dfrac{qpH}{c(p,q)H+L} - \gamma I_2, \\ I_M' = bp_{HM}\bar{M}\dfrac{qI_2+I_1}{c(p,q)H+L} - \mu I_M. \end{cases} \tag{5.2}$$

The system (5.2) can be written as

$$\begin{pmatrix} I_1 \\ I_2 \\ I_M \end{pmatrix}' = A\begin{pmatrix} I_1 \\ I_2 \\ I_M \end{pmatrix},$$

where, following [21, 22], we write $A = B - D$, with $B(p,q)$ accounting for infection,

$$B(p,q) := \begin{pmatrix} 0 & 0 & bp_{MH}\dfrac{(1-p)H}{c(p,q)H+L} \\ 0 & 0 & bp_{MH}\dfrac{qpH}{c(p,q)H+L} \\ \dfrac{bp_{HM}\bar{M}}{c(p,q)H+L} & \dfrac{qbp_{HM}\bar{M}}{c(p,q)H+L} & 0 \end{pmatrix}, \quad p,q \in [0,1],$$

and D accounting for transition

$$D := \begin{pmatrix} \gamma & 0 & 0 \\ 0 & \gamma & 0 \\ 0 & 0 & \mu \end{pmatrix}.$$

According to [24] , $\mathcal{R}_0^{\text{het}}(p,q)$ is the spectral radius of the NGM

$$K(p,q) := B(p,q)D^{-1},$$

and it explicitly reads [60]

$$\mathcal{R}_0^{\text{het}}(p,q) = \mathcal{R}_0^{\text{hom}} \frac{H}{c(p,q)H + L} \sqrt{1 - p + q^2 p}, \tag{5.3}$$

where

$$\mathcal{R}_0^{\text{hom}} = b \sqrt{\frac{p_{MH} p_{HM}}{\gamma \mu} \frac{\bar{M}}{H}}$$

is the expression found in (2.4) with no heterogeneity in main hosts and no secondary hosts.

There are two simple cases in which one can see the implications of (5.3): when there is no heterogeneity in hosts, and when there are no secondary hosts. In the first case ($p = 0$ or $q = 1$), Expression (5.3) becomes $\mathcal{R}_0^{\text{hom}} \frac{H}{H+L}$. This means that the presence of secondary hosts that do not transmit the infection decreases $\mathcal{R}_0$, and hence, the potential for infection outbreak. This rather intuitive conclusion has been named the *dilution effect*, especially in the context of tick-borne infections [63–66].

The second case occurs when $L = 0$, that is, in the absence of secondary noncompetent hosts. Then

$$\mathcal{R}_0^{\text{het}}(p,q) = \mathcal{R}_0^{\text{hom}} \frac{\sqrt{1 - p + q^2 p}}{1 - p + qp}.$$

Letting

$$f(p,q) = \frac{\mathcal{R}_0^{\text{het}}(p,q)}{\mathcal{R}_0^{\text{hom}}} = \frac{\sqrt{1 - p + q^2 p}}{1 - p + qp},$$

an easy computation shows that

$$\frac{\partial f}{\partial q} = -\frac{p(1-p)(1-q)}{(1 - p + qp)^2 \sqrt{1 - p + q^2 p}} < 0$$

if $q \in [0, 1)$ and $p \in (0, 1)$. Since, obviously, $f(p, 1) = 1$, it follows that $f(p, q) > 1$ for any $q \in [0, 1)$ and $p \in (0, 1)$.

Thus, it has been shown that when there are no secondary hosts, host heterogeneity increases $\mathcal{R}_0$; furthermore, the smaller is q, that is, the relative attractiveness of H_2, the larger is $\mathcal{R}_0$. Note that the expression of $f(p, q)$ is undefined when $p = 1$ and $q = 0$, that is, all hosts are completely unattractive; this is an extreme case that is not compatible with the assumption of Ross–Macdonald model.

Finally, it is worth looking at Expression (5.3) when there are both secondary hosts and heterogeneity in main hosts ($L > 0$, $p \in (0, 1)$ and $q < 1$). As discussed above, the presence of secondary hosts

tends to decrease $\mathcal{R}_0$, whereas host heterogeneity tends to increase it. A simple computation (see [60]) shows that $\mathcal{R}_0^{\text{het}}(p, q) > \mathcal{R}_0^{\text{het}}(p, 1)$ (i.e., when there is no heterogeneity in attractiveness) when

$$\frac{L}{H} > \frac{(1-p)(1-q)}{\sqrt{1-p(1-q^2)}+q}.$$

Depending on the values of L, p, and q, it is then possible that heterogeneity increases or decreases $\mathcal{R}_0$.

The fact that host heterogeneity (in the absence of secondary hosts) always increases $\mathcal{R}_0$ had first been found by Dye and Hasibeder [67] and in a subsequent paper explored the effect of heterogeneity on final epidemic size [68].

5.2. Implications for human protective behavior adoption

In recent years, the field of *behavioral epidemiology* of infectious diseases has attracted increasing attention, leading to the development of different mathematical frameworks to incorporate behavioral dynamics into infectious disease models, drawing approaches from different behavioral sciences (e.g., economics and sociology) [69]. There is extensive literature dealing with the mathematical modeling of the interplay between the spread of directly transmitted infectious diseases and human behavior; see the monograph [69] and recent reviews [70–72] for an overview of the topic. In contrast, the role of human behavior in the modeling of vector-borne diseases has received less attention, and has often been underrated.

In this setting, the results of Section 5 have raised a renewed attention, as H_2 may be interpreted as individuals who adopt a preventive behavior. In fact, a model similar to (5.1) (without secondary hosts but with mosquito biting rate depending on the number of hosts according to a Holling-type function) has been studied in [73]. Therein, the authors identified parameter regions in which preventive behavior with incomplete coverage may increase coverage risk. Several recent papers build on these ideas; for instance, in [74], the authors showed that a dynamical shift of individuals from preventive to unprotected behavior, and vice versa, reduces the potential increase of $\mathcal{R}_0$, relative to models where behavior is fixed. Although [73, 74] mostly argued in terms of $\mathcal{R}_0$, other recent papers proposed mathematical models to investigate the role of behavioral changes in the host population (either induced by public health policies or influenced by peers) on the transient dynamic of a vector-host epidemic [60, 75–79].

Modeling human protective behavior in the context of vector-borne infections raises many challenges, as its effectiveness on the overall dynamic and controllability of an epidemic depends on the model assumptions on the interaction between vectors and hosts (e.g., how the mosquito biting rates depend on host availability) [74]; see, for instance, [80–82].

To conclude this short excursus on measures of personal protection, we remark that there is a potential conflict of interest. In fact, we showed that, accepting the main ideas of the Ross–Macdonald model, there are parameter regions in which the adoption of preventive behaviors from part of the population may increase the risk of an outbreak, especially if the preventive measures are very effective (q close to 0). On the other hand, in the same parameter regions, the final epidemic size in the protected group may decrease with q. This means that preventive behaviors may be favorable for the individuals that adopt them, but cause a higher risk for the general population. It seems useful to better understand the circumstances in which this may occur, and also to appropriately guide public health communication on this issue.

6. Conclusions

From this brief overview, it should emerge that, by and large, models for vector-borne infections share many properties with the corresponding models for directly-transmitted infectious diseases. An interesting fact is that some quantities, as, for instance, the final epidemic size, seem to depend not so much on $\mathcal{R}_0$, which is defined as the spectral radius of the NGM (and more in general of the next-generation operator [24]), but rather on $\mathcal{R}_0^2$, the quantity used by Ross, which in current language is a type-reproduction number.

An important feature of mosquito abundance and behavior is their seasonality. Consequently, the study of vector-borne infections has led to an appropriate definition of $\mathcal{R}_0$ in epidemic models with periodic coefficients; it can be proved that an epidemic can persist in a periodic environment if and only if $\mathcal{R}_0 > 1$. On the other hand, the analysis of outbreak risk generally concentrates on the values of $\mathcal{R}_0$ computed using the parameter values at a given t, as if an epidemic outbreak were instantaneous. However, relying only on the value of $\mathcal{R}_0$ computed, using a formula such as (4.3), on the basis of the parameter values over a whole period (generally one year), neglects the risk that outbreaks that may last only a few months pose to public health. It would be useful to find a practical way to bridge these scales, and to be able to find a way to quantify outbreak risks, taking into account the seasonality of parameters but on a timescale appropriate to an epidemic outbreak.

Host heterogeneity itself introduces features specific of vector-borne infections. Although for directly-transmitted infections, preventive behavior by some individuals is beneficial to everybody, as it reduces the contact rates [69], in vector-borne infections, protective measures by some individuals may concentrate mosquito bites on the unprotected individuals [83, 84] and thus facilitate the transmission cycle from a mosquito to a host and then to another mosquito [73]. This always occurs under the idealized assumptions of the Ross–Macdonald model, but the same principle holds if one relaxes the assumptions, for instance by including other potential hosts that do not transmit the infection, or by letting the mosquito biting rate depend on host population size. This potentially raises a conflict of interest, as we showed that the adoption of preventive protective behaviors from part of the population may increase the risk for the general population. It seems useful to better understand the circumstances in which this may occur and to appropriately guide public health communication on this issue. As the role of human behavior in the modeling of vector-borne diseases has often been underrated, this area of research could bear fruitful future investigations.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there is no conflict of interest.

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