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BIFURCATION ANALYSIS OF A MATHEMATICAL MODEL FOR MALARIA TRANSMISSION*

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Abstract. We present an ordinary differential equation mathematical model for the spread of malaria in human and mosquito populations. Susceptible humans can be infected when they are bitten by an infectious mosquito. They then progress through the exposed, infectious, and recovered classes, before reentering the susceptible class. Susceptible mosquitoes can become infected when they bite infectious or recovered humans, and once infected they move through the exposed and infectious classes. Both species follow a logistic population model, with humans having immigration and disease-induced death. We define a reproductive number, R_0, for the number of secondary cases that one infected individual will cause through the duration of the infectious period. We find that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. We prove the existence of at least one endemic equilibrium point for all $R_0 > 1$. In the absence of disease-induced death, we prove that the transcritical bifurcation at $R_0 = 1$ is supercritical (forward). Numerical simulations show that for larger values of the disease-induced death rate, a subcritical (backward) bifurcation is possible at $R_0 = 1$.

Key words. malaria, epidemic model, reproductive number, bifurcation theory, endemic equilibria, disease-free equilibria

AMS subject classifications. Primary, 92D30; Secondary, 37N25

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1. Introduction.

Malaria is an infectious disease caused by the *Plasmodium* parasite and transmitted between humans through the bite of the female *Anopheles* mosquito. An estimated 40% of the world's population live in malaria endemic areas. The disease kills about 1 to 3 million people a year, 75% of whom are African children. The incidence of malaria has been growing recently due to increasing parasite drug-resistance and mosquito insecticide-resistance. Therefore, it is important to understand the important parameters in the transmission of the disease and develop effective solution strategies for its prevention and control.

Mathematical modeling of malaria began in 1911 with Ross's model [25], and major extensions are described in Macdonald's 1957 book [20]. The first models were two-dimensional with one variable representing humans and the other representing mosquitoes. An important addition to the malaria models was the inclusion of acquired immunity proposed by Dietz, Molineaux, and Thomas [11]. Further work on acquired immunity in malaria has been conducted by Aron [2] and Bailey [5]. Anderson and May [1], Aron and May [3], Koella [15] and Nedelman [21] have written some good reviews on the mathematical modeling of malaria. Some recent papers have also included environmental effects [19], [27], and [28]; the spread of resistance to drugs

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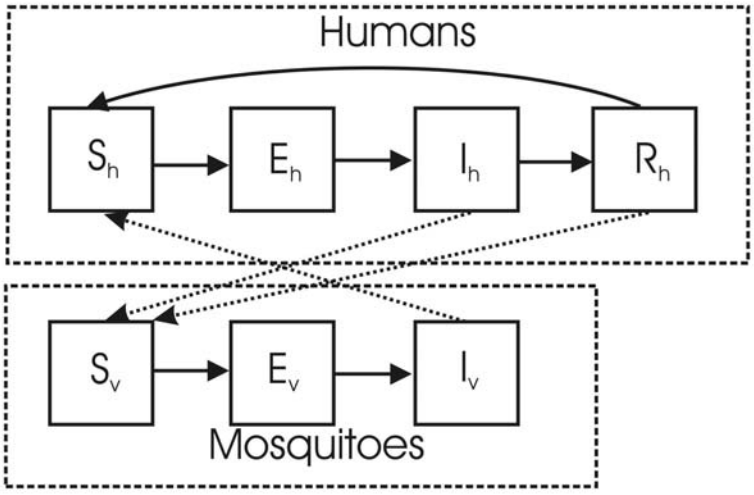

FIG. 1.1. *Susceptible humans, S_h, can be infected when they are bitten by infectious mosquitoes. They then progress through the exposed, E_h, infectious, I_h, and recovered, R_h, classes, before re-entering the susceptible class. Susceptible mosquitoes, S_v, can become infected when they bite infectious or recovered humans. The infected mosquitoes then move through the exposed, E_v, and infectious, I_v, classes. Both species follow a logistic population model, with humans having additional immigration and disease-induced death. Birth, death, and migration into and out of the population are not shown in the figure.*

[4] and [16]; and the evolution of immunity [17].

Recently, Ngwa and Shu [23] and Ngwa [22] proposed an ordinary differential equation (ODE) compartmental model for the spread of malaria with a susceptible-exposed-infectious-recovered-susceptible (SEIRS) pattern for humans and a susceptible-exposed-infectious (SEI) pattern for mosquitoes. In a Ph.D. dissertation, Chitnis [7] analyzed a similar model for malaria transmission. In this paper we extend the Chitnis model.

The new model (Figure 1.1) divides the human population into four classes: susceptible, S_h; exposed, E_h; infectious, I_h; and recovered (immune), R_h. People enter the susceptible class either through birth (at a constant per capita rate) or through immigration (at a constant rate). When an infectious mosquito bites a susceptible human, there is some finite probability that the parasite (in the form of sporozoites) will be passed on to the human and that the person will move to the exposed class. The parasite then travels to the liver where it develops into its next life stage. After a certain period of time, the parasite (in the form of merozoites) enters the blood stream, usually signaling the clinical onset of malaria. In our model, people from the exposed class enter the infectious class at a rate that is the reciprocal of the duration of the latent period. After some time, the infectious humans recover and move to the recovered class. The recovered humans have some immunity to the disease and do not get clinically ill, but they still harbor low levels of parasite in their blood streams and can pass the infection to mosquitoes. After some period of time, they lose their immunity and return to the susceptible class. Humans leave the population through a density-dependent per capita emigration and natural death rate, and through a per capita disease-induced death rate.

We divide the mosquito population into three classes: susceptible, S_v; exposed,

E_v; and infectious, I_v. Female mosquitoes (we do not include male mosquitoes in our model because only female mosquitoes bite animals for blood meals) enter the susceptible class through birth. The parasite (in the form of gametocytes) enters the mosquito with some probability when the mosquito bites an infectious human or a recovered human (the probability of transmission of infection from a recovered human is much lower than that from an infectious human), and the mosquito moves from the susceptible to the exposed class. After some period of time, dependent on the ambient temperature and humidity, the parasite develops into sporozoites and enters the mosquito's salivary glands, and the mosquito moves from the exposed class to the infectious class. The mosquito remains infectious for life. Mosquitoes leave the population through a per capita density-dependent natural death rate.

The extension of the Ngwa and Shu model [23] includes human immigration, excludes direct human recovery from the infectious to the susceptible class, and generalizes the mosquito biting rate so that it applies to wider ranges of populations. In [23], the total number of mosquito bites on humans depends only on the number of mosquitoes, while in our model, the total number of bites depends on both the human and mosquito population sizes. Human migration is present throughout the world and plays a large role in the epidemiology of diseases, including malaria. In many parts of the developing world, there is rapid urbanization as many people leave rural areas and migrate to cities in search of employment. We include this movement as a constant immigration rate into the susceptible class. We do not include immigration of infectious humans, as we assume that most people who are sick will not travel. We also exclude the movement of exposed humans because, given the short time of the exposed stage, the number of exposed people is small. We make the simplifying assumption that there is no immigration of recovered humans. We also exclude the direct infectious-to-susceptible recovery that the model of Ngwa and Shu [23] contains. This is a realistic simplifying assumption because most people show some period of immunity before becoming susceptible again. As our model includes an exponential distribution of movement from the recovered to the susceptible class, it will include the quick return to susceptibility of some individuals. The model in Chitnis [7] is the same as the model in this paper except for the mosquito biting rate, which is the same as in [23].

We first describe the mathematical model including the definition of a domain where the model is mathematically and epidemiologically well-posed. Next, we prove the existence and stability of a disease-free equilibrium point, define the reproductive number, and describe the existence and stability of the endemic equilibrium point(s).

2. Malaria model. The state variables (Table 2.1) and parameters (Table 2.2) for the malaria model (Figure 1.1) satisfy the equations in (2.1). All parameters

TABLE 2.1
The state variables for the malaria model (2.1).

S_h:	Number of susceptible humans
E_h:	Number of exposed humans
I_h:	Number of infectious humans
R_h:	Number of recovered (immune and asymptomatic, but slightly infectious) humans
S_v:	Number of susceptible mosquitoes
E_v:	Number of exposed mosquitoes
I_v:	Number of infectious mosquitoes
N_h:	Total human population
N_v:	Total mosquito population

TABLE 2.2
The parameters for the malaria model (2.1) *and their dimensions.*

Λ_h:	Immigration rate of humans. Humans $\times$ Time^{-1}.
ψ_h:	Per capita birth rate of humans. Time^{-1}.
ψ_v:	Per capita birth rate of mosquitoes. Time^{-1}.
σ_v:	Number of times one mosquito would want to bite humans per unit time, if humans were freely available. This is a function of the mosquito's gonotrophic cycle (the amount of time a mosquito requires to produce eggs) and its anthropophilic rate (its preference for human blood). Time^{-1}.
σ_h:	The maximum number of mosquito bites a human can have per unit time. This is a function of the human's exposed surface area. Time^{-1}.
β_{hv}:	Probability of transmission of infection from an infectious mosquito to a susceptible human, given that a contact between the two occurs. Dimensionless.
β_{vh}:	Probability of transmission of infection from an infectious human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless.
$\tilde{\beta}_{vh}$:	Probability of transmission of infection from a recovered (asymptomatic carrier) human to a susceptible mosquito, given that a contact between the two occurs. Dimensionless.
ν_h:	Per capita rate of progression of humans from the exposed state to the infectious state. $1/\nu_h$ is the average duration of the latent period. Time^{-1}.
ν_v:	Per capita rate of progression of mosquitoes from the exposed state to the infectious state. $1/\nu_v$ is the average duration of the latent period. Time^{-1}.
γ_h:	Per capita recovery rate for humans from the infectious state to the recovered state. $1/\gamma_h$ is the average duration of the infectious period. Time^{-1}.
δ_h:	Per capita disease-induced death rate for humans. Time^{-1}.
ρ_h:	Per capita rate of loss of immunity for humans. $1/\rho_h$ is the average duration of the immune period. Time^{-1}.
μ_{1h}:	Density-independent part of the death (and emigration) rate for humans. Time^{-1}.
μ_{2h}:	Density-dependent part of the death (and emigration) rate for humans. Humans^{-1} $\times$ Time^{-1}.
μ_{1v}:	Density-independent part of the death rate for mosquitoes. Time^{-1}.
μ_{2v}:	Density-dependent part of the death rate for mosquitoes. Mosquitoes^{-1} $\times$ Time^{-1}.

are strictly positive with the exception of the disease-induced death rate, δ_h, which is nonnegative. The mosquito birth rate is greater than the density-independent mosquito death rate, $\psi_v > \mu_{1v}$, ensuring that we have a stable positive mosquito population.

$$\frac{dS_h}{dt} = \Lambda_h + \psi_h N_h + \rho_h R_h - \lambda_h(t) S_h - f_h(N_h) S_h, \tag{2.1a}$$

$$\frac{dE_h}{dt} = \lambda_h(t) S_h - \nu_h E_h - f_h(N_h) E_h, \tag{2.1b}$$

$$\frac{dI_h}{dt} = \nu_h E_h - \gamma_h I_h - f_h(N_h) I_h - \delta_h I_h, \tag{2.1c}$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \rho_h R_h - f_h(N_h) R_h, \tag{2.1d}$$

$$\frac{dS_v}{dt} = \psi_v N_v - \lambda_v(t) S_v - f_v(N_v) S_v, \tag{2.1e}$$

$$\frac{dE_v}{dt} = \lambda_v(t) S_v - \nu_v E_v - f_v(N_v) E_v, \tag{2.1f}$$

$$\frac{dI_v}{dt} = \nu_v E_v - f_v(N_v) I_v, \tag{2.1g}$$

where $f_h(N_h) = \mu_{1h} + \mu_{2h} N_h$ is the per capita density-dependent death and emigration rate for humans and $f_v(N_v) = \mu_{1v} + \mu_{2v} N_v$ is the per capita density-dependent death rate for mosquitoes. The total population sizes are $N_h = S_h + E_h + I_h + R_h$ and

$N_v = S_v + E_v + I_v$, with

$$\frac{dN_h}{dt} = \Lambda_h + \psi_h N_h - f_h(N_h)N_h - \delta_h I_h, \tag{2.2a}$$

$$\frac{dN_v}{dt} = \psi_v N_v - f_v(N_v)N_v, \tag{2.2b}$$

and the infection rates are

$$\lambda_h = b_h(N_h, N_v) \cdot \beta_{hv} \cdot \frac{I_v}{N_v} \quad \text{and} \quad \lambda_v = b_v(N_h, N_v) \cdot \left(\beta_{vh} \cdot \frac{I_h}{N_h} + \tilde{\beta}_{vh} \cdot \frac{R_h}{N_h}\right). \tag{2.3}$$

We define the force of infection from mosquitoes to humans, λ_h, as the product of the number of mosquito bites that one human has per unit time, b_h, the probability of disease transmission from the mosquito to the human, β_{hv}, and the probability that the mosquito is infectious, I_v/N_v. We define the force of infection from humans to mosquitoes, λ_v, as the sum of the force of infection from infectious humans and from recovered humans. These are defined as the number of human bites one mosquito has per unit time, b_v; the probability of disease transmission from the human to the mosquito, β_{vh} and $\tilde{\beta}_{vh}$; and the probability that the human is infectious or recovered, I_h/N_h and R_h/N_h. Here, we model the total number of mosquito bites on humans as

$$b = b(N_h, N_v) = \frac{\sigma_v N_v \sigma_h N_h}{\sigma_v N_v + \sigma_h N_h} = \frac{\sigma_v \sigma_h}{\sigma_v (N_v/N_h) + \sigma_h} N_v, \tag{2.4}$$

so that the total number of mosquito-human contacts depends on the populations of both species. We define $b_h = b_h(N_h, N_v) = b(N_h, N_v)/N_h$ as the number of bites per human per unit time, and $b_v = b_v(N_h, N_v) = b(N_h, N_v)/N_v$ as the number of bites per mosquito per unit time. In the limit that the mosquito population goes to zero or the human population goes to infinity, the model reduces to that in Chitnis [7] and has the same mosquito-human interaction as in Ngwa and Shu [23] and the Ross–Macdonald model (as described by Anderson and May [1]), where the total number of bites is limited by the mosquito population. The number of bites per mosquito is then σ_v (denoted by σ_{vh} in [7]), and the number of bites per human is $\sigma_v N_v/N_h$. We show a summary of the model of mosquito-human interactions and its limits in Table 2.3.

TABLE 2.3
Number of mosquito bites on humans in the malaria transmission model (2.1) *and its limiting cases with population changes.*

	Number of bites per human, b_h	Number of bites per mosquito, b_v	Total number of bites, b
General model	$\frac{\sigma_v N_v \sigma_h}{\sigma_v N_v + \sigma_h N_h}$	$\frac{\sigma_v \sigma_h N_h}{\sigma_v N_v + \sigma_h N_h}$	$\frac{\sigma_v N_v \sigma_h N_h}{\sigma_v N_v + \sigma_h N_h}$
As $N_h \to \infty$ or $N_v \to 0$	$\frac{\sigma_v N_v}{N_h}$	σ_v	$\sigma_v N_v$
As $N_h \to 0$ or $N_v \to \infty$	σ_h	$\frac{\sigma_h N_h}{N_v}$	$\sigma_h N_h$

To simplify the analysis of the malaria model (2.1), we work with fractional quantities instead of actual populations by scaling the population of each class by the

total species population. We let

$$e_h = \frac{E_h}{N_h}, \quad i_h = \frac{I_h}{N_h}, \quad r_h = \frac{R_h}{N_h}, \quad e_v = \frac{E_v}{N_v}, \quad \text{and} \quad i_v = \frac{I_v}{N_v}, \tag{2.5}$$

with

$$S_h = s_h N_h = (1 - e_h - i_h - r_h)N_h \quad \text{and} \quad S_v = s_v N_v = (1 - e_v - i_v)N_v. \tag{2.6}$$

Differentiating the scaling equations (2.5) and solving for the derivatives of the scaled variables, we obtain

$$\frac{de_h}{dt} = \frac{1}{N_h}\left[\frac{dE_h}{dt} - e_h\frac{dN_h}{dt}\right] \quad \text{and} \quad \frac{de_v}{dt} = \frac{1}{N_v}\left[\frac{dE_v}{dt} - e_v\frac{dN_v}{dt}\right] \tag{2.7}$$

and so on for the other variables.

This creates a new seven-dimensional system of equations with two dimensions for the two total population variables, N_h and N_v, and five dimensions for the fractional population variables with disease, e_h, i_h, r_h, e_v, and i_v:

$$\frac{de_h}{dt} = \left(\frac{\sigma_v\sigma_h N_v\beta_{hv}i_v}{\sigma_v N_v + \sigma_h N_h}\right)(1 - e_h - i_h - r_h) - \left(\nu_h + \psi_h + \frac{\Lambda_h}{N_h}\right)e_h + \delta_h i_h e_h, \tag{2.8a}$$

$$\frac{di_h}{dt} = \nu_h e_h - \left(\gamma_h + \delta_h + \psi_h + \frac{\Lambda_h}{N_h}\right)i_h + \delta_h i_h^2, \tag{2.8b}$$

$$\frac{dr_h}{dt} = \gamma_h i_h - \left(\rho_h + \psi_h + \frac{\Lambda_h}{N_h}\right)r_h + \delta_h i_h r_h, \tag{2.8c}$$

$$\frac{dN_h}{dt} = \Lambda_h + \psi_h N_h - (\mu_{1h} + \mu_{2h}N_h)N_h - \delta_h i_h N_h, \tag{2.8d}$$

$$\frac{de_v}{dt} = \left(\frac{\sigma_v\sigma_h N_h}{\sigma_v N_v + \sigma_h N_h}\right)\left(\beta_{vh}i_h + \tilde{\beta}_{vh}r_h\right)(1 - e_v - i_v) - (\nu_v + \psi_v)e_v, \tag{2.8e}$$

$$\frac{di_v}{dt} = \nu_v e_v - \psi_v i_v, \tag{2.8f}$$

$$\frac{dN_v}{dt} = \psi_v N_v - (\mu_{1v} + \mu_{2v}N_v)N_v. \tag{2.8g}$$

The model (2.8) is epidemiologically and mathematically well-posed in the domain

$$\mathcal{D} = \left\{ \begin{pmatrix} e_h \\ i_h \\ r_h \\ N_h \\ e_v \\ i_v \\ N_v \end{pmatrix} \in \mathbb{R}^7 \;\middle|\; \begin{array}{c} e_h \geq 0, \\ i_h \geq 0, \\ r_h \geq 0, \\ e_h + i_h + r_h \leq 1, \\ N_h > 0, \\ e_v \geq 0, \\ i_v \geq 0, \\ e_v + i_v \leq 1, \\ N_v > 0 \end{array} \right\}. \tag{2.9}$$

This domain, $\mathcal{D}$, is valid epidemiologically as the fractional populations e_h, i_h, r_h, e_v, and i_v are all nonnegative and have sums over their species type that are less than or equal to 1. The human and mosquito populations, N_h and N_v, are positive. We use the notation f' to denote df/dt. We denote points in $\mathcal{D}$ by $x = (e_h, i_h, r_h, N_h, e_v, i_v, N_v)$.

THEOREM 2.1. *Assuming that the initial conditions lie in $\mathcal{D}$, the system of equations for the malaria model* (2.8) *has a unique solution that exists and remains in $\mathcal{D}$ for all time $t \geq 0$.*

Proof. The right-hand side of (2.8) is continuous with continuous partial derivatives in $\mathcal{D}$, so (2.8) has a unique solution. We now show that $\mathcal{D}$ is forward-invariant. We can see from (2.8) that if $e_h = 0$, then $e_h' \geq 0$; if $i_h = 0$, then $i_h' \geq 0$; if $r_h = 0$, then $r_h' \geq 0$; if $e_v = 0$, then $e_v' \geq 0$; and if $i_v = 0$, then $i_v' \geq 0$. It is also true that if $e_h + i_h + r_h = 1$, then $e_h' + i_h' + r_h' < 0$; and if $e_v + i_v = 1$, then $e_v' + i_v' < 0$. Finally, we note that if $N_h = 0$, then $N_h' > 0$ and if $N_v = 0$, then $N_v' = 0$. If $N_h > 0$ at time $t = 0$, then $N_h > 0$ for all $t > 0$. Similarly, if $N_v > 0$ at time $t = 0$, then $N_v > 0$ for all $t > 0$. Therefore, none of the orbits can leave $\mathcal{D}$, and a unique solution exists for all time. ∎

3. Disease-free equilibrium point and reproductive number.

3.1. Existence of the disease-free equilibrium point. Disease-free equilibrium points are steady-state solutions where there is no disease. We define the "*diseased*" classes as the human or mosquito populations that are either exposed, infectious, or recovered, that is, e_h, i_h, r_h, e_v, and i_v. We denote the positive orthant in $\mathbb{R}^7$ by $\mathbb{R}^7_+$, and the boundary of $\mathbb{R}^7_+$ by $\partial\mathbb{R}^7_+$. The positive equilibrium human and mosquito population values, in the absence of disease, for (2.8) are

$$N_h^* = \frac{(\psi_h - \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}}{2\mu_{2h}} \quad \text{and} \quad N_v^* = \frac{\psi_v - \mu_{1v}}{\mu_{2v}}. \tag{3.1}$$

THEOREM 3.1. *The malaria model* (2.8) *has exactly one equilibrium point, $x_{dfe} = (0, 0, 0, N_h^*, 0, 0, N_v^*)$, with no disease in the population (on $\mathcal{D} \cap \partial\mathbb{R}^7_+$).*

Proof. We need to show that x_{dfe} is an equilibrium point of (2.8) and that there are no other equilibrium points on $\mathcal{D} \cap \partial\mathbb{R}^7_+$. Substituting x_{dfe} into (2.8) shows all derivatives equal to zero, so x_{dfe} is an equilibrium point. We know from Lemma A.1 that on $\mathcal{D} \cap \partial\mathbb{R}^7_+$, $e_h = i_h = r_h = e_v = i_v = 0$. For $i_h = 0$, the only equilibrium point for N_h from (2.8d) is N_h^*, and the only equilibrium point for N_v in $\mathcal{D}$ from (2.8g) is N_v^*. Thus, the only equilibrium point on $\mathcal{D} \cap \partial\mathbb{R}^7_+$ is x_{dfe}. ∎

3.2. Reproductive number. We use the next generation operator approach as described by Diekmann, Heesterbeek, and Metz in [10] to define the reproductive number, R_0, as the number of secondary infections that one infectious individual would create over the duration of the infectious period, provided that everyone else is susceptible. We define the next generation operator, K, which provides the number of secondary infections in humans and mosquitoes caused by one generation of infectious humans and mosquitoes, as

$$K = \begin{pmatrix} 0 & K_{hv} \\ K_{vh} & 0 \end{pmatrix}, \tag{3.2}$$

where we use the following definitions:

K_{hv}: The number of humans that one mosquito infects through its infectious lifetime, assuming all humans are susceptible.

K_{vh}: The number of mosquitoes that one human infects through the duration of the infectious period, assuming all mosquitoes are susceptible.

Using the ideas of Hyman and Li [14], we define K_{hv} and K_{vh} as products of the probability of surviving till the infectious state, the number of contacts per unit

time, the probability of transmission per contact, and the duration of the infectious period:

$$K_{hv} = \left(\frac{\nu_v}{\nu_v + \mu_{1v} + \mu_{2v}N_v^*}\right) \cdot b_v^* \cdot \beta_{hv} \cdot \left(\frac{1}{\mu_{1v} + \mu_{2v}N_v^*}\right), \tag{3.3a}$$

$$\begin{aligned} K_{vh} = &\left(\frac{\nu_h}{\nu_h + \mu_{1h} + \mu_{2h}N_h^*}\right) \cdot b_h^* \cdot \beta_{vh} \cdot \left(\frac{1}{\gamma_h + \delta_h + \mu_{1h} + \mu_{2h}N_h^*}\right) \\ &+ \left(\frac{\nu_h}{\nu_h + \mu_{1h} + \mu_{2h}N_h^*} \cdot \frac{\gamma_h}{\gamma_h + \delta_h + \mu_{1h} + \mu_{2h}N_h^*}\right) \\ &\cdot b_h^* \cdot \tilde{\beta}_{vh} \cdot \left(\frac{1}{\rho_h + \mu_{1h} + \mu_{2h}N_h^*}\right). \end{aligned} \tag{3.3b}$$

In (3.3a), $\nu_v/(\nu_v + \mu_{1v} + \mu_{2v}N_v^*)$ is the probability that a mosquito will survive the exposed state to become infectious;[1] $b_v^* = b_v(N_h^*, N_v^*)$ is the number of contacts that one mosquito has with humans per unit time; and $1/(\mu_{1v} + \mu_{2v}N_v^*)$ is the average duration of the infectious lifetime of the mosquito. In (3.3b), the total number of mosquitoes infected by one human is the sum of the new infections from the infectious and from the recovered states of the human; $\nu_h/(\nu_h + \mu_{1h} + \mu_{2h}N_h^*)$ is the probability that a human will survive the exposed state to become infectious; $\gamma_h/(\gamma_h + \delta_h + \mu_{1h} + \mu_{2h}N_h^*)$ is the probability that the human will then survive the infectious state to move to the recovered state; $b_h^* = b_h(N_h^*, N_v^*)$ is the number of contacts that one human has with mosquitoes per unit time; $1/(\gamma_h + \delta_h + \mu_{1h} + \mu_{2h}N_h^*)$ is the average duration of the infectious period of a human; and $1/(\rho_h + \mu_{1h} + \mu_{2h}N_h^*)$ is the average duration of the recovered period of a human.

We define R_0 as the spectral radius of the next generation operator, K, i.e., $R_0^2 = K_{vh}K_{hv}$. Then, R_0^2 is the number of humans that one infectious human will infect, through a generation of infections in mosquitoes, assuming that previously all other humans and mosquitoes were susceptible.

DEFINITION 3.2. *We define the reproductive number, R_0, as*

$$R_0 = \sqrt{K_{vh}K_{hv}}, \tag{3.4}$$

where K_{vh} and K_{hv} are defined in (3.3).

The original definition of the reproductive number of the Ross–Macdonald model [1] and [3], and the Ngwa and Shu model [23], is equivalent to the square of this R_0. They ([1], [3], and [23]) use the traditional definition of the reproductive number, which approximates the number of secondary infections in humans caused by one infected human, while the R_0 in Definition 3.2 is consistent with the definition given by the next generation operator approach [10], which approximates the number of secondary infections due to one infected individual (be it human or mosquito). Our definition of R_0 includes the generation of infections in mosquitoes, so is the square root of the original definition. The threshold condition for both definitions is the same.

3.3. Stability of the disease-free equilibrium point.

THEOREM 3.3. *The disease-free equilibrium point, x_{dfe}, is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

The proof of this theorem is in the appendix section A.1.

[1] In defining periods of time and probabilities for R_0, we use the original system of equations (2.1) and not the scaled equations (2.8). As the two models are equivalent, the reproductive number is the same with either definition: $\mu_{1h} + \mu_{2h}N_h^* = \psi_h + \Lambda_h/N_h^*$ and $\mu_{1v} + \mu_{2v}N_v^* = \psi_v$.

4. Endemic equilibrium points. Endemic equilibrium points are steady-state solutions where the disease persists in the population (all state variables are positive). We use general bifurcation theory to prove the existence of at least one endemic equilibrium point for all $R_0 > 1$. We prove that the transcritical bifurcation at $R_0 = 1$ is supercritical (forward) when $\delta_h = 0$ (there is no disease-induced death). However, numerical results show that the bifurcation can be subcritical (backward) for some positive values of δ_h, giving rise to endemic equilibria for $R_0 < 1$.

We first rewrite the equilibrium equations for $u = (e_h, e_v)$ in (2.8) as a nonlinear eigenvalue problem in a Banach space:

$$u = G(\zeta, u) = \zeta L u + h(\zeta, u), \tag{4.1}$$

where $u \in Y \subset \mathbb{R}^2$, with Euclidean norm $\|\cdot\|$; $\zeta \in Z \subset \mathbb{R}$ is the bifurcation parameter; L is a compact linear map on Y; and $h(\zeta, u)$ is $\mathcal{O}(\|u\|^2)$ uniformly on bounded ζ intervals. We require that both Y and Z be open and bounded sets, and that Y contain the point 0. We define Z as the open and bounded set $Z = \{\zeta \in \mathbb{R} | -M_Z < \zeta < M_Z\}$. This set is defined to include the characteristic values (reciprocals of eigenvalues) of L, so there is minimum value that M_Z can have, but M_Z may be arbitrarily large. We use

$$\zeta = \frac{\sigma_v \sigma_h}{\sigma_v N_v^* + \sigma_h N_h^*} \tag{4.2}$$

for the bifurcation parameter. We also define $\Omega = Z \times Y$ so that the pair $(\zeta, u) \in \Omega$. We denote the boundary of Ω by $\partial\Omega$.

A corollary by Rabinowitz [24, Corollary 1.12] states that if $\zeta_0 \in Z$ is a characteristic value of L of odd multiplicity, then there exists a continuum of nontrivial solution-pairs (ζ, u) of (4.1) that intersects the trivial solution (that is, $(\zeta, 0)$ for any ζ) at $(\zeta_0, 0)$ and either meets $\partial\Omega$ or meets $(\hat{\zeta}_0, 0)$, where $\hat{\zeta}_0$ is also a characteristic value of L of odd multiplicity. We use this corollary to show that there exists a continuum of solution-pairs $(\zeta, u) \in \Omega$ for the eigenvalue equation (4.1). To each of these solution-pairs there corresponds an equilibrium-pair (ζ, x^*). We define the equilibrium-pair, $(\zeta, x^*) \in Z \times \mathbb{R}^7$, as the collection of a parameter value, ζ, and the corresponding equilibrium point, x^*, for that parameter value, of the malaria model (2.8).

THEOREM 4.1. *The model* (2.8) *has a continuum of equilibrium-pairs,* $(\zeta, x^*) \in Z \times \mathbb{R}^7$*, that connects the point* (ξ_1, x_{dfe}) *to the hyperplane* $\zeta = M_Z$ *in* $\mathbb{R} \times \mathbb{R}^7$ *on the boundary of* $Z \times \mathbb{R}^7$ *for any* $M_Z > \xi_1$*, where* x^* *is in the positive orthant of* $\mathbb{R}^7$*. Here* $\xi_1 = 1/\sqrt{AB}$*, where* A *and* B *are defined in* (A.19).

We show the proof of this theorem and related lemmas in appendix section A.2.

THEOREM 4.2. *The transcritical bifurcation point at* $\zeta = \xi_1$ *corresponds to* $R_0 = 1$*. For the set of* ζ *for which there exists an equilibrium-pair* (ζ, x^*)*, the corresponding set of values for* R_0 *includes, but is not necessarily identical to, the interval* $1 < R_0 < \infty$*. Thus, there exists at least one endemic equilibrium point of the malaria model* (2.8) *for all* $R_0 > 1$*.*

Proof. Using the definition of ζ, (4.2), some algebraic manipulations of R_0 (see (3.4)) produce

$$R_0 = \zeta \sqrt{AB}. \tag{4.3}$$

Thus, R_0 is linearly related to ζ, and when $\zeta = \xi_1$, $R_0 = 1$. For any $R_0 > 1$, (4.3) defines a corresponding ζ. We pick an M_Z larger than this ζ. Then, Theorem 4.1

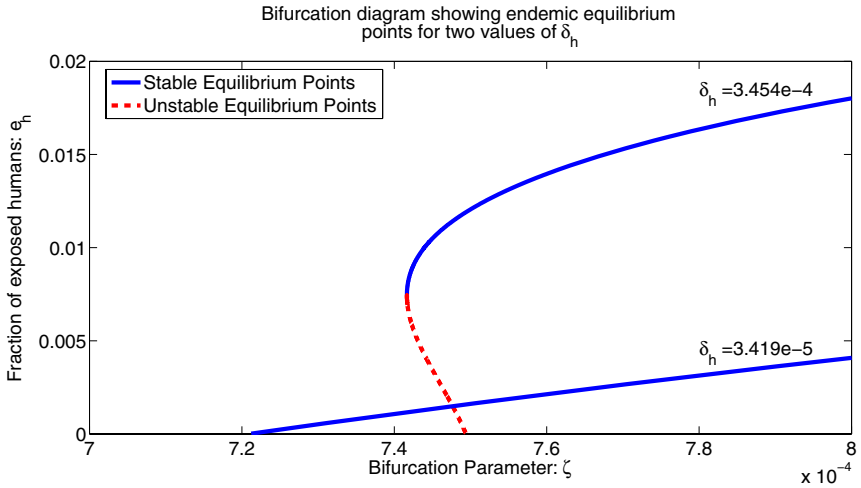

FIG. 4.1. *Bifurcation diagrams for* (2.8) *showing the endemic equilibrium values for the fraction of exposed humans,* e_h*, plotted for the parameters in Table* 4.1 *(except for* σ_v *and* σ_h*, which vary with* ζ*) and two values of the disease-induced death rate* ($\delta_h = 3.454 \times 10^{-4}$ *and* $\delta_h = 3.419 \times 10^{-5}$)*. For the parameter values in Table* 4.1*, there are three equilibrium points in* $\mathcal{D}$*: a locally asymptotically stable disease-free equilibrium point,* x_{dfe}*, on the boundary of the positive orthant of* $\mathbb{R}^7$*, and two endemic equilibrium points inside the positive orthant. Linear stability analysis shows that the "larger" endemic equilibrium point is locally asymptotically stable, while the "smaller" point is unstable. Further linear analysis with an increased value of* $\sigma_v = 0.7000$*,* $\sigma_h = 21.00$*, and all other parameters as in Table* 4.1 *(with* $R_0 = 1.155$*) shows that* x_{dfe} *is unstable, and there is one locally asymptotically stable endemic equilibrium point.*

guarantees the existence of an endemic equilibrium point for ζ, and thereby for the corresponding value of R_0. It is possible, though not necessary, for the continuum of equilibrium-pairs to include values of $\zeta < \xi_1$ ($R_0 < 1$). □

Typically in epidemiological models, bifurcations at $R_0 = 1$ tend to be supercritical (i.e., positive endemic equilibria exist for $R_0 > 1$ near the bifurcation point). In this model (2.8), in the absence of disease-induced death ($\delta_h = 0$), we prove, using the Lyapunov–Schmidt expansion as described by Cushing [9], that the bifurcation is supercritical (forward).

THEOREM 4.3. *In the absence of disease-induced death* ($\delta_h = 0$)*, the transcritical bifurcation at* $R_0 = 1$ *is supercritical (forward).*

Details of this proof are in appendix section A.2.

In the general case, a subcritical (backward) bifurcation can occur for some parameter values, where near the bifurcation point, positive endemic equilibria exist for $R_0 < 1$. Other examples of epidemiological models with subcritical bifurcations at $R_0 = 1$ include those described by Castillo-Chavez and Song [6], Gómez-Acevedo and Yi [13], and van den Driessche and Watmough [26]. The model of Ngwa and Shu [23] exhibits only a supercritical bifurcation at $R_0 = 1$. Although we cannot prove the existence of a subcritical bifurcation, we show through numerical examples that it is possible for some positive values of δ_h. This is important because it implies that there can be a stable endemic equilibrium even if $R_0 < 1$.

We use the bifurcation software program AUTO [12] to create two bifurcation diagrams around $R_0 = 1$ (Figure 4.1) with parameter values in Table 4.1, except for σ_h, σ_v, and δ_h. σ_h and σ_v change as ζ is varied, as shown in the figure; however, their ratio, $\theta = \sigma_h/\sigma_v = 30$, remains constant. One curve has δ_h as in Table 4.1,

TABLE 4.1

The parameter values for which there exist positive endemic equilibrium points when $R_0 < 1$: $R_0 = 0.9898$. *The unit of time is days.*

$\Lambda_h = 3.285 \times 10^{-2}$	
$\psi_h = 7.666 \times 10^{-5}$	$\psi_v = 0.4000$
$\beta_{vh} = 0.8333$	$\beta_{hv} = 2.000 \times 10^{-2}$
$\tilde{\beta}_{vh} = 8.333 \times 10^{-2}$	
$\sigma_v = 0.6000$	$\sigma_h = 18.00$
$\nu_h = 8.333 \times 10^{-2}$	$\nu_v = 0.1000$
$\gamma_h = 3.704 \times 10^{-3}$	
$\delta_h = 3.454 \times 10^{-4}$	
$\rho_h = 1.460 \times 10^{-2}$	
$\mu_{1h} = 4.212 \times 10^{-5}$	$\mu_{1v} = 0.1429$
$\mu_{2h} = 1.000 \times 10^{-7}$	$\mu_{2v} = 2.279 \times 10^{-4}$

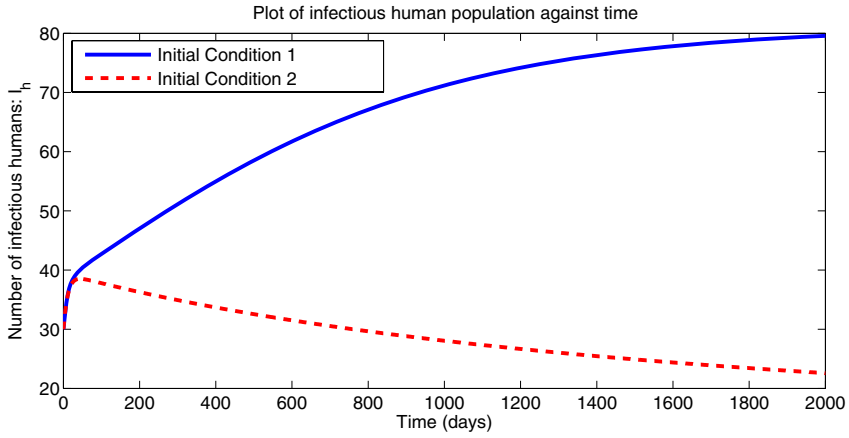

FIG. 4.2. *Solutions of the malaria model* (2.1) *with parameter values defined in Table* 4.1 *showing only the number of infectious humans,* I_h, *for two different initial conditions. The parameters correspond to* $R_0 = 0.9898$. *Initial condition* 1 *is* $S_h = 400$, $E_h = 10$, $I_h = 30$, $R_h = 0$, $S_v = 1000$, $E_v = 100$, *and* $I_v = 50$. *Initial condition* 2 *is* $S_h = 700$, $E_h = 10$, $I_h = 30$, $R_h = 0$, $S_v = 1000$, $E_v = 100$, *and* $I_v = 50$. *The solution for initial condition* 1 *approaches the locally asymptotically stable endemic equilibrium point, while the solution for initial condition* 2 *approaches the locally asymptotically stable disease-free equilibrium point.*

while the other has $\delta_h = 3.419 \times 10^{-5}$. The curve with $\delta_h = 3.454 \times 10^{-4}$ has both unstable and stable endemic equilibrium points. There is a subcritical bifurcation at $\zeta = 7.494 \times 10^{-4}$ ($R_0 = 1$), and a saddle-node bifurcation at $\zeta = 7.417 \times 10^{-4}$ ($R_0 = 0.9897$). Thus a locally asymptotically stable endemic equilibrium is possible for values of R_0 below 1. Further bifurcation analysis (not presented here) indicates that as ζ is increased to $\zeta = 50$ ($R_0 = 66719$), the size of the projection of the endemic equilibrium on the fractional infected groups increases monotonically, and the equilibrium point remains stable. For comparison we show the bifurcation diagram with $\delta_h = 3.419 \times 10^{-5}$. Here, we see only a stable branch of endemic equilibrium points. There is a supercritical bifurcation at $\zeta = 7.209 \times 10^{-4}$ ($R_0 = 1$). There are no endemic equilibrium points for R_0 less than 1. As ζ is increased to $\zeta = 50$ ($R_0 = 69358$), the size of the projection of the endemic equilibrium on the fractional infected groups increases monotonically, and the equilibrium point remains stable.

Figure 4.2 shows the infectious human population, for two different initial condi-

tions, of the solutions to the unscaled equations (2.1) for parameter values in Table 4.1 with $R_0 < 1$. One solution approaches the locally asymptotically stable endemic equilibrium point, while the other approaches the locally asymptotically stable disease-free equilibrium point.

The parameter values in Table 4.1 are within the bounds of a realistically feasible range, except for the mosquito birth and death rates, ψ_v and μ_{1v}, which have been increased to lower R_0 below 1. More realistic values are $\psi_v = 0.13$ and $\mu_{1v} = 0.033$, which result in (with all other parameters as in Table 4.1) $R_0 = 1.6$. More lists of realistic parameter values, and their references, can be found in [7] and [8]. $\delta_h = 3.454 \times 10^{-4}$ corresponds to a death rate of 12.62% of infectious humans per year.

5. Summary and conclusions. We analyzed an ordinary differential equation model for the transmission of malaria, with four variables for humans and three variables for mosquitoes. We showed that there exists a domain where the model is epidemiologically and mathematically well-posed. We proved the existence of an equilibrium point with no disease, x_{dfe}. We defined a reproductive number, R_0, that is epidemiologically accurate in that it provides the expected number of new infections (in mosquitoes or humans) from one infectious individual (human or mosquito) over the duration of the infectious period, given that all other members of the population are susceptible. We showed that if $R_0 < 1$, then the disease-free equilibrium point, x_{dfe}, is locally asymptotically stable, and if $R_0 > 1$, then x_{dfe} is unstable.

We also proved that an endemic equilibrium point exists for all $R_0 > 1$ with a transcritical bifurcation at $R_0 = 1$. The analysis and the numerical simulations showed that for $\delta_h = 0$ (no disease-induced death), and for some small positive values of δ_h, there is a supercritical transcritical bifurcation at $R_0 = 1$ with an exchange of stability between the disease-free equilibrium and the endemic equilibrium. For larger values of δ_h, there is a subcritical transcritical bifurcation at $R_0 = 1$, with an exchange of stability between the endemic equilibrium and the disease-free equilibrium; and there is a saddle-node bifurcation at $R_0 = R_0^*$ for some $R_0^* < 1$. Thus, for some values of $R_0 < 1$, there exist two endemic equilibrium points, the smaller of which is unstable, while the larger is locally asymptotically stable.

Although we cannot prove in general that the endemic equilibrium point is unique and stable for $R_0 > 1$, numerical results for particular parameter sets suggest that there is a unique stable endemic equilibrium point for $R_0 > 1$. Also, from Theorem 2.1 it follows that all orbits of the malaria model (2.8) are bounded. Thus, if there were no stable endemic equilibria in $\mathcal{D}$, then there would exist a nonequilibrium attractor (such as a limit cycle or strange attractor), though for this model we have no evidence for nonequilibrium attractors.

The possible existence of a subcritical bifurcation at $R_0 = 1$ and a saddle-node bifurcation at some $R_0^* < 1$ can have implications for public health, when the epidemiological parameters are close to those in Table 4.1. Simply reducing R_0 to a value below 1 is not always sufficient to eradicate the disease; it is now necessary to reduce R_0 to a value less than R_0^* to ensure that there are no endemic equilibria. The existence of a saddle-node bifurcation also implies that in some areas with endemic malaria, it may be possible to significantly reduce prevalence or eradicate the disease with small increases in control programs (a small reduction in R_0 so that it is less than R_0^*). In some areas where malaria has been eradicated it is possible for a slight disruption, like a change in environmental or control variables or an influx of infectious humans or mosquitoes, to cause the disease to reestablish itself in the population with a significant increase in disease prevalence (increasing R_0 above R_0^*

or moving the system into the basin of attraction of the stable endemic equilibrium).

As we have an explicit expression for R_0, we can analytically evaluate its sensitivity to the different parameter values. We can also numerically evaluate the sensitivity of the endemic equilibrium to the parameter values. This allows us to determine the relative importance of the parameters to disease transmission and prevalence. As each malaria intervention strategy affects different parameters to different degrees, we can thus compare different control strategies for efficiency and effectiveness in reducing malaria mortality and morbidity. This analysis, in the limiting case of the Chitnis model [7] shows that malaria transmission is most sensitive to the mosquito biting rate, and prevalence is most sensitive to the mosquito biting rate and the human recovery rate. The sensitivity analysis for the new model (2.8) is forthcoming [8].

We are extending the model to include the effects of the environment on the spread of malaria. Some parameters, such as the mosquito birth rate and the incubation period in mosquitoes, depend on seasonal environmental factors such as rainfall, temperature, and humidity. We can include these effects by modeling these parameters as periodic functions of time. We would like to explore this periodically forced model for features not seen in the autonomous model, including the modifications to the definition of the reproductive number and the endemic states. This would provide a more accurate picture of malaria transmission and prevalence than that obtained from models using parameter values that are averaged over the seasons. Other planned improvements to the model include the addition of age and spatial structure.

An ultimate goal is to validate this model by applying it to a particular malaria-endemic region of the world to compare the predicted endemic states with the prevalence data.

Appendix. Lemmas and proofs of theorems.

LEMMA A.1. *For all equilibrium points on* $\mathcal{D} \cap \partial\mathbb{R}^7_+$, $e_h = i_h = r_h = e_v = i_v = 0$.

Proof. We need to show that for an equilibrium point in $\mathcal{D}$, if any one of the diseased classes is zero, all the rest are also equal to zero. We show, by setting the right-hand side of (2.8) equal to 0, that if any one of e_h, i_h, r_h, e_v, or i_v is equal to 0, then $e_h = i_h = r_h = e_v = i_v = 0$. For $i'_h = 0$, $e_h = 0$ if and only if $i_h = 0$.[2] Similarly, for $r'_h = 0$, $i_h = 0$ if and only if $r_h = 0$. Thus, if $e_h = 0$, $i_h = 0$, or $r_h = 0$, then $e_h = i_h = r_h = 0$. From $e'_h = 0$, we see that if $e_h = i_h = r_h = 0$, then $i_v = 0$. Also, for $i'_v = 0$, $e_v = 0$ if and only if $i_v = 0$. Thus, if $e_v = 0$ or $i_v = 0$, then $e_v = i_v = 0$. Finally, for $e'_v = 0$, if $e_v = i_v = 0$, then $i_h = r_h = 0$. □

A.1. Proof of Theorem 3.3.

Proof. The Jacobian of the malaria model (2.8) evaluated at x_{dfe} is of the form

$$J = \begin{pmatrix} J_{11} & 0 & 0 & 0 & 0 & J_{16} & 0 \\ J_{21} & J_{22} & 0 & 0 & 0 & 0 & 0 \\ 0 & J_{32} & J_{33} & 0 & 0 & 0 & 0 \\ 0 & J_{42} & 0 & J_{44} & 0 & 0 & 0 \\ 0 & J_{52} & J_{53} & 0 & J_{55} & 0 & 0 \\ 0 & 0 & 0 & 0 & J_{65} & J_{66} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & J_{77} \end{pmatrix}. \tag{A.1}$$

[2] As the right-hand side of (2.8b) is a quadratic function of i_h, there are two possible solutions of i_h when $i'_h = 0$ and $e_h = 0$. However, the nonzero solution of i_h is greater than 1 and is thus outside of $\mathcal{D}$.

As the fourth and seventh columns (corresponding to the total human and mosquito populations) contain only the diagonal terms, these diagonal terms form two eigenvalues of the Jacobian:

$$\eta_6 = \psi_h - \mu_{1h} - 2\mu_{2h}N_h^* = -\sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}, \tag{A.2a}$$

$$\eta_7 = \psi_v - \mu_{1v} - 2\mu_{2v}N_v^* = -(\psi_v - \mu_{1v}). \tag{A.2b}$$

As we have assumed that $\psi_v > \mu_{1v}$, both η_6 and η_7 are always negative. The other five eigenvalues are the roots of the characteristic equation of the matrix formed by excluding the fourth and seventh rows and columns of the Jacobian (A.1):

$$A_5\eta^5 + A_4\eta^4 + A_3\eta^3 + A_2\eta^2 + A_1\eta + A_0 = 0 \tag{A.3}$$

with

$$\begin{aligned}
A_5 &= 1,\\
A_4 &= B_1 + B_2 + B_3 + B_4 + B_5,\\
A_3 &= B_1B_2 + B_1B_3 + B_1B_4 + B_1B_5 + B_2B_3 + B_2B_4 + B_2B_5 + B_3B_4\\
&\quad + B_3B_5 + B_4B_5,\\
A_2 &= B_1B_2B_3 + B_1B_2B_4 + B_1B_2B_5 + B_1B_3B_4 + B_1B_3B_5 + B_1B_4B_5 + B_2B_3B_4\\
&\quad + B_2B_3B_5 + B_2B_4B_5 + B_3B_4B_5,\\
A_1 &= B_1B_2B_3B_4 + B_1B_2B_3B_5 + B_1B_2B_4B_5 + B_1B_3B_4B_5 + B_2B_3B_4B_5\\
&\quad - B_6B_7B_8B_9,\\
A_0 &= B_1B_2B_3B_4B_5 - (B_3B_6B_7B_8B_9 + B_6B_7B_9B_{10}B_{11}),
\end{aligned}$$

and $B_1 = \nu_h + \psi_h + \Lambda_h/N_h^*$, $B_2 = \gamma_h + \delta_h + \psi_h + \Lambda_h/N_h^*$, $B_3 = \rho_h + \psi_h + \Lambda_h/N_h^*$, $B_4 = \nu_v + \psi_v$, $B_5 = \psi_v$, $B_6 = b_h^*\beta_{hv}$, $B_7 = \nu_h$, $B_8 = b_v^*\beta_{vh}$, $B_9 = \nu_v$, $B_{10} = \gamma_h$, and $B_{11} = b_v^*\tilde{\beta}_{vh}$.

To evaluate the signs of the roots of (A.3), we first use the Routh–Hurwitz criterion to prove that when $R_0 < 1$, all roots of (A.3) have negative real part. Then, using Descartes's rule of sign, we prove that when $R_0 > 1$, there is one positive real root.

The Routh–Hurwitz criterion [18, section 1.6-6(b)] for a real algebraic equation

$$a_nx^n + a_{n-1}x^{n-1} + \cdots + a_1x + a_0 = 0 \tag{A.4}$$

states that, given $a_n > 0$, all roots have negative real part if and only if $T_0 = a_n$, $T_1 = a_{n-1}$,

$$T_2 = \begin{vmatrix} a_{n-1} & a_n \\ a_{n-3} & a_{n-2} \end{vmatrix}, T_3 = \begin{vmatrix} a_{n-1} & a_n & 0 \\ a_{n-3} & a_{n-2} & a_{n-1} \\ a_{n-5} & a_{n-4} & a_{n-3} \end{vmatrix}, \ldots, T_n = \begin{vmatrix} a_{n-1} & \cdots & 0 \\ \vdots & \ddots & \vdots \\ 0 & \cdots & a_0 \end{vmatrix}$$

are all positive, with $a_i = 0$ for $i < 0$. This is true if and only if all a_i and either all even-numbered T_k or all odd-numbered T_k are positive (the Liénard–Chipart test). Korn and Korn [18] in section 1.6-6(c) state Descartes's rule of sign as the number of positive real roots of a real algebraic equation (A.4) is equal to the number, N_a, of sign changes in the sequence, $a_n, a_{n-1}, \ldots, a_0$, of coefficients, where the vanishing terms are disregarded, or it is less than N_a by a positive even integer.

We show that when $R_0 < 1$, all the coefficients, A_i, of the characteristic equation (A.3), and T_0, T_2, and T_4, are positive, so by the Routh–Hurwitz criterion, all the eigenvalues of the Jacobian (A.1) have negative real part. We then show that when $R_0 > 1$, there is one and only one sign change in the sequence $A_5, A_4, \ldots, A_0$, so by Descartes's rule of sign there is one eigenvalue with positive real part, and the disease-free equilibrium point is unstable.

The expression for R_0^2 in (3.4) can be written, in terms of B_i, as

$$R_0^2 = \frac{B_3B_6B_7B_8B_9 + B_6B_7B_9B_{10}B_{11}}{B_1B_2B_3B_4B_5}. \tag{A.5}$$

For $R_0 < 1$, by (A.5),

$$B_3B_6B_7B_8B_9 + B_6B_7B_9B_{10}B_{11} < B_1B_2B_3B_4B_5, \tag{A.6}$$

$$B_6B_7B_8B_9 < B_1B_2B_4B_5. \tag{A.7}$$

As all the B_i are positive, A_5, A_4, A_3, and A_2 are always positive. From (A.7) we see that $A_1 > 0$, and from (A.6) we see that $A_0 > 0$. Thus, for $R_0 < 1$, all A_i are positive. We now show that the even-numbered T_k are positive for $R_0 < 1$. For the fifth-degree polynomial (A.3), $T_0 = A_5$, which is always positive. $T_2 = A_3A_4 - A_2A_5$, which we can show to be a positive sum of products of B_i's, so $T_2 > 0$. Lastly,

$$T_4 = A_1[A_2A_3A_4 - (A_1A_4^2 + A_2^2A_5)] - A_0[A_3(A_3A_4 - A_2A_5) - (2A_1A_4A_5 - A_0A_5^2)].$$

For ease of notation, we introduce

$$C_1 = A_2A_3A_4 - (A_1A_4^2 + A_2^2A_5),$$
$$C_2 = A_3(A_3A_4 - A_2A_5) - (2A_1A_4A_5 - A_0A_5^2),$$

where we can show that $C_1 > 0$ and $C_2 > 0$, so that $T_4 = A_1C_1 - A_0C_2$. We define

$$C_2^{(1)} = C_2 + B_6B_7B_9B_{10}B_{11}.$$

As $C_2^{(1)} > C_2$ and $A_0 > 0$, for $T_4^{(1)} = A_1C_1 - A_0C_2^{(1)}$, $T_4 > T_4^{(1)}$. Similarly, we define

$$A_0^{(1)} = A_0 + (B_3B_6B_7B_8B_9 + B_6B_7B_9B_{10}B_{11}).$$

As $A_0^{(1)} > A_0$ and $C_2^{(1)} > 0$, for $T_4^{(2)} = A_1C_1 - A_0^{(1)}C_2^{(1)}$, $T_4^{(1)} > T_4^{(2)}$. Finally, we define

$$A_1^{(1)} = A_1 - (B_1B_2B_4B_5 - B_6B_7B_8B_9).$$

As $A_1^{(1)} < A_1$ (for $R_0 < 1$) and $C_1 > 0$, for $T_4^{(3)} = A_1^{(1)}C_1 - A_0^{(1)}C_2^{(1)}$, $T_4^{(2)} > T_4^{(3)}$. We can show that $T_4^{(3)}$ is a sum of positive terms, so $T_4^{(3)} > 0$. As $T_4 > T_4^{(1)} > T_4^{(2)} > T_4^{(3)}$, $T_4 > 0$. Thus, for $R_0 < 1$, all roots of (A.3) have negative real parts.

When $R_0 > 1$

$$B_3B_6B_7B_8B_9 + B_6B_7B_9B_{10}B_{11} > B_1B_2B_3B_4B_5,$$

and so $A_0 < 0$. As A_5, A_4, A_3, and A_2 are positive, the sequence, $A_5, A_4, A_3, A_2, A_1, A_0$ has exactly one sign change. Thus, by Descartes's rule of sign, (A.3) has one positive real root when $R_0 > 1$.

Thus, the disease-free equilibrium point, x_{dfe}, is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. If $R_0 < 1$, on average each infected individual infects fewer than one other individual, and the disease dies out. If $R_0 > 1$, on average each infected individual, infects more than one other individual, so we would expect the disease to spread. The Jacobian of (2.8) at x_{dfe} has one eigenvalue equal to 0 at $R_0 = 1$. □

A.2. Proofs of theorems and lemmas for the existence of endemic equilibrium points. The equilibrium equations for (2.8) are shown below in (A.8). In this analysis, we use the terms e_h, i_h, r_h, N_h, e_v, i_v, and N_v to represent their respective equilibrium values and not their actual values at a given time, t.

$$\left(\frac{\sigma_v\sigma_h N_v\beta_{hv}i_v}{\sigma_v N_v+\sigma_h N_h}\right)(1-e_h-i_h-r_h)-(\nu_h+\psi_h+\Lambda_h/N_h)e_h+\delta_h i_h e_h=0, \tag{A.8a}$$

$$\nu_h e_h-(\gamma_h+\delta_h+\psi_h+\Lambda_h/N_h)i_h+\delta_h i_h^2=0, \tag{A.8b}$$

$$\gamma_h i_h-(\rho_h+\psi_h+\Lambda_h/N_h)r_h+\delta_h i_h r_h=0, \tag{A.8c}$$

$$\Lambda_h+\psi_h N_h-(\mu_{1h}+\mu_{2h}N_h)N_h-\delta_h i_h N_h=0, \tag{A.8d}$$

$$\left(\frac{\sigma_v\sigma_h N_h}{\sigma_v N_v+\sigma_h N_h}\right)\left(\beta_{vh}i_h+\tilde{\beta}_{vh}r_h\right)(1-e_v-i_v)-(\nu_v+\psi_v)e_v=0, \tag{A.8e}$$

$$\nu_v e_v-\psi_v i_v=0, \tag{A.8f}$$

$$\psi_v N_v-(\mu_{1v}+\mu_{2v}N_v)N_v=0. \tag{A.8g}$$

We rewrite (A.8a) and (A.8e) in terms of the bifurcation parameter, ζ (4.2), and a new parameter, $\theta=\sigma_h/\sigma_v$, to obtain

$$\zeta\left(\frac{N_v^*+\theta N_h^*}{N_v+\theta N_h}\right)N_v\beta_{hv}i_v(1-e_h-i_h-r_h)-(\nu_h+\psi_h+\Lambda_h/N_h-\delta_h i_h)e_h=0, \tag{A.9a}$$

$$\zeta\left(\frac{N_v^*+\theta N_h^*}{N_v+\theta N_h}\right)N_h\left(\beta_{vh}i_h+\tilde{\beta}_{vh}r_h\right)(1-e_v-i_v)-(\nu_v+\psi_v)e_v=0. \tag{A.9b}$$

We can vary the bifurcation parameter, ζ, while keeping all other parameters fixed. In terms of the original variables, this corresponds to changing σ_h and σ_v while keeping the ratio between them fixed. We can pick θ, the ratio between them, and sweep out the entire parameter space.

We reduce the equilibrium equations to a two-dimensional system for e_h and e_v by solving for the other variables, either explicitly as functions of the parameters, or in terms of e_h and e_v. We solve (A.8g) for N_v, explicitly expressing the positive equilibrium for the total mosquito population in terms of parameters (exactly as in the disease-free case (3.1)):

$$N_v=\frac{\psi_v-\mu_{1v}}{\mu_{2v}}. \tag{A.10}$$

Solving for i_v in (A.8f) in terms of e_v, we find

$$i_v=\frac{\nu_v}{\psi_v}e_v. \tag{A.11}$$

We write the positive equilibrium for N_h in terms of i_h from (A.8d) as

$$N_h=\frac{(\psi_h-\mu_{1h}-\delta_h i_h)+\sqrt{(\psi_h-\mu_{1h}-\delta_h i_h)^2+4\mu_{2h}\Lambda_h}}{2\mu_{2h}}. \tag{A.12}$$

Using (A.12) in (A.8c), we solve for r_h in terms of i_h:

$$r_h = \frac{2\gamma_h i_h}{2\rho_h + (\psi_h + \mu_{1h} - \delta_h i_h) + \sqrt{(\psi_h - \mu_{1h} - \delta_h i_h)^2 + 4\mu_{2h}\Lambda_h}}. \tag{A.13}$$

Given the nonlinear nature of (A.8b), it is not feasible (or useful) to solve for i_h in terms of e_h explicitly. We therefore use (A.12) to rewrite (A.8b), and define the function $e_h = g(i_h)$ as

$$g(i_h) = \frac{\gamma_h + \delta_h + \frac{1}{2}\left((\psi_h + \mu_{1h} - \delta_h i_h) + \sqrt{(\psi_h - \mu_{1h} - \delta_h i_h)^2 + 4\mu_{2h}\Lambda_h}\right)}{\nu_h} i_h.$$

We note that $g(0) = 0$, and label the positive constant $g(1) = e_h^{max}$. As $g(i_h)$ is a smooth function of i_h with $g'(i_h) > 0$ for $i_h \in [0,1]$ and $e_h \in [0, e_h^{max}]$, there exists a smooth function $i_h = y(e_h)$ with domain $[0, e_h^{max}]$ and range $[0,1]$. As $g'(0) > 0$, the smooth function $y(e_h)$ would extend to some small $e_h < 0$. Substituting $i_h = y(e_h)$ into (A.12) and (A.13), we can also express N_h and r_h as functions of e_h.

We now introduce the bounded open subset of $\mathbb{R}^2$,

$$Y = \left\{ \begin{pmatrix} e_h \\ e_v \end{pmatrix} \in \mathbb{R}^2 \;\middle|\; \begin{array}{l} -\epsilon_h < e_h < e_h^{max} \\ -\epsilon_v < e_v < 1 \end{array} \right\}, \tag{A.14}$$

for some $\epsilon_v > 0$ and some $\epsilon_h > 0$. By substituting (A.10), (A.11), (A.12), (A.13), and $i_h = y(e_h)$ into (A.8a) and (A.8e), we reformulate the seven equilibrium equations (A.8) equivalently as two equations for the components $(e_h, e_v) \in Y$. To place these two equations into the Rabinowitz form (4.1), we need to determine lower order terms. We rewrite (A.8b) as $f(e_h, i_h) = 0$, where $f(e_h, i_h) =$

$$\nu_h e_h - \left[\gamma_h + \delta_h + \frac{1}{2}\left((\psi_h + \mu_{1h} - \delta_h i_h) + \sqrt{(\psi_h - \mu_{1h} - \delta_h i_h)^2 + 4\mu_{2h}\Lambda_h}\right)\right] i_h,$$

and use implicit differentiation to write $i_h = y(e_h)$ as a Taylor polynomial of the form

$$i_h = y_1 e_h + \mathcal{O}(e_h^2), \tag{A.15}$$

where

$$y_1 = -\left.\frac{\frac{\partial f}{\partial e_h}}{\frac{\partial f}{\partial i_h}}\right|_{i_h = e_h = 0} = \frac{\nu_h}{\gamma_h + \delta_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)}.$$

Finally, we substitute the Taylor approximation for i_h (A.15) into r_h (A.13) and N_h (A.12), and then all three, along with i_v (A.11) and N_v (A.10) into the equilibrium equations for e_h (A.9a) and e_v (A.9b), to provide first order approximations to the equilibrium equations:

$$\begin{pmatrix} 0 \\ 0 \end{pmatrix} = \begin{pmatrix} f_{1\_10} & f_{1\_01} \\ f_{2\_10} & f_{2\_01} \end{pmatrix} \begin{pmatrix} e_h \\ e_v \end{pmatrix} + \mathcal{O}\left(\begin{pmatrix} e_h \\ e_v \end{pmatrix}^2\right), \tag{A.16}$$

where

$$f_{1\_10} = -\left[\nu_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)\right], \tag{A.17a}$$

$$f_{1\_01} = \zeta \cdot \frac{\nu_v \beta_{hv}(\psi_v - \mu_{1v})}{\psi_v \mu_{2v}}, \tag{A.17b}$$

$$f_{2\_10} = \zeta \cdot \frac{\nu_h\left((\psi_h - \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\Lambda_h\mu_{2h}}\right)}{2\mu_{2h}\left(\gamma_h + \delta_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)\right)} \times \left[\beta_{vh} + \frac{\gamma_h\tilde{\beta}_{vh}}{\rho_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)}\right], \tag{A.17c}$$

$$f_{2\_01} = -\left(\psi_v + \nu_v\right). \tag{A.17d}$$

To apply Corollary 1.12 of Rabinowitz [24], we algebraically manipulate (A.16) to produce

$$u = \zeta L u + h(\zeta, u), \tag{A.18}$$

where

$$u = \begin{pmatrix} e_h \\ e_v \end{pmatrix} \quad \text{and} \quad L = \begin{pmatrix} 0 & A \\ B & 0 \end{pmatrix} \quad \text{with}$$

$$A = \frac{\nu_v \beta_{hv}(\psi_v - \mu_{1v})}{\psi_v \mu_{2v}\left(\nu_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)\right)}, \tag{A.19a}$$

$$B = \left(\beta_{vh} + \frac{\gamma_h\tilde{\beta}_{vh}}{\rho_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)}\right) \times \frac{\nu_h\left((\psi_h - \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)}{2\mu_{2h}(\psi_v + \nu_v)\left(\gamma_h + \delta_h + \frac{1}{2}\left((\psi_h + \mu_{1h}) + \sqrt{(\psi_h - \mu_{1h})^2 + 4\mu_{2h}\Lambda_h}\right)\right)}, \tag{A.19b}$$

and $h(\zeta, u)$ is $\mathcal{O}(u^2)$. The matrix, L, has two distinct eigenvalues: $\pm\sqrt{AB}$. Characteristic values of a matrix are the reciprocals of its eigenvalues. We denote the two characteristic values of L by $\xi_1 = 1/\sqrt{AB}$ and $\xi_2 = -1/\sqrt{AB}$. As both A and B are always positive (because we have assumed that $\psi_v > \mu_{1v}$), ξ_1 is real and corresponds to the dominant eigenvalue of L. The right and left eigenvectors corresponding to ξ_1 are, respectively,

$$v = \begin{pmatrix} \sqrt{A} \\ \sqrt{B} \end{pmatrix} \quad \text{and} \quad w = \begin{pmatrix} \sqrt{B} & \sqrt{A} \end{pmatrix}. \tag{A.20}$$

For $M_Z > \xi_1$, as $0 \in Y$, $(\xi_1, 0) \in \Omega$. By Corollary 1.12 of Rabinowitz [24], we know that there is a continuum of solution-pairs $(\zeta, u) \in \Omega$, whose closure contains the point $(\xi_1, 0)$, that either meets the boundary of Ω, $\partial\Omega$, or the point $(\xi_2, 0)$. We

denote the continuum of solution-pairs emanating from $(\xi_1, 0)$ by $\mathscr{C}_1$, where $\mathscr{C}_1 \subset \Omega$, and from $(\xi_2, 0)$ by $\mathscr{C}_2$, where $\mathscr{C}_2 \subset \Omega$. We introduce the sets

$$Z_1 = \{\zeta \in Z |\ \exists u \text{ such that } (\zeta, u) \in \mathscr{C}_1\}, \tag{A.21a}$$

$$U_1 = \{u \in Y |\ \exists \zeta \text{ such that } (\zeta, u) \in \mathscr{C}_1\}, \tag{A.21b}$$

$$Z_2 = \{\zeta \in Z |\ \exists u \text{ such that } (\zeta, u) \in \mathscr{C}_2\}, \tag{A.21c}$$

$$U_2 = \{u \in Y |\ \exists \zeta \text{ such that } (\zeta, u) \in \mathscr{C}_2\}. \tag{A.21d}$$

We denote the part of Y in the positive quadrant of $\mathbb{R}^2$ by $Y^+ = \{(e_h, e_v) \in Y \,|\, e_h > 0 \text{ and } e_v > 0\}$, and the internal boundary of Y^+ by

$$\partial Y^+ = \left\{ \begin{pmatrix} e_h \\ e_v \end{pmatrix} \in Y \,\middle|\, \begin{pmatrix} e_h > 0 \\ \text{and} \\ e_v = 0 \end{pmatrix} \text{ or } \begin{pmatrix} e_h = 0 \\ \text{and} \\ e_v > 0 \end{pmatrix} \text{ or } \begin{pmatrix} e_h = 0 \\ \text{and} \\ e_v = 0 \end{pmatrix} \right\}.$$

We can determine the initial direction of the continua of solution-pairs, $\mathscr{C}_1$ and $\mathscr{C}_2$, using the Lyapunov–Schmidt expansion, as described by Cushing [9]. Although we show the proofs only for the expansion of $\mathscr{C}_1$ around the bifurcation point at $\zeta = \xi_1$ in Lemmas A.2 and A.3, the results for $\mathscr{C}_2$ around $\zeta = \xi_2$ are similar. We begin by expanding the terms of the nonlinear eigenvalue equation (A.18) about the bifurcation point, $(\xi_1, 0)$. The expanded variables are

$$u = 0 + \varepsilon u^{(1)} + \varepsilon^2 u^{(2)} + \cdots, \tag{A.22a}$$

$$\zeta = \xi_1 + \varepsilon\zeta_1 + \varepsilon^2\zeta_2 + \cdots, \tag{A.22b}$$

$$\begin{aligned} h(\zeta, u) &= h(\xi_1 + \varepsilon\zeta_1 + \varepsilon^2\zeta_2 + \cdots, \varepsilon u^{(1)} + \varepsilon^2 u^{(2)} + \cdots) \\ &= \varepsilon^2 h_2(\xi_1, u^{(1)}) + \cdots. \end{aligned} \tag{A.22c}$$

We substitute the expansions (A.22) into the eigenvalue equation (A.18) and evaluate at different orders of ε. Evaluating the substitution of the expansions (A.22) into the eigenvalue equation (A.18) at $\mathcal{O}(\varepsilon^0)$ produces $0 = 0$, which gives us no information.

LEMMA A.2. *The initial direction of the branch of equilibrium points, $u^{(1)}$, near the bifurcation point, $(\xi_1, 0)$, is equal to the right eigenvector of L corresponding to the characteristic value, ξ_1.*

Proof. Evaluating the substitution of the expansions (A.22) into the eigenvalue equation (A.18) at $\mathcal{O}(\varepsilon^1)$, we obtain $u^{(1)} = \xi_1 L u^{(1)}$. This implies that $u^{(1)}$ is the right eigenvector of L corresponding to the eigenvalue $1/\xi_1$, v (A.20). Thus, close to the bifurcation point, the equilibrium point can be approximated by $e_h = \varepsilon\sqrt{A}$ and $e_v = \varepsilon\sqrt{B}$. ∎

LEMMA A.3. *The bifurcation at $\zeta = \xi_1$ of the nonlinear eigenvalue equation* (A.18) *is supercritical if $\zeta_1 > 0$ and subcritical if $\zeta_1 < 0$, where*

$$\zeta_1 = -\frac{w \cdot h_2}{w \cdot Lv}, \tag{A.23}$$

where v and w are the right and left eigenvectors of L corresponding to the characteristic value ξ_1, respectively.

Proof. Evaluating the substitution of the expansions (A.22) into the eigenvalue equation (A.18) at $\mathcal{O}(\varepsilon^2)$, we obtain $u^{(2)} = \xi_1 L u^{(2)} + \zeta_1 L u^{(1)} + h_2$, which we can

rewrite as

$$(\mathbb{I} - \xi_1 L)u^{(2)} = \zeta_1 Lv + h_2, \tag{A.24}$$

where $\mathbb{I}$ is the 2×2 identity matrix. As ξ_1 is a characteristic value of L, $(\mathbb{I} - \xi_1 L)$ is a singular matrix. Thus, for (A.24) to have a solution, $\zeta_1 Lv + h_2$ must be in the range of $(\mathbb{I} - \xi_1 L)$; i.e., it must be orthogonal to the null space of the adjoint of $(\mathbb{I} - \xi_1 L)$. The null space of the adjoint of $(\mathbb{I} - \xi_1 L)$ is spanned by the left eigenvector of L (corresponding to the eigenvalue $1/\xi_1$), w (A.20). The Fredholm condition for the solvability of (A.24) is $w \cdot (\zeta_1 Lv + h_2) = 0$. Solving for ζ_1 provides (A.23). If ζ_1 is positive, then for small positive ε, $u > 0$ and $\zeta > \xi_1$, and the bifurcation is supercritical. Similarly, if ζ_1 is negative, then for small positive ε, $u > 0$ and $\zeta < \xi_1$, and the bifurcation is subcritical. □

LEMMA A.4. *For all $u \in U_1$, $e_h > 0$ and $e_v > 0$.*

Proof. By Lemma A.1, there are no equilibrium points on ∂Y^+ other than $e_h = e_v = 0$, so $U_1 \cap \partial Y^+ = 0$. We know from Lemma A.2 that close to the bifurcation point $(\xi_1, 0)$, the direction of U_1 is equal to v, the right eigenvector corresponding to the characteristic value, ξ_1. As v contains only positive terms, U_1 is entirely contained in Y^+. Thus, for all $u \in U_1$, $e_h > 0$ and $e_v > 0$. □

LEMMA A.5. *The point $u = 0 \in Y$ corresponds to $x_{dfe} \in \mathbb{R}^7$ (on the boundary of the positive orthant of $\mathbb{R}^7$). For every solution-pair $(\zeta, u) \in \mathscr{C}_1$, there corresponds one equilibrium-pair $(\zeta, x^*) \in Z \times \mathbb{R}^7$, where x^* is in the positive orthant of $\mathbb{R}^7$.*

Proof. We first show that $u = 0$ corresponds to x_{dfe}. As $e_h = e_v = 0$, by Theorem 3.1 we know that the only possible equilibrium point is x_{dfe}. We now show that for every $\zeta \in Z_1$ there exists an x^* in the positive orthant of $\mathbb{R}^7$ for the corresponding $u \in U_1$. By Lemma A.4, we know that $e_h > 0$ and $e_v > 0$. We now need to show that for every positive e_h and e_v there exist corresponding positive i_h, r_h, i_v, N_h, and N_v. By looking at the equilibrium equation for i_v (A.11), we see that for every positive e_v there exists a positive i_v. The equilibrium equation for N_v has a positive and bounded solution, depending only on parameter values (A.10). From $i_h = y(e_h)$, we see that for every positive e_h there exists a positive i_h. The equilibrium equations for r_h (A.13) and N_h (A.12) show that for every positive i_h there exists a positive r_h and N_h. □

LEMMA A.6. *The set U_1 does not meet the boundary of Y.*

Proof. As Lemma A.4 shows us that for all $u \in U_1$, $e_h > 0$ and $e_v > 0$, we need to show that $e_h < e_h^{max}$ and $e_v < 1$. By Lemma A.5, we know that all state variables are positive. Therefore, for (A.8e) to have a solution, $e_v + i_v < 1$ so $e_v < 1$. From the properties of $e_h = g(i_h)$, we know that as i_h increases, e_h increases monotonically, reaching e_h^{max} at $i_h = 1$. However, we have already shown that when $e_h + i_h + r_h = 1$, $e_h' + i_h' + r_h' < 0$, and thus there can be no equilibrium point at $e_h + i_h + r_h = 1$. Therefore, i_h is always less than 1, and e_h is always less than e_h^{max}. □

Proof of Theorem 4.1. As shown in Lemma A.4, $U_1 \cap \partial Y^+ = 0$ and U_1 is entirely contained in Y^+. We can similarly show that U_2 is entirely outside of Y^+ because the right eigenvector corresponding to ξ_2 is $\begin{pmatrix} -\sqrt{A} & \sqrt{B} \end{pmatrix}^{\mathrm{T}}$. Therefore, $\mathscr{C}_1$ and $\mathscr{C}_2$ do not intersect, and by Corollary 1.12 of Rabinowitz [24], $\mathscr{C}_1$ meets $\partial\Omega$. By Lemma A.6, the set U_1 does not meet the boundary of Y, so $\mathscr{C}_1$ meets $\partial\Omega$ only at $\zeta = M_Z$.

By Lemma A.5, for every $u \in U_1$, there corresponds an x^* in the positive orthant of $\mathbb{R}^7$, and $u = 0$ corresponds to x_{dfe} (on the boundary of the positive orthant of $\mathbb{R}^7$). Thus, there exists a continuum of equilibrium-pairs $(\zeta, x^*) \in Z \times \mathbb{R}^7$ that connects the point (ξ_1, x_{dfe}) to the hyperplane $\zeta = M_Z$ in $\mathbb{R} \times \mathbb{R}^7$. □

Proof of Theorem 4.3. When $\delta_h = 0$, we can explicitly evaluate $h(\zeta, u)$ in the nonlinear eigenvalue equation (A.18) from the equilibrium equations (A.8) as

$$h = \zeta \begin{pmatrix} C_{(\delta_h=0)} e_h e_v \\ D_{(\delta_h=0)} e_h e_v \end{pmatrix} \tag{A.25}$$

since the coefficients of all the other higher order terms are zero. Although we do not show the explicit representations for $C_{(\delta_h=0)}$ and $D_{(\delta_h=0)}$, they are both negative. From (A.25) and (A.22) we can evaluate the second order expansion

$$h_2 = \xi_1 \begin{pmatrix} C_{(\delta_h=0)} \sqrt{A}\sqrt{B} \\ D_{(\delta_h=0)} \sqrt{A}\sqrt{B} \end{pmatrix} = \begin{pmatrix} C_{(\delta_h=0)} \\ D_{(\delta_h=0)} \end{pmatrix}. \tag{A.26}$$

As h_2 contains only negative terms and w, v, and L contain only nonnegative terms, (A.23) implies that ζ_1 is positive. Thus, by Lemma A.3, with no disease-induced death, for any positive values of the other parameters there is a supercritical bifurcation at $R_0 = 1$. □

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