THE IMPACT OF RELEASING STERILE MOSQUITOES ON MALARIA TRANSMISSION

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ABSTRACT. The sterile mosquitoes technique in which sterile mosquitoes are released to reduce or eradicate the wild mosquito population has been used in preventing the malaria transmission. To study the impact of releasing sterile mosquitoes on the malaria transmission, we first formulate a simple SEIR (susceptible-exposed-infected-recovered) malaria transmission model as our baseline model, derive a formula for the reproductive number of infection, and determine the existence of endemic equilibria. We then include sterile mosquitoes in the baseline model and consider the case of constant releases of sterile mosquitoes. We examine how the releases affect the reproductive numbers and endemic equilibria for the model with interactive mosquitoes and investigate the impact of releasing sterile mosquitoes on the malaria transmission.

1. Introduction. Mosquito-borne diseases, such as malaria, transmitted between humans by mosquitoes, are big concerns for the public health. Malaria is the fifth cause of death from infectious diseases worldwide (after respiratory infections, HIV/AIDS, diarrheal diseases, and tuberculosis), and the second leading cause of death from infectious diseases in Africa after HIV/AIDS. About three billion and four thousand million people (half of the world’s population) are at risk of malaria transmission living in 106 countries and territories. There were 207 million cases of malaria and 627,000 deaths in 2012, mostly young children in sub-Saharan Africa.

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In 2015, an estimated 214 million cases of malaria occurred worldwide and 438,000 people died due to malaria infection [20]. Between 1957 and 2015, 63 outbreaks of locally transmitted mosquito-borne malaria have occurred, and about 1,700 cases of malaria are diagnosed each year in the United States [7].

Malaria is not spread from a human to a human as influenza or HIV, but transmitted between humans by blood-feeding mosquitoes. No vaccines are available and an effective way to prevent malaria is to control mosquitoes. Among the mosquitoes control measures, the sterile insect technique (SIT) has been proved to be useful and effective [3]. The SIT is a method of biological control in which the natural reproductive process of mosquitoes is disrupted. Utilizing radical or other chemical or physical methods, male mosquitoes are genetically modified to be sterile so that they are incapable of producing offspring despite being sexually active. These sterile male mosquitoes are then released into the environment to mate with the present wild female mosquitoes. A wild female that mates with a sterile male will either not reproduce, or produce eggs, but the eggs will not hatch. Repeated releases of genetically modified mosquitoes or releasing a significantly large number of sterile mosquitoes may eventually wipe out a wild mosquito population, although it is often more realistic to consider controlling the population rather than eradicating it [1,4,21].

While the new development of the SIT has been promising, particularly in laboratories, many questions have to be answered before the techniques can be implemented and the sterile mosquitoes can be deployed in the field. Such questions include the investigation and assessment of the impact of releasing sterile mosquitoes on the malaria transmission.

Mathematical models have proven useful in population dynamics and epidemiology. To gain insight into such challenging questions, mathematical models have been formulated for the interactive dynamics of the wild and sterile mosquitoes and the dynamical features of these models have been well studied [3,6,8,9,14,15,19]. However, the impact of releases of sterile mosquitoes on the malaria transmission is to be studied.

In this paper, we focus on the impact of releases of sterile mosquitoes on the malaria transmission. We first formulate a simple SEIR malaria transmission model as our baseline model in Section 2. We derive formulas of the reproductive number for the baseline model and prove that there exists an endemic equilibrium as the reproductive number exceeds one. We then include the sterile mosquitoes in the baseline malaria model such that the susceptible mosquitoes consist of the wild and sterile mosquitoes in Section 3. We consider the case where the releasing rate of sterile mosquitoes is constant and derive a formula for the reproductive number, and explore the existence of an endemic equilibrium for the extended disease model. We investigate the impact of the releases of sterile mosquitoes on the malaria transmission based on the reproductive number and the endemic equilibrium in Section 4. Numerical examples are also provided to illustrate our findings. We finally give brief discussions in Section 5.

2. Baseline model for malaria transmission. Before studying the impact of the interactive wild and sterile mosquitoes on the malaria transmission, we consider the following simple SEIR malaria model as our baseline model [12,13].

We divide the human population into groups of susceptible, latent or incubating, infective, and recovered individuals. Here the latent or incubating period is defined
as the time from initial infection to the appearance of gametocytes in the blood [2].
Using index \( h \) for the humans, we let \( S_h \) be the number of susceptible humans, \( E_h \)
the number of latent or incubating humans who are infected but not infectious yet, \( I_h \) the number of infective humans who are infected and also infectious, and \( R_h \) the number of the humans who are recovered from infection but partly lose their
immunity [11,16–18]. The model equations for humans are given by

\[
\begin{align*}
\frac{dS_h}{dt} &= \Lambda_h - (\mu_h + \lambda_h) S_h + \theta_h R_h, \\
\frac{dE_h}{dt} &= \lambda_h S_h - (\mu_h + \gamma_h) E_h, \\
\frac{dI_h}{dt} &= \gamma_h E_h - (\mu_h + \delta_h + \eta_h) I_h, \\
\frac{dR_h}{dt} &= \eta_h I_h - (\mu_h + \theta_h) R_h,
\end{align*}
\]

(1)

where \( \Lambda_h \) is the input flow of the susceptible humans, \( \mu_h \) and \( \delta_h \) are the natural
and disease-induced death rates for humans, respectively, \( \theta_h \) is the rate of immunity
loss, \( \gamma_h \) is the developing rate of incubating humans to become infectious, such that
\( 1/\gamma_h \) is the incubation period, \( \eta_h \) is the recovery rate for infectious humans, and \( \lambda_h \)
is the infection rate or the incidence of infection from an infective mosquito to a
susceptible human. Notice that if there is no infection, the human population has
an asymptotically stable steady state \( \lim_{t \to \infty} S_h = \Lambda_h/\mu_h \).

For the mosquito population, we divide it into groups of susceptible, latent,
and infective individuals. Since the mosquito lifespan is usually shorter than their
infective period, no recovered mosquitoes are included. We denote the numbers
of susceptible, latent, and infective mosquitoes by \( S_v \), \( E_v \), and \( I_v \), respectively.
We further follow the line in [15] and assume, in the absence of infection, that the
density dependence occurs in the larvae stages progression, and density independent
death rate for wild mosquitoes. Then model equations for the mosquitoes are given by

\[
\begin{align*}
\frac{dS_v}{dt} &= C(N_v)\alpha_v(1 - \xi_v N_v) N_v - (\mu_v + \lambda_v) S_v, \\
\frac{dE_v}{dt} &= \lambda_v S_v - (\mu_v + \gamma_v) E_v, \\
\frac{dI_v}{dt} &= \gamma_v E_v - \mu_v I_v,
\end{align*}
\]

(2)

where \( C(N_v) \) is the mating rate, \( \alpha_v \) is the maximum number of offspring reproduced
per individual, the term \( 1 - \xi_v N_v \) characterizes the density-dependent larvae stages
progression with \( \xi_v \) being an early-stage carrying capacity parameter, \( N_v = S_v + \\
E_v + I_v \) is the total mosquito population size, \( \mu_v \) is the natural death rate of the
mosquitoes, \( \gamma_v \) is the rate of incubating mosquitoes becoming infectious or \( 1/\gamma_v \) is
the extrinsic incubation period of the parasite within the mosquito or the period
of sporogony, and \( \lambda_v \) is the infection rate from an infective human to a susceptible
mosquito. As in [15], we assume that the intrinsic growth rate of the wild mosquito
population \( r_0 := \alpha_v - \mu_v > 0 \) such that, in the absence of sterile mosquitoes, the
wild mosquito population approaches a positive steady state.
Let $N_h = S_h + E_h + I_h + R_h$ be the total human population size. The infection rates are given by

$$\lambda_v = r_v N_h I_h$$

(3)

and

$$\lambda_h = \beta_v r_v N_h I_v$$

(4)

where $r$ is the number of bites on humans by a single mosquito per unit of time, $\beta_h$ is the transmission probability per bite to a susceptible mosquito from an infective human, and $\beta_v$ is the transmission probability per bite from an infective mosquito to a susceptible human.

Suppose that mosquitoes have no difficulty to find their mates such that the mating rate is constant denoted by $C(N_v) := c$. Then system (2) becomes

$$dS_v dt = \alpha_v (1 - \xi_v N_v) N_v - (\mu_v + \lambda_v) S_v,$$

$$dE_v dt = \lambda_v (N_v - E_v - I_v) - (\mu_v + \gamma_v) E_v,$$

$$dI_v dt = \gamma_v E_v - \mu_v I_v,$$

(5)

where we merge $c$ and $\alpha_v$ but still write it as $\alpha_v$.

Notice that the total population size of mosquitoes satisfies the following logistic equation

$$dN_v dt = (r_0 - \alpha_v \xi_v N_v) N_v,$$

and thus

$$\lim_{t \to \infty} N_v(t) = \frac{r_0}{\alpha_v \xi_v} := N^0_v,$$

for all $N_v(0) > 0$. Instead of system (5), we hereafter consider the following system for the mosquito part in the baseline model

$$dN_v dt = (r_0 - \alpha_v \xi_v N_v) N_v,$$

$$dE_v dt = \lambda_v (N_v - E_v - I_v) - (\mu_v + \gamma_v) E_v,$$

$$dI_v dt = \gamma_v E_v - \mu_v I_v.$$

(6)

Define set

$$\Omega := \left\{(N_h, N_v) : 0 \leq N_h \leq N^0_h, 0 \leq N_v \leq \frac{r_0}{\alpha_v \xi_v}\right\},$$

where $N^0_h := \Lambda_h$. Then $\Omega$ is a positive invariant set for system (1) and (6). We assume $(N_h, N_v) \in \Omega$.

2.1. The reproductive number. We derive a formula for the reproductive number by investigating the local stability of the infection-free equilibrium. The Jacobian matrix at the infection-free equilibrium $(S_h, R_h, I_h, E_h, I_v, E_v, N_v) = (N^0_h, 0, 0, 0, 0, N^0_v)$ has the form of

$$\begin{pmatrix}
J_{21} & 0 \\
0 & J_{22} \\
0 & -r_0
\end{pmatrix}.$$
where

\[
J_{21} := \begin{pmatrix} -\mu_h & \theta_h \\ 0 & -\sigma_3 \end{pmatrix},
\]

\[
J_{22} := \begin{pmatrix} -\sigma_2 & \gamma_h & 0 & 0 \\ 0 & -\sigma_1 & r\beta_v & 0 \\ 0 & 0 & -\mu_v & \gamma_v \\ r\beta_h N_v^0 / N_h^0 & 0 & 0 & -\sigma_v \end{pmatrix},
\]

and we write \(\sigma_1 := \mu_h + \gamma_h\), \(\sigma_2 := \mu_h + \delta_h + \eta_h\), \(\sigma_3 := \mu_h + \theta_h\), and \(\sigma_v := \mu_v + \gamma_v\). Then the infection-free equilibrium is locally asymptotically stable if the eigenvalues of \(J_{22}\) all have negative real part.

Notice that all off-diagonal elements of \(-J_{22}\) are non-positive, and the first three leading principal minors of \(-J_{22}\), \(\sigma_2\), \(\sigma_1\sigma_2\) and \(\sigma_1\sigma_2\mu_v\), are all positive. Then it follows from the M-matrix theory \([5, 10]\) that all eigenvalues of \(J_{22}\) have negative real part and thus the infection-free equilibrium is locally asymptotically stable if the determinant of \(J_{22}\),

\[
\det J_{22} = \sigma_1\sigma_2\mu_v \left(1 - \frac{r^2\beta_h\beta_v\gamma_h\gamma_v N_v^0}{\sigma_1\sigma_2\sigma_v \mu_v N_h^0} \right),
\]

is positive.

Define the reproductive number of infection for system (1) and (6) by

\[
R_0 := \sqrt{\frac{r^2\beta_h\beta_v\gamma_h\gamma_v N_v^0}{(\mu_h + \gamma_h)(\mu_h + \delta_h + \eta_h)(\mu_v + \gamma_v)\mu_v N_h^0}},
\]

(7)

Then

\[
\det J_{22} = \sigma_1\sigma_2\sigma_v \mu_v \left(1 - R_0^2\right).
\]

If \(R_0 < 1\), \(\det J_{22} > 0\), and thus the infection-free equilibrium is locally asymptotically stable. On the other hand, if \(R_0 > 1\), the determinant of \(J_{22}\) is negative which implies that there exists at least one positive eigenvalue of \(J_{22}\). Then the infection-free equilibrium is unstable.

We note that the mean duration of infection within the human population is

\[
\bar{\tau}_h = \frac{\gamma_h}{(\mu_h + \gamma_h)(\mu_h + \delta_h + \eta_h)},
\]

the mean number of bites per human from a mosquito is

\[
\bar{r}_h = \frac{N_v^0}{N_h^0},
\]

and we write \(\bar{\beta}_h = \beta_h\). Then the reproductive number of infection for the human population can be expressed as

\[
R_h^0 := \bar{r}_h \bar{\beta}_h \bar{\tau}_h.
\]

Similarly, we define the mean duration of infection within the mosquito population as

\[
\bar{\tau}_v = \frac{\gamma_v}{\mu_v(\mu_v + \gamma_v)}.
\]

By writing \(\bar{r}_v = r\) and \(\bar{\beta}_v = \beta_v\), the reproductive number of infection for the mosquito population can be expressed as

\[
R_v^0 := \bar{r}_v \bar{\tau}_v \bar{\beta}_v.
\]
Then the reproductive number can be rewritten as
\[ R_0 = (R_0^h R_0^v)^{1/2}. \]

### 2.2. Endemic equilibrium

We next investigate the existence of endemic equilibrium when \( R_0 > 1 \).

The components for the humans at an endemic equilibrium can be solved, in terms of \( \lambda_h \), as

\[ S_h = \frac{\Lambda_h}{\mu_h (1 + K_1 \lambda_h)}, \quad E_h = \frac{\Lambda_h \lambda_h}{\mu_h \sigma_1 (1 + K_1 \lambda_h)}, \quad R_h = \frac{\Lambda_h \lambda_h \gamma_h \eta_h}{\mu_h \sigma_2 \sigma_3 (1 + K_1 \lambda_h)}, \]

where \( K_1 := \frac{1}{\mu_h} - \theta_h \frac{\eta_h \gamma_h}{\mu_h \sigma_1 \sigma_2 \sigma_3} > 0 \). We further write \( K_2 := \frac{\sigma_2 \sigma_3 (1 + K_1 \lambda_h)}{\mu_h \sigma_1 \sigma_2 \sigma_3} \) and have

\[ N_h = \frac{\Lambda_h (1 + K_2 \lambda_h)}{\mu_h (1 + K_1 \lambda_h)}. \]

**Remark 1.** We notice that it follows from

\[ \mu_h \sigma_1 \sigma_2 \sigma_3 (K_1 - K_2) = \sigma_1 \sigma_2 \sigma_3 - \theta \eta_h \gamma_h - \mu_h (\sigma_2 \sigma_3 + \sigma_3 \gamma_h + \gamma_h \eta_h) \]
\[ = \sigma_2 \sigma_3 (\sigma_1 - \mu_h) - \mu_h \sigma_3 \gamma_h - \eta_h \gamma_h (\theta_h + \mu_h) \]
\[ = \gamma_h \sigma_2 \sigma_3 - \gamma_h \sigma_3 (\mu_h + \eta_h) \]
\[ = \gamma_h \sigma_3 (\sigma_2 - (\mu_h + \eta_h)) = \gamma_h \sigma_3 \delta_h, \]

that

\[ K_1 - K_2 = \frac{\gamma_h \delta_h}{\mu_h \sigma_1 \sigma_2} > 0. \]

Substituting \( I_h \) in (8) and \( N_h \) in (9) into (3) yields

\[ \lambda_v = B_1 \frac{\lambda_h}{1 + K_2 \lambda_h}, \]

where \( B_1 := \frac{r \beta_h \gamma_h}{\sigma_1 \sigma_2} \). It follows from (6) that

\[ I_v = \frac{\gamma_v}{\mu_v} E_v, \]

and then

\[ \lambda_v N_v = \lambda_v (E_v + I_v) + \sigma_v E_v = \left( \lambda_v + \sigma_v + \frac{\lambda_v \gamma_v}{\mu_v} \right) E_v \]
\[ = \frac{E_v}{\mu_v} \left( \lambda_v \sigma_v + \mu_v \sigma_v \right) = \frac{\sigma_v E_v}{\mu_v} (\lambda_v + \mu_v). \]

Hence

\[ E_v = \frac{\mu_v N_v}{\sigma_v} \frac{\lambda_v}{\lambda_v + \mu_v}, \]

and

\[ I_v = \frac{\gamma_v N_v}{\sigma_v} \frac{\lambda_v}{\lambda_v + \mu_v} = \frac{\gamma_v r_0}{\alpha_v \xi_v \sigma_v} \frac{B_1 \lambda_h}{\mu_v (1 + \mu_v \lambda_h)}. \]
Substituting (10) into (4), we have
\[ \lambda_h = \frac{r \beta_v \mu_v (1 + K_1 \lambda_h)}{\lambda_h (1 + K_2 \lambda_h)} \quad \gamma_v r_0 B_1 \lambda_h \\
= \frac{r^2 \beta_v \mu_v \gamma_v \gamma_h r_0 \mu_v (1 + K_1 \lambda_h) \lambda_h}{\lambda_h \sigma \mu_v (1 + K_2 \lambda_h) \mu_v (B_1 + \mu_v K_2) \lambda_h} \\
= R^2_0 \frac{\mu_v (1 + K_1 \lambda_h) \lambda_h}{(1 + K_2 \lambda_h) (\mu_v + (B_1 + \mu_v K_2) \lambda_h)}.
\]

Write \( \lambda_h = \lambda \) for convenience and define the quadratic function
\[ g(\lambda) := (1 + K_2 \lambda) (\mu_v + (B_1 + \mu_v K_2) \lambda) - R^2_0 \mu_v (1 + K_1 \lambda) \\
= (B_1 + \mu_v K_2) (1 + K_2 \lambda) \lambda + \mu_v \left( (1 + K_2 \lambda) - R^2_0 (1 + K_1 \lambda) \right) \\
= K_2 (B_1 + \mu_v K_2) \lambda^2 + \left( B_1 + \mu_v (2 K_2 - R^2_0 K_1) \right) \lambda + (1 - R^2_0) \mu_v.
\]

Then there exists a positive endemic equilibrium if and only if there exists a positive solution \( \lambda > 0 \) to equation \( g(\lambda) = 0 \).

Clearly, if \( R_0 > 1 \), there exists a unique positive solution to \( g(\lambda) = 0 \) and hence a unique endemic equilibrium.

For \( R_0 = 1 \), we have
\[ g(\lambda) = K_2 (B_1 + \mu_v K_2) \lambda^2 + \left( B_1 + \mu_v (2 K_2 - R^2_0 K_1) \right) \lambda \\
= (B_1 + \mu_v K_2) (1 + K_2 \lambda) \lambda - \mu_v (K_1 - K_2) \lambda.
\]

Since \( K_1 - K_2 > 0 \), there exists a positive solution
\[ \lambda = \frac{\mu_v (K_1 - 2 K_2) - B_1}{K_2 (B_1 + \mu_v K_2)}
\]
to \( g(\lambda) = 0 \) if and only if
\[ \mu_v (K_1 - 2 K_2) > B_1.
\]

That is
\[ \frac{\gamma_h \delta_h}{\mu_h \sigma_2} > \frac{r \beta_h \gamma_h}{\mu_v \sigma_2} + \frac{\sigma_2 \sigma_3 + \sigma_3 \gamma_h + \gamma_h \eta_h}{\sigma_1 \sigma_2 \sigma_3},
\]
or
\[ \gamma_h \delta_h \mu_v \sigma_3 > r \beta_h \gamma_h \mu_v \sigma_3 + \mu_h \mu_v \left( \sigma_2 \sigma_3 + \sigma_3 \gamma_h + \gamma_h \eta_h \right),
\]
or
\[ \mu_h \gamma_h \eta_h \mu_v + \sigma_3 \left( r \beta_h \gamma_h \mu_h + \mu_h \mu_v \gamma_h + \mu_h \mu_v \delta_h \mu_v \right) \\
= \mu_h \gamma_h \eta_h \mu_v + \sigma_3 \left( r \beta_h \gamma_h \mu_h + \mu_h \mu_v \gamma_h + \mu_h \mu_v (\mu_h + \delta_h + \eta_h) - \gamma_h \delta_h \mu_v \right) \\
= \mu_h \gamma_h \eta_h \mu_v + \sigma_3 \left( r \beta_h \gamma_h \mu_h + \mu_v \left( \mu_h \gamma_h + \mu_h \mu_v + \mu_h \delta_h + \mu_h \eta_h - \gamma_h \delta_h \right) \right) < 0.
\]

For \( R_0 < 1 \), if
\[ \mu_v (K_1 - 2 K_2) - B_1 \leq 0. \quad \text{Hence } \mu_v (R^2_0 K_1 - 2 K_2) \leq B_1, \text{ that is, } B_1 + \mu_v (2 K_2 - R^2_0 K_1) \geq 0, \]
then \( \mu_v (K_1 - 2 K_2) - B_1 \leq 0. \)

In summary, we have the following results.

**Theorem 2.1.** **System (1) and (6) has a unique endemic equilibrium if \( R_0 > 1 \). Under condition (11), there exists no endemic equilibrium for system (1) and (6) if \( R_0 < 1 \).**
Remark 2. Condition (11) should be satisfied for feasible biological parameter sets and hence we don’t anticipate the existence of endemic equilibria for system (1) and (6) when $R_0 < 1$.

3. The models with sterile mosquitoes. We now include the sterile mosquito population in the baseline model. The part of the model equations for the humans is the same as in system (1). We let $S_g$ be the number of sterile mosquitoes and assume that the rate of releases is a constant, denoted by $b$. Then the part for the mosquitoes in the disease transmission model is given by the following system

\[
\begin{align*}
\frac{dS_v}{dt} &= \frac{\alpha_v N_v}{N_v + S_g} (1 - \xi_v N_v) N_v - (\mu_v + \lambda_v) S_v, \\
\frac{dE_v}{dt} &= \lambda_v S_v - (\mu_v + \gamma_v) E_v, \\
\frac{dI_v}{dt} &= \gamma_v E_v - \mu_v I_v, \\
\frac{dS_g}{dt} &= b - \mu_v S_g,
\end{align*}
\]

(12)

where we assume that the sterile mosquitoes have the same death rate as the wild mosquitoes.

Notice that the dynamics of the total wild mosquitoes are determined by

\[
\frac{dN_v}{dt} = \frac{\alpha_v N_v}{N_v + S_g} (1 - \xi_v N_v) N_v - \mu_v N_v.
\]

Thus, instead of system (12), we use the following system for the part of the mosquitoes in the disease transmission model

\[
\begin{align*}
\frac{dN_v}{dt} &= \frac{\alpha_v N_v}{N_v + S_g} (1 - \xi_v N_v) N_v - \mu_v N_v, \\
\frac{dE_v}{dt} &= \lambda_v (N_v - E_v - I_v) - (\mu_v + \gamma_v) E_v, \\
\frac{dI_v}{dt} &= \gamma_v E_v - \mu_v I_v, \\
\frac{dS_g}{dt} &= b - \mu_v S_g.
\end{align*}
\]

(13)

Since the interactive dynamics between the total wild and sterile mosquitoes are governed by the following system

\[
\begin{align*}
\frac{dN_v}{dt} &= \frac{\alpha_v N_v}{N_v + S_g} (1 - \xi_v N_v) N_v - \mu_v N_v, \\
\frac{dS_g}{dt} &= b - \mu_v S_g,
\end{align*}
\]

(14)

it follows from [15] that the results for system (14) can be stated as follows.

Lemma 3.1 (Theorem 3.1 [15]). Assume that the intrinsic growth rate of the wild mosquito population $r_0 := \alpha_v - \mu_v > 0$. System (14) has a boundary equilibrium $E_c^0 = (0, g^0)$ with $g^0 = b/\mu_v$, which is globally asymptotically stable if there exists no positive equilibrium, and locally asymptotically stable if a positive equilibrium
exists. System (14) has no, one, or two positive equilibria if \( b > b_c, b = b_c, \) or \( b < b_c, \) respectively, where the release threshold \( b_c \) is given by

\[
b_c := \frac{r_0^2}{4\alpha_v\xi_v}.
\]

For given \( b < b_c, \) the two positive equilibria of system (14) are \( E_c^- = (N_{eb}^-, g^0) \) and \( E_c^+ = (N_{eb}^+, g^0), \) where

\[
N_{eb}^\pm = \frac{r_0 \pm \sqrt{r_0^2 - 4\alpha_v\xi_v b}}{2\alpha_v\xi_v}.
\]

Equilibrium \( E_c^- \) is an unstable saddle and \( E_c^+ \) is a locally asymptotically stable node.

For \( b < b_c, \) the stable manifolds of \( E_c^- \) divides the first quadrant of the \( S_gN_v \)-plane into a region, denoted \( D_b, \) and its complement \( \mathbb{R}^2_+ \setminus D_b \) such that solutions of (14) with initial values in \( D_b \) approach \( E_c^0 \) as \( t \to \infty; \) that is, the wild mosquitoes are eventually wiped out if the initial sizes of the interactive wild and sterile mosquitoes are in \( D_b. \) The two types of mosquitoes coexist if their initial sizes are in \( \mathbb{R}^2_+ \setminus D_b. \)

We approximate region \( D_b \) by the region determined by the eigenvectors of the Jacobian matrix of system (14) at \( E_c^- = (N_{eb}^-, g^0) \) as follows.

Write the Jacobian matrix of (14) at the equilibrium \( E_c^- \) as \( J. \) Then we have

\[
J = \begin{pmatrix} J_{11} & J_{12} \\ 0 & -\mu_v \end{pmatrix},
\]

where

\[
J_{11} = \frac{\alpha_v N_{eb}^-}{(N_{eb}^- + g^0)^2} (g^0 (1 - \xi_v N_{eb}^-) - \xi_v N_{eb}^- (N_{eb}^- + g^0)) > 0,
\]

\[
J_{12} = -\frac{\alpha_v (N_{eb}^-)^2}{(N_{eb}^- + g^0)^2} (1 - \xi_v N_v) < 0.
\]

The eigenvalues of matrix \( J \) are \( \lambda_1 = -\mu_v \) and \( \lambda_2 = J_{11}. \) Let \( \vec{\eta}_1 \) and \( \vec{\eta}_2 \) be the eigenvectors corresponding to eigenvalues \( \lambda_1 \) and \( \lambda_2, \) respectively. Then we have \( \vec{\eta}_1 = (1, 0)^T \) and \( \vec{\eta}_2 = \left(1, \frac{-J_{11} + \mu_v}{J_{12}}\right)^T. \) Define region

\[
\tilde{\Omega}_b := \left\{(N_v, S_g) : 0 \leq S_g \leq \frac{b}{\mu_v}, 0 \leq N_v \leq N_{eb}^- - \frac{J_{12}}{J_{11} + \mu_v} (S_g - \frac{b}{\mu_v}) \right\}.
\]

Then clearly region \( D_b \) is a subset of \( \tilde{\Omega}_b. \)

Our objective in this study is to investigate the impact of releases of sterile mosquitoes on the transmission dynamics of malaria. If \( b > b_c, \) all wild mosquitoes will eventually go extinct and then there will be no malaria transmission. Thus we only consider \( b < b_c \) and for given \( b < b_c, \) we assume that the initial sizes of the interactive wild and sterile mosquitoes are not in \( \tilde{\Omega}_b \) hereafter. Moreover, it is clearly that \( \tilde{\Omega}_b \) is related to \( b. \) If we consider \( b \) as a variable, then \( \lim_{b \to 0} N_{eb}^-(b) = 0 \) and \( \lim_{b \to 0} g^0(b) = 0, \) which implies that region \( \tilde{\Omega}_b \) is small as \( b > 0 \) is small.

3.1. **The reproductive number and disease spread.** We derive, in this section, a formula for the reproductive number of infection after the sterile mosquitoes are released into the wild mosquito population with \( b < b_c \) and the initial sizes of the two types of mosquitoes in \( \mathbb{R}^2_+ \setminus \tilde{\Omega}_b. \)
The Jacobian matrix at an equilibrium \((S_h, R_h, E_h, I_v, E_v, N_v, S_g)\) for system (1) and (13) is given by
\[
\left( \begin{array}{cc}
\hat{J} & J_{33} \\
0 & J_{33}
\end{array} \right),
\]
where \(J_{33}\) is the Jacobian matrix of system (14) at either a boundary or a positive equilibrium with \(N_v\) evaluated at the equilibrium. Hence the equilibrium of system (1) and (13) corresponding to \(E_c^- = (N_{vb}^-, g^0)\), if exists, is always unstable, and thus we only consider the Jacobian matrix evaluated at the equilibrium involving the positive equilibrium \(E_c^+ = (N_{vb}^+, g^0)\) of system (14).

At the infection-free equilibrium \((S_h, R_h, I_h, E_h, I_v, E_v, N_v, S_g) = (N_0^h, 0, 0, 0, 0, 0, N_{vb}, S_g)\), the Jacobian matrix of system (1) and (13) has the form of
\[
\hat{J} = 
\left( \begin{array}{cc}
J_{31} & J_{32} \\
0 & J_{32}
\end{array} \right),
\]
where
\[
J_{31} = \begin{pmatrix}
-\mu_h & \theta_h \\
0 & -\sigma_3
\end{pmatrix},
\]
and
\[
J_{32} = 
\begin{pmatrix}
-\sigma_2 & \gamma_h & 0 & 0 \\
0 & -\sigma_1 & \gamma_v & 0 \\
0 & 0 & -\mu_v & \gamma_v \\
r\beta_h N_{vb}/N_h^0 & 0 & 0 & -\sigma_v
\end{pmatrix}.
\]

Since the eigenvalues of matrix \(J_{33}\) at equilibrium \(E_c^+\) both have negative real part, the local stability of the infection-free equilibrium is determined by matrix \(J_{32}\). Denote the reproductive number for system (1) and (13) associated with \(E_c^+\) by \(R_c^0\). Then, it follows from Section 2.1 that
\[
R_c^0 := \sqrt{rac{r^2 \beta_h \beta_v \gamma_h \gamma_v N_{vb}^+}{(\mu_h + \gamma_h)(\mu_h + \delta_h + \eta_h)(\mu_v + \gamma_v)\mu_v N_h}},
\]
where \(N_{vb}^+\) is given in (16). The biological interpretation of \(R_c^0\) is similar to that for \(R_0\) in Section 2.1.

Using \(b\) as a variable, we note that \(N_{vb}^+\) is a function of \(b\) and so is \(R_c^0\). When \(b = 0\), it is clear that \(N_{vb}^+ = N_v^0\) and \(R_c^0 = R_0\) given in (7). Then we have
\[
R_c^0(b) = \sqrt{\frac{N_{vb}^+(b)}{N_v^0}} R_0.
\]
Moreover, for \(0 < b < b_c\), we have \(0 < N_{vb}^+ < N_v^0\) and thus \(R_c^0(b) < R_0\).

Suppose \(R_0 = R_c^0(0) > 1\). Since \(N_{vb}^+(b)\) is a decreasing function of \(b\), it follows from (16) that there is a threshold value \(\bar{b}\) determined by \(R_c^0(\bar{b}) = 1\), that is,
\[
N_{vb}^+(\bar{b}) = \frac{N_v^0}{R_c^0},
\]
such that
\[
R_c^0(b) \begin{cases} > 1, & \text{if } b < \bar{b}, \\
< 1, & \text{if } b > \bar{b}.
\end{cases}
\]
Indeed, threshold \(\bar{b}\) can be explicitly solved as follows.
Write $C := \frac{N^0_v}{R^2_0}$. Then $N^+_v(b) = C$, which is equivalent to

$$r_0 + \sqrt{r_0^2 - 4 \alpha_v \xi_v b} = 2 \alpha_v \xi_v C,$$

that is,

$$\sqrt{r_0^2 - 4 \alpha_v \xi_v b} = 2 \alpha_v \xi_v C - r_0. \quad (18)$$

Square both sides of (18). Then simple algebra yields

$$b = C (r_0 - \alpha_v \xi_v C). \quad (19)$$

It follows from $N^0_v = \frac{r_0}{\alpha_v \xi_v}$ and (19) then that

$$\bar{b} := \frac{\alpha_v \xi_v (N^0_v)^2}{R^2_0} \left(1 - \frac{1}{R^2_0}\right). \quad (20)$$

Thus, the infection-free equilibrium of system (1) and (13) associated with the positive equilibrium $E^+_c$ of system (14) is locally asymptotically stable if $b > \bar{b}$ and unstable if $b < \bar{b}$.

We also note that if $b = b_c$, the unique positive equilibrium of system (14) is unstable and if $b > b_c$, there exists no positive equilibrium. That is to say that all wild mosquitoes will be wiped out if $b \geq b_c$ and hence $\bar{b} < b_c$. We summarize the results as follows.

**Theorem 3.2.** Assume sterile mosquitoes are released into the wild mosquito population constantly with the rate of releases $b$. Define two threshold values of releases $b_c$ and $\bar{b}$ in (15) and (20), respectively. Then we have the following.

- If $b > b_c$, there exists no positive equilibrium of the interactive mosquitoes system (14) and the only boundary equilibrium $E^0_v$ of (14) is globally asymptotically stable. All wild mosquitoes are wiped out and there is no infection.
- If $b = b_c$, the unique positive equilibrium of system (14) is unstable and boundary equilibrium $E^0_v$ is also globally asymptotically stable. All wild mosquitoes are wiped out as well and there is no infection.
- If $b < b < b_c$, the sterile and wild mosquitoes coexist, but the reproductive number $R^2_0 < 1$ and the infection-free equilibrium of system (1) and (13) associated with the locally asymptotically stable positive equilibrium $E^+_c$ is asymptotically stable. Thus the infection eventually goes extinct.
- If $b < \bar{b} < b_c$, then $R^2_0 > 1$ and the infection-free equilibrium of system (1) and (13) associated with $E^+_c$ is unstable. The disease spreads when the initial sizes of the wild and sterile mosquitoes are not in region $\bar{\Omega}_b$, that is, $(N_v(0), S_g(0)) \notin \bar{\Omega}_b$.

3.2. **Endemic equilibrium.** Similarly as in Section 2.2, we determine the existence of endemic equilibria of system (1) and (13) as follows.

The components of wild mosquitoes at an endemic equilibrium satisfy the following system

$$0 = \frac{\alpha_v \mu_v N_v}{\mu_v N_v + b} (1 - \xi_v N_v) N_v - \mu_v N_v, \quad (21a)$$

$$0 = \lambda_v S_v - (\mu_v + \gamma_v) E_v, \quad (21b)$$

$$0 = \gamma_v E_v - \mu_v I_v. \quad (21c)$$
which leads to
\[
\frac{\alpha_v \mu_v N_v}{\mu_v N_v + b} (1 - \xi_v N_v) N_v = \mu_v N_v.
\]
It follows from (21b) and (21c) that
\[
E_v = \frac{\lambda_v}{\sigma_v} S_v, \quad I_v = \frac{\gamma_v \lambda_v}{\mu_v \sigma_v} S_v,
\]
and then
\[
N_v = \left(1 + \frac{\lambda_v}{\sigma_v} + \frac{\gamma_v \lambda_v}{\mu_v \sigma_v}\right) S_v = \frac{\mu_v + \lambda_v}{\mu_v} S_v.
\]
Thus
\[
S_v = \frac{\mu_v}{\mu_v + \lambda_v} N_v,
\]
and
\[
I_v = \frac{\gamma_v \lambda_v}{\sigma_v (\mu_v + \lambda_v)} N_v = \frac{\gamma_v B_1 \lambda_h N_v}{\sigma_v (\mu_v + (B_1 + \mu_v K_2) \lambda_h)}.
\]
Substituting (22) into (4) yields
\[
\lambda_h = \frac{r \beta_v N_v}{N_h} = \frac{r \beta_v \mu_h (1 + K_1 \lambda_h)}{\Lambda_h (1 + K_2 \lambda_h)} \frac{\gamma_v B_1 \lambda_h N_v}{\sigma_v (\mu_v + (B_1 + \mu_v K_2) \lambda_h)},
\]
which leads to
\[
1 = \frac{r \beta_v \mu_h (1 + K_1 \lambda_h)}{\Lambda_h (1 + K_2 \lambda_h)} \frac{\mu_v \gamma_v B_1 N_v}{\sigma_v (\mu_v + (B_1 + \mu_v K_2) \lambda_h) \mu_v} \frac{r^2 \beta_v \mu_h \gamma_h \mu_v (1 + K_1 \lambda_h) N_v}{\Lambda_h \sigma_v \sigma_v \sigma_v \mu_v (1 + K_2 \lambda_h) (\mu_v + (B_1 + \mu_v K_2) \lambda_h)} \left((R_0^c)^2 \frac{N_v}{N_v} (1 + K_2 \lambda_h) (\mu_v + (B_1 + \mu_v K_2) \lambda_h) \right).
\]
Define
\[
G(\lambda_h, N_v) := K_2 (B_1 + \mu_v K_2) \lambda_h^2 + \left(B_1 + \mu_v \left(2K_2 - (R_0^c)^2 K_1 \frac{N_v}{N_v} + (R_0^c)^2 \mu_v \right) \right) \lambda_h
\]
\[
+ \left(1 - (R_0^c)^2 \frac{N_v}{N_v} \right) \mu_v.
\]
Then the endemic equilibria of system (1) and (13) correspond to the positive roots of \(G(\lambda_h, N_v) = 0\).
For \(N_v = N_{vb}^1\), (23) becomes
\[
G(\lambda_h, N_{vb}^1) := K_2 (B_1 + \mu_v K_2) \lambda_h^2 + \left(B_1 + \mu_v \left(2K_2 - (R_0^c)^2 K_1 \right) \right) \lambda_h
\]
\[
+ \left(1 - (R_0^c)^2 \right) \mu_v.
\]
Equation \(G(\lambda_h, N_{vb}^1) = 0\) has unique positive root and thus system (1) and (13) has a unique endemic equilibrium if \(R_0^c > 1\).
The existence of backward bifurcation for system (1) and (12) can be discussed in a similar way as in Section 2.2 and is skipped.
4. Impact of releases of sterile mosquitoes. To study the impact of releases of sterile mosquitoes, we consider the interval $(0, \hat{b})$. For each $b \in (0, \hat{b})$, the corresponding reproductive number $R_0^0(b) > 1$ and there exists a unique endemic equilibrium associated with $N_v^+(b)$ given in (16).

Again, for $N_v = N_v^+$ and in $G(\lambda_h(b), N_v^+(b)) = 0$, that is, in

\[
K_2(B_1 + \mu_vK_2)\lambda_h^2(b) + \left(B_1 + \mu_v \left(2K_2 - (R_0^0(b))^2 K_1\right)\right) \lambda_h(b) + \left(1 - (R_0^0(b))^2\right) \mu_v = 0,
\]

by taking the derivative with respect to $b$, we have

\[
(2K_2(B_1 + \mu_vK_2)\lambda_h(b) + B_1 + \mu_v(2K_2 - (R_0^0(b))^2 K_1)) \lambda_h'(b) - 2\mu_v R_0^0(b) R_0^{-1}(b)(1 + K_1 \lambda_h(b)) = 0.
\]

Solving for $\lambda_h'(b)$ then yields

\[
\lambda_h'(b) = \frac{2\mu_v R_0^0(b) R_0^{-1}(b)(1 + K_1 \lambda_h(b))}{2K_2(B_1 + \mu_vK_2)\lambda_h(b) + B_1 + \mu_v \left(2K_2 - (R_0^0(b))^2 K_1\right)}
\]

\[
= \frac{2\mu_v R_0^0(b) R_0^{-1}(b)(1 + K_1 \lambda_h(b)) \lambda_h(b)}{K_2(B_1 + \mu_vK_2)\lambda_h^2(b) - \left(1 - (R_0^0(b))^2\right) \mu_v} < 0
\]

for $R_0^0(b) > 1$ since $R_0^0(b) < 0$, and thus it follows from (8) that

\[
I_v'(b) = \frac{\Lambda \gamma_h \lambda_h^2(b)}{\mu_h \sigma_1 \sigma_2 (1 + K_1 \lambda_h(b))^2} < 0.
\]

Moreover, it follows from (16) that $N_v^+(b) < 0$, and follows from (22) that

\[
I_v'(b) = \frac{\gamma_v B_1 \left(\mu_v (\lambda_h'(b) N_v^-(b) + \lambda_h(b) N_v^+(b)) + (B_1 + \mu_vK_2)\lambda_h^2 N_v^+(b)\right)}{\sigma_v \left(\mu_v + (B_1 + \mu_vK_2)\lambda_h(b)\right)^2} < 0.
\]

Therefore, for $b \in (0, \hat{b})$, even though $R_0^0(b) > 1$ such that the disease spreads and goes to a positive steady state as $t \to \infty$, with the increase of the releases of sterile mosquitoes, we can reduce the components of the infected humans and mosquitoes to get the transmission under control.

We provide an example below to demonstrate our findings.

Example 1. We use the following parameters for the malaria transmission

\[
\alpha_v = 10, \quad \mu_v = 0.2, \quad \xi_v = 0.3, \quad \Lambda_h = 20, \quad \mu_h = 0.12, \quad \theta_h = 0.5, \quad \delta_h = 0.5, \quad \gamma_h = 0.6, \quad \eta_h = 0.7, \quad \gamma_v = 0.7, \quad r = 21, \quad \beta_h = 0.2, \quad \beta_v = 0.3.
\]

(25)

Before the sterile mosquitoes are introduced, the reproductive number of infection for system (1) and (6) is $R_0 = 1.1284 > 1$ and hence the malaria infection spreads. After the releases of sterile mosquitoes, we have the existence threshold value $b_c = 8.0033$ such that if $b < 8.0033$, there exist two positive equilibria

\[
N_v^\pm(b) = 1.6333 \pm 0.1667\sqrt{96.04 - 12b},
\]

where $N_v^-(b)$ is unstable and $N_v^+(b)$ is asymptotically stable for all $b < 8.0033$. Equilibrium $N_v^+(b)$ is a decreasing function of $b$. However, the threshold value for the disease spread is $\hat{b} = 5.3960$ such that the reproductive number $R_0^0(b) < 1$ and thus the disease dies out eventually if $b > 5.3960$. If $b < 5.3960$, the reproductive
number $R^*_0(b) > 1$, the infection-free equilibrium becomes unstable, and thus the disease spreads. It is shown in Figure 1.

![Figure 1](image1.png)

**Figure 1.** With the parameters given in (25), the threshold values are $\bar{b} = 5.3960$ and $b_c = 8.0033$. By using $b$ as an independent variable, the horizontal axis is for $b$ and the vertical axis is for $R^*_0$. The curve in this figure represents the reproductive number $R^*_0(b)$ for $0 \leq b \leq b_c$. The reproductive number $R^*_0(0) = R_0 = 1.1284 > 1$ at $b = 0$. At $b = \bar{b}$, the curve for $R^*_0(b)$ crosses the horizontal line $R^*_0 = 1$ so that $R^*_0(b) < 1$ for $\bar{b} < b \leq b_c$.

For $0 < b < 5.3960$, $R^*_0(b) > 1$, and the corresponding $\lambda_h(b)$ determined in (24) is a positive and decreasing function as shown in the left figure in Figure 2. Corresponding to $\lambda_h(b)$, there exists a unique endemic equilibrium for each $b$ whose components $I_h(b)$ and $I_v(b)$ are also decreasing functions of $b$ as shown in the right figure in Figure 2, which indicates that increasing of the releases of sterile mosquitoes reduces the disease spread.

![Figure 2](image2.png)

**Figure 2.** With the parameters given in (25), the threshold values are $\bar{b} = 5.3960$ and $b_c = 8.0033$, respectively. The curve on the left figure is for $\lambda_h(b)$ at the endemic equilibrium for each $b$. The upper and lower curves are for $I_h(b)$ and $I_v(b)$, respectively, at the endemic equilibrium for each $b$ as well in the right figure. Clearly, $\lambda_h(b)$, $I_h(b)$, and $I_v(b)$ all become negative for $b > \bar{b}$ which implies that no endemic equilibrium exists for $b \geq \bar{b}$ although positive $N^+_v(b)$ exist for $\bar{b} < b < b_c$.

To show the transit dynamical impact of the releases of the sterile mosquitoes, we also present the solutions of the disease transmission system versus $t$ in Figure 3.
When no sterile mosquitoes are released, the reproductive number $R_0 = 1.1284 > 1$ and the disease spreads as shown in the left figure. For $b = 6 > \bar{b} = 5.3960$, the reproductive number is reduced to $R_0^c = 0.9773 < 1$ and hence the infection goes extinct eventually, as shown in the right figure in Figure 3.

**Figure 3.** With the parameters given in (25), the reproductive number for system (1) and (6) is $R_0 = 1.1284 > 1$ and hence the infection spreads when there are no sterile mosquitoes released as shown in the left figure. After the sterile mosquitoes are introduced, for $b = 6 > \bar{b} = 5.3960$, the reproduction number becomes $R_0^c = 0.9773 < 1$ and hence the infection goes extinct as shown in the right figure.

5. **Concluding remarks.** To study the impact of releasing sterile mosquitoes on malaria transmission, we first formulated a simple compartmental SEIR model for malaria transmission in (1) and (6) as our baseline model in Section 2. We derived a formula for the reproductive number of infection $R_0$ in (7) for the baseline model (1) and (6), and showed that the infection-free equilibrium of the baseline model is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. We also showed that if $R_0 > 1$, there exists a unique endemic equilibrium for the baseline model, and established a condition to exclude the existence of backward bifurcation for the system when $R_0 < 1$, which would be satisfied for any biologically feasible set of parameters.

We then included the sterile mosquitoes in the baseline model in Section 3, and arrived at the models with sterile mosquitoes whose human component are the same as those in (1) but the mosquito components are given in (12). The interactive dynamics of the wild and sterile mosquitoes are governed by the same model equations in [15], and we only considered the case of constant releases. We derived a formula for the reproductive number $R_0^c$, presented in (17), for the model with sterile mosquitoes (1) and (13) where system (13) is equivalent to (12). We showed that the infection-free equilibrium of system (1) and (13) is asymptotically stable if $R_0^c < 1$ and unstable if $R_0^c > 1$. Using the constant rate of releases $b$ as an independent variable, we determined threshold value $b_c$ for the existence of positive equilibrium for the interactive wild and sterile mosquitoes and threshold value $\bar{b}$ that ascertains whether $R_0 < 1$ or $R_0^c > 1$: that is, whether the disease dies out or spreads. We also showed the existence of a unique endemic equilibrium when $R_0^c > 1$.

We studied the impact of releases of sterile mosquitoes on the transmission dynamics in Section 4 by investigating the changes of the reproductive number $R_0^c(b)$,
and the infective components $I_h(b)$ and $I_v(b)$, induced by $\lambda_h(b)$, as $b$ varies, based on the threshold values $b_c$ and $\bar{b}$. We provided Example 1 to confirm and demonstrate our findings. If the rate of releases $b$ is greater than threshold $b_c$, boundary equilibrium $E^b_0$ for the interactive wild and sterile mosquitoes is the only equilibrium and is globally asymptotically stable. All wild mosquitoes are wiped out and thus there is no infection. On the other hand, if $b < b_c$, while the wild mosquitoes cannot be wiped out and the interactive wild and sterile mosquitoes coexist, the disease can still go extinct when there are sufficient sterile mosquitoes released with $\bar{b} < b < b_c$ which leads to $R^b_0 < 1$. Even if we are unable to release enough sterile mosquitoes with $b < \bar{b}$ which results to $R^b_0 > 1$ and thus the disease spreads when the initial wild and sterile mosquitoes are not in region $\Omega_b$, the infective components $I_h(b)$ and $I_v(b)$ are decreased; that is, the infection is reduced, as we increase the rate of releases $b$.

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