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A Mathematical Modelling Approach to the Transmission Dynamics of Malaria

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ABSTRACT

This study presents a modified mathematical model for malaria transmission dynamics, refining an existing SEIR model to account for disease dynamics in human populations. The modified model incorporates assumptions on susceptibility, mortality and disease progression in human populations. Specifically, the model considers that newborns are initially susceptible, all human compartments are subject to natural death rates and infected humans face an additional disease-induced mortality rate. Unique aspects of the modified model include the assumption that recovered individuals without immunity may become susceptible again, allowing the model to reflect realistic malaria relapse patterns. The study further refines the transmission dynamics by incorporating a schematic representation and corresponding equations to describe the flow between susceptible, exposed, infected and recovered compartments for both humans and mosquitoes. This model is analytically validated by demonstrating the existence and uniqueness of the solution and the stability of the disease-free equilibrium point is analyzed through the computation of the basic reproduction number, R_0. Numerical simulations are conducted to observe malaria infection trends, highlighting the implications of the model parameters on infection rates. This refined model provides an enhanced framework for understanding malaria transmission and potential interventions, contributing valuable insights for policymakers and public health professionals.

1.Introduction

Malaria is a vector-borne infectious disease primarily transmitted to humans by the bite of infected female *Anopheles* mosquitoes (Zongo [17]). The disease, caused by *Plasmodium* parasites, is prevalent in tropical and subtropical regions, posing a major public health challenge globally, particularly in Sub-Saharan Africa, where a large proportion of cases occur (Chiyaka et al. [3]). Mathematical modeling is pivotal in understanding malaria transmission dynamics, offering insights into the interactions between the parasite, vector, and host (Dan and Zhiongyi [5]). Such models allow researchers to simulate various scenarios, predict outcomes and evaluate the potential impact of control strategies under different environmental and socio-economic conditions (Ndamuzi and Gahungu [10]; Bakare and Nwozo [2]).
Malaria, a severe illness caused by the Plasmodium parasite and transmitted by infected female Anopheles mosquitoes, poses a significant health challenge globally, especially in Sub-Saharan Africa (Ibezim and Odo [6]). Among the four species that infect humans Plasmodium vivax, Plasmodium falciparum, Plasmodium malariae and Plasmodium ovale-Plasmodium falciparum is the deadliest and is predominantly found across the African continent, while Plasmodium vivax is more common in non-Sub-Saharan African countries (WHO, [16]). Recent reports indicate that the burden of malaria is most severe among the Sub-Saharan African population, exacerbated by low socio-economic status, limited educational opportunities and conducive climatic conditions that promote the survival and reproduction of malaria vectors (Chiyaka et al. [3]; Tumwiine et al. [14]; Li [9]; Labadin et al.[8]; Faniran et al.[6]).
Mathematical models have emerged as crucial tools for studying malaria transmission dynamics, surpassing traditional statistical models due to their predictive capabilities (Singh et al. [13]). These models can be constructed using experimental data and biological insights, allowing for various epidemiological incidence rates to be represented, including bilinear, standard and saturated incidence, depending on the population size and specific parameters (Van den Driessche and Watmough [15]; Zongo [17]). Research by Chiyaka et al. [3] emphasizes the importance of sensitivity analysis in determining key parameters influencing malaria transmission, thereby facilitating targeted intervention strategies. However, such reliance on sensitivity analysis may limit the models' ability to capture the full complexity of malaria transmission across diverse ecological and social contexts.
Various studies have explored different aspects of malaria modeling, revealing both strengths and limitations in their approaches. For instance, Ngwa and Shu [11] present a model incorporating variable human and mosquito populations, reflecting more realistic transmission dynamics.

2 Model Formulation

In this section, a mathematical modeling of malaria transmission dynamics of malaria is formulated. The formulation of the new model from the existing model, variables and parameters, assumptions, description, schematic diagram, and the model equations, among others.

2.1 The Existing Model

The study Mathematical Modeling of Malaria Transmission Dynamics: Case of Burundi by Ndamuzi and Gahungu [10] employs a deterministic SEIR compartmental model to analyze malaria transmission dynamics in Burundi, considering interactions between human and mosquito populations.

2.2 Schematic diagram of Existing Model

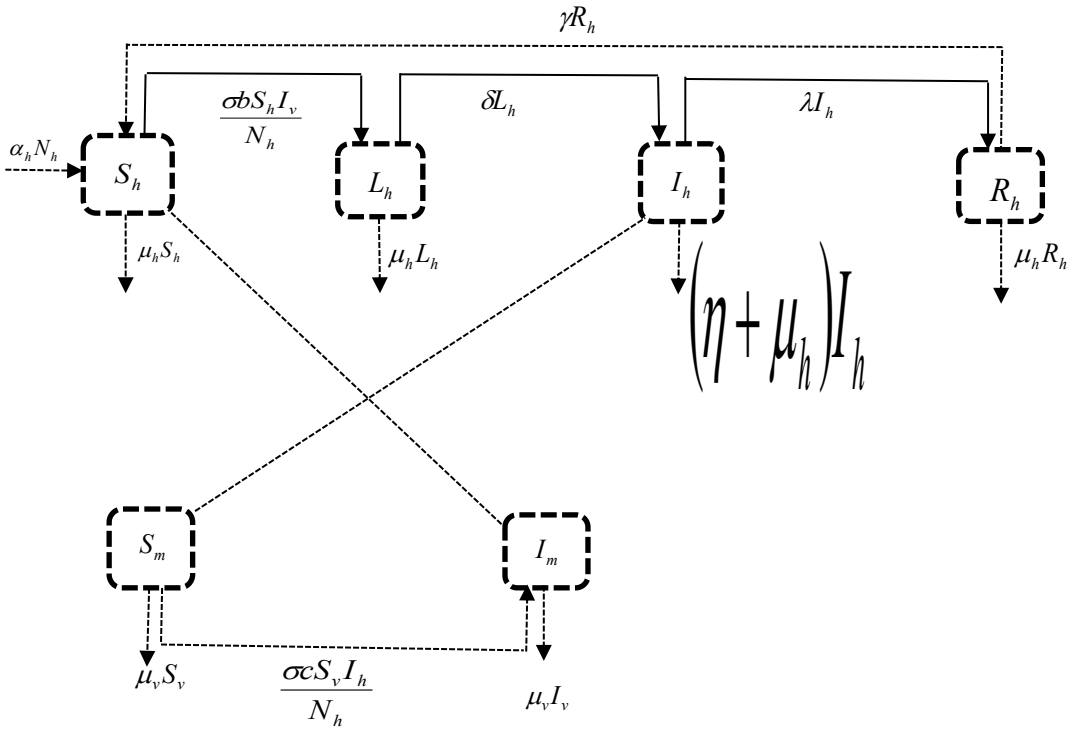

Figure 2.1: Flow diagram of the existing model for the transmission of dynamics of malaria. Source: Ndamuzi and Gahungu [10].

2.3 The Existing Model Equations

The following equation is derived from figure 2.1.

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \alpha_h N_h + \gamma R_h - \frac{\sigma b S_h I_v}{N_h} - \mu_h S_h \\
\frac{dL_h}{dt} &= \frac{\sigma b S_h I_h}{N_h} - (\mu_h + \delta) L_h \\
\frac{dI_h}{dt} &= \delta L_h - (\eta + \mu_h + \lambda) I_h \\
\frac{dR_h}{dt} &= \delta I_h - (\mu_h + \lambda) R_h \\
\frac{dS_v}{dt} &= \alpha_v N_v - \frac{\sigma c S_v I_h}{N_h} - \mu_v S_v \\
\frac{dI_v}{dt} &= \frac{\sigma c S_v I_h}{N_v} - (\mu + \delta)\mu_v I_v
\end{aligned}\right\} \qquad (2.1)$$

3 Modified Model

In this section, a modified mathematical model for malaria transmission dynamics is developed, incorporating assumptions and corresponding equations to quantitatively describe disease dynamics among humans and mosquitoes.

In developing the modified mathematical model for the transmission dynamics of malaria, several key assumptions are not were made. First, it was assumed that all newborns in both human and mosquito populations are initially susceptible to the disease. In the human population, all compartments have a natural death rate, except for the infected humans, who also face an additional death rate due to the disease. Both susceptible and infected mosquito populations experience the same natural mortality rate. The populations of humans and mosquitoes are not constant, indicating that birth and death rates are considered. Recovered humans can still transmit malaria, albeit at a reduced rate, and humans who recover without gaining immunity can revert to the susceptible state.

3.1 Variables and Parameters of the Modified Model

Table 3.1: Variables of the Modified Model

Variables	Meaning
S_h	Susceptible human
E_h	Exposed human
I_h	Infected human
R_h	Recovered human
R_{hA}	Recovered human without immunity
R_{hw}	Recovered human with immunity
S_m	Susceptible mosquito
I_m	Infected mosquito

Table 3.2: Parameters of the Modified Model

Parameters	Meaning
α_h	birth rate for humans
α_v	birth rate for mosquitoes
σ	number of bites of a mosquito to a human per unit of time
b	the probability of being infected for a human bitten by an infectious mosquito per unit of time
c	the probability of being infected for a mosquito biting an infectious human per unit of time
δ	rate of progression from latent to infectious state for humans
λ	rate of progression from infectious to recovered state for humans
γ	rate of progression from recovered to susceptible state for humans
μ_h	human (natural) mortality rate
η	human malaria-induced death rate
μ_v	mosquito mortality rate

ξ	Number of people who recovered without immunity
$1-\xi$	Preposition of those who recovered with immunity

3.2 Assumptions for the Modified Model

The following assumptions were made for the modified model:

i. All newborns are susceptible to the disease in these two populations.
ii. All the compartments in human have natural death rate μ_h except the infections human which has additional death rate denoted η caused by the disease
iii. Both compartments of mosquitoes die at the same natural rate μ_v
iv. The population of both human and mosquito are not constant not constant.
v. Recovered humans are still able to transmit the disease but at a lower rate,
vi. Human recover without immunity can move back to susceptible compartment

3.4 Schematic Diagram of the Modified Model

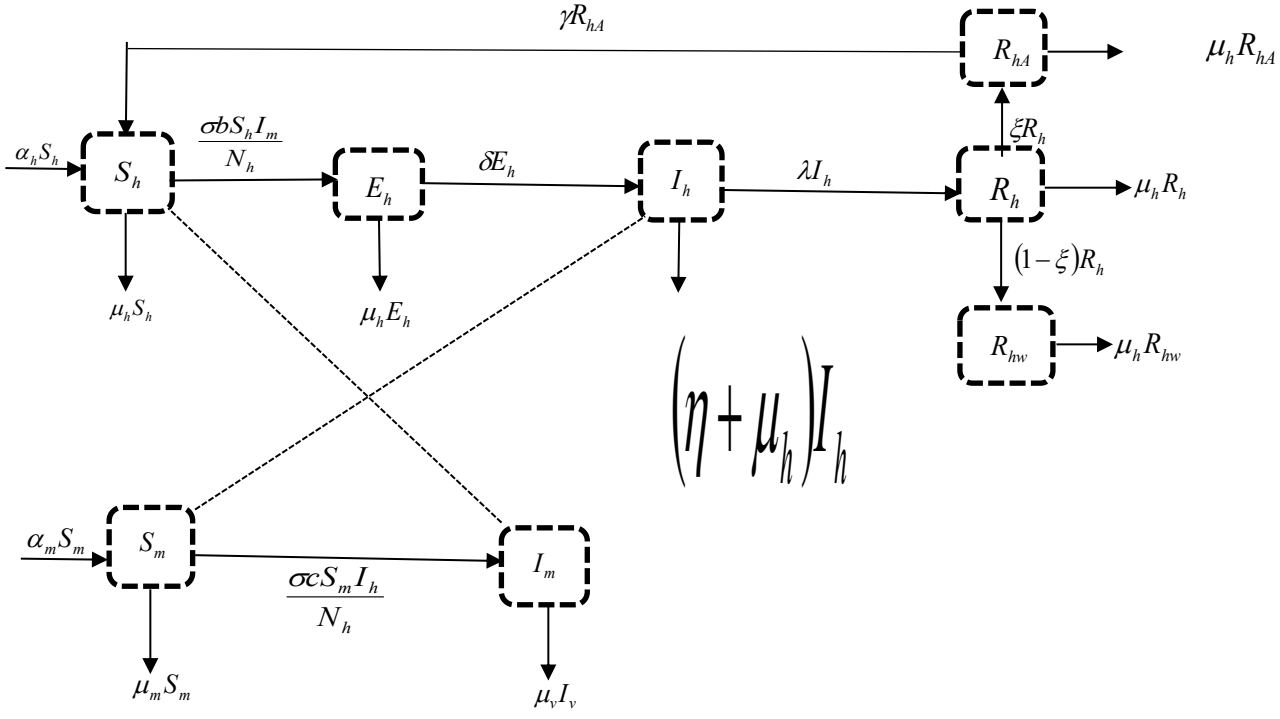

Figure 3.2: Flow diagram of modified model for the transmission of dynamics of malaria.

3.5 The Modified Model Equation

The following equations were derived from Figure 3.2.

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \alpha_h S_h + \gamma R_{hA} - \frac{\sigma b S_h I_m}{N_h} - \mu_h S_h \\
\frac{dE_h}{dt} &= \frac{\sigma b S_h I_m}{N_h} - (\mu_h + \delta) E_h \\
\frac{dI_h}{dt} &= \delta E_h - (\mu_h + \eta + \lambda) I_h \\
\frac{dR_h}{dt} &= \lambda I_h - (\mu_h + 1) R_h \\
\frac{dR_{hA}}{dt} &= \xi R_h - (\mu_h + \gamma) R_{hA} \\
\frac{dR_{hw}}{dt} &= (1 - \xi) R_h - \mu_h R_{hA} \\
\frac{dS_m}{dt} &= \alpha_m S_m - (\mu_m + \sigma c) S_m \\
\frac{dI_m}{dt} &= \frac{\sigma c S_m I_h}{N_h} - \mu_m I_m
\end{aligned}\right\} \qquad (3.2)$$

Model Analysis

In this section, we first discuss the analytical findings of our Modified model (3.2), demonstrating the existence and uniqueness of the solution. We then analyze the stability of the disease-free equilibrium point and determine the reproduction number.

4.1 Existence and Uniqueness

To demonstrate the uniqueness of the model solution, the following representations were made:

Let

$$y_1(t) = S_h(t),\ y_2(t) = E_h(t),\ y_3(t) = I_h(t),\ y_4(t) = R_h(t),\ y_5(t) = R_{hA}(t),\ y_6(t) = R_{hw}(t),\ y_7(t) = S_m(t),\ y_8(t) = I_m(t)$$

so that the model equation given by equation (3.2) can be re-written in a complex form as

$$\frac{dy}{dt} = f(t, y_1, y_2, y_3, y_4, y_5, y_6, y_7, y_8),\ y_1(t_0) = y_{10},\ y_2(t_0) = y_{20},\ y_3(t_0) = y_{30},\ y_4(t_0) = y_{40},\ y_5(t_0) = y_{50},\ y_6(t_0) = y_{60},\ y_7(t_0) = y_{70},\ y_8(t_0) = y_{80} \qquad (4.1)$$

Theorem 4.1

Suppose that the function $f(t, y_1, y_2, y_3, y_4, y_5, y_6, y_7, y_8)$ in the model equation given by system (4.1) satisfies Lipchitz condition in the region $D = \{(t, y): 0 \le |t - t_0| \le \|y - y_0\| \le b\}$ for some $a > 0, b > 0, a, b \in D$, then, there exist a natural constant number $\delta_1 > 0$, such that a unique continuous vector solution $y(t)$ of the model equation given by equation (4.1) exists in the interval $|t - t_0| < \delta_1$.

Proof

From the modified model equation (3.2), let

$$\left.\begin{aligned}
f_1(t\ y_1) &= \frac{dS_h}{dt} = \alpha_h S_h + \gamma R_{hA} - \frac{\sigma b S_h I_m}{N_h} - \mu_h S_h \\
f_2(t\ y_2) &= \frac{dE_h}{dt} = \frac{\sigma b S_h I_m}{N_h} - (\mu_h + \delta) E_h \\
f_3(t\ y_3) &= \frac{dI_h}{dt} = \delta E_h - (\mu_h + \eta + \lambda) I_h \\
f_4(t\ y_4) &= \frac{dR_h}{dt} = \lambda I_h - (\mu_h + 1) R_h \\
f_5(t\ y_5) &= \frac{dR_{hA}}{dt} = \xi R_h - (\mu_h + \gamma) R_{hA} \\
f_6(t\ y_6) &= \frac{dR_{hw}}{dt} = (1-\xi) R_h - \mu_h R_{hA} \\
f_7(t\ y_7) &= \frac{dS_m}{dt} = \alpha_m S_m - (\mu_m + \sigma c) S_m \\
f_8(t\ y_8) &= \frac{dI_m}{dt} = \frac{\sigma c S_m I_h}{N_h} - \mu_m I_m
\end{aligned}\right\} \qquad (4.2)$$

Lemma 3.1

If $f(t, y)$ has continuous partial derivative $\frac{\partial f_i}{\partial y_i}$ for $i = 1, 2, \cdots, n$ on a bounded convex domain R, then it satisfies a Lipchitz condition in R

$\|f(t, y) - f(t, y_{n-1})\| \le k\|y_n - y_{n-1}\|, i = 1, 2, 3, \cdots$

Proof

For the functions given by the equation (3.2) to satisfy Lipchitz condition.

To show that $\frac{\partial f_i}{\partial y_j}, i, j = 1, 2, 3, \cdots, 8$ are continuous and bounded in the region D.

We consider the partial derivatives of equation (4.2) are:

$$\begin{aligned}
&\frac{\partial f_1}{\partial S_h} = \alpha_h - \frac{\sigma b I_m}{N_h} - \mu_h < \infty, \frac{\partial f_1}{\partial E_h} = 0 < \infty, \ \frac{\partial f_1}{\partial I_h} = 0 < \infty, \ \frac{\partial f_1}{\partial R_h} = 0 < \infty, \ \frac{\partial f_1}{\partial R_{hA}} = \gamma < \infty, \\
&\frac{\partial f_1}{\partial R_{h\omega}} = 0 < \infty, \frac{\partial f_1}{\partial S_m} = 0 < \infty, \ \frac{\partial f_1}{\partial I_m} = -\frac{\sigma b S_h}{N_h} < \infty
\end{aligned} \qquad (4.3)$$

Also, taking the partial derivatives of the second component of equation (4.2) we obtained

$$\begin{aligned}
&\frac{\partial f_2}{\partial S_h} = \frac{\sigma b I_m}{N_h} < \infty, \ \frac{\partial f_2}{\partial E_h} = -(\mu_h + \delta) < \infty, \ \frac{\partial f_2}{\partial I_h} = 0 < \infty, \ \frac{\partial f_2}{\partial R_h} = 0 < \infty, \ \frac{\partial f_2}{\partial R_{hA}} = 0 < \infty, \\
&\frac{\partial f_2}{\partial R_{h\omega}} = 0 < \infty, \ \frac{\partial f_2}{\partial S_m} = 0 < \infty, \ \frac{\partial f_2}{\partial I_m} = \frac{\sigma b S_h}{N_h} < \infty
\end{aligned} \qquad (4.4)$$

Consider the partial derivatives of the third component of equation (4.2) we get

$$\frac{\partial f_3}{\partial S_h}=0<\infty,\ \frac{\partial f_3}{\partial E_h}=\delta<\infty,\ \frac{\partial f_3}{\partial I_h}=-(\mu_h+\eta+\lambda)<\infty,\ \frac{\partial f_3}{\partial R_h}=0<\infty,\ \frac{\partial f_3}{\partial R_{hA}}=0<\infty,$$
$$\frac{\partial f_3}{\partial R_{h\omega}}=0<\infty,\ \frac{\partial f_3}{\partial S_m}=0<\infty,\ \frac{\partial f_3}{\partial I_m}=0<\infty \quad (4.5)$$

The partial derivatives of the fourth component of equation (4.2) are as follows

$$\frac{\partial f_4}{\partial S_h}=0<\infty,\ \frac{\partial f_4}{\partial E_h}=0<\infty,\ \frac{\partial f_4}{\partial I_h}=\lambda<\infty,\ \frac{\partial f_4}{\partial R_h}=-(\mu+1)<\infty,\ \frac{\partial f_4}{\partial R_{hA}}=0<\infty,$$
$$\frac{\partial f_4}{\partial R_{h\omega}}=0<\infty,\ \frac{\partial f_4}{\partial S_m}=0<\infty,\ \frac{\partial f_4}{\partial I_m}=0<\infty \quad (4.6)$$

The partial derivatives of the fifth component of equation (4.2), we obtained

$$\frac{\partial f_5}{\partial S_h}=0<\infty,\ \frac{\partial f_5}{\partial E_h}=0<\infty,\ \frac{\partial f_5}{\partial I_h}=0<\infty,\ \frac{\partial f_5}{\partial R_h}=\xi<\infty,\ \frac{\partial f_5}{\partial R_{hA}}=-(\mu_h+\gamma)<\infty,$$
$$\frac{\partial f_5}{\partial R_{h\omega}}=0<\infty,\ \frac{\partial f_5}{\partial S_m}=0<\infty,\ \frac{\partial f_5}{\partial I_m}=0<\infty \quad (4.7)$$

The partial derivatives of the sixth component of equation (4.2), we obtained

$$\frac{\partial f_6}{\partial S_h}=0<\infty,\ \frac{\partial f_6}{\partial E_h}=0<\infty,\ \frac{\partial f_6}{\partial I_h}=0<\infty,\ \frac{\partial f_6}{\partial R_h}=(1-\xi)<\infty,\ \frac{\partial f_6}{\partial R_{hA}}=\gamma<\infty,$$
$$\frac{\partial f_6}{\partial R_{h\omega}}=0<\infty,\ \frac{\partial f_6}{\partial S_m}=0<\infty,\ \frac{\partial f_6}{\partial I_m}=-\frac{\sigma b S_h}{N_h}<\infty \quad (4.8)$$

The partial derivatives of the seventh component of equation (4.2), we obtained

$$\frac{\partial f_7}{\partial S_h}=0<\infty,\ \frac{\partial f_7}{\partial E_h}=0<\infty,\ \frac{\partial f_7}{\partial I_h}=0<\infty,\ \frac{\partial f_7}{\partial R_h}=0<\infty,\ \frac{\partial f_7}{\partial R_{hA}}=0<\infty,$$
$$\frac{\partial f_7}{\partial R_{h\omega}}=0<\infty,\ \frac{\partial f_7}{\partial S_m}=\alpha_m-(\mu_m+\sigma c)<\infty,\ \frac{\partial f_7}{\partial I_m}=0<\infty \quad (4.9)$$

The partial derivatives of the eighth component of equation (4.2), we obtained

$$\frac{\partial f_8}{\partial S_h}=\frac{\sigma c I_h}{N_h}<\infty,\ \frac{\partial f_8}{\partial E_h}=0<\infty,\ \frac{\partial f_8}{\partial I_h}=0<\infty,\ \frac{\partial f_8}{\partial R_h}=0<\infty,\ \frac{\partial f_8}{\partial R_{hA}}=0<\infty,$$
$$\frac{\partial f_8}{\partial R_{h\omega}}=0<\infty,\ \frac{\partial f_8}{\partial S_m}=0<\infty,\ \frac{\partial f_8}{\partial I_m}=-\mu<\infty \quad (4.10)$$

From equations (4.3) to (4.10), the partial derivatives of the model equations are continuous and bounded in the interval, $0<D<\infty$ by Lemma 3.1. The functions given by equation (3.2) satisfy the Lipchitz condition and hence, there exists a unique solution to the model equation (3.2) in the region *D*.

4.2 Positivity of Solution

To determine the positivity of the solution for the model, we need to ensure that the populations (susceptible, exposed, infected, and recovered individuals in both human and mosquito populations) remain non-negative for all time $t\geq 0$. This is crucial since negative populations are not physically meaningful. This will be achieved using the following theorem.

Theorem 4.2. For a nonnegative initial conditions, the model equation given in equation (3.2) "the solution" will remain positive for all time $(S_h, E_h, I_h, R_h, R_{hA}, R_{hw}, S_m, I_m)$ of the equation are non-negative for all time $t \geq 0$.

Proof:

For the model equations (3.2), let t be the maximum time for the epidemics. This implies that $t = \sup\{t > 0 : (S_h > E_h > I_h > R_h > R_{hA} > R_{hw} > S_m > I_m)\}$ that is $t^* \geq 0$. From the first equation of (3.2), we have

$$\frac{dS_h}{dt} = \alpha_h S_h + \gamma R_{hA} - \frac{\sigma b S_h I_m}{N_h} - \mu_h S_h \geq \frac{\sigma b I_m}{N_h} - \mu_h \tag{4.11}$$

For second equation of (3.2) we have

$$\frac{dE_h}{dt} = \frac{\sigma b S_h I_m}{N_h} - (\mu_h + \delta)E_h \geq -(\mu_h + \delta) \tag{4.12}$$

Similarly, for third equation of (3.2) we have

$$\frac{dI_h}{dt} = \delta E_h - (\mu_h + \eta + \lambda)I_h \geq -(\mu_h + \eta + \lambda) \tag{4.13}$$

From the fourth equation of (3.2) we have

$$\frac{dR_h}{dt} = \lambda I_h - (\mu_h + 1)R_h \geq -(\mu + 1) \tag{4.14}$$

Form For fifth equation of (3.2) we have

$$\frac{dR_{hA}}{dt} = \xi R_h - (\mu_h + \gamma)R_{hA} \geq -(\mu_h + \gamma) \tag{4.15}$$

Form For sixth equation of (3.2) we have

$$\frac{dR_{hw}}{dt} = (1 - \xi)R_h - \mu_h R_{hA} \geq -\mu_h \tag{4.16}$$

Form For seventh equation of (3.2) we have

$$\frac{dS_m}{dt} = \alpha_m S_m - (\mu_m + \sigma c)S_m \geq (\mu_m + \sigma c) \tag{4.17}$$

Form For eight equation of (3.2) we have

$$\frac{dI_m}{dt} = \frac{\sigma c S_m I_h}{N_h} - \mu_m I_m \geq -\mu_m \quad . \tag{4.18}$$

4.4 Basic Reproduction Number (R_0)

The basic reproduction number is defined as the average number of the secondary infections cases generated by a typical infected person in an otherwise disease free population. The basic reproduction number (R_0) of the system (3.2) "is computed using the next generation matrix method". Here the next generation matrix F_i denotes the rate of appearance of new infections and V_i represents the transfer of infection into and out of any compartment respectively (Andest & Daniel, 2025[1]).

To find the basic reproduction number, (R_0), "for equation (3.2)" we use the next-generation matrix approach, which involves linearizing the model around the Disease-Free Equilibrium (DFE).

The infected compartments in the model are I_h (infected humans), E_h (exposed humans) and I_m (infected mosquitoes).

Consider only the terms related to the infected compartments I_h, E_h and I_m.

The relevant equations are:

$$\left.\begin{aligned}\frac{dE_h}{dt} &= \frac{\sigma_m b S_h I_m}{N_h} - (\mu_h + \delta)E_h \\ \frac{dI_h}{dt} &= \delta E_h - (\mu_h + \eta + \lambda)I_h \\ \frac{dI_m}{dt} &= \frac{\sigma c S_m I_h}{N_h} - \mu_m I_m .\end{aligned}\right\} \tag{4.20}$$

At the DFE, the susceptible populations S_h and S_m are at their equilibrium values

$S_h = \frac{\alpha_h}{\mu_h}$ and $S_m = \frac{\alpha_m}{\mu_m}$.

The infected Matrix (F)

$$F = \begin{pmatrix} 0 & 0 & \frac{\sigma b S_h}{N_h} \\ 0 & 0 & 0 \\ 0 & \frac{\sigma_m S_m}{N_h} & 0 \end{pmatrix}.$$

Transition Matrix (V)

$$V = \begin{pmatrix} \mu_h + \delta & 0 & 0 \\ -\delta & \mu_h + \eta + \lambda & 0 \\ 0 & 0 & \mu_m \end{pmatrix}$$

The next-generation matrix G of the F (new infection terms) and V (transition terms) is given by:

$G = F \cdot V^{-1}$

To calculate G we first need to find the inverse of V.

The inverse of V is:

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu_h + \delta} & 0 & 0 \\ \frac{\delta}{(\mu_h + \delta)(\mu_h + \eta + \lambda)} & \frac{1}{\mu_h + \eta + \lambda} & 0 \\ 0 & 0 & \frac{1}{\mu_m} \end{pmatrix}$$

Then the next-generation matrix G is:

$$G=\begin{pmatrix} 0 & 0 & \frac{\sigma b S_h}{N_h} \\ 0 & 0 & 0 \\ 0 & \frac{\sigma_m S_m}{N_h} & 0 \end{pmatrix} \cdot \begin{pmatrix} \frac{1}{\mu_h+\delta} & 0 & 0 \\ \frac{\delta}{(\mu_h+\delta)(\mu_h+\eta+\lambda)} & \frac{1}{\mu_h+\eta+\lambda} & 0 \\ 0 & 0 & \frac{1}{\mu_m} \end{pmatrix}$$

After performing the matrix multiplication:

$$G=\begin{pmatrix} 0 & 0 & \frac{\sigma b S_h}{\mu_m N_h} \\ 0 & 0 & 0 \\ 0 & \frac{\sigma_m S_m}{(\mu_h+\eta+\lambda)N_h} & 0 \end{pmatrix}$$

The basic reproduction number R_0 is the spectral radius (dominant eigenvalue) of the next-generation matrix G.

From the structure of G, the eigenvalues are the entries:

$$R_0=\sqrt{\frac{\sigma b S_h \cdot \sigma_m S_m}{\mu_m(\mu_h+\eta+\lambda)N_h^2}}$$

4.5 Local Stability of the DFE($\mathbf{x_0}$)

To determine the local stability of the Disease-Free Equilibrium (DFE) using the model equations (3.2), with reference to following theorem.

4.6 Disease Free Equilibrium (DEF) of the Model

To determine the Disease-Free Equilibrium (DFE) for the model using the Next Generation Matrix (NGM) approach, the theorem 4.3 was considered as stated below:

Theorem 4.3

According to Andest et al. [1], a disease-free equilibrium state of the model (3.2) exists where $S_h=E_h=I_h=R_h=R_{hA}=R_{hw}=S_m=I_m=0$ point and is locally asymptotically stable if $R_0<1$, and unstable if $R_0>1$. Therefore, the disease free-equilibrium (DFE)

$$E_0=\left(\frac{\alpha_h N_h}{\mu_h},0,0,0,0,0,\frac{\alpha_m N_m}{\mu_h},0\right) \tag{3.19}$$

Proof

Here, we take the partial derivative of (3.2) with respect to $S_h, E_h, I_h, R_h, R_{hA}, R_{hw}, S_m, I_m$ at the disease-free equilibrium which gives us:

First, we compute the Jacobian matrix J of the system at the DFE. The Jacobian matrix J is the matrix of first-order partial derivatives of the right-hand side of the system with respect to the variables in equation (3.2).

The DFE occurs when there are no infections in the population, meaning $E_h=I_h=R_h=R_{hw}=I_m=0$.

At the DFE, the susceptible populations S_h and S_m are at their equilibrium values:

$$S_h = \frac{\alpha_h}{\mu_h}, S_m = \frac{\alpha_m}{\mu_m}$$

The Jacobian matrix J is computed by taking the partial derivatives of each equation with respect to the state variables

$S_h, E_h, I_h, R_h, R_{hA}, R_{hw}, S_m$ and I_m

The Jacobian matrix E_0 at the DFE simplifies to:

$$J(E_0) = \begin{pmatrix} \alpha_h - \frac{\sigma b I_m}{N_h} - \mu_h & 0 & 0 & 0 & \gamma & 0 & 0 & -\frac{\sigma b S_h}{N_h} \\ -\frac{\sigma b I_m}{N_h} & -(\mu_h + \delta) & 0 & 0 & 0 & 0 & 0 & \frac{\sigma b S_h}{N_h} \\ 0 & \delta & -(\mu_h + \eta + \lambda) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda & -(\mu + 1) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi & -(\mu_h + \gamma) & 0 & 0 & 0 \\ 0 & 0 & 0 & (1-\xi) & \gamma & -\mu_h & 0 & -\frac{\sigma b S_h}{N_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m - (\mu_m + \sigma c) & 0 \\ \frac{\sigma c I_h}{N_h} & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m \end{pmatrix} \quad (4.21)$$

Consider $P(\lambda)$ the characteristic polynomial of $J(E_0)$, we then have:

$$P(\lambda) = |J(E_0) - \lambda I_8| = \begin{pmatrix} \alpha_h - \frac{\sigma b I_m}{N_h} - \mu_h - \lambda & 0 & 0 & 0 & \gamma & 0 & 0 & -\frac{\sigma b S_h}{N_h} \\ -\frac{\sigma b I_m}{N_h} & -(\mu_h + \delta + \lambda) & 0 & 0 & 0 & 0 & 0 & \frac{\sigma b S_h}{N_h} \\ 0 & \delta & -(\mu_h + \eta + 2\lambda) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda & -(\mu + 1 + \lambda) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi & -(\mu_h + \gamma + \lambda) & 0 & 0 & 0 \\ 0 & 0 & 0 & (1-\xi) & \gamma & -\mu_h & 0 & -\frac{\sigma b S_h}{N_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m - (\mu_m + \sigma c + \lambda) & 0 \\ \frac{\sigma c I_h}{N_h} & 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_m + \lambda) \end{pmatrix}$$

5 Numerical Simulation

In this section, we will analyze the model equation (3.2) numerically. In particular, we focus on the numerical analysis of the infected human population with malaria. To do this, we collect a reasonable set of data for the parameters of the model. Most of these are obtained from models developed in the literature and other parameters will be assumed.

Table 4.1: Parameters for the Modified Model

Parameters	Values	References
α_h	38.377	Ndamuzi and Gahungu [10]
α_m	130	Zongo [17]
σ	20.425	Assumed
b	0.0181	Ministry of Public Health and the Fight against HIV/AIDS (2018)

c	0.11	Olaniyi et al. [12]
δ	0.321	Assumed
λ	0.0022	Chitnis et al. [4]
γ	0.00055	Chitnis et al. [4]
μ_h	7.766	Ndamuzi and Gahungu [10]
η	0.001	Assumed
μ_m	0.003	Zongo [17]

The data of confirmed cases, are obtained from the Ministry of Public Health and the Fight against HIV/AIDS for the period 2012-2018. As given in the literature, we initialize parameters and use minimize function with noise in Python for obtaining parameter values corresponding to the data under study. Initial of the state are

Table 4.2: Variables for the Existing Model

Variables	Values	Ndamuzi and Gahungu [10]
N_h	11890781	Zongo [17]
N_v	1000	Assumed
S_h	498901	Chitnis et al [4]
L_h	374176	Chitnis et al. [4]
I_h	823	Assumed
R_h	560	Chitnis et al. [4]
S_v	3582	Chitnis et al. [4]
I_v	66	Ndamuzi and Gahungu [10]

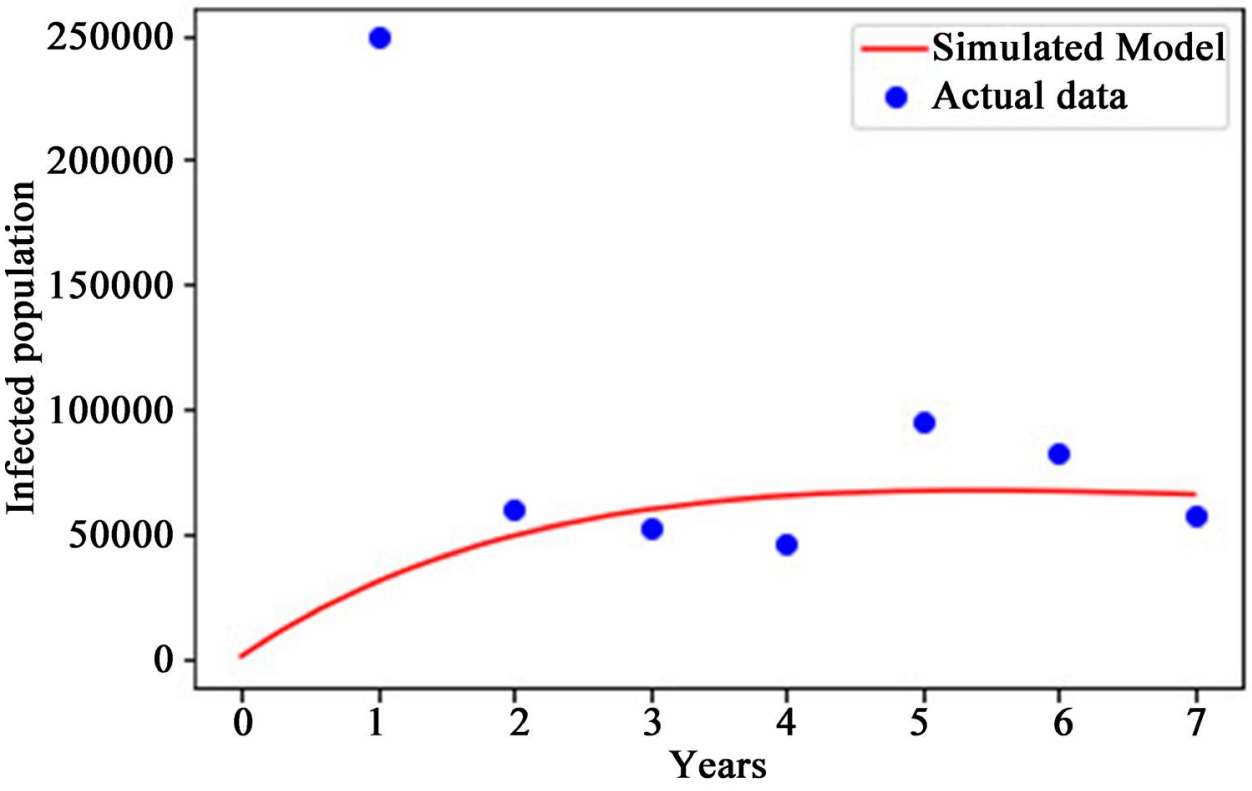

Figure 4.1: Graphical illustration of infected population.

6 Discussion of Results

The disease-free equilibrium occurs when there are no infected individuals in the population, meaning that all compartments representing the infected or exposed individuals are zero. At the DFE, only the susceptible populations S_h^* and S_m^* have positive values, reflecting the scenario where no disease is present, and the populations are at equilibrium. The DFE is critical as it represents a baseline state where the disease could potentially be eradicated if no new infections are introduced. Mathematically, this equilibrium is found by setting $E_h = I_h = R_h = R_{hw} = I_m$ and solving for S_h and S_m. The resulting DFE values provide insight into the conditions under which the population can remain disease-free, serving as a reference point for stability analysis.

The basic reproduction number, R_0, is a key epidemiological metric that indicates the average number of secondary infections produced by a single infected individual in a completely susceptible population. It serves as a threshold parameter: if $R_0 < 1$, the disease is expected to die out over time, leading to the stability of the DFE. Conversely, if $R_0 > 1$, the disease can spread and potentially cause an epidemic, rendering the DFE unstable. R_0 is derived from the model equations by analyzing the infection terms and transmission dynamics between susceptible and infected populations. It encapsulates factors such as transmission rates, recovery rates, and the contact patterns within the population, making it a fundamental determinant of the disease's potential to spread.

The local stability of the DFE is assessed by analyzing the eigenvalues of the Jacobian matrix, which is derived from the model equations at the DFE. If all eigenvalues have negative real parts, the DFE is

locally asymptotically stable, meaning that small perturbations in the population (such as the introduction of a few infected individuals) will not lead to an outbreak, and the system will return to the DFE over time. This stability is directly related to the basic reproduction number R_0. When $R_0 < 1$, the eigenvalues of the Jacobian indicate that the DFE is stable, confirming that the disease cannot sustain itself in the population. However, if $R_0 > 1$, at least one eigenvalue will have a positive real part, leading to the instability of the DFE and the potential for disease spread. Thus, the stability analysis provides a critical understanding of the conditions under which the population remains disease-free or experiences an outbreak.

Figure 4.1 illustrates the dynamics infected population over time, with the y-axis showing the number of infected individuals and the x-axis representing time. This curve reveals critical insights into disease progression, showing an initial slow increase in cases as the infection begins to spread, followed by a steep ascent during the exponential growth phase, where transmission rates are high. The peak indicates the point of maximum infection and healthcare strain. Subsequently, the curve declines, reflecting a reduction in new infections due to factors like the depletion of susceptible individuals, public health interventions, or the disease's natural course. The tail of the curve showing a gradual drop in cases, suggests effective disease control, potentially leading to eradication. The curve's shape and progression offer valuable information on the impact and timing of interventions and the likelihood of future outbreaks.

7 Summary and Conclusion

This research developed an epidemiological model to study disease transmission dynamics in a population by analyzing compartments such as susceptible, infected, and recovered individuals, ensuring the model's biological realism through positivity of solutions. The Disease-Free Equilibrium (DFE) is identified, representing a state where the disease is entirely absent. The basic reproduction number (R_0) was derived as a critical threshold, predicting whether the disease will spread or naturally dissipate. Stability analysis of the DFE indicated that when $R_0 < 1$, the DFE remains locally stable, suggesting the disease cannot sustain itself within the population.

The findings underscore the importance of keeping R_0 below 1 to maintain a disease-free state, making it a crucial target for public health interventions. The study emphasizes the DFE's significance in guiding control measures, offering a framework for effective outbreak management. By ensuring the model's positivity and applicability to real scenarios, this research demonstrates the potential for disease eradication through strategic interventions, providing key insights into controlling infectious diseases and predicting their spread under specific conditions.

8 Conflict of interest statement

This statement serves to declare that the authors have no conflicts of interest regarding the work submitted for publication consideration in your journal, *Al-Qa*disiyah of Pure Science

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