International Journal of
Mathematical Modelling & Computations
Vol. 12, No. 02, Spring 2022, 115- 130

Stability Analysis of a Malaria Transmission Model for the Effect of Infected Immigrants with Temperature and Rainfall Dependent Parameters

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Abstract. A human host-mosquito vector model for transmission of malaria with inflow of infected immigrants is formulated. The mosquito population includes aquatic stages (eggs, larvae, and pupae) and mature stages which have highly temperature and rainfall dependent life cycles. Model analysis reveals that the model only attains two (2) endemic equilibria; one in absence of the vector population and the other in presence of the vector population. The endemic equilibrium without the mosquito vector population is unstable. The endemic equilibrium with the vector population is locally stable and globally unstable. Numerical simulations of the model reveal that the proportion of infected humans introduced into the community does not significantly change the pattern of malaria transmission.

Received: 27 July 2021, Revised: 04 April 2022, Accepted: 24 April 2022.

Keywords: Equilibrium points; Local stability; Global stability; Numerical simulation.

AMS Subject Classification: 49K15, 65L07, 93C15.

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1. Introduction

Malaria remains a major public health problem globally, it is one of the leading causes of morbidity and mortality. According to the World Health Organisation (WHO) [42], an estimated 219 million cases of malaria occurred in 2017 worldwide with about 436000 deaths globally. Sub-Saharan Africa shares at least 80% and

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78% of the global malaria cases and deaths respectively [43]. Malaria was once eradicated in some parts of the world, most of which are developed countries, however, recently disease out break has been observed in previously disease free areas [34].

Human migration is believed to be the major factor leading to the re-emergence of malaria as infected travelers infect susceptible mosquitoes in disease free areas (see [11, 20, 21, 37]). Human population movements have significant impact on the transmission of vector borne diseases across spatial and temporal scales that exceed the limits of mosquito dispersal [33, 39]. Human migrations include voluntary and forced migrations such as refugees, it leads to the opening of new areas for settlement and urbanisation. Migrants most likely lack immunity against malaria, moreover acquired immunity to severe disease is life-long under conditions of repeated human exposure to biting *Anopheles* mosquitoes and might be lost with time without repeated re-exposure [13, 14, 44]. Rapid infrastructure development has enhanced human migration, most especially in developing countries which have high unprecedented population growth. Individuals mainly travel due to the search for greener economic opportunities, civil unrest and adventure. An increasing number of imported malaria cases due to migrant inflows from malaria-endemic regions, together with competent mosquito vectors species, favourable climatic conditions and changing climate underscores the risk of re-emergence of autochthonous cases in regions where malaria was previously eradicated [35]. Mobile and migrant populations, because of their higher risk to infection contribute to the spread of the drug-resistance problem from endemic areas to malaria-free regions. They have difficulties in staying in one region due to the nature of their work which requires seasonal migration [10, 11, 20, 41]. The inflow of infected individuals from higher to lower transmission areas presents a risk of initiating malaria epidemics and may also sustain malaria parasite populations in areas where they would otherwise be absent or being targeted for elimination [30, 39].

Climatic conditions such as temperature, rainfall patterns, and humidity affect the life cycle and survival of parasites and vectors. Seasonal factors influence malaria transmission because mosquito vectors and malaria parasites are sensitive to climatic conditions. The *Anopheles* mosquitoes that are responsible for transmission of the *Plasmodium* parasites often breed in aquatic habitats to complete their life cycle [3, 8]. The length of the *Anopheles* mosquito lifecycle and the sporogenic development of the *Plasmodium* parasites that cause malaria depend on temperature [12, 15, 27]. Water temperature is an important determinant of malaria transmission because it is responsible for both aquatic survival and development [4, 5, 18]. It has a major effect on the rate at which the immature stages of mosquitoes develop into adults. The temperature of the environment is one of the most important abiotic factors that affects the life of mosquitoes and also impacts on the mosquitoes' flight activity and host-seeking behaviour [31]. The increase in mosquito density and incidence of malaria cases usually occur in rainy seasons. However, immature stages of the *Anopheles* mosquitoes are frequently exposed to high frequency of extreme rainfall that may cause an increase in their mortality rate [28, 32]. Seasonally dry conditions and low ambient temperatures prevent sporogonic malaria development although abnormally warm temperatures and wet conditions favour malaria epidemic. However, in natural situation in tropical environments perennial malaria transmission occurs during hot dry seasons [25]. It is thus important to include temperature and rainfall dependence in a malaria transmission model.

Mathematical modelling and analysis is an important part of infectious disease epidemiology. Numerous authors (see [1, 16, 23, 24, 26, 27, 29, 36, 38, 40]) have developed models to quantify the impact of temperature and rainfall on malaria transmission dynamics. Agusto *et al.* [2] used a deterministic model for assessing the impact of changing temperature and temperature variability on short-term malaria transmission dynamics. A delay-differential model for immature mosquito development was developed by Beck-Johnson *et al.* [6] with development time inversely proportional to temperature. Bayoh and Lindsay [4] used non-linear models to describe the relationship between developmental rate of the aquatic stages of the *Anopheles gambiae sensu stricto* and temperature. It was revealed that adult development rate was greatest between 28°C and 32°C, while adult emergence was highest between 22°C and 26°C. However, there was no emergence of adults below 18°C or above 34°C. Makinde and Abiodun [19] formulated linear models based on stepwise regression based on some climate variables and number of susceptible individuals to malaria. They found out that an increase in daily rain amount and mean temperature significantly raises the chance of exposure to malaria while number of susceptible and exposed individuals affects transmission of malaria infection. Lunde *et al.* [17] compared six temperature dependent mortality models for the malaria vector *Anopheles gambiae sensu stricto* to examine how mosquito mortality is related to temperature. Their model suggests that transmission is most efficient at around 25°C. Most of the models with infective migrants (see [9, 21, 37]) ignore seasonal factor dependence in the mosquito vector population.

The influx of infective humans and with no seasonal factors have been considered in the following studies. Brauer and van den Driessche [9] formulated simple models for disease transmission that included immigration of infective individuals and demographic effects. It was revealed that, there is no disease free equilibrium with a fraction of infective immigrants and there is always a unique endemic equilibrium which is asymptotically stable. Tumwiine *et al.* [37] developed a susceptible-infective-recovered-susceptible (SIRS) and susceptible-infective (SI) in the human population and mosquito population, respectively for malaria transmission with influx of infective migrants. They showed that if the fraction of infective humans is sufficiently small there exists a threshold for which the disease can be eliminated. Mukandavire *et al.* [21] investigated the effect of seasonality in the transmission rates in a malaria model with infective immigrants, which revealed that the strength of seasonality increases the number of infective humans. Sunita and Nisha [34] incorporated the exposed class in a malaria transmission model with infective immigrants, they found that there are two disease free equilibrium points which exist only if there are no infective immigrants entering into the population, otherwise there is a unique endemic equilibrium.

In the previous work [9, 34, 37], they used deterministic models to analyse malaria transmission in presence of infective immigrants, however the effect of seasonal factors was ignored. Mukhtar *et al.* [22], Ngarakana - Gwasira *et al.*[24] and Yiga *et al.* [45] investigated the effect of seasonal factors in absence of infective immigrants. Temperature and rainfall dependence in the mosquito population have been ignored in infective immigrant malaria models. Therefore, this study is an extension of Yiga *et al.* [45] in which the effect of seasonal factors on malaria transmission in absence of infected immigrants was analysed. This study incorporates the inflow of a proportion of infected humans and mosquitoes' aquatic stages (eggs, larva, and pupae) that are sensitive to rainfall and temperature.

The rest of this paper is organised as follows: In Section 2 the model formulation is presented , the model is analysed for steady states and their stability in Section 3. In Section 4, numerical simulations are presented and the paper is concluded in

Section 5.

2. Model formulation

In this section, a human host- mosquito vector model with inflow of infected humans and infection through an interaction coefficient between infected and susceptible individuals is formulated. Susceptible humans become infected with interaction coefficient β_H between susceptible humans and infectious mosquitoes, similarly susceptible mosquitoes get infected with interaction coefficient β_V between susceptible mosquitoes and infected humans [34]. An SEIRS and SI model for the human and mosquito populations, respectively is formulated. The total human population N_H is divided into four compartments; susceptible S_H, exposed E_H, infected I_H and recovered R_H humans such that

$$N_H = S_H + E_H + I_H + R_H. \tag{1}$$

The total mosquito population M_T is divided into aquatic mosquitoes M_A (eggs, pupae, larvae) and adult female *Anopheles* mosquitoes N_V such that

$$M_T = M_A + N_V.$$

Adult female *Anopheles* mosquito population N_V is divided into two compartments; susceptible S_V and infectious I_V such that

$$N_V = S_V + I_V. \tag{2}$$

The populations are governed by a system of ordinary differential equations below

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + (1-p-q)A_H - \beta_H S_H I_V - \mu_H S_H + \sigma R_H,\\
\frac{dE_H}{dt} &= pA_H + \beta_H S_H I_V - (\rho + \mu_H)E_H,\\
\frac{dI_H}{dt} &= qA_H + \rho E_H - (\mu_H + \nu + \delta)I_H,\\
\frac{dR_H}{dt} &= \nu I_H - (\sigma + \mu_H)R_H,\\
\frac{dM_A}{dt} &= L(T)\left(1 - \frac{M_A}{K}\right)(S_V + I_V) - (\lambda(T,R) + \mu_A(T))M_A,\\
\frac{dS_V}{dt} &= \lambda(T,R)M_A - \beta_V S_V I_H - \mu_V(T)S_V,\\
\frac{dI_V}{dt} &= \beta_V S_V I_H - \mu_V(T)I_V,
\end{aligned} \tag{3}$$

where Λ_H is the human birth rate, A_H is the constant immigration rate of humans with a proportion of exposed p and infective q. μ_H is the natural death rate of the human population, σ is the rate of loss of immunity by humans, ρ is the progression rate of humans from exposed to the infectious class, ν is the recovery rate of infected humans and δ is the rate at which infected humans die from malaria infection. $L(T)$ is the egg deposition rate of adult mosquitoes, the population growth of mosquitoes is constrained by the carrying capacity of the environment K. $\lambda(T,R)$

is the temperature and rainfall dependent maturation rate of aquatic mosquitoes, $\mu_A(T)$ and $\mu_V(T)$ are temperature dependent death rates of the aquatic and adult mosquitoes respectively. In the above formulation it is assumed that all parameters are positive except $\delta \geqslant 0$.

3. Model analysis

3.1 *Invariant region*

The region in which the solution of the system (3) is bounded is obtained. Differentiating equation (1) gives

$$\frac{dN_H}{dt} = \Lambda_H + (1-p-q)A_H - \mu_H N_H - \delta I_H. \tag{4}$$

For the disease induced death rate $\delta \geqslant 0$ equation (4) becomes

$$\frac{dN_H}{dt} \leqslant \Lambda - \mu_H N_H, \tag{5}$$

where $\Lambda = \Lambda_H + (1-p-q)A_H$.
Solving equation (5) gives

$$N_H(t) \leqslant \frac{\Lambda}{\mu_H} + \left(N_H(0) - \frac{\Lambda}{\mu_H}\right) exp(-\mu_H t).$$

Evaluating as $t \to \infty$, shows that $N_H(t) \to \frac{\Lambda}{\mu_H}$. Therefore the total human population is bounded by $\frac{\Lambda}{\mu_H}$, and the solution is bounded in $\mathcal{D}_H = \{(S_H, E_H, I_H, R_H) \in \mathbb{R}^4_+ : 0 \leqslant N_H \leqslant \frac{\Lambda}{\mu_H}\}$.
Similarly, for the mosquito vector population differentiating equation (2) and substituting for $\frac{dS_V}{dt}$ and $\frac{dI_V}{dt}$ from system (3) gives

$$\frac{dN_V}{dt} = \lambda(T,R)M_A - \mu_V(T)N_V. \tag{6}$$

Solving equation (6) gives

$$N_V(t) = \frac{\lambda(T,R)M_A}{\mu_V(T)} - \frac{\lambda(T,R)}{\mu_V(T)} \exp(-\mu_V(T)t) \int \exp(\mu_V(T)t) \frac{dM_A}{dt} dt.$$

As $t \to \infty$, $N_V(t) \to \frac{\lambda(T,R)M_A}{\mu_V(T)}$, thus the total mosquito population is bounded by $\frac{\lambda(T,R)M_A}{\mu_V(T)}$. Therefore all the solution set of system (3) is bounded in $\mathcal{D} = \{(S_H, E_H, I_H, R_H) \in \mathbb{R}^4_+ : 0 \leqslant N_H \leqslant \frac{\Lambda}{\mu_H}, (M_A, S_V, I_V) \in \mathbb{R}^3_+ : 0 \leqslant N_V \leqslant \frac{\lambda(T,R)M_A}{\mu_V(T)}\}$. The model is epidemiological and mathematically well posed.

3.2 *Positivity of solutions*

In this subsection, it is shown that the solution of system (3) is always positive for all time, $t > 0$ if the respective initial values of the populations are positive.

Theorem 3.1 *For system* (3) *suppose* $S_H(0) > 0, E_H(0) > 0, I_H(0) > 0, R_H(0) > 0, M_A(0) > 0, S_V(0) > 0, I_V(0) > 0$, *then* $S_H(t) > 0, E_H(t) > 0, I_H(t) > 0, R_H(t) > 0, M_A(t) > 0, S_V(t) > 0$ *and* $I_V(t) > 0$ *for all* $t > 0$.

Proof Define a set $\mathcal{H} = \{t > 0 : S_H(t) > 0, E_H(t) > 0, I_H(t) > 0, R_H(t) > 0, M_A(t) > 0, S_V(t) > 0, I_V(t) > 0\}$.
It is assumed by contradiction that if the set $\mathcal{H}$ defined above is bounded, then $\mathcal{H}$ has a supremum τ. Now define τ as
$\tau = sup\{t > 0 : S_H(t) > 0, E_H(t) > 0, I_H(t) > 0, R_H(t) > 0, M_A(t) > 0, S_V(t) > 0, I_V(t) > 0, 0 \leqslant t \leqslant \tau\}$.
Since $S_H(t), E_H(t), I_H(t), R_H(t), M_A(t), S_V(t)$ and $I_V(t)$ are continuous then $\tau > 0$. If $\tau < \infty$ then necessarily $S_H(\tau) = 0$ or $E_H(\tau) = 0$ or $I_H(\tau) = 0$ or $R_H(\tau) = 0$ or $M_A(\tau) = 0$ or $S_V(\tau) = 0$ or $I_V(\tau) = 0$.
From the first equation of system (3),

$$\frac{dS_H}{dt} = a + \sigma R_H - (\beta_H I_V + \mu_H)S_H,$$

where $a = \Lambda_H + (1 - p - q)A_H$.
Let $P(t) = exp\left(\mu_H t + \int_0^t \beta_H I_V(s)ds\right)$ and note that $P(0) = 1$ and $P(t) > 0$ for all $t > 0$.

Consider,

$$\begin{aligned}
\frac{d}{dt}[S_H(t)P(t)] &= \dot{S}_H(t)P(t) + S_H(t)\dot{P}(t),\\
&= P(t)\dot{S_H}(t) + (\mu_H + \beta_H I_V(t))P(t)S_H(t),\\
&= P(t)[\dot{S}_H(t) + (\mu_H + \beta_H I_V(t))S_H(t)],\\
&= P(t)[a + \sigma R_H],\\
\int_0^\tau \frac{d}{dt}[S_H(t)P(t)]dt &= \int_0^\tau P(t)[a + \sigma R_H]dt,\\
S_H(\tau)P(\tau) - S_H(0)P(0) &= \int_0^\tau (a + \sigma R_H(t))P(t)dt,\\
S_H(\tau) &= P(\tau)^{-1}\left[S_H(0) + \int_0^\tau (a + \sigma R_H(t))P(t)dt\right].
\end{aligned}$$

Therefore, $S_H(\tau) > 0$ since all parameters are positive. Applying the above reasoning to the remaining equations shows that $E_H(\tau) > 0, I_H(\tau) > 0, R_H(\tau) > 0, M_A(\tau) > 0, S_V(\tau) > 0, I_V(\tau) > 0$ thus $\tau = \infty$. This contradicts τ being a supremum of $\mathcal{H}$, thus $\mathcal{H}$ is not bounded.
This confirms the positivity of solutions for all $t > 0$. ■

3.3 *Equilibrium points*

In this subsection, system (3) is analysed for steady states. There is no disease free equilibrium due to the inflow of infected immigrants, thus, the disease is always present in the population. At the equilibrium point, the right hand side (RHS) of equations in system (3) are set to zero to give

$$M_A\left[L(T)\left(1-\frac{M_A}{K}\right)\frac{\lambda(T,R)}{\mu_V(T)}-(\lambda(T,R)+\mu_A(T))\right]=0 \tag{7}$$

From equation (7) either $M_A = 0$ or $M_A = \frac{K[L(T)\lambda(T,R)-\mu_V(T)(\lambda(T,R)\mu_A(T))]}{L(T)\lambda(T,R)}$, the aquatic mosquito population is independent of the infected mosquito and human populations. This implies that the infected populations do not influence the size of the aquatic mosquito population.
For $M_A = 0$ it implies that $S_V = I_V = 0$ and there exists an endemic equilibrium point without both aquatic and adult mosquito population. This means malaria will be sustained in the population only because of the inflow of infected humans. $E_1(S_H, E_H, I_H, R_H, M_A, S_V, I_V) = [S_H^*, E_H^*, I_H^*, R_H^*, 0, 0, 0]$, where

$$\begin{aligned}
S_H^* &= \frac{1}{\mu_H}\left(\Lambda_H+(1-p-q)A_H+\frac{\sigma\nu[q(\rho+\mu_H)+\rho pA_H]}{(\rho+\mu_H)(\sigma+\mu_H)(\mu_H+\nu+\delta)}\right),\\
E_H^* &= \frac{pA_H}{\rho+\mu_H},\\
I_H^* &= \frac{q(\rho+\mu_H)+\rho pA_H}{(\rho+\mu_H)(\mu_H+\nu+\delta)},\\
R_H^* &= \frac{\nu[q(\rho+\mu_H)+\rho pA_H]}{(\sigma+\mu_H)(\rho+\mu_H)(\mu_H+\nu+\delta)}.
\end{aligned}$$

It is observed from the endemic equilibrium point E_1 that the population in the infected subsystem is due to inflow of infectives, that is, if the inflow of infectives is blocked such that $p = q = 0$ then the exposed, infected and recovered populations become zero which yields a disease free equilibrium. The resulting disease free equilibrium persists as long as there is no inflow of infectives allowed into the community, otherwise the disease persists.
For $M_A = \frac{K[L(T)\lambda(T,R)-\mu_V(T)(\lambda(T,R)+\mu_A(T))]}{L(T)\lambda(T,R)}$, it follows that $S_V \neq 0$ and $I_V \neq 0$ provided $\frac{L(T)\lambda(T,R)}{\mu_V(T)(\lambda(T,R)+\mu_A(T))} > 1$. Thus there is an equilibrium point in presence of mosquito vectors.

Theorem 3.2 *For the system* (3) *if* $M_A = \frac{K[L(T)\lambda(T,R)-\mu_V(T)(\lambda(T,R)+\mu_A(T))]}{L(T)\lambda(T,R)}$ *with* $L(T)\lambda(T,R) > \mu_V(T)(\lambda(T,R)+\mu_A(T))$ *then there exists a unique endemic equilibrium.*

Proof Setting the RHS of the equations in system (3) to zero gives

$$S_H = \frac{\Lambda_H+(1-p-q)A_H+\sigma R_H}{\beta_H I_V+\mu_H}, \tag{8}$$

$$E_H = \frac{pA_H + \beta_H S_H I_V}{\rho + \mu_H}, \tag{9}$$

$$I_H = \frac{qA_H + \rho E_H}{\mu_H + \nu + \delta}, \tag{10}$$

$$R_H = \frac{\nu I_H}{\sigma + \mu_H}, \tag{11}$$

$$S_V = \frac{\lambda(T,R)M_A}{\beta_V I_H + \mu_V(T)}, \tag{12}$$

$$I_V = \frac{\beta_V I_H S_V}{\mu_V(T)}. \tag{13}$$

For $M_A = \frac{K[L(T)\lambda(T,R) - \mu_V(T)(\lambda(T,R)+\mu_A(T))]}{L(T)\lambda(T,R)}$, let $d = L(T)\lambda(T,R) - \mu_V(T)(\lambda(T,R)+\mu_A(T))$ thus

$$M_A = \frac{Kd}{L(T)\lambda(T,R)}.$$

Then using equations (12) and (13)

$$S_V = \frac{Kd}{L(T)(\beta_V I_H + \mu_V(T))},$$

and

$$I_V = \frac{\beta_V I_H Kd}{L(T)\mu_V(T)(\beta_V I_H + \mu_V(T))}. \tag{14}$$

Substituting equations (10) and (13) into equation (8) gives

$$S_H = \frac{L(T)\mu_V(T)\left((\sigma+\mu_H)[\Lambda_H + (1-p-q)A_H] + \sigma\nu I_H\right)(\beta_V I_H + \mu_V(T))}{(\sigma+\mu_H)[(\beta_H\beta_V Kd + L(T)\mu_V(T)\mu_H\beta_V)I_H + L(T)\mu_H\mu_V(T)^2]}. \tag{15}$$

From equation (9)

$$E_H = \frac{pA_H + \beta_H S_H I_V}{\rho + \mu_H}.$$

Substituting for S_H and I_V using equations (14) and (15) gives

$$E_H = \frac{1}{(\rho+\mu_H)}\left(pA_H + \frac{\beta_H\beta_V Kd((\sigma+\mu_H)[\Lambda_H + (1-p-q)A_H] + \sigma\nu I_H)I_H}{(\sigma+\mu_H)[(\beta_H\beta_V Kd + L(T)\mu_V(T)\mu_H\beta_V)I_H + L(T)\mu_H\mu_V(T)^2]}\right). \tag{16}$$

From equation (10)

$$(\mu_H + \nu + \delta)I_H = qA_H + \rho E_H,$$

substituting for E_H gives that the number of infectives at equilibrium $\hat{I}_H^*$ is given

by the roots of the polynomial

$$F(\hat{I}_H^*) = r_1(\hat{I}_H^*)^2 + r_2(\hat{I}_H^*) + r_3, \tag{17}$$

where

$$\begin{aligned}
r_1 &= (\mu_H + \nu + \delta)(\rho + \mu_H)(\sigma + \mu_H)\beta_V(K\beta_H d + L(T)\mu_H\mu_V(T)) - \rho\beta_H K\beta_V d\sigma\nu,\\
r_2 &= -(A + B - C),\\
r_3 &= -L(T)\mu_H\mu_V(T)^2(\sigma + \mu_H)A_H[q(\rho + \mu_H) + \rho p],\\
A &= (\sigma + \mu_H)A_H\beta_V(q(\rho + \mu_H) + \rho p)[(K\beta_H d + L(T)\mu_H\mu_V(T))],\\
B &= \rho\beta_H K\beta_V d(\sigma + \mu_H)[\Lambda_H + (1 - p - q)A_H],\\
C &= (\mu_H + \nu + \delta)(\rho + \mu_H)(\sigma + \mu_H)L(T)\mu_H\mu_V(T)^2.
\end{aligned}$$

It is observed that $r_1 > 0$ because $\rho\beta_H K\beta_V d\sigma\nu$ is a term in the expansion of $(\mu_H + \nu + \delta)(\rho + \mu_H)(\sigma + \mu_H)\beta_V(K\beta_H d + L(T)\mu_H\mu_V(T))$, and $r_3 < 0$. Therefore the polynomial $F(I_H^{**})$ has real roots since $r_3 < 0$. For the roots to be positive there are three possible cases.

Case I : If $A + B < C$ then $r_2 > 0$, there exists one positive root if $-r_2 < \sqrt{(}r_2^2 - 4r_1r_3)$ which yields $4r_1r_3 < 0$ which is true since $r_1 > 0$ and $r_3 < 0$ thus there is one positive root if $r_2 > 0$.
Case II : If $A + B = C$ then $r_2 = 0$, there exists one positive root.
Case III : If $A + B > C$ then $r_2 < 0$, there exists two positive roots if $-r_2 > \sqrt{(}r_2^2 - 4r_1r_3)$ else there is only one positive root . This yields $4r_1r_3 > 0$ which is not possible since $r_1 > 0$ and $r_3 < 0$ thus there is one positive root if $r_2 < 0$.
Equation (17) has one positive root for any value of r_2, which implies that there exists one endemic equilibrium point when $M_A \neq 0$.
System (3) has an endemic equilibrium point given by

$$E_2(S_H, E_H, I_H, R_H, M_A, S_V, I_V) = (\hat{S}_H^*, \hat{E}_H^*, \hat{I}_H^*, \hat{R}_H^*, \hat{M}_A^*, \hat{S}_V^*, \hat{I}_V^*),$$

where

$$\begin{aligned}
\hat{S}_H^* &= \frac{L(T)\mu_V(T)[(\sigma + \mu_H)(\Lambda_H + (1 - p - q)A_H) + \sigma\nu\hat{I}_H^*](\beta_V\hat{I}_H^* + \mu_V(T))}{(\sigma + \mu_H)[(K\beta_H d + \mu_H\mu_V(T))\beta_V\hat{I}_H^* + L(T)\mu_H\mu_V(T)^2]},\\
\hat{E}_H^* &= \frac{1}{\rho + \mu_H}\left(p\Lambda + \frac{K\beta_H\beta_V d\hat{I}_H^*[(\sigma + \mu_H)(\Lambda_H + (1 - p - q)A_H) + \sigma\nu\hat{I}_H^*]}{(\sigma + \mu_H)[(K\beta_H d + L(T)\mu_H\mu_V(T))\beta_V\hat{I}_H^* + L(T)\mu_H\mu_V(T)^2]}\right),\\
\hat{R}_H^* &= \frac{\nu\hat{I}_H^*}{\sigma + \mu_H},\\
\hat{M}_A^* &= \frac{Kd}{L(T)\lambda(T, R)},\\
\hat{S}_V^* &= \frac{Kd}{L(T)(\beta_V\hat{I}_H^* + \mu_V(T))},
\end{aligned}$$

$$\hat{I}_V^* = \frac{K\beta_V d\hat{I}_H^*}{L(T)\mu_V(T)(\beta_V \hat{I}_H^* + \mu_V(T))}.$$

■

3.4 *Stability of the equilibrium points*

The Jacobian matrix for system (3) evaluated at the endemic equilibrium E_1 is given by

$$\mathcal{J}_{E_1} = \begin{bmatrix} -\mu_H & 0 & 0 & \sigma & 0 & 0 & -\beta_H S_H^* \\ 0 & -\alpha_1 & 0 & 0 & 0 & 0 & \beta_H S_H^* \\ 0 & \rho & -\alpha_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & \nu & -\alpha_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\alpha_4 & L(T) & L(T) \\ 0 & 0 & 0 & 0 & \lambda(T,R) & -\alpha_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_V I_H^* & -\mu_V(T) \end{bmatrix},$$

where $\alpha_1 = \rho + \mu_H$, $\alpha_2 = \mu_H + \nu + \delta$, $\alpha_3 = \mu_H + \sigma$, $\alpha_4 = \lambda(T,R) + \mu_A(T)$ and $\alpha_5 = \beta_V I_H^* - \mu_V(T)$. The eigenvalues of the Jacobian matrix $\mathcal{J}_{E_1}$ are $-\mu_H$, $-(\mu_H + \sigma)$, $-(\mu_H + \nu + \sigma)$, $-(\rho + \mu_H)$ and the zero points of the polynomial;

$\mathcal{Z}^3 + [\beta_V I_H^* + 2\mu_V(T) + \lambda(T,R) + \mu_A(T)]\mathcal{Z}^2 + [(\lambda(T,R) + \mu_A(T))(\beta_V I_H^* + 2\mu_V(T)) - L(T)\lambda(T,R)]\mathcal{Z} + (\beta_V I_H^* + \mu_V(T))[\mu_V(T)(\lambda(T,R) + \mu_A(T)) - L(T)\lambda(T,R)] = 0$, where $\mathcal{Z}$ is the eigenvalue.
By the Routh-Hurwitz criterion, for local stability of the endemic equilibrium $E_1, (\lambda(T,R) + \mu_A(T))(\beta_V I_H^* + 2\mu_V(T)) > L(T)\lambda(T,R)$ and $\mu_V(T)(\lambda(T,R) + \mu_A(T)) > L(T)\lambda(T,R)$ must be satisfied. However it has already been established that the endemic equilibrium E_1 only exists if $\mu_V(T)(\lambda(T,R) + \mu_A(T)) < L(T)\lambda(T,R)$ which violets the condition of stability hence the endemic equilibrium E_1 is locally unstable.

The local and global stability of the endemic equilibrium E_2 is investigated using numerical simulations and illustrated in section 4.

4. Numerical simulation

In this section, numerical simulation of the model with initial conditions $S_H(0) = 5000$, $E_H(0) = 200$, $I_H(0) = 500$, $R_H(0) = 0$, $M_A(0) = 2000$, $S_V(0) = 3000$, $I_V(0) = 100$ is performed. According to Mukhtar *et al.* [22], the maturation rate of aquatic mosquitoes to adulthood $\lambda(T,R)$ is temperature and rainfall dependent, governed by the total number of eggs laid per adult per oviposition $\omega(T)$, the daily survival probability of the rainfall dependent eggs $P_1(R)$, the daily survival probability of the rainfall dependent larvae $P_2(R)$, the daily survival probability of the rainfall dependent pupae $P_3(R)$, daily survival probability of the temperature dependent larvae $T_{EA}(T)$, and the temperature dependent duration of the immature mosquito development $T_{EA}(T)$. The temperature and rainfall dependent

parameters obtained from Mukhtar *et al.* [22] are given by

$$\begin{aligned}
\omega(T) &= \frac{-0.153T^2 + 8.61T - 97.7}{\mu_V(T)},\\
P_1(R) &= \frac{4 * 0.93}{2500} R(50 - R),\\
P_2(R) &= \frac{4 * 0.25}{2500} R(50 - R),\\
P_3(R) &= \frac{4 * 0.75}{2500} R(50 - R),\\
P_2(T) &= exp(0.06737 - 0.00554T),\\
T_{EA}(T) &= \frac{1}{-0.00094T^2 + 0.049T - 0.552}.\\
L(T) &= -0.153T^2 + 8.61T - 97.7\\
\lambda(T, R) &= \frac{\omega(T)P_1(R)P_2(R)P_3(R)P_2(T)}{T_{EA}(T)}\\
\mu_A(T) &= 1.0257 - 0.094T + 0.0025T^2\\
\mu_V(T) &= -ln(0.522 - 0.000828T^2 + 0.0367T)
\end{aligned}$$

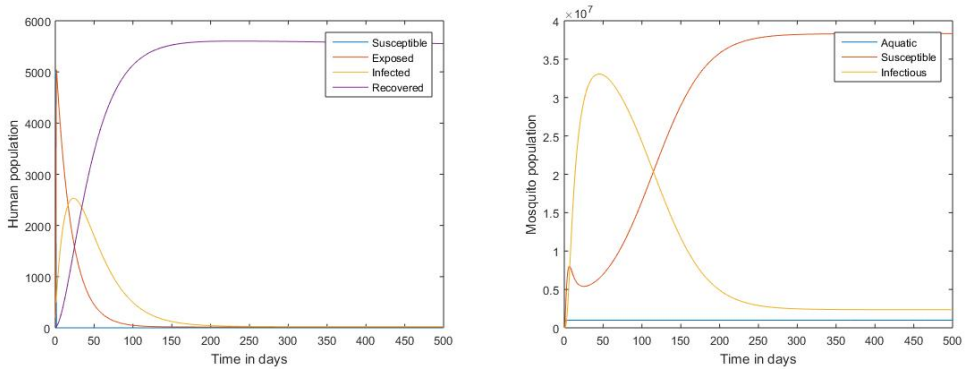
Temperature 25^o C and rainfall 30 mm are used because these were found to be the most effective values for malaria transmission in [45]. The rest of the parameters used are indicated in Table 1.

Table 1. Summary of the parameter values used.

Parameter	**Symbol**	**Value**	**Reference**
Birth rate of humans	Λ_H	0.03/day	[34]
Human immigration rate	A_H	0.001/day	Assumed
Natural death rate of humans	μ_H	1/21900/day	[7]
Interaction coefficient between susceptible humans and infected mosquitoes	β_H	0.00021	[34]
Rate of loss of immunity	σ	$1/(20 * 365)$/day	[7]
Progression rate from exposed class	ρ	1/20/day	[7]
Recovery rate	ν	1/30/day	[7]
Disease induced death rate	δ	0.001/day	[7]
Interaction coefficient between susceptible mosquitoes and infected humans	β_V	0.00021	[34]
Carrying capacity of the environment	K	1000000	[22]

Figure 1 (a) shows the human population change with time, it is observed that the populations in all the four compartments attain equilibrium. Similary, it is observed in Figure 1 (b) that the mosquito population in all the three compartments attains equilibrium. Therefore Figure 1 shows that the endemic equilibrium point E_2 is locally stable.

In Figure 2, initial conditions I_1, I_2, I_3, I_4 and I_5 are used to investigate the



(a) (b)

Figure 1. Human population change with time (a) and mosquito population change with time (b) over a period of 500 days.

global stabilty of the endemic equilibrium point E_2. Stability is observed for the susceptible human population in 2 (a), exposed human popultion in 2 (b), infected human population in 2 (c), aquatic mosquito population in 2 (e) and the susceptible mosquito population in 2 (f). However, Figure 2 (d) showing the recovered human population and Figure 2 (g) showing the infectious mosquito population indicate that the endemic equilibrium point E_2 is not globally stable. This is because different initial conditions result in different values at the equilibrium.

To investigate the effect of infected immigrants, different values of p (proportion of exposed immigrants) and q (proportion of infected immigrants) are used to perform a numerical simulation shown in Figure 3. Four cases are considered, that is, $p = q = 0$, $p = q = 0.2$, $p = 0.4 > q = 0.2$ and $p = 0.2 < q = 0.4$. It is observed that the proportion of infected immigrants introduced into the community does not significantly affect the malaria infection pattern as the infection curve does not swift neither upwards nor downwards.

5. Conclusion

A mathematical model that captures temperature and rainfall dependence in the mosquito population and inflow of infected immigrants has been presented. Analysis of the model reveals that the infected population does not affect the size of the aquatic mosquito population. The model has two endemic equilibria, one free of the mosquito vector population E_1 and the other with the mosquito vector population E_2. The endemic equilibrium E_1 without the mosquito vector population represents an area of no transmission since transmission is dependent on the mosquito vector population. The endemic equilibrium E_1 is locally unstable. It is observed that this is due to the inflow of infected immigrants such that if the infected immigrants are blocked the endemic equilibrium E_1 reduces to a disease free equilibrium. However as long as there is inflow of infected humans into the community the disease will persist. Therefore, for areas of no transmission it is important that those entering into the community are screened and those found infected treated in-order to maintain a disease free state. The endemic equilibrium E_2 is due to both inflow of infected migrants and transmission by the mosquito vectors. The endemic equilibrium E_2 is locally stable which shows that in the long run the disease persists in the population.

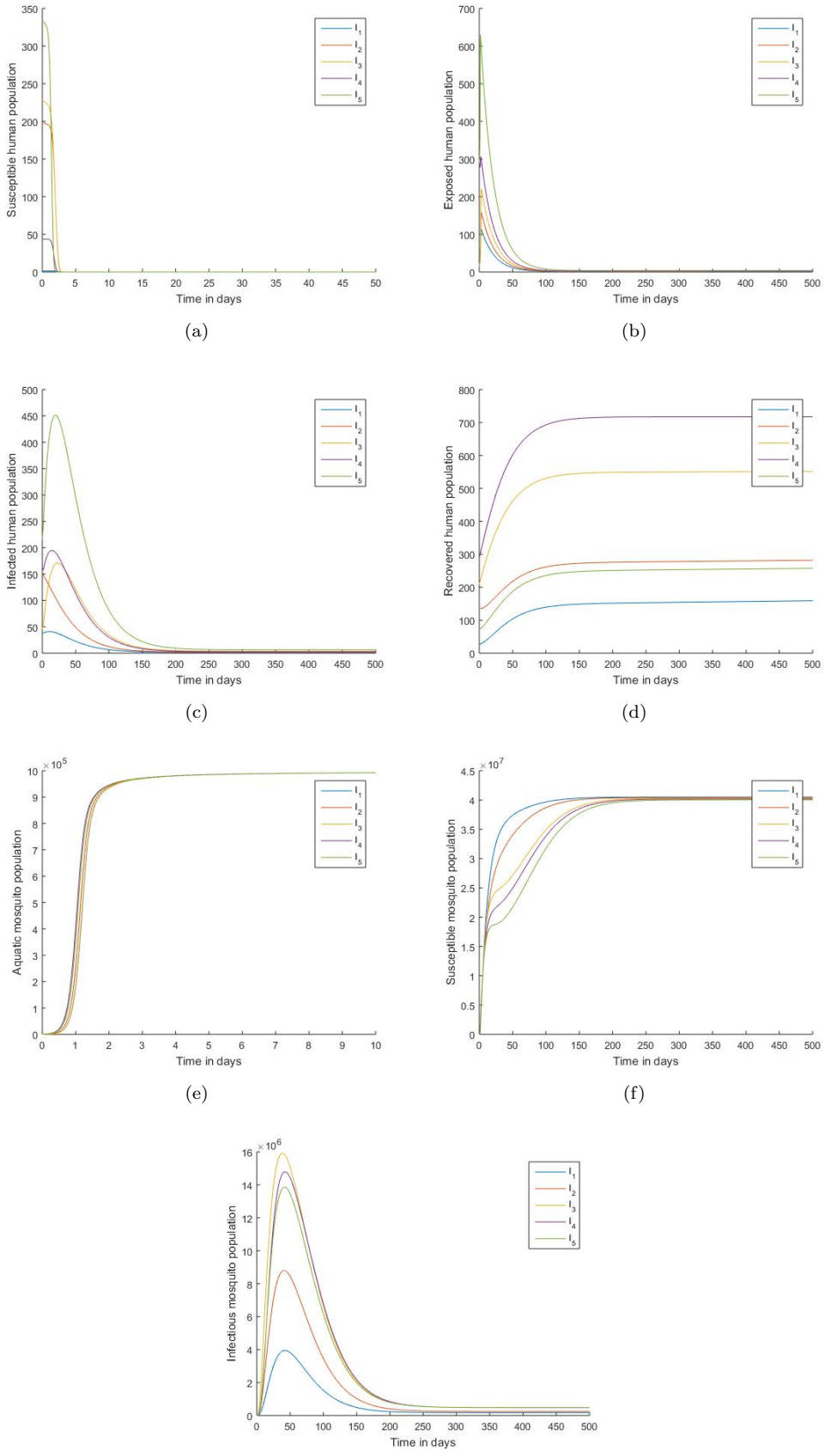


Figure 2. Showing the global stability of the endemic equilibrium point for all the human and mosquito population compartments.

Numerical simulations show that the endemic equilibrium E_2 is not globally stable. It is also shown that a proportion of infected immigrants introduced into the community does not significantly affect the malaria transmission pattern. Thus, over-emphasising the role of infected immigrants and its impact on malaria patterns is misleading for malaria eradication.

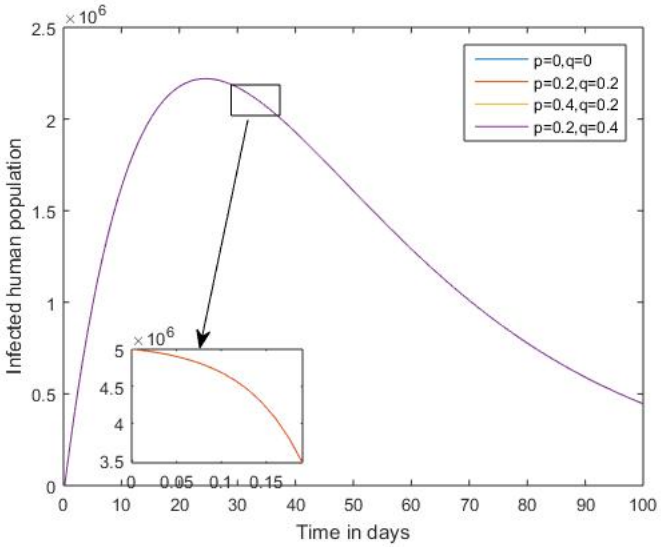


Figure 3. Showing the effect of the proportion of exposed and infected immigrants to the total number of infectives in the population over a period of 100 days.

Acknowledgments

Victor Yiga acknowledges the contribution of the Regional University Forum for Capacity Building in Agriculture (RUFORUM), Kyambogo University and Mbarara University of Science and Technology towards this research.

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