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Modelling the co-infection of malaria and zika virus disease

Emmanuel Chidiebere Duru[a,*], Godwin Christopher Ezike Mbah[b], Michael Chimezie Anyanwu[a], Nnamani Nicholas Topman[b]

[a]*Department of Mathematics, Michael Okpara University of Agriculture, P.M.B. 7267 Umudike, Nigeria*
[b]*Department of Mathematics, University of Nigeria, P.M.B. 3147 Nsukka, Nigeria*

Abstract

In this work, a new model for the co-infection of malaria and zika virus disease incorporating vaccination, treatment and vector control using sterile-insect technology (SIT) is formulated. The importance of this study is to highlight the possibility of the co-infection of humans with malaria and zika virus disease in any environment where both diseases co-circulate. Also, to suggest a new and comprehensive method for controlling the individual diseases and their co-infection. Through stability analysis, we showed that the disease-free equilibrium, (DFE) point of the co-infection model is locally asymptotically stable when the basic reproduction numbers, R_{mz} is less than one, and unstable otherwise. But, the DFE failed to be globally stable when $R_{mz} < 1$ which is an indication of existence of backward bifurcation in the model. This shows that bringing down the reproduction number, R_{mz} to less than one is not enough to eradicate the co-infection of the two diseases. Furthermore, it is shown that the two diseases have positive impact on the spread of each other, which could be attributed to misdiagnoses of one disease as the other. We also showed that effective treatment of infectious humans, increasing the rate of vaccination and employing sterile-insect technique to control the vectors significantly helped to control the individual diseases as well as the co-infection. From the results obtained in the study, it can be concluded that effective control of malaria and zika virus disease requires measures that will control their spread in both human and mosquito populations.

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1. Introduction

Malaria is a deadly endemic disease in Asia and many sub-Saharan African Countries. It is caused by the *Plasmodium parasite* and transmitted through the bites of the female Anopheles mosquito [1, 2]. Malaria can also be transmitted by blood transfusions, organ transplants and sharing of needles by intravenous drug (IV drugs) users [2]. About 40% of the population of the world are estimated to live in malaria endemic areas and the disease causes about 1 to 3 million deaths per year, with 75% of them African children [3]. Also, about 435,000 malaria-induced deaths occurred globally in 2017 [3]. Out of the five parasites that cause malaria, the most severe are *Plasmodium falciparum* and *Plasmodium vivax* [4]. Common symptoms of malaria include headache, profuse sweating, nausea, severe anaemia, high or moderate fever, severe or moderate shaking chills, morbidity, infant mortality, abdominal pain, muscle pain, etc. [5]. Recent studies have shown there is in-

*Corresponding author: Tel. No.: +234-706-532-8417.
Email address: duru.emmanuel@mouau.edu.ng (Emmanuel Chidiebere Duru)

crease in the rate at which the malaria parasites resist drug and also the mosquitoes resist insecticide thus leading to the prevalence of the disease [1]. This underlines the need to study and understand the various parameters involved in the disease dynamics. R21/Matrix-M vaccine was investigated in 2021 as a second vaccine for malaria after RTS,S, vaccine and World Health Organization, (WHO) declared that it has a minimum efficacy limit of 75% [6]. This means that it can reduce the probability of humans being infected by an infectious female Anopheles mosquito [5].

Zika virus disease on other hand is a *flavivirus disease* transmitted amongst humans through the bites of the Aedes aegypti mosquitoes, blood trnsfusion, sex and during pregnancy [7, 8]. Zika virus was first reported in Uganda in 1947 [7] and the first human infections were discovered in Nigeria in 1954 [9]. In 2015, zika virus infection by mosquitoes were recorded in 69 countries, with reported cases of congenital transmission in 29 countries, 13 countries had human-to-human transmission while reported cases of Guillain-Barre syndrome (GBs) was reported in 20 countries [10]. The Aedes mosquito that transmits zika virus is also responsible for transmitting yellow fever, dengue and chikungunya [7].

Some of the symptoms of zika virus disease include maculopapular rash, headache, malaise, mild fever, joint pain, muscle pain, conjunctivitis, arthralgia, etc. [10, 11]. Zika virus disease can be misdiagnosed as malaria, dengue or chikungunya because they have similar symptoms. Zika infection can cause congenital abnormalities of the brain like microcephaly during pregnancy which made the disease become a major global concern [12, 13]. Zika virus also triggers the Guillain-Barre syndrome [9]. Zika virus infection, dengue and chikungunya are among the major causes of ill-health in the tropics and subtropics and it causes significant health, economic and social burdens [14].

The sterile-insect technology, (SIT) is a biological control measure for insects and pests [15]. It involves releasing a large number of sterile male insects into a target wild population to mate with the females [16]. Females are not used to prevent them from laying eggs and increasing the population further. Using female mosquitoes as a control is not advisable because they can transmit plasmodium while feeding on human blood. When a sterile male released into the wild mates with a female mosquito, they female lay eggs without hatching thus producing no offspring [17, 18]. This technique has been used to eradicate the screw-worm fly from Mexico, Puerto Rico, the United States, Libya and Central America successfully [15]. It was also used to eradicate the Mexican fruit fly in northern Mexico and tsetse fly from Zanzibar in 1998 and from Senegal in 2014 [19, 20]. Currently, SIT is targeted to eradicate and control the population of mosquitoes. [21].

The mathematical modelling of malaria began as far back as 1911 with Ross's model which was later extended by MacDonald [1]. Their models had one variable representing humans and the other representing mosquitoes. Thereafter, Ref. [22] improved the two-dimensional work by including acquired immunity to the human compartment. Many other models have since been developed and studied on malaria diseases such as Ref. [23] who investigated the malaria transmission model with physical control. There was no endemic equilibrium in their model confirming the possibility of a malaria – free society. Ref. [24] worked on the optimal control of a malaria model incorporating seasonal factor in mosquitoes. They showed that seasonal factor has more influence on the dynamics of human population and infected mosquitoes in hot climate regions. Ref. [5] considered a malaria model that incorporates a preventive treatment against relapse as well as saturated fumigation. Specifically, the work suggested using tafenoquine treatment which when it is increasing, the basic reproduction number decreases and vice versa. Other works include Refs. [25–29], etc. In modelling zika virus disease, Ref. [30] used bilinear incidence rate with no inhibiting factors of infections. Ref. [31] used stochastic agent-based model to simulate the transmission dynamics of zika virus mosquitoes in 11 islands. Ref. [32] evaluated the basic reproduction number as a measurement of transmission potential and reanalyzing past epidemic data from the south pacific in studying zika virus model. Ref. [33] developed a mathematical model to analyze the zika virus from Rio de Janeiro to Miami during Carnival. Ref. [17] developed a model that employed SIT to reduce the population of the Aedes mosquitoes responsible for the zika virus disease. Further works done on zika virus disease modelling include Refs. [9, 34–37], etc.

For co-infection models, Ref. [38] proposed a co-infection model of malaria and zika virus diseases with nine (9) compartments. Their sensitivity analysis showed that the best approach to control both diseases is to improve their recovery rate which will bring down the basic reproduction number. Ref. [39] published a paper on the prevalence of malaria and zika virus diseases in patients with fever in secondary healthcare facilities in south-east of Nigeria using a stratified survey of nine (9) secondary facilities with 100 patients. Their work did not incorporate any model nor mathematical analysis. They tested blood samples of the patients and found out that 55% of the patients had malaria, 20% had zika infection while about 15% had the co-infection. Their work showed the high probability of the existence of the co-infection of both disease which is part of the motivation for our work. Ref. [40] conducted a clinical and sero-epidemiological studies to help understand the hidden endemicity and disease burden of zika virus, malaria and other arbovirus in humans. There study showed an increase in hidden endemicity, antibody seropositivity as well as the possibility of zika virus, malaria and other flavivirus co-circulating in Nigeria. Other works on co-infection of malaria and zika virus disease include Refs. [41, 42], etc.

In this work, we looked at a new transmission model for the co-infection of malaria and zika virus diseases that incorporates vaccination, treatment and use of SIT to control the population of the mosquitoes. It is expected that as more people are vaccinated and the mosquito population is reduced, the disease will be contained. The rest of this paper is arranged thus; in section 2, we propose the new model describing the disease dynamics in both human and mosquito populations as well as obtaining the disease-free equilibrium (DFE) point of the system; section 3 shows the basic reproduction number for the sys-

tem; in section 4, we looked at the local and global stability of the DFE; while section 5 discusses the impart of each disease on the spread of the other; section 6 centred on the sensitivity analysis of parameters of the reproduction number; in section 7, we performed the numerical simulation for the model and concluded the work in section 8.

2. Mathematical model

We present a new mathematical model for the co-infection with twenty-two (22) compartments made up of the human and mosquito populations. The human population is divided into sixteen (16) compartments; Susceptible humans S_h, Vaccinated humans for malaria S_{hv}, Unvaccinated humans for malaria S_{hu}, Exposed humans to malaria E_{hm}, Exposed humans to zika E_{hz}, co-infected humans with both diseases E_{mz}, Infectious humans with malaria I_{hm}, symptomatic infectious humans with zika I_{hzS}, asymptomatic infectious humans with zika I_{hzA}, co-infectious humans with both diseases I_{mz}, Infectious humans undergoing treatment for malaria I_{hmT}, Infectious humans undergoing treatment for zika I_{hzT}, co-infectious humans undergoing treatment for both diseases I_{mzT}, Infectious humans not undergoing treatment for malaria I_{hmU}, co-infectious humans not undergoing treatment for both disease I_{mzU} and Recovered humans R_h.

The mosquito population is subdivided into six (6) compartments; susceptible Anopheles mosquitoes S_{mv}, exposed Anopheles mosquitoes E_{mv}, Infectious Anopheles Mosquitoes I_{mv}, susceptible Aedes mosquitoes S_{zv}, exposed Aedes mosquitoes E_{zv} and infectious Aedes mosquitoes I_{zv}. We assumed that the infectious humans with malaria not undergoing treatment who can suppress the disease with time due to their immunity return to the exposed class with malaria only [43, 44]. Similar assumption is made for co-infectious humans with both diseases where those who recover from zika and can suppress the malaria disease due to their immunity returns to the exposed class with malaria only. We also assumed that the malaria vaccine does not grant total and permanent immunity to the disease [45]. We also assumed that recovered humans are reintroduced into susceptible human population since recovering from malaria does not grant permanent immunity to the disease like zika virus disease as seen in all the works cited in this paper.

2.1. Disease dynamics in human population

The susceptible human population, S_h is increased by level of recruitment through births and migration denoted by Λ_h and rate of recovery θ from the disease. It is reduced by movement into the vaccinated and unvaccinated class as well as natural death rate, τ_1. We assume that the vaccination does not grant permanent immunity to malaria but wanes with time. The unvaccinated human population, S_{hu} is increased by the proportion of the susceptible human population that are not vaccinated ρ_1 and reduced by the proportion exposed to malaria parasite and zika virus respectively as well as natural death rate, τ_1. The vaccinated human population, S_{hv} is increased by the proportion of the susceptible human population that are vaccinated ρ_2 and reduced by the proportion exposed to malaria parasite at the rate, φ and zika virus respectively as well as natural death rate, τ_1. The exposed human population to malaria, E_{hm} is increased by rate of movement from both the vaccinated and unvaccinated class as humans are bitten by mosquitoes with malaria parasites and infected. It is reduced by rate of development of infectiousness, δ_1 as well as natural death rate, τ_1. When the exposed humans to malaria are bitten by Aedes mosquitoes and infected with zika virus, they become co-infected with both diseases and move to the compartment exposed to both infections.

The exposed human population to zika virus, E_{hz} is increased by rate of movement of infected persons from both the vaccinated and unvaccinated class as humans are bitten by mosquitoes with zika virus and infected. It is reduced by rate of development of infectiousness, δ_2 as well as natural death rate, τ_1. When the exposed humans to zika virus disease are bitten by Anopheles mosquitoes and infected with malaria parasite, they become co-infected with both diseases and move to the compartment exposed to both infections. As infectiousness develops, the proportion that are symptomatic, χ_1 moves to the symptomatic class I_{hzS} and the proportion that are asymptomatic, χ_2 moves to the asymptomatic class I_{hzA}. The co-infected human population, E_{hmz} is increased by the proportion of exposed human to malaria and exposed humans to zika that becomes co-infected with both diseases. It is reduced by rate of becoming infectious with zika and malaria, δ_3 as well as natural death rate, τ_1. The infectious human population to malaria, I_{hm} is increased by rate of development of infectiousness from E_{hm} represented by δ_1 and reduced by natural death rate, τ_1 as well as death due to the disease τ_2. It is partitioned into two; the proportion that accepts to go for treatment, ε_1 and the proportion that refuses to go for treatment, ε_2. When the infectious humans with malaria are bitten by Aedes mosquitoes and infected with zika virus, we assume that they will become co-infectious with both disease with time. The infectious human population undergoing treatment for malaria, I_{hmT} is increased by the proportion of infectious humans with malaria that accepts to go treatment, ε_1 and reduced by natural death rate, τ_1 death rate due to the disease, τ_2 as well as rate of recovery from malaria, γ_1.

The untreated human population, I_{hmU} is increased by proportion of those that refuse to go for treatment, ε_2, reduced by natural death rate, τ_1 level of death due to the disease τ_2 as well as level of movement into the exposed class with malaria, ϕ_1 by those who loses their infectiousness due to their immune system with time but has not recovered due to lack of treatment. The symptomatic infectious human population to zika, I_{hzS} is increased by the proportion of infectious humans, χ_1 who show symptoms of the disease and reduced by natural death rate, τ_1 as well as death due to the disease, τ_3. The proportion of the population that accepts to go for treatment, ψ moves into the treated compartment while those who refuse treatment and are undecided can recover naturally at the rate, ω_1.

The asymptomatic infectious human population to zika, I_{hzA} is increased by the proportion of infectious humans, χ_2 who show no symptoms of the disease and reduced by natural death rate, τ_1 as well as death due to the disease, τ_3 and rate at which they recover naturally, ω_3. The infectious human population

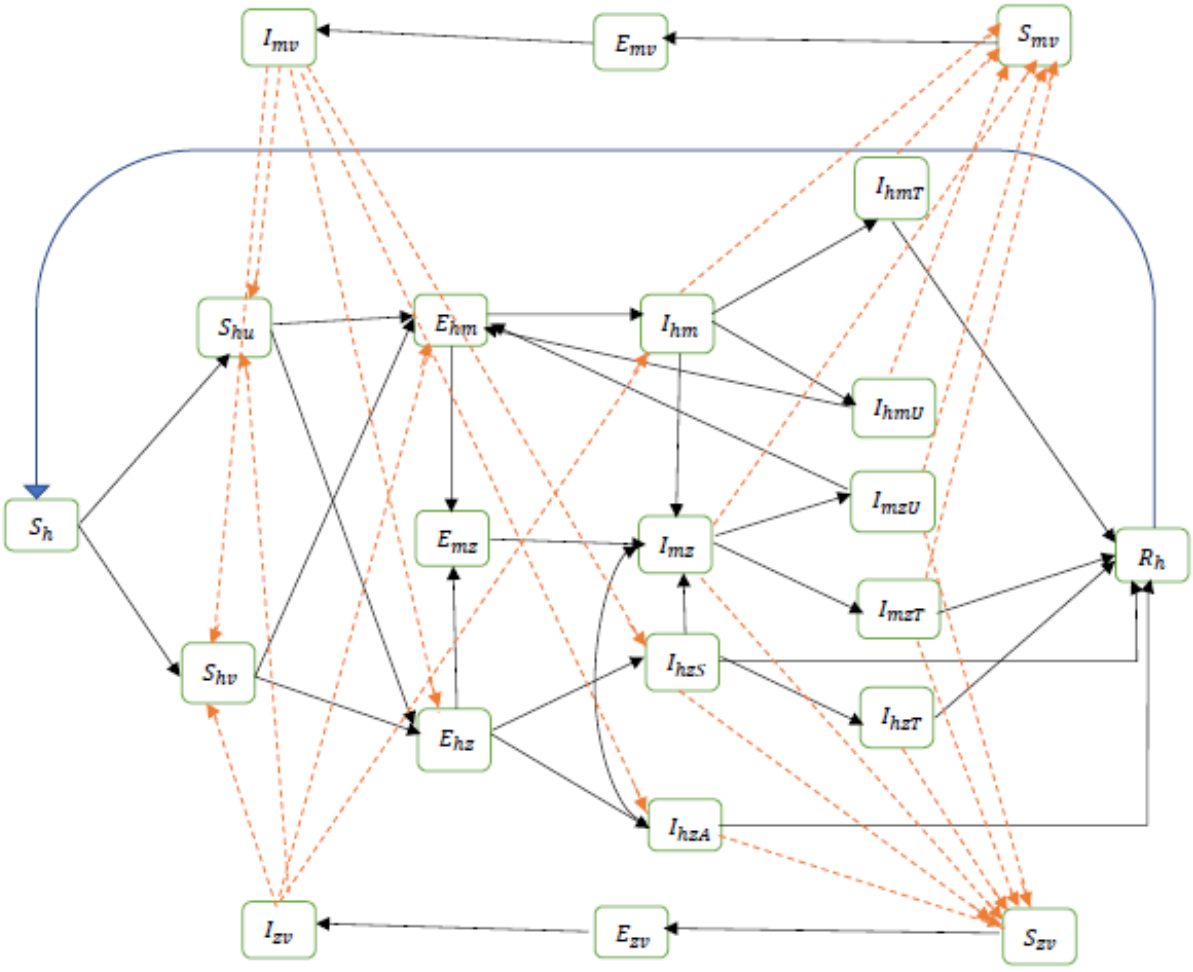

Figure 1. The flow diagram for the co-infection of malaria and zika virus disease. The reddish breaking lines signifies interactions between humans and mosquitoes leading to infections while the unbroken lines represents movement from one compartment to another in the system.

undergoing treatment for zika, I_{hzT} is increased by the proportion of symptomatic infectious humans with zika that accepts to go treatment, ψ and reduced by natural death rate, τ_1 as well as rate of recovery from zika ω_2. The co-infectious human population with malaria and zika I_{mz} is increased by rate of development of co-infectiousness from E_{mz} represented by δ_3 and rate of development of co-infectiousness in the infectious malaria and zika classes respectively. It is reduced by natural death rate, τ_1 and death due to co-infection τ_4. It is partitioned into two; the proportion that accepts to go for treatment, σ_1 and the proportion that refuses to go for treatment, σ_2. The co-infectious human population undergoing treatment for malaria and zika virus disease, I_{mzT} is increased by the proportion of co-infectious humans that accepts to go for treatment, σ_1 and reduced by natural death rate, τ_1 death rate due to co-infection, τ_4 as well as rate of recovery γ_2 from the disease co-infection.

The co-infectious human population not undergoing treatment for malaria and zika virus disease, I_{mzU} is increased by the proportion of co-infectious humans that refuses to go for treatment, σ_2 and reduced by natural death rate, τ_1 death rate due to co-infection, τ_4 as well as level of movement into the exposed human population to malaria, ϕ_2 by those who recover from zika naturally but has not recovered from malaria rather loses their infectiousness due to their immune system with time. The recovered human population R_h is increased by rate of recovery of infectious humans with malaria undergoing treatment, γ_1, rate of recovery from infectious humans with zika undergoing treatment, ω_2, rate of recovery of co-infection humans undergoing treatment, γ_2, rate of recovery of asymptomatic humans with zika naturally, ω_3 and rate of natural recovery of symptomatic humans with zika who refuses treatment, ω_1. It is reduced by natural death rate, τ_1 and rate at which the recovered humans lose immunity against malaria and return to the susceptible population.

2.2. *Disease dynamics in mosquito population*

The susceptible anopheles population, S_{mv} is increased by the level of recruitment denoted by Λ_{mv} and reduced by natural death rate μ, rate of reduction κ_1 in mosquito population due to SIT as well as rate of exposure of Anopheles mosquitoes

to infectious and co-infectious humans with malaria. The SIT technique reduces the mosquito population with time since the sterile male introduced into the environment mating with the Anopheles mosquitoes produces no offspring. The exposed anopheles population, E_{mv} is increased by rate at which the susceptible mosquitoes gets infected through contacts with infectious humans with malaria and reduced by natural death rate, μ and rate of infectiousness, ν_1. The infectious anopheles population, I_{mv} is increased by rate of development of infectiousness, ν_1 and reduced by natural death rate, μ.

The susceptible aedes population, S_{zv} is increased by the level of recruitment denoted by Λ_{zv} and reduced by natural death rate μ, rate of reduction κ_2 in mosquito population due to SIT as well as as well as rate of exposure of zika mosquitoes to infectious and co-infectious humans with zika virus. The exposed aedes population, E_{zv} is increased by rate at which the susceptible mosquitoes gets infected through contacts with infectious humans and reduced by natural death rate, μ and rate of development of infectiousness, ν_2. The infectious aedes population, I_{zv} is increased by rate of development of infectiousness, ν_2 and reduced by natural death rate, μ. Both κ_1 and κ_2 are dependent on the mating rate and mating probability of the SIT mosquitoes with the females in the wild.

Thus, the model for the system becomes:

$$
\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \theta R_h - (\rho_1 + \rho_2 + \tau_1)S_h.\\
\frac{dS_{hu}}{dt} &= \rho_1 S_h - \alpha_1\beta_1 I_{mv} S_{hu} - \alpha_2\eta_1 I_{zv} S_{hu} - \tau_1 S_{hu}.\\
\frac{dS_{hv}}{dt} &= \rho_2 S_h - \alpha_1\beta_2\varphi I_{mv} S_{hv} - \alpha_2\eta_1 I_{zv} S_{hv} - \tau_1 S_{hv}.\\
\frac{dE_{hm}}{dt} &= \alpha_1\beta_1 I_{mv} S_{hu} + \alpha_1\beta_2\varphi I_{mv} S_{hv} + \phi_1 I_{hmU} + \phi_2 I_{mzU}\\
&\quad - \alpha_2\eta_1 I_{zv} E_{hm} - (\delta_1 + \tau_1)E_{hm}.\\
\frac{dE_{hz}}{dt} &= \alpha_2\eta_1 I_{zv}(S_{hu} + S_{hv}) - \alpha_1\beta_1 I_{mv} E_{hz}\\
&\quad - ((\chi_1 + \chi_2)\delta_2 + \tau_1)E_{hz}.\\
\frac{dE_{mz}}{dt} &= \alpha_1\beta_1 I_{mv} E_{hz} + \alpha_2\eta_1 I_{zv} E_{hm} - (\delta_3 + \tau_1)E_{mz}.\\
\frac{dI_{hm}}{dt} &= \delta_1 E_{hm} - \alpha_2\eta_1 I_{zv} I_{hm} - (\tau_1 + \tau_2 + \varepsilon_1 + \varepsilon_2)I_{hm}.\\
\frac{dI_{hmT}}{dt} &= \varepsilon_1 I_{hm} - (\gamma_1 + \tau_1 + \tau_2)I_{hmT}.\\
\frac{dI_{hmU}}{dt} &= \varepsilon_2 I_{hm} - (\phi_1 + \tau_1 + \tau_2)I_{hmU}.\\
\frac{dI_{hzS}}{dt} &= \delta_2\chi_1 E_{hz} - \alpha_1\beta_1 I_{mv} I_{hzS} - (\tau_1 + \tau_2 + \psi + \omega_1)I_{hzS}.\\
\frac{dI_{hzA}}{dt} &= \delta_2\chi_2 E_{hz} - \alpha_1\beta_1 I_{mv} I_{hzA} - (\tau_1 + \tau_2 + \omega_3)I_{hzA}.\\
\frac{dI_{hzT}}{dt} &= \psi I_{hzS} - (\tau_1 + \tau_2 + \omega_2)I_{hzT}.\\
\frac{dI_{mz}}{dt} &= \alpha_1\beta_1 I_{mv}(I_{hzS} + I_{hzA}) + \alpha_2\eta_1 I_{zv} I_{hm} + \delta_3 E_{mz}\\
&\quad - (\tau_1 + \tau_4 + \sigma_1 + \sigma_2)I_{mz}.\\
\frac{dI_{mzT}}{dt} &= \sigma_1 I_{mz} - (\tau_1 + \tau_4 + \gamma_2)I_{mzT}.\\
\frac{dI_{mzU}}{dt} &= \sigma_2 I_{mz} - (\tau_1 + \tau_4 + \phi_2)I_{mzU}.\\
\frac{dR_h}{dt} &= \gamma_1 I_{hmT} + \gamma_2 I_{mzT} + \omega_1 I_{hzS} + \omega_2 I_{hzT} + \omega_3 I_{hzA}\\
&\quad - (\tau_1 + \theta)R_h.\\
\frac{dS_{mv}}{dt} &= \Lambda_{mv} - \alpha_1(\beta_3 I_{hm} + \beta_4 I_{hmT} + \beta_5 I_{hmU} + \beta_6 I_{mz} + \beta_7 I_{mzT}.\\
&\quad + \beta_8 I_{mzU})S_{mv} - (\kappa_1 I_{SIT} + \mu)S_{mv}.\\
\frac{dE_{mv}}{dt} &= \alpha_1(\beta_3 I_{hm} + \beta_4 I_{hmT} + \beta_5 I_{hmU} + \beta_6 I_{mz} + \beta_7 I_{mzT}\\
&\quad + \beta_8 I_{mzU})S_{mv} - (\nu_1 + \mu)E_{mv}.\\
\frac{dI_{mv}}{dt} &= \nu_1 E_{mv} - \mu I_{mv}.\\
\frac{dS_{zv}}{dt} &= \Lambda_{zv} - \alpha_2(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT} + \eta_5 I_{mz} + \eta_6 I_{mzT}\\
&\quad + \eta_7 I_{mzU})S_{zv} - (\kappa_2 I_{SIT} + \mu)S_{zv}.\\
\frac{dE_{zv}}{dt} &= \alpha_2(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT} + \eta_5 I_{mz} + \eta_6 I_{mzT}\\
&\quad + \eta_7 I_{mzU})S_{zv} - (\nu_2 + \mu)E_{zv}.\\
\frac{dI_{zv}}{dt} &= \nu_2 E_{zv} - \mu I_{zv}, \qquad (1)
\end{aligned}
$$

where $S_h(0) = S_h^0, S_{hu}(0) = S_{hu}^0, S_{hv}(0) = S_{hv}^0, E_{hm}(0) = E_{hm}^0, E_{hz}(0) = E_{hz}^0, E_{mz}(0) = E_{mz}^0, I_{hm}(0) = I_{hm}^0, I_{mz}(0) = I_{mz}^0, I_{hmT}(0) = I_{hmT}^0, I_{hmU}(0) = I_{hmU}^0, I_{hzS}(0) = I_{hzS}^0, I_{hzA}(0) = I_{hzA}^0, I_{hzT}(0) = I_{hzT}^0, I_{mzT}(0) = I_{mzT}^0, I_{mzU}(0) = I_{mzU}^0, R_h(0) = R_h^0, S_{mv}(0) = S_{mv}^0, E_{mv}(0) = E_{mv}^0, I_{mv}(0) = I_{mv}^0, S_{zv}(0) = S_{zv}^0$, $E_{zv}(0) = E_{zv}^0$ and $I_{zv}(0) = I_{zv}^0$ are the initial conditions of the system with

$$
\begin{aligned}
N_h &= S_h + S_{hu} + S_{hv} + E_{hm} + E_{hz} + E_{mz} + I_{hm} + I_{hmT} + I_{hmU}\\
&\quad + I_{hzS} + I_{hzA} + I_{hzT} + I_{mz} + I_{mzT} + I_{mzU} + R_h.\\
N_{mv} &= S_{mv} + E_{mv} + I_{mv}.\\
N_{zv} &= S_{zv} + E_{zv} + I_{zv}.
\end{aligned}
$$

We assume that the solution to the system exists in the region $\Omega = \Omega_1 + \Omega_2 + \Omega_3$ described by $S_h, S_{hu}, S_{hv}, E_{hm}, E_{hz}, E_{mz}, I_{hm}, I_{hmT}, I_{hmU}, I_{hzS}, I_{hzA}, I_{hzT}, I_{mz}$, $I_{mzT}, I_{mzU}, R_h \in \mathbb{R}^{16} | S_h + S_{hu} + S_{hv} + E_{hm} + E_{hz} + E_{mz} + I_{hm} + I_{hmT} + I_{hmU} + I_{hzS} + I_{hzA} + I_{hzT} + I_{mz} + I_{mzT} + I_{mzU} + R_h \leq \frac{\Lambda_h}{\tau_1}$, $S_{mv}, E_{mv}, I_{mv} \in \mathbb{R}^3 | S_{mv} + E_{mv} + I_{mv} \leq \frac{\Lambda_{mv}}{\mu}$ and $S_{zv}, E_{zv}, I_{zv} \in \mathbb{R}^3 | S_{zv} + E_{zv} + I_{zv} \leq \frac{\Lambda_{zv}}{\mu}$.

2.3. Positivity of solutions and invariant region

Theorem 1: Let the initial data set for the model be $S_h^0, S_{hu}^0, S_{hv}^0, E_{hm}^0, E_{hz}^0, E_{mz}^0, I_{hm}^0, I_{mz}^0, I_{hmT}^0, I_{hmU}^0, I_{hzS}^0, I_{hzA}^0, I_{hzT}^0, I_{mzT}^0, I_{mzU}^0, R_h^0, S_{mv}^0, E_{mv}^0, I_{mv}^0, S_{zv}^0, E_{zv}^0$ which are all non-negative at $t = 0$. Then, the solution $S_h(t)$, $S_{hu}(t)$, $S_{hv}(t)$, $E_{hm}(t)$, $E_{hz}(t)$, $E_{mz}(t)$, $I_{hm}(t)$, $I_{mz}(t)$, $I_{hmT}(t)$, $I_{hmU}(t)$, $I_{hzS}(t)$, $I_{hzA}(t)$, $I_{hzT}(t)$, $I_{mzT}(t)$, $I_{mzU}(t)$, $R_h(t)$, $S_{mv}(t)$, $E_{mv}(t)$, $I_{mv}(t)$, $S_{zv}(t)$, $E_{zv}(t)$, $I_{zv}(t)$ of the model with the given initial data will remain positive for all time $t > 0$.

Table 1. Description of parameters.

Parameters	Description
Λ_h	Level of recruitment of humans into susceptible population
Λ_{mv}	Level of recruitment of Anopheles mosquitoes
Λ_{zv}	Level of recruitment of Aedes mosquitoes
φ	Rate at which vaccinated humans loses immunity
θ	Rate of loss of immunity to malaria from recovered class
ρ_1	Proportion of the susceptible humans that are not vaccinated
ρ_2	Proportion of the susceptible humans that are vaccinated
τ_1	Natural death rate of humans
τ_2	Death due to malaria disease
τ_3	Death due to zika disease
τ_4	Death due to co-infection
α_1	Contact rate of Anopheles mosquitoes with humans
α_2	Contact rate of Aedes mosquitoes with humans
β_1	Rate of transmission from infectious mosquitoes to unvaccinated humans
β_2	Rate of transmission from infectious mosquitoes to vaccinated humans
β_3	Rate of transmission from infectious humans to mosquitoes
β_4	Rate of transmission from humans undergoing treatment to mosquitoes
β_5	Rate of transmission from humans not undergoing treatment to mosquitoes
β_6	Rate of transmission from co-infectious humans to mosquitoes
β_7	Rate of transmission from co-infectious humans undergoing treatment to mosquitoes
β_8	Rate of transmission from co-infectious humans not undergoing treatment to mosquitoes
η_1	Rate of transmission from infectious mosquitoes to humans
η_2	Rate of transmission from symptomatic humans to mosquitoes
η_3	Rate of transmission from asymptomatic humans to mosquitoes
η_4	Rate of transmission from humans undergoing treatment to mosquitoes
η_5	Rate of transmission from co-infectious humans to mosquitoes
η_6	Rate of transmission from co-infectious humans undergoing treatment to mosquitoes
η_7	Rate of transmission from co-infectious humans not undergoing treatment to mosquitoes
δ_1	Rate at which exposed humans with malaria parasite become infectious with malaria
δ_2	Rate at which exposed humans with zika virus become infectious with zika disease
δ_3	Rate at which co-infected humans become infectious with both diseases
ε_1	Rate at which infectious humans with malaria accepts to be treated
ε_2	Rate at which infectious humans with malaria refuse to be treated
σ_1	Rate at which co-infectious humans accept to be treated
σ_2	Rate at which co-infectious humans refuse to be treated
χ_1	Proportion of infectious humans with zika that are symptomatic
χ_2	Proportion of infectious humans with zika that are asymptomatic
ϕ_1	Proportion of infectious humans with malaria not undergoing treatment that loses their infectiousness
ϕ_2	Proportion of co-infectious humans not undergoing treatment that loses their malaria infectiousness
γ_1	Rate of recovery of infectious humans with malaria undergoing treatment
γ_2	Rate of recovery of co-infectious humans with malaria and zika virus disease undergoing treatment
ω_1	Rate of recovery of symptomatic infectious humans with zika virus not undergoing treatment
ω_2	Rate of recovery of infectious humans with zika virus undergoing treatment
ω_3	Rate of natural recovery of asymptomatic humans with zika virus
ψ	Rate at which symptomatic infectious humans with zika accepts to be treated
κ_1	Rate of reduction of Anopheles mosquitoes
κ_2	Rate of reduction of Aedes mosquitoes
μ	Natural death of mosquitoes
ν_1	Rate of development of Infectiousness of malaria parasite in Anopheles mosquitoes
ν_2	Rate of development of Infectiousness of zika virus in Aedes mosquitoes
I_{SIT}	Population of Sterile males to control Anopheles population
I^*_{SIT}	Population of Sterile males to control Aedes population

The purpose of establishing this theorem is to show that the proposed system is epidemiological correct in a biological sense [46].

Proof: It can be shown from Eq. (1) that

$$\frac{dS_h}{dt} \geq -(\rho_1 + \rho_2 + \tau_1)S_h.$$

$$\begin{aligned}
\frac{dS_{hu}}{dt} &\geq -(\alpha_1\beta_1 I_{mv} + \alpha_2\eta_1 I_{zv} + \tau_1)S_{hu},\\
\frac{dS_{hv}}{dt} &\geq -(\alpha_1\beta_1\varphi I_{mv} + \alpha_2\eta_1 I_{zv} + \tau_1)S_{hv},\\
&\vdots\\
\frac{dI_{zv}}{dt} &\geq -\mu I_{zv}.
\end{aligned}$$

Solving the differential inequality with the given initial solution gives

$$\begin{aligned} S_h(t) &\geq S_h^0 e^{-(\rho_1+\rho_2+\tau_1)t}, \\ S_{hu}(t) &\geq S_{hu}^0 e^{-(\alpha_1\beta_1 I_{mv}^0+\alpha_2\eta_1 I_{zv}^0+\tau_1)t}, \\ S_{hv}(t) &\geq S_{hv}^0 e^{-(\alpha_1\beta_1\varphi I_{mv}^0+\alpha_2\eta_1 I_{zv}^0+\tau_1)t}, \\ &\vdots \\ I_{zv}(t) &\geq I_{zv}^0 e^{-\mu t}. \end{aligned}$$

Hence, given initial solutions that are individually positive, the solution to the model system remains positive $\forall t > 0$.

Furthermore, the total human and mosquito populations satisfy the differential equations:

$$\begin{aligned} \frac{N_h}{dt} &= \Lambda_h - \tau_1 N_h - \tau_2(I_{hm} + I_{hmT} + I_{hmU}) \\ &\quad - \tau_3(I_{hzS} + I_{hzA} + I_{hzT}) - \tau_4(I_{mz} + I_{mzT} + I_{mzU}) \\ &\leq \Lambda_h - \tau_1 N_h, \\ \frac{N_{mv}}{dt} &= \Lambda_{mv} - \mu I_{mv}, \\ \frac{N_{zv}}{dt} &= \Lambda_{zv} - \mu I_{zv}, \end{aligned} \tag{2}$$

respectively. Integrating the differential equations and solving as $t \to \infty$ gives $0 \leq N_{hm} \leq \frac{\Lambda_h}{\tau_1}$, $0 \leq N_{mv} \leq \frac{\Lambda_{mv}}{\mu}$ and $0 \leq N_{zv} \leq \frac{\Lambda_{zv}}{\mu}$, respectively. This shows that all the solutions of the system are positive and bounded in the region $\Omega = \Omega_1 \times \Omega_2 \times \Omega_3$ and proves that the region, Ω is positively invariant with respect to the flow generated by Eq. (2). Thus, we conclude that the co-infection model Eq. (1) is biologically well-posed and defined since all the state variables remain non-negative for all $t > 0$.

2.4. Disease-free equilibrium of the system

The disease-free equilibrium (DFE) of the model is the steady state solution of Eq. (1) where there is no malaria infection, zika virus infection nor co-infection in the human and mosquito populations. If we denote the DFE by E^0, then we have

$$E^0 = \left(S_h^0, S_{hu}^0, S_{hv}^0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_{mv}}{\mu}, 0, 0, \frac{\Lambda_{zv}}{\mu}, 0, 0\right),$$

where $S_h^0 = \frac{\Lambda_h}{\rho_1+\rho_2+\tau_1}$, $S_{hu}^0 = \frac{\rho_1\Lambda_h}{\tau_1(\rho_1+\rho_2+\tau_1)}$, and $S_{hv}^0 = \frac{\rho_2\Lambda_h}{\tau_1(\rho_1+\rho_2+\tau_1)}$.

3. The basic reproduction number

The basic reproduction number generally given as R_0 is defined as the average number of new cases of an infection caused by one typical infectious individual, in a population consisting of susceptibles only [47–49]. According to Ref. [48], the basic reproduction number, R_0 can be considered as a threshold such that when $R_0 > 1$, the disease persists and when $R_0 < 1$, the disease dies out . The method for obtaining the basic reproduction number is defined in Refs. [50–52]. For our co-infection model, the infectious class, $Y = (E_{hm}, E_{hz}, E_{mz}, I_{hm}, I_{hmT}, I_{hmU}, I_{hzS}, I_{hzA}, I_{hzT}, I_{mz}, I_{mzT}, I_{mzU}, E_{mv}, I_{mv}, E_{zv}, I_{zv})$ consists of those state variables regarded as disease compartments and their corresponding differential equations are:

$$\begin{aligned} \frac{dE_{hm}}{dt} &= \alpha_1\beta_1 I_{mv} S_{hu} + \alpha_1\beta_2\varphi I_{mv} S_{hv} + \phi_1 I_{hmU} + \phi_2 I_{mzU} \\ &\quad - \alpha_2\eta_1 I_{zv} E_{hm} - (\delta_1 + \tau_1)E_{hm}, \\ \frac{dE_{hz}}{dt} &= \alpha_2\eta_1 I_{zv}(S_{hu} + S_{hv}) - \alpha_2\eta_1 I_{zv} E_{hm} \\ &\quad - ((\chi_1 + \chi_2)\delta_2 + \tau_1)E_{hz}. \\ \frac{dE_{mz}}{dt} &= \alpha_1\beta_1 I_{mv} E_{hz} + \alpha_2\eta_1 I_{zv} E_{hm} - (\delta_3 + \tau_1)E_{mz}. \\ \frac{dI_{hm}}{dt} &= \delta_1 E_{hm} - \alpha_2\eta_1 I_{zv} I_{hm} - (\tau_1 + \tau_2 + \varepsilon_1 + \varepsilon_2)I_{hm}, \\ \frac{dI_{hmT}}{dt} &= \varepsilon_1 I_{hm} - (\gamma_1 + \tau_1 + \tau_2)I_{hmT}, \\ \frac{dI_{hmU}}{dt} &= \varepsilon_2 I_{hm} - (\phi_1 + \tau_1 + \tau_2)I_{hmU}, \\ \frac{dI_{hzS}}{dt} &= \delta_2\chi_1 E_{hz} - \alpha_1\beta_1 I_{mv} I_{hzS} - (\tau_1 + \tau_2 + \psi + \omega_1)I_{hzS}, \\ \frac{dI_{hzA}}{dt} &= \delta_2\chi_2 E_{hz} - \alpha_1\beta_1 I_{mv} I_{hzA} - (\tau_1 + \tau_2 + \omega_3)I_{hzA}. \end{aligned}$$

$$\begin{aligned} \frac{dI_{hzT}}{dt} &= \psi I_{hzS} - (\tau_1 + \tau_2 + \omega_2)I_{hzT}. \\ \frac{dI_{mz}}{dt} &= \alpha_1\beta_1 I_{mv}(I_{hzS} + I_{hzA}) + \alpha_2\eta_1 I_{zv} I_{hm} + \delta_3 E_{mz} - \\ &\quad (\tau_1 + \tau_4 + \sigma_1 + \sigma_2)I_{mz}, \\ \frac{dI_{mzT}}{dt} &= \sigma_1 I_{mz} - (\tau_1 + \tau_4 + \gamma_2)I_{mzT}, \\ \frac{dI_{mzU}}{dt} &= \sigma_2 I_{mz} - (\tau_1 + \tau_4 + \phi_2)I_{mzU}, \\ \frac{dE_{mv}}{dt} &= \alpha_1(\beta_3 I_{hm} + \beta_4 I_{hmT} + \beta_5 I_{hmU} + \beta_6 I_{mz} + \beta_7 I_{mzT} \\ &\quad + \beta_8 I_{mzU})S_{mv} - (\nu_1 + \mu)E_{mv}, \\ \frac{dI_{mv}}{dt} &= \nu_1 E_{mv} - \mu I_{mv}, \\ \frac{dE_{zv}}{dt} &= \alpha_2(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT} + \eta_5 I_{mz} + \eta_6 I_{mzT} \\ &\quad + \eta_7 I_{mzU})S_{zv} - (\nu_2 + \mu)E_{zv}, \\ \frac{dI_{zv}}{dt} &= \nu_2 E_{zv} - \mu I_{zv}. \end{aligned}$$

The rate of appearance of new infections in the disease compartments is denoted by $\mathcal{F}$, while the rate of transfer of individuals into and out of the disease compartments by any other means is denoted by $\mathcal{V}$. They are given by:

$$\mathcal{F} = \begin{bmatrix} \alpha_1 I_{mv}(\beta_1 S_{hu} + \beta_2 \varphi S_{hv}) \\ \alpha_2\eta_1 I_{zv}(S_{hu} + S_{hv}) \\ \alpha_1\beta_1 I_{mv}E_{hz} + \alpha_2\eta_1 I_{zv}E_{hm} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ \alpha_1\beta_1 I_{mv}(I_{hzS} + I_{hzA}) + \alpha_2\eta_1 I_{zv} I_{hm} \\ 0 \\ 0 \\ \alpha_1(\beta_3 I_{hm} + \beta_4 I_{hmT} + \beta_5 I_{hmU} + \beta_6 I_{mz} + \beta_7 I_{mzT} + \beta_8 I_{mzU})S_{mv} \\ 0 \\ \alpha_2(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT} + \eta_5 I_{mz} + \eta_6 I_{mzT} + \eta_7 I_{mzU})S_{zv} \\ 0 \end{bmatrix}, \mathcal{V} = \begin{bmatrix} -\phi_1 I_{hmU} - \phi_2 I_{mzU} + (\alpha_2\eta_1 I_{zv} + \delta_1 + \tau_1)E_{hm} \\ (\alpha_2\eta_1 I_{zv} + (\chi_1 + \chi_2)\delta_2 + \tau_1)E_{hz} \\ (\delta_3 + \tau_1)E_{mz} \\ -\delta_1 E_{hm} + (\alpha_2\eta_1 I_{zv} + \tau_1 + \tau_2 + \varepsilon_1 + \varepsilon_2)I_{hm} \\ \delta_3 E_{mz} + (\tau_1 + \tau_4 + \sigma_1 + \sigma_2)I_{mz} \\ -\varepsilon_1 I_{hm} + (\gamma_1 + \tau_1 + \tau_2)I_{hmT} \\ -\varepsilon_2 I_{hm} + (\phi_1 + \tau_1 + \tau_2)I_{hmU} \\ -\delta_2\chi_1 E_{hz} + (\alpha_1\beta_1 I_{mv} + \tau_1 + \tau_2 + \psi + \omega_1)I_{hzS} \\ -\delta_2\chi_2 E_{hz} + (\alpha_1\beta_1 I_{mv} + \tau_1 + \tau_2 + \omega_3)I_{hzA} \\ -\psi I_{hzS} + (\tau_1 + \tau_2 + \omega_2)I_{hzT} \\ -\sigma_1 I_{mz} + (\tau_1 + \tau_4 + \gamma_2)I_{mzT} \\ -\sigma_2 I_{mz} + (\tau_1 + \tau_4 + \phi_2)I_{mzU} \\ (\nu_1 + \mu)E_{mv} \\ -\nu_1 E_{mv} + \mu I_{mv} \\ (\nu_2 + \mu)E_{zv} \\ -\nu_2 E_{zv} + \mu I_{zv} \end{bmatrix}.$$

The Jacobians of $\mathcal{F}$ and $\mathcal{V}$ gotten by differentiating $\mathcal{F}$ and $\mathcal{V}$ with respect to $Y = (E_{hm}, E_{hz}, E_{mz}, I_{hm}, I_{hmT}, I_{hmU}, I_{hzS}, I_{hzA}, I_{hzT}, I_{mz}, I_{mzT}, I_{mzU}, E_{mv}, I_{mv}, E_{zv}, I_{zv})$ are denoted by F and V, respectively, and given as:

$$F = \frac{\partial\mathcal{F}}{\partial Y} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & A_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & C_1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & A_2 & A_3 & A_4 & 0 & 0 & 0 & A_5 & A_6 & A_7 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & C_2 & C_3 & C_4 & C_5 & C_6 & C_7 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \frac{\partial\mathcal{V}}{\partial Y} = \begin{bmatrix} B_1 & 0 & 0 & 0 & 0 & -\phi_1 & 0 & 0 & 0 & 0 & 0 & -\phi_2 & 0 & 0 & 0 & 0 \\ 0 & D_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & B_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\delta_1 & 0 & 0 & B_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\varepsilon_1 & B_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\varepsilon_2 & 0 & B_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -D_6 & 0 & 0 & 0 & 0 & D_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -D_7 & 0 & 0 & 0 & 0 & 0 & D_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\psi & 0 & D_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\delta_3 & 0 & 0 & 0 & 0 & 0 & 0 & B_7 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\sigma_1 & B_8 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\sigma_2 & 0 & B_9 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & B_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\nu_1 & \mu & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & D_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\nu_2 & \mu \end{bmatrix},$$

where $A_1 = \dfrac{\alpha_1\Lambda_h(\beta_1\rho_1 + \beta_2\varphi\rho_2)}{\tau_1(\rho_1 + \rho_2 + \tau_1)}$, $A_2 = \dfrac{\alpha_1\beta_3\Lambda_{mv}}{\mu}$, $A_3 = \dfrac{\alpha_1\beta_4\Lambda_{mv}}{\mu}$, $A_4 = \dfrac{\alpha_1\beta_5\Lambda_{mv}}{\mu}$, $A_5 = \dfrac{\alpha_1\beta_6\Lambda_{mv}}{\mu}$,

$A_6 = \frac{\alpha_1\beta_7\Lambda_{mv}}{\mu}, A_7 = \frac{\alpha_1\beta_8\Lambda_{mv}}{\mu}, B_1 = \delta_1 + \tau_1,$
$B_2 = \tau_1 + \tau_2 + \varepsilon_1 + \varepsilon_2, B_3 = \gamma_1 + \tau_1 + \tau_2, B_4 = \phi_1 + \tau_1 + \tau_2,$
$B_5 = \nu_1 + \mu, B_6 = \delta_3 + \tau_1, B_7 = \tau_1 + \tau_4 + \sigma_1 + \sigma_2,$
$B_8 = \tau_1 + \tau_4 + \omega_2, B_9 = \tau_1 + \tau_4 + \phi_2, C_1 = \frac{\alpha_2\eta_1\Lambda_h}{\tau_1},$
$C_2 = \frac{\alpha_2\eta_2\Lambda_{zv}}{\mu}, \quad C_3 = \frac{\alpha_2\eta_3\Lambda_{zv}}{\mu}, \quad C_4 = \frac{\alpha_2\eta_4\Lambda_{zv}}{\mu},$
$C_5 = \frac{\alpha_2\eta_5\Lambda_{zv}}{\mu}, \quad C_6 = \frac{\alpha_2\eta_6\Lambda_{zv}}{\mu}, \quad C_7 = \frac{\alpha_2\eta_7\Lambda_{zv}}{\mu},$
$D_1 = (\chi_1 + \chi_2)\delta_2 + \tau_1, D_2 = \tau_1 + \tau_3 + \psi + \omega_1, D_3 = \tau_1 + \tau_3 + \omega_3, D_4 = \tau_1 + \tau_3 + \omega_2, D_5 = \nu_2 + \mu, D_6 = \chi_1\delta_2, D_7 = \chi_2\delta_2.$

The basic reproduction number which corresponds to the dominant eigenvalue of FV^{-1} denoted in this work by R_{mz} is given by

$$R_{mz} = max(R_m, R_z), \tag{3}$$

where

$$R_m = \left(\frac{\nu_1\delta_1 A_1(A_2B_4B_3 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)}{\mu B_3B_5(B_1B_2B_4 - \varepsilon_2\delta_1\phi_1)}\right)^{\frac{1}{2}}.$$

and

$$R_z = \left(\frac{C_1\nu_2\delta_2(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)}{\mu D_1D_2D_3D_4D_5}\right)^{\frac{1}{2}}.$$

R_m is the average number of humans and Anopheles mosquitoes that can be infected by one infectious human or mosquito with malaria in a population of susceptible humans or mosquitoes. Similarly, R_z is the average number of humans and Aedes mosquitoes that can be infected by one infectious humans or mosquito with zika virus in a population of susceptible humans or mosquitoes.

4. Stability analysis

4.1. Local stability of the disease-free equilibrium

The DFE is locally asymptotically stable if $R_{mz} < 1$ and unstable if otherwise. The Jacobian, J of the system evaluated at the DFE is given by $J(E^0)$

$$\begin{bmatrix}
-A_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta & 0 & 0 & 0 & 0 & 0 & 0 \\
\rho_1 & -\tau_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -A_9 & 0 & 0 & -A_{10} \\
\rho_2 & 0 & -\tau_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -C_8 & 0 & 0 & -C_9 \\
0 & 0 & 0 & -B_1 & 0 & 0 & 0 & 0 & \phi_1 & 0 & 0 & 0 & 0 & 0 & \phi_2 & 0 & 0 & 0 & A_1 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -D_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & C_1 \\
0 & 0 & 0 & 0 & 0 & -B_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \delta_1 & 0 & 0 & -B_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \varepsilon_1 & -B_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \varepsilon_2 & 0 & -B_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & D_6 & 0 & 0 & 0 & -D_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & D_7 & 0 & 0 & 0 & 0 & -D_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi & 0 & -D_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \delta_3 & 0 & 0 & 0 & 0 & 0 & -B_7 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_1 & -B_8 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \sigma_2 & 0 & -B_9 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_1 & 0 & \omega_1 & \omega_2 & \omega_3 & 0 & \gamma_2 & 0 & -D_8 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -A_2 & -A_3 & -A_4 & 0 & 0 & 0 & -A_5 & -A_6 & -A_7 & 0 & -\mu & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & A_2 & A_3 & A_4 & 0 & 0 & 0 & A_5 & A_6 & A_7 & 0 & 0 & -B_5 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \nu_1 & -\mu & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -C_2 & -C_3 & -C_4 & -C_5 & -C_6 & -C_7 & 0 & 0 & 0 & 0 & -\mu & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & C_2 & C_3 & C_4 & C_5 & C_6 & C_7 & 0 & 0 & 0 & 0 & 0 & -D_5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \nu_2 & -\mu
\end{bmatrix},$$

where $A_8 = \rho_1 + \rho_2 + \tau_1$, $A_9 = \frac{\alpha_1\beta_1\Lambda_h\rho_1}{\tau_1(\rho_1 + \rho_2 + \tau_1)}, D_8 = (\tau_1 + \theta)$
$A_{10} = \frac{\alpha_2\eta_1\Lambda_h\rho_1}{\tau_1(\rho_1 + \rho_2 + \tau_1)}, C_8 = \frac{\alpha_1\beta_2\Lambda_h\varphi\rho_2}{\tau_1(\rho_1 + \rho_2 + \tau_1)},$
$C_9 = \frac{\alpha_2\eta_1\Lambda_h\rho_2}{\tau_1(\rho_1 + \rho_2 + \tau_1)}$ and all the $A_i^{'}s$, $B_i^{'}s$, $C_i^{'}s$ and $D_i^{'}s$ are as described in section 3.

The eigenvalues of the Jacobian matrix, $J(E^0)$ are $(-A_8, -\tau_1, -\tau_1, -D_6, -\mu, -\mu)$ and the roots of the characteristics polynomial:

$$P(\lambda) = Q_7(Q_0\lambda^6 + Q_1\lambda^5 + Q_2\lambda^4 + Q_3\lambda^3 + Q_4\lambda^2 + Q_5\lambda + Q_6)$$
$$(Q_0^*\lambda^6 + Q_1^*\lambda^5 + Q_2^*\lambda^4 + Q_3^*\lambda^3 + Q_4^*\lambda^2 + Q_5^*\lambda + Q_6^*),$$

where

$Q_7 = (\lambda + B_6)(\lambda + B_7)(\lambda + B_8)(\lambda + B_9),$
$Q_0 = 1,$

$$Q_1 = B_1 + B_2 + B_3 + B_4 + B_5 + \mu,$$
$$Q_2 = B_1(\mu + B_2 + B_3 + B_4 + B_5) + B_2(\mu + B_3 + B_4 + B_5) + B_3(\mu + B_4 + B_5) + B_4(B_5 + \mu) + \mu B_5,$$
$$Q_3 = B_1B_2[\mu + B_3 + B_5] + B_3[\mu + B_4 + B_5][B_1 + B_2] + B_4[B_1 + B_2 + B_3][\mu + B_5] + \mu B_5[B_1 + B_2 + B_3 + B_4] + \frac{\nu_1\delta_1A_1(A_2B_3B_4 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)}{\mu B_3B_5R_{0m}^2},$$
$$Q_4 = \mu B_1B_3[B_2 + B_4 + B_5] + B_3B_5[\mu + B_1][B_2 + B_4] + \mu B_4B_5[B_1 + B_2] + B_2B_3B_4[\mu + B_5] + \mu B_1B_2B_5[1 - R_{0m}^2] + \frac{\nu_1\delta_1A_1(A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)}{B_3B_4} + \mu B_1B_2B_5\phi_1\delta_1\varepsilon_2R_{0m}^2 + \frac{\nu_1\delta_1A_1(A_2B_3B_4 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)[\mu + B_3 + B_5]}{\mu B_3B_5R_{0m}^2},$$

$$Q_5 = \frac{\nu_1\delta_1 A_1(A_2B_3B_4 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)[\mu + B_5]}{\mu B_5 R_{0m}^2}$$

$$+ \mu B_3B_4B_5[B_1 + B_2] + \frac{\nu_1\delta_1A_1(A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)[B_4 + B_3]}{B_3B_4}$$

$$+ \mu B_1B_2B_3B_5[1 - R_{0m}^2] + \frac{\mu B_3B_5\phi_1\delta_1\varepsilon_2R_{0m}^2}{B_4}$$

$$+ \frac{\nu_1\delta_1A_1(A_2B_3B_4 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)[1 - R_{0m}^2]}{\mu B_3R_{0m}^2},$$

$$Q_6 = \mu B_1B_2B_3B_4B_5[1 - R_{0m}^2],$$

$$Q_1^* = D_1 + D_2 + D_3 + D_4 + D_5 + \mu,$$

$$Q_2^* = D_1(\mu + D_2 + D_3 + D_4 + D_5) + D_2(\mu + D_3 + D_4 + D_5) + D_3(\mu + D_4 + D_5) + D_4(D_5 + \mu) + \mu D_5,$$

$$Q_3^* = D_1D_2(\mu + D_3 + D_4 + D_5) + D_1D_3(\mu + D_4 + D_5) + D_1D_4(\mu + D_5) + D_2D_3(\mu + D_4 + D_5) + \mu D_1D_5 + D_3D_4(D_5 + \mu) + D_2D_4(D_5 + \mu) + \mu D_5(D_2 + D_3 + D_4),$$

$$Q_4^* = D_1D_2D_3(\mu + D_4 + D_5) + D_1D_2D_4(\mu + D_5) + \mu D_2D_3D_5 + \mu D_4D_5(D_1 + D_2 + D_3) + D_2D_3D_4(\mu + D_5) + D_1D_3D_4(\mu + D_5) + \frac{C_1\nu_2\delta_2C_4\chi_1\psi(D_2 + D_3)}{D_2D_4} + \frac{C_1\nu_2\delta_2(C_3\chi_2D_2D_2 + C_2\chi_1D_3D_3)}{D_2D_3} + \mu D_1D_2D_5[1 - R_z^2] + \mu D_1D_3D_5[1 - R_z^2],$$

$$Q_5^* = D_2D_3D_4[\mu D_1 + \mu D_5 + D_1D_5] + \frac{C_1\nu_2\delta_2\chi_1D_3(C_2D_4 + C_4\psi)}{D_2} + \frac{C_1\nu_2\delta_2(C_3\chi_2D_2D_4D_4 + C_4\chi_1\phi D_3D_3}{D_3D_4D_5} + \mu D_1D_3D_4D_5[1 - R_z^2] + \mu D_1D_2D_4D_5[1 - R_z^2] + \mu D_1D_2D_3D_5[1 - R_z^2],$$

$$Q_6^* = \mu D_1D_2D_3D_4D_5[1 - R_z^2].$$

According to Refs. [48, 53], Routh-Hurwitz criterion for stability states that all roots of the characteristic polynomial $P(\lambda)$ will have negative real parts if $Q_7 > 0, Q_6 > 0, Q_5 > 0, Q_4 > 0, Q_3 > 0, Q_2 > 0, Q_1 > 0, Q_0 > 0, Q_6^* > 0, Q_5^* > 0, Q_4^* > 0, Q_3^* > 0, Q_2^* > 0, Q_1^* > 0, Q_0^* > 0$ and the following are satisfied; $\frac{Q_1}{Q_0} > \frac{Q_3}{Q_2}, \frac{Q_4}{Q_3} > \frac{Q_6}{Q_5}, \frac{Q_2}{Q_0} > \frac{Q_6}{Q_4}, \frac{Q_1^*}{Q_0^*} > \frac{Q_3^*}{Q_2^*}, \frac{Q_4^*}{Q_3^*} > \frac{Q_6^*}{Q_5^*}$ and $\frac{Q_2^*}{Q_0^*} > \frac{Q_6^*}{Q_4^*}$. From Q_7, we have four more eigenvalues $-B_6, -B_7, -B_8$, and $-B_9$ which are all negative. The remaining twelve eigenvalues will be negative if Routh-Hurwitz criterion for stability is satisfied. The stability criterion will be satisfied if $R_m < 1$ and $R_z < 1$ respectively. Thus, the DFE will be locally asymptotically stable if $R_m < 1$ and $R_z < 1$.

Epidemiologically, this signifies that an influx of a small number of malaria and zika virus infected humans into the population will not lead to an outbreak if the reproduction number is less than one. This result however depends on the initial sizes of the infected individuals.

4.2. Global stability of the disease-free equilibrium

We employed the method of Castillo-Chavez [54] to check if the DFE is globally asymptotically stable (GAS). Consider the system of differential equations

$$\frac{dX}{dt} = F_1(X, 0), \tag{4}$$

$$\frac{dY}{dt} = F_2(X, Y), F_2(X, 0) = 0, \tag{5}$$

where Eq. (4) is the system of differential equations, satisfied by non-disease classes such that $X = (S_h, S_{hu}, S_{hv}, R_h, S_{mv}, S_{zv})$ and Eq. (5) is the system of differential equations satisfied by the disease classes so that $Y = (E_{hm}, E_{hz}, E_{mz}, I_{hm}, I_{hmT}, I_{hmU}, I_{hzS}, I_{hzA}, I_{hzT}, I_{mz}, I_{mzT}, I_{mzU}, E_{mv}, I_{mv}, E_{zv}, I_{zv})$. The DFE, E^0 is GAS if Eq. (4) is GAS, and if in Eq. (5), $BX_2 - F_2(X, Y) \geq 0$, where B is the Jacobian matrix of $F_2(X, Y)$, evaluated at E^0.

Solving the resulting equations of Eq. (4) at the DFE gives

$$S_h = \frac{\Lambda_h}{\rho_1 + \rho_2 + \tau_1} + \frac{\theta R_h^0 e^{-(\tau_1+\theta)t}}{(\rho_1 + \rho_2 - \theta)} + c_1e^{-(\rho_1+\rho_2+\tau_1)t},$$

$$S_{hu} = \frac{\rho_1\Lambda_h}{\tau_1(\rho_1 + \rho_2 + \tau_1)} - \frac{\rho_1R_h^0e^{-(\tau_1+\theta)t}}{\rho_1 + \rho_2 - \theta} - \frac{\rho_1c_1e^{(\rho_1+\rho_2+\tau_1)t}}{(\rho_1 + \rho_2)} + c_2e^{-\tau_1t},$$

$$S_{hu} = \frac{\rho_2\Lambda_h}{\tau_1(\rho_1 + \rho_2 + \tau_1)} - \frac{\rho_2R_h^0e^{-(\tau_1+\theta)t}}{\rho_1 + \rho_2 - \theta} - \frac{\rho_2c_1e^{(\rho_1+\rho_2+\tau_1)t}}{(\rho_1 + \rho_2)} + c_3e^{-\tau_1t},$$

$$R_h = R_h^0e^{-(\tau_1+\theta)t},$$

$$S_{mv} = \frac{\Lambda_{mv}}{\mu} + \left(S_{mv}^0 - \frac{\Lambda_{mv}}{\mu}\right)e^{-\mu t},$$

$$S_{zv} = \frac{\Lambda_{zv}}{\mu} + \left(S_{zv}^0 - \frac{\Lambda_{zv}}{\mu}\right)e^{-\mu t}.$$

As $t \to \infty$, we will have $S_h \to \frac{\Lambda_h}{\rho_1 + \rho_2 + \tau_1}$, $S_{hu} \to \frac{\rho_1\Lambda_h}{\tau_1(\rho_1 + \rho_2 + \tau_1)}$, $S_{hv} \to \frac{\rho_2\Lambda_h}{\tau_1(\rho_1 + \rho_2 + \tau_1)}$, $R_h \to 0$, $S_{mv} \to \frac{\Lambda_{mv}}{\mu}$ and $S_{zv} \to \frac{\Lambda_{zv}}{\mu}$ respectively which corresponds to the values of these state variables at the DFE. Thus, Eq. (4) is GAS. Also, the matrix B corresponds to our Jacobian submatrix, $J_1(E^0)$. Hence, the expression $BX_2 - F_2(X, Y)$ becomes

$$BX_2 - F_2(X,Y) = \begin{pmatrix} \alpha_1\beta_2\varphi\left(\frac{\rho_2\Lambda_h}{\tau_1(\rho_1+\rho_2+\tau_1)} - S_{hv}\right)I_{mv} + \alpha_1\beta_1\left(\frac{\rho_1\Lambda_h}{\tau_1(\rho_1+\rho_2+\tau_1)} - S_{hu}\right)I_{mv} + \alpha_2\eta_1 I_{zv}E_{hm} \\ \alpha_2\eta_1\left(\frac{\rho_2\Lambda_h}{\tau_1(\rho_1+\rho_2+\tau_1)} - S_{hv}\right)I_{zv} + \alpha_2\eta_1\left(\frac{\rho_1\Lambda_h}{\tau_1(\rho_1+\rho_2+\tau_1)} - S_{hu}\right)I_{zv} + \alpha_1\beta_1 I_{mv}E_{hz} \\ -\alpha_1\beta_1 I_{mv}E_{hz} - \alpha_2\eta_1 I_{zv}E_{hm} \\ \alpha_2\eta_1 I_{zv}I_{hm} \\ 0 \\ 0 \\ \alpha_1\beta_1 I_{mv}I_{hzS} \\ \alpha_1\beta_1 I_{mv}I_{hzA} \\ 0 \\ -\alpha_1\beta_1 I_{mv}(I_{hzS} + I_{hzA}) - \alpha_2\eta_1 I_{zv}I_{hm} \\ 0 \\ 0 \\ \alpha_1\left(\frac{\Lambda_{mv}}{\mu} - S_{mv}\right)(\beta_3 I_{hm} + \beta_4 I_{hmT} + \beta_5 I_{hmU} + \beta_6 I_{mz} + \beta_7 I_{mzT} + \beta_8 I_{mzU}) \\ 0 \\ \alpha_2\left(\frac{\Lambda_{zv}}{\mu} - S_{zv}\right)(\eta_2 I_{hzS} + \eta_3 I_{hzA} + \eta_4 I_{hzT} + \eta_5 I_{mz} + \eta_6 I_{mzT} + \eta_7 I_{mzU}) \\ 0 \end{pmatrix}.$$

It is obvious that $BX_2 - G(X_1, X_2) < 0$, due to the entries in the third and tenth rows respectively. Hence, the DFE is not globally stable. This guarantees that there will be at least two endemic equilibrium point (EEP) in the co-infection model. This means that bringing the reproduction number to less than one is not enough to control the co-infection of malaria and zika virus disease hence the necessity for a better control measure which SIT offers.

5. Impart of malaria on zika virus disease and vice versa

The impact of malaria on the spread zika virus disease, and vice versa can be investigated by expressing their basic reproduction numbers a function of each other, and taking partial derivatives with respect to the reproduction numbers. From Eq. (3), obtaining an expression from both reproduction numbers in terms of μ gives

$$\mu = \frac{\nu_1\delta_1 A_1(A_2B_4B_3 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)}{B_3B_5(B_1B_2B_4 - \varepsilon_2\delta_1\phi_1)R_m^2} = \frac{C_1\nu_2\delta_2(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)}{D_1D_2D_3D_4D_5R_z^2}.$$

We can write

$$R_z^2 = \Phi_{mz}R_m^2,$$

where

$$\Phi_{mz} = \frac{C_1\nu_2\delta_2(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)B_3B_5(B_1B_2B_4 - \varepsilon_2\delta_1\phi_1)}{D_1D_2D_3D_4D_5\nu_1\delta_1A_1(A_2B_4B_3 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)}.$$

Partial differentiation of R_z with respect to R_m shows that Φ_{mz} is the constant rate at which zika virus disease affects the spread of malaria. Specifically, $\frac{\partial R_z}{\partial R_m} = \Phi_{mz}$.

On the other hand,

$$\Phi_{mz}^{-1} = \frac{D_1D_2D_3D_4D_5\nu_1\delta_1A_1(A_2B_4B_3 + A_3\varepsilon_1B_4 + A_4\varepsilon_2B_3)}{C_1\nu_2\delta_2(C_2\chi_1D_3D_4 + C_3\chi_2D_2D_4 + C_4\chi_1\psi D_3)B_3B_5(B_1B_2B_4 - \varepsilon_2\delta_1\phi_1)}$$

is the constant rate at which malaria affects the spread of zika virus disease. Since $\Phi_{mz} > 0$, it means that malaria has a positive impact on the spread of zika virus disease, and vice versa. However, the degree of impact depends on whether $\Phi_{mz} > 1$ or $\Phi_{mz} < 1$.

This result means that in any environment where Anopheles and Aedes mosquitoes coexist and malaria co-circulate with zika virus disease, an increase in the population of one mosquito will lead to an increase in the population of the other mosquito as both mosquitoes are affected by the same environmental factors. Thus, an increase in the spread of malaria will definitely mean an increase in the spread of zika virus disease in the absence of any intervention strategy for both diseases.

6. Sensitivity analysis of the co-infection model

Here, we compute the sensitivity indices of the parameters in the co-infection reproduction number using the normalized forward sensitivity index [25] of R_{mz} that depends on the parameter q which is given by

$$S_q^{R_{mz}} = \frac{\partial R_{mz}}{\partial q} \times \frac{q}{R_{mz}}. \tag{6}$$

The sensitivity indices shown in Tables 2 and 3 helps us ascertain the parameters that affects the value of R_{mz} more for effective intervention measure in controlling the co-infection.

The endemicity of the co-infection is increased by the parameters with positive sensitivity index and decreased by the parameters with negative sensitivity index. The parameters with negative values need to be increased in order to control the co-infection while those with positive values need to be reduced. This means that those parameters with positive values needs much attention as controlling them will significantly reduce the spread of the disease. The parameter with the highest impact

Table 2. Sensitivity indices for R_m.

Parameter	Values	Sensitivity index
Λ_h	100	0.5
Λ_{mv}	100	0.5
α_1	0.4	1
β_1	0.034	0.4899
β_2	0.013	0.0010
β_3	0.0044	0.1083
β_4	0.0022	0.1341
β_5	0.0044	0.2576
τ_1	0.00004	-0.5006
τ_2	0.00032338	-0.0017
δ_1	0.0833	0.0004
ρ_1	0.65	0.1405
ρ_2	0.28	-0.1405
ν_1	0.1	0.1787
φ	0.0125	0.0101
μ	0.0556	-1.1787
ε_1	0.62	-0.3648
ε_2	0.31	0.2568
γ_1	0.25	-0.1339
ϕ_1	0.13	-0.2562

Table 3. Sensitivity indices for R_z.

Parameter	Values	Sensitivity index
Λ_h	30	0.5
Λ_{zv}	100	0.5
α_2	0.4	1
η_1	0.0009	0.5
η_2	0.07	0.0233
η_3	0.07	0.3918
η_4	0.05	0.0848
τ_1	0.00004	-0.5.003
τ_3	0.0003	-0.0012
δ_2	0.125	0.0002
χ_1	0.31	-0.0584
χ_2	0.62	0.0586
ν_2	0.1	0.1787
ψ	0.85	-0.0077
μ	0.0556	-1.1787
ω_1	0.1429	-0.0156
ω_2	0.1667	-0.0847
ω_3	0.118	-0.3907

on the spread of the disease is the contact rate of humans with the mosquitoes denoted by α_1 and α_2 with sensitivity index of 1 respectively. Thus, the co-infection or the individual infections will be successfully controlled by ensuring that the mosquitoes have minimal contacts with humans. The recruitment rates for humans Λ_h, anopheles mosquitoes, Λ_{mv} and aedes mosquitoes, Λ_{zv} respectively have sensitive indices of 0.5 which showed that controlling the co-infection will require humans to avoid areas where these mosquitoes are prevalent and efforts should be made to reduce the recruitment of more mosquitoes into human environment.

We could also see that proportion of humans not vaccinated, ρ_1 and proportion not treated for malaria, ε_2 also increase the endemicity of the co-infection. Also, the more asymptomatic cases for zika virus infection is recorded, the more the co-infection persists as those proportion will remain undetected and continue to spread the virus. The transmission rates of both diseases from humans to mosquitoes and vice versa also affects the endemicity of the co-infection. The parameters with negative values do not increase the persistence of the co-infection. The parameter with the least negative value is μ which represents natural death rate of mosquitoes. This means that to control the disease, efforts should also be focused on ensuring that the mosquitoes die more often as this will also reduce the contact rate of the mosquitoes with humans.

7. Numerical experiment

In this section, we perform simulation analysis on the co-infection model using the following values; $S_h = 500, S_{hu} = 320, S_{hv} = 19, E_{hm} = 48, E_{hz} = 30, E_{hmz} = 20, I_{hm} = 35, I_{hmU} = 8, I_{hmT} = 21, I_{hzS} = 20, I_{hzA} = 40, I_{hzT} = 15, I_{hmz} = 15, I_{hmzU} = 3, I_{hmzT} = 9, R_h = 20, S_{mv} = 500, E_{mv} = 60, I_{mv} = 50, S_{zv} = 500, E_{zv} = 25, I_{zv} = 10$. The results of the numerical experiments are shown in Figures 2 - 31. The Figures show the behaviour of the state variables under various controls.

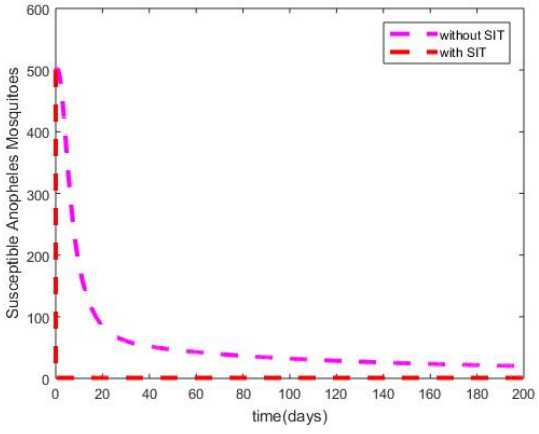

Figure 2. Susceptible anopheles mosquitoes.

7.1. Effects of SIT on the co-infection model

Using the parameters in Table 4, we investigate how the application of SIT will affect some of the various population studied especially the mosquitoes populations. This analysis is done by varying the level of application of SIT while keeping the level of treatment and vaccination constant for all cases.

Figures 2-4 show the trajectories of the Anopheles mosquitoes under the application of SIT. The susceptible mosquito population shown in Figure 2 describes how SIT successfully reduces the number of Anopheles mosquitoes with time. That reduction leads to a corresponding reduction in the populations of the exposed and infectious anopheles mosquitoes as shown in Figure 3 and Figure 4, respectively.

Table 4. Parameters, values and sources.

Parameters	Values	Sources
Λ_h	100	Assumed
Λ_{mv}	100	Assumed
Λ_{zv}	100	Assumed
φ	0.0125	Assumed
θ	0.0146	[2]
ρ_1	0.65	Assumed
ρ_2	0.28	Assumed
τ_1	0.00004	[1, 24, 28]
τ_2	0.00032338	[26]
τ_3	0.0003	Assumed
τ_4	0.0006454	Assumed
α_1	0.4	[2]
α_2	0.1	Assumed
β_1	0.034	[38]
β_2	0.013	Assumed
β_3	0.0044	[24, 28]
β_4	0.0022	Assumed
β_5	0.0044	[24, 28]
β_6	0.0022	Assumed
β_7	0.0044	Assumed
β_8	0.0022	Assumed
η_1	0.0009	[7]
η_2	0.07	[7]
η_3	0.07	[7]
η_4	0.05	Assumed
η_5	0.03	Assuned
η_6	0.02	Assumed
η_7	0.03	Assumed
δ_1	0.0833	[2, 5, 11]
δ_2	0.125	[7, 8]
δ_3	0.0833	Assumed
ε_1	0.62	Assumed
ε_2	0.31	Assumed
σ_1	0.72	Assumed
σ_2	0.18	Assumed
χ_1	0.31	Assumed
χ_2	0.62	[11]
ϕ_1	0.13	Assumed
ϕ_2	0.1	Assumed
γ_1	0.25	[2]
γ_2	0.111	Assumed
ω_1	0.1429	[36]
ω_2	0.1667	[7]
ω_3	0.118	[55]
ψ	0.85	[8]
κ_1	0.25	Assumed
κ_2	0.25	Assumed
μ	0.0556	[7]
ν_1	0.1	[2]
ν_2	0.1	[8]
I_{SIT}/I^*_{SIT}	500	Assumed

Similarly, Figures 5-7 show the trajectories of the Aedes

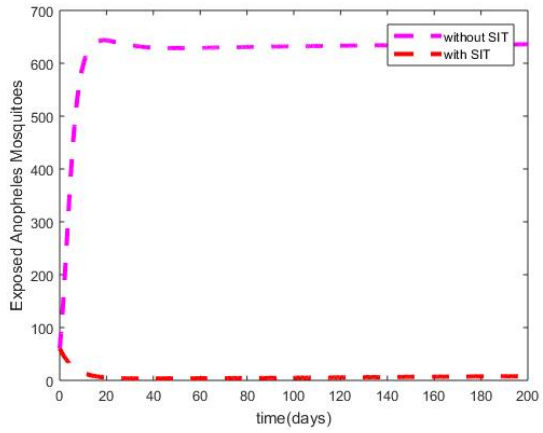

Figure 3. Exposed anopheles mosquitoes.

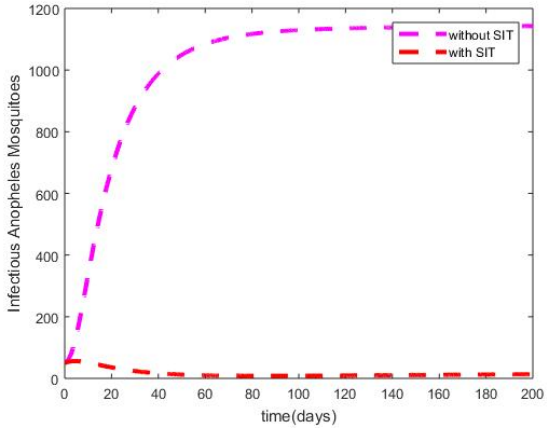

Figure 4. Infectious anopheles mosquitoes.

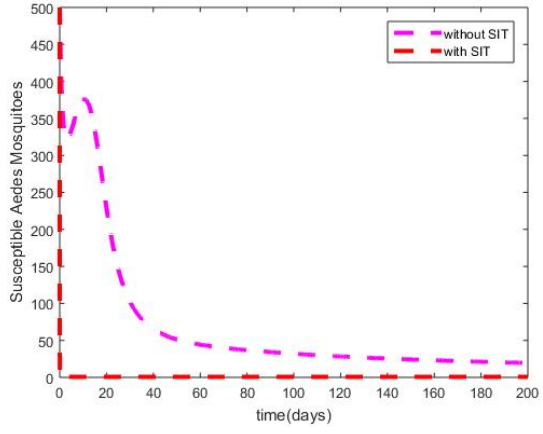

Figure 5. Susceptible aedes mosquitoes.

mosquitoes under the application of SIT. The susceptible mosquito population shown in Figure 5 describes how SIT successfully reduces the number of Aedes mosquitoes. This leads to a corresponding reduction in the populations of the exposed and infectious aedes mosquitoes as shown in Figure 6 and Fig-

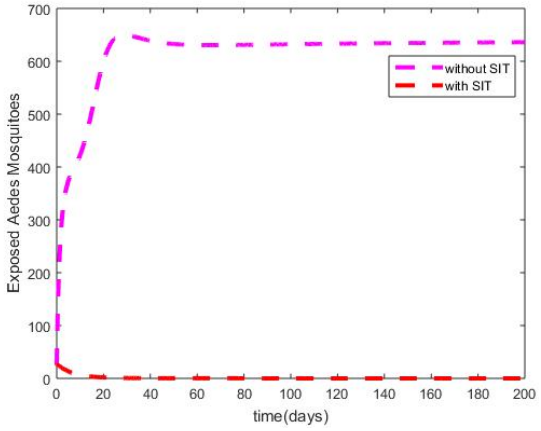

Figure 6. Exposed aedes mosquitoes.

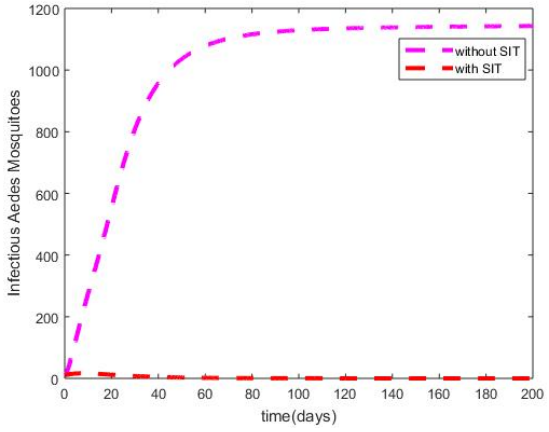

Figure 7. Infectious aedes mosquitoes.

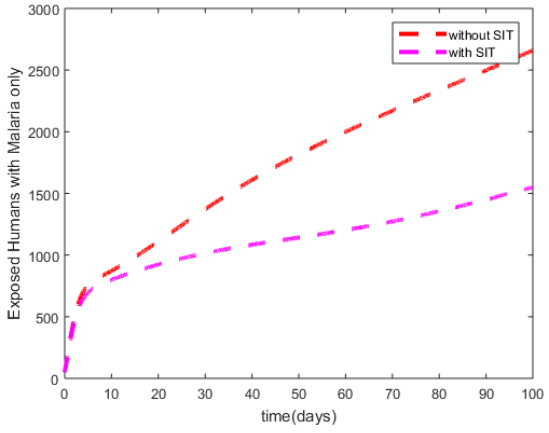

Figure 8. Exposed humans with malaria only.

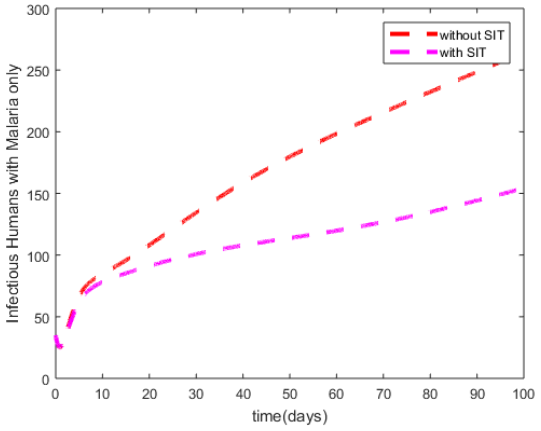

Figure 9. Infectious humans with malaria only.

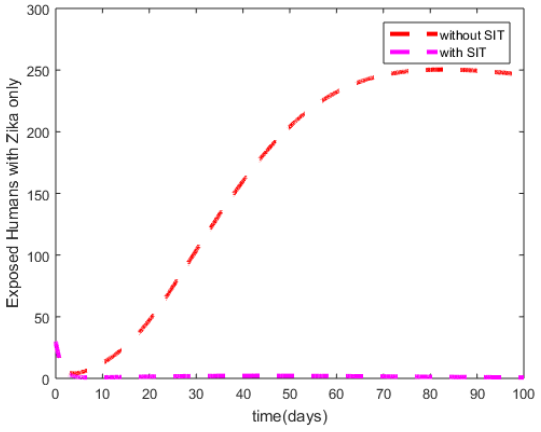

Figure 10. Exposed humans with Zika only.

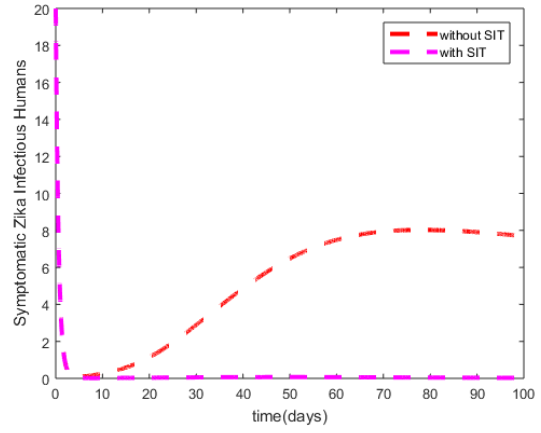

Figure 11. Symptomatic humans with Zika only.

ure 7, respectively.

In Figures 8 and 9, we showed the effects of SIT on the exposed and infectious human population with malaria only. Both populations reduce greatly under SIT because of the reduction of the populations of the Anopheles mosquitoes which are vectors responsible for transmitting malaria.

In Figures 10-12, we showed the effects of SIT on the exposed and infectious human population with zika virus disease only. The three populations reduce greatly under SIT because of the reduction of the populations of the Aedes mosquitoes

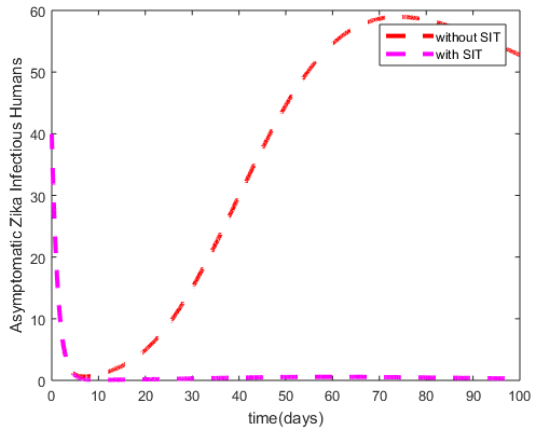

Figure 12. Asymptomatic humans with Zika only.

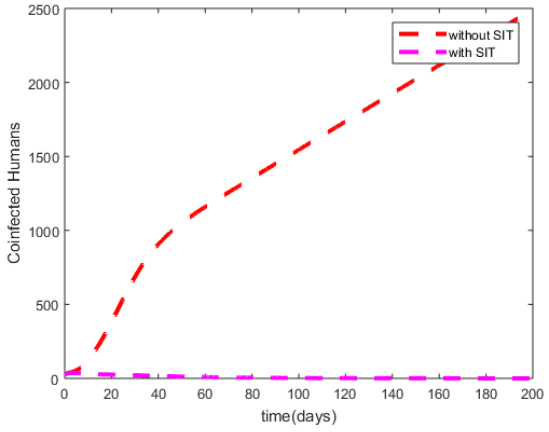

Figure 13. Co-infected humans.

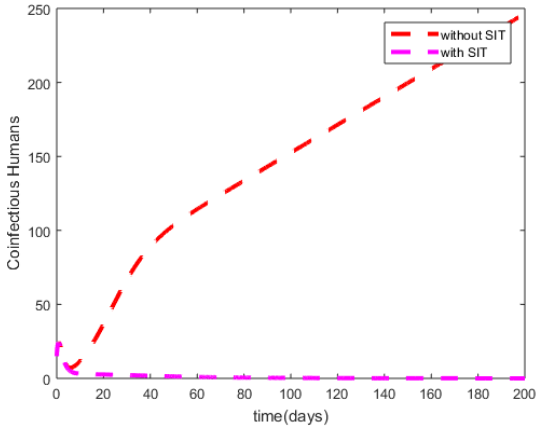

Figure 14. Co-infectious humans.

which are vectors responsible for transmitting zika virus disease.

In Figures 13 and 14, we showed how application of SIT affects the co-infected and co-infectious human population respectively. Both populations reduce sharply under SIT due to the fact that SIT as demonstrated helped to reduce the populations of the mosquitoes which are vectors responsible for transmitting malaria and zika virus disease. We could also see that the reductions in the co-infected and co-infectious humans are a direct consequence of the reductions in the diseased classes of the individual diseases.

Thus, the existence of the co-infection is directly proportional to the prevalence of these mosquitoes. Thus, successful control of the individual or co-infection disease is largely dependent on controlling the mosquito populations and SIT offers a unique way of doing that.

7.2. Effects of vaccination on the co-infection model

In this section, we will investigate how vaccination will affect some of the various population studied especially the human populations using the parameters in Table 4. This analysis is done by varying the level of vaccination while keeping the level of treatment and application of SIT constant for all cases.

The susceptible humans shown in Figure 15 is seen to reduce gradually as the rate of vaccination increases. Thus, the more people are vaccinated, the more the number of humans at risk of being infected with malaria reduces. Similarly, the exposed and infectious humans with malaria only shown in Figures 16 and 17 also experienced a decline in population due to the increased level of vaccination. This is so because when more humans are protected from malaria through vaccination, it reduces the rate at which they become infected with the disease or become infectious.

The co-infected classes are shown in Figures 18 and 19. The trajectories showed that vaccination has no much effect in reducing the population of the co-infected classes since the vaccination is targeted only against malaria and not zika. Due to the fact that vaccination wanes with time, the co-infected classes experienced little increase as people lose their immunity against malaria and become infected. The effect of vaccination in the co-infected classes is seen as it prevented drastic increase in the classes but kept it negligible. In Figure 20, we see that the number of persons recovering from the system continues to reduce as the level of vaccination continues to increase. This is because vaccination protects humans from contracting malaria and thus reduces the overall infectious classes in the system, hence the recovered population.

Vaccination of humans against malaria also affects the anopheles mosquito population as shown in Figures 21 and 22. As more people are protected against malaria, it reduces the possibility of the anopheles mosquitoes contracting the disease, hence reduces also their ability to re-infect humans. The trajectories in the mosquito populations all emanate from the same point and flows at the same pace to a point before the diverges. This is because we used same initial data and kept the effect of SIT and treatment constant for all cases. The point of divergence is where the effect of vaccination sets in.

Thus, vaccination has been shown to be a good strategy to control infectious diseases and when employed together with other control measures such as treatment and vector control measures such as use of SIT in this case will produce a better result.

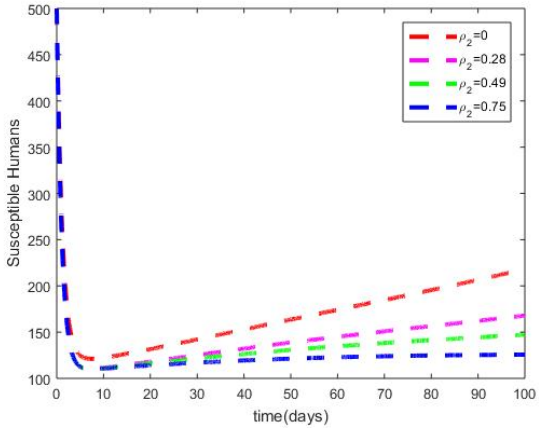

Figure 15. Susceptible humans.

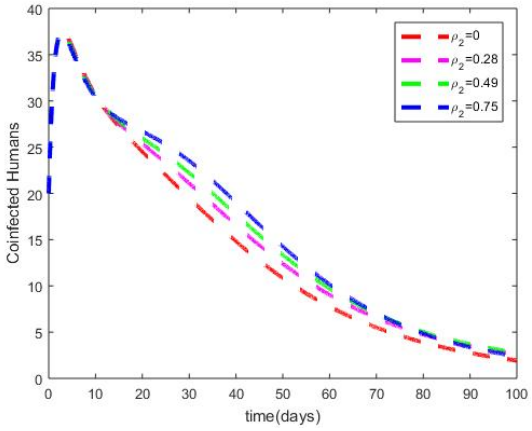

Figure 18. co-infected humans.

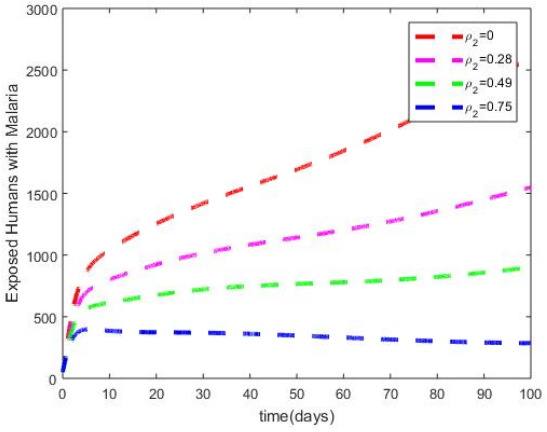

Figure 16. Exposed humans with malaria only.

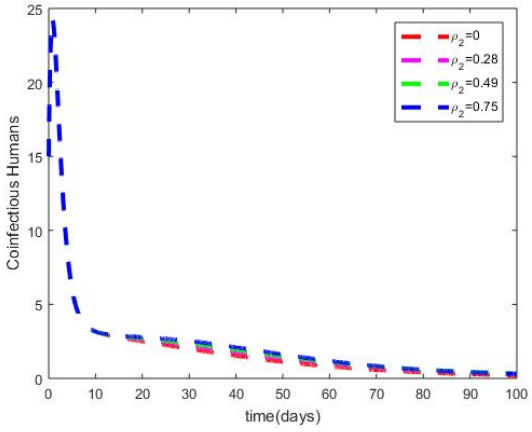

Figure 19. co-infectious humans.

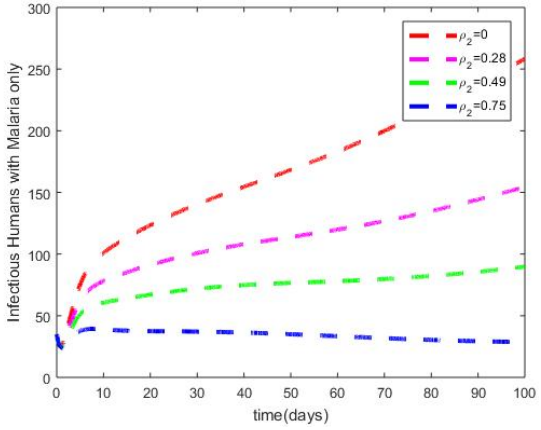

Figure 17. Infectious humans with malaria only.

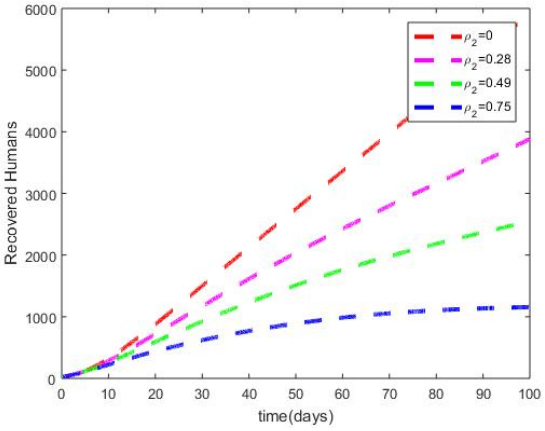

Figure 20. Recovered humans.

7.3. Effects of treatment on the co-infection model

Here, we also used the parameters in Table 4 to investigate how treatment of humans infectious with the individual diseases or co-infectious with both will affect the system especially the infectious classes. Just like in the first two numerical analysis, the effect of treatment is investigated by varying the level of treatment while keeping the level of vaccination and application of SIT constant for all cases.

The exposed and infectious humans with malaria only shown in Figures 23 and 24 experienced a significant decrease

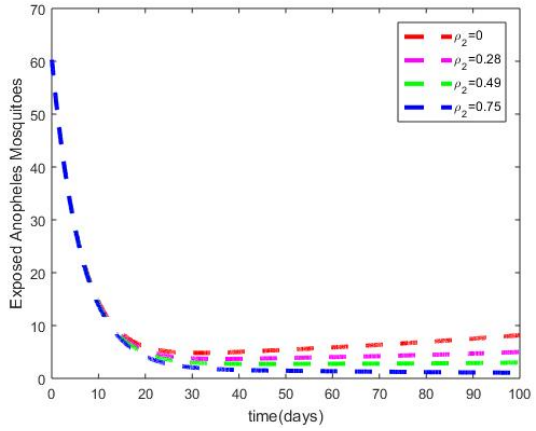

Figure 21. Exposed Anopheles mosquitoes.

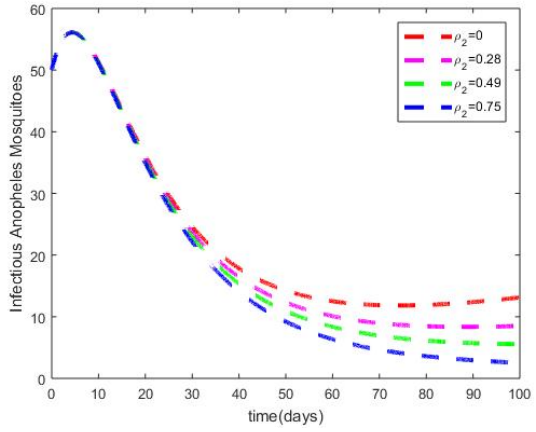

Figure 22. Infectious Anopheles mosquitoes.

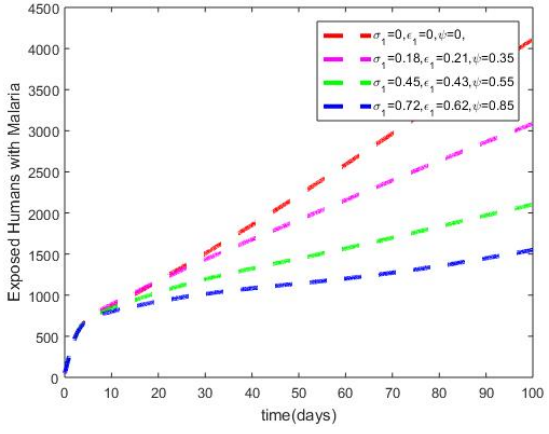

Figure 23. Exposed humans with malaria only.

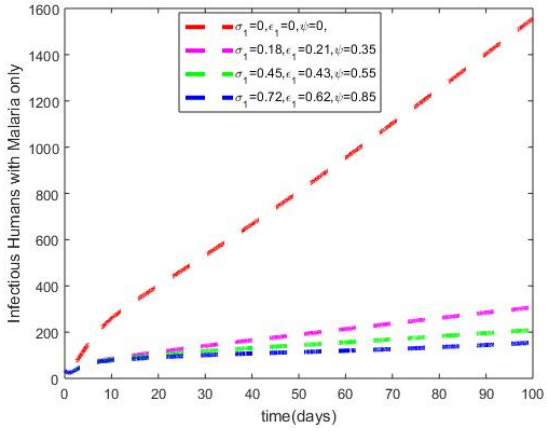

Figure 24. Infectious humans with malaria only.

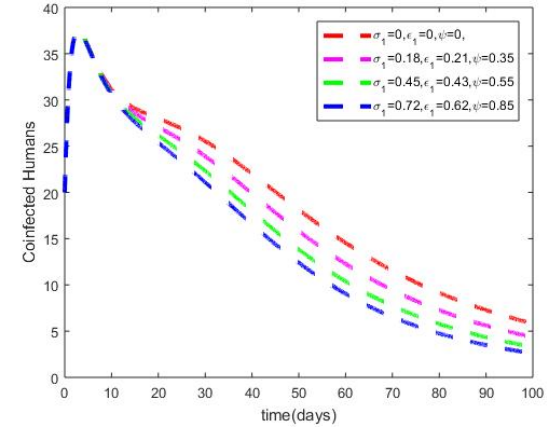

Figure 25. co-infected humans.

in population as more people in the infectious classes with the disease are treated. We could see that the higher the level of treatment, the quicker and higher the level of recovery and thus reducing the time spent in the infectious class as well as the number of persons in the infectious classes. The effect of treatment on the co-infected, co-infectious and recovered humans are shown in Figures 25, 26 and 27, respectively. The result showed that while the recovered humans increased significantly under treatment, the co-infected and co-infectious populations decreased slightly under treatment. The increase in the recovered class is because treatment increases the rate of recovery and as the level of treatment increases, more infectious human recover faster. The co-infected class experienced more decrease under treatment than the co-infectious class because the co-infected class receive entries only from the exposed classes of individual infections which treatment had significantly reduced. But, the co-infectious class receives entries from both the co-infected class and the infectious classes of each individual disease. The effect of treatment on the mosquito populations underlines how important treatment is in the control of infectious diseases. In Figures 28 and 29, the populations of the exposed and infectious female anopheles mosquitoes experienced sharp decrease under treatment. We could see that as more infectious people with malaria are treated, the population of the Anopheles mosquitoes exposed or infectious with malaria continues to drop. This is because treatment speeds up level of recovery and reduces the time infectious humans stay

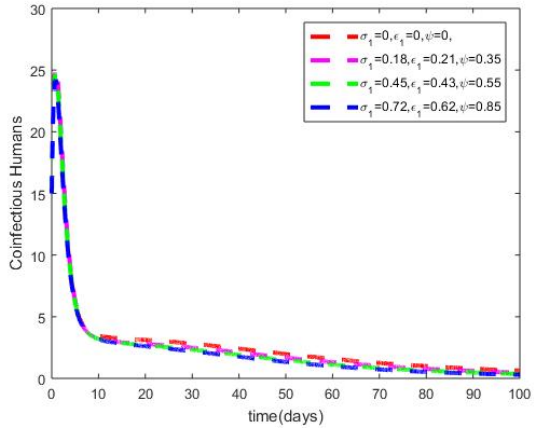

Figure 26. co-infectious humans.

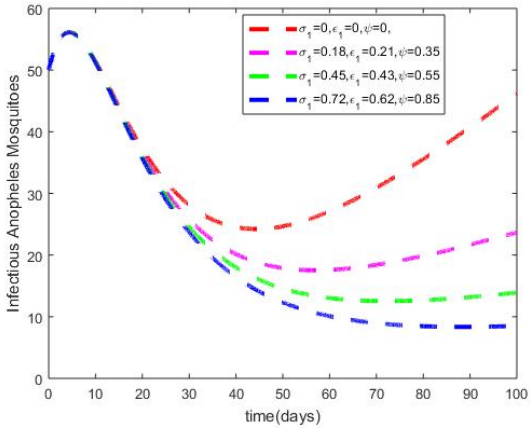

Figure 29. Infectious anopheles mosquitoes.

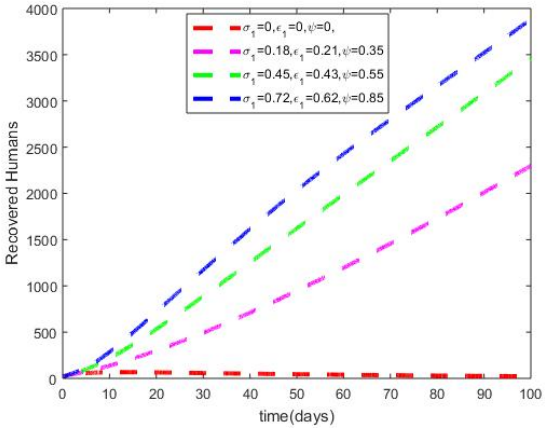

Figure 27. Recovered humans.

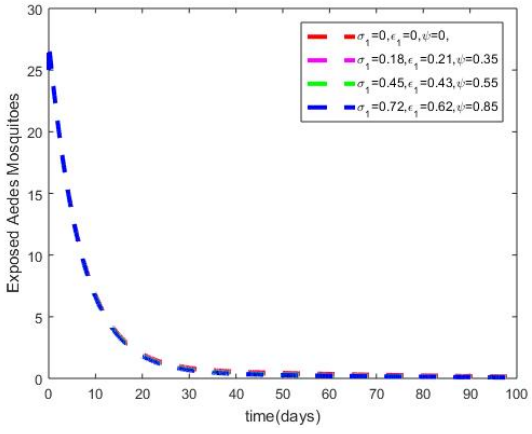

Figure 30. Exposed aedes mosquitoes.

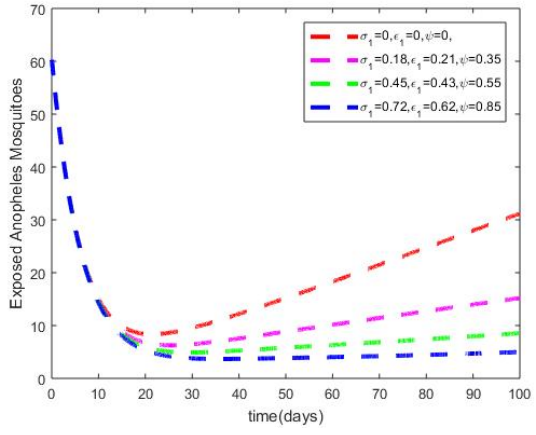

Figure 28. Exposed anopheles mosquitoes.

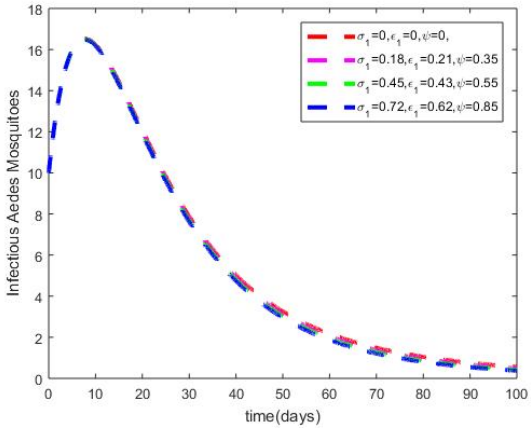

Figure 31. Infectious aedes mosquitoes.

in the infectious classes. Consequently, there will be fewer infectious humans to infect the mosquitoes.

Unlike in the Anopheles mosquito population, treatment has no much effect on the population of the Aedes mosquitoes shown in Figures 30 and 31. The occurrence of more asymptomatic cases of Zika virus disease guarantees a steady presence of infectious humans that will always infect the Aedes mosquitoes. Thus, treatment will have impact in the control of Zika virus disease if there are fewer cases of asymptomatic patients.

8. Conclusion

This work presents a new mathematical model to study the transmission dynamics of the co-infection of malaria and zika virus disease which incorporates vaccination of humans against malaria, treatment of infectious humans as well as the use of SIT to control the vectors. The analysis shows that the DFE of the model is locally asymptotically stable when the basic reproduction number, R_{mz} is less than one and unstable if otherwise but not globally asymptotically stable. The global instability of the disease-free equilibrium shows that there is existence of possible backward bifurcation in the model and the existence of at least two endemic equilibrium points. The analysis of the model shows that the spread of malaria has a positive impact on the spread of zika virus disease. This means that in any environment where the Anopheles mosquitoes coexist with Aedes mosquitoes, the co-circulation of malaria and zika virus is possible and since both mosquitoes are affected by the same environmental factors, an increase in the population of one of the mosquitoes will cause an increase in the other too. The effects of treatment and vaccination on the human population were shown as well as the effect of SIT on the mosquito population. The use of vaccination as a protective measure against malaria was effective in reducing the transmission of malaria to humans. Treatment of infectious humans did not only reduce the number of infectious humans but also the number of infectious mosquitoes which is a significant contribution of this work. As more people are treated, it reduces the infectious population of humans that could infect the mosquitoes, thus reducing the number of mosquitoes in the infected class. The application of SIT was seen to effectively reduce the population of the mosquitoes and thus reduce the spread of the disease. Thus, we conclude that if the three control measures are effectively employed, the co-infection of both diseases vis-a-vis the individual diseases will be reduced drastically and controlled. We also advocate that for total protection of humans against malaria, efforts should be made by health professionals and the global world to support developing vaccines that will grant total and effective immunity against the disease. Furthermore, campaigns should continue on the importance of early treatment of malaria and zika virus diseases as this work highlighted its significant role in controllong the spread of the diseases. People should also be educaated and assisted to reduce contacts with mosquitoes by protecting themselves as our sensitivity analysis shows that contact rate of humans with mosquitoes had the greatest impart on whether the disease will persist or die out. Further research in this regard should consider the optimal control analysis, comparison of effectiveness of SIT and other mosquito control measures, possibility of incorporating biological and physical measures in controlling mosquitoes simultaneously as well as the cost effective analysis of the different controls.

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