



Stability Analysis of the Disease Free Equilibrium of Malaria, Dengue and Typhoid Triple Infection Model

T. J. Oluwafemi^{1*}, E. Azuaba² and Y. M. Kura³

¹Department of General Studies, Newgate College of Health Technology, Minna, Niger State, Nigeria.

²Department of Mathematics, Bingham University Karu, Nassarawa State, Nigeria.

³Department of Mathematics, Federal Polytechnic Nasarawa, Nasarawa State, Nigeria.

Authors' contributions

This work was carried out in collaboration among all authors. Author TJO designed the study, performed the analysis, wrote the protocol and wrote the first draft of the manuscript. Authors EA and YMK managed the analyses. Author YMK managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/ARJOM/2020/v16i1130238

Editor(s):

(1) Dr. Nikolaos D. Bagis, Aristotle University of Thessaloniki, Greece.

Reviewers:

(1) Simonescu Claudia Maria, University POLITEHNICA of Bucharest, Romania.

(2) Asep K. Supriatna, Padjadjaran University, Indonesia.

Complete Peer review History: <http://www.sdiarticle4.com/review-history/62581>

Received: 28 August 2020

Accepted: 01 November 2020

Published: 28 November 2020

Original Research Article

Abstract

A Mathematical model of a system of non-linear differential equation is developed to study the transmission dynamics of malaria, dengue and typhoid triple infection. In this work, the basic reproduction number is derived using the Next Generation Matrix, also we computed the disease free equilibrium point. The disease free equilibrium (DFE) point is analyzed and was found that the DFE is locally stable but may be globally unstable when $R_0 < 1$.

Keywords: Malaria; dengue; typhoid; co-infection; reproduction number; stability analysis; disease-free equilibrium.

1 Introduction

Malaria is an infectious disease caused by Plasmodium parasite, spread through the bites of an infected female Anopheles mosquito [1,2]. According to the World Health Organization (WHO), the estimated

*Corresponding author: E-mail: dayofemi985@gmail.com;

number of death due to malaria is 435,000 with Africa recording 93% of the death cases [3]. The various species of the parasite causing malaria includes *P. falciparum*, *P. vivax*, *P. malariae*, *P. knowlesi*, *P. ovale wallikeri*, *P. ovale curtisi* [4].

Dengue (DENV), is a viral disease transmitted by an infected female *Aedes aegypti* mosquito to human, it is also known as ‘breakbone fever’, ‘tropical flu’ [5,6]. The disease is caused by any of the four DENV virus (DENV-1, DENV-2, DENV-3 and DENV-4). The infection ranges from mild illness to more severe forms such as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS) [6]. According to the World Health Organization (WHO), the incidence of dengue has grown globally, from 505,430 cases in 2000 to over 4.2 million cases in 2019. In 2019, the highest reported cases of dengue were recorded with Afghanistan recording her first case [7].

Typhoid, an infectious disease caused by a bacteria *Salmonella Typhi* is spread through contaminated food and water [8]. The signs and symptoms includes; sustained fever, poor appetite, severe headache, fatigue and vomiting. Typhoid Fever has an incubation period of between 7 – 14 days [9]. It is estimated that 11 – 20 million cases of typhoid fever is reported annually with 128000 – 161 000 deaths yearly. The risk of typhoid is higher in population with inadequate access to safe water and poor sanitation [10].

Ogunmiloro [2] considered a mathematical model of malaria-toxoplasmosis co-infection dynamics. The analysis shows that the model is locally and globally stable, sensitivity analysis revealed that the spread malaria-toxoplasmosis can be achieved via the availability of treated bed nets, proper environmental sanitation, availability of drugs etc. Amoah-Mensah [11] proposed a mathematical model to study stability analysis of the disease-free and endemic equilibria of zika-malaria co-infection in malaria-endemic region. Sensitivity analysis on the basic reproduction number reveals that recovery from the diseases simultaneously will eliminate the disease. Aldila [12] developed a model to study Dengue-Chikungunya co-infection. In the work, the basic reproduction was computed and the local stability of the equilibrium points were computed. Bonyah [13] proposed a co-infection model of zika and dengue virus. The local stability of the disease free equilibrium was analyzed and the computation of the basic reproduction number. Bifurcation analysis proved that the model experiences backward bifurcation. Oluwafemi [14] formulated a co-infection model of malaria and dengue fever transmission dynamics. In the work, the basic reproduction number was computed and analysis established the local and global stability of the disease-free equilibrium of the model.

There are reports of Malaria, Dengue and Typhoid triple infection. Suresh [15] reported the case of a 24 year man with malaria, dengue fever and typhoid infection, Deshkar [16] reported a 38 year old male with the triple infection, Basha [17] report the triple infection among children. However no literature to the best of the author knowledge is available to study mathematically the dynamics of malaria, dengue and typhoid triple infection. Few mathematical model has been proposed and analyzed to study triple infections [18,19].

In this paper, we develop a mathematical model to study the transmission dynamics of Malaria, Dengue and Typhoid triple infection and the stability analysis of the disease free equilibrium were carried out.

2 Model Formulation

The proposed model divides the human population into Susceptible human S_h ; Malaria infected human I_{hm} ; Dengue Infected human I_{hd} ; Typhoid infected human I_{ht} ; co-infection of Malaria and Dengue I_{md} ; Malaria and Typhoid I_{mt} ; Dengue and Typhoid I_{dt} ; Malaria, Dengue and Typhoid I_{mdt} ; Recovered class R ; the vector population is subdivided into; Non-disease carrier vector S_v ; Malaria parasite vector carrier I_{vm} ; Dengue virus vector carrier I_{vd} and the Typhoid carrier Bacteria W .

The system of equations representing the transmission dynamics of the triple infection is presented as follows:

$$\left. \begin{aligned}
 \frac{dS_h}{dt} &= \Lambda + \delta R - (\alpha_{hm} + \alpha_{hd} + \alpha_{ht} + \mu_h)S_h \\
 \frac{dI_{hm}}{dt} &= \alpha_{hm}S_h + \rho_{hd}I_{md} + \rho_{ht}I_{mt} - (\alpha_{hd} + \alpha_{ht} + \rho_{hm} + \eta_{hm} + \mu_h)I_{hm} \\
 \frac{dI_{hd}}{dt} &= \alpha_{hd}S_h + \rho_{hm}I_{md} + \rho_{ht}I_{dt} - (\alpha_{hm} + \alpha_{ht} + \rho_{hd} + \eta_{hd} + \mu_h)I_{hd} \\
 \frac{dI_{ht}}{dt} &= \alpha_{ht}S_h + \rho_{hm}I_{mt} + \rho_{hd}I_{dt} - (\alpha_{hm} + \alpha_{hd} + \rho_{ht} + \eta_{ht} + \sigma_1 + \mu_h)I_{ht} \\
 \frac{dI_{md}}{dt} &= \alpha_{hm}I_{hd} + \alpha_{hd}I_{hm} + \rho_{ht}I_{mdt} - (\alpha_{ht} + \rho_{hd} + \rho_{hm} + \eta_{hm} + \eta_{hd} + \mu_h)I_{md} \\
 \frac{dI_{mt}}{dt} &= \alpha_{hm}I_{ht} + \alpha_{ht}I_{hm} + \rho_{hd}I_{mdt} - (\alpha_{hd} + \rho_{ht} + \rho_{hm} + \eta_{hm} + \eta_{ht} + \sigma_2 + \mu_h)I_{mt} \\
 \frac{dI_{dt}}{dt} &= \alpha_{ht}I_{hd} + \alpha_{hd}I_{ht} + \rho_{hm}I_{mdt} - (\alpha_{hm} + \rho_{hd} + \rho_{ht} + \eta_{ht} + \eta_{hd} + \sigma_3 + \mu_h)I_{dt} \\
 \frac{dI_{mdt}}{dt} &= \alpha_{ht}I_{md} + \alpha_{hd}I_{mt} + \alpha_{hm}I_{dt} - (\rho_{hm} + \rho_{hd} + \rho_{ht} + \eta_{hm} + \eta_{ht} + \eta_{hd} + \sigma_4 + \mu_h)I_{mdt} \\
 \frac{dR}{dt} &= \rho_{hm}I_{hm} + \rho_{hd}I_{hd} + \rho_{ht}I_{ht} - \delta R \\
 \frac{dS_v}{dt} &= \Lambda_v - (\beta_1 + \beta_2 + \mu_v)S_v \\
 \frac{dI_{vm}}{dt} &= \beta_1S_v - \mu_vI_{vm} \\
 \frac{dI_{vd}}{dt} &= \beta_2S_v - \mu_vI_{vd} \\
 \frac{dW}{dt} &= \sigma_1I_{ht} + \sigma_2I_{mt} + \sigma_3I_{dt} + \sigma_4I_{mdt} - \mu_bW
 \end{aligned} \right\} \tag{1}$$

Where

$$\left. \begin{aligned}
 \alpha_{hm} &= \frac{b_m \vartheta_m I_{vm}}{N_h}; \alpha_{hd} = \frac{b_d \vartheta_d I_{vd}}{N_h}; \alpha_{ht} = \frac{vW}{K+W} \\
 \beta_1 &= \frac{b_m \vartheta_m (I_{hm} + I_{md} + I_{mt} + I_{mdt})}{N_h}; \beta_2 = \frac{b_d \vartheta_d (I_{hd} + I_{md} + I_{dt} + I_{mdt})}{N_h}
 \end{aligned} \right\} \tag{2}$$

The Tables below contains the description of the variables and parameters of the model.

Table 1. Model variables

Variables	Description
S_h	Susceptible human
I_{hm}	Malaria infected human
I_{hd}	Dengue Infected human
I_{ht}	Typhoid infected human
I_{md}	Malaria and Dengue Co-infection
I_{mt}	Malaria and Typhoid Co-infection
I_{dt}	Dengue and Typhoid Co-infection
I_{mdt}	Malaria, Dengue and Typhoid co-infection
R	Recovered human
S_v	Non-disease carrier vector
I_{vm}	Malaria parasite vector carrier
I_{vd}	Dengue virus vector carrier
W	Typhoid carrier Bacteria

Table 2. Model parameters

Parameter	Description
Λ	Recruitment rate
δ	Rate at which recovered become susceptible
ρ_{hm}	Recovery rate for malaria only
ρ_{hd}	Recovery rate for dengue fever only
ρ_{ht}	Recovery rate for typhoid only
α_{hm}	Rate at which one acquires malaria
α_{hd}	Rate at which one acquires dengue
α_{ht}	Rate at which one acquires typhoid
η_{hm}	Malaria induced death
η_{hd}	Dengue induced death
η_{ht}	Typhoid induced death
μ_h	Human Natural death rate
Λ_v	Vector recruitment rate
μ_v	Vector natural death
σ	Typhoid Bacteria discharge rate
Λ_b	Bacteria growth rate
μ_b	Bacteria death rate
b_m	Probability of transmission of malaria
b_d	Probability of transmission of dengue
ϑ_m	Number of bites of malaria carrier vector per time
ϑ_d	Number of bites of dengue carrier vector per time

3 Methodology

3.1 Positivity of solution

Theorem 3.1:

Let the initial state variable be non-negative i.e. $\{(S_h(0), I_{hm}(0), I_{hd}(0), I_{ht}(0), I_{md}(0), I_{mt}(0), I_{dt}(0), I_{mdt}(0), R(0), S_v(0), I_{vm}(0), I_{vd}(0), W(0)) \geq 0\}$

Then the solution set $\{(S_h, I_{hm}, I_{hd}, I_{ht}, I_{md}, I_{mt}, I_{dt}, I_{mdt}, R, S_v, I_{vm}, I_{vd}, W)\}(t)$ is positive for all $t > 0$.
 $\limsup N_h(t) \leq \frac{\Lambda}{\mu_h}$ and $\limsup N_m(t) \leq \frac{\Lambda_m}{\mu_m}$.

If $N_h(0) \leq \frac{\Lambda}{\mu_h}$ and $N_m(0) \leq \frac{\Lambda_m}{\mu_m}$ then $N_h(t) \leq \frac{\Lambda}{\mu_h}$ and $N_m(t) \leq \frac{\Lambda_m}{\mu_m}$.

The region,

$$\Omega_h = \left\{ (S_h, I_{hm}, I_{hd}, I_{ht}, I_{md}, I_{mt}, I_{dt}, I_{mdt}, R) \in R_+^9 : N_h(t) \leq \frac{\Lambda}{\mu_h} \right\}$$

$$\Omega_m = \left\{ (S_v, I_{vm}, I_{vd}) \in R_+^3 : N_m(t) \leq \frac{\Lambda_m}{\mu_m} \right\}$$

Is positively invariant. The theorem indicates that the model is biologically and epidemiologically well posed in the region and thus, the dynamics of the model can be sufficiently studied in Ω .

3.2 Disease Free Equilibrium (DFE)

The DFE point is the state where there are no infections. To compute the DFE, the right hand side of the model is set at zero. We solve for the non-infected and non-carrier state and variables at $I_{hm} = I_{hd} = I_{ht} = I_{md} = I_{mt} = I_{dt} = I_{mtd} = I_{vm} = I_{vd} = 0$.

Hence the DFE is given as

$$E^0 = (S_h, I_{hm}, I_{hd}, I_{ht}, I_{md}, I_{mt}, I_{dt}, I_{mtd}, R, S_v, I_{vm}, I_{vd}, W)$$

$$= \left(\frac{\Lambda}{\mu_h}, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0, 0 \right) \tag{3}$$

3.3 Basic reproduction number and local stability of Disease Free Equilibrium (DFE)

The basic reproduction number is defined as the average number of secondary infections that a single infected individual will produce in a susceptible population. To compute the Basic Reproduction Number, the next generation method will be applied. By the method, the Basic reproduction number is the maximum value of the spectral radius of matrix FV^{-1} . Where F and V are the rate of appearance of new infections and rate of in or out of a compartment respectively. The matrices at DFE are given by

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ a_4 & 0 & 0 & a_4 & a_4 & 0 & a_4 & 0 & 0 & 0 & 0 \\ 0 & a_5 & 0 & a_5 & 0 & a_5 & a_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{4}$$

$$V = \begin{pmatrix} u_1 & 0 & 0 & -\rho_{hd} & -\rho_{ht} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & u_2 & 0 & -\rho_{hm} & 0 & -\rho_{ht} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & u_3 & 0 & -\rho_{hm} & -\rho_{hd} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & u_4 & 0 & 0 & -\rho_{ht} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & u_5 & 0 & -\rho_{hd} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & u_6 & -\rho_{hm} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & u_7 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_v & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_v & 0 & 0 \\ 0 & 0 & -\sigma_1 & 0 & -\sigma_2 & -\sigma_3 & -\sigma_4 & 0 & 0 & \mu_b & 0 \end{pmatrix} \tag{5}$$

Where

$$a_1 = \frac{b_m \vartheta_m S_h}{N_h}; a_2 = \frac{b_d \vartheta_d S_h}{N_h}; a_3 = \frac{v S_h}{K}; a_4 = \frac{b_m \vartheta_m S_v}{N_h}; a_5 = \frac{b_d \vartheta_d S_v}{N_h}; \tag{6}$$

$$\left. \begin{aligned} u_1 &= (\rho_{hm} + \eta_{hm} + \mu_h); u_2 = (\rho_{hd} + \eta_{hd} + \mu_h); u_3 = (\rho_{ht} + \eta_{ht} + \sigma_1 + \mu_h); \\ u_4 &= (\rho_{hd} + \rho_{hm} + \eta_{hm} + \eta_{hd} + \mu_h); u_5 = (\rho_{ht} + \rho_{hm} + \eta_{hm} + \eta_{ht} + \sigma_2 + \mu_h); \\ u_6 &= (\rho_{hd} + \rho_{ht} + \eta_{ht} + \eta_{hd} + \sigma_3 + \mu_h); \\ u_7 &= (\rho_{hm} + \rho_{hd} + \rho_{ht} + \eta_{hm} + \eta_{ht} + \eta_{hd} + \sigma_4 + \mu_h) \end{aligned} \right\} \tag{7}$$

The basic reproduction number is given as

$$R_0 = \max \left\{ \sqrt{\frac{\Lambda_v \mu_h b_m^2 \theta_m^2}{(\rho_{hm} + \eta_{hm} + \mu_h) \Lambda \mu_v^2}}, \sqrt{\frac{\Lambda_v \mu_h b_d^2 \theta_d^2}{(\rho_{hd} + \eta_{hd} + \mu_h) \Lambda \mu_v^2}}, \sqrt{\frac{v \Lambda \sigma_1}{(\rho_{ht} + \eta_{ht} + \sigma_1 + \mu_h) K \mu_b \mu_h}} \right\} \quad (8)$$

Theorem 3.2: The DFE is locally asymptotically stable if $R_0 < 1$ and unstable when otherwise.

3.4 Global stability of the Disease-Free Equilibrium (DFE)

The theorem [20] is used to study the global asymptotic stability of the disease-free equilibrium of the model. The model is re-written as

$$\frac{dX}{dt} = H(X, Z), \quad (9)$$

$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0 \quad (10)$$

Where $X = (S_h, R, S_v)$ and $Z = (I_{hm}, I_{hd}, I_{ht}, I_{md}, I_{mt}, I_{dt}, I_{mdt}, I_{vmt}, I_{vdt})$, with $X \in \mathbb{R}^3$ denoting non-infected class and the components $Z \in \mathbb{R}^9$ denoting infected class.

The disease-free equilibrium is denoted as

$$E^0 = (X^0, 0), X^0 = \left(\frac{\Lambda}{\mu_h}, \frac{\Lambda_v}{\mu_v} \right). \quad (11)$$

The conditions (H_1) and (H_2) must be satisfied to guarantee global asymptotic stability:

$$H_1: \frac{dX}{dt} = H(X, 0), X^0 \text{ is globally asymptotically stable (GAS)} \quad (12)$$

$$H_2: G(X, Z) = PZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0 \text{ for } (X, Z) \in \Omega \quad (13)$$

Where $P = D_z G(X^0, 0)$ is an M-matrix (the off-diagonal elements of P are non-negative) and Ω is the region where the model makes biological sense.

Theorem 3.3: The DFE (E^0) of the model equation is globally asymptotically stable if $R_0 < 1$ and the conditions (H_1) and (H_2) are satisfied.

Proof: We have $H(X, Z)$ and $G(X, Z)$ given as

$$H(X, Z) = \begin{pmatrix} \Lambda + \delta R - (\alpha_{hm} + \alpha_{hd} + \alpha_{ht} + \mu_h) S_h \\ \rho_{hm} I_{hm} + \rho_{hd} I_{hd} + \rho_{ht} I_{ht} - \delta R \\ \Lambda_v - (\beta_1 + \beta_2 + \mu_v) S_v \end{pmatrix} \quad (14)$$

$$G(X, Z) = \begin{pmatrix} \alpha_{hm} S_h + \rho_{hd} I_{md} + \rho_{ht} I_{mt} - (\alpha_{hd} + \alpha_{ht} + \rho_{hm} + \eta_{hm} + \mu_h) I_{hm} \\ \alpha_{hd} S_h + \rho_{hm} I_{md} + \rho_{ht} I_{dt} - (\alpha_{hm} + \alpha_{ht} + \rho_{hd} + \eta_{hd} + \mu_h) I_{hd} \\ \alpha_{ht} S_h + \rho_{hm} I_{mt} + \rho_{hd} I_{dt} - (\alpha_{hm} + \alpha_{hd} + \rho_{ht} + \eta_{ht} + \sigma_1 + \mu_h) I_{ht} \\ \alpha_{hm} I_{hd} + \alpha_{hd} I_{hm} + \rho_{ht} I_{mdt} - (\alpha_{ht} + \rho_{hd} + \rho_{hm} + \eta_{hm} + \eta_{hd} + \mu_h) I_{md} \\ \alpha_{hm} I_{ht} + \alpha_{ht} I_{hm} + \rho_{hd} I_{mdt} - (\alpha_{hd} + \rho_{ht} + \rho_{hm} + \eta_{hm} + \eta_{ht} + \sigma_2 + \mu_h) I_{mt} \\ \alpha_{ht} I_{hd} + \alpha_{hd} I_{ht} + \rho_{hm} I_{mdt} - (\alpha_{hm} + \rho_{hd} + \rho_{ht} + \eta_{ht} + \eta_{hd} + \sigma_3 + \mu_h) I_{dt} \\ \alpha_{ht} I_{md} + \alpha_{hd} I_{mt} + \alpha_{hm} I_{dt} - (\rho_{hm} + \rho_{hd} + \rho_{ht} + \eta_{hm} + \eta_{ht} + \eta_{hd} + \sigma_4 + \mu_h) I_{mdt} \\ \beta_1 S_v - \mu_v I_{vm} \\ \beta_2 S_v - \mu_v I_{vd} \end{pmatrix} \quad (15)$$

The reduced system

$$H(X, 0) = \begin{pmatrix} \Lambda - \mu_h S_h \\ 0 \\ \Lambda_v - \mu_v S_v \end{pmatrix} \tag{16}$$

Let

$$P = \begin{pmatrix} -u_1 & 0 & 0 & \rho_{hd} & \rho_{ht} & 0 & 0 & b_m \vartheta_m & 0 \\ 0 & -u_2 & 0 & \rho_{hm} & 0 & \rho_{ht} & 0 & 0 & b_d \vartheta_d \\ 0 & 0 & -u_3 & 0 & \rho_{hm} & \rho_{hd} & 0 & 0 & 0 \\ 0 & 0 & 0 & -u_4 & 0 & 0 & \rho_{ht} & 0 & 0 \\ 0 & 0 & 0 & 0 & -u_5 & 0 & \rho_{hd} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -u_6 & \rho_{hm} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -u_7 & 0 & 0 \\ a_4 & 0 & 0 & a_4 & a_4 & 0 & a_4 & -\mu_v & 0 \\ 0 & a_5 & 0 & a_5 & 0 & a_5 & a_5 & 0 & -\mu_v \end{pmatrix} \tag{17}$$

Then

$$\hat{G}(X, Z) = \begin{pmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \\ \hat{G}_3(X, Z) \\ \hat{G}_4(X, Z) \\ \hat{G}_5(X, Z) \\ \hat{G}_6(X, Z) \\ \hat{G}_7(X, Z) \\ \hat{G}_8(X, Z) \\ \hat{G}_9(X, Z) \end{pmatrix} = \begin{pmatrix} b_m \vartheta_m \left(1 - \frac{S_h}{N_h}\right) + (\alpha_{hd} + \alpha_{ht}) I_{hm} \\ b_d \vartheta_d \left(1 - \frac{S_h}{N_h}\right) + (\alpha_{hm} + \alpha_{ht}) I_{hd} \\ -\left(\frac{vW}{K+W}\right) S_h + (\alpha_{hm} + \alpha_{hd}) I_{ht} \\ -(\alpha_{hm} I_{hd} + \alpha_{hd} I_{hm}) + \alpha_{ht} I_{md} \\ -(\alpha_{hm} I_{ht} + \alpha_{ht} I_{hm}) + \alpha_{hd} I_{mt} \\ -(\alpha_{ht} I_{hd} + \alpha_{hd} I_{ht}) + \alpha_{hm} I_{dt} \\ -(\alpha_{ht} I_{md} + \alpha_{hd} I_{mt} + \alpha_{hm} I_{dt}) \\ b_m \vartheta_m (I_{hm} + I_{md} + I_{mt} + I_{mdt}) \left(1 - \frac{S_v}{N_h}\right) \\ b_d \vartheta_d (I_{hd} + I_{md} + I_{dt} + I_{mdt}) \left(1 - \frac{S_v}{N_h}\right) \end{pmatrix} \tag{18}$$

It is observed that $\hat{G}_7(X, Z) < 0$ so the conditions (H_1) and (H_2) are not satisfied, hence (E^0) may not be globally asymptotically stable when $R_0 < 1$.

4 Conclusion

In this paper, we developed and carried out the stability analysis of the disease free equilibrium model of the transmission dynamics of malaria, dengue and typhoid triple infection.

We compute the disease free equilibrium (DFE) point, after which the basic reproduction number (R_0) of the model is derived which also shows that the system is locally stable. The global stability of the model was also carried out and it was observed that the disease free equilibrium point may not be globally stable given that $R_0 < 1$. However, if maximum protection is provided again the triple infection, global stability of the disease free can be achieved.

Competing Interests

Authors have declared that no competing interests exist.

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