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DYNAMICAL SYSTEM FOR EBOLA OUTBREAK WITHIN VACCINATION TREATMENT

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Abstract

Ebola Virus Disease (EVD) is a deadly disease caused by Ebola virus. The mathematical model of Ebola virus transmission dynamics is formulated by considering both human and vector populations. This research aims to analyse dynamic systems of EVD transmission considering vaccination treatment. The equilibrium points and basic reproduction number $(\mathcal{R}_0)$ are determined. There are two equilibrium points, namely, disease-free equilibrium and endemic equilibrium points. The results of model analysis show that the disease-free equilibrium is locally asymptotically stable if $\mathcal{R}_0 < 1$. The endemic equilibrium is found to be unique, positive and asymptotically stable if $\mathcal{R}_0 > 1$. Numerical simulation is performed for showing the population dynamic of both human and vector for time.

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1. Introduction

Ebola virus is one of the Filoviridae families that causes severe hemorrhagic fever in human and non-human primates. Animal which is considered as natural vector are fruits bat of Pteropodidae family. EVD spread in human populations occurred after the susceptible individual had direct contacted with infected human blood, body fluids or skin, or comes in contained environmental Ebola virus.

EVD had occurred in the region of Central Africa to West Africa which began in Guinea on December 2013, then spread to the territory of Liberia, Sierra Leone, and Nigeria. On June 10th, 2016, World Health Organization (WHO) states that there are 28.616 people confirmed, probable and suscepted cases have been reported in Guinea, Liberia, and Sierra Leone, with 11.310 deaths [7].

Mathematical model is an important tool to analyze the characteristics of epidemiology of this infectious disease. Kalu et al. [2] formulated a mathematical model for Ebola disease into SEIR type. Osemwinyen and Diakhaby [4] formulated a mathematical model for Ebola disease into SIQRD type, and the result shows that quarantine increased the number of recovered people. Martins et al. [3] developed a mathematical model of Ebola disease involving the vector population, namely, SEIQR-SI type, and the result shows that disease-free equilibrium is locally and globally stable.

In this article, Ebola disease compartment is formulated by modifying model SEIQR-SI type by involving vaccine compartment model [3], in order to obtain SVEIQR-SI type model. In addition, Ebola disease compartment is formulated by involving assumption that exposed population can be diagnosed prior to symptoms and infected populations recover naturally [5]. Furthermore, this model is called *SVEIQR-SI model*.

This article is divided into five sections: Section 1 is the background and purpose of this article. Section 2 describes the model formulation. Section 3 describes model analysis. Section 4 shows numerical simulation. Finally, the conclusions are provided in Section 5.

2. Model Formulation

The total human population is categorized into six compartments: susceptible human population (S_H), vaccinated human population (V_H), exposed human population (E_H), infected human population (I_H), quarantined human population (Q_H) and recovered human population (R_H). Total vector population is categorized into two compartments: susceptible (S_V) and infected vector population (I_V).

The underlying assumptions establishing this model are as follows. The rate of incoming human individuals into the susceptible population is constant Λ_H. The rate of change of infected human population is dependent on the level of population of infected human (I_H), quarantined human (Q_H) and the infected vector (I_V), and mathematically expressed as $\alpha_1(I_H + \eta Q_H + I_V)$. The susceptible population receives the vaccine at level ξ. The rate of change of infected human population from the vaccinated human is dependent on the level of population of infected human (I_H), quarantined human (Q_H) and the infected vector (I_V), and mathematically expressed as $(1-l)\alpha_1(I_H + \eta Q_H + I_V)$, where l is the vaccine effectiveness $(0 < l < 1)$. The exposed human can move into the infected human individual with rate σ_1 or be diagnosed prior to symptoms with rate γ_r. The infected human individual becomes the quarantined human individual with rate σ_2 or recovers naturally with rate γ_r. The quarantined human individual can infect the susceptible or vaccinated human population with probability η. The infected and quarantined human individual can be killed by Ebola virus with rate δ_1. The natural mortality rate for all human compartments is μ_1. We do not consider the exposed vector population. The rate of incoming vector individuals into the susceptible is constant Λ_V. New infectious occur only produced due to contact between the susceptible vector individual and the infected vector individual with an incidence rate $\alpha_2 I_V$.

The infected vector individual infects the susceptible vector individual or the susceptible and vaccinated human individuals. The infected vector individual can be killed by Ebola virus with rate δ_2. The natural mortality rate for all vector compartments is μ_2.

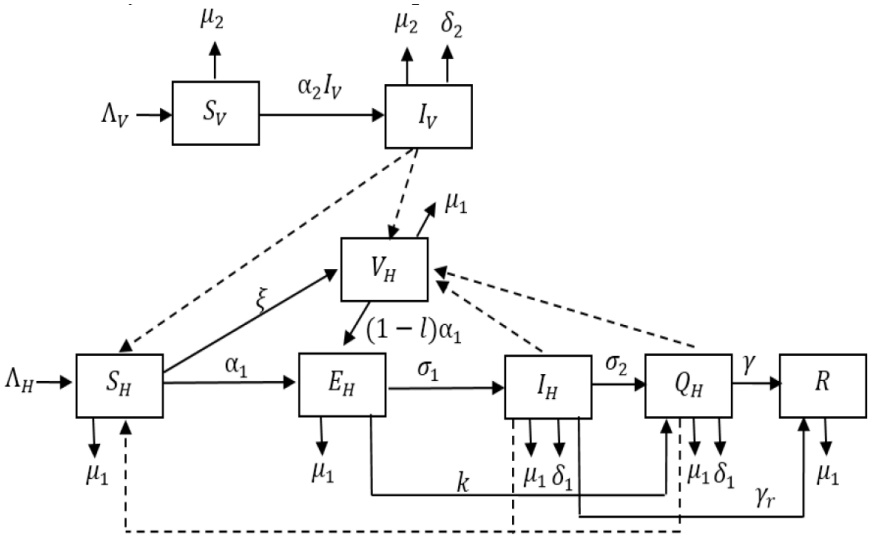


Figure 1. Compartment of Ebola transmission model.

Based on the assumptions, the model of Ebola virus transmission is given by eight ordinary differential equations as follows:

$$\frac{dS_H}{dt} = \Lambda_H - \alpha_1(I_H + \eta Q_H + I_V)S_H - \mu_1 S_H - \xi S_H,$$

$$\frac{dV_H}{dt} = \xi S_H - (1-l)\alpha_1(I_H + \eta Q_H + I_V)V_H - \mu_1 V_H,$$

$$\frac{dE_H}{dt} = \alpha_1(I_H + \eta Q_H + I_V)S_H - (\mu_1 + \sigma_1 + k)E_H + (1-l)\alpha_1(I_H + \eta Q_H + I_V)V_H,$$

$$\frac{dI_H}{dt} = \sigma_1 E_H - (\mu_1 + \sigma_2 + \delta_1)I_H - \gamma_r I_H,$$

$$\frac{dQ_H}{dt} = \sigma_2 I_H - (\mu_1 + \delta_1 + \gamma)Q_H + kE_H,$$

$$\frac{dR_H}{dt} = \gamma Q_H - \mu_1 R_H + \gamma_r I_H,$$

$$\frac{dS_V}{dt} = \Lambda_V - \alpha_2 I_V S_V - \mu_2 S_V,$$

$$\frac{dI_V}{dt} = \alpha_2 I_V S_V - (\mu_2 + \delta_2) I_V, \tag{1}$$

with $N_H(t) = S_H(t) + V_H(t) + E_H(t) + I_H(t) + Q_H(t) + R_H(t)$ is the total human population at time t and $N_V(t) = S_V(t) + I_V(t)$ is the total vector population at time t. The initial value for the system (1) is $S_H(0) = S_{H_0}$, $V_H(0) = V_{H_0}$, $E_H(0) = E_{H_0}$, $I_H(0) = I_{H_0}$, $Q_H(0) = Q_{H_0}$, $R_H(0) = R_{H_0}$, $S_V(0) = S_{V_0}$ and $I_V(0) = I_{V_0}$. All parameters are non-negative constants.

Lemma 1. *The set*

$$D = \left\{ (S_H, V_H, E_H, I_H, Q_H, R_H, S_V, I_V) \in \mathbb{R}_+^8 \,|\, 0 \le N_H \le \frac{\Lambda_H}{\mu_1} + N_{H_0}, \right.$$

$$\left. 0 \le S_H \le \frac{\Lambda_H}{\mu_1 + \xi} + S_{H_0}, 0 \le N_V \le \frac{\Lambda_V}{\mu_2} + N_{V_0} \right\}$$

is the positive bounded region from the system (1), *where* N_{H_0} *and* N_{V_0} *are the total human and vector populations at* $t = 0$, *respectively.*

3. Model Analysis

The disease-free equilibrium of the system (1) is given by

$$\boldsymbol{T_0}(S_H, V_H, E_H, I_H, Q_H, R_H, S_V, I_V)$$

$$= \left(\frac{\Lambda_H}{\mu_1 + \xi}, \frac{\Lambda_H \xi}{\mu_1(\mu_1 + \xi)}, 0, 0, 0, 0, \frac{\Lambda_V}{\mu_2}, 0 \right),$$

and the endemic equilibrium of the system (1) is given by

$$\boldsymbol{T}^*(S_H, V_H, E_H, I_H, Q_H, R_H, S_V, I_V)$$

$$= (S_H^*, V_H^*, E_H^*, I_H^*, Q_H^*, R_H^*, S_V^*, I_V^*),$$

where

$$S_H^* = \frac{\Lambda_H}{\alpha_1(I_H^* + \eta Q_H^* + I_V^*) + \mu_1 + \xi}, \quad Q_H^* = \frac{KE_H^* + I_H^*\sigma_2}{\mu_1 + \sigma_1 + \gamma},$$

$$V_H^* = \frac{S_H^*\xi}{\alpha_1(1-l)(I_H^* + \eta Q_H^* + I_V^*) + \mu_1}, \quad R_H^* = \frac{\gamma Q_H^* + I_H^*\gamma_r}{\mu_1},$$

$$E_H^* = \frac{\alpha_1((1-l)V_H^* + S_H^*)(I_H^* + \eta Q_H^* + I_V^*)}{k + \sigma_1 + \mu_1}, \quad S_V^* = \frac{\Lambda_V}{\alpha_2 I_V^* + \mu_2},$$

$$I_H^* = \frac{\sigma_1 E_H^*}{\gamma_r + \sigma_2 + \mu_1 + \delta_1}, \quad I_V^* = 0. \tag{2}$$

We calculate the basic reproduction number by using the next generation operator approach by van den Driessche and Watmough [6]. The next generation matrix at the disease-free equilibrium $\boldsymbol{T_0}$ is given by:

$$\boldsymbol{F} = \begin{pmatrix} 0 & \frac{\alpha_1\Lambda_H((1-l)\xi + \mu_1)}{\mu_1(\mu_1 + \xi)} & \frac{\alpha_1\Lambda_H((1-l)\xi + \mu_1)}{\mu_1(\mu_1 + \xi)} & \frac{\alpha_1\Lambda_H((1-l)\xi + \mu_1)}{\mu_1(\mu_1 + \xi)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\alpha_2\Lambda_V}{\mu_2 + \xi} \end{pmatrix},$$

$$\boldsymbol{V} = \begin{pmatrix} k + \sigma_1 + \mu_1 & 0 & 0 & 0 \\ -\sigma_1 & \gamma_r + \delta_1 + \sigma_2 + \mu_1 & 0 & 0 \\ -k & -\sigma_2 & \gamma + \delta_1 + \mu_1 & 0 \\ 0 & 0 & 0 & \delta_2 + \mu_2 \end{pmatrix}.$$

The basic reproduction number $\mathcal{R}_0$ is dominant eigenvalue of $\boldsymbol{FV}^{-1}$, thus we get

$$\mathcal{R}_0 = \left(\frac{\begin{array}{c} \alpha_1 \Lambda_H ((1-l)\xi + \mu_1)(k\eta(\gamma_r + \delta_1 + \sigma_2 + \mu_1) \\ + \sigma_1(\mu_1 + \delta_1 + \gamma + \eta\sigma_2)) \end{array}}{\mu_1(\mu_1 + \delta_1 + \gamma)(\mu_1 + \xi)(k + \sigma_1 + \mu_1)(\gamma_r + \sigma_2 + \mu_1 + \delta_1)} \right). \quad (3)$$

The stability of system (1) is dependent on the basic reproduction number $\mathcal{R}_0$. The stability analysis of both equilibrium $\boldsymbol{T_0}$ and $\boldsymbol{T}^*$ will be provided through the following theorems:

Theorem 1. *The disease-free equilibrium* $\boldsymbol{T_0}$ *is locally asymptotically stable if* $\mathcal{R}_0 < 1$, *and unstable if* $\mathcal{R}_0 > 1$.

Proof. The Jacobian matrix at $\boldsymbol{T_0}$ for system (1) is given by

$$\boldsymbol{J}_{\boldsymbol{T_0}} = \begin{pmatrix} -\mu_1 - \xi & 0 & 0 & -\frac{\alpha_1 \Lambda_H}{\mu_1 + \xi} & -\frac{\alpha_1 \eta \Lambda_H}{\mu_1 + \xi} & 0 & 0 & -\frac{\alpha_1 \Lambda_H}{\mu_1 + \xi} \\ \xi & -\mu_1 & 0 & -\frac{\alpha_1(1-l)\Lambda_H \xi}{\mu_1(\mu_1 + \xi)} & -\frac{\alpha_1(1-l)\eta \Lambda_H \xi}{\mu_1(\mu_1 + \xi)} & 0 & 0 & J_{28} \\ 0 & 0 & J_{33} & J_{34} & J_{35} & 0 & 0 & J_{38} \\ 0 & 0 & \sigma_1 & J_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & k & \sigma_2 & -\gamma - \delta_1 - \mu_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_r & \gamma & -\mu_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_2 & -\frac{\alpha_2 \Lambda_V}{\mu_2} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & J_{88} \end{pmatrix},$$

where

$$J_{28} = -\frac{\alpha_1(1-l)\Lambda_H \xi}{\mu_1(\mu_1 + \xi)},$$

$$J_{33} = -k - \sigma_1 - \mu_1,$$

$$J_{34} = \frac{\alpha_1(1-l)\Lambda_H \xi}{\mu_1(\mu_1 + \xi)} + \frac{\alpha_1 \Lambda_H}{\mu_1 + \xi},$$

$$J_{35} = \frac{\alpha_1(1-l)\eta\Lambda_H\xi}{\mu_1(\mu_1+\xi)} + \frac{\alpha_1\eta\Lambda_H}{\mu_1+\xi},$$

$$J_{38} = \frac{\alpha_1(1-l)\Lambda_H\xi}{\mu_1(\mu_1+\xi)} + \frac{\alpha_1\Lambda_H}{\mu_1+\xi},$$

$$J_{44} = -\gamma_r - \delta_1 - \sigma_2 - \mu_1,$$

$$J_{88} = \alpha_2\frac{\Lambda_V}{\mu_2} - \delta_2 - \mu_2.$$

The characteristic polynomial of the matrix $\boldsymbol{J_{T_0}}$ is

$$(\lambda - J_{11})(\lambda - J_{22})(\lambda - J_{66})(\lambda - J_{77})(\lambda - J_{88})$$
$$\cdot(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0, \tag{4}$$

where

$$a_1 = k + \sigma_1 + \mu_1 + \gamma_r + \delta_1 + \sigma_2 + \mu_1 + \gamma + \delta_1 + \mu_1,$$

$$a_2 = -\left(\alpha_1(1-l)\frac{\Lambda_H\xi}{\mu_1(\mu_1+\xi)} + \alpha_1\frac{\Lambda_H}{\mu_1+\xi}\right)(\sigma_1+\eta k)$$
$$+ (k+\sigma_1+\mu_1)(\gamma_r+\delta_1+\sigma_2+\mu_1) + (k+\sigma_1+\mu_1)(\gamma+\delta_1+\mu_1)$$
$$+ (\gamma_r+\delta_1+\sigma_2+\mu_1)(\gamma+\delta_1+\mu_1),$$

$$a_3 = -\frac{\alpha_1\Lambda_H((1-l)\xi+\mu_1)((\gamma_r+\delta_1+\sigma_2+\mu_1)\eta k + \sigma_1(\gamma+\delta_1+\mu_1+\eta\sigma_2))}{\mu_1(\mu_1+\xi)}$$
$$+ (k+\sigma_1+\mu_1)(\gamma_r+\delta_1+\sigma_2+\mu_1)(\gamma+\delta_1+\mu_1).$$

Based on (4), eight eigenvalues can be determined. The five eigenvalues are $\lambda_1 = J_{11} = -(\mu_1+\xi)$, $\lambda_2 = J_{22} = -\mu_1$, $\lambda_3 = J_{66} = -\mu_1$, $\lambda_4 = J_{77} = -\mu_2$, $\lambda_5 = J_{88} = \alpha_2\frac{\Lambda_V}{\mu_2} - \delta_2 - \mu_2$. Four of them are found to be negative. Assume $\lambda_5 < 0$. The other three eigenvalues of (4) can be determined as follows:

$$\lambda_6 + \lambda_7 + \lambda_8 = -a_1,$$

$$\lambda_6\lambda_7 + \lambda_7\lambda_8 + \lambda_6\lambda_8 = a_2,$$

$$\lambda_6\lambda_7\lambda_8 = -a_3. \tag{5}$$

As $a_1 > 0$, then $\lambda_6 + \lambda_7 + \lambda_8 < 0$. Let $\lambda_6 < 0$. The disease-free equilibrium $\boldsymbol{T_0}$ stability will be dependent on the values of λ_7 and λ_8. If $\lambda_7 < 0$ and $\lambda_8 < 0$, then the equilibrium is stable. But, if $\lambda_7 > 0$ and $\lambda_8 > 0$, then the disease-free equilibrium $\boldsymbol{T_0}$ is not stable.

If $\mathcal{R}_0 < 1$, then $a_2 > 0$ and $a_3 > 0$ (in equation (4)). As $a_2 > 0$ and $a_3 > 0$, then

$$\lambda_6(\lambda_7 + \lambda_8) + \lambda_7\lambda_8 > 0 \quad \text{and} \quad \lambda_6\lambda_7\lambda_8 < 0. \tag{6}$$

Since $\lambda_6 < 0$, by condition (6),

$$\lambda_7\lambda_8 > 0 \quad \text{and} \quad \lambda_7 + \lambda_8 < 0. \tag{7}$$

This implies that $\lambda_7 < 0$ and $\lambda_8 < 0$. This concludes that if $\mathcal{R}_0 < 1$, then disease-free equilibrium $\boldsymbol{T_0}$ is local asymptotically stable.

Next, if $\mathcal{R}_0 > 1$, then the disease-free equilibrium $\boldsymbol{T_0}$ will be proved not stable. Based on equation (3), if $\mathcal{R}_0 > 1$, then $a_3 < 0$ (in equation (4)). As $a_3 < 0$, the inequity (5) implies

$$\lambda_6\lambda_7\lambda_8 > 0. \tag{8}$$

If condition (8) holds, then $\lambda_7\lambda_8 < 0$ given that $\lambda_6 < 0$, which concludes that λ_7 and λ_8 must have opposite signs. As a consequence, either the value of λ_7 or λ_8 must be positive, which concludes that the disease-free equilibrium $\boldsymbol{T_0}$ is unstable.

As a conclusion, the disease-free equilibrium $\boldsymbol{T_0}$ for system (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

Theorem 2. *For system* (1), *the disease-free equilibrium* $\boldsymbol{T_0}$ *exists. Moreover, endemic equilibrium* $\boldsymbol{T}^*$ *is unique and positive if and only if* $\mathcal{R}_0 > 1$.

Proof. From equation (2), we have

$$E_H^*(A_1 E_H^{*2} + A_2 E_H^* + A_3) = 0, \tag{9}$$

where

$$A_1 = \frac{\alpha_1\alpha_1(1-l)}{\mu_1(\mu_1+\xi)}\left(\frac{\mu_1(\mu_1+\xi)(k+\sigma_1+\mu_1)\mathcal{R}_0}{\alpha_1\Lambda_H((1-l)\xi+\mu_1)}\right)^2,$$

$$\begin{aligned} A_2 = {} & ((1-l)(\mu_1+\xi)(k+\sigma_1+\mu_1)(\gamma_r+\sigma_2+\mu_1+\delta_1)(\mu_1+\delta_1+\gamma) \\ & + \mu_1(k+\sigma_1+\mu_1)(\gamma_r+\sigma_2+\mu_1+\delta_1)(\mu_1+\delta_1+\gamma) \\ & - \alpha_1\Lambda_H(1-l)(\sigma_1(\mu_1+\sigma_1+\gamma)+\eta k(\gamma_r+\sigma_2+\mu_1+\delta_1) \\ & + \eta\sigma_2\sigma_1))\frac{\mathcal{R}_0}{\Lambda_H((1-l)\xi+\mu_1)}, \end{aligned}$$

$$A_3 = 1-\mathcal{R}_0.$$

Based on (9), three values of E_H^* can be obtained. The first root was found to be $E_H^* = 0$. This gives $I_H^* = 0$, $Q_H^* = 0$, $R_H^* = 0$, $S_H^* = \frac{\Lambda_H}{\mu_1+\xi}$, $V_H^* = \frac{\Lambda_H\xi}{(\mu_1+\xi)\mu_1}$, $S_V^* = \frac{\Lambda_V}{\mu_2}$ and $I_V^* = 0$. As a consequence, the existence of the disease-free equilibrium $\boldsymbol{T_0}$ was successfully proved. Other two roots of (9) can be determined as follows:

$$E_{H_1}^* + E_{H_2}^* = -\frac{A_2}{A_1},$$

$$E_{H_1}^* E_{H_2}^* = \frac{A_3}{A_1}. \tag{10}$$

The value of A_3 in (9) was defined previously, i.e., $A_3 = 1 - \mathcal{R}_0$. If $\mathcal{R}_0 > 1$, then $A_3 < 0$. As $A_1 > 0$ (in equation (9)), then $E^*_{H_1} E^*_{H_2} < 0$ given that $A_3 < 0$, which concludes that $E^*_{H_1}$ and $E^*_{H_2}$ must have opposite signs. As a consequence, either the value of $E^*_{H_1}$ or $E^*_{H_2}$ must be positive. This concludes that there is only one positive root E^*_H so that S^*_H, V^*_H, I^*_H, Q^*_H, R^*_H, S^*_V and I^*_V exist and are positive unique.

If the endemic equilibrium $\boldsymbol{T}^*$ is unique and positive, then $\mathcal{R}_0 > 1$.

If $\mathcal{R}_0 < 1$, then $A_2 > 0$ and $A_3 = 1 - R_0 > 0$ (in equation (9)). As $A_2 > 0$, then $E^*_{H_1} + E^*_{H_2} < 0$ and $E^*_{H_1} E^*_{H_2} > 0$ given that $A_3 > 0$, which concludes that $E^*_{H_1} < 0$ and $E^*_{H_2} < 0$. As a consequence, there is not positive endemic equilibrium $\boldsymbol{T}^*$. This concludes that endemic equilibrium $\boldsymbol{T}^*$ is positive and unique, and therefore $\mathcal{R}_0 > 1$.

If $\mathcal{R}_0 = 1$, then $A_2 > 0$ and $A_3 = 1 - \mathcal{R}_0 = 0$ (in equation (9)). As $A_2 > 0$, then $E^*_{H_1} + E^*_{H_2} < 0$ and $E^*_{H_1} E^*_{H_2} = 0$ given that $A_3 = 0$, which concludes that one of the roots of (9) is zero and the other root is negative. As a consequence, there is not positive unique endemic equilibrium $\boldsymbol{T}^*$. This concludes that endemic equilibrium $\boldsymbol{T}^*$ is positive unique if $\mathcal{R}_0 > 1$.

As a result, the supposition is wrong. It is proven that if the endemic equilibrium $\boldsymbol{T}^*$ is unique and positive, then $\mathcal{R}_0 > 1$.

As a consequence, the endemic equilibrium $\boldsymbol{T}^*$ is unique and positive if and only if $\mathcal{R}_0 > 1$ was successfully proved. □

Theorem 3. *If $\mathcal{R}_0 > 1$, then the endemic equilibrium $\boldsymbol{T}^*$ is locally asymptotically stable.*

Proof. Based on Castillo-Chaves and Song [1], let $\varphi = \alpha_1$ be the bifurcation parameter. Based on condition $\mathcal{R}_0 = 1$, we have

$$\varphi = \varphi^*$$

$$= \frac{\mu_1(\mu_1 + \delta_1 + \gamma)(\mu_1 + \xi)(k + \sigma_1 + \mu_1)(\gamma_r + \sigma_2 + \mu_1 + \delta_1)}{\Lambda_H((1-l)\xi + \mu_1)(k\eta(\gamma_r + \delta_1 + \sigma_2 + \mu_1) + \sigma_1(\mu_1 + \delta_1 + \gamma + \eta\sigma_2))}. \quad (11)$$

Consider $\boldsymbol{J_{T_0}}$. Disease-free equilibrium $\boldsymbol{T_0}$ has one zero eigenvalue and seven negative eigenvalues if $\mathcal{R}_0 = 1$ or $\varphi = \varphi^*$. The zero eigenvalue has right eigenvector $(u_1, u_2, u_3, u_4, u_5, u_6, u_7, u_8)$ and left eigenvector $(v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8)$. As indicated previously that u_4 is arbitrary positive, then $u_7 = u_8 = 0$,

$$u_1 = -\left(\frac{\alpha_1\Lambda_H(\gamma + \delta_1 + \mu_1)\sigma_1 + \alpha_1\eta\Lambda_H k(\gamma_r + \delta_1 + \sigma_2 + \mu_2) + \alpha_1\eta\Lambda_H\sigma_2\sigma_1}{(\mu_1 + \xi)(\mu_1 + \xi)(\gamma + \delta_1 + \mu_1)\sigma_1} \right) u_4 < 0,$$

$$u_2 = -\left(\frac{\alpha_1\Lambda_H\xi((\gamma + \delta_1 + \mu_1 + \sigma_2\eta)\sigma_1 + k\eta(\gamma_r + \delta_1 + \sigma_2 + \mu_2)) \cdot (\mu_1 + (1-l)(\mu_1 + \xi))}{(\mu_1 + \xi)(\mu_1 + \xi)(\gamma + \delta_1 + \mu_1)\sigma_1\mu_1\mu_1} \right) u_4 < 0,$$

$$u_3 = \left(\frac{\gamma_r + \delta_1 + \sigma_2 + \mu_1}{\sigma_1} \right) u_4 > 0,$$

$$u_5 = \left(\frac{k(\gamma_r + \delta_1 + \sigma_2 + \mu_1) + \sigma_1\sigma_2}{(\gamma + \delta_1 + \mu_1)\sigma_1} \right) u_4 > 0,$$

$$u_6 = \left(\frac{\gamma_r}{\mu_1} + \frac{\gamma k(\gamma_r + \delta_1 + \sigma_2 + \mu_1) + \gamma\sigma_2\sigma_1}{\mu_1\sigma_1(\gamma + \delta_1 + \mu_1)} \right) u_4 > 0,$$

as indicated previously that $v_8 > 0$ is arbitrary positive, then $v_1 = v_2 = v_6 = v_7 = 0$,

$$v_3 = -\frac{(\alpha_2\Lambda_V - \mu_2\delta_2 - \mu_2\mu_2)\mu_1(\mu_1 + \xi)}{\mu_2(\alpha_1(1-l)\Lambda_H\xi + \mu_1\alpha_1\Lambda_H)}v_8 > 0,$$

$$v_4 = \begin{pmatrix} -(\alpha_2\Lambda_V - \mu_2\delta_2 - \mu_2\mu_2)(\mu_1 + \xi)\mu_1 \\ \frac{((k + \sigma_1 + \mu_1)(\gamma + \delta_1 + \mu_1) + \eta k)}{(\gamma + \delta_1 + \mu_1)(\alpha_1(1-l)\Lambda_H\xi + \mu_1\alpha_1\Lambda_H)\mu_2\sigma_1} \end{pmatrix} v_8,$$

$$v_5 = -\frac{(\alpha_2\Lambda_V - \mu_2\delta_2 - \mu_2\mu_2)(\mu_1 + \xi)\mu_1\eta}{\mu_2(\gamma + \delta_1 + \mu_1)(\alpha_1(1-l)\Lambda_H\xi + \alpha_1\Lambda_H\mu_1)}v_8 > 0.$$

Define

$$a = \sum_{k,i,j=1}^{8} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\boldsymbol{T_0}, \varphi^*),$$

$$b = \sum_{k,i,j=1}^{8} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(\boldsymbol{T_0}, \varphi^*), \tag{12}$$

where

$$x_1 = S_H, \quad x_2 = V_H, \quad x_3 = E_H, \quad x_4 = I_H,$$

$$x_5 = Q_H, \quad x_6 = R_H, \quad x_7 = S_V, \quad x_8 = I_V,$$

$$f_1 = \frac{dx_1}{dt} = \Lambda_H - \varphi(x_4 + \eta x_5 + x_7)x_1 - \mu_1 x_1 - \xi x_1,$$

$$f_2 = \frac{dx_2}{dt} = \xi x_1 - (1-l)\alpha_1(x_4 + \eta x_5 + x_8)x_2 - \mu_1 x_2,$$

$$f_3 = \frac{dx_3}{dt} = \varphi(x_4 + \eta x_5 + x_8)x_1 - (\mu_1 + \sigma_1 + k)x_3 + (1-l)\varphi(x_4 + \eta x_5 + x_8)x_2,$$

$$f_4 = \frac{dx_4}{dt} = \sigma_1 x_3 - (\mu_1 + \sigma_2 + \delta_1)x_4 - \gamma_r x_4,$$

$$f_5 = \frac{dx_5}{dt} = \sigma_2 x_4 - (\mu_1 + \delta_1 + \gamma)x_5 + kx_3,$$

$$f_6 = \frac{dx_6}{dt} = \gamma x_5 - \mu_1 x_6 + \gamma_r x_4,$$

$$f_7 = \frac{dx_7}{dt} = \Lambda_V - \alpha_2 x_8 x_7 - \mu_2 x_7,$$

$$f_8 = \frac{dx_8}{dt} = \alpha_2 x_8 x_7 - (\mu_2 + \delta_2)x_8.$$

Based on equation (12), we have

$$a = 2v_3u_1u_4\varphi^* + 2v_3u_1u_5\varphi^*\eta + 2v_3u_2u_4(1-l)\varphi^* + 2v_3u_2u_5(1-l)\varphi^*\eta$$

and

$$b = v_3u_4\frac{\Lambda_H(\mu_1 + \xi(1-l))}{\mu_1(\mu_1+\xi)} + v_3u_5\frac{\Lambda_H\eta(\mu_1+\xi(1-l))}{\mu_1(\mu_1+\xi)}.$$

As $v_3, u_4, u_5 > 0$, $u_1, u_2 < 0$, and $\varphi^* > 0$, then $a < 0$. As $u_4, u_5, v_3 > 0$, then $b > 0$. Consequently, when φ changes from $\varphi < \varphi^*$ to $\varphi > \varphi^*$, the disease-free equilibrium $\boldsymbol{T_0}$ changes from stable and becomes unstable, while endemic equilibrium $\boldsymbol{T}^*$ changes from negative and becomes positive and thus becomes local asymptotically stable. As a consequence, the endemic equilibrium $\boldsymbol{T}^*$ is locally asymptotically stable if $\mathcal{R}_0 > 1$. □

4. Numerical Simulation

The numerical simulations were performed to visualize stability properties of the equilibrium points of both $\boldsymbol{T_0}$ and $\boldsymbol{T}^*$ based on the theorem in Section 3. The initial values used are $S_H(0) = 100$, $V_H(0) = 10$, $E_H(0) = 20$, $I_H(0) = 20$, $Q_H(0) = 20$, $R_H(0) = 0$, $S_V(0) = 100$ and $I_V(0) = 10$. The parameter values used in this simulation are $\Lambda_V = 0.65$,

$\alpha_1 = 0.016$, $\eta = 0.8$, $\alpha_2 = 0.02$, $\sigma_1 = 8$, $\sigma_2 = 6$, $\gamma = 0.3$, $\mu_1 = 0.2$, $\mu_2 = 0.6$, $\delta_1 = 0.6$, $\delta_2 = 0.5$ [3]. $k = 14.697$, $\gamma_r = 5$ [5]. $\Lambda_H = 50$ (assumed).

For the disease free equilibrium, additional values of $\xi = 0.39$ and $l = 0.9487$ give $\mathcal{R}_0 = 0.933156 < 1$. But, for the endemic equilibrium, additional values of $\xi = 0.039$ and $l = 0.5487$ give $\mathcal{R}_0 = 2.278 > 1$.

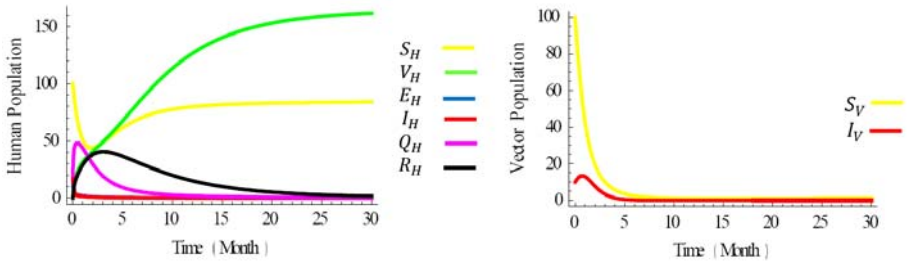


Figure 2. The population dynamics of human and vector for the disease-free equilibrium.

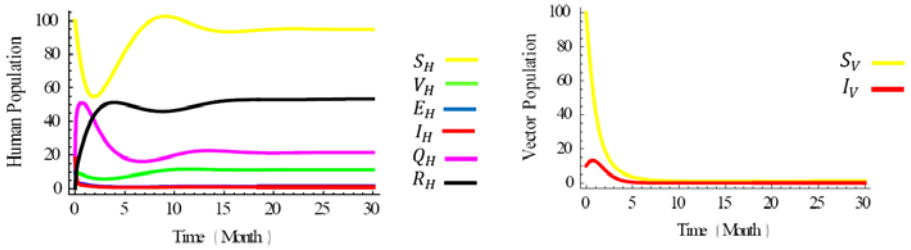


Figure 3. The population dynamics of human and vector for the endemic equilibrium.

Exposed population (E_H) is almost the same with infected population (I_H) (invisible). Figure 2 supports Theorem 1 and Figure 3 supports Theorems 2 and 3. This simulation shows that the system will be stable at around disease-free equilibrium when $\mathcal{R}_0 < 1$ and the system will be stable at around endemic equilibrium when $\mathcal{R}_0 > 1$.

5. Conclusions

This work carried out dynamical analysis for mathematical model of Ebola outbreak to consider vaccination. The results of the model analysis obtained two equilibria, namely, disease-free equilibrium and endemic equilibrium. The basic reproduction number $(\mathcal{R}_0)$ was determined. The disease-free equilibrium is locally asymptotically stable on condition $\mathcal{R}_0 < 1$, whereas the endemic equilibrium is locally asymptotically stable on condition $\mathcal{R}_0 > 1$. The numerical simulation of population dynamics of both humans and vectors showed similar patterns as expected.

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