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## Mathematical analysis of sensitive parameters due to dynamic transmission of Ebola virus disease

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### Abstract

A mathematical model of Ebola virus disease was formulated, it was shown that the model was well- posed, both disease and endemic equilibria for the models were obtained. The models were analysed for stability and it was established that the disease free equilibrium of model is locally asymptotically stable whenever the basic reproduction number is less than unity. Similarly, there exist endemic point when the basic reproduction numbers of Ebola virus disease is greater than unity. The results obtained so far from sensitivity analysis strongly shows that the spread of Ebola virus disease in the population depend on effective contact rate. Conclusively, in the numerical simulation where Runge –Kutta method of order four via MAPPLE (18) software was adopted it shows that the best way to control the Ebola virus disease in the population is to minimize the contact rate.

**Keywords:** Ebola virus disease; Boundedness of solutions; Basic reproduction number; Existence of endemic equilibrium point; Sensitivity indices

### 1. Introduction

Ebola is a deadly virus that attacks healthy cells and replicates itself in a host's body. The virus, previously known as Ebola hemorrhagic fever, is the deadliest pathogen for humans which affected several African countries. [1-10] performed numerical simulation and sensitivity analysis on a mathematical model of Ebola transmission to determine the biological significance of key model parameters in relation to disease transmissions and prevalence. Result from sensitivity analysis affirm that average contacts and transmission rates championed the disease outbreaks. Similarly, a model with multi-intervention strategies was proved to effectively reduce the contact and prevalence of Ebola virus disease than the models with one intervention at a time. They suggests that strategies targeting contact reduction (such as education and isolation) and those that focus on recovery rates (such as prompt treatment of the infected persons) can be successful in curtailing the Ebola epidemic but they did not consider Ebola-malaria co-infections. [9-19] proposed a mathematical model of Ebola virus (EBOV) using susceptible exposed infected recovered (SEIR) model, their model, the population is affected by animals, EBOV is an infectious agent causing hemorrhagic fever, a severe infectious disease characterized by high fever and bleeding, in humans and some monkeys. The conditions to investigate all possible equilibria of the model in terms of the basic reproduction number (local and global stability) was calculated. [4] formulated a compartmental model of susceptible, exposed, undetected, detected and recovered model to study the dynamical spread of Ebola virus disease which conclude that the rate of public enlightenment and availability of isolation centers can reduce the spread of Ebola virus disease. [10-20] considered a deterministic model of Ebola virus disease incorporating contact tracing and quarantine as interventions. The model was analysed for the existence and

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stability of disease free equilibrium and endemic equilibrium points and numerical simulations were carried out to examine the impact of contact tracing and quarantine measures on the transmission dynamics of Ebola virus disease, the result indicates that Ebola virus disease could be eliminated faster when contact tracing and quarantine measures were implemented together.[13-17] developed a susceptible-exposed-infected-treatment (SEIT) model of Ebola virus transmission. They assumed that some treated individuals will die of the disease while some will recover and loose immunity. [3-19] formulated a susceptible infected-recovered-death model to study the spread of Ebola virus disease transmission in Sub-Saharan African countries. It was assumed that recovered individuals lost immunity and become susceptible again with natural death in susceptible infected recovered (SIR) compartments. [1-2, 20] worked on mathematical analysis of effects of isolation on Ebola transmission dynamics, it shows that if the detection rate of infected undetected individuals is sufficiently large, then the isolation technique can lead to elimination of Ebola in the population, this work is modified by adding the treated class into their model and performed the stability analysis and sensitivity indices on formulated model.

## 2. Descriptions of Mathematical Model of Ebola Virus Disease

A mathematical model is proposed to consider the dynamics spread of Ebola virus disease The human population is divided into six classes, susceptible individuals  $S_H(t)$ , Ebola virus disease latently infected individuals  $L_E(t)$ , Ebola virus disease infected undetected Individuals  $I_U(t)$ , infected detected Ebola virus disease individuals  $I_D(t)$ , individuals under treatment for Ebola virus disease  $I_T(t)$ , individuals isolated for Ebola virus disease  $J$ .

Therefore the total population of human denoted

$$N_H(t) = S_H(t) + L_E(t) + I_U(t) + I_D(t) + I_T(t) + J(t) \quad (1)$$

It assumed that susceptible humans are recruited into the population at the constant rate  $\pi_H$ , it acquires Ebola virus diseases infection following the effective contact with infected undetected, infected detected, infected treated and isolated individual. The population increases by fraction of isolated individual who move to susceptible after test negative of Ebola virus disease infection but natural death occurs in all human (at the rate  $\mu$ ), which decreases the population. The force of infection associated with Ebola virus disease, denoted by;

$$\lambda_E = \beta_E \left( \frac{I_U + \eta_D I_D + \eta_T I_T + \eta_J J}{N_H} \right) \quad (2)$$

where  $\beta_E$  represents the effective contact rate,  $\eta_D$  is modification parameter comparing the individual transmissibility of detected infected individuals in relationship to latently infected. Since detected individuals are under treatment and isolation, it is intuitive to assume that  $\eta_D \leq 1$ . Similarly  $\eta_T$  and  $\eta_J$  are modification parameters comparing the transmissibility of infected individuals in the treated class and isolated class respectively. Putting these assumptions together, the model is given as;

$$\left. \begin{aligned} \frac{dS_H}{dt} &= \pi_H - \lambda_E S_H - \mu S_H + \alpha \theta J \\ \frac{dL_E}{dt} &= \varepsilon_1 \lambda_E S_H - (\kappa_E + \sigma_1 + \mu) L_E + \phi_2 I_T + (1 - \alpha) \theta J \\ \frac{dI_U}{dt} &= (1 - \varepsilon_1) \lambda_E S_H + \omega_1 \kappa_E L_E - (\gamma_{UE} + \mu + \delta_{UE}) I_U \\ \frac{dI_D}{dt} &= (1 - \omega_1) \kappa_E L_E - (\tau_1 + \mu + \delta_{DE} + \sigma_2) I_D + \gamma_{UE} I_U \\ \frac{dI_T}{dt} &= \tau_1 I_D - (\phi_2 + \mu) I_T \\ \frac{dJ}{dt} &= \sigma_1 L_E + \sigma_2 I_D - (\mu + \delta_j) J - \theta J \end{aligned} \right\} \quad (3)$$

where  $\lambda_E$  is the force of infection,  $\mu$  is natural death rate,  $\alpha$  is the rate of certified Ebola free,  $\theta$  is the discharge rate,  $\kappa_E$  is progression rate,  $\sigma_1$  is isolation rate,  $\omega_1$  fraction of detection rate,  $\gamma_{UE}$  is the detection rate,  $\delta$  is the disease death rate,  $\tau_1$  is the treatment rate and  $\varepsilon_1$  is fraction of individual with low immunity.

### 2.1 Boundedness Solutions of Ebola Virus Disease

For the Ebola virus disease transmission model (3) to be epidemiologically meaningful, it is important to prove that all solutions with nonnegative initial data will remain nonnegative for all time  $t \geq 0$ .

Theorem 1.

If  $S_H(0), L_E(0), I_U(0), I_D(0), I_T(0)$  and  $J(0)$  are non-negative, then the solutions  $S_H, L_E, I_U, I_D, I_T$  and  $J$  of the Ebola virus disease model (3) are non-negative for all  $t \geq 0$ .

Proof:

Consider the biologically-feasible region,  $\Pi = \left\{ (S_H, L_E, I_U, I_D, I_T, J) \in \mathcal{R}_+^6 : N_H \leq \frac{\pi_H}{\mu} \right\}$ , it will be proved that  $\Pi$  is positively invariant.

The total population of Ebola virus disease transmission of sub-model is obtained by adding all the model equation (9) is given by;

$$\frac{dN_H}{dt} = \pi_H - \mu N_H - (\delta_{UE} I_U + \delta_{DE} I_D + \delta_j J) \quad (4)$$

In the absence of mortality due to Ebola virus disease, equation (4) become

$$\frac{dN_H}{dt} \leq \pi_H - \mu N_H \quad (5)$$

Solve (5) and re-arrange, upon taking the limit as  $t \rightarrow \infty$  obtain,

$$\left. \begin{aligned} \frac{dS_H}{dt} &= \pi_H - \lambda_E S_H - \mu S_H + \alpha \theta J \\ \frac{dL_E}{dt} &= \varepsilon_1 \lambda_E S_H - (\kappa_E + \sigma_1 + \mu) L_E + \phi_2 I_T + (1 - \alpha) \theta J \\ \frac{dI_U}{dt} &= (1 - \varepsilon_1) \lambda_E S_H + \omega_1 \kappa_E L_E - (\gamma_{UE} + \mu + \delta_{UE}) I_U \\ \frac{dI_D}{dt} &= (1 - \omega_1) \kappa_E L_E - (\tau_1 + \mu + \delta_{DE} + \sigma_2) I_D + \gamma_{UE} I_U \\ \frac{dI_T}{dt} &= \tau_1 I_D - (\phi_2 + \mu) I_T \\ \frac{dJ}{dt} &= \sigma_1 L_E + \sigma_2 I_D - (\mu + \delta_j) J - \theta J \end{aligned} \right\} \quad (3)$$

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## 2.2 Boundedness Solutions of Ebola Virus Disease

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$$\frac{dN_H}{dt} \leq \pi_H - \mu N_H \quad (5)$$

Solve (5) and re-arrange, upon taking the limit as  $t \rightarrow \infty$  obtain,

$$R_E = \beta_E \frac{\left( \begin{aligned} & a_1 a_7 b_1 \eta_D \gamma_{UE} + a_1 a_7 \eta_T \gamma_{UE} \tau_1 + a_1 b_1 \eta_j \gamma_{UE} \sigma_2 - a_2 b_1 \eta_D \gamma_{UE} \sigma_1 - a_2 \eta_T \gamma_{UE} \sigma_1 \tau_1 + \eta_j \gamma_{UE} \phi_2 \sigma_1 \tau_1 - \\ & \varepsilon_1 a_1 a_6 a_7 b_1 + \varepsilon_1 a_2 a_5 b_1 \sigma_2 + \varepsilon_1 a_2 a_6 b_1 \sigma_1 + \varepsilon_1 a_5 a_7 \phi_2 \tau_1 + a_3 a_6 a_7 b_1 \varepsilon_1 - \varepsilon_1 a_1 a_7 b_1 \eta_D \gamma_{UE} - \varepsilon_1 a_1 a_7 \eta_T \gamma_{UE} \tau_1 \\ & - \varepsilon_1 a_1 b_1 \eta_j \gamma_{UE} \sigma_2 + \varepsilon_1 a_2 b_1 \eta_D \gamma_{UE} \sigma_1 + \varepsilon_1 a_2 \eta_T \gamma_{UE} \sigma_1 \tau_1 - \varepsilon_1 \eta_j \gamma_{UE} \phi_2 \sigma_1 \tau_1 + \varepsilon_1 a_4 a_5 b_1 \eta_j \sigma_2 + \varepsilon_1 a_4 a_6 b_1 \eta_j \sigma_1 \\ & a_1 a_6 a_7 b_1 - a_2 a_5 \sigma_2 b_1 - a_2 a_6 b_1 \sigma_1 - a_5 a_7 \phi_2 \tau_1 \end{aligned} \right)}{a_1 a_4 a_6 a_7 b_1 - a_2 a_3 b_1 \gamma_{UE} \sigma_2 - a_2 a_4 a_5 b_1 \sigma_2 - a_2 a_4 a_6 b_1 \sigma_1 - a_3 a_7 \gamma_{UE} \phi_2 \tau_1 - a_4 a_5 a_7 \phi_2 \tau_1}$$

The reproduction number for Ebola virus disease is given as  $R_E$ , where denotes the spectral radius of the dominant eigenvalue of the next generation matrix. Therefore, this measure the average number of new infectious generated by a single infectious individual in a population consisting of susceptible.

### 2.3 Local Stability of Disease Free of Ebola Virus Disease

**Theorem 2.** Disease free equilibrium point is locally asymptotically stable if  $R_E < 1$  and unstable if  $R_E > 1$ . Then the theorem implies the disease can be eliminated from the community.

**Proof:** To prove local stability of disease free equilibrium, obtain the Jacobian matrix of the system (3) at disease free equilibrium.

$$J(E_1) = \begin{vmatrix} -\mu & 0 & -\beta_E & -\beta_E \eta_D & -\beta_E \eta_T & \alpha\theta - \beta_E \eta_j \\ 0 & -K_1 & \varepsilon_1 \beta_E & \varepsilon_1 \beta_E \eta_D & \varepsilon_1 \beta_E \eta_T + \phi_2 & \varepsilon_1 \beta_E \eta_j + (1-\alpha)\theta \\ 0 & \omega_1 \kappa_E & -K_2 + (1-\varepsilon_1)\beta_E & (1-\varepsilon_1)\beta_E \eta_D & (1-\varepsilon_1)\beta_E \eta_T & (1-\varepsilon_1)\beta_E \eta_j \\ 0 & (1-\omega_1)\kappa_E & \gamma_{UE} & -K_3 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & -K_4 & 0 \\ 0 & \sigma_1 & 0 & \sigma_2 & 0 & -K_5 \end{vmatrix}$$

For simplicity

$$K_1 = \kappa_E + \mu + \sigma_1, K_2 = \gamma_{UE} + \mu + \delta_{UE}, K_3 = \tau_1 + \mu + \delta_{DE} + \sigma_2, K_4 = \phi_2 + \mu, K_5 = \mu + \delta_j + \theta$$

Therefore, the eigenvalue of the Jacobian matrix are the solution of the characteristic equation  $|J - \lambda I| = 0$

Expand along the first column obtain

$$-\mu - \lambda \begin{vmatrix} -K_1 - \lambda & \varepsilon_1 \beta_E & \varepsilon_1 \beta_E \eta_D & \varepsilon_1 \beta_E \eta_T + \phi_2 & \varepsilon_1 \beta_E \eta_j + (1-\alpha)\theta \\ \omega_1 \kappa_E & -K_2 + (1-\varepsilon_1)\beta_E - \lambda & (1-\varepsilon_1)\beta_E \eta_D & (1-\varepsilon_1)\beta_E \eta_T & (1-\varepsilon_1)\beta_E \eta_j \\ (1-\omega_1)\kappa_E & \gamma_{UE} & -K_3 - \lambda & 0 & 0 \\ \sigma_1 & 0 & \sigma_2 & 0 & -K_5 - \lambda \end{vmatrix} = 0$$

$$\Rightarrow \lambda_1 = \mu, \text{ or}$$

$$\begin{vmatrix} -\mu - \lambda & 0 & -\beta_E & -\beta_E \eta_D & -\beta_E \eta_T & \alpha\theta - \beta_E \eta_J \\ 0 & -K_1 - \lambda & \varepsilon_1 \beta_E & \varepsilon_1 \beta_E \eta_D & \varepsilon_1 \beta_E \eta_T + \phi_2 & \varepsilon_1 \beta_E \eta_J + (1-\alpha)\theta \\ 0 & \omega_1 \kappa_E & -K_2 + (1-\varepsilon_1)\beta_E - \lambda & (1-\varepsilon_1)\beta_E \eta_D & (1-\varepsilon_1)\beta_E \eta_T & (1-\varepsilon_1)\beta_E \eta_J \\ 0 & (1-\omega_1)\kappa_E & \gamma_{UE} & -K_3 - \lambda & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & -K_4 - \lambda & 0 \\ 0 & \sigma_1 & 0 & \sigma_2 & 0 & -K_5 - \lambda \end{vmatrix} = 0$$

$$\begin{vmatrix} -K_1 - \lambda & \varepsilon_1 \beta_E & \varepsilon_1 \beta_E \eta_D & \varepsilon_1 \beta_E \eta_T + \phi_2 & \varepsilon_1 \beta_E \eta_J + (1-\alpha)\theta \\ \omega_1 \kappa_E & -K_2 + (1-\varepsilon_1)\beta_E - \lambda & (1-\varepsilon_1)\beta_E \eta_D & (1-\varepsilon_1)\beta_E \eta_T & (1-\varepsilon_1)\beta_E \eta_J \\ (1-\omega_1)\kappa_E & \gamma_{UE} & -K_3 - \lambda & 0 & 0 \\ \sigma_1 & 0 & \sigma_2 & 0 & -K_5 - \lambda \end{vmatrix} = 0$$

Again expand along second column obtain

$$\left\{ \begin{array}{l} \omega_1 \kappa_E \quad (1-\varepsilon_1)\beta_E \eta_D \quad (1-\varepsilon_1)\beta_E \eta_T \quad (1-\varepsilon_1)\beta_E \eta_J \\ (1-\omega_1)\kappa_E \quad -K_3 - \lambda \quad 0 \quad 0 \\ 0 \quad \tau_1 \quad -K_4 - \lambda \quad 0 \\ \sigma_1 \quad \sigma_2 \quad 0 \quad -K_5 - \lambda \end{array} \right\} - \varepsilon_1 \beta_E$$

$$-K_2 + (1-\varepsilon_1)\beta_E - \lambda)$$

$$\begin{vmatrix} -K_1 - \lambda & \varepsilon_1 \beta_E \eta_D & \varepsilon_1 \beta_E \eta_T + \phi_2 & \varepsilon_1 \beta_E \eta_J + (1-\alpha)\theta \\ (1-\omega_1)\kappa_E & K_3 - \lambda & 0 & 0 \\ 0 & \tau_1 & -K_4 - \lambda & 0 \\ \sigma_1 & \sigma_2 & 0 & -K_5 - \lambda \end{vmatrix}$$

$$-\gamma_{UE}$$

$$\begin{bmatrix} -K_1 - \lambda & \varepsilon_1 \beta_E \eta_D & \varepsilon_1 \beta_E \eta_T + \phi_2 & \varepsilon_1 \beta_E \eta_J + (1 - \alpha)\theta \\ \omega_1 K_E & (1 - \varepsilon_1) \beta_E \eta_D & (1 - \varepsilon_1) \beta_E \eta_T & (1 - \varepsilon_1) \beta_E \eta_J \\ 0 & \tau_1 & -K_4 - \lambda & 0 \\ \sigma_1 & \sigma_2 & 0 & -K_5 - \lambda \end{bmatrix} = 0$$

The characteristics polynomial of the above matrix is given by;

$$B_5 \lambda^5 + B_4 \lambda^4 + B_3 \lambda^3 + B_2 \lambda^2 + B_1 \lambda + B_0 = 0 \tag{7}$$

where;

$$B_5 = 1,$$

$$B_4 = K_2 + K_4 + K_5 + K_3 + K_1$$

$$B_3 = -q_1 q_4 + b_1 K_2 + b_2 + q_3 \sigma_1 - \gamma_{UE} C_3 + a_3$$

$$\begin{aligned} B_2 = & -C_1 q_1 q_4 + K_2 b_2 + b_3 - q_3 \sigma_1 K_2 - q_3 \sigma_1 K_3 - q_3 \sigma_1 K_4 \\ & + q_3 q_4 \sigma_2 - q_2 q_4 \tau_1 + \gamma_{UE} - \gamma_{UE} \tau_1 C_5 - \gamma_{UE} C_3 d_1 - \sigma_2 \gamma_{UE} C_7 \\ & + a_4 + a_3 K_4 - 2K_5 a_3 \end{aligned}$$

$$\begin{aligned} B_1 = & q_1 q_4 C_2 + K_2 b_3 + b_4 - q_3 K_2 K_3 \sigma_1 - q_3 K_2 K_4 \sigma_1 - q_3 \sigma_1 K_3 K_4 - q_3 q_4 \sigma_2 K_4 - q_3 q_4 \tau_2 \sigma_2 - q_2 q_4 \tau_1 K_2 \\ & - q_2 q_4 \tau_1 K_5 + \sigma_1 C_7 C_1 \gamma_{UE} - \gamma_{UE} \sigma_1 C_6 C_3 + \gamma_{UE} C_2 C_4 \tau_1 + 2\gamma_{UE} K_5 + \gamma_{UE} C_2 C_6 \sigma_2 - \gamma_{UE} C_5 K_1 \tau_1 - \gamma_{UE} \tau_1 C_5 K_5 \\ & - \gamma_{UE} C_3 d_2 - \gamma_{UE} C_7 K_1 \sigma_2 - \gamma_{UE} C_7 K_4 \sigma_2 + a_5 + a_4 K_4 + a_4 K_5 + 2a_3 K_5 K_4 - a_2 - a_1 \end{aligned}$$

The Routh Hurwitz criterion will be applied to determine the nature of the roots of the polynomial, which state that the roots of the polynomial will be negative if the coefficient

$B_i$  (where  $i=0, 1, 2, \dots, 5$ ) are all positive and that the Hurwitz matrices are greater than zero. The coefficient  $B_5 > 0$ ,  $B_4 > 0$ ,  $B_3 > 0$ ,  $B_2 > 0$ ,  $B_1 > 0$ .

Also, the Hurwitz matrices are as follow:

$$\det(H_2) = \begin{pmatrix} B_1 & 1 \\ B_3 & B_2 \end{pmatrix} = B_1 B_2 - B_3 > 0, \det(H_3) = \begin{pmatrix} B_1 & 1 & 0 \\ B_3 & B_2 & B_1 \\ 0 & 0 & B_3 \end{pmatrix} = B_1 B_2 B_3 - B_3^2 > 0 \Rightarrow B_1 B_2 - B_3 > 0 \text{ and}$$

$$\det(H_4) = \begin{pmatrix} B_1 & 1 & 0 & 0 \\ B_3 & B_2 & B_1 & 1 \\ 0 & B_4 & B_3 & B_2 \\ 0 & 0 & 0 & B_4 \end{pmatrix} = B_3(B_2 B_1 - B_3) - B_4 B_1^2 > 0.$$

Now, that all determinants of the Hurwitz matrices are positive, then all the eigenvalues of the Jacobian matrix have negative real roots when  $R_E < 1$ , therefore, the disease free equilibrium is locally asymptotically stable.

### 2.4 Existence of Endemic Equilibrium Point of Ebola Virus Disease

Here we consider the possible existence and stability of endemic (positive) equilibria of the model (3) (that is, a case, where equilibria of one of the infected components of the model is non-zero) will be explored.

Let  $E_1^{**} = (S_H^{**}, L_E^{**}, I_U^{**}, I_D^{**}, I_T^{**}, J^{**})$  represents any arbitrary endemic equilibrium of model (3) so that  $N_H^{**} = S_H^{**} + L_E^{**} + I_U^{**} + I_D^{**} + I_T^{**} + J^{**}$ . Solve (3) at steady state obtain  $N_H^{**} = S_H^{**} + L_E^{**} + I_U^{**} + I_D^{**} + I_T^{**} + J^{**}$

$$S_H^{**} = \frac{\pi_H + \alpha \theta J^{**}}{\lambda_E^{**} + \mu}, L_E^{**} = \frac{\varepsilon_1 \lambda_E^{**} S_H^{**} + \phi^2 I_T^{**} + (1 - \alpha) \theta J^{**}}{C_2}, I_U^{**} = \frac{(1 - \varepsilon_1) \lambda_E^{**} S_H^{**} + \omega_1 \kappa_E L_E^{**}}{C_3}, \quad (8)$$

$$I_D^{**} = \frac{(1 - \omega_1) \kappa_E L_E^{**} + \gamma_{UE} I_U^{**}}{C_4}, I_T^{**} = \frac{\tau_1 I_D^{**}}{C_5}, J^{**} = \frac{\sigma_1 L_E^{**} + \sigma_2 I_D^{**}}{C_6}$$

Recall that the force of infection  $\lambda_E$ , defined in (3) can be expressed at endemic steady state as

$$\lambda_E^{**} = \beta_E \left( \frac{I_U^{**} + \eta_D I_D^{**} + \eta_T I_T^{**} + \eta_J J^{**}}{N_H^{**}} \right) \quad (9)$$

For computational simplicity (8) can be re-write in terms of  $\lambda_E^{**} S_H^{**}$  below

$$L_E^{**} = \frac{(C_3 C_4 M_1 + M_2 K_3) \lambda_E^{**} S_H^{**}}{C_3 C_4 M_4 - M_2 K_1 + C_3 C_4 M_3} = P_1 \lambda_E^{**} S_H^{**}, I_U^{**} = \frac{(K_3 + \omega_1 \kappa_E P_1) \lambda_E^{**} S_H^{**}}{C_3} = P_5 \lambda_E^{**} S_H^{**},$$

$$I_D^{**} = \frac{(K_1 P_1 + K_3) \lambda_E^{**} S_H^{**}}{C_3 C_4} = P_2 \lambda_E^{**} S_H^{**}, I_T^{**} = \frac{(\tau_1 K_1 P_1 + K_3 \tau_1) \lambda_E^{**} S_H^{**}}{C_3 C_4 C_5} = P_4 \lambda_E^{**} S_H^{**} \quad (10)$$

$$J^{**} = \frac{(K_2 P_1 + K_3) \lambda_E^{**} S_H^{**}}{C_3 C_4 C_6} = P_3 \lambda_E^{**} S_H^{**}.$$

Where  $P_1 = \frac{C_3 C_4 M_1 + M_2 K_3}{C_3 C_4 M_4 - M_2 K_1 + C_3 C_4 M_3}, P_2 = \frac{K_1 P_1 + K_3}{C_3 C_4}, P_4 = \frac{\tau_1 K_1 P_1 + K_3 \tau_1}{C_3 C_4 C_5},$

$$P_3 = \frac{K_2 P_1 + K_3}{C_3 C_4 C_6}, P_5 = \frac{K_3 + \omega_1 \kappa_E P_1}{C_3}$$

Now, substituting the above expression into (10) gives,

$$\lambda_E^{**} S_H^{**} \neq 0 \quad \lambda_E^{**} (S_H^{**} + P_1 \lambda_E^{**} S_H^{**} + P_5 \lambda_E^{**} S_H^{**} + P_2 \lambda_E^{**} S_H^{**} + P_4 \lambda_E^{**} S_H^{**} + P_3 \lambda_E^{**} S_H^{**}) \quad (11)$$

$$= \beta_E \lambda_E^{**} (P_5 S_H^{**} + \eta_D P_2 S_H^{**} + \eta_T P_4 S_H^{**} + \eta_J P_3 S_H^{**})$$

Dividing each term of (11) by  $\lambda_E^{**} S_H^{**}$  (and noting that at the endemic steady-state,) obtain  $1 + P_6 \lambda_E^{**} = R_E$

where  $P_6 = P_1 + P_2 + P_3 + P_4 + P_5 \geq 0$  and  $R_E = \beta_E (P_5 + \eta_D P_2 + \eta_T P_4 + \eta_J P_3)$

Hence,  $\lambda_E^{**} = \frac{R_E - 1}{P_6} > 0$ , whenever  $R_E > 1$  (12)



The components of  $E_1$  can be obtained by substituting the unique value of  $\lambda_E^{**}$ , obtain in (12) into the expression in (3), then the result is established.

**Lemma 1** The model (3) has a unique endemic (positive) equilibrium, given by  $E_1$ , whenever  $R_E > 1$ .

**2.5 Sensitivity Analysis of Model Parameters for Ebola Virus Disease**

Sensitivity analysis was carried out to determine the model robustness to parameter values. This helps us to identify the parameters that have a significant to the basic reproduction number  $R_E$ . Also, Sensitivity indices helps in developing efficient and effective intervention strategies in the control of Ebola virus disease in the community. This is calculated using normalized forward sensitivity method, which is defined as the ratio of the relative change in  $R_E$  to the relative change in the parameter “P”:

$$Z_P^{R_E} = \frac{\partial R_E}{\partial P} \times \frac{P}{R_E}.$$

**Table 1** Sensitivity values of basic reproduction number  $R_E$  of Ebola virus disease model

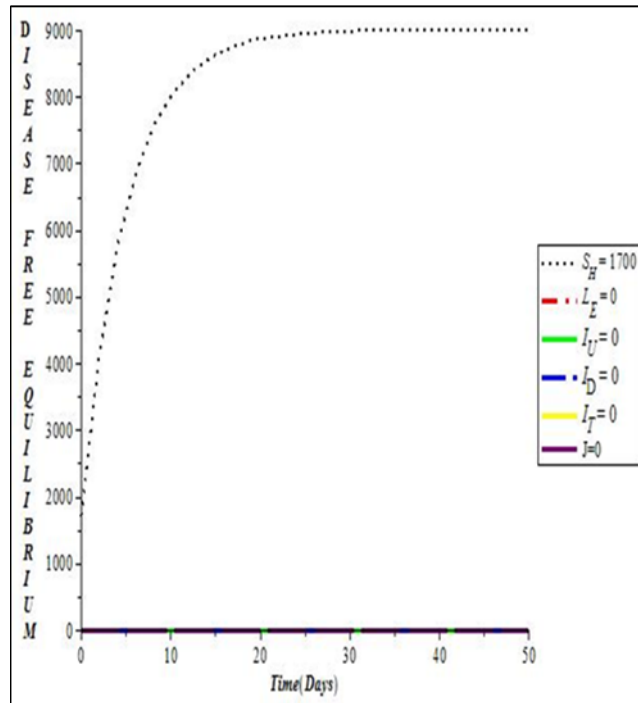
Parameters	Sensitivity indices
$\beta_E$	1.0000000000
$\sigma_2$	0.5252281866
$\theta$	0.1580601091
$\alpha$	0.1272806385
$\eta_D$	0.009595059744
$\kappa_E$	0.003178326922
$\mu$	0.0005554843456
$\omega_1$	0.002846541946
$\gamma_{UE}$	0.0003910511435
$\varepsilon_1$	0.0001295977841
$\phi_2$	0.00004396024358
$\sigma_1$	-0.2704057438
$\tau_1$	-0.0001999572217
$\eta_T$	-0.0002006352752
$\eta_J$	-0.000002002101128
$\delta_{UE}$	0
$\delta_{DE}$	0
$\delta_j$	0

**3. Numerical Simulation**

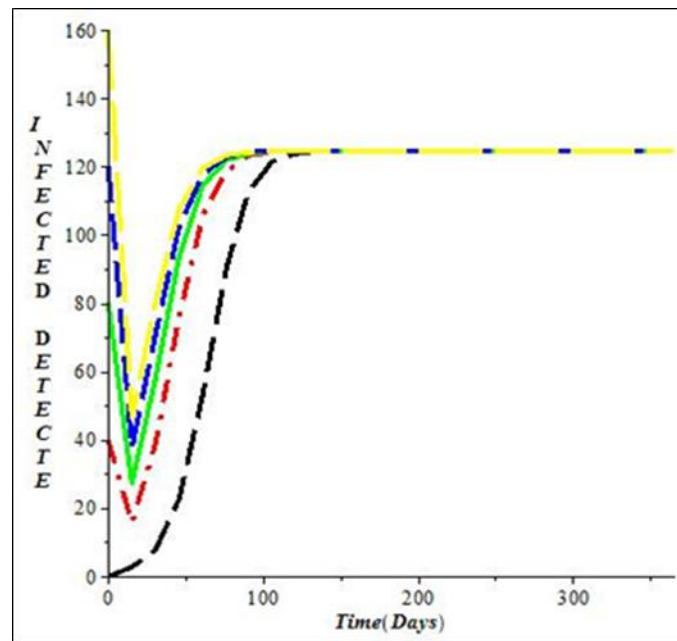
The analytical results of this study are illustrated by carrying out numerical simulations using parameter values in Table 2 with initial values  $S_H(0) = 12000, L_E(0) = 2000, I_U(0) = 200, I_D(0) = 300, I_T(0) = 350, J(0) = 180$ . The simulations are carried out with the help of MAPLE 18 software and the results are given below

**Table 2** Parameters values used for the numerical simulation

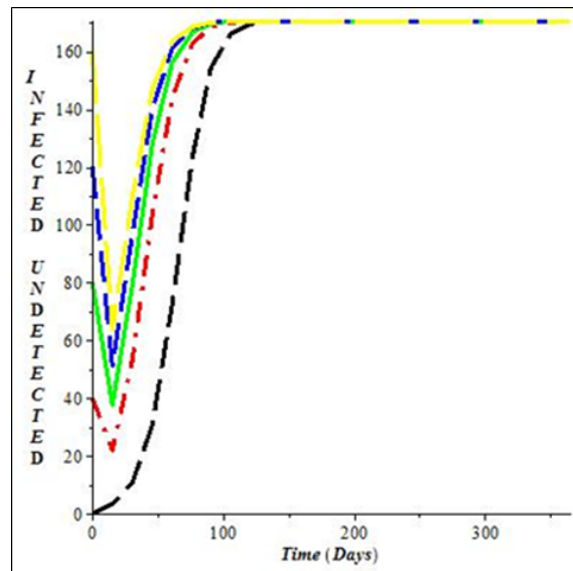
Parameters	Values	Sources
$\pi_H$	1800	Estimated
$\mu$	0.2	Estimated
$\tau_1$	0.3143	[2,20]
$\varepsilon_1$	0.92	[19, 20]
$\gamma_{UE}$	0.2	[2]
$\kappa_E$	0.2	[1]
$\sigma_2$	0.6	[1-2]
$\sigma_1$	0.712	[1-2]
$\beta_E$	0.8	Estimated
$\omega_1$	0.2	[2,20]
$\phi_2$	0.02	[1-2]
$\theta$	0.8	[20]
$\delta_{UE}$	0.01	Estimated
$\delta_{DE}$	0.008	[1,20]
$\delta_j$	0.06	Estimated
$\eta_T, \eta_D, \eta_J$	0.01	Estimated



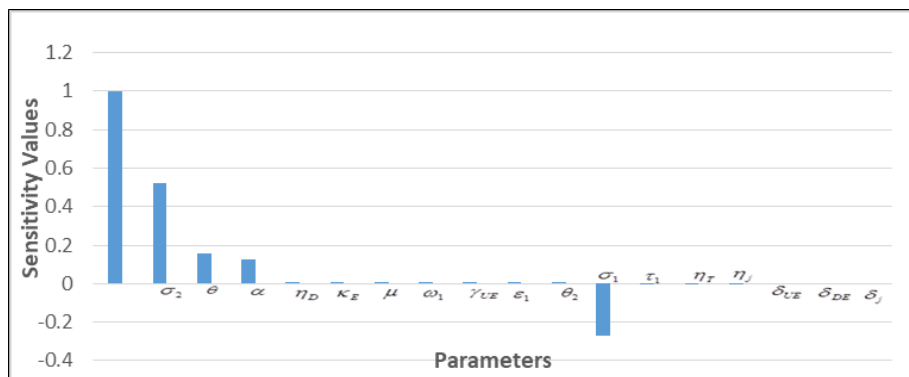
**Figure 1** Graph shows DFE point at different time



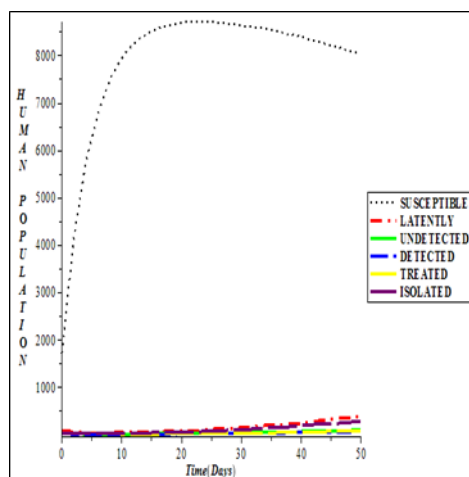
**Figure 2** Graph shows global stability of EE point at different time



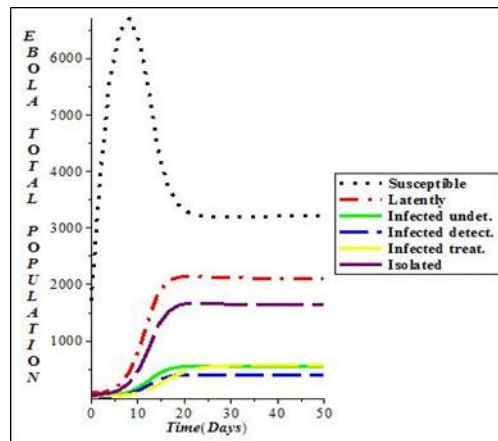
**Figure 3** Graph shows global stability of EE point at different time



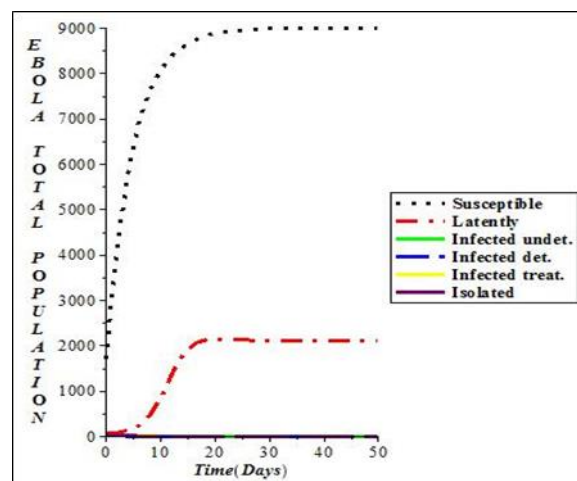
**Figure 4** Chart of sensitivity indices on basic reproduction number



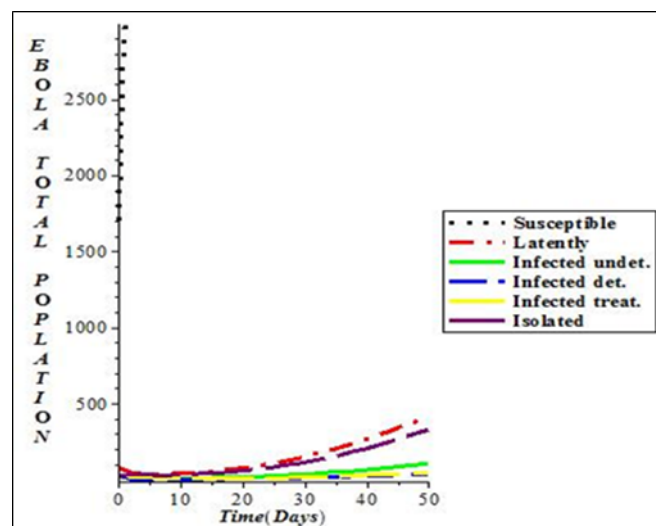
**Figure 5** Represents the initial behavior of the Ebola virus disease model



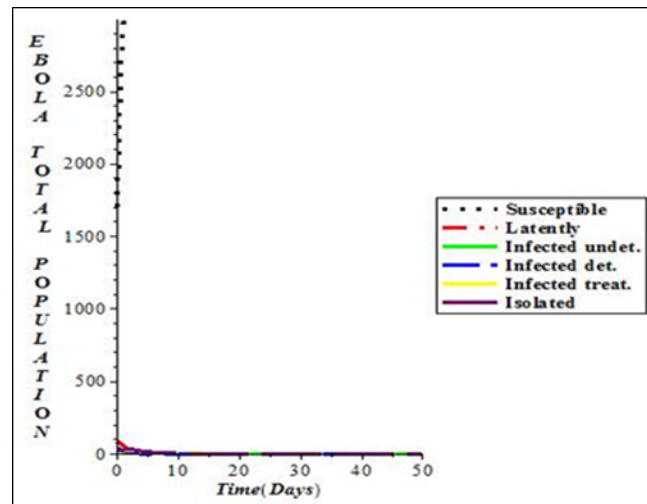
**Figure 6** Graph of increasing the most positive sensitive index value which is contact rate on Ebola virus disease model



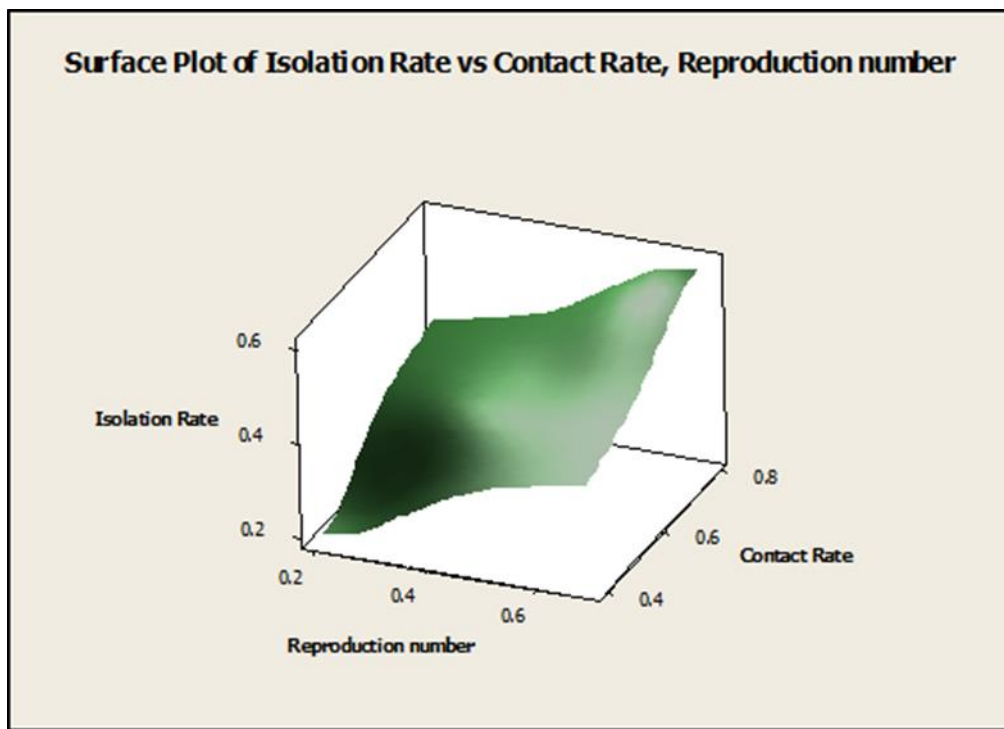
**Figure 7** Graph of reducing the most positive sensitive index value which is contact rate from Ebola virus disease model



**Figure 8** Graph of increasing the most second positive sensitive index value which is isolation rate on Ebola virus disease model



**Figure 9** Graph of eliminating the most positive sensitive index value (contact rate) and increasing the most second sensitive value index (isolation rate) on Ebola virus disease model



**Figure 10** Graph of decreasing the sensitive index value contact rate and isolation rate and their effect on reproduction number

#### 4. Results

Figure 1 depicts the disease-free equilibrium point of the Ebola virus disease, demonstrating that while latent, infected identified, infected undetected, infected treated, and isolated individuals die out, there is always a susceptible population as a whole. According to Figure 2, the system will converge to the same point at a maximum of 120 infected detected classes after 149 days, regardless of the beginning values. Figure 2 illustrates the global stability of endemic equilibrium point of infected detected person. Figure 3 depicts the global stability of the infected undiscovered individual endemic equilibrium point, which illustrates that regardless of the initial values, the system also converges after 149 days to the same position at a maximum of 165 infected undetected classes. The chart of sensitivity indices on the fundamental reproduction is shown in Figure 4. Figure 5 shows the initial behavior of the total population of the various Ebola virus classes. The susceptible population increases initially, then declines slightly due to the natural death rate and remains stable due to the percentage of infected isolated people who become susceptible after testing, while

the latently infected class decreases due to progression, isolation rate, and increases slightly due to discharge rate and treatment progression rate. Individuals' detection causes them to leave the class and join the discovered class, which diminishes the undetected class.

The treated class later reduces as a result of therapy, which causes them to migrate from the undetected class, which initially increases due to the rapid detection of undiscovered individuals. Due to advancement and death rate, the treated class decreases. Due to contributions from latent and observed class isolation rates, the isolated class initially falls and then occasionally increases. As the contact rate increases, it starts to decline due to an increase in infection in the compartment (Figure 6), while the initial susceptible population increases to a peak of 6,700 at just 10 days. Latent, infected undetected, infected detected, infected treated, and isolated individuals increase and remain constant after 20 days. Figure 7 when contact rate was abolished in the community, susceptible human grows to 8600 its highest at 11 days and remain consistent as the day advances while latent population reduces a bit and remain constant as a result of persistence of virus in latent individual. However, the number of isolated, treated, and infected people remained the same throughout and did not significantly affect the compartment.

Figure 8 shows that as the isolation rate rises, the susceptible population grows significantly while the latent and isolated populations also advance. However, the population has a very small number of infected who have been identified, undetected, and treated. Figure 9 shows the effects of reducing contact rates and raising isolation rates. The vulnerable human population reaches its peak without ever declining because there is no infection in the population, while other compartments do not actually exist.

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## 5. Conclusion

In order to incorporate the treated of infected class into [2], a mathematical model was built in this work. The results of the research demonstrated that the model is correctly posed; both endemic and disease equilibria for the models were found. The fundamental Ebola virus disease reproduction number is  $R_E = 0.6870878144$  obtained using the newest matrix technique. When the fundamental reproduction number is less than unity, the disease-free equilibrium of the model is locally asymptotically stable, according to the analysis of the models' stability. Similar to this, there are endemic areas where the Ebola virus disease's fundamental reproduction numbers are more than unity. The results of the sensitivity analysis thus far strongly suggest that the effective contact rate  $\beta_E$  is a key factor in the spread of the Ebola virus disease within the population. The elimination of contact rates is the greatest strategy to control the Ebola virus Disease in the population, according to the graphical results in Figure 9. The 3D plots of the inverse relationship between the fundamental reproduction number, contact rate, and isolation rate are shown in Figures 10 and 11.

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## Compliance with ethical standards

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### *Disclosure of conflict of interest*

No conflict of interest was declared by the authors.

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