Analysis of the Effect of Vaccination on Ebola Dynamics

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Abstract — A SVIR model that shows the effects of vaccination on Ebola virus has been developed. The existence of both disease-free and endemic equilibria has been established and proved to be both globally asymptotically stable whenever $R_V < 1$ and $R_V > 1$ respectively. Numerical simulation performed shows the effectiveness of vaccine in controlling Ebola virus infection.

Keywords — Ebola Virus, Endemic Equilibrium, Vaccination, Global Stability.

I. INTRODUCTION

Ebola virus disease (EVD), formerly known as Ebola hemorrhagic fever, is a severe, often fatal illness in humans. The virus is transmitted to people from wild animals and spreads in the human population through human-to-human transmission. The average EVD case fatality rate is around 50%. Case fatality rates have varied from 25% to 90% in past outbreaks [1, 2]. The virus derives its name after a river in the Democratic Republic of the Congo (formerly Zaire) in Africa, where it was first recognized in 1976. There are five identified subtypes of Ebola virus. Four of the five have caused disease in humans: Ebola Zaire, Ebola-Sudan, Ebola-Ivory Coast and Ebola-Bundibugyo. The fifth, Ebola Reston causes Ebola virus disease in non-human primates; unlike the other four Ebola viruses, it is not known to cause disease in humans, but has caused asymptomatic infections [3]. People can be exposed to Ebola virus from direct contact with the blood, secretions and/or other bodily fluids of dead or living infected persons. Thus, the virus is often spread through families and friends because they come in close contact with such secretions when caring for infected persons or handling dead bodies of Ebola victims. Exposure to Ebola virus may also occur through contact with objects, such as needles, that have been contaminated with infected secretions. Nosocomial transmission during outbreaks have also been reported.

Ebola infected individuals become symptomatic after an average incubation period mostly ranging from 2-21 days. Characteristic symptoms of EVD are nonspecific and include sudden onset of fever, weakness, vomiting, diarrhea, headache, and a sore throat and impaired kidney and liver function. Only a fraction of the symptomatic individuals present with hemorrhagic manifestations. There have been four major known outbreaks about four decades since the disease was first recognized. However, the largest and most devastating is the West African outbreak in Guinea, Liberia and Sierra Leone, believed to have started in Guinea in March 2014. Due to air travel and human mobility, the outbreak eventually spread to Nigeria, Senegal and the US. The US diagnosed its first imported travel-related Ebola case in September 2014, by a person who had come from Liberia. The imported case, who died the following month resulted in the infection of two health-care workers who cared for the deceased patient [9]. One of the cases flown to Spain also led to infection of health care workers [3]. Following this outbreak a total of 6553 cases, with 3083 deaths were reported to WHO as of September 2014. There is no standard treatment for Ebola disease. Patients receive supportive therapy. This consists of balancing the patients' fluids and electrolytes, maintaining their oxygen status and blood pressure, and treating them for any complicating infections. The search for a vaccine for the virus has been ongoing and a WHO news released for July 2015 indicate that vaccine trials had yielded interim results showing 100% efficacy in individuals.

The paper is organized as follows. In Section 1, we have provided background information about Ebola virus infection; in Section 2, we developed the model; Section 3 established a disease-free equilibrium, derived the basic reproduction number, R_V and finally established the existence of endemic equilibrium. In Section 5, the proof of global asymptotic stability is done. In Section 6, numerical simulation is performed. Finally, a discussion and conclusion is presented in Section 7.

II. MODEL FORMULATION AND DESCRIPTION

The total human population size N at any time is subdivided into the classes: susceptible S, vaccinated V, infectious with Ebola virus I and removed R. The removed class consists of individuals who have recovered, those who are vaccine protected as well as those who have died from the infection. We assume that the probability of survival till the infectious state for individuals exposed to Ebola virus is unity and therefore we exclude the exposure stage. The recruitment into the susceptible population takes place at the rate Λ, while the vaccination of susceptibles occurs at the rate ϕ. We assume that the vaccine induced immunity is not instantaneous because it takes time $\frac{1}{\alpha}$ to develop antibodies. We assume the mass action incidence transmission, where the infection of the susceptibles occurs due to contact at the rate β. We also assume that vaccine efficacy wanes at the rate ω, though the above interim results on vaccine trials show 100% efficacy in individuals. However during the development of antibodies, we assume that individuals may be infected but at lower contact rate $\rho\beta$ where $0 < \rho < 1$. Since the

development and trial of vaccines is ongoing, it may not be easy to factually conclude that the vaccine immunity wanes or does not. We assume the latter. Let μ define the per capita natural death rate. Disease mortality is assumed to take place at the rate ψ while the recovery takes place at the rate κ. From the above definitions and variables we have the following model with nonnegative initial conditions

$$\frac{dS}{dt} = \Lambda - \beta SI + \omega R - \phi S - \mu S$$

$$\frac{dV}{dt} = \phi S - \rho\beta VI - \alpha V - \mu V \qquad (1)$$

$$\frac{dI}{dt} = \beta SI + \rho\beta VI - (\psi + \kappa + \mu)I$$

$$\frac{dR}{dt} = \kappa I + \alpha V - \omega R - \mu R$$

Since $N = S + V + I + R$, we have

$$\frac{dN}{dt} = \Lambda - \mu N - \psi I \qquad (2)$$

In the absence of infection equation (2) becomes $\frac{dN}{dt} = \Lambda - \mu N$, therefore N approaches carrying capacity $\frac{\Lambda}{\mu}$. The associated state variables of model (1) are non-negative for all time $t \geq 0$ and the solutions of the model (1) with positive initial data remains positive for all time $t \geq 0$. Thus model (1) is mathematically well posed and its dynamics can be considered in a proper subset describing human population. Therefore it can be shown that the associated state variables $\Omega = \left\{ (S, V, I, R) \in R_+^4 : N \leq \frac{\Lambda}{\mu} \right\}$.

III. EQUILIBRIUM POINTS OF THE MODEL

i. Disease-free equilibrium, $E_0 \left(S^0, V^0, I^0, R^0 \right)$

At the disease-free equilibrium $I_0 = R_0 = 0$. Substituting these values into equation (1) we obtain:

$$\Lambda - \mu S^0 - \phi S^0 = 0 \qquad (3)$$

$$\phi S^0 - \alpha V^0 - \mu V^0 = 0$$

Solving for S^0 and V^0 in (3) we get $\frac{\Lambda}{\phi + \mu}$ and $\frac{\phi\Lambda}{(\phi + \mu)(\alpha + \mu)}$ respectively. The disease-free equilibrium of model (1) is thus given by

$$E^0 = \left(\frac{\Lambda}{\phi + \mu}, \frac{\phi\Lambda}{(\phi + \mu)(\alpha + \mu)}, 0, 0 \right).$$

ii. The Basic Reproduction Number, R_V

We define the basic reproduction number, R_V as the number of secondary Ebola virus infections caused by a single Ebola infected individual in the presence of vaccination. In the absence of vaccination, the basic

reproduction number is given as R_0. When the basic reproduction number is greater than one it means that an infectious individual is causing, on average, more than one new infection and thus the disease invades and persist in the population.

Using the next generation operator approach [4, 5], we determine R_V as

$$R_V = \frac{\beta\Lambda}{(\psi + \kappa + \mu)} \left[\frac{(\alpha + \mu) + \phi\rho}{(\phi + \mu)(\alpha + \mu)} \right] \qquad (4)$$

In the absence of vaccination $\phi = \alpha = \rho = 0$, then the reproduction number becomes

$$R_0 = \frac{\beta\Lambda}{\mu(\psi + \kappa + \mu)} \qquad (5)$$

From equations (4) and (5), it is easy to see that $R_V < R_0$ and $R_v = R_0$ if and only if $\phi = \rho = \alpha = 0$. We can therefore conclude that vaccination is important in controlling Ebola virus infection as it helps in reducing the basic reproduction number.

iii. Existence of a unique positive endemic equilibrium $E^* = \left(S^*, V^*, I^*, R^* \right)$

Lemma 3.1. An endemic equilibrium $E^* \left(S^*, V^*, I^*, R^* \right)$ exists provided that $R_V > 1$.

Proof. At an endemic state, equation (1) becomes

$$0 = \Lambda - \beta S^* I^* + \omega R^* - \phi S^* - \mu S^*$$

$$0 = \phi S^* - \rho\beta V^* I^* - \alpha V^* - \mu V^*$$

$$0 = \beta S^* I^* + \rho\beta V^* I^* - (\psi + \kappa + \mu) I^* \qquad (6)$$

$$0 = \kappa I^* + \alpha V^* - \omega R^* - \mu R^*$$

Solving for S^* from the third equation in (6) we obtain

$$S^* = \frac{(\psi + \kappa + \mu) - \rho\beta V^*}{\beta} \qquad (7)$$

From the second equation of (6), we also express S^* as

$$S^* = \frac{(\rho\beta I^* + \alpha + \mu)}{\phi} V^* \qquad (8)$$

Equating equations (7) and (8) and simplifying, we obtain

$$V^* = \frac{\phi}{\beta} \left(\frac{\psi + \kappa + \mu}{\rho\beta I^* + \alpha + \mu + \phi\rho} \right) > 0, \ \forall I^* > 0. \qquad (9)$$

Substituting (9) into (8)) and solving for S^*, we get

$$S^* = \frac{(\psi + \kappa + \mu)(\beta I^* + \alpha + \mu)}{\beta(\beta I^* + \alpha + \mu + \phi\rho)} > 0, \ \forall I^* > 0 \qquad (10)$$

Using the fourth equation of (6), we express R^* as

$$R^* = \frac{\kappa I^*}{\omega + \mu} + \frac{\alpha V^*}{\omega + \mu} \qquad (11)$$

Substituting (9) into (11)) and solving for R^*, we get

$$R^* = \frac{\beta\kappa I^* \left(\rho\beta I^* + \alpha + \mu + \phi\rho \right) + \phi\alpha(\psi + \kappa + \mu)}{\beta(\omega + \mu)\left(\rho\beta I^* + \alpha + \mu + \phi\rho \right)} > 0, \ \forall I^* > 0. \qquad (12)$$

To obtain the value of I^*, we substitute equations (10) and (12) into the first equation of (6), that is

$$\Lambda - \beta \frac{(\psi + \kappa + \mu)(\beta I^* + \alpha + \mu)}{\beta(\beta I^* + \alpha + \mu + \phi\rho)}$$

$$+ \omega \frac{\beta\kappa I^*(\rho\beta I^* + \alpha + \mu + \phi\rho) + \phi\alpha(\psi + \kappa + \mu)}{\beta(\omega + \rho)(\rho\beta I^* + \alpha + \mu + \phi\rho)} \quad (13)$$

$$- (\phi + \mu) \frac{(\psi + \kappa + \mu)(\beta I^* + \alpha + \mu)}{\beta(\beta I^* + \alpha + \mu + \phi\rho)} = 0$$

Simplifying equation (13), we obtain

$$AI^{*2} + BI^* + C = 0 \quad , \text{where,} \quad (14)$$

$$A = \left[\mu(\psi + \kappa + \mu) + \omega(\psi + \mu) \right] \rho\beta^2$$

$$B = \beta[(\alpha + \mu + \phi\rho)(\psi + \mu) + \mu(\alpha + \omega\rho)(\psi + \kappa)$$
$$+ \mu(\mu + \phi\rho)(\kappa + \mu) + \mu^2(\rho(\psi + \kappa + \mu) + \alpha + \psi)$$
$$+ \mu^2\rho(\omega + \phi) - \beta\Lambda(\omega\rho + \mu)]$$

$$C = (\omega + \mu)(\psi + \kappa + \mu)(\phi + \mu)(\alpha + \mu)\left[\frac{\mu\omega(\phi + \alpha + \mu)}{(\omega + \mu)(\alpha + \mu)(\phi + \mu)} + \frac{\mu}{\omega + \mu} \right]$$

Since $\dfrac{\mu\omega(\phi + \alpha + \mu)}{(\omega + \mu)(\alpha + \mu)(\phi + \mu)} + \dfrac{\mu}{\omega + \mu} < 1, \ C < 0,$

whenever $R_{V>1}$, equation (14) can either take the form of $AI^{*2} + BI^* - C = 0$ or $AI^{*2} - BI^* - C = 0$. It is therefore easy to show that (14) has two real roots given by $I^* > 0$ and $I^* < 0$. We ignore $I^* < 0$ since it is biologically meaningless. We therefore conclude that there exists a positive endemic equilibrium whenever $I^* > 0$.

IV. LOCAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM OF MODEL (1)

Theorem 4.1. The disease-free equilibrium of (1) is locally stable whenever $R_V < 1$ and unstable whenever $R_V > 1$.

Proof. The matrix of linearization of the system (1) at the disease-free equilibrium is given as:

$$J_{E^0} = \begin{bmatrix} -(\phi + \mu) & 0 & \frac{\beta\Lambda}{\phi + \mu} & \omega \\ \phi & -(\alpha + \mu) & \frac{-\phi\rho\beta\Lambda}{(\phi + \mu)(\alpha + \mu)} & 0 \\ 0 & 0 & (\psi + \kappa + \mu)(R_V - 1) & 0 \\ 0 & \alpha & \kappa & -(\omega + \mu) \end{bmatrix}$$

Note that one of the roots is given as
$\lambda = (\psi + \kappa + \mu)(R_V - 1) < 1 \quad \forall R_V < 1$. To determine the sign of the remaining roots, we consider the matrix

$$\begin{bmatrix} \lambda + \phi + \mu & 0 & \omega \\ \phi & \lambda + \alpha + \mu & 0 \\ 0 & \alpha & \lambda + \omega + \mu \end{bmatrix} = 0$$

This gives the polynomial equation $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$,

where,

$$a_1 = 3\mu + \alpha + \phi + \omega > 0$$

$$a_2 = \alpha\phi + 2\phi\mu + 2\alpha\mu + 2\omega\mu + \omega\alpha + \omega\phi + 2\mu^2 > 0$$

$$a_3 = 2\alpha\mu^2 + 2\omega\phi\alpha + \omega\alpha\mu + \omega\mu^2 + \mu\phi\alpha + \mu^2\phi + \mu^3 > 0$$

It is easy to show that $a_1a_2 - a_3 > 0$.

According to Hurwitz criterion, the disease-free equilibrium is locally asymptotically stable whenever $R_V < 1$ and unstable when $R_V > 1$.

V. GLOBAL STABILITY OF EQUILIBRIUM POINTS OF THE MODEL (1)

To find the global stability of disease-free and endemic equilibria, we reduce model (1) by using $R = \frac{\Lambda}{\mu} - S - V - I$. This will eliminate R from the first equation of model (1) leading to the following reduced three dimensional model

$$\frac{dS}{dt} = \frac{\Lambda}{\mu}(\omega + \mu) - \beta SI - (\omega + \phi + \mu)S - \omega V - \omega I$$

$$\frac{dV}{dt} = \phi S - \rho\beta VI - \alpha V - \mu V \quad (15)$$

$$\frac{dI}{dt} = \beta SI + \rho\beta VI - (\psi + \kappa + \mu)I$$

Note that the dynamics of model (1) and model (15) remain the same.

i. Global stability of disease-free equilibrium.

The global stability of the disease-equilibrium E^0 is easily proved by using a Lyapunov function and LaSalle's invariance principle.

Theorem 5.1. If $R_V \leq 1$, then the disease-free equilibrium of (15) is globally asymptotically stable in Ω.

Proof. Define $L : \{(S, V, I) \in \Omega : S, V > 0\} \to \Box$ by

$L(S, V, I) = (\omega + \mu)I$.

Then if $R_0 \leq 1$;

$$L' = (\omega + \mu)[\beta SI + \rho\beta VI - (\psi + \kappa + \mu)I]$$
$$= (\omega + \mu)[\beta(S + \rho V) - (\psi + \kappa + \mu)]I$$
$$\leq (\omega + \mu)[\beta(S + V) - (\psi + \kappa + \mu)]I$$
$$\leq (\omega + \mu)[\frac{\beta\Lambda}{\mu} - (\psi + \kappa + \mu)]I$$
$$\leq (R_0 - 1)(\omega + \mu)(\psi + \kappa + \mu)I$$
$$\leq 0$$

If $L' = 0$, then $I = 0$ or $R_V = 0$. Hence L is a Lyapunov function on Ω. Thus $I \to 0$ as $t \to \infty$. When we substitute $I = 0$ in (1), we obtain $S + V \to \frac{\Lambda}{\mu}$.

Therefore it follows from LaSalle's invariance principle [6, 7, 8] that every solution of (15), with initial conditions in Ω, approaches E_0 as $t \to \infty$

ii. Global stability of the endemic equilibrium

To prove global stability of the endemic equilibrium, E^* of model (15), we use the method of geometrical

approach developed by Li and Muldowney in [9]. For a brief outline of this approach, see [10].

Lemma 5.1. Model (1) is uniformly persistent and satisfies assumptions (H1), (H2) and (H3) as defined in [9].

For an assumption (H3), we have shown in **Lemma 3.1** that indeed $E^* = (S^*, V^*, I^*, R^*)$ is the only positive endemic equilibrium of model (1) and it exits whenever $R_V > 1$. Assumptions (H1) and (H2) also hold since by using the persistence property by [11], it can be verified that the solution of system (1) is uniformly persistent. Let $B = E^0$, **Theorem5.1** implies that when $R_0 > 1$, B^s is an isolated in Ω and is contained in the $S-$ axis in the boundary of Ω. When $R_0 > 1$, model (1) satisfies condition by [12]. Therefore, we conclude that model (1) is persistent in Ω when R0 > 1.

Since we have shown that all the assumptions are satisfied, we therefore apply **Theorem3.3** and **Theorem 3.4** of [10] to prove the following theorem:

Theorem 5.2. If $S \geq \dfrac{\Lambda(\omega + \mu)}{\mu(\omega + 2\phi + \mu)}$ and $V \geq \dfrac{\phi}{\alpha + \mu}S$ then the unique positive endemic equilibrium, E^* of system (15) is globally asymptotically stable whenever $R_V > 1$.

Proof. The Jacobian matrix of model (15) associated with the general solution (S, V, I) is given by

$$J = \begin{bmatrix} -\beta I - (\omega + \phi + \mu) & -\omega & -(\beta S + \omega) \\ \phi & -\rho\beta - (\alpha + \mu) & -\rho\beta V \\ \beta I & \rho\beta I & \beta S + \rho\beta V - (\psi + \kappa + \mu) \end{bmatrix} \quad (16)$$

The second compound additive matrix of (16) is given as

$$J^{[2]} = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix} \quad (17)$$

Where,

$a_{11} = -(\beta I + \rho\beta I + \omega + \phi + \alpha + 2\mu)$

$a_{12} = -\rho\beta V$

$a_{13} = \beta S + \omega$

$a_{21} = \rho\beta I$

$a_{22} = -(\beta I + \omega + \phi + \psi + \kappa + 2\mu) + \beta S + \rho\beta V$

$a_{23} = -\omega$

$a_{31} = -\beta I$

$a_{32} = \phi$

$a_{33} = -(\rho\beta I + \rho\beta + \alpha + \psi + \kappa + 2\mu) + \beta S + \rho\beta V$.

Set matrix $P(S, V, I) = \text{diag}\left(1, \dfrac{V}{I}, \dfrac{V}{I}\right)$.

Then,

$$P_f P = \left[0, \dfrac{V'}{V} - \dfrac{I'}{I}, \dfrac{V'}{V} - \dfrac{I'}{I}\right].$$

Matrix $PJ^{[2]}P^{-1}$ is given as $\begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix}$, where:

$a_{11} = -(\beta I + \rho\beta I + \omega + \phi + \alpha + 2\mu)$

$a_{12} = -\rho\beta I$

$a_{13} = (\beta S + \omega)\dfrac{1}{V}$

$a_{21} = \rho\beta V$

$a_{22} = -(\beta I + \omega + \phi + \psi + \kappa + 2\mu) + \beta S + \rho\beta V$

$a_{23} = -\omega$

$a_{31} = -\beta V$

$a_{32} = \phi$

$a_{33} = -(\rho\beta I + \alpha + \psi + \kappa + 2\mu) + \beta S + \rho\beta V$.

The matrix $P_f P + PJ^{[2]}P^{-1}$ as defined in equation (3.7) of [10] can be written in block form as:

$$Q = \begin{bmatrix} Q_{11} & Q_{12} \\ Q_{21} & Q_{22} \end{bmatrix}, \text{ where}$$

$Q_{11} = -(\beta I + \rho\beta I + \omega + \phi + \alpha + 2\mu)$

$Q_{12} = \left(-\rho\beta I \quad \dfrac{1}{V}(\beta S + \omega)\right)$

$Q_{21} = \begin{pmatrix} -\rho\beta V \\ -\beta V \end{pmatrix}$

$Q_{22} = \begin{bmatrix} q_{11} & q_{12} \\ q_{21} & q_{22} \end{bmatrix}, \text{ where}$

$q_{11} = \dfrac{V'}{V} - \dfrac{I'}{I} - (\beta I + \omega + \phi + \kappa + 2\mu) + \beta S + \rho\beta V$

$q_{12} = -\omega$

$q_{21} = \phi$

$q_{22} = \dfrac{V'}{V} - \dfrac{I'}{I} - (\rho\beta I + \alpha + \psi + \kappa + 2\mu) + \beta S + \rho\beta V$

Let the vector norm $|.|$ in $\mathbb{R}^3 \cong \mathbb{R}^{\binom{3}{2}}$ be chosen $|(u, v, w)| = \text{Sup}\{|u|, |v| + |w|\}$, for $(u, v, w) \in \mathbb{R}^3$ any vector. We can then estimate the Lozinskii measure $k(B)$ with respect to $|.|$ by $k(Q) \leq \sup(g_1 \quad g_2)$, with

$$g_1 = k_1(Q_{11}) + |Q_{12}|$$
, where $|Q_{12}|$ and
$$g_2 = k_1(Q_{22}) + |Q_{21}|$$

$|Q_{21}|$ are matrix norms induced by L_1 vector norm and k_1 being the Lozinskii measure with respect to the L_1 norm, (see [13]). Specifically,

$$g_1 = -(\beta I + \rho\beta I + \omega + \phi + \alpha + 2\mu) + \dfrac{\beta SI}{V} + \dfrac{\omega I}{V} \quad (19)$$

From the second equation of (15) we obtain

$$-(\alpha + \mu) = \dfrac{V'}{V} + \rho\beta I - \dfrac{\phi S}{V} \quad (20)$$

Substituting (20) into (19), we get

$$g_1 = \dfrac{V'}{V} - \beta I - \dfrac{\phi S}{V} - \omega - \phi - \mu + \dfrac{\beta SI}{V} + \dfrac{\omega I}{V} \quad (21)$$

From the first equation of (15), we have

$$\frac{\beta SI}{V} + \frac{\omega I}{V} = \frac{\Lambda(\omega+\mu)}{\mu V} - (\omega+\phi+\mu)\frac{S}{V} - \frac{S'}{V} - \omega \quad (22)$$

Substituting (22) into (21), we get

$$g_1 = \frac{V'}{V} - \beta I - \phi\frac{S}{V} - 2\omega - \phi - \mu + \frac{\Lambda(\omega+\mu)}{V} - \frac{(\omega+\phi+\mu)}{V} - \frac{S'}{V}$$

$$\leq \frac{V'}{V} - (\omega+\mu),$$

provided that $S \geq \dfrac{\Lambda(\omega+\mu)}{\mu(\omega+2\phi+\mu)}$.

g_2 can be expressed as

$$g_2 = \frac{V'}{V} - \frac{I'}{I} - (\rho\beta I + \omega + \psi + \kappa + 2\mu) + \beta S + \rho\beta V + \beta V \quad (23)$$

$$\leq \frac{V'}{V} - \frac{I'}{I} - (\omega + \psi + \kappa + 2\mu) + \beta S + \rho\beta V + \beta V$$

From the third equation of (15), we have

$$\beta S + \rho\beta V = \frac{I'}{I} + (\psi + \kappa + \mu) \quad (24)$$

Substituting this equation into (24) into (23), we obtain

$$g_2 = \frac{V'}{V} - (\omega+\mu) + \beta V \quad (25)$$

From the second equation of (15), we have

$$\beta V = -\frac{V'}{\rho I} - \frac{(\alpha+\mu)V}{\rho I} + \frac{\phi S}{\rho I} \quad (26)$$

Substituting (26) into (25) we obtain

$$g_2 = \frac{V'}{V} - (\omega+\mu) - \frac{V'}{\rho I} - \frac{(\alpha+\mu)}{\rho I} + \frac{\phi S}{\rho I} \quad (27)$$

$$\leq \frac{V'}{V} - (\omega+\mu),$$

provided that $V \geq \dfrac{\phi}{\alpha+\mu}S$.

Therefore, $k(Q) \leq \dfrac{V'}{V} - (\omega+\mu)$.

Since $0 \leq V \leq N$, there exists a $T > 0$ such that when

$t > T$, $\dfrac{\ln V(t) - \ln V(0)}{t} < \dfrac{\omega+\mu}{2}$. As a result

$$\frac{1}{t}\int_0^t k(Q)\,dt \leq \frac{1}{t}\int_0^t \left(\frac{V'}{V} - (\omega+\mu)\right) = \frac{\ln V(t) - \ln V(0)}{t}$$

$$-(\omega+\mu) < \frac{(\omega+\mu)}{2}$$

which implies that $\bar{q} \leq -\dfrac{(\omega+\mu)}{2} < 0$. Hence, we have

shown that the endemic equilibrium E^* of system (15) is globally asymptotically stable in Ω.

VI. NUMERICAL SIMULATIONS

In order to validate the analysis of model (15), numerical simulations were carried out using a set of parameter values given in Table 1.

Table 1: Parameter values

| Parameter	Symbol	Value	Source
Rate of recruitment into susceptible class	Λ	$9.6274 \times 10^{-5}\text{day}^{-1}$	[14]
Effective contact rate	β	Variable	Variable
Vaccination rate	ϕ	0.1429day^{-1}	Assumed
Time needed to develop antibodies	$\frac{1}{\alpha}$	14 days	Assumed
Scaling factor	ρ	$0.27 - 0.99$	[15]
Disease induced mortality rate	ψ	$\frac{1}{75}\text{day}^{-1}$	[15]
Recovery rate	κ	$\frac{1}{79}$ day^{-1}	[15]
Per capita natural death rate	μ	$\frac{1}{22}\text{day}^{-1}$	[15]

(a) (b)
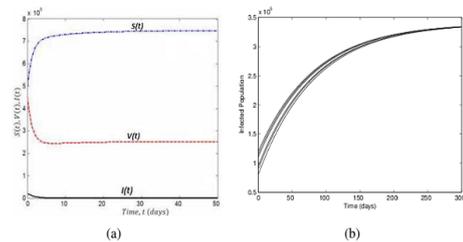
Figure 1: Simulations of model (15) showing (a) global stability of disease-free equilibrium with $R_v = 0.5967, \beta = 0.0000125, \rho = 0.27$, (b) global stability of the endemic equilibrium of model (15) with $\mathbb{R}_v = 1.7254, \beta = 0.000625, \rho = 0.525$. Other parameters remain as shown in Table 1.
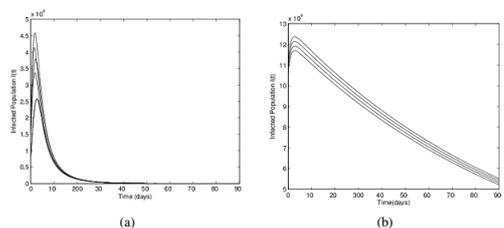

(a) (b)

Figure 2: Simulations of model (15) with various initial values showing plots for trend of infected individuals $(I(t))$, with (a) $R_v = 0.4215, \beta = 0.000125, \rho = 0.3215$ (b) $R_v = 0.7316, \beta = 0.000275$ and $\rho = 0.6125$. Other parameters remain as in Table1.
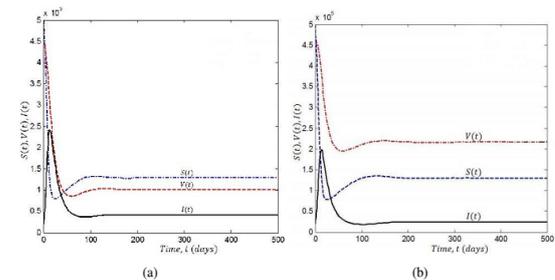

(a) (b)

Figure 3: Simulations of model (15) showing both global stability of endemic equilibrium and how varying ρ affects the population with (a) $R_v = 2.1683, \rho = 0.7235, \beta = 0.0002731$, (b) $R_v = 1.6618, \rho = 0.3721, \beta = 0.0002731$. Other parameters remain as in Table1.

VII. DISCUSSION AND CONCLUSION

Figure 1 shows the global stability of both disease-free and endemic equilibria. Figure1 (a), shows the global stability of the disease-free equilibrium when Rv= 0.5967. From the figure it is observed that when the number of susceptibles increase, the vaccinated population reduce. This happens until the global stability is attained. Figure 1(b) shows the global stability of the endemic equilibrium when Rv = 1.7254. In the figure, we observe that after a given period of time, the infected population converge towards this equilibrium regardless of the initial point. Simulations in Figure 2 shows that when Rv < 1, the disease can easily be controlled or can naturally dies out. It

takes a slightly shorter time to control the disease in Figure 2(a) than in Figure 2(b). This is simply because of differences in Rv, that is the basic reproduction number of Figure 2(b) is higher that of Figure 2(a). From this observation, it is therefore recommended that measures that can reduce Rv be undertaken. Measures such as vaccination, minimizing contacts between infected and uninfected are highly recommended. Figure 3 shows the global stability of the endemic equilibrium. It also shows how varying ρ affects the population. When ρ = 0.7235, we observe that the number of susceptibles are more than that of the vaccinated and more people are infected, see Figure 3(a). When ρ = 0.3721, the vaccinated population are more than the susceptibles and now few people are infected. This is a confirmation that effective vaccine is necessary in controlling Ebola virus. This also supports our analysis where we showed that vaccination effectively reduces the basic reproduction number of the model.

In summary, a SVIR model has been developed. It has been shown that it has both a disease-free and endemic equilibria which are globally asymptotically stable. Numerical simulations have also been carried out to show the positive impact of vaccination on Ebola virus infections. This work recommends that more effective vaccines need to be develop in order to help in fighting Ebola epidemics.

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