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Vertical dynamics of Ebola with media impact

Nita H. Shah [a,*], Zalak A. Patel [b], Bijal M. Yeolekar [a]

[a] Department of Mathematics, Gujarat University, Ahmedabad 380009, Gujarat, India
[b] L.D. College of Engineering, Ahmedabad 380015, Gujarat, India

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ABSTRACT

The Ebola virus disease (EVD) is one of the deadly diseases amongst human and non-human primates caused by most virulent pathogens Ebola virus. Socio-economic impacts of Ebola is very high. In 2014, the latest and largest major outbreak occurred in West African countries Guinea, Sierra Leone, and Liberia. During the outbreak, cases were reported about presence of Ebola virus in semen and breast milk of individuals after recovery. Vertical transmission of disease spread and its impacts on population are studied in this research. How effectively early diagnosis, isolation, awareness campaigns break the chain of infectious cases and control disease spread is studied. Mathematical model for vertical transmission of Ebola with media effect is developed using seven compartments namely susceptible, exposed, infectious, Hospitalised/Isolated, Convalescent, Dead (not properly buried dead bodies of Ebola victims), and Recovered. Basic reproduction number, Disease free equilibrium, Endemic equilibrium are derived. Stability of DFE and EE is established. Numerical simulations are carried out. It is observed that mass media is one of the most effective ways of creating awareness about such type of high mortality disease. Informative awareness amongst public may curb the disease spread and help to control disease in initial stage.

1. Introduction

Ebola virus disease (EVD) having fatality rate around 50%–90% (Kucharski and Edmunds, 2014) is a serious major threat to human population. Ebola virus diseases (EVD), formerly known as Ebola haemorrhagic fever get spread by infection from virus of the family *Filoviridae*, genus *Ebolavirus*. As per WHO Ebola situation report March 2015, around 10,311 deaths with total 24,872 infection cases of Ebola were reported from western African countries. In 1976, two simultaneous outbreaks of Ebola were occurred in South Sudan and Democratic Republic of Congo. Thereafter, outbreak was occurred in a village near river Ebola, and then it was started to identify as EVD. As a Zoonotic disease Ebola virus can transmit amongst animal and human population.

Various study of Ebola virus transmission amongst animal population suggests that fruit bats are the reservoir hosts for the Ebola virus. Baize et al. (2014) observed that from handling bushmeat and contacts with fruit Bats, the virus can transmit to humans and nonhuman primates like animals, apes, monkeys. Pourrut et al. (2005) and, Martin-Moreno et al. (2014) studied that after the first infection in human, transmission of the virus amongst human population occur through contact with broken skin or mucous membranes of the blood or body fluids and surfaces of materials contaminated with such fluids of EVD infected people and dead but not buried bodies of those who have died because of EVD (Kupferschmidt, 2014).

Chowell et al. (2015) studied that the early diagnosis of pre-symptomatic individuals helps to decrease basic reproduction number. Effective isolation may improve result as isolated class has lower transmission rate. Individual stays long in isolated class, which increases possibility of disease spread if effective precautions are not taken.

As per Legrand et al. (2007), Adebamowo et al.(2014) and Pandey et al.(2014), the incubation period from infection with the virus to development of symptoms is 2–21 days. Feldmann and Geisbert (2011) studied that humans remain uninfectious until they develop symptoms like fever, severe headache, muscle pain, weakness, fatigue, diarrhoea, vomiting, stomach pain, and unexplained hemorrhage (bleeding or bruising).

* Corresponding author.
E-mail address: nitahshah@gmail.com (N.H. Shah).

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Studies of Rodriguez et al. (1999), Bausch et al. (2007), and Center for Disease Controls and Prevention (2015) Morbidity and Mortality weekly report suggest that semen from man survived from Ebola (Convalescent Ebola patients) also contains virus up to nine months of infection and its spread is also possible through sexual activities. As per WHO (2014), the transmission of Ebola virus can occur via up to 7 weeks after recovery from an Ebola virus infection from the semen of a convalescent men. Center for Disease Control and Prevention (2015), Morbidity and mortality weekly report also indicates that there were no traces of virus spread due to vaginal fluids during intercourse from a woman who had Ebola. Bausch et al. (2007) also studied that Ebola virus can survive in breast milk. This leads to study of vertical transmission of EVD. Studies indicates that sexual and congenital transmission are relative congruent. If mother get infection due to sexual activity, baby will not survive. If mother do not get infection then virus will direct move to placenta. Baby gets infected and directly moves toward infectious class.

As per WHO factsheet (2016), there is no vaccines or medicine available for cure of Ebola. Only supportive care-rehydration with oral or intravenous fluids and treatment of specific symptoms helps to improve survival. As per Center for Disease Controls and Prevention report (2016), recovery from Ebola depends on good supportive clinical care and the patient's immune response. People who recover from Ebola infection develop antibodies that last for at least 10 years. Chowell et al. (2015), Dhillon et al. (2015) and Chowell and Nishiura (2014) studied the effect of quick diagnosis and Isolation with rapid testing onset of symptoms on disease spread. Galvani et al. (2014) observed that in absence of medicine and vaccines, early diagnosis, isolation of suspected, awareness campaigns and sanitary burial practices are effective controls to stop disease spread.

Liu et al.(2007), Pang and Cui (2009), Safi and Gumel (2011) observed that during outbreak of infectious disease like Ebola, the informative reporting by media highly affects belief of people about risk and threat of disease. Detail media coverage develops sense about precautions like wearing mask, avoiding travel and visit of public places during disease outbreaks. Wang and Xiao (2014) observed that effective news coverage with detail and faster flow of information increase the knowledge about disease in public domain. Sun et al. (2011) studied that, during early stage of outbreak when medicine or vaccines are not developed effective media coverage including warning and advisory helps in curb of disease. The transmission dynamics of EVD is studied by different researchers like Smailhodvic et al. (2014), Love et al. (2014), Basch et al. (2014) and Househ (2015) with various strategies. Mathematical modeling is an effective tool to analyse the epidemiological characteristics of infectious diseases. Shen et al. (2015a,b) studied model involving different intervention strategies like effects of early diagnosis, isolation, improved media coverage, restricted burial procedures and vaccination on the epidemic of Ebola infection. Shau and Dhar (2015) studied SEQIRHS model and observed that media coverage did affect basic reproduction number but it helps to lowering infectious individuals at endemic stage.

The paper is organised as follow. In Section 2, mathematical model with notations, assumptions, and detail discussion of dynamics of disease spread with population flow amongst compartments followed by a suggested system of ordinary differential equations is formulated. In Section 3, basic reproduction number, disease free equilibrium and endemic equilibrium points with and without media effects are derived. In Section 4, stability of model has been discussed. In Section 5, numerical simulation has been carried out and sensitivity of model is discussed using graphical analysis of various parameters for both with and without media effects. For the model, the graphical comparisons of the various populations for both with and without media effects are elaborated. Results obtained support calculations made in Sections 2 and 3. In Section 6, conclusions emphasize the role of media, isolation of exposed, proper burial process and safe sexual activities to control disease spread.

2. Mathematical model

The mathematical model is developed with following notations (Table 1).

A non-linear dynamical system of differential equations is suggested to study the spread of EVD with vertical transmission and media effect. Total population is divided in to seven compartments viz. Susceptible, Exposed, Infectious, Hospitalised/Isolated, Convalescent, Dead (not properly buried dead bodies of Ebola victims), and Recovered adults.

To prepare the model, the following possibilities of disease spread are considered.

(1) Vertical Transmission of infection to new-borns.
(2) Nonsexual transmission amongst total population.
(3) Heterosexual transmissions amongst adults (from man to women).
(4) Transmission from Dead body of EVD victims.

To formulate the model following assumptions are taken in account.

Table 1
Notations with model parameters.

Parameters	Description	Range
$\bar{N}(\bar{t})$	Total Population at time t	>0
$\bar{S}(\bar{t})$	Number of Susceptible individuals at time t	>0
$\bar{E}(\bar{t})$	Number of Exposed individuals at time t	>0
$\bar{I}(\bar{t})$	Number of Infected individuals at time t	>0
$\bar{H}(\bar{t})$	Number of Hospitalised/Isolated individuals at time t	>0
$\bar{C}(\bar{t})$	Number of Convalescent individuals at time t	>0
$\bar{D}(\bar{t})$	Number of Dead bodies of Ebola victim individuals at time t	>0
$\bar{R}(\bar{t})$	Number of Recovered individuals at time t	>0
$\bar{B}$	New recruitments	>0
$\bar{\beta}$	Disease transmission rate due to contacts of infected and hospitalised/isolated with susceptible	(0, 1)
$\bar{\beta}_C$	Disease transmission rate due to contacts during sexual activities of convalescent with susceptible	(0, 1)
$\bar{\beta}_D$	Disease transmission rate due to contacts of infected dead bodies	(0, 1)
$\bar{\varepsilon}$	Probability of disease spread due to leakage in isolation (Shen et al., 2015a,b)	(0, 1)
$\bar{\psi}$	Probability of a female individual get infected due to sexual activity	(0, 1)
$\bar{\delta}$	Probability of virus entering direct in placenta due to sexual activity	(0, 1)
$\bar{\mu}$	Mortality rate	(0, 1)
$\bar{\alpha}_1$	Disease induced death rate in infected class	(0, 1)
$\bar{\alpha}_2$	Disease induced death rate in hospitalised/ isolated class	(0, 1)
$\bar{\sigma}$	Transmission rate from exposed class to infected class	(0, 1)
$\bar{\eta}$	Rate at which infected are hospitalised/ isolated	(0, 1)
$\bar{\theta}$	Rate at which exposed are hospitalised/ isolated due to early detection of symptoms	(0, 1)
$\bar{\gamma}_1$	Recovery rate from infected class	(0, 1)
$\bar{\gamma}_2$	Recovery rate from hospitalised/ isolated class	(0, 1)
$\bar{\gamma}_3$	Recovery rate from convalescent class	(0, 1)
$\bar{m}_1$	Number of individuals are aware by media coverage for risk of horizontal transmission of EVD	(0, 10000)
$\bar{m}_2$	Number of individuals are aware by media coverage for risk of transmission of disease due to contact with dead bodies of EVD victims	(0, 10,000)
$\bar{m}_3$	Number of individuals are aware by media coverage for risk of sexual transmission of EVD	(0, 10,000)

It is assumed that the infected, exposed, hospitalised/isolated individuals are not taking part in sexual activities and hence not responsible for disease spread via sexual activity. It is also assumed that, recovered individuals should not persist infection due to immunity. As dead bodies of EVD victims are responsible for disease transmission the dead individuals are also considered as part of total population till these bodies are not properly buried. For simplicity, rate of bury of EVD victims dead bodies is considered as equal to natural mortality rate.

The flow of population amongst the compartments is shown in Fig. 1.

New recruitments in observed population are entering in system at fixed number $\bar{B}$. The convalescent (not fully recovered) sexually active man (contains virus in their semen) transmits the Ebola virus to susceptible women at rate $\bar{\psi}\bar{\beta}_C\frac{\bar{C}}{\bar{N}}$ or direct placenta of women which leads birth of infected new-born at rate $\overline{\delta\beta}_C\frac{\bar{C}}{\bar{N}}$ and such susceptible individuals moves toward exposed class or new-born babies towards infected class respectively.

Susceptible individuals get infection by contacts of body fluid of infected individuals at rate $\bar{\beta}\frac{\bar{I}}{\bar{N}}$, by Leakage in hospitalisation/isolation at rate $\bar{\varepsilon}\bar{\beta}\frac{\bar{H}}{\bar{N}}$ and by contacts of dead bodies of EVD victims at rate $\bar{\beta}_D\frac{\bar{D}}{\bar{N}}$ and hence they move towards exposed class. Exposed individuals either move toward infected class at rate $\bar{\sigma}$ or hospitalised/isolated class at rate $\bar{\theta}$. Infected individuals either move toward hospitalised/ isolated class at rate $\bar{\eta}$ or move toward Dead bodies' class at rate α_1 or get partial recovery and move towards convalescent class at rate $\bar{\gamma}_1$. Hospitalised/Isolated individuals get move toward Dead bodies' class at rate α_2 or get partial recovery and move towards convalescent class at rate $\bar{\gamma}_2$. Convalescent individuals get full recovery and move toward recovered class at rate $\bar{\gamma}_3$. The mortality rate $\bar{\mu}$ affects the population from each compartment.

Thus, the dynamics of the disease can be expressed by the system of non-linear differential equations as

$$\frac{d\bar{S}(\bar{t})}{d\bar{t}} = \bar{B} - \bar{\beta}\left(\frac{\bar{I}}{\bar{N}} + \bar{\varepsilon}\frac{\bar{H}}{\bar{N}}\right)\bar{S} - \bar{\beta}_D\frac{\bar{D}}{\bar{N}}\bar{S} - \bar{\beta}_C\bar{\psi}\frac{\bar{C}}{\bar{N}}\bar{S} - \bar{\mu}\bar{S} \tag{1}$$

$$\frac{d\bar{E}(\bar{t})}{d\bar{t}} = \bar{\beta}\left(\frac{\bar{I}}{\bar{N}} + \bar{\varepsilon}\frac{\bar{H}}{\bar{N}}\right)\bar{S} + \bar{\beta}_D\frac{\bar{D}}{\bar{N}}\bar{S} + \bar{\beta}_C\bar{\psi}\frac{\bar{C}}{\bar{N}}\bar{S} + \bar{\beta}_C\bar{\delta}\frac{\bar{C}}{\bar{N}}\bar{S} - (\bar{\theta} + \bar{\sigma} + \bar{\mu})\bar{E} \tag{2}$$

$$\frac{d\bar{I}(\bar{t})}{d\bar{t}} = \bar{\sigma}\bar{E} - \bar{\alpha}_1\bar{I} - \bar{\eta}\bar{I} - \bar{\gamma}_1\bar{I} - \bar{\mu}\bar{I} \tag{3}$$

$$\frac{d\bar{H}(t)}{d\bar{t}} = \bar{\eta}\bar{I} + \bar{\theta}\bar{E} - \bar{\alpha}_2\bar{H} - \bar{\gamma}_2\bar{H} - \bar{\mu}\bar{H} \tag{4}$$

$$\frac{d\bar{C}(t)}{d\bar{t}} = \bar{\gamma}_1 I + \bar{\gamma}_2\bar{H} - \bar{\gamma}_3\bar{C} - \bar{\mu}\bar{C} \tag{5}$$

$$\frac{d\bar{D}(t)}{d\bar{t}} = \bar{\alpha}_1\bar{I} + \bar{\alpha}_2\bar{H} - \bar{\mu}\bar{D} \tag{6}$$

$$\frac{d\bar{R}(t)}{d\bar{t}} = \bar{\gamma}_3\bar{C} - \bar{\mu}\bar{R} \tag{7}$$

where $\bar{N}(\bar{t}) = \bar{S}(\bar{t}) + \bar{E}(\bar{t}) + \bar{I}(\bar{t}) + \bar{H}(\bar{t}) + \bar{C}(\bar{t}) + \bar{R}(\bar{t})$ denotes total population and

$$\bar{\beta} = \bar{\beta}_0 + (\bar{\beta}_1 - \bar{\beta}_0)e^{-m_1\left(\frac{1}{N} + \bar{\varepsilon}\frac{\bar{H}}{\bar{N}}\right)}$$

$$\bar{\beta}_D = \bar{\beta}_{D0} + (\bar{\beta}_{D1} - \bar{\beta}_{D0})e^{-m_2\frac{\bar{D}}{\bar{N}}}$$

$$\bar{\beta}_C = \bar{\beta}_{C0} + (\bar{\beta}_{C1} - \bar{\beta}_{C0})e^{-m_3\frac{\bar{C}}{\bar{N}}}$$

As solutions of (1)–(7) are positive real numbers, solution set of (1)–(7) is subset of $\hbar_+^7$.

Also $\left(\frac{d\bar{S}(\bar{t})}{d\bar{t}}\right)_{\bar{S}=0} > 0$, $\left(\frac{d\bar{E}(\bar{t})}{d\bar{t}}\right)_{\bar{E}=0} > 0$, $\left(\frac{d\bar{I}(\bar{t})}{d\bar{t}}\right)_{\bar{I}=0} > 0$, $\left(\frac{d\bar{H}(\bar{t})}{d\bar{t}}\right)_{\bar{H}=0} > 0$, $\left(\frac{d\bar{C}(\bar{t})}{d\bar{t}}\right)_{\bar{C}=0} > 0$, $\left(\frac{d\bar{D}(\bar{t})}{d\bar{t}}\right)_{\bar{D}=0} > 0$, and $\left(\frac{d\bar{R}(\bar{t})}{d\bar{t}}\right)_{\bar{R}=0} > 0$ verifies that all solutions trajectories of (1)–(7) initiating in $\hbar_+^7$ will remain inside $\hbar_+^7$.

Adding Eqs. (1)–(7), the total change in population can be expressed as $\frac{d\bar{N}(\bar{t})}{d\bar{t}} = \bar{B} + \bar{\delta}\bar{\beta}_C\frac{\bar{C}}{\bar{N}}\bar{S} - \bar{\mu}\bar{N} - \bar{\alpha}_1\bar{I} - \bar{\alpha}_2\bar{H}$. i.e. $\frac{d\bar{N}(\bar{t})}{d\bar{t}} \leqslant \bar{B} - \bar{\mu}\bar{N}$.

Hence, by Brikhoff and Rota's theorem, $\bar{N}(\bar{t}) \leqslant \frac{\bar{B}}{\bar{\mu}}$ as $\bar{t} \to \infty$.

Thus, $\bar{\Omega} = \left\{(\bar{S},\bar{E},\bar{I},\bar{H},\bar{C},\bar{D},\bar{R}) \in \hbar_+^7 : \bar{S},\bar{E},\bar{I},\bar{H},\bar{C},\bar{D},\bar{R} \geqslant 0,\ \bar{S}+\bar{E}+\bar{I}+\bar{H}+\bar{C}+\bar{D}+\bar{R} \leqslant \frac{\bar{B}}{\bar{\mu}}\right\}$ is positively invariant region, in which existence, uniqueness of solution and continuation of solutions hold.

Now, reducing the system of equations in to non-dimensional form (Shau and Dhar, 2015) using $S = \frac{\bar{S}}{\bar{N}}$, $E = \frac{\bar{E}}{\bar{N}}$, $I = \frac{\bar{I}}{\bar{N}}$, $H = \frac{\bar{H}}{\bar{N}}$, $R = \frac{\bar{R}}{\bar{N}}$, $D = \frac{\bar{D}}{\bar{N}}$, $N = \frac{\bar{N}}{\bar{N}_0}$, $t = \frac{\bar{\mu}}{\bar{t}}$ and $S + E + I + H + D + R = 1$.

Hence, required non-dimensional system can be given by,

$$\frac{dS}{dt} = \frac{1}{N} - [\beta(I + \varepsilon H) + \beta_D D + \beta_C\psi C]S - S - \frac{S}{N}\frac{dN}{dt} \tag{8}$$

$$\frac{dE}{dt} = [\beta(I + \varepsilon H) + \beta_D D + \beta_C(\psi + \delta)C]S - (\theta + \sigma + 1)E - \frac{E}{N}\frac{dN}{dt} \tag{9}$$

$$\frac{dI}{dt} = \sigma E - (1 + \alpha_1 + \eta + \gamma_1)I - \frac{I}{N}\frac{dN}{dt} \tag{10}$$

$$\frac{dH}{dt} = \eta I + \theta E - (1 + \alpha_2 + \gamma_2)H - \frac{H}{N}\frac{dN}{dt} \tag{11}$$

$$\frac{dC}{dt} = \gamma_1 I + \gamma_2 H - (1 + \gamma_3)C - \frac{C}{N}\frac{dN}{dt} \tag{12}$$

$$\frac{dD}{dt} = \alpha_1 I + \alpha_2 H - D - \frac{D}{N}\frac{dN}{dt} \tag{13}$$

$$\frac{dR}{dt} = \gamma_3 C - R - \frac{R}{N}\frac{dN}{dt} \tag{14}$$

$$\frac{dN}{dt} = \frac{1}{N_0}\left(\frac{d\bar{N}}{d\bar{t}}\right) = 1 - (1 + \alpha_1 I + \alpha_2 H + \delta\beta_C CS)N \tag{15}$$

where $\beta = \frac{\bar{\beta}}{\bar{\mu}}$, $\beta_D = \frac{\bar{\beta}_D}{\bar{\mu}}$, $\alpha_1 = \frac{\bar{\alpha}_1}{\bar{\mu}}$, $\alpha_2 = \frac{\bar{\alpha}}{\bar{\mu}}$, $\beta_C = \frac{\bar{\beta}_C}{\bar{\mu}}$, $\delta = \frac{\bar{\delta}}{\bar{\mu}}$, $\theta = \frac{\bar{\theta}}{\bar{\mu}}$, $\sigma = \frac{\bar{\sigma}}{\bar{\mu}}$, $\eta = \frac{\bar{\eta}}{\bar{\mu}}$, $\gamma_1 = \frac{\bar{\gamma}_1}{\bar{\mu}}$, $\gamma_2 = \frac{\bar{\gamma}_2}{\bar{\mu}}$, $\gamma_3 = \frac{\bar{\gamma}_3}{\bar{\mu}}$ and the feasible region for solutions of (8)–(13) is considered as

$$\Omega_0 = \{(E,I,H,C,D,R,N) :\ 0 \leqslant E,I,H,C,D,R,N \leqslant 1\}.$$

3. Disease free equilibrium (DFE) and basic reproduction number

The DFE of the system (8)–(14) can be given by $E_0 = (0,0,0,0,0,0,1)$.

To study local stability of DFE, Basic Reproduction number is required.

Apply next generation matrix method suggested by van den Driessche and Watmough (2002) at DFE. Let $X' = \frac{dX}{dt} = \Im(X) - \upsilon(X)$, with $X = (E,I,H,C,D)$ where $\Im(X)$ is the appearance rate of new infected individuals in different class and $\upsilon(X)$ represents the rate of infection transmission in the system, which is given by

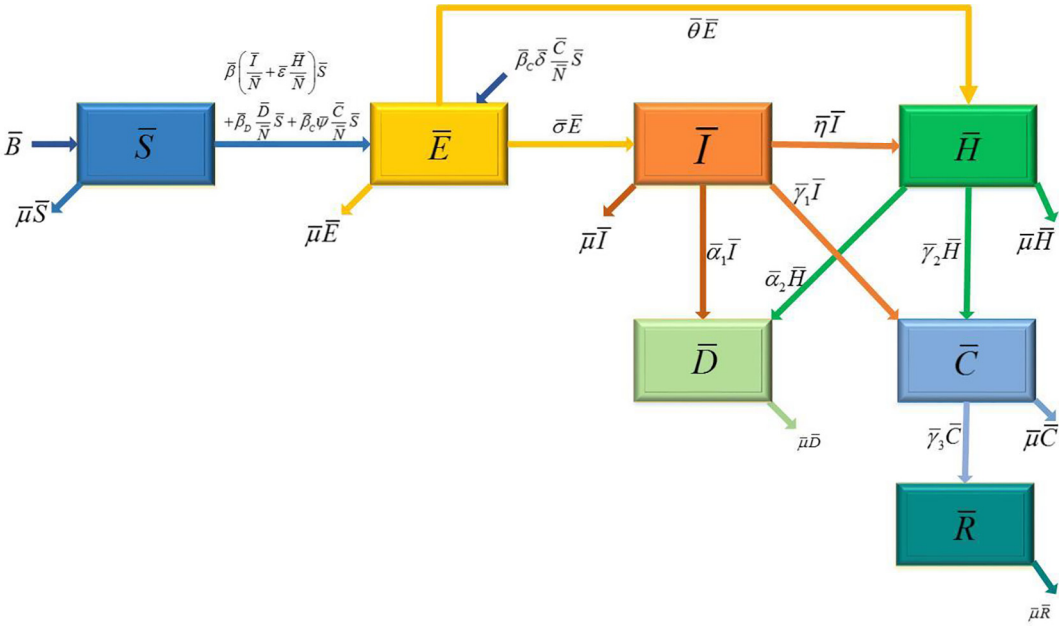

Fig. 1. Disease dynamics in population.

$$\Im(X)=\begin{bmatrix}[\beta(I+\varepsilon H)+\beta_D D+\beta_C(\psi+\delta)C](1-E-I-H-C-D-R)\\0\\0\\0\\0\end{bmatrix}$$

and

$$\upsilon(X)=\begin{bmatrix}(\theta+\sigma+1)E+\left(\frac{1}{N}-1+\alpha_1 I+\alpha_2 H+\delta\beta_C C(1-E-I-H-C-D-R)\right)E\\-\sigma E+(1+\alpha_1+\eta+\gamma_1)I+\left(\frac{1}{N}-1+\alpha_1 I+\alpha_2 H+\delta\beta_C C(1-E-I-H-C-D-R)\right)I\\-\eta I-\theta E+(1+\alpha_2+\gamma_2)H+\left(\frac{1}{N}-1+\alpha_1 I+\alpha_2 H+\delta\beta_C C(1-E-I-H-C-D-R)N\right)H\\-\gamma_1 I-\gamma_2 H+(1+\gamma_3)C+\left(\frac{1}{N}-1+\alpha_1 I+\alpha_2 H+\delta\beta_C C(1-E-I-H-C-D-R)N\right)C\\-\alpha_1 I-\alpha_2 H+D+\left(\frac{1}{N}-1+\alpha_1 I+\alpha_2 H+\delta\beta_C C(1-E-I-H-C-D-R)N\right)D\end{bmatrix}$$

Defining, $F=\left[\frac{\partial\Im_i(X_0)}{\partial X_j}\right]=\begin{bmatrix}0&\beta&\beta\varepsilon&(\psi+\delta)\beta_C&\beta_D\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\end{bmatrix}$ and

$V=\left[\frac{\partial\upsilon_i(X_0)}{\partial X_j}\right]=\begin{bmatrix}\sigma+\theta+1&0&0&0&0\\-\sigma&\alpha_1+\gamma_1+\eta+1&0&0&0\\-\theta&-\eta&\alpha_2+\gamma_2+1&0&0\\0&-\gamma_1&-\gamma_2&\gamma_3+1&0\\0&-\alpha_1&-\alpha_2&0&1\end{bmatrix}$

The basic reproduction number R_0 is spectral radius of FV^{-1}.

$$R_0=\frac{(\beta_1+\beta_{D1}\alpha_1)+(\beta_1\varepsilon+\beta_{D1}\alpha_2)b_3+(\psi+\delta)\beta_{C1}b_5}{b_1 b_{10}}$$

where $b_1=\frac{1+\alpha_1+\gamma_1+\eta}{\sigma}$, $b_{10}=\sigma(\sigma+\theta+1)$.

3.1. Endemic equilibrium

Solving, $\left(\frac{dE(t)}{dt}\right)=0$, $\left(\frac{dI(t)}{dt}\right)=0$, $\left(\frac{dH(t)}{dt}\right)=0$, $\left(\frac{dC(t)}{dt}\right)=0$, $\left(\frac{dD(t)}{dt}\right)=0$ and $\left(\frac{dR(t)}{dt}\right)=0$ simultaneously.

Endemic equilibrium point is $(E^*,H^*,I^*,C^*,D^*,R^*,N^*)=\left(b_1I^*,b_3I^*,I^*,b_5I^*,b_6I^*,b_7I^*,\frac{1}{b_9I^*}\right)$

where $b_2=\sigma(1+\alpha_2+\gamma_2)$, $b_3=\frac{(\theta b_1+\eta)\sigma}{b_2}$, $b_4=1+\gamma_3$, $b_5=\frac{b_3\gamma_2+\gamma_1}{b_4}$, $b_6=\alpha_1+\alpha_2 b_3$, $b_7=\gamma_3 b_5$, $b_8=1+b_1+b_3+b_5+b_6+b_7$ and $b_9=(1+\alpha_1+\alpha_2 b_3+\delta\beta_C b_4(1-b_8)I^*)$ as $S^*=1-b_8I^*$.

Since, $[\beta(I^*+\varepsilon H^*)+\beta_D D^*+\beta_C(\psi+\delta)C^*]S^*-(\sigma+\theta+1)E^*=0$ for $m_1=m_2=m_3=0$ can be converted to $[\beta_1(1+\varepsilon b_3)+\beta_D(\alpha_1+\alpha_2 b_3)+(\psi+\delta)\beta_C b_5](1-b_8I^*)=(\sigma+\theta+1)b_1=0$. Finally, one can get value of I^* as $I^*=\frac{1}{b_8}\left(1-\frac{1}{R_0}\right)$ which proves existence of endemic equilibrium for $R_0>1$ in absence of media effect. For study of compile media impact, I^* can be approximated by solution of quadratic equation $b_{11}b_8(I^*)^2-(R_0b_1b_{10}b_8+b_{11})(I^*)+(R_0b_1b_8-(1+\theta+\sigma)b_1)=0$.

where $b_{11}=(\beta_1-\beta_0)\left(m_1(1+\varepsilon b_2)^2\right)+(\beta_{D1}-\beta_{D0})\left(m_2(\alpha_1+\alpha_2 b_3)^2\right)+(\beta_{C1}-\beta_{C0})\left(m_3b_5^2\right)$.

Clearly, $(R_0b_1b_8-(1+\theta+\sigma)b_1)$ is positive only if $R_0>\frac{1+\theta+\sigma}{b_8}>1$ as b_8 is too small.

Hence, endemic equilibrium of system (9)–(15) exists for $R_0>1$.

4. Stability

4.1. Local stability of DFE

The Jacobian matrix for the system (9)–(15) at DFE can be given by

Table 2
Parametric values.

Parameter	Value	Parameter	Value	Parameter	Value
$\bar{N}(0)$	100,000	$\bar{\beta}_0$	0.05	$\bar{\mu}$	0.14
$\bar{S}(0)$	10,000	$\bar{\beta}_{C0}$	0.05	$\bar{\alpha}_1$	0.2
$\bar{E}(0)$	5000	$\bar{\beta}_{D0}$	0.15	$\bar{\alpha}_2$	0.2
$\bar{I}(0)$	3000	$\bar{\beta}_1$	0.26	$\bar{\sigma}$	0.4
$\bar{H}(0)$	5000	$\bar{\beta}_{C1}$	0.15	$\bar{\eta}$	0.3
$\bar{C}(0)$	1000	$\bar{\beta}_{D1}$	0.3	$\bar{\theta}$	0.5
$\bar{D}(0)$	3000	$\bar{\varepsilon}$	0.05	$\bar{\gamma}_1$	0.3
$\bar{R}(0)$	1000	$\bar{\psi}$	0.2	$\bar{\gamma}_2$	0.4
$\bar{B}$	500	$\bar{\delta}$	0.1	$\bar{\gamma}_3$	0.7
$\bar{m}_1$	0–10,000	$\bar{m}_2$	0–10,000	$\bar{m}_3$	0–10,000

$$J=\begin{pmatrix} -1-\sigma-\theta & \beta & \beta\varepsilon & (\psi+\delta)\beta_C & \beta_D & 0 & 0 \\ \sigma & -1-\alpha_1-\gamma_1-\eta & 0 & 0 & 0 & 0 & 0 \\ \theta & \eta & -1-\alpha_2-\gamma_2 & 0 & 0 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -1-\gamma_3 & 0 & 0 & 0 \\ 0 & \alpha_1 & \alpha_2 & 0 & -1 & 0 & 0 \\ 0 & 0 & 0 & \gamma_3 & 0 & -1 & 0 \\ \delta\beta_C & -\alpha_1+\delta\beta_C & -\alpha_2+\delta\beta_C & \delta\beta_C & \delta\beta_C & \delta\beta_C & -1-\delta\beta_C \end{pmatrix}$$

$trace(J) = -3-\sigma b_1-\frac{b_2}{\sigma}-b_3-\frac{b_{10}}{\sigma}-\delta\beta_C<0$ and principal minors of J are

$$A_1=-\frac{b_{10}}{\sigma}<0,\ A_2=b_{10}b_1-B\sigma>0,$$

$$A_3=-\left(\frac{b_{10}b_1b_2-(1+b_3\varepsilon)\sigma\beta b_2}{\sigma}\right)<0,$$

$$A_4=\frac{b_{10}b_1b_2b_4-(1+b_3\varepsilon)\sigma\beta b_2b_4-b_5b_4b_2\sigma\beta_C(\psi+\delta)}{\sigma}>0,$$

$$A_5=-\left(\frac{b_{10}b_1b_2b_4-(1+b_3\varepsilon)\sigma\beta b_2b_4-b_5b_4b_2\sigma\beta_C(\psi+\delta)-b_6b_2b_4\sigma\beta_D}{\sigma}\right)<0,\ A_6=-A_5>0$$

$$A_7=b_1b_2b_4b_{10}(R_0-1)(1+\delta\beta_C).$$

$A_7<0\iff R_0<1$. It leads to the conclusion that the system (8)–(13) is locally stable if and only if $R_0<1$, as all eigenvalues of J are negative if and only if it's all principal minors have opposite signs.

4.2. Global stability of DFE

Applying the method used by Shau and Dhar (2015), at DFE $\alpha_1=\alpha_2=C=0$, hence $\frac{dN}{dt}=1-N$. As $t\to\infty,\ N\to 1$. So for limiting case the system of Eqs. (8)–(13) will reduce to,

$$\frac{dE}{dt}=[\beta(I+\varepsilon H)+\beta_D D+\beta_C(\psi+\delta)C]S-(\theta+\sigma+1)E \tag{16}$$

$$\frac{dI}{dt}=\sigma E-(1+\alpha_1+\eta+\gamma_1)I \tag{17}$$

$$\frac{dH}{dt}=\eta I+\theta E-(1+\alpha_2+\gamma_2)H \tag{18}$$

$$\frac{dC}{dt}=\gamma_1 I+\gamma_2 H-(1+\gamma_3)C \tag{19}$$

$$\frac{dD}{dt}=\alpha_1 I+\alpha_2 H-D \tag{20}$$

$$\frac{dR}{dt}=\gamma_3 C-R \tag{21}$$

Let $X=(R)$ and $Z=(E,I,H,C,D)$.

At DFE, $X^0=(0)$ and $Z^0=(0,0,0,0,0)$.

Then, $\frac{dX}{dt}\left(=\frac{dR}{dt}\right)=F(X,Z)=\gamma_3 C-R$. Hence, at $Z=Z^0,\ F(X,0)=-X(=-R)$.

So, as $t\to\infty,\ \frac{dX}{dt}\to X^0=0$. Thus, $X=X^0$ is globally asymptotically stable.

From Eqs. (16)–(20), $\frac{dZ}{dt}\left(=\frac{d(E,I,H,C,D)}{dt}\right)=G(X,Z)=BZ-\hat{G}(X,Z)$.

The Jacobian matrix for the system (8)–(13) at DFE can be given by

$$B=\begin{pmatrix} -1-\sigma-\theta & \beta & \beta\varepsilon & (\psi+\delta)\beta_C & \beta_D \\ \sigma & -1-\gamma_1-\eta & 0 & 0 & 0 \\ \theta & \eta & -1-\gamma_2 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -1-\gamma_3 & 0 \\ 0 & 0 & 0 & 0 & -1 \end{pmatrix}$$

B is an M-Matrix.

$$\hat{G}(X,Z)=\begin{pmatrix} (\beta(I+\varepsilon H)+\beta_D D+\beta_C(\psi+\delta)C)(1-S) \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}\geqslant 0 \text{ as } S<1.$$

which satisfies conditions (H_1) and (H_2) of Castillo-Chavez et al. (2002). Therefore, DFE is globally asymptotically stable if $R_0<1$.

4.3. Global stability of endemic equilibrium

Consider, Lyapunov function$V:\mathcal{R}_+^7\to R$, defined as

$$V(\bar{X})=\left[E-E^*-E^*\ln\left(\frac{E}{E^*}\right)\right]+\left[I-I^*-I^*\ln\left(\frac{I}{I^*}\right)\right]+\left[H-H^*-H^*\ln\left(\frac{H}{H^*}\right)\right]+\left[D-D^*-D^*\ln\left(\frac{D}{D^*}\right)\right]+\left[C-C^*-C^*\ln\left(\frac{C}{C^*}\right)\right]+\left[R-R^*-R^*\ln\left(\frac{R}{R^*}\right)\right]$$

where $\bar{X}=(E,I,H,C,D,R)$.

Here, $V(\bar{X})>0$ for $\forall\ \bar{X}\in\Omega-\{0\}$ and $V(0)=0$.

Also, $\frac{dV}{dX}=1-b_8I^*\left((R_0\frac{E}{b_1}-I)-1\right)$.

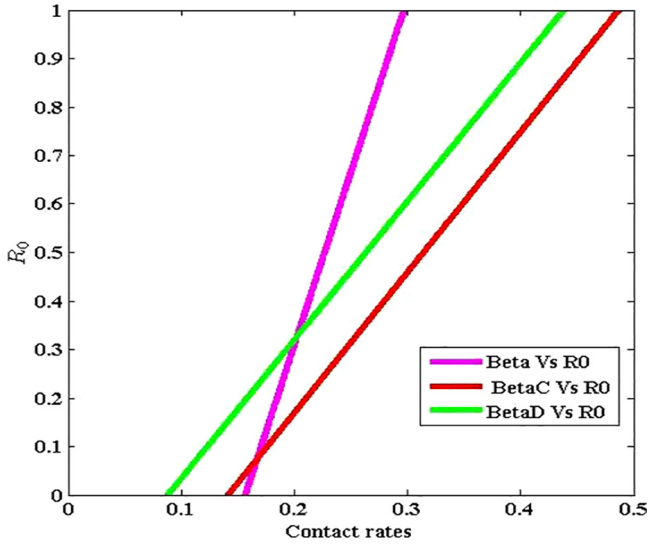

Fig. 2. Effect of change in contact rates on R_0.

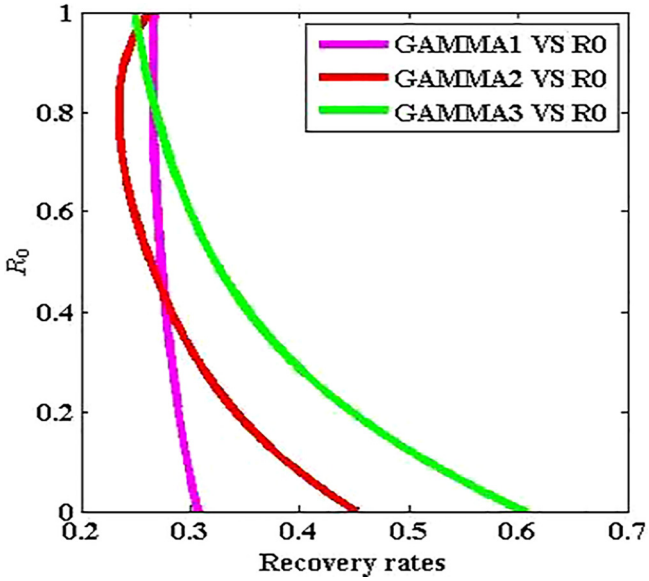

Fig. 3. Effect of change in recovery rates on R_0.

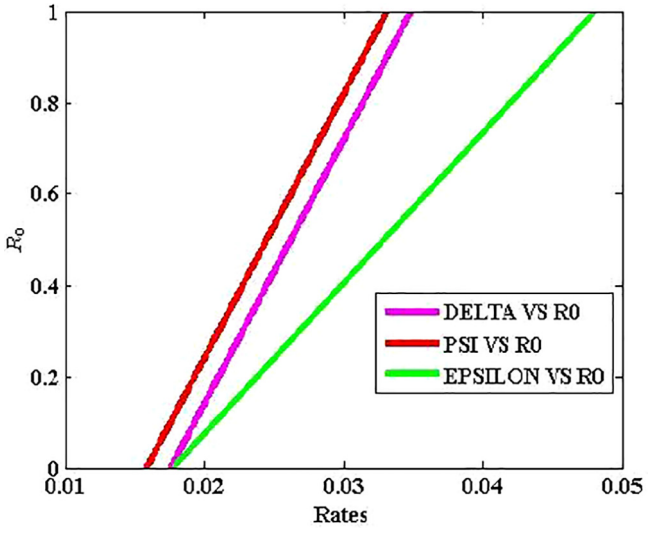

Fig. 4. Effect of change in $\bar{\delta}$, $\bar{\psi}$, $\bar{\varepsilon}$ on R_0.

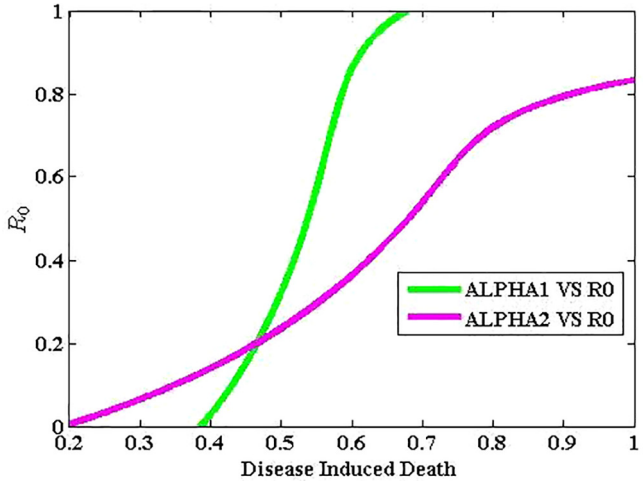

Fig. 6. Effect of change in $\bar{\alpha}_1$ and $\bar{\alpha}_2$ on R_0.

Hence, $\frac{dV}{dX} < 0 \iff R_0 > 1$.

Thus, stability criteria of Lyapunov (1992), guarantees that endemic equilibrium is stable when $R_0 > 1$.

5. Numerical simulation and observation

Numerical simulation is carried out with the parametric values of disease parameters as tabulated in Table 2 in appropriate units. For tabulated parameters, the basic reproduction number $R_0 = 0.966032$. Next sensitivity of R_0 with different disease parameters is verified to understand disease spread possibilities.

In Fig. 2, the effects of change in contact rates $\bar{\beta}$, $\bar{\beta}_C$ and $\bar{\beta}_D$ on R_0 are shown. It is observed that increase in these contact rates will also increase R_0 and hence disease will persist for long time in population. Also it can be seen that sensitivity of R_0 with $\bar{\beta}$ is much higher than $\bar{\beta}_C$ and $\bar{\beta}_D$.

In Fig. 3, the effects of change in recovery rates $\bar{\gamma}_1$, $\bar{\gamma}_2$ and $\bar{\gamma}_3$ on R_0 are described. It is observed that increase in recovery rates will decrease R_0, which finally makes stable and control system.

In Fig. 4, the effects of changes in parameters $\bar{\delta}$, $\bar{\psi}$, $\bar{\varepsilon}$ on R_0 are plotted. It can be observed that increase in probability of a female individual get infected due to sexual activity $\bar{\delta}$, probability of virus enter direct in placenta due to sexual activity $\bar{\psi}$ and probability of disease spread due to leakage in isolation $\bar{\varepsilon}$ will also increase R_0.

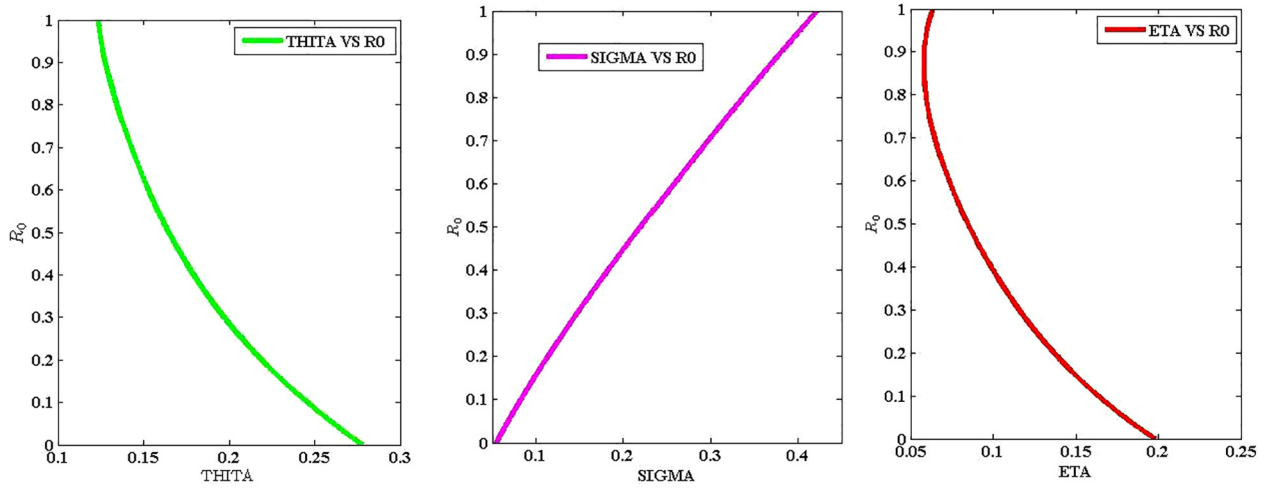

Fig. 5. Effect of change in $\bar{\theta}$, $\bar{\sigma}$, $\bar{\eta}$ on R_0.

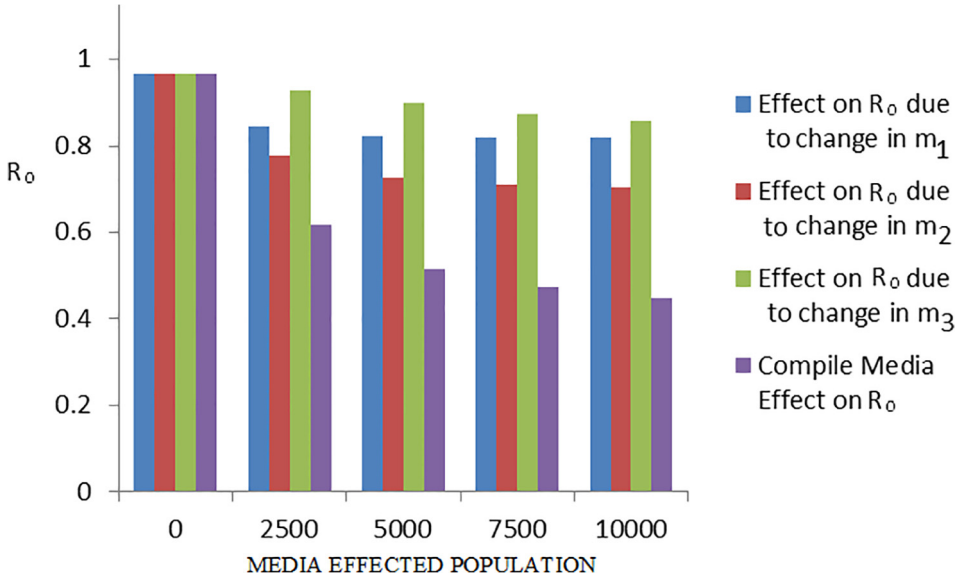

Fig. 7. Effect of change in m_1, m_2, m_3 on R_0 through β, β_C, β_D.

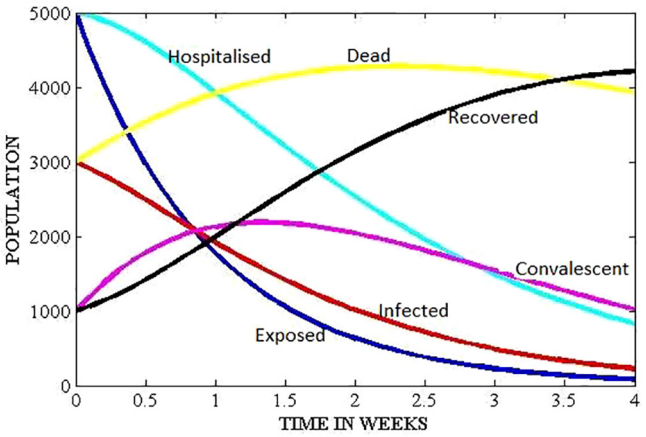

Fig. 8. Global stability of the system (1) for $R_0 < 1$.

Fig. 5 indicates the effects of change in parameters $\bar{\theta}$, $\bar{\sigma}$, $\bar{\eta}$ on R_0. It can be observed that increase in early hospitalisation of exposed individuals with rate $\bar{\theta}$ and hospitalisation of infected with rate $\bar{\eta}$ will help to control disease resulting decrease of R_0. Increase in movement of exposed towards infected class with rate $\bar{\sigma}$ will also spread Ebola at a faster rate.

From Fig. 6, it can be noted that as disease induced death will increase R_0 will also increase. This is due to more infected deaths will create chaos on proper burial process, which finally results in higher disease spread.

As R_0 is not directly depended on media but contact rates are depended on media hence in Fig. 7 the effects of change in m_1, m_2, m_3 on R_0 through $\bar{\beta}$, $\bar{\beta}_C$, $\bar{\beta}_D$ are plotted. It can be noted that as awareness through media i.e. number of aware individuals by media increase, R_0 will decrease. It can also be observed that the compile effect of media is much higher than separate media effects to control R_0 and hence to control disease spread. Media plays vital role in controlling disease.

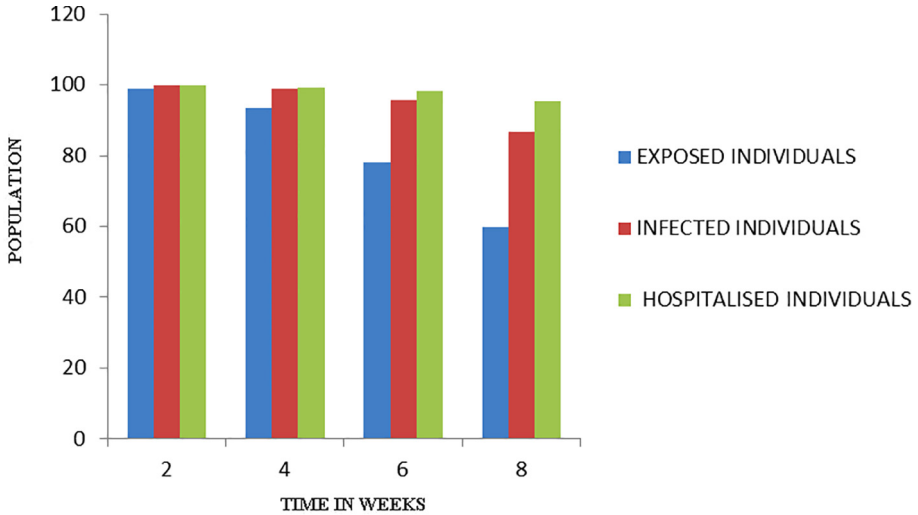

Fig. 9. Decrease in population percentage of various classes due to media effect with time.

In Fig. 8, flow of population with time is described, it can be observed that if proper preventive measures like isolation, media awareness, proper burial process are taken to keep $R_0 < 1$, then it will effectively control disease spred and reduce exposed, infected, convolecent, death individuals and increase recovered individuals successively.

In Fig. 9, population percentage of various classes like exposed, infected and hospitalised with media effect are compared with fixed hundred value of each individuals without media effect. It is observed that with media effect at a gap of 2, 4, 6 and 8 weeks, exposed individuals reduce to 98, 95, 80, 60, infected individuals reduce to 99, 97, 93, 90 while hospitalised individuals reduce to 99, 98, 96, 94 with respect to 100 fixed exposed, infected, hospitalised individuals without media effect.

6. Conclusions

In this paper, vertical dynamics of Ebola with media impact is discussed. The dimensionless system is derived. The basic reproduction number is calculated using the next generation matrix method. Stability at the equilibrium states for model parameters along with numerical simulation is carried out. The effects of media awareness, isolation or early hospitalisation of exposed individuals, sanitary burial process on endemic behaviour of the model are investigated. It is observed that the mass media is one of the most effective ways of creating awareness on Ebola disease spread. Informative awareness of disease amongst public may curb the disease spread and help to control disease in initial stage. Early diagnosis of pre-symptomatic individuals and their effective isolation helps to decrease basic reproduction number. It is suggested to avoid leakage in isolation class as it increases disease spread. Unsafe sexual activities during disease breakout may infect placenta which promotes disease spread amongst offspring. Early diagnosis, isolation of exposed, safe sexual activities, sanitary burial practices with effective mass media awareness are crucial controls to stop disease spread. This research can be extended to study the negative impact of media on the population about the disease and cost effective control can be incorporated. One can also study the health sectors role in medication and burial process by providing proper kit.

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References

Adebamowo, C., Bah-Sow, O., Binka, F., et al., 2014. Randomised controlled trials for Ebola: practical and ethical issues. Lancet 384, 1423–1424.
Baize, S., Pannetier, D., Oestereich, L., Rieger, T., Koivogui, L., Magassouba, N., Soropogui, B., Sow, M.S., Keïta, S., DeClerck, H., et al., 2014. Emergence of Zaire Ebola virus disease in Guinea – Preliminary Report. New Engl. J. Med. 371 (15), 1418–1422. https://doi.org/10.1056/NEJMoa1404505. PMID: 24738640.
Basch, C.H., Basch, C.E., Redlener, I., 2014. Coverage of the Ebola virus disease epidemic in three widely circulated United States newspapers: implications for preparedness and prevention. Health Promotion Perspect. 4, 247–251.
Bausch, D.G., Towner, J.S., Dowell, S.F., et al., 2007. Assessment of the risk of Ebola virus transmission from bodily fluids and fomites. J. Infect. Dis. 196 (Suppl. 2), S142–147.
Castillo-Chavez, C., Feng, Z., Huang, W., 2002. On the computation $\mathscr{R}_0$ and its role on global stability. In: Castillo-Chavez, Carlos, Blower, Sally, Driessche, Pauline, Kirschner, Denise, Yakubu, Abdul-Aziz (Eds.), Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction, The IMA Volumes in Mathematics and its Applications, vol. 125. Springer-Verlag, Berlin-Heidelberg-New York, pp. 229–250.
Centers for Disease Controls and Prevention, 2015. Possible sexual transmission of Ebola virus – Liberia. MMWR Morbid. Mortality Weekly Rep. 64 (17), 479–481.
Centers for Disease Controls and Prevention, 2016. Ebola Virus Disease, https://www.cdc.gov/vhf/ebola/ (accessed on 16 February 2017).
Chowell, D., Castillo-Chavez, C., Krishna, S., Qiu, X.G., Anderson, K.S., 2015. Modelling the effect of early detection of Ebola. Lancet Infect. Dis. 15, 148–149.
Chowell, G., Nishiura, H., 2014. Transmission dynamics and control of Ebola virus disease (EVD): a review. BMC Med. 12, 196.
Dhillon, R.S., Srikrishna, D., Garry, R.F., Chowell, G., 2015. Ebola control: rapid diagnostic testing. Lancet Infect. Dis. 15, 147–148.
Feldmann, H., Geisbert, T.W., 2011. Ebola haemorrhagic fever. Lancet 377, 849–862.
Galvani, A.P., Ndeffo-Mbah, M.L., Wenzel, N., Childs, J.E., 2014. Ebola vaccination: if not now, when? Ann. Intern. Med. 161, 749–751.
Househ, M., 2015. Communicating Ebola through social media and electronic news media outlets: a cross-sectional study. Health Inform. J., 1–9 https://doi.org/10.1177/1460458214568037.
Kucharski, A.J., Edmunds, W.J., 2014. Case fatality rate for Ebola virus disease in West Africa. Lancet 384, 1260.
Kupferschmidt, K., 2014. Estimating the Ebola epidemic. Science 345, 1108.
Legrand, J., Grais, R.F., Boelle, P.Y., et al., 2007. Understanding the dynamics of Ebola epidemics. Epidemiol. Infect. 135, 610–621.
Liu, R., Wu, J., Zhu, H., 2007. Media/psychological impact on multiple outbreaks of emerging infectious diseases. Comput. Math. Methods Med. 8 (3), 153–164.
Love, C.B., Arnesen, S.J., Phillips, S.J., 2014. Ebola outbreak response: the role of information resources and the national library of medicine. Disaster Med. Publ. Health Preparedness 9, 82–85.
Lyapunov, A.M., 1992. The General Problem of the Stability of Motion Translated and edited by A. T. Fuller. Taylor & Franscis, London.
Martin-Moreno, J.M., Llinás, G., Hernández, J.M., 2014. Is respiratory protection appropriate in the Ebola response? Lancet 384, 856.
Shen, Mingwang, Xiao, Yanni, Rong, Libin, 2015a. Modeling the effect of comprehensive interventions on Ebola virus transmission. Sci. Rep. https://doi.org/10.1038/srep15818.
Pandey, A., Atkins, K.E., Medlock, J., et al., 2014. Strategies for containing Ebola in West Africa. Science 346, 991–995.
Pang, J., Cui, I.-A., 2009. An SIRS epidemiological model with nonlinear incidence rate incorporating media coverage. In: Second International Conference on Information and Computing Science, IEEE, pp. 116–119.
Pourrut, X., Kumulungui, B., Wittmann, T., et al., 2005. The natural history of Ebola virus in Africa. Microbes Infect. 7, 1005–1014.
van den Driessche, P., Watmough, James, 2002. Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission. Math. Biosci. 180 (2002), 29–48.
Rodriguez, L.L., De Roo, A., Guimard, Y., et al., 1999. Persistence and genetic stability of Ebola virus during the outbreak in Kikwit, Democratic Republic of the Congo, 1995. J. Infect. Dis. 179 (Suppl. 1), S170–176.
Safi, M.A., Gumel, A.B., 2011. Mathematical analysis of a disease transmission model with quarantine, isolation and an imperfect vaccine. Comput. Math. Appl. 61 (10), 3044–3070.
Shau, G.P., Dhar, J., 2015. Dynamics of an SEQIHRS epidemic model with media coverage, quarantine and isolation in a community with pre-existing immunity. J. Math. Anal. Appl. 421, 1651–1672. https://doi.org/10.1016/j.jmaa.2014.08.0190022-247X/.
Shen, M., Xiao, Y., Rong, L., 2015b. Modeling the effect of comprehensive interventions on Ebola virus transmission. Sci. Rep. 2015. https://doi.org/10.1038/srep15818.
Smailhodvic A., Andrew K., Hahn L., Womble, P.C., Webb, C., 2014. Sample NLPDE and NLODE Social-Media Modeling of Information Transmission for Infectious Diseases: Case Study Ebola. http://arxiv.org/ftp/arxiv/papers/1501/1501.00198.pdf.
Sun, C., Yang, W., Arino, J., Khan, K., 2011. Effect of media-induced social distancing on disease transmission in a two patch setting. Math. Biosci. 230 (2), 87–95.
Wang, A., Xiao, Y., 2014. A Filippov system describing media effects on the spread of infectious diseases. Nonlinear Anal.: Hybrid Syst. 11 (1), 84–97.
WHO, 2014. Global Alert and Response (GAR), Ebola Outbreak. Pandemic and Epidemic Diseases Ebola-Situation report. http://www.who.int/csr/disease/ebola/en/.
World Health Organisation, 2016. Ebola Virus Disease Fact Sheet, January 2016. http://www.who.int/mediacentre/factsheets/fs103/en/.
World Health Organization, 2015. Ebola Situation Report-25 March 2015. http://apps.who.int/ebola/current-situation/ebola-situationreport-25-march-2015.