Dynamical interplay between $UARA$ awareness and $SVI_1I_2..I_nR$ epidemic model over multiplex network: A case study on EVD

Padmavathi Ramamoorthi, Anuradha Arumugam, Senthilkumar Muthukrishnan

Assistant Professor,PG & Research department of Mathematics,
Sri Ramakrishna college of Arts & Science,
Coimbatore, India. padmavathi@srcas.ac.in
Assistant Professor,PG & Research department of Mathematics,
Sri Ramakrishna college of Arts & Science,
Coimbatore, India. anuradha@srcas.ac.in
Professor, Department of Applied Mathematics and Computational Sciences,
PSG College of Technology,Coimbatore,India. msk.amcs@psgtech.ac.in

Abstract

Evolution of real world networks capturing the spreading dynamics of epidemic and human awareness sheds light on modeling of various diseases/ rumours / memes spreading over network. The awareness through mass media and social media play crucial role in controlling the epidemic. In this paper,we propose the dynamical interplay between spreading dynamics of epidemic over physical contact layer and human awareness diffusion over virtual contact layer,over a multiplex network framework. Spreading process of epidemic follows $SVI_1I_2.....I_nR$ process and human awareness diffusion follows UARA process. The awareness induced vaccination is taken for modeling. Competing dynamics of spreading process of epidemic and human awareness diffusion are studied based on Microscopic Markov Chain Approach. Next Generation Matrix is used to find R_0 and global asymptotic stability of disease free equilibrium is proved. The finding of the epidemic threshold of epidemic spread with various number of infectious stages and the control of epidemic spread through vaccination are corroborated through numerical simulations. Fraction of recovered for different number of infected stages and the controlling of vaccination for various rate of vaccination are illustrated with several real multiplex networks. The influence of awareness factor in the epidemic spread is illustrated. The fraction of recovered and vaccinated for various rate of infection spread and awareness diffusion are also found. The model is well suitable to show the Ebola Virus Disease(EVD) spread, by taking n=3. The theoretical values are compared with EVD spread in Guinea and Liberia during 2014 - 2015.

Keywords: Epidemic spread; Awareness diffusion; Optimum Control; Social mobility; Ebola.

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1 Introduction

Spreading dynamics of diseases, rumours, opinions and memes over on-line social networks has become an emerging field of research. In the past few years, plenty of research works focus on the way to control the disease spread and to eradicate infection. Mathematical modelling helps to incorporate critical issues like immunization and vaccination policies in the epidemic theory [1]-[2]. In [3]-[4], spreading dynamics of multi stage infection SIS model with propagation vector is discussed. In [5]-[18], MMCA is used to represent disease and awareness diffusion in multiplex network.

Many works investigate UAU process in the information layer [5]-[16]. In [17], epidemic threshold for UAR-SIR process is found numerically but it is not derived analytically. In [18], only the outbreak threshold of information diffusion is found for UAR-SVIR process. This paper investigates $SVI_1I_2\dots I_nR$ process in the epidemic layer and $UARA$ process in the information layer, awareness via both mass media and social media are considered. Our contributions are i) Epidemic with n-stage infection($I_1I_2\dots I_n$) is analysed which is used to model diseases like EVD ii) When a disease starts to spread in a region, government creates awareness, prevention measure and the importance of vaccination through mass media. People by their own interest spread awareness to their neighbours either via direct or via social media. Due to awareness, people show interest(l) to go for vaccination iii) When people receive awareness about a disease, prevention measures become part of their routine but they spread the awareness to their neighbours in a small period of time. After that they do not show interest to spread awareness. If they continuously get awareness from mass media, they once again spread awareness to their neighbours. R-Removed state is used to denote the state at which people follow prevention measures and not spread awareness to their neighbours. iv) Next generation matrix(NGM) is used to derive epidemic threshold.

The layout of the paper is as follows: In section 2, EVD symptoms, propagation and preventions are discussed. In Section 3, mathematical model consisting of epidemic layer, information layer and multiplex layer are described. Probability transition tree is constructed and using it, MMCA equations are derived and epidemic threshold using NGM is found in Section 4. In Section 5, numerical simulation is carried out and the results are discussed. The effect of the awareness factors are also discussed. Section 6 compares the theoretical values with WHO EVD spreading data in the countries Guinea, Liberia during 2014 to 2015. Section 7 concludes with the concluding remarks.

2 Ebola Virus Disease(EVD) symptoms, propagation and preventions

Ebola is a deadly disease caused by virus. It takes 2 to 21 days, after getting contact with the virus, to show the symptoms like fever, headache, sore throat, tiredness, vomiting, diarrhoea. The virus affects the immune system and causes heavy bleeding inside the body hence damages almost all the inner organs. It spreads through the direct contact of blood, secretions or bodily fluids of infected persons or animals. Contact of virus contaminated surfaces or objects also transmit the infection. The virus is able to survive on objects for a few hours in a dried state, and can survive for a few days within body fluids outside of a person. It enters into healthy persons via nose, mouth, eyes, open wounds, cuts and abrasions. The WHO advised the people who take of the infected people and person who handle the contaminated objects should wear protective clothing including masks, gloves, gowns and goggles. Individuals

are advised to avoid contact with infected people and follow frequent hand washing using soap and water. The travellers are asked to isolate them for 21 days[19],[20].

3 Mathematical Model

This model is a multiplex network formed by two layers, Epidemic layer is formed by network of physical contacts and Information layer is formed by network of social contacts via Facebook, Twitter, WhatsApp etc. Nodes in the two layers represent the same entities, edges represents contacts between them and the information layer has additional edges to represents contacts via social media. A node at the top represents mass media which continuously creates awareness.

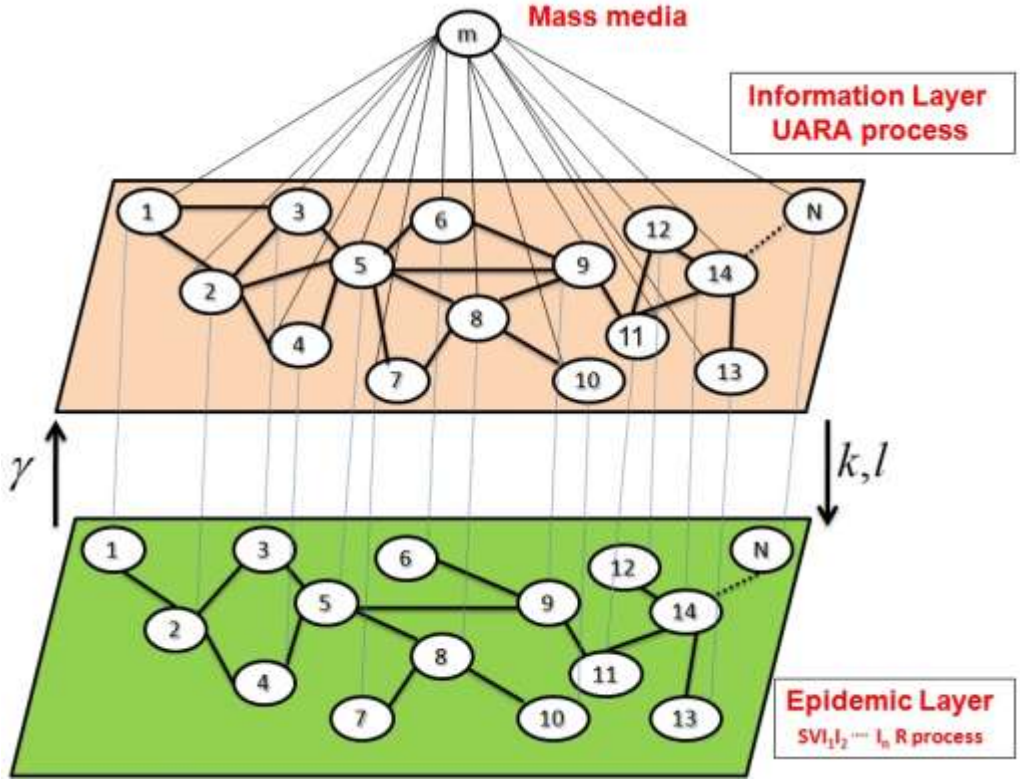


Figure 1: Model description for $UARA - SVI_1I_2I_3 \ldots I_nR$ dynamics in two layer networks.

The upper layer supports information spreading and the lower layer supports epidemic spreading. A node at the top represents mass media, which provide awareness to all the nodes.

Table 1: Parameters and their description

Para meters	**Description**
a_{ij} (b_{ij})	Adjacency matrix in the information (epidemic) layer.
d	average degree of the network in the epidemic layer.
n	number of infected stages.
$\beta^A(\beta^U)$	Infection rate for an aware (unaware) and susceptible individual.
η_i	transmission rate from i^{th} infected stage to $(i+1)^{th}$ infected stage.
δ_E	recovery rate for an infected individual.
λ	transmission rate from the unaware state to aware state.
δ_I	transmission rate from the aware state to removed state.
γ	infection reduction factor when a susceptible agent is aware about the epidemics.
m	rate of an agent being informed periodically by mass media during the epidemics.
k	rate of infected individual spreads awareness to their neighbours.
l	rate of aware individual go for vaccination.
$r_i(t)$	probability of a node i not being informed in the information layer.
$q_i^U(t)$ $(q_i^A(t))$	probability of an unaware(aware) agent i not being infected in the epidemic layer.

3.1 Epidemic Layer

The WHO states that people who are very sick are able to spread Ebola disease through bodily fluids[19]. It is assumed that infected in n^{th} stage can transmit the disease and the probability that susceptible node gets infection after a contact with another infected node in stage I_n is β. The probability of none of the neighbours of i infect it is $q_i(t) = \prod_j (1 - b_{ji}p_j^{I_n}(t)\beta)$, $\beta = \beta^U$ for unaware node, $\beta = \gamma\beta^U = \beta^A$ for aware node, $0 \leq \gamma \leq 1$. It is suitable to describe the spreading dynamics of ebola virus in a region, when n is taken as 3, I_1, I_2, I_3 respectively represent Suspected, Probable, Confirmed.

3.2 Information Layer

The probability of an unaware node becoming aware after getting contact with aware node is λ. The probability of none of the neighbours of i providing information to it is $r_i(t) = \prod_j (1 - a_{ji}p_j^A(t)\lambda)$.

3.3 Multiplex Network

People who are in susceptible state and got awareness about the disease will go for vaccination with probability l. Some people would like to prevent their neighbours from infection which they got. They spread awareness to their neighbours. The probability of infected people spread awareness about the disease to their neighbours is $k, 0 \leq k \leq 1$. A node which is aware in the information layer will take measures for preventing infection, the parameter γ regulates the probability of a node to get infection. $\gamma = 0$ represents total immunization, $\gamma = 1$ represents no effects for awareness on the epidemics. Fig 2 shows the flow between states in multiplex network.

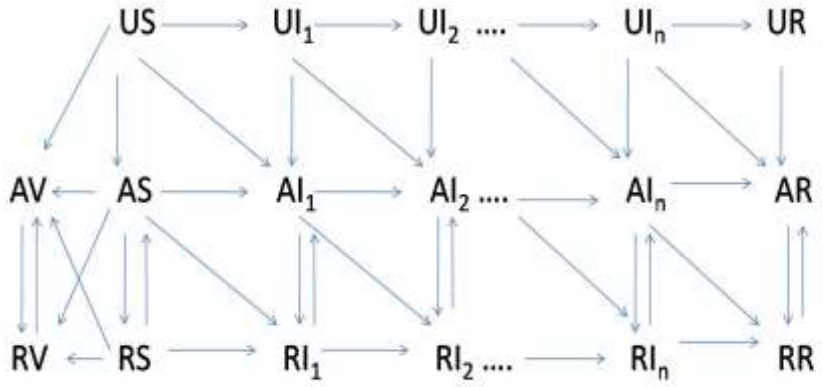


Figure 2: Flow of states in multiplex network

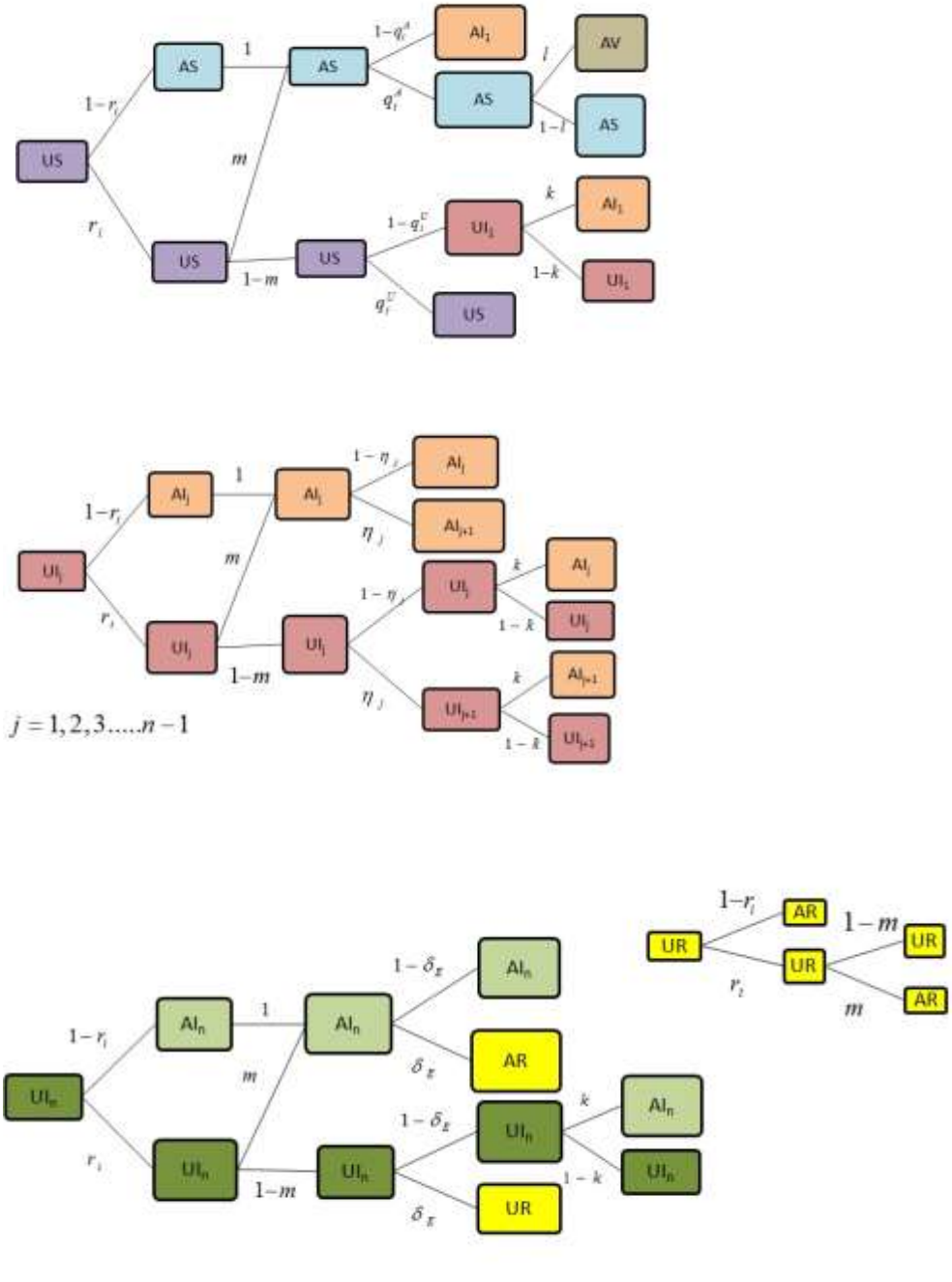


Figure 3: Probability transition trees for unaware node

4 Theoretical MMCA

The possible states of a node are $US, AS, RS, AV, RV, UI_j, AI_j, RI_j, UR, AR, RR$, $j = 1,2,3,\ldots n$, the infection has n stages. The probability trees, figures 3,4, are used to understand the transmission from one state to another state [5]-[16],[21].

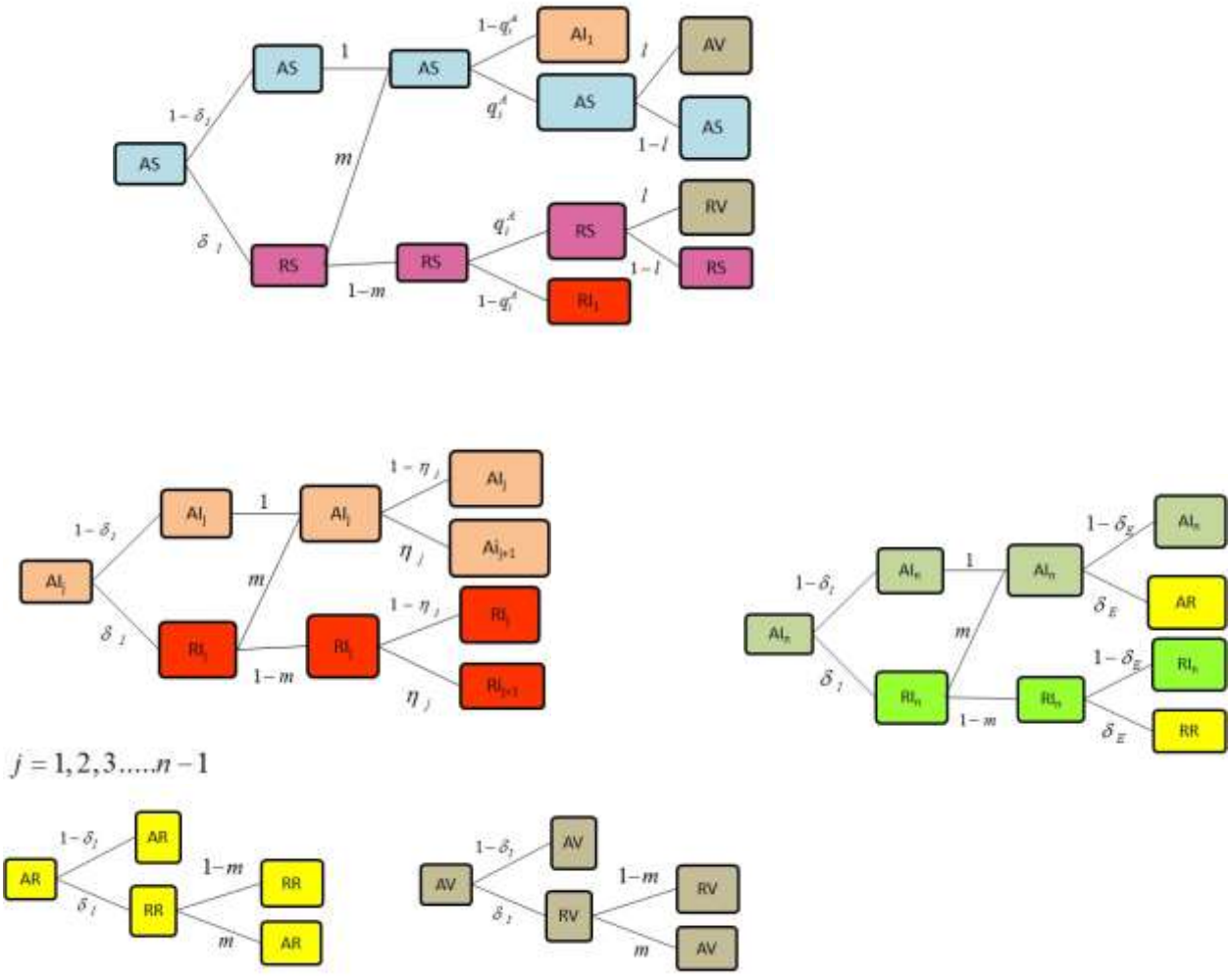
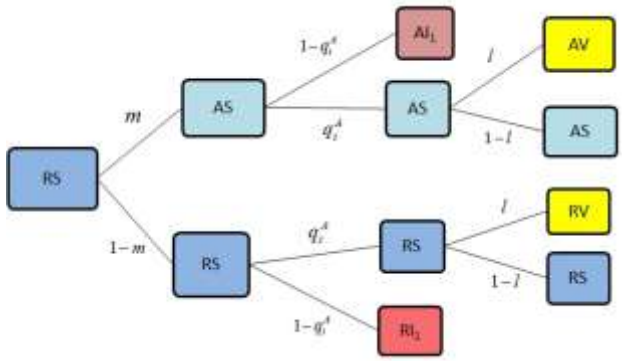


Figure 4: Probability transition trees for aware node



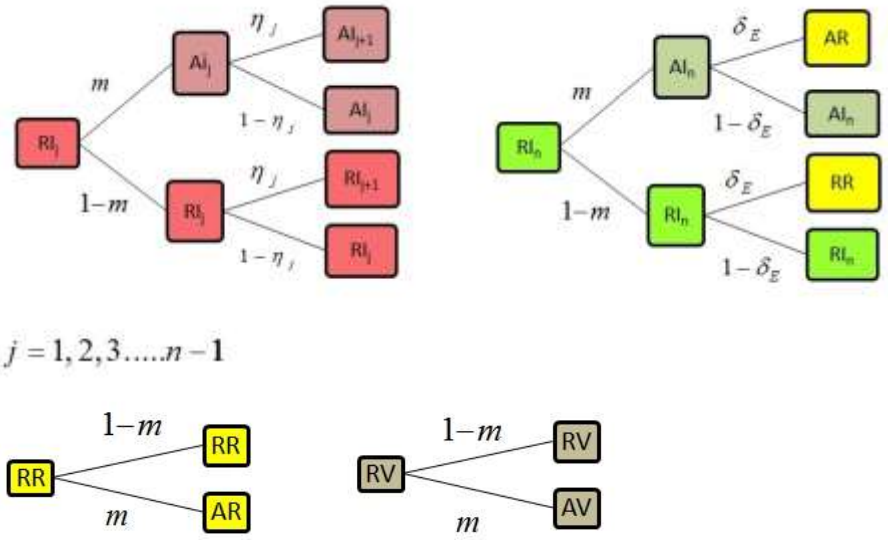


Figure 5: Probability transition trees for removed node

$p_i^{US}(t)$ and $p_i^{US}(t+1)$ denote probability of node i to be in state US at time t and t+1 respectively. Similar notation is used for all the other states. The MMCA equations ?? to ?? represent the probability of every node being in each of the possible states. Solving the system of equations (1) to (18) iteratively, we can find the time evolution of the awareness and the epidemics for any initial condition.

$$p_i^{US}(t+1) = p_i^{US}(t)[r_i(t)(1-m)q_i^U(t)] \quad (1)$$

$$\begin{aligned} p_i^{AS}(t+1) = \; & p_i^{US}(t)[r_i(t)mq_i^A(t)(1-l) + (1-r_i(t))q_i^A(t)(1-l)] + p_i^{AS}(t) \\ & [\delta_I mq_i^A(t)(1-l) + (1-\delta_I)q_i^A(t)(1-l)] + p_i^{RS}(t)[mq_i^A(t)(1-l)] \end{aligned} \quad (2)$$

$$p_i^{RS}(t+1) = p_i^{AS}(t)[\delta_I(1-m)q_i^A(t)(1-l)] + p_i^{RS}(t)[(1-m)q_i^A(t)(1-l)] \quad (3)$$

$$\begin{aligned} p_i^{UI_1}(t+1) = \; & p_i^{US}(t)[r_i(t)(1-m)(1-q_i^U(t))(1-k)] + p_i^{UI_1}(t) \\ & [r_i(t)(1-m)(1-\eta_1)(1-k)] \end{aligned} \quad (4)$$

$$\begin{aligned} p_i^{AI_1}(t+1) = \; & p_i^{US}(t)[r_i(t)m(1-q_i^A(t)) + r_i(t)(1-m)(1-q_i^U(t))k + \\ & (1-r_i(t))(1-q_i^A(t))] + p_i^{AS}(t)[\delta_I m(1-q_i^A(t)) + (1-\delta_I) \\ & (1-q_i^A(t))] + p_i^{RS}(t)[m(1-q_i^A(t))] + p_i^{UI_1}(t)[r_i(t)m(1-\eta_1) + \\ & r_i(t)(1-m)(1-\eta_1)k + (1-r_i(t))(1-\eta_1)] + p_i^{AI_1}(t)[\delta_I m \\ & (1-\eta_1) + (1-\delta_I)(1-\eta_1)] + p_i^{RI_1}(t)[m(1-\eta_1)] \end{aligned} \quad (5)$$

$$\begin{aligned} p_i^{RI_1}(t+1) = \; & p_i^{AS}(t)[\delta_I(1-m)(1-q_i^A(t))] + p_i^{RS}(t)[(1-m)(1-q_i^A(t))] + \\ & p_i^{AI_1}(t)[\delta_I(1-m)(1-\eta_1)] + p_i^{RI_1}(t)[(1-m)(1-\eta_1)] \end{aligned} \quad (6)$$

$$p_i^{UI_j}(t+1) = p_i^{UI_{j-1}}(t)[r_i(t)(1-m)\eta_{j-1}(1-k)] + p_i^{UI_j}(t)[r_i(t)(1-m)(1-\eta_j)(1-k)] \quad (7)$$

$$p_i^{AI_j}(t+1) = p_i^{UI_{j-1}}(t)[r_i(t)m\eta_{j-1} + r_i(t)(1-m)\eta_{j-1}k + (1-r_i(t))\eta_{j-1}] + p_i^{AI_{j-1}}(t)[\delta_I m\eta_{j-1} + (1-\delta_I)\eta_{j-1}] + p_i^{RI_{j-1}}(t)[m\eta_{j-1}] + p_i^{UI_j}(t)[r_i(t)m(1-\eta_j) + r_i(t)(1-m)(1-\eta_j)k + (1-r_i(t))(1-\eta_j)] + p_i^{AI_j}(t)[\delta_I m(1-\eta_j) + (1-\delta_I)(1-\eta_j)] + p_i^{RI_j}(t)[m(1-\eta_j)] \quad (8)$$

$$p_i^{RI_j}(t+1) = p_i^{AI_{j-1}}(t)[\delta_I(1-m)\eta_{j-1}] + p_i^{RI_{j-1}}(t)[(1-m)\eta_{j-1}] + p_i^{AI_j}(t)[\delta_I(1-m)(1-\eta_j)] + p_i^{RI_j}(t)[(1-m)(1-\eta_j)] \quad (9)$$

$j = 2,3,4\ldots\ldots(n-1)$

$$p_i^{UI_n}(t+1) = p_i^{UI_{n-1}}(t)[r_i(t)(1-m)\eta_{n-1}(1-k)] + p_i^{UI_n}(t)[r_i(t)(1-m)(1-\delta_E)(1-k)] \quad (10)$$

$$p_i^{AI_n}(t+1) = p_i^{UI_{n-1}}(t)[r_i(t)m\eta_{n-1} + r_i(t)(1-m)\eta_{n-1}k + (1-r_i(t))\eta_{n-1}] + p_i^{AI_{n-1}}(t)[\delta_I m\eta_{n-1} + (1-\delta_I)\eta_{n-1}] + p_i^{RI_{n-1}}(t)[m\eta_{n-1}] + p_i^{UI_n}(t)[r_i(t)m(1-\delta_E) + r_i(t)(1-m)(1-\delta_E)k + (1-r_i(t))(1-\delta_E)] + p_i^{AI_n}(t)[\delta_I m(1-\delta_E) + (1-\delta_I)(1-\delta_E)] + p_i^{RI_n}(t)[m(1-\delta_E)] \quad (11)$$

$$p_i^{RI_n}(t+1) = p_i^{AI_{n-1}}(t)[\delta_I(1-m)\eta_{n-1}] + p_i^{RI_{n-1}}(t)[(1-m)\eta_{n-1}] + p_i^{AI_n}(t)[\delta_I(1-m)(1-\delta_E)] + p_i^{RI_n}(t)[(1-m)(1-\delta_E)] \quad (12)$$

$$p_i^{UR}(t+1) = p_i^{UI_n}(t)[r_i(t)(1-m)\delta_E] + p_i^{UR}(t)[r_i(t)(1-m)] \quad (13)$$

$$p_i^{AR}(t+1) = p_i^{UI_n}(t)[r_i(t)m\delta_E + (1-r_i(t))\delta_E] + p_i^{AI_n}(t)[\delta_I m\delta_E + (1-\delta_I)\delta_E] + p_i^{RI_n}(t)[m\delta_E] + p_i^{UR}(t)[(1-r_i(t)) + r_i(t)m] + p_i^{AR}(t)[\delta_I m + (1-\delta_I)] + p_i^{RR}(t)[m] \quad (14)$$

$$p_i^{RR}(t+1) = p_i^{AI_n}(t)[\delta_I(1-m)\delta_E] + p_i^{RI_n}(t)[(1-m)\delta_E] + p_i^{AR}(t)[\delta_I(1-m)] + p_i^{RR}(t)[1-m] \quad (15)$$

$$p_i^{AV}(t+1) = p_i^{US}(t)[r_i(t)mq_i^A(t)l + (1-r_i(t))q_i^A(t)l] + p_i^{AS}(t)[\delta_I mq_i^A(t)l + (1-\delta_I)q_i^A(t)l] + p_i^{RS}(t)[mq_i^A(t)l] + p_i^{AV}(t)[(1-\delta_I) + \delta_I m] + p_i^{RV}(t)[m] \quad (16)$$

$$p_i^{RV}(t+1) = p_i^{AS}(t)[\delta_I(1-m)q_i^A(t)l] + p_i^{RS}(t)[(1-m)q_i^A(t)l] + p_i^{AV}(t)[\delta_I(1-m)] + p_i^{RV}(t)[1-m] \quad (17)$$

where

$$p_i^{US}(t) + p_i^{AS}(t) + p_i^{RS}(t) + \sum_{f=1}^{n} (p_i^{UI_f}(t) + p_i^{AI_f}(t) + p_i^{RI_f}(t)) + p_i^{UR}(t) + p_i^{AR}(t) + p_i^{RR}(t) + p_i^{AV}(t) + p_i^{RV}(t) = 1 \quad (18)$$

Theorem 1 finds epidemic threshold using NGM[22].

4.1 Theorem 1

If infection probability of each node goes to zero, then $\beta < \frac{\delta_E}{\lambda_{max}(G)}$, where $'G'$ is a $N \times N$ matrix given by $g_{ij} = \{p_i^U(r_i(1-m) + \gamma[1 - r_i(1-m)]) + \gamma p_i^A + \gamma p_i^R\}b_{ji}$, where p_i^U, p_i^A and p_i^R are probability of i^{th} node in unaware, aware and removed state.

4.2 Proof

When there is no epidemic $S \approx 1, I = R = V \approx 0$. In steady state it is assumed that $p_i^{US} = p_i^U, p_i^{AS} = p_i^A, p_i^{RS} = p_i^R$. The linearized differential equations corresponding to disease free equilibrium for i^{th} node in k^{th} infected stage are

$$\frac{dI_{i1}}{dt} = -\eta_1 I_{i1} + C_i b_{1i}\beta I_{1n} + C_i b_{2i}\beta I_{2n} + .. + C_i b_{Ni}\beta I_{Nn}$$

$$\frac{dI_{ik}}{dt} = \eta_{k-1} I_{ik-1} - \eta_k I_{ik}, k = 2,3,\ldots n-1 \quad (19)$$

$$\frac{dI_{in}}{dt} = \eta_{n-1} I_{in-1} - \delta_E I_{in}$$

Where, $C_i = p_i^U\{r_i(1-m) + \gamma[1 - r_i(1-m)]\} + \gamma(p_i^A + p_i^R)$.

Let $X = (I_{11}, I_{21}, .. I_{N1}, I_{12}, I_{22}, \ldots . I_{N2}, I_{13}, I_{23}, \ldots . I_{N3} \ldots\ldots\ldots I_{1n}, I_{2n}, \ldots I_{Nn})^T$.

Equation 19 can be written as $\frac{dX}{dt} = (F - V)X$ where

$$F = \begin{pmatrix} 0_{N\times N} & 0_{N\times N} & 0_{N\times N} & \cdots & H_{N\times N} \\ 0_{N\times N} & 0_{N\times N} & 0_{N\times N} & \cdots & 0_{N\times N} \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ 0_{N\times N} & 0_{N\times N} & 0_{N\times N} & \cdots & 0_{N\times N} \end{pmatrix}$$

$$V = \begin{pmatrix} D(\eta_1)_{N\times N} & 0_{N\times N} & \cdots & 0_{N\times N} \\ D(-\eta_1)_{N\times N} & D(\eta_2)_{N\times N} & \cdots & 0_{N\times N} \\ 0_{N\times N} & D(-\eta_2)_{N\times N} & \cdots & 0_{N\times N} \\ \vdots & \vdots & \ddots & \vdots \\ 0_{N\times N} & 0_{N\times N} & \cdots & D(\delta_E)_{N\times N} \end{pmatrix}$$

$h_{ij} = C_i b_{ji}\beta$. $R_0 = \Lambda_{max}(FV^{-1}) = \frac{\lambda_{max}(H)}{\delta_E}$. The epidemic threshold is given by

$$R_0 < 1 \Rightarrow \frac{\lambda_{max}(H)}{\delta_E} < 1 \Rightarrow \beta < \frac{\delta_E}{\lambda_{max}(G)}, g_{ij} = C_i b_{ji}.$$

Next, the global asymptotic stability of disease free equilibrium(DFE)is proved.

4.3 Theorem 2

If $R_0 < 1$, the DFE $E_0 = (1,1..1,0,0..0)$ is globally asymptotically stable on $\Gamma = \{S_1, S_2 \ldots . S_N, I_{1.}, I_{2.} \ldots I_{N.})/S_i + I_{i.} \leq 1, 0 \leq S_i, I_{i.} \leq 1, i = 1,2..N\}$.

4.4 Proof

The Lyapunov function L:$\Gamma \rightarrow R^N$ is defined as $L(S(t), I(t)) = I(t)$, where $I_{i.}(t) = \sum_{k=1}^{n} I_{ik}$.

$\frac{dL_i}{dt} = \frac{dI_{i.}}{dt} = C_i b_{1i} \beta I_{1n} + C_i b_{2i} \beta I_{2n} + .. + C_i b_{Ni} \beta I_{Nn} - \delta_E I_{in}$ $\frac{dL}{dt} = HI_n^T - \delta_E \Delta I_n^T = (H - \delta_E \Delta) I_n^T$, where Δ is $N \times N$ identity matrix, $I_n = (I_{1n}, I_{2n}, .. I_{Nn})$.

Hence, $\frac{dL}{dt} < 0$ only if $R_0 < 1$. When $R_0 < 1$, all the solutions in Γ are globally asymptotically stable to E_0.

If $R_0 > 1$ all solutions in Γ will move away from E_0, which makes the system unstable. Hence, the infection will spread into the population.

5 Results and discussion

For numerical simulation, a scale-free network with 1000 nodes is used. Both epidemic and information layer have same number of nodes, but the information layer has additionally (Average degree d $\times$ N) edges to represent the contacts via social media. The nodes in the multiplex network follows $UARA - SVI_1I_2 \ldots I_nR$ process. Initially, the network has two arbitrary n^{th} stage infected nodes(I_n), in that, one is aware about the disease and the other is not. These two infected node spread disease to their neighbours with rate β and the aware node spread awareness with rate λ.

The comparison between Monte Carlo simulation and MMCA analysis is given in Fig 6. For MC simulation 100 runs are taken for fixed parameter values and for different networks with various initial values.

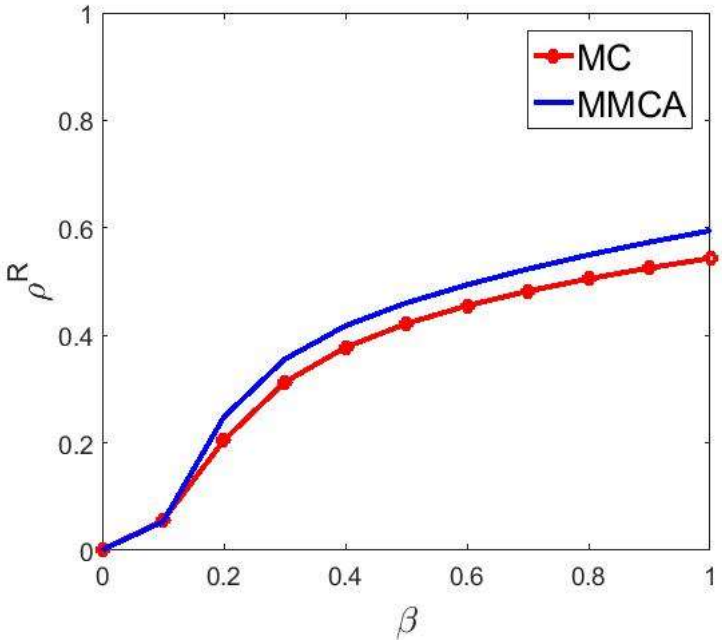


Figure 6: Comparison of MC simulation and MMCA analysis for fraction of recovered. The parameter values are λ=0.7; δ_I=0.6; β=0.6; η_i=0.8; n=5; k=0.3; m=0.3; γ=0.5; $\delta_E = 0.6$; l=0.1.

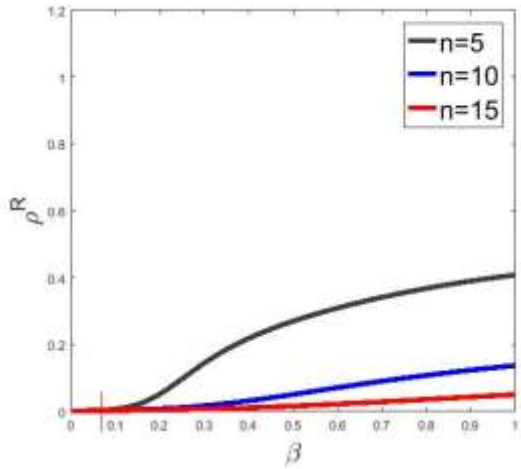
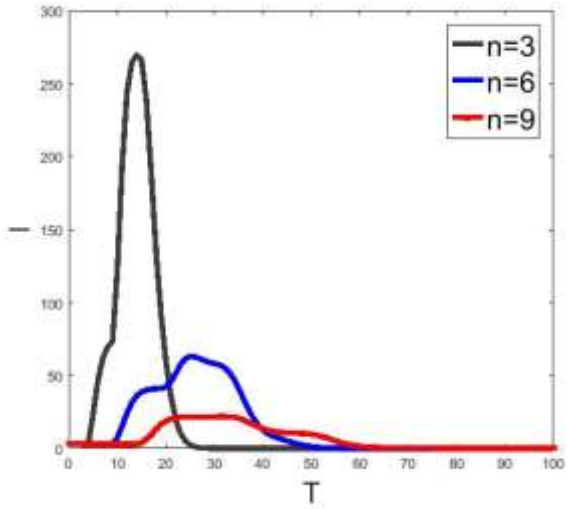


Figure 7 (a):[ρ^R vs β for $n = 5,10,15$]



[I vs time for $n = 3,6,9$]

Figure 7(b): Effect of various number n of infected stages I_n.

Fig7(a) shows how the fraction of recovered varies for various number of infected stages n. An increase in number of infected stages increases the vaccination because time taken for a newly infected node to spread the infection is increased when n is increased. During that time, due to the awareness, susceptible node goes for vaccination. Hence, number of recovered decreases as n increases. Fig 7(b) shows the epidemic at various time for different number of infected stages n. When n is small, it infects more number of people and saturates earlier. For smaller n epidemic spreads faster than vaccination.

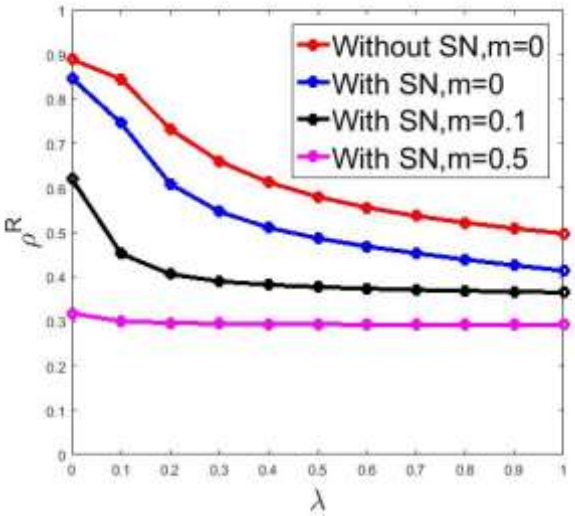


Figure 8 (a): [ρ^R vs λ]

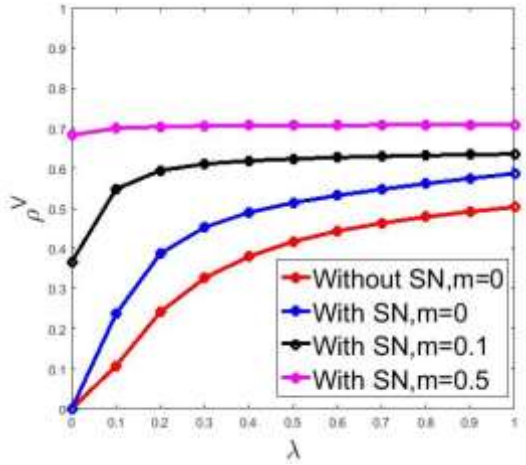


Figure 8(b): [ρ^V vs λ]

Figure 8: Effect of mass media and social networks. Analysis without SN and m=0, with SN and m=0,0.1,0.5

Fig 8(a) shows fraction of recovered for model with and without social network and mass media. Absence of mass media and social network reduces the awareness spread. Hence, more people got infection. When m=0 and m=0.1, social media has some impact on awareness spreading. When m=0.5 the mass media dominates social networks. Hence, ρ^R decreases with respect to λ for m=0 and m=0.1, and it does not vary with respect to λ for m=0.5.Fig 8(b) shows fraction of vaccinated for the same. Vaccination increases as awareness increases.

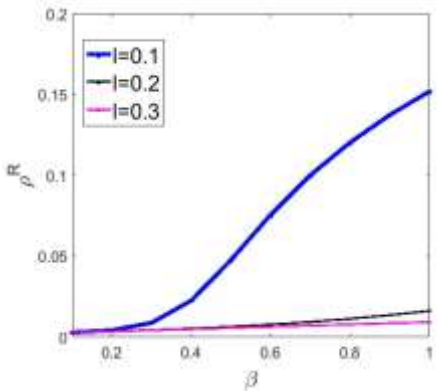


Figure 9(a) : [ρ^R vs β]

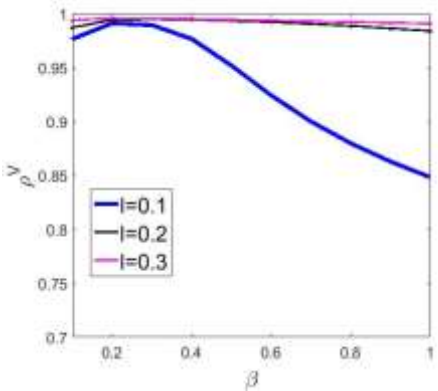


Figure 9(b) : [ρ^V vs β]

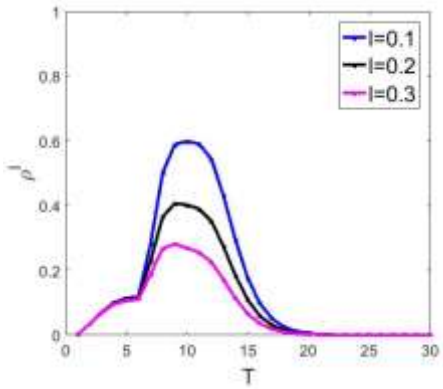


Figure 9(c) : [ρ^I vs T]

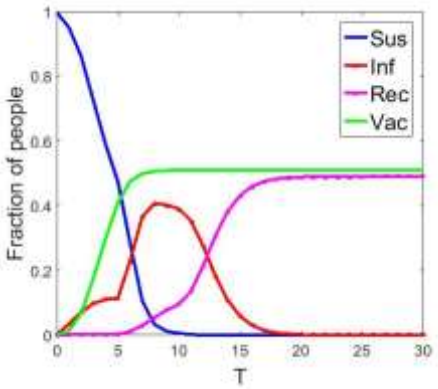


Figure 9(d) :[$\rho^S, \rho^I, \rho^R, \rho^V$ vs T for $l = 0.2$]

Figure 9: Effect of vaccination for $l = 0.1, l = 0.2, l = 0.3.$

Fig9(a)and (b) shows fraction of recovered and vaccinated for $l = 0.1, l = 0.2, l = 0.3.$ When $l = 0.1$ the infection spread faster than vaccination. When $l = 0.2,0.3$ the vaccination spreads faster than infection and controls the epidemic. Fig9(c) shows fraction of infected vs time for $l = 0.1.0.2,0.3.$ Fig 9(d) shows fraction of susceptible, infected, recovered and vaccinated vs time when $l = 0.2.$

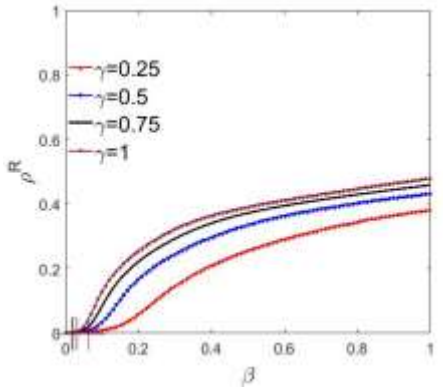


Figure 10(a) : [ρ^R vs β]

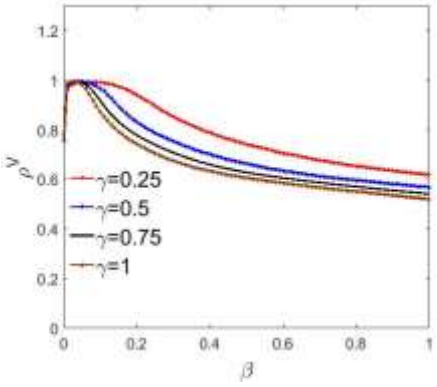


Figure 10(b) : [ρ^V vs β]

Figure 10: Effect of various values of infection attenuation factor $\gamma = 0.25,0.5,0.75,1.$

Fig10(a) and (b) shows the fraction of recovered and vaccinated for various values of γ. $1 - \gamma$ denotes the effect of preventive measures. When $\gamma < 1$ the infection rate is reduced, due to the preventing measures taken by aware people. $\gamma = 1$ denotes epidemic without awareness. Increase in effect of preventive measures decreases the epidemic and increases the epidemic threshold and vaccination.

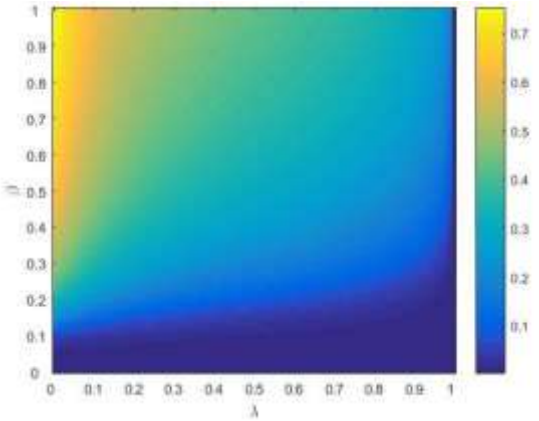


Figure 11(a) : [ρ^R]

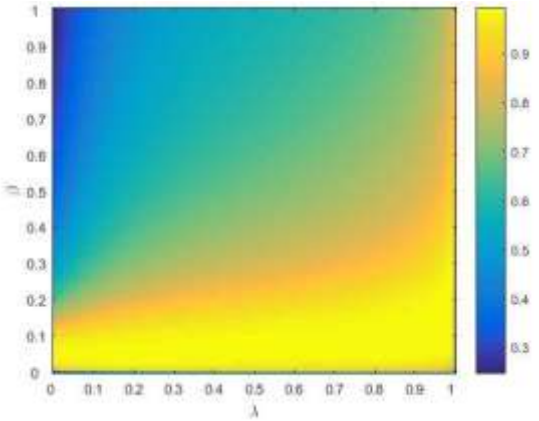


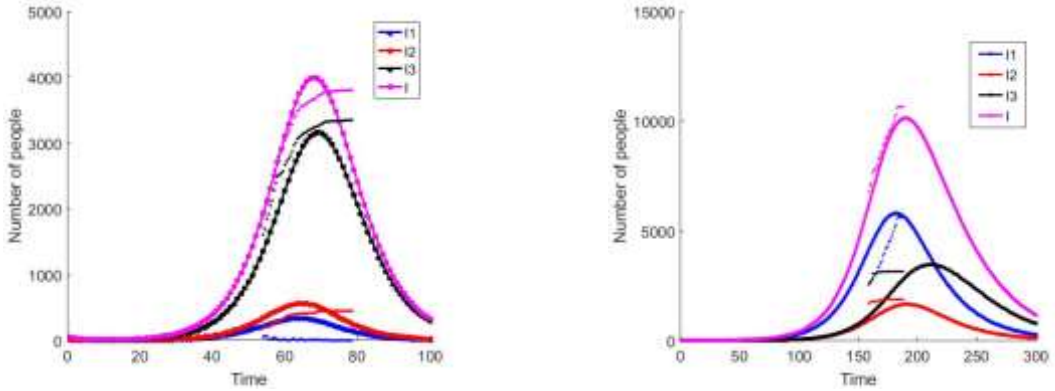
Figure 11(b) : [ρ^V]

Figure 11: The MMCA analysis for various β and λ.

Fig11 shows the fraction of vaccinated and recovered for various β and λ. People getting awareness increases as λ increases, hence people going for vaccination increases. Because of the preventing measures due to awareness fraction of people recovered decreases as λ increases.

6 Data fitting of EVD

Ebola virus causes more number of deaths in West Africa during 2014 to 2015. Using SPSS, the parameter values which are fitted to the outbreak of this disease in Guinea, Liberia from November 2014 to November 2015 (first, second, third and fourth week of each month), from World Health Organization(WHO), are estimated [19]. Model with n=3 is taken for estimating theoretical data. I_1, I_2, I_3 respectively represents Suspected,Probable,Confirmed people. The awareness reduction factor and vaccination parameter are taken as $\gamma = 0.5$ and l=0 respectively; The rate at which I_1 goes to $I_2(\eta_1)$ and I_2 goes to $I_3(\eta_2)$ and infection rate(β) are 1,0.3,0.012 for Guinea, and 0.03,0.05,0.02 for Liberia. In Guinea Suspected is lesser than probable and the chance for suspected goes to probable is 1 hence the disease did not spread into more people. But in Liberia suspected is larger than probable and the chance for suspected goes to probable is less than 1, hence it affects more number people. The correct identification of suspected reduces the infection spread. In fig 12 the theoretical values (line) and WHO data values (dots)of these countries are shown.



[Guinea] [Liberia]

Figure 12: Comparison of theoretical values with real data (I_1 Suspected, I_2 Probable, I_3 Confirmed and I total infected).

7 Conclusion

In this paper,a two-layered networking $UARA - SVI_1I_2 \ldots I_nR$ model with mass media is used to mimic the disease spreading and disease-related information diffusion among the same population. By utilizing the MMCA method, the probability trees are generated to characterize the possible state transitions between the information and disease spreading. NGM is used to derived the epidemic threshold. Global asymptotic stability of DFE is proved. Numerical simulation shows that increase in n reduces the epidemic spread, social network and mass media plays significant roll in spreading awareness, hence reduce the epidemic spread. Vaccination provides significant control over the epidemic spread. The model with 3 stage infection is well suitable for EVD spread in Guinea and Liberia. The correct identification of suspected reduces the infection spread.

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