



## An SIRS Model of Virus Epidemic on a Computer Network

Olubadeji Bukola<sup>1\*</sup>, A. O. Adetunmbi<sup>1</sup> and T. T. Yusuf<sup>2</sup>

<sup>1</sup>Department of Computer Science, Federal University of Technology, Akure, Ondo State, Nigeria.

<sup>2</sup>Department of Mathematical Science, Federal University of Technology, Akure, Ondo State, Nigeria.

### Authors' contributions

This work was carried out in collaboration between all authors. Author OB designed the study, wrote the protocol and supervised the work. Authors OB, AOA and TTY carried out all laboratories work and performed the statistical analysis. Author TTY managed the analyses of the study. Author OB wrote the first draft of the manuscript. Author OB managed the literature searches and edited the manuscript. All authors read and approved the final manuscript.

### Article Information

DOI: 10.9734/BJMCS/2016/24816

*Editor(s):*

(1) Junjie Chen, Department of Electrical Engineering, University of Texas at Arlington, USA.

*Reviewers:*

(1) Rachana Pathak, University of Lucknow, Lucknow, Uttar Pradesh, India.

(2) R. Roy, University of Texas-Pan American, Edinburg, USA.

(3) Olukayode Adebimpe, Landmark University, Omu-Aran, Nigeria.

Complete Peer review History: <http://sciencedomain.org/review-history/15413>

Received: 1<sup>st</sup> February 2016

Accepted: 25<sup>th</sup> April 2016

Published: 17<sup>th</sup> July 2016

Original Research Article

## Abstract

Recently, intense research has been on how to reduce the spread of virus on a network of computer systems, which involves the mathematical modelling of the spread of virus based on mathematical epidemiological approach. This is necessary because a threshold cannot be discerned from the data generated on the network, rather it requires a mathematical model to analyze and simulate the virus dynamics on the network. It also enables the calculation of the basic reproductive number ( $R_0$ ) which is an important threshold for determining whether the network is at risk or not. In this paper, we adopt the susceptible- infected-recovered-susceptible (SIRS) model to depict the spread of virus on the network. We qualitatively analyze the model and establish that the virus-free state is locally asymptotically stable provided the basic reproduction number is less than unity. We solved the model numerically and simulate the solution for different scenarios on the network. The findings from our simulations are discussed.

*Keywords:* Mathematical model; basic reproductive number; vaccination; equilibrium solution; local asymptotic stability.

\*Corresponding author: E-mail: [bolubadeji@futa.edu.ng](mailto:bolubadeji@futa.edu.ng);

## 1 Introduction

Computer viruses have evolved in time, adopting different strategies that take advantage of different weak points of computers and software. These viruses are independent of the independent of the platform's hardware and infect data files such as documents produced with spreadsheets or word processors. The spreading of computer viruses has been studied for long years, in close analogy with the models developed for study of the transmission of biological disease [1]. In the biological framework, the key point is the description of the epidemic process in terms of individuals and their interactions. In this simplified formalism, individuals can only exist in a discrete set of states, such as susceptible (or healthy), infected (and ready to spread the disease), immune, dead (or removed). The interactions among individuals are schematized in structure of the contacts along which the epidemics can propagate. This type of system can be described as a network or graph [2] in which the nodes represent the individuals and the links are the connections along which the epidemics propagates. Epidemics model are heavily affected by the connectivity patterns characterizing the population in which the infective agent spreads, so as to illustrate the features of epidemic spreading on the computer networks, we used the SIRS model. It is important to stress, however that, the analysis on the computer networks of different models such as that of SIRS model confirm the presented epidemiological model.

Several authors have suggested many nonlinear incidence rates to model the disease transmission process [3,4,5].

Moreover, in the SIRS model, the population of host is divided into three classes, susceptible computers, infected computers and recovered computers and several computer simulations are performed using different initial conditions. The Susceptible – Infected - Recovered (SIR) model was introduced by Kermack and McKendrick, in 1927 [6]. In the model, they divided the population into three distinct groups of: the Susceptible S, the Infected I, and the Recovered R, where S, I and R represents the number of systems in each of the groups respectively and the total population  $N = S + I + R$ . The Susceptible are those who are not infected and not immune, the Infected are those who are infected and can transmit the disease, and the Recovered are those who are immune, either due to vaccination or recovery with immunity after infection.

## 2 Vaccination

Immunization in the computational realm is the ability to prevent a viral program from executing and replicating further to other hosts. There are many reasons a node might be immune to a virus. For example, a host running Unix is immune to Windows-based viruses, or a node can become immunized against a particular virus if the ways that the virus exploits the underlying host are disabled.

It is our intent to know the ways in which immunization can be achieved. Rather, assuming that immunization techniques exist, our goal is to examine what the effectiveness of immunization on the computer network, because models for infectious diseases lead to a better understanding of how vaccination programs affect the control or eradication of the disease. Several popular articles by [7] used optimal control to study nonlinear SIR epidemic model with a vaccination program. Also, [8] investigated a disease transmission model by considering the impact of a protective vaccine and found the optimal vaccine coverage threshold required for disease control. In [9] considered an SIR epidemic model using vaccination as control. Clearly, it is often not feasible to immunize the entire network. A more realistic approach would be to immunize a subset of the population, and so choosing the appropriate size and membership of that subset becomes an important question. Thus, if disease eradication can be achieved by partially vaccinating some fraction  $p$  of the population, an advantage is gained. The fraction to be immunized must be such that the remaining population,  $(1 - p)N$  where  $N$  depicts the total population, will no longer exceed the threshold level necessary to perpetuate the disease. In the terminology, the reproductive factor  $R_0$  of the infection is to be reduced below 1. The percentage of the population to be vaccinated thus depends strongly on the infectiousness of the disease.

### 3 Mathematical Model Formulation

One of the simplest epidemiological model one can consider is the susceptible-infected-recovered-susceptible (SIRS) model [10]. In SIRS model, individuals can only exist in three discrete states, namely, susceptible, infected and recovered.

At each time step, each susceptible node is infected with probability  $\beta$  if it is connected to one or more infected nodes. At the same time, infected nodes are recovered (cured) and become again susceptible.

The SIRS model take into account the possibility of individuals removal due to purging or crash or acquired immunization which would lead to the so-called susceptible-infected-removed (SIR) model. Using the case of virus spread on the network, there is an arrival of new susceptible systems into the network. For this type of situation births and deaths rate must be included in the model. The following differential equations represent the model which indicates the rate of change of number of systems/individuals in each compartment with respect to time.

$$\frac{dS}{dt} = \Lambda - \beta SI - kS - \mu S + \rho R \tag{3.1}$$

$$\frac{dI}{dt} = \beta SI - \gamma I - kI - \delta I \tag{3.2}$$

$$\frac{dR}{dt} = \gamma I - \rho R - kR + \mu S \tag{3.3}$$

It is important to note that the total number of systems/individuals under consideration at any point in time can be obtained by  $N(t) = S(t) + I(t) + R(t)$ , and its dynamics is given by:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda - dN + \delta I \tag{3.4}$$

Several assumptions were made in the formulation of these equations: First, a system on the network must be considered as having an equal probability as every other system of contracting the disease/virus at a rate  $\beta$ , which is considered the contact or infection rate of the disease/virus. Therefore, an infected individual makes contact and is able to transmit the disease with  $\beta N$  others per unit time and fraction of contacts by an infected with a susceptible is  $S/N$ . The number of new infections in unit time per infective then is  $\beta N(S/N)$ , giving the rate of new infections (or those leaving the susceptible category) as  $\beta N \left(\frac{S}{N}\right) I = \beta SI$  [11].

**Table 1. The description of parameters used in the model**

Parameter	Description	Unit
$\Lambda$	Constant rate of replacement of new system on network	Number / unit time
$\beta$	Rate at which the infection is transmitted on the network	Rate / unit time
$\mu$	Vaccination rate of susceptible system	Rate / unit time
$\gamma$	Troubleshooting success rate	Rate / unit time
$\rho$	Antivirus effectiveness warning rate	Rate / unit time
$\delta$	Purging rate i.e rate at which system's get damaged or crashed due too virus infection	Rate / unit time
$k$	The rate at which system becomes obsolete/crashed and are removed from the network	Rate / unit time

*The unit time is (per day)*

## 4 Model Analysis

### 4.1 Model well – posedness

Since the model monitors the number of computer system on the network, all the variables and parameters of the model are non-negative. Thus, we consider the mathematically-feasible region

$$\Omega = \{[S, I, R] \in \mathbb{R}_+^3 : N \leq \frac{A}{k}\}$$

The rate of change of the total number of system on the network per unit time is given by

$$\frac{dN}{dt} = \lambda - kN - \delta I \leq \lambda - kN \quad (4.1)$$

Theorem 1: Every solution of the model equations, (3.1) to (3.3) with initial conditions in  $\Omega$ , is a member of  $\Omega$  (i.e the region  $\Omega$  is positive invariant and attracting).

Proof:

Based on eqn (4.1), we have

$$\begin{aligned} \frac{dN}{dt} &\leq \lambda - dN \\ \text{then } \frac{dN}{dt} + dN &\leq \lambda \end{aligned} \quad (4.2)$$

This gives integrating factor (I.F) =  $e^{\int k dt} = e^{kt}$  multiplying the given equation (4.1) by  $e^{kt}$ , we have

$$e^{kt} \frac{dN}{dt} + e^{kt} dN \leq \lambda e^{kt} \quad (4.3)$$

However, eqn (4.3) is equivalent to

$$\frac{d}{dt} (Ne^{kt}) \leq \lambda e^{kt} \quad (4.4)$$

Integrating the proceeding equation with respect to time gives:

$$\begin{aligned} \int_0^t \frac{d}{dt} (Ne^{kt}) dt &\leq \int_0^t \lambda e^{kt} dt \\ \text{so, } N(t)e^{kt} - N(0) &\leq \left[ \frac{\lambda}{k} e^{kt} \right]_0^t \\ \therefore N(t)e^{kt} - N(0) &\leq \left[ \frac{\lambda}{k} e^{kt} \right]_0^t \leq \left( \frac{\lambda}{k} e^{kt} - \frac{\lambda}{k} \right) \\ \Rightarrow N(t)e^{kt} &\leq N(0) + \frac{\lambda}{k} (e^{kt} - 1) \\ \therefore N(t) &\leq N(0)e^{-kt} + \frac{\lambda}{k} (1 - e^{-kt}) \end{aligned} \quad (4.5)$$

In particular,  $N(t) \leq \frac{A}{k}$  if  $N(0) \leq \frac{A}{k}$ .

Therefore, every solution of the model with initial conditions in  $\Omega$  remains there for all  $t \geq 0$ . so the region  $\Omega$  is positive invariant and attracting. Consequently, it is sufficient to consider the dynamics of the model in  $\Omega$ . As a result, the model is mathematically and epidemiologically well posed.

## 4.2 Model equilibrium solutions

At equilibrium point

$$\frac{dS}{dt} = 0, \frac{dI}{dt} = 0, \frac{dR}{dt} = 0$$

Thus, we have

$$A - \beta SI - kS - \mu S + \rho R = 0 \tag{4.6}$$

$$\beta SI - \gamma I - kI - \delta I = 0 \tag{4.7}$$

$$\gamma I - \rho R - kR + \mu S = 0 \tag{4.8}$$

From eqns 4.6, 4.7 and 4.8 simultaneously for S(t), I(t) and R(t), we obtained the Virus – free equilibrium

$$E_1 = (S^*, I^*, R^*)$$

Where

$$E_1 = \left[ S^* = \frac{A(\rho+k)}{d(\mu+\rho+k)}, I^* = 0, R^* = \frac{A\mu}{k(\mu+\rho+k)} \right]$$

and the virus endemic equilibrium  $E_2$  is

$$E_2 = \left[ S^{**} = \frac{\gamma + k + \delta}{\beta}, I^{**} = \frac{k(\rho + k + \mu k)(\gamma + k + \delta)(R_0 - 1)}{\beta(\rho k + \rho \delta + \gamma k + k^2 + k \delta)}, \right. \\ \left. R^{**} = \frac{\beta \gamma A + \mu(k + \delta)(\gamma + k + \delta) - \gamma k(\gamma + k + \delta)}{\beta(\rho k + \rho \delta + \gamma k + k^2 + k \delta)} \right]$$

## 4.3 Local stability of the virus – free equilibrium

We linearize the system of equations given, using the Jacobian matrix approach to obtain:

Evaluating the Jacobian matrix at the virus – free equilibrium  $E_1$  gives

$$(JE_1) = \begin{bmatrix} -k - \mu & -\beta \left[ \frac{A(\rho + k)}{k(\mu + \rho + k)} \right] & \rho \\ 0 & \beta \left[ \frac{A(\rho + k)}{k(\mu + \rho + k)} \right] - \gamma - k - \delta & 0 \\ \mu & \gamma & -\rho - k \end{bmatrix}$$

We defined the characteristic polynomial equation for the  $J(E_1)$  solve for the eigen valves, to get:

$$\lambda_1 = -k, \quad \lambda_2 = -\mu - \rho - k, \quad \lambda_3 = \frac{A\beta(\rho + k) - k(\mu + \rho + k)(\gamma + k + \delta)}{k(\mu + \rho + k)}$$

As we can see,  $\lambda_1 < 0$ ,  $\lambda_2 < 0$ , So, for the virus – free – equilibrium to be locally asymptotically stable,  $\lambda_3$  must be less than zero.

This is so if 
$$\Lambda\beta(\rho + k) - k(\mu + \rho + k)(\gamma + k + \delta) < 0$$

which implies that

$$\Lambda\beta(\rho + k) < k(\mu + \rho + k)(\gamma + k + \delta)$$

and this is equivalent to

$$R_0 = \frac{\Lambda\beta(\rho + k)}{k(\mu + \rho + k)(\gamma + k + \delta)} < 1$$

Where  $R_0$  is the basic reproduction number.

It is imperative to note that the *Basic Reproductive Number*, denoted as  $R_0$ , is an important threshold in modelling of infectious diseases since it tells us if a population is at risk from a disease or not. Thus, whenever  $R_0 < 1$  the new cases (i.e. incidence) of the disease will be on the decrease and the disease will eventually be eliminated.

Based on foregoing, the Basic Reproduction number ( $R_0$ ) for our model is less than unity i.e

$$R_0 = \frac{\Lambda\beta(\rho+k)}{k(\mu+\rho+k)(\gamma+k+\delta)} < 1$$

Then,  $I(t)$  decreases monotonically to zero as  $t \rightarrow \infty$ . Therefore, the virus – free equilibrium is locally stable. The implication of this result is that we can drive the virus epidemic situation on the network to a virus – free state provided we can put control measures in place on the network that would drive the situations on the network sufficiently close to the virus – free state.

## 5 Numerical Solution and Simulation

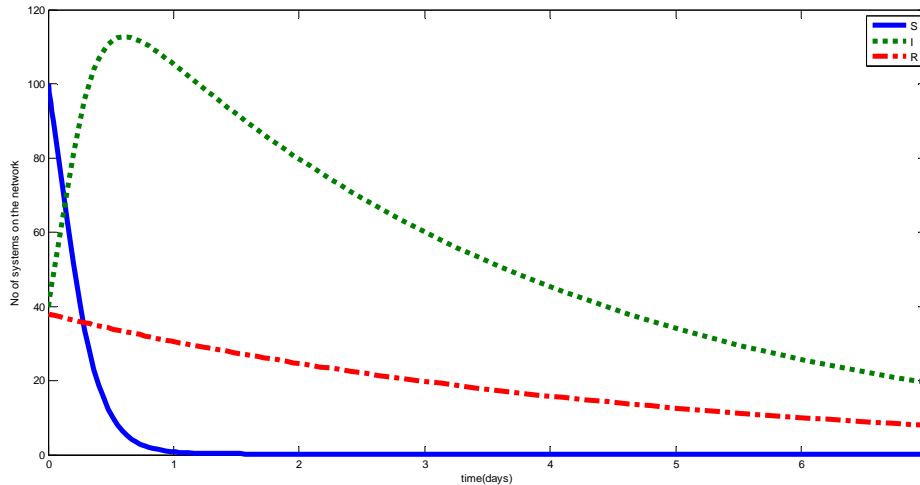
The SIRS model was solved numerically using Runge – Kutta method. We adopted Matlab ode45 program, which is based on an explicit Runge Kutta (4, 5) formula. It is a one-step solver used in solving a system of first – order ordinary differential equation (ODE). So, in computing  $y(t_n)$ , it needs only the solution at the immediately preceding time point,  $y(t_{n-1})$ . In general, ode45 is the best function to apply as a first try for most problems involving systems of first order ODES. Runge kutta of order four is also used in plotting the graphs; it's a powerful and popular method because of its accuracy and stability. Also, its simplicity and stability make it one of the most widely used numerical algorithms for stiff and non-stiff equations, while it converges faster than that of order two or three.

**Table 2. Simulating the model using the following parameters values**

Parameters	$\Lambda$	$\beta$	$k$	$\rho$	$\delta$	$\gamma$	$\mu$
Value for Fig. 1	5	0.50	0.25	$3.3 \times 10^{-2}$	$2.3 \times 10^{-2}$	0.02	0.50
Value for Fig. 2	5	0.25	0.25	$3.3 \times 10^{-2}$	$2.3 \times 10^{-2}$	0.02	1.00
Value for Fig. 3	5	0.125	0.25	$3.3 \times 10^{-2}$	$2.3 \times 10^{-2}$	0.02	1.50

These are the parameters used in plotting the graphs; although, some of it changes due to the fact that they are the major factors determining the situations of the network. This implies that some of these parameters determine whether virus would persist or be eradicated on the network.

In Fig. 1, there are 178 susceptible systems on the network, the infected systems increases on the first day, because the virus propagate fast without the knowledge of the user and it is able to subvert the systems before the introduction of vaccine, while the vaccine introduced is not strong enough to subvert the effects of the virus on the systems on the network.



**Fig. 1. Solutions of SIRS model with  $S(0) = 100$ ,  $I(0) = 40$ ,  $R(0) = 38$**

First, an initial worm infects one machine (computer system) in the network. For the next few days (or hours), the worm propagates freely in the network without being noticed by most users. After some time, users realise that there is an outbreak, and take appropriate action, by introducing a virus signature to computers in the network which is in the form of an antivirus but in these case the antivirus used was not strong enough to subdue the effect of the virus that had already infected some of the systems on the networks. The challenge is that the virus spread so fast that recovering the systems is almost not possible. Thus, the infected systems need to be purged from the network instead of trying to manage it. This may be because the virus has interfered with the vaccine thereby preventing detection, and the virus effect can be severe such that which the virus could corrupt the virus database files thereby leading to a misleading effect on vaccine behaviour. Moreover, if care is not taking, the vaccine can damage the system it intends to protect (that is it can cause autoimmunity).

These kinds of challenges can be overcome by having multiple simultaneous vaccinated systems with different signature files which is referred to as vaccine redundancy. This is based on Biodiversity concept which could be applied to information technology (IT) environment. However, the IT environment should avoid the danger of software monoculture by using different software not the same at all time. Also, multiple different operating systems should be on the key and clients servers to keep the data safe, if virus attack one operating system the others will still be safe.

This scenario of an increasing numbers of infected systems is called an epidemic. This is often the case when the basic reproduction number  $R_0$ , of virus on infected system is greater than unity ( $R_0 > 1$ ). Thus, leading to continuous increase in the number of infected system on the network until it reaches its maximum, after wish the number of infected systems start to decrease. This graph depicts a scenario which could lead into an endemic situation in the network where  $R_0 > 1$ .

Since,

$$R_0 \Rightarrow \frac{\beta A(\rho + k)}{k(\rho + k + \mu)(\gamma + k + \delta)} > 1$$

Using specified parameters values to simulate the endemic situation on the network,

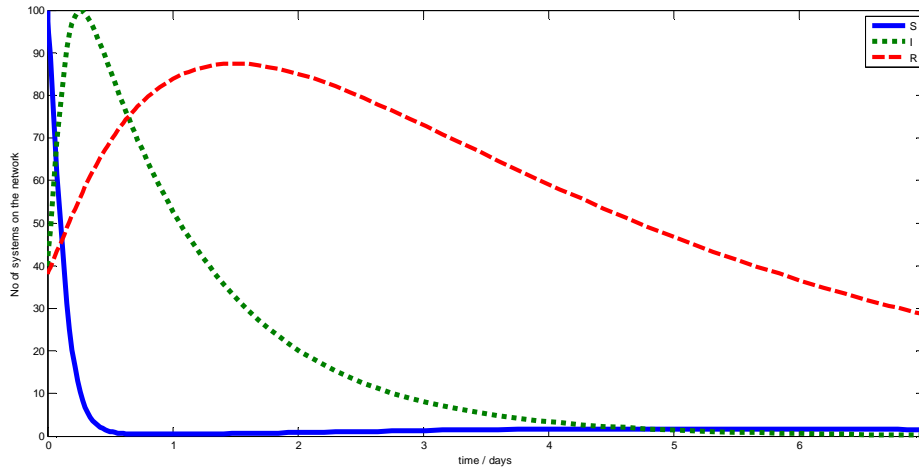
$$\beta = 5, \lambda = 0.5, \rho = 3.3 * 10^{-2}, k = 0.25, \mu = 0.50, \gamma = 0.02, \delta = 2.3 * 10^{-2}$$

The basic reproduction number is as computed below:

$$R_0 = \frac{0.5 * 5(3.3 * 10^{-2} + 0.25)}{0.25(3.3 * 10^{-2} + 0.25 + 0.50)(0.02 + 0.25 + 2.3 * 10^{-2})}$$

$$R_0 = \frac{1.45}{0.135} = 10.76$$

We can infer that the situation of the systems on the network is endemic and it follows that an infection can invade the entire network of computer systems wrecking havoc in majority of the system on the network.



**Fig. 2. Solutions of SIRS model with S(0) = 100, I(0) = 40, R(0) = 38**

In Fig. 2 above, there 178 susceptible systems on the network, the number of infected systems rate increases sharply. The virus attacks on the network nearly subdue all the system before the introduction of vaccine at mid-day. Immediately a strong vaccine was introduced the infection rate dropped and the systems on the network recovered fully from the virus attack. Our suspicion in this situation is that, the worm propagates freely in the network without being noticed by most users. After some time, the worm is detected on some machines (by scanning the systems to know if there are viruses) and immediate action is taken to prevent further spread and to cure infected computers. A worm signature is extracted and included at a specified rate in the antivirus (AV) software of most machines in the network. Machines that were not infected then become automatically immune to the worm, and previously infected machines are being detected at a rate which is depending on how often the AV update is made. These machines are then isolated, cured and immunized against further infection.

Similarly, with

$$R_0 \Rightarrow \frac{\beta \lambda (\rho + k)}{k(\rho + k + \mu)(\gamma + k + \delta)} < 1$$



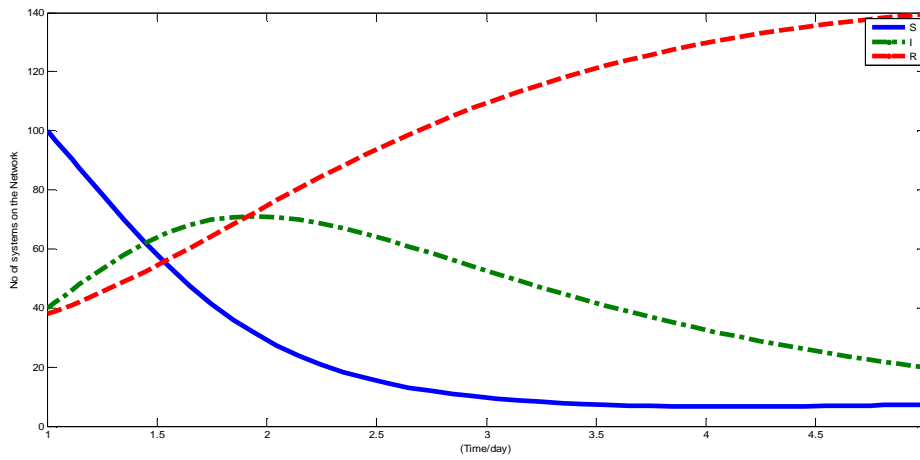
and the parameters values used are

$$\beta = 0.25, \lambda = 5, \rho = 3.3 * 10^{-2}, k = 0.25, \mu = 1.0, \gamma = 0.75, \delta = 2.3 * 10^{-2}$$

$$\text{We have } R_0 = \frac{0.25 * 5 (3.3 * 10^{-2} + 0.25)}{0.25 (3.3 * 10^{-2} + 0.25 + 1.0) (0.02 + 0.25 + 2.3 * 10^{-2})} = 0.52$$

The situation of the systems on this network is that of virus – free equilibrium, it follows that there is an outbreak of infection, but with the introduction of strong vaccine (an anti-virus) all the systems were recovered, the systems on the network is total free of virus, the basic reproduction number is now  $R_0 < 1$ .

In this scenario,  $R_0 < 1$  when compared with Fig.1 where  $R_0 > 1$ . The observed difference in the two graphs is due to the impact of the vaccination success rate ( $\mu$ ). In the latter case, the vaccination is strong enough to ensure that initially infected systems are recovered while blocking new infection.



**Fig. 3. Solutions of SIRS model with  $S(0) = 100, I(0) = 40, R(0) = 38$**

The graph in Fig. 3 depicts a situation that would eventually result in virus – free network situation. This is so because the number of infected system on the network will continue to decrease, until the virus is eventually eliminated on the network, since  $R_0 \ll 1$ .

$$R_0 \Rightarrow \frac{\beta \lambda (\rho + k)}{k (\rho + k + \mu) (\gamma + k + \delta)} < 1$$

The parameters values used in this simulation are:

$$\beta = 0.125, \lambda = 5, \rho = 3.3 * 10^{-2}, k = 0.25, \mu = 2.0, \gamma = 0.75, \delta = 2.3 * 10^{-2}$$

Therefore,

$$R_0 = \frac{0.125 * 5 (3.3 * 10^{-2} + 0.25)}{0.25 (3.3 * 10^{-2} + 0.25 + 2.0) (0.02 + 0.25 + 2.3 * 10^{-2})} = 0.16$$

The situation of the systems on this network is that of virus – free equilibrium. It follows that if there is an outbreak of infection, through the introduction of strong vaccine (an anti-virus), all the infected systems will be recovered. The systems on the network will eventually be free of virus, whenever the basic reproduction number is  $R_0 \ll 1$ .

Comparing the three graphs, Fig. 1 is totally of that of an endemic equilibrium because the virus spread freely at first before the introduction of vaccine, the vaccine has little or no effect on the virus (i.e. the antivirus is too weak to stop the spread of the virus) thereby subverting the systems on the network. However, Figs. 2 and 3 is of a virus – free equilibrium in which the vaccine was able to control the spread of the virus and it imputed the signature of the virus on the network (antivirus) in case the systems fall back into susceptible state.

## 6 The Model Simulation with Varying Parameters

Below are the graphs of SIRS model which monitor the dynamics of systems on the network based on changes on some of the model parameters.

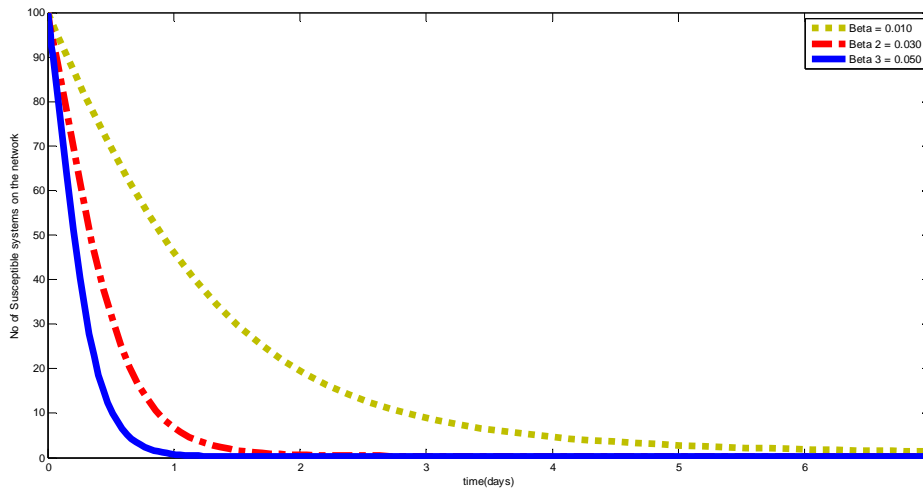


Fig. 4. Effects of changes in infection rate ( $\beta$ ) on the susceptible systems population

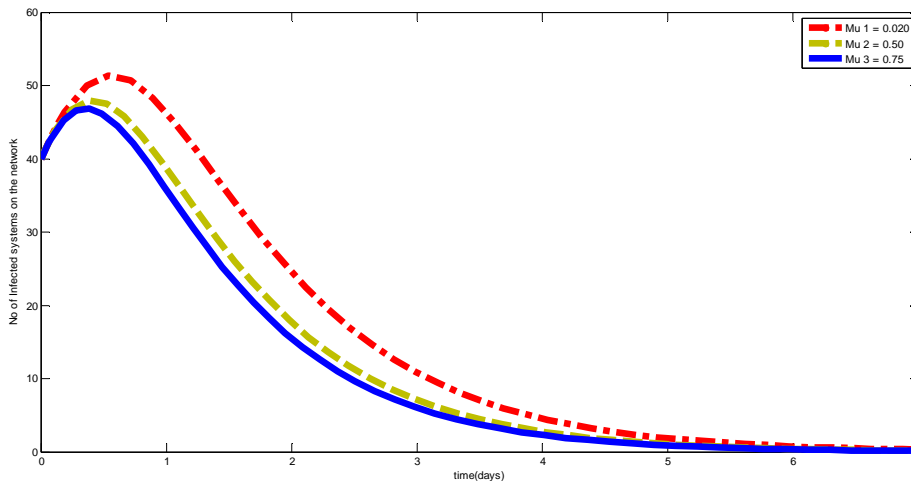
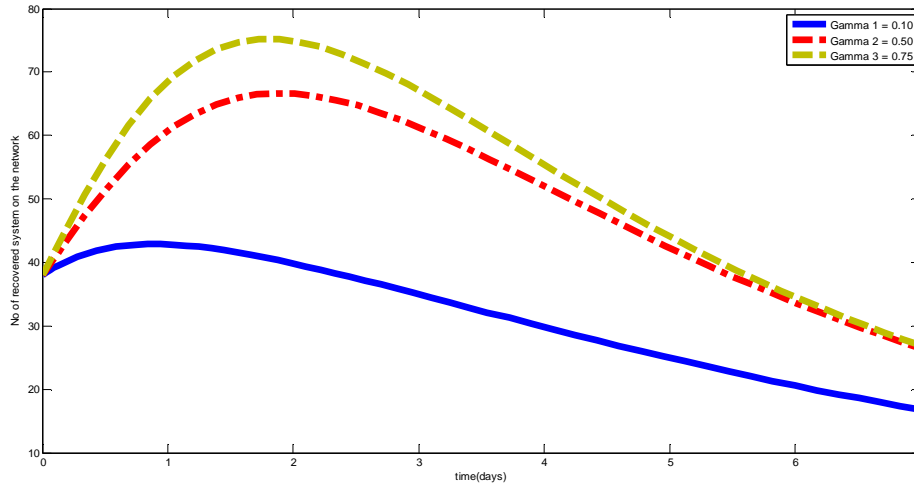


Fig. 5. Effects of changes in vaccine rate ( $\mu$ ) on the infected system population

The graph in Fig. 4 shows the sensitivity of the susceptible systems in the network to changes in the infection rate ( $\beta$ ). As the rate of  $\beta$  increases, the virus was able to infects a larger population of systems on the network before the infected number of systems start decreasing. This decrease in the number of infected systems due to the introduction of strong vaccine  $\mu$  which helps in subverting the actions of the virus on the systems in the computer network.

The graph in Fig. 5 shows the effects of  $\mu$  on the spread of the virus on the network. The graph shows the potency of the vaccine based on the changes in the parameter values, from 0.02% to 0.75%, this means that there is need to equip the systems on the network with strong antivirus against virus attack.



**Fig. 6. The troubleshooting success rate ( $\gamma$ ) on the infected systems population**

The graph in above Fig. 6 shows the troubleshooting success rate  $\gamma$  on the spread of virus on the systems on the network. The success rate  $\gamma$  shows that there are chances for the infected systems on the network to fully recover if they are properly managed.

## 7 Conclusion

In this paper, we formulated an SIRS model to depict the dynamics of virus spread on a network of computer systems. The model virus-free equilibrium and virus-endemic equilibrium were obtained. Thereafter, we derive the model basic reproductive number ( $R_0$ ) and showed that the virus-free equilibrium is locally asymptotically stable if  $R_0 < 1$ . We solved the model equations numerically using Matlab ode45 solver which is based on Runge-Kunta forth-order scheme. The simulations of the model solutions confirm that the situation on the network tends to the virus-free state whenever  $R_0 < 1$ . Also, the simulations show that with strong and effective vaccine on each of the computer system on the network coupled with high trouble shooting success rate for infected systems, a virus-free network is attainable and sustainable.

## Competing Interests

Authors have declared that no competing interests exist.

## References

- [1] Pastor-Satorras, Vespignani A. Epidemics and immunization in scale-free networks. In handbook of graphs and networks. From the Genome to the internet. S. Bornholdt S, Schuster H. G. ed.s. Wiley-VCH, Berlin. 2002;113-132.
- [2] Moreno Y, Pastor-Satorras R, Vespignani A. Epidemic outbreaks in complex heterogeneous networks. Eur. Phys. J. b. 2002;26:521-529.
- [3] Kaddar A. Stability analysis in a delayed SIR epidemic model with a saturated incidence rate. Nonlin. Ana. Model. And Con. 2010;15:299–306.
- [4] Gumel AB, Moghadas SM. A qualitative study of vaccination model with nonlinear incidence. Appl. Math. Comput. 2003;143:409-419.
- [5] Wang J, Xue Y. Bifurcation analysis of a stage structured epidemic model with a nonlinear incidence. Int. J. Inf. Sys. Sci. 2011;7:61–72.
- [6] Edelstein-Keshet L. Mathematical models in biology. Proc. Roy. Soc. London A. 2005;115:700-721.
- [7] Kar TK, Batabyal A. Stability analysis and optimal control of an SIR epidemic model with vaccination. BioSystems. 2011;104:127-135.
- [8] Hattaf K, Lashari AA, Louartassi Y, Yousfi N. A delayed SIR epidemic model with general incidence rate. Electronic Journal of Qualitative Theory of Differential Equations. 2013;3:1-9.
- [9] Zaman G, Kang YH, Jung IH. Stability analysis and optimal vaccination of an sir epidemic model. BioSystems. 2008;93:240–249.
- [10] Diekmann O, Heesterbeek JAP. Mathematical epidemiology of infectious disease: Model building, analysis and interpretation. New York: John Wiley & Sons; 2000.
- [11] Brauer F, Castillo-Chavez C. Mathematical models in population biology and epidemiology. New York: Springer; 2001.

---

© 2016 Bukola et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

**Peer-review history:**

The peer review history for this paper can be accessed here (Please copy paste the total link in your browser address bar)

<http://sciencedomain.org/review-history/15413>