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# Analysis of a Mathematical Model for the Dynamics of Smartphone Virus Propagation: a guide from epidemiological model

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# Abstract

Virus attack is a serious threat in smartphone network environment and is still multiplying as time continues to pass. There are many virus applications in smartphone and users may not be aware of most of these virus intrusions. Thus, controlling these virus attacks in smartphone become necessary, hence we proposed an SVEIQRS model for the control of the virus attack in smartphones through vaccination and quarantine of infected smartphones prior to treatment. The model has a globally asymptotically stable virus free equilibrium when  $R_0 < 1$  and a unique endemic equilibrium when  $R_0 > 1$ . The numerical analysis of the different properties was conducted in MATLAB and the result showed that in other to suppress the propagation of smartphone virus attack in the network, vaccination rate should be increased.

**Keywords:** Smartphone virus, Susceptible Smartphone, epidemic model, mobile device, vaccination, Quarantine.

## Introduction

Smartphone is mobile device with integrated features of cellular and computing capabilities. It uses mobile operating systems with touch screen keyboard and can access the internet [1]. Thousands of applications (apps) can be installed on smartphone platforms which users can conveniently use to transfer funds, transact businesses, conduct training, communicate verbally especially with conference call, etc. The proliferation of mobile application has enhanced transactions across the globe [2]. Developers had eased the installation of these applications, thereby raising the security concerns [3]. Viveros [4] said that the enhancement of computational and communication capabilities of smartphones attracts viruses to these smartphones. Virus are malicious or destructive codes that lack the capability of self-reproduction and self-propagation in a network [5].

Most of the mobile environment for malicious codes are bundled in application software's and have a high risk to learn or steal personal information data [6]. Moreover, the current smartphones and other mobile devices both have a large number of vulnerabilities, which makes the virus outbreaks become a great potential threat. There are two methods used by intruders to steal data from smartphone and it includes trojan infected apps and malicious apps [7]. The former is where the cyber criminals downloads an app

suppress it [5].

from mobile store and then re-upload it again into the app site with injected malicious malware[8]. Where as in the later, the cyber criminals create malicious app under the disguise of popular mobile app and upload them to the mobile store [9]. Virus causes unwanted behavior and degradation of the smartphone performance. The performance issues include frozen apps, failure to reboot, and difficulty in connecting to network. They can eat up battery or processing power, hijack the browser, send unauthorized SMS messages and freeze or brick the device entirely [10].

These has resulted in the rapid growth of malware which ranges from pop up advertisements to inimical encroachment of individuals[11], businesses. and governments systems [12]. Malware attacks smartphones by compromising personal information, deleting data or draining the battery[13]. Malicious software uses expensive features to steal phone services [5]. Smartphones can become infected either through downloading infected files using the phone internet browser, transferring files between phones using the Bluetooth interface, synchronizing with an infected computer, accessing an infected physical memory card, or opening infected files attached to Multimedia Message Service (MMS) messages[6]. Security issues of smartphones have become greatly of important since the large population of smartphone users and wide coverage of mobile communication network [14] create a breeding ground for the propagation of smartphone virus. The propagation of smartphone virus may be more potentially destructive than the computer virus[15]. In this regard, it is necessary to research the propagation behavior of smartphone virus attack in the real world and design effective containment strategies to

Zhang [16] discovered that smartphone virus can also propagate through multi-layered network. He concluded that smartphone viruses can use any network as their communication network. (3G, 4G, Wi-Fi, or Bluetooth).The smartphonevirus propagation starts with infection on a single phone. The virus on the infected phone send MMS message with an infected attachment file to other phones[17].When an unsuspected user accepts the infected attachment file, using a phone susceptible to the virus, then the virus is installed and infected the target phone which then begins to perform as an attacked phone. Smartphone virus can seize the victim's mobile device by running a malicious exploit, and this infected device will, in turn, scan and infect other smartphones in the mobile network [18]

Although the phone user education response mechanism strives to dissuade the user from accepting infected messages, other response mechanism, such as immunization, can prevent infection even if the user accepts the MMS message attachment [19]. The immunization response mechanism operates using software placed directly on each smartphone [13]. After the service provider detects virus that exploits a vulnerable smartphone, the service provider begins developing a patch to fix that vulnerability[20]. Once the patch is developed, the immunization software resident in each smartphone automatically installs any immunization patch available [16]. Bandwidth constraints prevent most smartphones from receiving the patch simultaneously, so the patch is rolled out to the entire smartphone population uniformly over a period of time[21]. The more servers that are dedicated to distributing these patches, the faster the deployment to all susceptible smartphones in the network [10]. When the deployed patch arrives at a particular smartphone, the smartphone become immunized from the virus if not infected, or the patch stops further propagation attempts from the smartphone if it is already infected [17].

Due to the specific features of smartphone virus propagation, attention has been focused on development of new techniques for the detection and elimination of viruses [22]. So far, previous models on propagation of computer viruses considered susceptible, infected, removed, recovered, exposed, patched, and external. The researchers combined them in different order to form the model, such as susceptible–infected–susceptible (SIS) models [23; 24], susceptible–infected–removed (SIR) models [25],

susceptible–infected–recovered–susceptible (SIRS) models [8], susceptible–exposed–infected–removed– susceptible (SEIRS) models [10], susceptible–infected–patched–susceptible (SIPS) models [20], susceptible–infected–external–susceptible (SIES) [16] and Susceptible–Vaccinated–Exposed–Infectious– Recovered (SVEIR) model[26].Only Upadhyay et al [26] included vaccinated in their model but quarantined is missing.This paper proposes Susceptible–Vaccinated–Exposed–Infectious– Recovered–Susceptible (SVEIQRS) model for the control of the virus attack in smartphones. MATLAB simulation experiments were performed to draw inference from this model, and then recommend strategies for controlling virus propagation on smartphone.

# **Related Work**

Smartphone users communicate and share files with their friends and they also take part in some activities or join groups online [27]. This characteristic gives hackers the opportunity to attack mobile users. As a result, the smartphone virus can spread quickly. Smartphone virus is similar to biological virus like HIV or HBV [28,29,30] as such some epidemical models used in representing malware propagation are presented to show the propagation of malware in smartphone. Recently, smartphone virus stability and propagation models have turn out to be an attractive research field to facilitate virus detection, analysis, prediction, and prevention. There are other proposed models to simulate the smartphone virus propagation. Yao, Xang, Qu, Tu and Geo[31] researched the worm propagation model by considering the time delay. They found that time delay may lead to Hopf bifurcation phenomenon which will make the worm propagation system unstable and uncontrollable.

To study the fundamental spreading patterns that characterize a mobile virus outbreak, Wang, Chen, Xu and Zhan [18]modeled the mobile benign worm based on two stages of repairing mechanisms.

Yan and Eidenbenz [32] built an analytical model to study the spread of Bluetooth worms. In their model, the impact of mobility patterns on Bluetooth worm propagation can be investigated by introducing the input parameters, such as average node degree, average node meeting rate and the link duration distribution. Xia, Chen and Yuan [33] built a susceptible - exposed - infected - recovered - dormancy (SEIRD) model for the Bluetooth and MMS hybrid spread model according to comister worm. This simply implies that they divided phone nodes into five states such as S, E, I, R, D.

Rhodes and Nekovee [34] explored the effect of device behavior and population characteristics on the dynamic outbreak of Bluetooth worm using the SIP model. They proposed that if a worm is introduced into the system, the system device can be either susceptible (S), infected (I), or recovered (R).

Fan, Zheng and Yang [19]formulated a Susceptible - Exposed - Infected - Recovered (SEIR) model to hybridize Bluetooth and SMS/MMS, which was based on preventive immunity and maturation of the mobile phone virus. They also discussed the influence of propagation parameters like virus mutation, preventive immunity of mobile phone users, SMS/MMS network immunity structure, and average degree of nodes in the Bluetooth.

Lanz, et al[5] proposed a malware transmission model in a network of mobile devices by considering the treatment effectiveness based on the type of malware infections accrued (hostile malware or malicious malware). The purposed model considers six classes of mobile devices based on their epidemiological status: Susceptible, exposed, and infected by hostile malware, infected by malicious malware, quarantined and recovered.

Martin, Burge, Gill, Washington and Alfred [35] used the SIS model from mathematical epidemiology to predict the mobile phone viruses. But Peng et al [14] observed that Martin et al [35] did not take into account the impact of individual differences on the propagation dynamics of proximity-based viruses, and then stated that epidemic models are usually classified into three categories: deterministic models, stochastic models and spatial temporal models.

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Wang et al [18] proposed a model for the spread and control of mobile benign worm based on two stage repairing mechanism. Their work focused on the spread of worm in the mobile environment and control process of mobile benign worms was divided into stages as rapid repair control and post repair that uses passive mode to optimize the environment for the purpose of controlling the mobile network.

Yang et al [5] developed two different propagation models for mobile phone virus namely: user-tricking virus which is intended to describe the propagation of user, and vulnerability-exploiting virus that describes the vulnerability propagation. Based on the traditional epidemic model, the characteristics of mobile phone viruses and the network topology structure are incorporated into their model.

Coronel et al [15] proposed a generalized SEIR-KS model. They assumed that virus propagation is time dependent and then classified the population as susceptible, exposed, infected, recovered, and kill signals. The computer population on the network is viewed as nodes which interact with each other at a given time.

In this research, we proposed virus transmission model in a network of smartphones by considering vaccination and treatment effectiveness based on the virus infection. The proposed model consists of six compartments of smartphones based on their epidemiological status: Susceptible, Vaccinated, Exposed, Infected, Quarantined and Recovered. Here, vaccinated implies smartphones with temporal immunity due to the antivirus software, where quarantined in this case means an isolation of the smartphone from the network while going through a treatment (scanning) process to remove the virus. It is also assumed that once the virus is removed, the smartphone employs temporary immunity which allow them to become susceptible again to the infection as the immunity wanes of.

## **Model Formulation**

The total population of smartphone at time t, denoted by N(t), is split into the mutually exclusive compartments of susceptible smartphones (S(t)), exposed smartphones to virus infection(E(t)),vaccinated smartphones (V(t)), Infectious smartphones (I(t)), quarantined smartphones (Q(t)), recovered Smartphones (R(t)), that is

$$N(t) = S(t) + V(t) + E(t) + I(t) + Q(t) + R(t)$$
(1)

The population of smartphones in the susceptible compartment is generated by the introduction of new smartphones into the network at the rate  $\theta$  and the recovered that become susceptible again at a rate  $\rho$ . This population is diminished by smartphones whose temporary immunity (vaccination) is updated at a rate  $\xi$ . It is also diminished by smartphones that had effective contact with infected ones at a rate  $\sigma\beta I$  and natural death at a rate  $\mu$  (that is, death other than virus attack). So, we have

$$\frac{dS}{dt} = \theta + \rho R - \xi S - \sigma \beta S I - \mu S$$

The population of smartphones in the vaccinated compartment is generated by susceptible smartphones whose temporary immunity (vaccination) is updated at a rate  $\xi$ , but is diminished natural death at a rate  $\mu$  and effective contact with infected smartphones by those whose temporary immunity is not updated at a rate  $\psi\beta I$ . Then we have

$$\frac{dV}{dt} = \xi S - \psi \beta V I - \mu V$$

The population of smartphones in the exposed compartment is generated by susceptible smartphones that had effective contact with infected ones at a rate  $\sigma\beta I$  and by those whose temporary immunity is not updated, who also had effective contact with infected smartphones at a rate  $\psi\beta I$ . This population is decreased by natural death at a rate  $\mu$  and progression from exposed compartment to infectious compartment at a rate  $\varphi$ . So we obtain

$$\frac{dE}{dt} = \sigma\beta SI + \psi\beta VI - (\mu + \varphi)E$$

The population of smartphones in the infectious compartment is generated by progression from exposed compartment at a rate  $\varphi$ . It is diminished by natural death at a rate  $\mu$ , death due to virus attack at a rate  $\alpha$ , progression from infectious compartment to quarantine compartment at a rate  $\gamma$  and progression from infectious compartment to recovered compartment at a rate  $\pi$ . Therefore, we obtain

$$\frac{dI}{dt} = \varphi E - (\mu + \alpha + \gamma + \pi)I$$

The population of smartphones in the quarantined compartment is generated by progression from infectious compartment at a rate  $\varphi$ . It is diminished by natural death at a rate  $\mu$ , death due to virus attack at a rate  $\alpha_1$ , and progression from Quarantined compartment to recovered compartment at a rate  $\delta$ . Therefore, we obtain

$$\frac{dQ}{dt} = \gamma I - (\mu + \alpha_1 + \delta)Q$$

The population of smartphones in the infectious compartment is generated by progression from infectious compartment to recovered compartment at a rate  $\pi$  and progression from Quarantined compartment to recovered compartment at a rate  $\delta$ . It is also decreased by natural death at a rate  $\mu$  and the recovered that become susceptible again at a rate  $\rho$ . Then we get

$$\frac{dR}{dt} = \pi I + \delta Q - (\mu + \rho)$$

The flow diagram is presented below.



Figure 1: Flow diagram for the model.

We itemize some of the main assumptions of the formulation of the model as follows:

- 1. All newly connected smartphones are virus free and susceptible.
- 2. Susceptible smartphones are vaccinated (which may be updated or not).
- 3. Each virus free smartphones and additional (old updated) smartphones get contact with infected smartphones.
- 4. Death rate other than the attack of virus is constant.
- 5. Exposed smartphones become infected.
- 6. Infected smartphones are treated.
- 7. Infectious smartphones are quarantined.

- 8. Quarantined smartphones are treated.
- 9. Recovered smartphones become susceptible again.
- 10. Smartphones clash due to virus attack.

Based on the above formulations, assumptions and flow chart, the following deterministic system of non-linear differential equations is the model for the transmission dynamics of virus attack; the associated state variables and parameters of the model are depicted below.

$$\frac{dS}{dt} = \theta + \rho R - \xi S - \sigma \beta I S - \mu S$$

$$\frac{dV}{dt} = \xi S - \psi \beta I V - \mu V$$

$$\frac{dE}{dt} = \sigma \beta I S + \psi \beta I V - (\mu + \varphi) E$$

$$\frac{dI}{dt} = \varphi E - (\mu + \alpha + \gamma + \pi) I$$

$$\frac{dQ}{dt} = \gamma I - (\mu + \alpha_1 + \delta) Q$$

$$\frac{dR}{dt} = \pi I + \delta Q - (\mu + \rho) R$$
(2)

## **Description of variables**

Variables	Description
S	Susceptible smartphones having no immunity.
V	Vaccinated smartphones having susceptibility.
Ε	Exposed smartphones having contact with infected ones.
Ι	Infected smartphones that have to be treated.
Q	Infected smartphones that are quarantined.
R	Recovered smartphones having temporary immunity.

## Description of Parameters

Parameters	Description				
β	Effective contact (transmission) rate				
γ	Progression rate from infectious compartment to quarantined compartment				
θ	Recruitment rate of susceptible smartphones				
δ	Progression rate from quarantined compartment to recovered compartment				
μ	Natural death rate				
ξ	Progression rate from susceptible compartment to vaccinated compartment				
ρ	Progression rate from recovered compartment to susceptible compartment				
σ	Progression rate from susceptible compartment to exposed compartment				
π	Progression rate from infectious compartment to recovered compartment				
$\varphi$	Progression rate from exposed compartment to infectious compartment				
$\psi$	Progression rate from vaccinated compartment to exposed compartment				

The force of infection is given by

$$\lambda = \beta I$$

(3)

## **Invariant Properties**

Since the population under study relates to that of physical quantities that cannot be negative, there is a need to prove that all state variables are non-negative for all time (t), for the model system (2) to be epidemiologically meaningful. In other words, the solution of the model system (2) with positive initial data will remain positive for all  $t \ge 0$ , we do this as follows;

The feasible region for this model is

$$\mathfrak{O} = \left\{ (S, V, E, I, Q, R) \in \mathbb{R}^6_{\ge 0}, S + V + E + I + Q + R \le \frac{\theta}{\mu} \right\}$$
(4)

Next, we show that the region  $\mathfrak{D}$  is positively invariant, so that it is sufficient to consider the dynamics of the above model in  $\mathfrak{D}$ .

Adding sub-equations in system (2) gives

$$N(t) = \theta - \mu N - \alpha I - \alpha_1 Q$$

Since the right side of the above equation is bounded by  $\theta - \mu N$ , it follows that

$$N(t) \le \frac{\theta}{\mu} + c e^{-\mu t}$$

Using a standard comparison theorem by [36 and 16], it can be shown that

$$N(t) \le N(0)e^{-\mu t} + \frac{\theta}{\mu}(1 - e^{-\mu t}).$$

Thus,  $\mathfrak{D}$  is positively invariant. If  $N(0) > \frac{\theta}{\mu}$ , then it is either that the solution enters  $\mathfrak{D}$  in finite time, or N(t) approaches  $\frac{\theta}{\mu}$  asymptotically, and the smartphone virus attack variables E, I, Q and R approaches zero. Hence  $\mathfrak{D}$  is attracting.

One of the most important concerns in the analysis of epidemiological models is the determination of the asymptotic behavior of their solution which is usually based on the stability of the associated equilibria [37]. This model typically consists of virus attack free equilibrium and the endemic equilibrium. The local stability of the virus attack-free equilibrium (VFE) is determined based on a threshold parameter, known as the basic reproduction number [38,39].

Local Stability of the Virus attack Free Equilibrium (VFE)

The VFE of model system (2) is given by

$$\mathcal{P}_0 = (S_0, V_0, 0, 0, 0, 0)$$

Where

$$S_0 = \frac{\theta}{\mu + \xi} \text{ and } V_0 = \frac{\xi \theta}{\mu(\mu + \xi)}$$

The local stability of VFE of model system (2) is determined by its basic reproduction number  $\mathcal{R}_0$ , which is computed using the next generation operator method proposed by [39]. Let

Where  $A = \mu + \varphi$ ,  $B = \mu + \alpha + \gamma + \pi$ ,  $C = \mu + \alpha + \delta$ ,  $D = \mu + \rho$ Then,

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$$\mathbf{v}^{-1} \rightarrow \begin{pmatrix} \frac{1}{A} & 0 & 0 & 0 \\ \frac{\phi}{A \cdot B} & \frac{1}{B} & 0 & 0 \\ \frac{\gamma \cdot \phi}{A \cdot B \cdot C} & \frac{\gamma}{B \cdot C} & \frac{1}{C} & 0 \\ \frac{\pi \cdot C \cdot \phi + \gamma \cdot \phi \cdot \delta}{A \cdot B \cdot C \cdot D} & \frac{\pi \cdot C + \gamma \cdot \delta}{B \cdot C \cdot D} & \frac{\delta}{C \cdot D} & \frac{1}{D} \end{pmatrix}$$

It follows that the reproduction number of smartphone virus attack of system (2) denoted by  $\mathcal{R}_0$  is given by

$$\mathcal{R}_0 = \beta \varphi \theta \left( \frac{\sigma \mu + \xi \psi}{\mu (\mu + \xi) AB} \right) \tag{5}$$

Interpretation of the Reproduction Number for the Model System (2)

there is need to interpret the reproduction number of model system (2), so that we can clearly see the meaning of the quantity:

$$\mathcal{R}_0 = \beta \varphi \theta \left( \frac{\sigma \mu + \xi \psi}{\mu (\mu + \xi) AB} \right)$$

this simply means that

$$\mathcal{R}_0 = \beta \theta \left( \frac{\sigma \mu + \xi \psi}{\mu(\mu + \xi)} \right) \times \frac{\varphi}{A} \times \frac{1}{B}$$

where  $\beta\theta\left(\frac{\sigma\mu+\xi\psi}{\mu(\mu+\xi)}\right)$  is the transmission probability,  $\frac{\varphi}{A}$  is that a smartphone has become infected (that is, moved from exposed to the infectious compartment) and is the average time that the smartphone is infectious.

**Lemma 1:** The VFE  $(\mathcal{P}_0)$  of the model system (2) is locally asymptotically stable (LAS) if  $\mathcal{R}_0 < 1$ , and unstable if  $\mathcal{R}_0 > 1$ . Epidemiologically, this implies that virus attack will be eliminated from the population (smartphones). Whenever  $\mathcal{R}_0 < 1$ , if the initial size of the population is in the basin of attraction of VFE, i.e., a small influx of virus attack smartphones through the network will not generate a large virus attack outbreak and the attack dies out in time.

From the reproduction number  $\mathcal{R}_0$ , we observe that the parameter  $\xi$  is important for the prevalence of virus attack. It controls the dynamic of virus attack most especially the equilibrium states, and the state of susceptibility, infected and exposed. Then from equation (5) we have

$$\lim_{\xi \to 1} \mathcal{R}_0 = \beta \varphi \theta \left( \frac{\sigma \mu + \psi}{\mu(\mu + 1)AB} \right) > 0 \tag{6}$$

Therefore, an effective antivirus software (vaccination) can positively influence virus attack ones the right-hand side of (6) is less than unity.

$$\psi < \frac{\mu(\mu+1)AB - \beta\varphi\theta\sigma\mu}{\beta\varphi\theta} \tag{7}$$

More so, the sensitivity analysis on the vaccination parameter of the susceptible smartphones and quarantine parameter were carried out by computing the partial derivative of  $\mathcal{R}_0$  with respect to  $\xi$  and  $\gamma$  respectively yields

$$\frac{\partial \mathcal{R}_{0}}{\partial \xi} = -\frac{\beta \varphi \theta \mu (\psi - \sigma)}{\mu (\mu + \xi)^{2} AB} \\
\frac{\partial \mathcal{R}_{0}}{\partial \gamma} = -\frac{\beta \varphi \theta (\mu \sigma + \psi \xi)}{\mu (\mu + \xi) AB^{2}}$$
(8)

This simply implies that the endemicity of the smartphone virus attack will be reduced if there is increase in vaccination and quarantine since their sensitivity indices are negative.

## **Global Stability Analysis**

Theorem 1: The VFE of Model system (2) denoted by  $\mathcal{P}_0$  is globally asymptotically stable (GAS) in  $\mathfrak{D}$  if and only if  $\mathcal{R}_0 \leq 1$ .

One of the effective methods used in addressing the problem associated with global stability analysis of epidemiological models is the use of LaSalle Lyapunov candidate function as in [40; 41]. Therefore, we have

$$\mathcal{L}(E,I) = aE + bI$$
(9)
where  $a = B$  and  $b = \beta \left( \frac{\sigma\theta}{\mu + \xi} + \frac{\psi\xi\theta}{\mu(\mu + \xi)} \right).$ 

The time derivative of  $\mathcal{L}$  given in (9) along the solution of the model (2) yields

$$\dot{\mathcal{L}}(E,I) = a\dot{E} + b\dot{I}$$

$$\dot{\mathcal{L}}(E,I) = a\left[\beta\left(\frac{\sigma\theta}{\mu+\xi} + \frac{\psi\xi\theta}{\mu(\mu+\xi)}\right)I - AE\right] + b[\varphi E - BI] \qquad (10)$$

$$\dot{\mathcal{L}} \leq \left[B\beta\left(\frac{\sigma\theta}{\mu+\xi} + \frac{\psi\xi\theta}{\mu(\mu+\xi)}\right)I - B\beta\left(\frac{\sigma\theta}{\mu+\xi} + \frac{\psi\xi\theta}{\mu(\mu+\xi)}\right)I\right] + \left[\varphi\beta\left(\frac{\sigma\theta}{\mu+\xi} + \frac{\psi\xi\theta}{\mu(\mu+\xi)}\right)E - ABE\right]$$

$$\dot{\mathcal{L}} \leq \left[\varphi\beta\theta\left(\frac{\sigma\mu+\psi\xi}{\mu(\mu+\xi)}\right) - AB\right]E \qquad (11)$$

Therefore,  $\dot{\mathcal{L}} \leq 0$  for  $\mathcal{R}_0 \leq 1$  with  $\dot{\mathcal{L}} = 0$  if and only if E = 0. By Lyapunov LaSalle invariance principle, the largest compact invariant set in  $\{(S, V, E, I, Q, R) \in \mathfrak{O}: \dot{\mathcal{L}} = 0\}$  is reduced to the VFE. This proves the global asymptotic stability on  $\mathfrak{D}[42]$ . The above result implies that the virus attack elimination is possible irrespective of the initial sizes of the sub-populations of the model whenever the threshold parameter,  $\mathcal{R}_0$ , is less than unity.

## **Existence of Endemic Equilibrium point (EEP)**

Endemic equilibrium defines the point where the virus attack cannot totally be eradicated from the population. we let  $\mathcal{P}_0^*$  be the endemic equilibrium point. To calculate the endemic equilibrium point we have

$$\mathcal{P}_0^* = (S^*, V^*, E^*, I^*, Q^*, R^*) \neq 0$$

Thus, model system (2) are solved in terms of the force of infection at the steady state  $(\lambda^*)$ yielding  $\lambda^* = \beta I^*$  (12)

Thus, we have

$$\begin{array}{l} \theta + \rho R^{*} - \xi S^{*} - \sigma \beta I^{*} S^{*} - \mu S^{*} = 0 \\ \xi S^{*} - \psi \beta I^{*} V^{*} - \mu V^{*} = 0 \\ \sigma \beta I^{*} S^{*} + \psi \beta I^{*} V^{*} - (\mu + \varphi) E^{*} = 0 \\ \varphi E^{*} - (\mu + \alpha + \gamma + \pi) I^{*} = 0 \\ \gamma I^{*} - (\mu + \alpha_{1} + \delta) Q^{*} = 0 \\ \pi I^{*} + \delta Q^{*} - (\mu + \rho) R^{*} = 0 \end{array}$$

$$(13)$$

Solving the above equations in (13) at steady state gives

$$S^* = \frac{\theta CD + (\rho \pi C + \rho \delta \gamma)I^*}{CD((\xi + \mu) + \sigma \lambda^*)}, \qquad V^* = \frac{\xi(\theta CD + (\rho \pi C + \rho \delta \gamma)I^*)}{CD((\xi + \mu) + \sigma \lambda^*)(\mu + \psi \lambda^*)}, \qquad E^* = \frac{BI^*}{\varphi}$$
$$Q^* = \frac{\gamma I^*}{C}, \qquad R^* = \frac{(\pi C + \delta \gamma)I^*}{CD}$$

and  $I^*$  is obtained by solving

$$\sigma[\rho\pi C + \rho\delta\gamma - \psi ABCD]\beta^{2}I^{*2} + [\sigma\mu\rho\pi C + \sigma\mu\rho\delta\gamma + \beta\theta\sigma CD + \psi\xi\rho\pi C + \psi\rho\delta\gamma\xi - ABCD((\mu + \xi)\psi + \mu\sigma)]\beta I^{*} + \frac{1}{\varphi}(\mathcal{R}_{0} - 1) = 0$$
(14)

Equation (14) can be written as

$$b_2(\lambda^*)^2 + b_1\lambda^* + b_0 = 0 \tag{15}$$

where

$$b_{2} = \sigma[\rho\pi C + \rho\delta\gamma - \psi ABCD]$$
  

$$b_{1} = \sigma\mu\rho\pi C + \sigma\mu\rho\delta\gamma + \beta\theta\sigma CD + \psi\xi\rho\pi C + \psi\rho\delta\gamma\xi - ABCD((\mu + \xi)\psi + \mu\sigma)$$
  

$$b_{0} = \frac{1}{\varphi}(\mathcal{R}_{0} - 1)$$

It is worth noting that the coefficient  $b_0$  is always positive if and only if  $\mathcal{R}_0 > 1$  and negative if and only if  $\mathcal{R}_0 < 1$ . Therefore, the number of possible real roots of equation (15) can have depends on the signs of  $b_2$ ,  $b_1$ , and  $b_0$ . This can be analyzed using the Descartes Rule of sign on the polynomial

$$f(\lambda^*) = b_2(\lambda^*)^2 + b_1\lambda^* + b_0 \tag{1}$$

Since  $\lambda^* \in [0, \beta]$ , one can easily verify that

$$f(0) = \frac{1}{\varphi}(\mathcal{R}_0 - 1) = b_0$$
$$f(\beta) = b_2(\beta)^2 + b_1\beta + b_0$$

A simple calculation proves that  $f(\beta) > 0$ . it is now a trivial matter to see that f(0) > 0 when  $\mathcal{R}_0 > 1$ . The existence follows the intermediate value theorem. Now there is a unique endemic equilibrium whenever  $b_0 > 0$ . There are two endemic equilibria when  $b_0 < 0$ ,  $b_1 > 0$ ,  $b_1^2 - 4b_2b_0 > 0$  and there are no endemic equilibria otherwise.

Lemma 2: When  $\mathcal{R}_0 > 1$ , the model system (2) has a unique endemic equilibrium  $\mathcal{P}_0^* = (S^*, V^*, E^*, I^*, Q^*, R^*)$  with  $S^*, V^*, E^*, I^*, Q^*$  and  $R^*$  all non-negative.

Now, let us study the stability of the endemic equilibrium guaranteed by lemma 2. Using the center manifold theory [43] as discussed in[44], we establish the local asymptotic stability (LAS) of the endemic equilibrium [45]. More precisely, we look for conditions on the parameter values that cause a forward or a backward bifurcation to occur. In order to do that, we will make use of the result summarized below, which has been obtained in [44] and is based on the use of general center manifold theory [46].

To apply this theory, the following simplification and change of variables are made first of all. Let  $x_1 = S, x_2 = V, x_3 = E, x_4 = I, x_5 = Q, x_6 = R$  so that  $N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$ . Further, by using vector notation  $x = (x_1, x_2, x_3, x_4, x_5, x_6)^T$ , the smartphone viral attack model system (2) can be written in the form  $\dot{x} = f(x)$  with  $f = (f_1, f_2, f_3, f_4, f_5, f_6)^T$ , as follows:

$$\dot{x}_1 = f_1 = \theta + \rho x_6 - \xi x_1 - \sigma \lambda x_1 - \mu x_1$$

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$$\dot{x}_{2} = f_{2} = \xi x_{1} - \psi \lambda x_{2} - \mu x_{2}$$

$$\dot{x}_{3} = f_{3} = \sigma \lambda x_{1} + \psi x_{2} - (\mu + \varphi) x_{3}$$

$$\dot{x}_{4} = f_{4} = \varphi x_{3} - (\mu + \alpha + \gamma + \pi) x_{4}$$

$$\dot{x}_{5} = f_{5} = \gamma x_{4} - (\mu + \alpha_{1} + \delta) x_{5}$$

$$\dot{x}_{6} = f_{6} = \pi x_{4} + \delta x_{5} - (\mu + \rho) x_{6}$$
(17)

where  $\lambda = \beta x_4$ 

The Jacobian of system (17), at the smartphone virus attack free equilibrium (VFE)  $\mathcal{P}_0$  is given by

$$J(\mathbf{P_0}) \coloneqq \begin{pmatrix} -\mathbf{C_1} & \mathbf{0} & \mathbf{0} & \mathbf{y_1} & \mathbf{0} & \mathbf{\rho} \\ \xi & -\mu & \mathbf{0} & \mathbf{y_2} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & -\mathbf{A} & \mathbf{y_3} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{\phi} & -\mathbf{B} & \mathbf{0} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{\gamma} & -\mathbf{C} & \mathbf{0} \\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{\pi} & \mathbf{\delta} & -\mathbf{D} \end{pmatrix}$$

Where 
$$C_1 = \xi + \mu$$
,  $A = \mu + \varphi$ ,  $B = \mu + \alpha + \gamma + \pi$ ,  $C = \mu + \alpha + \delta$ ,  $D = \mu + \rho$ ,  
 $y_1 = \sigma \beta x_{01}$ ,  $y_2 = \psi \beta x_{02}$ ,  $y_3 = \beta (\sigma x_{01} + \psi x_{02})$ ,  $x_{01} = \frac{\theta}{\xi + \mu}$ ,  $x_{02} = \frac{\xi \theta}{\mu (\xi + \mu)}$ .

Considering, next, the case when  $\mathcal{R}_0 = 1$  and suppose further that  $\beta = \beta^*$  is chosen as a bifurcation parameter, then we solve for  $\beta$  from  $\mathcal{R}_0 = 1$  which gives

$$\beta = \frac{\mu(\xi + \mu)AB}{\varphi\theta(\mu\sigma + \xi\psi)} \tag{18}$$

It follows that the Jacobian  $J(\mathcal{P}_0)$  of system (17) at smartphone virus free equilibrium, with  $\beta = \beta^*$  denoted by  $J_{\beta^*}$  has a simple zero eigenvalue (with all other eigenvalues having negative real part). Hence the center manifold theorem [43] can be used to analyze the dynamics of the model (17). The theorem in [41] will be used to chow that the unique endemic equilibrium of the model (17) (or, equivalently, Model (2)) is locally asymptotically stable for  $\mathcal{R}_0 = 1$ .

**Theorem 2:** Castillo-Chavez and Song [44] consider the following general system of ordinary differential equations with parameter  $\phi$ .

$$\frac{dx}{dt} = f(x,\phi) \quad f: \mathbb{R}^n \times \mathbb{R} \longrightarrow \mathbb{R} \text{ and } f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R})$$
(19)

Without loss of generality, it is assumed that 0 is an equilibrium for system (19) for all values of the parameter  $\phi$ , (that is  $f(0, \phi) \equiv 0$ ).

Assume

- 1.  $\mathcal{A} = \mathcal{D}, f(0, 0)$  is the linearization matrix of system (17) around the equilibrium 0 with  $\phi$  evaluated at 0. Zero is a simple eigenvalue of  $\mathcal{A}$  and other eingevalues of  $\mathcal{A}$  have negative real parts.
- 2. Matrix  $\mathcal{A}$  has a right eigenvector w and a left eigenvector v (each corresponding to the zero eigenvalue)

let  $f_k$  be the  $k^{th}$  component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0)$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi} (0,0)$$

Then the local dynamics of the system around the equilibrium point 0 is totally determined by the signs of a and b as describe in [41].

- 1. a > 0, b > 0. When  $\phi < 0$  with  $|\phi| \ll 1, 0$  is locally asymptotically stable and there exist a positive unstable equilibrium; when  $0 < \phi \ll 0, 0$  is unstable and there exists a negative locally stable equilibrium;
- 2. a < 0, b < 0. When  $\phi < 0$  with  $|\phi| \ll 1, 0$  is unstable; when  $0 < \phi \ll 1, 0$  is locally asymptotically stable equilibrium and there exists a positive locally unstable equilibrium;
- 3. a > 0, b < 0. When  $\phi < 0$  with  $|\phi| \ll 1, 0$  is unstable and there exist a locally asymptotically stable negative equilibrium; when  $0 < \phi \ll 1, 0$  is stable and a positive unstable equilibrium appears;
- 4. a < 0, b > 0. When  $\phi$  changes fro negative to positive, 0 changes from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Particularly, if a > 0 and b > 0, then a backward bifurcation occurs at  $\phi = 0$ .

We shall apply the above theorem by making the following necessary computations having in mind that our bifurcation parameter is  $\beta^*$  and not  $\phi$  as in theorem 2.

Eigenvectors of  $J_{\beta^*}$ : For the case when  $\mathcal{R}_0 = 1$ , it can be shown that the Jacobian of system (17) at  $\beta = \beta^*$  (denoted by  $J_{\beta^*}$ ) has a right eigenvector (corresponding to zero eigenvalue), given by  $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ , where

$$\begin{pmatrix} -C_1 & 0 & 0 & y_1 & 0 & \rho \\ \xi & -\mu & 0 & y_2 & 0 & 0 \\ 0 & 0 & -A & y_3 & 0 & 0 \\ 0 & 0 & \phi & -B & 0 & 0 \\ 0 & 0 & 0 & \gamma & -C & 0 \\ 0 & 0 & 0 & \pi & \delta & -D \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \end{pmatrix}$$

Gives

$$w_{1} = \frac{\rho \pi \varphi C + \rho \delta \gamma \varphi - \varphi y_{1} CD}{BCC_{1}D} w_{3}$$

$$w_{2} = \frac{\varphi \xi [\rho \pi C + \rho \delta \gamma - y_{1} CD] - \varphi y_{2} CC_{1}D}{BCC_{1}D} w_{3}$$

$$w_{3} = w_{3} > 0$$

$$w_{4} = \frac{\varphi}{B} w_{3}$$

$$w_{5} = \frac{\varphi \gamma}{BC} w_{3}$$

$$w_{6} = \frac{\pi \varphi C + \varphi \gamma \delta}{BCD} w_{3}$$

Similarly, the components of the left eigenvectors of  $J_{\beta^*}$  (corresponding to zero eigenvalue), denoted by  $v = (v_1, v_2, v_3, v_4, v_5, v_6)$  are given by

$$\begin{pmatrix} v_1 & v_2 & v_3 & v_4 & v_5 & v_6 \end{pmatrix}$$

(_(	21	0	0	У <sub>1</sub>	0	p )
ξ	-	-μ	0	У <sub>2</sub>	0	0
(	)	0 -	-A	У <sub>3</sub>	0	0
(	)	0	φ	-B	0	0
(	)	0	0	γ -	-C	0
	)	0	0	π	δ -	-D)

From which we obtain  $v_1 = v_2 = v_5 = v_6 = 0$ ,  $v_3 = v_3 > 0$ ,  $v_4 = \frac{y_3}{B}v_3$ 

Further, we computer the second order non zero partial derivative of system (17) at the virus attack free equilibrium point and obtain

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_4} = \frac{\partial^2 f_1}{\partial x_4 \partial x_1} = -\sigma\beta, \qquad \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = -\psi\beta$$
$$\frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial x_1} = \sigma\beta, \qquad \frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \psi\beta$$

Similarly

$$\frac{\partial^2 f_1}{\partial \beta \partial x_4} = \frac{\partial^2 f_1}{\partial x_4 \partial \beta} = -\sigma x_{01} = -\frac{\sigma \theta}{\xi + \mu}, \qquad \frac{\partial^2 f_2}{\partial \beta \partial x_4} = \frac{\partial^2 f_2}{\partial \beta \partial x_4} = -\psi x_{02} = -\frac{\psi \xi \theta}{\mu (\xi + \mu)}$$
$$\frac{\partial^2 f_3}{\partial \beta \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial \beta} = \sigma x_{01} + \psi x_{02} = \frac{\sigma \theta}{\xi + \mu} + \frac{\psi \xi \theta}{\mu (\xi + \mu)}$$

And all the other second order partial derivatives are zero. Thus, we can compute the coefficients a and b defined in theorem 2, that is,

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0)$$
$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi} (0,0)$$

Taking into account the system (17) and considering in *a* and bonly the non – zero derivatives for the term  $\frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0)$  and  $\frac{\partial^2 f_k}{\partial x_i \partial \beta}(0,0)$ , it follows that

$$a = 2v_1 w_1 w_4 \frac{\partial^2 f_1}{\partial x_1 \partial x_4}(0,0) + 2v_2 w_2 w_4 \frac{\partial^2 f_2}{\partial x_2 \partial x_4}(0,0) + 2v_3 w_1 w_4 \frac{\partial^2 f_3}{\partial x_1 \partial x_4}(0,0) + 2v_3 w_2 w_4 \frac{\partial^2 f_3}{\partial x_2 \partial x_4}(0,0)$$

$$a = 2v_{3}w_{4}(w_{1} + w_{2})$$

$$a = 2v_{3}\left(\frac{\varphi}{B}w_{3}\right) \left[ \left(\frac{\rho\pi\varphi C + \rho\delta\gamma\varphi - \varphi y_{1}CD}{BCC_{1}D}w_{3}\right)(\sigma\beta) + \left(\frac{\varphi\xi[\rho\pi C + \rho\delta\gamma - y_{1}CD] - \varphi y_{2}CC_{1}D}{BCC_{1}D}w_{3}\right)(\psi\beta) \right]$$

$$a = \frac{2v_{3}\varphi\beta}{B} \left[ \sigma\left(\frac{\rho\pi\varphi C + \rho\delta\gamma\varphi - \varphi y_{1}CD}{BCC_{1}D}\right) + \psi\left(\frac{\varphi\xi[\rho\pi C + \rho\delta\gamma - y_{1}CD] - \varphi y_{2}CC_{1}D}{BCC_{1}D}\right) \right] w_{3}^{2}$$

$$= \frac{2v_{3}\varphi\beta}{B} \left[ \left( \frac{(\rho\pi\varphi C + \rho\delta\gamma\varphi)(\sigma + \xi\psi)}{BCC_{1}D} \right) - \left( \frac{\varphi CD\left(\frac{\sigma\beta\theta}{\xi+\mu}\right)(\sigma + \xi\psi) + \psi\varphi CC_{1}D\frac{\psi\beta\xi\theta}{\mu(\xi+\mu)}}{BCC_{1}D} \right) \right] w_{3}^{2}$$

$$= \frac{2v_{3}\varphi\beta}{B} \left[ \left( \frac{(\rho\pi\varphi C + \rho\delta\gamma\varphi)(\sigma + \xi\psi)}{BCC_{1}D} \right) - \left( \frac{\varphi\beta\theta CD\left(\frac{\sigma^{2}}{\xi+\mu} + \frac{\xi\sigma\psi}{\xi+\mu} + \frac{\psi^{2}\xi}{\mu}\right)}{BC_{1}} \right) \right] w_{3}^{2}$$

$$= \frac{2v_{3}\varphi\beta}{B} \left[ \left( \frac{(\rho\pi\varphi C + \rho\delta\gamma\varphi)(\sigma + \xi\psi)}{BCC_{1}D} \right) - \left( \frac{\varphi\beta\theta\left(\frac{\mu\sigma^{2} + \mu\xi\sigma\psi + \psi^{2}\xi^{2} + \psi^{2}\mu\xi}{\mu(\xi+\mu)}\right)}{BC_{1}} \right) \right] w_{3}^{2}$$

$$= \frac{2v_{3}\varphi\beta}{B} \left[ \left( \frac{(\rho\pi\varphi C + \rho\delta\gamma\varphi)(\sigma + \xi\psi)}{BCC_{1}D} \right) - \left( \frac{\varphi\beta\theta\xi\psi(\mu\sigma + \xi\psi)}{\mu(\xi+\mu)BC_{1}} + \frac{\varphi\beta\theta\mu(\sigma^{2} + \psi^{2}\xi)}{\mu(\xi+\mu)BC_{1}} \right) \right] w_{3}^{2}$$

$$= \frac{2v_{3}\varphi\beta}{B} \left[ \left( \frac{(\rho\pi\varphi C + \rho\delta\gamma\varphi)(\sigma + \xi\psi)}{BCC_{1}D} \right) - \left( \frac{\xi\psi A}{C_{1}} \mathcal{R}_{0} + \frac{\varphi\beta\theta\mu(\sigma^{2} + \psi^{2}\xi)}{\mu(\xi+\mu)BC_{1}} \right) \right] w_{3}^{2}$$

Similarly,

$$b = 2v_1w_4 \frac{\partial^2 f_1}{\partial x_4 \partial \beta}(0,0) + 2v_2w_4 \frac{\partial^2 f_2}{\partial x_4 \partial \beta}(0,0) + 2v_3w_4 \frac{\partial^2 f_3}{\partial x_4 \partial \beta}(0,0)$$
  
$$= -2v_1w_4 \frac{\theta\sigma}{\xi + \mu} - 2v_2w_4 \frac{\theta\xi\psi}{\mu(\xi + \mu)} + 2v_3w_4 \left[\frac{\theta\sigma}{\xi + \mu} + \frac{\theta\xi\psi}{\mu(\xi + \mu)}\right]$$
  
$$b = 2v_3 \left(\frac{\varphi}{B}w_3\right) \left[\frac{\theta\sigma}{\xi + \mu} + \frac{\theta\xi\psi}{\mu(\xi + \mu)}\right]$$
  
$$b = \frac{2v_3\varphi\theta}{B} \left[\frac{\mu\sigma + \xi\psi}{\mu(\xi + \mu)}\right]w_3 > 0$$

Observe that the coefficient *b* is always greater than zero, so that, according to theorem 2, it is the sum of the coefficient *a* which decides the local dynamics around the virus attack free equilibrium for  $\beta = \beta^*$ . Base on theorem 2, model system (2) will undergo bifurcation.

### **Numerical Simulation**

Here, a control model is proposed for virus propagation based on the smartphone network using vaccination and quarantine. Several numerical simulations were performed with Runge Kutta (RK4) in MATLAB R2019a (9.6.0.1072779) to illustrate the dynamics of virus in smartphone network.

In figure 2, we numerically simulated the basic reproduction to show that as the control parameter  $\xi$  which is denoted by a4, the virus attack will be eradicated from the network. This implies that vaccination influences how much the virus attack can spread in the smartphones.



Figure 2: Simulation of the Basic Reproduction Number.



Figure 3: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.000057$ ,  $\alpha = \alpha_1 = 0.001$ ,  $\delta = 0.017$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.83 \gamma = 0.038$ ,  $\psi = 0.75$ ,  $\sigma = 0.8$ ,  $\rho = 0.00069$ .



Figure 4: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.000057$ ,  $\alpha = \alpha_1 = 0.001$ ,  $\delta = 0.017$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.83 \gamma = 0.038$ ,  $\psi = 0.75$ ,  $\sigma = 0.8$ ,  $\rho = 0.00069$ .



Figure 5: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.057$ ,  $\alpha = \alpha_1 = 0.1$ ,  $\delta = 0.17$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.83 \gamma = 0.00038$ ,  $\psi = 0.0075$ ,  $\sigma = 0.0008$ ,  $\rho = 0.069$ .



Figure 6: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.057$ ,  $\alpha = \alpha_1 = 0.1$ ,  $\delta = 0.17$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.83 \gamma = 0.00038$ ,  $\psi = 0.0075$ ,  $\sigma = 0.0008$ ,  $\rho = 0.069$ .



Figure 7: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.057$ ,  $\alpha = \alpha_1 = 0.1$ ,  $\delta = 0.017$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.083 \gamma = 0.0038$ ,  $\psi = 0.0075$ ,  $\sigma = 0.0008$ ,  $\rho = 0.069$ .



Figure 8: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.057$ ,  $\alpha = \alpha_1 = 0.1$ ,  $\delta = 0.017$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.083 \gamma = 0.0038$ ,  $\psi = 0.0075$ ,  $\sigma = 0.0008$ ,  $\rho = 0.069$ .



Figure 9: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.057$ ,  $\alpha = \alpha_1 = 0.1$ ,  $\delta = 0.17$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.83 \gamma = 0.00038$ ,  $\psi = 0.0075$ ,  $\sigma = 0.0008$ ,  $\rho = 0.069$ .



Figure 10: Simulation of the dynamic behavior of model system (2), generated in Matlab with parameter values  $\theta = 100$ ,  $\beta = 0.085$ ,  $\mu = 0.057$ ,  $\alpha = \alpha_1 = 0.1$ ,  $\delta = 0.17$ ,  $\xi = 0.65$ ,  $\varphi = 0.45$ ,  $\pi = 0.83 \gamma = 0.00038$ ,  $\psi = 0.0075$ ,  $\sigma = 0.0008$ ,  $\rho = 0.069$ .

Figure 3(a-f) and figure 4 shows the trajectory of model system (2) when there is no control and we observe that if the value of the parameters  $\sigma$  and  $\psi$  are increased, there will be a corresponding rise in the number of exposed and infected smartphones thereby causing the virus to continuously exist in the population. Figure 5(a-f) and figure 6, shows the trajectories of the model system (2) with effective control. We observed that when control is effective, (i.e., increasing the value of the parameters,  $\xi, \pi$  and  $\delta$ ), the number of vaccinated smartphones will subsequently rise causing the exposed and infected compartments to decrease to a minimal level. Figure 7(a-f) and figure 8 indicated that the control is not effective to eradicate the virus attack from the population. It is seen that the susceptible, vaccinated, exposed, infected, quarantined and recovered smartphones after oscillating for some time became constant which implies that the virus attack cannot be totally eradicated from the smartphone population if the control is effective. The performance of the vaccination rate to the propagation of vulnerability of smartphone to virus is shown in figure 9. It will be observed that the vaccination rate can affect the propagation of virus in smartphones. Certainly, the more vaccination rate  $\xi$  is, the lesser the smartphones are vulnerable to virus and it will subsequently be controlled from the smartphone population. This is because figure 10 depicted the recovery of the infected smartphones through treatment. In other words, to assure normal applications of smartphones and also suppress the propagation of smartphone virus attack, we should increase vaccination rate  $\xi$ .

## Discussion

A realistic deterministic system of nonlinear ordinary differential equation based compartmental model for transmission of viruses in smartphone has been proposed and analyzed. The main theoretical results obtained are as follows:

- 1. We are able to compute the basic reproduction number for smartphone virus attack,  $R_0$ ;
- 2. The model has a globally asymptotically stable virus free equilibrium whenever a certain epidemiological threshold ( $R_0$ ) is less than unity and unstable if this threshold exceeds unity;

3. The model has a unique endemic equilibrium whenever the basic reproduction number( $R_0$ ) exceeds unity. This endemic equilibrium is locally asymptomatically stable at least near  $R_0 = 1$  whenever it exists;

The simulation results provided many interesting insights into the effect of the dynamics of smartphone virus.

- i. The smartphone virus model has a VFE which is GAS.
- ii. The smartphone VFE is stable when  $R_0 < 1$ . However, the numerical results suggest that to reduce or control the impact of the virus propagation, increasing vaccination (that is, providing temporary immunity to smartphones and constantly updating it) can be an effective option. Such reductions would not be easy due to the waning vaccine-induced immunity. More so, significant reductions may be obtained through quarantine and subsequently treating both the quarantined and the infected that were not successfully quarantined. That is the only necessary option one's the smartphone losses it's temporary immunity and were infected.

## Conclusion

Epidemiology of smart phone virus is different from the model of computer malware because of the mobility of smart phone. We made use of deterministic system of non-linear differential equations to formulate the model for the transmission dynamics of virus attack in smart phone.

We considered the vaccination of susceptible and quarantine of infected smart phones. Through the analysis of this model, we can conclude that these measures, that is, proper vaccination and quarantine can effectively control the spread of smart phone virus.

#### References

- [1] Barrera, D. and Van Oorschot, P. Secure software installation on smartphones. IEEE Security & Privacy, 9(3), 42-48. (2010).
- [2] Lanz A, Roger D and Alford T L. An Epidemic Model of Malware Virus with Quarantine. Journal of Advances in Mathematics and Computer Science, 33(4), 1-10 (2019). <u>https://doi.org/10.9734/jamcs/2019/v33i430182</u>
- [3] Yang, L. X., & Yang, X. A new epidemic model of computer viruses. Communications in Nonlinear Science and Numerical Simulation, 19(6), 1935-1944(2014).
- [4] Viveros, S. The economic impact of malicious code in wireless mobile networks. In 4th International Conference on 3G Mobile Communication Technologies (pp. 1-6). IET. (June, 2003).
- [5] Yang, W., Wei, X. L., Guo, H., An, G., Guo, L., and Yao, Y. Modeling the propagation of mobile phone virus under complex network. The Scientific World Journal, 2014.
- [6] Chandrasekar S. and Jayaprakasan V. Automatic Detection and Restraining Mobile Virus Propagation using Android. International Journal of Computer Science and Mobile Computing, 3(1), 526-532(2014)
- [7] Fan, W. and Yeung, K. H. Online social networks—Paradise of computer viruses. *Physica A: Statistical Mechanics and its Applications*, 390(2), 189-197 (2011).
- [8] Zhao, J, Wang, L and Han, Z Stability analysis of two new SIRS models with two viruses International Journal of Computer Mathematics, 95(10), 2026-2035, (2018)DOI: <u>10.1080/00207160.2017.1364369</u>
- [9] Manjunath, V. and Colley, M. Reverse engineering of malware on android. SANS Institute InfoSec Reading Room (2011).
- [10] Batista, F. K., del Rey, Á. M., Quintero-Bonilla, S., and Queiruga-Dios, A. A SEIR Model for Computer Virus Spreading Based on Cellular Automata. In *International Joint Conference SOCO'17-CISIS'17-ICEUTE'17 León, Spain, September 6–8, 2017, Proceeding* (pp. 641-650). Springer, Cham (2017).
- [11] Weinberger, S. Computer security: Is this the start of cyberwarfare? Nature News, 474(7350), 142-145 (2011).
- [12] Gen C, Tang X, Liu W, Zhu Q and Zhang X, An Epidermic Model of Computer Viruses with Vaccination and generalized Nonlinear incidence rate, Applied Mathematics and Computation, 222, 265-274 (2013).
- [13] Racic, R., Ma, D. and Chen, H. Exploiting MMS vulnerabilities to stealthily exhaust mobile phone's battery. In 2006 Securecomm and Workshops (pp. 1-10). IEEE. (August ,2006).
- [14] Peng, S., Yu, S. and Yang, A. Smartphone malware and its propagation modeling: A survey. IEEE Communications Surveys & Tutorials, 16(2), 925-941(2013).
- [15] Coronel, A., Huancas, F., Hess, I., Lozada, E., and Novoa-Munoz, F. Analysis of a SEIR-KS Mathematical Model for Computer Virus Propagation in a Periodic Environment. *Mathematics*, 8(5), 761 (2020).
- [16] Zhang, C. Global behavior of a computer virus propagation model on multilayer networks. Security and Communication Networks, 2018.

- [17] Van Ruitenbeek E, Courtney T, Sander W H and Stevens F. Quantifying the Effectiveness of Mobile Phone Viruse response mechanism. In preceeding of the 37th Annual IEEE/IFTP International Conference on Dependable System and Networks (DNS 07), 790-799 (2007).
- [18] Wang, M., Chen, Z., Xu, L. and Zhan, H. Spread and control of mobile benign worm based on two-stage repairing mechanism. *Journal of Applied Mathematics*, 2014.
- [19] Fan Y, Zheng K and Yang Y. Epidemic model of mobile phone virus for hybrid spread mode with preventive immunity and mutation", in proc. 6th international conference on wireless communications networking and mobile computing (WiCOM 2010), Chengdu, China, 1-5 (2010).
- [20] Yang, L. X., Li, P., Yang, X., and Tang, Y. Y. Distributed interaction between computer virus and patch: A modeling study. arXiv preprint arXiv:1705.04818 (2017).
- [21] MadhuSudanan, V. and Geetha, R. Dynamics of epidemic computer virus spreading model with delays. Wireless Personal Communications, 115(3), 2047-2061 (2020).
- [22] Zhu, Q., Yang, X., Yang, L. X. and Zhang, C. Optimal control of computer virus under a delayed model. Applied Mathematics and Computation, 218(23), 11613-11619. (2012).
- [23] Kermack W. O, and McKendrick, A. G. A contribution to the mathematical theory of epidemics. Proceedings of the royal society of london. Series A, Containing papers of a mathematical and physical character, 115(772), 700-721 (1927). https://doi.org/10.1098/rspa.1927.0118
- [24] Nakamura, G. M., and Martinez, A. S. Hamiltonian dynamics of the SIS epidemic model with stochastic fluctuations. *Scientific reports*, 9(1), 1-9 (2019).
- [25] Zimeras, S., and Diomidous, MComputer Virus Models-The Susceptible Infected Removed (SIR) Model. *Studies in health technology and informatics*, 251, 75-77 (2018).
- [26] Upadhyay, R. K., Kumari, S. and Misra, A. K. Modeling the virus dynamics in computer network with SVEIR model and nonlinear incident rate. *Journal of Applied Mathematics and Computing*, 54(1-2), 485-509 (2017). <u>https://doi.org/10.1007/s12190-016-1020-0</u>
- [27] Iliev, A., Kyurkchiev, N., Rahnev, A., and Terzieva, T. Some models in the theory of computer virus propagation. *LAP LAMBERT Academic Publishing* (2019).
- [28] Mbah G.C.E and Chinebu T.I., Mathematical Model on the Viral Load/Burden of HIV/AIDS in the body of a Host. Journal of the Association of mathematical Physics, Vol. 17 (2010):299-310.
- [29] Chinebu T.I, Aja R.O and Mbah G.C.E., An intracellular Differential Equation Model for the dynamics of Hepatitis B Virus (Hbv) Infection with Immunity Control. International Journal of Precious Engineering Research and Application 2(5): 01-11, 2021 ISSN:2456-2734.
- [30] Chinebu T.I, Udegbe I.V and Eberendu A.C., Epidemic Model and Mathematical Study of Impact of Vaccination for the Control of Malware in Computer Network. Journal of Avances in Mathematics and Computer Science. 36(3): 72-96, 2021 ISSN: 2456-9968.
- [31] Yao, Y., Xiang, W., Qu, A., Yu, G. and Gao, FHopf bifurcation in an SEIDQV worm propagation model with quarantine strategy. *Discrete Dynamics in Nature and Society*, 2012.
- [32] Yan, G. and Eidenbenz, S. Modeling propagation dynamics of Bluetooth worms. In 27th International Conference on Distributed Computing Systems (ICDCS'07) (pp. 42-42). IEEE. (2007)
- [33] Xia W, Li Z, Chen Z and Yuan Z, Commwarrior worm propagation model for smartphone networks. *The J. China university of posts and telecommunications, vol. 15 (2)* 60-66. (2008).
- [34] Rhodes C J and Nekovee M. The opportunistic transmission of wireless worms between mobile devices. *Physica A. statistical mechanics* and its applications, 387(27) 6837-6844 (2008).
- [35] Martin J C, Burge L L I,Gill T I, Washington A N and Alfred M. Modelling the spread of Mobile Malware. Internal journal of Computing Aided Engineering and Technology (IJCAET) 2, 3-14 (2010).
- [36] Lakshmikantham V, Leela S and Martynyuk A. A stability Analysis of Nonlinear Systems. Marcel Dekker Inc. New York and Basel, 31 (1989)
- [37] Moghadas, S. M. Modelling the effect of imperfect vaccines on disease epidemiology. Discrete & Continuous Dynamical Systems-B, 4(4), 999 (2004).
- [38] Diekmann, O., Heesterbeek, J. A. P., and Metz, J. A. On the definition and the computation of the basic reproduction ratio R 0 in models for infectious diseases in heterogeneous populations. *Journal of mathematical biology*, 28(4), 365-382 (1990).
- [39] Van den Driessche, P. and Watmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences*, 180(1-2), 29-48 (2002).
- [40] Olaniyi, S., Lawal, M. A. and Obabiyi, O. S. Stability and sensitivity analysis of a deterministic epidemiological model with pseudorecovery. *IAENG International Journal of Applied Mathematics*, 46(2), 160-167 (2016).
- [41] Bowong, S., & Kurths, J. Modelling tuberculosis and hepatitis b co-infections. *Mathematical Modelling of Natural Phenomena*, 5(6), 196-242. (2010).
- [42] Bhatia, N. P., and Szegö, G. P. Stability theory of dynamical systems. Springer Science & Business Media. (2002)
- [43] Carr, J., and Muncaster, R. G. The application of centre manifolds to amplitude expansions. I. Ordinary differential equations. *Journal of differential equations*, 50(2), 260-279. (1983).
- [44] Castillo-Chavez, C., and Song, B. Dynamical models of tuberculosis and their applications. *Mathematical Biosciences & Engineering*, 1(2), 361 (2004).
- [45] Dushoff, J., Huang, W. and Castillo-Chavez, C. Backwards bifurcations and catastrophe in simple models of fatal diseases. *Journal of mathematical biology*, 36(3), 227-248 (1998).
- [46] Guckenheimer, J., and Holmes, P. Local bifurcations. In *Nonlinear oscillations, dynamical systems, and bifurcations of vector fields* (pp. 117-165) (1983). Springer, New York, NY.