Propagation Model with Varying Population Size of Removable Memory Device Virus

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Abstract

Removable memory device (RMD) is one of main way for propagating computer virus. In this paper, a dynamic propagation model of RMD-virus with varying population size is discussed. Unlike other computer virus propagation models, the proposed model mainly considers the RMD-virus propagation between host and RMD, which is embodied by introducing the RMD state and new propagation rate. Furthermore, to control the RMD-virus, three threshold parameters are obtained. The simulation results show that the proposed model can serve as a basis for understanding and simulating RMD-virus.

Keywords: Computer virus, propagation model, RMD-SIR model, RMD-virus

1 Introduction

Computer virus propagation mainly depends on two ways: network and removable memory device (RMD). In this paper, RMD represents all mobile devices related to computer, including flash disk, mobile phones, digital cameras, mobile hard disk, memory card etc. The virus to propagate through network and RMD is called as network virus and RMD-virus respectively.

There are significant similarities between the propagation of computer virus and biological epidemics [3, 10, 13]. Epidemiological propagation model of computer virus was based on two simplifications: (1) At any given time, each host in the total population is one of a finite number of states, e.g., susceptible, removed, exposed, infected and detected etc. (2) The virus transmission can be translated into a probability of a host infecting another one. Some epidemiological models, such as Susceptible-Infected-Recovered (SIR) model, have been considered by possible ways for obtaining the propagation behavior of computer virus with various states (e.g., susceptible (S), infected (I), and recovered (R)) and their transitions [2, 6, 7].

In this paper, we will discuss a RMD-virus propagation model, namely RMD-SIR, which is an extension of the SIR [8] model by adding the RMD state and transmission RMD state. We will analyze the RMD-virus-free equilibrium (RVFE) to derive the important parameter. The RMD-SIR provides an opportunity to study the behavior of RMD-virus propagation.

In former studies of computer virus propagation, the total number of hosts always is assumed constant. However, in reality, the population size (i.e., the number of hosts) is varying with time due to the network and off network of hosts. To study the propagation of RMD-virus with different host size, we assume the total host size is varying with time. Similarity, the total RMD number is also varying with time.

The remainder of this paper is organized as follows. Section 2 proposes a dynamic propagation model of RMD-virus based on dynamic propagation rates. Section 3 analyzes the stability of the RMD-SIR system, and Section 4 represents the dynamic property of the host population sizes. Section 5 represents some numerical results. Finally, conclusions are given in Section 6.

2 Description of RMD-SIR Model

For RMD-SIR model, we have following assumptions: (1). The total population size \( N \) of hosts and total population size \( n \) of RMDs are varying with time \( t \). (2) All RMDs can exchange information with more than one host. This assumption is reasonable for RMD. (3). Even user has found the host or RMD to be infected, and he can still continue to acquire information from this host or RMD, such as the computers in print shop or internet bar. (4). Once hosts are vaccinated, they can gain permanent immunity and no longer be infected by this RMD-virus.
In RMD-SIR model, the two new RMD states, $S_u$ and $I_u$ corresponding to the host states $S_c$ and $I_c$ in SIR model, are added into the traditional SIR model. The new propagation rates are $\beta_{cu}$ and $\beta_{uc}$ respectively, where $\beta_{cu}$ is the the propagation rate from host to RMD and $\beta_{uc}$ propagation rate from RMD to host. So, there are five states and state transitions in RMD-SIR model, which is shown in Figure 1.

$\begin{align*}
&\text{Figure 1: RMD-SIR model} \\
&\end{align*}$

### 2.1 States and State Transition in RMD-SIR Model

The total population of hosts is partitioned into three groups, and any host can potentially be one of these groups at any time $t$: (1) $S$ (Susceptible): All hosts in this group have not been infected by RMD-virus, only when these hosts contact with an infected RMD, they can be infected by the RMD-virus, indicated by $S_c$. (2) $I$ (Infected): All hosts in this group have been infected by the RMD-virus, indicated by $I_c$. (3) $R$ (Removed): All hosts in this group have been vaccinated and immunized to the RMD-virus, indicated by $R$.

The total population of RMD is partitioned into two groups: (1) $S$ (Susceptible): All RMDs in this group easily infect virus, only when RMDs insert into an infected host, RMDs are infected by the virus, indicated by $S_u$. (2) $I$ (Infected): All RMDs in this group have been infected by the virus, indicated by $I_u$. Where, $c$ and $u$ as the subscripts, indicate the states of the host and RMD, respectively.

Initially, all hosts are in $S$-state. Once RMD-virus intrudes into the system, these hosts may change their states according to the following rules:

- $S_c \to R$, using countermeasure of immunization.
- $S_c \to I_c$, infected by an infected RMD.
- $I_c \to R$, using immunization after virus stopping and cleaning in host.
- $I_c \to S_c$, do not capture immunization ability after RMD-virus stopping and cleaning in host.
- $S_u \to I_u$, infected by an infected host.
- $I_u \to S_u$, after RMD-virus cleaning, because RMD dose not have immunization ability itself, the remove RMD-virus in RMD must depend on clearing RMD-virus capability of host.

In Figure 1, each circular represents the states of host or RMD. And the directed lines denote the potential transition paths from one state to another.

We assume the hosts subject to the uniformly distribution. More specifically, we assume that the local density of the total hosts is a constant though the total population size $N(t)=S_c(t)+I_c(t)+R(t)$ may vary with time $t$. Here $S_c(t)$, $I_c(t)$ and $R(t)$ denote the sizes of the $S_c(t)$, $I_c(t)$ and $R$ classes at any time $t$, respectively. Similarly, the total population size $n(t)$ of RMD is varying with time $t$, denoted by $n(t)=S_u(t)+I_u(t)$.

Let $\epsilon>0$ and $\mu>0$ be birth rate and mortality rate not due to the RMD-virus respectively. Many researchers assume the birth rate and mortality rate are same [12]. In practice, these two parameters are not equivalent. It is assumed that all newborns are susceptible and direct transmission can be neglected. Let $\alpha$ be the death rate for the infectious hosts suffer a RMD-virus, it means the probability of infected hosts not to be used due to the RMD-virus. For convenience, we explain the notations used in this paper. The details are listed in Table 1.

1/n is the proportion of contacting between susceptible hosts and infective RMDs, and 1/N is the proportion of contacting between susceptible RMDs and infective hosts. For convenience, the replacement rates of host and RMD are set to be $\mu$. This assumption is consistent with the actual situation, because in practice, the replacement frequency between the host and RMD is very close.

### 2.2 RMD-virus Propagation Model

The RMDs in the RMD-SIR model can be formulated by following equation:

$$
\begin{align*}
\frac{dS_u(t)}{dt} &= en(t) - \beta_{cu}S_u(t)I_c(t)/N + \delta_uI_u(t) - \mu S_u(t) \\
\frac{dI_u(t)}{dt} &= \beta_{cu}S_u(t)I_c(t)/N - \delta_uI_u(t) - \mu I_u(t).
\end{align*}
$$

Where, $\frac{dS_c(t)}{dt}$ and $\frac{dI_c(t)}{dt}$ are added into the traditional SIR model. The new propagation rate from host to RMD and $\beta_{uc}$ propagation rate from RMD to host. So, there are five states and state transitions in RMD-SIR model, which is shown in Figure 1.

The hosts in RMD-SIR model can be formulated by following equation:

$$
\begin{align*}
\frac{dS_c(t)}{dt} &= en(t) + \delta_cI_c(t) - \beta_{uc}S_c(t)I_u(t)/n - \mu S_c(t) - \rho_{SR}S_c(t) \\
\frac{dI_c(t)}{dt} &= \beta_{uc}S_c(t)I_u(t)/n - (\mu + \omega + \delta_c + \alpha)I_u(t) \\
\frac{dR(t)}{dt} &= \omega I_c(t) + \rho_{SR}S_c(t) - \mu R(t).
\end{align*}
$$
Table 1: Notations and definitions

<table>
<thead>
<tr>
<th>Notation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>$S_u(t)$</td>
<td>Number of susceptible RMD at time $t$</td>
</tr>
<tr>
<td>$I_u(t)$</td>
<td>Number of infected RMD at time $t$</td>
</tr>
<tr>
<td>$S_c(t)$</td>
<td>Number of susceptible hosts at time $t$</td>
</tr>
<tr>
<td>$I_c(t)$</td>
<td>Number of infected hosts at time $t$</td>
</tr>
<tr>
<td>$R(t)$</td>
<td>Number of vaccinated hosts at time $t$</td>
</tr>
<tr>
<td>$N$</td>
<td>Total number of hosts under consideration</td>
</tr>
<tr>
<td>$n$</td>
<td>Total number of RMD under consideration</td>
</tr>
<tr>
<td>$e$</td>
<td>Birth rate</td>
</tr>
<tr>
<td>$\mu$</td>
<td>Replacement rate not due to the RMD-virus</td>
</tr>
<tr>
<td>$\beta_{cu}$</td>
<td>RMD-virus propagation rate from host to RMD</td>
</tr>
<tr>
<td>$\beta_{uc}$</td>
<td>RMD-virus propagation rate from RMD to host</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>Recovery rate of host from state $I$ to $S_c$</td>
</tr>
<tr>
<td>$\omega$</td>
<td>Recovery rate of RMD from state $I$ to $S_u$</td>
</tr>
<tr>
<td>$\rho_{SR}$</td>
<td>Rate at which infected hosts are vaccinated or treated</td>
</tr>
</tbody>
</table>

Combine (1) and (2) as:

\[
\begin{aligned}
\frac{dS_u(t)}{dt} &= eN(t) - \mu S_u(t) - \beta_{cu} S_u(t) I_u(t)/N + \delta_c I_c(t) - \delta_u I_u(t) \\
\frac{dI_u(t)}{dt} &= \beta_{cu} S_u(t) I_u(t)/N - \mu I_u(t) - \delta_u I_u(t) \\
\frac{dS_c(t)}{dt} &= eN(t) + \delta_c I_c(t) - \beta_{uc} S_c(t) I_u(t)/n - \mu S_c(t) - \rho_{SR} S_c(t) \\
\frac{dI_c(t)}{dt} &= \beta_{uc} S_c(t) I_u(t)/n - (\mu + \delta_c + \alpha) I_c(t) \\
\frac{dR(t)}{dt} &= \omega I_c(t) + \rho_{SR} S_c(t) - \mu R(t).
\end{aligned}
\]  

(3)

Where

\[
\begin{aligned}
\frac{dN(t)}{dt} &= (e - \mu)N(t) - \alpha I_c(t) \\
\frac{dn(t)}{dt} &= (e - \mu)n(t).
\end{aligned}
\]  

(4)

When $N(t)$ is not constant, it is often necessary to consider the proportions of individuals in five epidemiological classes, namely, $s_u = S_u/n$, $i_u = I_u/n$, $s_c = S_c/n$, $i_c = I_c/N$, $r = R/N$. We use $\nu$ to replace $d/dt$, according to the derivation formula $s_u' = (\frac{\partial}{\partial u})' = (\frac{s_u}{\nu} S_u N)$, we get $s_u' = e - \beta_{cu} s_u i_c + \delta_u i_u - \delta_s s_u$, then it is easy to verify that $s_u$, $i_u$, $s_c$, $i_c$, $r$ satisfy the following system of differential equations:

\[
\begin{aligned}
s_u' &= e - \beta_{cu} s_u i_c + \delta_u i_u - \delta_s s_u \\
i_u' &= \beta_{cu} s_u i_c - \beta_{uc} s_c i_u - \delta_u i_u \\
s_c' &= e - \beta_{uc} s_c i_u - (\rho_{SR} + \omega) s_c + \alpha i_c s_c \\
i_c' &= \beta_{uc} s_u i_c - (\omega + \delta_c + \alpha + \rho_{SR}) i_c + \alpha i_c^2 \\
r' &= \omega i_c + \rho_{SR} S_c - \omega + \alpha i_c r
\end{aligned}
\]  

(5)

Let $i_c' = 0$, we have

\[
i_c = i_u' = 0, s_u = 1, s_c = \frac{e}{e + \rho_{SR}}.
\]  

(6)

For the case of $i_c = i_u = 0$, we have the RVFE as follows

\[
EQ_{of} = P_0 = (s_u^*, i_u^*, s_c^*, i_c^*) = (1, 0, \frac{e}{e + \rho_{SR}}, 0).
\]  

(7)

For the case of $i_c > 0$, $i_u > 0$, we will verify the existence of the RMD-virus-epidemic equilibrium (RVEE) in Subsection 3.3.

3.1 Basic Reproduction Number of Model

It was easy to calculate the RVFE $EQ_{of} = (1, 0, \frac{e}{e + \rho_{SR}}, 0)$ of model (6). Let $X = (s_u, i_u, s_c, i_c)$, then the model (6)
can be written as $X' = F(X) - Z(X)$. Where
\[
F(X) = \begin{pmatrix}
\beta_{cu} s u_i c \\
0 \\
0
\end{pmatrix}
\quad Z(X) = \begin{pmatrix}
\delta_u i u + e i u \\
-\beta_{uc} s c i u + (\omega + \delta_c + \alpha + e) i c + \alpha i c^2 \\
-e - \beta_{uc} s c i u + (\rho_{SR} + e) s c - \alpha c e i c.
\end{pmatrix}
\]

The stability of $P_0$ is equivalent to all eigenvalues of (16) being with negative real parts, which can be guaranteed by
\[
R_0 = \frac{e \beta_{cu} \beta_{uc}}{\delta_u + e(\omega + \delta_c + \alpha + e)(e + \rho_{SR})} < 1
\]
when $R_0 > 1$, Equation (10) has one positive root and three negative roots. So, the RVFE $P_0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. So, we have following Lemma 1.

**Lemma 1.** RVFE $P_0$ is locally asymptotically stable if $R_0 < 1$.

**3.2.2 Global Stability of the RVFE**

In this subsection, we show that the parameter restrictions of local stability of the RVFE guarantee its global stability. Here we define another threshold parameter
\[
\hat{R}_0 = \frac{\beta_{cu} \beta_{uc}}{(\delta_u + e)(\omega + \delta_c + \alpha + e)}
\]
It notices that $\hat{R}_0 < 1$ guarantees $R_0 < 1$.

**Theorem 1.** RVFE $P_0$ of (6) is globally asymptotically stable $\hat{R}_0 \leq 1$; it is unstable if $R_0 > 1$. In the latter case, the solutions of (6) starting sufficiently close to $P_0$ move away from $P_0$.

**Proof.** We proof the global stability of $P_0$ by constructing a suitable Lyapunov function [11] $V = \beta_{uc} i u + (\delta_u + e)i c$. Differentiating $V$ along (6), we obtains Equation (19).

The maximum value of (19) is achieved at the extreme points: $A_1(0, 0, 0, 0)$, $A_2(1, 0, 0, 0)$, $A_3(0, 1, 0, 0)$, $A_4(0, 0, 1, 0)$, $A_5(0, 0, 0, 1)$. It is easy to verify that the situations at these five points are shown in Table 2.

**Table 2: Situations of five points**

<table>
<thead>
<tr>
<th>$A_1(0,0,0,0)$</th>
<th>$V''(5)=0$, when $R_0 \leq 1$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A_2(1,1,0,0)$</td>
<td>$V''(5)=0$, when $R_0 \leq 1$</td>
</tr>
<tr>
<td>$A_3(1,0,0,0)$</td>
<td>$V''(5)=0$, when $R_0 \leq 1$</td>
</tr>
<tr>
<td>$A_4(0,1,0,0)$</td>
<td>$V''(5)=0$, when $R_0 \leq 1$</td>
</tr>
<tr>
<td>$A_5(0,0,1,0)$</td>
<td>$V''(5)=0$, if $R_0=1$</td>
</tr>
</tbody>
</table>

Let (19) be 0, i.e., $V''(5) = 0$ if $i_c = i_u = 0$ or $\hat{R}_0 = 1$. The maximum invariant set in $\{(s_u, i_u, s_c, i_c) \in \Delta | V''(5) = 0\}$ is the singleton $\{P_0\}$. By LaSalle Invariance Principle ([4], Chapter 2, Theorem 6.4), the RVFE $P_0$ is globally asymptotically stable when $\hat{R}_0 \leq 1$.

If $R_0 > 1$, we define $V_2 = \beta_{uc} i u + (\delta_u + e)i c$. So, we have Equation (20).

Observe that $V_2''(5) > 0$ for $s_c$ sufficiently close to $e/(\rho_{SR} + e)$ except when $i_u = i_c = 0$. Solutions starting sufficiently close to $P_0$ leave a neighborhood of $P_0$ except those on the invariant $s_u$-axis, where (4) reduces to $s'_c = e - (\rho_{SR} + e)s_c$ and thus $s_c(t) \to e/(\rho_{SR} + e)$ as $t \to \infty$, completing the proof. $\square$
Theorem 1 points out that the threshold parameters $R_0$ and $R_0^*$ give whether the infected hosts and RMDs to be promptly eliminated locally and globally respectively. Reducing $R_0$ value to less than unity can eradicate virus with a small magnitude. However, when $R_0^*$ is adjusted less than or equal to unity, the virus can be eradicated even with a large magnitude. $R_0$ and $R_0^*$ can be explained as the virus propagate by increasing inflow of susceptible and the infectious individuals, while weakening the outflow of the infected and susceptible.

### 3.3 Existence of the Local Equilibrium (LE)

Subsection 3.2.2 shows that the RVFE is globally asymptotically stable when $R_0 \leq 1$. This implies that, if there is not LE, the virus can be eliminated in the end. From the view of virus propagation, it is more important to investigate the existence of LE.

Suppose that $P_c(s_*, i_*, s_c, i_c)$ is a LE. From Equation (6), its coordinate should satisfy

\[
\begin{align*}
& e - \beta_{cu}s_u i_c + \delta_u i_u + e s_u = 0 \\
& \beta_{cu}s_u i_c - \delta_u i_u - e i_u = 0 \\
& e - \beta_{uc} s_c i_u - (\rho_{SR} + e) s_c + \alpha i_c s_c = 0 \\
& \beta_{uc} s_c i_u - (\omega + \delta_c + \alpha + e) i_c + \alpha i_c^2 = 0
\end{align*}
\]

Where $s_u > 0, i_u > 0, s_c > 0, i_c > 0$. Using these inequalities, we have $(\rho_{SR} + e - \alpha i_c)(1 - s_u - i_u) = \omega i_c + \delta_u(1 - s_u)$, which gives the following range of $i_c$,

\[0 < i_c < \min\{1, (\rho_{SR} + e)/\alpha\}\]

We notice that, from Equation (22), when $\alpha$ is less that the $\rho_{SR}$, or $e$, or $\rho_{SR} + e, i_c$ will lie in the interval $(0, 1)$. Eliminating $s_u$, $i_u$, $s_c$, from Equation (12), $i_c$ satisfies Equation (23).

Where $R_0$ is defined by Equation (12). Furthermore, $s_u, i_u, s_c$ can be uniquely determined by Equation (24).

In (24), $s_u > 0, i_u > 0$, and $s_c > 0$ are satisfied respectively, they can be guaranteed by the fact that $0 < i_c < 1$ and $R_0 > 1$. Let $x \rightarrow f(x) \in R^n$ be a smooth vector field defined for $x$ in an open set $D \subset R^n$, where define

\[
f(i_c) = R_0,
\]

\[
f(i_c) = (1 - \frac{\alpha}{\omega + \delta_c + \alpha + e})i_c(1 - \frac{\alpha - \beta_{uc} \beta_{cu}}{\rho_{SR} + e}i_c)(1 + \frac{\beta_{cu}}{\delta_u + e}i_c)
\]

Case 1: When $(\rho_{SR} + e) < \alpha$ and $0 < i_c^* < 1$, the three roots of $f(i)$ are $i_1 = (\omega + \delta_c + \alpha + e)/\alpha, i_2 = (\rho_{SR} + e)/(\alpha - \beta_{uc} \beta_{cu})$, and $i_3 = (-\delta_u + e)/\beta_{cu}$. They all lie outside $[0, 1]$ when $R_0 > 1$. Furthermore, $f(0)=1$ and $f(1) = (\omega + \delta_c + e)(\rho_{SR} + e + \beta_{cu} \beta_{uc} - \alpha)(\delta_u + e + \beta_{cu})(\omega + \delta_c + \alpha + e)(\delta_u + e) > R_0$.

Case 2: When $0 < i_c^* < (\rho_{SR} + e)/\alpha$, the three roots of $f(i)$ all lie outside $[0, (\rho_{SR} + e)/\alpha]$ when $\rho_{SR} < (\omega + \delta_c + \alpha)$ and $R_0 > 1$. Furthermore, $f(0)=1$ and $f((\rho_{SR} + e)/\alpha) = \beta_{cu} \beta_{uc} (\omega + \delta_c + \alpha - \rho_{SR})(\alpha(\delta_u + e + \beta_{cu})(\rho_{SR} + e))/\alpha^2(\omega + \delta_c + \alpha + e)(\delta_u + e) > R_0$.

These two cases lead to the conclusion that, when $R_0 > 1$, the line $y=R_0$ has exactly on intersection $(i_{c*}, f(i_{c*}))$ with the graph of $f(i_c)$ that satisfies Equation (22). So, we can obtain the following result.

Theorem 2. Suppose that $R_0 > 1$, then model (6) has an interior equilibrium $P_c(s_*, i_*, s_c, i_c)$ and its coordinates satisfy Equations (21)-(23).

### 4 Analysis of the Population Sizes

In the previous sections, we investigate the global dynamics of the system (5) and obtain restrictive conditions of the parameter for RMD-virus. Are these conclusions compatible with the original system (3)? In this section, we focus on finding out the correlations between them.

We now study the dynamics of $(S_u(t), I_u(t), S_c(t), I_c(t), R(t)), n(t) = S_u(t) + I_u(t)$ and $N(t) = S_c(t) + I_c(t) + R(t)$, which are governed by systems (3) and (4). In fact, $R$ does not appear in the first four equation system (3),
\[ |\lambda E - J(P_0)| = \begin{bmatrix} \lambda + e & -\delta_u & 0 & \beta_{cu}c \varepsilon_1 \\ 0 & \lambda + (\delta_u + e) & 0 & -\alpha_{e1}c \\ 0 & \beta_{uc}S_{e1}^* & \lambda + (\rho_{SR} + e) & 0 \\ 0 & -\beta_{uc}S_{e1}^* & 0 & \lambda + (\omega + \delta_e + \alpha + e) \end{bmatrix} \] (15)

\[ V'(5) = \beta_{uc}[\beta_{cu}s_u c - (\delta_u + e)i_u] + (\delta_u + e)[\beta_{uc}s_e c - (\omega + \delta_e + \alpha + e)i_e + \alpha c^2] \]

\[ = \beta_{uc}\beta_{cu}s_u c - \beta_{uc}(\delta_u + e)i_u + (\delta_u + e)\beta_{uc}s_e c - (\delta_u + e)(\omega + \delta_e + \alpha + e)i_e + (\delta_u + e)\alpha c^2 \] (19)

which allows us to study the equivalent system

\[
\begin{align*}
\frac{dS_u(t)}{dt} &= eN(t) - \beta_{cu}S_u(t)I_c(t)/N + \delta_uI_u(t) \\
\frac{dI_u(t)}{dt} &= -\mu S_u(t) \\
\frac{dS_e(t)}{dt} &= \beta_{uc}S_u(t)I_c(t)/N - \delta_uI_u(t) - \mu I_u(t) \\
\frac{dI_c(t)}{dt} &= \beta_{uc}S_e(t)I_u(t)/N - (\mu + \omega + \delta_e + \alpha)I_c(t) \\
\frac{dN(t)}{dt} &= (e - \mu)N(t) - \alpha I_c(t)
\end{align*}
\] (26)

In its feasible region, \( \sum \{ (S_u, I_u, S_e, I_c, N, n) \in R^6_+ | 0 \leq S_u + I_u \leq n, 0 \leq S_e + I_c \leq N \} \). Where, \( R^6_+ \) is a non-negative real number of 6-dimensional space.

If \( e, \mu \) and \( \alpha > 0 \), or \( e, \mu \) and \( \alpha > 0 \), Equation (26) implies that total host population \( N(t) \)→0 monotonically as \( t \to \infty \) for all solutions with \( I_u \to 0 \) and \( I_c \to 0 \), namely the RMD-virus is initially present. If \( e = \mu \) and \( \alpha = 0 \), \( N(t) \) and \( n(t) \) remain constant so that (26) degeneracy to a model with constant population, whose dynamic behaviors are very similar to (5) and (6).

In the rest of this section, we assume that \( e > \mu, \alpha > 0 \) or \( e > \mu, \alpha = 0 \). The latter does not incorporate RMD-virus related death in host population, and the whole population will increase exponentially.

Let \( f(x) = \left(1 - \frac{\alpha}{\omega + \alpha + e + x}\right) \left(1 - \frac{\delta_u}{\rho_{SR} + x}\right) \left(1 + \frac{\alpha}{\delta_u + e}\right) \), where \( f(x) \) is the cubic polynomial defined in (25). Let \( \tilde{R}_0 = f((e - \mu)/\alpha) \), the parameters \( \tilde{R}_0 \) play key roles in the dynamics of the population sizes.

**Theorem 3.** If \( \tilde{R}_0 \neq R_0 \), system (26) has only a trivial equilibrium \( P_0(0, 0, 0, 0, 0) \). If \( \tilde{R}_0 = R_0 \) system (26) has a line of equilibrium points:

\[
\begin{align*}
\left( \frac{eN_u - \theta}{\mu} \right) &+ \left( \frac{\omega + \alpha + \mu + \delta_e}{\beta_{uc}[e + (\omega + \alpha + \mu + 2\delta_e)(e - \mu)]/\alpha} \right)(e - u)N_u - \alpha N_c, \\
\left( \frac{eN_e - \theta}{\mu} \right) &+ \left( \frac{\omega + \alpha + \mu + 2\delta_e}{\beta_{uc}[e + (\omega + \alpha + \mu + 2\delta_e)(e - \mu)]/\alpha} \right)(e - u)N_e - \alpha N_u
\end{align*}
\]

\( \tilde{N}_u, \tilde{N}_e \).

Where, \( \tilde{N}_u \) and \( \tilde{N}_e \) are arbitrary positive number. positive number.

**Proof.** We now consider the case \( e, \mu > 0 \) and \( \alpha > 0 \). System (26) also has trivial equilibrium \( P_0(0, 0, 0, 0, 0) \).

Our interest is to determine whether there is a LE in system (26). Suppose \( \tilde{P}_e(S_{u*}, I_{u*}, S_{e*}, I_{c*}, N_*, \tilde{n}_*) \) is a LE of system (26). Let the right-hand side of (21) equal to zero, we have

\[ 0 = e - \beta_{cu} \frac{S_{u*}}{N_*} I_{c*} + \delta_u \frac{I_{c*}}{N_*} - \mu \frac{S_{u*}}{N_*} \]

\[ \tilde{S}_{u*} = \frac{eI_{c*} - \theta}{\mu} \]

\[ \tilde{I}_{u*} = \frac{eN_u - \theta}{\mu} \]

\[ \tilde{S}_{c*} = \frac{eN_e - \theta}{\mu} \]

\[ \tilde{I}_{c*} = \frac{eN_e - \theta}{\mu} \]

Eliminating \( \tilde{S}_{u*}, \tilde{I}_{u*}, \tilde{S}_{c*}, \tilde{I}_{c*}, \tilde{N}_u, \tilde{N}_e \) from these following condition

\[ \beta_{uc}e(e - \mu) = \frac{(\omega + \alpha + \mu + \delta_e)(e - \mu)(\mu + \rho_{SR})}{\beta_{uc}[e + (\omega + \alpha + \mu + 2\delta_e)(e - \mu)]/\alpha} \]

\[ + \frac{\beta_{uc}(e - \mu)}{\alpha} \]

(27)

which is equivalent to Equation (28).

If \( R_0 \leq 1 \), then the infected hosts vanish. The RMD-virus does not suppress the growth of the host population so that \( N(t) \), \( n(t) \), \( S_e(t) \), \( S_u(t) \) tend to infinity exponentially with an exponential rate \( e - \mu \) as \( t \to \infty \).

Theorem 1 shows that when \( \tilde{R}_0 \leq 1 \), \( (s_u(t), i_u(t), s_e(t), i_c(t), r(t)) \to (1, 0, e/(e + \rho_{SR}), 0, \rho_{SR}/(e + \rho_{SR}) \) exponentially as \( t \to \infty \). According to system (4), we let

\[ N'(t) = ((e - \mu) - a\alpha_e)N. \]

(29)

We therefore claim that \( N(t) \) increases exponentially due to the fact that \( i_c(t) \to 0 \) as \( t \to \infty \) [1]. The trajectories of \( S_e(t) \) and \( R(t) \) tend to infinity due to the facts that \( s_e(t) = S_e(t)/N(t) \to e/(e + \rho_{SR}) \) and \( r(t) = R(t)/N(t) \to \rho_{SR}/(e + \rho_{SR}) \) respectively, which are independent of \( \tilde{R}_0 \). So, we can see the behavior of \( I_u(t), I_c(t) \) and \( R(t) \). \qed
\( V_t^{(5)} = \beta_uc[\beta_cu_su_{ic} - (\delta_u + e)i_u] + (\delta_u + e)[\beta_uc sc_iu - (\omega + \delta_c + \alpha + e)i_c + \alpha i_c^2] \\
= \beta_uc[\beta_cu_su_{ic} - \beta_uc(\delta_u + e)i_u + (\delta_u + e)\beta_uc sc_iu - (\delta_u + e)(\omega + \delta_c + \alpha + e)i_c + (\delta_u + e)\alpha i_c^2 \\
= i_c[\beta_uc \beta_cu_su - (\delta_u + e)(\omega + \delta_c + \alpha + e) + (\delta_u + e)\alpha i_c] + i_u \beta_uc(\delta_u + e)(s_c - 1) \) (20)

\[
e^\beta_uc \beta_cu_i c_e - [(\omega + \delta_c + \alpha + e)i_{ce} - \alpha i_{ce}^2] \times [(\rho_{SR} + e) - \alpha i_{ce} + \beta_uc \beta_cu_i c_e] \times [(\delta_u + e) - \beta_cu_i c_e] = 0 \\
\Rightarrow [(\omega + \delta_c + \alpha + e) - \alpha i_{ce}^2] \times [(\rho_{SR} + e) - \alpha i_{ce} + \beta_uc \beta_cu_i c_e] \times [(\delta_u + e) + \beta_cu_i c_e] = \epsilon \beta_uc \beta_cu \\
\Rightarrow \left(1 - \frac{\alpha}{\omega + \delta_c + \alpha + e}\right) \left(1 - \frac{\beta_uc \beta_cu_{ce}}{\rho_{SR} + e}\right) \left(1 + \frac{\beta_cu_i c_e}{\delta_u + e}\right) = R_0 \) (23)

**Theorem 4.** Suppose \( R_0 > 1 \) and \( \alpha < \beta_{uc} \). As \( t \to \infty \):

When \( R_0 > R_0 \), \( (S_u(t), I_u(t), S_c(t), I_c(t), R(t), N(t), n(t)) \to (\infty, \infty, \infty, \infty, \infty, \infty, \infty) \);

When \( R_0 < R_0 \), \( (S_u(t), I_u(t), S_c(t), I_c(t), R(t), N(t), n(t)) \to (0, 0, 0, 0, 0, 0, 0) \);

When \( R_0 = R_0 \), \( (S_u(t), I_u(t), S_c(t), I_c(t), R(t), N(t), n(t)) \to (N^{(1)}, N^{(2)}, \bar{N}_c, \bar{N}_s) \).

Proof. Since \( \bar{R}_0 = f(\frac{(e - \mu)}{\alpha}) \) and \( R_0 = f(i_{ce}) \), from the analysis of function \( f \) in Subsection 3.3, we know that \( \bar{R}_0 > R_0 \) or \( \bar{R}_0 < R_0 \) is equivalent to the relations \( e - \mu - \alpha i_{ce} > 0 \) or \( e - \mu - \alpha i_{ce} < 0 \) respectively. \( N' = (e - \mu)N - \alpha_i L \) can be rewritten as

\[
N' = [(e - \mu - \alpha i_{ce}) - \alpha (i_{ce} - i_{ce})]N. \) (30)

If \( e - \mu - \alpha i_{ce} > 0 \), \( N(t) \to \infty(t \to \infty) \) in (30), which implies \( (S_u(t), I_u(t), S_c(t), I_c(t), R(t)) \to (\infty, \infty, \infty, \infty, \infty) \) as \( t \to \infty \); if \( e - \mu - \alpha i_{ce} < 0 \), \( N(t) \to 0(t \to \infty) \) in (30), which implies \( (S_u(t), I_u(t), S_c(t), I_c(t), R(t)) \to (0, 0, 0, 0, 0) \) as \( t \to \infty \); if \( e - \mu - \alpha i_{ce} = 0 \), \( N(t) \to \bar{N}_c(t \to \infty) \) in (30), which implies as \( (S_u(t), I_u(t), S_c(t), I_c(t), R(t)) \to (\infty, \infty, \infty, \infty, \infty) \). Hence the trivial equilibrium \( \bar{P}_c \) is unstable.

5 Simulation Experiment

We first introduce the numerical experiment environments and then provide some results based on RMD-virus propagation control. As shown in Table 3, the parameters of RMD-SIR have two types: the system and the state transition parameters.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( N(0) )</td>
<td>10000</td>
<td>( \rho_{SR} )</td>
<td>Not fixed</td>
</tr>
<tr>
<td>( n(0) )</td>
<td>5000</td>
<td>( \alpha )</td>
<td>Not fixed</td>
</tr>
<tr>
<td>( \mu )</td>
<td>0.0002</td>
<td>( e )</td>
<td>0.005</td>
</tr>
<tr>
<td>( R(0) )</td>
<td>( N-S(0)-I(0) )</td>
<td>( I(0) )</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 3: Parameters used in experiments

In Figure 2, the host \( I_c \) and \( S_c \) states eventually tend to 0, and \( R \) state increases with time \( t \) and the total number \( N \). The proposed model is stable with \( R_0 < 1 \). From the Figure 2, we can draw several conclusions:

1) \( I_u \) and \( S_u \) are basic opposite trend, there are two reasons: one is RMD only has two states, another is...
the value of $\mu$ is very small, only 0.00022. But $I_u$ does not tend to 0 with time $t$, because new RMDs are assumed that $S_u$ state and RMD dose not have $R$ state.

2) When almost all of RMD will be infected, the number $n$ in Figure 2 is close to 5000 at climax, it is the total number of RMD. As RMD can not detect and remove viruses, when outbreak of RMD-virus takes place, almost all users have not taken countermeasures, or there are no countermeasures appearing. So at this moment, all RMDs that contact with the infected hosts will be infected.

3) With the moment of $I_c$ climax, peak time of $I_u$ also will come. They are basically at the same time, even early than $I_c$. That is, when outbreak of RMD-virus propagation between $I_c$ and $I_u$ takes place, the two objects $I_c$ and $I_u$ are the same important, the RMD-virus propagation will not outbreak if lack any one.

4) The peak time of $I_u$ sustains longer than $I_c$, because it depends on hosts to clear RMD-virus or the format of RMD-virus. That is to say, resistant RMD-virus is more difficult than host virus.

5.2 Impact of $e$ and $u$ on $N(t)$ and $n(t)$

We know that, if $e < \mu$ and $\alpha \geq 0$ or $e \leq \mu$ and $\alpha > 0$, the total host population $N(t) \to 0$ monotonically as $t \to \infty$ for all solutions with $I_s>0$ and $I_c>0$. As shown in Figure 3, $N(t)$ and $n(t)$ descent with time $t$, and $R$ and $S_u$ also descent with time $t$, they all tend to 0 as $t \to \infty$.

If $e=\mu$ and $\alpha=0$, $N(t)$ and $n(t)$ remain constant so that system (26) degenerates into a model with constant population, their dynamic behaviors are very similar to (5) and (6). As shown in Figure 4, two straight lines of $N(t)$ and $n(t)$ are horizontal, namely they are constant. $R$ state and $N(t)$ are almost parallel, $S_u$ and $I_u$ are also almost parallel with $n(t)$ to later. However, the number of hosts in $R$ state does not equal the $N$, which indicates that not all the hosts have the ability of immunity, even the RMD-virus has been controlled.

This situation shows that the host population does not consider RMD-virus-related death, so impacting factors on $N(t)$ and $n(t)$ are same, namely $e-\mu$. On other hand, in the whole simulation process, the values of $e$ and $\mu$ do not change, and the increasing speed of $N(t)$ and $n(t)$

$$
\begin{align*}
    &s_{u*} = 1 - i_{u*} \\
    &i_{u*} = \frac{\beta_{cu}i_{cs}}{\beta_{cu}i_{cs} + (\delta_u + e)} \\
    &s_{cs} = \frac{\beta_{cu}\beta_{uc}i_{cs}}{\beta_{cu}\beta_{uc}i_{cs} + (\delta_u + e)} \\
    &\tilde{R}_0 = f\left(\frac{e - \mu}{\alpha}\right) = \left(1 - \frac{e - \mu}{\omega + e}\right) \left(1 - \frac{(\alpha - \beta_{uc}\beta_{cu})e}{(\beta_{SE} + e)\alpha}\right) \left(1 + \frac{\beta_{cu}(e - \mu)}{(\delta_u + e)\alpha}\right) = R_0
\end{align*}
$$

Figure 3: RMD-virus propagation results with $e=0.005$, $\mu=0.0055$, $\alpha=0.06$, $e-\mu<0$

Figure 4: RMD-virus propagation results with $e-\mu=0$ and $\alpha=0$
is uniform. As shown in Figure 5, the increasing rate of \( N(t) \) and \( n(t) \) are essentially the same.

Figure 5: RMD-virus propagation results with \( e - \mu > 0 \) and \( \alpha = 0 \)

5.3 Impact of \( \beta_{uc} \) and \( \beta_{cu} \) on Model

We have known that \( \beta_{uc} \) indicates the RMD-virus propagate propagate probability from RMD to host, \( \beta_{cu} \) indicates propagate probability from host to RMD. The bigger \( \beta_{uc} \) means that few users take countermeasures against the RMD-virus and little ability to remove the RMD-virus in initial period. The initial period. The bigger \( \beta_{cu} \) means that more and more infected, but the solution countermeasures is inefficient, and which indicates that the virus outbreak is coming. The coming. The relationship between \( \beta_{uc} \) and \( \beta_{cu} \) whole periods of propagation process

These analytic results can help researchers to study various fact of RMD-virus propagation and new effective countermeasure to clear RMD-virus. So, the study of the relationship between \( \beta_{uc} \) and \( \beta_{cu} \) is necessary. If constant, we let \( \lambda = \beta_{cu}/\beta_{uc} \). The value of \( \lambda \) impacts simulation results of the RMD-SIR model. Figure 6 shows simulation results when changing \( \lambda \).

From Figure 6, we can see that \( \lambda \) can impact the speed of \( I_c \) declining. The relationship between \( \beta_{uc} \) and \( \beta_{cu} \) impacts the whole speed of removing RMD-virus. Therefore, this key point in the RMD-virus propagation model. Through correctly handling this relationship, we can quickly eliminate RMD-virus. In Figure 6(a), the peak value of \( I_e \) is about 7800, but in Figure 6(b), it is only about 6300, and the minimum value of \( I_u \) is less than Figure 6(a)’s. The bigger \( \lambda \) indicates \( \beta_{cu} \) is larger than \( \beta_{uc} \); that is to say, the RMD-virus propagation from host to RMD is faster than propagation speed from RMD to host, which can cause more RMDs infected by infected hosts. After RMD-virus breaking out, \( I_c \) and \( I_u \) should decline with with effective countermeasure, but the higher \( \beta_{cu} \) can cause more RMDs infected, at this time, remove RMD-virus from RMDs need some time. For example, in actual life, the RMDs also are infected after being cleared. In other words, we should keep \( \lambda \) with a smaller value; which can faster eliminate RMD-virus. To this purpose, \( \beta_{cu} \) should keep smaller. So relative to \( \beta_{uc} \), \( \beta_{cu} \) is more worth considerable.

6 Conclusion

The objective of this paper is to establish the RMD-virus propagation model, and then to find out control methods of RMD-virus propagation for eliminating virus. We proposed the RMD-SIR model with varying population size based on epidemiologic model SIR and obtained the conditions by the asymptotically stability of RVFE. We get the basic reproduction number \( R_0 \), furthermore, other threshold parameters, e.g. \( \bar{R}_0 \) and \( \bar{R}_0 \), are also obtained govern the RMD-virus propagation, which involve the total number of infective nodes and their proportion in all nodes. We analyzed the trend of each state in RMD-SIR model with varying \( N(t) \) and \( n(t) \). The simulation results show that the proposed model can help us for understanding and simulating RMD-virus propagation. The future work will focus on exploring more complex RMD-virus propagation model, which may require more specific parameters to analyze the more effective method of controlling RMD-virus propagation. We will also study some countermeasures by building up a practical and effective defense system against RMD-virus.

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References

Figure 6: RMD-virus propagation results impacted by $\beta_{uc}$ and $\beta_{cu}$


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