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Study of the stability of a SEIRS model for computer worm propagation

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HIGHLIGHTS

- A new mathematical model for computer worm propagation is proposed.
- It is an improvement of the model due to Toutonji et al.
- A more realistic basic reproductive number, R_0, has been derived.
- Efficient control strategies are stated from the expression of the R_0.

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ABSTRACT

Nowadays, malware is the most important threat to information security. In this sense, several mathematical models to simulate malware spreading have appeared. They are compartmental models where the population of devices is classified into different compartments: susceptible, exposed, infectious, recovered, etc. The main goal of this work is to propose an improved SEIRS (Susceptible–Exposed–Infectious–Recovered–Susceptible) mathematical model to simulate computer worm propagation. It is a continuous model whose dynamic is ruled by means of a system of ordinary differential equations. It considers more realistic parameters related to the propagation; in fact, a modified incidence rate has been used. Moreover, the equilibrium points are computed and their local and global stability analyses are studied. From the explicit expression of the basic reproductive number, efficient control measures are also obtained.

1. Introduction

Malware is software created to carry out activities in a device (computer, laptop, smartphone, tablet, etc.) without the consent of its owner. The consequences of malware affect both physical materials and the logical structure of devices. In fact, malware is one of the most important security threats and the estimation of the cost of their malicious effects exceeds millions of dollars, so this could cause high economic and social impacts [1].

Consequently it is very important not only to detect the presence of malware in a network, but also to simulate its propagation. The majority of efforts in this subject are associated with development of techniques to detect malware [2], whereas the design of mathematical models to simulate malware propagation has received less attention [3]. The importance

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of these models reside in both the obtaining of relevant information about the behavior of malware and the determination of the efficiency of control measures.

The great majority of mathematical models to simulate malware spreading are global and deterministic [3] and they are usually based on systems of ordinary differential equations [4]; nevertheless, also interesting individual-based proposals based on cellular automata has appeared [5]. Furthermore, they are also compartmental models since the population of devices are classified into several types according to the relationship with the malware. Thus, we can consider among other types, SEIRS models where susceptible, exposed, infectious and recovered devices are taken into account.

The susceptible devices are those devices that have not been infected by the malware. Exposed devices are those that have been successfully infected but the malware remains latent (that is, it cannot perform neither its payload nor the spreading process). Infectious devices are those exposed devices where the malware is activated and ready to propagate, and finally, recovered devices are those infected devices where the malware has been detected and successfully removed. Note that when the malware reaches a susceptible device, it becomes exposed. The exposed devices change into infectious when the malware is activated (the malicious code is ready to perform its payload and/or to propagate). Infectious or exposed devices become recovered when the malware is successfully detected and an efficient recovery process removes it from the device. Furthermore, also susceptible devices can change into recovered when they are supplied with adequate antivirus software. Finally, some recovered devices become susceptible again after a temporary immunity period.

A few works proposing SEIRS models have appeared in the literature (see [6–9]). In [6] Hosseini et al. proposed a discrete-time SEIRS model to study the dynamical behavior of malware propagation in scale-free networks considering software diversity. Mishra and Keshri [7] introduced a SEIRS model considering vaccinated devices to simulate malware spreading in a wireless sensor network, and Mishra and Pandey proposed an e-epidemic SEIRS model for the transmission of computer worms in computer network through vertical transmission [8]. Nevertheless, our work is focused in the model due to Toutonji et al. [9]. The importance of this model is that it considers accurate positions for dysfunctional hosts and their replacements in state transition.

Although this is an influential model, we have detected some drawbacks and consequently we have set as the main goal of this work its improvement. Specifically, in this paper an improved model is introduced and the local and global stabilities in both, the worm-free equilibrium state and the worm-endemic equilibrium state, are derived in detail. Furthermore, efficient control strategies are also proposed taking into account the explicit expression of the basic reproductive number obtained.

The rest of the paper is organized as follows: in Section 2 the model due to Toutonji et al. [9] is revisited; its critical analysis and the mathematical description of the new model is shown in Section 3; Section 4 is devoted to the stability analysis of the proposed model, and some control strategies are studied in Section 5. Finally, the conclusions and further work is presented in Section 6.

2. The model proposed by Toutonji, Yoo and Park

As was mentioned above, the model proposed by O.A. Toutonji, S.M. Yoo and M.Y. Park [9] is a SEIRS model that takes into consideration security countermeasures in order to prevent and protect from computer worms, and the effect of adjusting them on the exposed and infectious compartments: in this model the abnormal functioning of devices occurs in the infectious state, and the hosts replaced are fully up-to-date with the security countermeasures such that the replacement occurs in the recovered state.

Specifically the establishment of security countermeasures rules (1) the transition from susceptible to recovered devices by the rate $\psi_1 \geq 0$, (2) the transition from exposed hosts to recovered by the rate $\psi_2 \geq 0$, and (3) the transition from infectious to recovered devices by means of the rate $\gamma \geq 0$. In addition, these security countermeasures provide temporary immunity to some hosts and permanent immunity to the rest of devices; in this sense, $\phi \geq 0$ is the transition rate from recovered devices to susceptible hosts.

On the other hand, all hosts are vulnerable to malware attacks and the force of incident is defined as $f = \frac{\beta\alpha}{N}$ where $\beta \geq 0$ stands for the number of incidents per unit of time, $\alpha \geq 0$ is the state transition rate from exposed to infectious host and N is the total number of devices (that is, $\alpha E(t)$ is the number of attacked hosts moved to the infectious compartment per unit of time, where $E(t)$ stands for the number of exposed hosts at step of time t).

Finally, $\theta \geq 0$ is the dysfunctional rate and $\mu \geq 0$ is the replacement rate. In this sense, as the total number of hosts N is considered fixed, the number of replaced hosts, μN, must be equal to the number of dysfunctional hosts, $\theta I(t)$, where $I(t)$ is the number of infectious devices at t.

Thus, taking into account the last mentioned considerations and parameters, and setting $S(t)$ and $R(t)$ as the number of susceptible and recovery devices at step of time t respectively, the system of differential equations that governs the model is the following:

$$\frac{dS}{dt} = -fES - \psi_1 S + \phi R, \tag{1}$$

$$\frac{dE}{dt} = fES - (\alpha + \psi_2)E, \tag{2}$$

$$\frac{dI}{dt} = \alpha E - (\gamma + \theta)I, \tag{3}$$

$$\frac{dR}{dt} = \mu N + \psi_1 S + \psi_2 E + \gamma I - \phi R, \tag{4}$$

where the total number of devices is fixed: $N = S(t) + E(t) + I(t) + R(t)$ for every $t \geq 0$.

The basic reproductive number associated to this model is

$$R_0 = \frac{\alpha\beta\phi}{(\psi_1 + \phi)(\alpha + \psi_2)}. \tag{5}$$

Moreover, the explicit expressions of both the worm-free equilibrium point E_f^* and the worm-endemic equilibrium point E_e^* are the following:

$$E_f^* = \left(S_0^*, E_0^*, I_0^*, R_0^*\right) = \left(\frac{\phi N}{\psi_1 + \phi}, 0, 0, \frac{\psi_1 N}{\psi_1 + \phi}\right), \tag{6}$$

$$\begin{aligned} E_e^* &= \left(S_1^*, E_1^*, I_1^*, R_1^*\right) \\ &= \left(\frac{\alpha + \psi_2}{\beta\alpha}N, \frac{\phi - \frac{\alpha+\psi_2}{\beta\alpha}(\psi_1 - \phi)}{\alpha + \psi_2 + \phi\left(1 + \frac{\alpha}{\gamma+\theta}\right)}N, \frac{\alpha}{\gamma + \theta}E_1^*, N - S_1^* - E_1^* - I_1^*\right). \end{aligned} \tag{7}$$

The stability analysis of this model yields the following results [9,10]:

Theorem 1. *The worm-free equilibrium point E_f^* is locally asymptotically stable and globally asymptotically stable if $R_0 \leq 1$.*

Theorem 2. *The worm-endemic equilibrium point E_e^* is locally asymptotically stable if $R_0 > 1$.*

Theorem 3. *If $R_0 < 1$ and one of the following conditions holds:*

(i) $\psi_1 > \alpha + \psi_2$ *and* $\psi + \gamma + \theta > \alpha$,
(ii) $\psi_1 \geq \alpha + \psi_2$ *and* $\psi_1 + \psi + \gamma + \theta > 2\alpha + \psi_2$,

then the worm-endemic equilibrium point E_e^* is globally asymptotically stable.

As a consequence, the following statement also holds in order to prevent the widespread of computer worm:

Corollary 1. *To stop the computer worm propagation, the recovery rate associated to the susceptible compartment should satisfy the following inequality:*

$$\psi_1 > \phi\left(\frac{\beta\alpha}{\alpha + \psi_2} - 1\right). \tag{8}$$

3. The new model

3.1. Critical analysis of the model due to Toutonji et al.

A critical analysis of the model introduced in the last section exhibits some drawbacks that must be overcome in order to enhance the realism of the model. In what follows, these drawbacks are shown and how to solve them is also introduced.

There are two recovery rates in the original model not related to infectious devices: ψ_1 associated to susceptible devices, and ψ_2 corresponding to exposed devices. Moreover, it is supposed that these two coefficients are different: $\psi_1 \neq \psi_2$. As exposed devices are those infected devices in which the malware is not active (it is in the latent period), the malicious code is not able to perform its payload and its propagation to other hosts neither [11]. Consequently, it is difficult to detect it not only by human perception but also for malware detection techniques. This is, for example, the case of zero-days malware that exploit unknown vulnerabilities and, consequently, have unknown signatures. Then, it seems to be reasonable to suppose that there is no significant difference between the behavior of a susceptible and an exposed device and therefore the numeric values of ψ_1 and ψ_2 must be very similar: $\psi_1 \approx \psi_2$, but also supposing $\psi_1 < \psi_2$. Note that in the original paper (see [9]) the values considered in the simulations are very different: $\psi_1 = 0.0003 \ll \psi_2 = 2.8$.

In the model [9] the population dynamic is considered when the dysfunctional infectious hosts, $\theta I(t)$, are replaced and they are fully up-to-date with security measures, having been replaced in the recovery state. In this sense, the authors assumed that $\theta I(t) = \mu N$ and consequently $I(t) = \frac{\mu}{\theta}N$ thus $I(t)$ remains constant over the time. Obviously, this is not a realistic situation so that in the improved model it will not be considered (in fact, the number of replaced dysfunctional devices will be $\theta I(t)$).

On the other hand, in the model due to Toutonji et al. the incidence (that is, the new infected hosts – exposed in our case – per unit of time) is defined by

$$fE(t)S(t) = \frac{\beta\alpha}{N}E(t)S(t) = \beta\frac{S(t)}{N}(\alpha E(t)). \tag{9}$$

This follows the traditional *mass action* law considering the factors $S(t)$ and $E(t)$, and consequently, the unique devices capable of transmitting the malware are $\alpha E(t)$, that is, the new infected devices per unit of time (which are the new

exposed devices per unit of time). Thus, the infectious devices are not involved in the propagation process and their roles are undervalued. In order to improve this, the incidence will be defined as $\frac{\beta}{N} I(t) S(t)$ in the model proposed in the next section.

3.2. Mathematical description

Considering the reasoning made in the last subsection, the system of ordinary differential equations that rules the dynamic of the improved model (see Fig. 1) is the following:

$$\left.\begin{aligned} \frac{dS}{dt} &= -\frac{\beta}{N} SI - \psi_1 S + \phi R \\ \frac{dE}{dt} &= \frac{\beta}{N} SI - (\alpha + \psi_2) E \\ \frac{dI}{dt} &= \alpha E - (\gamma + \theta) I \\ \frac{dR}{dt} &= \theta I + \psi_1 S + \psi_2 E + \gamma I - \phi R \end{aligned}\right\} \tag{10}$$

where $\psi_1 \approx \psi_2$ and

$$N = S(t) + E(t) + I(t) + R(t). \tag{11}$$

It is easy to check that the set

$$\Omega = \left\{(S, E, I, R) \in \mathbb{R}^4 : 0 \leq S, E, I, R \leq N, S + E + I + R = N\right\} \tag{12}$$

is positively invariant with respect to the system (10). Then, we can focus our attention on the following reduced system of three ordinary differential equations by considering the condition (11):

$$\left.\begin{aligned} \frac{dS}{dt} &= \phi N - \frac{\beta}{N} SI - (\psi_1 + \phi) S - \phi E - \phi I \\ \frac{dE}{dt} &= \frac{\beta}{N} SI - (\alpha + \psi_2) E \\ \frac{dI}{dt} &= \alpha E - (\gamma + \theta) I \end{aligned}\right\}. \tag{13}$$

Finally, note that from the first equation of the system (13) it is:

$$\frac{dS}{dt} \leq \phi N - (\psi_1 + \phi) S, \tag{14}$$

and a simple computation shows that:

$$S(t) \leq \frac{e^{-(\phi+\psi_1)t} + \phi N}{\phi + \psi_1}, \tag{15}$$

and, consequently,

$$\lim_{t \to \infty} S(t) \leq \frac{\phi N}{\phi + \psi_1}. \tag{16}$$

4. Stability analysis

The equilibrium points are the solutions of the following system:

$$\left.\begin{aligned} 0 &= \phi N - \frac{\beta}{N} SI - (\psi_1 + \phi) S - \phi E - \phi I \\ 0 &= \frac{\beta}{N} SI - (\alpha + \psi_2) E \\ 0 &= \alpha E - (\gamma + \theta) \end{aligned}\right\}. \tag{17}$$

A simple computation shows that there are two solutions: the *disease free equilibrium point* defined by

$$E_f^* = \left(S_0^*, E_0^*, I_0^*\right) = \left(\frac{N\phi}{\phi + \psi_1}, 0, 0\right), \tag{18}$$

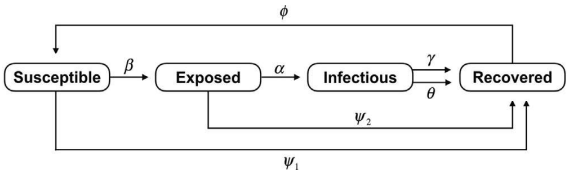

Fig. 1. Diagram of the improved model.

and the *endemic equilibrium point*, whose explicit expression is $E_e^* = \left(S_1^*, E_1^*, I_1^*, R_1^*\right)$, with:

$$S_1^* = \frac{N(\gamma+\theta)(\alpha+\psi_2)}{\alpha\beta}, \tag{19}$$

$$E_1^* = \frac{\gamma+\theta}{\alpha} I_1^*, \tag{20}$$

$$I_1^* = \frac{N(\alpha\beta\phi - (\alpha+\psi_2)(\gamma+\theta)(\phi+\psi_1))}{\beta(\alpha(\gamma+\theta+\phi)+(\gamma+\theta)(\phi+\psi_2))}. \tag{21}$$

Note that, taking into account these expressions, the number of recovered devices of the equilibrium points are:

$$R_0^* = \frac{N\psi_1}{\phi+\psi_1}, \tag{22}$$

$$R_1^* = \frac{\alpha\beta + (\alpha+\gamma+\theta)\psi_1 - \frac{\alpha\beta}{N}S_1^*}{\alpha(\gamma+\theta+\phi)+(\gamma+\theta)(\phi+\psi_2)} S_1^*. \tag{23}$$

In order to compute the *basic reproductive number* R_0, the Next Generation method [12] is applied to the system (13). Then:

$$F_E = \frac{\beta}{N} SI, F_I = 0, \tag{24}$$

$$V_E = (\alpha+\psi_2)E, V_I = (\gamma+\theta)I - \alpha E, \tag{25}$$

and consequently:

$$\begin{aligned}\left(F\cdot V^{-1}\right)_{E_f^*} &= \begin{pmatrix} \frac{\partial F_E}{\partial E} & \frac{\partial F_E}{\partial I} \\ \frac{\partial F_I}{\partial E} & \frac{\partial F_I}{\partial I} \end{pmatrix}_{E_f^*} \cdot \begin{pmatrix} \frac{\partial V_E}{\partial E} & \frac{\partial V_E}{\partial I} \\ \frac{\partial V_I}{\partial E} & \frac{\partial V_I}{\partial I} \end{pmatrix}^{-1}_{E_f^*} \\ &= \begin{pmatrix} 0 & \frac{\beta\phi}{\phi+\psi_1} \\ 0 & 0 \end{pmatrix} \cdot \begin{pmatrix} \alpha+\psi_2 & 0 \\ -\alpha & \gamma+\theta \end{pmatrix}^{-1} \\ &= \begin{pmatrix} \frac{\alpha\beta\phi}{(\gamma+\theta)(\phi+\psi_1)(\alpha+\psi_2)} & \frac{\beta\phi}{(\gamma+\theta)(\phi+\psi_1)} \\ 0 & 0 \end{pmatrix}. \end{aligned} \tag{26}$$

A simple calculus shows that the basic reproductive number is given by:

$$R_0 = \frac{\alpha\beta\phi}{(\gamma+\theta)(\phi+\psi_1)(\alpha+\psi_2)}. \tag{27}$$

4.1. Stability of the disease-free equilibrium point

The Jacobian matrix at the disease-free equilibrium point E_f^* is:

$$J\left(E_f^*\right) = \begin{pmatrix} -\phi-\psi_1 & -\phi & -\frac{\beta\phi}{\phi+\psi_1} - \phi \\ 0 & -\alpha-\psi_2 & \frac{\beta\phi}{\phi+\psi_1} \\ 0 & \alpha & -\gamma-\theta \end{pmatrix}, \tag{28}$$

and, as a simple calculus shows, their eigenvalues are the following:

$$\lambda_1 = -\phi - \psi_1, \tag{29}$$

$$\lambda_2 = -\frac{\alpha+\gamma+\theta+\psi_2}{2} - \frac{1}{2}\sqrt{(\alpha+\psi_2-\gamma-\theta)^2 + \frac{4\alpha\beta\phi}{\psi_1+\phi}}, \tag{30}$$

$$\lambda_3 = -\frac{\alpha+\gamma+\theta+\psi_2}{2} + \frac{1}{2}\sqrt{(\alpha+\psi_2-\gamma-\theta)^2 + \frac{4\alpha\beta\phi}{\psi_1+\phi}}. \tag{31}$$

Note that they are real numbers and it is easy to check that $\lambda_1 < 0$, and $\lambda_2 < 0$. Moreover, setting $A = \alpha+\psi_2$, and $B = \gamma+\theta$, it is:

$$\lambda_3 = -\frac{A+B}{2} + \frac{1}{2}\sqrt{(A-B)^2 + 4ABR_0}. \tag{32}$$

As $A > 0$ and $B > 0$ then $\lambda_3 < 0$ if and only if $(A-B)^2 + 4ABR_0 < (A+B)^2$. A simple calculus shows that this inequality holds iff $R_0 < 1$.

Consequently, the following theorem is obtained:

Theorem 4. *The disease-free equilibrium point* $E_f^* = \left(\frac{N\phi}{\phi+\psi_1}, 0, 0, \frac{N\psi_1}{\phi+\psi_1}\right)$ *is locally asymptotically stable iff* $R_0 \leq 1$.

Furthermore, the following result also holds:

Theorem 5. *The disease-free equilibrium point* E_f^* *is globally asymptotically stable if* $R_0 \leq 1$.

Proof. Let $\mathcal{L} : \Omega \subset \mathbb{R}^2 \to \mathbb{R}$ the function defined by $\mathcal{L}(E, I) = \alpha E + (\alpha+\psi_2) I$. It is a Lyapunov function if $R_0 \leq 1$ because it satisfies the following properties:

(1) $\mathcal{L}$ is a continuously differentiable function.
(2) $\mathcal{L}$ is positive definite since $\alpha > 0$, $\psi_2 > 0$ and $E \geq 0$, $I \geq 0$, and $\mathcal{L}\left(E_f^*\right) = 0$.
(3) $\dot{\mathcal{L}} \leq 0$ if $R_0 \leq 1$ since taking into account (10) and Eq. (16), we obtain:

$$\begin{aligned}
\dot{\mathcal{L}} &= \alpha\dot{E} + (\alpha+\psi_2)\dot{I} \\
&= \alpha\left[\frac{\beta}{N}SI - (\alpha+\psi_2)E\right] + (\alpha+\psi_2)[\alpha E - (\gamma+\theta)I] \\
&= \left[\frac{\alpha\beta}{N}S - (\alpha+\psi_2)(\gamma+\theta)\right]I \\
&\leq \left[\frac{\alpha\beta}{N}\frac{\phi N}{\phi+\psi_1} - (\alpha+\psi_2)(\gamma+\theta)\right]I = (R_0-1)(\alpha+\psi_2)(\gamma+\theta)I.
\end{aligned} \tag{33}$$

Then the orbital derivative of $\mathcal{L}$ is negative semidefinite if $R_0 \leq 1$ because $\alpha+\psi_2 > 0$, $\gamma+\theta > 0$, and $I \geq 0$.

It is shown that the largest invariant set in $\{(E, I) \in \Omega | \dot{\mathcal{L}}(E, I) = 0\}$ is a singleton containing the origin. Effectively, if $\dot{\mathcal{L}}(E, I) = 0$ then

$$0 = \dot{\mathcal{L}}(E, I) = \alpha\dot{E} + (\alpha+\psi_2)\dot{I} = \left[\frac{\alpha\beta}{N}S - (\alpha+\psi_2)(\gamma+\theta)\right]I, \tag{34}$$

and consequently either $S = \frac{(\alpha+\psi_2)(\gamma+\theta)N}{\alpha\beta}$ or $I = 0$. In the first case a simple calculus shows that:

$$0 = \frac{dS}{dt} = -\left[\frac{(\alpha+\psi_2)(\gamma+\theta)}{\alpha} + \phi\right]I - \phi E + \phi\left(1 - \frac{1}{R_0}\right)N < 0, \tag{35}$$

when $R_0 < 1$, which is a contradiction. On the other hand, if $I = 0$ then $0 = \frac{dI}{dt} = \alpha E$ and consequently $E = 0$, and the origin is obtained.

Moreover, from (16) $\lim_{t\to\infty}(S(t), E(t), I(t)) = E_f^*$, and applying the LaSalle's invariance principle [13], the disease-free equilibrium point is globally asymptotically stable if $R_0 \leq 1$. □

4.2. Stability of the endemic equilibrium point

Let

$$J\left(E_e^*\right)=\begin{pmatrix} -\dfrac{\phi\left(\alpha\beta+(\alpha+\gamma+\theta)\phi+(\alpha+\gamma+\theta)\psi_1\right)}{(\gamma+\theta)\phi+\alpha(\gamma+\theta+\phi)+(\gamma+\theta)\psi_2} & -\phi & -\phi-\dfrac{(\gamma+\theta)(\alpha+\psi_2)}{\alpha} \\ \dfrac{\alpha(\beta-\gamma-\theta)\phi-(\gamma+\theta)(\phi\psi_2+\psi_1(\alpha+\psi_2))}{(\gamma+\theta)\phi+\alpha(\gamma+\theta+\phi)+(\gamma+\theta)\psi_2} & -\alpha-\psi_2 & \dfrac{(\gamma+\theta)(\alpha+\psi_2)}{\alpha} \\ 0 & \alpha & -\gamma-\theta \end{pmatrix} \tag{36}$$

be the Jacobian matrix at the endemic equilibrium point. The explicit expressions of its eigenvalues are too long to be handled and, consequently, the Routh–Hurwitz criterion [14] will be used to study the local stability. In this sense, the characteristic polynomial of $J\left(E_e^*\right)$ is given by $P(x)=p_0x^3+p_1x^2+p_2x+p_3$, where:

$$\begin{aligned} p_0 &= 1, \\ p_1 &= \alpha+\gamma+\theta+\psi_2+\frac{\phi\left(\alpha\beta+\psi_1(\alpha+\gamma+\theta)+\phi(\alpha+\gamma+\theta)\right)}{\phi(\alpha+\gamma+\theta)+\alpha(\gamma+\theta)+\psi_2(\gamma+\theta)}, \\ p_2 &= \frac{\phi}{\alpha(\gamma+\theta+\phi)+\psi_2(\gamma+\theta)+\phi(\gamma+\theta)}\left[\phi\left(\alpha^2+\alpha(\beta+\gamma+\theta)+(\gamma+\theta)^2\right)\right. \\ &\quad \left.+\psi_1\left(\alpha^2+\alpha(\gamma+\theta)+(\gamma+\theta)^2\right)+\alpha\beta(\alpha+\gamma+\theta)+\alpha\psi_2\left(\beta+\psi_1+\phi\right)\right], \\ p_3 &= \alpha\phi(\beta-\gamma-\theta)-(\gamma+\theta)\left(\psi_1(\alpha+\psi_2)+\psi_2\phi\right). \end{aligned} \tag{37}$$

Note that $p_0>0$, $p_1>0$ and $p_2>0$ since the parameters of the model are positive and all terms of the expressions of p_0, p_1, p_2 are also positive. Moreover, a simple calculus shows that $p_2p_1-p_3>0$ and, consequently $p_2p_1>p_3=p_3p_0$. Finally, as $p_3=(R_0-1)(\alpha+\psi_2)(\gamma+\theta)(\psi_1+\phi)$, and $\alpha+\psi_2>0$, $\gamma+\theta>0$, $\psi_1+\phi>0$, then $p_3>0$ if and only if $R_0-1>0$. As a consequence the following result holds:

Theorem 6. *The endemic equilibrium point E_e^* is locally and asymptotically stable iff $R_0>1$.*

Now, the global stability of the endemic equilibrium point will be studied taking into account the classic geometric approach.

Lemma 1. *If $R_0>1$ the system (13) is uniformly persistent.*

Proof. Taking into account that E_f^* is unstable if $R_0>1$ and $E_f^*\in\partial\Omega$, from Theorem 4.3 of [15] it yields that the system (13) is uniformly persistent for $R_0>1$. □

This result implies the existence of a compact absorbing set in int (Ω) and, consequently, the geometric approach can be used [16]. Thus, the following result holds:

Theorem 7. *E_e^* is globally and asymptotically stable in* int (Ω) *if $R_0>1$.*

Proof. The Jacobian matrix of the system (13) is:

$$J=\begin{pmatrix} -\dfrac{\beta}{N}I-\phi-\psi_1 & -\phi & -\dfrac{\beta}{N}S-\phi \\ \dfrac{\beta}{N}I & -\alpha-\psi_2 & \dfrac{\beta}{N}S \\ 0 & \alpha & -\gamma-\theta \end{pmatrix}, \tag{38}$$

and, consequently, its second additive compound matrix is given by:

$$J^{[2]}=\begin{pmatrix} -\dfrac{\beta I+(\alpha+\phi+\psi_1+\psi_2)N}{N} & \dfrac{\beta}{N}S & \dfrac{\beta}{N}S+\phi \\ \alpha & -\dfrac{\beta I+(\gamma+\theta+\phi+\psi_1)N}{N} & -\phi \\ 0 & \dfrac{\beta}{N}I & -\alpha-\gamma-\theta-\psi_2 \end{pmatrix}. \tag{39}$$

Set

$$A = \begin{pmatrix} 1 & 0 & 0 \\ 0 & \frac{E}{I} & 0 \\ 0 & 0 & \frac{E}{I} \end{pmatrix}, \tag{40}$$

then

$$A_f A^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ 0 & -\frac{\alpha\,(E+I)}{I} + \frac{\beta SI}{NE} + \gamma + \theta - \psi_2 & 0 \\ 0 & 0 & -\frac{\alpha\,(E+I)}{I} + \frac{\beta SI}{NE} + \gamma + \theta - \psi_2 \end{pmatrix}, \tag{41}$$

where A_f stands for the directional derivative of A along (S, E, I). A simple computation yields:

$$B = A_f A^{-1} + AJ^{[2]}A^{-1} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix} \tag{42}$$

where:

$$B_{11} = -\frac{\beta I + (\alpha + \phi + \psi_1 + \psi_2)\,N}{N}, \tag{43}$$

$$B_{12} = \left(\frac{\beta SI}{NE}, \left(\frac{\beta S}{N} + \phi\right)\frac{I}{E}\right), \tag{44}$$

$$B_{21} = \begin{pmatrix} \frac{\alpha E}{I} \\ 0 \end{pmatrix}, \tag{45}$$

$$B_{22} = \begin{pmatrix} \frac{-\alpha\,(E+I)}{I} + \frac{\beta I\,(S-E)}{NE} - \phi - \psi_1 - \psi_2 & -\phi \\ \frac{\beta I}{N} & \frac{-\alpha\,(E+2I)}{I} + \frac{\beta SI}{NE} - 2\psi_2 \end{pmatrix}. \tag{46}$$

It is easy to check that

$$\mu(B) \leq \sup\left\{\mu_1(B_{11}) + \|B_{12}\|_1, \mu_1(B_{22}) + \|B_{21}\|_1\right\}, \tag{47}$$

where $\|z\| = \sup(\|z_1\|, \|z_2\| + \|z_3\|)$ with $z = (z_1, z_2, z_3) \in \mathbb{R}^3$, μ is the Lozinskil measure with respect this norm, and μ_1 is the Lozinskil measure with respect to L_1 norm.

As a consequence:

$$\mu_1(B_{11}) = -\frac{\beta I + (\alpha + \phi + \psi_1 + \psi_2)\,N}{N}, \tag{48}$$

$$\|B_{12}\|_1 = \left(\frac{\beta S}{N} + \phi\right)\frac{I}{E}, \tag{49}$$

$$\mu_1(B_{22}) = \frac{\alpha E}{I}, \tag{50}$$

$$\begin{aligned} \|B_{21}\|_1 &= \max\left\{-\frac{\alpha\,(E+I)}{I} + \frac{\beta I\,(S-E)}{NE} + \frac{\beta I}{N} - \psi_1 - \psi_2 - \phi\ ,\ -\frac{\alpha\,(E+2I)}{I} + \frac{\beta SI}{NE} - 2\psi_2 + \phi\right\} \\ &= -\alpha - \frac{\alpha E}{I} + \frac{\beta SI}{NE} - \psi_2 + \max\{-\psi_1 - \phi, -\alpha - \psi_2 + \phi\}. \end{aligned} \tag{51}$$

Furthermore, the following is obtained:

$$\mu_1(B_{11}) + \|B_{12}\|_1 = \frac{E'}{E} - \frac{I\phi}{E} - \frac{\beta I}{N} - \psi_1 - \phi, \tag{52}$$

$$\mu_1(B_{22}) + \|B_{21}\|_1 = \frac{E'}{E} + \max\{-\psi_1 - \phi, -\alpha - \psi_2 + \phi\}, \tag{53}$$

and consequently:

$$\mu(B) \leq \frac{E'}{E} + \max\{-\psi_1 - \phi, -\alpha - \psi_2 + \phi\} + \sup\left\{0, -\frac{\phi I}{E} - \frac{\beta I}{N}\right\}. \tag{54}$$

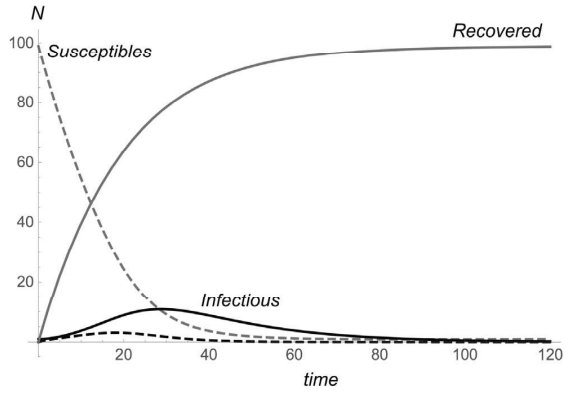

Fig. 2. Evolution of the different compartments of the model when $R_0 < 1$.

Now, suppose that:

(1) $\phi < \alpha + \psi_2$, then $\max\{-\psi_1 - \phi, -\alpha - \psi_2 + \phi\} < 0$.
(2) $\frac{\phi}{E} - \frac{\beta}{N} < 0$, then $\sup\left\{0, -\frac{\phi I}{E} - \frac{\beta I}{N}\right\} = 0$.

Therefore $\mu(B) \leq \frac{E'}{E} - \theta$ with $\theta > 0$. Thus, there exists $T > 0$ such that for $t > T$ it is $\frac{E(t)}{E(0)} < e^{\frac{\theta t}{4}}$, that is:

$$\frac{1}{t}\log\left(\frac{E(t)}{E(0)}\right) < \frac{\theta}{4}. \tag{55}$$

Then we have:

$$\frac{1}{t}\int_0^t \mu(B)\,dt < \frac{1}{t}\log\left(\frac{E(t)}{E(0)}\right) - \theta < -\frac{1}{2}\theta, \tag{56}$$

and, consequently:

$$\overline{q}_2 = \overline{\lim}_{t\to\infty} \sup_{(S(0),E(0),I(0))\in \mathrm{int}(\Omega)} \frac{1}{t}\int_0^t \mu(B)\,dt < -\frac{1}{2}\theta < 0. \tag{57}$$

As $\overline{q}_2 < 0$, E_e^* is global and asymptotically stable in int (Ω) for $R_0 > 1$. □

4.3. Numerical analysis

In this section we will perform a numerical integration for two sets of parameters that illustrate the behavior of the model depending on the value of the basic reproductive number. In both cases, it is assumed that $0 \leq t \leq 120$, and the total number of devices is $N = 100$ with $S(0) = 99$ and $I(0) = 1$. Moreover, the numeric values of the coefficients are the following: $\alpha = 0.3$, $\beta = 0.5$, $\psi_1 = 0.05$, $\psi_2 = 0.075$, $\theta = 0.003$, and $\gamma = 0.05$. In the first simulation (see Fig. 2) it is supposed that the loss of immunity coefficient is $\phi = 0.0005$, thus a simple calculus shows that $R_0 \approx 0.0747 < 1$ and the disease-free steady state is obtained.

On the other hand, in the second simulation, it is considered $\phi = 0.015$ and consequently $R_0 \approx 1.7417 > 1$. As a consequence, the system evolves to the endemic steady state (see Fig. 3).

Note that from Eq. (27), we can obtain

$$\phi = \frac{(\gamma+\theta)(\alpha+\psi_2)\psi_1}{\alpha\beta - (\gamma+\theta)(\alpha+\psi_2)}. \tag{58}$$

As a consequence the disease-free steady state is locally and globally asymptotically stable if

$$\phi \leq \frac{(\gamma+\theta)(\alpha+\psi_2)\psi_1}{\alpha\beta - (\gamma+\theta)(\alpha+\psi_2)}, \tag{59}$$

whereas the endemic steady state is locally and globally asymptotically stable if

$$\phi > \frac{(\gamma+\theta)(\alpha+\psi_2)\psi_1}{\alpha\beta - (\gamma+\theta)(\alpha+\psi_2)}. \tag{60}$$

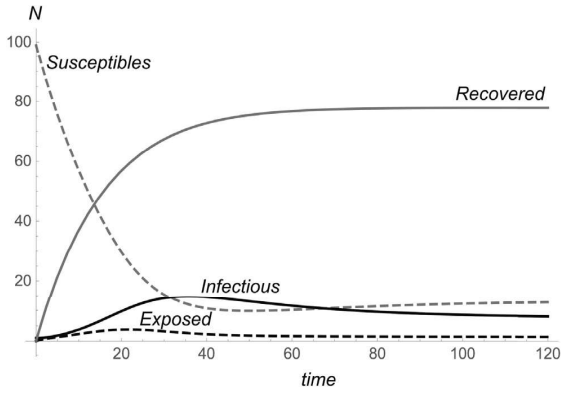

Fig. 3. Dynamic of the model when $R_0 > 1$.

Taking into account the numeric values used in the examples, the transition from the disease-free regimen to the endemic regimen occurs when $\phi = 0.0076$.

5. Control strategies

As is well-known, the basic reproductive number R_0 plays an important role in the control of an epidemic: in order to prevent that a computer worm outbreak becomes an epidemic process, it is mandatory to reduce R_0 as necessary. In our case, this threshold parameter depends on all parameters of the system: the recovery coefficients ψ_1, ψ_2 and γ, the infection rate β, the dysfunctional rate θ, the infectious rate α, and loss of immunity rate ϕ. Note that, this threshold parameter does not depend on the total number of devices N.

A simple computation shows that:

$$\frac{\partial R_0}{\partial \alpha} = \frac{\beta\psi_2\phi}{(\alpha+\psi_2)^2(\gamma+\theta)(\psi_1+\phi)} > 0, \tag{61}$$

$$\frac{\partial R_0}{\partial \beta} = \frac{\alpha\phi}{(\alpha+\psi_2)(\gamma+\theta)(\psi_1+\phi)} > 0, \tag{62}$$

$$\frac{\partial R_0}{\partial \gamma} = -\frac{\alpha\beta\phi}{(\alpha+\psi_2)(\gamma+\theta)^2(\psi_1+\phi)} < 0, \tag{63}$$

$$\frac{\partial R_0}{\partial \phi} = \frac{\alpha\beta\psi_1}{(\alpha+\psi_2)(\gamma+\theta)(\psi_1+\phi)^2} > 0, \tag{64}$$

$$\frac{\partial R_0}{\partial \theta} = -\frac{\alpha\beta\phi}{(\alpha+\psi_2)(\gamma+\theta)^2(\psi_1+\phi)} < 0, \tag{65}$$

$$\frac{\partial R_0}{\partial \psi_1} = -\frac{\alpha\beta\phi}{(\alpha+\psi_2)(\gamma+\theta)(\psi_1+\phi)^2} < 0, \tag{66}$$

$$\frac{\partial R_0}{\partial \psi_2} = -\frac{\alpha\beta\phi}{(\alpha+\psi_2)^2(\gamma+\theta)(\psi_1+\phi)} < 0. \tag{67}$$

As a consequence, if we consider all variables of R_0 constant except only one, the function R_0 decreases as the coefficients α, β, and ϕ decrease or the coefficients γ, θ, ψ_1 and ψ_2 increase. Then, to reduce the value of R_0 it is necessary to reduce the numeric value of α, β, and ϕ, or to increase the value of γ, θ, ψ_1 and ψ_2.

In short, from this analysis of the basic reproductive number, the following control measures are obtained to control the malware outbreak:

(1) Reducing the infectious rate α.
(2) Reducing the infection rate β by installing efficient anti-virus software.
(3) Reducing the loss of immunity coefficient ϕ by using efficient malware-remove software.
(4) Increasing the recovery rate γ by improving the performance of antivirus software.
(5) Increasing the recovery rates ψ_1 and ψ_2 by sensitizing users to install security countermeasures.

Furthermore, $R_0 < 1$ if and only if

$$\frac{\alpha\beta\phi}{(\gamma+\theta)(\phi+\psi_1)(\alpha+\psi_2)} < 1, \tag{68}$$

that is, the malware outbreak does not become epidemic iff

$$\alpha\beta\phi < (\gamma+\theta)(\phi+\psi_1)(\alpha+\psi_2). \tag{69}$$

Since $\alpha < \alpha+\psi_2$ and $\phi < \phi+\psi_1$, then $R_0 < 1$ if $\beta < \gamma+\theta$.

6. Conclusions

In this work, a critical analysis of the malware epidemiological model proposed by Toutonji et al. has been performed and an improved mathematical model has been introduced.

A qualitative study of the proposed new model has been done: the disease-free and the endemic equilibrium points are derived and the basic reproductive number has been computed. Moreover, the stability of the model has been stated taking into account such threshold parameter.

In our opinion, the model introduced in this work seems to be more realistic than the early one. Specifically, in the new model the vector transmission is given by the compartment of infectious devices and the population dynamic paradigm has been adapted to a more realistic situation.

From the analysis of the basic reproductive number associated to the model, the main efficient security countermeasures are presented. They include the reduction of the infectious rate, infection rate and the loss of immunity coefficient, and the increase of the recovery rates. Moreover, it is also obtained that the malware outbreak does not become epidemic if the portion of infectious devices that are recovered at every step of time is greater than the infection rate.

The basic reproductive number, R_0, associated to the improved model is greater that the basic reproductive number associated to the model by Toutonji et al., $\bar{R}_0$. Specifically, $\bar{R}_0 = (\gamma+\theta)R_0$, where obviously $\gamma+\theta \leq 1$. As a consequence, in the earliest model the threshold parameter was underestimated.

Further work aimed at improving this model by considering the propagation of computer worm over networks considering different non-linear incident rates. Moreover, a detailed study about the existence of damped oscillations must be performed.

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