

Article

Nonsmooth Modeling of Computer Virus Propagation

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Abstract

Most previous computer virus propagation (CVP) models are smooth, meaning that their right-hand sides are continuously differentiable. However, recovery resources for compromised hosts are often limited, and the aggregate recovery rate may decrease once the number of bursting nodes exceeds a defense threshold. To describe this resource-constrained mechanism, this article proposes a nonsmooth susceptible–latent–bursting–susceptible (SLBS) model with a two-level recovery function and a Holling-II saturated infection rate. Well-posedness, positivity, and positive invariance of the feasible region are first proved. The basic reproduction number is derived by the next-generation matrix method, and its normalized sensitivity indices is provided. The virus-endemic equilibria are obtained by reducing the equilibrium equations to a strictly increasing scalar equation, with special attention to the threshold case at the nonsmooth switching surface. Local stability is established by piecewise linearization and explicit Routh–Hurwitz criteria. Finally, vector-graphic numerical simulations, convergence checks, and parameter robustness tests are reported. The results clarify how limited recovery capacity and saturated infection jointly affect hierarchical control of network viruses.

Keywords: computer virus propagation (CVP) model; nonsmoothness; hierarchical recovery rate; nonlinear infection rate; basic reproduction number; equilibrium; asymptotic stability

MSC: 34D20; 34D23



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1. Introduction

A computer virus is a type of harmful, malicious software that can replicate itself and spread from one device to another [1]. Viruses may damage system data, slow computers, steal personal information, or cause system crashes. For example, XWorm 7.2 is a recently updated Remote Access Trojan that mainly targets Windows computers. Attackers spread it via fake business emails with malicious Excel attachments that exploit a software vulnerability to stealthily install the virus. XWorm 7.2 can cause severe and multifaceted losses, ranging from a full system takeover and large-scale data theft to privacy breaches and financial losses. Therefore, reducing the damage caused by computer-virus propagation is of considerable practical importance.

Computer viruses also threaten modern multimedia and data-security infrastructures. Networked cameras, mobile devices, cloud storage platforms, and content-distribution systems continuously exchange images, audio, and video. Once malware enters such systems, it can compromise the confidentiality, integrity, and availability of content, not merely cause system crashes or data corruption. Recent research on video cryptosystems shows that multimedia protection mechanisms may contain exploitable structural weaknesses if their security design is not sufficiently robust [2]. Accordingly, understanding how computer viruses spread can help clarify how limited defense resources may be allocated for multimedia content protection, privacy-preserving services, and complex network defense.

Studying the propagation mechanisms of computer viruses and formulating effective defense strategies are important ways to resist virus attacks. Because the spread of infectious diseases is mathematically similar to the spread of computer viruses, epidemic modeling ideas [3] have been widely used to develop computer virus propagation (CVP) models [4–6]. These models can be used to predict virus trends, analyze transmission mechanisms, design targeted defense strategies, and assess risk. Since the early 1990s, many CVP models have been proposed, ranging from ordinary models [7–9] and delayed models [10–12] to stochastic models [13,14] and fractional models [15]. Most existing CVP models are smooth. In nonsmooth models, however, standard linearization cannot be applied directly at switching surfaces, which increases the difficulty of equilibrium stability analysis.

Epidemic models with limited treatment resources are modified epidemic models that impose an upper bound on available medical treatment capacity. Such models are widely used to assess healthcare pressure, predict epidemic trends, and guide the optimal allocation of scarce medical resources. See [16,17] for the earliest foundational works on epidemic models with limited treatment resources. See [18–20] for recent advances in this field. Because compromised hosts are recovered under limited defensive resources, the overall recovery rate is not constant. Instead, it may decrease as the number of infected nodes increases. Hence, several CVP models with limited repair capabilities have been reported. See [21] for a CVP model with limited anti-virus ability. See [22] for a CVP model based on security classification. These CVP models are nonsmooth.

Most CVP models assume bilinear infection rates, i.e., the infection force increases linearly with the number of infected nodes [7–9]. This assumption has the following two limitations: (1) It ignores the restriction of network capacity. (2) It cannot reflect the fact that the infection growth will slow down when infected nodes reach a certain scale. Nonlinear infection rates can overcome these shortcomings by accounting for saturation and inhibition effects during transmission and by describing the slowing of infection speed as the number of infected nodes increases. Consequently, CVP models with nonlinear infection rates have received considerable attention [10–12,23,24].

One important class of CVP models is the susceptible–latent–bursting–susceptible (SLBS) model. In this class, each internal node is in one of three states: susceptible, latent, or bursting. This structure reflects the infection characteristics of many viruses by distinguishing the latent, non-destructive stage from the bursting, destructive stage. See [25,26] for traditional SLBS models and [27–29] for delayed SLBS models. By adding a recovered compartment to describe nodes with temporary immunity, SLBS models have also been extended to Susceptible–Latent–Bursting–Recovered–Susceptible (SLBRS) models [30–32]. These models are all smooth.

In this article, a nonsmooth SLBS model is established. The main feature of the model is a hierarchical recovery rate that accounts for limited antivirus resources. The model also contains a Holling-II infection rate that represents saturation in virus propagation. The remainder of this paper is organized as follows. Section 2 formulates the model. Section 3

proves the basic properties of the model. Section 4 derives the basic reproduction number and its sensitivity indices. Section 5 determines all virus-endemic equilibria. Section 6 examines local asymptotic stability by dividing the state space into smooth regions and applying linearization in each region. Section 7 validates the theoretical results through numerical experiments. Section 8 investigates the influence of different parameters on virus propagation. Section 9 concludes the article.

2. Modeling of Computer Virus Propagation

For a protected computer network, all nodes in the world may be classified into two categories: internal nodes (i.e., nodes within the network) and external nodes (i.e., nodes outside the network). Assume that each internal node is either susceptible (i.e., not infected by a computer virus) or latent (i.e., infected by a virus but not performing destructive operations) or bursting (i.e., infected by a virus and performing destructive operations). Assume that each external node is susceptible.

Let $S(t)$ denote the number of susceptible internal nodes at time t . Let $L(t)$ (resp. $B(t)$) denote the number of latent (resp. bursting) nodes at time t . For modeling purposes, the following assumptions are imposed:

- (A1) The number of internal nodes is constant.
- (A2) External nodes are connected to the network at the entrance rate $\mu > 0$.
- (A3) Each internal node is disconnected from the network at the exit rate $\delta > 0$.
- (A4) Through contact with latent nodes, each susceptible internal node becomes latent at time t at the infection rate $\frac{\beta L(t)}{1+\theta L(t)}$. Here, $\beta > 0$ is the infection force and $\theta > 0$ is the infection saturation factor.
- (A5) Each latent node becomes bursting at time t at the burst rate $\eta > 0$.
- (A6) After virus removal, each bursting node becomes susceptible at time t at the recovery rate $\gamma_1 > 0$ or $\gamma_2 > 0$ according to whether $B(t) \leq \hat{B}$ or $B(t) > \hat{B}$. Here, $\hat{B} > 0$ is the recovery threshold; $\gamma_1 > \gamma_2$, γ_1 is the first recovery rate; and γ_2 is the second recovery rate.

Let

$$h(B) = \begin{cases} \gamma_1 B & \text{if } 0 \leq B \leq \hat{B}, \\ \gamma_2 B + (\gamma_1 - \gamma_2)\hat{B} & \text{if } B > \hat{B}. \end{cases} \quad (1)$$

It is obvious that $h(B)$ is continuous.

The above assumptions lead to the three-dimensional SLBS model:

$$\begin{cases} \frac{dS(t)}{dt} = \mu - \frac{\beta S(t)L(t)}{1+\theta L(t)} + h(B(t)) - \delta S(t), \\ \frac{dL(t)}{dt} = \frac{\beta S(t)L(t)}{1+\theta L(t)} - \eta L(t) - \delta L(t), \\ \frac{dB(t)}{dt} = \eta L(t) - h(B(t)) - \delta B(t), \\ S(0), L(0), B(0) \geq 0, S(0) + L(0) + B(0) = \frac{\mu}{\delta}. \end{cases} \quad (2)$$

This model naturally reduces to the two-dimensional SLBS model:

$$\begin{cases} \frac{dL(t)}{dt} = \frac{\beta [\frac{\mu}{\delta} - L(t) - B(t)]L(t)}{1+\theta L(t)} - \eta L(t) - \delta L(t), \\ \frac{dB(t)}{dt} = \eta L(t) - h(B(t)) - \delta B(t), \\ L(0), B(0) \geq 0, L(0) + B(0) \leq \frac{\mu}{\delta}. \end{cases} \quad (3)$$

3. Basic Properties

This section establishes the well-posedness and feasible region of the model (3). Let

$$\Omega = \left\{ (L, B) \in \mathbb{R}_+^2 : L + B \leq \frac{\mu}{\delta} \right\}, \quad (4)$$

$$\Omega_1 = \{ (L, B) \in \mathbb{R}_+^2 : B < \hat{B}, L + B \leq \frac{\mu}{\delta} \}, \quad (5)$$

$$\Omega_2 = \{ (L, B) \in \mathbb{R}_+^2 : B > \hat{B}, L + B \leq \frac{\mu}{\delta} \}, \quad (6)$$

$$\Omega_3 = \{ (L, B) \in \mathbb{R}_+^2 : B = \hat{B}, L + B \leq \frac{\mu}{\delta} \}. \quad (7)$$

Then

$$\Omega = \Omega_1 \cup \Omega_2 \cup \Omega_3. \quad (8)$$

Lemma 1. For every initial condition $(L(0), B(0)) \in \mathbb{R}_+^2$, model (3) admits a unique nonnegative solution. Moreover, if $(L(0), B(0)) \in \Omega$, then $(L(t), B(t)) \in \Omega$ for all $t \geq 0$.

Proof of Lemma 1. The function $h(B)$ is continuous and globally Lipschitz on $\mathbb{R}_+$, because it is piecewise linear with slopes γ_1 and γ_2 . Since $1 + \theta L > 0$ for $L \geq 0$, the right-hand side of model (3) is locally Lipschitz on $\mathbb{R}_+^2$. Hence, the model admits a unique local solution.

Next, the vector field points inward on the boundary of $\mathbb{R}_+^2$. Indeed, if $L = 0$, then

$$\frac{dL(t)}{dt} = 0, \quad (9)$$

and if $B = 0$, then

$$\frac{dB(t)}{dt} = \eta L(t) \geq 0. \quad (10)$$

Therefore, a solution starting from $\mathbb{R}_+^2$ cannot leave $\mathbb{R}_+^2$.

It remains to prove the positive invariance of Ω . Let $Z(t) = L(t) + B(t)$. From model (3),

$$\frac{dZ(t)}{dt} = \frac{\beta[\frac{\mu}{\delta} - Z(t)]L(t)}{1 + \theta L(t)} - \delta Z(t) - h(B(t)). \quad (11)$$

On the boundary $Z(t) = \frac{\mu}{\delta}$, we have

$$\frac{dZ(t)}{dt} = -\mu - h(B(t)) \leq 0. \quad (12)$$

Consequently, no solution starting in Ω can cross the boundary $L + B = \frac{\mu}{\delta}$ outward. The solution is bounded in Ω , so the local solution extends to all $t \geq 0$. The proof is complete. $\square$

Lemma 1 shows that the model (3) is mathematically and computationally meaningful: latent and bursting node numbers remain nonnegative, and the total number of infected internal nodes cannot exceed the limiting network size μ/δ when the initial condition is feasible. In particular, the model always admits the virus-free equilibrium $E_0 = (0, 0)$.

4. Basic Reproduction Number

Theorem 1. Let R_0 denote the basic reproduction number of model (3). Then,

$$R_0 = \frac{\beta\mu}{\delta(\eta + \delta)}. \quad (13)$$

Proof of Theorem 1. The infected compartments are the latent compartment L and the bursting compartment B . New infections enter only the latent compartment because assumption (A4) states that susceptible nodes become latent after effective contact with latent nodes. Thus, near $E_0 = (0, 0)$,

$$\mathcal{F}(L, B) = \begin{bmatrix} \frac{\beta(\frac{\mu}{\delta} - L - B)L}{1 + \theta L} \\ 0 \end{bmatrix}. \quad (14)$$

All other transfers are collected in $\mathcal{V} = \mathcal{V}^- - \mathcal{V}^+$. Since $E_0 \in \Omega_1$ and $h(B) = \gamma_1 B$ in a neighborhood of E_0 ,

$$\mathcal{V}(L, B) = \begin{bmatrix} (\eta + \delta)L \\ (\gamma_1 + \delta)B - \eta L \end{bmatrix}. \quad (15)$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at E_0 are

$$F = \begin{bmatrix} \frac{\beta\mu}{\delta} & 0 \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \eta + \delta & 0 \\ -\eta & \gamma_1 + \delta \end{bmatrix}. \quad (16)$$

Therefore,

$$FV^{-1} = \begin{bmatrix} \frac{\beta\mu}{\delta(\eta + \delta)} & 0 \\ 0 & 0 \end{bmatrix}. \quad (17)$$

By the next-generation matrix method [33,34], the basic reproduction number is the spectral radius of FV^{-1} . Hence,

$$R_0 = \rho(FV^{-1}) = \frac{\beta\mu}{\delta(\eta + \delta)}. \quad (18)$$

The proof is complete. $\square$

To quantify the dependence of R_0 on the model parameters, define the normalized forward sensitivity index

$$Y_p^{R_0} = \frac{p}{R_0} \frac{\partial R_0}{\partial p}. \quad (19)$$

The sensitivity indices are listed in Table 1. The threshold parameter $\hat{B}$, the infection saturation factor θ , and the recovery rates γ_1, γ_2 do not appear directly in R_0 because R_0 counts secondary latent infections generated near the virus-free equilibrium, before bursting-node recovery affects transmission. These parameters still influence endemic equilibria and transient dynamics.

Table 1. Normalized forward sensitivity indices of R_0 .

Parameter	Meaning	$Y_p^{R_0}$
μ	entrance rate	1
β	infection force	1
δ	exit rate	$-1 - \frac{\delta}{\eta + \delta}$
η	burst rate	$-\frac{\eta}{\eta + \delta}$
θ	infection saturation factor	0
$\hat{B}$	recovery threshold	0
γ_1	first recovery rate	0
γ_2	second recovery rate	0

5. Virus-Endemic Equilibria

An equilibrium of model (3) is virus-endemic if $L > 0$ or $B > 0$. The possible endemic types are latency-free burst-endemic, latency-endemic burst-free, and latency-endemic burst-endemic. The following two results exclude the first two possibilities.

Theorem 2. *The model (3) admits no latency-free burst-endemic equilibrium.*

Proof of Theorem 2. Assume that $E^* = (L^*, B^*)$ is latency-free burst-endemic. Then $L^* = 0$ and $B^* > 0$. The second equation of (3) gives

$$h(B^*) + \delta B^* = 0. \quad (20)$$

If $B^* \leq \widehat{B}$, then $(\gamma_1 + \delta)B^* = 0$, which contradicts $B^* > 0$. If $B^* > \widehat{B}$, then

$$(\gamma_2 + \delta)B^* + (\gamma_1 - \gamma_2)\widehat{B} = 0, \quad (21)$$

which is impossible because all terms are positive. The proof is complete. $\square$

Theorem 3. *The model (3) admits no latency-endemic burst-free equilibrium.*

Proof of Theorem 3. Assume that $E^* = (L^*, B^*)$ is latency-endemic burst-free. Then $L^* > 0$ and $B^* = 0$. The second equation of (3) gives $\eta L^* = 0$, which contradicts $L^* > 0$. The proof is complete. $\square$

Thus, every virus-endemic equilibrium must be latency-endemic and burst-endemic.

Theorem 4. *If $R_0 \leq 1$, then the model (3) admits no virus-endemic equilibrium.*

Proof of Theorem 4. Assume that $E^* = (L^*, B^*)$ is a virus-endemic equilibrium. Then $L^* > 0$ and $B^* > 0$. From the first equation of (3),

$$\frac{\beta(\frac{\mu}{\delta} - L^* - B^*)}{1 + \theta L^*} = \eta + \delta. \quad (22)$$

This implies

$$\frac{\beta\mu}{\delta} > \eta + \delta, \quad (23)$$

and hence $R_0 > 1$, which contradicts $R_0 \leq 1$. The proof is complete. $\square$

Theorem 5. *Suppose $R_0 > 1$. Let*

$$a = \beta + (\eta + \delta)\theta, \quad (24)$$

$$b = \beta\eta + \delta[\beta + (\eta + \delta)\theta], \quad (25)$$

$$c = \eta \left[\frac{\beta\mu}{\delta} - (\eta + \delta) \right] = \eta(\eta + \delta)(R_0 - 1). \quad (26)$$

The model (3) admits exactly one virus-endemic equilibrium. More precisely:

(A1) *If $\frac{c}{a\gamma_1 + b} \leq \widehat{B}$, then the unique virus-endemic equilibrium is*

$$E_1 = (L_1, B_1), \quad (27)$$

where

$$L_1 = \frac{(\gamma_1 + \delta)c}{\eta(a\gamma_1 + b)}, \quad B_1 = \frac{c}{a\gamma_1 + b}. \quad (28)$$

(A2) If $\frac{c}{a\gamma_1 + b} > \widehat{B}$, then the unique virus-endemic equilibrium is

$$E_2 = (L_2, B_2), \quad (29)$$

where

$$L_2 = \frac{c(\gamma_2 + \delta) + (b - a\delta)(\gamma_1 - \gamma_2)\widehat{B}}{\eta(a\gamma_2 + b)}, \quad B_2 = \frac{c - a(\gamma_1 - \gamma_2)\widehat{B}}{a\gamma_2 + b}. \quad (30)$$

Proof of Theorem 5. Let $E^* = (L^*, B^*)$ be a virus-endemic equilibrium. Then $L^* > 0$ and $B^* > 0$. The equilibrium equations give

$$L^* = \frac{\left[\frac{\beta\mu}{\delta} - (\eta + \delta)\right] - \beta B^*}{\beta + (\eta + \delta)\theta} \quad (31)$$

and

$$L^* = \frac{h(B^*) + \delta B^*}{\eta}. \quad (32)$$

Eliminating L^* yields the scalar equation

$$G(B^*) \equiv ah(B^*) + bB^* - c = 0. \quad (33)$$

Because $R_0 > 1$, we have $c > 0$ and hence $G(0) = -c < 0$. Furthermore, $G(B) \rightarrow \infty$ as $B \rightarrow \infty$.

It remains to address the nonsmooth threshold $B = \widehat{B}$. The recovery function is continuous at the threshold because

$$h(\widehat{B}^-) = \gamma_1 \widehat{B} = \gamma_2 \widehat{B} + (\gamma_1 - \gamma_2)\widehat{B} = h(\widehat{B}^+). \quad (34)$$

Consequently, $G(B)$ is continuous on $\mathbb{R}_+$. Moreover, G is strictly increasing, with slope $a\gamma_1 + b > 0$ on $0 \leq B < \widehat{B}$ and slope $a\gamma_2 + b > 0$ on $B > \widehat{B}$. Therefore, $G(B) = 0$ has exactly one positive root.

If the root lies in $0 < B \leq \widehat{B}$, then $h(B) = \gamma_1 B$ and

$$B^* = \frac{c}{a\gamma_1 + b} = B_1, \quad L^* = \frac{(\gamma_1 + \delta)B_1}{\eta} = L_1. \quad (35)$$

The condition $B_1 \leq \widehat{B}$ is exactly $\frac{c}{a\gamma_1 + b} \leq \widehat{B}$. The equality case corresponds to an endemic equilibrium located on the switching surface $B = \widehat{B}$.

If $\frac{c}{a\gamma_1 + b} > \widehat{B}$, then $G(\widehat{B}) < 0$, and the unique root lies in $B > \widehat{B}$. In this region, $h(B) = \gamma_2 B + (\gamma_1 - \gamma_2)\widehat{B}$, which gives

$$B^* = \frac{c - a(\gamma_1 - \gamma_2)\widehat{B}}{a\gamma_2 + b} = B_2 \quad (36)$$

and

$$L^* = \frac{(\gamma_2 + \delta)B_2 + (\gamma_1 - \gamma_2)\widehat{B}}{\eta} = L_2. \quad (37)$$

The proof is complete. $\square$

6. Local Asymptotic Stability

On Ω_1 and Ω_2 , the recovery function is differentiable. Therefore, equilibria located strictly inside these two regions can be studied by standard linearization. The threshold case $B = \widehat{B}$ is covered in the existence-uniqueness analysis above; its stability requires one-sided nonsmooth analysis and is not needed for the generic strict cases considered below.

6.1. The Virus-Free Equilibrium E_0

Theorem 6. *The following assertions hold.*

(A1) *If $R_0 < 1$, then E_0 is locally asymptotically stable.*

(A2) *If $R_0 > 1$, then E_0 is unstable.*

Proof of Theorem 6. Because $E_0 \in \Omega_1$, the Jacobian matrix of (3) at E_0 is

$$J_0 = \begin{bmatrix} (\eta + \delta)(R_0 - 1) & 0 \\ \eta & -(\gamma_1 + \delta) \end{bmatrix}. \quad (38)$$

Its eigenvalues are

$$\lambda_1 = (\eta + \delta)(R_0 - 1), \quad \lambda_2 = -(\gamma_1 + \delta) < 0. \quad (39)$$

The assertion follows from Lyapunov's indirect method [35]. $\square$

6.2. The Virus-Endemic Equilibrium E_1

Suppose $R_0 > 1$ and $\frac{c}{a\gamma_1 + b} < \widehat{B}$. Then $E_1 = (L_1, B_1) \in \Omega_1$. The Jacobian matrix of (3) at E_1 is

$$J_1 = \begin{bmatrix} M_1 & -M_2 \\ \eta & -(\gamma_1 + \delta) \end{bmatrix}, \quad (40)$$

where

$$M_1 = \frac{\beta(\frac{\mu}{\delta} - 2L_1 - B_1 - \theta L_1^2)}{(1 + \theta L_1)^2} - (\eta + \delta), \quad (41)$$

and

$$M_2 = \frac{\beta L_1}{1 + \theta L_1} > 0. \quad (42)$$

The characteristic polynomial is

$$\lambda^2 + p_1\lambda + q_1, \quad (43)$$

with the explicit Routh–Hurwitz coefficients

$$p_1 = \gamma_1 + \delta - M_1, \quad (44)$$

$$q_1 = \eta M_2 - (\gamma_1 + \delta)M_1. \quad (45)$$

Thus, the two-dimensional Routh–Hurwitz criterion states that E_1 is locally asymptotically stable if and only if $p_1 > 0$ and $q_1 > 0$.

Theorem 7. *If $R_0 > 1$ and $\frac{c}{a\gamma_1 + b} < \widehat{B}$, then the virus-endemic equilibrium E_1 is locally asymptotically stable.*

Proof of Theorem 7. At E_1 , the first equilibrium equation implies

$$\frac{\beta(\frac{\mu}{\delta} - L_1 - B_1)}{1 + \theta L_1} = \eta + \delta. \quad (46)$$

Substituting this identity into M_1 gives

$$M_1 = -\frac{\beta L_1 [1 + \theta(\frac{\mu}{\delta} - B_1)]}{(1 + \theta L_1)^2} < 0. \quad (47)$$

Since $M_2 > 0$, it follows that

$$p_1 = \gamma_1 + \delta - M_1 > 0, \quad (48)$$

and

$$q_1 = \eta M_2 - (\gamma_1 + \delta)M_1 > 0. \quad (49)$$

Therefore, both roots of the characteristic polynomial have negative real parts by the Routh–Hurwitz criterion. The proof is complete. $\square$

6.3. The Virus-Endemic Equilibrium E_2

Suppose $R_0 > 1$ and $\frac{c}{a\gamma_1+b} > \widehat{B}$. Then $E_2 = (L_2, B_2) \in \Omega_2$. The Jacobian matrix of (3) at E_2 is

$$J_2 = \begin{bmatrix} M_3 & -M_4 \\ \eta & -(\gamma_2 + \delta) \end{bmatrix}, \quad (50)$$

where

$$M_3 = \frac{\beta(\frac{\mu}{\delta} - 2L_2 - B_2 - \theta L_2^2)}{(1 + \theta L_2)^2} - (\eta + \delta), \quad (51)$$

and

$$M_4 = \frac{\beta L_2}{1 + \theta L_2} > 0. \quad (52)$$

The characteristic polynomial is

$$\lambda^2 + p_2\lambda + q_2, \quad (53)$$

where

$$p_2 = \gamma_2 + \delta - M_3, \quad (54)$$

$$q_2 = \eta M_4 - (\gamma_2 + \delta)M_3. \quad (55)$$

The explicit Routh–Hurwitz stability conditions are $p_2 > 0$ and $q_2 > 0$.

Theorem 8. *If $R_0 > 1$ and $\frac{c}{a\gamma_1+b} > \widehat{B}$, then the virus-endemic equilibrium E_2 is locally asymptotically stable.*

Proof of Theorem 8. At E_2 , the first equilibrium equation implies

$$\frac{\beta(\frac{\mu}{\delta} - L_2 - B_2)}{1 + \theta L_2} = \eta + \delta. \quad (56)$$

Substituting this identity into M_3 gives

$$M_3 = -\frac{\beta L_2 [1 + \theta(\frac{\mu}{\delta} - B_2)]}{(1 + \theta L_2)^2} < 0. \quad (57)$$

Since $M_4 > 0$, we obtain

$$p_2 = \gamma_2 + \delta - M_3 > 0, \quad (58)$$

and

$$q_2 = \eta M_4 - (\gamma_2 + \delta)M_3 > 0. \quad (59)$$

Thus, both eigenvalues of J_2 have negative real parts. The proof is complete. $\square$

7. Numerical Simulations

This section verifies the analytical results and explains the numerical parameter choices. The parameters are non-dimensionalized rates per unit time. They are illustrative settings rather than empirical estimates fitted to one particular malware trace. Specifically, they are selected to cover the analytically distinct threshold regimes $R_0 < 1$, $R_0 > 1$ with $E_1 \in \Omega_1$, and $R_0 > 1$ with $E_2 \in \Omega_2$. Their network-security meanings are summarized in Table 2. The simulation code used to generate all figures and numerical tests is provided as Supplementary File S1. See Table 3 for the generated parameters sets.

Table 2. Interpretation of model parameters used in the numerical simulations.

Parameter	Name	Interpretation in a Real Network Context
μ	entrance rate	Average rate at which susceptible external nodes connect to the protected network.
δ	exit rate	Average disconnection or removal rate of internal nodes.
$\widehat{B}$	recovery threshold	Bursting-node level at which the defense system switches from high to constrained recovery capacity.
β	infection force	Effective contact rate from latent nodes to susceptible nodes before saturation.
θ	infection factor	Saturation strength caused by limited bandwidth, contact opportunities, or congestion.
η	burst rate	Rate at which latent infected nodes become bursting/destructive nodes.
γ_1	first recovery rate	Per-node recovery rate when recovery resources are sufficient, i.e., $B \leq \widehat{B}$.
γ_2	second recovery rate	Per-node recovery rate when recovery resources are constrained, i.e., $B > \widehat{B}$, with $0 < \gamma_2 < \gamma_1$.

Table 3. Parameter sets and theoretical predictions for Figures 1–6.

Exp.	μ	δ	$\widehat{B}$	β	θ	η	γ_1	γ_2	Prediction
1	50	1	10	0.015	0.6	0.1	0.06	0.002	$R_0 = 0.682 < 1$; E_0 is locally asymptotically stable.
2	100	1	10	0.1	0.6	1	0.06	0.02	$R_0 = 5.000 > 1$; E_0 is unstable and the endemic equilibrium is $E_1 = (5.737, 5.413)$.
3	200	1	10	0.1	0.6	1	0.6	0.2	$R_0 = 10.000 > 1$; $E_1 = (13.211, 8.257)$ is locally asymptotically stable.
4	40	0.9	10	0.05	0.6	0.9	0.06	0.002	$R_0 = 1.235 > 1$; $E_1 = (0.359, 0.336)$ is locally asymptotically stable.
5	50	0.5	10	0.5	0.06	0.5	0.06	0.02	$R_0 = 50.000 > 1$; $E_2 = (47.450, 44.856)$ is locally asymptotically stable.
6	40	1	10	0.2	0.05	0.8	0.08	0.01	$R_0 = 4.444 > 1$; $E_2 = (14.136, 10.503)$ is locally asymptotically stable.

All trajectories are computed with an adaptive Runge–Kutta solver with relative tolerance 10^{-9} and absolute tolerance 10^{-11} . To check numerical convergence, we repeat the computations with relative tolerance 10^{-12} and absolute tolerance 10^{-14} and compare the two solutions on the same time grid. For robustness, each parameter set is perturbed independently by $\pm 5\%$ for 200 random trials. All figures are regenerated as vector PDF graphics with enlarged labels, non-overlapping legends, distinguishable colors and line styles, and LaTeX/Palatino figure fonts consistent with the manuscript body text. See Table 4 for the convergence and robustness checks.

Table 4. Convergence and robustness checks. Here $z(t) = (L(t), B(t))$, $T = 200$, and E is the attracting equilibrium predicted by the theory.

Exp.	$\max_t \ z_{10^{-9}}(t) - z_{10^{-12}}(t)\ $	$\ z(T) - E\ $	$\pm 5\%$ Robustness Outcome in 200 Trials
1	6.22×10^{-10}	6.64×10^{-12}	200 cases remained in the $R_0 < 1$ regime and converged to E_0 .
2	1.34×10^{-8}	3.33×10^{-10}	200 cases remained in the $R_0 > 1$ endemic regime with equilibrium E_1 .
3	1.83×10^{-8}	6.58×10^{-10}	198 cases had endemic equilibrium E_1 and 2 cases crossed to E_2 ; all remained locally stable endemic cases.
4	5.90×10^{-10}	3.08×10^{-11}	200 cases remained in the $R_0 > 1$ endemic regime with equilibrium E_1 .
5	8.07×10^{-8}	7.00×10^{-9}	200 cases remained in the $R_0 > 1$ endemic regime with equilibrium E_2 .
6	3.55×10^{-7}	2.09×10^{-9}	145 cases had endemic equilibrium E_2 and 55 cases crossed to E_1 ; all remained locally stable endemic cases.

Figures 1–6 show the corresponding phase portraits. Here, each coloured line represents a phase trajectory, and each symbol (circle, square, triangle) represents an initial condition. Figure 1 confirms that trajectories converge to E_0 when $R_0 < 1$. Figures 2–6 confirm that trajectories move away from the virus-free state when $R_0 > 1$ and approach the unique endemic equilibrium predicted by Theorems 5, 7, and 8. See Table 3 for the predictions.

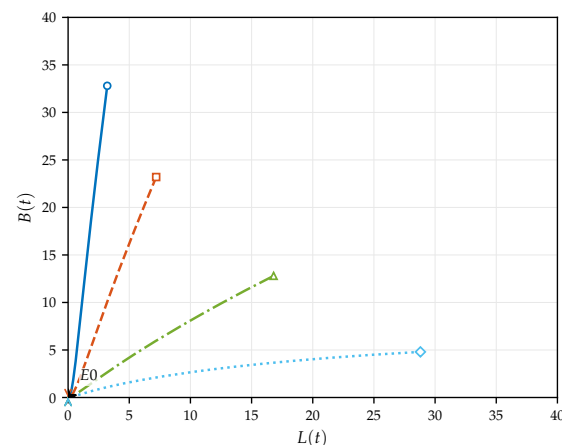


Figure 1. Phase portrait for Experiment 1, showing convergence to the virus-free equilibrium E_0 for the parameter set in Table 3.

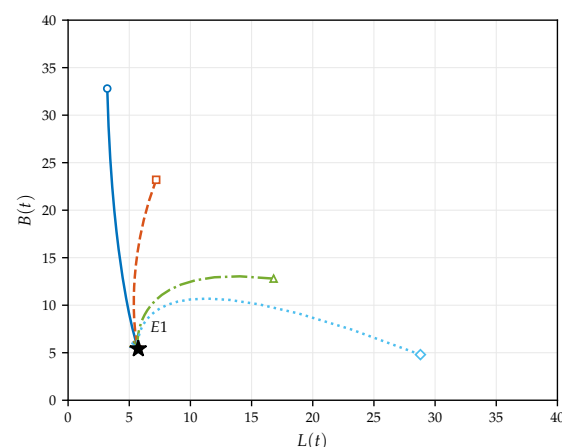


Figure 2. Phase portrait for Experiment 2, showing convergence to the endemic equilibrium E_1 for the parameter set in Table 3.

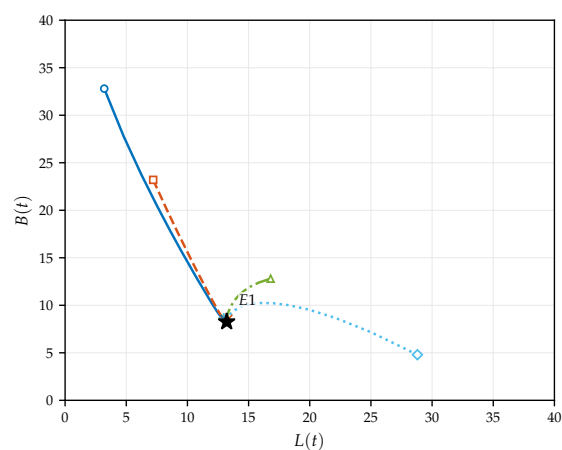


Figure 3. Phase portrait for Experiment 3, showing convergence to the endemic equilibrium E_1 for the parameter set in Table 3.

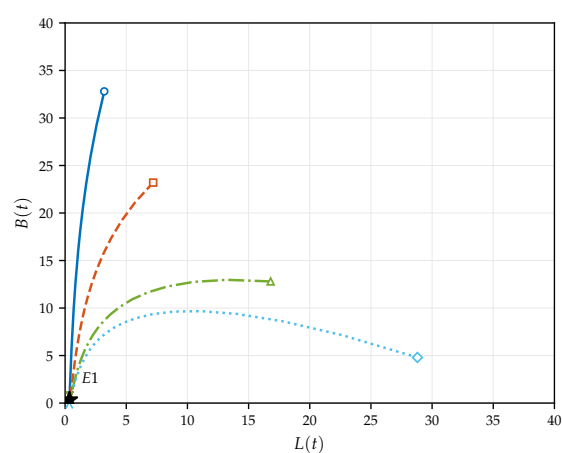


Figure 4. Phase portrait for Experiment 4, showing convergence to the endemic equilibrium E_1 for the parameter set in Table 3.

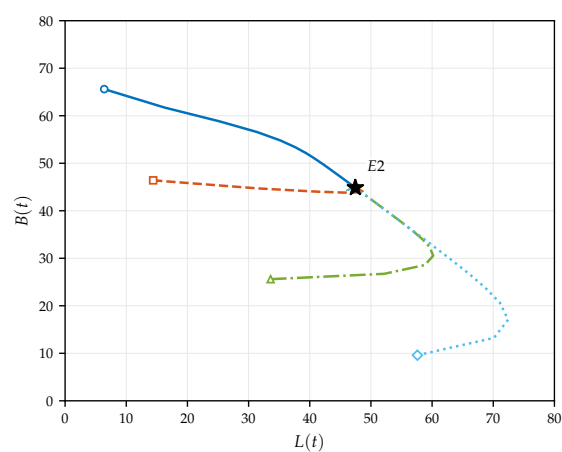


Figure 5. Phase portrait for Experiment 5, showing convergence to the endemic equilibrium E_2 for the parameter set in Table 3.

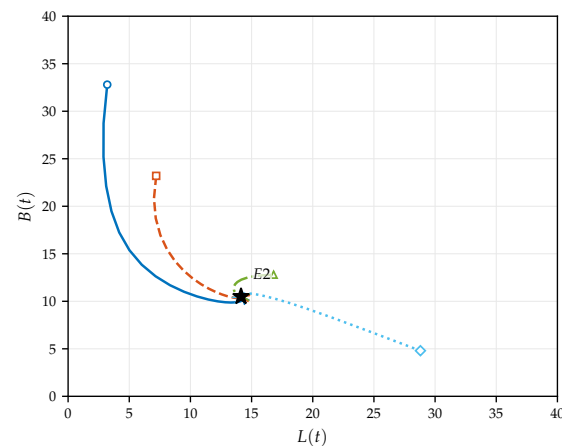


Figure 6. Phase portrait for Experiment 6, showing convergence to the endemic equilibrium E_2 for the parameter set in Table 3.

8. Influence of Different Model Parameters on Virus Propagation

Model (3) involves eight parameters. The sensitivity analysis in Section 4 explains the threshold dependence of R_0 , while this section illustrates how each parameter affects the long-time numbers of latent and bursting nodes. Unless otherwise stated, the initial condition is $(L(0), B(0)) = (2, 1)$, and all simulations use the same numerical solver as in Section 7. In Figures 7–14, each figure contains two full-trajectory panels, one for $L(t)$ and one for $B(t)$. Each panel includes a small inset that magnifies the late-time regime where several curves lie close together, while the main axis remains complete.

8.1. Entrance Rate

Consider the models (3) with $\mu \in \{20, 40, 60, 80, 100\}$, $\delta = 0.5$, $\hat{B} = 10$, $\beta = 0.5$, $\theta = 0.06$, $\eta = 0.5$, $\gamma_1 = 0.06$, and $\gamma_2 = 0.02$. Figure 7 shows that both $L(t)$ and $B(t)$ approach steady states, and these steady states increase as μ increases. Thus, a larger entrance rate facilitates both latent and bursting virus propagation.

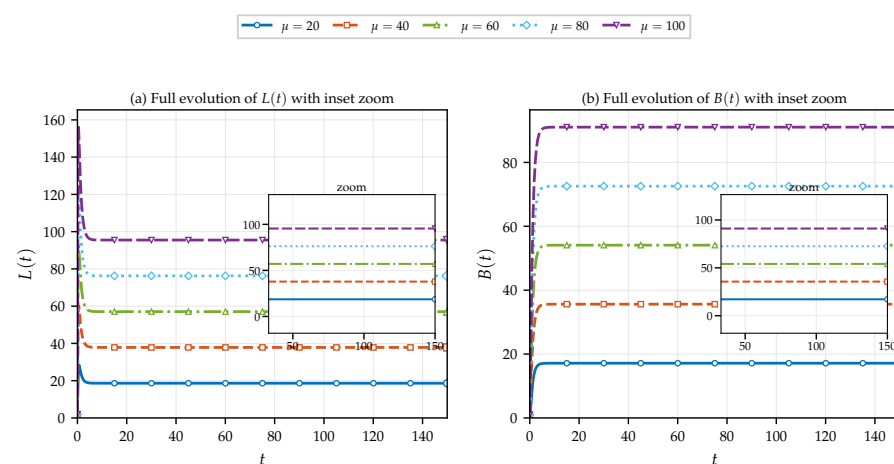


Figure 7. Time-series response to variations in the entrance rate μ : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.2. Exit Rate

Consider the models (3) with $\mu = 50$, $\delta \in \{0.2, 0.4, 0.6, 0.8, 1.0\}$, $\hat{B} = 10$, $\beta = 0.5$, $\theta = 0.06$, $\eta = 0.5$, $\gamma_1 = 0.06$, and $\gamma_2 = 0.02$. Figure 8 shows that the steady states of $L(t)$

and $B(t)$ decrease as δ increases. Therefore, increasing the exit rate inhibits both latent and bursting virus propagation.

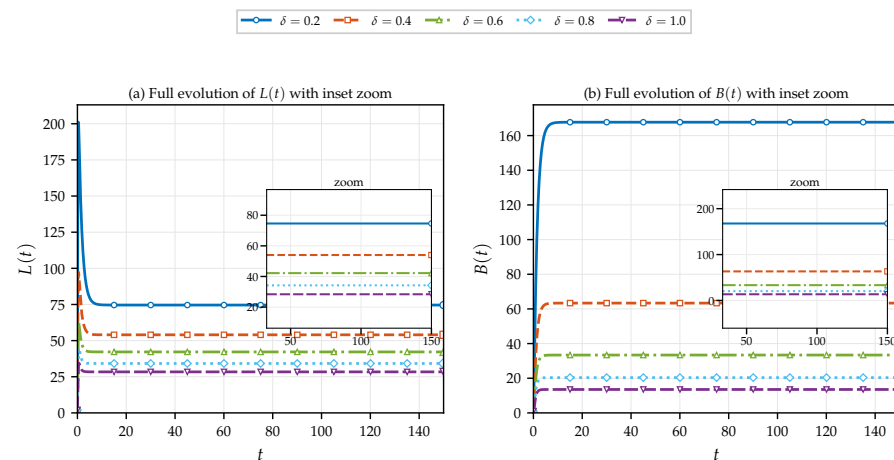


Figure 8. Time-series response to variations in the exit rate δ : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.3. Recovery Threshold

Consider the models (3) with $\mu = 50$, $\delta = 0.5$, $\hat{B} \in \{10, 20, 30, 40, 50\}$, $\beta = 0.5$, $\theta = 0.06$, $\eta = 0.5$, $\gamma_1 = 0.06$, and $\gamma_2 = 0.02$. Figure 9 shows that increasing $\hat{B}$ raises the latent steady state and lowers the bursting steady state. This reflects the fact that a higher threshold allows the high-recovery regime to remain active over a larger range of bursting infections.

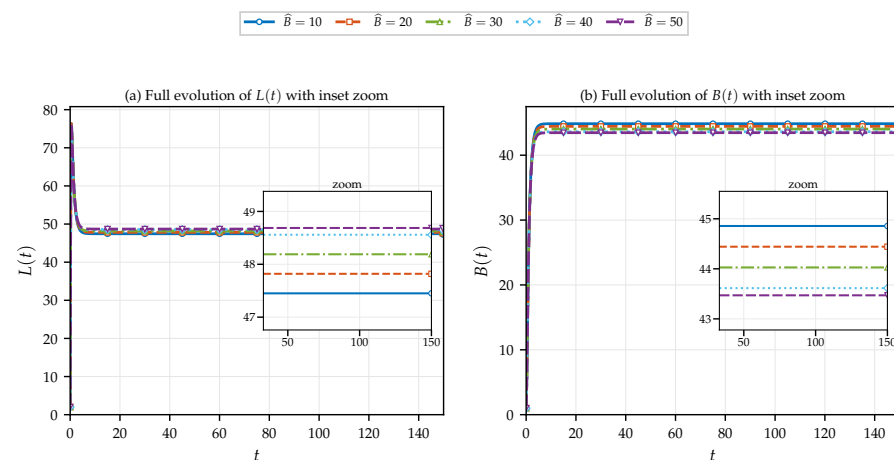


Figure 9. Time-series response to variations in the recovery threshold $\hat{B}$: (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.4. Infection Force

Consider the models (3) with $\mu = 50$, $\delta = 0.5$, $\hat{B} = 10$, $\beta \in \{0.2, 0.4, 0.6, 0.8, 1.0\}$, $\theta = 0.06$, $\eta = 0.5$, $\gamma_1 = 0.06$, and $\gamma_2 = 0.02$. Figure 10 shows that increasing β increases both steady-state components. Hence, a larger infection force facilitates propagation in both infected compartments.

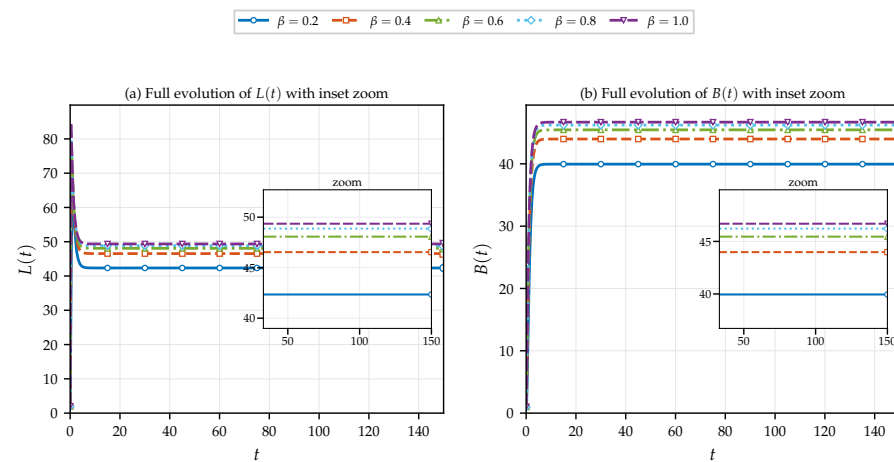


Figure 10. Time-series response to variations in the infection rate β : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.5. Infection Saturation Factor

Consider the models (3) with $\mu = 50$, $\delta = 0.5$, $\hat{B} = 10$, $\beta = 0.5$, $\theta \in \{0.02, 0.04, 0.06, 0.08, 0.10\}$, $\eta = 0.5$, $\gamma_1 = 0.06$, and $\gamma_2 = 0.02$. Figure 11 shows that increasing θ decreases both steady-state components. Thus, stronger saturation in the infection process inhibits propagation.

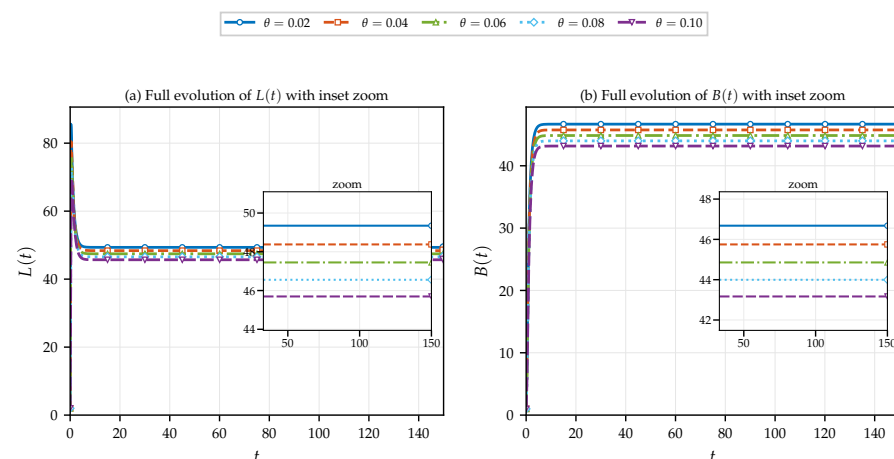


Figure 11. Time-series response to variations in the infection factor θ : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.6. Burst Rate

Consider the models (3) with $\mu = 200$, $\delta = 1$, $\hat{B} = 10$, $\beta = 0.1$, $\theta = 0.6$, $\eta \in \{0.2, 0.4, 0.6, 0.8, 1.0\}$, $\gamma_1 = 0.6$, and $\gamma_2 = 0.2$. Figure 12 shows that increasing η lowers the latent steady state and raises the bursting steady state. Hence, a larger burst rate accelerates the transition from the latent stage to the bursting stage.

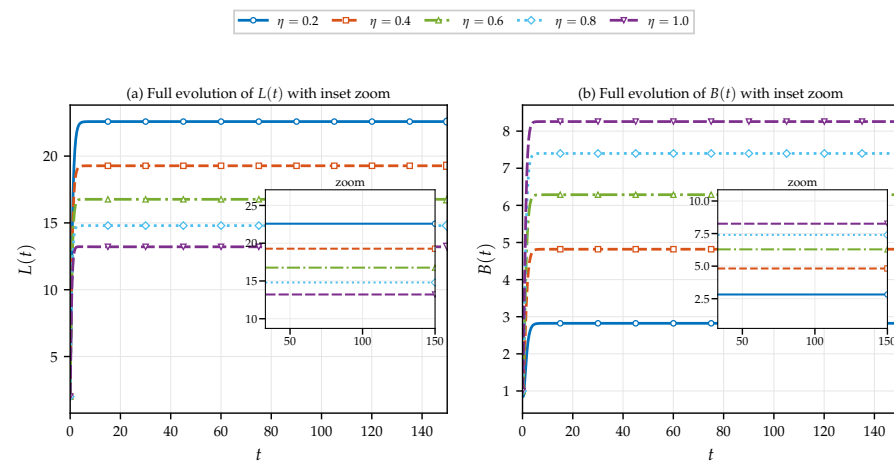


Figure 12. Time-series response to variations in the burst rate η : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.7. First Recovery Rate

Consider the models (3) with $\mu = 40$, $\delta = 0.5$, $\hat{B} = 10$, $\beta = 0.5$, $\theta = 0.06$, $\eta = 0.5$, $\gamma_1 \in \{0.04, 0.06, 0.08, 0.10, 0.12\}$, and $\gamma_2 = 0.02$. Figure 13 shows that increasing γ_1 reduces the bursting steady state. The latent component may increase because faster recovery from the bursting state returns nodes to susceptibility, which can subsequently feed the latent compartment through the infection process.

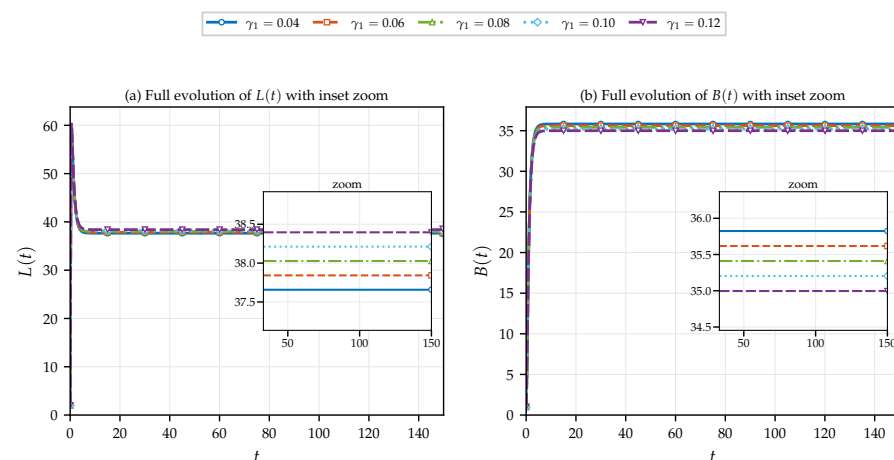


Figure 13. Time-series response to variations in the first recovery rate γ_1 : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

8.8. Second Recovery Rate

Consider the models (3) with $\mu = 40$, $\delta = 0.5$, $\hat{B} = 10$, $\beta = 0.5$, $\theta = 0.06$, $\eta = 0.5$, $\gamma_1 = 0.06$, and $\gamma_2 \in \{0.005, 0.010, 0.015, 0.020, 0.025\}$ so that $0 < \gamma_2 < \gamma_1$ is preserved. Figure 14 shows that increasing γ_2 reduces the bursting steady state in the constrained-recovery regime and correspondingly changes the latent steady state.

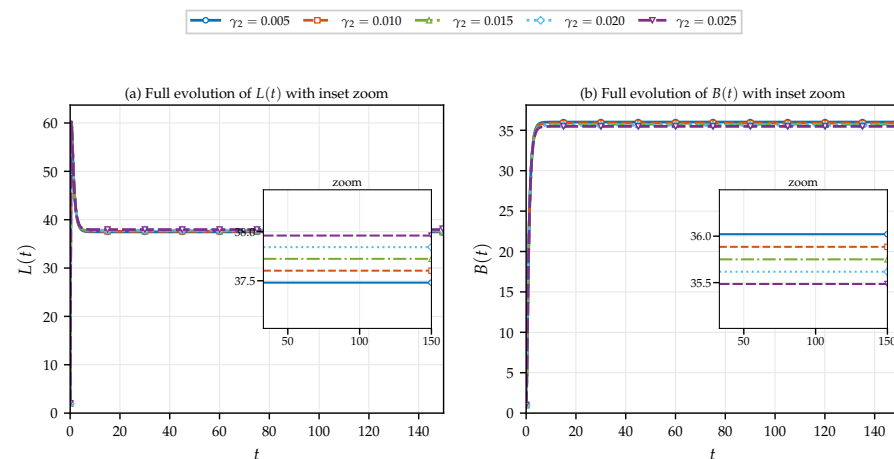


Figure 14. Time-series response to variations in the second recovery rate γ_2 : (a) full evolution of $L(t)$ with an inset zoom of the late-time region; (b) full evolution of $B(t)$ with an inset zoom of the late-time region.

9. Conclusions

In this article, we propose a nonsmooth SLBS-based computer-virus propagation model. The model combines a Holling-II saturated infection rate with a two-level recovery function that captures constrained recovery resources. We have proved positivity and positive invariance of the feasible region, derived the basic reproduction number by the next-generation matrix method, obtained a unique endemic equilibrium when $R_0 > 1$, and established local asymptotic stability by explicit Routh–Hurwitz criteria. Numerical simulations, convergence checks, and robustness tests support the theoretical conclusions and illustrate how entrance, exit, infection, saturation, bursting, and recovery parameters shape virus propagation.

The model has several limitations. First, it uses a homogeneous-mixing approximation and therefore does not explicitly represent heterogeneous degree distributions, community structures, or different security policies across subnetworks. Second, the two-level recovery function is an idealized representation of defense resources; real administrators may deploy time-varying, adaptive, or multi-level strategies. Third, sudden large-scale attacks, malware mutations, and coordinated adversarial behavior may abruptly change parameters, whereas the present model treats them as fixed during each simulation.

These limitations suggest several future directions. It is worthwhile to consider CVP models with multi-level or continuous recovery rates, more complex infection rates such as Beddington–DeAngelis and Monod–Haldane forms [11,23,36–39], network heterogeneity, adaptive defense, and optimal control [40–42]. The methodology developed here may also be extended to other nonsmooth spreading processes, such as rumor propagation with saturation and threshold-dependent intervention [43].

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/math14142664/s1>. File S1, Python script used to reproduce Figures 1–14 and the convergence/robustness checks.

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Data Availability Statement: The analytical results are included in the article. The Python v3.13 simulation script is used to reproduce Figures 1–14, and the numerical convergence and robustness checks are provided as Supplementary File S1. Further inquiries can be directed to the corresponding author.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Szor, P. *The Art of Computer Virus Research and Defense*; Addison-Wesley Professional: Boston, MA, USA, 2005.
2. Lin, Y.; Wei, Y.; Chen, D.; Li, Y.; Erkan, U.; Toktas, A.; Gao, S.; Zhang, Y. Cryptanalysis and improvement of a video cryptosystem via chaos and S-box. *ACM Trans. Multimed. Comput. Commun. Appl.* **2026**, *22*, 173.
3. Britton, N.F. *Essential Mathematical Biology*; Springer: Berlin/Heidelberg, Germany, 2003.
4. Kephart, J.O.; White, S. R. Directed epidemiological models of computer viruses. In Proceedings of 1999 IEEE Computer Society Symposium on Research in Security and Privacy, Los Alamitos, CA, USA, 20–22 May 1991; pp. 343–359.
5. Kephart, J.O.; White, S. R. Measuring and modeling computer virus prevalence. In Proceedings of 1999 IEEE Computer Society Symposium on Research in Security and Privacy, Los Alamitos, CA, USA, 24–26 May 1993; pp. 2–15.
6. Kephart, J. O.; White, S. R.; Chess, D. M. Computers and epidemiology. *IEEE Spectr.* **1993**, *30*, 20–26.
7. Toutonji, O.A.; Yoo, S.M.; Park, M. Stability analysis of VEISV propagation modeling for network worm attack. *Appl. Math. Model.* **2012**, *36*, 2751–2761.
8. Xiao, X.; Fu, P.; Dou, C.; Li, Q.; Hu, G.; Xia, S. Design and analysis of SEIQR worm propagation model in mobile internet. *Commun. Nonlinear Sci. Numer. Simul.* **2017**, *43*, 341–350.
9. Singh, A.; Awasthi, A. K.; Singh, K.; Srivastava, P. K. Modeling and analysis of worm propagation in wireless sensor networks. *Wirel. Pers. Commun.* **2018**, *98*, 2535–2551.
10. MadhuSudanan, V.; Geetha, R. Dynamics of epidemic computer virus spreading model with delays. *Wirel. Pers. Commun.* **2020**, *115*, 2047–2061.
11. Zhang, Z.; Madhusudanan, V.; Murthy, B.S.N. Effect of delay in SMS worm propagation in mobile network with saturated incidence rate. *Wirel. Pers. Commun.* **2023**, *131*, 659–678.
12. Zhang, W.; Yang, X.; Yang, L. A delayed malware propagation model under a distributed patching mechanism: Stability analysis. *Mathematics* **2025**, *13*, 2266.
13. Amador, J.; Artalejo, J. R. Stochastic modeling of computer virus spreading with warning signals. *IEEE Spectr.* **2013**, *350*, 1112–1138.
14. Khan, U.; Jamel, L.; Zubair, M. H.; Khalifa, H. E.; Aljarboa, S. Mathematical analysis of computer viruses through deterministic and stochastic models. *Comput. Secur.* **2026**, *160*, 104734.
15. Ayaz, A.; Rehamn, M. A. U.; Rafiq, M.; Iqbal, Z.; Ahmed, N.; Akgül, A.; Iqbal, M. S.; Raza, A.; Ceesay, B. Stochastic fractional order model for the computational analysis of computer virus. *Sci. Rep.* **2025**, *15*, 33951.
16. Wang, W.; Ruan, S. Bifurcation in an epidemic model with constant removal rate of the infectives. *J. Math. Anal. Appl.* **2004**, *291*, 775–793.
17. Wang, W. Backward bifurcation of an epidemic model with treatment. *Math. Biosci.* **2006**, *201*, 58–71.
18. Yang, J.; Chen, Z.; Tan, Y.; Liu, Z.; Cheke, R.A. Threshold dynamics of an age-structured infectious disease model with limited medical resources. *Math. Comput. Simul.* **2023**, *214*, 114–132.
19. Wei, W.; Xu, W.; Liu, J. A regime-switching stochastic SIR epidemic model with a saturated incidence and limited medical resources. *Int. J. Biomath.* **2023**, *16*, 2250124.
20. Hao, Y.; Luo, Y.; Teng, Z. Role of limited medical resources in an epidemic model with media report and general birth rate. *Infect. Dis. Model.* **2025**, *10*, 522–545.
21. Xu, Y.; Ren, J. Propagation effect of a virus outbreak on a network with limited anti-virus ability. *PLoS One* **2016**, *11*, e0164415.
22. Zhu, Q.; Cen, C. A novel computer virus propagation model under security classification. *Discret. Dyn. Nat. Soc.* **2017**, *2017*, 8609082.
23. Upadhyay, R.K.; Kumari, S.; Misra, A.K. Modeling the virus dynamics in computer network with SVEIR model and nonlinear incident rate. *J. Appl. Math. Comput.* **2017**, *54*, 485–509.
24. Upadhyay, R.K.; Kumari, S. Bifurcation analysis of an e-epidemic model in wireless sensor. *Int. J. Comput. Math.* **2018**, *95*, 1775–1805.
25. Yang, L. X.; Yang, X.; Wen, L.; Liu, J. A novel computer virus propagation model and its dynamics. *Int. J. Comput. Math.*, **2012**, *89*, 2307–2314.
26. Yang, L.X.; Yang, X. A new epidemic model of computer viruses. *Commun. Nonlinear Sci. Numer. Simul.* **2014**, *19*, 1935–1944.

27. Ren, J.; Xu, Y. Stability and bifurcation of a computer virus propagation model with delay and incomplete antivirus ability. *Math. Probl. Eng.* **2014**, 2014, 475934.
28. Zhao, T.; Wei, S.; Bi, D. Hopf bifurcation of a computer virus propagation model with two delays and infectivity in latent period. *Syst. Sci. Control Eng.* **2018**, 6, 90–101.
29. Zhang, Z.; Kumari, S.; Upadhyay, R.K. A delayed e-epidemic SLBS model for computer virus. *Adv. Differ. Equ.* **2019**, 2019, 414.
30. Li, C.; Liao, X. The impact of hybrid quarantine strategies and delay factor on viral prevalence in computer networks. *Math. Model. Nat. Phenom.* **2016**, 11, 105–119.
31. Zhang, Z.; Upadhyay, R.K.; Bi, D.; Wei, R. Stability and Hopf bifurcation of a delayed epidemic model of computer virus with impact of antivirus software. *Discret. Dyn. Nat. Soc.* **2018**, 2018, 8239823.
32. Ding, J.; Gul, N.; Liu, G.; Saeed, T. Dynamical aspects of a delayed computer viruses model with horizontal and vertical dissemination over internet. *Fractals* **2023**, 31, 2340092.
33. Diekmann, O.; Heesterbeek, J. A. P.; Metz, J. A. J. On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. *J. Math. Biol.* **1990**, 28, 365–382.
34. van den Driessche, P.; Watmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.* **2002**, 180, 29–48.
35. Robinson, R. *An Introduction to Dynamical Systems: Continuous and Discrete*; Pearson Education, Inc.: London, UK, 2005.
36. Zhao, T.; Zhang, Z.; Upadhyay, R.K. Delay-induced Hopf bifurcation of an SVEIR computer virus model with nonlinear incidence rate. *Adv. Differ. Equ.* **2018**, 2018, 256.
37. Chu, Y.; Xia, W.; Wang, Z. A delayed computer virus model with nonlinear incidence rate. *Syst. Sci. Control Eng.* **2019**, 7, 389–406.
38. Lu, H.; Zhu, E. A novel SIR-based computer virus propagation model with Beddington-DeAngelis functional response: Stability analysis and practical implications. *AIMS Math.* **2025**, 10, 27412–27439.
39. Liu, J.; Saeed, T.; Zeb, A. Delay effect of an e-epidemic SEIRS malware propagation model with a generalized non-monotone incidence rate. *Results Phys.* **2022**, 39, 105672.
40. Zhang, C.; Huang, H. Optimal control strategy for a novel computer virus propagation model on scale-free networks. *Physica A* **2016**, 451, 251–265.
41. Feng, L.; Han, R.; Wang, H.; Qingshan Zhao, Q.; Fu, C.; Han, Q. Virus propagation model and optimal control strategy in the point-to-group Network to information security investment. *Complexity* **2021**, 2021, 6612451.
42. Omar, O.A.M.; Ahmed, H.M.; Nofal, T.A.; Darwish, A.; Ahmed, A.M.S. Analysis and optimal control of propagation model for malware in multi-cloud environments with impact of Brownian motion process. *Math. Comput. Appl.* **2025**, 30, 8.
43. Niu, Y.; Muhammadhaji, A. Dynamics of a fractional-order IDSR rumor propagation model with time delays. *Fractal Fract.* **2025**, 9, 242.

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