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Stability and Hopf Criteria in a Malware Dissemination Model for Wireless Sensor Networks with Distributed Recovery Delays

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Abstract

A delayed malware dissemination model for wireless sensor networks is extended by replacing the single fixed anti-virus cycle time with distributed recovery memories. Two normalized gamma kernels are considered: a one-stage exponential memory (weak kernel) and a two-stage gamma memory (strong kernel), both representing heterogeneous anti-virus removal times across carrier and infectious nodes. The linear chain trick transforms the corresponding integro-differential systems into six- and eight-dimensional ordinary differential equation models. For these distributed-delay formulations, we derive the common disease-free equilibrium and its local stability conditions, prove that the endemic equilibrium and the basic reproduction number are preserved, and show that the inherited delayed-removal mechanism does not generate a positively invariant nonnegative orthant; in its place, we establish the strongest valid conditional boundedness and continuation result. Local endemic stability is reduced to a Routh–Hurwitz problem, and Hopf thresholds are obtained from simple purely imaginary roots together with transversality. For the reference parameter set, full spectral verification and first-Lyapunov-coefficient evaluation show that the first weak- and strong-kernel Hopf points are subcritical. Numerical simulations illustrate convergence below threshold and loss of equilibrium stability above it, while comparison with the discrete-delay benchmark shows that a broader recovery-time distribution postpones instability.

Keywords: malware propagation; wireless sensor networks; distributed delay; Hopf bifurcation

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1. Introduction

Wireless sensor networks (WSNs) are at the core of many emerging Internet-of-Things (IoT) deployments, from environmental monitoring and smart cities to industrial control systems, and they remain particularly exposed to malicious software because sensor nodes have severely limited power, processing capacity, transmission range, and built-in defenses. A comprehensive overview of the security issues introduced by integrating WSNs into IoT ecosystems is given in [1,2], while ref. [3] documents how vulnerable and unpatched IoT devices can be compromised and recruited into large-scale botnet attacks. The pertinence

of these concerns is reinforced by the persistent activity of Mirai-class IoT botnets, whose variants continue to recruit weakly protected devices through scanning and credential exploitation [4]. Mathematical modeling of how malware enters and propagates through a WSN therefore offers both a theoretical lens for understanding which network parameters drive containment and a practical tool for evaluating anti-virus policies.

The dominant modeling paradigm extends classical compartmental epidemiology to the digital setting. The susceptible-carrier-infectious-recovered (SCIRS) model proposed by Hernandez Guillen and Martin del Rey [5] explicitly introduces a carrier class to account for devices that transmit malware without being damaged themselves and constitutes the analytical starting point for many subsequent works. A novel revision of the SCIRS framework, with sharper biological assumptions, appears in [6], while ref. [7] proposes a Susceptible-Unexposed-Infected-Isolation-Removed structure tailored to WSN dynamics. Reviews and comparative analyses of WSN malware compartmental models can be found in [8,9]. A cluster-based variant employing game-theoretic infection and recovery rates is developed in [10], and Lévy-noise extensions are studied in [11].

Time-delay versions of compartmental WSN models have attracted sustained attention because delays naturally encode latency, scanning windows, and removal cycles. The reference model of the present paper is the delayed carrier-infectious system of Zhang, Zhang, Nisar, Gul, and Ahmed [12], in which the time delay represents the cycle time required by the anti-virus mechanism to clean carrier and infectious nodes. The same group and several closely related contributions have analyzed Hopf bifurcation, optimal control, and global exponential stability for delayed WSN malware models [13–15]. Multiple-delay and diffusion-based extensions appear in [16–18], while ref. [19] studies a ratio-dependent SEIRV model with delay awareness in IoT systems. Dual-delay industrial-control variants [20], SEI₂RS cyber-physical models with saturated incidence [21], and impulsive heterogeneous WSN models [9] all confirm that the choice of delay structure has a non-trivial effect on equilibrium stability. The role of fractional-order memory in this class of problems has been investigated in [22,23], and broader fractional-delay perspectives in epidemiology are surveyed in [24–27]. Stochastic counterparts that include random environmental fluctuations are developed in [28–30], and Turing-type spatial extensions for mobile WSNs are studied in [17,18].

A common analytical assumption in the literature above is that the cleaning time is a single deterministic number τ . While analytically convenient, this assumption is restrictive: in a real WSN, scanning, detection, cleaning, rebooting, and reconnection times vary from node to node and depend on the available power and the network topology. A more realistic representation is therefore obtained by replacing the discrete delay with a distributed memory kernel, that is, by letting the rate of removal at time t depend on a weighted average of past states. This idea is classical in mathematical biology and population dynamics [31,32] and has recently been the object of renewed methodological interest [33–38]. The most tractable family of kernels is the gamma family, because integer-shape gamma distributions can be turned into finite ordinary differential systems by the so-called linear chain trick [32,33]; this turns the original transcendental characteristic equation into an ordinary polynomial. The two simplest cases are the exponential (weak) and the two-stage gamma (strong) kernels: the weak kernel spreads the past more uniformly, while the strong kernel is more concentrated around its mean and therefore closer to a discrete delay. Distributed-delay treatments of related epidemic problems have been developed in [39–44], and Hopf-bifurcation arguments built on these representations are now standard [45,46].

The purpose of this paper is to determine how the distribution of anti-virus recovery times changes the dynamics of the delayed SCIRS malware model of [12]. The method-

ological ingredients used below, Erlang kernels, the linear chain trick, the Routh–Hurwitz criterion, and Hopf bifurcation theory, are established. The contribution of the present work is instead the following model-specific combination of results.

1. We introduce distributed memory simultaneously in the two distinct recovery channels of carrier and infectious nodes. This produces coupled six- and eight-dimensional augmented systems whose Jacobian structures and characteristic polynomials are specific to the SCIRS malware mechanism.
2. We prove that the fixed-delay, one-stage Erlang, and two-stage Erlang formulations preserve the same disease-free and endemic equilibria and the same basic reproduction number. Consequently, their comparison is performed at equal equilibrium and equal mean recovery time so that differences in stability can be attributed to the shape and dispersion of the recovery-time distribution rather than to changes in the endemic level.
3. We derive explicit local stability conditions for the disease-free equilibrium and rigorous Routh–Hurwitz/Hopf criteria for the endemic equilibrium. We also identify a structural limitation of the inherited delayed-removal law: the nonnegative orthant is not positively invariant. Rather than asserting a false positivity result, we prove the strongest valid conditional boundedness and global-continuation theorem.
4. At the first weak- and strong-kernel Hopf points, we verify the complete critical spectrum and transversality, compute the first Lyapunov coefficient, and classify both bifurcations as subcritical for the reference parameter set. The local periodic branches are then continued numerically and their instability is independently confirmed by Floquet multipliers.
5. The equal-mean comparison reveals a qualitative dispersion effect: the broader exponential memory postpones the first oscillatory instability more strongly than the more concentrated two-stage Erlang memory, which remains closer to the deterministic-delay threshold. This gives the model-specific operational conclusion that heterogeneity in node-cleaning times can smooth recovery feedback, whereas synchronized recovery can promote recurrent malware waves.

The rest of this paper is organized as follows. Section 2 recalls the fixed-delay reference model, derives its endemic equilibrium in a form convenient for the comparison with distributed delays, and writes the discrete-delay characteristic equation. Sections 3 and 4 introduce, respectively, the weak and the strong gamma extensions, prove the chain-trick equivalence in detail, and derive the linearizations and characteristic polynomials. Section 6 states and proves the stability and Hopf bifurcation criteria. Section 7 compares the fixed-delay threshold with the two distributed-memory thresholds. Sections 8 and 9 present the numerical simulations, and Sections 10 and 11 contain the discussion and the conclusion.

2. Reference Fixed-Delay Model

The reference model divides the sensor-node population into susceptible nodes $S(t)$, carrier nodes $C(t)$, infectious nodes $I(t)$, and recovered nodes $R(t)$. The delayed model is

$$\begin{cases} \frac{dS(t)}{dt} = A + \varepsilon R(t) - aI(t)S(t) - (v + \mu)S(t), \\ \frac{dC(t)}{dt} = a(1 - \delta)I(t)S(t) - \eta_1 C(t - \tau) - \mu C(t), \\ \frac{dI(t)}{dt} = a\delta I(t)S(t) - \eta_2 I(t - \tau) - \mu I(t), \\ \frac{dR(t)}{dt} = \eta_1 C(t - \tau) + \eta_2 I(t - \tau) + vS(t) - (\varepsilon + \mu)R(t). \end{cases} \quad (1)$$

Here, $\tau > 0$ is the anti-virus cycle time. Carrier and infectious nodes are removed according to their past states. All parameters are positive: A is the recruitment rate, ε the immunity-loss rate, a the contact rate, v the temporary immunity rate, μ the natural removal rate, $\delta \in (0, 1)$ the fraction of susceptibles targeted by the malware, and η_1, η_2 the carrier and infectious recovery rates.

Figure 1 provides a schematic representation of the delayed SCIRS malware model, following the compartmental-flow representation proposed in [47] and described by system (1). The infection incidence aIS is divided into the carrier and infectious pathways, with rates $a(1 - \delta)IS$ and $a\delta IS$, respectively. The recovery transfers are represented by $\eta_1 C$ and $\eta_2 I$, where $C = C(t - \tau)$ and $I = I(t - \tau)$ for the fixed-delay formulation, while they denote the corresponding gamma-distributed recovery memories in the distributed-delay models. The diagram also shows recruitment, temporary immunization, immunity loss, and natural removal from every compartment.

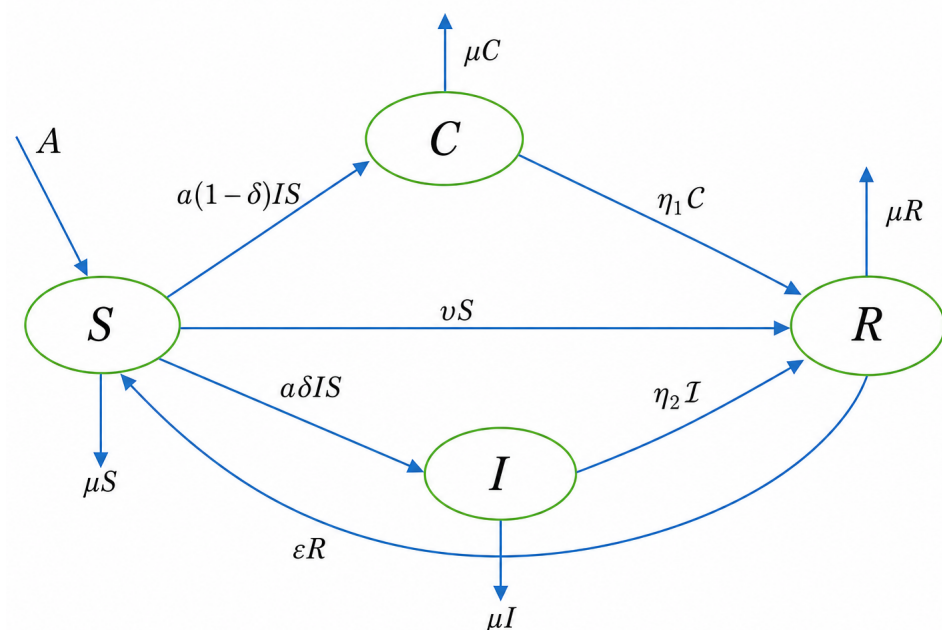


Figure 1. Schematic diagram of the SCIRS malware model. The quantities C and I represent the delayed carrier and infectious states in the fixed-delay model and the corresponding distributed recovery memories in the gamma-kernel models.

A constant equilibrium of (1) satisfies $C(t - \tau) = C(t)$ and $I(t - \tau) = I(t)$; the delay therefore cannot affect the equilibrium coordinates. Setting the right-hand sides equal to zero, the equation for dI/dt gives $I^*[a\delta S^* - (\eta_2 + \mu)] = 0$. Excluding the disease-free fixed point, the endemic equilibrium satisfies

$$S^* = \frac{\eta_2 + \mu}{a\delta},$$

which corresponds, in next-generation matrix terms, to the basic reproduction number

$$R_0 = \frac{Aa\delta(\varepsilon + \mu)}{\mu(\eta_2 + \mu)(\varepsilon + v + \mu)}. \quad (2)$$

Substituting S^* into $dC/dt = 0$ yields

$$C^* = \frac{a(1 - \delta)S^*I^*}{\eta_1 + \mu}, \quad (3)$$

and $dR/dt = 0$ yields

$$R^* = \frac{\eta_1 C^* + \eta_2 I^* + v S^*}{\varepsilon + \mu}. \quad (4)$$

To determine I^* , we use the conservation identity obtained by summing the four equations of (1) at equilibrium. The delayed terms $\eta_1 C(t - \tau)$ and $\eta_2 I(t - \tau)$ appear with opposite signs in the C/R and I/R equations, respectively, and cancel; the bilinear aIS terms also cancel because $-aIS + a(1 - \delta)IS + a\delta IS = 0$. One is left with

$$0 = A - \mu(S^* + C^* + I^* + R^*), \quad \text{hence} \quad S^* + C^* + I^* + R^* = \frac{A}{\mu}.$$

Substituting C^* and R^* from (3)–(4) into this identity, and collecting the linear terms in I^* , one finds

$$I^* = \frac{A/\mu - S^* - vS^*/(\varepsilon + \mu)}{1 + \frac{a(1 - \delta)S^*}{\eta_1 + \mu} + \frac{1}{\varepsilon + \mu} \left[\frac{\eta_1 a(1 - \delta)S^*}{\eta_1 + \mu} + \eta_2 \right]}. \quad (5)$$

The denominator in (5) is strictly positive. Moreover, since

$$A/\mu - S^* - \frac{vS^*}{\varepsilon + \mu} = \frac{S^*(\varepsilon + v + \mu)}{\varepsilon + \mu}(R_0 - 1),$$

the numerator in (5) is positive if and only if $R_0 > 1$. Hence, $R_0 > 1$ guarantees $I^* > 0$, and then (3) and (4) give $C^* > 0$ and $R^* > 0$. Formulas (3)–(5) agree, by construction, with the conservation identity $S^* + C^* + I^* + R^* = A/\mu$; they are algebraically equivalent to the closed-form expression in [12] and have the advantage that the dependence of I^* on S^* is exposed in a way that simplifies the distributed-delay comparison. Set $s = S - S^*$, $c = C - C^*$, $i = I - I^*$, and $r = R - R^*$. A direct computation of the Jacobian of (1) at $E^* = (S^*, C^*, I^*, R^*)$ splits into the current-state part B_0 and the delayed-state part B_1 :

$$B_0 = \begin{pmatrix} -(aI^* + v + \mu) & 0 & -aS^* & \varepsilon \\ a(1 - \delta)I^* & -\mu & a(1 - \delta)S^* & 0 \\ a\delta I^* & 0 & a\delta S^* - \mu & 0 \\ v & 0 & 0 & -(\varepsilon + \mu) \end{pmatrix}, \quad B_1 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & -\eta_1 & 0 & 0 \\ 0 & 0 & -\eta_2 & 0 \\ 0 & \eta_1 & \eta_2 & 0 \end{pmatrix}.$$

The corresponding characteristic equation is

$$\det(\lambda I_4 - B_0 - B_1 e^{-\lambda \tau}) = 0. \quad (6)$$

Equation (6) is the transcendental quasi-polynomial used in [12]; for the numerical parameter set fixed below in Section 7, the first Hopf point reported in [12] is $\tau_0 \simeq 9.3347$ with imaginary part $\omega_0 \simeq 0.4952$. This is the discrete-delay benchmark for the distributed-memory analysis.

Disease-Free Equilibrium and Threshold Conditions

The disease-free equilibrium is common to the fixed- and distributed-delay formulations and is given by

$$E_0 = (S_0, 0, 0, R_0^{(s)}), \quad S_0 = \frac{A(\varepsilon + \mu)}{\mu(\varepsilon + v + \mu)}, \quad R_0^{(s)} = \frac{vS_0}{\varepsilon + \mu}.$$

For the weak chain, the auxiliary coordinates are $X_0 = Y_0 = 0$, whereas for the strong chain $X_{1,0} = X_{2,0} = Y_{1,0} = Y_{2,0} = 0$. To avoid confusing the recovered-state coordinate $R_0^{(s)}$ with the basic reproduction number, the latter continues to be denoted by R_0 as in (2).

Proposition 1 (Local stability of the disease-free equilibrium). Let $\beta = a\delta S_0 - \mu$.

(1) For the weak gamma model, E_0 is locally asymptotically stable if and only if

$$R_0 < 1, \quad \alpha + \mu - a\delta S_0 > 0.$$

(2) For the strong gamma model, E_0 is locally asymptotically stable if and only if

$$b_1 = 2\alpha - \beta > 0, \quad b_2 = \alpha^2 - 2\alpha\beta > 0, \quad b_3 = \alpha^2(\eta_2 - \beta) > 0, \quad b_1b_2 > b_3.$$

In particular, $b_3 > 0$ is equivalent to $R_0 < 1$.

Proof. At E_0 , the (S, R) block is Hurwitz and the carrier subsystem is a stable linear filter driven by the infectious subsystem. For the weak kernel, the homogeneous (I, Y) block has characteristic polynomial

$$q_w(\lambda) = \lambda^2 + (\alpha - \beta)\lambda + \alpha(\eta_2 - \beta).$$

The quadratic Routh–Hurwitz criterion gives $\alpha - \beta > 0$ and $\eta_2 - \beta > 0$. Since $\eta_2 - \beta = \mu + \eta_2 - a\delta S_0 = (\mu + \eta_2)(1 - R_0)$, the stated conditions follow. For the strong kernel, the homogeneous (I, Y_1, Y_2) block has

$$q_s(\lambda) = (\lambda - \beta)(\lambda + \alpha)^2 + \eta_2\alpha^2 = \lambda^3 + b_1\lambda^2 + b_2\lambda + b_3.$$

The cubic Routh–Hurwitz criterion is precisely $b_1, b_2, b_3 > 0$ and $b_1b_2 > b_3$. The remaining diagonal blocks are Hurwitz, so these conditions are also sufficient for the full linearization. $\square$

3. Weak Gamma Distributed Removal Delay

The weak gamma memory replaces the discrete delay by the normalized kernel

$$K_w(u) = \alpha e^{-\alpha u}, \quad u \geq 0, \quad \alpha = \frac{1}{T}, \quad T > 0, \quad (7)$$

which is the probability density function of an exponential law with mean T . The fact that $\int_0^\infty K_w(u) du = 1$ will play a decisive role in the equilibrium analysis. The delayed removal terms in (1) are now replaced by weighted averages of the past:

$$\mathcal{C}_w(t) = \int_0^\infty K_w(u)C(t-u) du, \quad \mathcal{I}_w(t) = \int_0^\infty K_w(u)I(t-u) du.$$

The weak distributed-delay model reads

$$\begin{cases} \frac{dS}{dt} = A + \varepsilon R - aIS - (v + \mu)S, \\ \frac{dC}{dt} = a(1 - \delta)IS - \eta_1\mathcal{C}_w - \mu C, \\ \frac{dI}{dt} = a\delta IS - \eta_2\mathcal{I}_w - \mu I, \\ \frac{dR}{dt} = \eta_1\mathcal{C}_w + \eta_2\mathcal{I}_w + vS - (\varepsilon + \mu)R. \end{cases} \quad (8)$$

Lemma 1. For any sufficiently smooth function $g(\cdot)$ such that the convolution $Y(t) = \int_0^\infty K_w(u)g(t-u) du$ is well defined, Y satisfies the linear ordinary differential equation

$$\frac{dY}{dt} = \alpha(g(t) - Y(t)). \quad (9)$$

Proof. Set $v = t - u$, so that

$$Y(t) = \alpha e^{-\alpha t} \int_{-\infty}^t e^{\alpha v} g(v) dv.$$

Differentiating with respect to t ,

$$\frac{dY}{dt} = -\alpha^2 e^{-\alpha t} \int_{-\infty}^t e^{\alpha v} g(v) dv + \alpha e^{-\alpha t} (e^{\alpha t} g(t)) = -\alpha Y(t) + \alpha g(t).$$

□

Applying Lemma 1 with $g = C$ and $g = I$ and setting $X(t) = \mathcal{C}_w(t)$, $Y(t) = \mathcal{I}_w(t)$, the integro-differential system (8) is equivalent to the augmented ordinary differential system

$$\begin{cases} \frac{dS}{dt} = A + \varepsilon R - aIS - (v + \mu)S, \\ \frac{dC}{dt} = a(1 - \delta)IS - \eta_1 X - \mu C, \\ \frac{dI}{dt} = a\delta IS - \eta_2 Y - \mu I, \\ \frac{dR}{dt} = \eta_1 X + \eta_2 Y + vS - (\varepsilon + \mu)R, \\ \frac{dX}{dt} = \alpha(C - X), \\ \frac{dY}{dt} = \alpha(I - Y). \end{cases} \quad (10)$$

The equivalence is exact in the sense that any solution of (8) satisfying the (mild) integrability conditions defines, by setting $X = \mathcal{C}_w$ and $Y = \mathcal{I}_w$, a solution of (10); conversely, given initial data $X(0) = \int_0^\infty K_w(u)C(-u)du$ and $Y(0) = \int_0^\infty K_w(u)I(-u)du$, the ODE solution $X(t)$ recovers the convolution $\mathcal{C}_w(t)$ by the uniqueness of the solution to (9), and similarly for Y .

Proposition 2. The augmented system (10) admits the endemic fixed point

$$(S^*, C^*, I^*, R^*, X^*, Y^*) = (S^*, C^*, I^*, R^*, C^*, I^*),$$

where S^*, C^*, I^*, R^* are given by (3)–(5) together with $S^* = (\eta_2 + \mu)/(a\delta)$. In particular, the endemic equilibrium and the reproduction number R_0 of the fixed-delay model are preserved.

Proof. At a constant solution $C(t) \equiv C^*$, the convolution $\mathcal{C}_w(t)$ becomes

$$\mathcal{C}_w(t) = C^* \int_0^\infty K_w(u) du = C^*,$$

because K_w is normalized. Similarly, $\mathcal{I}_w(t) = I^*$. Substituting these identities into the C equation of (8) reproduces $(\eta_1 + \mu)C^* = a(1 - \delta)I^*S^*$, which is (3); the I equation reproduces $a\delta S^* = \eta_2 + \mu$; the R equation reproduces (4); and the S equation, combined with the conservation identity, yields (5). The chain identifications $X^* = \mathcal{C}_w^* = C^*$ and $Y^* = \mathcal{I}_w^* = I^*$ follow immediately. □

Proposition 2 shows that the kernel only changes the dynamics around the equilibrium, not the equilibrium itself. Whether stability is preserved is the question addressed in Section 6. Setting $s = S - S^*$, $c = C - C^*$, $i = I - I^*$, $r = R - R^*$, $x = X - X^*$, $y = Y - Y^*$, the Jacobian of (10) at the endemic equilibrium is the 6×6 matrix

$$M_w = \begin{pmatrix} -(aI^* + v + \mu) & 0 & -aS^* & \varepsilon & 0 & 0 \\ a(1 - \delta)I^* & -\mu & a(1 - \delta)S^* & 0 & -\eta_1 & 0 \\ a\delta I^* & 0 & a\delta S^* - \mu & 0 & 0 & -\eta_2 \\ v & 0 & 0 & -(\varepsilon + \mu) & \eta_1 & \eta_2 \\ 0 & \alpha & 0 & 0 & -\alpha & 0 \\ 0 & 0 & \alpha & 0 & 0 & -\alpha \end{pmatrix}. \quad (11)$$

The characteristic equation is therefore the ordinary polynomial

$$P_w(\lambda; \alpha) = \det(\lambda I_6 - M_w) = 0, \quad (12)$$

of degree six in λ , with coefficients that depend smoothly on $\alpha = 1/T$. A Hopf point for the weak kernel is therefore a pair (T, ω) with $\omega > 0$ and $T > 0$ such that $P_w(i\omega; 1/T) = 0$ and the additional simplicity/transversality conditions of Section 6 hold.

Remark 1. A simple expansion along the last two rows of M_w yields

$$P_w(\lambda; \alpha) = (\lambda + \alpha)^2 \det(\lambda I_4 - B_0) + \alpha \mathcal{R}(\lambda, \alpha),$$

where $\mathcal{R}$ collects the contributions of the off-diagonal α -entries in column 5 and column 6 of (11). In particular, in the rigid limit $\alpha \rightarrow \infty$ (i.e., $T \rightarrow 0$) the dominant balance forces the auxiliary variables to satisfy $X = C$, $Y = I$ instantaneously, and $P_w(\lambda; \alpha)$ reduces, after dividing by α^2 , to the no-delay characteristic polynomial $\det(\lambda I_4 - B_0 - B_1)$. In the opposite limit $\alpha \rightarrow 0$ ($T \rightarrow \infty$), the auxiliary variables evolve on an increasingly slow time scale. Thus the recovery feedback becomes dynamically delayed over a very long memory horizon, and the limiting characteristic structure must be interpreted as a singular limit. Both limits are consistent with the intuition that the weak gamma kernel interpolates between instantaneous recovery feedback and an arbitrarily slow recovery response.

4. Strong Gamma Distributed Removal Delay

The strong gamma memory is

$$K_s(u) = \alpha^2 u e^{-\alpha u}, \quad u \geq 0, \quad \alpha = \frac{2}{T}, \quad T > 0, \quad (13)$$

which is the probability density function of a gamma law with shape 2 and rate α and therefore has mean $2/\alpha = T$. The kernel (13) is more concentrated around its mean than the weak kernel (7) (variance $T^2/2$ versus T^2) and is therefore closer, in this qualitative sense, to a discrete delay. Define

$$C_s(t) = \int_0^\infty K_s(u) C(t - u) du, \quad \mathcal{I}_s(t) = \int_0^\infty K_s(u) I(t - u) du.$$

The strong distributed-delay model is

$$\begin{aligned} \frac{dS}{dt} &= A + \varepsilon R - aIS - (v + \mu)S, \\ \frac{dC}{dt} &= a(1 - \delta)IS - \eta_1 C_s - \mu C, \\ \frac{dI}{dt} &= a\delta IS - \eta_2 \mathcal{I}_s - \mu I, \\ \frac{dR}{dt} &= \eta_1 C_s + \eta_2 \mathcal{I}_s + vS - (\varepsilon + \mu)R. \end{aligned}$$

Lemma 2. The strong kernel (13) is the convolution of the weak kernel with itself: $K_s = K_1 * K_1$, where $K_1(u) = \alpha e^{-\alpha u}$. Consequently, for any sufficiently smooth function g such that the convolutions below are well defined,

$$\int_0^\infty K_s(u)g(t-u) du = \int_0^\infty K_1(u) Y_1(t-u) du, \quad Y_1(t) = \int_0^\infty K_1(u)g(t-u) du,$$

and $Y_2(t) := \int_0^\infty K_s(u)g(t-u) du$ satisfies

$$\frac{dY_1}{dt} = \alpha(g(t) - Y_1(t)), \quad \frac{dY_2}{dt} = \alpha(Y_1(t) - Y_2(t)). \quad (14)$$

Proof. A direct computation gives

$$(K_1 * K_1)(u) = \int_0^u \alpha e^{-\alpha s} \alpha e^{-\alpha(u-s)} ds = \alpha^2 e^{-\alpha u} \int_0^u ds = \alpha^2 u e^{-\alpha u} = K_s(u).$$

Therefore, $Y_2 = K_s * g = K_1 * (K_1 * g) = K_1 * Y_1$. The ODE for Y_1 is Lemma 1, and the same argument applied to $Y_2 = K_1 * Y_1$ (with g replaced by Y_1) yields the ODE for Y_2 . $\square$

Applying Lemma 2 with $g = C$ and $g = I$, and writing X_1, X_2 for the auxiliary variables associated with C and Y_1, Y_2 for those associated with I , the strong distributed-delay model is equivalent to the eight-dimensional ODE system

$$\begin{cases} \frac{dS}{dt} = A + \varepsilon R - aIS - (v + \mu)S, \\ \frac{dC}{dt} = a(1 - \delta)IS - \eta_1 X_2 - \mu C, \\ \frac{dI}{dt} = a\delta IS - \eta_2 Y_2 - \mu I, \\ \frac{dR}{dt} = \eta_1 X_2 + \eta_2 Y_2 + vS - (\varepsilon + \mu)R, \\ \frac{dX_1}{dt} = \alpha(C - X_1), \quad \frac{dX_2}{dt} = \alpha(X_1 - X_2), \\ \frac{dY_1}{dt} = \alpha(I - Y_1), \quad \frac{dY_2}{dt} = \alpha(Y_1 - Y_2). \end{cases} \quad (15)$$

Proposition 3. The augmented system (15) admits the endemic equilibrium

$$(S^*, C^*, I^*, R^*, X_1^*, X_2^*, Y_1^*, Y_2^*) = (S^*, C^*, I^*, R^*, C^*, C^*, I^*, I^*).$$

Proof. At a constant solution $C \equiv C^*$, the chain identities (14) have unique fixed points $X_1^* = C^*$ and $X_2^* = X_1^* = C^*$. The same argument applied to I gives $Y_1^* = Y_2^* = I^*$. Hence, $C_s^* = X_2^* = C^*$ and $I_s^* = Y_2^* = I^*$, and substituting into the C, I, R, S equations recovers (3)–(5). $\square$

The Jacobian of (15) at the endemic equilibrium is the 8×8 matrix

$$M_s = \begin{pmatrix} -(aI^* + v + \mu) & 0 & -aS^* & \varepsilon & 0 & 0 & 0 & 0 \\ a(1 - \delta)I^* & -\mu & a(1 - \delta)S^* & 0 & 0 & -\eta_1 & 0 & 0 \\ a\delta I^* & 0 & a\delta S^* - \mu & 0 & 0 & 0 & 0 & -\eta_2 \\ v & 0 & 0 & -(\varepsilon + \mu) & 0 & \eta_1 & 0 & \eta_2 \\ 0 & \alpha & 0 & 0 & -\alpha & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -\alpha & 0 & 0 \\ 0 & 0 & \alpha & 0 & 0 & 0 & -\alpha & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \alpha & -\alpha \end{pmatrix}. \quad (16)$$

The characteristic equation is the polynomial

$$P_s(\lambda; \alpha) = \det(\lambda I_8 - M_s) = 0, \quad (17)$$

of degree eight in λ . A Hopf point for the strong kernel is a pair (T, ω) with $\omega > 0$ and $\alpha = 2/T$ such that $P_s(i\omega; 2/T) = 0$ and the simplicity/transversality conditions of Section 6 hold.

5. Nonnegativity Limitation and Conditional Boundedness

The delayed-removal mechanism inherited from the reference model does not generate a positively invariant nonnegative orthant. This is not a technical gap of the chain representation. For the weak chain, at a boundary point with $C = 0$ and $X > 0$ one has $C' = -\eta_1 X < 0$; likewise, $I = 0$ and $Y > 0$ imply $I' = -\eta_2 Y < 0$. For the strong chain the same conclusion follows with X_2 and Y_2 . Consequently, even nonnegative, convolution-consistent histories do not by themselves provide a general positivity theorem for the physical compartments. A claim of unconditional positive invariance would therefore be false unless the recovery mechanism were reformulated, for example, by using a positivity-preserving removal flux.

Proposition 4 (Conditional boundedness and continuation). *Consider either augmented chain system and let $z(t)$ be a classical solution on its maximal interval $[0, t_{\max})$. Assume that the four physical components satisfy*

$$S(t), C(t), I(t), R(t) \geq 0, \quad 0 \leq t < t_{\max}.$$

Then $t_{\max} = \infty$ and

$$N(t) := S(t) + C(t) + I(t) + R(t) = \frac{A}{\mu} + \left(N(0) - \frac{A}{\mu} \right) e^{-\mu t}.$$

Hence, with $M_N = \max\{N(0), A/\mu\}$,

$$0 \leq S(t), C(t), I(t), R(t) \leq M_N \quad (t \geq 0).$$

If the auxiliary initial values are nonnegative, then all auxiliary variables remain nonnegative and satisfy explicit uniform bounds depending only on M_N and their initial values.

Proof. Summing the first four equations of either model cancels all infection and recovery-transfer terms and gives the scalar equation $N' = A - \mu N$. Its explicit solution is the identity stated above, and the assumed nonnegativity of the physical compartments implies the componentwise bound by M_N . For the weak chain,

$$X(t) = e^{-\alpha t} X(0) + \alpha \int_0^t e^{-\alpha(t-s)} C(s) ds,$$

so $0 \leq X(t) \leq \max\{X(0), M_N\}$; the same argument applies to Y . For the strong chain, the estimate is applied successively to X_1, X_2, Y_1, Y_2 . Thus, every component is bounded on the maximal interval. Since the vector fields are polynomial and hence locally Lipschitz, the standard continuation theorem for ODEs excludes finite-time blow-up, so $t_{\max} = \infty$. $\square$

Proposition 4 must not be read as a positivity theorem: nonnegativity is an explicit hypothesis, not a conclusion. This limitation is now stated because it is intrinsic to the delayed-removal formulation. All numerical trajectories reported below were checked a posteriori to remain nonnegative over the displayed intervals.

6. Stability and Hopf Criteria

The distributed-delay characteristic Equations (12) and (17) are ordinary polynomials with real coefficients. Write

$$P(\lambda; \alpha) = \lambda^n + b_1(\alpha)\lambda^{n-1} + \dots + b_{n-1}(\alpha)\lambda + b_n(\alpha),$$

with $n = 6$ for the weak kernel and $n = 8$ for the strong kernel. Each coefficient $b_k(\alpha)$ is a polynomial in α (and a polynomial in the equilibrium coordinates and the model parameters). In particular, $b_n(\alpha) = \det(-M)$ where $M \in \{M_w, M_s\}$, and $b_0 \equiv 1$. Recall the Routh–Hurwitz criterion: all roots of P have strictly negative real parts if and only if the leading principal minors $\Delta_1, \Delta_2, \dots, \Delta_n$ of the associated Hurwitz matrix are strictly positive [45].

Theorem 1 (Weak gamma kernel). *Assume $R_0 > 1$, with R_0 defined by (2). Fix $T > 0$ and let $\alpha = 1/T$. Then:*

- (i) *The endemic equilibrium of the weak gamma model (10) is locally asymptotically stable if and only if all the Hurwitz determinants $\Delta_1(\alpha), \dots, \Delta_6(\alpha)$ of $P_w(\lambda; \alpha)$ are strictly positive.*
- (ii) *Suppose that at $T = T_{0,w}$ (i.e., $\alpha = \alpha_{0,w} = 1/T_{0,w}$) the characteristic equation $P_w(\lambda; \alpha_{0,w}) = 0$ has a simple pair of purely imaginary roots $\lambda = \pm i\omega_{0,w}$, with $\omega_{0,w} > 0$, and all remaining four roots have strictly negative real parts. If the transversality condition*

$$\left. \frac{d}{dT} \operatorname{Re} \lambda(T) \right|_{T=T_{0,w}} = \frac{1}{T_{0,w}^2} \operatorname{Re} \left[\frac{\partial P_w / \partial \alpha}{\partial P_w / \partial \lambda} \right]_{\substack{\lambda = i\omega_{0,w} \\ \alpha = \alpha_{0,w}}} \neq 0 \quad (18)$$

holds, then a Hopf bifurcation occurs at $T = T_{0,w}$.

Proof. (i) By Proposition 2, the endemic equilibrium of (10) is well defined whenever $R_0 > 1$. The linearized system $z' = M_w z$ is a finite-dimensional autonomous linear ODE with constant coefficients depending continuously on α ; local asymptotic stability of the equilibrium of (10) is therefore equivalent to all six eigenvalues of M_w having strictly negative real parts. Because $P_w(\lambda; \alpha)$ is an ordinary polynomial of degree six in λ with real coefficients, the Routh–Hurwitz criterion applies and the claim follows.

(ii) We verify the smooth dependence of the eigenvalues on T and derive the transversality formula. The polynomial $P_w(\lambda; \alpha)$ is smooth in both arguments. If $\lambda_0 = i\omega_{0,w}$ is a simple root at $\alpha = \alpha_{0,w}$, then $\partial P_w / \partial \lambda(\lambda_0; \alpha_{0,w}) \neq 0$, and the implicit function theorem produces a smooth function $\alpha \mapsto \lambda(\alpha)$ defined on a neighbourhood of $\alpha_{0,w}$, with $\lambda(\alpha_{0,w}) = \lambda_0$ and

$$\left. \frac{d\lambda}{d\alpha} \right|_{\alpha=\alpha_{0,w}} = - \frac{\partial P_w / \partial \alpha}{\partial P_w / \partial \lambda} \Big|_{(\lambda_0, \alpha_{0,w})}.$$

Composing with $\alpha = 1/T$, and using $d\alpha/dT = -1/T^2$, one obtains

$$\left. \frac{d\lambda}{dT} \right|_{T=T_{0,w}} = - \frac{1}{T_{0,w}^2} \cdot \left. \frac{d\lambda}{d\alpha} \right|_{\alpha=\alpha_{0,w}}.$$

Substituting $d\lambda/d\alpha = -(P_{w,\alpha}/P_{w,\lambda})$ into this identity gives the positive sign in (18). Taking real parts yields (18). By assumption, $\operatorname{Re}(d\lambda/dT) \neq 0$ at $T = T_{0,w}$, so the pair of purely imaginary eigenvalues crosses the imaginary axis transversally. Together with the simplicity of the pair and the negativity of all other roots, this satisfies the hypotheses of the classical Hopf bifurcation theorem for ODEs [45], which therefore yields a Hopf bifurcation at $T = T_{0,w}$. $\square$

Theorem 2 (Strong gamma kernel). Assume $R_0 > 1$. Fix $T > 0$ and let $\alpha = 2/T$. Then,

- (i) The endemic equilibrium of the strong gamma model (15) is locally asymptotically stable if and only if all the Hurwitz determinants $\Delta_1(\alpha), \dots, \Delta_8(\alpha)$ of $P_s(\lambda; \alpha)$ are strictly positive.
- (ii) Suppose that at $T = T_{0,s}$ (i.e., $\alpha = \alpha_{0,s} = 2/T_{0,s}$) the characteristic equation $P_s(\lambda; \alpha_{0,s}) = 0$ has a simple pair of purely imaginary roots $\lambda = \pm i\omega_{0,s}$, all remaining six roots have strictly negative real parts, and the transversality condition

$$\left. \frac{d}{dT} \operatorname{Re} \lambda(T) \right|_{T=T_{0,s}} = \frac{2}{T_{0,s}^2} \operatorname{Re} \left[\frac{\partial P_s / \partial \alpha}{\partial P_s / \partial \lambda} \right]_{\substack{\lambda=i\omega_{0,s} \\ \alpha=\alpha_{0,s}}} \neq 0 \quad (19)$$

holds. Then a Hopf bifurcation occurs at $T = T_{0,s}$.

Proof. By Proposition 3, the endemic equilibrium of (15) is well defined whenever $R_0 > 1$, and the linearization is the finite-dimensional system $z' = M_s z$. Local asymptotic stability is equivalent to all eigenvalues of M_s having strictly negative real parts; this gives the Routh–Hurwitz conditions of (i) since P_s is a real-coefficient polynomial of degree eight. The transversality formula (19) is obtained as in the proof of Theorem 1, replacing $\alpha = 1/T$ by $\alpha = 2/T$, so that $d\alpha/dT = -2/T^2$. Since $d\lambda/d\alpha = -(P_{s,\alpha}/P_{s,\lambda})$, the two minus signs cancel and yield the positive factor $2/T_{0,s}^2$ in (19). The conclusion follows from the Hopf bifurcation theorem for ODEs [45]. $\square$

First Lyapunov Coefficient and Hopf Direction

Although the original formulations involve distributed delays, the weak and strong gamma kernels considered here admit exact finite-dimensional realizations through the linear chain trick, namely the six-dimensional system (10) and the eight-dimensional system (15). Therefore, the Hopf normal-form calculation below is carried out on these equivalent autonomous ODE systems, rather than by applying an ODE formula directly to an infinite-dimensional delay equation. Consequently, the standard finite-dimensional first-Lyapunov-coefficient formula used in Equation (20) is directly applicable to these equivalent augmented ODE systems and their Jacobian matrices M_w and M_s . Because both augmented systems contain only the bilinear incidence term IS , their Taylor expansion about the endemic equilibrium has the form

$$\dot{z} = Mz + \frac{1}{2}B(z, z),$$

with no cubic term. In the coordinate order used in (11) and (16), the only nonzero components of the symmetric bilinear map are

$$B_1(x, y) = -a(x_1 y_3 + x_3 y_1), \quad B_2(x, y) = a(1 - \delta)(x_1 y_3 + x_3 y_1), \quad B_3(x, y) = a\delta(x_1 y_3 + x_3 y_1).$$

Let q and p satisfy $Mq = i\omega_0 q$, $M^\top p = -i\omega_0 p$, and $\bar{p}^\top q = 1$. The first Lyapunov coefficient in the standard ODE normalization [48] is

$$\ell_1 = \frac{1}{2\omega_0} \operatorname{Re} \left\langle p, -2B(q, M^{-1}B(q, \bar{q})) + B(\bar{q}, (2i\omega_0 I - M)^{-1}B(q, q)) \right\rangle. \quad (20)$$

This formula is well defined because zero and $2i\omega_0$ are not eigenvalues at a simple Hopf point. If $\sigma = d \operatorname{Re} \lambda / dT$ and $\ell_1 \neq 0$, the center-manifold radial equation is $\dot{r} = \sigma(T - T_0)r + \ell_1 r^3 + O(r^5 + |T - T_0|r^3)$. Therefore, $\ell_1 < 0$ gives a supercritical Hopf bifurcation with stable small-amplitude cycles for $T > T_0$ when $\sigma > 0$, whereas $\ell_1 > 0$ gives a subcritical Hopf bifurcation with unstable small-amplitude cycles for $T < T_0$.

Formula (20) is an analytical normal-form identity, whereas its sign for the present models is determined numerically at the two reference Hopf points. Using the parameter values of Section 7, double-precision evaluation gives

$$\ell_{1,w} = 0.0425744535 > 0, \quad \ell_{1,s} = 0.0165411999 > 0.$$

The corresponding eigenvector residuals are

$$\|M_w q - i\omega_{0,w} q\|_2 = 1.1 \times 10^{-11}, \quad \|M_w^\top p + i\omega_{0,w} p\|_2 = 5.6 \times 10^{-11},$$

$$\|M_s q - i\omega_{0,s} q\|_2 = 1.6 \times 10^{-11}, \quad \|M_s^\top p + i\omega_{0,s} p\|_2 = 9.1 \times 10^{-11},$$

and $|\bar{p}^\top q - 1| < 5 \times 10^{-16}$ in both computations. Thus, for the reference parameter set, the first Hopf bifurcation is subcritical for both the weak and strong gamma kernels. Indeed, $\ell_{1,w} > 0$ and $\ell_{1,s} > 0$, and the corresponding eigenvalue crossing speeds are positive; hence, in both cases, the local branch of small-amplitude periodic solutions lies on the side $T < T_0$ and is unstable. This is a parameter-specific conclusion, not a theorem asserting subcriticality for all admissible parameter values. The forward simulations for $T > T_0$ illustrate loss of equilibrium stability only; numerical continuation and Floquet verification of the unstable periodic branches are provided in Section 7.

7. Numerical Comparison with the Reference Paper

The parameter set is the one used in the numerical section of [12]:

$$A = 2, \varepsilon = 0.004, a = 0.007, v = 0.05, \mu = 0.04, \delta = 0.91, \eta_1 = 0.005, \eta_2 = 0.1.$$

Using $S^* = (\eta_2 + \mu)/(a\delta)$ together with (3)–(5), the endemic equilibrium is

$$E^* = (21.9780, 0.2593, 0.8428, 26.9199).$$

These values agree with the equilibrium reported in [12] to four decimal places and satisfy the conservation identity $S^* + C^* + I^* + R^* = A/\mu = 50$ up to round-off. All numerical computations were performed in Python 3.12 using NumPy and SciPy in double precision. For each T , the complete spectrum of $M_w(T)$ or $M_s(T)$ was computed with `numpy.linalg.eigvals`. The first crossing was bracketed on a grid of step 10^{-3} and refined by Brent's method applied to the real part of the continuously tracked rightmost eigenvalue, with absolute tolerance 10^{-12} . Transversality was checked by centered finite differences with steps 10^{-4} , 10^{-5} , and 10^{-6} . The first Lyapunov coefficient was evaluated from (20) by solving the indicated complex linear systems and checking the eigenvector and normalization residuals reported above. Time integrations used `scipy.integrate.solve_ivp` with DOP853, relative tolerance 10^{-10} , absolute tolerance 10^{-12} , and maximum step 0.1. In every time-series experiment the physical initial state was

$$(S(0), C(0), I(0), R(0)) = (S^*, C^*, I^*, R^*) + (10^{-2}, 10^{-3}, 10^{-3}, -1.2 \times 10^{-2}),$$

which preserves the total population. For the weak chain, $X(0) = C(0)$ and $Y(0) = I(0)$; for the strong chain, $X_1(0) = X_2(0) = C(0)$ and $Y_1(0) = Y_2(0) = I(0)$. Repeating the integrations with maximum step 0.05 and tolerances 10^{-12} and 10^{-14} changed the computed trajectories by less than 10^{-8} in the sup norm over the displayed time window and did not change the plotted curves at graphical resolution.

The distributed thresholds were obtained by continuing the rightmost eigenvalue of (11) and (16) as a function of T , or equivalently by solving $P_w(i\omega; 1/T) = 0$ and $P_s(i\omega; 2/T) = 0$. The threshold comparison and spectral verification are given in Table 1.

Table 1. Hopf thresholds for the reference fixed-delay model and for the two distributed-memory models.

Model	Critical Memory	Critical Pair	Largest Remaining Real Part	$d \operatorname{Re} \lambda / dT$
Fixed delay [12]	$\tau_0 \simeq 9.3347$	$\pm 0.4952i$	not recomputed	not reported
Weak gamma	$T_{0,w} = 9.98892596$	$\pm 0.02921690i$	-0.04000000	0.00460662
Strong gamma	$T_{0,s} = 9.72363580$	$\pm 0.03357325i$	-0.04000000	0.00628894

At $T_{0,w}$ the remaining weak-kernel eigenvalues are

$$-0.04000000, -0.05013710, -0.09006729, -0.09991676,$$

and at $T_{0,s}$ the remaining strong-kernel eigenvalues are

$$\begin{aligned} &-0.04000000, -0.04876102, -0.10395415, \\ &-0.16446501, -0.23834040, -0.30711636. \end{aligned}$$

These lists verify that the imaginary pair is simple and that every noncritical eigenvalue lies strictly in the open left half-plane.

Numerical Continuation of the Local Periodic Branches

To provide direct numerical evidence of the periodic solutions, we solved the periodic boundary-value problem

$$\frac{dz}{ds} = PF(z; T), \quad z(0) = z(1), \quad s \in [0, 1],$$

where P and T are unknowns. Time-translation invariance was removed by

$$\langle z(0) - E^*, \operatorname{Im} q \rangle = 0,$$

and the branch amplitude was fixed by

$$\langle z(0) - E^*, \operatorname{Re} q \rangle = \rho, \quad \rho = 2 \times 10^{-2}.$$

The initial guess was the leading Hopf approximation

$$z_{\text{guess}}(s) = E^* + \rho(\operatorname{Re} q \cos(2\pi s) - \operatorname{Im} q \sin(2\pi s)).$$

The collocation tolerance was 10^{-9} . Floquet multipliers were obtained by integrating the variational equation over one period and computing the eigenvalues of the monodromy matrix. The numerical results for the continued local periodic solutions are summarized in Table 2.

Table 2. Numerically continued local periodic solutions. The value $\mu_{\max}$ is the largest nontrivial Floquet multiplier in the modulus.

Model	T	Period P	Residual	$\mu_{\max}$
Weak gamma	$9.9888581306 < T_{0,w}$	215.0767341	5.63×10^{-10}	1.00013444
Strong gamma	$9.7236030328 < T_{0,s}$	187.1727594	6.82×10^{-10}	1.00007715

For the weak cycle,

$$21.9554304 \leq S \leq 22.0008976, \quad 0.2555809 \leq C \leq 0.2629919,$$

$$0.8277956 \leq I \leq 0.8576151, \quad 26.9139943 \leq R \leq 26.9257291.$$

For the strong cycle,

$$21.9535289 \leq S \leq 22.0028555, \quad 0.2553746 \leq C \leq 0.2631867,$$

$$0.8263075 \leq I \leq 0.8590774, \quad 26.9130198 \leq R \leq 26.9266998.$$

Thus, both computed cycles remain strictly positive. In each case, the largest nontrivial Floquet multiplier exceeds one, while the trivial multiplier equals one to numerical precision. Therefore, both local periodic orbits are unstable, in agreement with $\ell_{1,w} > 0$ and $\ell_{1,s} > 0$.

The weak kernel gives the largest critical mean memory, while the strong kernel is closer to the fixed-delay threshold. This ordering is consistent with the shape of the two kernels: the weak exponential memory has variance T^2 and therefore averages the past more broadly, with a stronger smoothing effect, while the strong kernel has variance $T^2/2$ and is more concentrated around its mean, hence closer to a discrete delay. The Hopf frequencies for the distributed models are much smaller than the fixed-delay frequency, so the emerging oscillations are slower when the removal process is represented by a distributed memory.

8. Weak-Kernel Simulations

The weak-kernel simulations illustrate the stabilizing role of a broad exponential memory. Since the weak kernel distributes the recovery action over a relatively wide portion of the past, the critical mean memory is larger than in the strong-kernel case. The two panels below and above threshold are therefore intended to show how the same equilibrium response changes when the mean anti-virus recovery time crosses $T_{0,w}$.

Figure 2 shows the weak gamma model for $T = 9.80 < T_{0,w}$. The left panel reports deviations from the endemic state, not the absolute state variables, so convergence is read from the decay of all curves toward zero. The oscillations are initially visible mainly in the infectious component and then are progressively transferred to the carrier and recovered components through the recovery feedback. Their envelope decreases throughout the integration interval, which is consistent with the fact that the rightmost eigenvalue still has negative real part below the Hopf threshold. The right panel gives the same information geometrically: the projected trajectory forms an inward spiral toward E^* , showing that the transient swings in S , C , and I are not persistent. In practical terms, the average recovery memory is still short enough for the anti-virus feedback to compensate for infection growth: carrier and infectious nodes exhibit transient peaks, but the recovery class absorbs these deviations and the system returns to the endemic operating level. The rescaling of C and I is purely graphical and is used only to place variables with different magnitudes on a common vertical scale.

Figure 3 uses $T = 10.05 > T_{0,w}$, slightly above the first critical value. The perturbation no longer decays; instead, the envelope of the deviations increases slowly and the phase portrait moves away from the equilibrium along a spiral-like path. This visual behaviour is the local signature of the Hopf crossing predicted by Theorem 1. The dominant oscillatory mode is especially clear in the infectious class, while the susceptible and recovered classes react with smaller but coherent fluctuations because they are coupled to infection and removal. The growth is gradual because the Hopf frequency in Table 1 is small, so a long integration window is needed before the instability becomes visually pronounced.

This feature is important for WSN monitoring: near the threshold, instability may first appear as low-frequency recurrent waves of carrier and infectious nodes rather than as an abrupt outbreak. Therefore, a defense policy based only on short-time observations could incorrectly classify a near-critical network as stable.

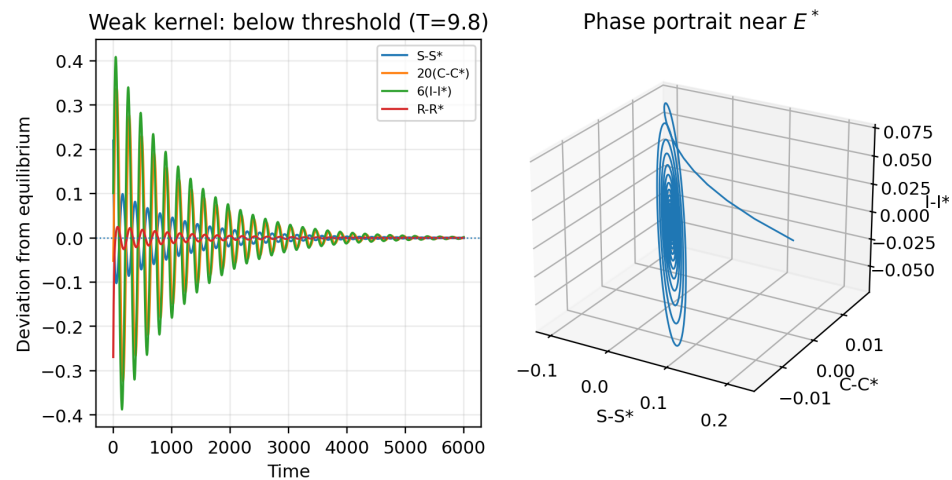


Figure 2. Weak gamma kernel below the critical mean memory: $T = 9.80 < T_{0,w} \simeq 9.9889$. The time series show damped deviations from equilibrium, and the phase portrait shows an inward spiral toward E^* . Variables C and I are rescaled in the time plot only to make all curves visible.

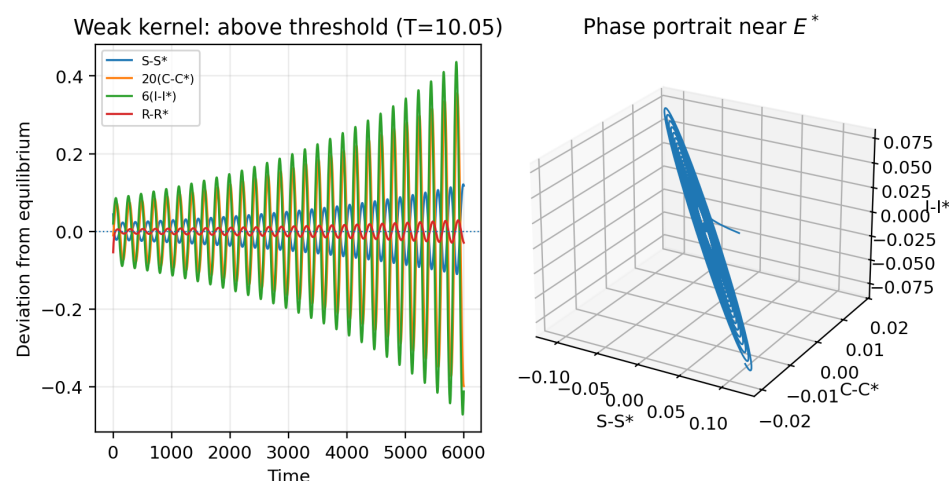


Figure 3. Weak gamma kernel just above the critical mean memory: $T = 10.05 > T_{0,w} \simeq 9.9889$. The equilibrium is locally unstable: the time-series envelope grows slowly and the projected trajectory moves away from E^* .

9. Strong-Kernel Simulations

The strong-kernel simulations give the corresponding picture for a two-stage gamma memory. This memory is less dispersed than the weak one and places more weight around the mean recovery time. Consequently, it is dynamically closer to a deterministic delay and loses stability at a smaller mean value. The comparison with the weak case is therefore useful because the equilibrium is unchanged, while the shape of the recovery-time distribution changes the local response to perturbations.

Figure 4 shows the strong gamma model for $T = 9.55 < T_{0,s}$. The transient oscillations decay, and the phase trajectory returns to the endemic equilibrium. The left panel again plots deviations from equilibrium; hence, the relevant diagnostic is the shrinking envelope around the zero line. The right panel confirms this interpretation by showing an inward motion in the projected $(S-S^*, C-C^*, I-I^*)$ space. Compared with the weak kernel,

the strong kernel concentrates more weight around the mean cleaning time, so the stabilizing averaging effect is weaker and the critical value is lower. Nevertheless, for T below the critical value, recovery feedback still dominates and the deviations shrink. The figure therefore represents the stable side of the Hopf threshold for a two-stage memory, where the cleaning process is less dispersed than in the weak case but not yet sufficiently delayed to destabilize the endemic state.

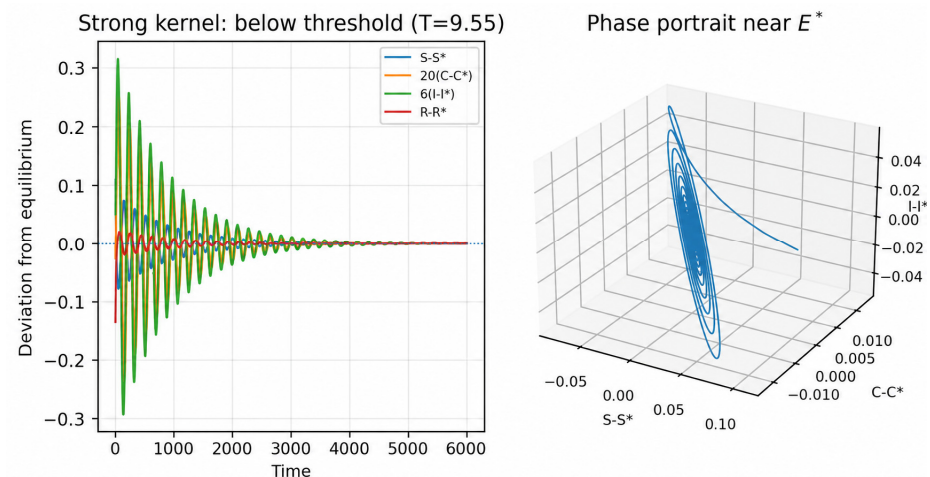


Figure 4. Strong gamma kernel below the critical mean memory: $T = 9.55 < T_{0,s} \simeq 9.7236$. Oscillatory deviations are damped and the projected trajectory returns to the endemic equilibrium.

Figure 5 uses $T = 9.75 > T_{0,s}$, again just above the first critical value. The oscillations are slow and their amplitude grows, in accordance with the positive real part of the leading eigenvalue. The outward phase portrait is more compact than in the weak-kernel unstable case because the chosen value of T is only slightly beyond $T_{0,s}$, but the trend is unambiguous: the trajectory does not spiral back to E^* . The strong kernel crosses earlier than the weak kernel, consistently with the interpretation that a more sharply concentrated memory resembles the destabilizing fixed-delay mechanism more closely. Operationally, this means that when many nodes are cleaned after almost the same mean response time, the recovery action can become synchronized with the infection dynamics and amplify recurrent waves instead of damping them. As in the weak case, the numerical evidence is used only to illustrate the local loss of stability; the stability of the bifurcating periodic orbit is not asserted without a normal-form computation.

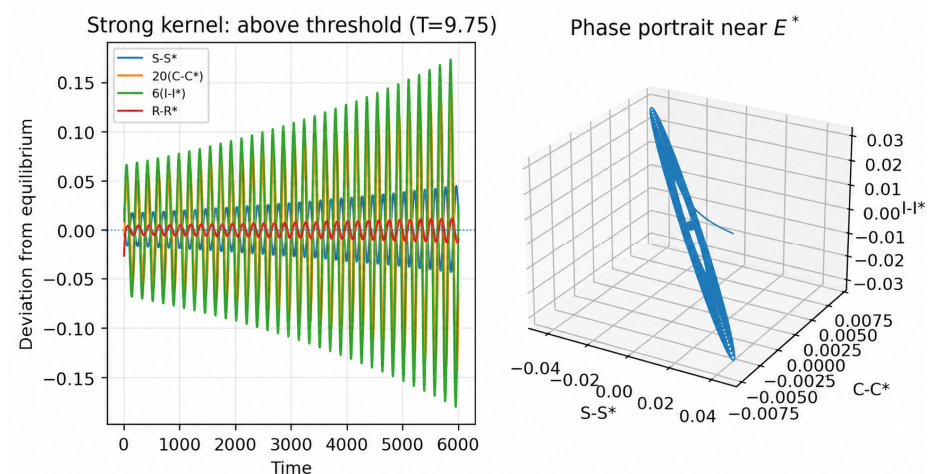


Figure 5. Strong gamma kernel just above the critical mean memory: $T = 9.75 > T_{0,s} \simeq 9.7236$. Oscillatory deviations grow and the phase portrait shows departure from the endemic equilibrium.

10. Discussion

The fixed-delay model of [12] assumes a single deterministic cleaning time. The weak and strong gamma models replace this by a distribution of cleaning times. Because both kernels are normalized, Propositions 2 and 3 show that this change does not alter the endemic equilibrium; what it does alter is the characteristic equation and hence the local dynamics around that equilibrium. Thus the comparison isolates the effect of memory shape: the endemic level of susceptible, carrier, infectious and recovered nodes is the same, but the response of the system to perturbations changes.

The numerical thresholds in Table 1 show that distributed delay is not a cosmetic modification of the reference model. For the same mean memory, the stability boundary depends on the shape of the memory law. The weak kernel spreads the removal effect over a broader portion of the past and gives the largest stability threshold. The strong kernel is more concentrated and produces a threshold closer to the fixed-delay value. This comparison supports the cybersecurity interpretation that not only the average anti-virus response time, but also the variability of response times across nodes, may affect whether malware prevalence settles or oscillates. Broadly distributed cleaning times can smooth the feedback and postpone oscillatory instability, whereas a narrowly concentrated response behaves more like a synchronized delayed reaction and can destabilize the endemic state earlier.

The figures complement the eigenvalue calculations by showing how the theoretical threshold is expressed in the trajectories. Below the critical mean memory, both kernels produce damped oscillations and inward phase motion; above it, the perturbations grow slowly and generate recurrent waves in the carrier and infectious classes. The small distributed-delay Hopf frequencies reported in Table 1 explain why these oscillations have long periods. From a practical viewpoint, this suggests that near-critical WSNs may not show immediate explosive growth but rather delayed recurrent fluctuations, which could be missed by short observation windows.

From a modeling viewpoint, the gamma formulation also has a practical advantage. The fixed-delay system leads to a transcendental characteristic in Equation (6), whereas the weak and strong gamma kernels lead to ordinary polynomial eigenvalue problems (12) and (17) after the chain transformation. This makes stability testing and numerical continuation easier and more reliable: the Routh–Hurwitz criterion can be applied directly, and the implicit function theorem gives a closed-form transversality condition (18) and (19). The price is an increase in dimension: the weak kernel adds two auxiliary variables, while the strong kernel adds four.

For the reference parameter set, the normal-form evaluation resolves the local direction of the first Hopf point of each kernel: both are subcritical and their nearby periodic orbits are unstable. The analysis remains local and does not determine the global fate of trajectories after they leave a neighbourhood of the endemic equilibrium. Moreover, the same memory law is used for carrier and infectious recovery. In real WSNs, carrier nodes may be detected and cleaned with a different time distribution from infectious nodes, because the latter are more visible but may also require longer remediation. These points motivate the extensions mentioned in the conclusion.

11. Conclusions

This paper generalized a delayed malware dissemination model for wireless sensor networks by replacing the fixed anti-virus cycle time with weak and strong gamma distributed memories. The construction keeps the equilibrium analysis transparent: because both kernels have unit mass, the endemic equilibrium and the reproduction threshold remain those of the reference model. The local stability problem, however, changes substantially because carrier and infectious recovery now depend on weighted histories rather

than on a single past value. The linear chain trick converts the weak and strong distributed-delay formulations into finite-dimensional ODE systems. This allows the characteristic equations to be written as sixth- and eighth-degree polynomials and makes it possible to apply Routh–Hurwitz stability tests and standard Hopf bifurcation theory. The proofs show that a Hopf transition occurs when a simple complex-conjugate pair crosses the imaginary axis transversally, while all remaining eigenvalues stay in the left half-plane. For the reference parameter set of [12], the fixed-delay Hopf threshold is approximately $\tau_0 \simeq 9.3347$. The weak and strong gamma thresholds are $T_{0,w} \simeq 9.9889$ and $T_{0,s} \simeq 9.7236$, respectively. The weak kernel exerts the strongest stabilizing effect, while the strong kernel lies closer to the discrete-delay case. For the reference parameter set, the numerically evaluated first Lyapunov coefficients are positive at the first weak- and strong-kernel Hopf points. Hence, these two parameter-specific crossings are subcritical and generate local unstable small-amplitude periodic orbits on the $T < T_0$ side. The numerical figures confirm convergence below the critical mean memory and the onset of oscillatory instability above it. Hence, in addition to reducing the average cleaning time, a WSN defense policy should also consider how synchronized or heterogeneous the cleaning times are across nodes.

Relative to the fixed-delay model and to existing applications of the linear chain trick, the principal contribution is not the transformation itself but the model-specific combination of: disease-free threshold analysis, identification of the failure of unconditional positive invariance, rigorous conditional boundedness, complete Hopf spectral and transversality verification, first-Lyapunov-coefficient classification, and direct continuation with Floquet verification of the local periodic branches. The equal-mean and equal-equilibrium comparison further isolates the effect of recovery-time dispersion: the broader weak kernel delays the first instability more than the more concentrated strong kernel.

A natural continuation is to extend the presently computed local periodic branches globally and detect possible folds of cycles or secondary bifurcations. Other extensions include heterogeneous kernels for carrier and infectious nodes, gamma kernels with non-integer shape parameter (which require pseudospectral methods beyond the linear chain trick [38]), stochastic fluctuations in the cleaning process, and contact structures depending on the topology of the wireless sensor network.

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