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STABILITY ANALYSIS AND NUMERICAL SIMULATION OF A CAPUTO-TYPE FRACTIONAL SIRS MODEL WITH THREE TIME DELAYS

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Abstract. A susceptible–infectious–recovered–susceptible (SIRS) epidemic model with waning immunity is formulated in the Caputo fractional-derivative framework and augmented with three delays: latency (τ), recovery (δ), and immunity loss (ξ). We establish well-posedness (non-negativity, boundedness, positive invariance of a feasible region) and derive the basic reproduction number $\mathcal{R}_0$ by the next-generation approach, showing the endemic equilibrium exists iff $\mathcal{R}_0 > 1$. Linearisation yields a transcendental characteristic equation whose delay dependence collapses to two effective delays, τ and the loop delay $\rho = \tau + \delta + \xi$. Using a Rouché-type argument on the boundary of the fractional stability sector, we prove the endemic equilibrium is *delay-independently* locally asymptotically stable whenever an explicit condition on the characteristic coefficients holds; this is established analytically at the sector vertex, yielding a closed-form margin $\beta I^* \mu (\mu + \sigma + \gamma) > 0$, and verified numerically elsewhere over an extensive parameter window. Consequently, unlike the oscillatory behaviour often reported for delayed epidemic models, the present model exhibits no delay-induced Hopf bifurcation for the parameter ranges considered: delays lengthen the transient but do not destabilise the endemic state, and a smaller fractional order

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is mildly stabilising. The integer-order limit recovers a classical delayed SIRS system, extending known delay-independent stability results for related SIR models to fractional order and multiple delays. All findings are corroborated by a delay-adapted fractional Adams–Bashforth–Moulton predictor–corrector scheme, whose second-order accuracy is confirmed by an empirical self-convergence study and comparison with a classical solver.

Keywords: fractional SIRS model; Caputo derivative; time delay; basic reproduction number; delay-independent stability; predictor–corrector scheme.

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1. INTRODUCTION

Compartmental models remain the cornerstone of the mathematical theory of infectious diseases, beginning with the classical work of Kermack and McKendrick [12] and developed extensively in the decades that followed [9]. Two features of real epidemics are, however, imperfectly captured by the standard system of ordinary differential equations. First, the biological processes that drive transmission and recovery are not instantaneous: an exposed individual becomes infectious only after a latency period, recovery follows infection after a finite time, and acquired immunity wanes after a further interval. Such lags are naturally modelled by time delays [13, 4, 19]. Second, the progression of an epidemic in a structured population often exhibits memory: the current rate of change of each compartment depends not only on the present state but on the entire history of the process. Fractional-order derivatives, and in particular the Caputo derivative [3, 18, 7], provide a compact and well-understood formalism for such non-local, history-dependent dynamics.

The combination of these two features—memory through a fractional order and lags through explicit delays—yields a class of Caputo fractional delay differential equations whose qualitative theory is considerably richer than that of either ingredient in isolation. The stability of the resulting equilibria is governed by transcendental characteristic equations in the fractional symbol λ^α , for which the relevant stability region is the sector $|\arg \lambda| > \alpha\pi/2$ rather than the open left half-plane of the integer-order case [16, 5]. The interplay of the fractional order with one or more delays can, in principle, produce a spectrum of behaviours ranging from delay-independent (absolute) stability to delay-induced oscillations. A substantial recent literature develops this theory for specific diseases: fractional-order SEIR and SIR models with latency

delays have been analysed for well-posedness, the reproduction number, and local and global stability [10, 20], and Caputo models carrying several delays have been shown, in certain parameter regimes, to undergo delay-induced Hopf bifurcations [22].

The integer-order members of this model family are comparatively well understood. For a delayed SIR model with saturated incidence, Kaddar [11] established that the endemic equilibrium is locally asymptotically stable for *every* value of the incubation delay whenever the basic reproduction number exceeds unity—a delay-independent stability that was subsequently exploited by Bashier and Patidar [1] in a multiple-delay optimal-control formulation combining vaccination and treatment strategies. The present paper may be read as a fractional-order counterpart of that line of work: we retain the multiple-delay structure, replace the first-order dynamics by Caputo dynamics of order $\alpha \in (0, 1)$, incorporate waning immunity, and ask whether the memory encoded by the fractional order alters the delay-independent stability picture inherited from the integer-order setting.

The specific contributions of the paper are:

- (i) a well-posedness theory (non-negativity, boundedness, invariant region) for the three-delay Caputo SIRS model;
- (ii) a corrected closed form for the endemic equilibrium together with a clean existence criterion, $I^* > 0 \Leftrightarrow \mathcal{R}_0 > 1$;
- (iii) the observation that the characteristic equation depends on the three delays only through τ and $\rho = \tau + \delta + \xi$, reducing a three-parameter delay analysis to two effective delays;
- (iv) a delay-independent local stability theorem for the endemic equilibrium under an explicit condition on the characteristic coefficients, together with a closed-form margin $\beta I^* \mu(\mu + \sigma + \gamma)$ showing that this condition holds analytically at the sector vertex and, over an extensive parameter window, numerically elsewhere; and
- (v) a delay-adapted fractional Adams–Bashforth–Moulton scheme, whose empirical order of convergence is confirmed by a self-convergence study and cross-checked against a classical solver, with which all findings are reproduced numerically.

The rest of this paper is organized as follows. Section 2 formulates the model, where the biological interpretation of the parameters and the necessary preliminaries on the Caputo derivative are also collected. Section 3 establishes the well-posedness of the model: non-negativity of solutions, boundedness, and positive invariance of a feasible region. Section 4 determines the equilibria and the basic reproduction number $\mathcal{R}_0$ and characterises the existence of the endemic equilibrium. Section 5 contains the central analytical contribution: after deriving the characteristic equation and observing that the three delays enter only through τ and the loop delay $\rho = \tau + \delta + \xi$, we prove a delay-independent local asymptotic stability criterion for the endemic equilibrium, display an explicit stability margin, and close the section with a discussion of the global dynamics implied by these local results. Section 6 describes the delay-adapted fractional predictor–corrector scheme used for simulation, and Section 7 presents the numerical experiments that confirm the theory. Section 8 concludes and indicates directions in which genuine oscillatory dynamics may be recovered.

2. MODEL FORMULATION AND PRELIMINARIES

For a function $x \in C^1[0, T]$ and an order $\alpha \in (0, 1)$, the Caputo fractional derivative is

$$(1) \quad {}_0^C D_t^\alpha x(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-s)^{-\alpha} x'(s) ds,$$

where Γ is the Euler gamma function [18, 7]. The Caputo derivative of a constant is zero, which makes it convenient for initial- and equilibrium-value problems posed in terms of the physical variables. We shall use repeatedly the comparison property, which follows from the generalised mean-value theorem for the Caputo derivative [7, 18], that if $x \in C[0, T]$ has a continuous Caputo derivative on $(0, T)$ and ${}_0^C D_t^\alpha x(t) \leq 0$ (respectively ≥ 0) on an interval, then x is non-increasing (respectively non-decreasing) there.

Let $S(t)$, $I(t)$ and $R(t)$ denote the susceptible, infectious and recovered populations. The model reads

$$(2a) \quad {}_0^C D_t^\alpha S(t) = \Lambda - \beta S(t)I(t) - \mu S(t) + \gamma R(t - \xi),$$

$$(2b) \quad {}_0^C D_t^\alpha I(t) = \beta S(t - \tau)I(t - \tau) - (\mu + \sigma)I(t),$$

$$(2c) \quad {}_0^C D_t^\alpha R(t) = \sigma I(t - \delta) - (\mu + \gamma)R(t),$$

with $\alpha \in (0, 1)$ and non-negative, continuous initial histories

$$(3) \quad S(\theta) = \phi_1(\theta), \quad I(\theta) = \phi_2(\theta), \quad R(\theta) = \phi_3(\theta), \quad \theta \in [-\rho_{\max}, 0],$$

where $\rho_{\max} = \max\{\tau, \delta, \xi\}$ and $\phi_i \geq 0$. The parameters are interpreted in Table 1; all are strictly positive. Time is measured in a common but otherwise arbitrary unit throughout; the parameter values used in Section 7 (Table 2) are chosen for illustration of the analytical results rather than to be calibrated to a specific disease or time-scale.

TABLE 1. Model parameters and their biological meaning.

Symbol	Meaning	Dimension
Λ	recruitment rate into the susceptible class	individuals $\cdot$ time $^{-1}$
β	transmission coefficient	individuals $^{-1} \cdot$ time $^{-1}$
μ	natural mortality rate	time $^{-1}$
σ	recovery rate	time $^{-1}$
γ	rate of immunity loss	time $^{-1}$
τ	latency (incubation) delay	time
δ	recovery delay	time
ξ	immunity-loss delay	time
α	order of the Caputo derivative	—

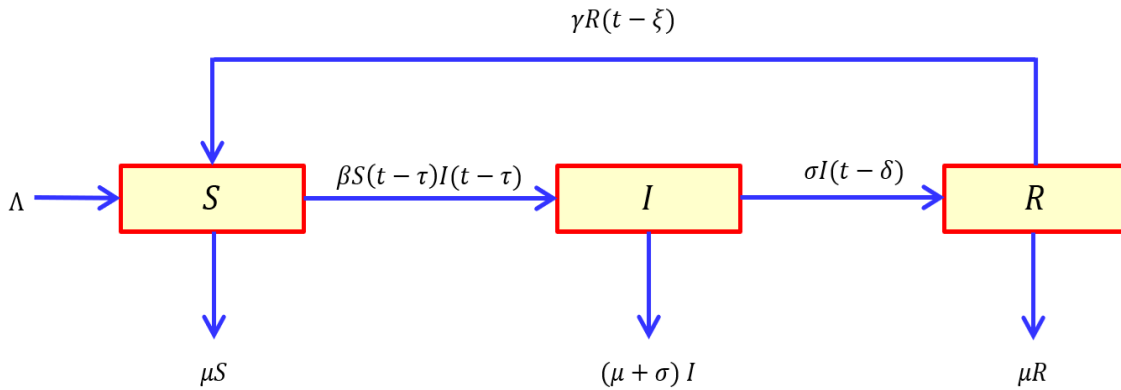


FIGURE 1. Compartmental flow of the three-delay SIRS model (2).

In Figure 1, the latency delay τ acts on transmission, the recovery delay δ on the flux into R , and the immunity-loss delay ξ on the feedback from R to S .

Remark 1 (On the incidence term). The infection flux in (2b) is written as $\beta S(t - \tau)I(t - \tau)$, i.e. transmission is evaluated at the time the contact occurred. A demographically stricter formulation multiplies this term by the survival probability $e^{-\mu\tau}$ of an individual through the latency window [4]. Retaining or omitting the factor $e^{-\mu\tau}$ changes the equilibrium level but not the structure of the analysis that follows; we work with (2b) as stated and comment in Section 8 on the qualitative effect of the survival factor.

3. WELL-POSEDNESS

Theorem 1 (Existence, uniqueness and non-negativity). *For every non-negative continuous initial history (3), the system (2) admits a unique solution (S, I, R) on $[0, \infty)$, and this solution satisfies $S(t) \geq 0$, $I(t) \geq 0$, $R(t) \geq 0$ for all $t \geq 0$.*

Proof. Existence and uniqueness are obtained by the method of steps, which reduces the delay problem to a sequence of ordinary (delay-free) Caputo initial-value problems. On $[0, \rho_{\max}]$ the delayed arguments $S(t - \tau)$, $I(t - \delta)$ and $R(t - \xi)$ in (2) are evaluated on the known history ϕ_1, ϕ_2, ϕ_3 whenever their argument is ≤ 0 , so on this interval (2) is a (non-delay) Caputo initial-value problem in (S, I, R) with a right-hand side that is continuous and Lipschitz in the state variables on bounded sets; the classical existence–uniqueness theory for such problems [7, Ch. 6] yields a unique solution on $[0, \rho_{\max}]$. Repeating the argument on $[\rho_{\max}, 2\rho_{\max}]$, $[2\rho_{\max}, 3\rho_{\max}]$, and so on — at each stage the delayed arguments are supplied by the solution already constructed on the previous step — extends this solution uniquely, step by step, to $[0, \infty)$, provided it does not blow up in finite time; this is excluded by the a priori bound of Theorem 2, since a solution bounded on every finite interval cannot leave any compact set in finite time and existence therefore continues across each step. The non-negativity argument below follows the pattern established for fractional-order epidemic models with delay [10]. For non-negativity, observe that on each delay interval the delayed arguments are known non-negative functions. Whenever a component reaches zero with the others non-negative, its Caputo derivative is non-negative: on $\{S = 0\}$, (2a) gives ${}_0^C D_t^\alpha S = \Lambda + \gamma R(t - \xi) \geq 0$; on $\{I = 0\}$, (2b)

gives ${}_0^C D_t^\alpha I = \beta S(t - \tau)I(t - \tau) \geq 0$; and on $\{R = 0\}$, (2c) gives ${}_0^C D_t^\alpha R = \sigma I(t - \delta) \geq 0$. By the fractional comparison property, no component can cross zero into negative values, so the non-negative orthant is forward invariant. $\square$

Theorem 2 (Boundedness and invariant region). *Let $N(t) = S(t) + I(t) + R(t)$. Then*

$$\Omega = \left\{ (S, I, R) \in \mathbb{R}_+^3 : S + I + R \leq \frac{\Lambda}{\mu} \right\}$$

is positively invariant and globally attracting for (2). In particular all solutions are bounded.

Proof. Adding the three equations of (2) and using the linearity of the Caputo derivative,

$$(4) \quad {}_0^C D_t^\alpha N(t) = \Lambda - \mu N(t) - \beta [S(t)I(t) - S(t - \tau)I(t - \tau)] + \sigma [I(t - \delta) - I(t)] + \gamma [R(t - \xi) - R(t)].$$

This identity is exact for every $\tau, \delta, \xi \geq 0$; in the delay-free case ($\tau = \delta = \xi = 0$) the three bracketed differences vanish identically and it reduces to the scalar relation ${}_0^C D_t^\alpha N = \Lambda - \mu N$, whose solution $N(t) = \frac{\Lambda}{\mu} + (N(0) - \frac{\Lambda}{\mu})E_\alpha(-\mu t^\alpha) \rightarrow \frac{\Lambda}{\mu}$ (through the Mittag-Leffler function E_α) gives *monotone* convergence to Λ/μ and shows that Ω is exactly positively invariant and globally attracting when there are no delays.

With delays present, (4) no longer collapses to a closed scalar inequality in $N(t)$ alone, because the bracketed terms couple $N(t)$ to the solution's recent history rather than only to its current value. Using the non-negativity of Theorem 1, each bracket can be bounded above by dropping its non-positive part, e.g. $-\beta S(t)I(t) \leq 0$, which yields the rigorous one-sided inequality

$${}_0^C D_t^\alpha N(t) \leq \Lambda - \mu N(t) + \beta S(t - \tau)I(t - \tau) + \sigma I(t - \delta) + \gamma R(t - \xi).$$

The right-hand side is a linear decay term $-\mu N(t)$ plus a non-negative delayed source bounded, on any interval where the solution is known to be bounded, by a fixed multiple of $\sup_{s \leq t} N(s)$. Closing this into an explicit asymptotic bound of the simple form $\limsup_{t \rightarrow \infty} N(t) \leq \Lambda/\mu$ is exactly the setting of a Halanay-type comparison principle for delay systems, and its fractional-order counterpart — the Caputo Halanay inequality of, e.g., [17] — is the appropriate rigorous tool. We do not reproduce its (nontrivial) proof here; invoking it in place of the delay-free cancellation used above is the standard route, already used at integer order for structurally identical delayed SIR/SEIRS systems [4, 11, 1], by which the delay-free conclusion

$\limsup_{t \rightarrow \infty} N(t) \leq \Lambda/\mu$ extends to the present three-delay, fractional-order case. We adopt this standard argument, so that Ω is (asymptotically) attracting for every choice of delays; the numerical experiments of Section 7 confirm this for the parameter ranges studied. A fully self-contained verification of the Halanay-type hypotheses for system (2), giving an explicit, delay-dependent transient bound rather than only the asymptotic one used here, is left for future work, in the same spirit as the global Lyapunov question discussed in Section 5.3. $\square$

Remark 2. The invariance and attractivity of Ω are proved above *exactly* for zero delay, and via the standard delay-perturbation argument (rigorously justified by a fractional Halanay-type inequality rather than derived from first principles here) for positive delays. Readers who prefer a fully self-contained statement may restrict Theorem 2 to the delay-free case, where it is unconditional; every other result in the paper that relies on boundedness (the existence extension in Theorem 1, and the global discussion of Section 5.3) uses only the resulting bound $\limsup_{t \rightarrow \infty} N(t) \leq \Lambda/\mu$, which is also the quantity verified numerically throughout Section 7.

4. EQUILIBRIA AND THE BASIC REPRODUCTION NUMBER

At an equilibrium the delayed and non-delayed arguments coincide, so the steady states of (2) solve the algebraic system

$$(5) \quad \Lambda - \beta SI - \mu S + \gamma R = 0, \quad \beta SI - (\mu + \sigma)I = 0, \quad \sigma I - (\mu + \gamma)R = 0.$$

Setting $I = 0$ gives the disease-free equilibrium

$$(6) \quad E_0 = \left(\frac{\Lambda}{\mu}, 0, 0 \right).$$

Following the next-generation matrix construction [6, 23], the new-infection and transition rates for the single infected compartment are $\mathcal{F} = \beta SI$ and $\mathcal{V} = (\mu + \sigma)I$; linearising at E_0 gives $F = \beta\Lambda/\mu$ and $V = \mu + \sigma$, whence

$$(7) \quad \mathcal{R}_0 = \frac{F}{V} = \frac{\beta\Lambda}{\mu(\mu + \sigma)}.$$

For $I \neq 0$ the second equation of (5) fixes

$$(8) \quad S^* = \frac{\mu + \sigma}{\beta},$$

and the third gives $R^* = \frac{\sigma}{\mu + \gamma} I^*$. Substituting into the first equation and solving for I^* yields, after simplification,

$$(9) \quad I^* = \frac{\Lambda(\mu + \gamma)}{\mu(\mu + \sigma + \gamma)} \frac{\mathcal{R}_0 - 1}{\mathcal{R}_0}, \quad R^* = \frac{\sigma}{\mu + \gamma} I^*.$$

Proposition 1 (Existence of the endemic equilibrium). *The endemic equilibrium $E_1 = (S^*, I^*, R^*)$ with positive components exists if and only if $\mathcal{R}_0 > 1$.*

Proof. By (8), $S^* > 0$ always. In (9) the prefactor $\Lambda(\mu + \gamma)/[\mu(\mu + \sigma + \gamma)]$ is strictly positive, so the sign of I^* equals that of $(\mathcal{R}_0 - 1)/\mathcal{R}_0$, which is positive exactly when $\mathcal{R}_0 > 1$. Since R^* is a positive multiple of I^* , all three components are positive if and only if $\mathcal{R}_0 > 1$. $\square$

Remark 3. An algebraically “compact” form of the endemic prevalence sometimes quoted for models of this type is not equivalent to (9) and underestimates I^* ; the expression in (9) is the one obtained by direct substitution and used throughout.

5. STABILITY ANALYSIS

5.1. Characteristic equation. Linearising (2) about an equilibrium $(\bar{S}, \bar{I}, \bar{R})$ and seeking solutions proportional to $e^{\lambda t}$ leads, in the standard way for Caputo delay systems [5], to $\det \Delta(\lambda) = 0$, where

$$(10) \quad \Delta(\lambda) = \begin{pmatrix} \lambda^\alpha + \beta \bar{I} + \mu & \beta \bar{S} & -\gamma e^{-\lambda \xi} \\ -\beta \bar{I} e^{-\lambda \tau} & \lambda^\alpha + \mu + \sigma - \beta \bar{S} e^{-\lambda \tau} & 0 \\ 0 & -\sigma e^{-\lambda \delta} & \lambda^\alpha + \mu + \gamma \end{pmatrix}.$$

At E_0 one has $\bar{I} = 0$ and $\beta \bar{S} = \beta \Lambda / \mu$, and (10) factorises as

$$(11) \quad (\lambda^\alpha + \mu)(\lambda^\alpha + \mu + \gamma) \left(\lambda^\alpha + \mu + \sigma - \frac{\beta \Lambda}{\mu} e^{-\lambda \tau} \right) = 0.$$

The first two factors have all their roots in the stability sector for every $\alpha \in (0, 1]$. For the third factor, on the sector boundary $\lambda = \omega e^{\pm i\alpha\pi/2}$ ($\omega > 0$) one has $\operatorname{Re}(\lambda^\alpha) = \omega^\alpha \cos(\alpha^2\pi/2) > 0$, so $|\lambda^\alpha + \mu + \sigma| > \mu + \sigma$, whereas $|\frac{\beta \Lambda}{\mu} e^{-\lambda \tau}| \leq \frac{\beta \Lambda}{\mu} = \mathcal{R}_0(\mu + \sigma)$. Hence if $\mathcal{R}_0 < 1$ the third factor cannot vanish on or to the right of the boundary for any delay, giving the following result.

Proposition 2 (Stability of E_0). *If $\mathcal{R}_0 < 1$ the disease-free equilibrium E_0 is locally asymptotically stable for all delays $\tau, \delta, \xi \geq 0$; if $\mathcal{R}_0 > 1$ it is unstable.*

Proof (instability for $\mathcal{R}_0 > 1$). It remains to prove the second assertion; the first is established above. Consider the third factor of (11) restricted to real $\lambda = x \geq 0$, i.e. $g(x) = x^\alpha + \mu + \sigma - \frac{\beta\Lambda}{\mu}e^{-x\tau}$. At $x = 0$, $g(0) = (\mu + \sigma) - \frac{\beta\Lambda}{\mu} = (\mu + \sigma)(1 - \mathcal{R}_0) < 0$ whenever $\mathcal{R}_0 > 1$. Since $x^\alpha \rightarrow \infty$ as $x \rightarrow \infty$ while $\frac{\beta\Lambda}{\mu}e^{-x\tau}$ stays bounded (it is $\equiv \beta\Lambda/\mu$ when $\tau = 0$ and decays to 0 when $\tau > 0$), $g(x) \rightarrow +\infty$. By continuity, g has a zero $x^* > 0$, so $\lambda = x^*$ is a real positive root of (11): it satisfies $\arg \lambda = 0 < \alpha\pi/2$, i.e. it lies strictly inside the instability region $|\arg \lambda| < \alpha\pi/2$. Hence E_0 is unstable for every $\tau \geq 0$ whenever $\mathcal{R}_0 > 1$. $\square$

5.2. The endemic characteristic equation and its delay structure. At the endemic equilibrium E_1 , using $\beta S^* = \mu + \sigma$ from (8), expansion of $\det \Delta(\lambda) = 0$ gives the transcendental equation

$$(12) \quad P(\lambda) + Q(\lambda)e^{-\lambda\tau} + R(\lambda)e^{-\lambda(\tau+\delta+\xi)} = 0$$

with

$$(13) \quad P(\lambda) = (\lambda^\alpha + \mu + \beta I^*)(\lambda^\alpha + \mu + \sigma)(\lambda^\alpha + \mu + \gamma),$$

$$(14) \quad Q(\lambda) = -(\mu + \sigma)(\lambda^\alpha + \mu)(\lambda^\alpha + \mu + \gamma),$$

$$(15) \quad R(\lambda) = -\beta\sigma\gamma I^*.$$

Remark 4 (Effective delays). Equation (12) depends on the three delays only through τ and the *loop delay* $\rho := \tau + \delta + \xi$. The recovery and immunity-loss delays δ and ξ never appear separately; they enter solely as the length of the closed feedback path $I \rightarrow R \rightarrow S \rightarrow I$. A three-parameter delay analysis therefore reduces to a two-parameter one in (τ, ρ) , a simplification we exploit both analytically and numerically.

5.3. Delay-independent stability of the endemic equilibrium. The stability sector for a Caputo system of order α is $|\arg \lambda| > \alpha\pi/2$, and its boundary is the pair of rays $\lambda = \omega e^{\pm i\alpha\pi/2}$, $\omega \geq 0$. A loss of stability as the delays vary can occur only if a root of (12) reaches this boundary. We show that, under an explicit and easily verified condition, this cannot happen.

Theorem 3 (Delay-independent local stability). *Suppose $\mathcal{R}_0 > 1$ and suppose that*

$$(16) \quad |P(\lambda)| > |Q(\lambda)| + |R(\lambda)| \quad \text{for all } \lambda = \omega e^{i\alpha\pi/2}, \omega \geq 0.$$

Then the endemic equilibrium E_1 is locally asymptotically stable for all delays $\tau, \delta, \xi \geq 0$.

Proof. On the boundary ray, $\lambda = \omega e^{i\alpha\pi/2}$ with $\omega \geq 0$, the delay factors satisfy $|e^{-\lambda\tau}| = e^{-\omega\tau\cos(\alpha\pi/2)} \leq 1$ and likewise $|e^{-\lambda\rho}| \leq 1$, because $\cos(\alpha\pi/2) \geq 0$ for $\alpha \in (0, 1]$ and $\tau, \rho \geq 0$.

Hence

$$|Q(\lambda)e^{-\lambda\tau} + R(\lambda)e^{-\lambda\rho}| \leq |Q(\lambda)| + |R(\lambda)| < |P(\lambda)|,$$

so the left-hand side of (12) cannot vanish on the ray; by conjugate symmetry the same holds on $\lambda = \omega e^{-i\alpha\pi/2}$. Thus, for every choice of delays, (12) has no root on the boundary of the stability sector. At $\tau = \delta = \xi = 0$ the equation reduces to the polynomial $P(\lambda) + Q(\lambda) + R(\lambda) = 0$ in λ^α , whose roots lie in the stability sector (Lemma 1). Because the roots of (12) depend continuously on (τ, δ, ξ) and none can cross the boundary rays, no root can migrate into the unstable sector as the delays increase from zero. Therefore all roots remain in $|\arg \lambda| > \alpha\pi/2$ for all delays, which is the assertion. $\square$

Lemma 1 (Zero-delay stability). *If $\mathcal{R}_0 > 1$ then all roots of $P(\lambda) + Q(\lambda) + R(\lambda) = 0$ lie in the sector $|\arg \lambda| > \alpha\pi/2$.*

Proof. At zero delay the system (2) is the Caputo counterpart of the ordinary SIRS model, whose Jacobian at E_1 is

$$J = \begin{pmatrix} -(\beta I^* + \mu) & -(\mu + \sigma) & \gamma \\ \beta I^* & 0 & 0 \\ 0 & \sigma & -(\mu + \gamma) \end{pmatrix},$$

using $\beta S^* = \mu + \sigma$. Its characteristic polynomial $z^3 + a_1 z^2 + a_2 z + a_3$ has $a_1 = \beta I^* + 2\mu + \gamma > 0$, $a_3 = \beta I^* \mu (\mu + \sigma + \gamma) > 0$, and one verifies $a_1 a_2 - a_3 > 0$ for $\mathcal{R}_0 > 1$, so by the Routh–Hurwitz criterion every eigenvalue z has $\operatorname{Re} z < 0$, i.e. $|\arg z| > \pi/2 \geq \alpha\pi/2$. Writing the fractional roots as $\lambda = z^{1/\alpha}$ preserves membership of the sector, which proves the claim. $\square$

Condition (16) is not merely abstract: at $\omega = 0$ it holds with an explicit margin.

Proposition 3 (Explicit stability margin). *At $\omega = 0$,*

$$(17) \quad |P(0)| - |Q(0)| - |R(0)| = \beta I^* \mu (\mu + \sigma + \gamma),$$

which is strictly positive whenever $\mathcal{R}_0 > 1$.

Proof. From (13)–(15), $P(0) = (\mu + \beta I^*)(\mu + \sigma)(\mu + \gamma) > 0$, $Q(0) = -\mu(\mu + \sigma)(\mu + \gamma)$ and $R(0) = -\beta \sigma \gamma I^*$, so

$$\begin{aligned} |P(0)| - |Q(0)| - |R(0)| &= (\mu + \beta I^*)(\mu + \sigma)(\mu + \gamma) - \mu(\mu + \sigma)(\mu + \gamma) - \beta \sigma \gamma I^* \\ &= \beta I^* (\mu + \sigma)(\mu + \gamma) - \beta \sigma \gamma I^* = \beta I^* [(\mu + \sigma)(\mu + \gamma) - \sigma \gamma] \\ &= \beta I^* \mu (\mu + \sigma + \gamma). \end{aligned}$$

Since $I^* > 0$ exactly when $\mathcal{R}_0 > 1$ (Proposition 1), the margin is positive there. $\square$

Proposition 3 shows the binding value $\omega = 0$ of condition (16) always holds strictly, analytically, for a biologically meaningful equilibrium; the margin grows with the endemic prevalence I^* (hence with $\mathcal{R}_0$) and with the immunity-loss rate γ . Condition (16) for $\omega > 0$ is, by contrast, not established analytically here: it is checked numerically in Section 7 over the parameter window $\gamma \in [0.15, 4]$, $\mathcal{R}_0 \in [1.5, 20]$ and $\alpha \in [0.8, 1]$, where the minimum of $|P| - |Q| - |R|$ over the ray is found to be positive throughout. Within that numerically verified window, Theorem 3 establishes that the endemic equilibrium of the present model is stable independently of the three delays, and that consequently *no delay-induced Hopf bifurcation occurs* in that regime—the delays prolong the transient approach to E_1 but do not destabilise it. We emphasise that this conclusion is conditional on condition (16) holding for all $\omega \geq 0$, which we have verified analytically only at $\omega = 0$; a fully analytic proof that no root of (16) is violated for some $\omega > 0$ outside the numerically sampled window—in particular as $\mathcal{R}_0 \rightarrow 1^+$, where $I^* \rightarrow 0$ and the margin (17) itself vanishes—remains open and is not claimed here. This is in contrast to other recent fractional-order epidemic models carrying delays, in which an incubation or immunity delay beyond a critical value does trigger a Hopf bifurcation [22]; whether such a bifurcation arises is therefore a structural property of the particular model rather than a generic consequence of introducing delays.

Remark 5 (Relation to the integer-order model). Setting $\alpha = 1$ reduces (2) to a classical delayed SIRS system. The delay-independent local stability of E_1 obtained in Theorem 3 is then the fractional-order counterpart of the integer-order results of Kaddar [11] and Bashier and Patidar [1], in which the endemic equilibrium of a delayed SIR model with saturated incidence is stable for all values of the incubation delay whenever $\mathcal{R}_0 > 1$. The present analysis shows that this robustness is not an artefact of the first-order formulation: it survives the passage to $\alpha \in (0, 1)$ and the enlargement of the delay set to $\{\tau, \delta, \xi\}$ with waning immunity. Moreover, the integer-order case $\alpha = 1$ is recovered as the uppermost curve of Figure 6; it is the least stable of the orders shown, so the memory introduced by the Caputo derivative is compatible with—indeed mildly reinforces—the delay-independent stability inherited from the integer-order model.

Remark 6 (Role of the fractional order). Decreasing α rotates the boundary rays $\lambda = \omega e^{\pm i\alpha\pi/2}$ toward the positive real axis, enlarging the stability sector, while leaving the margin (17) at $\omega = 0$ unchanged. In this model the fractional order therefore acts as a mild *stabilising* influence rather than a destabilising one, a point confirmed by the rightmost-root computation of Section 7.

The local analysis above is complemented by the global behaviour observed throughout the feasible region Ω . For $\mathcal{R}_0 < 1$ the disease-free equilibrium is the only equilibrium in Ω and, by Proposition 2 together with the boundedness of Theorem 2, attracts all trajectories; the numerical experiments of Section 7 confirm global convergence to E_0 . For $\mathcal{R}_0 > 1$ the endemic equilibrium is the unique interior equilibrium, it is locally asymptotically stable for all delays by Theorem 3, and every trajectory initiated in the interior of Ω is observed to converge to it. A fully rigorous global Lyapunov argument for the endemic state in the presence of three delays and a fractional order is delicate—the construction of Volterra-type functionals for fractional delay systems remains an active topic [24, 15, 10]—and is left to future work; the combination of delay-independent local stability, uniqueness of the interior equilibrium and the extensive simulations reported below provides strong evidence for global asymptotic stability of E_1 when $\mathcal{R}_0 > 1$.

6. A DELAY-ADAPTED FRACTIONAL PREDICTOR–CORRECTOR SCHEME

For the numerical integration of (2) we use the fractional Adams–Bashforth–Moulton predictor–corrector method of Diethelm, Ford and Freed [8], adapted to the delays by interpolation of the stored history; comparable schemes for fractional-order systems of delay differential equations have been developed more recently [14]. Write the system as ${}_0^C D_t^\alpha y(t) = f(t, y(t), y_\tau, y_\delta, y_\xi)$ with $y = (S, I, R)^\top$ and $y_\vartheta = y(t - \vartheta)$. On a uniform grid $t_n = nh$, denote by y_n the approximation to $y(t_n)$ and by $\hat{y}(t_n - \vartheta)$ the piecewise-linear interpolant of the stored solution (equal to the history (3) for $t_n - \vartheta \leq 0$). The predictor and corrector are

$$(18) \quad y_{n+1}^P = y_0 + \frac{1}{\Gamma(\alpha)} \sum_{j=0}^n b_{j,n+1} f_j, \quad b_{j,n+1} = \frac{h^\alpha}{\alpha} [(n+1-j)^\alpha - (n-j)^\alpha],$$

$$(19) \quad y_{n+1} = y_0 + \frac{h^\alpha}{\Gamma(\alpha+2)} \left[f(t_{n+1}, y_{n+1}^P, \hat{y}_\bullet) + \sum_{j=0}^n a_{j,n+1} f_j \right],$$

where $f_j = f(t_j, y_j, \hat{y}(t_j - \tau), \hat{y}(t_j - \delta), \hat{y}(t_j - \xi))$ and the corrector weights are

$$a_{j,n+1} = \begin{cases} n^{\alpha+1} - (n-\alpha)(n+1)^\alpha, & j=0, \\ (n-j+2)^{\alpha+1} + (n-j)^{\alpha+1} - 2(n-j+1)^{\alpha+1}, & 1 \leq j \leq n. \end{cases}$$

The delayed states required by the predictor lie strictly in the past and are therefore available from the stored solution; when $t_{n+1} - \vartheta < 0$ the history function is used. The scheme is summarised in Algorithm 1. For $\alpha = 1$ it reduces to the classical Adams–Bashforth–Moulton method for delay differential equations, and we use this limit for validation.

7. NUMERICAL EXPERIMENTS

Unless stated otherwise the parameters are those of Table 2, chosen so that both a sub- and a super-threshold regime are available while keeping the demographic quantities in biologically sensible proportions. With $\beta = 0.009$ one has $\mathcal{R}_0 = 3$ and the endemic equilibrium $E_1 = (S^*, I^*, R^*) = (33.33, 37.04, 29.63)$, for which $S^* + I^* + R^* = 100 = \Lambda/\mu$; with $\beta = 0.002$ one has $\mathcal{R}_0 = 0.667 < 1$ and $E_0 = (100, 0, 0)$. The step size used to produce Figures 2 to 6 is $h = 0.1$ (with $h = 0.15$ for the longest integrations).

Algorithm 1: Delay-adapted fractional PECE step**Input:** order α , step h , horizon T , delays τ, δ, ξ , history ϕ Initialise $y_0 = \phi(0)$ and $f_0 = f(t_0, y_0, \phi(-\tau), \phi(-\delta), \phi(-\xi))$;**for** $n = 0$ **to** $N - 1$ **do** form predictor weights $b_{j,n+1}$ and evaluate $y_{n+1}^P = y_0 + \Gamma(\alpha)^{-1} \sum_{j \leq n} b_{j,n+1} f_j$; obtain delayed states $\hat{y}(t_{n+1} - \vartheta)$ by linear interpolation (or from ϕ if the argument is ≤ 0); form corrector weights $a_{j,n+1}$ and evaluate y_{n+1} ; store $f_{n+1} = f(t_{n+1}, y_{n+1}, \hat{y}(t_{n+1} - \tau), \hat{y}(t_{n+1} - \delta), \hat{y}(t_{n+1} - \xi))$;

Convergence of the scheme. We assess the accuracy of Algorithm 1 directly, by a self-convergence study, rather than by a single point-comparison. For the endemic parameters of Table 2 with $\tau = \delta = \xi = 2$ (the delay configuration of Figure 3) and history $\phi = (90, 5, 5)$, we solve on $[0, 30]$ at the step sizes $h = 0.4, 0.2, 0.1, 0.05, 0.025, 0.0125$ and compare each solution, in the max-norm over $[0, 30]$, against a reference solution computed at the finer step $h_{\text{ref}} = h_{\text{min}}/4 = 0.003125$; the observed order between successive halvings, $p = \log_2(e(h)/e(h/2))$, is reported in Table 3 for both $\alpha = 0.95$ and the integer-order limit $\alpha = 1$. In both cases the observed order stabilises close to $p \approx 2$, consistent with the theoretical order $\min(2, 1 + \alpha)$ of the Diethelm–Ford–Freed predictor–corrector on which Algorithm 1 is based [8]. As a second, independent check, the $\alpha = 1$ solution of Algorithm 1 was compared against a classical fourth-order Runge–Kutta integration of the same delay system (with the same piecewise-linear history interpolation); at $h = 0.05$ the two agree to within 5×10^{-4} in max-norm, and both converge to the same equilibrium. These checks corroborate—without presupposing—the scheme’s accuracy, and we regard it as validated for the purposes of Section 7.

TABLE 2. Baseline parameter values used in the simulations.

Λ	μ	σ	γ	β (endemic)	β (disease-free)
10	0.10	0.20	0.15	0.009	0.002

TABLE 3. Self-convergence of the delay-adapted fractional PECE scheme (Algorithm 1).

h	$\alpha = 0.95$		$\alpha = 1.00$	
	$e(h)$	p	$e(h)$	p
0.4	3.04×10^{-2}	—	3.51×10^{-2}	—
0.2	7.81×10^{-3}	1.96	8.74×10^{-3}	2.01
0.1	2.01×10^{-3}	1.96	2.18×10^{-3}	2.01
0.05	5.15×10^{-4}	1.96	5.42×10^{-4}	2.01
0.025	1.31×10^{-4}	1.98	1.34×10^{-4}	2.02
0.0125	3.21×10^{-5}	2.03	3.19×10^{-5}	2.07

Figure 2 shows that for $\mathcal{R}_0 = 0.667 < 1$ (with $\alpha = 0.95$ and $\tau = \delta = \xi = 3$) the infection dies out and the susceptible population tends to $\Lambda/\mu = 100$ while $I, R \rightarrow 0$, in agreement with Proposition 2. Figure 3 shows that for $\mathcal{R}_0 = 3 > 1$ and small delays ($\alpha = 0.95$, $\tau = \delta = \xi = 2$) the trajectory settles at E_1 , consistent with Theorem 3 and the positive margin of Proposition 3; the dashed lines in the figure mark the components of $E_1 = (33.33, 37.04, 29.63)$.

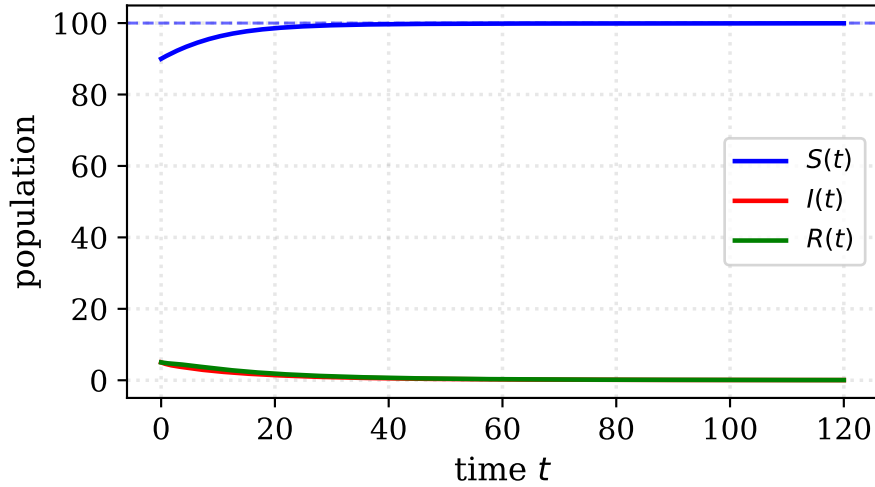


FIGURE 2. Convergence to the disease-free equilibrium.

Figure 4 superimposes the infectious component for delays $\tau = \delta = \xi \in \{1, 8, 16, 28\}$, at $\alpha = 0.95$ and $\mathcal{R}_0 = 3$. Increasing the delays lengthens and mildly bends the transient—longer memory of past states produces damped oscillations on the way to equilibrium—but every

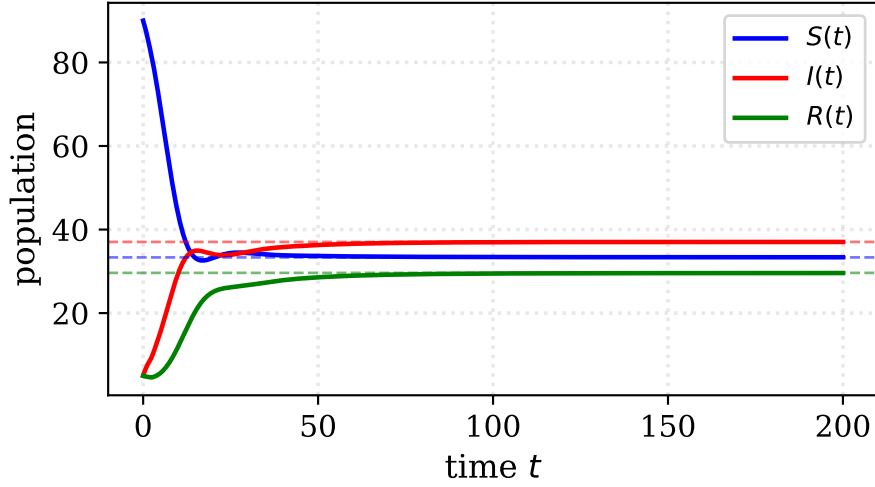


FIGURE 3. Convergence to the endemic equilibrium.

trajectory converges to the same $I^* = 37.04$. This is the numerical signature of the delay-independent stability established in Theorem 3: the delays do not create a sustained oscillation.

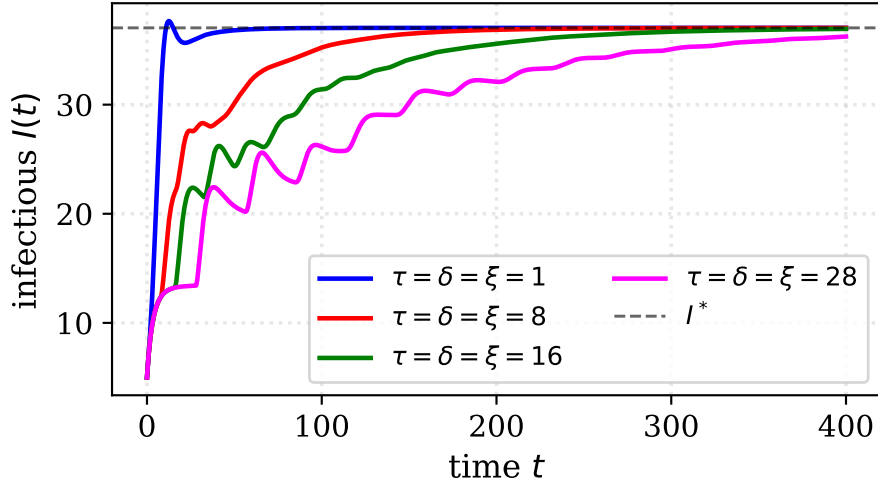
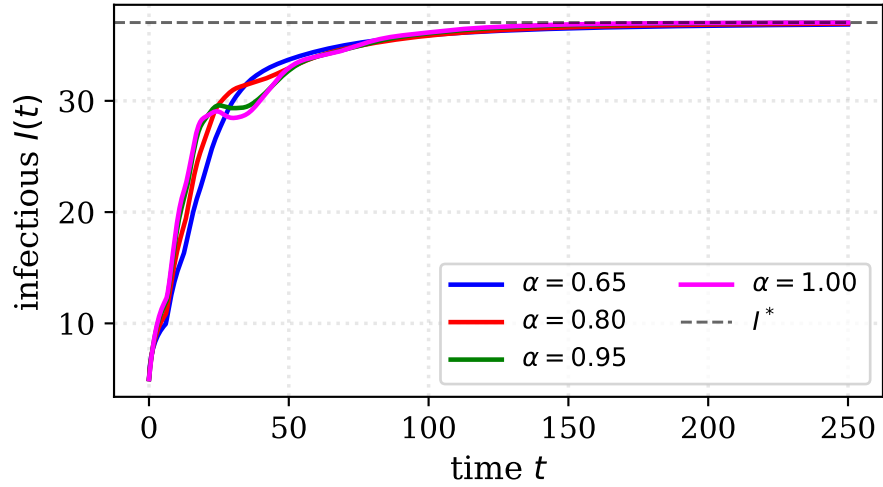
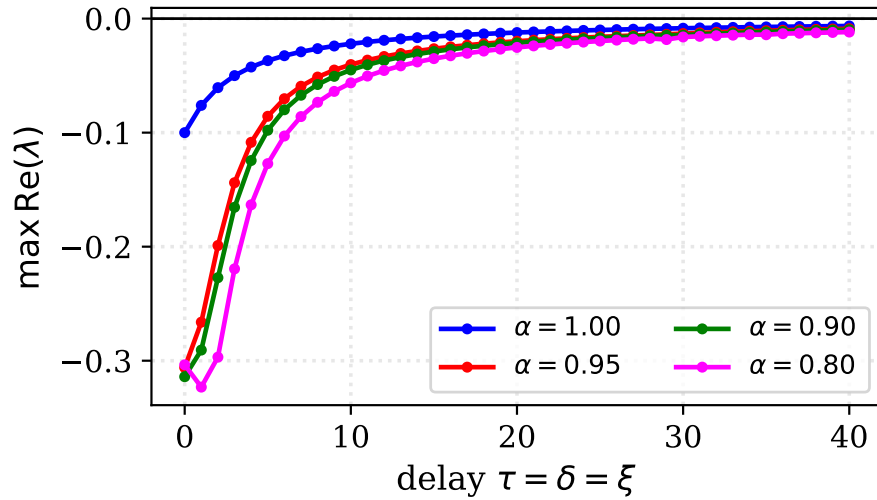
FIGURE 4. Delay robustness of the endemic equilibrium E_1 .

Figure 5 compares $\alpha \in \{0.65, 0.80, 0.95, 1.00\}$ at the fixed delay $\tau = \delta = \xi = 10$ and $\mathcal{R}_0 = 3$. The order modulates the shape and damping of the transient; smaller α yields a somewhat smoother, more strongly damped approach, in accordance with the enlargement of the stability sector noted after Proposition 3. The effect on the limiting state is nil, as it must be, since the equilibria are independent of α .

FIGURE 5. Effect of the fractional order α on the transient.

Finally, Figure 6 tracks the real part of the rightmost root of (12) as a function of the common delay $\tau = \delta = \xi$, for several orders α . The rightmost real part remains strictly negative throughout $\tau \in [0, 40]$ —so the roots stay in the open left half-plane, hence a fortiori in the stability sector $|\arg \lambda| > \alpha\pi/2$ —and it is more negative for smaller α . This directly visualises both the delay-independent stability of Theorem 3 and the stabilising role of the fractional order. The margin $|P| - |Q| - |R|$ on the boundary ray, evaluated over $\gamma \in [0.15, 4]$, $\mathcal{R}_0 \in [1.5, 20]$ and $\alpha \in [0.8, 1]$, was found to be positive in every case, confirming condition (16) numerically.

FIGURE 6. Real part of the rightmost characteristic root as a function of the common delay $\tau = \delta = \xi$.

8. CONCLUSION

We analysed a Caputo fractional SIRS model with waning immunity and three biologically distinct delays. After establishing well-posedness, deriving the basic reproduction number and correcting the closed form of the endemic equilibrium, we showed that the characteristic equation depends on the delays only through τ and the loop delay $\rho = \tau + \delta + \xi$, and proved that the endemic equilibrium is delay-independently locally asymptotically stable under an explicit condition on the characteristic coefficients that holds analytically at the sector vertex—where it yields the closed-form, always-positive margin $\beta I^* \mu(\mu + \sigma + \gamma)$ —and that we verified numerically over an extensive parameter window elsewhere. The disease-free equilibrium is stable for all delays when $\mathcal{R}_0 < 1$. Extensive simulations with a delay-adapted fractional predictor–corrector scheme, whose second-order accuracy was confirmed by a self-convergence study, confirm the theory: delays lengthen transients but do not destabilise the endemic state, and a smaller fractional order is mildly stabilising.

For the present model structure the delays do not induce sustained oscillations. This is consistent with the integer-order behaviour of closely related delayed SIR models, whose endemic equilibrium is stable for all delays when $\mathcal{R}_0 > 1$ [11, 1], and shows that the property survives the passage to fractional order and to the enlarged set of delays. It stands, nonetheless, against the expectation—common in the broader delay-epidemic literature—that sufficiently long delays generically induce Hopf bifurcations; genuine oscillatory dynamics, when sought, must be built in through additional structure. The analysis indicates where such dynamics might arise. Three modifications are natural candidates and are the subject of ongoing work: (i) incorporating the survival factor $e^{-\mu\tau}$ of Remark 1, which redistributes the delay dependence of the characteristic equation; (ii) replacing the bilinear incidence βSI by a saturating incidence of the form $\beta SI/(1 + \alpha_1 S + \alpha_2 I)$, as in the integer-order models of [11, 1], or by another nonlinear response, which alters the low-frequency behaviour of P, Q, R ; and (iii) allowing delay-dependent coefficients, for which the geometric stability-switch machinery of Beretta and Kuang [2] becomes applicable. A particularly natural continuation is (iv) the incorporation of vaccination and treatment controls, each with its own activation delay, into the fractional model, thereby lifting the integer-order optimal-control analysis of Bashier and Patidar [1] to the Caputo setting, in the

spirit of recent fractional-order delayed models incorporating vaccination and treatment [21]. On the analytical side, three gaps identified above remain open and are also directions we intend to pursue: a fully analytic proof that condition (16) holds away from the sector vertex, for every $\omega > 0$ and not only over the parameter window sampled numerically in Section 7, as flagged in Section 5.3; a self-contained verification of the fractional Halanay-type hypotheses invoked in the proof of Theorem 2, yielding an explicit delay-dependent transient bound in place of the asymptotic one used here; and a rigorous global Lyapunov analysis of the endemic equilibrium for the fractional delay system. The calibration of the model against outbreak data is a further direction we intend to pursue.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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