



Research article

Backward bifurcation in a seasonal conjunctivitis model

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Abstract: This study develops a non-autonomous compartmental model for conjunctivitis transmission with seasonal forcing, treatment failure, temporary immunity, modified infectiousness during treatment, and limited treatment capacity. The model is shown to be mathematically well posed, and the disease threshold is characterized using the periodic next-generation approach. The analysis shows that the disease-free periodic solution is locally stable below the seasonal threshold and unstable above it, while uniform persistence and the existence of at least one positive periodic solution are established when the threshold is exceeded. Although saturated treatment does not affect the local invasion threshold when the baseline treatment rate is fixed, it changes the nonlinear dynamics and can generate backward bifurcation under specified parameter conditions. In the autonomous limiting case, this mechanism is confirmed through an explicit endemic branch calculation, numerical equilibrium-stability checks, and continuation methods; backward branching can also occur when treated individuals are noninfectious under sufficient saturation. A comparison argument establishes global elimination under a sufficient condition. In the seasonal model, simulations from multiple initial conditions provide evidence of subthreshold bistability, where disease extinction and persistent seasonal infection may coexist below the invasion threshold depending on the initial state. The simulation-estimated transition is not an exact seasonal fold. These results show that reducing transmission below the invasion threshold alone may not guarantee global elimination when treatment capacity is limited. The numerical findings describe assumed scenarios rather than outbreak-calibrated predictions.

Keywords: conjunctivitis; seasonal epidemic model; saturated treatment; treatment failure; periodic reproduction number; backward bifurcation; bistability; MatCont

1. Introduction

Compartmental epidemic models provide a rigorous mathematical framework for describing disease transmission, identifying threshold parameters, and understanding qualitative epidemic behavior [1–3]. In many classical formulations, treatment is assumed to occur at a constant per-capita rate, transmission is taken to be time-independent, and clinical outcomes are represented through simplified recovery pathways. Although such assumptions are useful for analytical tractability, real outbreaks may also be influenced by environmental variability, heterogeneous clinical responses, imperfect treatment, and limitations in healthcare capacity. These mechanisms can modify epidemic thresholds and may generate nonlinear phenomena such as multiple endemic states, recurrent oscillations, persistence, or backward bifurcation.

Conjunctivitis, commonly known as “pink eye” or “bloodshot eyes”, is an inflammation of the conjunctiva and may present with redness, irritation, tearing, discharge, or a gritty sensation [4]. Allergic conjunctivitis includes acute seasonal and perennial forms, emphasizing the relevance of seasonal triggers in ocular inflammation [5]. Acute hemorrhagic conjunctivitis is highly contagious and is characterized by rapid onset and a short incubation period [6]. Environmental variability has also been incorporated directly into conjunctivitis transmission models; in particular, rainfall has been investigated as a driver of transmission dynamics [7].

The mathematical modeling of conjunctivitis has developed from classical deterministic models to frameworks incorporating data fitting, nonlinear incidence, indirect transmission, fractional operators, stochasticity, public-health interventions, optimal control, fuzzy logic, delay effects, and bifurcation analysis. Early modeling of acute hemorrhagic conjunctivitis used outbreak data from Mexico to estimate transmission dynamics and examine the effects of underreporting and behavioral changes [8]. Threshold quantities and equilibrium stability have subsequently been central components of conjunctivitis models [9, 10]. Direct and indirect transmission routes, together with optimal-control strategies, have also been considered [11]. More recent studies have used fractional-order formulations and stochastic effects to represent memory, uncertainty, and media awareness [12, 13]. Public-health education and optimal-control interventions have been investigated in data-supported conjunctivitis models [14, 15]. Fuzzy logic and time-delay effects have also been incorporated to represent uncertainty and latent processes [16]. Bifurcation and deterministic–stochastic approaches have further expanded the analysis of conjunctivitis and adenovirus transmission [17, 18].

Beyond conjunctivitis, advanced mathematical methods continue to broaden the analysis of infectious-disease systems. Fractional epidemic models based on nonclassical operators have been used to incorporate memory effects and real-data dynamics [19, 20]. Fractional-order delay SIR dynamics have also been investigated using a Mittag–Leffler kernel [21]. Stability and sensitivity have been analyzed in a fractional Chlamydia transmission model [22]. Residual power series methods provide a complementary semi-analytical tool for nonlinear reaction–diffusion–convection systems [23]. Discrete quarantine/isolation dynamics have been studied through mathematical analysis [24], while a delayed nonlinear population model has been examined for bifurcation and chaotic behavior [25]. Optimal control and real-data parameterization have also been used to evaluate intervention strategies in epidemic models [26, 27]. Of particular relevance to the present work, limited treatment capacity has long been recognized as a mechanism capable of producing backward bifurcation [28]. Backward bifurcation has also been established for models with saturated treatment

functions [29], while imperfect interventions can likewise generate subthreshold endemic behavior [30]. The center-manifold framework of Castillo-Chavez and Song provides methodological context for determining the direction of local epidemic bifurcations [31].

Despite this substantial progress, the combined effects of two mechanisms remain insufficiently explored in conjunctivitis modeling. The first is seasonal forcing. Many existing models assume constant transmission, although infection risk may vary with environmental conditions, population mixing, human behavior, and pathogen survival. For conjunctivitis specifically, rainfall-dependent transmission has already been examined, supporting the biological relevance of environmentally varying contact or transmission conditions [7]. These observations motivate the periodic transmission rate specified in Section 2, with baseline rate β_0 , forcing amplitude η , and period $\mathcal{T}$.

The second mechanism is limited treatment capacity. Many epidemic models assume that the treatment rate increases linearly with the number of infectious individuals, which implicitly represents unlimited treatment resources. During large outbreaks, however, treatment delivery may become constrained by finite clinical capacity. Models with saturated treatment have shown that such constraints can produce backward bifurcation and additional nonlinear behavior [28, 29]. More recent saturated-treatment models also exhibit saddle-node and Hopf bifurcations and sustained oscillatory dynamics [32]. Accordingly, the treatment function specified in Section 2 uses the baseline rate κ and saturation parameter ϖ . Linear treatment is recovered when $\varpi = 0$, while $\varpi > 0$ represents constrained treatment capacity in the model.

In this paper, we develop a non-autonomous susceptible–exposed–infectious–treated–treatment-failure–recovered (SEITFR) model for conjunctivitis transmission that incorporates seasonal forcing, treatment failure, temporary immunity, modified infectiousness during treatment, and saturated treatment capacity. Treated individuals are allowed to transmit infection at a reduced rate, while the treatment-failure class is assumed to be non-infectious due to isolation, reduced contact, or advanced clinical management. This structure allows the model to capture both epidemiological transmission and clinical limitations during large outbreaks.

Because the transmission rate is periodic, the model is non-autonomous and the classical autonomous next-generation matrix is not sufficient to characterize disease invasion. We therefore define the seasonal reproduction number using the periodic next-generation operator [33]. This threshold determines the local stability of the disease-free periodic solution: The disease-free periodic solution is locally asymptotically stable when the seasonal reproduction number is less than one and unstable when it is greater than one. Furthermore, when the seasonal threshold exceeds one, uniform persistence is established and the model admits at least one positive seasonal periodic solution.

We examine the combined effects of seasonal transmission and limited treatment in the proposed conjunctivitis model, building on established work on saturated-treatment bifurcation and periodic next-generation theory. Saturation leaves the local invasion threshold unchanged but, under derived parameter conditions, produces an autonomous backward branch with a lower fold. The explicit branch, eigenvalue calculations, and numerical continuation distinguish subthreshold coexistence from demonstrated local stability; an ablation also examines whether treated infectiousness is necessary for backward branching. For the seasonal model, we establish a sufficient global-elimination condition and use simulations from multiple initial states to investigate basin-dependent outcomes. The resulting finite-horizon, initial-state-specific transition estimate is distinguished from the uncomputed fold of seasonal periodic orbits. Thus, a reproduction number

below one guarantees local disease-free stability, but does not by itself establish global elimination outside the sufficient region. Numerical parameter choices are exploratory scenarios, not an outbreak calibration.

The remainder of the paper is organized as follows. Section 2 presents the model and its basic mathematical properties. Section 3 develops the periodic invasion threshold, comparison bounds, and persistence analysis. Section 4 derives the autonomous endemic branch, lower fold, and sufficient global-elimination condition. Section 5 formulates the seasonal Poincaré-map framework, a sufficient extinction criterion, and the simulation-based transition algorithm. Section 6 connects these results to the reproduction-number calculations, equilibrium eigenvalues and MatCont continuation, seasonal basin experiments, mechanism ablation, robustness analysis, and finite-horizon transition estimates. Section 7 summarizes the findings, limitations, and remaining questions.

2. Model formulation and basic properties

We investigate the transmission dynamics of conjunctivitis in a periodically varying environment. At time t , the total human population is denoted by $N(t)$ and is subdivided into six epidemiological compartments: susceptible individuals $S(t)$, exposed individuals $E(t)$, infectious individuals $I(t)$, treated individuals $T(t)$, individuals experiencing treatment failure $F(t)$, and temporarily immune recovered individuals $R(t)$. Hence,

$$N(t) = S(t) + E(t) + I(t) + T(t) + F(t) + R(t). \quad (1)$$

Susceptible individuals are recruited into the population at the constant rate $\Pi > 0$, and all individuals are subject to natural mortality at the rate $\mu > 0$. Disease transmission is governed by the time-dependent force of infection

$$\lambda(t) = \frac{\beta(t)(I(t) + \epsilon T(t))}{N(t)}, \quad (2)$$

which is defined on the biologically feasible region where $N(t) > 0$. Here, $\epsilon \in (0, 1]$ represents the relative infectiousness of treated individuals. When $\epsilon < 1$, treatment reduces infectiousness, whereas $\epsilon = 1$ indicates that treated and untreated infectious individuals have equal infectiousness. The limiting case $\epsilon = 0$ is considered separately in Sections 4 and 6; the persistence proof in Section 3 assumes $\epsilon > 0$.

The transmission rate is assumed to vary periodically in time according to

$$\beta(t) = \beta_0 \left(1 + \eta \cos \left(\frac{2\pi t}{\mathcal{T}} \right) \right), \quad (3)$$

where $\beta_0 > 0$ is the baseline transmission intensity, $\eta \in [0, 1)$ is the amplitude of seasonal forcing, and $\mathcal{T} = 365$ days is the seasonal period. Since $0 \leq \eta < 1$, it follows that

$$\beta_0(1 - \eta) \leq \beta(t) \leq \beta_0(1 + \eta), \quad (4)$$

and therefore $\beta(t)$ remains strictly positive for all $t \geq 0$.

To incorporate limited healthcare capacity, the transition from the infectious class $I(t)$ to the treated class $T(t)$ is modeled by the saturated treatment function

$$h(I) = \frac{\kappa I}{1 + \varpi I}, \quad (5)$$

where $\kappa > 0$ is the baseline treatment rate and $\varpi \geq 0$ is the treatment saturation parameter. If $\varpi = 0$, then

$$h(I) = \kappa I,$$

and treatment occurs linearly with respect to the number of infectious individuals. If $\varpi > 0$, then $h(I)$ is increasing and bounded, with

$$\lim_{I \rightarrow \infty} h(I) = \lim_{I \rightarrow \infty} \frac{\kappa I}{1 + \varpi I} = \frac{\kappa}{\varpi}.$$

Thus, for $\varpi > 0$, the quantity κ/ϖ represents the asymptotic maximum treatment capacity.

The parameter ϖ has unit population^{-1} . Its biological interpretation can be clarified using the half-saturation level. For $\varpi > 0$, the treatment function reaches one half of its asymptotic maximum value when

$$h(I) = \frac{1}{2} \frac{\kappa}{\varpi}.$$

Substituting $h(I) = \kappa I / (1 + \varpi I)$ gives

$$\frac{\kappa I}{1 + \varpi I} = \frac{\kappa}{2\varpi}.$$

Since $\kappa > 0$, this is equivalent to

$$\frac{I}{1 + \varpi I} = \frac{1}{2\varpi}.$$

Hence,

$$2\varpi I = 1 + \varpi I,$$

and therefore

$$I_{1/2} = \frac{1}{\varpi}. \quad (6)$$

Consequently, larger values of ϖ correspond to lower half-saturation levels and hence stronger limitations in treatment capacity. For example, $\varpi = 0.01$ corresponds to half-saturation at $I = 100$ infectious individuals, whereas $\varpi = 0.25$ corresponds to half-saturation at $I = 4$ infectious individuals, representing a highly constrained treatment-capacity scenario. When $\varpi = 0$, no finite half-saturation level exists because the treatment function is linear and unbounded.

The remaining transitions are described as follows. Exposed individuals become infectious at rate Φ . Infectious individuals recover spontaneously at rate α_1 . Treated individuals recover at rate α_2 or move to the treatment-failure class at rate Ψ . Individuals in the treatment-failure class recover at rate α_3 . Recovered individuals lose temporary immunity at rate δ and return to the susceptible class. Individuals in the treatment-failure class are assumed to be non-infectious due to isolation, reduced contact, or advanced symptom management.

Combining these assumptions yields the following non-autonomous periodic system:

$$\begin{cases} \frac{dS}{dt} = \Pi - \lambda(t)S - \mu S + \delta R, \\ \frac{dE}{dt} = \lambda(t)S - (\Phi + \mu)E, \\ \frac{dI}{dt} = \Phi E - \frac{\kappa I}{1 + \varpi I} - (\alpha_1 + \mu)I, \\ \frac{dT}{dt} = \frac{\kappa I}{1 + \varpi I} - (\alpha_2 + \Psi + \mu)T, \\ \frac{dF}{dt} = \Psi T - (\alpha_3 + \mu)F, \\ \frac{dR}{dt} = \alpha_1 I + \alpha_2 T + \alpha_3 F - (\mu + \delta)R. \end{cases} \quad (7)$$

Write the state vector as

$$X(t) = (S(t), E(t), I(t), T(t), F(t), R(t))^T. \quad (8)$$

The biological descriptions and units of the model parameters are summarized in Table 1.

Table 1. Description and units of the model parameters.

Parameter	Description	Unit
Π	Recruitment rate of susceptible individuals	population day ⁻¹
μ	Natural mortality rate	day ⁻¹
β_0	Baseline transmission intensity	day ⁻¹
ϵ	Relative infectiousness of treated individuals	dimensionless
Φ	Progression rate from exposed to infectious class	day ⁻¹
κ	Baseline treatment rate	day ⁻¹
Ψ	Treatment-failure rate	day ⁻¹
α_1	Spontaneous recovery rate of infectious individuals	day ⁻¹
α_2	Recovery rate of treated individuals	day ⁻¹
α_3	Recovery rate of treatment-failure individuals	day ⁻¹
δ	Rate of loss of temporary immunity	day ⁻¹
ϖ	Treatment saturation parameter	population ⁻¹
η	Seasonal forcing amplitude	dimensionless
$\mathcal{T}$	Seasonal period	days

2.1. Positivity of solutions

Theorem 1. Let $X(0) = (S(0), E(0), I(0), T(0), F(0), R(0))^T \in \mathbb{R}_+^6$ with $N(0) > 0$. Then every solution of system (7) remains nonnegative on its maximal forward existence interval $0 \leq t < t_{\max}$. Moreover, $N(t) > 0$ on this interval.

Proof. It is sufficient to verify that the vector field is quasipositive on the boundary of the nonnegative orthant, provided $N(t) > 0$. Evaluating system (7) on the coordinate hyperplanes gives

$$\left. \frac{dS}{dt} \right|_{S=0} = \Pi + \delta R \geq \Pi > 0,$$

$$\begin{aligned}\frac{dE}{dt}\Big|_{E=0} &= \lambda(t)S \geq 0, \\ \frac{dI}{dt}\Big|_{I=0} &= \Phi E \geq 0, \\ \frac{dT}{dt}\Big|_{T=0} &= \frac{\kappa I}{1 + \varpi I} \geq 0, \\ \frac{dF}{dt}\Big|_{F=0} &= \Psi T \geq 0,\end{aligned}$$

and

$$\frac{dR}{dt}\Big|_{R=0} = \alpha_1 I + \alpha_2 T + \alpha_3 F \geq 0.$$

Thus, no solution starting in $\mathbb{R}_+^6$ can cross any coordinate hyperplane into the negative region. Hence, $\mathbb{R}_+^6$ is positively invariant as long as the solution is defined and $N(t) > 0$.

Adding the six equations in system (7) gives

$$\frac{dN}{dt} = \Pi - \mu N. \quad (9)$$

Its solution is

$$N(t) = \frac{\Pi}{\mu} + \left(N(0) - \frac{\Pi}{\mu}\right)e^{-\mu t}. \quad (10)$$

Since $N(0) > 0$, this expression implies $N(t) > 0$ on the maximal existence interval. Consequently, the force of infection remains well-defined, and the solution remains in $\mathbb{R}_+^6$ throughout that interval. $\square$

2.2. Boundedness and positively invariant region

In the population equation (9), all internal transfers cancel. Its explicit solution is given by (10). Once global existence is established in Theorem 3, Eq (10) also yields the limiting population

$$N^* := \frac{\Pi}{\mu} = \lim_{t \rightarrow \infty} N(t). \quad (11)$$

Theorem 2. Let $X(0) \in \mathbb{R}_+^6$ with $N(0) > 0$. Then $0 < N(t) \leq \mathcal{M}$, $0 \leq t < t_{\max}$, where

$$\mathcal{M} = \max \left\{ N(0), \frac{\Pi}{\mu} \right\}. \quad (12)$$

In particular, for any fixed $\mathcal{M} \geq \Pi/\mu$, the set

$$\Omega_{\mathcal{M}}^+ = \{(S, E, I, T, F, R) \in \mathbb{R}_+^6 : 0 < S + E + I + T + F + R \leq \mathcal{M}\}$$

is positively invariant for system (7) on the maximal forward existence interval.

Proof. From (10), if $N(0) > \Pi/\mu$, then $N(t)$ decreases monotonically to Π/μ . If $0 < N(0) < \Pi/\mu$, then $N(t)$ increases monotonically to Π/μ . If $N(0) = \Pi/\mu$, then $N(t) = \Pi/\mu$ on the maximal existence interval. Hence,

$$0 < N(t) \leq \mathcal{M},$$

for $0 \leq t < t_{\max}$.

Now let $\mathcal{M} \geq \Pi/\mu$ and suppose $X(0) \in \Omega_{\mathcal{M}}^+$. Then $0 < N(0) \leq \mathcal{M}$, and (10) implies $0 < N(t) \leq \mathcal{M}$ throughout the maximal existence interval. Together with Theorem 1, this shows that $\Omega_{\mathcal{M}}^+$ is positively invariant on that interval. $\square$

The smaller region

$$0 < N \leq \frac{\Pi}{\mu}$$

is positively invariant only for initial data satisfying $N(0) \leq \Pi/\mu$. For arbitrary nonnegative initial data with $N(0) > 0$, use the forward-invariant bound (12).

2.3. Existence and uniqueness

Theorem 3. *For every initial condition $X(0) \in \mathbb{R}_+^6$ with $N(0) > 0$, system (7) admits a unique solution defined for all $t \geq 0$. Moreover, the solution remains in $\Omega_{\mathcal{M}}^+$ with $\mathcal{M}$ defined in (12).*

Proof. The right-hand side of system (7) is continuous in t because $\beta(t)$ is continuous and periodic. We now verify local Lipschitz continuity with respect to the state variables on the biologically relevant domain where $N > 0$.

The only term requiring special attention is the force of infection (2). Since $N(0) > 0$, there exists a neighborhood of the initial condition on which N is bounded away from zero. On such a neighborhood, $\lambda(t)$ is continuously differentiable with respect to the state variables and is therefore locally Lipschitz.

The saturated treatment function (5) is continuously differentiable for $I \geq 0$, with derivative

$$h'(I) = \frac{\kappa}{(1 + \varpi I)^2}. \quad (13)$$

Since $1 + \varpi I \geq 1$ for $I \geq 0$ and $\varpi \geq 0$, we have

$$0 < h'(I) \leq \kappa.$$

Thus, h is Lipschitz continuous on $\mathbb{R}_+$.

It follows that the vector field is locally Lipschitz with respect to the state variables in a neighborhood of every biologically feasible point with $N > 0$. Hence, by the Picard–Lindelöf theorem, system (7) has a unique local solution.

It remains to show that this solution can be extended globally. From (10),

$$N(t) \geq \gamma := \min \left\{ N(0), \frac{\Pi}{\mu} \right\} > 0, \quad 0 \leq t < t_{\max}.$$

Thus, the denominator in the force of infection remains bounded away from zero along the solution. Furthermore, by Theorems 1 and 2, the solution remains nonnegative and bounded on its maximal existence interval. Together with $N(t) \geq \gamma > 0$, these bounds rule out a finite maximal existence time by the continuation theorem. Hence the local solution extends uniquely to all $t \geq 0$. $\square$

3. Disease-free threshold dynamics

This section derives the disease-free periodic solution and the seasonal basic reproduction number. Since the transmission rate is time-periodic, the epidemic threshold is defined using the next-generation operator for periodic epidemic systems. The autonomous threshold obtained by replacing the seasonal transmission rate with its average value is introduced only for comparison.

3.1. Disease-free periodic solution

On the disease-free boundary, the infected compartments vanish. The following theorem identifies the unique disease-free periodic solution.

Theorem 4. *System (7) admits the unique disease-free $\mathcal{T}$ -periodic solution, with N^* defined by (11):*

$$P_0 = (N^*, 0, 0, 0, 0, 0).$$

Proof. On the disease-free boundary, $E = I = T = F = 0$, and the remaining equations reduce to

$$S' = \Pi - \mu S + \delta R, \quad R' = -(\mu + \delta)R.$$

If a solution on this boundary is $\mathcal{T}$ -periodic, then $R(t)$ must also be $\mathcal{T}$ -periodic. Since

$$R(t) = R(0)e^{-(\mu+\delta)t},$$

periodicity implies $R(0) = 0$, and hence $R(t) \equiv 0$. The equation for S then becomes

$$S' = \Pi - \mu S.$$

The only $\mathcal{T}$ -periodic solution of this scalar equation is $S(t) \equiv N^*$ by (11). Thus, the unique disease-free $\mathcal{T}$ -periodic solution is P_0 . $\square$

3.2. Seasonal basic reproduction number $\mathcal{R}_0$

Let

$$x(t) = (E(t), I(t), T(t))^T \tag{14}$$

denote the vector of transmission-relevant infected compartments. The treatment-failure class $F(t)$ is excluded from this subsystem because individuals in this class are assumed to be non-infectious.

At the disease-free periodic solution P_0 from Theorem 4, $S_0 = N_0 = N^*$, so $S_0/N_0 = 1$. Thus, near P_0 , the incidence term in the exposed equation satisfies

$$\lambda(t)S = \frac{\beta(t)(I + \epsilon T)}{N}S = \beta(t)(I + \epsilon T) + \text{higher-order terms}.$$

By the treatment derivative (13), $h'(0) = \kappa$. Therefore,

$$h(I) = \kappa I + O(I^2) \quad \text{as } I \rightarrow 0.$$

In particular, the saturation parameter ϖ does not appear in the linearized infected subsystem.

The linearized infected subsystem of system (7) at P_0 is therefore

$$\begin{cases} E' = \beta(t)I + \epsilon\beta(t)T - (\Phi + \mu)E, \\ I' = \Phi E - (\alpha_1 + \kappa + \mu)I, \\ T' = \kappa I - (\alpha_2 + \Psi + \mu)T. \end{cases} \quad (15)$$

This system can be written in the next-generation form

$$x' = [\mathcal{F}(t) - \mathcal{V}]x, \quad (16)$$

where

$$\mathcal{F}(t) = \begin{pmatrix} 0 & \beta(t) & \epsilon\beta(t) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad (17)$$

and

$$\mathcal{V} = \begin{pmatrix} d_1 & 0 & 0 \\ -\Phi & d_2 & 0 \\ 0 & -\kappa & d_3 \end{pmatrix}, \quad (18)$$

with

$$d_1 = \Phi + \mu, \quad d_2 = \alpha_1 + \kappa + \mu, \quad d_3 = \alpha_2 + \Psi + \mu. \quad (19)$$

Let $Y(t, s)$ denote the evolution operator generated by

$$y' = -\mathcal{V}y.$$

Since $\mathcal{V}$ is constant, we have

$$Y(t, s) = e^{-\mathcal{V}(t-s)}, \quad t \geq s.$$

Let $C_{\mathcal{T}}$ be the ordered Banach space of continuous $\mathcal{T}$ -periodic functions from $\mathbb{R}$ to $\mathbb{R}^3$, equipped with the cone of nonnegative functions. The next-generation operator $\mathcal{L} : C_{\mathcal{T}} \rightarrow C_{\mathcal{T}}$ is therefore defined by

$$(\mathcal{L}u)(t) = \int_0^\infty e^{-\mathcal{V}a} \mathcal{F}(t-a)u(t-a) da. \quad (20)$$

The seasonal basic reproduction number is defined by

$$\mathcal{R}_0 = \rho(\mathcal{L}), \quad (21)$$

where $\rho(\mathcal{L})$ denotes the spectral radius of $\mathcal{L}$.

Theorem 5. *The disease-free periodic solution P_0 of system (7) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. For $r > 0$, consider the auxiliary periodic linear system

$$z' = \left[\frac{1}{r} \mathcal{F}(t) - \mathcal{V} \right] z.$$

Let $Z_r(t)$ be the fundamental matrix solution satisfying

$$Z'_r(t) = \left[\frac{1}{r} \mathcal{F}(t) - \mathcal{V} \right] Z_r(t), \quad Z_r(0) = I_{3 \times 3}. \quad (22)$$

Then $Z_r(\mathcal{T})$ is the monodromy matrix over one period.

By the standard next-generation theory for periodic epidemic systems, if $\mathcal{R}_0 > 0$, then $r = \mathcal{R}_0$ is the unique positive solution of

$$\rho(Z_r(\mathcal{T})) = 1.$$

Moreover,

$$\mathcal{R}_0 < 1 \iff \rho(Z_1(\mathcal{T})) < 1,$$

and

$$\mathcal{R}_0 > 1 \iff \rho(Z_1(\mathcal{T})) > 1.$$

The matrix $Z_1(\mathcal{T})$ is the monodromy matrix associated with the linearized infected subsystem (16). Therefore, $\rho(Z_1(\mathcal{T})) < 1$ means that all infected Floquet multipliers lie inside the unit disk, and hence infected perturbations decay.

The remaining non-infected components are governed, at the disease-free solution, by linear equations with strictly negative decay rates, possibly forced by infected variables. Hence, when the infected variables decay, the non-infected perturbations also decay toward the disease-free periodic solution. Therefore, P_0 is locally asymptotically stable when $\mathcal{R}_0 < 1$.

If $\mathcal{R}_0 > 1$, then $\rho(Z_1(\mathcal{T})) > 1$, so at least one infected Floquet multiplier lies outside the unit disk. Hence the linearized infected subsystem has an unstable Floquet direction, and the disease-free periodic solution P_0 is unstable. $\square$

For numerical computation, define

$$g(r) = \rho(Z_r(\mathcal{T})) - 1. \quad (23)$$

Then $\mathcal{R}_0$ is the unique positive root of

$$g(r) = 0.$$

A bracketing interval can be obtained adaptively. Choose $r_{\min} > 0$ sufficiently small so that $g(r_{\min}) > 0$, and choose an initial value $r_{\max} > r_{\min}$. If $g(r_{\max}) > 0$, then increase $r_{\max}$, for example by repeated doubling, until

$$g(r_{\max}) < 0.$$

This procedure is justified because increasing r decreases the effective infection term $(1/r)\mathcal{F}(t)$ and consequently decreases $\rho(Z_r(\mathcal{T}))$.

Algorithm 1 Numerical computation of the seasonal basic reproduction number $\mathcal{R}_0$

Require: Periodic matrix $\mathcal{F}(t)$, transition matrix $\mathcal{V}$, period $\mathcal{T}$, tolerance $\text{tol} > 0$

Ensure: Seasonal reproduction number $\mathcal{R}_0$

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1: Use the fundamental system (22) and root function (23).
2: Choose  $r_{\min} > 0$  sufficiently small and compute  $g(r_{\min})$ .
3: while  $g(r_{\min}) \leq 0$  do
4:   Set  $r_{\min} = r_{\min}/2$  and recompute  $g(r_{\min})$ .
5: end while
6: Set  $r_{\max} = 1$  and compute  $g(r_{\max})$ .
7: while  $g(r_{\max}) > 0$  do
8:   Set  $r_{\max} = 2r_{\max}$ .
9:   Recompute  $g(r_{\max})$ .
10: end while
11: while  $r_{\max} - r_{\min} > \text{tol}$  do
12:   Set  $r_{\text{mid}} = (r_{\min} + r_{\max})/2$ .
13:   Compute  $g(r_{\text{mid}})$  by integrating the matrix system for  $Z_{r_{\text{mid}}}(t)$  over one period.
14:   if  $g(r_{\text{mid}}) > 0$  then
15:     Set  $r_{\min} = r_{\text{mid}}$ .
16:   else
17:     Set  $r_{\max} = r_{\text{mid}}$ .
18:   end if
19: end while
20: return  $\mathcal{R}_0 = (r_{\min} + r_{\max})/2$ .

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3.3. Averaged autonomous reproduction number and comparison bounds

Averaging the seasonal transmission rate (3) over one period gives

$$\bar{\beta} = \frac{1}{\mathcal{T}} \int_0^{\mathcal{T}} \beta(t) dt = \beta_0.$$

Replacing $\beta(t)$ by $\bar{\beta} = \beta_0$ in the infected subsystem (16) gives the autonomous linearization with transition matrix (18) and new-infection matrix

$$\mathcal{F}_{\text{avg}} = \begin{pmatrix} 0 & \beta_0 & \epsilon\beta_0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \quad (24)$$

The averaged autonomous reproduction number is

$$\mathcal{R}_0^{\text{avg}} = \rho(\mathcal{F}_{\text{avg}} \mathcal{V}^{-1}).$$

A direct calculation gives

$$\mathcal{F}_{\text{avg}} \mathcal{V}^{-1} = \begin{pmatrix} \frac{\beta_0 \Phi(d_3 + \epsilon\kappa)}{d_1 d_2 d_3} & \frac{\beta_0}{d_2} + \frac{\epsilon\beta_0 \kappa}{d_2 d_3} & \frac{\epsilon\beta_0}{d_3} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Since this matrix has rank one, its spectral radius is the nonzero diagonal entry. Hence,

$$\mathcal{R}_0^{\text{avg}} = \frac{\beta_0 \Phi(d_3 + \epsilon \kappa)}{d_1 d_2 d_3}. \quad (25)$$

In general,

$$\mathcal{R}_0 \neq \mathcal{R}_0^{\text{avg}},$$

when $\eta > 0$, because the true seasonal reproduction number $\mathcal{R}_0$ is defined by the periodic next-generation operator rather than by the averaged autonomous system.

From (4), the periodic new-infection matrix satisfies

$$(1 - \eta)\mathcal{F}_{\text{avg}} \leq \mathcal{F}(t) \leq (1 + \eta)\mathcal{F}_{\text{avg}}, \quad t \in \mathbb{R}. \quad (26)$$

We now justify how this pointwise matrix inequality yields comparison bounds for $\mathcal{R}_0$.

Lemma 1. *Let $\mathcal{F}_1(t)$ and $\mathcal{F}_2(t)$ be continuous $\mathcal{T}$ -periodic new-infection matrices satisfying*

$$0 \leq \mathcal{F}_1(t) \leq \mathcal{F}_2(t), \quad t \in \mathbb{R}. \quad (27)$$

Let $\mathcal{L}_1$ and $\mathcal{L}_2$ be the corresponding periodic next-generation operators on $C_{\mathcal{T}}$, defined by

$$(\mathcal{L}_i u)(t) = \int_0^\infty e^{-\mathcal{V}a} \mathcal{F}_i(t-a) u(t-a) da, \quad i = 1, 2.$$

Then

$$0 \leq \mathcal{L}_1 \leq \mathcal{L}_2.$$

Consequently,

$$\rho(\mathcal{L}_1) \leq \rho(\mathcal{L}_2).$$

Proof. From (18), we have

$$-\mathcal{V} = \begin{pmatrix} -d_1 & 0 & 0 \\ \Phi & -d_2 & 0 \\ 0 & \kappa & -d_3 \end{pmatrix}.$$

Thus, $-\mathcal{V}$ is a Metzler matrix because all its off-diagonal entries are nonnegative. Therefore,

$$e^{-\mathcal{V}a} \geq 0, \quad a \geq 0.$$

Let $u \in C_{\mathcal{T}}$ satisfy $u(t) \geq 0$ for all t . From (27), we have

$$0 \leq \mathcal{F}_1(t-a)u(t-a) \leq \mathcal{F}_2(t-a)u(t-a).$$

Multiplication by the nonnegative matrix $e^{-\mathcal{V}a}$ preserves the ordering, so

$$0 \leq e^{-\mathcal{V}a} \mathcal{F}_1(t-a)u(t-a) \leq e^{-\mathcal{V}a} \mathcal{F}_2(t-a)u(t-a).$$

Integrating over $a \in [0, \infty)$ gives

$$0 \leq (\mathcal{L}_1 u)(t) \leq (\mathcal{L}_2 u)(t).$$

Thus,

$$0 \leq \mathcal{L}_1 u \leq \mathcal{L}_2 u$$

for every nonnegative $u \in C_T$. Hence,

$$0 \leq \mathcal{L}_1 \leq \mathcal{L}_2$$

as positive linear operators.

By the monotonicity of the spectral radius for positive bounded linear operators on an ordered Banach space,

$$0 \leq \mathcal{L}_1 \leq \mathcal{L}_2 \implies \rho(\mathcal{L}_1) \leq \rho(\mathcal{L}_2).$$

□

Applying Lemma 1 to (26), we obtain

$$\rho((1 - \eta)\mathcal{L}_{\text{avg}}) \leq \rho(\mathcal{L}) \leq \rho((1 + \eta)\mathcal{L}_{\text{avg}}),$$

where $\mathcal{L}$ is the periodic next-generation operator corresponding to $\mathcal{F}(t)$, and $\mathcal{L}_{\text{avg}}$ is the next-generation operator corresponding to $\mathcal{F}_{\text{avg}}$. Using the homogeneity of the spectral radius,

$$\rho(c\mathcal{L}_{\text{avg}}) = c\rho(\mathcal{L}_{\text{avg}}), \quad c \geq 0,$$

we obtain

$$(1 - \eta)\rho(\mathcal{L}_{\text{avg}}) \leq \rho(\mathcal{L}) \leq (1 + \eta)\rho(\mathcal{L}_{\text{avg}}).$$

Since

$$\rho(\mathcal{L}) = \mathcal{R}_0, \quad \rho(\mathcal{L}_{\text{avg}}) = \mathcal{R}_0^{\text{avg}},$$

it follows that

$$(1 - \eta)\mathcal{R}_0^{\text{avg}} \leq \mathcal{R}_0 \leq (1 + \eta)\mathcal{R}_0^{\text{avg}}. \quad (28)$$

These inequalities provide useful validation bounds for the numerical computation of the seasonal reproduction number. They are comparison bounds and should not be interpreted as sharp approximations, particularly when the seasonal forcing amplitude η is large.

3.4. Uniform persistence for $\mathcal{R}_0 > 1$

Let

$$\rho_0(X) = E + I + T \quad (29)$$

measure the total density of the transmission-relevant infected compartments. In the biologically relevant state space $\mathcal{D} = \{X \in \mathbb{R}_+^6 : N > 0\}$, define the disease-free boundary by

$$\partial\Omega_0 = \{X \in \mathcal{D} : \rho_0(X) = 0\} = \{X \in \mathcal{D} : E = I = T = 0\}.$$

The disease-present region is

$$\Omega_0 = \mathcal{D} \setminus \partial\Omega_0 = \{X \in \mathcal{D} : E + I + T > 0\}.$$

For the persistence argument, consider the compact set

$$\mathcal{K} = \left\{ X \in \mathbb{R}_+^6 : \frac{\Pi}{2\mu} \leq N \leq \frac{2\Pi}{\mu} \right\}.$$

Since $N' = \Pi - \mu N$, the set $\mathcal{K}$ is positively invariant and absorbs every solution with $N(0) > 0$ after a finite time. We apply the Poincaré map to $\mathcal{K}$ and use the disease-present region $\mathcal{K} \cap \Omega_0$ and its disease-free boundary $\mathcal{K} \cap \partial\Omega_0$.

Theorem 6. *If $\mathcal{R}_0 > 1$, then system (7) is uniformly persistent with respect to the transmission-relevant infected compartments. In particular, there exists $\varepsilon_p > 0$ such that every solution starting in Ω_0 satisfies*

$$\liminf_{t \rightarrow \infty} \rho_0(X(t)) \geq \varepsilon_p.$$

Moreover, there exist positive constants ε_E , ε_I , and ε_T such that

$$\liminf_{t \rightarrow \infty} E(t) \geq \varepsilon_E, \quad \liminf_{t \rightarrow \infty} I(t) \geq \varepsilon_I, \quad \liminf_{t \rightarrow \infty} T(t) \geq \varepsilon_T.$$

Furthermore, system (7) admits at least one $\mathcal{T}$ -periodic solution with $E(t), I(t), T(t) > 0$ for all t .

Proof. Since system (7) is $\mathcal{T}$ -periodic, we use the state vector (8) to define the one-period Poincaré map

$$\mathcal{P} : \mathcal{K} \rightarrow \mathcal{K}, \quad \mathcal{P}(X_0) = X(\mathcal{T}; X_0),$$

where $X(\mathcal{T}; X_0)$ is the solution evaluated after one full period with initial condition $X(0) = X_0$. By positivity, boundedness, and continuous dependence on initial data, $\mathcal{P}$ is well defined and continuous on $\mathcal{K}$.

The disease-free boundary $\partial\Omega_0$ is positively invariant. Indeed, if $E = I = T = 0$, then $\lambda(t) = 0$ and $h(0) = 0$. Therefore, the equations for F and R reduce to

$$F' = -(\alpha_3 + \mu)F, \quad R' = \alpha_3 F - (\mu + \delta)R.$$

Hence,

$$F(t) \rightarrow 0, \quad R(t) \rightarrow 0 \quad \text{as } t \rightarrow \infty.$$

The equation for S then approaches

$$S' = \Pi - \mu S,$$

and consequently

$$S(t) \rightarrow \frac{\Pi}{\mu}.$$

Every disease-free boundary trajectory therefore converges to the disease-free state P_0 defined in Theorem 4.

When $\mathcal{R}_0 > 1$, Theorem 5 implies that P_0 is unstable with respect to infected perturbations. We now verify that no disease-present solution can converge to P_0 . Suppose that $\mathcal{P}^n(X_0) \rightarrow P_0$ for some $X_0 \in \mathcal{K} \cap \Omega_0$. Continuous dependence over one period gives $X(t; X_0) \rightarrow P_0$ as $t \rightarrow \infty$. The model equations and $\Pi, \Phi, \kappa, \epsilon > 0$ imply that $E(t), I(t), T(t) > 0$ for all sufficiently large t .

Choose $0 < \zeta < 1$ and $r > 0$. Since $S(t)/N(t) \rightarrow 1$ and $I(t) \rightarrow 0$, for sufficiently large $t_0 = m\mathcal{T}$,

$$\frac{S(t)}{N(t)} \geq 1 - \zeta, \quad I(t) \leq r, \quad t \geq t_0.$$

Using $h(I) \leq \kappa I$ and $h(I) \geq \kappa I/(1 + \varpi r)$ when $I \leq r$, the infected vector (14) satisfies the componentwise differential inequality

$$x'(t) \geq A_{\zeta,r}(t)x(t), \quad A_{\zeta,r}(t) = \begin{pmatrix} -d_1 & (1 - \zeta)\beta(t) & (1 - \zeta)\epsilon\beta(t) \\ \Phi & -d_2 & 0 \\ 0 & \frac{\kappa}{1 + \varpi r} & -d_3 \end{pmatrix}.$$

As $(\zeta, r) \rightarrow (0, 0)$, $A_{\zeta,r}(t)$ converges uniformly to $\mathcal{F}(t) - \mathcal{V}$. Since $\mathcal{R}_0 > 1$, the monodromy matrix of the latter system has spectral radius greater than one. By continuity, $\zeta, r > 0$ can be chosen so that the monodromy matrix $M_{\zeta,r}$ of $y' = A_{\zeta,r}(t)y$ satisfies $\rho(M_{\zeta,r}) > 1$. This cooperative system is irreducible, and hence $M_{\zeta,r}$ has a strictly positive Perron eigenvector v . Since $x(t_0) \gg 0$, choose $c > 0$ with $cv \leq x(t_0)$. The comparison principle and periodicity then give

$$x(t_0 + n\mathcal{T}) \geq cM_{\zeta,r}^n v = c\rho(M_{\zeta,r})^n v, \quad n \geq 0.$$

This contradicts boundedness. Thus,

$$W_{\varphi}^s(P_0) \cap (\mathcal{K} \cap \Omega_0) = \emptyset.$$

The same comparison shows that no infected complete orbit can remain in a sufficiently small neighborhood of P_0 for all forward time; on the disease-free boundary, the only complete orbit contained in $\mathcal{K}$ for all positive and negative times is P_0 . Hence P_0 is isolated as an invariant set in $\mathcal{K}$.

The boundary invariant set $\{P_0\}$ is isolated and acyclic. Together with the stable-set exclusion above, the acyclicity theorem for uniform persistence of compact maps [34] applies on $\mathcal{K}$. Consequently, there exists $\varepsilon_d > 0$ such that

$$\liminf_{n \rightarrow \infty} \rho_0(\mathcal{P}^n(X_0)) \geq \varepsilon_d$$

for every $X_0 \in \mathcal{K} \cap \Omega_0$. For continuous time, summing the infected equations gives $\rho'_0 \geq -C\rho_0$, where $C = \max\{\mu, \alpha_1 + \mu, d_3\}$. Hence $\rho_0(t) \geq e^{-C\mathcal{T}}\rho_0(n\mathcal{T})$ for $n\mathcal{T} \leq t \leq (n+1)\mathcal{T}$. Since $\mathcal{K}$ absorbs all positive-population solutions, this implies

$$\liminf_{t \rightarrow \infty} \rho_0(X(t)) \geq \varepsilon_p := e^{-C\mathcal{T}}\varepsilon_d > 0.$$

It remains to note that the infected subsystem is strongly coupled in the transmission-relevant variables. Specifically, exposed individuals progress to the infectious class at rate $\Phi > 0$, infectious individuals enter treatment at rate $\kappa > 0$, and treated individuals contribute to new infections through the term $\epsilon\beta(t)T$ with $\epsilon > 0$ and $\beta(t) > 0$. For all sufficiently large t , $\rho_0(X(t)) \geq \varepsilon_p/2$. For a fixed $\tau > 0$, every initial state in the compact set

$$\mathcal{K}_* = \{X \in \mathcal{K} : \rho_0(X) \geq \varepsilon_p/2\}$$

has strictly positive E , I , and T components after time τ , for every forcing phase, by $\Pi, \Phi, \kappa, \epsilon > 0$ and $\beta(t) > 0$. Continuity and compactness of $[0, \mathcal{T}] \times \mathcal{K}_*$ give uniform positive minima of these components

at time τ . Applying this bound to the trajectory from time $t - \tau$ establishes the componentwise bounds in Theorem 6.

Finally, $\mathcal{K}$ is compact and convex, and the disease-present set $\mathcal{K} \cap \Omega_0$ is convex, nonempty, relatively open, and positively invariant. Moreover,

$$\text{dist}(X, \mathcal{K} \cap \partial\Omega_0) \geq \sqrt{E^2 + I^2 + T^2} \geq \frac{\rho_0(X)}{\sqrt{3}},$$

so the uniform lower bound for ρ_0 implies uniform persistence in the distance-to-boundary sense. Since all solutions are globally defined and ultimately bounded in $\mathcal{K}$, Zhao's periodic-ODE coexistence theorem [35, Theorem 3.1] applies and yields at least one $\mathcal{T}$ -periodic solution with initial state $X^* \in \mathcal{K} \cap \Omega_0$. Equivalently, $\mathcal{P}(X^*) = X^*$, or $X(\mathcal{T}; X^*) = X^*$. By uniqueness of solutions and the $\mathcal{T}$ -periodicity of the vector field,

$$X(t + \mathcal{T}; X^*) = X(t; X^*) \quad \text{for all } t \geq 0.$$

Thus, system (7) admits at least one $\mathcal{T}$ -periodic solution with $E(t), I(t), T(t) > 0$ for all t . $\square$

4. Autonomous backward bifurcation mechanism

The saturated treatment term (5) does not affect the local disease-invasion threshold because $h'(0) = \kappa$ by (13). Thus, near the disease-free equilibrium, saturated treatment contributes the same linear term as ordinary linear treatment. However, when the infectious population is large, the nonlinear saturation term can substantially alter the global equilibrium structure. To isolate this nonlinear mechanism, we first consider the autonomous limiting case obtained by setting $\eta = 0$.

When $\eta = 0$, Eq (3) gives $\beta(t) = \beta_0$. In this case, the seasonal reproduction number reduces to the autonomous reproduction number, which coincides with the averaged value derived previously. Hence,

$$\mathcal{R}_0^{(0)} = \mathcal{R}_0^{\text{avg}} = \frac{\beta_0 \Phi(d_3 + \epsilon\kappa)}{d_1 d_2 d_3} = \frac{\beta_0}{\beta_0^*}, \quad \beta_0^* = \frac{d_1 d_2 d_3}{\Phi(d_3 + \epsilon\kappa)}, \quad (30)$$

where d_1, d_2 , and d_3 are defined in (19). The value β_0^* is the critical autonomous transmission intensity corresponding to $\mathcal{R}_0^{(0)} = 1$.

4.1. Endemic equilibrium branch

Theorem 7. *In the autonomous limiting system, namely system (7) with $\eta = 0$, every endemic equilibrium with $I^* > 0$ can be parameterized by the infectious component I^* . For each admissible value of $I^* > 0$ satisfying $S^*(I^*) > 0$, the corresponding equilibrium is*

$$P^*(I^*) = (S^*(I^*), E^*(I^*), I^*, T^*(I^*), F^*(I^*), R^*(I^*)),$$

where

$$E^* = \frac{1}{\Phi} \left(\alpha_1 + \mu + \frac{\kappa}{1 + \varpi I^*} \right) I^*, \quad (31)$$

$$T^* = \frac{\kappa I^*}{(1 + \varpi I^*) d_3}, \quad (32)$$

$$F^* = \frac{\Psi\kappa I^*}{(1 + \varpi I^*)d_3(\alpha_3 + \mu)}, \quad (33)$$

and

$$R^* = \frac{\alpha_1 I^* + \frac{\alpha_2 \kappa I^*}{(1 + \varpi I^*)d_3} + \frac{\alpha_3 \Psi \kappa I^*}{(1 + \varpi I^*)d_3(\alpha_3 + \mu)}}{\mu + \delta}. \quad (34)$$

Moreover,

$$S^*(I^*) = N^* - I^* \left[A + \frac{B}{1 + \varpi I^*} \right], \quad (35)$$

where

$$A = \frac{\alpha_1 + \mu}{\Phi} + 1 + \frac{\alpha_1}{\mu + \delta}, \quad (36)$$

and

$$B = \frac{\kappa}{\Phi} + \frac{\kappa}{d_3} + \frac{\Psi\kappa}{d_3(\alpha_3 + \mu)} + \frac{\alpha_2 \kappa}{d_3(\mu + \delta)} + \frac{\alpha_3 \Psi \kappa}{d_3(\alpha_3 + \mu)(\mu + \delta)}. \quad (37)$$

The corresponding value of the autonomous reproduction number along the endemic branch is

$$\mathcal{R}_0^{(0)}(I^*) = \frac{N^*(d_3 + \epsilon\kappa) [(\alpha_1 + \mu)(1 + \varpi I^*) + \kappa]}{d_2 [d_3(1 + \varpi I^*) + \epsilon\kappa] S^*(I^*)}. \quad (38)$$

Proof. At an endemic equilibrium, all derivatives in system (7) vanish and $I^* > 0$. Summing the equilibrium equations gives the population value N^* in (11).

From the I -equation, we obtain

$$0 = \Phi E^* - \frac{\kappa I^*}{1 + \varpi I^*} - (\alpha_1 + \mu)I^*,$$

which gives (31). Similarly, the T -equation yields

$$0 = \frac{\kappa I^*}{1 + \varpi I^*} - d_3 T^*,$$

and hence (32). The F -equation gives

$$0 = \Psi T^* - (\alpha_3 + \mu)F^*,$$

which yields (33). Finally, the R -equation gives (34).

Using the population identity (1) and the equilibrium value (11), we obtain

$$S^* = N^* - E^* - I^* - T^* - F^* - R^*. \quad (39)$$

Substituting (31)–(34) into (39) and collecting terms gives (35).

It remains to determine the reproduction-number value associated with a given point on the endemic branch. At equilibrium, the exposed equation satisfies

$$\frac{\beta_0(I^* + \epsilon T^*)S^*}{N^*} = d_1 E^*.$$

Using (11), this gives

$$\beta_0 = \frac{N^* d_1 E^*}{(I^* + \epsilon T^*) S^*}.$$

Substituting this expression into (30), we obtain

$$\mathcal{R}_0^{(0)}(I^*) = \frac{N^* \Phi(d_3 + \epsilon \kappa) E^*}{d_2 d_3 (I^* + \epsilon T^*) S^*}.$$

Using (31) and (32), this expression simplifies to (38).

Therefore, every positive endemic equilibrium lies on the branch described above. Conversely, any admissible value $I^* > 0$ satisfying $S^*(I^*) > 0$ and

$$\mathcal{R}_0^{(0)} = \mathcal{R}_0^{(0)}(I^*)$$

defines an endemic equilibrium of the autonomous system. $\square$

4.2. Lower autonomous critical threshold

The admissible endemic branch is determined by

$$\mathcal{I} = \{I^* > 0 : S^*(I^*) > 0\}. \quad (40)$$

Along this branch, the autonomous reproduction number is given by (38). Since

$$\lim_{I^* \rightarrow 0^+} \mathcal{R}_0^{(0)}(I^*) = 1,$$

the endemic branch emanates from the disease-free threshold at $\mathcal{R}_0^{(0)} = 1$. The direction of the endemic branch at $\mathcal{R}_0^{(0)} = 1$ must be checked to establish backward branching.

We define the lower autonomous critical threshold by

$$\mathcal{R}_c^{(0)} = \inf_{I^* \in \mathcal{I}} \mathcal{R}_0^{(0)}(I^*). \quad (41)$$

If the infimum is attained at an interior admissible point $I_c \in \mathcal{I}$, then

$$\mathcal{R}_c^{(0)} = \mathcal{R}_0^{(0)}(I_c), \quad (42)$$

where

$$\left. \frac{d}{dI^*} \mathcal{R}_0^{(0)}(I^*) \right|_{I^*=I_c} = 0, \quad S^*(I_c) > 0. \quad (43)$$

If this interior point is a nondegenerate strict local minimum, then additionally

$$\left. \frac{d^2}{d(I^*)^2} \mathcal{R}_0^{(0)}(I^*) \right|_{I^*=I_c} > 0. \quad (44)$$

The role of saturation can be seen directly from (38). Differentiating the branch at $I^* = 0$ gives

$$\left. \frac{d\mathcal{R}_0^{(0)}}{dI^*} \right|_{I^*=0} = \frac{A + B}{N^*} - \frac{\varpi \kappa [d_3 - \epsilon(\alpha_1 + \mu)]}{d_2(d_3 + \epsilon \kappa)}.$$

If $d_3 > \epsilon(\alpha_1 + \mu)$, the derivative is negative when

$$\varpi > \varpi_{\text{BB}} := \frac{A+B}{N^*} \frac{d_2(d_3 + \epsilon\kappa)}{\kappa[d_3 - \epsilon(\alpha_1 + \mu)]}. \quad (45)$$

Under this condition, the endemic branch is directed into $\mathcal{R}_0^{(0)} < 1$ near the disease-free threshold. If $\varpi = 0$, then

$$\mathcal{R}_0^{(0)}(I^*) = \frac{N^*}{S^*(I^*)} > 1 \quad \text{for all } I^* > 0,$$

because $0 < S^*(I^*) < N^*$ on the endemic branch. Therefore, the branch cannot enter the region $\mathcal{R}_0^{(0)} < 1$ when treatment is linear. Hence, any backward-bifurcation mechanism arising from this treatment structure requires genuine saturation, namely $\varpi > 0$.

Setting $\epsilon = 0$ in the branch derivative yields

$$\left. \frac{d\mathcal{R}_0^{(0)}}{dI^*} \right|_{I^*=0, \epsilon=0} = \frac{A+B}{N^*} - \frac{\varpi\kappa}{d_2}.$$

Thus, $\varpi > (A+B)d_2/(N^*\kappa)$ gives a backward direction even when treated individuals do not transmit infection. This is an analytical limiting-case test; it does not establish endemic-equilibrium stability.

Theorem 8. Suppose that $d_3 > \epsilon(\alpha_1 + \mu)$, the saturation condition (45) holds, and the autonomous branch has an interior fold point $I_c > 0$ satisfying (43) and (44), with the value in (42) below one. Then the endemic branch is directed backward at the disease-free threshold. In particular, for

$$\mathcal{R}_c^{(0)} < \mathcal{R}_0^{(0)} < 1, \quad (46)$$

the locally stable disease-free equilibrium and at least one positive endemic equilibrium coexist. The existence statement alone does not establish stability of an endemic equilibrium or bistability.

Proof. The disease-free equilibrium changes local stability at $\mathcal{R}_0^{(0)} = 1$. For $\mathcal{R}_0^{(0)} < 1$, the disease-free equilibrium is locally asymptotically stable. The derivative obtained above is negative under the theorem assumptions; hence the endemic branch initially enters the subthreshold region.

By the assumed fold conditions (42)–(44), the branch has an admissible interior minimum below one. Its limit at the disease-free threshold was established above. By continuity of $\mathcal{R}_0^{(0)}(I^*)$ on the admissible branch, for every value in (46) there exists at least one admissible $I^* > 0$ such that

$$\mathcal{R}_0^{(0)} = \mathcal{R}_0^{(0)}(I^*).$$

By Theorem 7, this value of I^* defines a positive endemic equilibrium.

Thus, in the interval (46), the disease-free equilibrium is locally asymptotically stable, while at least one positive endemic equilibrium also exists. The branch-direction condition establishes backward branching; coexistence alone does not imply bistability. $\square$

Algorithm 2 Computation of the autonomous lower critical threshold $\mathcal{R}_c^{(0)}$

Require: Model parameters and the endemic-branch function $\mathcal{R}_0^{(0)}(I)$

Ensure: Autonomous lower critical threshold $\mathcal{R}_c^{(0)}$

- 1: Compute $E(I)$, $T(I)$, $F(I)$, $R(I)$, and $S(I)$ using (31)–(35), with I^* replaced by I .
- 2: Determine the admissible set (40) with I^* replaced by I .
- 3: Find the positive root $I_{\max}$ of $S(I) = 0$, so that the admissible interval is $0 < I < I_{\max}$.
- 4: Compute $\mathcal{R}_0^{(0)}(I)$ using (38).
- 5: Use a bounded scalar minimization method, such as MATLAB's `fminbnd`, to compute

$$I_c = \arg \min_{0 < I < I_{\max}} \mathcal{R}_0^{(0)}(I).$$

- 6: Evaluate the fold value using (42).
 - 7: Verify that I_c lies in the interior and is a minimum; otherwise report a boundary infimum instead of a fold.
 - 8: Check whether $\mathcal{R}_c^{(0)} < 1$.
 - 9: **if** $\mathcal{R}_c^{(0)} < 1$ **then**
 - 10: Report subthreshold endemic equilibria; establish backward branching only after checking the branch direction, and assess endemic-equilibrium stability separately.
 - 11: **else**
 - 12: Conclude that no backward bifurcation is detected through this endemic-branch criterion.
 - 13: **end if**
 - 14: **return** $\mathcal{R}_c^{(0)}$.
-

The absence of positive equilibria below $\mathcal{R}_c^{(0)}$ does not by itself establish global stability. Set

$$d_{2,G} = \alpha_1 + \mu + \frac{\kappa}{1 + \varpi N^*}, \quad \mathcal{R}_{\text{up}}^{(0)} = \frac{\beta_0 \Phi(d_3 + \epsilon \kappa)}{d_1 d_{2,G} d_3}. \quad (47)$$

If $\mathcal{R}_{\text{up}}^{(0)} < 1$, the disease-free equilibrium is globally asymptotically stable for $N(0) > 0$. Indeed, for every $q > 0$, the population identity gives $N(t) \leq N^* + q$ for all sufficiently large t . Consequently, the infected vector $x = (E, I, T)^T$ satisfies $x' \leq A_q x$ componentwise, where

$$A_q = \begin{pmatrix} -d_1 & \beta_0 & \epsilon \beta_0 \\ \Phi & -\alpha_1 - \mu - \frac{\kappa}{1 + \varpi(N^* + q)} & 0 \\ 0 & \kappa & -d_3 \end{pmatrix}.$$

The matrix A_q is Metzler, and $\mathcal{R}_{\text{up}}^{(0)} < 1$ ensures that A_q is Hurwitz for sufficiently small $q > 0$ by continuity of eigenvalues and the autonomous next-generation criterion. Comparison gives $E(t), I(t), T(t) \rightarrow 0$. The F - and R -equations then imply $F(t), R(t) \rightarrow 0$, and $S(t) \rightarrow N^*$. Local stability also follows because $d_{2,G} \leq d_2$ and hence $\mathcal{R}_0^{(0)} \leq \mathcal{R}_{\text{up}}^{(0)} < 1$. Equivalently, a sufficient condition is $\mathcal{R}_0^{(0)} < d_{2,G}/d_2$. This bound need not coincide with the lower fold $\mathcal{R}_c^{(0)}$. Below $\mathcal{R}_c^{(0)}$, local stability of the disease-free equilibrium and the absence of positive equilibria are consistent with elimination. In the interval below $\mathcal{R}_c^{(0)}$ not covered by this condition, however, equilibrium nonexistence alone does not exclude non-equilibrium invariant sets, and global elimination remains unproved.

5. Seasonal bistability

The autonomous analysis identifies a nonlinear mechanism through which saturated treatment can generate backward bifurcation. When $\eta > 0$, however, system (7) is non-autonomous. A positive $\mathcal{T}$ -periodic solution corresponds to an infected fixed point of the one-period map; periodic forcing does not exclude more complicated long-term behavior. The seasonal analogue of an autonomous fold, when it exists, is a fold of positive periodic solutions.

With the state vector (8), for fixed η and β_0 , define the one-period Poincaré map by

$$\mathcal{P}_{\eta, \beta_0}(X_0) = X(\mathcal{T}; X_0, \eta, \beta_0),$$

where $X(\mathcal{T}; X_0, \eta, \beta_0)$ denotes the solution of system (7) evaluated after one seasonal period with initial condition $X(0) = X_0$. A positive seasonal endemic solution corresponds to a fixed point of this map:

$$\mathcal{P}_{\eta, \beta_0}(X_0) = X_0, \quad X_0 \in \Omega_0.$$

Equivalently, the corresponding solution is $\mathcal{T}$ -periodic and has positive transmission-relevant infected components.

If a fold of positive $\mathcal{T}$ -periodic solutions exists, denote its associated lower threshold by $\mathcal{R}_c(\eta)$. A necessary condition at such a fold is that the linearization of the Poincaré map has a unit Floquet multiplier:

$$1 \in \sigma(D_X \mathcal{P}_{\eta, \beta_{0,c}}(X_c(0))),$$

where $X_c(t)$ is the corresponding critical periodic orbit and $\beta_{0,c}$ is the associated critical transmission intensity. This provides the conceptual link between the autonomous backward-bifurcation mechanism and seasonal bistability.

In the present work, the fold of positive seasonal periodic solutions is not computed by numerical continuation. Instead, seasonal bistability is investigated using direct numerical simulation and threshold bracketing. We therefore distinguish the putative seasonal lower threshold $\mathcal{R}_c(\eta)$ from the simulation-estimated transition $\widehat{\mathcal{R}}_c^{\text{sim}}(\eta)$. The latter is not an exact bifurcation value. Rather, it is a finite-time numerical estimate of the transition between apparent extinction and apparent persistence for a specified high-infection initial condition, simulation horizon, and extinction tolerance; its dependence on these choices must be checked.

The comparison bounds derived from the monotonicity of the periodic next-generation operator provide useful constraints on the seasonal reproduction number. From (28) and (30), we obtain

$$(1 - \eta) \frac{\beta_0}{\beta_0^*} \leq \mathcal{R}_0(\eta, \beta_0) \leq (1 + \eta) \frac{\beta_0}{\beta_0^*}. \quad (48)$$

Consequently,

$$\beta_0 < \frac{\beta_0^*}{1 + \eta} \implies \mathcal{R}_0(\eta, \beta_0) < 1,$$

whereas

$$\beta_0 > \frac{\beta_0^*}{1 - \eta} \implies \mathcal{R}_0(\eta, \beta_0) > 1.$$

The intermediate interval

$$\frac{\beta_0^*}{1+\eta} \leq \beta_0 \leq \frac{\beta_0^*}{1-\eta} \quad (49)$$

cannot be classified from the comparison bounds alone. In this interval, the periodic reproduction number must be computed using Algorithm 1.

The autonomous comparison condition in Section 4 has a seasonal counterpart. Using $d_{2,G}$ from (47), set

$$\mathcal{V}_G = \begin{pmatrix} d_1 & 0 & 0 \\ -\Phi & d_{2,G} & 0 \\ 0 & -\kappa & d_3 \end{pmatrix}.$$

Define $\mathcal{R}_{\text{up}} = \rho(\mathcal{L}_G)$, where $\mathcal{L}_G$ is the periodic next-generation operator constructed as in Section 3 using $\mathcal{F}(t)$ and $\mathcal{V}_G$. If $\mathcal{R}_{\text{up}} < 1$, then the disease-free solution is globally attractive for every nonnegative initial state with $N(0) > 0$. Indeed, for every $q > 0$ and all sufficiently large t , the population identity gives $I(t) \leq N(t) \leq N^* + q$ and $S(t)/N(t) \leq 1$. Consequently, the infected vector (14) satisfies $x' \leq A_{G,q}(t)x$ componentwise, with

$$A_{G,q}(t) = \begin{pmatrix} -d_1 & \beta(t) & \epsilon\beta(t) \\ \Phi & -\alpha_1 - \mu - \frac{\kappa}{1 + \varpi(N^* + q)} & 0 \\ 0 & \kappa & -d_3 \end{pmatrix}.$$

This periodic matrix is cooperative. The condition $\mathcal{R}_{\text{up}} < 1$ makes its $q = 0$ monodromy matrix exponentially stable by the threshold criterion of Section 3. Continuity gives the same property for sufficiently small $q > 0$, and comparison yields $E(t), I(t), T(t) \rightarrow 0$. The equations for F and R then give $F(t), R(t) \rightarrow 0$, while $S(t) \rightarrow N^*$. This is a sufficient elimination criterion, not a proof of global extinction throughout the entire region below an unknown seasonal fold.

For fixed η , the simulation-based bracketing is performed in terms of the baseline transmission parameter β_0 . For each value of β_0 , the corresponding periodic reproduction number $\mathcal{R}_0(\eta, \beta_0)$ is computed using Algorithm 1. The finite-horizon outcome is classified using the final-year mean of the infected burden (29):

$$\bar{\rho}_{\text{final}}(\beta_0; X_0) = \frac{1}{\mathcal{T}} \int_{t_{\text{end}} - \mathcal{T}}^{t_{\text{end}}} \rho_0(X(t; \beta_0, X_0)) dt. \quad (50)$$

Given a small numerical tolerance $\xi > 0$, the trajectory is classified as numerically extinct if

$$\bar{\rho}_{\text{final}}(\beta_0; X_0) < \xi,$$

and as numerically nonextinct if

$$\bar{\rho}_{\text{final}}(\beta_0; X_0) \geq \xi.$$

Let $X_H \in \Omega_0$ be a fixed high-infection initial condition. Choose β_L and β_U satisfying the endpoint classifications

$$\bar{\rho}_{\text{final}}(\beta_L; X_H) < \xi, \quad \bar{\rho}_{\text{final}}(\beta_U; X_H) \geq \xi. \quad (51)$$

To ensure that the detected persistent outcome occurs below the invasion threshold, additionally require

$$\mathcal{R}_0(\eta, \beta_U) < 1. \quad (52)$$

This condition places the apparent persistent outcome at β_U in the subcritical region $\mathcal{R}_0 < 1$. Numerical evidence of bistability additionally requires a low-infection initial condition at the same parameter values to approach the disease-free periodic solution while the high-infection trajectory approaches a positive seasonal regime.

Algorithm 3 Simulation-based bracketing of the seasonal trajectory-transition threshold

Require: Seasonal amplitude η , high-infection initial condition X_H , extinction tolerance $\xi > 0$, bisection tolerance $\tau_\beta > 0$, simulation horizon t_{end} , extended horizon t_{check} , periodicity residual tolerance τ_{per} , initial bracket $[\beta_L, \beta_U]$, and additional feasible low- and intermediate-infection initial conditions

Ensure: Simulation-estimated seasonal transition values $\widehat{\beta}_{0,c}^{\text{sim}}(\eta)$ and $\widehat{\mathcal{R}}_c^{\text{sim}}(\eta)$

- 1: Verify both endpoint classifications in (51).
 - 2: Compute the periodic reproduction number using Algorithm 1 and verify (52).
 - 3: At $\beta_0 = \beta_U$, simulate a low-infection initial condition and several intermediate initial conditions at an extended simulation horizon; record the outcomes before claiming bistability.
 - 4: **while** $\beta_U - \beta_L > \tau_\beta$ **do**
 - 5: Set $\beta_M = \frac{\beta_L + \beta_U}{2}$.
 - 6: Simulate system (7) from X_H up to t_{end} using $\beta_0 = \beta_M$.
 - 7: Evaluate $\bar{\rho}_{\text{final}}(\beta_M; X_H)$ using (50).
 - 8: **if** $\bar{\rho}_{\text{final}}(\beta_M; X_H) < \xi$ **then**
 - 9: Set $\beta_L = \beta_M$.
 - 10: **else**
 - 11: Set $\beta_U = \beta_M$.
 - 12: **end if**
 - 13: **end while**
 - 14: Set $\widehat{\beta}_{0,c}^{\text{sim}}(\eta) = \frac{\beta_L + \beta_U}{2}$.
 - 15: Compute $\widehat{\mathcal{R}}_c^{\text{sim}}(\eta) = \mathcal{R}_0(\eta, \widehat{\beta}_{0,c}^{\text{sim}}(\eta))$ using Algorithm 1.
 - 16: At both final bracket endpoints, repeat the integration to t_{check} and compare the last-period burden with ξ . For the nonextinct upper endpoint also compute the period-one residual $D_{\mathcal{T}}$ between successive final-period full-state profiles. If either classification changes, or $D_{\mathcal{T}} > \tau_{\text{per}}$, flag the estimate as horizon-dependent or unresolved; do not interpret it as a verified transition or a seasonal fold.
 - 17: **return** $\widehat{\beta}_{0,c}^{\text{sim}}(\eta)$ and $\widehat{\mathcal{R}}_c^{\text{sim}}(\eta)$.
-

The following algorithm estimates a finite-horizon transition value for the chosen high-infection initial state. It assumes that, for the chosen high-infection initial condition X_H , the extinction-persistence classification changes once on the selected bracket. In practice, this bracket should first be verified by a preliminary parameter scan. If the single-transition assumption fails, the procedure must not report a unique transition value.

For the reference computations, $\mathcal{T} = 365$ days, $\eta = 0.25$, $\varpi = 0.25$, $\xi = 10^{-8}$, and the bisection width is $\tau_\beta = 10^{-6} \text{ day}^{-1}$. The primary horizon is $t_{\text{end}} = 30\mathcal{T} = 10,950$ days, and endpoint outcomes are checked at $t_{\text{check}} = 60\mathcal{T} = 21,900$ days. The residual tolerance for successive whole-period state profiles is $\tau_{\text{per}} = 10^{-5}$. Numerical integration uses a relative tolerance of 10^{-9} , absolute tolerance

of 10^{-11} , and maximum step of one day. For the transition scan, $X_H = (900, 150, 180, 120, 30, 20)^T$ and the initial reproduction-number bracket is $[0.65, 0.926]$, converted to a β_0 bracket using the periodic next-generation number; all state vectors have total population $N^* = 1400$. These settings define a reproducible, initial-condition-specific numerical diagnostic, not a general fold calculation.

For fixed η , the new-infection matrix is proportional to β_0 . Hence the periodic next-generation operator is also proportional to β_0 , and $\mathcal{R}_0(\eta, \beta_0)$ is monotone increasing in β_0 . Therefore, the final bracketing interval yields

$$\mathcal{R}_0(\eta, \beta_L) \leq \widehat{\mathcal{R}}_c^{\text{sim}}(\eta) \leq \mathcal{R}_0(\eta, \beta_U), \quad (53)$$

where $\beta_U - \beta_L \leq \tau_\beta$. This interval records bisection precision for the specified finite-time classification and is not an error bound on an actual fold of periodic orbits. Applying the comparison bounds (48) at each bracket endpoint gives validation bounds, not error estimates for a seasonal fold. The reported values of $\mathcal{R}_0(\eta, \beta_L)$, $\mathcal{R}_0(\eta, \beta_U)$, and $\widehat{\mathcal{R}}_c^{\text{sim}}(\eta)$ are computed using the periodic next-generation method in Algorithm 1.

This simulation-based procedure does not replace a rigorous continuation analysis of folds of periodic orbits. It locates a transition along one initial-condition path and does not determine the full basin boundary or establish a periodic-orbit saddle-node. However, it provides a transparent numerical test for seasonal bistability. In particular, if for the same parameter value satisfying $\mathcal{R}_0 < 1$ a low-infection initial condition converges to the disease-free periodic solution while a high-infection initial condition approaches a positive seasonal regime, then the simulations provide evidence consistent with bistability at that parameter value, subject to verification over longer horizons and additional initial conditions.

To assess dependence on the initial condition at fixed subthreshold parameter values, consider $X_\theta = (1 - \theta)X_{\text{low}} + \theta X_H$ for several $0 < \theta < 1$, using feasible states with the same total population. Compare the final-year mean infectious burden at the original and extended simulation horizons and, for apparent positive seasonal solutions, compare successive final-period state profiles.

The autonomous lower threshold $\mathcal{R}_c^{(0)}$ is defined through an equilibrium-branch fold, whereas $\widehat{\mathcal{R}}_c^{\text{sim}}(\eta)$ is a finite-time, initial-condition-dependent estimate. Even at $\eta = 0$, a trajectory transition for a specified X_H need not equal the autonomous fold because the chosen initial state may lie outside the endemic basin. To assess the effect of seasonality on the same diagnostic, repeat the protocol at the same X_H , numerical tolerances, and horizon while varying η and converting β_0 to the correct periodic $\mathcal{R}_0$. Observed trends over a finite η grid do not establish monotonicity of an exact seasonal fold or of the entire bistability region.

6. Numerical simulations and bifurcation analysis

This section evaluates the thresholds and dynamical regimes established in Sections 2–5 and examines the parameter-dependent behavior that the analytical results do not determine. The numerical experiments are illustrative scenarios, not a fit to conjunctivitis outbreak data. Unless specified otherwise, simulations use MATLAB ode45 with relative tolerance 10^{-9} , absolute tolerance 10^{-11} , and maximum step of one day; the periodic reproduction-number computation uses Algorithm 1 with root tolerance 10^{-10} and monodromy maximum step $\mathcal{T}/400$. The autonomous minimization follows Algorithm 2 with tolerance 10^{-12} . The seasonally forced experiments use $\mathcal{T} = 365$ days.

6.1. Parameter values and interpretation of treatment saturation

Table 2 gives the reference scenario. In particular, $\alpha_1 = 0.14$, $\Psi = 0.02$, and $\delta = 0.015$ are assumed numerical inputs: The earlier references do not provide measurements of these three rates for the present compartments. They are retained to examine the mathematical mechanism under a reproducible scenario, not represented as parameter estimates. In isolation, the three rates correspond to characteristic times of approximately 7.14, 50, and 66.7 days, respectively; actual compartment residence times also depend on competing transitions. These times are interpretations of the assumptions, not clinical measurements. Their variation is examined below rather than claimed to validate their empirical accuracy.

Table 2. Reference inputs for system (7). Values describe a qualitative scenario, not a data-calibrated outbreak. The disputed rates α_1 , Ψ , and δ are explicitly identified as assumptions; β_0 , ϖ , ϵ , and η are varied in the experiments described below.

Parameter	Reference value	Unit	Numerical role
Π	0.056	persons day ⁻¹	demographic input
μ	0.00004	day ⁻¹	demographic input
β_0	varied	day ⁻¹	transmission parameter
ϵ	0.05	dimensionless	relative treated infectiousness
Φ	0.25	day ⁻¹	exposed-to-infectious progression
κ	0.15	day ⁻¹	treatment initiation parameter
Ψ	0.02	day ⁻¹	assumed treatment-failure rate
α_1	0.14	day ⁻¹	assumed spontaneous recovery rate
α_2	0.30	day ⁻¹	recovery during treatment
α_3	0.10	day ⁻¹	recovery after treatment failure
δ	0.015	day ⁻¹	assumed waning-immunity rate
ϖ	0.25	persons ⁻¹	strong-saturation scenario
$\omega = \varpi N^*$	350	dimensionless	normalized saturation strength
η	0.25	dimensionless	principal seasonal scenario
$\mathcal{T}$	365	days	forcing period

The demographic equilibrium $N^* = \Pi/\mu = 1400$ represents an illustrative community scale, not an estimate of a regional population. As explained in Section 2, $\varpi = 0.25$ places treatment half-saturation at only four infectious individuals; the asymptotic treatment capacity is $\kappa/\varpi = 0.6$ persons per day. This deliberately constrained-capacity scenario makes the nonlinear mechanism visible but is not an empirically established healthcare capacity. Because saturation also depends on population scale, results should not be extrapolated to other populations without reparameterization. The ranges examined below are scenario stress ranges rather than clinical confidence intervals.

6.2. Numerical computation of the seasonal reproduction number

Algorithm 1 was applied over $0 \leq \eta \leq 0.5$ at the autonomous reference transmission $\beta_0^* \approx 0.283444$. Figure 1(a) shows that the computed periodic reproduction number remains within the bounds established in Section 3 and equals the autonomous value to numerical accuracy at $\eta = 0$.

Figure 1(b) shows the relative difference from the averaged autonomous reproduction number. At $\eta = 0.25$, the computed value is 0.99211097 when the averaged value is one; at $\eta = 0.50$, the corresponding value is 0.96708149, a difference of -3.291851% . These results validate the numerical threshold computation for this parameter set; they do not locate a nonlinear fold of seasonal periodic solutions.

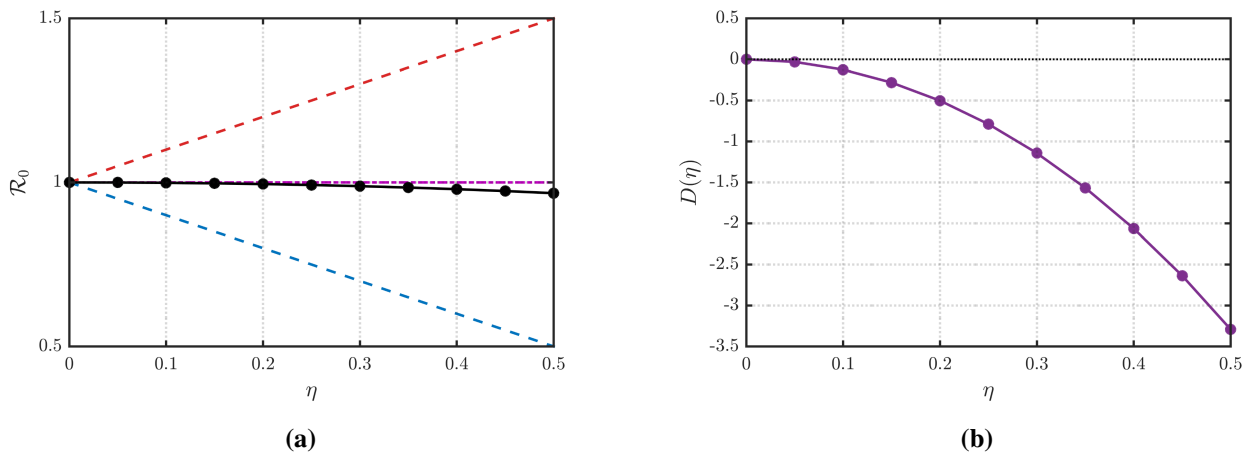


Figure 1. Seasonal invasion-threshold calculation at $\beta_0 = \beta_0^*$. (a) Periodic reproduction number (black markers), autonomous reference (magenta dash-dot), and the lower and upper bounds (blue and red dashed curves). (b) Percentage difference between the periodic and autonomous reproduction numbers; the dotted line marks zero. These are invasion thresholds, not nonlinear lower-fold estimates.

6.3. Numerical verification of local stability and approach to a seasonal periodic regime

For $\eta = 0.25$, five initial conditions near the disease-free state were simulated at $R_0 = 0.90$; all displayed infectious trajectories decay (Figure 2(a)), consistent with the local stability result of Section 3. At $R_0 = 1.05$, five positive initial conditions yield sustained seasonal oscillations with the same numerically observed final-period profile (Figure 2(b),(c)). Their final-year mean infected burden at $30\mathcal{T}$ is approximately 92.4414 for each trajectory. The full-state period-one residuals at that horizon range from 8.89×10^{-9} to 1.44×10^{-8} , below the prescribed tolerance 10^{-5} . This provides numerical evidence consistent with approach to a positive periodic regime; it does not establish uniqueness or global attraction. The residuals are reported here rather than occupying a separate plot.

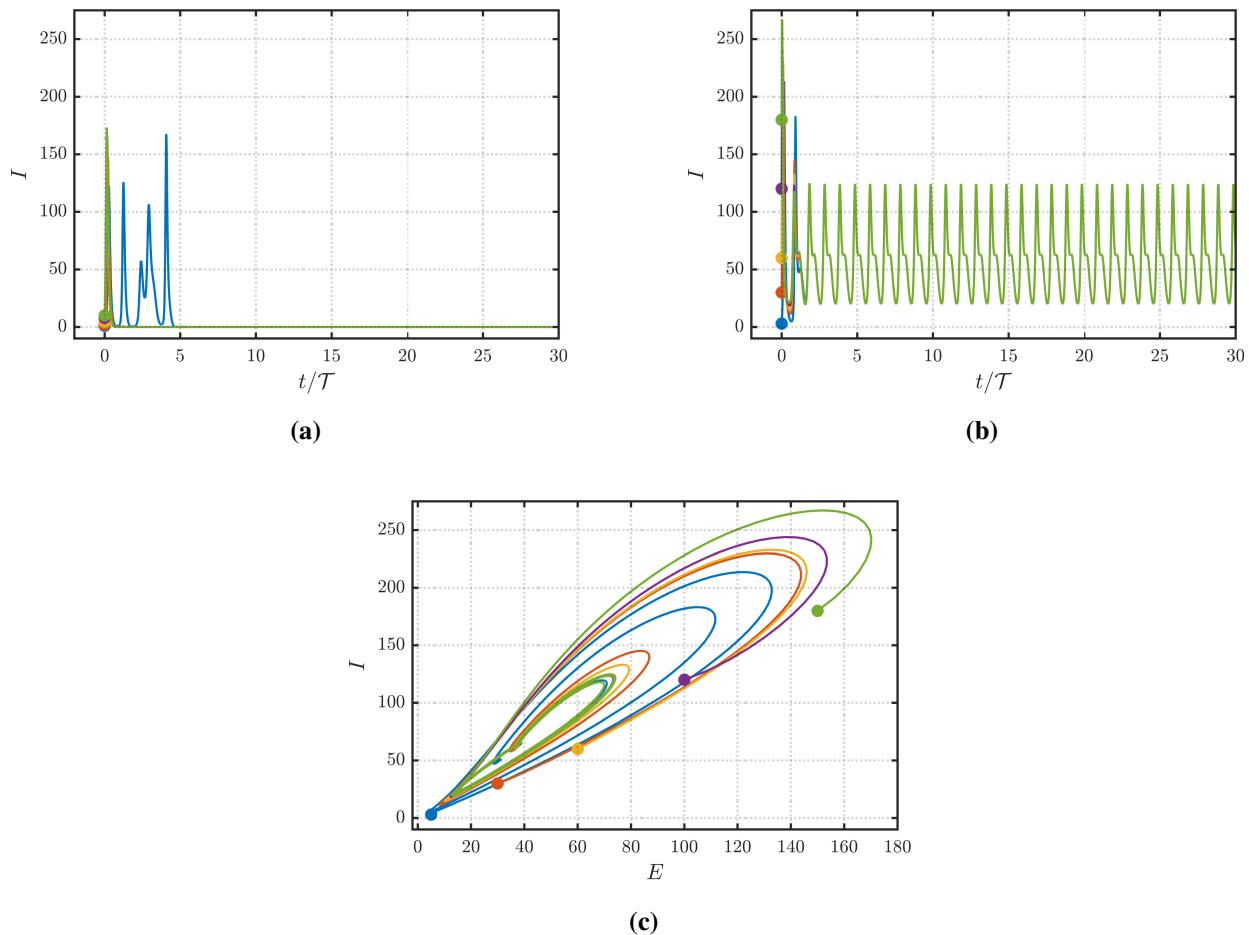


Figure 2. Threshold-dynamics simulations at $\eta = 0.25$ from five initial conditions. (a) Decay of $I(t)$ at $\mathcal{R}_0 = 0.90$. (b) Sustained oscillations at $\mathcal{R}_0 = 1.05$. (c) Corresponding (E, I) state-space trajectories in the superthreshold experiment, numerically consistent with a common seasonal periodic regime. The initial values are marked by filled circles; all trajectory curves are solid.

6.4. Autonomous backward bifurcation diagram

The explicit branch in Section 4 is minimized using Algorithm 2. At the reference parameters, the disease-free threshold is $\beta_0^* \approx 0.283444018$ and the lower turning point is $\beta_{0,c}^{(0)} \approx 0.198970988$, with $I_c \approx 14.970348$. This gives $\mathcal{R}_c^{(0)} \approx 0.701976320$. The positive computed curvature (6.65668×10^{-4}) is consistent with the nondegenerate local minimum in Section 4; see Figure 3(a),(b).

At $\mathcal{R}_0^{(0)} = 0.926$, the full 6×6 Jacobian has a positive eigenvalue 9.146074×10^{-3} at the lower endemic equilibrium ($I^* \approx 0.826673$). At the upper equilibrium ($I^* \approx 51.048961$), its largest real eigenvalue is -4.0×10^{-5} . Thus, the lower equilibrium is unstable and the upper equilibrium is locally asymptotically stable, while the disease-free equilibrium is also locally stable. This establishes numerical bistability at the specified autonomous parameter value rather than inferring it from equilibrium coexistence alone.

The MatCont continuation gives a disease-free branch-point discrepancy of 6.7×10^{-16} in β_0 and a maximum discrepancy of 2.60×10^{-12} between the continuation and the explicit transmission branch. A local quadratic fit places the turning point within 4.4×10^{-6} in I_c and 3.5×10^{-13} in $\beta_{0,c}^{(0)}$ of the explicit-branch calculation (Figure 3(c),(d)). The fold was identified from a turning point of the pseudo-arclength continuation, not from a MatCont “NE” detected point. This comparison verifies agreement with the MatCont continuation.

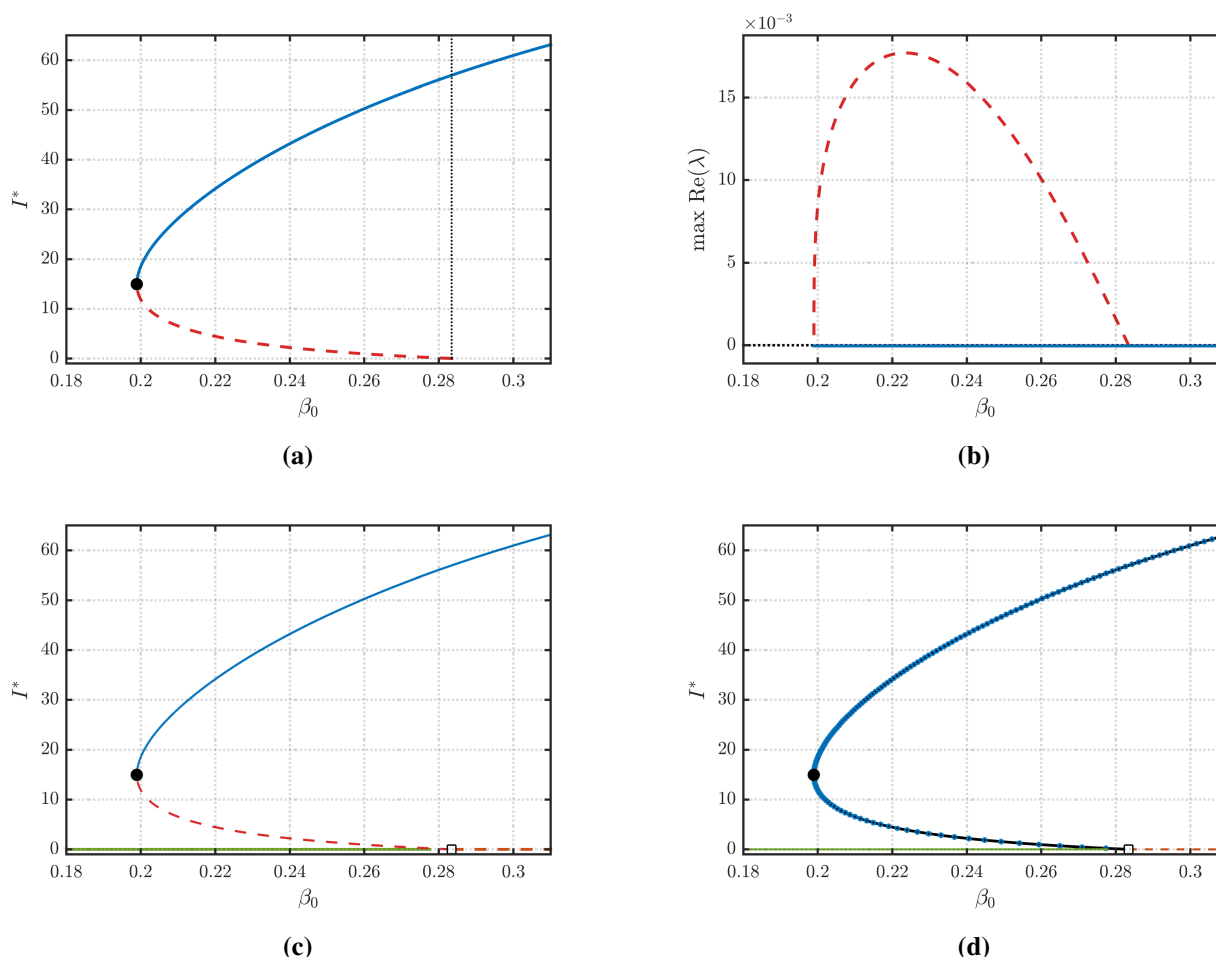


Figure 3. Autonomous branch and stability verification. (a) Explicit endemic branch (blue solid: stable; red dashed: unstable) and lower turning point (filled black circle). (b) Largest Jacobian real part along the endemic branch, with zero indicated by the dotted reference. (c) MatCont equilibrium continuation, with the disease-free and endemic branches and their stability classifications; the turning point is reconstructed from the continuation, not an “NE” label. (d) Agreement of sampled MatCont points with the explicit branch.

6.5. Verification of three dynamical regimes

The seasonal global-elimination criterion proved in Section 5 applies at $\eta = 0.25$ and $\mathcal{R}_0 = 0.45$ ($\beta_0 \approx 0.128564054$), for which $\mathcal{R}_{\text{up}} \approx 0.929703 < 1$. The two trajectories in Figure 4(a) illustrate this theorem-supported case; the proof, rather than these two simulations, supplies the global conclusion.

For the subthreshold experiment $\mathcal{R}_0 = 0.926$ ($\beta_0 \approx 0.264556253$), the low- and high-infection initial states are $X_{\text{low}} = (1385, 5, 3, 2, 0, 5)^T$ and $X_{\text{high}} = (900, 150, 180, 120, 30, 20)^T$. As prescribed in Section 5, the additional 21 initial states are sampled along the segment joining these vectors. At $30\mathcal{T}$, the numerical classification is extinction for the 11 samples with $\theta \leq 0.50$ and approach to a positive period-one regime for the ten samples with $\theta \geq 0.55$. The positive final-period means are approximately 69.781, with period-one residuals below 1.3×10^{-7} ; classifications are consistent with the $20\mathcal{T}$ checks. Figure 4(b),(c) display the sampled basin dependence, not a complete basin boundary or a seasonal fold.

At $\mathcal{R}_0 = 1.05$ the trajectories display persistent seasonal oscillations (Figure 4(d)), in agreement with the persistence theorem. The corresponding (E, I) projections for all three cases are given in Figure 5; these projections are numerical illustrations and are not additional stability proofs.

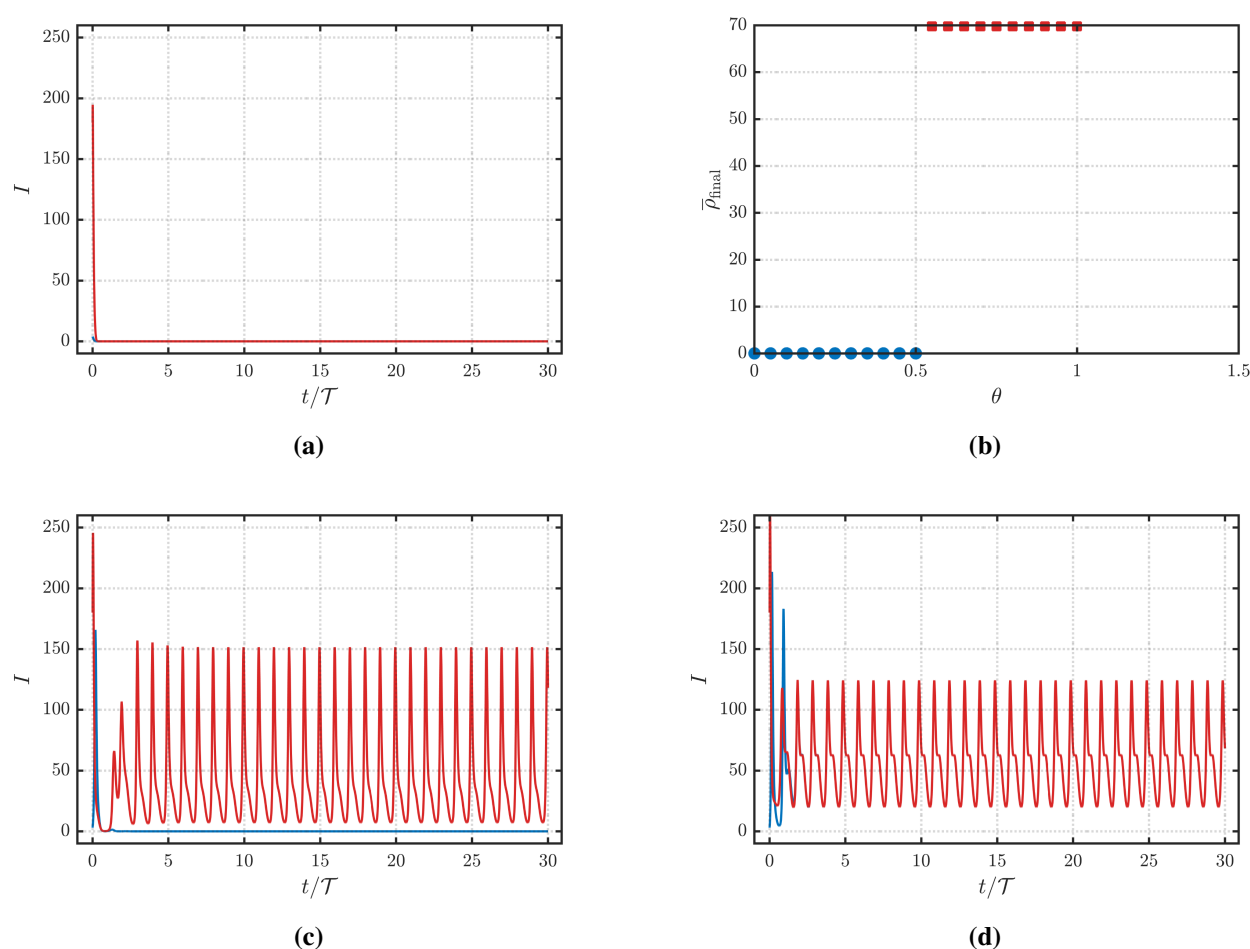


Figure 4. Seasonal outcomes at $\eta = 0.25$. (a) Extinction example at $\mathcal{R}_0 = 0.45$, satisfying the sufficient global-elimination condition. (b) Final-year infected burden for 21 initial states at $\mathcal{R}_0 = 0.926$: 11 numerically extinct and ten approaching a positive period-one regime. (c) Contrasting outcomes from low- and high-infection initial states at the same subthreshold value. (d) Persistent seasonal oscillations at $\mathcal{R}_0 = 1.05$. All time-series trajectories are drawn with solid lines; colors distinguish initial states.

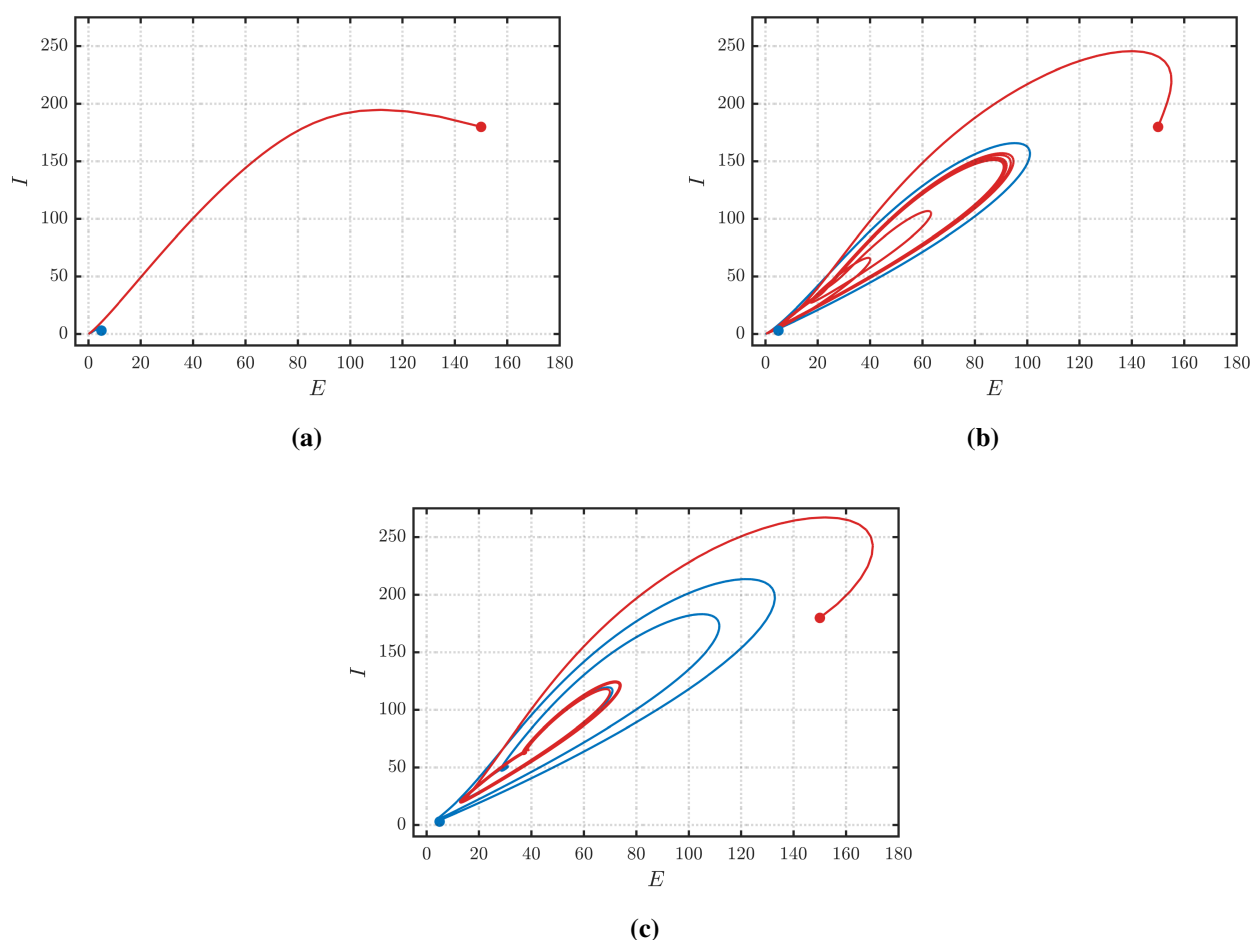


Figure 5. (E, I) projections of the regimes in Figure 4: (a) approach to the disease-free state at $\mathcal{R}_0 = 0.45$; (b) different outcomes for the low- and high-infection states at $\mathcal{R}_0 = 0.926$; and (c) approach to a positive oscillatory regime at $\mathcal{R}_0 = 1.05$. Filled circles mark initial conditions. Solid curves show the saved trajectories; their final periods are emphasized.

6.6. Mechanism isolation and two-parameter threshold diagrams

The limiting ablation $\epsilon = 0$ tests whether infectiousness during treatment is necessary for the autonomous backward branch. At the reference normalized saturation $\omega = 350$, the computed backward-direction threshold is $\omega_{\text{BB}}(0) \approx 42.5449$ and the fold is $\mathcal{R}_c^{(0)} \approx 0.689268$ ($\beta_{0,c}^{(0)} \approx 0.199947$). Thus, a subthreshold branch persists even when treated individuals are noninfectious, under the saturation condition established in Section 4. Saturation can generate this mechanism; arbitrary nonzero saturation is not sufficient.

Figure 6 reports the autonomous lower-fold transmission value versus ω and ϵ , together with the corresponding invasion threshold. The ω scan covers 0–450, equivalently $0 \leq \varpi \leq 0.32143$ at $N^* = 1400$; the ϵ scan covers 0–1.

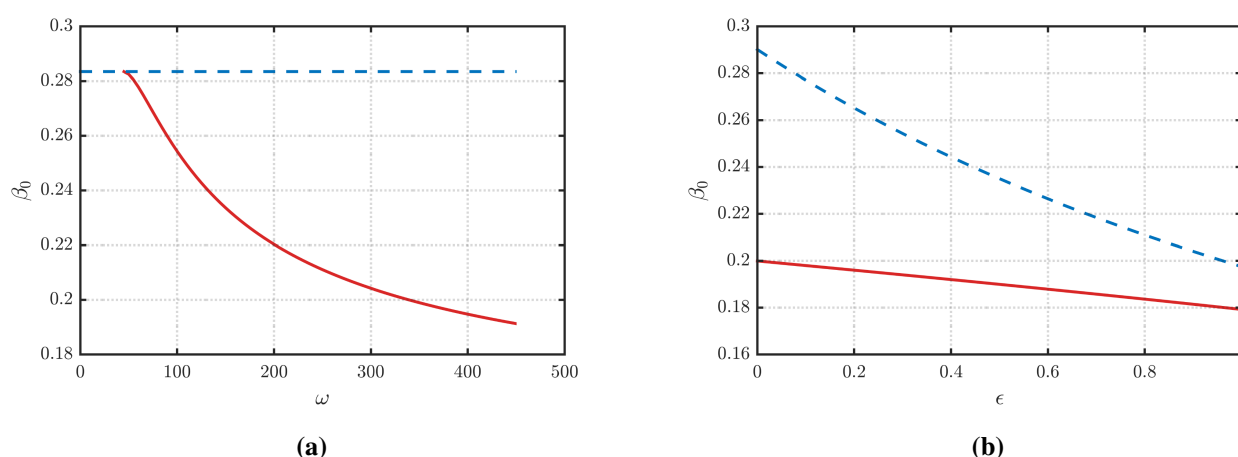


Figure 6. Autonomous two-parameter calculations: (a) lower-fold transmission value versus normalized saturation ω ; (b) its dependence on treated infectiousness ϵ at $\omega = 350$. Solid red curves denote the lower fold where present and dashed blue curves the disease-free transmission threshold.

6.7. Structural robustness and global sensitivity

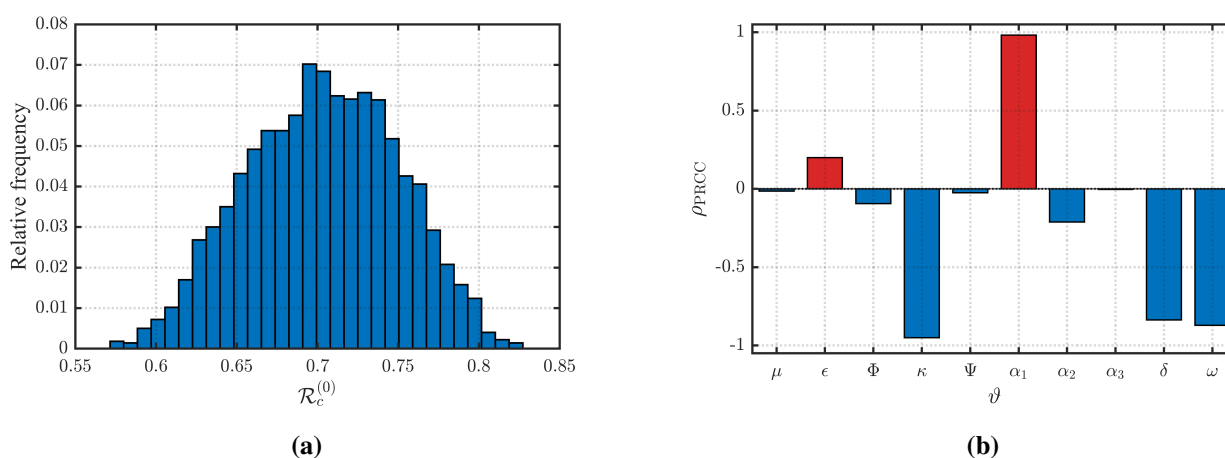


Figure 7. Structural robustness of the autonomous lower-fold threshold $\mathcal{R}_c^{(0)}$ under independent $\pm 20\%$ variations of ten parameters at fixed $N^* = 1400$. (a) Relative-frequency histogram from 5000 Latin-hypercube samples; relative frequency is the fraction of samples in each bin. (b) Partial rank correlation coefficients (PRCCs) for the same ten inputs, with positive and negative values indicating the direction of association. These are scenario sensitivity calculations, not empirical parameter calibration or confidence intervals for a population.

To evaluate dependence on the assumed inputs, ten parameters (μ , ϵ , Φ , κ , Ψ , α_1 , α_2 , α_3 , δ , and ω) were varied independently over $\pm 20\%$ of their reference values. The population scale $N^* = 1400$

was fixed, with $\Pi = \mu N^*$ when varying demographic turnover. A 5000-point Latin-hypercube sample produced autonomous lower-fold values between 0.571480 and 0.827213, with median 0.704156 and central sampled 95% range 0.613753–0.789717 (Figure 7(a)). Each of the 5000 sampled scenarios retained a subthreshold fold and $\omega > \omega_{\text{BB}}$; these frequencies describe the specified sample, not the probability of backward bifurcation in a real population.

A seven-level conditional monotonicity screen at 100 background combinations passed for each input. Figure 7(b) displays the partial rank correlation coefficients (PRCCs) for all ten inputs. The largest absolute PRCCs with the autonomous lower threshold were α_1 (0.9815), κ (−0.9506), ω (−0.8714), and δ (−0.8371).

6.8. Simulation-estimated seasonal transition

Algorithm 3 was executed using the protocol and numerical settings specified in Section 5 at each sampled value of η . The MATLAB calculation returned $\widehat{\mathcal{R}}_c^{\text{sim}} = 0.797399, 0.848395, 0.875647, 0.900705, \text{ and } 0.924028$ for $\eta = 0, 0.10, 0.15, 0.20, \text{ and } 0.25$, respectively. All five extended-check flags passed. Figure 8 displays the resulting sampled trend.

The analytical autonomous fold $\mathcal{R}_c^{(0)} \approx 0.701976$ and the $\eta = 0$ trajectory transition 0.797399 are different quantities even without seasonal forcing: The latter depends on the chosen initial state and finite-horizon classification. Comparing that *same* trajectory diagnostic at $\eta = 0$ and 0.25 gives 0.797399 and 0.924028, respectively. The earlier estimate near 0.88 was not reproduced under the specified protocol and is superseded by the numerical MATLAB calculation. The upward trend for these five sampled amplitudes is consistent with a narrower detected subthreshold-persistence interval for this protocol. It neither demonstrates monotonicity for every η nor identifies the fold of positive seasonal periodic orbits; changing transmission timing relative to nonlinear treatment demand is a possible interpretation, not an independently established causal decomposition.

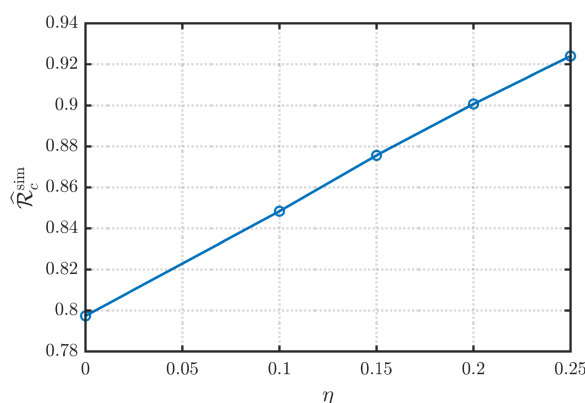


Figure 8. Seasonal forcing amplitude versus the simulation-estimated extinction–persistence transition for the fixed high-infection initial condition in Algorithm 3. Points are the five numerical MATLAB console estimates after $30\mathcal{T}$ classification and successful $60\mathcal{T}$ endpoint checks.

Together, the computations connect the seasonal invasion threshold, autonomous backward branch, sufficient extinction criterion, and initial-condition-dependent seasonal outcomes to their respective

theoretical results without identifying the simulated transition with an uncomputed seasonal fold.

7. Conclusions

This study developed a non-autonomous SEITFR model for conjunctivitis transmission incorporating seasonal forcing, modified infectiousness during treatment, treatment failure, temporary immunity, and saturated treatment capacity. The model was shown to be mathematically well posed by proving positivity, boundedness, and global existence and uniqueness of solutions.

Because the transmission rate is periodic, the disease threshold was defined using the periodic next-generation operator. The resulting seasonal reproduction number determines local stability of the disease-free periodic solution; above unity, the model is uniformly persistent and admits at least one positive seasonal periodic solution. A separate comparison criterion provides a sufficient condition for global disease elimination, illustrated numerically at $\mathcal{R}_0 = 0.45$.

The analysis also showed that treatment saturation can change the nonlinear threshold structure without changing the local invasion threshold. In the autonomous limit, the explicitly parameterized endemic branch has a lower fold at $\mathcal{R}_c^{(0)} \approx 0.701976$. At the selected subthreshold value $\mathcal{R}_0^{(0)} = 0.926$, the lower endemic equilibrium is unstable and the upper one locally stable, as determined from the full Jacobian; the branch and MatCont continuation agree. Backward branching persists for noninfectious treated individuals in the $\epsilon = 0$ ablation under the derived saturation condition, although arbitrary positive saturation is not sufficient.

In the seasonal model, simulations at $\mathcal{R}_0 = 0.926$ identify 11 extinction outcomes and ten positive-periodic-regime outcomes among 21 initial states. The finite-horizon transition for the same high-infection initial state changes from approximately 0.797399 without forcing to 0.924028 at $\eta = 0.25$. Neither estimate is an exact periodic-orbit fold: In particular, the analytical autonomous fold and the autonomous trajectory diagnostic are different quantities. The sampled upward trend does not establish monotonicity for all forcing amplitudes, and the observed basin dependence does not constitute a global classification.

The numerical procedures for the seasonal reproduction number, autonomous fold, and initial-state-specific transition provide reproducible ways to investigate the model. The $\pm 20\%$ sensitivity analysis supports the autonomous backward branch only within the tested scenario ranges and fixed population scale; it does not validate the assumed clinical rates or establish robustness of a seasonal fold. Accordingly, the findings are qualitative, not calibrated forecasts or quantitative treatment recommendations. Future work should use outbreak data to constrain the parameters, examine other population scales, and locate the exact seasonal fold and its dependence on treatment saturation and forcing amplitude.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

Bashir Al-Hdaibat: Conceptualization, Methodology, Software, Formal analysis, Investigation, Writing – original draft, Supervision. **A. Alameer:** Conceptualization, Methodology, Software, Investigation, Writing – original draft. All authors read and approved the final manuscript.

Use of AI tools declaration

The authors did not use artificial intelligence (AI) tools in developing the mathematical model, conducting the research, performing the analyses, or generating the scientific results.

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