

Article

Modeling Influenza–*Streptococcus pneumoniae* Co-Infection: Multistage Progression and Competitive Exclusion

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Abstract

Co-infection between influenza and *Streptococcus pneumoniae* is an important public health concern, since influenza can increase susceptibility to secondary bacterial invasion and enhance bacterial transmission. We develop and analyze a compartmental model of influenza–pneumococcal co-infection with eleven epidemiological compartments, incorporating imperfect vaccination, quarantine, and a three-stage pneumococcal progression from colonization to invasive disease. We establish the positivity and boundedness of solutions, determine the equilibria, and derive, via the next-generation matrix method, the sub-model reproduction numbers R_0^I and R_0^P together with the composite threshold $R_0 = \max\{R_0^I, R_0^P\}$. Each single infection undergoes a forward bifurcation: the disease-free equilibrium is locally asymptotically stable when the corresponding reproduction number is below unity, and a unique endemic equilibrium—globally asymptotically stable in the pneumococcal sub-model, established by a Goh–Volterra Lyapunov function—emerges above it. When both thresholds exceed unity, the two infections compete for the shared susceptible pool and undergo competitive exclusion: one infection persists while the other, together with the co-infected class, is eliminated. We show that the outcome is governed not by the disease-free reproduction numbers but by the invasion reproduction numbers evaluated at the single-infection boundary equilibria, so that even equal disease-free thresholds do not yield coexistence. Numerical simulations confirm the analytical results and quantify the effect of quarantine and vaccination, providing a framework for assessing control of interacting respiratory infections.

Keywords: influenza; *Streptococcus pneumoniae*; co-infection; basic reproduction number; global stability; competitive exclusion

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1. Introduction

Global health is currently challenged by a significant burden of morbidity and mortality, which reduces human life expectancy and imposes substantial socio-economic costs on healthcare systems worldwide [1,2]. Among these global threats, infectious diseases remain a primary public health concern, with respiratory infections acting as a leading cause of illness in humans [3–5]. Pathogenic microbial agents, particularly viruses and bacteria, shape the global disease burden through complex transmission dynamics and

varying clinical severities [1,6]. Two of the most significant contributors to this burden are the influenza virus and *Streptococcus pneumoniae* (pneumococcus) [6–8].

Influenza is one of the most prevalent recurrent respiratory diseases worldwide and continues to pose a major public health burden [9,10]. Its persistence is driven by antigenic drift, which helps the virus evade host immunity, together with the temporary nature of post-infection immunity, which allows recovered individuals to become susceptible again over time [10–12]. These mechanisms support repeated outbreaks and the long-term circulation of the disease in human populations [12]. As a result, seasonal influenza causes annual epidemics and contributes to an estimated 290,000 to 650,000 deaths globally each year from severe respiratory illness [13].

In parallel, *Streptococcus pneumoniae* remains a major bacterial pathogen associated with substantial morbidity and mortality worldwide [1,6,14]. It has been reported to cause approximately 1,600,000 deaths annually, particularly among young children and older adults, and is recognized as a leading cause of community-acquired pneumonia, bacterial meningitis, and septicemia [6,8,15]. A key feature of its pathogenesis is that nasopharyngeal colonization is a necessary precursor to invasive disease development [6,14,16]. Although colonization is often asymptomatic, the bacteria may spread to the lungs or bloodstream when host defenses are weakened [14].

The pathogenesis of influenza–*S. pneumoniae* co-infection is characterized by a strong synergistic interaction in which primary influenza infection alters the respiratory environment in ways that facilitate secondary bacterial invasion [7,17,18]. Historical evidence suggests that most deaths during the 1918 influenza pandemic were attributable to secondary bacterial pneumonia rather than to influenza alone [10]. This synergy is driven by several mechanisms, including virus-induced disruption of the epithelial barrier, enhanced bacterial adherence and proliferation, and suppression of innate immune defenses such as neutrophil function and alveolar macrophage phagocytic activity [14,17,19,20]. Clinical and experimental studies further indicate that this vulnerability is strongly time-dependent, with the highest risk of severe secondary pneumonia occurring around one week after influenza infection [20].

Importantly, the interaction between influenza and *S. pneumoniae* operates at the population level as well as within the individual host. Epidemiological surveillance indicates that influenza activity is followed, after a short lag, by a marked rise in invasive pneumococcal disease, with seasonal analyses attributing a substantial excess of pneumococcal cases to preceding influenza and pointing to enhanced invasion in colonized individuals as a principal driver [21]. This interaction is also heterogeneous: its magnitude varies with pneumococcal serotype and host comorbidity, being most pronounced for less-invasive serotypes and among individuals with fewer comorbidities [22]. Experimental work further supports a transmission-level effect: influenza infection increases pneumococcal shedding and facilitates host-to-host spread of the bacterium, so that the virus modulates not only susceptibility to disease but also the transmission of its bacterial partner [23]. These findings establish influenza–pneumococcal co-infection as a genuinely coupled transmission process that warrants explicit population-level modeling. For tractability, the model developed here treats pneumococcus as a single homogeneous strain, so that its parameters represent population-averaged rates; incorporating serotype structure is a natural extension.

At the level of the individual patient, the clinical management of severe pneumonia and mixed respiratory infections remains challenging, especially when drug-resistant or multiple pathogens are involved. Inhaled polymyxin B combined with systemic therapy has been associated with improved microbiological clearance in stroke-associated pneumonia caused by carbapenem-resistant Gram-negative bacteria [24], and omadacycline has been used successfully in severe *Chlamydia psittaci* pneumonia complicated by

multidrug-resistant bacterial co-infection [25]. Individualized antimicrobial dosing guided by therapeutic drug monitoring may further improve outcomes, as suggested by the association between trimethoprim–sulfamethoxazole blood concentrations and clinical outcomes in *Pneumocystis jirovecii* pneumonia [26], while emerging influenza treatments that jointly inhibit viral replication and excessive host inflammation—for example, dual targeting of the viral PB2 protein and the host JAK2 pathway—may offer additional strategies against severe influenza and respiratory co-infections [27]. Such advances act on the individual patient, however, and do not describe how co-infection spreads and persists across a population, or how population-level interventions such as vaccination and quarantine reshape that spread.

Mathematical modeling has become an essential framework for analyzing the complex dynamics of co-infection across a wide range of viral and bacterial diseases [5]. While earlier research often focused on single-pathogen dynamics, contemporary studies employ compartmental frameworks to represent two interacting pathogens explicitly, as in models of influenza and *S. pneumoniae* [7], HIV/AIDS and tuberculosis [28], pneumonia and meningitis [4], and influenza and SARS-CoV-2 [3]. Such models typically incorporate interventions such as quarantine, isolation, and vaccination [29,30], and emphasize threshold quantities such as the basic reproduction number [11,31,32] together with environmental and structural drivers including seasonality [10,33,34] and contact-network clustering [18,35]. To support robust risk assessment and evidence-based public health policy, these multi-pathogen models are increasingly combined with analytical tools such as sensitivity analysis [4], parameter identifiability analysis [3,36], and Bayesian inference [37].

In this paper, we propose and investigate a deterministic model for the co-infection transmission dynamics of influenza and *Streptococcus pneumoniae*. The model is formulated as a system of eleven nonlinear ordinary differential equations describing the movement of individuals among the epidemiological compartments, and it incorporates imperfect vaccination, quarantine, and the three-stage progression of pneumococcal infection through colonization, transmission, and invasion. After establishing the positivity and boundedness of solutions, we determine the equilibrium points and derive the basic reproduction number by the next-generation matrix method, obtaining the composite threshold $R_0 = \max\{R_0^I, R_0^P\}$ for the full system. We then analyze the stability of the disease-free and endemic equilibria and show that each single infection undergoes a forward bifurcation: the disease-free equilibrium is locally asymptotically stable below threshold, and a unique endemic equilibrium—proved globally asymptotically stable for the pneumococcal sub-model by means of a Goh–Volterra Lyapunov function—emerges above it. Our main contribution concerns the coupled dynamics: when both single-infection thresholds exceed unity, the two infections compete for the shared susceptible pool and undergo competitive exclusion, one persisting while the other, together with the co-infected class, is eliminated. We show that the outcome is governed by the invasion reproduction numbers evaluated at the single-infection boundary equilibria, rather than by the disease-free reproduction numbers, so that even equal disease-free thresholds do not lead to coexistence. Numerical simulations corroborate the analytical results, confirming convergence to the disease-free equilibrium below threshold and to the appropriate single-infection endemic state above it, and quantify the effect of vaccination and quarantine on the epidemic.

The remainder of this paper is organized as follows. Section 2 formulates the co-infection model and establishes the positivity and boundedness of its solutions. Sections 3 and 4 analyze the influenza and *Streptococcus pneumoniae* sub-models, respectively, deriving their reproduction numbers and the stability of the disease-free and endemic equilibria. Section 5 analyzes the full co-infection model, computes the basic reproduction number, and examines the stability of the disease-free equilibrium. Section 6 presents

the numerical simulations, including the forward bifurcation, the competitive-exclusion behavior governed by the invasion reproduction numbers, and the effects of the control parameters. Finally, Section 7 summarizes the findings and discusses their implications and directions for future work.

2. Model Formulation

In this study, we propose a co-infection model for influenza and *Streptococcus pneumoniae* by subdividing the total human population into eleven disjoint compartments. These include susceptible individuals $S(t)$, vaccinated individuals $V(t)$, influenza-exposed individuals $E(t)$, symptomatic influenza-infected individuals $I(t)$, quarantined influenza cases $Q_I(t)$, pneumococcal-colonized individuals $P_C(t)$, individuals in the transmission stage of pneumococcal infection $P_T(t)$, individuals in the invasion stage $P_V(t)$, quarantined pneumococcal-infected individuals $Q_P(t)$, co-infected individuals $C(t)$, and recovered individuals $R(t)$; the compartments and the transitions among them are shown in Figure 1. Hence, the total population at time t is given by:

$$N(t) = S(t) + V(t) + E(t) + I(t) + Q_I(t) + P_C(t) + P_T(t) + P_V(t) + Q_P(t) + C(t) + R(t).$$

We assume that individuals are recruited into the susceptible class at a constant rate Λ , and all individuals die at a natural mortality rate μ . Susceptible individuals acquire influenza through contact with exposed, infected, and co-infected individuals with a force of infection $\lambda_I(t)$, given by:

$$\lambda_I(t) = \beta_1 E(t) + \beta_2 I(t) + \beta_3 C(t),$$

where β_1 , β_2 , and β_3 are the effective contact rates for influenza transmission via the respective classes. Likewise, individuals acquire pneumococcal infection through a separate force of infection $\lambda_P(t)$, defined as:

$$\lambda_P(t) = \beta_4 P_C(t) + \beta_5 P_T(t) + \beta_6 C(t),$$

with β_4 , β_5 , and β_6 as pneumococcal contact coefficients.

We assume that susceptible individuals are vaccinated against influenza at rate η and move to the vaccinated class $V(t)$. Vaccination reduces, but does not eliminate, the risk of influenza infection by a factor ϕ with $0 < \phi < 1$. Because the influenza vaccine confers no protection against *S. pneumoniae*, vaccinated individuals remain fully susceptible to pneumococcal colonization at the same force of infection λ_P as susceptible individuals; upon colonization they enter the class $P_C(t)$. Individuals in the exposed class $E(t)$ progress to symptomatic influenza infection $I(t)$ at rate ψ .

In our model, pneumococcal infection progresses through three distinct clinical stages. Individuals first enter the colonization class $P_C(t)$ upon exposure. They progress to the transmission stage $P_T(t)$ at a rate θ , and finally to the invasion stage $P_V(t)$ at a rate π . Each stage includes possibilities for co-infection, recovery, quarantine, and disease-induced mortality.

The co-infected class $C(t)$ receives individuals from multiple inflow routes involving both influenza and pneumococcal dynamics. Individuals may recover from either infection, die due to complications, or proceed to further disease states depending on their health and treatment access.

Several structural assumptions of the model warrant clarification. First, exposed individuals E are taken to be infectious—they appear in λ_I —reflecting the well-documented pre-symptomatic transmission of influenza during the incubation period. Second, the invasion stage P_V does not contribute to the pneumococcal force of infection, since inva-

sive pneumococcal disease, such as bacteremia or meningitis, corresponds to severe and typically hospitalized cases that contribute negligibly to community-level colonization and transmission. Third, recovered individuals R are assumed to retain immunity to both infections over the epidemic time scale considered here; although post-influenza immunity is ultimately temporary, its waning occurs on a longer time scale than the outbreak dynamics studied here and is therefore neglected. Finally, the recovery-partition parameters satisfy $m \geq 0$, $n \geq 0$, and $m + n \leq 1$, so that the fraction $1 - (m + n)$ of co-infected individuals recovering directly into R is non-negative.

Based on the assumptions above, the transitions between compartments are depicted in the flow diagram of Figure 1, and the full governing system of nonlinear differential equations is given by:

$$\begin{aligned}
 \frac{dS}{dt} &= \Lambda - (\lambda_I + \lambda_P + \eta + \mu)S, \\
 \frac{dV}{dt} &= \eta S - (\phi\lambda_I + \lambda_P + \mu)V, \\
 \frac{dE}{dt} &= \lambda_I S + \phi\lambda_I V - (\psi + \mu)E, \\
 \frac{dI}{dt} &= \psi E + m\alpha C - (\epsilon + \varsigma\lambda_P + \varphi + \mu + \delta_I)I, \\
 \frac{dQ_I}{dt} &= \varphi I - (\xi + \mu + \sigma_I)Q_I, \\
 \frac{dP_C}{dt} &= \lambda_P(S + V) - (\kappa\lambda_I + \theta + \mu)P_C, \\
 \frac{dP_T}{dt} &= \theta P_C - (\nu\lambda_I + \zeta + \pi + \omega + \mu + \delta_T)P_T, \\
 \frac{dP_V}{dt} &= \pi P_T + n\alpha C - (\rho\lambda_I + \gamma + \chi + \mu + \delta_V)P_V, \\
 \frac{dQ_P}{dt} &= \omega P_T + \gamma P_V - (\tau + \mu + \sigma_P)Q_P, \\
 \frac{dC}{dt} &= \varsigma\lambda_P I + \kappa\lambda_I P_C + \nu\lambda_I P_T + \rho\lambda_I P_V - (\alpha + \mu + \delta_{IP})C, \\
 \frac{dR}{dt} &= \xi Q_I + \epsilon I + \zeta P_T + \chi P_V + \tau Q_P + (1 - (m + n))\alpha C - \mu R.
 \end{aligned} \tag{1}$$

The model parameters and their epidemiological interpretations are described as follows. The parameter Λ denotes the recruitment rate into the population, while μ represents the natural death rate. The quantity η is the vaccination rate against influenza, and ϕ measures the reduction in influenza infection due to vaccination.

The parameters β_1 , β_2 , and β_3 denote the influenza transmission rates generated by exposed individuals E , infected individuals I , and co-infected individuals C , respectively. The parameter ψ represents the progression rate from the exposed class to symptomatic influenza infection, whereas ϵ denotes the recovery rate from influenza infection. Moreover, φ is the quarantine rate for influenza-infected individuals, ξ is the recovery rate from influenza quarantine, σ_I denotes the death rate during influenza quarantine, and δ_I is the disease-induced death rate due to influenza.

For the pneumococcal component, β_4 , β_5 , and β_6 represent the pneumococcal transmission rates from colonized individuals P_C , transmission-stage individuals P_T , and co-infected individuals C , respectively. The parameter θ denotes the progression rate from colonization to the transmission stage, and π is the progression rate from the transmission stage to the invasion stage. The quantity ω represents the quarantine rate for pneumococcal transmission-stage individuals, while ζ denotes the recovery rate from this stage. In addition, γ is the quarantine rate for pneumococcal invasion-stage individuals, χ is the corresponding recovery rate, and τ denotes the recovery rate from pneumococcal quarantine.

The parameter σ_P represents the death rate during pneumococcal quarantine, while δ_T and δ_V denote the disease-induced death rates associated with the pneumococcal transmission and invasion stages, respectively.

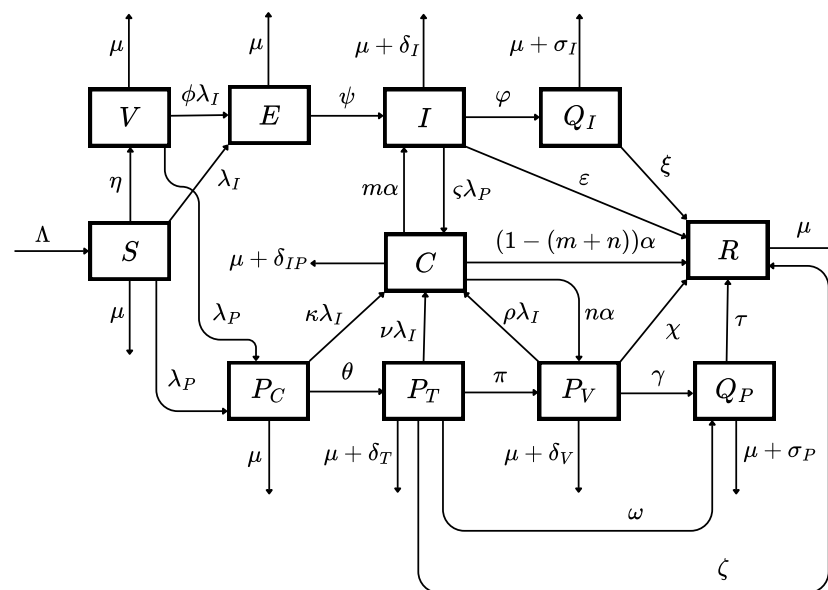


Figure 1. Flow diagram of the influenza–*Streptococcus pneumoniae* co-infection model (1). Boxes denote the eleven epidemiological compartments and arrows denote transitions between them, with the associated per-capita rates shown alongside. New recruits enter the susceptible class S at rate Λ , and all compartments exit the population through natural death at rate μ . The infected classes I , P_T , P_V , and C carry additional disease-induced death at rates δ_I , δ_T , δ_V , and δ_{IP} , respectively, and the quarantined classes Q_I and Q_P carry additional death at rates σ_I and σ_P . The influenza and pneumococcal forces of infection are $\lambda_I = \beta_1 E + \beta_2 I + \beta_3 C$ and $\lambda_P = \beta_4 P_C + \beta_5 P_T + \beta_6 C$. Both susceptible (S) and vaccinated (V) individuals acquire pneumococcal colonization at the force of infection λ_P and enter the colonization class P_C , since influenza vaccination confers no protection against *S. pneumoniae*. The co-infected class C is fed from the singly infected classes at rates $\zeta \lambda_P I$, $\kappa \lambda_I P_C$, $\nu \lambda_I P_T$, and $\rho \lambda_I P_V$, and returns individuals to I , P_V , and R at rates $m\alpha$, $n\alpha$, and $(1 - (m + n))\alpha$, respectively.

For the co-infection process, δ_{IP} denotes the disease-induced death rate due to co-infection, and α is the recovery rate from co-infection. The parameters m and n represent the proportions of co-infected individuals who recover into the influenza-infected class I and the pneumococcal invasion-stage class P_V , respectively, with $m \geq 0$, $n \geq 0$ and $m + n \leq 1$. Furthermore, κ , ν , and ρ denote the co-infection rates arising from the pneumococcal classes P_C , P_T , and P_V under influenza infection, respectively, while ζ represents the risk factor for co-infection from influenza infection to pneumococcal infection.

Positivity and Boundedness of Solutions

To ensure biological feasibility, all model variables representing population classes must remain non-negative and bounded over time. In this section, we show that the system admits non-negative solutions for non-negative initial values and that the total population stays bounded within a realistic region.

Theorem 1. For any non-negative initial data, the corresponding solution of system (1) remains non-negative for all $t \geq 0$ and satisfies

$$N(t) \leq \max \left\{ N(0), \frac{\Lambda}{\mu} \right\} \quad \text{for all } t \geq 0, \quad \limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

In particular, the region

$$\Omega = \left\{ (S, V, E, I, Q_I, P_C, P_T, P_V, Q_P, C, R) \in \mathbb{R}_+^{11} : 0 \leq N \leq \frac{\Lambda}{\mu} \right\}$$

is positively invariant and attracts all solutions of (1).

Proof. Consider non-negative initial conditions $S(0) \geq 0, V(0) \geq 0, E(0) \geq 0, I(0) \geq 0, Q_I(0) \geq 0, P_C(0) \geq 0, P_T(0) \geq 0, P_V(0) \geq 0, Q_P(0) \geq 0, C(0) \geq 0, R(0) \geq 0$. On each face of the non-negative orthant where a state variable vanishes, the corresponding vector field points inward, since

$$\begin{aligned} \left. \frac{dS}{dt} \right|_{S=0} &= \Lambda \geq 0, \\ \left. \frac{dV}{dt} \right|_{V=0} &= \eta S \geq 0, \\ \left. \frac{dE}{dt} \right|_{E=0} &= \lambda_I S + \phi \lambda_I V \geq 0, \\ \left. \frac{dI}{dt} \right|_{I=0} &= \psi E + m \alpha C \geq 0, \\ \left. \frac{dQ_I}{dt} \right|_{Q_I=0} &= \varphi I \geq 0, \\ \left. \frac{dP_C}{dt} \right|_{P_C=0} &= \lambda_P (S + V) \geq 0, \\ \left. \frac{dP_T}{dt} \right|_{P_T=0} &= \theta P_C \geq 0, \\ \left. \frac{dP_V}{dt} \right|_{P_V=0} &= \pi P_T + n \alpha C \geq 0, \\ \left. \frac{dQ_P}{dt} \right|_{Q_P=0} &= \omega P_T + \gamma P_V \geq 0, \\ \left. \frac{dC}{dt} \right|_{C=0} &= \varsigma \lambda_P I + \kappa \lambda_I P_C + \nu \lambda_I P_T + \rho \lambda_I P_V \geq 0, \\ \left. \frac{dR}{dt} \right|_{R=0} &= \xi Q_I + \epsilon I + \zeta P_T + \chi P_V + \tau Q_P + (1 - (m + n)) \alpha C \geq 0, \end{aligned}$$

where the last inequality uses $m + n \leq 1$. Hence the non-negative orthant $\mathbb{R}_+^{11}$ is positively invariant, and all state variables remain non-negative for $t \geq 0$.

Adding the eleven equations of (1), the total population satisfies

$$\frac{dN}{dt} = \Lambda - \mu N - (\delta_I I + \delta_T P_T + \delta_V P_V + \delta_{IP} C + \sigma_I Q_I + \sigma_P Q_P) \leq \Lambda - \mu N,$$

since all state variables and disease-induced death rates are non-negative. By the standard comparison theorem, the solution of the linear differential inequality gives

$$N(t) \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}, \quad t \geq 0.$$

If $N(0) \leq \Lambda/\mu$, the right-hand side never exceeds Λ/μ , so $N(t) \leq \Lambda/\mu$ for all $t \geq 0$; consequently Ω is positively invariant. If $N(0) > \Lambda/\mu$, the right-hand side decreases monotonically from $N(0)$ toward Λ/μ , so $N(t) \leq N(0)$ for all $t \geq 0$. In either case $N(t) \leq \max\{N(0), \Lambda/\mu\}$, and letting $t \rightarrow \infty$ yields $\limsup_{t \rightarrow \infty} N(t) \leq \Lambda/\mu$. Therefore every solution eventually enters and remains in Ω , so Ω is positively invariant and attracts

all solutions of (1). This establishes that the model is mathematically and epidemiologically well posed. $\square$

3. Analysis of the Influenza Sub-Model

For a better understanding of the dynamics of co-infection, we analyse the proposed model by first computing the equilibrium points and then examining the behaviour of the solutions around these stationary points. The analysis is organized into three parts: the influenza sub-model, the *Streptococcus pneumoniae* sub-model, and the full co-infection model.

Throughout the three analyses, we use the following shorthand for the compound removal rates:

$$\begin{aligned} c_1 &= \psi + \mu, & c_2 &= \epsilon + \varphi + \mu + \delta_I, & c_3 &= \xi + \mu + \sigma_I, \\ d_1 &= \theta + \mu, & d_2 &= \zeta + \pi + \omega + \mu + \delta_T, & d_3 &= \gamma + \chi + \mu + \delta_V, \\ d_4 &= \tau + \mu + \sigma_P, & d_5 &= \alpha + \mu + \delta_{IP}, \end{aligned}$$

and we write S^0 and V^0 for the disease-free susceptible and vaccinated levels of the model under consideration.

Excluding the *Streptococcus pneumoniae* infection and the co-infected class, we retain only the influenza dynamics. The force of infection is

$$\lambda_I(t) = \beta_1 E(t) + \beta_2 I(t),$$

and the governing system is

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - (\beta_1 E + \beta_2 I + \eta + \mu)S, \\ \frac{dV}{dt} &= \eta S - (\phi(\beta_1 E + \beta_2 I) + \mu)V, \\ \frac{dE}{dt} &= (\beta_1 E + \beta_2 I)S + \phi(\beta_1 E + \beta_2 I)V - (\psi + \mu)E, \\ \frac{dI}{dt} &= \psi E - (\epsilon + \varphi + \mu + \delta_I)I, \\ \frac{dQ_I}{dt} &= \varphi I - (\xi + \mu + \sigma_I)Q_I, \\ \frac{dR}{dt} &= \xi Q_I + \epsilon I - \mu R. \end{aligned} \tag{2}$$

3.1. Local Stability of the Disease-Free Equilibrium of the Influenza Sub-Model

All the steady-state solutions of the influenza sub-model are computed by setting the right-hand sides of (2) to zero.

$$\begin{aligned} \Lambda - (\beta_1 E + \beta_2 I + \eta + \mu)S &= 0, \\ \eta S - (\phi(\beta_1 E + \beta_2 I) + \mu)V &= 0, \\ (\beta_1 E + \beta_2 I)S + \phi(\beta_1 E + \beta_2 I)V - (\psi + \mu)E &= 0, \\ \psi E - (\epsilon + \varphi + \mu + \delta_I)I &= 0, \\ \varphi I - (\xi + \mu + \sigma_I)Q_I &= 0, \\ \xi Q_I + \epsilon I - \mu R &= 0. \end{aligned}$$

The disease-free equilibrium point E_0^I is computed by setting the disease states $E = 0$ and $I = 0$, which gives

$$E_0^I = \left(\frac{\Lambda}{\eta + \mu}, \frac{\eta \Lambda}{\mu(\eta + \mu)}, 0, 0, 0, 0 \right), \quad S^0 = \frac{\Lambda}{\eta + \mu}, \quad V^0 = \frac{\eta \Lambda}{\mu(\eta + \mu)}.$$

The basic reproduction number, defined as the number of secondary infections produced by a single, initially infected individual in a completely susceptible population, is computed for the influenza sub-model and denoted R_0^I , using the next-generation matrix method [38]. From Equation (2), we have

$$\mathcal{F} = \begin{bmatrix} (\beta_1 E + \beta_2 I)S + (\beta_1 E + \beta_2 I)\phi V \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} (\psi + \mu)E \\ (\epsilon + \varphi + \mu + \delta_I)I - \psi E \end{bmatrix}.$$

The Jacobian matrices of $\mathcal{F}(x)$ and $\mathcal{V}(x)$ at E_0^I are, respectively,

$$F = D\mathcal{F}(E_0^I) = \begin{bmatrix} \beta_1 S^0 + \beta_1 \phi V^0 & \beta_2 S^0 + \beta_2 \phi V^0 \\ 0 & 0 \end{bmatrix}, \quad V = D\mathcal{V}(E_0^I) = \begin{bmatrix} c_1 & 0 \\ -\psi & c_2 \end{bmatrix}.$$

The inverse of V is

$$V^{-1} = \begin{bmatrix} \frac{1}{c_1} & 0 \\ \frac{\psi}{c_1 c_2} & \frac{1}{c_2} \end{bmatrix},$$

and hence the next-generation matrix FV^{-1} has the single nonzero eigenvalue

$$R_0^I = \frac{\beta_1 \Lambda (\phi \eta + \mu)}{\mu(\eta + \mu)(\psi + \mu)} + \frac{\beta_2 \psi \Lambda (\phi \eta + \mu)}{\mu(\eta + \mu)(\psi + \mu)(\epsilon + \varphi + \mu + \delta_I)}.$$

To examine the local stability of E_0^I , we employ the linearization approach. The Jacobian matrix of (2) evaluated at the disease-free equilibrium E_0^I is

$$J(E_0^I) = \begin{bmatrix} -(\eta + \mu) & 0 & -\beta_1 S^0 & -\beta_2 S^0 & 0 & 0 \\ \eta & -\mu & -\beta_1 \phi V^0 & -\beta_2 \phi V^0 & 0 & 0 \\ 0 & 0 & \beta_1 S^0 + \beta_1 \phi V^0 - c_1 & \beta_2 S^0 + \beta_2 \phi V^0 & 0 & 0 \\ 0 & 0 & \psi & -c_2 & 0 & 0 \\ 0 & 0 & 0 & \varphi & -c_3 & 0 \\ 0 & 0 & 0 & \epsilon & \zeta & -\mu \end{bmatrix}. \quad (3)$$

Four eigenvalues of (3) are $\lambda_1 = -(\eta + \mu)$, $\lambda_{2,3} = -\mu$, and $\lambda_4 = -c_3$; the remaining two are obtained from

$$J_1 = \begin{bmatrix} \beta_1 S^0 + \beta_1 \phi V^0 - c_1 & \beta_2 S^0 + \beta_2 \phi V^0 \\ \psi & -c_2 \end{bmatrix},$$

whose characteristic equation is the quadratic $a_0 \lambda^2 + a_1 \lambda + a_2 = 0$ with

$$a_0 = 1, \quad a_1 = c_2 + c_1(1 - R_0^I) + \frac{\beta_2 \psi (S^0 + \phi V^0)}{c_2}, \quad a_2 = c_1 c_2 (1 - R_0^I).$$

By the Routh–Hurwitz criterion [39], both roots have negative real parts if $a_1 > 0$ and $a_2 > 0$, which hold when $R_0^I < 1$. Hence the disease-free equilibrium E_0^I is locally asymptotically stable for $R_0^I < 1$.

3.2. Endemic Equilibrium and Forward Bifurcation

The endemic equilibrium point of the influenza-only model is obtained by solving

$$\begin{aligned}\Lambda - (\lambda_I + \eta + \mu) S &= 0, \\ \eta S - (\phi \lambda_I + \mu) V &= 0, \\ \lambda_I S + \phi \lambda_I V - (\psi + \mu) E &= 0, \\ \psi E - (\epsilon + \varphi + \mu + \delta_I) I &= 0, \\ \varphi I - (\xi + \mu + \sigma_I) Q_I &= 0, \\ \xi Q_I + \epsilon I - \mu R &= 0.\end{aligned}\tag{4}$$

Let $\Sigma_I = (S^*, V^*, E^*, I^*, Q_I^*, R^*)$ be the endemic equilibrium point. Solving in terms of the endemic force of infection $\lambda_I^* = \beta_1 E^* + \beta_2 I^*$, we obtain

$$\begin{aligned}S^* &= \frac{\Lambda}{\lambda_I^* + \eta + \mu}, \\ V^* &= \frac{\eta \Lambda}{(\lambda_I^* + \eta + \mu)(\lambda_I^* \phi + \mu)}, \\ E^* &= \frac{\Lambda \lambda_I^* ((\lambda_I^* \phi + \mu) + \phi \eta)}{(\lambda_I^* + \eta + \mu)(\lambda_I^* \phi + \mu) c_1}, \\ I^* &= \frac{\psi \Lambda \lambda_I^* ((\lambda_I^* \phi + \mu) + \phi \eta)}{(\lambda_I^* + \eta + \mu)(\lambda_I^* \phi + \mu) c_1 c_2}, \\ Q_I^* &= \frac{\varphi \psi \Lambda \lambda_I^* ((\lambda_I^* \phi + \mu) + \phi \eta)}{(\lambda_I^* + \eta + \mu)(\lambda_I^* \phi + \mu) c_1 c_2 c_3}, \\ R^* &= \frac{\psi \Lambda \lambda_I^* ((\lambda_I^* \phi + \mu) + \phi \eta) (\xi \varphi + \epsilon c_3)}{\mu (\lambda_I^* + \eta + \mu) (\lambda_I^* \phi + \mu) c_1 c_2 c_3}.\end{aligned}\tag{5}$$

Substituting (5) into the definition of λ_I^* shows that a nonzero endemic force of infection must satisfy

$$A_0 (\lambda_I^*)^2 + A_1 \lambda_I^* + A_2 = 0,\tag{6}$$

where

$$A_0 = \phi c_1, \quad A_1 = \mu c_1 + \phi(\eta + \mu) V^0 \left(\beta_1 \phi + \frac{\beta_2 \psi}{c_2} \right) + \phi(\eta + \mu) c_1 (1 - R_0^I), \quad A_2 = \mu(\eta + \mu) c_1 (1 - R_0^I).$$

We now determine the number of admissible (positive) roots of (6). For all positive parameter values, $A_0 = \phi c_1 > 0$.

- Case $R_0^I > 1$: here $A_2 = \mu(\eta + \mu) c_1 (1 - R_0^I) < 0$, so the product of the two roots, A_2/A_0 , is negative. Equation (6) therefore has exactly one positive root,

$$\lambda_I^* = \frac{-A_1 + \sqrt{A_1^2 - 4A_0 A_2}}{2A_0} > 0,$$

and, by (5), a unique endemic equilibrium Σ_I exists.

- Case $R_0^I < 1$: here $1 - R_0^I > 0$, so every term of A_1 is positive and $A_1 > 0$; likewise $A_2 = \mu(\eta + \mu) c_1 (1 - R_0^I) > 0$. All three coefficients A_0, A_1, A_2 are then strictly positive, so (6) admits no positive root, and no endemic equilibrium exists.

- Case $R_0^I = 1$: here $A_2 = 0$ and $A_1 = \mu c_1 + \phi(\eta + \mu)V^0(\beta_1\phi + \beta_2\psi/c_2) > 0$, so the only roots are $\lambda_I^* = 0$ (the disease-free equilibrium) and $\lambda_I^* = -A_1/A_0 < 0$; hence no positive endemic equilibrium bifurcates below threshold.

Consequently, the influenza sub-model possesses no endemic equilibrium for $R_0^I < 1$ and a unique endemic equilibrium for $R_0^I > 1$. The two branches meet at $R_0^I = 1$, where the disease-free and endemic equilibria exchange stability. Because all coefficients of (6) are positive when $R_0^I < 1$, no sub-threshold (subcritical) endemic branch can occur; the model therefore undergoes a forward (supercritical) transcritical bifurcation at $R_0^I = 1$, and no backward bifurcation arises from the imperfect vaccination structure.

3.3. Local Stability of the Endemic Equilibrium

Since the bifurcation at $R_0^I = 1$ is forward, the unique endemic equilibrium Σ_I that emerges for $R_0^I > 1$ inherits the stability lost by the disease-free equilibrium at the threshold. More precisely, at a supercritical transcritical bifurcation the emerging branch is locally asymptotically stable, so Σ_I is locally asymptotically stable for R_0^I in a right neighbourhood of unity. Numerical simulations reported in Section 6 indicate that Σ_I remains locally asymptotically stable and attracts all positive trajectories for the broader parameter ranges considered, so that once influenza has invaded it settles at a persistent positive level. In contrast to a backward bifurcation, the forward bifurcation established above guarantees that reducing R_0^I below unity is sufficient to eliminate influenza in the sub-model, since no endemic equilibrium then exists.

4. Analysis of the *Streptococcus pneumoniae* Sub-Model

Excluding the influenza infection and the co-infected class, we retain only the *Streptococcus pneumoniae* dynamics. Since vaccination in the full model is specific to influenza, it plays no epidemiological role in the absence of influenza; we therefore set $\eta = 0$ in this sub-model, so that the vaccinated class is empty and the susceptibles form a single pool. The force of infection is

$$\lambda_P(t) = \beta_4 P_C(t) + \beta_5 P_T(t),$$

where β_4 and β_5 denote the effective transmission rates from the colonization-stage and transmission-stage pneumococcal classes, respectively. The sub-model of Equation (1) without influenza infection is

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - (\lambda_P + \mu) S, \\ \frac{dP_C}{dt} &= \lambda_P S - (\theta + \mu) P_C, \\ \frac{dP_T}{dt} &= \theta P_C - (\zeta + \pi + \omega + \mu + \delta_T) P_T, \\ \frac{dP_V}{dt} &= \pi P_T - (\gamma + \chi + \mu + \delta_V) P_V, \\ \frac{dQ_P}{dt} &= \omega P_T + \gamma P_V - (\tau + \mu + \sigma_P) Q_P, \\ \frac{dR}{dt} &= \zeta P_T + \chi P_V + \tau Q_P - \mu R. \end{aligned} \tag{7}$$

4.1. Local Stability of the Disease-Free Equilibrium of the Pneumococcal Sub-Model

The disease-free equilibrium point of (7) is

$$E_0^P = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0 \right), \quad S^0 = \frac{\Lambda}{\mu},$$

and the basic reproduction number of the pneumococcus-only model is

$$R_0^P = \frac{\beta_4 \Lambda}{\mu d_1} + \frac{\beta_5 \Lambda \theta}{\mu d_1 d_2}.$$

We note that this disease-free susceptible pool of size Λ/μ coincides with the combined susceptible-and-vaccinated pool $S^0 + V^0 = \Lambda/\mu$ that is available for pneumococcal colonization in the full model, where vaccinated individuals are colonized at the same force of infection λ_P as susceptibles. Consequently, R_0^P equals the pneumococcal invasion number R_0^{PC} derived for the full system in Section 5, and we use R_0^P for both throughout.

Theorem 2. *The disease-free equilibrium point E_0^P of the *Streptococcus pneumoniae* sub-model is locally asymptotically stable if $R_0^P < 1$.*

Proof. The Jacobian matrix of the *Streptococcus pneumoniae*-only model at E_0^P is

$$J(E_0^P) = \begin{bmatrix} -\mu & -\beta_4 S^0 & -\beta_5 S^0 & 0 & 0 & 0 \\ 0 & \beta_4 S^0 - d_1 & \beta_5 S^0 & 0 & 0 & 0 \\ 0 & \theta & -d_2 & 0 & 0 & 0 \\ 0 & 0 & \pi & -d_3 & 0 & 0 \\ 0 & 0 & \omega & \gamma & -d_4 & 0 \\ 0 & 0 & \zeta & \chi & \tau & -\mu \end{bmatrix}. \quad (8)$$

The matrix is block lower-triangular. The S -column yields the eigenvalue $-\mu$; the lower-right block in (P_V, Q_P, R) is triangular with eigenvalues $-d_3$, $-d_4$, and $-\mu$; thus four eigenvalues of (8) are $\lambda_{1,2} = -\mu$, $\lambda_3 = -d_4$, and $\lambda_4 = -d_3$, all negative. The remaining two are obtained from

$$J_2 = \begin{bmatrix} \beta_4 S^0 - d_1 & \beta_5 S^0 \\ \theta & -d_2 \end{bmatrix},$$

whose characteristic equation is the quadratic $a_0 \lambda^2 + a_1 \lambda + a_2 = 0$ with

$$a_0 = 1, \quad a_1 = d_2 + d_1(1 - R_0^P) + \frac{\beta_5 \theta S^0}{d_2}, \quad a_2 = d_1 d_2 (1 - R_0^P).$$

By the Routh–Hurwitz criterion, $a_1 > 0$ and $a_2 > 0$ hold when $R_0^P < 1$. Hence, E_0^P is locally asymptotically stable if $R_0^P < 1$. $\square$

4.2. Endemic Equilibrium

The endemic equilibrium point of the *Streptococcus pneumoniae* sub-model is obtained by solving

$$\begin{aligned} \Lambda - (\lambda_P + \mu) S &= 0, \\ \lambda_P S - (\theta + \mu) P_C &= 0, \\ \theta P_C - (\zeta + \pi + \omega + \mu + \delta_T) P_T &= 0, \\ \pi P_T - (\gamma + \chi + \mu + \delta_V) P_V &= 0, \\ \omega P_T + \gamma P_V - (\tau + \mu + \sigma_P) Q_P &= 0, \\ \zeta P_T + \chi P_V + \tau Q_P - \mu R &= 0. \end{aligned} \quad (9)$$

Let $\Sigma_P = (S^*, P_C^*, P_T^*, P_V^*, Q_P^*, R^*)$ be the endemic equilibrium point. Solving in terms of the endemic force of infection $\lambda_P^* = \beta_4 P_C^* + \beta_5 P_T^*$, we obtain

$$\begin{aligned}
S^* &= \frac{\Lambda}{\lambda_p^* + \mu}, \\
P_C^* &= \frac{\lambda_p^* \Lambda}{(\lambda_p^* + \mu) d_1}, \\
P_T^* &= \frac{\theta \lambda_p^* \Lambda}{(\lambda_p^* + \mu) d_1 d_2}, \\
P_V^* &= \frac{\pi \theta \lambda_p^* \Lambda}{(\lambda_p^* + \mu) d_1 d_2 d_3}, \\
Q_P^* &= \frac{\omega \theta \lambda_p^* \Lambda d_3 + \gamma \pi \theta \lambda_p^* \Lambda}{(\lambda_p^* + \mu) d_1 d_2 d_3 d_4}, \\
R^* &= \frac{\zeta \theta \lambda_p^* \Lambda d_3 d_4 + \chi \pi \theta \lambda_p^* \Lambda d_4 + \tau (\omega \theta \lambda_p^* \Lambda d_3 + \gamma \pi \theta \lambda_p^* \Lambda)}{\mu (\lambda_p^* + \mu) d_1 d_2 d_3 d_4}.
\end{aligned} \tag{10}$$

Substituting these expressions back into λ_p^* yields the simplified relation

$$\lambda_p^* = \frac{\beta_4 \Lambda d_2 + \beta_5 \Lambda \theta}{d_1 d_2} - \mu = \mu \left(\frac{\beta_4 \Lambda d_2 + \beta_5 \Lambda \theta}{\mu d_1 d_2} - 1 \right) = \mu (R_0^P - 1).$$

Hence, the endemic equilibrium point exists and is unique if and only if $R_0^P > 1$, since λ_p^* is a strictly increasing linear function of R_0^P that is positive precisely for $R_0^P > 1$. In particular, the pneumococcus-only model undergoes a forward bifurcation at $R_0^P = 1$ and exhibits no backward bifurcation.

4.3. Global Stability of the Endemic Equilibrium

Theorem 3. If $R_0^P > 1$, the endemic equilibrium point Σ_P of the *Streptococcus pneumoniae*-only model is globally asymptotically stable in the interior of the feasible region.

Proof. The force of infection $\lambda_P = \beta_4 P_C + \beta_5 P_T$ depends only on the transmission core (S, P_C, P_T) ; the downstream classes P_V, Q_P , and R do not appear in it and do not feed back into the first three equations of (7). We therefore first establish convergence of the core and then treat the downstream classes as an asymptotically forced linear cascade.

Following the Goh–Volterra approach, define

$$\mathcal{L} = \left(S - S^* - S^* \ln \frac{S}{S^*} \right) + \left(P_C - P_C^* - P_C^* \ln \frac{P_C}{P_C^*} \right) + \frac{\beta_5 S^*}{d_2} \left(P_T - P_T^* - P_T^* \ln \frac{P_T}{P_T^*} \right).$$

The function $\mathcal{L}$ is continuously differentiable on the interior of the feasible region, positive definite with respect to (S^*, P_C^*, P_T^*) , and attains its minimum value zero only when $S = S^*$, $P_C = P_C^*$, and $P_T = P_T^*$. Using the equilibrium relations $\Lambda = \lambda_p^* S^* + \mu S^*$, $d_1 P_C^* = \lambda_p^* S^* = \beta_4 P_C^* S^* + \beta_5 P_T^* S^*$, and $d_2 P_T^* = \theta P_C^*$, the time derivative of $\mathcal{L}$ along the solutions of (7) reduces to

$$\begin{aligned}
\frac{d\mathcal{L}}{dt} &= -\mu \frac{(S - S^*)^2}{S} + \beta_4 P_C^* S^* \left(2 - \frac{S^*}{S} - \frac{S}{S^*} \right) \\
&\quad + \beta_5 P_T^* S^* \left(3 - \frac{S^*}{S} - \frac{S P_T P_C^*}{S^* P_T^* P_C} - \frac{P_C P_T^*}{P_C^* P_T} \right).
\end{aligned}$$

Each parenthesized bracket has the form $k - \sum_{j=1}^k u_j$ whose arguments have unit product $\prod_j u_j = 1$; by the arithmetic mean–geometric mean inequality, $\sum_j u_j \geq k$, so both brackets are non-positive, and the first term is manifestly non-positive. Hence $\frac{d\mathcal{L}}{dt} \leq 0$ on the feasible region, with equality if and only if $S = S^*$, $P_C = P_C^*$, and $P_T = P_T^*$.

By Theorem 2 the compact set Ω is positively invariant and absorbing, so LaSalle's invariance principle applies. Let M be the largest invariant set contained in $\{d\mathcal{L}/dt = 0\} = \{S = S^*, P_C = P_C^*, P_T = P_T^*\}$. On M , the variables S , P_C , and P_T are constant at their equilibrium values; in particular $P_T \equiv P_T^*$. Substituting this into the P_V equation of (7) gives $\dot{P}_V = \pi P_T^* - d_3 P_V$, whose only invariant (constant) solution is $P_V \equiv \pi P_T^*/d_3 = P_V^*$. Substituting $P_T \equiv P_T^*$ and $P_V \equiv P_V^*$ into the Q_P equation forces $Q_P \equiv Q_P^*$, and then the R equation forces $R \equiv R^*$. Therefore $M = \{\Sigma_P\}$ is a singleton.

By LaSalle's invariance principle, every solution starting in the interior of Ω converges to Σ_P . Since Σ_P is the unique interior equilibrium and $\mathcal{L}$ is a Lyapunov function for the core, Σ_P is globally asymptotically stable whenever it exists, i.e., for $R_0^P > 1$. Biologically, once pneumococcal infection establishes itself, it persists at a positive endemic level irrespective of the initial sizes of the compartments. $\square$

5. Analysis of the Co-Infection Model

We now determine the equilibrium points of the full model (1) by solving the steady-state system (11).

$$\begin{aligned}
 \Lambda - (\lambda_I + \lambda_P + \eta + \mu)S &= 0, \\
 \eta S - (\phi\lambda_I + \lambda_P + \mu)V &= 0, \\
 \lambda_I S + \phi\lambda_I V - (\psi + \mu)E &= 0, \\
 \psi E + m\alpha C - (\epsilon + \zeta\lambda_P + \varphi + \mu + \delta_I)I &= 0, \\
 \varphi I - (\xi + \mu + \sigma_I)Q_I &= 0, \\
 \lambda_P(S + V) - (\kappa\lambda_I + \theta + \mu)P_C &= 0, \\
 \theta P_C - (\nu\lambda_I + \zeta + \pi + \omega + \mu + \delta_T)P_T &= 0, \\
 \pi P_T + n\alpha C - (\rho\lambda_I + \gamma + \chi + \mu + \delta_V)P_V &= 0, \\
 \omega P_T + \gamma P_V - (\tau + \mu + \sigma_P)Q_P &= 0, \\
 \zeta\lambda_P I + \kappa\lambda_I P_C + \nu\lambda_I P_T + \rho\lambda_I P_V - (\alpha + \mu + \delta_{IP})C &= 0, \\
 \xi Q_I + \epsilon I + \zeta P_T + \chi P_V + \tau Q_P + (1 - (m + n))\alpha C - \mu R &= 0.
 \end{aligned} \tag{11}$$

The disease-free equilibrium point of the full model is

$$E_0 = \left(\frac{\Lambda}{\eta + \mu}, \frac{\eta\Lambda}{\mu(\eta + \mu)}, 0, 0, 0, 0, 0, 0, 0, 0 \right), \quad S^0 = \frac{\Lambda}{\eta + \mu}, \quad V^0 = \frac{\eta\Lambda}{\mu(\eta + \mu)}.$$

Since vaccinated individuals are colonized by *S. pneumoniae* at the same force of infection as susceptibles, the pool eligible for pneumococcal colonization at the disease-free equilibrium is $S^0 + V^0 = \Lambda/\mu$.

5.1. The Basic Reproduction Number

Using the next-generation matrix method with the vector of infected states $X = (E, I, P_C, P_T, P_V, C)$, the new-infection and transition functions are

$$\mathcal{F}(X) = \begin{bmatrix} \lambda_I S + \phi\lambda_I V \\ 0 \\ \lambda_P(S + V) \\ 0 \\ 0 \\ \zeta\lambda_P I + \kappa\lambda_I P_C + \nu\lambda_I P_T + \rho\lambda_I P_V \end{bmatrix} \quad \text{and} \quad \mathcal{V}(X) = \begin{bmatrix} (\psi + \mu)E \\ (\epsilon + \zeta\lambda_P + \varphi + \mu + \delta_I)I - \psi E - m\alpha C \\ (\kappa\lambda_I + \theta + \mu)P_C \\ (\nu\lambda_I + \zeta + \pi + \omega + \mu + \delta_T)P_T - \theta P_C \\ (\rho\lambda_I + \gamma + \chi + \mu + \delta_V)P_V - \pi P_T - n\alpha C \\ (\alpha + \mu + \delta_{IP})C \end{bmatrix}.$$

The Jacobian matrices of $\mathcal{F}(X)$ and $\mathcal{V}(X)$ at E_0 are, using $S^0 + V^0 = \Lambda/\mu$,

$$F = D\mathcal{F}(E_0) = \begin{bmatrix} \beta_1 S^0 + \beta_1 \phi V^0 & \beta_2 S^0 + \beta_2 \phi V^0 & 0 & 0 & 0 & \beta_3 S^0 + \beta_3 \phi V^0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_4 \frac{\Lambda}{\mu} & \beta_5 \frac{\Lambda}{\mu} & 0 & \beta_6 \frac{\Lambda}{\mu} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$V = D\mathcal{V}(E_0) = \begin{bmatrix} c_1 & 0 & 0 & 0 & 0 & 0 \\ -\psi & c_2 & 0 & 0 & 0 & -m\alpha \\ 0 & 0 & d_1 & 0 & 0 & 0 \\ 0 & 0 & -\theta & d_2 & 0 & 0 \\ 0 & 0 & 0 & -\pi & d_3 & -n\alpha \\ 0 & 0 & 0 & 0 & 0 & d_5 \end{bmatrix}.$$

The next-generation matrix FV^{-1} is

$$FV^{-1} = \begin{bmatrix} \frac{(\beta_1 c_2 + \beta_2 \psi)\Lambda(\eta\phi + \mu)}{\mu(\eta + \mu)c_1 c_2} & \frac{\beta_2 \Lambda(\eta\phi + \mu)}{\mu(\eta + \mu)c_2} & 0 & 0 & 0 & \frac{(\beta_2 m\alpha + \beta_3 c_2)\Lambda(\eta\phi + \mu)}{\mu(\eta + \mu)c_1 d_5} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{(\beta_4 d_2 + \beta_5 \theta)\Lambda}{\mu d_1 d_2} & \frac{\beta_5 \Lambda}{\mu d_2} & 0 & \frac{\beta_6 \Lambda}{\mu d_5} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

Three eigenvalues of FV^{-1} are zero, and the remaining nonzero eigenvalues are

$$\lambda_1 = \frac{(\beta_1 c_2 + \beta_2 \psi)\Lambda(\eta\phi + \mu)}{\mu(\eta + \mu)c_1 c_2} = R_0^I, \quad \lambda_2 = \frac{(\beta_4 d_2 + \beta_5 \theta)\Lambda}{\mu d_1 d_2} = R_0^P.$$

Hence the basic reproduction number of the full model (1) is the spectral radius of FV^{-1} ,

$$R_0 = \max\{R_0^I, R_0^P\}.$$

Because vaccinated individuals remain fully susceptible to pneumococcal colonization, the pneumococcal invasion number of the full system draws on the entire pool $S^0 + V^0 = \Lambda/\mu$ and therefore coincides with the stand-alone pneumococcal reproduction number R_0^P ; the two need not be distinguished.

5.2. Local Stability of the Disease-Free Equilibrium of the Full Model

The Jacobian matrix of the full model (1) evaluated at E_0 is given in Equation (12), where $S^0 + V^0 = \Lambda/\mu$.

$$J(E_0) = \begin{bmatrix} -(\eta + \mu) & 0 & -\beta_1 S^0 & -\beta_2 S^0 & 0 & -\beta_4 S^0 & -\beta_5 S^0 & 0 & 0 & -(\beta_3 + \beta_6) S^0 & 0 \\ \eta & -\mu & -\beta_1 \phi V^0 & -\beta_2 \phi V^0 & 0 & -\beta_4 V^0 & -\beta_5 V^0 & 0 & 0 & -(\beta_3 \phi + \beta_6) V^0 & 0 \\ 0 & 0 & \beta_1 S^0 + \beta_1 \phi V^0 - c_1 & \beta_2 S^0 + \beta_2 \phi V^0 & 0 & 0 & 0 & 0 & 0 & \beta_3 S^0 + \beta_3 \phi V^0 & 0 \\ 0 & 0 & \psi & -c_2 & 0 & 0 & 0 & 0 & 0 & m\alpha & 0 \\ 0 & 0 & 0 & \varphi & -c_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_4 \frac{\Lambda}{\mu} - d_1 & \beta_5 \frac{\Lambda}{\mu} & 0 & 0 & \beta_6 \frac{\Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & -d_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \pi & -d_3 & 0 & n\alpha & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \omega & \gamma & -d_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -d_5 & 0 \\ 0 & 0 & 0 & \epsilon & \xi & 0 & 0 & \chi & \tau & (1 - (m + n))\alpha & -\mu \end{bmatrix} \quad (12)$$

Ordering the variables as infected (E, I, P_C, P_T, P_V, C) followed by uninfected (S, V, Q_I, Q_P, R), the matrix $J(E_0)$ is block triangular, because the infected equations do not depend on the uninfected variables at E_0 (all such dependencies enter through the forces of infection, which vanish at E_0). The uninfected block contributes the negative eigenvalues $-(\eta + \mu)$, $-\mu$ (twice), $-c_3$, and $-d_4$, while the non-transmitting infected classes P_V and C contribute $-d_3$ and $-d_5$. The remaining four eigenvalues are obtained from the reduced matrix

$$J_3 = \begin{bmatrix} \beta_1 S^0 + \beta_1 \phi V^0 - c_1 & \beta_2 S^0 + \beta_2 \phi V^0 & 0 & 0 \\ \psi & -c_2 & 0 & 0 \\ 0 & 0 & \beta_4 \frac{\Lambda}{\mu} - d_1 & \beta_5 \frac{\Lambda}{\mu} \\ 0 & 0 & \theta & -d_2 \end{bmatrix}.$$

The matrix J_3 is block-diagonal, decoupling into an influenza block and a pneumococcal block. This block-diagonal structure expresses a symmetry of the linearized dynamics at the disease-free equilibrium: the two infection modules are mutually decoupled to first order, each governed by its own reproduction number, and the diseases are linked only through the co-infection interactions, whose contributions are of second order and hence vanish at E_0 . The influenza block has eigenvalues with negative real parts if and only if $R_0^I < 1$ (the condition already obtained for J_1), while the pneumococcal block does so if and only if $R_0^P < 1$ (the Routh–Hurwitz argument used for J_2 , now with the colonization pool Λ/μ). Consequently, all eleven eigenvalues of $J(E_0)$ have negative real parts precisely when

$$R_0 = \max\{R_0^I, R_0^P\} < 1,$$

so the disease-free equilibrium E_0 of the full model is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

5.3. Global Stability of the Disease-Free Equilibrium

Owing to the coupling between the two infections through the co-infected class, the endemic equilibrium of the full model cannot be written in closed form; its existence and the persistence of the co-infection for $R_0 > 1$ are demonstrated numerically in Section 6. For the disease-free equilibrium we apply the method of Castillo-Chavez et al. Writing the full model (1) as

$$\begin{cases} \frac{dX}{dt} = F(X, Z), \\ \frac{dZ}{dt} = G(X, Z), \quad G(X, 0) = 0, \end{cases} \quad X = (S, V, R), \quad Z = (E, I, Q_I, P_C, P_T, P_V, Q_P, C),$$

with $X \in \mathbb{R}_+^3$ the uninfected compartments and $Z \in \mathbb{R}_+^8$ the infected compartments, the disease-free state is $U_0 = (X_0, 0)$ with $X_0 = (\frac{\Lambda}{\eta + \mu}, \frac{\eta \Lambda}{\mu(\eta + \mu)}, 0)$. Global asymptotic stability of E_0 for $R_0 < 1$ is guaranteed if the following two conditions hold:

- (H1) for $\frac{dX}{dt} = F(X, 0)$, the equilibrium X_0 is globally asymptotically stable;
 (H2) $G(X, Z) = AZ - \widehat{G}(X, Z)$ with $\widehat{G}(X, Z) \geq 0$ for $(X, Z) \in \Omega$ and $A = D_Z G(X_0, 0)$ a Metzler matrix.

From the model,

$$F(X, Z) = \begin{bmatrix} \Lambda - (\lambda_I + \lambda_P + \eta + \mu)S \\ \eta S - (\phi\lambda_I + \lambda_P + \mu)V \\ \xi Q_I + \epsilon I + \zeta P_T + \chi P_V + \tau Q_P + (1 - (m + n))\alpha C - \mu R \end{bmatrix}, \quad F(X, 0) = \begin{bmatrix} \Lambda - (\eta + \mu)S \\ \eta S - \mu V \\ -\mu R \end{bmatrix}.$$

The reduced system $\dot{X} = F(X, 0)$ is linear with the globally asymptotically stable equilibrium X_0 (its coefficient matrix has eigenvalues $-(\eta + \mu)$, $-\mu$, $-\mu$), so condition (H1) holds. Next,

$$G(X, Z) = \begin{bmatrix} \lambda_I S + \phi\lambda_I V - (\psi + \mu)E \\ \psi E + m\alpha C - (\epsilon + \zeta\lambda_P + \phi + \mu + \delta_I)I \\ \phi I - (\xi + \mu + \sigma_I)Q_I \\ \lambda_P(S + V) - (\kappa\lambda_I + \theta + \mu)P_C \\ \theta P_C - (\nu\lambda_I + \zeta + \pi + \omega + \mu + \delta_T)P_T \\ \pi P_T + n\alpha C - (\rho\lambda_I + \gamma + \chi + \mu + \delta_V)P_V \\ \omega P_T + \gamma P_V - (\tau + \mu + \sigma_P)Q_P \\ \zeta\lambda_P I + \kappa\lambda_I P_C + \nu\lambda_I P_T + \rho\lambda_I P_V - (\alpha + \mu + \delta_{IP})C \end{bmatrix}.$$

The associated Metzler matrix $A = D_Z G(X_0, 0)$ is

$$A = \begin{bmatrix} \beta_1 S^0 + \beta_1 \phi V^0 - c_1 & \beta_2 S^0 + \beta_2 \phi V^0 & 0 & 0 & 0 & 0 & 0 & \beta_3 S^0 + \beta_3 \phi V^0 \\ \psi & -c_2 & 0 & 0 & 0 & 0 & 0 & m\alpha \\ 0 & \phi & -c_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_4 \frac{\Lambda}{\mu} - d_1 & \beta_5 \frac{\Lambda}{\mu} & 0 & 0 & \beta_6 \frac{\Lambda}{\mu} \\ 0 & 0 & 0 & \theta & -d_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \pi & -d_3 & 0 & n\alpha \\ 0 & 0 & 0 & 0 & \omega & \gamma & -d_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -d_5 \end{bmatrix},$$

and $\widehat{G}(X, Z) = AZ - G(X, Z)$ evaluates to

$$\widehat{G}(X, Z) = \begin{bmatrix} \lambda_I(S^0 - S) + \phi\lambda_I(V^0 - V) \\ \zeta\lambda_P I \\ 0 \\ \lambda_P((S^0 - S) + (V^0 - V)) + \kappa\lambda_I P_C \\ \nu\lambda_I P_T \\ \rho\lambda_I P_V \\ 0 \\ -(\zeta\lambda_P I + \kappa\lambda_I P_C + \nu\lambda_I P_T + \rho\lambda_I P_V) \end{bmatrix}.$$

Within the feasible region Ω we have $S + V \leq N \leq \Lambda/\mu = S^0 + V^0$, so the fourth component of $\widehat{G}$ is non-negative; the first, second, fifth, and sixth components are likewise non-negative. However, the last component,

$$-(\zeta\lambda_P I + \kappa\lambda_I P_C + \nu\lambda_I P_T + \rho\lambda_I P_V),$$

is strictly negative whenever any infected class is positive. Hence $\widehat{G}(X, Z) \geq 0$ fails and condition (H2) is not satisfied.

We emphasize that the failure of (H2) does not imply instability of E_0 , the existence of a sub-threshold endemic equilibrium, or a backward bifurcation; it means only that this particular sufficient condition cannot be used to certify global stability. Such a failure is intrinsic to co-infection models in which singly infected individuals acquire the second pathogen and pass directly into the co-infected class C: the infected-to-infected infection flows $\zeta\lambda_P I$, $\kappa\lambda_I P_C$, $\nu\lambda_I P_T$, and $\rho\lambda_I P_V$ cannot be absorbed into the Metzler decomposition required by (H2). Accordingly, we do not claim global stability of E_0 through this theorem. Nevertheless, both single-infection sub-models undergo forward bifurcations with no endemic equilibrium below threshold (Sections 3 and 4), and E_0 is locally asymptotically stable whenever $R_0 < 1$; the numerical simulations in Section 6 show that trajectories starting from a range of initial conditions converge to E_0 for $R_0 < 1$ and to a co-infection endemic state for $R_0 > 1$. These results indicate that E_0 is globally attracting when $R_0 < 1$, a full analytical proof of which remains open. Biologically, they indicate that reducing R_0 below unity is expected to eliminate the co-infection, while the persistence of infection for $R_0 > 1$ underscores the role of vaccination and quarantine in lowering R_0 .

6. Numerical Simulation

In this section we illustrate the analytical results of the preceding sections through numerical simulations of the full co-infection model (1). All computations were performed with the ode45 routine in MATLAB 2023a, an adaptive one-step solver for non-stiff problems based on the explicit Runge–Kutta (4,5) Dormand–Prince pair. This solver was chosen because the system (1) is non-stiff over the epidemic horizon considered, and ode45 provides fifth-order accuracy with automatic step-size control at negligible cost for an eleven-dimensional system; its accuracy is quantified in Section 6.7. The relative and absolute error tolerances were set to $\text{RelTol} = 10^{-8}$ and $\text{AbsTol} = 10^{-10}$, and non-negativity of all state variables was enforced through the solver options. Time is measured in weeks, and all rate parameters are expressed per week.

Unless stated otherwise, the simulations use the single initial condition

$$\begin{aligned} S(0) &= 16760, V(0) = 1471, E(0) = 368, I(0) = 653, Q_I(0) = 165, P_C(0) = 1744, \\ P_T(0) &= 399, P_V(0) = 204, Q_P(0) = 136, C(0) = 75, R(0) = 1025, \end{aligned}$$

for which the initial total population is $N(0) = 23,000$, lying inside the feasible region since $N(0) \leq \Lambda/\mu = 23,437.5$.

6.1. Parameter Values

The parameter values are listed in Tables 1 and 2 and are classified by source. Parameters accompanied by a citation were adopted from published studies. The remaining parameters are marked Assumed: they describe transition processes—such as progression between the pneumococcal colonization, transmission, and invasion stages, the co-infection risk factors, and the stage-specific quarantine and recovery rates—that are not routinely recorded in surveillance data, and were therefore assigned biologically plausible values rather than fitted to observations. Because the aim of the simulations is to confirm the analytical findings rather than to forecast a specific outbreak, the qualitative conclusions depend on the sign of $R_0 - 1$ and the invasion thresholds below, not on the precise numerical values of the assumed parameters.

The parameter μ requires comment. Since the model is analysed over an epidemic horizon rather than a demographic lifetime, μ represents the per-capita rate of exit (turnover) from the active population—encompassing background mortality, out-migration, and loss to follow-up—rather than the reciprocal of the human life expectancy. With

$\mu = 0.064 \text{ week}^{-1}$, the mean residence time in the active population is about $1/\mu \approx 15.6$ weeks, and the turnover-balanced population size is $\Lambda/\mu \approx 23,438$.

Table 1. Influenza-related parameter values (rates per week).

Parameter	Value	Source
Λ	1500	Assumed
μ	0.064	Assumed (turnover)
η	0.25	Assumed
ϕ	0.6	Assumed
β_1	2.0×10^{-6}	Assumed
β_2	2.1×10^{-6}	Assumed
β_3	1.9×10^{-6}	Assumed
ψ	0.994	[40]
ϵ	0.067	[41]
φ	0.4	Assumed
ζ	0.045	Assumed
σ_I	0.00025	Assumed
δ_I	0.021	[42]

All rates are per week. Source: Assumed = selected within a biologically plausible range (not fitted to data); turnover = per-capita exit rate from the active population over the epidemic horizon; a citation denotes a value adopted from the literature.

Table 2. Pneumococcal- and co-infection-related parameter values (rates per week).

Parameter	Value	Source
β_4	1.7×10^{-6}	Assumed
β_5	1.5×10^{-6}	Assumed
β_6	1.2×10^{-6}	Assumed
θ	0.5	Assumed
π	0.11	Assumed
ω	0.55	Assumed
ζ	0.0714	Assumed
γ	0.7	Assumed
χ	0.12	Assumed
τ	0.09	Assumed
σ_P	0.00021	Assumed
δ_T	0.08	Assumed
δ_V	0.05	Assumed
δ_{IP}	0.07	Assumed
α	0.11	Assumed
m	0.0018	Assumed
n	0.0023	Assumed
κ	0.16	Assumed
ν	0.11	Assumed
ρ	0.18	Assumed
ς	0.12	Assumed

All rates are per week. Assumed = selected within a biologically plausible range (not fitted to data).

6.2. Threshold Dynamics

With the baseline values of Tables 1 and 2, the reproduction numbers are $R_0^I = 0.0873$ and $R_0^P = 0.1062$, so that $R_0 = \max\{R_0^I, R_0^P\} = 0.1062 < 1$. Notably, the baseline value is pneumococcus-driven ($R_0^P > R_0^I$): because vaccination transfers a large fraction of the population into V , and vaccinated individuals remain fully susceptible to pneumococcal colonization while only partially susceptible to influenza, the effective colonization pool favours pneumococcus. Figure 2 shows that, for $R_0 < 1$, all infected compartments decay

to zero and the solution approaches the disease-free equilibrium, consistent with its local asymptotic stability established in Section 5.

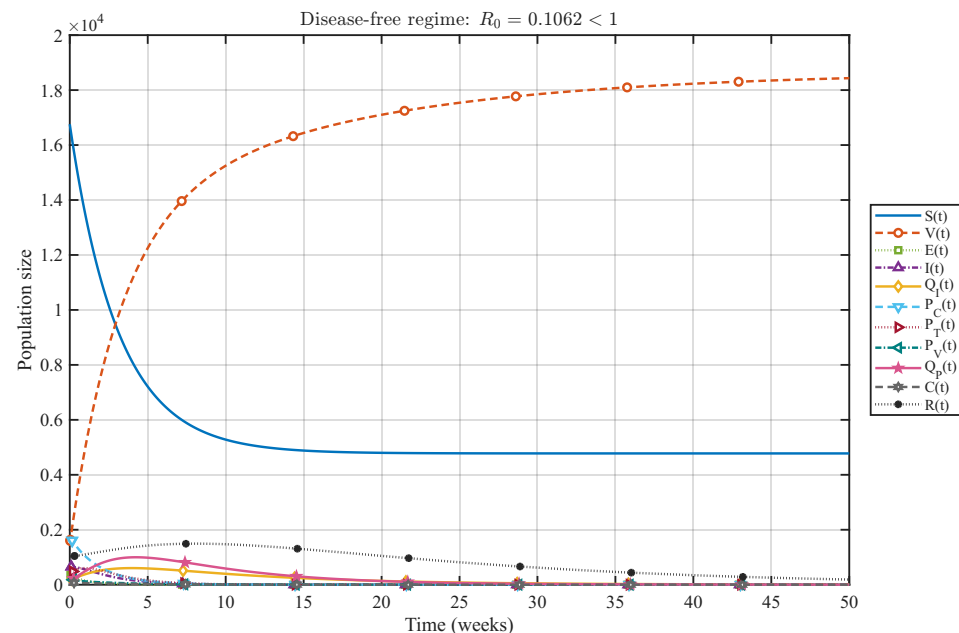


Figure 2. Time evolution of all eleven compartments in the disease-free regime $R_0 = 0.1062 < 1$. Every infected class tends to zero, and the trajectory converges to the disease-free equilibrium.

To illustrate the endemic regime we increase the influenza transmission coefficient to $\beta_2 = 9.0 \times 10^{-6} \cdot 10 = 9.0 \times 10^{-5}$, keeping all other parameters at their baseline values; this single change raises R_0^I to 2.477 while leaving $R_0^P = 0.1062$ unchanged, so $R_0 = 2.477 > 1$. Figure 3 shows that the influenza-related compartments settle at positive endemic levels while the pneumococcal compartments vanish, because $R_0^P < 1$. The computed disease-free and endemic equilibria, together with the corresponding reproduction numbers, are reported in Table 3.

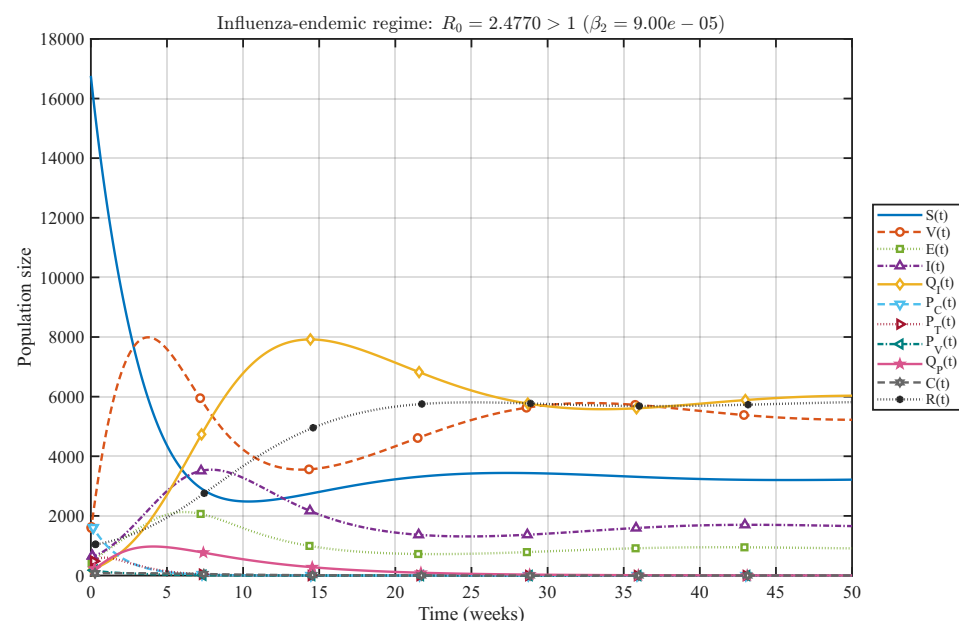


Figure 3. Time evolution of all eleven compartments in the influenza-endemic regime $R_0 = 2.477 > 1$, obtained by setting $\beta_2 = 9.0 \times 10^{-5}$ (all other parameters at baseline). The influenza classes persist at positive levels; the pneumococcal classes vanish because $R_0^P < 1$.

Table 3. Computed reproduction numbers and equilibrium values (to four significant figures) for the two baseline scenarios. Values below 10^{-6} are reported as 0.

	Disease-Free ($R_0 = 0.106$)	Influenza-Endemic ($R_0 = 2.477$)
S^*	4777.1	3250.6
V^*	18,660	5330.0
E^*	0	898.7
I^*	0	1618.3
Q_I^*	0	5925.2
P_C^*	0	0
P_T^*	0	0
P_V^*	0	0
Q_P^*	0	0
C^*	0	0
R^*	0	5860.4
R_0^I	0.0873	2.477
R_0^P	0.1062	0.1062
R_0	0.1062	2.477

6.3. Forward Bifurcation

The corrected analysis of Section 3 shows that the influenza sub-model undergoes a forward (supercritical) transcritical bifurcation at $R_0^I = 1$, with no sub-threshold endemic equilibrium. Figure 4 confirms this numerically: the endemic level I^* is zero for $R_0^I < 1$ and increases continuously from zero as R_0^I crosses unity. Across the sub-threshold range the computed endemic level never exceeds 10^{-11} (numerical zero), so no bistable or backward-bifurcation behaviour is present.

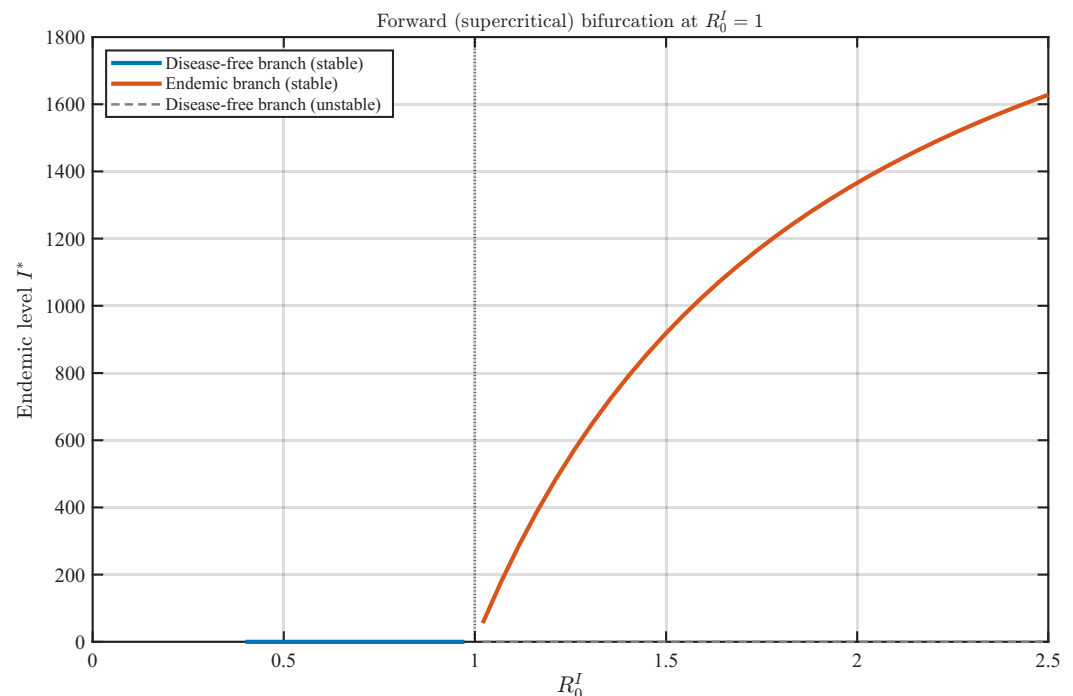


Figure 4. Endemic infected level I^* as a function of R_0^I (varied through β_2). The disease-free branch is stable for $R_0^I < 1$ and unstable for $R_0^I > 1$, and the endemic branch emerges continuously at $R_0^I = 1$, confirming a forward bifurcation.

6.4. Competitive Exclusion and Invasion Numbers

When both single-infection thresholds exceed unity, the two infections compete for the shared susceptible pool. Because the model contains no mechanism that favours the doubly

infected state, this competition results in competitive exclusion: one infection persists while the other, together with the co-infected class, is driven to extinction. The outcome is not determined by the disease-free reproduction numbers, which are evaluated at the pathogen-free state, but by the invasion reproduction numbers $\mathcal{R}_{P|I}$ and $\mathcal{R}_{I|P}$. Here $\mathcal{R}_{P|I}$ is the reproduction number of pneumococcus when rare in a population held at the influenza-endemic boundary equilibrium, and $\mathcal{R}_{I|P}$ is defined symmetrically; each is computed as the spectral radius of the next-generation matrix of the invading infection evaluated at the resident equilibrium. Pneumococcus persists if $\mathcal{R}_{P|I} > 1$, influenza persists if $\mathcal{R}_{I|P} > 1$, and the surviving infection is the one able to invade the other's equilibrium.

Table 4 reports the invasion numbers for several parameter sets in which both disease-free thresholds exceed unity, together with the simulated outcome. In every case exactly one invasion number exceeds unity, and it correctly predicts the surviving infection; the co-infected level C converges to numerical zero throughout. Importantly, the disease-free reproduction numbers do not govern the competition: when $R_0^I = R_0^P$ (rows with targets (3,3) and (4,4)), the infections neither coexist nor tie—influenza excludes pneumococcus, as predicted by $\mathcal{R}_{I|P} > 1 > \mathcal{R}_{P|I}$. Figure 5 displays two representative runs from the same initial condition: in panel (a) influenza excludes pneumococcus, and in panel (b) the reverse occurs.

Table 4. Invasion reproduction numbers at the single-infection boundary equilibria and the resulting competitive outcome. Both disease-free thresholds exceed unity in every row. The final column reports the simulated co-infected level, which converges to numerical zero, confirming that co-infection does not persist.

R_0^I	R_0^P	$\mathcal{R}_{P I}$	$\mathcal{R}_{I P}$	Outcome	C (Simulated)
2	3	1.359	0.741	pneumococcus persists	$\sim 10^{-12}$
2	4	1.811	0.571	pneumococcus persists	$\sim 10^{-12}$
3	3	0.842	1.109	influenza persists	$\sim 10^{-117}$
3	4	1.122	0.853	pneumococcus persists	$\sim 10^{-11}$
4	4	0.793	1.136	influenza persists	$\sim 10^{-157}$

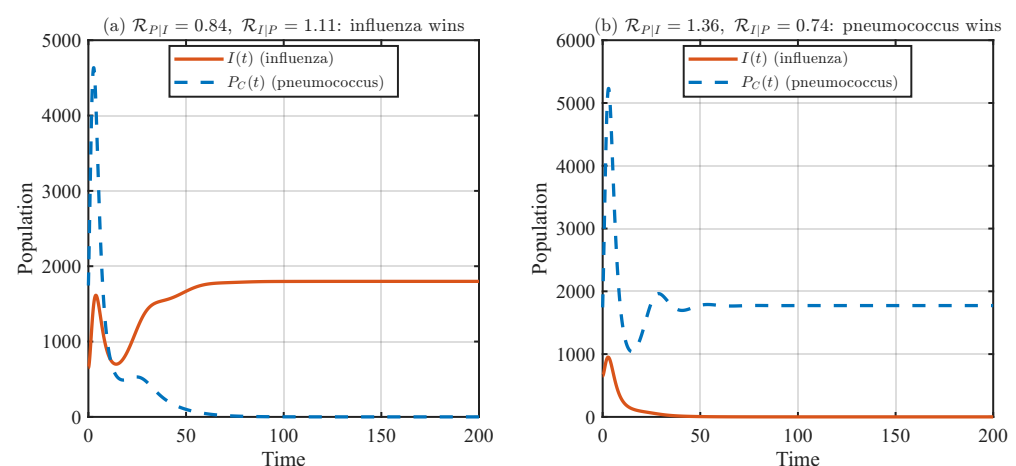


Figure 5. Competitive exclusion from a common initial condition. (a) With $\mathcal{R}_{P|I} = 0.84 < 1 < \mathcal{R}_{I|P} = 1.11$, influenza persists ($I(t)$) and pneumococcal colonization ($P_C(t)$) is excluded. (b) With $\mathcal{R}_{P|I} = 1.36 > 1 > \mathcal{R}_{I|P} = 0.74$, the outcome is reversed. Extinction of the excluded infection is exponential; the residual reflects numerical, not biological, persistence.

6.5. Effect of Control Parameters

We next examine how the transmission and quarantine parameters shape the epidemic. In each experiment one parameter is varied while the others are held at baseline, and the

target reproduction numbers are obtained analytically by inverting the R_0 expressions of Section 5. Figure 6 shows the effect of the influenza transmission coefficient β_2 on $I(t)$, of the influenza quarantine rate φ on $I(t)$, and of the pneumococcal transmission coefficient β_4 on $P_C(t)$; the corresponding peak, terminal, and cumulative values are summarized in Table 5.

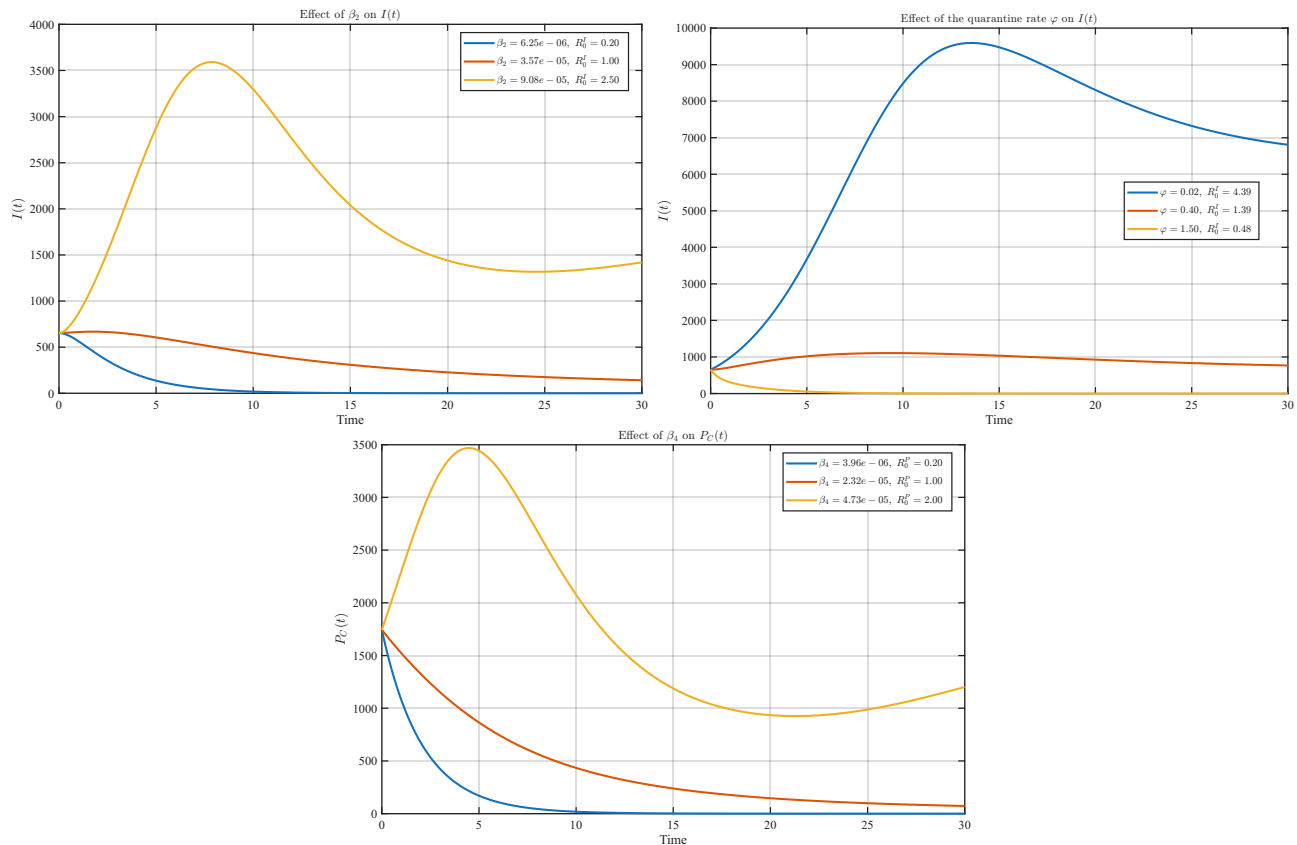


Figure 6. Parameter-variation study. Increasing β_2 (top left) or β_4 (bottom) raises the corresponding reproduction number and shifts the dynamics from decay to persistence, whereas increasing the quarantine rate φ (top right) lowers R_0^I and suppresses the influenza epidemic.

Table 5. Effect of the control parameters on the reproduction number and on the epidemic burden. Peak, terminal, and cumulative values refer to $I(t)$ for the β_2 and φ experiments and to $P_C(t)$ for the β_4 experiment.

Parameter	Value	R_0^I	Peak	Terminal	Cumulative
β_2	6.25×10^{-6}	0.20	653.0	0.02	2228
β_2	3.57×10^{-5}	1.00	668.3	141.5	10,811
β_2	9.08×10^{-5}	2.50	3590.9	1420.1	60,256
φ	0.02	4.39	9592.7	6807.8	2.06×10^5
φ	0.40	1.39	1106.7	767.5	28,295
φ	1.50	0.48	653.0	0.006	1141
R_0^P					
β_4	3.96×10^{-6}	0.20	1744.0	0.009	3721
β_4	2.32×10^{-5}	1.00	1744.0	72.6	12,978
β_4	4.73×10^{-5}	2.00	3468.5	1201.2	51,610

The quarantine experiment is the most informative from a control standpoint: raising φ from 0.02 to 1.50 week⁻¹ reduces R_0^I from 4.39 to 0.48, crossing the threshold, and lowers

the cumulative influenza burden by more than two orders of magnitude (from 2.06×10^5 to 1.14×10^3). Combined with the exclusion analysis of Section 6.4, this indicates that interventions targeting the dominant infection can, in principle, allow the competing infection to establish, an effect that vaccination and quarantine policies should account for.

6.6. Effect of Initial Conditions

To confirm that the long-term behaviour is independent of the starting state, we integrate the model from six initial conditions, each summing to the same total population $N(0) = 23,000$. Figures 7–17 show the trajectories of the individual compartments. Although the transient dynamics differ, all solutions converge to the same equilibrium determined by R_0 , in agreement with the stability results.

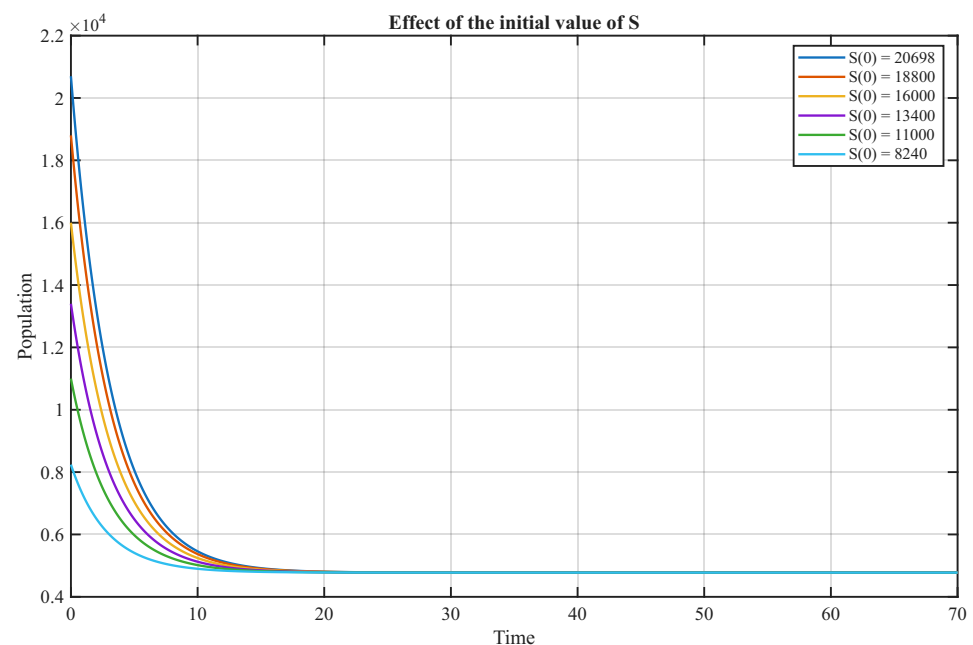


Figure 7. Susceptible population $S(t)$ for different initial values $S(0)$.

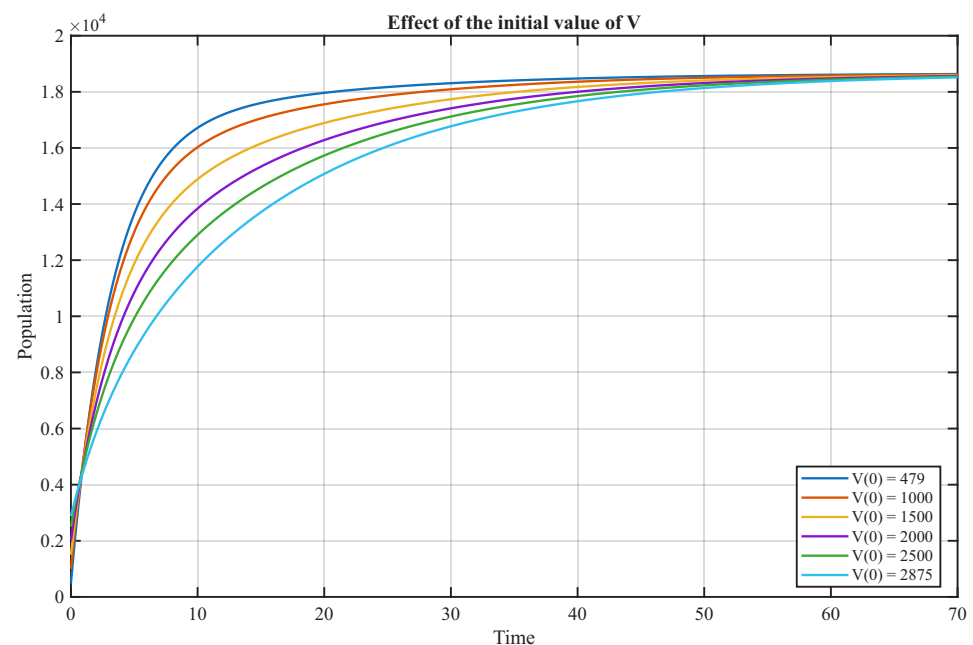


Figure 8. Vaccinated population $V(t)$ for different initial values $V(0)$.

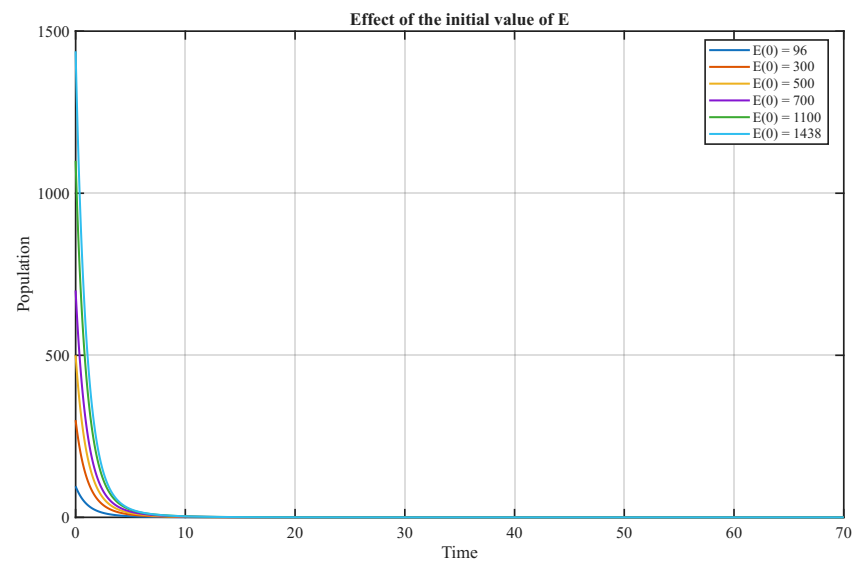


Figure 9. Exposed population $E(t)$ for different initial values $E(0)$.

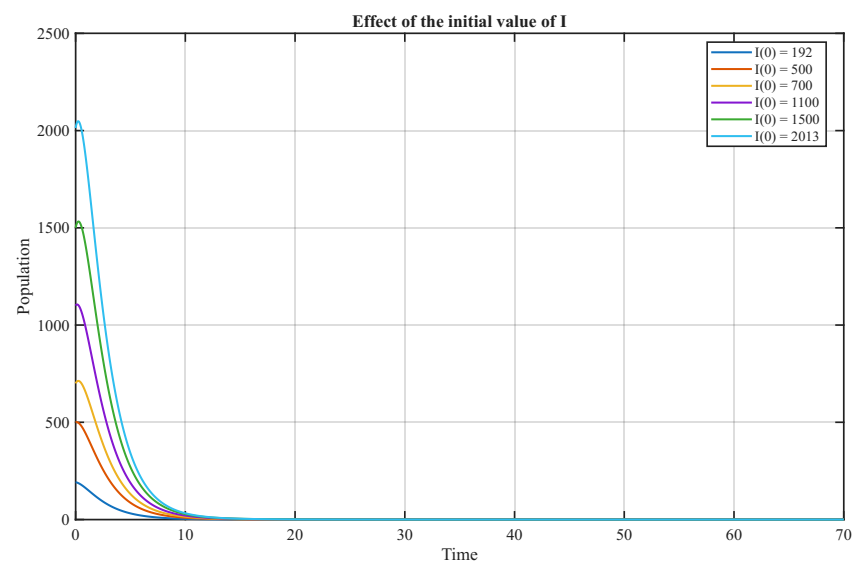


Figure 10. Influenza-infected population $I(t)$ for different initial values $I(0)$.

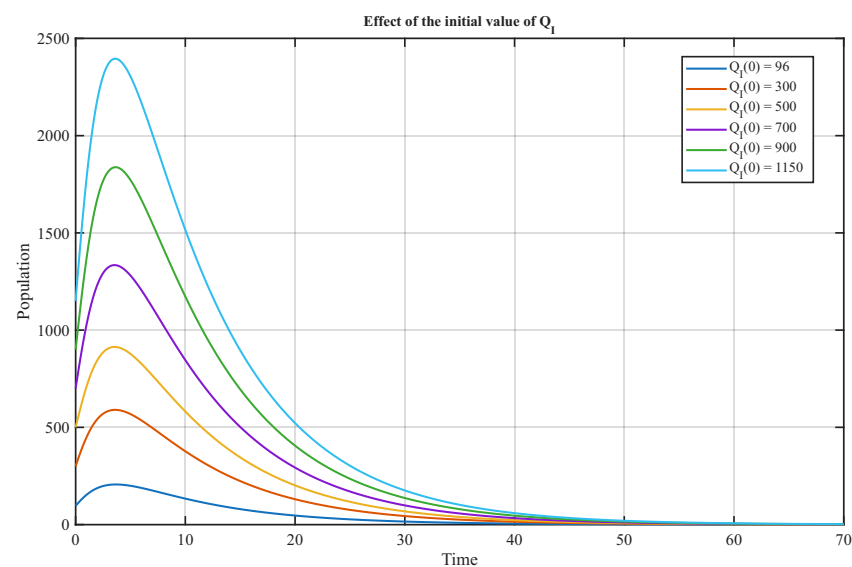


Figure 11. Quarantined influenza population $Q_I(t)$ for different initial values $Q_I(0)$.

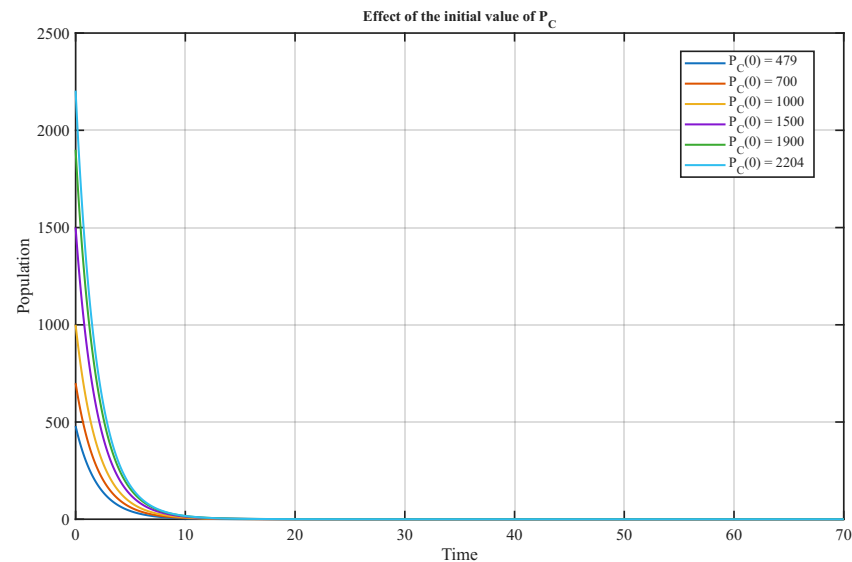


Figure 12. Colonization-stage pneumococcal population $P_C(t)$ for different initial values $P_C(0)$.

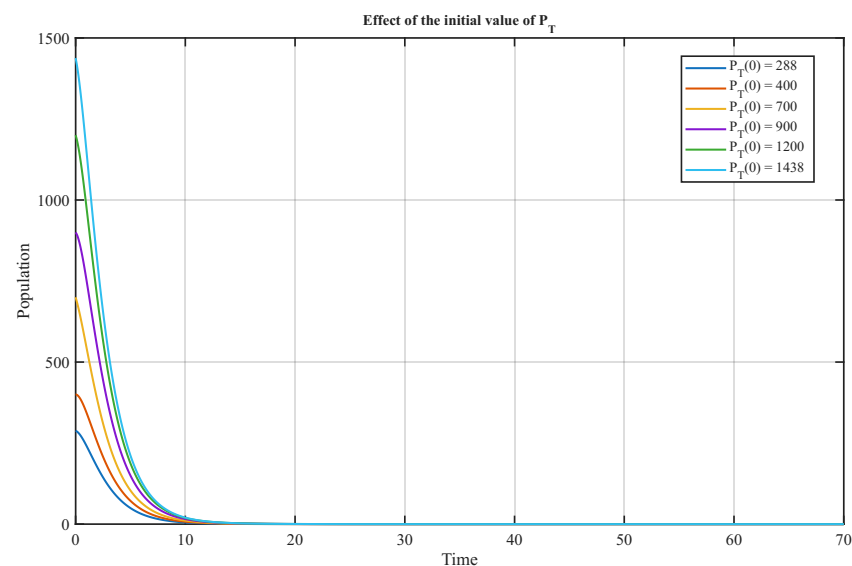


Figure 13. Transmission-stage pneumococcal population $P_T(t)$ for different initial values $P_T(0)$.

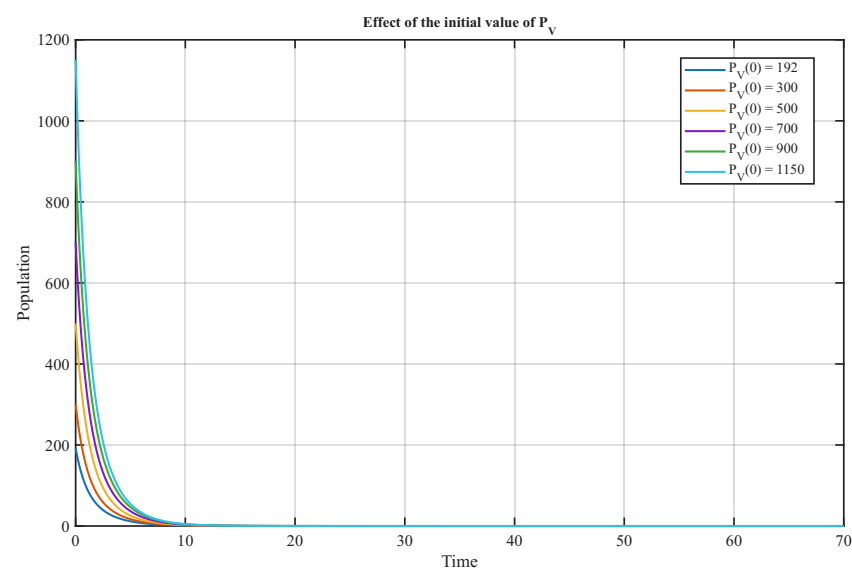


Figure 14. Invasion-stage pneumococcal population $P_V(t)$ for different initial values $P_V(0)$.

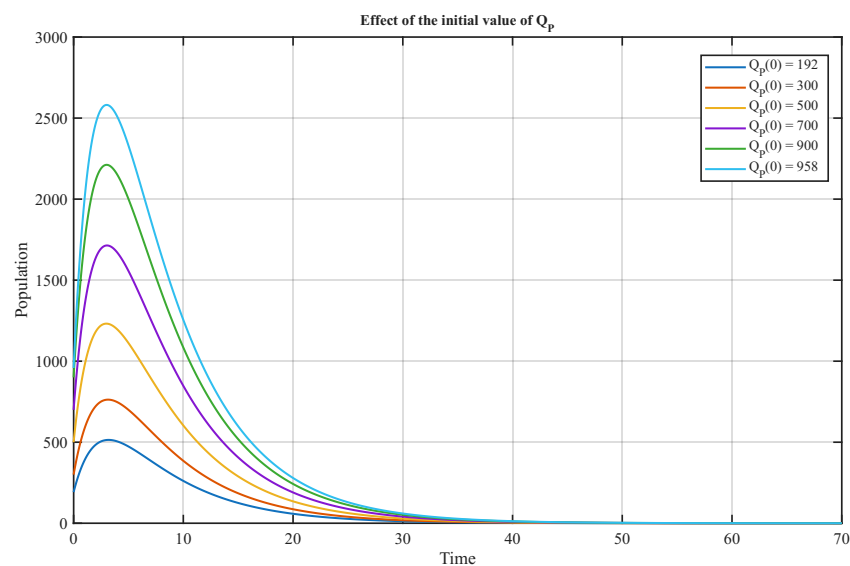


Figure 15. Quarantined pneumococcal population $Q_p(t)$ for different initial values $Q_p(0)$.

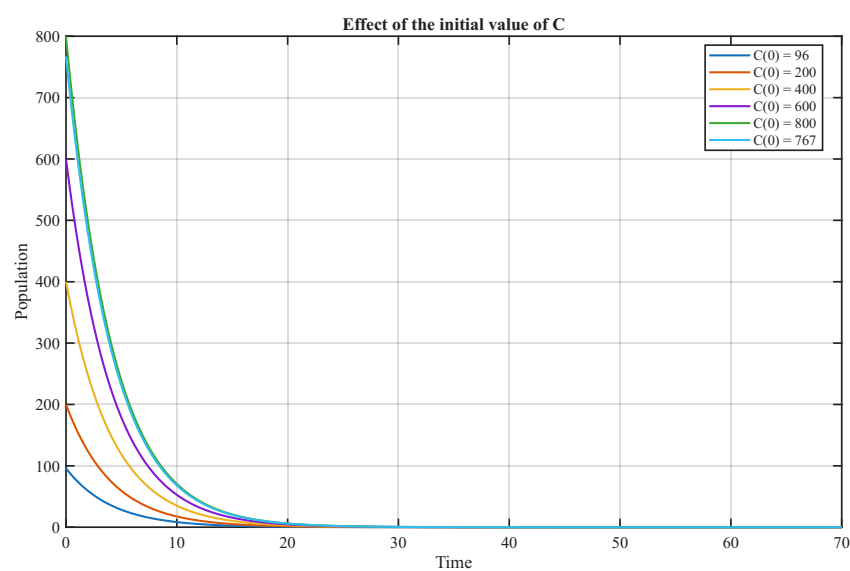


Figure 16. Co-infected population $C(t)$ for different initial values $C(0)$.

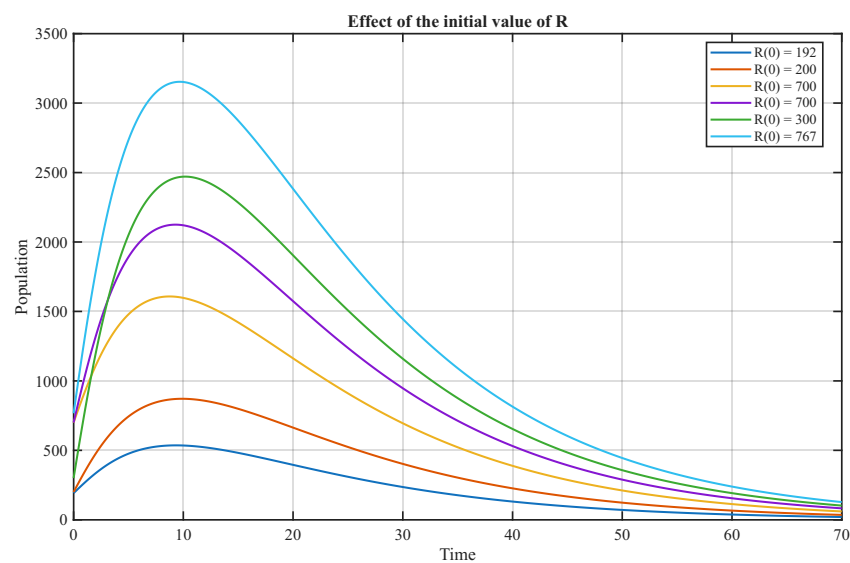


Figure 17. Recovered population $R(t)$ for different initial values $R(0)$.

6.7. Numerical Accuracy and Verification

To assess the accuracy of ode45, the solution in the endemic regime was compared against a high-accuracy reference obtained with $\text{RelTol} = 10^{-12}$ and $\text{AbsTol} = 10^{-14}$. Table 6 reports the maximum and mean absolute errors, the maximum relative error, and the CPU time as the tolerance is tightened; Figure 18 shows the corresponding convergence. The maximum absolute error decreases from 1.05 at $\text{RelTol} = 10^{-3}$ to 2.8×10^{-8} at $\text{RelTol} = 10^{-10}$, confirming the expected high-order convergence, while the CPU time remains at the millisecond scale throughout. The working tolerance $\text{RelTol} = 10^{-8}$ yields a maximum absolute error below 3×10^{-6} individuals, which is more than sufficient for the present study.

Table 6. Absolute and relative errors of ode45 at decreasing tolerances, relative to a reference solution computed with $\text{RelTol} = 10^{-12}$, $\text{AbsTol} = 10^{-14}$.

RelTol	Max abs. Error	Mean abs. Error	Max rel. Error	CPU (s)
10^{-3}	1.055	3.48×10^{-2}	1.15×10^{-3}	0.006
10^{-4}	7.12×10^{-2}	2.58×10^{-3}	7.56×10^{-5}	0.003
10^{-6}	3.23×10^{-4}	1.24×10^{-5}	5.30×10^{-7}	0.005
10^{-8}	2.88×10^{-6}	9.81×10^{-8}	4.92×10^{-9}	0.011
10^{-10}	2.78×10^{-8}	9.01×10^{-10}	4.74×10^{-11}	0.025

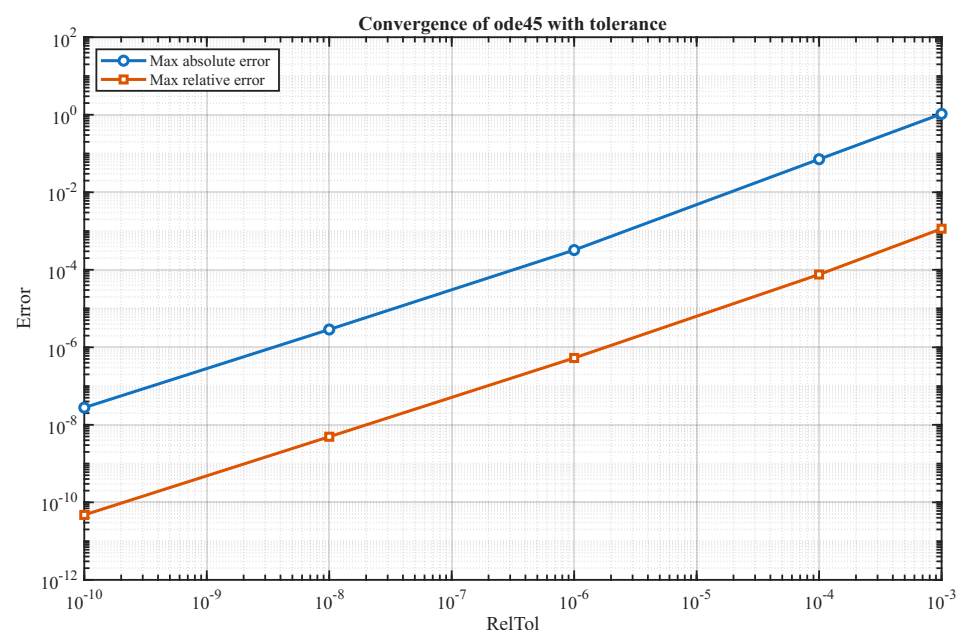


Figure 18. Convergence of ode45: maximum absolute and relative errors against the solver tolerance, on logarithmic axes.

As additional verification, all state variables remained non-negative throughout every simulation, and the total population satisfied the balance law of Section 2 to within an integrated error of 2.7×10^{-4} , confirming that the model was transcribed and integrated correctly.

7. Conclusions

We proposed and analysed a deterministic model of influenza–*Streptococcus pneumoniae* co-infection, formulated as eleven nonlinear ordinary differential equations incorporating imperfect vaccination, quarantine, and a three-stage pneumococcal progression. We established the positivity and boundedness of solutions, derived the reproduction

numbers R_0^I and R_0^P and the composite threshold $R_0 = \max\{R_0^I, R_0^P\}$, and showed that each single infection undergoes a forward bifurcation: the disease-free equilibrium is locally asymptotically stable for a reproduction number below unity, and a unique endemic equilibrium—globally asymptotically stable in the pneumococcal sub-model, by a Goh–Volterra Lyapunov function—emerges above it, with no sub-threshold endemic state. For the full model the disease-free equilibrium is locally asymptotically stable if and only if $R_0 < 1$, with global attraction supported numerically.

The central feature of the coupled dynamics is competitive exclusion: when both single-infection thresholds exceed unity, the two infections compete for the shared susceptible pool and one is driven to extinction together with the co-infected class, since the model contains no mechanism favouring co-infection. We showed that the outcome is governed not by the disease-free reproduction numbers but by the invasion numbers $\mathcal{R}_{P|I}$ and $\mathcal{R}_{I|P}$ evaluated at the single-infection boundary equilibria, so that even equal disease-free thresholds do not yield coexistence. Numerically, increasing the quarantine rate lowered the influenza reproduction number below unity and reduced the cumulative burden by more than two orders of magnitude, while the exclusion analysis warns that suppressing the dominant infection may release the other. Sustaining endemic co-infection would require an explicit synergy mechanism, such as influenza-enhanced pneumococcal susceptibility, which—together with sensitivity analysis, optimal control, and data calibration—is a natural direction for future work.

Author Contributions: Conceptualization, D.P. and K.T.; Data Curation, I.C.; Formal Analysis, D.P., K.T. and S.P.; Investigation, D.P., K.T. and I.C.; Methodology, D.P., K.T., S.P. and I.C.; Software, D.P., K.T., S.P. and I.C.; Validation, K.T. and I.C.; Visualization, D.P., S.P. and I.C.; Writing—Original Draft, D.P., K.T., S.P. and I.C.; Writing—Review & Editing, D.P., K.T. and I.C. All authors have read and agreed to the published version of the manuscript.

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