



Mathematical model for an infectious disease with quarantine

F. Iranzad, H.R. Marasi, H. Kheiri*, and A. Jabbari

Abstract

This article presents a novel compartmental mathematical model for an infectious disease that uniquely distinguishes susceptible individuals into quarantined and nonquarantined groups, while explicitly capturing dynamic transitions between these compartments. Unlike traditional models, ours integrates the combined effects of quarantine effectiveness and population mobility on disease transmission dynamics. We rigorously define the biologically feasible region and prove its positive invariance, ensuring model consistency. Using the next-generation matrix method, we derive a precise expression for the basic reproduction number ($\mathcal{R}_0$). Our

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analytical contributions include establishing both local and global stability of the disease-free equilibrium for $\mathcal{R}_0 < 1$, and, notably, developing a novel Lyapunov function to prove the global asymptotic stability of the endemic equilibrium when $\mathcal{R}_0 > 1$. Through comprehensive numerical simulations, we validate the theoretical results and provide new insights into the critical role of quarantine measures in epidemic control. These findings offer valuable guidance for designing more effective public health intervention strategies.

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1 Introduction

Historically, infectious diseases have been a major source of morbidity and mortality among human populations, often devastating entire communities. Beyond individual health implications, these diseases significantly disrupt community health systems and severely impact economic and social infrastructures. From the bubonic plague to recent pandemics such as COVID-19, outbreaks have profoundly shaped human history and public health responses.

Over the last decade, with the rise of global epidemics, quarantine has gained renewed importance as an effective nonpharmaceutical intervention for infectious disease control. Quarantine aims to isolate individuals who may have been exposed to the pathogen, thereby reducing transmission potential [1]. This intervention not only curtails the spread of disease but also alleviates strain on healthcare facilities by modulating the flow of infectious and susceptible individuals.

Mathematical modeling serves as a fundamental tool to understand infectious disease transmission dynamics and evaluate control strategies [6]. While prior models have addressed interventions such as treatment and contact tracing [8, 5], many do not explicitly differentiate between quarantined and nonquarantined susceptibles or incorporate the role of hospitalization distinctly within the population compartments.

In this paper, we propose a novel compartmental model that subdivides the total population into six epidemiological classes: nonquarantined susceptible, quarantined susceptible, latent, infectious, hospitalized, and recovered individuals. Our model explicitly incorporates the dynamics of quarantine not only for susceptibles but also distinguishes hospitalized individuals as a separate compartment, capturing their unique role in disease progression and control. The force of infection is modeled by effective contacts with both infectious and hospitalized individuals, reflecting realistic transmission pathways.

The originality of this work lies in the detailed representation of quarantine processes, including transfer rates between susceptible and quarantined classes, and an explicit hospitalized class, which is often aggregated or omitted in existing models. This framework allows a more nuanced analysis of quarantine's impact on reducing infection spread and healthcare burden. Rigorous qualitative analyses, including stability and threshold dynamics, are conducted to provide insights into the disease behavior and control under quarantine measures.

By integrating these aspects, our model enhances the understanding of how quarantine and hospitalization jointly influence infectious disease dynamics. The insights gained could inform public health strategies tailored to optimize quarantine implementation and hospital resource management in the context of controlling infectious diseases.

2 Model formulation

We denote the total population at time t by $N(t)$, which is subdivided into six disjoint classes of nonquarantined susceptible $S(t)$, quarantined susceptible $S_q(t)$, latent $L(t)$, infectious $I(t)$, hospitalized $H(t)$, and recovered $R(t)$ individuals. So, it can be written

$$N(t) = S(t) + S_q(t) + L(t) + I(t) + H(t) + R(t).$$

The population of susceptible individuals grows through the recruitment of the new population (at the rate of Λ). Also, quarantined susceptible may leave their class with rate of ψ and move into susceptible individuals. It is reduced by infected following efficient contacts with infected individuals, at rate of λ , given by

$$\lambda = \frac{\gamma(\eta_I I + \eta_H H)}{N(t)}, \quad (1)$$

where γ is effective contact rate, and η_I and η_H are modifier parameters for infectious and hospitalized. This individual is reduced by transferring from susceptible individuals to quarantined susceptible individuals with rate of γ , and natural death (with the rate of θ). Then, the dynamic of susceptible is obtained as follows:

$$\frac{dS}{dt} = \Lambda - \lambda S - (\theta + \gamma)S + \psi S_q.$$

In a similar way, other dynamic equations can be obtained. We have

$$\frac{dS}{dt} = \Lambda - \lambda S - (\theta + \gamma)S + \psi S_q,$$

Table 1: Description of parameters in the model (2)

Variable	Description
S	Population of nonquarantined susceptible individuals
S_q	Population of quarantined susceptible individuals
L	Population of latent individuals
I	Population of infected symptomatic individuals
H	Population of hospitalized individuals
R	Population of effectively-treated individuals
Parameter	Description
Λ	Recruitment rate
η_I	Modification parameter for infectious
η_H	Modification parameter for hospitalized
γ	Effective contact rate
θ	Per-capita natural mortality rate
δ_I	Per-capita death rate due to infectious
δ_H	Per-capita death rate due to hospitalized
θ	Per-capita rate of infected people hospitalized
ψ	Transfer rate of quarantined susceptible to nonquarantined susceptible
γ	Transfer rate of nonquarantined susceptible to quarantined susceptible
r	Fixed number between 0 and 1
σ	Endogenous re-activation rate of latent individuals
ψ_I	Effective treatment rate of infectious individuals
ψ_H	Effective treatment rate of hospitalized individuals

$$\begin{aligned}
\frac{dS_q}{dt} &= -r\lambda S_q + \gamma S - (\psi + \theta)S_q, \\
\frac{dL}{dt} &= \lambda S + r\lambda S_q - (\theta + \sigma)L, \\
\frac{dI}{dt} &= \sigma L - (\theta + \psi_I + \theta + \delta_I)I, \\
\frac{dH}{dt} &= \theta I - (\psi_H + \theta + \delta_H)H, \\
\frac{dR}{dt} &= \psi_I I + \psi_H H - \theta R.
\end{aligned} \tag{2}$$

The parameters used in the model are given in Table 1, and the graphical representation of the model is shown in Figure 1.

3 Basic properties of the model

We establish that system (2) is biologically well-posed, demonstrating that its solutions remain nonnegative for all time.

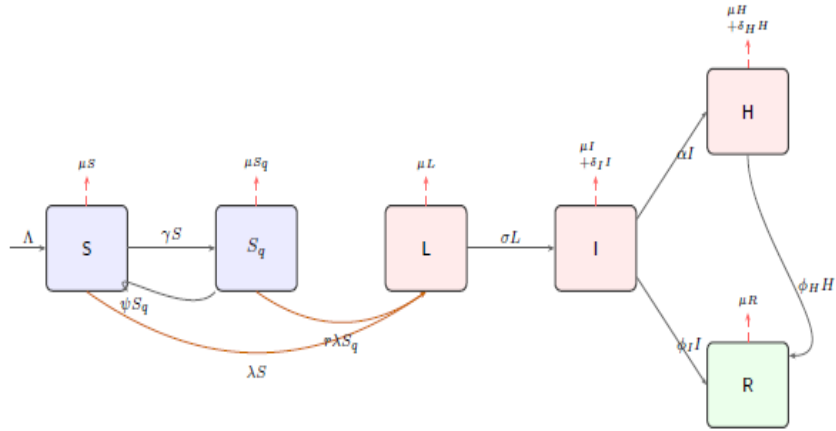


Figure 1: Susceptible (S, S_q), Latent (L), Infectious (I), Hospitalized (H), and Recovered (R). Transmission flows, orange arrows show infection, solid black arrows show transitions, and dashed red arrows show mortality.

Theorem 1. The domain

$$\Omega_+ = \{(S, S_q, L, I, H, R) \in \mathbb{R}_+^6 \mid S > 0, S_q, L, I, H, R \geq 0\}$$

remains positively invariant under the dynamics of system (2).

Proof. The results in [9] guarantee the existence and uniqueness of solutions to (2) on the interval $(0, \infty)$. Restricting the system to the set Ω_+ , it follows that

$$\begin{aligned} \frac{dS}{dt} \Big|_{S=0} &= \Lambda + \psi S_q \geq 0, \\ \frac{dS_q}{dt} \Big|_{S_q=0} &= \gamma S \geq 0, \\ \frac{dL}{dt} \Big|_{L=0} &= \lambda S + r \lambda S_q \geq 0, \\ \frac{dI}{dt} \Big|_{I=0} &= \sigma L \geq 0, \\ \frac{dH}{dt} \Big|_{H=0} &= \theta I \geq 0, \\ \frac{dR}{dt} \Big|_{R=0} &= \psi_I I + \psi_H H \geq 0. \end{aligned} \tag{3}$$

Now, if $(S(0), S_q(0), L(0), I(0), H(0), R(0)) \in \Omega_+$, by (3), then the solution (S, S_q, L, I, H, R) cannot leave the hyper-planes $S = 0$, $S_q = 0$, $L = 0$, $I = 0$, $H = 0$, or $R = 0$. The vector field is either tangent to each hyperplane or directed inward relative to Ω_+ . Consequently, all solutions remain confined within Ω_+ , proving its positive invariance. $\square$

Theorem 2. The set Ω_+ is positively invariant, and all trajectories with initial conditions in $\mathbb{R}_+^6$ eventually enter Ω_+ under the dynamics of (2).

Proof. Summing all the right-hand side expressions of model (2) yields

$$\begin{aligned}\frac{dN}{dt} &= \Lambda - \theta(S + S_q + L + I + H + R) - \delta_I I - \delta_H H \\ &\leq \Lambda - \theta(S + S_q + L + I + H + R) = \Lambda - \theta N,\end{aligned}$$

which can be written as

$$N(t) \leq \frac{\Lambda}{\theta}(1 - e^{-\theta t}) + N(0)e^{-\theta t}.$$

By choosing initial values from Ω_+ , it results that $N(t) \leq \frac{\Lambda}{\theta}$. Hence, the set Ω_+ is positively invariant. $\square$

3.1 Disease-free equilibrium

The disease-free equilibrium (DFE) of system (2) is obtained by setting all right-hand side terms to zero and solving for the state variables under the condition that all infection-related components are zero:

$$\varepsilon_0 = (S^0, S_q^0, L^0, I^0, H^0, R^0) = \left(\frac{\Lambda(\theta + \psi)}{\theta(\theta + \psi + \gamma)}, \frac{\Lambda\gamma}{\theta(\theta + \psi + \gamma)}, 0, 0, 0, 0 \right). \quad (4)$$

Applying the next-generation matrix method [3, 13], we derive the basic reproduction number $\mathcal{R}_0$ as the spectral radius of the matrix product $\mathcal{F}\mathcal{V}^{-1}$, which governs the local stability of the DFE ε_0 . Here, $\mathcal{F}$ represents the new infection terms and $\mathcal{V}$ the transition terms for system (2), defined as follows:

$$\mathcal{F} = \begin{bmatrix} 0 & \frac{\gamma\eta_I[(\theta+\psi)+r\gamma]}{(\theta+\psi+\gamma)} & \frac{\gamma\eta_H[(\theta+\psi)+r\gamma]}{(\theta+\psi+\gamma)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad (5)$$

$$\mathcal{V} = \begin{bmatrix} \theta + \sigma & 0 & 0 & 0 \\ -\sigma & \theta + \psi_I + \theta + \delta_I & 0 & 0 \\ 0 & -\theta & \psi_H + \theta + \delta_H & 0 \\ 0 & -\psi_I & -\psi_H & \theta \end{bmatrix}. \quad (6)$$

The basic reproduction number $\mathcal{R}_0$ for system (2) is given by

$$\mathcal{R}_0 = \rho(\mathcal{F}\mathcal{V}^{-1}) = \frac{\gamma\sigma(\theta + \psi + r\gamma)(\eta_H\theta + \eta_I k_3)}{k_1 k_2 k_3(\theta + \psi + \gamma)},$$

where

$$\begin{aligned} k_1 &= \sigma + \theta, \\ k_2 &= \theta + \psi_I + \theta + \delta_I, \\ k_3 &= \psi_H + \theta + \delta_H. \end{aligned} \quad (7)$$

For more details see [3].

Theorem 3. The DFE of system (2) is locally asymptotically stable (L.A.S) when $\mathcal{R}_0 < 1$, and unstable when $\mathcal{R}_0 > 1$.

Proof. The Jacobian matrix of system (2) evaluated at the DFE point ε_0 is

$$\mathcal{J}_{\varepsilon_0} = \begin{bmatrix} -(\theta + \gamma) & \psi & 0 & 0 & 0 & 0 \\ \gamma & -(\theta + \psi) & 0 & 0 & 0 & 0 \\ -\frac{\gamma(\theta + \psi)}{\theta + \psi + \gamma} - \frac{\gamma r \gamma}{\theta + \psi + \gamma} & -(\theta + \sigma) & \frac{\gamma \theta \eta_I [(\theta + \psi) + r \gamma]}{\theta(\theta + \psi + \gamma)} & \frac{\gamma \theta \eta_H [(\theta + \psi) + r \gamma]}{\theta(\theta + \psi + \gamma)} & 0 & 0 \\ 0 & 0 & \sigma & -(\theta + \psi_I + \theta + \delta_I) & 0 & 0 \\ 0 & 0 & 0 & \theta & -(\theta + \psi_H + \delta_H) & 0 \\ 0 & 0 & 0 & \psi_I & \psi_H & -\theta \end{bmatrix}.$$

This matrix can be decomposed as

$$\mathcal{J}_{\varepsilon_0} = \begin{bmatrix} A & 0 \\ B & \mathcal{F} - \mathcal{V} \end{bmatrix},$$

where

$$A = \begin{bmatrix} -(\theta + \gamma) & \psi \\ \gamma & -(\theta + \psi) \end{bmatrix}, \quad B = \begin{bmatrix} -\frac{\gamma(\theta + \psi)}{\theta + \psi + \gamma} - \frac{\gamma r \gamma}{\theta + \psi + \gamma} & \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{bmatrix}.$$

The DFE is L.S.A if and only if all eigenvalues of $\mathcal{J}_{\varepsilon_0}$ have negative real parts. Since $\mathcal{J}_{\varepsilon_0}$ is block lower-triangular, its eigenvalues consist of those of A and those of $\mathcal{F} - \mathcal{V}$. The eigenvalues of A are explicitly

$$-\theta \quad \text{and} \quad -(\theta + \psi + \gamma),$$

both of which are negative. Hence, stability depends on the spectral properties of $\mathcal{F} - \mathcal{V}$. The conditions reduce to

$$\lambda(\mathcal{J}_{\varepsilon_0}) < 0 \iff \lambda(\mathcal{F} - \mathcal{V}) < 0 \iff \rho(\mathcal{F}\mathcal{V}^{-1}) < 1,$$

where $\rho(\cdot)$ denotes the spectral radius and the invertibility of $\mathcal{V}$ is assumed.

Since the basic reproduction number $\mathcal{R}_0$ is defined by $\mathcal{R}_0 = \rho(\mathcal{F}\mathcal{V}^{-1})$, we conclude the threshold behavior:

- If $\mathcal{R}_0 < 1$, then the DFE is L.A.S.
- If $\mathcal{R}_0 > 1$, then the DFE is unstable.

□

To analyze the global stability of the DFE, we rigorously apply [12, Theorem 2.1], which provides sufficient conditions for the global asymptotic stability of an equilibrium in epidemiological models.

We first rewrite system (2) in the form

$$x' = (\mathcal{F} - \mathcal{V})x,$$

where $x = [L, I, H, R]^T$ denotes the infected state variables, $\mathcal{F}$ represents the matrix of new infection terms, and $\mathcal{V}$ aggregates transition and removal terms as defined in (5) and (6), respectively. We verify the required conditions for the method:

1. By the model construction and equations (5) and (6), the matrix $\mathcal{F}$ satisfies $\mathcal{F} \geq 0$ (componentwise nonnegative), and the matrix $\mathcal{V}$ is a nonsingular M -matrix with inverse $\mathcal{V}^{-1} \geq 0$, explicitly given by

$$\mathcal{V}^{-1} = \begin{bmatrix} \frac{1}{k_1} & 0 & 0 & 0 \\ \frac{\sigma}{k_1 k_2} & \frac{1}{k_2} & 0 & 0 \\ \frac{\theta \sigma}{k_1 k_2 k_3} & \frac{\theta}{k_2 k_3} & \frac{1}{k_3} & 0 \\ \frac{\sigma(\theta \psi_H + \delta_H \psi_I + \theta \psi_I + \psi_H \psi_I)}{\theta k_1 k_2 k_3} & \frac{\theta \psi_H + \delta_H \psi_I + \theta \psi_I + \psi_H \psi_I}{\theta k_2 k_3} & \frac{\psi_H}{\theta k_3} & \frac{1}{\theta} \end{bmatrix}.$$

2. The next-generation matrix $\mathcal{K} = \mathcal{V}^{-1}\mathcal{F}$ has a nonnegative left eigenvector $\omega^T = [0, \eta_I, \eta_H, 0] \geq 0$ corresponding to the spectral radius $\mathcal{R}_0$, that is,

$$\omega^T \mathcal{K} = \mathcal{R}_0 \omega^T.$$

3. Define the Lyapunov function

$$Q(x) = \omega^T \mathcal{V}^{-1}x,$$

which is nonnegative for $x \geq 0$.

4. Differentiating Q along solutions of the system gives

$$\begin{aligned} Q' &= \omega^T \mathcal{V}^{-1}x' = \omega^T \mathcal{V}^{-1}((\mathcal{F} - \mathcal{V})x) \\ &= \omega^T (\mathcal{K} - I)x = (\mathcal{R}_0 - 1)\omega^T x, \end{aligned}$$

where we used the eigenvector relation $\omega^T \mathcal{K} = \mathcal{R}_0 \omega^T$.

Since $\omega^T x \geq 0$, it follows that, if $\mathcal{R}_0 < 1$, then $Q' \leq 0$, with equality only at $x = 0$, that is, the DFE. By LaSalle's Invariance Principle, the largest invariant set where $Q' = 0$ is the singleton $\{0\}$.

Therefore, all trajectories of the infectious subsystem converge to the DFE, that means the DFE is globally asymptotically stable when $\mathcal{R}_0 < 1$.

Theorem 4. System (2) at the DFE is globally asymptotically stable if and only if $\mathcal{R}_0 < 1$.

3.2 Endemic equilibrium point

The endemic equilibrium point (EEP), denoted by

$$\varepsilon^* = (S^*, S_q^*, L^*, I^*, H^*, R^*),$$

of system (2) is found by solving the steady-state conditions

$$\dot{S} = \dot{S}_q = \dot{L} = \dot{I} = \dot{H} = \dot{R} = 0.$$

The equilibrium values are given by

$$\begin{aligned}
 S^* &= \frac{\Lambda(r\lambda^* + \theta + \psi)}{(\theta + r\lambda^* + \psi)(\theta + \lambda^* + \gamma) - \psi\gamma}, \\
 S_q^* &= \frac{\Lambda\gamma}{(\theta + r\lambda^* + \psi)(\theta + \lambda^* + \gamma) - \psi\gamma}, \\
 L^* &= \frac{\Lambda\lambda^*[\Lambda(r(\lambda^* + \gamma) + \theta + \psi)]}{k_1[(\theta + r\lambda^* + \psi)(\theta + \lambda^* + \gamma) - \psi\gamma]}, \\
 I^* &= \frac{\sigma\Lambda\lambda^*[\Lambda(r(\lambda^* + \gamma) + \theta + \psi)]}{k_1k_2[(\theta + r\lambda^* + \psi)(\theta + \lambda^* + \gamma) - \psi\gamma]}, \\
 H^* &= \frac{\theta\sigma\Lambda\lambda^*[\Lambda(r(\lambda^* + \gamma) + \theta + \psi)]}{k_1k_2k_3[(\theta + r\lambda^* + \psi)(\theta + \lambda^* + \gamma) - \psi\gamma]}, \\
 R^* &= \frac{\sigma\Lambda\lambda^*[\Lambda(r(\lambda^* + \gamma) + \theta + \psi)][\psi_Ik_3 + \psi_H\theta]}{\theta k_1k_2k_3[(\theta + r\lambda^* + \psi)(\theta + \lambda^* + \gamma) - \psi\gamma]}.
 \end{aligned} \tag{8}$$

The endemic steady-state value λ^* , defined in (1), satisfies the quadratic equation

$$a_2(\lambda^*)^2 + a_1\lambda^* + a_0 = 0,$$

with coefficients

$$\begin{aligned}
 a_2 &= k_1k_2k_3 > 0, \\
 a_1 &= k_1k_2k_3[r(\theta + \gamma) + (\theta + \psi)] - \gamma\theta r[k_3(k_2 + \eta_I\sigma) + \eta_H\theta\sigma], \\
 a_0 &= \theta(1 - \mathcal{R}_0)(\theta + \psi + \gamma)k_1k_2k_3,
 \end{aligned}$$

where k_1, k_2, k_3 are defined in (7). Using the relation $\lambda_1\lambda_2 = \frac{a_0}{a_2}$, the discriminant analysis of this quadratic shows that a positive EEP exists if and only if $\mathcal{R}_0 > 1$.

Theorem 5. The EEP of system (2), given in (8), is globally asymptotically stable in the interior of the feasible region when $\mathcal{R}_0 > 1$.

Proof. To prove the globally asymptotically stable of EEP, when $\mathcal{R}_0 > 1$, we construct a Lyapunov function V

$$\begin{aligned}
 V &= \left(S - S^* - S^* \ln \frac{S}{S^*} \right) + \left(S_q - S_q^* - S_q^* \ln \frac{S_q}{S_q^*} \right) \\
 &\quad + \left(L - L^* - L^* \ln \frac{L}{L^*} \right) + \frac{k_1}{\sigma} \left(I - I^* - I^* \ln \frac{I}{I^*} \right) \\
 &\quad + \frac{k_1}{\sigma} \cdot \frac{\eta_H\theta}{\eta_{\text{eff}}k_3} \left(H - H^* - H^* \ln \frac{H}{H^*} \right),
 \end{aligned}$$

where $\eta_{eff} = \eta_I + \eta_H \frac{\theta}{k_3}$. By introducing $g(u) = u - 1 - \ln u$, it is clear that $g(u) \geq 0$ for $u > 0$, with equality only at $g(1) = 0$. Since $g(u)$ is convex and has a global minimum at $u = 1$. The Lyapunov function V can be expressed in terms of the function $g(u)$ as follows:

$$V = g\left(\frac{S}{S^*}\right) S^* + g\left(\frac{S_q}{S_q^*}\right) S_q^* + g\left(\frac{L}{L^*}\right) L^* \\ + \frac{k_1}{\sigma} g\left(\frac{I}{I^*}\right) I^* + \frac{k_1 \eta_H \theta}{\sigma \eta_{eff} k_3} g\left(\frac{H}{H^*}\right) H^*.$$

It is clear that $V(X) \geq 0$ for $X \geq 0$, with equality only at $V(\varepsilon^*) = 0$.

Now, we analyze the time derivative of the composite Lyapunov function $V = V_1 + V_2 + V_3 + V_4 + V_5$ to establish global stability of the EEP. Each component corresponds to a specific compartment in our epidemic model. For the susceptible compartment, we examine

$$\frac{dV_1}{dt} = \left(1 - \frac{S^*}{S}\right) \frac{dS}{dt}.$$

Substituting the $\frac{dS}{dt}$ from (2), we have

$$\frac{dV_1}{dt} = \left(1 - \frac{S^*}{S}\right) [\Lambda - \lambda S - (\theta + \gamma)S + \psi S_q].$$

Applying the equilibrium condition $\Lambda - \lambda^* S^* - (\theta + \gamma)S^* + \psi S_q^* = 0$, we can get

$$\frac{dV_1}{dt} = \left(1 - \frac{S^*}{S}\right) [\lambda^* S^* - \lambda S + (\theta + \gamma)(S^* - S) + \psi(S_q - S_q^*)].$$

Expanding this expression yields

$$\frac{dV_1}{dt} = (\lambda^* S^* - \lambda S) + \psi(S_q - S_q^*) \\ - \frac{S^*}{S} [\lambda^* S^* - \lambda S + \psi(S_q - S_q^*)] - (\theta + \gamma) \frac{(S - S^*)^2}{S}.$$

Similarly, for the other components, it can be calculated

$$\frac{dV_2}{dt} = -(\psi + \theta) \frac{(S_q - S_q^*)^2}{S_q} + \gamma(S - S^*) \left(1 - \frac{S_q^*}{S_q}\right) \\ + r\lambda^*(S_q^* - S_q) \left(1 - \frac{S_q^*}{S_q}\right) + r(\lambda^* - \lambda)S_q \left(1 - \frac{S_q^*}{S_q}\right), \\ \frac{dV_3}{dt} = (\lambda S + r\lambda S_q) - k_1(L - L^*) \\ - \frac{L^*}{L} [\lambda S + r\lambda S_q - (\lambda^* S^* + r\lambda^* S_q^*) - k_1(L - L^*)],$$

Table 2: Key parameter values for model (2).

Parameter	Value	Source
Λ	11400	[11]
η_I	0.5	[11]
η_H	0.15	[11]
γ	Changes	Assumed
θ	0.00021	[4]
δ_I	0.00999	[2]
δ_H	0.0007	[2]
θ	0.0267	[2]
ψ	0.4947	[2]
γ	0.2422	[2]
r	0.37	Assumed
σ	0.25	[10]
ψ_I	0.27	[10]
ψ_H	0.5	[10]

$$\begin{aligned}\frac{dV_4}{dt} &= k_1 \left(1 - \frac{I^*}{I}\right) (L - L^*) - \frac{k_1 k_2}{\sigma} \frac{(I - I^*)^2}{I}, \\ \frac{dV_5}{dt} &= \frac{k_1 \eta_H \theta}{\sigma \eta_{\text{eff}} k_3} \left(1 - \frac{H^*}{H}\right) [\theta(I - I^*) - k_3(H - H^*)].\end{aligned}$$

After algebraic calculations and systematic cancellation of all cross-interaction terms, the complete time derivative becomes

$$\begin{aligned}\frac{dV}{dt} &= -(\theta + \gamma) \frac{(S - S^*)^2}{S} - (\psi + \theta) \frac{(S_q - S_q^*)^2}{S_q} - k_1 \frac{(L - L^*)^2}{L} \\ &\quad - \frac{k_1 k_2}{\sigma} \frac{(I - I^*)^2}{I} - \frac{k_1 \eta_H \theta}{\sigma \eta_{\text{eff}}} \frac{(H - H^*)^2}{H}.\end{aligned}\tag{9}$$

Therefore $\frac{dV}{dt} \leq 0$ for all trajectories, with equality if and only if

$$(S, S_q, L, I, H) = (S^*, S_q^*, L^*, I^*, H^*).\tag{10}$$

By using the LaSalle Theorem [7], every bounded trajectory approaches the invariant set Ω_+ to EEP when $\mathcal{R}_0 > 1$. $\square$

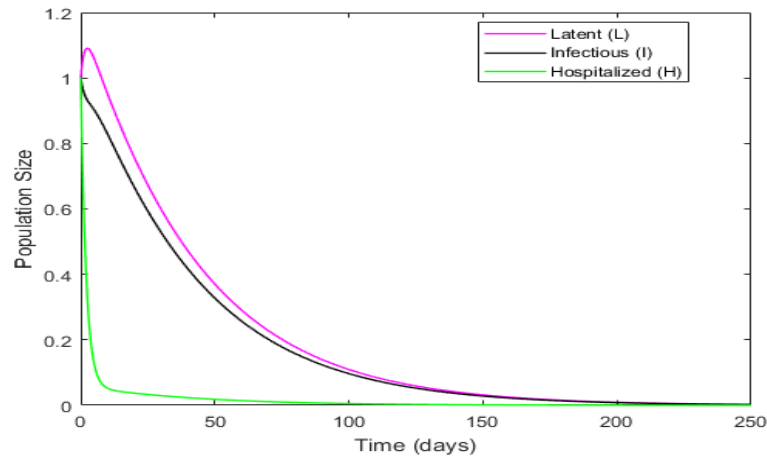


Figure 2: The population of latent, infectious and hospitalized individuals for $\mathcal{R}_0 < 1$.

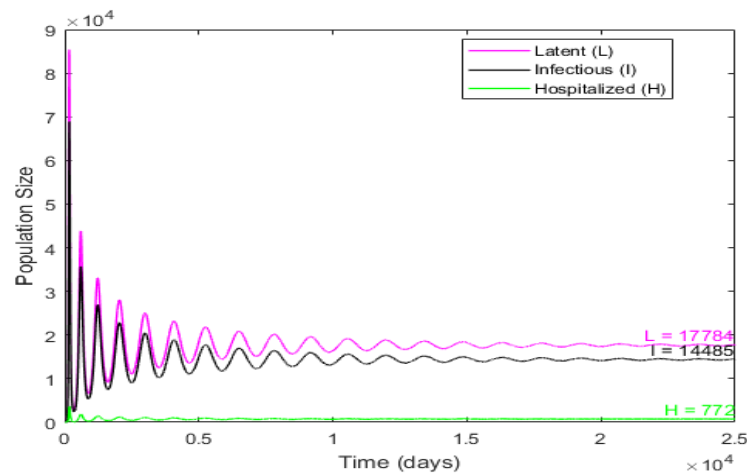


Figure 3: The population of latent, infectious and hospitalized individuals for $\mathcal{R}_0 > 1$.

4 Numerical simulations

The numerical simulations for system (2) are performed in MATLAB using the ODE45 solver. For Figure 1, the time interval $[0, 250]$ is discretized into $N = 1000$ uniform steps to ensure adequate resolution of the system dynamics. For Figure 2, the time interval $[0, 2.5 \times 10^4]$ is discretized into $N = 10^6$ uniform steps to achieve sufficient resolution of the system dynamics.

Using the parameter values listed in Table 2, we computed the basic reproduction number $\mathcal{R}_0$ for two transmission rates: $\gamma = 0.63$ and $\gamma = 1.23$. The corresponding reproduction numbers

were found to be $\mathcal{R}_0 = 0.8262$ and $\mathcal{R}_0 = 1.6131$, respectively. These values allow us to analyze the system's behavior under different epidemiological scenarios.

DFE Stability ($\mathcal{R}_0 < 1$)

For $\gamma = 0.63$ ($\mathcal{R}_0 = 0.8262 < 1$), the system exhibits convergence to the DFE. As illustrated in Figure 2, the population of latent individuals, infectious individuals, and hospitalized individuals consistently decline over time, eventually approaching zero. This numerical observation confirms the theoretical stability of the DFE when $\mathcal{R}_0 < 1$, indicating that the infection cannot sustain itself and will eventually die out under these conditions.

EEP Stability ($\mathcal{R}_0 > 1$)

Conversely, for $\gamma = 1.23$ ($\mathcal{R}_0 = 1.6131 > 1$), the system transitions to an EEP. Figure 3 demonstrates that the subpopulations of latent, infectious, and hospitalized individuals stabilize at nonzero equilibrium values over time. This persistent behavior suggests that the disease remains endemic in the population, with the EEP exhibiting asymptotic stability. The convergence of trajectories to fixed values further supports the existence of a stable endemic state when $\mathcal{R}_0 > 1$.

Theoretical and numerical consistency

The numerical simulations align precisely with our analytical stability analysis. The clear distinction in system dynamics decaying populations for $\mathcal{R}_0 < 1$ versus sustained endemic levels for $\mathcal{R}_0 > 1$ validates our theoretical predictions.

These findings have important implications for public health strategies. When $\mathcal{R}_0 < 1$, control measures need only to maintain conditions below this threshold to ensure disease eradication. However, if $\mathcal{R}_0 > 1$, then long-term management strategies must account for the stable endemic state, emphasizing the need for sustained interventions such as vaccination, improved treatment rates, or reduced transmission.

5 Conclusion

In this study, we developed and analyzed an infectious disease model that incorporates both quarantine and nonquarantine groups. Our analysis shows that when the basic reproduction number is below one, the DFE is globally stable, emphasizing the critical role of targeted public health measures in controlling outbreaks. Moreover, by utilizing a Lyapunov function approach, we demonstrated the global asymptotic stability of the endemic equilibrium, providing insight into the complex behavior of sustained infections. These theoretical results are further validated by numerical simulations, reinforcing the robustness of our findings and their potential implications for disease control strategies.

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Conflicts of Interest

The authors declare no conflicts of interest.

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