Journal of Mathematical Modeling
Vol. 14, No. 2, 2026, pp. 741-760. Research Article

JMM

Dynamical analysis of novel minimal SEIR model incorporating asymptomatic transmission

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Abstract. We propose a modified SEIR model that includes asymptomatic transmission directly in the infection term, avoiding the need for a separate asymptomatic compartment while keeping the model realistic. The basic reproduction number ($\mathscr{R}_0$) is calculated to measure the potential for disease spread. Local stability analysis shows that the disease-free equilibrium is stable when $\mathscr{R}_0 < 1$ and unstable when $\mathscr{R}_0 > 1$, while the endemic equilibrium is locally stable in the latter case. A forward bifurcation at $\mathscr{R}_0 = 1$ is identified, indicating a smooth transition from the disease-free equilibrium to a unique endemic equilibrium without coexistence of the two equilibria. Global stability results show that the disease-free state is globally asymptotically stable for $\mathscr{R}_0 \leq 1$, and the endemic state is globally asymptotically stable for $\mathscr{R}_0 > 1$. Simulations using early COVID-19 data support these findings, showing that higher asymptomatic transmission prolongs outbreaks, increases peaks, and delays elimination. Evaluation of control strategies reveals that isolation is more effective than testing alone, and their combination produces the greatest overall reduction in disease spread under appropriate assumptions.

Keywords: Modified *SEIR* model, asymptomatic infection, stability analysis, forward bifurcation, COVID-19 simulation
AMS Subject Classification 2010: 37N25, 92-10, 92D30

1 Introduction

Mathematical modeling has played a crucial role in understanding the dynamics of infectious diseases. Over the years, deterministic models have provided significant insights into disease spread, control strategies, and public health interventions. One of the earliest and most influential models is the *SIR* (Susceptible–Infectious–Recovered) model [14–16], which was later extended to the *SEIR* (Susceptible–Exposed–

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Received: 25 August 2025/ Revised: 10 November 2025/ Accepted: 17 December 2025
DOI: 10.22124/jmm.2025.31474.2832

Infectious–Recovered) framework to account for the latent period of infection—when individuals are infected but not yet infectious [8]. Depending on the nature of specific diseases, several modified versions of the *SEIR* model have been studied, including *SEAIR*, *SEIAQR*, SEI_WIHR, SEI_sI_aUR among others [1–3, 9, 17, 21, 22]. These extensions aim to capture additional epidemiological features such as asymptomatic transmission, quarantine, and vaccination.

The mechanisms of disease transmission become significantly more complex in the presence of asymptomatic infections. In some cases—particularly during the COVID-19 pandemic—asymptomatic transmission has accounted for up to 100% of symptomatic infections [32]. For most infectious diseases, symptoms serve as the primary indicators for detection and diagnosis. However, asymptomatic individuals—those who can transmit the infection without exhibiting symptoms—pose a serious challenge for disease control. Their presence undermines symptom-based screening and isolation strategies. To better capture asymptomatic transmission, various studies have modified the SEIR framework by introducing separate compartments for asymptomatic individuals, thereby allowing for a more detailed representation of transmission dynamics [1, 12, 13, 33]. However, such advanced models make the mathematical formulation considerably more complex, and the resulting analysis often depends on restrictive assumptions or numerical methods.

Respiratory viruses such as SARS-CoV-2 (COVID-19) and influenza can be transmitted by individuals who exhibit no symptoms, yet still pose a significant risk to public health. Asymptomatic carriers may unknowingly spread the virus to others, some of whom may subsequently develop symptoms or even severe illness. For instance, during the COVID-19 pandemic, a major outbreak in a call center in Seoul, South Korea, illustrated how an undetected or asymptomatic carrier likely initiated widespread transmission among coworkers, resulting in symptomatic cases in the days that followed [23]. Similarly, a study in South Africa found that individuals with asymptomatic influenza transmitted the virus to approximately 6% of their household contacts, some of whom became symptomatic [7]. Detailed case investigations have also documented instances where asymptomatic individuals directly infected others who later developed typical COVID-19 symptoms such as fever and cough [34]. These findings underscore the epidemiological importance of asymptomatic transmission and raise questions about how best to represent it in mathematical models. While adding separate compartments for asymptomatic individuals can improve realism, it often increases analytical and computational complexity. Moreover, the incubation period for asymptomatic cases is frequently ill-defined or even indeterminable, making it difficult to model their progression accurately. In practice, once an individual tests positive, they are typically classified within the infected compartment regardless of symptom presentation. Furthermore, studies have shown that asymptomatic individuals can transmit infections that result in either symptomatic or asymptomatic outcomes in susceptible hosts [7, 11, 23, 34] and a symptomatic individual can transmit infection to others, who may develop either symptomatic or asymptomatic disease based on infected persons immune response, age or health status [19]. This dual transmission dynamic suggests that a separate compartment for asymptomatic individuals may not be essential for capturing the core mechanisms of disease spread. These observations motivate the development of streamlined epidemiological models that retain the ability to reflect the critical role of asymptomatic transmission without introducing excessive structural complexity.

Motivated by these observations, this study proposes a novel modified SEIR model that incorporates asymptomatic transmission without introducing a separate compartment. Instead, the effect of asymptomatic carriers is embedded directly into the transmission process—from susceptible to infected individuals—thereby preserving model simplicity while maintaining biological realism. This approach

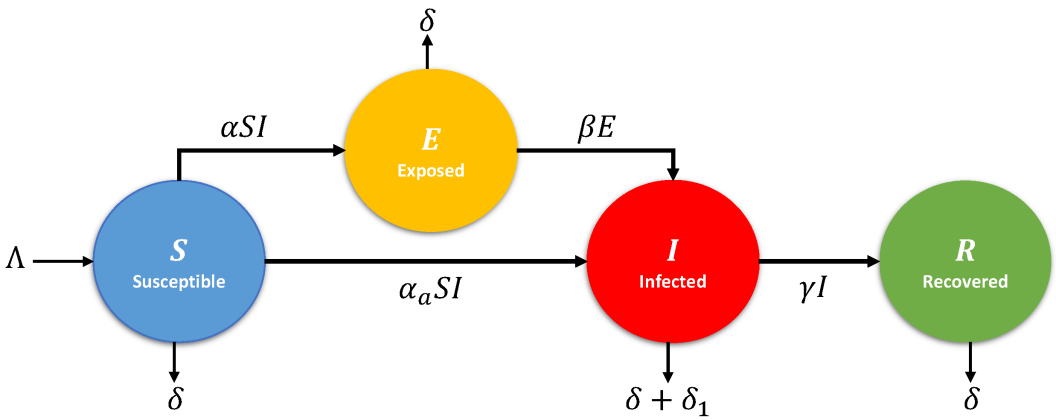

Figure 1: Schematic diagram of the model

balances analytical tractability with the need to accurately represent key aspects of disease spread. In this paper, we develop and analyse a proposed SEIR model. We investigate its mathematical properties, including well-posedness, the existence of equilibria, and the basic reproduction number ($\mathscr{R}_0$). We further examine the local and global stability of equilibria, explore potential bifurcation, and conduct numerical simulations with a focus on COVID-19 dynamics. Finally, we assess the effectiveness of public health interventions—such as rapid testing and isolation—during outbreaks involving asymptomatic transmission, demonstrating the practical utility of our approach.

2 Model formulation and positivity of solutions

We discuss a modified SEIR compartmental mathematical model to study the dynamics of epidemics of a disease with asymptomatic infection. Total population $N(t)$ at a time t is divided into four compartments: the susceptible $(S(t))$, exposed or latent $(E(t))$, infected $(I(t))$, and recovered $(R(t))$. The parameters of transmissions between compartments of the model are listed in Table 1 with their description. Along with these, we have taken following assumptions in formation of the model:

1. In real-world settings, it is rarely possible to identify all infected individuals. When modelling disease spread with both symptomatic and asymptomatic transmissions, we therefore use separate parameters for each, rather than assuming the asymptomatic value is simply the complement of the symptomatic one, as asymptomatic infections are generally detected less frequently (for example, rapid antigen tests detect 72–93 % of symptomatic infections but only 38–63% of asymptomatic infections [4, 24, 27]).

2. We model the spread of a single, stable pathogen strain capable of both symptomatic and asymptomatic transmission. We assume no mutation, antigenic variation, or strain replacement during

Table 1: Parameters table

Parameter	Description
Λ	Recruitment rate
δ	Natural death rate
α	Symptomatic infection rate
α_a	Asymptomatic infection rate
β	Exposed-to-infected progression rate
δ_1	Disease caused death rate
γ	Recovery rate

the course of the disease. Accordingly, the transmission rates and recovery rate remain constant, and the model does not include additional compartments for multiple strains.

Based on these assumptions, the schematic diagram given in Figure 1 and parameters described in Table 1, we have following system of equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha SI - \alpha_a SI - \delta S, \\
\frac{dE}{dt} &= \alpha SI - (\beta + \delta)E, \\
\frac{dI}{dt} &= \beta E + \alpha_a SI - (\gamma + \delta + \delta_1)I, \\
\frac{dR}{dt} &= \gamma I - \delta R,
\end{aligned} \tag{1}$$

where $S \geq 0, E \geq 0, I \geq 0,$ and $R \geq 0$.
Since there is no transmission from recovered (R) compartment to another compartment and we want to study dynamics of infection, the following reduced system is used:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha SI - \alpha_a SI - \delta S, \\
\frac{dE}{dt} &= \alpha SI - (\beta + \delta)E, \\
\frac{dI}{dt} &= \beta E + \alpha_a SI - (\gamma + \delta + \delta_1)I,
\end{aligned} \tag{2}$$

where $S \geq 0, E \geq 0,$ and $I \geq 0$.

2.1 Positivity of the solutions

Theorem 1. *Any solution of system* (1) *with non-negative initial values remains non-negative over the time.*

Proof. Let $(S(t) + E(t) + I(t) + R(t))$ be any solution of (1) with

$$S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, \tag{3}$$

and let $\tau > 0$.
Form equation of susceptible compartment of (1), we have

$$\frac{dS}{dt} \geq -(\alpha I + \alpha_a I + \delta)S.$$

This gives

$$\int_0^\tau \frac{1}{S} dS \geq \int_0^\tau -(\alpha I + \alpha_a I + \delta)dt,$$

then

$$S(\tau) \geq S(0)\exp\left(-\delta\tau - \int_0^\tau [\alpha I(t) + \alpha_a I(t)]dt\right) \geq 0. \tag{4}$$

Similarly, using equations of all other compartments of equation (1), we get

$$\begin{aligned} E(\tau) &\geq E(0)\exp(-(\beta+\delta)\tau) \geq 0, \\ I(\tau) &\geq I(0)\exp(-(\gamma+\delta+\delta_1)\tau) \geq 0, \\ R(\tau) &\geq R(0)\exp(-\delta\tau) \geq 0. \end{aligned} \tag{5}$$

Since τ is arbitrary, by combining (3), (4), and (5), we are done. □

Lemma 1. *The closed set*

$$\mathscr{D} = \{(S,E,R) : S \geq 0, E \geq 0, I \geq 0, S+E+I \leq \Lambda/\delta\}$$

is invariant. In particular, any solution of (2) with initial value in $\mathscr{D}$ remains in $\mathscr{D}$.

Proof. Let $N(t) = S(t) + E(t) + I(t)$, for all $t \geq 0$ with $N(0) = S(0) + E(0) + I(0) \in \mathscr{D}$.
By adding all equations of (2), we get

$$\frac{dN(t)}{dt} \leq \Lambda - \delta N(t).$$

By solving this linear inequality, we have

$$N(t) \leq \frac{\Lambda}{\delta} + \left(N(0) - \frac{\Lambda}{\delta}\right)e^{-\delta t}.$$

As $N(0) \in \mathscr{D}$, we can conclude that

$$N(t) \leq \frac{\Lambda}{\delta}, \text{ for all } t \geq 0.$$

Thus the set $\mathscr{D}$ is invariant. That is, any solution of (2) with initial value in $\mathscr{D}$ remains in $\mathscr{D}$. □

3 Basic reproduction number and existence of equilibria

3.1 Disease-free equilibrium

The disease-free equilibrium is a steady state solution of model (2) with no infectious individuals in population. Let $\mathscr{E}_0$ be denoted disease-free equilibrium, then

$$\mathscr{E}_0 = (S_0^*, E_0^*, I_0^*) = \left(\frac{\Lambda}{\delta}, 0, 0\right). \tag{6}$$

3.2 Basic reproduction number

We know that basic reproduction number is pivotal in disease dynamics. Now, we will find it using Next-Generation Matrix [10] method. For that let $\mathscr{F}$ denotes column vectors of new infections and $\mathscr{V}$ denotes the vector of other transitions involved in model (2). That is,

$$\mathscr{F} = \begin{pmatrix} 0 \\ \alpha SI, \\ \alpha_a SI \end{pmatrix}, \ \mathscr{V} = \begin{pmatrix} -\Lambda + \alpha SI + \alpha_a SI + \delta S \\ (\beta+\delta)E \\ (\gamma+\delta+\delta_1)I - \beta E \end{pmatrix}.$$

Let F and V denotes Jacobian matrix of $\mathscr{F}$ and $\mathscr{V}$, respectively, both obtained at $\mathscr{E}_0$, then

$$F = \mathscr{F}(\mathscr{E}_0) = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & \alpha S_0^* \\ 0 & 0 & \alpha_a S_0^* \end{pmatrix}, \ V = \mathscr{V}(\mathscr{E}_0) = \begin{pmatrix} \delta & 0 & (\alpha+\alpha_a)S_0^* \\ 0 & \beta+\delta & 0 \\ 0 & -\beta & \delta+\delta_1+\gamma \end{pmatrix}.$$

Thus, the next generation matrix

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 \\ 0 & \frac{\alpha\beta}{(\beta+\delta)(\gamma+\delta+\delta_1)} \times S_0^* & \frac{\alpha}{\gamma+\delta+\delta_1} \times S_0^* \\ 0 & \frac{\alpha_a\beta}{(\beta+\delta)(\gamma+\delta+\delta_1)} \times S_0^* & \frac{\alpha_a}{\gamma+\delta+\delta_1} \times S_0^* \end{pmatrix}.$$

The spectral radius of the matrix FV^{-1} is given by

$$\rho(FV^{-1}) = \frac{\alpha\beta + \alpha_a\beta + \alpha_a\delta}{\beta\delta + \delta\delta_1 + \delta\gamma + \delta^2 + \beta\delta_1 + \beta\gamma} \times S_0^*. \tag{7}$$

Since the basic reproduction number $\mathscr{R}_0$ of model (2) is the spectral radius of FV^{-1}, we have

$$\mathscr{R}_0 = \frac{\alpha\beta + \alpha_a(\beta+\delta)}{(\beta+\delta)(\gamma+\delta+\delta_1)} \times S_0^* = \frac{\Lambda\alpha\beta + \Lambda\alpha_a(\beta+\delta)}{\delta(\beta+\delta)(\gamma+\delta+\delta_1)}. \tag{8}$$

For the convenience, take $b = \beta+\delta$ and $c = \gamma+\delta+\delta_1$. So, using $S_0^* = \frac{\Lambda}{\delta}$ we can rewrite $\mathscr{R}_0$ as

$$\mathscr{R}_0 = \frac{\alpha\beta + \alpha_a b}{bc} \times S_0^* = \frac{\Lambda\alpha\beta + \Lambda\alpha_a b}{\delta bc}. \tag{9}$$

That is,

$$\mathscr{R}_0 = \mathscr{R}_0^s + \mathscr{R}_0^a, \tag{10}$$

where $\mathscr{R}_0^s$ and $\mathscr{R}_0^a$ represents reproduction numbers of symptomatic and asymptomatic infections respectively, as follows

$$\mathscr{R}_0^s = \frac{\Lambda\alpha\beta}{\delta bc}, \ \mathscr{R}_0^a = \frac{\Lambda\alpha_a}{\delta c}.$$

Remark 1. *We have following immediate consequences:*

1. *In absence of asymptomatic infection, that is when $\alpha_a = 0$, then*

$$\mathscr{R}_0 = \mathscr{R}_0^s.$$

2. *In the presence of asymptomatic infection, that is when $\alpha_a > 0$, we get $\mathscr{R}_0^a > 0$ and so*

$$\mathscr{R}_0^s < \mathscr{R}_0^s + \mathscr{R}_0^a = \mathscr{R}_0.$$

3. *Consider $\mathscr{R}_0$ as a function of asymptomatic infection rate α_a, assuming all other parameters are held constant, we have*

$$\mathscr{R}_0(\alpha_a) = \frac{\Lambda\alpha\beta}{\delta bc} + \frac{\Lambda}{\delta c} \times \alpha_a.$$

   Observe that $\mathscr{R}_0$ is an increasing function of α_a.

That is, asymptomatic infection rate (α_a) is crucial in disease dynamics and it is necessary to restrict α_a along with α to control the spread of disease with asymptomatic infection.

3.3 Endemic equilibrium

The endemic equilibrium is the steady state solution of model (2) in which infectious individual persists in the population. Let $\mathscr{E}$ be denote endemic equilibrium, then

$$\mathscr{E} = (S^*, E^*, I^*), \tag{11}$$

where

$$\begin{aligned} S^* &= \frac{bc}{\alpha\beta + \alpha_a b} = \frac{\Lambda}{\delta} \times \frac{1}{\mathscr{R}_0}, \\ E^* &= \frac{\alpha c}{\alpha_a b + \alpha\beta} I_1^* = \frac{\delta\alpha c}{(\alpha + \alpha_a)(\alpha\beta + \alpha_a b)} \times (\mathscr{R}_0 - 1), \\ I^* &= \frac{1}{\alpha + \alpha_a}\left(\frac{\Lambda}{S^*} - \delta\right) = \frac{\delta}{\alpha + \alpha_a} \times (\mathscr{R}_0 - 1), \end{aligned} \tag{12}$$

by direct calculation and using (9). From endemic equilibrium state, we have the following lemma as an immediate conclusion.

Lemma 2. *The endemic equilibrium (11) of the model (2) exists and epidemiologically meaningful if and only if $\mathscr{R}_0 > 1$.*

4 Mathematical analysis of model

We will take $b=\beta+\delta$ and $c=\gamma+\delta+\delta_1$ in this section. We will check the possibility of the phenomenon of Backward bifurcation to verify that can endemic equilibrium co-exist with disease-free equilibrium when $\mathscr{R}_0<1$. Further, we will establish local stability of equilibria.

4.1 Local stability and bifurcation analysis

Lemma 3. *The disease-free equilibrium* (6) *of model* (2) *is locally asymptotically stable if* $\mathscr{R}_0<1$, *and unstable if* $\mathscr{R}_0>1$.

Proof. Using the next generation method discussed earlier, it is clear form Theorem 2 of [29]. □

To determine the possibility of forward or backward bifurcation, we use the Center Manifold Theory approach [6]. For ease of algebraic calculations, we consider the following change of variables:

$$S(t)=x_1,\quad E(t)=x_2,\quad \text{and}\quad I(t)=x_3.$$

Consider a vector, $x=(x_1,x_2,x_3)^T$, where T denotes transpose. Then equation (2) can be written as $\frac{dx}{dt}=f(x)$ such that $f=(f_1,f_2,f_3)^T$ with

$$\begin{aligned}
\dot{x_1} &= f_1(x)=\Lambda-\alpha x_1x_3-\alpha_a x_1x_3-\delta x_1,\\
\dot{x_2} &= f_2(x)=\alpha x_1x_3-bx_2,\\
\dot{x_3} &= f_3(x)=\beta x_2+\alpha_a x_1x_3-cx_3.
\end{aligned}\tag{13}$$

The Jacobian matrix of system (13) at disease-free equilibrium (6) is given by

$$J(\mathscr{E}_0)=\begin{pmatrix}\delta & 0 & -S_0^*(\alpha+\alpha_a)\\ 0 & -b & S_0^*\alpha\\ 0 & \beta & S_0^*\alpha_a-c\end{pmatrix}.$$

At $\mathscr{R}_0=1$, equation (9) gives

$$\frac{\alpha\beta+\alpha_a b}{bc}\times S_0^*=1 \iff S_0^*\alpha_a=c-\frac{\alpha\beta S_0^*}{b}.$$

By considering α as a bifurcation parameter and taking $\alpha=\alpha^*$, we have

$$S_0^*\alpha_a=c-\frac{\alpha^*\beta S_0^*}{b},$$

and thus the Jacobian matrix at $\mathscr{R}_0=1$ and $\alpha=\alpha^*$ is given by

$$J(\mathscr{E}_0)_{\alpha^*}=\begin{pmatrix}\delta & 0 & \frac{S_0^*\alpha^*\beta}{b}-S_0^*\alpha^*-c\\ 0 & -b & S_0^*\alpha^*\\ 0 & \beta & -\frac{S_0^*\alpha^*\beta}{b}\end{pmatrix}.$$

At $\mathscr{R}_0=1$, the Jacobian matrix $J(\mathscr{E}_0)_{\alpha^*}$ of the system (13) with $\alpha=\alpha^*$ has followings conclusions:

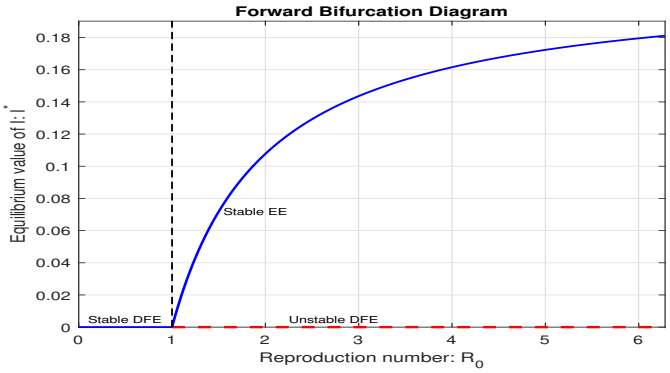

Figure 2: Forward bifurcation at $\mathscr{R}_0 = 1$ with critical value of the bifurcation parameter $\alpha^* = 0.1592 \approx 0.159$. That is, if $\alpha \leq \alpha^*$ then $\mathscr{R}_0 \leq 1$ and if $\alpha > \alpha^*$ then $\mathscr{R}_0 > 1$.

1. 0 is a simple eigenvalue of the matrix $J(\mathscr{E}_0)_{\alpha^*}$ and remaining eigenvalues are negative real numbers.
2. The right eigenvector of the eigenvalue (in view of $b = \beta + \delta$) is given by $w = [w_1, w_2, w_3]^T$, where
$$w_1 = -\frac{bc + S_0^* \alpha^* b - S_0^* \alpha^* \beta}{b\delta} = -\left(\frac{c}{\delta} + \frac{S_0^* \alpha^*}{b}\right), \qquad w_2 = \frac{S_0^* \alpha^*}{b}, \qquad w_3 = 1.$$
3. The left eigenvector of the eigenvalue 0 is given by $u = [v_1, v_2, v_3]^T$, where
$$v_1 = 0, \qquad v_2 = \frac{\beta}{b}, \qquad v_3 = 1.$$

To identify the direction of bifurcation, we need to determine signs of bifurcation coefficients $\mathscr{A}$ and $\mathscr{B}$ provided by

$$\mathscr{A} = \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathscr{E}_0, \alpha^*) = -\frac{c(bc + S_0^* \alpha^* b - S_0^* \alpha^* \beta)}{\Lambda b} = -\frac{c(bc + S_0^* \alpha^* \delta)}{\Lambda b},$$
$$\mathscr{B} = \sum_{k,i=1}^{3} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha}(\mathscr{E}_0, \alpha^*) = \frac{S_0^* b \beta}{b^2 + S_0^* \alpha^* \beta}.$$

We have $\mathscr{A} < 0$ and $\mathscr{B} > 0$, by Theorem 4.1 in [6], model (2) does not have backward bifurcation at $\mathscr{R}_0 = 1$. Consequently (2) gives away the possibility of co-existence of endemic equilibrium along with disease-free equilibrium. Thus, at $\mathscr{R}_0 = 1$, forward (or transcritical) bifurcation exists and plotted in Figure 2.

Lemma 4. *The endemic equilibrium (11) of system (2) is unique and locally asymptotically stable if* $\mathscr{R}_0 > 1$.

Proof. From the above discussion of forward bifurcation, Theorem 4.1 of [6] gives this lemma. □

Remark 2. *If* $\mathscr{R}_0 > 1$ *then Lemma 4 indicates that eventually the trajectories of (2) are either monotone or damped oscillatory. So, no sustained periodic solutions of system (2) exists as local stability of endemic equilibrium (11) omits possibilities of Hopf bifurcation.*

4.2 Global stability analysis of equilibria

This section discusses global stability of equilibria using Lyapunov functions method as surveyed in [5, 25]. Appropriate Lyapunov functions has been constructed to establish global stability.

Theorem 2. *The disease-free equilibrium* (6) *of model* (2) *is globally asymptotically stable if* $\mathscr{R}_0 \leq 1$.

Proof. Consider a Lyapunov candidate on $\mathscr{D}$ as follow:

$$\mathscr{L}_0(S,E,I) = S - S_0^* - S_0^* \ln\left(\frac{S}{S_0^*}\right) + E + \frac{b}{\beta} I. \tag{14}$$

Since the function $f(x) = x - 1 - \ln(x), x > 0$, attains its global minimum value at 1 and $f(1) = 0$, then $f(x) > 0$ for all $x \neq 1$. Applying this for (14), we have

$$\mathscr{L}_0(S,E,I) > 0, \text{ for all } (S,E,I) \in \mathscr{D} \text{ except } \mathscr{E}_0. \tag{15}$$

Also notice that

$$\mathscr{L}_0(\mathscr{E}_0) = 0, \quad \text{and} \quad \mathscr{L}_0(S,E,I) \to \infty \quad \text{as} \quad \|(S,E,I)\| \to \infty. \tag{16}$$

Equations (14), (15), and (16) indicate that $\mathscr{L}_0$ is an appropriate Lyapunov function.
By taking time derivative of $\mathscr{L}_0$, we have

$$\begin{aligned}
\dot{\mathscr{L}}_0(S,E,I) &= \left(1 - \frac{S_0^*}{S}\right)\dot{S} + \dot{E} + \frac{b}{\beta}\dot{I} \\
&= \left(1 - \frac{S_0^*}{S}\right)(\Lambda - \alpha SI - \alpha_a SI - \delta S) + (\alpha SI - bE) + \frac{b}{\beta}(\alpha_a SI + \beta E - cI) \\
&= \Lambda - \alpha SI - \alpha_a SI - \delta S - \frac{S_0^*}{S}(\Lambda) + \alpha S_0^* I + \alpha_a S_0^* I + \delta S_0^* + \alpha SI - bE + bE + \frac{\alpha_a b}{\beta} SI - \frac{bc}{\beta} I.
\end{aligned} \tag{17}$$

Here, b and c are as mentioned at the beginning of this section.
Plunging $\Lambda = \delta S_0^*$, $b = \beta + \delta$ in (17) and by cancelling and collecting appropriate terms, we have

$$\begin{aligned}
\dot{\mathscr{L}}_0(S,E,I) &= \delta S_0^* \left(2 - \frac{S}{S_0^*} - \frac{S_0^*}{S}\right) + \left(\alpha S_0^* + \alpha_a S_0^* + \frac{\alpha_a \delta S}{\beta} - \frac{bc}{\beta}\right) I \\
&= \delta S_0^* \left(2 - \frac{S}{S_0^*} - \frac{S_0^*}{S}\right) + \frac{bc}{\beta}\left(\frac{\alpha\beta S_0^* + \alpha_a b S_0^*}{bc} - 1\right) I \\
&= \delta S_0^* \left(2 - \frac{S}{S_0^*} - \frac{S_0^*}{S}\right) + \frac{bc}{\beta}(\mathscr{R}_0 - 1) I.
\end{aligned} \tag{18}$$

Using the relation between arithmetic and geometric progression, we have

$$\left(2 - \frac{S}{S_0^*} - \frac{S_0^*}{S}\right) \leq 0.$$

From (18) we have $\dot{\mathscr{L}_0} \leq 0$, whenever $\mathscr{R}_0 \leq 1$. Moreover

$$\{(S,E,I) \in \mathscr{D} : \mathscr{L}_0(S,E,I) = 0\} = \left\{ \left(\frac{\Lambda}{\delta}, 0, 0 \right) \right\}.$$

Hence, by by Lasalle's invariance principle [18] disease-free equilibrium is globally asymptotically stable on $\mathscr{D}$. □

Proposition 1. *The set* $\mathscr{D}^* = \{(S,E,I) \in \mathscr{D} : (S-S^*)(I-I^*) \leq 0\}$ *is attracting and positively invariant whenever* $\mathscr{R}_0 > 1$.

Proof. Let $\mathscr{R}_0 > 1$. Then Lemma 4 indicates that system (2) does not admit sustained periodic solutions at endemic equilibrium ((11)), we conclude that trajectories of (2) eventually become either monotone or exhibit damped oscillations and so does I. As a pandemic subside in some finite time, trajectories—particularly I—become monotone after a limited number of oscillations. Since our primary interest lies in the long-term or asymptotic behavior, we may assume that either $I \geq I^*$ or $I \leq I^*$.
We first take $I \geq I^*$. As $\mathscr{R}_0 > 1$, by (11) we have $I^* = \frac{\delta}{\alpha+\alpha_a} \times (\mathscr{R}_0 - 1)$ and $S^* = \frac{\Lambda}{\delta} \times \frac{1}{\mathscr{R}_0}$. Thus form first equation of (2), we get

$$\begin{aligned}
\frac{dS}{dt} \leq & \Lambda - (\alpha + \alpha_a) S I^* - \delta S \\
= & \Lambda - ((\alpha + \alpha_a) I^* + \delta) S \\
= & \Lambda - (\delta(\mathscr{R}_0 - 1) + \delta) S \\
= & \delta \mathscr{R}_0 \left(\frac{\Lambda}{\delta \mathscr{R}_0} - S \right) \\
= & \delta \mathscr{R}_0 (S^* - S).
\end{aligned}$$

This implies

$$S(t) \leq S^* - (S^* - S(0)) e^{-t\delta\mathscr{R}_0}.$$

Thus, either S converges to S^* asymptotically or $S \leq S^*$ after some finite time (similar conclusion is in an equation of proof of Theorem 2 of [28] p. 443). So, I converges to I^* asymptotically or since $I \geq I^*$, $(S-S^*)(I-I^*)$ becomes and remains non-positive. The same hold for the later case of $I \leq I^*$.
Hence the set D^* is attracting and positively-invariant. □

Next, we discuss global stability of endemic equilibrium over the set $\mathscr{D}^*$ using Lyapunov function method.

Theorem 3. *The endemic equilibrium* (11) *of model* (2) *is globally asymptotically stable on* $\mathscr{D}^*$ *as follows:*

Proof. Consider a Lyapunov candidate on $\mathscr{D}^*$

$$\mathscr{L}(S,E,I) = S - S^* \left(1 + \ln \left(\frac{S}{S^*} \right) \right) + E - E^* \left(1 + \ln \left(\frac{E}{E^*} \right) \right) + \frac{b}{\beta} \left[I - I^* \left(1 + \ln \left(\frac{I}{I^*} \right) \right) \right] \quad (19)$$

Since, the function $f(x) = x - 1 - \ln(x)$, $x > 0$, attains its global minimum value at 1 and $f(1) = 0$, then $f(x) > 0$ for all $x \neq 1$. Applying it to (19), we get

$$\mathscr{L}(S,E,I) > 0, \text{ for all } (S,E,I) \in \mathscr{D}^* \text{ except } \mathscr{E}. \tag{20}$$

Also notice that

$$\mathscr{L}(\mathscr{E}) = 0, \quad \text{and} \quad \mathscr{L}(S,E,I) \to \infty \quad \text{as} \quad \|(S,E,I)\| \to \infty. \tag{21}$$

Equations (19), (20) and, (21) indicate that $\mathscr{L}$ is an appropriate Lyapunov function.
By taking time derivative of $\mathscr{L}$, we have

$$\begin{aligned}
\dot{\mathscr{L}} &= \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{E^*}{E}\right)\dot{E} + \frac{b}{\beta}\left(1 - \frac{I^*}{I}\right)\dot{I} \\
&= \left(1 - \frac{S^*}{S}\right)(\Lambda - \alpha SI - \alpha_a SI - \delta S) + \left(1 - \frac{E^*}{E}\right)(\alpha SI - bE) \\
&\quad + \frac{b}{\beta}\left(1 - \frac{I^*}{I}\right)(\beta E + \alpha_a SI - cI) \\
&= \Lambda - \alpha SI - \alpha_a SI - \delta S - \Lambda\frac{S^*}{S} + \alpha S^* I + \alpha_a S^* I + \delta S^* + \alpha SI - bE - \alpha E_1^*\frac{SI}{E} + bE_1^* + bE \\
&\quad + \frac{\alpha_a b}{\beta} SI - \frac{bc}{\beta} I - bI^*\frac{E}{I} - \frac{\alpha_a b}{\beta} SI^* + \frac{cb}{\beta} I^*.
\end{aligned} \tag{22}$$

Using endemic state conditions

$$\begin{aligned}
\Lambda &= \alpha S^* I^* + \alpha_a S^* I^* + \delta S^*, \\
bE^* &= \alpha S^* I^*, \\
cI^* &= \alpha_a S^* I^* + \beta E^*, \\
b &= \beta + \delta,
\end{aligned}$$

in equation (22) and by direct calculations, we have

$$\begin{aligned}
\dot{\mathscr{L}} &= (\delta S^* + \alpha_a S^* I^*)\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) + \alpha S^* I^*\left(3 - \frac{S^*}{S} - \frac{E^* SI}{ES^* I^*} - \frac{EI^*}{E^* I}\right) \\
&\quad + \frac{bc}{\beta}\left(\frac{\alpha\beta S^* + \alpha_a \beta S^* + \alpha_a \delta S^*}{bc} - 1\right) + \frac{\alpha_a \delta}{\beta}(SI - S^* I - SI^* - S^* I^*) \\
&= (\delta S^* + \alpha_a S^* I^*)\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) + \alpha S^* I^*\left(3 - \frac{S^*}{S} - \frac{E^* SI}{ES^* I^*} - \frac{EI^*}{E^* I}\right) \\
&\quad + \frac{bc}{\beta}\left(\frac{\alpha\beta + \alpha_a b}{bc} \times S^* - 1\right) + \frac{\alpha_a \delta}{\beta}(S - S^*)(I - I^*).
\end{aligned} \tag{23}$$

From the relation between arithmetic and geometric means, we have

$$\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right) \leq 0, \tag{24}$$

$$\left(3-\frac{S^*}{S}-\frac{E^*SI}{ES^*I^*}-\frac{EI^*}{E^*I}\right)\leq 0, \tag{25}$$

and by plug-in the value of S^*, we get

$$\left(\frac{\alpha\beta+\alpha_a b}{bc}\times S^*-1\right)\leq 0. \tag{26}$$

Also, note that $(S-S^*)(I-I^*)\leq 0$ on D^*. Combining this with (24), (25) and, (26) and finally applying it to (23), we have $\dot{\mathscr{L}}\leq 0$. Also, notice that

$$\{(S,E,I)\in\mathscr{D}^* : \dot{\mathscr{L}}(S,E,I)=0\}=\{\mathscr{E}\}.$$

Hence, by Lasalle's invariance principle [18] endemic equilibrium is globally asymptotically stable on $\mathscr{D}^*$. □

Remark 3. *If $\mathscr{R}_0>1$, then Proposition 1 implies that all trajectories of system (2) enter the positively invariant set $\mathscr{D}^*$ in finite time and remain there thereafter. Furthermore, Theorem 3 guarantees the global asymptotic stability of the endemic equilibrium (11) on $\mathscr{D}^*$. Hence, if $\mathscr{R}_0>1$ the endemic equilibrium (11) is globally asymptotically stable.*

5 Numerical simulations

In this section, we perform numerical simulations to support the analytical results derived in the previous section. The model equations given in system (2) are solved using ODE45 environment in MATLAB.

We use parameter values based on early COVID-19 data from Wuhan. The infection rate is taken as $\alpha=0.533$, and the average incubation period is 5.2 days, which implies $\beta=1/5.2\approx 0.192$ [20]. The global case fatality rate, as of 10 June 2020, is reported as $\delta_1=0.057$, computed as the ratio of total deaths to total reported cases [30]. According to [26], the median recovery period is 7 days, which gives $\gamma=1/7\approx 0.143$. The recruitment rate and natural death rate are both taken as $\mu=0.048$, based on the crude birth rate reported by WHO in 2020 [31]. We assume these two rates to be equal in order to keep the total population constant over time. This allows us to focus on the disease dynamics without the influence of natural population growth or decline. With these values, the basic reproduction number is estimated as $\mathscr{R}_0=3.3487>1$. To study the case when $\mathscr{R}_0<1$, we assume $\alpha=0.133$ and keep all other parameters unchanged and it gives $\mathscr{R}_0=0.8372$. To observe the impact of asymptomatic transmission, we assume $\alpha_a=p\alpha$, where $p=0.2,0.4,0.6,0.8,1.0$. This is based on the study of [32], which reports that the asymptomatic infection rate α_a can be up to 100% of the symptomatic infection rate α.
Using these parameter values, we carry out the following simulations:

- **Figure 3**: This figure indicates that when $\mathscr{R}_0\leq 1$, the disease dies out, and the disease-free equilibrium becomes globally asymptotically stable. This supports Theorem 2 (Here, $\alpha_a=0.4\alpha$).
- **Figure 4**: The solution of system (2) enters the set $\mathscr{D}^*$ in finite time and remains there for all future time. This confirms that $\mathscr{D}^*$ is positively invariant and attracting, thereby supporting Proposition 1 (Here, $\alpha_a=0.4\alpha$).

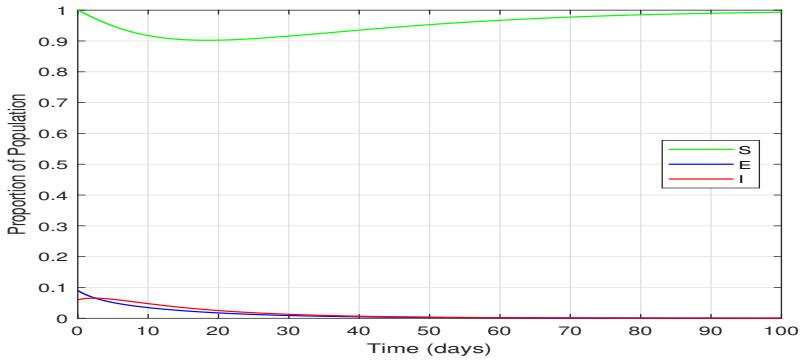

Figure 3: In case of $\mathscr{R}_0 = 0.8722 \leq 1$

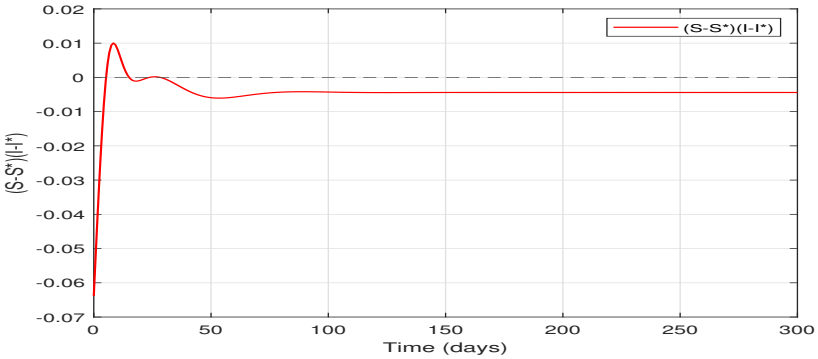

Figure 4: $\mathscr{D}^*$ is attracting and positive invariant

- **Figure 5**: When $\mathscr{R}_0 > 1$, the disease persists, and the endemic equilibrium becomes globally asymptotically stable. This validates Theorem 3 (Here, $\alpha_a = 0.4\alpha$).
- **Figure 6**: This figure illustrates the effect of asymptomatic infection on the disease-free equilibrium when α_a is taken as $20\%, 40\%, 60\%, 80\%$, and 100% of α. It is observed that higher asymptomatic transmission leads to a delay in reaching the disease-free state and, in some cases, may drive the system toward an endemic state.
- **Figure 7**: This figure demonstrates the impact of asymptomatic transmission on the endemic equilibrium using the same values of α_a as in Figure 6. The results show that higher values of α_a lead to faster and larger outbreaks.

These simulations confirm the analytical results and highlight the significant role of asymptomatic transmission in shaping the disease dynamics.

5.1 Testing and isolation as a control measures

Asymptomatic infections cannot be identified without testing, making strategies like contact tracing and rapid testing essential for detecting these hidden cases. To effectively control the spread of the disease,

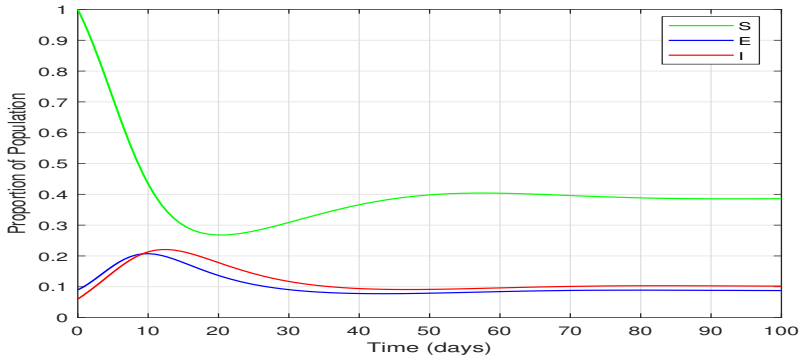

Figure 5: In case of $\mathscr{R}_0 = 2.9073 > 1$

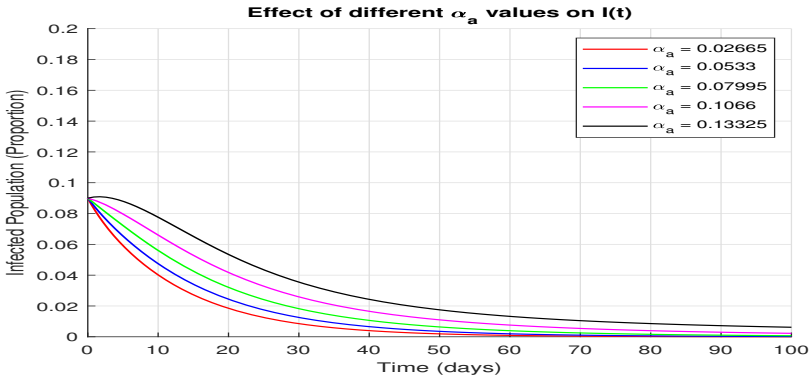

Figure 6: Impact of α_a on Disease-Free Equilibrium

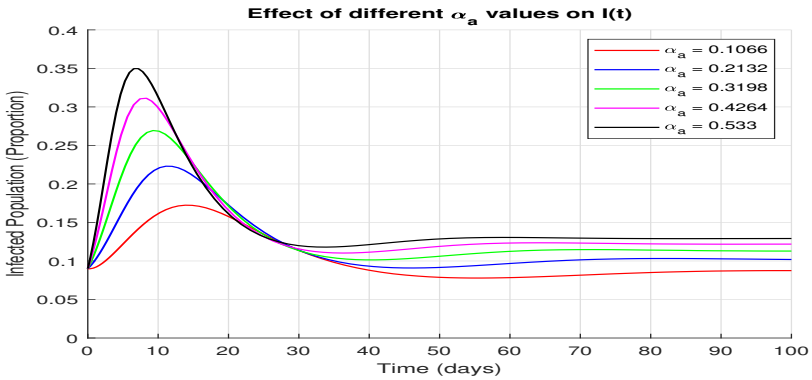

Figure 7: Impact of α_a on Endemic Equilibrium

it is also important to isolate both symptomatic and asymptomatic individuals once they are identified.

Since the demand for testing increases with the number of infected individuals, we assume that susceptible individuals test positive through rapid testing at a rate proportional to the infectious population, denoted by νI. Similarly, infected individuals are isolated at a rate μI. Those in isolation are assumed not to transmit the infection and remain isolated until they either recover or die, transitioning to the recovered compartment or deceased. To evaluate the effectiveness of testing and isolation, we incorporate these processes into (2) as:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha SI - \alpha_a SI - \delta S - \nu I, \\
\frac{dE}{dt} &= \alpha SI - (\beta + \delta)E, \\
\frac{dI}{dt} &= \beta E + \alpha_a SI - (\gamma + \delta + \delta_1)I - \mu I,
\end{aligned} \tag{27}$$

where $S \geq 0, E \geq 0,$ and $I \geq 0$.

System (27) is simulated for the case $\mathscr{R}_0 > 1$, using three different values—0, 0.05, and 0.1—for both the testing rate (ν) and the isolation rate (μ), as shown in Figure 8. This simulation uses the same parameter values as in the case $\mathscr{R}_0 = 3.387 > 1$, except for α_a, which is varied according to the set of values used in Figure 7.

The subfigures demonstrate that both testing and isolation contribute to reducing the peak of the infectious population (I). A comparison between subfigures (b) and (d), as well as (c) and (g), indicates that increasing the isolation rate μ has a stronger effect on lowering the peak than increasing the testing rate ν. This suggests that isolation is more effective than testing in controlling the spread.

Moreover, subfigure (i) shows that combining both testing and isolation yields a significantly greater reduction in the peak of infection compared to applying either measure alone, highlighting the synergistic benefit of implementing both interventions simultaneously.

Additionally, testing will helpful to find out the proportion of α_a compared to α, which is useful to predict the dynamics and improvement of preparedness to control the disease.

6 Conclusion and discussion

In this study, we formulated and analyzed a compartmental model to understand the transmission dynamics of COVID-19 like diseases, incorporating both symptomatic and asymptomatic infections. The model was studied analytically, and several key theoretical results were established, including the global stability of the disease-free equilibrium when $\mathscr{R}_0 \leq 1$, the occurrence of forward bifurcation at $\mathscr{R}_0 = 1$, and the global stability of the unique endemic equilibrium when $\mathscr{R}_0 > 1$. These results confirm that the minimal model is mathematically well-posed, dynamically stable, and epidemiologically meaningful.

To support the theoretical results, numerical simulations were carried out using parameter values informed by early COVID-19 data. The simulation results confirmed the analytical findings: the disease dies out when $\mathscr{R}_0 \leq 1$, and persists at a stable endemic level when $\mathscr{R}_0 > 1$. Furthermore, the impact of asymptomatic transmission was explored by varying the rate α_a relative to symptomatic transmission. It was observed that higher asymptomatic transmission significantly delays disease elimination and can shift the system from a disease-free state to an endemic one. Additionally, in endemic scenarios, larger values of α_a led to faster spread and higher peak infection levels. In line of this, simulation results of

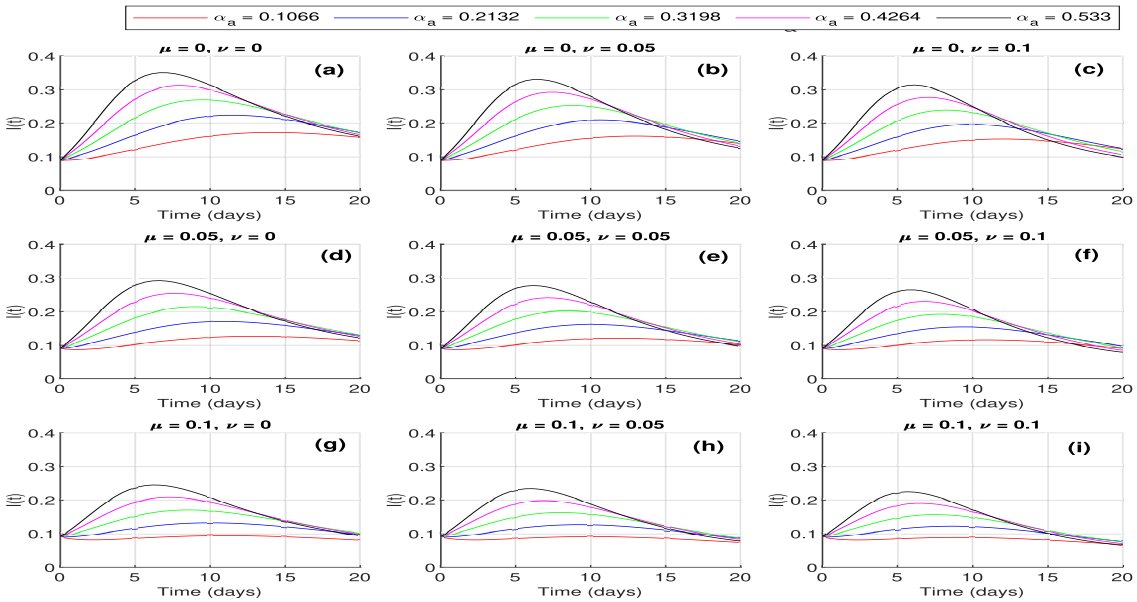

Figure 8: Impact of μ and ν on α_a on peak of I.

control measures demonstrate that while both testing and isolation reduce the peak of infections, isolation has a comparatively stronger impact. Moreover, combining testing and isolation yields a synergistic effect, leading to a more substantial reduction in disease spread. These simulations highlight that ignoring asymptomatic transmission can lead to underestimation of disease persistence and severity. It also show up crucial role of asymptomatic carriers in the spread of disease like COVID-19 along with comparison of prevention strategies.

Overall, this study provides minimal deterministic *SEIR* model with mathematically proven support to dynamics of infectious diseases with asymptomatic transmission and assures that even though asymptotic transmissions come up as challenge, transmission dynamics can be determined in term of the reproduction number as: when $\mathscr{R}_0 \leq 1$, the disease will be eradicated, whereas for $\mathscr{R}_0 > 1$, the disease will persist. Furthermore, the disease transmission and related casualties can be effectively reduced through interventions and control measures.

Acknowledgment

We thank the anonymous reviewers for thoughtful comments and constructive suggestions that strengthened the manuscript.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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