Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Computers in Biology and Medicine 143 (2022) 105264

Contents lists available at ScienceDirect

Computers in Biology and Medicine

journal homepage: www.elsevier.com/locate/compbiomed

Modeling the SARS-CoV-2 parallel transmission dynamics: Asymptomatic and symptomatic pathways

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ARTICLE INFO

Keywords:
SARS-CoV-2
COVID-19
Mathematical model
Symptomatic transmission
Asymptomatic transmission
Basic reproduction number

ABSTRACT

Asymptomatic transmission of the coronavirus disease and the infected individual prediction has become very important in the COVID-19 outbreak study. The asymptomatic and symptomatic transmission studies are still ongoing to assess their impacts on disease monitoring and burden. However, there has been limited research on how asymptomatic and symptomatic transmissions together can affect the coronavirus disease outbreak. A mathematical model is therefore needed to be developed in order to assess the effect of these transmissions on the coronavirus disease dynamics. This paper develops a mathematical model concerning asymptomatic and symptomatic disease transmission processes in the COVID-19 outbreak. The model sensitivity has been analysed in terms of the variance of each parameter, and the local stability at two equilibrium points have been discussed in terms of the basic reproduction number (R_0). It is found that the disease-free equilibrium gets stable for $R_0 < 1$ whereas the endemic equilibrium becomes stable for $R_0 > 1$ and unstable otherwise. The proportion of the effect of asymptomatic and symptomatic transmission rates on R_0 is calculated to be approximately between 1 and 3. The results demonstrate that asymptomatic transmission has a significant impact compared to symptomatic transmission in the disease outbreak. Outcomes of this study will contribute to setting an effective control strategy for the COVID-19 outbreak.

1. Introduction

The COVID-19 (coronavirus) disease has become the most infectious disease affecting the human society on a wide scale. Since 2019, this virus has been a serious public health concern [1]. Several pneumonia cases with unknown causes were primarily addressed by the health authority of the Wuhan municipal, and later on, the unknown cause was identified as a new type of coronavirus, namely COVID-19 by the World Health Organization (WHO). The pandemic was declared on 11 March 2020 by WHO, which had then spread across the globe within a very short time [2,3] and a preliminary guideline on public health management for the territories dealing with this outbreak was published on the WHO website [3,4]. A generic structure of coronavirus shows that it contains a number of different proteins: nucleocapsid, membrane, and spike proteins on its surface [5,6]. The spike proteins show active interactions with the host living cells and are mostly responsible for the spread of the disease. As time passed, this contagious infection has become a global threat to public health and has been spread to approximately 216 nations, areas and territories around the world [7].

Most of the COVID-19 infected individuals experience a mild to moderate respiratory illness with some active symptoms like cough, sore throat, fever, headache, tiredness, shortness of breath, and they recover without having any special treatment [3,8]. Individuals with underlying co-morbidities such as cancer, diabetes, cardiovascular problems, and chronic lung disease are more vulnerable to COVID-19. As reported in the New York Times [9], many COVID-19 infected individuals can

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https://doi.org/10.1016/j.compbiomed.2022.105264
Received 27 November 2021; Received in revised form 21 January 2022; Accepted 22 January 2022
Available online 25 January 2022

actively spread the disease before the symptom onset. The median time before the symptom onset is 2–14 days [10] and the longest period is found as 27 days [11]. However, in most cases, asymptomatic cases are not reported to the medical authorities and the worst effect of insufficient testing leads to the spread of disease in silence. Radha et al. [12] conducted a study between asymptomatic infectious but not diagnosed, and confirmed infectious cases in mainland China. The authors found a significant disparity between those cases. Symptomatic individuals are usually kept under surveillance and thus less likely to spread the infection. But without a diagnosis, asymptomatic individuals even do not know whether they are infectious and thus propagating the disease in course of time. Therefore, a new mathematical model becomes necessary to quantify the role of asymptomatic infectious individuals on the COVID-19 outbreak.

Mathematical models always play a significant role in the analysis of the contagious disease dynamics and the prediction of epidemic severity. The epidemic models are mainly compartmental where the total population is divided into different classes [13,14]. These models are very useful for quantifying possible disease spread. In the study of the compartmental model, the most classical one is the SIR model [15] and which was later extended to SIS [16,17], SEIR [18,19] to model different typical complex epidemic situations. These types of models are likewise proficient in determining the threshold potential of many outbreaks. Based on these models, a good number of compartmental models were proposed by different authors to study the COVID-19 outbreak [18, 20–28]. Tang et al. [22] developed a general epidemiological model of type SEIR where isolation, quarantine, and care were considered. Incorporating multiple pathways of transmission, Mojeeb et al. [18] proposed a COVID-19 mathematical model on the basis of the mass action law. The model was fitted with the data collected from Wuhan city, China. Effects of the mitigation strategies on the population level for New York City's COVID-19 outbreak were assessed by Ngonghala et al. [21]. The population-wide impact of asymptomatic infectious individuals was demonstrated by Eikenberry et al. [23]. The authors showed that the use of even relatively ineffective face covering can reasonably minimize the COVID-19 community transmission and reduce the hospitalizations rate. Also, many authors including Nishiura [29] and Mizumoto [30] conducted a statistical analysis of transmitting the COVID-19 disease by asymptomatic patients. Over a statistical study of 157 confirmed individuals and 30 asymptomatic infected persons, Chen et al. [31] reported that the differences in symptomatic and asymptomatic transmission are not statistically significant. However, in the study of Daihai et al. [32], a statistically significant difference was found between the transmissions using the same data set. Some other researcher [33–35] used clinical studies to assess the strength of asymptomatic transmission against symptomatic transmission.

Since coronavirus is a single RNA virus, there are no specific treatment strategies like antibiotics and other medications against this disease. Therefore, non-pharmaceutical interventions like social distancing, isolation, quarantine, awareness programs, and personal hygiene including wearing a mask, washing hands regularly become more useful in minimizing the transmission of the COVID-19 disease [36]. Surprisingly, it is noticed that people maintaining all practicable steps are sometimes infected and thus it becomes important to analyse the facts in human to human transmission processes. Although clinically nothing has yet been purely known on the transmission process of the COVID-19, many compartmental models provide simulation graphs very close to statistical data. In most cases, asymptomatic individuals remain out of the COVID-19 diagnosis test and play a significant role in spreading the disease. Also, there are many cases of individuals who died of other diseases but tested positive for COVID-19 after their death. Since COVID-19 is more of a contagious disease than a deadly one, a large proportion of the total population becomes a carrier in the long run. Asymptomatic illnesses' contributions to herd immunity and community transmission are critical to COVID-19 resurgence and control, but they are challenging to quantify using existing available models that disregard the way asymptomatic transmission occurs. A deterministic model that reliably estimates the fraction of asymptomatic cases in addition to symptomatic infections could assist epidemiologists in determining whether the basic reproduction number is adequate for predicting disease outbreaks.

There are numerous compartmental models on the spread of COVID-19 disease that include a variety of social and pharmacological intervention policies, and epidemiological data. However, none of them were designed to determine how well asymptomatic cases transmit compared to symptomatic cases. As a result, the models' basic reproduction numbers (R_0) remained incomplete, and so were failed to make precise predictions on community disease transmission. To address this fact and to provide a concise epidemiological understanding of the COVID-19 disease transmission, it becomes necessary to develop a mathematical model that integrates both the symptomatic and asymptomatic disease transmission simultaneously. Therefore, this paper develops such a mathematical model integrating the parallel transmission dynamics in the COVID-19 outbreak. Two parallel pathways for symptomatic and asymptomatic transmission across the exposed compartment are considered to construct the mathematical model, namely the parallel-SEIR model. The model analyses the asymptomatic transmission effect on the overall COVID-19 outbreak and the sensitivity of the model's parameter affecting the endemic. The systematic organization of this study is presented in Fig. 1.

2. The Parallel-SEIR model formulation

A parallel-SEIR model is proposed on COVID-19 disease transmission in this section, where the total population is classified into five compartments: susceptible compartment **S**, exposed compartment **E**, asymptomatic infected compartment ($\mathbf{I_1}$), symptomatic infected compartment ($\mathbf{I_2}$); and recovered compartment **R**. The model assumes the total population **N(t)** is not constant throughout the time and equal

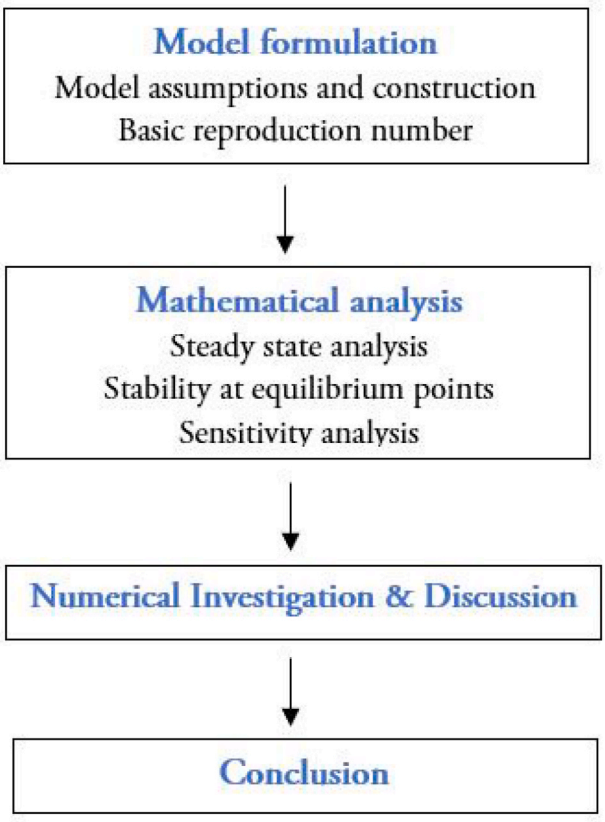

Fig. 1. Systematic overview of the study.

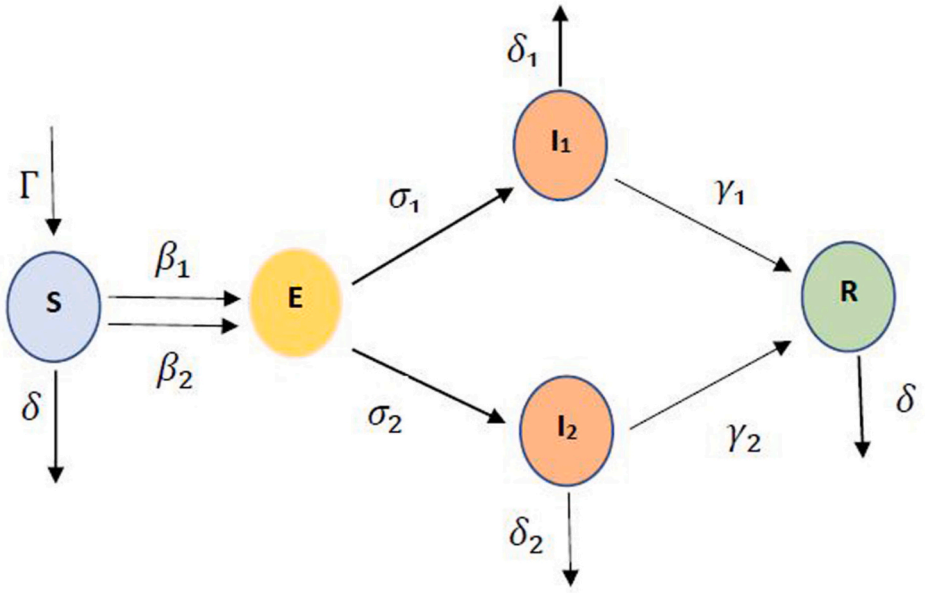

Fig. 2. Schematic diagram of the parallel-SEIR model.

to the sum of all compartment sizes at any time t, i.e. $\mathbf{N(t) = S(t) + E(t) + I_1(t) + I_2(t) + R(t)}$.The following assumptions are made to construct the model:

A1. The primary pathway SEI_1R represents the asymptomatic transmission where the asymptomatic infectious individuals spread the disease at the rate β_1. The second pathway SEI_2R signifies the indicative transmission where symptomatic individuals spread the illness at the rate β_2. The transmission and infection rate for the symptomatic case is considered to be higher than those in the asymptomatic case, i.e. $\beta_2 > \beta_1$, and $\sigma_2 > \sigma_1$.
A2. The recovery rate in asymptomatic patients is higher than that of symptomatic patients ($\gamma_1 > \gamma_2$), and the death rate of asymptomatic patients is lower than that in symptomatic individuals, ($\delta_2 > \delta_1$).
A3. Recruitment rate into the compartment S is equal to the natural death rate δ.
A4. In the interim compartments of this model, death is considered only because of the COVID-19 infection.

Using the assumptions (A1) − (A4), the following nonlinear system of differential equations is developed:

$$\begin{aligned}
\frac{d\mathbf{S}}{dt} &= \pi - \delta\mathbf{S} - \beta_1\mathbf{S}\frac{\mathbf{I_1}}{\mathbf{N}} - \beta_2\mathbf{S}\frac{\mathbf{I_2}}{\mathbf{N}} \\
\frac{d\mathbf{E}}{dt} &= \beta_1\mathbf{S}\frac{\mathbf{I_1}}{\mathbf{N}} + \beta_2\mathbf{S}\frac{\mathbf{I_2}}{\mathbf{N}} - \sigma_1\mathbf{E} - \sigma_2\mathbf{E} \\
\frac{d\mathbf{I_1}}{dt} &= \sigma_1\mathbf{E} - \delta_1\mathbf{I_1} - \gamma_1\mathbf{I_1} \\
\frac{d\mathbf{I_2}}{dt} &= \sigma_2\mathbf{E} - \delta_2\mathbf{I_2} - \gamma_2\mathbf{I_2} \\
\frac{d\mathbf{R}}{dt} &= \gamma_1\mathbf{I_1} + \gamma_2\mathbf{I_2} - \delta\mathbf{R}
\end{aligned} \tag{1}$$

where the recruitment rate π is assumed as a constant, representing an entrance into the compartment S in the form of birth or migration or recovered individuals and δ is the natural death rate. The parameters β_i, σ_i, γ_i, and δ_i (i = 1, 2) represents the transmission rate, infection rate, recovery rate, and death rate in asymptomatic and symptomatic pathways respectively.

In the first equation, the terms $\beta_1\mathbf{S}\frac{\mathbf{I_1}}{\mathbf{N}}$ and $\beta_2\mathbf{S}\frac{\mathbf{I_2}}{\mathbf{N}}$ denote the interaction of susceptible people with asymptomatic and symptomatic infectious individuals with rates β_1 and β_2 respectively. These interactions increase the number of exposed individuals as seen in the second equation. Also the terms $\sigma_1\mathbf{E}$ and $\sigma_2\mathbf{E}$ represent the number of people becoming infected through the exposure of susceptible individuals. In the third and fourth equations, $\delta_1\mathbf{I_1}$, $\gamma_1\mathbf{I_1}$ and $\delta_2\mathbf{I_2}$, $\gamma_2\mathbf{I_2}$ specify the number of deceased and recovered individuals in asymptomatic and symptomatic infected compartments. The terms $\delta\mathbf{S}$ and $\delta\mathbf{R}$ in the first and fifth equation represent the natural death of susceptible and recovered individuals respectively.

Dividing all the equations of the model (1) by (**N**) and setting $S = \frac{\mathbf{S}}{\mathbf{N}}$, $E_1 = \frac{\mathbf{E_1}}{\mathbf{N}}, I_1 = \frac{\mathbf{I_1}}{\mathbf{N}}, I_2 = \frac{\mathbf{I_2}}{\mathbf{N}}$, $R = \frac{\mathbf{R}}{\mathbf{N}}$ and $\Gamma = \frac{\pi}{\mathbf{N}}$ (recruitment rate), the original model (1) can be written as

$$\begin{aligned}
\frac{dS}{dt} &= \Gamma - \delta S - \beta_1 S I_1 - \beta_2 S I_2 \\
\frac{dE}{dt} &= \beta_1 S I_1 + \beta_2 S I_2 - \sigma_1 E - \sigma_2 E \\
\frac{dI_1}{dt} &= \sigma_1 E - \delta_1 I_1 - \gamma_1 I_1 \\
\frac{dI_2}{dt} &= \sigma_2 E - \delta_2 I_2 - \gamma_2 I_2 \\
\frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 I_2 - \delta R
\end{aligned} \tag{2}$$

where S + E + I_1 + I_2 + R = 1. The proposed parallel-SEIR model (Fig. 1) for COVID-19 outbreak is considered under the initial conditions: S(0), E(0), $I_1(0)$, $I_2(0)$, R(0) ≥ 0 and N(0) > 0. All of the parameters (β_i, σ_i, γ_i, δ_i) are assumed to be non-negative with appropriate epidemiological interpretation. The model is designed to investigate the role of asymptomatic transmission rate compared to symptomatic transmission rate in the COVID-19 outbreak.

To assess the feasibility of the proposed parallel-SEIR model, a quantitative measure, namely the basic reproduction number (R_0) needs to be evaluated which has been discussed in the following section.

2.1. Basic reproduction number

Basic reproduction number (R_0) is a potential threshold condition [37,38] determining whether the disease continues or becomes extinct in the population as time evolves. In general, it is known as the ratio of newly infected people to the total infected people [37]. Therefore, R_0 > 1 implies that a single primary infection causes more than one secondary

infection and disease spreads across the community causing an epidemic. In contrast, if $R_0 < 1$ the disease transmission rate is significantly low and can not persist in the population. Previous studies conducted on COVID-19 estimated R_0 in a number of ways and updated it continuously as new information emerged in time. Based on early evidence, WHO estimated R_0 in between 1.4 and 2.5 while Zhao et al. [39, 40] predicted R_0 in between 3.6 and 4.0 and 2.24–3.58. In other studies [41–43], R_0 was estimated in the range of 1.5–3.5.

In the current proposed parallel-SEIR model for COVID-19 endemic, the idea of the next generation matrix (NGM) [44] is used to determine R_0. Linearising equations (2)–(4) of the model (1) as a subsystem of infection [29], the following transmission matrix T and transition matrix Σ are obtained:

$$T = \begin{pmatrix} 0 & \beta_1 S & \beta_2 S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \Sigma = \begin{pmatrix} (\sigma_1+\sigma_2) & 0 & 0 \\ -\sigma_1 & (\delta_1+\gamma_1) & 0 \\ -\sigma_2 & 0 & (\delta_2+\gamma_2) \end{pmatrix}$$

The zero rows in the transmission matrix T imply that there is no individual in these states after infection, and thus the next generation matrix (NGM) [44], $K = P'T\Sigma^{-1}P$ will be of (1×1) order where P is an auxiliary matrix consisting of a unit vector $(1, 0, 0)'$. Using this next generation matrix K, R_0 is determined as below

$$R_0 = \frac{\beta_1 S\sigma_1}{(\delta_1+\gamma_1)(\sigma_1+\sigma_2)} + \frac{\beta_2 S\sigma_2}{(\delta_2+\gamma_2)(\sigma_1+\sigma_2)} \tag{3}$$

where $S = \frac{\Gamma}{\delta}$ represents the value of the susceptible individuals at disease-free equilibrium.

3. Mathematical analysis of the model

In this section, the feasibility of the solution trajectories of the model has been studied using well-posedness whereas the solution stability has been determined at steady states.

3.1. Boundedness and non-negativity

The solutions of the system are positively invariant and bounded under the model assumptions with the given initial conditions {S(0), E(0), $I_1(0)$, $I_2(0)$, R(0) ≥ 0}.

Let N(t) represents the total population size where N(t) = S(t) + E(t) + I_1(t) + I_2(t) + R(t). Computing the rate of change of the total population, the following equation is derived:

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_1}{dt} + \frac{dI_2}{dt} + \frac{dR}{dt} \\ &= \Gamma - \delta S - \delta_1 I_1 - \delta_2 I_2 - \delta R \\ &\le \Gamma - \delta R \end{aligned}$$

Using the fact, R ≤ N

$$\frac{dN}{dt} \le \Gamma - \delta N \tag{4}$$

The solution of the inequality (4), yields

$$N(t) \le \frac{\Gamma}{\delta} + \left(N(0) - \frac{\Gamma}{\delta}\right)e^{-\delta t} \tag{5}$$

where N(0) is the initial population size. Considering large time scale, $\lim_{t\to\infty} N(t) \le \frac{\Gamma}{\delta}$. Thus at any time τ < t during the outbreak, the total population size will be $N(\tau) \le \frac{\Gamma}{\delta}$, i.e. the total population size is bounded above by a positive constant $\frac{\Gamma}{\delta} \in \mathbb{R}$.

In order to check the non-negativity of the solution trajectories, the first equation from model (2), is considered

$$\frac{dS}{dt} = \Gamma - \delta S - \beta_1 S E_1 - \beta_2 S E_2$$

and is written as

$$\frac{dS}{dt} + \delta S \ge 0 \tag{6}$$

The integration of the inequality (6) gives

$$S(t) \ge S(0)e^{-\delta t} \tag{7}$$

The inequality (7) becomes S(t) ≥ 0 for every t ∈ [0, ∞). It indicates that the solution curve S(t) lies entirely in the positive quadrant. A similar approach for the rest of the equations in the model (2) yields S(t) ≥ 0, E(t) ≥ 0, I_1(t) ≥ 0, I_2(t) ≥ 0 and R(t) ≥ 0.

Therefore, the solution space for model (2) is given by

$$\Omega = \left(\left(S,E,I_1,I_2,R\right) \in \mathbb{R}^5_{\ge 0}; 0 < S(t)+E(t)+I_1(t)+I_2(t)+R(t) \le \frac{\Gamma}{\delta} \subset \mathbb{R}^5_{\ge 0}\right)$$

which ensures the feasibility of the model both epidemiologically and mathematically. Therefore, the results are summarized by proposing the following lemma:

Lemma 1. *The feasible region of the solutions of the system* (2) *is bounded and non-negative.*

3.2. Steady state analysis

Steady state analysis of an epidemic model plays a vital role to determine the local stability at the points of the endemic equilibrium (EE) and disease-free equilibrium (DFE) for large-scale time evolution. At any of the DFE or EE points the value of R_0 helps to understand whether the disease does exist or extinct or under control. At DFE, $R_0 > 1$ implies that the equilibrium is unstable resulting in an outbreak and $R_0 < 1$ indicates the equilibrium is asymptotically stable.In order to compute these equilibrium points, Eq. (2) is obtained by setting $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dR}{dt} = 0$

$$\begin{aligned} \Gamma - \delta S - \beta_1 S I_1 - \beta_2 S I_2 &= 0 \\ \beta_1 S I_1 + \beta_2 S I_2 - \sigma_1 E - \sigma_2 E &= 0 \\ \sigma_1 E - \gamma_1 I_1 - \delta_1 I_1 &= 0 \\ \sigma_2 E - \gamma_2 I_2 - \delta_2 I_2 &= 0 \\ \gamma_1 I_1 + \gamma_2 I_2 - \delta R &= 0 \end{aligned} \tag{8}$$

The disease-free equilibrium condition requires $E = I_1 = I_2 = 0$ and applying these conditions in the system of equation (8), the disease-free equilibrium point $(S^0, E^0, I_1^0, I_2^0, R^0)$ is obtained for this parallel-SEIR COVID 19 model as follows:

$$(S^0, E^0, I_1^0, I_2^0, R^0) = \left(\frac{\Gamma}{\delta}, 0, 0, 0, 0\right) \tag{9}$$

The endemic equilibrium means disease persists in the community that can spread after a while and therefore none of the state variables (S, E, I_1, I_2, R) can identically be zero. Thus the endemic equilibrium point $(S^*, E^*, I_1^*, I_2^*, R^*)$ of the system (8) is given by,

$$\begin{aligned} S^* &= \frac{\Gamma}{(\beta_1 I_1 + \beta_2 I_2 + \delta)} \\ E^* &= \frac{S(\beta_1 I_1 + \beta_2 I_2)}{(\sigma_1+\sigma_2)} \\ I_1^* &= \frac{\sigma_1 S(\beta_1 I_1 + \beta_2 I_2)}{(\delta_1+\gamma_1)(\sigma_1+\sigma_2)} \\ I_2^* &= \frac{\sigma_2 S(\beta_1 I_1 + \beta_2 I_2)}{(\delta_2+\gamma_2)(\sigma_1+\sigma_2)} \\ R^* &= \frac{1}{\delta}\left[\frac{\sigma_1 S(\beta_1 I_1 + \beta_2 I_2)}{(\delta_1+\gamma_1)(\sigma_1+\sigma_2)} + \frac{\sigma_2 S(\beta_1 I_1 + \beta_2 I_2)}{(\delta_2+\gamma_2)(\sigma_1+\sigma_2)}\right] \end{aligned} \tag{10}$$

The Jacobian matrix corresponding to model (2) is represented by

$$J = \begin{pmatrix} -(\delta+\beta_1 I_1+\beta_2 I_2) & 0 & -\beta_1 S & -\beta_2 S & 0 \\ (\beta_1 I_1+\beta_1 I_2) & -(\sigma_1+\sigma_2) & \beta_1 S & \beta_2 S & 0 \\ 0 & \sigma_1 & -(\delta_1+\gamma_1) & 0 & 0 \\ 0 & \sigma_2 & 0 & -(\delta_2+\gamma_2) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -\delta \end{pmatrix}$$

3.2.1. Stability at disease-free equilibrium state (E^0)

The Jacobian-matrix (J) at the disease-free equilibrium point $(\frac{\Gamma}{\delta}, 0, 0, 0, 0)$ is given by

$$J(E^0) = \begin{pmatrix} -\delta & 0 & -\Gamma\beta_1\delta^{-1} & -\Gamma\beta_2\delta^{-1} & 0 \\ 0 & -(\sigma_1+\sigma_2) & \Gamma\beta_1\delta^{-1} & \Gamma\beta_2\delta^{-1} & 0 \\ 0 & \sigma_1 & -(\delta_1+\gamma_1) & 0 & 0 \\ 0 & \sigma_2 & 0 & -(\delta_2+\gamma_2) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -\delta \end{pmatrix}$$

and the corresponding characteristic equation yields

$$(-\lambda-\delta)^2(\lambda^3+K_1\lambda^2+K_2\lambda+K_3)=0 \tag{11}$$

where

$$\begin{aligned} K_1 &= (\sigma_1+\sigma_2+\gamma_1+\gamma_2+\delta_1+\delta_2) \\ K_2 &= (\sigma_1+\sigma_2)(\gamma_1+\delta_1)+(\gamma_1+\delta_1)(\gamma_2+\delta_2)+(\sigma_1+\sigma_2)(\gamma_2+\delta_2) \\ &\quad -(\sigma_1\beta_1+\sigma_2\beta_2)\Gamma\delta^{-1} \\ K_3 &= (\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)-\beta_2\sigma_2\Gamma(\gamma_1+\delta_1)\delta^{-1}-\beta_1\sigma_1\Gamma(\gamma_2+\delta_2)\delta^{-1} \end{aligned}$$

It is obvious in the first factor of the above characteristic equation (11) that two eigenvalues λ_1 and λ_2 are always negative for $\delta > 0$. The computation for the remaining three eigenvalues gives a large expression that is very difficult to deal with two complex roots. But the approach used in the present paper is about the sign of the eigenvalues rather than its numerical value to determine the stability of the model at equilibrium points. Therefore, Descarte's rule [45] of number in changing the signs of polynomial coefficients is considered here instead of registering the eigenvalues straightforwardly.The second factor of Eq. (11), is taken into account and is compared to the following equation given with three sign-changes in the coefficient:

$$\lambda^3 - L_1\lambda^2 + L_2\lambda - L_3 = 0$$

where L_1, L_2, and L_3 are positive, and consequently, the remaining roots of the characteristic equation (11) will be negative if K_1, K_2, and K_3 are all positive:

$$\begin{aligned} &K_1: (\sigma_1+\sigma_2+\gamma_1+\gamma_2+\delta_1+\delta_2)>0 \\ &K_2: (\sigma_1+\sigma_2)(\gamma_1+\delta_1)+(\gamma_1+\delta_1)(\gamma_2+\delta_2)+(\sigma_1+\sigma_2)(\gamma_2+\delta_2)>(\sigma_1\beta_1+\sigma_2\beta_2)\Gamma\delta^{-1} \\ &K_3: (\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)>\beta_2\sigma_2\Gamma(\gamma_1+\delta_1)\delta^{-1}+\beta_1\sigma_1\Gamma(\gamma_2+\delta_2)\delta^{-1} \end{aligned}$$

Here, K_1 is positive and rewriting the K_3 in terms of the basic reproduction number R_0, K_3 becomes

$$K_3 : (\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)(1-R_0)$$

which reveals that K_3 will be positive if only if R_0 is less than 1. On the other hand, if $R_0 > 1$, K_3 will be negative which gives at least one positive eigenvalue of the Jacobian matrix at the disease-free equilibrium point, and therefore the system becomes unstable. This requirement leads to conclude the stability of the model (2) at DFE as follows:

Lemma 2. *The disease-free equilibrium of the system* (2) *is locally asymptotically stable if* K_2, $K_3 > 0$ *and* $R_0 < 1$. *Otherwise, the system is unstable.*

3.2.2. Stability at endemic equilibrium state (E*)

Asymptomatic but infectious individuals always play a major role in spreading the disease across the community. The endemic equilibrium state of model (2) is stable when $R_0 > 1$ that has been shown in this section.

Since the computation of the eigenvalues of the Jacobian-matrix (J) at endemic equilibrium point $(S^*, E^*, I_1^*, I_2^*, R^*)$ is very tedious, the sign of the eigenvalues is checked by applying the Descarte's rule [45] instead, in a similar way presented in the previous section.The characteristic equation of the Jacobian matrix at $(S^*, E^*, I_1^*, I_2^*, R^*)$ is written as

$$(-\lambda-\delta)(\lambda^4+P_1\lambda^3+P_2\lambda^2+P_3\lambda+P_4)=0 \tag{12}$$

where

$$\begin{aligned} P_1 &= (\sigma_1+\sigma_2+\gamma_1+\gamma_2+\delta_1+\delta_2+\delta+\beta_1 I_1^*+\beta_2 I_2^*) \\ P_2 &= (\gamma_2+\delta_2)(\delta+\sigma_1+\sigma_2+\gamma_1+\delta_1+\beta_1 I_1^*+\beta_2 I_2^*)+(\sigma_1+\sigma_2)(\delta+\beta_1 I_1^*+\beta_2 I_2^*) \\ &\quad +(\gamma_1+\delta_1)(\delta+\sigma_1+\sigma_2+\beta_1 I_1^*+\beta_2 I_2^*)-\beta_1\sigma_1 S^*-\beta_2\sigma_2 S^* \\ P_3 &= (\sigma_1+\sigma_2)(\gamma_2+\delta_2)(\delta+\gamma_1+\delta_1+\beta_1 I_1^*+\beta_2 I_2^*)+(\delta+\beta_1 I_1^*+\beta_2 I_2^*) \\ &\quad \{(\gamma_1+\delta_1)(\gamma_2+\delta_2)+(\sigma_1+\sigma_2)(\gamma_1+\delta_1)\}-\beta_1\sigma_1 S^*(\delta+\gamma_2+\delta_2) \\ &\quad -\beta_2\sigma_2 S^*(\delta+\gamma_1+\delta_1) \\ P_4 &= (\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)(\delta+\beta_1 I_1^*+\beta_2 I_2^*)-\beta_1\delta\sigma_1 S^*(\gamma_2+\delta_2) \\ &\quad -\beta_2\delta\sigma_2 S^*(\gamma_1+\delta_1) \end{aligned}$$

The P_1 is seen as positive and it is clear from the characteristic equation (12) that all of the eigenvalues are negative if P_2, P_3 and P_4 are all positive. In such a case, rewriting the P_4,

$$\begin{aligned} P_4 &= \delta(\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)\left(1-\frac{\beta_1\sigma_1 S^*(\gamma_2+\delta_2)+\beta_2\sigma_2 S^*(\gamma_1+\delta_1)}{(\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)}\right) \\ &\quad +(\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)(\beta_1 I_1^*+\beta_2 I_2^*) \\ &= \delta(\sigma_1+\sigma_2)(\gamma_1+\delta_1)(\gamma_2+\delta_2)\left(1-R_0+(\beta_1 I_1^*+\beta_2 I_2^*)\delta^{-1}\right) \end{aligned}$$

where P_4 is positive if

$$1-R_0+(\beta_1 I_1^*+\beta_2 I_2^*)\delta^{-1}>0$$

equivalently,

$$(\beta_1 I_1^*+\beta_2 I_2^*)\delta^{-1} > R_0-1$$

The left side of the above inequality can never be negative according to a realistic epidemiological model at the endemic equilibrium state. Therefore, for any positive quantity on the left side, this inequality holds only if $R_0 > 1$ which guarantees the negative eigenvalue. On the contrary, $R_0 < 1$ may give a negative value on the left side of above inequality which is not feasible in an epidemiological sense. In such a case, P_4 can be negative giving at least one positive eigenvalue at that point, and the system becomes unstable. Thus, the stability of the model (2) at EEP is concluded as follows:

Lemma 3. *The endemic equilibrium of the system* (2) *is locally asymptotically stable for* P_2, $P_3 > 0$ *and* $R_0 > 1$. *Otherwise, the system is unstable.*

4. Sensitivity analysis

A sensitivity analysis is usually studied to determine the effect of the parameters on the COVID-19 outbreak described by the model. In particular, the most sensitive parameters need to be identified that cause a perturbation in the dynamics of the model due to a small change in numeric value. In the study of sensitivity analysis, the derivatives refer to the rate of change in prediction with respect to the parameters and the normalized elasticity index of a variable is defined as the ratio of the apparent change in the variable to that in respective parameter [46]. Also, for a differentiable function, the elasticity index [46] may be

Table 1
List of parameters for numerical investigation.

Parameters	Values		Reference	Remarks
Γ	4.21×10^{-5}		Bugalia et al. [28]	$1/(65 \times 365)$
β_1	0.251 6	($\beta_1 < \beta_2$)	…	Assumed
β_2	0.311 0		Worldometer [47]	
σ_1	0.151 6	($\sigma_1 < \sigma_2$)	…	Assumed
σ_2	0.181 8		Worldometer [47,48]	
γ_1	0.07	($\gamma_1 > \gamma_2$)	…	Assumed
γ_2	0.03		…	Assumed
δ_1	3.5×10^{-3}	($\delta_1 < \delta_2$)	…	Assumed
δ_2	9.7×10^{-3}		Worldometer [47]	
δ	4.21×10^{-5}		Bugalia et al. [28]	$1/(65 \times 365)$

defined in terms of the partial derivative. This kind of analysis assists to establish the control strategies for the COVID-19 outbreak.

The sensitivity index of R_0 at DFE is calculated with the parameters Γ, δ, β_i, σ_i, γ_i, δ_i, ($i = 1, 2$) which yields:

$$\frac{\partial R_0}{\partial \Gamma} = \frac{\sigma_1\beta_1}{\delta(\delta_1+\gamma_1)(\sigma_1+\sigma_2)} + \frac{\sigma_2\beta_2}{\delta(\delta_2+\gamma_2)(\sigma_1+\sigma_2)}$$

$$\frac{\partial R_0}{\partial \beta_1} = \frac{\sigma_1\Gamma}{\delta(\delta_1+\gamma_1)(\sigma_1+\sigma_2)}$$

$$\frac{\partial R_0}{\partial \beta_2} = \frac{\sigma_2\Gamma}{\delta(\delta_2+\gamma_2)(\sigma_1+\sigma_2)}$$

$$\frac{\partial R_0}{\partial \sigma_1} = \left(\frac{\sigma_2\Gamma(\beta_1(\delta_2+\gamma_2)-\beta_2(\delta_1+\gamma_1))}{\delta(\delta_1+\gamma_1)(\delta_2+\gamma_2)(\sigma_1+\sigma_2)^2}\right)$$

$$\frac{\partial R_0}{\partial \sigma_2} = \left(\frac{\sigma_1\Gamma(\beta_2(\delta_1+\gamma_1)-\beta_1(\delta_2+\gamma_2))}{\delta(\delta_1+\gamma_1)(\delta_2+\gamma_2)(\sigma_1+\sigma_2)^2}\right)$$

$$\frac{\partial R_0}{\partial \delta} = -\left(\frac{\beta_1\Gamma\sigma_1}{\delta^2(\delta_1+\gamma_1)(\sigma_1+\sigma_2)^2} + \frac{\beta_2\Gamma\sigma_2}{\delta^2(\delta_2+\gamma_2)(\sigma_1+\sigma_2)^2}\right)$$

$$\frac{\partial R_0}{\partial \delta_1} = \frac{\partial R_0}{\partial \gamma_1} = -\frac{\beta_1\Gamma\sigma_1}{\delta(\delta_1+\gamma_1)^2(\sigma_1+\sigma_2)^2}$$

$$\frac{\partial R_0}{\partial \delta_2} = \frac{\partial R_0}{\partial \gamma_2} = -\frac{\beta_1\Gamma\sigma_1}{\delta(\delta_2+\gamma_2)^2(\sigma_1+\sigma_2)^2}$$

The positive result of the first three partial derivatives indicates that the increase in the value of the parameters Γ, β_1, β_2, increases the basic reproductive number R_0, and the sign of the term $\beta_1(\delta_2+\gamma_2) - \beta_2(\delta_1+\gamma_1)$ determines which of the 4th and 5th partials shows positive sensitivity to R_0. Based on the model assumptions and the parameters used in Table 1, the 5th partial derivative is computed positive and the normalized sensitivity indices corresponding to these parameters are estimated as follows:

$$\Theta_\Gamma = \frac{\Gamma}{R_0}\frac{\partial R_0}{\partial \Gamma} = 1$$

$$\Theta_{\beta_1} = \frac{\beta_1}{R_0}\frac{\partial R_0}{\partial \beta_1} = \frac{\beta_1\sigma_1(\delta_2+\gamma_2)}{\beta_1\sigma_1(\delta_2+\gamma_2)+\beta_2\sigma_2(\delta_1+\gamma_1)}$$

$$\Theta_{\beta_2} = \frac{\beta_2}{R_0}\frac{\partial R_0}{\partial \beta_2} = \frac{\beta_2\sigma_2(\delta_1+\gamma_1)}{\beta_1\sigma_1(\delta_2+\gamma_2)+\beta_2\sigma_2(\delta_1+\gamma_1)}$$

$$\Theta_{\sigma_2} = \frac{\sigma_2}{R_0}\frac{\partial R_0}{\partial \sigma_2} = \frac{\sigma_1\sigma_2\Gamma(\beta_2(\delta_1+\gamma_1)-\beta_1(\delta_2+\gamma_2))}{(\sigma_1+\sigma_2)}$$

The sensitivity index can be constant here depending on several parameters of the system or can be free of any independent parameters.

5. Numerical investigation and discussion

The numerical investigation is very useful in setting COVID-19 management strategies for a certain region. Therefore, the numerical simulation of the present model (2) was carried out to support the analytical result using the value of the parameters as tabulated in Table 1. Based on the value of the parameters, the sensitivity analysis was conducted by computing the elasticity indices to investigate the parameter effect on R_0. The propensity of the total infection ($I_1 + I_2$) curve corresponding to the sensitive elastic indices was also assessed.

The following elasticity indices are obtained by using the values of the corresponding parameters:

$$\Theta_\Gamma = 1,\ \Theta_{\beta_1} = 0.267,\ \Theta_{\beta_2} = 0.733,\ \Theta_{\sigma_2} = 6.2 \times 10^{-7}$$

The recruitment rate Γ was found as the most sensitive parameter while the transmission rates (β_1 and β_2) showed sensitivity in every situation satisfying the condition $\beta_2(\delta_1+\gamma_1) > \beta_1(\delta_2+\gamma_2)$ throughout the model. A similar result was also found in the study of Bugalia et al. [28]. From the computed elasticity indices, it can be stated that a 10%

Table 2
Loop iterations corresponding to the sensitive parameters.

Parameters	Loop iteration		Type of effect
	Total Infection Plot ($1 \le j \le 5$)	Reproduction Number Plot ($1 \le j \le 10$)	
β_1	β_1 + j*0.05	β_1 + j*0.001	Individual
β_2	β_2 + j*0.05	β_2 + j*0.001	Individual
σ_2	σ_2 + j*0.05	σ_2 + j*0.001	Individual
Γ	Γ + j*0.001	Γ + j*0.000 001	Individual
β_1, Γ	β_1 + j*0.005, Γ + j*0.001	β_1 + j*0.005, Γ + j*0.000 001	Combined effect of β_1 and Γ
β_2, Γ	β_2 + j*0.005, Γ + j*0.001	β_2 + j*0.005, Γ + j*0.000 001	Combined effect of β_1 and Γ

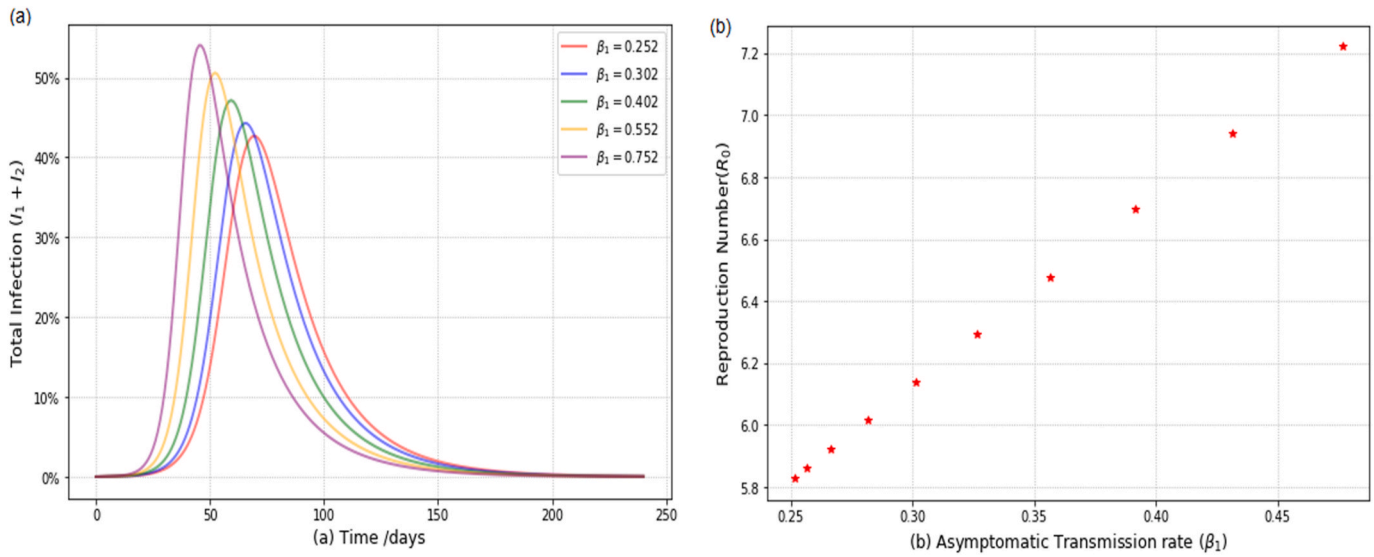

Fig. 3. (a) Variation in total infection ($I_1 + I_2$) and (b) reproduction number (R_0) with respect to asymptomatic transmission rate (β_1).

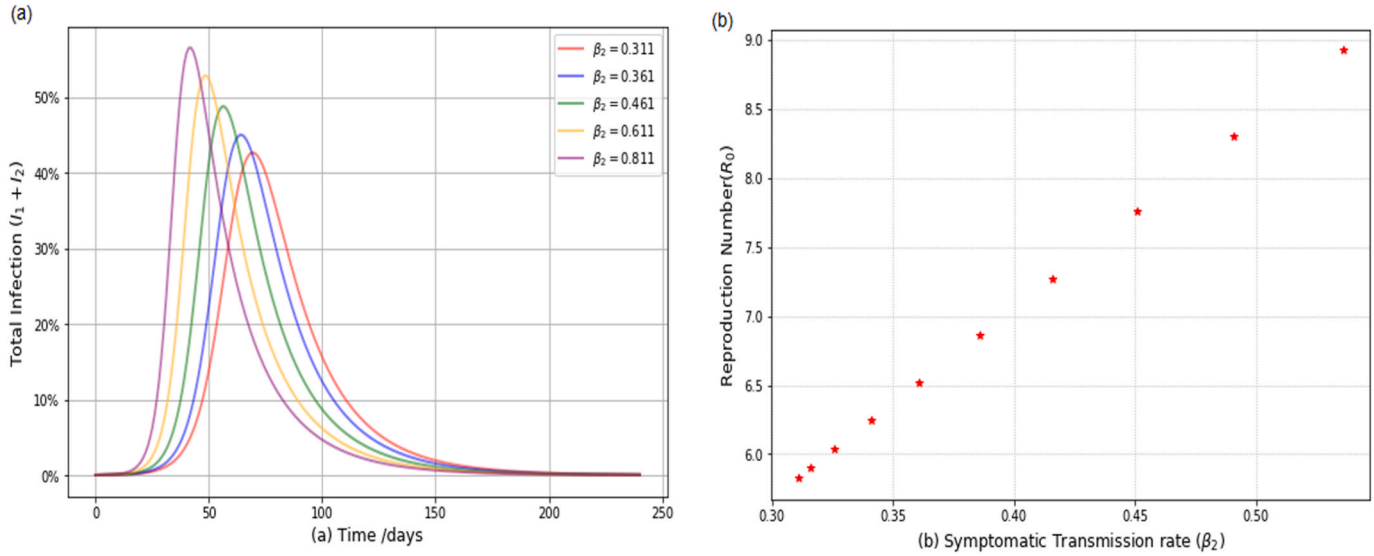

Fig. 4. (a) Variation in total infection ($I_1 + I_2$) and (b) reproduction number (R_0) with respect to symptomatic transmission rate (β_2).

increase in the recruitment rate Γ, asymptomatic transmission rate β_1, and symptomatic transmission rate β_2 cause to increase the value of R_0 by 10%, 2.4%, and 7.6% respectively, and can lead to an outbreak subsequently. However, the symptomatic infection rate (σ_2) does not affect R_0 significantly.

The non-linear ordinary differential equation (ODE) model developed in the present paper was numerically solved using Python (version 3.7) **scipy.integrate** package and **odeint** library function with the initial conditions. The initial conditions (E_0, I_{10}, I_{20}, R_0) = (5/20 000, 0, 0, 0), $S_0 = 1 - E_0 - I_{10} - I_{20} - R_0$ with $N = 1$, were considered to simulate the model. The loop iterations used to simulate the model (2) are summarized in Table 2.

To assess the COVID-19 outbreak pattern, the simulation comprised different values of a sensitive parameter in generating the graphs of total infection and basic reproduction number (R_0) while the other parameters remained constant. The graphs were presented in a panel where the left one shows the total infection trend and the right one displays the basic reproduction number profile with respect to a particular sensitive parameter. The R_0 increases steadily with the increase of β_1 as shown in Fig. 3. After a certain value of β_1, R_0 crossed the threshold 1 which specifies that disease-free equilibrium is stable up to a certain value of β_1 and becomes unstable beyond that value. Also, the total number of infected people gets higher as β_1 increases with time and the number of the highest infected people in each iteration grows exponentially.

In a study of quantifying asymptomatic transmissible cases along with the symptomatic transmission, Subramanian et al. [49] found that asymptomatic patients contribute significantly to the overall infection force leading to a substantial increase in the basic reproduction number which agrees with our findings (Fig. 3). The authors demonstrated that even when the basic reproductive number concerning symptomatic transmission is high, pre-symptomatic and asymptomatic infections together account for at least half of the overall infection force [49]. These results are consistent with model (2) findings that the proportion of the effect of asymptomatic and symptomatic transmission rates on R_0 is between 1 and 3. Figs. 3 and 4 also ensure that asymptomatic individuals play an important role in spreading COVID-19 disease like symptomatic individuals. As asymptomatic transmissions are not taken into account in many Covid-19 models, the basic reproduction number

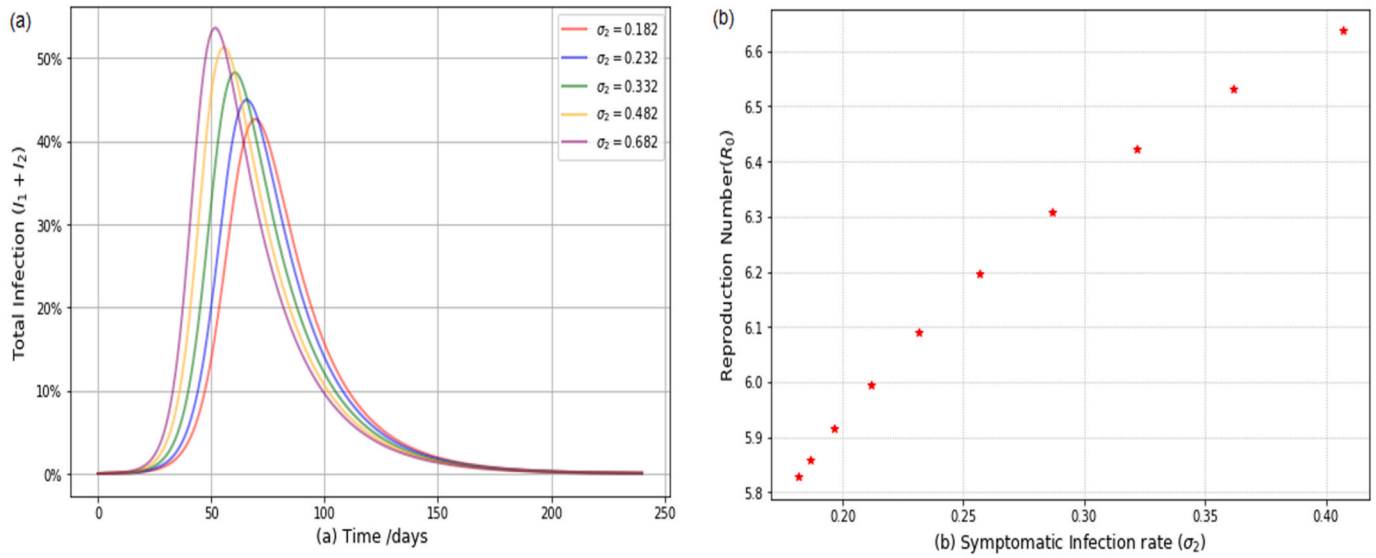

Fig. 5. (a) Variation in total infection ($I_1 + I_2$) and (b) reproduction number (R_0) with respect to symptomatic infection rate (σ_2).

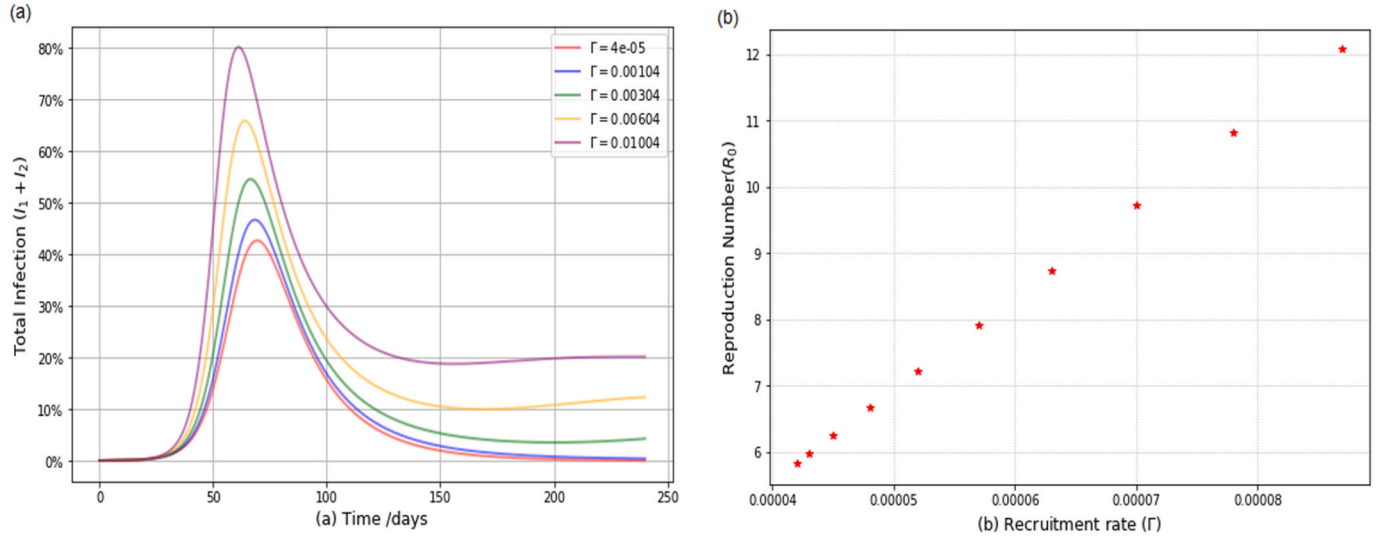

Fig. 6. (a) Variation in total infection ($I_1 + I_2$) and (b) reproduction number (R_0) with respect to recruitment rate (Γ).

R_0 may be underestimated resulting in a misleading prediction. Therefore, community-wide interventions taking into account the asymptomatic transmission are crucial for controlling the outbreak. Consequently, identifying the group of asymptomatic infectious people through a large-scale screening and taking different effective measures like wearing a mask, using sanitizer, and maintaining social distance will result in controlling in the outbreak.

The symptomatic transmission rate β_2 is the second most sensitive factor affecting the outbreak as found through the sensitivity analysis. The parameter β_2 has an almost similar impact on R_0 like β_1 shown in Fig. 3. However, β_2 causes a rapid and higher increase in the total infection graph compared to β_1. As seen in the figure, approximately 58% of the total population become infected on day 40 (approx.) for β_2 while the same for β_1 is about 52% percent on the day of 48 (approx). As a result, maintaining self-isolation and quarantine would be the most useful measures to control the COVID-19 disease which is spread by symptomatic transmission.

The role of the symptomatic infection rate σ_2 on the total infection and R_0 is displayed in Fig. 5. As mentioned earlier, the parameter σ_2 shows less sensitivity to the basic reproduction number R_0. However, it noticeably affects the COVID-19 outbreak as seen in Fig. 5.

The influence of the recruitment rate Γ on the COVID-19 infection is illustrated in Fig. 6. As seen in the figure, for a very small increase in Γ, the number of total infected people increases rapidly and the growth in R_0 is very significant. The basic reproduction number R_0 peaked from 5.8 to 12 for a very small change in Γ from 0.000 04 to 0.000 08 which indicates that the recruitment rate to an area during the spread of COVID-19 highly affects the outbreak. Thus imposing a bar on population migration from one place to another place would be the best effective measure in controlling the outbreak. Setting a management plan controlling Γ to keep R_0 below 1, should be the first strategic action policy.

Model (2) was further simulated to investigate the combined effect in the simultaneous variation of the parameters of recruitment (Γ) and

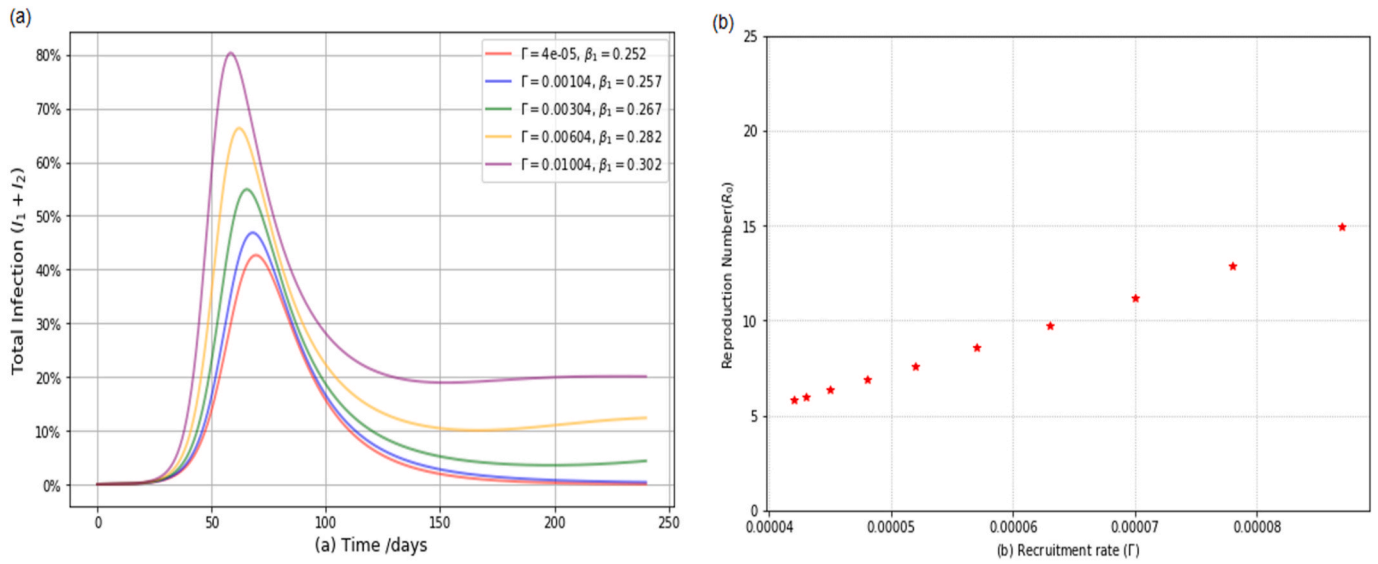

Fig. 7. (a) Variation in total infection ($I_1 + I_2$) and (b) reproduction number (R_0) with respect to recruitment rate (Γ) and asymptomatic transmission rate (β_1) simultaneously.

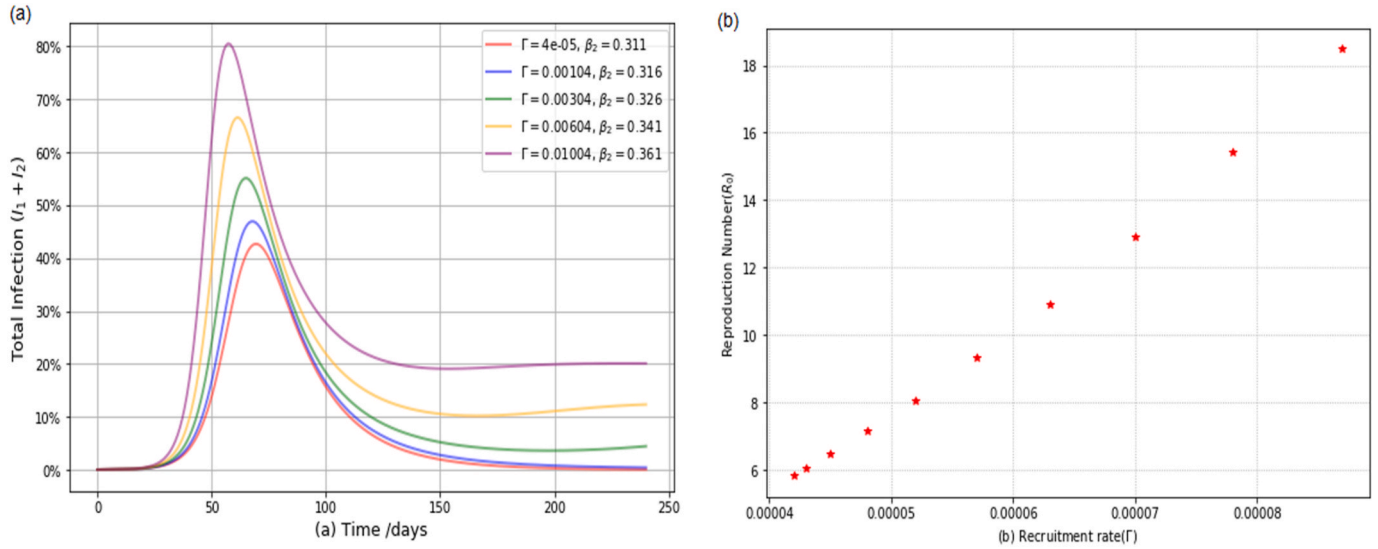

Fig. 8. (a) Variation in total infection ($I_1 + I_2$) and (b) reproduction number (R_0) with respect to recruitment rate (Γ) and symptomatic transmission rate (β_2) simultaneously.

transmission rates β_1, β_2. In each loop iteration, equal increase in the parameters values (β_1, Γ) and (β_2, Γ) keeping others constant, gives almost similar graphs as seen in Figs. 7 and 8. In both cases, a rapid rise in total infection curves indicates that approximately 80% percent of the total population become infected and remain nearly constant after a rapid fall. But, R_0 increased at a higher rate in Fig. 8 compared to Fig. 7 which revealed simultaneous changes in the symptomatic transmission and recruitment rate causes severe endemic.

The numerical results in the present model (2) indicate that asymptomatic infections contribute significantly to community transmission when combined with symptomatic cases. According to the study [50], symptomatic and asymptomatic patients may have similar viral counts, and a high viral load does not always suggest a high transmissibility. Due to the uncertainty surrounding individual disease transmission rates, the epidemic may spread faster than the existing available models predict if asymptomatic people spread at the same rate as symptomatic infections. In this decision analysis, the plausible model assumptions projected that asymptomatic persons would account for one-third of the overall transmission. The outcomes of this study imply that simply identifying and isolating people with symptomatic COVID-19 will not be sufficient to prevent SARS-CoV-2 from spreading. In addition, imposing restrictions on the movements of asymptomatic individuals together with the non-pharmaceutical interventions would be a good measure. To solve the non-identifiability of asymptomatic transmission's relative strength and slow down the outbreak's progression, a comprehensive vaccination program, community testing, and asymptomatic case contact tracing would be the most effective measures.

6. Conclusion

Coronavirus (COVID-19) is a highly infectious pandemic disease with interhuman transmission, is the greatest challenge for the whole world. There is not enough viable information and specific treatment on the coronavirus disease that would help in disease management and in setting a control plan to minimize the disease spread. A mathematical model was proposed in the present study by considering asymptomatic and symptomatic transmission pathways. The model dynamics were also analysed throughout the solution space. The results demonstrated that the asymptomatic transmission rate significantly affects R_0, and the recruitment rate rules the illness flare-up the most. Through the numerical simulation and analytical simplification, it was found that disease-free equilibrium is stable for $R_0 < 1$, up to a certain asymptomatic transmission rate, and becomes unstable when higher than that. The proportion of the effect of asymptomatic transmission and symptomatic transmission rate on R_0 was found to be approximately between 1 and 3 and so the present model would provide more accurate predictions on SARS-CoV-2 disease spread than the other models. The model also suggested that, the overall basic reproduction number could be higher than often assumed depending on how well asymptomatic cases might transmit virus particles. Therefore, the COVID-19 endemic can considerably be minimised by preventing the migration of people and strictly maintaining personal measures in both symptomatic and asymptomatic individuals simultaneously. Consideration of viral load in an asymptomatic and symptomatic group of people in this model would provide a more specific illustration of transmission dynamics and would be a good fit for future research.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

The authors are greatful to University of Chittagong, Bangladesh for their supports in carrying out this study.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.compbiomed.2022.105264.

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