Chapter 3
Modeling the Spread of COVID-19 Among Doctors from the Asymptomatic Individuals

M. H. A. Biswas, A. K. Paul, M. S. Khatun, S. Mandal, S. Akter, M. A. Islam, M. R. Khatun, and S. A. Samad

Abstract The present world is in dire straits due to the deadly SARS coronavirus-2 (CoV-2) outbreak, and the experts are trying heart and soul to discover any prevention and/or remedy. The people from all walks of life in the universe are fighting to defeat this novel coronavirus. In this case, doctors are in the front line fighters who have put themselves at a risk. In this paper, we have formulated a non-linear system of five differential equations of COVID-19 based on the tendency of doctors to be infected. The target of this study is to take a look at the transmission of COVID-19 from asymptomatic populations to the doctors. The model is analyzed with the determination of the basic reproduction number, equilibrium, and related stability analysis at both equilibrium points. The graph of the basic reproductive ratio for different parameters has been drawn to show the disease behavior. Finally, we have simulated our model numerically for visualizing the analytical findings. Our study shows that the asymptomatic population increases as the disease (COVID-19) transmission rate

M. H. A. Biswas (✉) · A. K. Paul · M. S. Khatun · M. A. Islam · M. R. Khatun · S. A. Samad
Mathematics Discipline, Khulna University, Khulna, Bangladesh
e-mail: mhabiswas@yahoo.com

A. K. Paul
e-mail: arindam017@gmail.com

M. S. Khatun
e-mail: shantajsku@gmail.com

M. A. Islam
e-mail: anarul1996@gmail.com

M. R. Khatun
e-mail: reshma161201@gmail.com

S. A. Samad
e-mail: abdussamadku41@gmail.com

S. Mandal · S. Akter
Department of Mathematics, Bangabandhu Sheikh Mujibur Rahman Science and Technology University, Gopalganj, Bangladesh
e-mail: sajibmandal1997@gmail.com

S. Akter
e-mail: soniaaa053@gmail.com

N. H. Shah and M. Mittal (eds.), *Mathematical Analysis for Transmission of COVID-19*, Mathematical Engineering, https://doi.org/10.1007/978-981-33-6264-2_3

increases. The number of infected population increases with the infection rate. These increasing asymptomatic and infected populations lead the doctors to get infected by contacting with them. Thus, the whole medical service system is getting down over time.

Keywords SARS CoV-2/COVID-19 · Doctors · Mathematical model · Basic reproductive ratio · Numerical simulation

MSC 2020 Primary: 92Bxx · 37M05 · 34D20 · 92D30 · Secondary: 34A34

Introduction

This century is experiencing one of the most devastating diseases called COVID-19. The cause of this disease is severe acute respiratory syndrome coronavirus-2 (SARS CoV-2), a newly immerged virus in 2019. COVID-19 is a highly infectious disease that has already spread irrespective of developed, developing, and under-developing countries across the globe. It has become a global health hazard because still now there is no specific medicine or drugs for remedy or even no other prevention like a vaccine that can prevent people from being infected by this disease. The curve of incidence rate and the death rate from coronavirus disease (COVID-19) is becoming skyrocketed day by day. In order to overcome this fatality, our scientists, researchers, government employers, politicians, defenses, experimentalists, intellectuals, health workers are working hand in hand. However, doctors are trying their utmost to rescue people in almost every way. These people are most vulnerable to infection by this disease as they are directly involved in the treatment of COVID-19 patients. Already an alarming number of doctors have died from this fatal disease. And, the death of consultant doctors is an irreversible loss. Because it takes a long time to be a consultant doctor. By this time in the United States, approximately, 600 health workers have died of novel coronavirus disease at the time of performing their duty [1]. In Bangladesh, the coronavirus infection rate among health workers is very high in comparison to other countries in the world. At present, there are about 3301 health workers living with coronavirus infections in Bangladesh, among which 1040 doctors and about 50 doctors have been died by this fatal disease by this time [2]. In the case of doctors' death rate of COVID-19, United States is in the first position followed by Russia (545), UK (540), Brazil (351), Mexico (248), Italy (188), Egypt (111), Iran (91), Ecuador (82), and Spain (63) [3].

Nowadays, mathematical modeling is playing an incredible role in providing quantitative insight into this type of mysterious infectious disease dynamics and maintenance. It has already contributed to a better understanding of the mechanisms of various critical phenomena and has gotten increasing attention because modeling and simulation allow for rapid, cost-effective, and illuminating assessment. In COVID-19, mathematical modeling can be used to better predictions, management, and control strategies.

Our previous work focused on the control policies where we showed that social distancing, educational campaign, and treatment are efficacious for controlling the spread of this horrible disease [4]. Many mathematical models of COVID-19 have been studied as well. Sheffield et al. [5] combined an epidemic model with a cost associated model with a lockdown in comparison to intermittent and moderate lockdown strategies of COVID-19. Bairagi et al. [6] presented an analytical model in order to control the outbreak of COVID-19 by augmenting isolation and social distancing features of populations. Chen and Yu [7] studied a second derivative mathematical model of COVID-19 in order to characterize it in China during the first two months of the outbreak. Chen et al. [8] worked on a Bats-Hosts-Reservoir-People transmission model where they simulated the potential transmission from the infection source to the human infection. Depending on the outbreak of novel coronavirus infections in Thailand, Sookaromdee and Wiwanitkit [9] developed and analyzed a mathematical model of this disease. Zhao et al. [10] proposed a model where they estimated the basic reproductive ratio of COVID-19 on the early detection of its outbreak in China. Ndairoua [11] investigated a compartmental model of COVID-19 by focusing on the transmissibility of super-spreaders populations. We refer Aguilar et al. [12], Carolina et al. [13], Casas-Rojo et al. [14], Chitnis et al. [15], Chen et al. [16], Huo and Feng [17], Kabir et al. [18], Queen Elizabeth Hospital Birmingham COVID-19 airway team [19], Magalhaesa et al. [20], Melliani et al. [21], Nadeem [22], Prompetchara et al. [23], Safi and Garba [24], Zhao et al. [10], Xu et al. [25], Zhang et al. [26], Zhang et al. [27], Biswas [4, 28–34], Khatun and Biswas [35–37] and all other references inside for gathering effective knowledge about this highly infectious disease and a very recent study of modeling and control methods.

The novel coronavirus disease (COVID-19) is a world pandemic. People from all walks of life are taking steps to defend the COVID-19 epidemic but there is no fruitful solution to this disease yet. As per our view, there is no work that highlights on a mathematical model of COVID-19 based on the tendency of doctors to be infected as a result, the medical service system is getting down over the time. Doctors are assets and front line fighters during this tiring situation but most of the countries in the world do not have adequate doctor as many as general people. In that case, the government of those countries has to hire additional doctors and health experts to serve COVID-19 patients. The present paper deals with the five compartmental model: susceptible populations, asymptomatic populations, infected populations, doctor populations, and recovered populations. The novelty of this study is to develop a mathematical model of COVID-19 in terms of a set of non-linear ordinary differential equations showing that doctors are affecting more frequently at the time of serving coronavirus infected patients. Thus, the medical service system has shuttered for the time being.

Mathematical Model

In our study, we formulate a five compartmental model of coronavirus disease (COVID-19) by showing how frequently doctors are affecting, and thus, the medical service system is getting down over time. Let the compartments are susceptible $S(t)$, asymptomatic $A_s(t)$, infected $I(t)$, doctors $D_r(t)$, and recovered $R(t)$. Here, the first compartment $S(t)$ denotes the susceptible populations and the second compartment $A_s(t)$ is for asymptomatic populations who are actually infected but have no symptoms of COVID-19. The third compartment $I(t)$ denotes the infected populations as those who are actually infected and identified. The fourth compartment $D_r(t)$ denotes the doctor individuals who are serving COVID-19 patients. For that reason, they come to close contact with COVID-19 patients. For this, some doctors are being infected at the time of performing the duty. After being infected, they become unable to provide treatment. While contacting COVID-19 patients, doctors are being infected by a rate of ξ. Since there is a non-linear relationship, we can say that if doctors decrease, then more infected people will die. And if the infected people come into contact with susceptible people, the susceptible people can be infected soon at a rate r. Another portion of susceptible populations get infected coming into contact with infected people but they do not show any symptoms of COVID-19. This phenomenon is denoted by σ. For that reason, they can easily contact susceptible people and lead them to become infected faster at a rate β. When the country demands more doctors for serving the COVID-19 patients, the government starts recruiting the doctors by the rate of μ from susceptible populations. Some infected populations are dying at a rate θ, and another part is being recovered at the rate δ after getting proper treatment. μ_0 denotes the natural death of populations. $\xi\gamma$ represents the infection rate of doctors at the time of serving the patients. $(1-\xi)\gamma$ indicates the recovery rate of COVID-19 patients for undertaking treatment at the hospital. The interacting transmissions among different compartments are shown in Fig. 3.1.

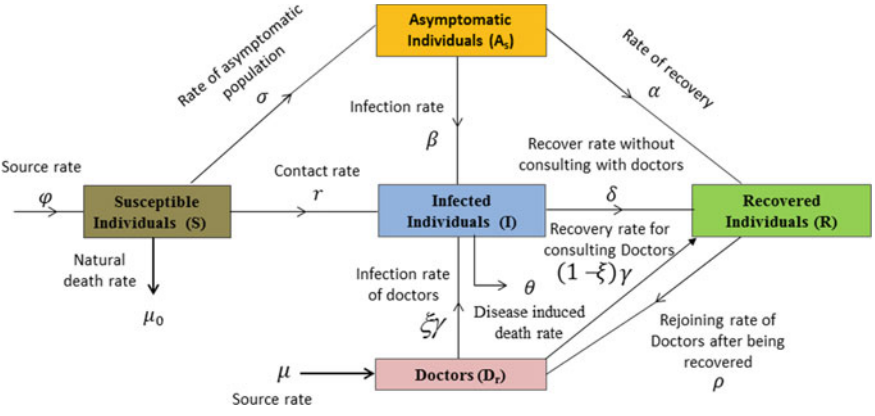

Fig. 3.1 The schematic diagram of the compartmental model of COVID-19

Table 3.1 Parameter specifications of COVID-19 model (3.1)

Symbol	Meaning	Values
r	Contact rate of susceptible and infected populations	0.002
ϕ	Source rate to susceptible populations	5,000,000
γ	Rate of infected cases while consulting with doctors	0.06
θ	Death rate from COVID-19	0.02
δ	Recover rate without consulting with doctors	0.05
ξ	Infection rate of doctors for contacting with COVID-19 patients	0.00006
ρ	Rejoining rate of doctors after getting recovered	0.00005
β	Infection rate of infected populations	0.005
μ	Source rate of doctors	150
σ	Disease transmission rate of asymptomatic individuals for contacting an infected and susceptible population	0.003
α	Recovery rate of asymptomatic populations	0.03

Source Estimated from [1–3]

Now taking all the situations in Fig. 3.1 into consideration, the proposed model can be formulated by the following set of non-linear ODE:

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \varphi - \mu_0 S(t) - rI(t)S(t) - \sigma I(t)S(t) \\ \dfrac{\mathrm{d}A_s(t)}{\mathrm{d}t} = \sigma I(t)S(t) - \beta A_s(t)S(t) - \alpha A_s(t) \\ \dfrac{\mathrm{d}I(t)}{\mathrm{d}t} = \beta A_s(t)S(t) - \theta I(t) - \delta I(t) + rI(t)S(t) + \gamma\xi D_r(t)I(t) \\ \dfrac{\mathrm{d}D_r(t)}{\mathrm{d}t} = \mu + \rho R(t) - \gamma D_r(t)I(t) \\ \dfrac{\mathrm{d}R(t)}{\mathrm{d}t} = \alpha A_s(t) + \delta I(t) - \rho R(t) + \gamma D_r(t)I(t)(\xi - 1) \end{cases} \tag{3.1}$$

with boundary conditions

$$S(0) = S_0 > 0, A_s(0) = A_{s0} \geq 0, I(0) = I_0 \geq 0, D_r(0) = D_{r0} \geq 0, R(0) = R_0 \geq 0$$

The brief description of the parameters used in the model are shown in Table 3.1.

Analysis of the Compartmental Model

We have done the positivity analysis of the solutions of the model in order to show the validation and well-posedness of this model. Further, we have calculated different

equilibria, basic reproduction ratio and have analyzed the stability at two different equilibrium points.

Positivity Analysis

Lemma 1 *If* $S(t) > 0$, $A_s(t) \geq 0$, $I(t) \geq 0$, $D_r(t) \geq 0$ *and* $R(t) \geq 0$ *then the solutions* $S(t)$, $A_s(t)$, $I(t)$, $D_r(t)$ *and* $R(t)$ *of the model* (3.1) *are non-negative.*

Proof In order to show the proof of Lemma 1, we use the set of equations (3.1).

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = \varphi - \mu_0 S(t) - rI(t)S(t) - \sigma I(t)S(t) \tag{3.2}$$

To seek positivity, we can write

$$\begin{aligned} &\frac{\mathrm{d}S(t)}{\mathrm{d}t} \geq \varphi - \mu_0 S(t) \\ &\Rightarrow \frac{\mathrm{d}S(t)}{\mathrm{d}t} + \mu_0 S(t) \geq \varphi \end{aligned} \tag{3.3}$$

The integrating factor of (3.3) is given by

$$\therefore I.F. = \mathrm{e}^{\int \mu_0 \mathrm{d}t} = \mathrm{e}^{\mu_0 t}$$

Multiplying $\mathrm{e}^{\mu_0 t}$ on both sides of (3.3), we get

$$\frac{\mathrm{d}}{\mathrm{d}t}\left(\mathrm{e}^{\mu_0 t} S(t)\right) \geq \varphi \mathrm{e}^{\mu_0 t} \tag{3.4}$$

Now, by integrating (3.4), we have

$$S(t) \geq \frac{\varphi}{\mu_0} + c\mathrm{e}^{-\mu_0 t} \tag{3.5}$$

where c is an integrating constant.

Considering the initial value at $t = 0$, $S(t) \geq S(0)$.

From (3.5), we attain

$$S(0) \geq \frac{\varphi}{\mu_0} + c \Rightarrow S(0) - \frac{\varphi}{\mu_0} \geq c$$

Substituting the value of c into (3.5), we obtain

$$S(t) \geq \frac{\varphi}{\mu_0} + \left(S(0) - \frac{\varphi}{\mu_0}\right)\mathrm{e}^{-\mu_0 t} \tag{3.6}$$

So, at $t = 0$ and $t \to \infty$, $S(t) > 0$. By repeating the above procedure, we can prove the positivity of all other state variables.

Consequently, it is clear that $\forall\, t \geq 0$.

$$S(t) > 0, A_s(t) \geq 0, I(t) \geq 0, D_r(t) \geq 0.$$

Thus, Lemma 1 is undoubtedly proven.

Equilibrium Points

Let, $E_{dfe}(S^*, A_s^*, I^*, D_r^*, R^*)$ be the disease-free equilibrium point of the model (3.1). In order to find the disease-free equilibrium point, we need to solve $\frac{dS^*}{dt} = \frac{dA_s^*}{dt} = \frac{dI^*}{dt} = \frac{dD_r^*}{dt} = \frac{dR^*}{dt} = 0$ of the model (3.1). At the disease-free equilibrium point $E_{dfe}(S^*, A_s^*, I^*, D_r^*, R^*)$, the model (3.1) takes the following form:

$$\begin{aligned}
&\varphi - \mu_0 S^*(t) - r I^*(t) S^*(t) - \sigma I^*(t) S^*(t) = 0\\
&\sigma I^*(t) S^*(t) - \beta A_s^*(t) S^*(t) - \alpha A_s^*(t) = 0\\
&\beta A_s^*(t) S^*(t) - \theta I^*(t) - \delta I^*(t) + r I^*(t) S^*(t) + \gamma \xi D_r^*(t) I^*(t) = 0\\
&\mu + \rho R^*(t) - \gamma D_r^*(t) I^*(t) = 0\\
&\alpha A_s^*(t) + \delta I^*(t) - \rho R^*(t) + \gamma D_r^*(t) I^*(t)(\xi - 1) = 0
\end{aligned} \tag{3.7}$$

Since infection not found at the $E_{dfe}(S^*, A_s^*, I^*, D_r^*, R^*)$ (i.e., $A_s^*(t) = 0,\ I^*(t) = 0,\ R^*(t) = 0$), from (3.7), we have

$$S^*(t) = \frac{\varphi}{\mu_0}, D_r^*(t) = 0.$$

Hence, $E_{dfe}(S^*, A_s^*, I^*, D_r^*, R^*) = \left(\frac{\varphi}{\mu_0}, 0, 0, 0, 0\right)$.

Similarly, we solve the system of equations (3.7) for finding $E_{ee}(S^*, A_s^*, I^*, D_r^*, R^*)$ where

$$\begin{aligned}
S^* &= \frac{\varphi\theta}{(\mu+\varphi)(r+\sigma)}, A_s^* = \frac{\sigma\varphi^2 + \mu\sigma\varphi}{\alpha\mu r + \alpha\mu\sigma + \alpha\varphi r + \alpha\varphi\sigma + \beta\varphi\theta}, I^* = \frac{\mu+\varphi}{\theta},\\
Dr^* &= \frac{\begin{array}{c}\alpha\delta\mu^2 r + \alpha\delta\mu^2\sigma + \alpha\delta\varphi^2 r + \alpha\delta\varphi^2\sigma + \beta\delta\varphi^2\theta + \beta\mu\varphi\theta^2 + \alpha\mu^2 r\theta\\ +\alpha\mu^2\sigma\theta + \alpha\varphi^2\sigma\theta + 2\alpha\delta\mu\varphi r + 2\alpha\delta\mu\varphi\sigma + \beta\delta\mu\varphi\theta + \alpha\mu\varphi r\theta + 2\alpha\mu\varphi\sigma\theta\end{array}}{\xi(\gamma\mu + \gamma\varphi)(\alpha\mu r + \alpha\mu\sigma + \alpha\varphi r + \alpha\varphi\sigma + \beta\varphi\theta)'},\\
R^* &= \frac{\begin{array}{c}\alpha\delta\mu^2 r + \alpha\delta\mu^2\sigma + \alpha\delta\varphi^2 r + \alpha\delta\varphi^2\sigma + \beta\delta\varphi^2\theta + \beta\mu\varphi\theta^2 + \alpha\mu^2 r\theta + \alpha\mu^2\sigma\theta\\ +\alpha\varphi^2\sigma\theta + 2\alpha\delta\mu\varphi r + 2\alpha\delta\mu\varphi\sigma + \beta\delta\mu\varphi\theta + \alpha\mu\varphi r\theta + 2\alpha\mu\varphi\sigma\theta\end{array}}{-\beta\mu\varphi\theta^2\xi - \alpha\mu^2 r\theta\xi - \alpha\mu^2\sigma\theta\xi - \alpha\mu\varphi r\theta\xi - \alpha\mu\varphi\sigma\theta\xi}.\\
&\qquad\qquad \rho\theta\xi(\alpha\mu r + \alpha\mu\sigma + \alpha\varphi r + \alpha\varphi\sigma + \beta\varphi\theta)'
\end{aligned}$$

Basic Reproduction Ratio

We have used the next-generation matrix method for finding the basic reproduction number of our model [38]. To employ this method, we have to seek the classes that are disease or infection related term. From the infection subsystem, we get transmission and transition matrix F and V.

Here, the matrix for the transmission terms:

$$F = \begin{pmatrix} 0 & \sigma \\ r & 0 \end{pmatrix}$$

And, the matrix for the transition terms:

$$V = \begin{pmatrix} -\alpha - \beta & 0 \\ \beta & -\delta - \gamma - \theta \end{pmatrix}$$

Then, we need to estimate a matrix G, so that $G = FV^{-1}$.

$$G = FV^{-1} = \begin{pmatrix} \frac{\beta\sigma}{(\alpha+\beta)(\delta+\theta+\gamma)} & \frac{\sigma}{\delta+\theta+\gamma} \\ \frac{r}{\alpha+\beta} & 0 \end{pmatrix}$$

Now, the characteristic polynomial is given by setting $|G - \lambda I| = 0$.

$$\Rightarrow \alpha\lambda^2\theta - r\sigma + \alpha\delta\lambda^2 + \alpha\gamma\lambda^2 + \beta\delta\lambda^2 - \beta\sigma\lambda + \beta\theta\lambda^2 + \beta\gamma\lambda^2 = 0$$

Solving this equation, we get the following basic reproduction number.

$$R_0 = \frac{\beta\sigma + \sqrt{\sigma\left(\beta^2\sigma + 4\alpha\delta r + 4\beta\delta r + 4\alpha\gamma r + 4\beta\gamma r + 4\alpha r\theta + 4\beta r\theta\right)}}{2(\alpha\delta + \beta\delta + \alpha\gamma + \beta\gamma + \alpha\theta + \beta\theta)}.$$

R_0 signifies an important role in disease modeling that if $R_0 > 1$, the disease will persist, and if $R_0 < 1$, the disease will die out.

Stability Analysis at DFE ($\mathbf{E_{dfe}}$)

Here, we have investigated the stability at E_{dfe} by establishing the Theorem 1.

Theorem 1 *The disease-free equilibrium (DEF) point of the model (3.1) is asymptotically stable if the eigenvalues of the Jacobian matrix are negative.*

Proof The Jacobian of (3.1) is given by

$$J = \begin{bmatrix} -\mu_0 - Ir - I\sigma & 0 & -Sr - S\sigma & 0 & 0 \\ I\sigma - A_s\beta & -\alpha - S\beta & S\sigma & 0 & 0 \\ A_s\beta + Ir & S\beta & Sr - \theta - D_r\gamma - \delta + D_r\gamma\xi & I\gamma\xi - I\gamma & 0 \\ 0 & 0 & -D_r\gamma\xi & -I\gamma\xi & \rho \\ 0 & \alpha & \delta - D_r\gamma(\xi - 1) & -I\gamma(\xi - 1) & -\rho \end{bmatrix} \tag{3.8}$$

The characteristic polynomial can be attained as $|J - \lambda I| = 0$.

$$\begin{vmatrix} -\mu_0 - Ir - I\sigma - \lambda & 0 & -Sr - S\sigma & 0 & 0 \\ I\sigma - A_s\beta & -\alpha - S\beta - \lambda & S\sigma & 0 & 0 \\ A_s\beta + Ir & S\beta & Sr - \theta - D_r\gamma - \delta + D_r\gamma\xi - \lambda & I\gamma\xi - I\gamma & 0 \\ 0 & 0 & -D_r\gamma\xi & -I\gamma\xi - \lambda & \rho \\ 0 & \alpha & \delta - D_r\gamma(\xi - 1) & -I\gamma(\xi - 1) & -\rho - \lambda \end{vmatrix} = 0$$

Substituting $S^* = \frac{\varphi}{\mu_0}$, we obtain

$$\begin{pmatrix} -\lambda - \mu_0 & 0 & -\frac{\varphi r}{\mu_0} - \frac{\varphi\sigma}{\mu_0} & 0 & 0 \\ 0 & -\alpha - \lambda - \frac{\beta\varphi}{\mu_0} & \frac{\varphi\sigma}{\mu_0} & 0 & 0 \\ 0 & \frac{\beta\varphi}{\mu_0} & \frac{\varphi r}{\mu_0} - \lambda - \theta - \delta & 0 & 0 \\ 0 & 0 & 0 & -\lambda & \rho \\ 0 & \alpha & \delta & 0 & -\lambda - \rho \end{pmatrix} = 0 \tag{3.9}$$

By taking determinant and solving it for λ.

$$\lambda_1 = 0, \lambda_2 = -\mu_0, \lambda_3 = -\rho, \lambda_3 = -\frac{\alpha\mu_0 + \delta\mu_0 + \beta\varphi + \mu_0\theta - \varphi r + \sqrt{\psi}}{2\theta},$$
$$\lambda_5 = -\frac{\alpha\mu_0 + \delta\mu_0 + \beta\varphi + \mu_0\theta - \varphi r - \sqrt{\psi}}{2\mu_0}.$$

where

$$\begin{aligned} \psi = {} & \alpha^2\mu_0^2 + 2\alpha\beta\mu_0\varphi - 2\alpha\delta\mu_0^2 - 2\alpha\mu_0^2\theta + 2\alpha\mu_0\varphi r \\ & + \beta^2\varphi^2 - 2\beta\delta\mu_0\varphi - 2\beta\mu_0\varphi\theta + 2\beta\varphi^2 r + 4\sigma\beta\varphi^2 \\ & + \delta^2\mu_0^2 + 2\delta\mu_0^2\theta - 2\delta\mu_0\varphi r + \mu_0^2\theta^2 - 2\mu_0\varphi r\theta + \varphi^2 r^2 \end{aligned}$$

Since $\lambda_1, \lambda_2, \lambda_3, \lambda_4$ and λ_5 are all negative, E_{dfe} point is asymptotically stable. Hence, Theorem 1 is proved.

Stability Analysis at EE (E_{ee})

In this section, we have investigated the stability at E_{dfe} by proving the Theorem 2.

Theorem 2 *The endemic equilibrium (DEF) point of the model (3.1) is asymptotically stable if the eigenvalues of the Jacobian matrix are negative.*

Proof The Jacobian of (3.1) at E_{ee} point is

$$J = \begin{bmatrix} -\mu_0 - rI^* - \sigma I^* & 0 & -rS^* - \sigma S^* & 0 & 0 \\ \sigma I^* - \beta A_s^* & -\beta S^* - \alpha & \sigma S^* & 0 & 0 \\ \beta A_s^* + rI^* & \beta S^* & -\theta - \gamma D_r^* - \delta + rS^* + \gamma\xi D_r^* & -\gamma I^* + \gamma\xi I^* & 0 \\ 0 & 0 & -\gamma\xi D_r^* & -\gamma\xi I^* & \rho \\ 0 & \alpha & \delta - \gamma D_r^* & -\gamma I^* & -\rho \end{bmatrix} \tag{3.10}$$

In Echelon form, the above matrix is written as

$$J = \begin{bmatrix} -\mu_0 - rI^* - \sigma I^* & 0 & -rS^* - \sigma S^* & 0 & 0 \\ 0 & -\beta S^* - \alpha & \frac{\beta A_s^* S^*}{I^*} & 0 & 0 \\ 0 & 0 & -a_{33} & -\gamma I^* + \gamma\xi I^* & 0 \\ 0 & 0 & 0 & -a_{44} & \rho \\ 0 & 0 & 0 & 0 & -a_{55} \end{bmatrix} \tag{3.11}$$

Equation (3.11) is a 5×5 matrix and the characteristic polynomial having eigenvalue λ is given by $|J - \lambda I| = 0$.

$$\begin{vmatrix} -\mu_0 - rI^* - \sigma I^* - \lambda & 0 & -rS^* - \sigma S^* & 0 & 0 \\ 0 & -\beta S^* - \alpha - \lambda & \frac{\beta A_s^* S^*}{I^*} & 0 & 0 \\ 0 & 0 & -a_{33} - \lambda & -\gamma I^* + \gamma\xi I^* & 0 \\ 0 & 0 & 0 & -a_{44} - \lambda & \rho \\ 0 & 0 & 0 & 0 & -a_{55} - \lambda \end{vmatrix} = 0$$

$$\Rightarrow (-\mu_0 - rI^* - \sigma I^* - \lambda)(-\beta S^* - \alpha - \lambda)(-a_{33} - \lambda)(-a_{44} - \lambda)(-a_{55} - \lambda) = 0$$

Here the eigenvalues are given by

$$\lambda_1 = -(\mu_0 + rI^* + \sigma I^*), \lambda_2 = -(\beta S^* + \alpha), \lambda_3 = -a_{33}, \lambda_4 = -a_{44}, \lambda_5 = -a_{55}.$$

where

$$a_{33} = \frac{\alpha(\theta + \delta + \gamma D_r^*) + \beta S^*(\theta + \delta + \gamma D_r^*) - \gamma\xi D_r^*(\alpha + \beta S^*)}{\beta S^* + \alpha} + \frac{\alpha\beta S^* A_s^*}{(\beta S^* + \alpha)Q^*}$$

$$a_{44} = \frac{\gamma\xi(\delta + \theta)Q^{*2}\{\alpha + \beta S^*\} + \alpha\beta\gamma\xi S^* A_s^* Q^*}{(\theta + \delta)\{\alpha + \beta S^*\}Q^* + (1 - \xi)\{\gamma\alpha + \gamma\beta S^*\}Q^* D_r^* + \alpha\beta S^* A_s^*}$$

$$a_{55} = \frac{\rho\left[\beta\{\xi\theta + 2\delta + \theta\}Q^*S^* + \alpha\{\xi\theta + 2\delta + \theta\}Q^* + 2\alpha\beta S^* A_s^*\right]}{\xi\left[(\delta + \theta)\{\alpha + \beta S^*\}Q^* + \alpha\beta S^* A_s^*\right]}$$

The eigenvalues of the characteristic polynomial are $\lambda_1 = -(rI^* + \sigma I^*)$, $\lambda_2 = -(\beta S^* + \alpha)$, $\lambda_3 = -a_{33}$, $\lambda_4 = -a_{44}$, and $\lambda_5 = -a_{55}$, which are all real numbers. Since all the eigenvalues ($\lambda_1, \lambda_2, \lambda_3, \lambda_4$ and λ_5) have a negative real part, E_{ee} is asymptotically stable. Hence, Theorem 2 is proved.

Results and Discussion

Computer simulations of any biological phenomena provide a rapid, cost-effective, and illuminating assessment. For this reason, numerical simulations of the developed COVID-19 model (3.1) have been carried out by the Runge–Kutta-Fehlberg method using MATLAB programming language. We have taken initial population at $S = 10,000,000$, $A_s = 30,000$, $I = 50,000$, $D_r = 3000$, $R = 20,000$. Firstly, we have solved the model (3.1) for the tabulated values in Table 3.1, representing all the parametric values considered for our model and the figure obtained from numerical simulation is presented in Fig. 3.2. Also, we simulated the model for different parameter values of σ and β keeping all other values the same to show their effects on the model. In this case, the graphs are shown in Figs. 3.3, 3.4, 3.5, 3.6, 3.7 and 3.8. The parameter σ represents disease transmission rate of asymptomatic individuals for contacting an infected and susceptible population. Thus, this parameter has great value in respect to the disease transmissions. So, we perform simulations for different values of σ and the graphs are presented in Figs. 3.4, 3.5, 3.6, and 3.7. Furthermore, β is the rate at which the infected populations get infectious for contacting COVID-19 patients, and this parameter has a significant role for disease transmission among the doctors. So, considering different values of this parameter, Figs. 3.8 and 3.9 have been drawn. Finally, we show the graphs of the basic reproductive ratio presented in Figs. 3.10, 3.11, 3.12 and 3.13. It helps us to predict whether the disease will persist or die out.

Figure 3.2 shows the dynamics of five compartments such as susceptible, asymptomatic, infected, doctors, and recovered populations. We have observed that the susceptible population decreases from the initial state and it reaches to zero steadily. The asymptomatic population decreases for the first week and quickly reaches to zero after that it gradually booms. The infected populations progressively soar from the initial state and reach to the peak level leading to decrease the number of doctors. As coronavirus disease is highly infectious, doctors are frequently getting infected at the time of performing their novel duty. So, the doctors are decreasing surprisingly from the initial state. Despite this, the recovered populations and death rate increase from the initial state. It shows that the infection rate of coronavirus disease increases leading to an enormous death every day and causing a massive number of infections among doctors. Thus, the whole medical service system is getting down over time.

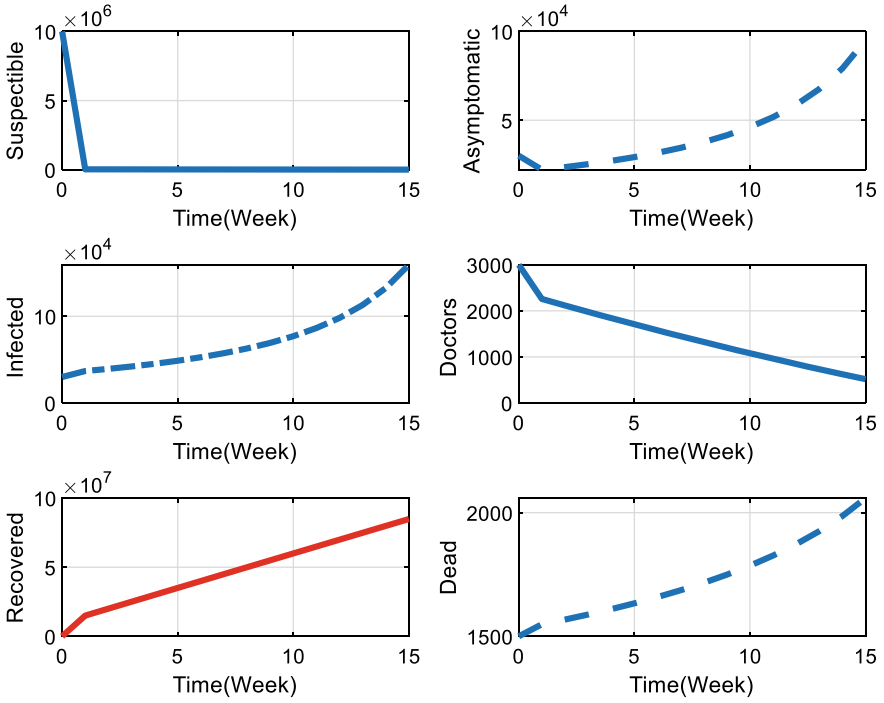

Fig. 3.2 Trajectories of all the compartments of the epidemic model (3.1) (*Source* Own)

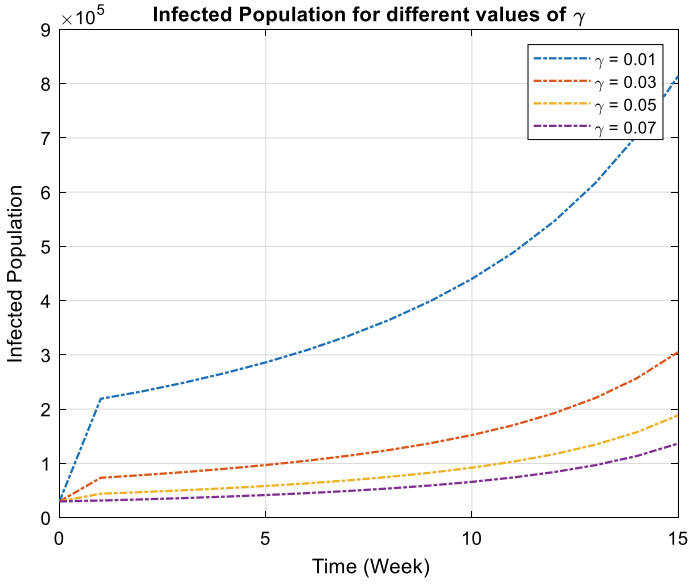

Fig. 3.3 Change of infected population over 15 weeks for $\gamma = 0.01, 0.03, 0.05, 0.07$ (*Source* Own)

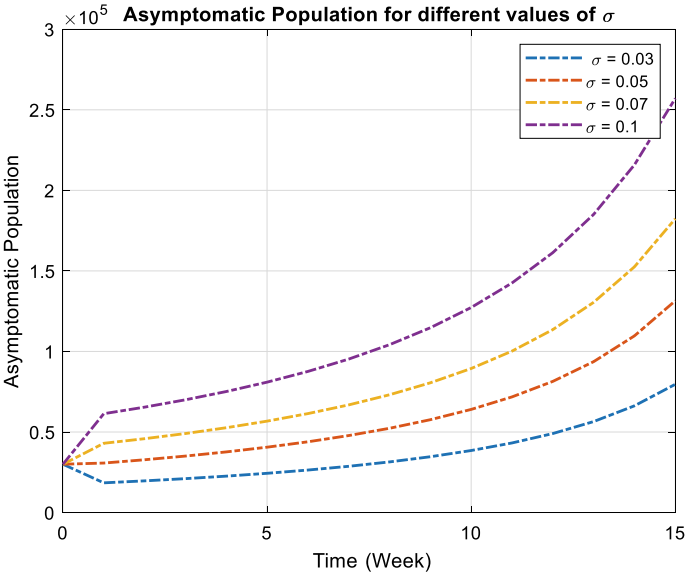

Fig. 3.4 Change of asymptomatic population over 15 weeks for $\sigma = 0.03, 0.05, 0.07, 0.1$ (*Source* Own)

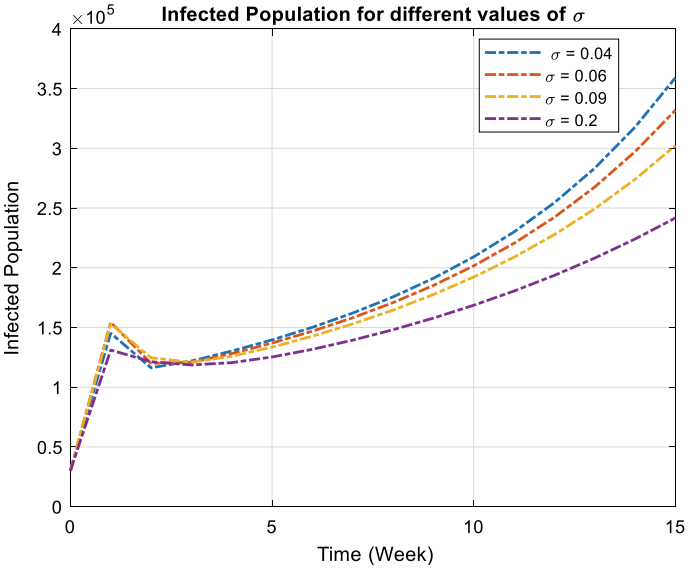

Fig. 3.5 Change of infected population over 15 weeks for $\sigma = 0.04, 0.06, 0.09, 0.2$ (*Source* Own)

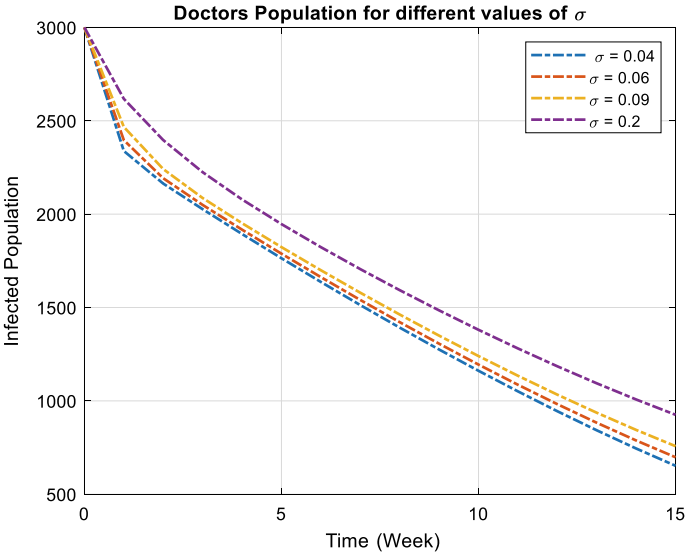

Fig. 3.6 Change of doctor population over 15 weeks for $\sigma = 0.04, 0.06, 0.09, 0.2$ (*Source* Own)

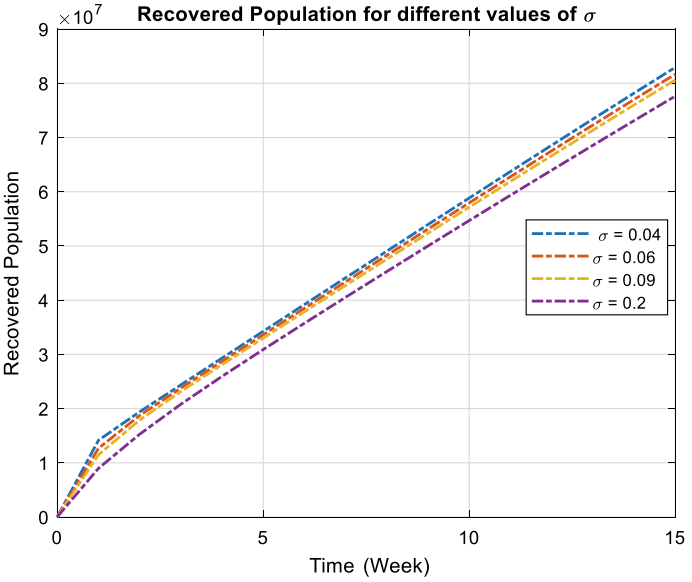

Fig. 3.7 Change of recovered population (15 weeks) for $\sigma = 0.04, 0.06, 0.09, 0.2$ (*Source* Own)

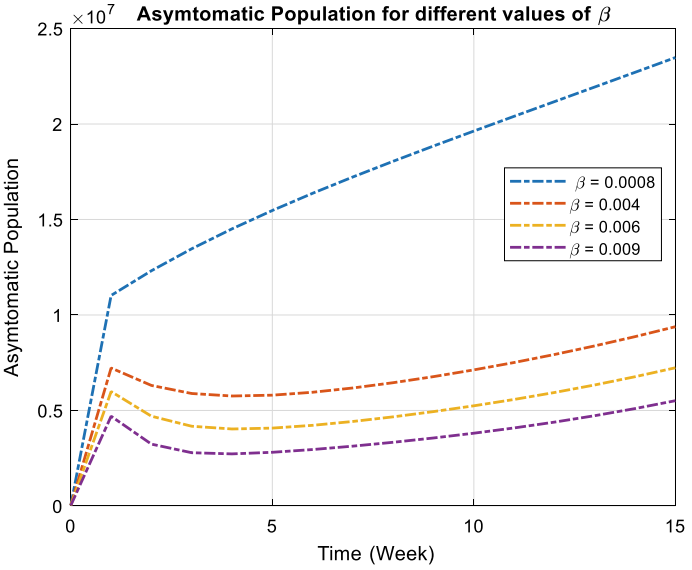

Fig. 3.8 Change of asymptomatic population over (15 weeks) for $\beta = 0.0008, 0.004, 0.006, 0.009$ (*Source* Own)

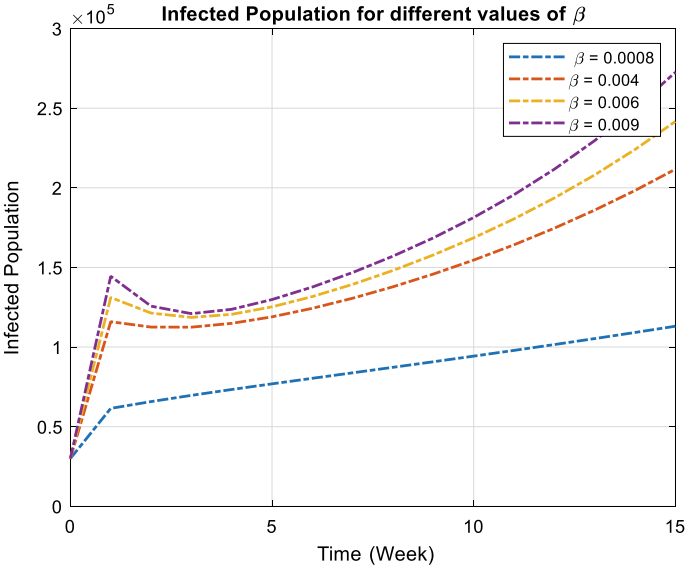

Fig. 3.9 Change of asymptomatic population over (15 weeks) for $\beta = 0.0008, 0.004, 0.006, 0.009$ (*Source* Own)

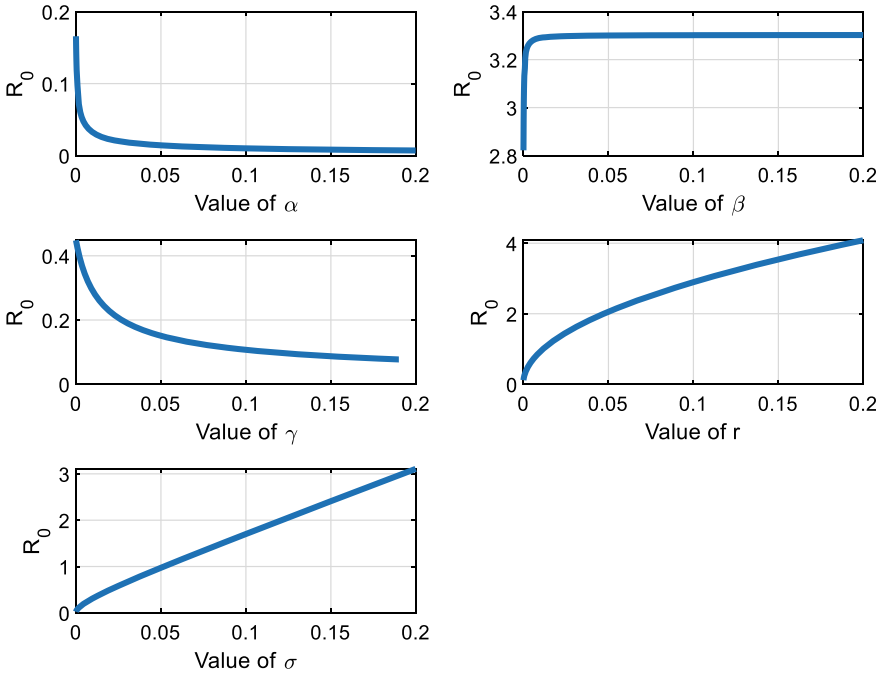

Fig. 3.10 Graphical illustration of the basic reproductive ratio for different parameter value range (*Source* Own)

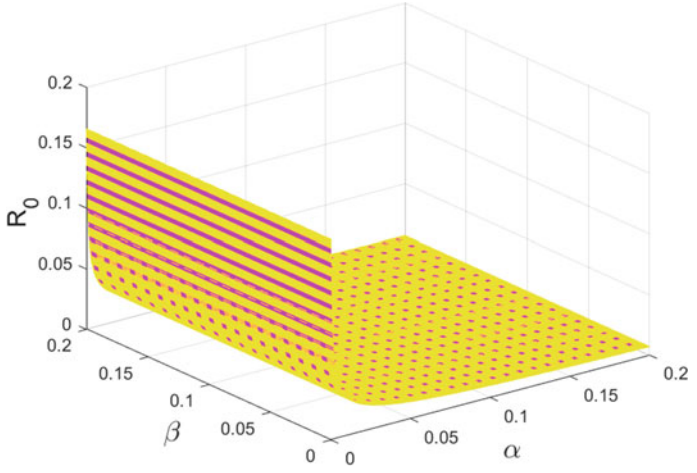

Fig. 3.11 The graph of basic reproductive ratio (R_0) with respect to the infection rate (β) of infected populations and recovery rate (α) of asymptomatic populations (*Source* Own)

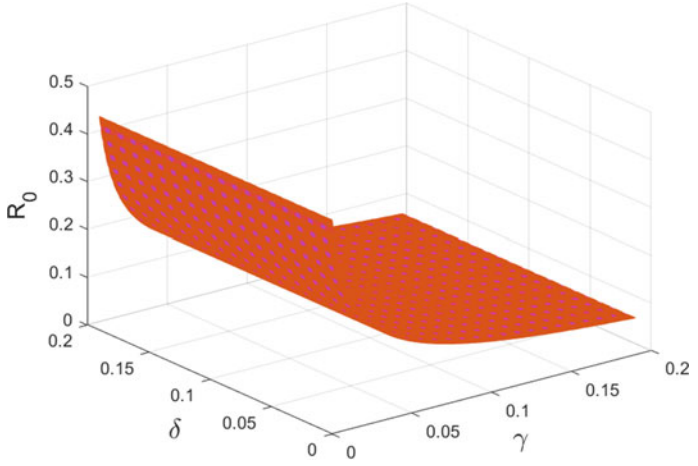

Fig. 3.12 The graph of basic reproductive ratio (R_0) with respect to the recover rate (δ) without consulting with doctors and recovered rate (γ) due to consulting with doctors (*Source* Own)

From Fig. 3.3, we can say that for a higher rate of γ, the infected population decrease with time because the majority of COVID-19 patients undertake treatment to relieve the disease. This leads to a less number of asymptomatic individuals. Hence, these reduced asymptomatic individuals do not infect the doctors massively.

Figure 3.4 indicates the influence of disease transmission rate (σ) on the asymptomatic populations for 15 weeks period. Asymptomatic populations are those who are infected by the coronavirus disease. The unique feature of these asymptomatic populations is that they don't reveal any signs or symptoms of COVID-19. For that reason, these asymptomatic populations spread the disease among people subconsciously. In Fig. 3.4, it has been noticed that the asymptomatic populations increase as the disease transmission rate (when σ rises from 0.03 to 0.1) increases.

Figure 3.5 represents the effect of the disease transmission rate (σ) on the infected populations for 15 weeks period. Infected populations are those who are actually infected by the coronavirus disease, identified, and able to transmit it among people. From Fig. 3.5, we have observed that the infected populations decrease sequentially as the disease transmission rate σ (when σ rises from 0.04 to 0.2) of asymptomatic populations increases. This shows that the asymptomatic populations increase as the disease transmission rate σ increases but the infected populations decrease as this parameter increases. This is because of the increasing number of asymptomatic populations lead to a decrease in the concentration of infected populations. As the more populations remain in asymptomatic condition, the more populations do not get identified as COVID-19 carriers. Thus, a massive population will remain unidentified and hence the number of identified infected populations will decrease.

Figure 3.6 shows the variation of doctor populations for different values of disease transmission rate (σ), the rate at which the susceptible populations becoming infectious by contacting COVID-19 patients. A remarkable number of doctors have been

infected worldwide at the time of serving COVID-19 patients, and as a result, they have died by this novel coronavirus infection. Most of them are getting infected with coronavirus by the asymptomatic populations. This scenario is presented in Fig. 3.6, and from this figure, it has been observed that the doctor populations decrease quickly as the disease transmission rate (σ) (when σ ranges from 0.04 to 0.2) of asymptomatic populations increases. The increasing number of asymptomatic populations leads the doctors to be infected. Thus, the number of doctors to serve patients is going down over time.

Figure 3.7 shows the effect of the disease transmission rate (σ) on the recovered populations for 15 weeks period. It has been observed that the recovered populations decrease as the disease transmission rate σ (when σ goes from 0.04 to 0.2) of asymptomatic population increases. Since the asymptomatic population increases with the increase of disease transmission rate σ, recovered individuals decrease with the increase of this parameter value.

Figure 3.8 presents the effect of infection rate (β) on the asymptomatic populations for 15 weeks period. From this figure, it has been noticed that the asymptomatic populations decrease as the infection rate β (when β goes from 0.0008 to 0.009) of the infected population increases. Since the infected population increases with the increase of infection rate β, the asymptomatic population decreases with the increase of this parameter value.

Figure 3.9 exhibits the effect of infection rate (β) on the infected populations for 15 weeks period. It has been observed that the infected populations increase swiftly for the first two weeks from the initial state but it slowly decreases to the next two weeks as the infection rate (β) (when β goes from 0.0008 to 0.006) increases. After that, the infected populations gradually increase as the infection rate (β) increases. It means that when the infection rate (β) increases, the number of infected individuals also increases.

The simulated graphs presented in Fig. 3.10, shows the schematic view of the basic reproductive ratio of the model (3.1). We have performed the simulation of basic reproductive ratio (R_0) with respect to α, β, γ, r, and σ. From Fig. 3.10, it has been noticed that the basic reproductive ratio $R_0 < 1$ for all values of α and γ whereas $R_0 > 1$ for all values β, r, and σ. Hence, the disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Whereas, the endemic equilibrium point is locally asymptotically stable if $R_0 > 1$ and unstable if $R_0 < 1$ [37]. Figures 3.11, 3.12 and 3.13 present a 3-dimensional plot of basic reproductive ratio.

Conclusions

People from the entire world are confined at the home due to life-threatening coronavirus disease (COVID-19). In spite of the deadliness of COVID-19, doctors are performing their novel duty to serve the coronavirus infected patients. Thus, they are acting as first-line soldiers keeping themselves vulnerable to coronavirus infections.

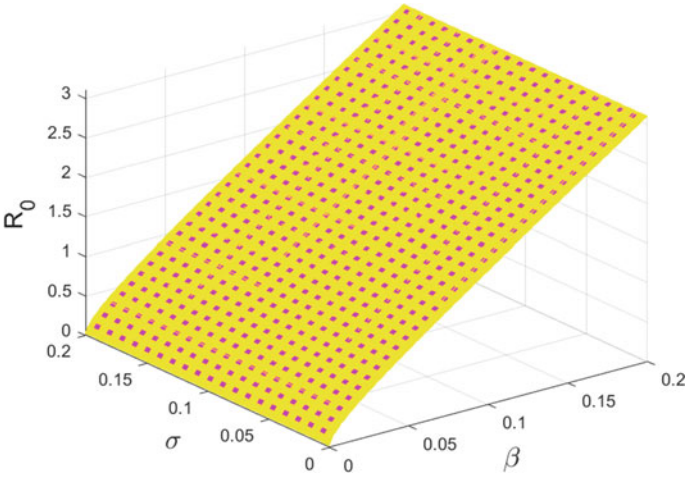

Fig. 3.13 The graph of basic reproductive ratio (R_0) with respect to the disease transmission rate (σ) of asymptomatic and the infection rate (β) of infected populations (*Source* Own)

In this paper, we have developed a mathematical model of COVID-19 in terms of a set of non-linear ordinary differential equations showing that doctors are affecting more frequently at the time of serving coronavirus infected patients. So, the medical service system is going down over time. We have analyzed the model by determining of the basic reproductive ratio and related stability analysis at the disease-free and endemic equilibrium points. The graph of the basic reproductive ratio for different parameters has been carried out to show the disease behavior. Finally, numerical simulations have been performed to illustrate the analytic results. We have observed that the asymptomatic population increases as the disease (COVID-19) transmission rate increases and also the number of infected population increases when the infection rate increases. These increasing asymptomatic and infected populations lead the doctors to be infected by contact with them. Thus, the whole medical service system is getting down over time. So, it is time to save our supreme warriors (doctors) during this coronavirus outbreak by ensuring their proper safety like life-saving protective equipment.

Acknowledgements The authors greatly acknowledge the partial financial support provided by the Ministry of Science and Technology, Government of the People's Republic of Bangladesh under special allocation in 2019-2020 with the research grant Ref. No.-39.00.0000.009.06.024.19-12/410(EAS). Supports with Ref.: 17-392 RG/MATHS/AS_I–FR3240297753 funded by TWAS, Italy, and Ref. no.-6(74) UGC/ST/Physical-17/2017/3169 funded by the UGC, Bangladesh is also acknowledged.

Data Availability The data used to support the findings of this study are included in the article.

Conflicts of Interest The authors declare that there are no conflicts of interest regarding the publication of this manuscript.

Authors' Contributions This research is a group work carried out in collaboration among all authors. Authors MHAB and AKP designed the study, performed the conceptualization and methodological analysis and model formulation of the first draft of the manuscript. Authors SM and SA analyzed the model analytically and wrote some literature on the study. Author MSK wrote the programming codes and performed some part of the computational analysis. Author SAS contributed to literature searches and calculated the real data to estimate the parameters, MAI and MRK verified the parameters and checked the literature. All authors have read and agreed to publish the final version of the manuscript.

References

1. Nearly 600 US health care workers have died of COVID-19. abc News (2020). https://abcnews.go.com/Health/600-us-health-care-workers-died-covid-19/story?id=71132667. Last accessed 2020/06/8.
2. Coronavirus: Doctors' mortality rate in Bangladesh 'highest in the world'. United News of Bangladesh (UNB). https://unb.com.bd/category/Special/coronavirus-doctors-mortality-rate-in-bangladesh-highest-in-the-world/53378. Last accessed 2020/06/21.
3. Global: Health workers silenced, exposed and attacked. Amnesty International. https://www.amnesty.org/en/latest/news/2020/07/health-workers-rights-covid-report/. Last accessed 2020/07/13.
4. Biswas, M. H. A., Khatun, M. S., Paul, A. K., Khatun, M. R., Islam, M. A., Samad, S. A., & Ghosh, U. (2020). Modeling the effective control strategy for the transmission dynamics of global pandemic COVID-19. Preprint (2020). https://doi.org/10.1101/2020.04.22.20076158.
5. Sheffield, S., York, A., Swartwood, N., Bilinski, A., Williamson, A., & Fitzpatrick, M. (2020). Strict physical distancing may be more efficient: A mathematical argument for making the lockdowns count. Preprint (2020). https://doi.org/10.1101/2020.05.19.20107045
6. Bairagi, A. K., Masud, M., Kim, D. H., Munir, M. S., Nahid, A. A., Abedin, S. F., et al. (2020). Controlling the outbreak of COVID-19: A noncooperative game perspective. Preprint (2020). arxiv:2007.13305.
7. Chen, X., & Yu, B. (2020). First two months of the 2019 coronavirus disease (COVID-19) epidemic in China: Real time surveillance and evaluation with a second derivative model. *Global Health Research and Policy, 5*(7), 1–9. https://doi.org/10.1186/s41256-020-00137-4.
8. Chen, N., Zhou, M., Dong, X., & Zhang, L. (2020). Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: A descriptive study. *The Lancet, 395*(10223), 507–513.
9. Sookaromdee, P., & Wiwanitkit, V. (2020). Imported cases of 2019-novel coronavirus (2019-nCoV) infections in Thailand: Mathematical modelling of the outbreak. *Asian Pacific Journal of Tropical Medicine, 13*(3), 139–140.
10. Zhao, S., Lin, Q., Ran, J., Musa, S. S., Yang, G., Wang, W., et al. (2020). Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in China, from 2019 to 2020: A data-driven analysis in the early phase of the outbreak. *International Journal of Infectious Diseases, 92,* 214–217.
11. Ndairou, F., Area, I., Nieto, J. J., & Torres, D. F. M. (2020). Mathematical modeling of COVID-19 transmission dynamics with a case study of Wuhan. Preprint (2020). arXiv:2004.10885v1.
12. Aguilar, J. B., Faust, G. S., Westafer, M. L. M., & Gutierrez, J. B. (2020). Investigating the impact of asymptomatic carriers on COVID-19 transmission. Preprint (2020). https://doi.org/10.1101/2020.03.18.20037994.
13. Carolina Garcia-Vidal, C., et al. (2020). Incidence of co-infections and superinfections in hospitalized patients with COVID-19: A retrospective cohort study. *Clinical Microbiology and Infection.* https://doi.org/10.1016/j.cmi.2020.07.041.

14. Casas-Rojo, J. M., et al. (2020). Clinical characteristics of patients hospitalized with COVID-19 in Spain: Results from the SEMI-COVID-19 registry. *Revista Clinica Espanola*. https://doi.org/10.1016/j.rce.2020.07.003.
15. Chitnis, N., Hyman, J. M., & Cushing, J. M. (2020). Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of Mathematical Biology, 70,* 1272–1296. https://doi.org/10.1007/s11538-008-9299-0.
16. Chen, T. M., Rui, J., Wang, Q., Zhao, Z., Cui, J., & Yin, L. (2020). A Mathematical model for simulating the phase-based transmissibility of a novel coronavirus. *Infectious Diseases of Poverty, 9*(24), 1–8. https://doi.org/10.1186/s40249-020-00640-3.
17. Huo, H. F., & Feng, L. X. (2012). Global stability of an epidemic model with incomplete treatment and vaccination. *Discrete Dynamics in Nature and Society, 2012,* 1–14. https://doi.org/10.1155/2012/530267.
18. Kabir, M. H., Gani, M. O., Mandal, S., & Biswas, M. H. A. (2020). Modeling the dispersal effect to reduce the infection of COVID-19 in Bangladesh. *Sensors International, 1,* 100043. https://doi.org/10.1016/j.sintl.2020.100043.
19. Queen Elizabeth Hospital Birmingham COVID-19 airway team, safety and 30-day outcomes of tracheostomy for COVID-19: A prospective observational cohort study. *British Journal of Anaesthesia*. https://doi.org/10.1016/j.bja.2020.08.023.
20. Magalhaesa, J. J. F. D., et al. (2020). Epidemiological and clinical characteristics of the first 557 successive patients with COVID-19 in Pernambuco state, Northeast Brazil. *Travel Medicine and Infectious Disease, 38,* 101884.
21. Melliani, S., Allaoui, A. E., & Chadli, L. S. (2020). A simple mathematical model for coronavirus (COVID-19). *Discrete Dynamics in Nature and Society*. Preprint (2020). https://doi.org/10.1101/2020.04.23.20076919.
22. Nadeem, S. (2020). Coronavirus Covid-19: Available free literature provided by various companies. *Journals and Organizations Around the World, 5*(1), 7–13.
23. Prompetchara, E., Ketloy, C., & Palaga, T. (2020). Immune responses in COVID-19 and potential vaccines: Lessons learned from SARS and MERS epidemic. *Asian Pacific Journal of Allergy and Immunology, 38*(1), 1–9. https://doi.org/10.12932/AP-200220-0772.
24. Safi, M. A., & Garba, S. M. (2012). Global stability analysis of SEIR model with Holling Type II incidence function. *Computational and Mathematical Methods in Medicine, 2012*(826052), 1–8. https://doi.org/10.1155/2012/826052.
25. Xu, X., Chen, P., Wang, J., Feng, J., Zhou, H., Li, X., et al. (2020). Evolution of the novel coronavirus from the ongoing wuhan outbreak and modeling of its spike protein for risk of human transmission. *Science China Life Sciences, 63,* 457–460.
26. Zhang, J., Weili, W., Zhao, X., & Zhang, W. (2020). Recommended psychological crisis intervention response to the 2019 novel coronavirus pneumonia outbreak in China: A model of West China hospital. *Precision Clinical Medicine, 3*(1), 3–8.
27. Zhang, S., Diao, M., Wenbo, Y., Pei, L., Lin, Z., & Chen, D. (2020). Estimation of the reproductive number of novel coronavirus (COVID-19) and the probable outbreak size on the diamond princess cruise ship: A data-driven analysis. *International Journal of Infectious Diseases, 93,* 201–214.
28. Biswas, M. H. A., Haque, M. M., & Mallick, U. K. (2019). Optimal control strategy for the immunotherapeutic treatment of HIV infection with state constraint. *Optimal Control, Applications and Methods, 40*(3), 1–12.
29. Biswas, M. H. A., Paiva, L. T., & de Pinho, M. D. R. (2014). A SEIR model for control of infectious diseases with constraints. *Mathematical Biosciences and Engineering, 11*(4), 761–784.
30. Biswas, M. H. A. (2014). On the immunotherapy of HIV infections via optimal control with constraint. In *Proceedings of the 18th International Mathematics Conference* (pp. 51–54), Dhaka, 20–22 March 2014.
31. Biswas, M. H. A. (2013). *Necessary conditions for optimal control problems with state constraints: Theory and applications* (Ph.D. thesis). Department of Electrical and Computer Engineering, Faculty of Engineering, University of Porto, Portugal.

32. Biswas, M. H. A. (2012). Model and control strategy of the deadly Nipah Virus (NiV) infections in Bangladesh. *Research & Reviews in BioSciences, 6*(12), 370–377.
33. Biswas, M. H. A. (2012). AIDS epidemic worldwide and the millennium development strategies: A light for lives. *HIV and AIDS Review, 11*(4), 87–94.
34. Biswas, M. H. A., Ara, M., Haque, M. N., & Rahman, M. A. (2011). Application of control theory in the efficient and sustainable forest management. *International Journal of Scientific & Engineering Research, 2*(3), 26–33.
35. Khatun, M. S., & Biswas, M. H. A. (2020). Mathematical analysis and optimal control applied to the treatment of leukemia. *Journal of Applied Mathematics and Computing, 64,* 331–354. https://doi.org/10.1007/s12190-020-01357-0.
36. Khatun, M. S., & Biswas, M. H. A. (2020). Optimal control strategies for preventing hepatitis B infection and reducing chronic liver cirrhosis incidence. *Infectious Disease Modelling, 5,* 91–110.
37. Khatun, M. S., & Biswas, M. H. A. (2019). Modeling the effect of adoptive T cell therapy for the treatment of leukemia. *Computational and Mathematical Method, 2,* e1069. https://doi.org/10.1002/cmm4.1069.
38. Driessche, P. V. D., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences, 180,* 29–48.