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Modeling the dispersal effect to reduce the infection of COVID-19 in Bangladesh



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ABSTRACT

In this paper, we propose a four compartmental model to understand the dynamics of infectious disease COVID-19. We show the boundedness and non-negativity of solutions of the model. We analytically calculate the basic reproduction number of the model and perform the stability analysis at the equilibrium points to understand the epidemic and endemic cases based on the basic reproduction number. Our analytical results show that disease free equilibrium point is asymptotically stable (unstable) and endemic equilibrium point is unstable (asymptotically stable) if the basic reproduction number is less than (greater than) unity. The dispersal rate of the infected population and the social awareness control parameter are the main focus of this study. In our model, these parameters play a vital role to control the spread of COVID-19. Our results reveal that regional lockdown and social awareness (e.g., wearing a face mask, washing hands, social distancing) can reduce the pandemic of the current outbreak of novel coronavirus in a most densely populated country like Bangladesh.

1. Introduction

Mathematical modeling is an important tool to analyze several complex phenomena surrounding us. When a real system change concerning time, then it is referred to as a dynamical system. A dynamical system can be described by using a system of ordinary differential equations (ODEs) or a system of partial differential equations (PDEs) depending on the real (physical or chemical or biological or social) problems. These are sometimes called the ODE or PDE models respectively. In this paper, we propose a new deterministic ODE model, which has four ODEs, for the study of spread and control of infectious disease of COVID-19. COVID-19 is an active research topic not only in the community of mathematical epidemiology group [1,6,9,28,41] but also the problem is of interests in various scientific communities including the group of modeling biological systems [8,13], biomedical engineering [5,33] and signal processing [46], etc. To prepare a new mathematical model for a specific disease, some population-specific assumptions are important to make the model simple. However, the principal of model parsimony is also important, which is simply states as "a mathematical model should be as simple as possible and as complex as necessary" [39]. It also includes the parameter estimation from the source of real outbreaks information. A new mathematical model grants us to know the asymptotic prediction of infectious outbreaks shortly of a particular area of a country or of a country [4,25, 35,37,40,45] by using the present data of outbreaks and also allow us to understand the basic epidemiological processes [2]. Thehe first, simplest and most basic proposed ODE model in the study of epidemiology is the three compartmental SIR (susceptible, infected, and recovered) model proposed by Kermack and McKendrick in 1927 [21]. Several modifications of the SIR model was published to understand the infectious outbreaks in humans and other animals [19,20,29]. Modeling approach was also used to predict and understand several infectious diseases such as HIV-infection [31], influenza pandemic [10], H1F1 epidemic [20], pseudo-periodic 1918 pandemic influenza (known as Spanish flu) [3,12, 30,39], 2002/2003 SARS epidemic in Asia [27,44], etc. The agents of

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Fig. 1. The schematic diagram of the model.

Table	1
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Compartmental variables.

Compartmental variables	Physical meaning
S_n S_q	Non-quarantined susceptible population Quarantined susceptible population
Ι	Infected population
R	Recovered or Immune population

infectious diseases are mainly viruses, bacteria, and protozoa. The fundamental characteristics or biological dynamics of different infectious diseases are different. A basic model needs to modify based on a particular disease so that the model behavior and results adapt to the field data. Therefore, in this study, we propose a new model to understand the dynamics and control mechanisms of an ongoing pandemic coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [32], emerging in December 2019 in Wuhan city of China [15]. There are already proposed mathematical models for COVID-19 [18,26,36] to understand the current global outbreaks. COVID-19 outbreaks transmitted rapidly throughout the world and causing subversive health problems. The non-pharmaceuticals interventions, such as social-distancing (three or six feet), isolation or hospitalization for confirmed cases, home quarantine for susceptible cases, contact-tracing, washing hands for 20 s, use of face-masks in public places and some cases at home, etc. are the only way to suppress the pandemic burden as no vaccine or antiviral medicines are not yet invented by the researchers or healthcare management [36]. As of May 29, 2020, COVID-19 has spread to about 216 countries, area or territories, causing 5704736 infectious and deaths 357736 across the world [42]. The United States is now the epicenter of the current pandemic of the coronavirus. The record shows that 1803622 is of confirmed total cases and 104987 deaths in US [43]. In Bangladesh COVID-19 causes 44844 infectious, 582 deaths and 9015 recovered cases [42]. The first three cases (two men and one woman) of COVID-19 in Bangladesh was confirmed on March 8, 2020 [17]. Two men were Italy returnees and the woman was a family member of one of them. It is clear from the data of the developed countries that people of 65 years older and above are at high risk of deaths, however, in the case of Bangladesh it is 40 years and above. COVID-19 has incubation period (susceptible to infectious) of range 2-14 days and common symptoms of the disease are fever or chills, shortness of breath for mild cases, coughing, fatigue, muscle or body aches, headache, sore throat, congestion or runny nose, nausea or vomiting, diarrhoea, loss of smell and pneumonia for severe cases [7,42]. The most dangerous cases are the corona positive people (about 80%) without any prior symptoms [42]. In a high density populated country like Bangladesh, it is not easy to maintain six-feet social-distancing in the public places (e.g., raw vegetable market, shopping-mall, festival gathering, etc.). As a result, the situation is becoming worse day by day here in Bangladesh.

In this paper, we propose a new mathematical model for the understanding of the transmission dynamics of COVID-19. We show the control mechanism of corona virus and make a possible prediction for the next six months in Bangladesh. We specially emphasize the influence of the

Table 2						
List of the	paramotore	involved	in	the	mod	0

Deremetere	Description Description			
Parameters	Description			
Α	Recruitment rate of non-quarantined susceptible population			
η	Number of individual having COVID-19 negative after quarantine			
α	Rate of non-quarantined individuals having COVID-19 symptoms			
β	Immunity increasing rate			
μ	Rate of non-quarantined population having COVID-19 positive			
ν	Social Awareness control parameter			
ρ	Natural death rate			
γ	Infection rate of quarantined population			
ε	Natural recovery rate of quarantined population			
σ	Recovery rate of infected individual			
δ	COVID-19 induced death rate of infected individual			

Table 3

d

Estimated values of the parameters. Source: Worldometer- COVID-19 data for Bangladesh.

Average spatial dispersal rate of infected population

Parameters	Values (in thousands per Day)
Α	2.0
η	0.03
α	0.747
β	1.0
μ	0.583
ν	1.0
ρ	0.2
γ	0.0752
ε	0.5
σ	0.153
κ	0.11
d	0.032

social distancing and regional lockdown on the spread of corona virus in Bangladesh through our proposed model. This result will help the government in order to prepare the country's medical system, hospitals, treatment management, etc. for the upcoming days.

In Section 2, a brief introduction of our mathematical model, local dynamics and its scope for the infectious disease of COVID-19 is presented. The current real data fit the spread of the corona virus in Bangladesh and using this result as an initial data our model prediction is demonstrated in Section 3. The paper is concluded with some general remarks and future perspectives in Section 4.

2. Mathematical model of COVID-19

To understand the transmission of COVID-19 as well as its outbreak, we propose a mathematical model that can predict the dynamics of transmission in four compartments. For this purpose, we assume $S_n(t)$ denotes non-quarantined susceptible population, $S_q(t)$ represents quarantined susceptible population, R(t) represents recovered or immune population. For the formulation of the model, we impose the following assumptions:

- (A1) Non-quarantined population undergoes to quarantine having COVID-19 symptoms with rate α , while β can control the exposure of such symptoms by increasing immune level and following personal consciousness.
- (A2) Non-quarantined population becomes infected with rate μ , while ν can control such infection by maintaining social distance, wearing face-mask and PPE, and washing hands with soap frequently, etc.
- (A3) Quarantined population having COVID-19 positive with rate γ , and after completion of quarantine, there is a possibility to go back to home with COVID-19 negative at a rate η . Moreover, quarantined population becomes cured underlying the quarantine protocol strictly with the rate ε .



(a) Non-quarantined susceptible population

Fig. 2. a. Non-quarantined population $S_n(t)$ increases as β increases, b. Quarantined population $S_q(t)$ decreases as β increases. Here the parameters are chosen from Table 3.

- (A4) Infected population recovers with rate σ through proper medication and dies with rate δ due to the COVID-19.
- (A5) All the compartments have natural death rate ρ .

The schematic representation of our assumptions underlying the model is depicted in Fig. 1.

Under the above-mentioned assumptions (A1-A5), the model leads to the following system of nonlinear ordinary differential equations:

$$\begin{cases} \frac{dS_n}{dt} = A + \eta S_q - \frac{\alpha S_n}{\beta + S_n} S_q - \frac{\mu S_n}{\nu + S_n} I - \rho S_n, \\ \frac{dS_q}{dt} = \frac{\alpha S_n}{\beta + S_n} S_q - \gamma S_q - \varepsilon S_q - \eta S_q - \rho S_q, \\ \frac{dI}{dt} = \gamma S_q + \frac{\mu S_n}{\nu + S_n} I - \sigma I - \delta I - \rho I + dI, \\ \frac{dR}{dt} = \sigma I + \varepsilon S_q - \rho R, \end{cases}$$
(2.1)

with the initial conditions $S_n(0) = S_{n0} \ge 0, S_q(0) = S_{q0} \ge 0, I(0) = I_0 \ge 0, R(0) = R_0 \ge 0.$

The description of compartmental variables and parameters are presented in the following tables Table 1.

All the parameters are assumed to be nonnegative having following physical interpretations mentioned in the following Table 2.

We do not make the compartmental variables non-dimensional, instead we use the following transformations to rescale system (2.1)

$$\begin{split} \tilde{t} &= mt, \tilde{A} = \frac{A}{m}, \tilde{\eta} = \frac{\eta}{m}, \tilde{\alpha} = \frac{\alpha}{m}, \tilde{\mu} = \frac{\mu}{m} \\ \tilde{\varepsilon} &= \frac{\varepsilon}{m}, \tilde{\sigma} = \frac{\sigma}{m}, \tilde{\rho} = \frac{\rho}{m}, m = \gamma + \varepsilon + \eta + \rho, \kappa = \frac{\sigma + \delta + \rho}{\gamma + \varepsilon + \eta + \rho}. \end{split}$$

Now, neglecting the \sim (tilde) sign, we obtain the following rescaled version of (2.1)

$$\begin{cases} \frac{dS_n}{dt} = A + \eta S_q - \frac{\alpha S_n}{\beta + S_n} S_q - \frac{\mu S_n}{\nu + S_n} I - \rho S_n, \\ \frac{dS_q}{dt} = \frac{\alpha S_n}{\beta + S_n} S_q - S_q, \\ \frac{dI}{dt} = \gamma S_q + \frac{\mu S_n}{\nu + S_n} I - \kappa I + dI, \\ \frac{dR}{dt} = \sigma I + \varepsilon S_q - \rho R, \end{cases}$$
(2.2)

(b) Quarantined susceptible population

with the initial conditions

$$\left(S_n(0), S_q(0), I(0), R(0)\right) = \left(S_{n0}, S_{q0}, I_0, R_0\right).$$
(2.3)

In order to analyze the model from analytical and numerical viewpoints, we estimate the parameters from the COVID-19 data for Bangladesh from 08 March to May 24, 2020 available in the Worldometer [43] which are listed in Table 3.

3. Mathematical analysis of the model

To investigate the local dynamics of the system, we first find the following non-negative equilibria by solving the following algebraic system

$$\begin{cases} A + \eta S_q - \frac{\alpha S_n}{\beta + S_n} S_q - \frac{\mu S_n}{\nu + S_n} I - \rho S_n = 0 \\ \frac{\alpha S_n}{\beta + S_n} S_q - S_q = 0 \\ \gamma S_q + \frac{\mu S_n}{\nu + S_n} I - \kappa I + dI = 0 \\ \sigma I + \varepsilon S_q - \rho R = 0. \end{cases}$$
(3.1)

We solve system (3.1) to obtain the disease free equilibrium (DFE) (S_q^0, I^0, R^0) , the quarantine population free equilibrium (QFE) $(\overline{S}_n, \overline{S}_q, \overline{I}, \overline{R})$ and the endemic equilibrium (EE) (S_n^*, S_q^*, I^*, R^*) . The equilibrium points take the following form

$$\begin{split} & \left(S_n^0, S_q^0, I^0, R^0\right) = \left(\frac{A}{\rho}, 0, 0, 0\right), \\ & \left(\overline{S}_n, \overline{S}_q, \overline{I}, \overline{R}\right) = \left(\frac{\nu(\kappa - d)}{\mu - \kappa + d}, 0, \frac{A(\mu - \kappa + d) - \nu\rho(\kappa - d)}{(\mu - k + d)(\kappa - d)}, \frac{A\sigma(\mu - \kappa + d) - \sigma\nu\rho(\kappa - d)}{\rho(\mu - k + d)(\kappa - d)}\right), \\ & \left(S_n^*, S_q^*, I^*, R^*\right) = \left(\frac{\beta}{\alpha - 1}, O, P, Q\right), \\ & \text{where} \end{split}$$

$$\begin{split} O &= \frac{\gamma (A\alpha - A - \rho\beta)(\nu\alpha - \nu + \beta)}{(\alpha - 1)[\gamma\mu\beta - (\eta - 1)((\kappa - d)(\nu\alpha - \nu + \beta) - \mu\beta)]}, \\ P &= \frac{1}{\gamma} \Big(\kappa - d - \frac{\mu\beta}{\nu\alpha - \alpha + \beta}\Big) S_q^* \quad \text{and} \quad Q = \frac{\alpha I^* + \varepsilon S_q^*}{\rho}. \end{split}$$



(a) Non-quarantined susceptible population



Fig. 3. a. Non-quarantined population $S_n(t)$ increases, as ν increases, b. Quarantined population $S_q(t)$ has no significant change with respect to ν , c. Infected population rapidly decreases, as ν increases, d. Recovered population also rapidly decreases, as ν increases. Here the other parameters are chosen from Table 2.

4



(b) Quarantined susceptible population



(a) Non-quarantined susceptible population



(b) Quarantined susceptible population



Fig. 4. a. Non-quarantimed population $S_n(t)$ decreases, as *d* increases, b. Quarantimed population $S_q(t)$ has no significant change with respect to *d*, c. Infected population rapidly increases, as *d* increases, d. Recovered population also rapidly decreases, as *d* increases. Here the other parameters are chosen from Table 2.

We perform mathematical analysis [22] of the model (2.2). We discuss the boundedness and non-negativity analysis, and determining the basic reproduction number for the model in the following manner.

3.1. Boundedness and non-negativity of solutions of the model

In this section, we propose Lemma 3.1 and Lemma 3.2 to confirm the boundedness and non-negativity [24] of solutions of system (2.2).

Lemma 3.1. The region $\Omega = \{(S_n(t), S_q(t), I(t), R(t)) \in \mathbb{R}^4_+\}$ is a positively invariant set of the model (2.2).

Proof. Let the total population size is N(t), where $N(t) = S_n(t) + S_q(t) + I(t) + R(t)$. Then the rate of change of the total population is $\frac{dN}{dt} = \frac{dS_n}{dt} + \frac{dS_q}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$. From the system (2.2), we find

$$\frac{dN}{dt} = A - \rho N + (\gamma + \eta + \varepsilon + \rho - 1)S_q + (d + \sigma + \rho - \kappa)I.$$
(3.2)

For large time, that is, for $t \to \infty$, the infected population and quarantined population become extinct. So, in the absence of quarantined susceptible population and infected population, that is, $S_q = I = 0$, we find from the equation (3.2)

$$\frac{dN}{dt} = A - \rho N, \tag{3.3}$$

which is analogous to a linear differential equation having the integrating factor, $e^{\int \rho dt}$. With this, solving (3.3) so that we find

$$N(t) = \frac{A}{\rho} + \left(N_0 - \frac{A}{\rho}\right) e^{-\rho t}.$$
(3.4)

Therefore we write, $\lim_{t\to\infty} N(t) = \frac{A}{\rho}$ which indicates that $N(t) \leq \frac{A}{\rho}$, that is, $\frac{A}{\rho}$ is the upper bound of N(t), that is, the solutions $(S_n(t), S_q(t), I(t), R(t))$ approach to the region Ω asymptotically. Hence the model (2.2) is mathematically and epidemiologically well-posed in the region Ω . This completes the proof of Lemma 3.1.



(a) Non-quarantined susceptible population



(b) Quarantined susceptible population



Fig. 5. a. Non-quarantined population $S_n(t)$ increases, as β , ν increases and d decreases, b. Quarantined population $S_q(t)$ decreases, as β , ν increases and d decreases, c. Infected population rapidly increases, as β , ν decreases and d increases, d. Recovered population also rapidly increases, β , ν decreases and d increases. Here the other parameters are chosen from Table 2.

Lemma 3.2. If $S_n(t) \ge 0$, $S_q(t) \ge 0$, $I(t) \ge 0$ and $R(t) \ge 0$, then the solutions of system (2.2) are non-negative.

Proof. To prove Lemma 3.2, we first consider the first equation of the system (2.2)

$$\frac{dS_n}{dt} = A + \eta S_q - \frac{\alpha S_n}{\beta + S_n} S_q - \frac{\mu S_n}{\nu + S_n} I - \rho S_n.$$
(3.5)

In order to find the non-negativity of the solution of (3.5), we find

$$\frac{dS_n}{dt} + \rho S_n \ge A. \tag{3.6}$$

Integrating yields,

$$S_n(t) \ge \frac{A}{\rho} + c e^{-\rho t} \tag{3.7}$$

where *c* is an integrating constant. Applying the initial condition at t = 0, we have $S_n(0) \ge \frac{A}{a} + c$. Finally, (3.7) takes the form

$$S_n(t) \ge \frac{A}{\rho} + \left(S_n(0) - \frac{A}{\rho}\right) e^{-\rho t}.$$
(3.8)

Hence (3.8) confirms that $S_n(t) \ge 0$ at t = 0 and $t \to \infty$. Therefore $S_n(t)$ is non-negative for all $t \ge 0$.

Similarly, we can show that $S_q(t) \ge 0$, $I(t) \ge 0$ and $R(t) \ge 0$ for all $t \ge 0$. This completes the proof of Lemma 3.2.

3.2. Basic reproduction number

In the case of the disease epidemic model, the basic reproduction number plays a vital role in the model because of the prediction of disease transmission [11,23,34]. We denote the basic reproduction number by Λ_0 , which is defined as the ratio of newly infected individuals with respect to total infected individuals. It is also widely used in epidemiology because it assures whether the disease persists or extinct. In this model (2.2), the non-quarantined susceptible population, quarantined susceptible population, and infected population contribute to spreading the infection among the non-infected population and here, there is no scope to spread the infection by the recovered or immune population. Therefore, to find out the basic reproduction number Λ_0 of the model, we consider the first three equations of system (2.2) as follows

$$\begin{cases} \frac{dS_n}{dt} = A + \eta S_q - \frac{\alpha S_n}{\beta + S_n} S_q - \frac{\mu S_n}{\nu + S_n} I - \rho S_n, \\ \frac{dS_q}{dt} = \frac{\alpha S_n}{\beta + S_n} S_q - S_q, \\ \frac{dI}{dt} = \gamma S_q + \frac{\mu S_n}{\nu + S_n} I - \kappa I + dI. \end{cases}$$
(3.9)

From (3.9), we can form the following matrices F and V for gain and loss terms, respectively

$$F = \begin{bmatrix} 0 & \eta & 0\\ \frac{\alpha S_q}{\beta + S_n} - \frac{\alpha S_n S_q}{(\beta + S_n)^2} & \frac{\alpha S_n}{\beta + S_n} & 0\\ 0 & 0 & 0 \end{bmatrix}$$
(3.10)

and

$$V = \begin{bmatrix} \frac{\alpha S_q}{\beta + S_n} - \frac{\alpha S_n S_q}{(\beta + S_n)^2} + \frac{\mu I}{\nu + S_n} - \frac{\mu I S_n}{(\nu + S_n)^2} + \rho & \frac{\alpha S_n}{\beta + S_n} & \frac{\mu S_n}{\nu + S_n} \\ 0 & 1 & 0 \\ -\frac{\mu I}{\nu + S_n} + \frac{\mu I S_n}{(\nu + S_n)^2} & -\gamma & -\frac{\mu S_n}{\nu + S_n} + \kappa - d \end{bmatrix}$$
(3.11)

At the disease free equilibrium point, $(S_n, S_q, I, R) = \left(\frac{A}{\rho}, 0, 0, 0\right)$, we find

$$F = \begin{bmatrix} 0 & \eta & 0 \\ 0 & \frac{\alpha A}{\beta \rho + A} & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \rho & \frac{\alpha A}{\beta \rho + A} & \frac{\mu A}{\nu \rho + A} \\ 0 & 1 & 0 \\ 0 & -\gamma & -\frac{\mu A}{\nu \rho + A} + \kappa - d \end{bmatrix} \text{ and}$$
(3.12)

$$V^{-1} = \begin{bmatrix} \frac{1}{\rho} & \frac{A}{\rho(\beta\rho + A)((d - \kappa)(A + \nu) + \mu A)} & \frac{\mu A}{\rho((d - \kappa)(A + \nu) + \mu A)} \\ 0 & 1 & 0 \\ 0 & \frac{\gamma(\nu\rho + A)}{(\kappa - d)(\nu\rho + A) - \mu A} & -\frac{\nu\rho + A}{(\kappa - d)(\nu\rho + A) - \mu A} \end{bmatrix}.$$

It is now possible to evaluate the next generation matrix FV^{-1} such that

$$FV^{-1} = \begin{bmatrix} 0 & \eta & 0 \\ 0 & \frac{\alpha A}{\beta \rho + A} & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$
 (3.13)

Thus the basic reproduction number of system (2.2) is

$$\Lambda_0 = \frac{\alpha A}{\beta \rho + A}.\tag{3.14}$$

4. Stability analysis of the model at equilibria

To perform the stability analysis of the model at the equilibrium points, we consider system (2.2) in vector form

$$\begin{cases} \dot{\mathbf{x}}(t) = f(t, \mathbf{x}) \\ \mathbf{x}(0) = \mathbf{x}_0 \end{cases}$$
(4.1)

where $x = (S_n(t), S_q(t), I(t), R(t)), f = (f_1, f_2, f_3, f_4)$ and $x_0 = (S_{n0}, S_{q0}, I_0, R_0)$ with

$$f_1(S_n, S_q, I, R) = A + \eta S_q - \frac{\alpha S_n}{\beta + S_n} S_q - \frac{\mu S_n}{\nu + S_n} I - \rho S_n$$

$$f_2(S_n, S_q, I, R) = \frac{\alpha S_n}{\beta + S_n} S_q - S_q$$

$$f_3(S_n, S_q, I, R) = \gamma S_q + \frac{\mu S_n}{\nu + S_n} I - \kappa I + dI$$

$$f_4(S_n, S_q, I, R) = \sigma I + \varepsilon S_q - \rho R.$$

For stability analysis, we linearize system (4.1) at an equilibrium point x^* so that the Jacobian matrix has the following form

$$J = \begin{bmatrix} \frac{\alpha\beta S_{q}}{(\beta + S_{n})^{2}} - \frac{\mu\nu I}{(\nu + S_{n})^{2}} - \rho & \eta - \frac{\alpha S_{n}}{\beta + S_{n}} & \frac{\mu S_{n}}{\nu + S_{n}} & 0\\ \frac{\alpha\beta S_{q}}{(\beta + S_{n})^{2}} & \frac{\alpha S_{n}}{\beta + S_{n}} - 1 & 0 & 0\\ \frac{\mu\nu I}{(\nu + S_{n})^{2}} & \gamma & \frac{\mu S_{n}}{\nu + S_{n}} - \kappa + d & 0\\ 0 & \varepsilon & \sigma & -\rho \end{bmatrix}.$$
(4.2)

4.1. Stability analysis of the model at disease free equilibrium point (S_n^0, S_q^0, I^0, R^0)

In order to investigate the stability of system (2.2) at the DFE (S_n^0, S_q^0, I^0, R^0) , we propose the following Theorem 4.1.

Theorem 4.1. The disease free equilibrium point of system (2.2) is locally asymptotically stable if $\Lambda_0 < 1$ and $\frac{\mu A}{\nu \rho + A} + d < \kappa$ and unstable otherwise.

Proof. The Jacobian matrix (4.2) can be evaluated at the DFE, (S_n^0, S_a^0, S_a^0)

$$I^{0}, R^{0}) = \begin{pmatrix} \frac{A}{\rho}, 0, 0, 0 \end{pmatrix} \text{ as follows}$$

$$J_{1} = \begin{bmatrix} -\rho & \eta - \frac{\alpha A}{\beta \rho + A} & -\frac{\mu A}{\nu \rho + A} & 0 \\ 0 & \frac{\alpha A}{\beta \rho + A} - 1 & 0 & 0 \\ 0 & \gamma & \frac{\mu A}{\nu \rho + A} - \kappa + d & 0 \\ 0 & \varepsilon & \sigma & -\rho \end{bmatrix}.$$
(4.3)

The eigenvalues of the matrix J_1 can be obtained in the following form

$$\lambda_1 = \frac{\alpha A}{\beta \rho + A} - 1, \ \lambda_2 = -\rho, \ \lambda_3 = \frac{\mu A}{\nu \rho + A} - \kappa + d, \quad \text{and} \ \lambda_4 = -\rho.$$

In terms of basic reproduction number Λ_0 , these can be rewritten as

$$\lambda_1 = \Lambda_0 - 1, \ \lambda_2 = -\rho, \ \lambda_3 = -\kappa + \frac{\mu A}{\nu \rho + A} + d, \quad \text{and} \ \lambda_4 = -\rho$$

It follows that the DFE is locally asymptotically stable if $\Lambda_0 < 1$ and $\frac{\mu A}{\nu \rho + A} + d < \kappa$ and unstable otherwise. According to the parameter values mentioned in Table 3, we find that DFE is unstable.

4.2. Stability analysis of the quarantined susceptible population free equilibrium point

For the stability of the quarantined susceptible population free equilibrium point $(\overline{S}_n, \overline{S}_q, \overline{I}, \overline{R}) = \begin{pmatrix} \nu(\kappa-d) \\ \mu-\kappa+d \end{pmatrix}, 0, \frac{A(\mu-\kappa+d)-\nu\rho(\kappa-d)}{(\mu-\kappa+d)(\kappa-d)}$

ив

$$\frac{A\sigma(\mu-\kappa+d)-\sigma\nu\rho(\kappa-d)}{\rho(\mu-k+d)(\kappa-d)}$$
, we propose the following Theorem 4.2.

Theorem 4.2. The quarantined susceptible population free equilibrium point of system (2.2) is locally asymptotically stable if $\Lambda_0 < 1$ and unstable if $\Lambda_0 > 1.$

Proof. At the quarantined susceptible population free equilibrium point $\left(\tfrac{\nu(\kappa-d)}{\mu-\kappa+d}, 0, \frac{A(\mu-\kappa+d)-\nu\rho(\kappa-d)}{(\mu-k+d)(\kappa-d)}, \frac{A\sigma(\mu-\kappa+d)-\sigma\nu\rho(\kappa-d)}{\rho(\mu-k+d)(\kappa-d)} \right), \text{ the Jacobian matrix (4.2) takes}$

the following form

Theorem 4.3. The endemic equilibrium point of system (2.2) is locally asymptotically stable if $\Lambda_0 > 1$ and unstable if $\Lambda_0 < 1$.

Proof. At the endemic equilibrium point $(S_n^*, S_q^*, I^*, R^*) = \left(\frac{\beta}{\alpha-1}, S_q^*, I^*, R^*\right)$

 R^*), the Jacobian matrix (4.2) becomes

$$J_{2} = \begin{bmatrix} -\frac{(\mu - \kappa + d)(A(\mu - \kappa + d) - \rho\nu(\kappa - d))}{\mu\nu(\kappa - d)} - \rho & \eta - \frac{\alpha\nu(\kappa - d)}{\beta(\mu - \kappa + d) + \nu(k - d)} & d - \kappa & 0\\ 0 & \frac{\alpha\nu(\kappa - d)}{\beta(\mu - \kappa + d) + \nu(\kappa - d)} - 1 & 0 & 0\\ \frac{(\mu - \kappa + d)(A(\mu - \kappa + d) - \rho\nu(\kappa - d))}{\mu\nu(\kappa - d)} & \gamma & 0 & 0\\ 0 & \varepsilon & \sigma & -\rho \end{bmatrix}.$$
(4.4)

The eigenvalues of the matrix J_2 can be obtained in the following form

The eigenvalues of the matrix
$$J_2$$
 can be obtained in the following form

$$\lambda_1 = -\frac{1}{2\mu\nu(\kappa-d)} \left(w_1 - \sqrt{w_2 + 2w_3 + w_4} \right),$$

$$\lambda_2 = -\frac{1}{2\mu\nu(\kappa-d)} \left(w_1 + \sqrt{w_2 + 2w_3 + w_4} \right),$$

$$J_3 = \begin{bmatrix} -\frac{(\alpha - 1)^2 S_q^*}{\alpha\beta} - \frac{\mu\nu I^*(\alpha - 1)^2}{(\nu\alpha - \nu + \beta)^2} - \rho & \eta - 1 & \frac{\mu\beta}{\nu\alpha - \nu + \beta} & 0 \\ \frac{(\alpha - 1)^2 S_q^*}{\alpha\beta} & 0 & 0 & 0 \\ \frac{\mu\nu I^*(\alpha - 1)^2}{(\nu\alpha - \nu + \beta)^2} & \gamma & \frac{\mu\beta}{\nu\alpha - \nu + \beta} - \kappa + d & 0 \\ 0 & \varepsilon & \sigma & -\rho \\ (4.5)$$

where

 $w_1 = A(\mu - \kappa + d)^2 + \nu \rho(\kappa - d)^2$

We determine the eigenvalues of the matrix
$$J_3$$
 in the following form

 $\lambda_{3} = \frac{\alpha \nu \eta (d - \kappa) + \beta \eta (d + \mu - \kappa) - d(\alpha \nu - \nu + \beta) + \nu \kappa (\eta + \alpha - 1) + \beta (\mu \gamma - \mu + \kappa)}{(\eta - 1)(\alpha \nu - \nu + \beta)},$

 $w_2 = A^2 \left(\mu - \kappa + d\right)^4$

$$w_3 = \nu A (d-\kappa)^2 (\mu-\kappa+d)^2 (2\mu+\rho)$$

$$w_4 = \rho \nu^2 (d - \kappa)^3 (4\mu^2 + (d - \kappa)(4\mu + \rho)).$$

It is observed that the eigenvalues have a complicated form that makes us unable to identify their nature (sign) applying Routh-Hurwitz criterion [16, 38]. In that case, we numerically confirm that the quarantined population free equilibrium of the model is locally asymptotically stable if $\Lambda_0 < 1$ and unstable if $\Lambda_0 > 1$ concerning the parameter values listed in the Table 3.

4.3. Stability analysis of the endemic equilibrium point

For the stability of the endemic equilibrium point (S_n^*, S_q^*, I^*, R^*) , we propose the following theorem.

$$\lambda_1 = \frac{u_1 + u_2}{\alpha \beta (\alpha \nu - \nu + \beta)^2}, \ \lambda_2 = \frac{(\alpha \nu - \nu + \beta)^2 (\alpha - 1)^2 (\eta - 1) S_q^*}{u_1 + u_2},$$

and $\lambda_4 = -\rho$

where

$$u_{1} = \alpha \beta \mu \nu I^{*} (\alpha - 1)^{2} + \alpha \beta \rho (\nu - \beta)^{2} + 2\nu \rho \alpha^{2} \beta (\beta - \nu)$$

$$u_{2} = 6\alpha \beta \nu S_{q}^{*} (1 - \alpha) + \alpha^{2} \nu^{2} S_{q}^{*} (\alpha^{2} - 4\alpha + 6) + \beta^{2} S_{q}^{*} (\alpha - 1)^{2} + \nu S_{q}^{*} (2\alpha^{3}\beta - 4\alpha\nu - 2\beta + \nu).$$

Due to the complexity of the explicit form of the eigenvalues, it is

impossible to confirm the nature (sign) of eigenvalues analytically using the Routh-Hurwitz criterion. However, we numerically confirm that the endemic equilibrium point is unstable ($\Lambda_0 < 1$) for the parameter values mentioned in Table 3 and it becomes asymptotically stable when we change the parameters *A*, *a*, and *b*, etc. so that $\Lambda_0 > 1$.

Finally, it is understood that the stability of the equilibria depends on the basic reproduction number Λ_0 . The basic reproductive ratio can also play a pivotal role to control the infection in epidemiology [14].

5. Numerical investigations

In order to investigate the dynamics of the model, we perform numerical simulations of the model as a standard Initial Value Problem (IVP) (2.2)–(2.3) incorporating a relevant initial condition.

For numerical solution of the IVP (2.2)–(2.3), we use Runge-Kutta fourth order method. The initial conditions are chosen from the same dataset [43] as $S_{n0} = 2.0, S_{q0} = 1.86, I_0 = 0.07, R_0 = 0.15$. We also take the parameter values from Table 2 so that $\kappa < d + \frac{\mu A}{\nu \rho + A}$ and the basic reproduction number, $\Lambda_0 = 0.6791$ (< 1). Under these conditions, it is expected that the quarantined susceptible population free equilibrium point, $(\overline{S}_n, \overline{S}_n, \overline{I}, \overline{R})$ is asymptotically stable.

We are mainly interested to investigate the influence of dispersal on the infection of COVID-19 pandemic in Bangladesh. In our model, the parameter *d* can explain the dispersal of infection of coronavirus in terms of the frequency of the inter-regional movement of the people (regional lockdown). In addition to that ν and β correspond to the effect of social distancing and immune growth of susceptible populations, respectively. That is why we emphasize these three parameters to understand the applicability of the model to the real situation of COVID-19.

We first focus on the parameter β which is responsible for immune growth through intaking vitamins and doing physical exercise although it has no significant influence on the infected population. People should increase their immunity during this pandemic so that COVID-19 symptoms do not come out and subsequently people do not undergo quarantine. This phenomenon can be reflected by the parameter β in the model. To investigate this situation, we increase β , that is, $\beta = 10.0$, we see that the quarantine population decreases and the non-quarantine population increases, as shown in Fig. 2. On the other hand, the quarantine population gradually increases as β decreases. This result suggests that COVID-19 symptoms can be prevented, that is, the frequency of quarantined people from non-quarantined can be controlled to improve the immunity level staying at home.

Fig. 3 exhibits the variations of the compartmental variables with respect to the time when the parameter ν varies. We emphasize the parameter ν which corresponds to the social awareness parameter, that is, wearing a mask, PPE and maintaining 3 m social distance from each other and frequently washing hands with soap, etc. when people are in the workplace or out of the home. When social awareness is well developed, that is, the parameter ν is suitably increased, it is observed that the infected population rapidly decreases, as shown in Fig. 3. It is also inspected that the solution of the IVP (4.1), $(S_n, S_q, I, R)(t)$ converges to (25.31, 0.0, 19.40, 0.15) for large time when the parameters chosen from Table 2. We also numerically confirm that for this parameter setting, the eigenvalues of the matrix J_2 in (4.4) are real and negative, that is, $\lambda_1 = -11.1659$, $\lambda_2 = -0.90$, $\lambda_3 = -0.2$, and $\lambda_4 = -0.07714$. This is a quite reasonable agreement to the stability analysis of $(\overline{S}_n, \overline{S}_q, \overline{I}, \overline{S}_q)$ \overline{R}), presented in Section 4.2. Consequently, we claim that the parameter ν has a significant influence to prevent the infection of novel coronavirus, that is, the COVID-19 outbreak.

We now focus on the main goal of this research, that is, the influence of dispersal on the infection of COVID-19 in Bangladesh. The dispersal effect is implemented by the parameter d in the model through the strict restriction of the movement of people from one region to another region. If the movement of people is highly restricted, that is, lockdown is strictly imposed, the parameter d is relatively smaller. On the other hand, a relatively larger value of *d* corresponds that the people can move often from one region to another region, that is, lockdown is not maintained. To this end, we can say that the parameter *d* controls the flexibility of the movement of people. To do so, we change the parameter *d* keeping the other parameters fixed to understand the spreading dynamics of COVID-19 all over the country. Fig. 4c demonstrates that the infected population rapidly increases due to the increase of the dispersal rate *d*. On the contrary, when we decrease the value of *d*, for instance *d* = 0.001, Fig. 4c shows that the infected population decreases than that for *d* = 0.1 and *d* = 0.032. Numerical results reveal that the spread of COVID-19 over the country could be controlled reasonably if we impose restrictions on the inter-district movements of the people. Finally, we can claim that regional lockdown can play an important role to reduce the infection of novel coronavirus and subsequently control the outbreak of COVID-19 in Bangladesh.

Fig. 5 exhibits the collective influence of the parameters β , ν and d on the COVID-19 epidemic. We see that the infected population and recovered population decreases when we increase β , ν and decrease d simultaneously. The non-quarantined population increases if the parameters β , ν are increased and d is decreased. When β , ν are increased and d is decreased. When β , ν are increased and d is decreases. Finally, we may conclude from the model that the outbreak of COVID-19 could be prevented by taking effective measures, that is, controlling the parameters β , ν and d simultaneously in terms of increasing immune level, proper social distancing, and regional lockdown, respectively.

6. Conclusions

COVID-19 has been declared as a global pandemic by the World Health Organization (WHO) [42] on March 11, 2020. The coronavirus COVID-19 pandemic, a major public health concern, is the greatest challenge we have faced since the second world war. In all the affected countries over the world, it has the potential to create devastating social, economic, and political crises and that will leave deep scars. The impact of the COVID-19 pandemic is not only felt on the national economy but also the household economy of millions of Bangladeshis. It is merely important to slow the spread of the novel coronavirus by testing and treating patients, carrying out contact tracing, limiting travel, quarantining citizens, and canceling all sorts of large gatherings for a reasonable period. To impose sustainable preventive measures, in this paper, we proposed a four-compartmental model by introducing the dispersal effect and social distancing effect on the infection of COVID-19. We analyzed the boundedness and non-negativity of solutions of the model. To confirm whether the infection of the coronavirus persists or extinct, we determined the basic reproduction number for the model. We performed a stability analysis at the equilibria to identify epidemic and endemic cases in terms of the basic reproduction number, Λ_0 . We numerically confirmed that the quarantined susceptible population-free equilibrium $(\overline{S}_n,\overline{S}_q,\overline{I},\overline{R})$ is locally asymptotically stable if $\Lambda_0<1$ and the endemic equilibrium $\left(\frac{\beta}{\alpha-1}, S_q^*, I^*, R^*\right)$ is locally asymptotically stable if $\Lambda_0 > 1$. Furthermore, we performed numerical simulations to illustrate the analytical findings. The infection of coronavirus is regulated if the movement of the people from one region to another region is highly restricted, that is, regional lockdown is strictly imposed and that was reflected in the model through the dispersal effect parameter d. We have seen that the infection is also controlled when the level of social distancing, such as keeping a 1-m distance from one to another, frequent handwashing with soap, wearing face mask and PPE, etc. is well developed, that is, ν is suitably increased. Finally, the numerical results suggest that the infection of novel coronavirus could be prevented and controlled in Bangladesh when the dispersal effect, that is, regional lockdown is strictly maintained and the level of social distancing is well-developed as non-pharmaceutical interventions.

Declaration of competing interest

The authors have no interests to declare.

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