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Prediction and mathematical analysis of the outbreak of coronavirus (COVID-19) in Bangladesh

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A B S T R A C T
In this study based on Bangladesh, a modified SIR model is produced and analysed for COVID-19. We have theoretically investigated the model along with numerical simulations. The reproduction number ($R_0$) has been calculated by using the method of the next-generation matrix. Due to the basic reproduction number, we have analysed the local stability of the model for disease-free and endemic equilibria. We have investigated the sensitivity of the reproduction number to parameters and calculate the sensitivity indices to determine the dominance of the parameters. Furthermore, we simulate the system in MATLAB by using the fourth-order Runge–Kutta (RK4) method and validate the results using fourth order polynomial regression (John Hopkins Hospital (JHH), 2020). Finally, the numerical simulation depicts the clear picture of the upward, and the downward trend of the spread of this disease along with time in a particular place, and the parameters in the mathematical model indicate this change of intensity. This result represents, the effect of COVID-19 from Bangladesh's perspective.

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1. Introduction

Nowadays, different countries of the world are waylaying with a new transmittable disease named novel coronavirus (COVID-19). The International Committee on Taxonomy of Viruses (ICTV) proclaimed the accredited classification of the new coronavirus (2019-nCoV) as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on 11 February 2020. Subsequently, the World Health Organisation (WHO) officially announced that Coronavirus Disease-19 (COVID-19) is responsible for this pandemic [1]. Along with MERS-nCoV and SARS-nCoV, COVID-19 is the seventh member of the coronavirus family that can be transmitted to humans [2]. Including the common symptoms, e.g., fever, cough, shortness of breath, diarrhoea, respiratory, digestive, liver, and nervous system disorders [1].

Recent studies showed that apart from the syndrome mentioned above, COVID-19 patients possess severe consequenses. It is now clear that respiratory failure in pneumonia or Acute Respiratory Distress Syndrome (ARDS) alone cannot explain these sudden deaths; there are more varied causes of sudden death [3]. Why are deaths happening so fast? Which again cannot be fully explained by X-ray or CT scan pneumonia. Some people start to have severe shortness of breath quickly due to lack of oxygen, which is again incompatible with a chest X-ray or CT scan. So, what is happening in the body of patients? As reported, Corona itself is a hypercoagulable state, meaning that it causes blood clots to form in blood vessels. Pulmonary embolism (PE) is the medical term for the blockage of blood vessels in the lungs. However, even if it does not cause sudden death, the frozen area becomes dark or gangrene [4]. Moreover, in Bangladesh, people aged 21 to

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30 are mostly affected, which is unusual in comparison to the statistics of other countries. On the other hand, patients above 60 mostly died from this disease [5].

Bangladesh is facing a severe shortage of testing kits — it does not have more than 100 thousand testing kits in stock and not more than 20-thousands of which have been distributed to different areas around the country [6–8]. As coronavirus has no vaccine yet, and hence, Bangladesh’s government is trying to apply quarantine, social distancing and, lockdown policy. As it is confirmed that the disease may still be infectious during the period of latent infection, and it can spread from person to person through respiratory droplets and close contact [9].

The pioneer epidemiologist Kermack and McKendrick (1927) extensively used mathematical models to get features of these infectious diseases [10]. Following the footprints of Kermack and McKendrick, some of the eminent models have been proposed for the transmission dynamics of some illnesses which include: HIV model [11]; Heathcote and Yorke (1984) model for the spread and control of gonorrhoea [12]; Ronald Ross model for control of malaria [13]; Glass network [14,15] etc. At present, researchers are trying to find a mathematical model of COVID-19. It is impossible to generate a unique model that can predict the adverse effect in different countries. As a result, people around the world are trying to get the root of this pandemic generated by a coronavirus [16].

Since the SIR model mainly describes three classes — susceptible, infected, and recovery. On the other hand, a modified SIR model includes another three sections, such as exposed quarantine and death class, as well as, the susceptible class is divided into two categories — exposed and quarantine. Exposed individuals mainly indicate the people are infected due to COVID-19 but not infectious. Quarantine plays a vital role in controlling the outbreak of the transmission. However, in the paper [17], Quarantine is a limitation method where persons assumed to have been presented to an infectious disease though yet to be ill because they are not infected or for, they are still in the development period.

Quarantine is functional in the case of an individual or a group and usually includes the restriction on movement to the home with the facility. So, a new compartment quarantine class can be induced to control the spreading of COVID-19 infectious disease. Conversely, the well-known SIR model only deals with infected individuals. If the people die by COVID-19, the death class connected with quarantine and the infected class. This technique mostly affects and predicts the outbreak of Bangladesh likewise a modified from the SIR model.

The coronavirus again attacks some people, and so are exhibited a mathematical model of recovery individuals are connected to the susceptible class, which is different from the SIR model. However, the infected class includes a parameter. That means the rate of infection is implemented that played an important role in reducing the spreading of COVID-19. Mathematically, in this paper, we have proved what will be the effect of COVID-19 in Bangladesh. We checked the numerical results utilizing MATLAB. Sometimes researchers used unique or a couple of software to validate numerical results [15,18–20], but in this manuscript, we utilize fancy software MATLAB.

Till today, the spread of coronavirus (COVID-19) contagious disease is announced among 216 countries. So far, many countries have experienced deadly consequences due to the outbreak of this disease. It does cause not only health problems but also social, educational, and economical problems along with causalities. As of now, WHO has pronounced that COVID-19 does not have an inoculation yet, and sagacious steps are induced through isolation and observatory systems. To analyse such event, compartmental models are ubiquitous in science [21–24]. The outbreak of COVID-19 that triggered the fact of defending infectious occurrence by means of mathematical modelling [25–27]. For reckoning [28], models search are the centrepieces for the effective posterior enhancement of the disease [29,30].

In brief, this work aims to develop a SEQIRP model based on the SIR model to describe the epidemic due to COVID-19 in Bangladesh [24]. The analysis of the model will give an insight into the stable and unstable situation based on the reproduction number [31,32]. Moreover, sensitivity analysis shows which parameter is more dominating for the system, and numerical simulation also depicts the variation of parameters at different times to predict the COVID-19 epidemic in Bangladesh.

2. Model formulation

One can get a piece of reliable and valuable information about disease control and spread employing epidemiological models. So, our goal is to investigate the situation of COVID-19 in Bangladesh. In this case, we have developed the SEQIRP (Susceptible–Exposed–Quarantine–Infected–Recovered–Death) model developed from the originator of the SIR model [10]. In a brief description, the SEQIRP model divides the total population into six compartments each of which is a representative of time $t$.

To be precise, $S(t)$ represents the susceptible class that is uninfected, $E(t)$ represents infected people but not infectious, $Q(t)$ represents the quarantine which may involve in uninfected, infected, and death. $I(t)$ represents the number of infected people that have to be cured or dead, and $R(t)$ represents the recovered individuals having a cure from the disease. Finally, $P(t)$ represents the disease dies the people: $N(t)$, the total number of populations at time $t$. The total number of population $(N)$ divided into six different classes: Susceptible $(S)$, Exposed $(E)$ Quarantine $(Q)$, Infected $(I)$, Recovered $(R)$, Death $(P)$. That is,

$$N(t) = S(t) + E(t) + Q(t) + I(t) + R(t) + P(t)$$

The compartmental description of the model is illustrated in Fig. 1. The model maintains the following properties:

(i) Susceptible people are transmitted into exposed class and quarantine class at rate of $\beta_1$ and $\beta_2$ respectively.
(ii) Exposed class people become infectious at a rate $\omega$ and exposed class people are dying at a rate $\mu$. 2
Table 1
Parameter description.

| Parameter | Description |
|-----------|-------------|
| $\beta_1$ | Contact rate of susceptible class to exposed class (1/day). |
| $\beta_2$ | Contact rate of susceptible class to quarantine class (1/day). |
| $\omega$  | Transmission coefficient of exposed class to infectious class (1/day). |
| $\mu$    | The death rate of exposed class to death class (1/day). |
| $\varepsilon$ | Transferred rate of quarantine class to susceptible class (1/day). |
| $\varphi$ | Transferred rate of quarantine class to infectious class (1/day). |
| $\sigma$  | The death rate of quarantine class to death class (1/day). |
| $\gamma$ | The rate of recovery (1/day). |
| $\delta$ | The rate of death (1/day). |
| $\alpha$ | The rate of susceptibility (1/day). |

(iii) Quarantine people are moved into susceptible class at $\varepsilon$ transferred rate because the people are uninfected due to COVID-19, also, the people are infectious and death class transmitted at a certain rate $\varphi$ and $\sigma$.

(iv) Infected people are removed from the disease at a rate $\gamma$.

(v) The disease dies infected people at rate $\delta$.

(vi) Recovered people become susceptible again at a rate $\alpha$.

(vii) We have assumed that the disease of COVID-19 spreads mainly from infected and the rate of incidence is of the form $\frac{(\beta_1+\beta_2)S(t)I(t)}{N}$.

Based on these assumptions, the compartmental structure and flow directions of the model can be described using a directed flow chart as illustrated as follows:

\[\begin{align*}
\frac{dS(t)}{dt} &= -\frac{(\beta_1+\beta_2)}{N}S(t)I(t) + \varepsilon Q(t) + \alpha R(t) \\
\frac{dE(t)}{dt} &= \frac{\beta_1 S(t) I(t)}{N} - \omega E - \mu E \\
\frac{dQ(t)}{dt} &= \frac{\beta_2 S(t) I(t)}{N} - \varepsilon Q(t) - \varphi Q(t) - \sigma Q(t) \\
\frac{dI(t)}{dt} &= \omega E - \gamma I(t) - \delta I(t) + \varphi Q(t) \\
\frac{dR(t)}{dt} &= \gamma I(t) - \alpha R(t) \\
\frac{dp(t)}{dt} &= \mu E + \sigma Q(t) + \delta I(t)
\end{align*}\]

where birth rate and natural death rate are constant. In Table 1, the parameters used in the model are defined:

Now, knowing that $S(t), E(t), Q(t), I(t), R(t)$ and $P(t)$ are fractions of the population, we can say that:

\[s(t) + e(t) + q(t) + i(t) + r(t) + p(t) = 1\]

where $s(t) = \frac{S(t)}{N}, e(t) = \frac{E(t)}{N}, q(t) = \frac{Q(t)}{N}, i(t) = \frac{I(t)}{N}, r(t) = \frac{R(t)}{N}, p(t) = \frac{P(t)}{N}$
Now we can assume the system (1) as follows:
\[
\begin{align*}
\frac{ds(t)}{dt} &= -(\beta_1 + \beta_2)s(t)i(t) + \varepsilon q(t) + \alpha r(t) \\
\frac{de(t)}{dt} &= \beta_1 s(t)i(t) - \omega e(t) - \mu e(t) \\
\frac{dq(t)}{dt} &= \beta_2 s(t)i(t) - \varepsilon q(t) - \varphi q(t) - \sigma q(t) \\
\frac{di(t)}{dt} &= \omega e(t) - \gamma i(t) - \delta i(t) + \varphi q(t) \\
\frac{dr(t)}{dt} &= \gamma i(t) - \alpha r(t) \\
\frac{dp(t)}{dt} &= \mu e(t) + \sigma q(t) + \delta i(t)
\end{align*}
\] (2)

3. Theoretical analysis of the model

In the following section, we carry out the theoretical analysis of the system (2).

3.1. Equilibria

At equilibrium, the Left-Hand Side (LHS) of the system equations of (2) will be zeros, i.e.
\[
\frac{ds(t)}{dt} = 0, \quad \frac{de(t)}{dt} = 0, \quad \frac{dq(t)}{dt} = 0, \quad \frac{di(t)}{dt} = 0, \quad \frac{dr(t)}{dt} = 0
\]

The equilibrium points without an infection called disease-free equilibria [33,34]. So, the system (2) has always the disease-free equilibrium at \(E^0 = (S^0, 0, 0, 0, 0) = (1, 0, 0, 0, 0)\). The equilibrium points with an infection called endemic equilibrium. So, the endemic equilibrium point from the system (2) is given by:
\[
s = \frac{\varepsilon q + \alpha r}{(\beta_1 + \beta_2)i} = S^*, \quad e = \frac{\beta_1 si}{(\omega + \mu)} = E^*, \quad q = \frac{\beta_2 si}{\varepsilon + \varphi + \sigma} = Q^*, \quad i = \frac{\omega e + \varphi q}{\gamma + \delta} = I^*, \quad r = \frac{\gamma i}{\alpha} = R^*
\]

Thus, \(E^* = (S^*, E^*, Q^*, I^*, R^*)\) is the unique endemic equilibrium of the system (2).

3.2. Basic reproduction number

In the study of disease transmission, the basic reproduction number shows the disease spread and control. If \(R_0 < 1\), then the disease-free equilibrium is stable, the disease ceases to exist in the community. If \(R_0 > 1\), the endemic equilibrium exists, and the disease forever stays in the community. Using the Next-generation matrix, we obtain the reproduction number [13,31,35,36]. Let \(x = (E, Q, I)\), then it follows from system (2):
\[
\frac{dx}{dt} = F - \nu,
\]
\[
F = \begin{pmatrix}
\beta_1 s & 0 \\
0 & \beta_2 s \\
0 & 0
\end{pmatrix}
\]
and \(\nu = \begin{pmatrix}
(\omega + \mu)e \\
(\varepsilon + \varphi + \sigma)q \\
-\omega e - \varphi q + \delta i + \gamma i
\end{pmatrix}
\]

The disease-free equilibrium point of the system (2) has coordinates \(E^0 = (0, 0, 0, 0, 0)\). The derivatives of \(F\) and \(\nu\) at \((0, 0, 0, 0, 0)\).
\[
F = \begin{pmatrix}
0 & 0 & \beta_1 \\
0 & 0 & \beta_2 \\
0 & 0 & 0
\end{pmatrix}, \quad \nu = \begin{pmatrix}
\omega + \mu & 0 & 0 \\
0 & \varepsilon + \varphi + \sigma & 0 \\
-\omega & -\varphi & \gamma + \delta
\end{pmatrix}
\]

The next-generation matrix for the system (2) is
\[
\begin{pmatrix}
\frac{\omega \beta_1}{(\omega + \mu)(\gamma + \delta)} & \frac{q \beta_1}{(\omega + \mu)(\gamma + \delta)} \\
\frac{\omega \beta_2}{(\omega + \mu)(\gamma + \delta)} & \frac{\omega \beta_2}{(\omega + \mu)(\gamma + \delta)} \\
0 & 0
\end{pmatrix}
\]
The eigenvalues of the matrix $FV^{-1}$ are $\lambda_1 = \lambda_2 = 0$ by and $\lambda_3 = \frac{1}{(\delta + \gamma)} \left[ \frac{\beta_1 \omega}{(\omega + \mu)} + \frac{\beta_2 \varphi}{(\epsilon + \varphi + \sigma)} \right]$. Hence $R_0$ is the maximum (dominant) of the two eigenvalues of $FV^{-1}$. Thus we have

$$R_0 = \frac{1}{(\delta + \gamma)} \left[ \frac{\beta_1 \omega}{(\omega + \mu)} + \frac{\beta_2 \varphi}{(\epsilon + \varphi + \sigma)} \right]$$

which is the required basic reproduction number for the system (2).

3.3 Stability analysis

In this section, we have discussed the local stability of the disease-free and endemic equilibrium.

**Theorem 1.** When $R_0 < 1$, the disease-free equilibrium $E_0$ is locally asymptotically stable. If $R_0 = 1$, $E_0$ is locally stable. When $R_0 > 1$, the infection-free equilibrium $E_0$ is an unstable saddle point.

**Proof.** The Jacobian matrix at the point $E_0$ given by

$$J (E_0) = \begin{pmatrix}
0 & 0 & \varepsilon & - (\beta_1 + \beta_2) & \alpha \\
0 & (\omega + \mu) & 0 & \beta_1 & 0 \\
0 & (\epsilon + \varphi + \sigma) & \beta_2 & 0 & 0 \\
0 & \varphi & (\gamma + \delta) & 0 & 0 \\
0 & 0 & \gamma & -\alpha & 0
\end{pmatrix}$$

(4)

By [17,33] $E_0$ is locally asymptotically stable if all the eigenvalues of $J (E_0)$ have a negative real part that means $R_0 < 1$. Again $E_0$ is unstable if at least one of the eigenvalues of $J (E_0)$ has a positive real part that means $R_0 > 1$. Now, from $J(E_0)$ we get the two eigenvalues: $\lambda_1 = 0$, $\lambda_2 = -\alpha$, the other eigenvalues of are $J(E_0)$ determined by the following equation

$$\lambda^3 + C_3 \lambda^2 + C_4 \lambda + C_5 = 0$$

(5)

where $A = (\omega + \mu), B = (\epsilon + \varphi + \sigma), C = (\gamma + \delta), C_3 = (A + B + C) > 0, C_4 = (AB + AC + BC - \beta_2 \varphi - \beta_1 \omega), C_5 = ABC - A\beta_2 \varphi - B\beta_1 \omega$

$$C_4 = \left(AB + AC (1 - R_0) + BC (1 - R_0) + \frac{B}{A} \omega \beta_1 \right) > 0 \text{ if } R_0 < 1$$

$$C_5 = ABC (1 - R_0) > 0 \text{ if } R_0 < 1$$

$$C_3 C_4 - C_5 = CC_4 + C \left(A^2 + B^2 \right) (1 - R_0) + \frac{B^2}{C} \omega \beta_1 + \frac{A^2}{C} \varphi \beta_2 + AB^2 + A^2 B + ABC > 0 \text{ if } R_0 < 1$$

The Routh–Hurwitz criteria is satisfied as $C_3 > 0, C_4 > 0, C_5 > 0$ and $C_3 C_4 - C_5 > 0$ if $R_0 < 1$.

Therefore, all the eigenvalues of Eq. (5) have a negative real part. On the other hand, one eigenvalue is zero that means the reproduction number $R_0 = \frac{1}{(\delta + \gamma)} \left[ \frac{\beta_1 \omega}{(\omega + \mu)} + \frac{\beta_2 \varphi}{(\epsilon + \varphi + \sigma)} \right]$ lead us to the value 1. precise information of the system (4) based on [37]. The rest eigenvalues of the system (4) have a negative real part. This concludes the analysis of disease-free equilibrium of the system (4), which means $E_0$ is locally stable [37–39].

**Theorem 2.** When $R_0 > 1$, the endemic equilibrium is $E^*$ locally asymptotically stable.

**Proof.** The Jacobian matrix of the model (2) at $E^* = (S^*, E^*, Q^*, I^*, R^*)$ is

$$J (E^*) = \begin{pmatrix}
- (\beta_1 + \beta_2) I^* & 0 & \varepsilon & - (\beta_1 + \beta_2) S^* & \alpha \\
\beta_1 I^* & - (\omega + \mu) & 0 & \beta_1 S^* & 0 \\
0 & (\epsilon + \varphi + \sigma) & \beta_2 S^* & 0 & 0 \\
0 & \varphi & (\gamma + \delta) & 0 & 0 \\
0 & 0 & \gamma & -\alpha & 0
\end{pmatrix}$$

$$\text{trace } J (E^*) = - (\beta_1 + \beta_2) I^* - (\epsilon + \varphi + \sigma) - (\gamma + \delta) - \alpha < 0$$

Now det

$$J (E^*) = -\alpha (\beta_1 + \beta_2) (\omega + \mu) (\gamma + \delta) (\epsilon + \varphi + \sigma) I^* + \varepsilon \alpha \beta_2 (\omega + \mu) (\gamma + \delta) I^* + \alpha \gamma (\omega + \mu) (\epsilon + \varphi + \sigma) (\gamma + \delta) I^* + (\beta_1 + \beta_2) ABCI^* + \alpha \gamma ABC I^* + \alpha \gamma (R_0 - 1) I^* > 0$$

$$= -\alpha (\beta_1 + \beta_2) ABCI^* + \varepsilon \alpha \beta_2 (\omega + \mu) ABCI^* + \alpha \gamma ABC I^* + \alpha \gamma (R_0 - 1) I^* > 0$$
provided the condition
If $R_0 > 1$ and $\varepsilon \alpha \beta_2 (\omega + \mu) ACI^* + \alpha \gamma ABCI^* + \alpha \gamma (R_0 - 1) I^* > -\alpha (\beta_1 + \beta_2) ABCI^*$

By [33], the endemic equilibrium of the system (3) $E^*$ has a negative real part. Thus, we conclude that the endemic equilibrium $E^*$ of the system (3) is locally asymptotically stable so $R_0 > 1$. This completes the proof.

4. Sensitivity analysis

Beginning disease transmission is straightforwardly identified with the reproduction number. We calculate the sensitivity indices of the basic reproduction number ($R_0$) to the parameters in the model to determine which of the parameter have a high impact on $R_0$, and consequently to the disease transmission. We follow the approach as in [11,40]. The normalized sensitivity indices of $R_0$ on parameter $P_i$ is given by

$$I_{R_0}^{P_i} = \frac{\partial R_0}{\partial P_i} \frac{P_i}{R_0}$$

Presently, we investigate the sensitivity analysis of $R_0$ with respect to the model parameters. Here we are utilizing the parameter values from Table 3. We have got $R_0$ from Eq. (3), which has an explicit expression $R_0 = \frac{1}{(\delta + \gamma)} [\frac{\phi \beta_2}{(\omega + \mu) + \gamma} + \frac{\alpha \gamma}{(\omega + \mu)}]$. In the following way, we have got the sensitivity of parameters.

- $I_{R_0}^{P_1} = \frac{\partial R_0}{\partial P_1} \frac{P_1}{R_0}$, we put the values of parameter from Table 3, we have obtained 0.44
- $I_{R_0}^{\gamma} = \frac{\partial R_0}{\partial \gamma} \frac{\gamma}{R_0}$, we plug the values of parameter from Table 3, we have found the value -0.80
- $I_{R_0}^{\delta} = \frac{\partial R_0}{\partial \delta} \frac{\delta}{R_0}$, we put the values of parameter from Table 3, we get the value -0.16.

We observe the following results from the analysis of the indices:

(i) From, $I_{R_0}^{P_1}$ we can say that if increasing $P_1$ by 10% increases by 10% $|0.44| = 0.044$ of $R_0$, which can prompt an outbreak and if $\beta_1$ decreases 10% so $R_0$ decreases by 10% $|0.44| = 0.044$ i.e. the value of $\beta_1$ plays a major role in reducing the value of $R_0$. Hence, it is required to make the rate of $\beta_1$ reduced in order to restrain the disease. This is why measures such as social distance and quarantine are being promoted by the WHO and the government to control the epidemic.

(ii) $I_{R_0}^{\gamma}$ is negative and from the value, we can say increasing (decreasing) $\gamma$ by 10% decreases (increases) $R_0$ by 10% $|0.80| = 0.080$. Therefore, COVID patients need to be isolated to restrict the spread of the disease.

(iii) Similarly, from $I_{R_0}^{\delta}$ is negative and from the value we can say increasing (decreasing) $\delta$ by 10% decreases (increases) $R_0$ by 10% $|0.16| = 0.016$. Therefore, the COVID patients need to be isolated to restrict the spread of the disease.

In Fig. 2, the actual infection rate will be at its peak at 150th days (July 2020) and the peak value will be around 0.36. On the other hand, if we change the initial value such as adding 0.01 then we see the perturb line gives the peak situation in 116th days (May 2020). According to the deviation results, on 135th days (July 2020) the deviation of the actual value and the perturb value is 0.33 and the actual line surpass the perturb line. From Fig. 2, we can see that after the 400th days (May 2021) the infection rate is heading below 0.1 which implies the disease will be stable in Bangladesh.

From Fig. 3, the actual recovery rate is upward after 400th days (April 2021) and the value is around 0.56 and the perturb line shows that the recovery rate is also upward and the value is around 0.57. That means if we change the initial value adding 0.001 along with the initial value, the recovery rate is increasing and the deviation is 0.007 on the 400th days (April 2021).

Fig. 4 shows the death rate is upward. The perturb graph of the death rate and the actual graph is very close after the 350th days (March 2021). The deviation result for two different initial values is 0.002 on around 400th days (April 2021). From Fig. 5, we can see that the susceptible people get recovered from the disease day by day so the infectious people are back to the original compartment. Adding 0.001 to the initial value then the recovery rate goes upward more quickly. Eventually, the people are back to the original position. The actual line of the rate of susceptible people and the perturb line is very close after the 200th days. Finally, on the 400th days (April 2021) the deviation value is very negligible.

5. Numerical simulations and discussion

We are going to discuss the numerical simulations and findings of the system (1) in this section. We have solved the system by using the fourth-order Runge–Kutta method in the ambiance of MATLAB. The epidemiologic information was taken from an open-source storehouse worked by the Johns Hopkins University Centre for Systems Science and Engineering [JHU CSSE] [41]. We also used data taken from the World meter [42]. The initial variables and parameters used in the implementation of the model are shown in Tables 2 and 3 below. We have considered the data from 18 March to 24 October of Bangladesh.

System (1) is composed of ten parameters. Due to the pandemic situation, it is difficult to find the desired parameter so we have used some assumed data. On the other hand, we have used some values of the parameter estimated from [41,42].
For example, we depicted analytics formulae for three values of the parameter that have been used in the following way:

Consider $\xi_i = \text{Average}(\sum_{j=1}^{n} (\eta_{1j+1} - (\eta_{1j}))$; $i = 1, 2, 3$, where $(\eta_{1j})_{j+1}$ and $(\eta_{1j})_{j}$ are the representative of the exposed people who are infected by COVID-19 in today and yesterday respectively. Similarly, $(\eta_{2j})_{j+1}$ and $(\eta_{2j})_{j}$ infectious people who are recovered from COVID-19 in today and yesterday respectively. Finally, $(\eta_{3j})_{j+1}$ and $(\eta_{3j})_{j}$ are the infectious people who are death due to COVID-19 in today and yesterday respectively. All these $\xi_1, \xi_2, \xi_3$ are entitled as $\beta_1, \gamma, \delta$ respectively.

Although, the hardest part of mathematical biology is the estimation of parameters. There are several issues, among them lack of proper information is the most exciting one. We do have some parameters that are assumed either from a reference or estimated from the information available in reliable sources of Bangladesh like i.e. [5].
Table 3
Values of the parameters used in the numerical experiments. Some Parameters value are calculated from data like $\beta_1$, $\gamma$ and $\delta$, some for-example infectiousness rate $\omega$, $\mu$, and $\phi$, etc take from WHO or other credible sources.

| Parameters | Values      | Data source  |
|------------|-------------|--------------|
| $\beta_1$  | 0.0517901   | Estimated    |
| $\beta_2$  | 0.2         | Assumed      |
| $\epsilon$ | 0.6         | Assumed      |
| $\omega$   | 0.07        | Assumed      |
| $\mu$      | 0.003370    | Assumed      |
| $\phi$     | 0.35        | Assumed      |
| $\sigma$   | 0.01        | Assumed      |
| $\gamma$   | 0.012227    | Estimated    |
| $\delta$   | 0.002522    | Estimated    |
| $\alpha$   | 0.000001    | Assumed      |
Fig. 6. Solutions of the system (top left panel) based on MATLAB. Superimposition of existing available data with fourth-order polynomial regression [41]. Simulation results show the rate of infection (right top panel), rate of recovery (left bottom panel), and death (right bottom panel) of the Bangladesh coronavirus pandemic that make ties with the analysis of this manuscript.

Utilizing values of (Tables 2 and 3) along with fourth-order RK method and fourth order polynomial regression, we found the following Fig. 6:

Fig. 6 shows that the effect of the infection rate is certainly upward. On the other hand, after a certain period of time, the rate of infection came downward. We can see that from the figure and predict that during July, the rate of the infection was going to be peak position then slowly reduce after 150 days i.e., middle month of July 2020 (counting from March 18). The infection rate of the disease is slowly reduced after 350 days i.e., March 2021. According to the real scenario of Bangladesh, the number of infected people was highest in July and then started decreasing slowly. The number of the infected population is largely depended on the rate of infection of the COVID-19.

Finally, in Fig. 6, The red line represents the current trends in the number of infections, recovered, and death cases. We used fourth-order polynomial regression to find the trendline and shows that the rate of death is almost constant. So, if we undertake prudent steps, we can fight against COVID-19. Otherwise, infected people will improve day by day, and people will die. Also, the figure manifests, the rate of death is going to rises around 0.18 at 400th days (April 2021). In the real scenario of Bangladesh, the number of deaths has been raised from the month of May 2020 which is matches our work. Unless any individual comes from other countries, our prediction will be correct, but we are afraid that lots of Bangladeshi people work in foreign countries. Due to the pandemic, the world is experiencing a substantial economic crisis, which may make many people working outside the country jobless, and, subsequently, they will come back here to Bangladesh.

We know, WHO has already declared no vaccine has come to light yet. If we maintain a prudent step in society, the rate of infection will decrease, which is more important for every country and also in Bangladesh. Also, in Fig. 6, it is
visible that the rate of recovery was also in a sharp increase after 100th days (May 2020). When the rate of infection is fluctuating, we see that rate of recovery rate is upward, which indicates the number of infectious people is going healthy every day.

We observe from Fig. 6, the rate of recovery is going to rise almost 0.56 at 350th days (March 2021). According to Fig. 6, in the real scenario of Bangladesh, the number of recovery people has increased after May 2020 and the number of recovery people is going on steady after the specified time which coincides with our research. Obviously, the recovery rate is essential for our research. Therefore, if the number of people does not remain constant due to the reason as mentioned earlier, the prediction may vary.

Eventually, temperature plays a tricky role in increasing coronavirus transmission. A study regarding such an issue is addressed from the perspectives of Bangladesh in [43]. Apart from Bangladesh, many other countries have been tracked out such issues. So, the winter season is not magniloquent for Bangladeshi people. Since coronavirus is quite comfortable with it, so that awareness will play a very crucial role in decreasing coronavirus transmission.

6. Discussion and conclusion

The simulation predicts the rate of infection will increase, and the disease is spreading in Bangladesh rapidly. Also, the parameters of infection present us when the condition is downward in Bangladesh. From this simulation, it can be predicted that the outbreak was at its peak during the month of July 2020, and the rate was around 0.36. After, this the value will be dipping.

Coronavirus has no drug or vaccine yet to come, so the Bangladesh government or the related authority should implement social awareness, quarantine, and social distancing. Also, prudent steps like lockdown-policies are valuable for decreasing the rate of infection. Bangladesh is a developing country, and lots of people live in the poverty level or below the poverty line, so long-time lockdown policy is not fruitful for the economy as well as for the people. Also, the healthcare infrastructure of Bangladesh is not capable of providing medication to a massive number of COVID-19 positive patients. Therefore, besides slowing down the spread of the COVID-19 virus during this pandemic (flattening the curve), the capacity of the healthcare facilities should also be increased. However, the rate of recovery will be rising position; it means that people will recover from the disease gradually. Also, the rate of death increases day by day.

A pandemic of coronavirus (COVID-19) outbreak, mostly in developing nations, could be a genuine danger to tranquil presence. As exhibited by our mathematical models, it is seen that an uncontrolled transmittable contact between the infected and the susceptible can hasten the disease, since no immunity and vaccine yet. In this study, the resulting model has been solved analytically as well as by numerical simulations. From the demonstrated modified SIR model, the equilibria and stabilities have also been presented, and the calculation of the sensitivity indices to determine which parameters are more dominating.

On the other hand, numerical simulations are a vital tool in dealing with predicting the outbreak situation of any disease. To simulate the mathematical model, we have applied the fourth-order Runge–Kutta method in MATLAB (programs are appended in the appendix). Moreover, we tested our results with fourth-order polynomial regression with the sources of data. We get ties in both scenarios which lead us to give a conjecture that the second web of coronavirus is about to hit by November 2020 and prolong its fatal consequences in all aspects of our outcome.

This simulation shows the variation of the rate of infection, rate of recovery, and the rate of death at different times. The number of infected populations is remarkably dependent on the rate of infection. The rate of infection will continue despite the increased rate of recovery and death since no vaccine has come yet at this moment. So, with the observatory system and quarantine, social distancing and social awareness are mandatory to control the rate of infection that means to control the spreading of the disease. We would like to demarcate the end line of this work. The model SEQIRP predicts the Bangladesh epidemic rate of infection that was thrust out its peak from July 2020, with the rate of recovery and death also climbing.

CRediT authorship contribution statement

Pabel Shahrear: Conceptualization, Formal analysis, Resources, Writing - original draft, Writing - review & editing, Supervision. S. M. Saydur Rahman: Conceptualization, Software, Validation, Formal analysis, Resources, Writing - original draft, Visualization. Md Mahadi Hasan Nahid: Software, Validation, Data curation, Visualization.

Declaration of competing interest

No author associated with this paper has disclosed any potential or pertinent conflicts which may be perceived to have impending conflict with this work. For full disclosure statements refer to https://doi.org/10.1016/j.rinam.2021.100145.

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Appendix. MATLAB program for SEQIRP model

```matlab
% The Fourth-Order Runge-Kutta code to solve SEQIRP problem
% Runge Kutta code to solve SEQIRP problem
clear clc
% Parameters
a = 0.00001;
b1 = 0.05790;
b2 = 0.2;
c = 0.6;
w=0.01;
n=0.003370;
p = 0.35;
s = 0.01;
l = 0.01227;
d = 0.002528;
N = 164375277;

% Define Functions
fS = @(t,S,I,R) - ((b1+b2)*S*(1/N)*w*S*R);
fI = @(t,S,I,R) (b1*S*(1/N)*w*S*R);
fR = @(t,S,I,R) l*I*

% Initial Conditions
t(1) = 0;
S(t) = 164285245;
I(t) = 14;
Q(t) = 90000;
R(t) = 14;
\%
% Step Size
h = 0.001;
tfina = 500;
\%
% Loop for t
for i=1:n;
    \%
    % Runge Kutta Constants Calculation
    k1S=ES(t(i), S(i), I(i), R(i), Q(i));
k1R=ER(t(i), S(i), I(i), R(i), Q(i));
k1I=EI(t(i), S(i), I(i), R(i), Q(i));
k1Q=EQ(t(i), S(i), I(i), R(i), Q(i));
k2S=ES(t(i)+h/2, S(i)+h/2*k1S, I(i)+h/2*k1I, R(i)+h/2*k1R, Q(i)+h/2*k1Q);
k2R=ER(t(i)+h/2, S(i)+h/2*k1S, I(i)+h/2*k1I, R(i)+h/2*k1R, Q(i)+h/2*k1Q);
k2I=EI(t(i)+h/2, S(i)+h/2*k1S, I(i)+h/2*k1I, R(i)+h/2*k1R, Q(i)+h/2*k1Q);
k2Q=EQ(t(i)+h/2, S(i)+h/2*k1S, I(i)+h/2*k1I, R(i)+h/2*k1R, Q(i)+h/2*k1Q);
k3S=ES(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k3R=ER(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k3I=EI(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k3Q=EQ(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k4S=ES(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k4R=ER(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k4I=EI(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
k4Q=EQ(t(i)+h, S(i)+h*k1S, I(i)+h*k1I, R(i)+h*k1R, Q(i)+h*k1Q);
\%
% Updating S, Q, I, R & D
S(i+1)=S(i)+h/6*(k1S + 2*k2S + 2*k3S + k4S);
I(i+1)=I(i)+h/6*(k1I + 2*k2I + 2*k3I + k4I);
Q(i+1)=Q(i)+h/6*(k1Q + 2*k2Q + 2*k3Q + k4Q);
R(i+1)=R(i)+h/6*(k1R + 2*k2R + 2*k3R + k4R);
\%
% Plotting Solution
figure(1);clf(1)
pcolormap('cmap'); lineWidth',2.0)
hold on
plot(t,S,'c','LineWidth',2.0)
hold on
plot(t,I,'b','LineWidth',2.0)
hold on
plot(t,R,'r','LineWidth',2.0)
hold on
plot(t,Q,'g','LineWidth',2.0)
hold on
xlabel('Time/Days')
ylabel('Proportion')
legend('Susceptible','Exposed','Quarantine','Infected','Recovered','Death')
yticklabels({'0','0.2','0.4','0.6','0.8','1.0'})
end
```

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