# A new Compartmental Mathematical Model for COVID 19 Transmission: Bangladesh Study

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#### Abstract

The outbreak of the Coronavirus COVID-19 has taken the lives of several thousands worldwide and locked out many countries and regions, with yet unpredictable global consequences. In this work, we propose a compartmental mathematical model for the spread of the COVID-19 disease with special focus on the transmissibility of symptomatic, asymptomatic or super-spreaders individuals. We estimate the basic reproduction number to our propose model using general and exponential growth rate formula.

A case study of transmission of Covid-19 in Bangladesh has been done by our proposed model. The efficiency and some limitations of our model also explained in the paper.

**Keywords:** Covid-19 Coronavirus, Mathematical Modeling, Basic Reproduction Number, SEIR Model, Exponential Growth Rate, Case Study

Mathematics Subject Classification Number 2020: 34A05, 34A07, 34B05, 34B07

Date of Submission: 24-06-2024 Date of Acceptance: 03-07-2024

Date of Submission. 24-00-2024 Date of Acceptance. 03-07-202-

#### I. Introduction

Since the outbreak of the COVID-19 coronavirus in early 2020, the virus has affected most countries and taken the lives of several thousands of people worldwide. By March 2020, the World Health Organization (WHO) declared the situation a pandemic, the first of its kind in our generation. From early 2020, many countries and regions have been locked-down and applied strict social distancing measures to stop the virus propagation. From a strategic and healthcare management perspective, the propagation pattern of the disease and the prediction of its spread over time is of great importance, to save lives and to minimize the social and economic consequences of the disease. The problem of interest has been studied in various communities including mathematical epidemiology [1], [2], biological systems modeling [3], [4], signal processing [5] and control engineering [6].

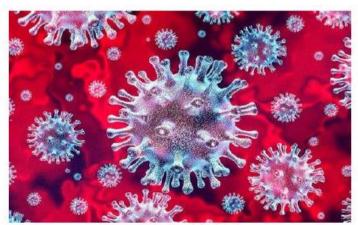


Figure 1: Covid-19 virus

A number of modeling studies have already been performed for the COVID-19 epidemic. Wu et al. [12] introduced a susceptible-exposed-infectious-recovered (SEIR) model to describe the transmission dynamics, and forecasted the national and global spread of the disease, based on reported data from December 31, 2019 to January 28, 2020. They also estimated that the basic reproductive number for COVID-19 was about 2.68. Wu et al. [12] reported a value of 3.1 for the basic reproductive number based on data fitting of a SEIR model, using an assumption of Poissondistributed daily time increments. Tang et al. [7, 9] proposed a deterministic compartmental model incorporating the clinical progression of the disease, the individual epidemiological status, and the intervention measures. They found that the control reproductive number could be as high as 6.47, and that intervention strategies such as intensive contact tracing followed by quarantine and isolation can effectively reduce the control reproduction number and the transmission risk. Imai et al [12] conducted computational modeling of potential epidemic trajectories to estimate the size of the disease outbreak in Wuhan, with a focus on the human-to-human transmission. Their results imply that control measures need to block well over 60% of transmission to be effective in containing the outbreak. In addition, Gao et al. [13] developed a deep learning algorithm to analyze the infectivity of the novel coronavirus and predict its potential hosts. Their results indicate that bats and minks may be two animal hosts of this virus. Most of these models have emphasized the significant role of the direct, human-to-human transmission pathway in this epidemic, as highlighted by the facts that the majority of the infected individuals did not have any contact with the marketplaces in Wuhan, that the number of infections has been rapidly increasing, and that the disease has spread to more than 213 other countries.

Meanwhile, the transmission rates in our model depend on the epidemiological status and environmental conditions which change with time. In particular, when the infection level is high, people would be motivated to take necessary action to reduce the contact with the infected individuals and contaminated environment so as to protect themselves and their families, leading to a reduction of the average transmission rates. Such varied transmission rates also reflect the strong disease control measures that the whole world has implemented, including large-scale quarantine, intensive tracking of movement and contact, strict isolation, and advising the public to stay home and avoid spreading infection. The remainder of this paper is organized as follows.

In section II, we describe the general mathematical and compartmental models, In section III, we explain about the measure and estimation of reproduction number. In section IV, we describe about the methodology of our work. In section V, we introduce our proposed model. In section VI, we explain the numerical case study to our proposed model in Bangladesh. In section VII, we discuss about the efficiency and some limitations of our model.

#### II General Mathematical and Compartmental Models

Mathematical modeling is the art of translating problems from an application area into tractable mathematical formulations whose theoretical and numerical analysis provides insight, answers, and guidance useful for the originating application. A model is an entity that resembles a system or object in certain aspects, but is easier to work with as compared to the original system. Models are used for the 1) identification and better understanding of systems, 2) simulation of a system's behavior, 3) prediction of its future behavior, and ultimately 4) system control.

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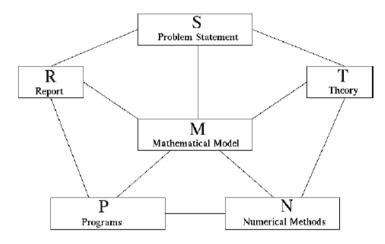


Figure 2: Diagram of mathematical modeling

The nodes of the following diagram represent information to be collected, sorted, evaluated, and organized.

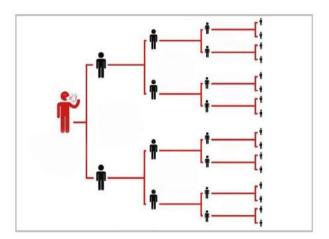
A compartmental model is a type of mathematical model (that is to say, a model that can be described by a set of mathematical equations) that simulates how individuals in different "compartments" in a population interact. The people (or animals) in each compartment are assumed to be the same as all the other people (or animals) in that compartment. The compartments of the model can either flow between each other (for instance live individuals can flow to a "dead" compartment with a certain rate, which is 1/lifetime), or they can interact (for instance, predators eat prey... the prey doesn't "flow" into the predator class, but obviously the number of predators that can survive is related to the number of prey available for them to eat... and the survival of prey is obviously related to the number of predators around to eat them). Differential (difference) equations arise in many modeling problems.

With this background, the basic steps of compartmental modeling are:

- 1) Identifying the quantities of interest as distinct compartments and selecting a variable for each quantity as a function of time. These variables are the state variables of the resulting state-space equations.
- 2) Linking the compartments with arrows indicating the rate of quantity flow from each compartment to another (visually denoted over the arrows connecting the compartments).
- 3) Writing the corresponding first-order (linear or nonlinear) differential equations for each of the state variables of the model. In writing the equations from the graph representation, the edge weights multiplied by the state variable of their start node are added to (subtracted from) the rate change equation of the end node (start node). External inputs can be considered to be originated from an external node with value 1.
- 4) Setting initial conditions and solving the system of equations (either analytically or numerically), which is in the form of a first-order state-space model.

# III Measuring and Estimating Reproduction $\mathcal{R}_0$

In epidemiology, the basic reproduction number, or basic reproductive number (sometimes called basic reproduction ratio or basic reproductive rate), denoted  $\mathcal{R}_0$  (pronounced R nought or R zero), of an infection can be thought of as the expected number of cases directly generated by one case in a population where all individuals are susceptible to infection. Counting the number of cases of infection during an epidemic can be extremely difficult, even when public health officials use active surveillance and contact tracing to attempt to locate all infected persons. Although measuring the true  $\mathcal{R}_0$  value is possible during an outbreak of a newly emerging infectious pathogen that is spreading through a wholly susceptible population, rarely are there sufficient data collection systems in place to capture the early stages of an outbreak when  $\mathcal{R}_0$  might be measured most accurately. As a result,  $\mathcal{R}_0$ is nearly always estimated retrospectively from sero-epidemiologic data or by using theoretical mathematical models. Data-driven approaches include the use of the number of susceptible persons at endemic equilibrium, average age at infection, final size equation, and intrinsic growth rate. When mathematical models are used,  $\mathcal{R}_0$  values are often estimated by using ordinary differential equations, but high-quality data are rarely available for all components of the model. The estimated values of  $\mathcal{R}_0$  generated by mathematical models are dependent on numerous decisions made by the modeler.



**Figure 3:** Disease transmission when Reproduction number  $\mathcal{R}_0 = 2$ 

The population structure of the model, such as the susceptible-infectious-recovered model or susceptible-exposed-infectious-recovered model, which includes compartments for persons who are exposed but not yet infectious, as well as assumptions about demographic dynamics (e.g., births, deaths, and migration over time), are critical model parameters. Population mixing and contact patterns must also be considered; for example, for homogeneous mixing, all population members are equally likely to come into contact with one another, and for heterogeneous mixing, variation in contact patterns are present among age subgroups or geographic regions. Other decisions include whether to use a deterministic (yielding the same outcomes each time the model is run) or stochastic (generating a distribution of likely outcomes on the basis of variations in the inputs) approach and which distributions (e.g., Gaussian or uniform distributions) to use to describe the probable values of parameters, such as effective contact rates and duration of contagiousness. Furthermore, many of the parameters included in the models used to estimate  $\mathcal{R}_0$  are merely educated guesses; the true values are often unknown or difficult or impossible to measure directly. This limitation is compounded as models become more complex and, thus, require more input parameters, such as when using models to estimate the value of  $\mathcal{R}_0$  for infectious pathogens with more complex transmission pathways, which can include vector borne infectious agents or those with environmental or wildlife reservoirs. In summary, although only 1 true  $\mathcal{R}_0$  value exists for an infectious disease event occurring in a particular place at a particular time, models that have minor differences in structure and assumptions might produce different estimates of that value, even when using the same epidemiologic data as inputs. In commonly used infection models, when  $\mathcal{R}_0 > 1$  the infection will be able to start spreading in a population, but not if  $\mathcal{R}_0 < 1\mathcal{R}_0 < 1$ . Generally, the larger the value of  $\mathcal{R}_0$ , the harder it is to control the epidemic.

# IV Methodology

Our proposed model has been made on base of the following SEIR model

#### SEIR Model

It is an extended SIR model [14] where a new compartment or state is added. It is known as Exposed or E which is positioned between the susceptible and infectious compartments. The Exposed individual is infected but not infectious, i.e. the disease remains in latent state. This concept can also be explained on the basis of the level of pathogen within the host and immunological status of the host. When the host is susceptible, it indicates that no pathogen is present and only a low level of non-specific immunity exists within the host. As soon as the susceptible encounters an infectious individual, he becomes infected. The pathogen increases in number and the infected host may not show any signs of infection and thus he enters the Exposed compartment. As soon as the pathogen burden is sufficiently high, the Exposed host becomes Infectious and disease is transmitted to another susceptible individual. When the Infectious individual can no longer transmit infection as the pathogen is cleared from his immunity system, he belongs to the Recovered category. The class distinction between Exposed-Infectious and Infectious-Recovered is not very distinct because of variability in responses between different individuals and variability in pathogen levels over the infectious period.

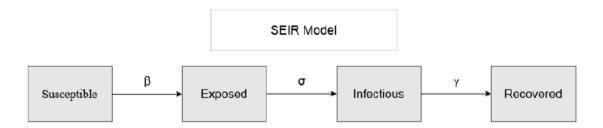


Figure 4: Diagram of SEIR Model

Many diseases have a latent phase during which the individual is infected but not yet infectious. This delay between the acquisition of infection and the infectious state can be incorporated within the SIR model by adding a latent/exposed population, E, and letting infected (but not yet infectious) individuals move from S to E and from E to I.

#### V Our Propose Model

General Description

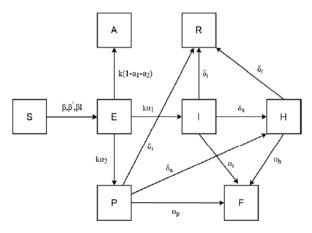


Figure 5: Flowchart of the model

We propose a new epidemiological compartment model that takes into account the super-spreading phenomenon of some individuals. Moreover, we consider a fatality compartment, related to death due to the virus infection. In doing so, the constant total population size N is subdivided into eight epidemiological classes: susceptible class (S), exposed class (E), symptomatic and infectious class (I), super-spreaders class (P), infectious but asymptomatic class (A), hospitalized (H), recovery class (R), and fatality class (F). Also we construct the qualitative analysis of the model as determining the reproduction number, local stability and sensitivity analysis.

We also assume that new births are susceptible people. We do not consider here movement of people between territories. Under those assumptions, the evolution of the compartments mentioned above is modeled by the following system of ordinary differential equations (which is simplified below):

$$\begin{split} &\frac{dS}{dt} = -\beta \frac{I}{N}S - I\beta \frac{H}{N}S - \beta' \frac{P}{N}S \\ &\frac{dE}{dt} = \beta \frac{I}{N}S + I\beta \frac{H}{N}S + \beta' \frac{P}{N}S - kE \\ &\frac{dI}{dt} = k\alpha_1 E - (\delta_a + \delta_i)I - \sigma_i I \\ &\frac{dP}{dt} = k\alpha_2 E - (\delta_a + \delta_i)P - \sigma_p P \\ &\frac{dA}{dt} = k(1 - \alpha_1 - \alpha_2)E \\ &\frac{dH}{dt} = \delta_a(1 + P) - \delta_r H - \sigma_h H \\ &\frac{dR}{dt} = \delta_i(1 + P) + \delta_r H \\ &\frac{dF}{dt} = \sigma_i I + \sigma_p P + \sigma_h H \end{split}$$

#### Where,

- $\beta =$  Human to human transmission coefficient per unit time per person.
- β = High transmission coefficient due to super spreaders.
- l = The relative transmissibility of hospitalized patients.
- k = Rate at which an individual leaves the exposed class by becoming infectious.
- α<sub>1</sub> Proportion of progression from exposed class E to infectious class I.
- α<sub>2</sub> = Relative very low rate at which exposed individual becomes super spreader.
- δ<sub>a</sub> = Average rate at which symptomatic and super spreader individuals become hospitalized.
- δ<sub>i</sub> = Recovery rate without being hospitalized.
- δ<sub>r</sub> = Recovery rate of hospitalized patients.
- $\sigma_i, \sigma_p, \sigma_h =$  Death rates due to infected, super spreaders and hospitalized patients.

Calculating the Basic Reproduction Number and Local Stability

It can be understood as the average number of cases one infected individual generates, over the course of its infectious period, in an otherwise uninfected population. Using the next generation matrix approach [15] to our propose model (1), the basic reproduction number can be computed by considering the below generation matrices F and V, that is, the Jacobian matrices associated to the rate of appearance of new infections and the net rate out of the corresponding compartments, respectively,

Where

$$w_i = \delta_a + \delta_i + \sigma_i$$
,  $w_p = \delta_a + \delta_i + \sigma_p$ ,  $w_h = \delta_r + \sigma_h$  (2)

The basic reproduction number  $\mathcal{R}_0$  is obtained as the spectral radius of F  $V^{-1}$  precisely,

$$\mathcal{R}_0 = \frac{\beta \alpha_1 (\delta_a l + w_h)}{w_i w_h} + \frac{(\beta \delta_a l + \beta' w_h) \alpha_2}{w_p w_h}$$
(3)

Noting that the two last equations and the fifth of system (1) are uncoupled to the remaining equations of the system, we can easily obtain, by direct integration, the following analytical results:

$$A(t) = k(1 - \alpha_1 - \alpha_2) \int_0^t E(s) ds$$

$$R(t) = \delta_i \int_0^t (I(s) + P(s)) ds + \delta_r \int_0^t H(s) ds$$

$$F(t) = \sigma_i \int_0^t I(s) ds + \sigma_p \int_0^t P(s) ds + \sigma_h \int_0^t H(s) ds$$

$$(4)$$

Since the total population size N is constant, one has

$$S(t) = N - [E(t) + I(t) + P(t) + A(t) + H(t) + R(t) + F(t)]$$
(5)

Therefore, the local stability of model (1) can be studied through the remaining coupled system of state variables, namely, the variables E, I, P, and H in (1). The Jacobian matrix associated to these variables of (1) is the following one:

$$J_{M} = \begin{bmatrix} -k & \beta & \beta l & \beta' \\ k\alpha_{1} & -w_{i} & 0 & 0 \\ k\alpha_{2} & 0 & -w_{p} & 0 \\ 0 & \delta_{2} & \delta_{3} & -w_{p} \end{bmatrix}$$
(6)

where  $w_i$ ,  $w_p$  and  $w_h$  are defined in (2). The eigenvalues of the matrix  $J_M$  are the roots of the following characteristic polynomial:

$$Z(\lambda) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4$$
 (7)

Where

$$\mathbf{a}_1 = \mathbf{k} + \mathbf{w}_1 + \mathbf{w}_p + \mathbf{w}_h$$

$$a_2 = -\beta k\alpha_1 - \beta' k\alpha_2 + kw_h + kw_i + kw_p + w_h w_i + w_h w_p + w_i w_p$$

$$\begin{aligned} a_3 &= -\beta \delta_a k l \alpha_1 - \beta \delta_a k l \alpha_2 - \beta k \alpha_1 w_h - \beta^{'} k \alpha_2 w_h - \beta k \alpha_1 w_p - \beta^{'} k \alpha_2 w_i + k w_h w_i + k w_h w_p \\ &+ k w_i w_p + w_h w_i w_p \end{aligned}$$

$$a_4 = -\beta \delta_a k l \alpha_2 w_i - \beta \delta_a k l \alpha_1 w_p - \beta' k \alpha_2 w_i w_h - \beta k \alpha_1 w_p w_h + k w_h w_i w_p$$

$$\tag{8}$$

Now by using the Liénard-Chipard test, all the roots of  $Z(\lambda)$  are negative or have negative real part if, and only if, the following conditions are satisfied:

1. 
$$a_i > 0$$
 ,  $i = 1, 2, 3, 4$   
2.  $a_1 a_2 > a_3$ 

In order to check these conditions of the Liénard-Chipard test [9], we rewrite the coefficients  $a_1, a_2, a_3, a_4$  of the characteristic polynomial in terms of the basic reproduction number given by (3)

$$\mathbf{a}_1 = \mathbf{k} + \mathbf{w}_i + \mathbf{w}_p + \mathbf{w}_h$$

$$\begin{split} a_2 &= (1-\mathcal{R}_0) \Big(kw_i + kw_p\Big) + kw_p \frac{\beta\alpha_1}{w_i} + kw_i \frac{\beta^{'}\alpha_2}{w_p} + \beta\delta_a kl\alpha_1 \Big(\frac{1}{w_h} + \frac{w_p}{w_h w_i}\Big) \\ &+ \beta\delta_a kl\alpha_2 \Big(\frac{1}{w_h} + \frac{w_i}{w_h w_p}\Big) + (k+w_i)w_h + (w_h+w_i)w_p \end{split} \tag{8}$$

$$\begin{split} a_3 &= k(1-\mathcal{R}_0) \Big( w_h w_p + w_h w_i + w_i w_p \Big) + k w_p \frac{\beta \alpha_1 w_h}{w_i} + k w_i \frac{\beta^{'} \alpha_2 w_h}{w_p} \\ &+ \beta \delta_a k l \alpha_1 w_p \Big( \frac{1}{w_h} + \frac{1}{w_i} \Big) + \beta \delta_a k l \alpha_2 w_i \Big( \frac{1}{w_h} + \frac{1}{w_p} \Big) + w_h w_i w_p \\ a_4 &= k w_h w_i w_p (1-\mathcal{R}_0) \end{split}$$

From the above expressions we can obtain that

$$\begin{split} a_{1}a_{2}-a_{3} &= (1-\mathcal{R}_{0})(k+w_{i})kw_{i}+(1-\mathcal{R}_{0})\Big(k+w_{p}+w_{h}\Big)kw_{p} \\ &+ \Big(k+w_{i}+w_{p}\Big)\bigg(\frac{\beta\alpha_{1}}{w_{p}}+\frac{\beta\delta_{a}l\alpha_{1}}{w_{i}}\bigg)kw_{p}+\Big(k+w_{i}+w_{p}\Big)\bigg(\frac{\beta^{'}\alpha_{2}}{w_{p}}+\frac{\beta\delta_{a}l\alpha_{2}}{w_{p}}\bigg)kw_{i} \\ &+ (k+w_{i}+w_{h})\frac{\beta\delta_{a}kl\alpha_{1}}{w_{h}}+\Big(k+w_{h}+w_{p}\Big)\frac{\beta\delta_{a}kl\alpha_{2}}{w_{h}}+(k+w_{i})w_{h} \\ &+ (w_{h}+w_{i})w_{p} \end{split} \label{eq:equation:equat$$

From these previous expressions, it is clear that if  $\mathcal{R}_0 < 1$ , then the conditions of the Liénard-Chipard test [10] are satisfied and, as a consequence, the disease free equilibrium is stable. In the case when  $\mathcal{R}_0 > 1$ , we have that  $a_4 < 0$  and, by using Descartes' rule of signs, we conclude that at least one of the eigenvalues is positive. Therefore, the system is unstable.

# VI Case Study in Bangladesh

The virus was confirmed to have spread to Bangladesh in March 2020. The first three known cases were reported on 8 March 2020 by the country's epidemiology institute, IEDC. After that some precautions like lockdown, social distancing and wearing mask campaign were taken by the government and the authorities. Though the spread of corona virus became alarming day by day in Bangladesh. From 7 April 2020 to 4 August 2020, we conduct a case study of 120 days measuring the basic reproduction number  $\mathcal{R}_0$  by (1) Exponential growth rate & (2) Our proposed model.

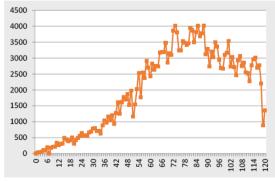


Figure 6: Daily confirmed infected cases (7/4/2020 to 04/08/2020)

(9)

Calculating reproduction number by Exponential Growth Rate and general formula

We estimates  $\mathcal{R}_0$  using epidemic data of covid-19 by the exponential growth rate r. Thus process requires two statistical process. First estimate r and then convert r into $\mathcal{R}_0$ .

J(t) be the number of new infections at time t.

Supposing that each infected individual on average generates secondary cases at a rate  $A(\tau)$  at time  $\tau$ since( $\tau$  is referred as the infection age).

$$J(t) = \int_0^\infty A(\tau)(t - \tau)d\tau \tag{11}$$

Since  $\mathcal{R}_0$  represent the total number of secondary cases that a primary case generates during the entire course of infection, the estimation is

$$\mathfrak{R}_0 = \int_0^\infty A(\tau) d\tau \tag{12}$$

When J(t) follows an exponential growth path, it is easy to extract the integral of  $A(\tau)$  from equation (7). So we have

$$J(t) = Ke^{rt}$$
, K is a constant  $J(t - \tau) = Ke^{rt}e^{-rt}$ 

$$J(t - \tau) = Ke^{rt}e^{-rt}$$

This simplifies equation (11) to the so called Eular-Lotka equation

$$I = \int_0^\infty A(\tau) e^{-r\tau} d\tau \tag{13}$$

 $g(\tau)$  represents the frequency of secondary transmission relative to infection age  $\tau$ .

$$g(\tau) = \frac{A(\tau)}{\int_0^\infty A(s) \, ds} = \frac{A(\tau)}{\Re_0} \tag{14}$$

Replacing  $A(\tau)$  in the right hand side of equation (9) by that of equation (10),  $\mathcal{R}_0$  is obtained

$$\mathfrak{R}_0 = \frac{1}{\int_0^\infty g(\tau)e^{(-r\tau)} d\tau} = \frac{r}{g(\tau)} \tag{15}$$

Now from our collected data of Bangladesh, we can construct the below table with required information:

Notation	Value	Description
X	242022	Total confirmed cases of period t
P	164,689,383	Total population of the area
X <sub>0</sub>	35	Initial confirmed case when t=1
t	120	Total number of days

Table 1: Parameter values for Exponential growth rate

Now we know by exponential growth rate formula

$$X = X_0(1+r)^t$$

$$\Rightarrow$$
 242022 = 35(1 + r)<sup>120</sup>

$$\rightarrow r - 0.0765$$

Also  $g(\tau)$  = the frequency of secondary transmission of period  $\tau$ .

$$\label{eq:gtau} \begin{cal} $ \dot{\cdot} \ g(\tau) = \frac{\mbox{Total confirmed cases of a period}}{\mbox{Total population of the area}} \times 100 \end{cases}$$

$$\Rightarrow g(\tau) = \frac{242022}{164689383} \times 100 = 0.147$$

From equation (15) putting the values of r &  $g(\tau)$  we find

$$\mathcal{R}_0 = \frac{\mathbf{r}}{\mathbf{g}(\tau)} = \frac{0.0765}{0.147}$$
$$= 0.52$$

Here the value of  $\mathcal{R}_0$  is less than 1. So the virus will die out after a certain period.

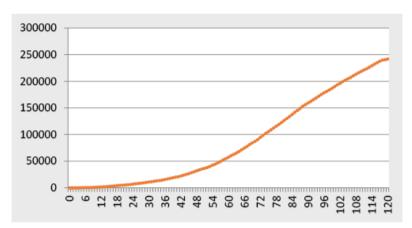


Figure 6(A): Cumulative number of confirmed infected cases (7/4/2020 to 04/08/2020)

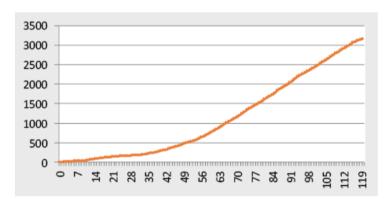


Figure 6(B): Cumulative number of deaths (7/4/2020 to 04/08/2020)

β	3.9787	Day <sup>-1</sup>
β΄	11.9361	Day <sup>-1</sup>
1	1	dimensionless
k	0.25	Day <sup>-1</sup>
α <sub>1</sub>	0.7	dimensionless
$\alpha_2$	0.001	dimensionless
$\delta_a$	0.05	Day <sup>-1</sup>
$\delta_{\rm i}$	0.9	Day <sup>-1</sup>
$\delta_{ m r}$	0.9	Day <sup>-1</sup>
$\sigma_{\mathbf{i}}$	2.1040	Day <sup>-1</sup>
$\sigma_{\rm p}$	1	Day <sup>-1</sup>
$\sigma_{\mathbf{h}}$	0.1	Day <sup>-1</sup>

Table 2: Calculated and Assumed parameter values for our proposed model

We put these parameter values in equation (3) and calculate the basic reproduction number

$$\mathcal{R}_0 = \frac{\beta \alpha_1 (\delta_a l + w_h)}{w_i w_h} + \frac{\left(\beta \delta_a l + \beta' w_h\right) \alpha_2}{w_p w_h} = 0.96$$

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#### VII Results and Discussion

SIR & SEIR model divide the whole population into 3 or 4 compartments where the compartments are susceptible, exposed, infected and removed/recovered. But in the cases of very infectious disease, there can be other compartments in the population like symptomatic, asymptomatic and super spreaders which we found in Covid-19 transmission played a very important role and could make vital differences in the calculation. Also SIR & SEIR model do not take different compartment for recovered and removed population, but we took them as different compartment in our model to calculate the more accurate picture of the transmission of Covid-19. The basic reproduction number  $\mathcal{R}_0$  for SIR model is  $\mathcal{R}_0 = \frac{\beta}{\gamma}$  from which we can see  $\mathcal{R}_0$  depends on only two factor  $\beta$  (infection rate) &  $\gamma$  (recovery rate). But in our proposed model the basic reproduction number  $\mathcal{R}_0 = \frac{\beta \alpha_1(\delta_a l + w_h)}{w_l w_h} + \frac{(\beta \delta_a l + \beta w_h)\alpha_2}{w_p w_h}$  in which  $\mathcal{R}_0$  is dependent on some more factors like high transmission coefficient due to super spreaders, the relative transmissibility of hospitalized patients, rate at which an individual leaves the exposed class by becoming infectious etc. These factors help to determine more accurate numerical value of the reproduction number.

At the time of writing, well-documented limitations in testing capacity in Bangladesh and a lack of information on exposed individuals, super spreaders and hospitalized patients, made it challenging to know where on the epidemic situation we currently find ourselves. The challenges in both scope of testing and pace of testing make case counts a poor metric of underlying disease activity. Any model involves trade-offs between simplicity and realism, and in this work we have not attempted to model physical distancing. Our understanding of the natural history of Covid-19 infection continues to evolve, and the precise role of presymptomatic and subclinical transmission is uncertain. Physical distancing becomes a more important control measure in the face of incomplete case ascertainment owing to asymptomatic or mildly symptomatic cases. Lastly, the model does not include seasonality; it is possible that transmission will attenuate in the summer, resulting in a decline in cases that would be expected to resurge with the return of colder weather. Therefore, the accuracy and the validity of the estimation would be better if the models fit the first-hand data on the population mobility and the data on the natural history, the epidemiological characteristics, and the transmission mechanism of the virus.

# VIII Conclusion

We proposed a mathematical model for Covid-19 transmission taking into account some parameters and developed a route of transmission for the disease. We estimated the theoretical formula of the basic reproduction number  $\mathcal{R}_0$  through our model. We also conducted an optimal control theory for our model by which the control measurement can be taken with the help of proper data. A case study of transmission of Covid-19 in Bangladesh was also done by two different methods including our proposed model. We calculated the basic reproduction number  $\mathcal{R}_0$  for Bangladesh by Exponential growth rate model & our proposed model.

# Acknowledgement

The University Grants Commission Bangladesh for providing the financial support to work with this article.

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