Transmission Dynamics of COVID-19 in Nepal

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Abstract

In this paper, we use the basic SEIR model to study the transmission dynamics of COVID-19 in Nepal. Fitting the model with the data of Nepal, we estimated 26% of Seroprevalence from 13 March, 2021 and the level of control strategies to reduce COVID-19 cases in Nepal. The value of β is approximated 0.0536 for the study period. Reduction of β by 25%, 50%, 75%, reduces reported cumulative cases by the end of 13th March, 2021 by 34.5%, 55.6% and 68.7% respectively.

Introduction

Corona virus disease first appeared in Wuhan, China in December 2019 (Lin, et al., 2020). In a short time, many people become infected and many lost their lives. A few months later the virus began to appear in many other countries, eventually spreading to all countries in the world. Nepal also becomes a victim of COVID-19 disease in 2020. The virus was first detected in Nepal on 23 January 2020 when a 31- years old student, who had returned to Kathmandu from Wuhan on 9 January (Shrestha, Shrestha, Khanal , & Kc , 2020). The first case of local transmission was confirmed on 4 April and the first death occurred on 14 May. COVID-19 disease began to grow rapidly as well as the number of deaths. Consequently, to prevent the transmission of COVID-19 disease, the government of Nepal introduced a nationwide lockdown from 24 March 2020. Lockdown did control the disease transmission from person to person to some extent but it had affected daily life and it was ended on 21 July 2020 (MoHP, 2020).

Researchers around the world have studied the spread of COVID-19 through compartmental mathematical modeling (Ndairou, Nieto, & Torres, 2020) (Kyrychko, Blyuss, & Brovchenko, 2020) (Kim, Kim, & Oh, 2020) (Singh, Bajpai, & Gupta, 2020). Y. Fang et al. investigated the outbreak of COVID-19 in China (Fang, Nie, &

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Penny, 2020). S. Tobias et al. studied the impracticality of COVID-19 herd immunity strategies in the UK (Brett & Rohani, 2020). Pandey, Subedi, Khanal, Koirala (Pandey, Subedee, Khanal, & Koirala, 2020), Bhuju, Phaijo, and Gurung (Bhuju, Phaijoo, Gurung, 2020) had studied transmission dynamics of COVID-19 in Nepal using the *SIR* model. Adhikari et.al studied the transmission dynamics of COVID-19 and found some insightful results during the control phase of the first wave (22 March, 2020) to 21 July, 2020) (Adhikari, Gautam, Pokharel, Uprety, & . Vaidya, 2021)

In this paper, a *SEIR* model with vital dynamics is taken to study the transmission dynamics of COVID-19 in Nepal. At first, we analyzed showing the positivity and boundness of solution. We derived the expression of disease-free and endemic equilibrium points, and basic reproduction number. Then, we estimate and approximate some model parameters taking the data from 17th September 2020 to 13th March 2021 which provided that official website of the government of Nepal (MoHP, 2020) and then, we predict the peak of the outbreak, eradicating time of outbreak without the implementation of any control measure.

Method

Data

This work is based on the data taken from 17th September 2020 to 13th March 2021 published by MOHP, Nepal (MoHp,2020).

Model Formulation

In the model, the total population N(t) is divided into *Susceptible*, *Exposed*, *Infectives*, and *Recovered* and denoted by S(t), E(t), I(t), R(t) respectively. The human requirement rate is Λ and natural death rate is μ . The population who become exposed leave the *Susceptible* compartment *S* and enter in to *Exposed* compartment *E* at the rate β called transmission rate. Likewise, when the exposed individuals become infectious, they move into *Infectious* compartment *I* from the *Exposed* compartment *E* at the rate η , called the rate of becoming infectious. Finally, when the infectious individuals become free from disease, they move into *Recovered* compartment *R* at the recovery rate γ . Those who die due to disease leave the compartment *I* at the rate δ , called mortality rate due to disease. The visualized schematic transfer diagram of the *SEIR* model is below, where the arrow shows the direction of movement between the compartments:

Figure 1: Compartmental diagram of SEIR epidemic model



The system of ordinary differential equations:

$$\frac{dS}{dt} = \Lambda - \beta S \frac{I}{N} - \mu S,$$

$$\frac{dE}{dt} = \beta S \frac{I}{N} - (\eta + \mu) E,$$
(1)
$$\frac{dI}{dt} = \eta E - (g + \mu + \delta) I,$$

$$\frac{dR}{dt} = \gamma I - \mu R.$$

Model Analysis

Positivity and boundedness of solution

For the problems associated with population dynamics, all the state variables involved in the model must be positive and bounded at any time.

Theorem 3.1: For all non-negative initial value, the solutions of model (1) exist, remain non-negative and bounded.

Proof:

In order to show positivity of state variables, it is straightforward by the fundamental theorem of differential equation for t > 0, we have from the first equation of (1)

$$\frac{dS}{dt} = \Lambda - \beta S \frac{I}{N} - \mu S$$

or,

$$\frac{dS}{dt} \ge -\left(\beta \frac{I}{N} + \mu\right)S$$
or,

$$\frac{dS}{S} \ge -\left(\beta \frac{I}{N} + \mu\right)dt$$
On integration,

$$\left[\log S\right]_{0}^{t} \ge \int_{0}^{t} \left(\beta \frac{I}{N} + \mu\right)dt$$
or,

$$\log S(t) - \log S(0) \ge -\int_{0}^{t} \left(\beta \frac{I}{N} + \mu\right)dt$$

or,
$$\log \frac{\mathbf{S}(t)}{S(0)} \ge -\int_0^t \left(\beta \frac{\mathbf{I}}{N} + \mu\right) dt$$

That is, $S \ge S(0) \exp\left(-\int_{0}^{t} \left(\beta \frac{I}{N} + \mu\right) dt\right)$. Taking limit $t \to \infty$. Then, we get S > 0 for all k > 0. Also, we have from second equation of equation (i)

$$\frac{dE}{dt} = \beta S \frac{I}{N} - (\eta + \mu) E$$
$$\frac{dE}{dt} \ge -(\eta + \mu) E$$

or,

or,

or,

$$\frac{dE}{dt} \ge -(\eta + \mu) dt$$

On integration with limit t = 0 to t,

$$\begin{bmatrix} \log E(t) \end{bmatrix}_{0}^{t} \ge -\int_{0}^{t} (\eta + \mu) dt \\\\ \log \frac{E(t)}{E(0)} \ge -\int_{0}^{t} (\eta + \mu) dt \\\\ \frac{E(t)}{E(0)} \ge \exp\left(-\int_{0}^{t} (\eta + \mu) d\right) t$$

Taking limit $t \to \infty$, $E(t) \ge 0$ for all $t \in [0, \infty)$. Similarly, $I(t) \ge 0$ and $R(t) \ge 0$.

For boundedness: From the model (1), we have,

$$\frac{dN}{dt} = \Lambda - \mu \left(S + E + I + R\right) - \delta I$$
$$\frac{dN}{dt} = \Lambda - \mu \left(S + E + I + R\right)$$
$$\frac{dN}{dt} \le \Lambda - \mu N$$
$$\frac{dN}{dt} + \mu N \le \Lambda$$

The solution is,

$$\left[N e^{\mu t}\right]_0^t \le -\int_0^t \Lambda e^{\mu t} dt$$

or,,
$$N(t) e^{\mu t} - N(0) \leq \frac{\Lambda}{\mu} e^{\mu t} - \frac{\Lambda}{\mu}$$

or,,
$$N(t) e^{\mu t} \leq N(0) + \frac{\Lambda}{\mu} e^{\mu t} - \frac{\Lambda}{\mu}$$

or,
$$N(t) \le N(0) e^{-\mu t} + \frac{\Lambda}{\mu} (1 - \mu e^{-\mu t})$$

 $t \to \infty, \ N(t) \le \frac{\Lambda}{\mu}$

As

This implies that N(t) is bounded, and hence S(t), E(t), I(t), and R(t) are bounded.

Equilibria analysis

Here, we discuss about the disease-free equilibrium point (DFE) and endemic equilibrium point (EE). DFE is the point at which there is no disease in the system and all the state variables remains constant whereas EE is the point at which disease persist in the population with constant state variables. For both type of equilibrium point the common condition is

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

or,

For simplicity, we define $\lambda = I/N$. Solving the equilibria equations, we get the equilibrium point (*S*^{*}, *E*^{*}, *I*^{*}, *R*^{*}) as follows.

$$S^{*} = \frac{\Lambda}{\beta\lambda + \mu'}$$

$$E^{*} = \frac{\beta\lambda\Lambda}{(\eta + \mu)(\beta\lambda + \mu)'}$$

$$I^{*} = \frac{\beta\eta\lambda\Lambda}{(\gamma + \delta + \mu)(\eta + \mu)(\beta\lambda + \mu)'}$$

$$R^{*} = \frac{\beta\eta\lambda\Lambda}{\mu(\gamma + \delta + \mu)(\eta + \mu)(\beta\lambda + \mu)}$$
(2)

Since, $\lambda - \frac{I}{N} = \lambda - \frac{I}{S + E + I + R} = 0$, then by some manipulation on equation, we have $(\beta\gamma\eta + \beta\gamma\mu + \beta\delta\mu + \beta\eta\mu + \beta\eta\mu + \beta\mu^2)\lambda^2 + (\gamma\eta\mu + \delta\eta\mu + \gamma\mu^2 + \delta\mu^2 + \eta\mu^2 - \beta\eta\mu)\lambda = 0$. Then roots of equation are, $\lambda = 0$ and, $-\frac{\mu(-\beta\eta + (\gamma + \delta + \mu)(\eta + \mu))}{\beta(\gamma(\eta + \mu) + \mu(\delta + \eta + \mu))}$.

Substituting $\lambda = 0$ in equation (2), we get $S^* = \frac{\Lambda}{\mu}$, we get $E^* = 0$, $I^* = 0$, and $R^* = 0$. Therefore, $\lambda = 0$ gives disease free equilibrium point (DFE).

Thus,
$$DFE = (S^*, E^*, I^*, R^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right)$$
.

Basic reproduction number (R_0)

For a simple model, the basic reproduction number (R_0) can formulate directly or by using specific methods. Here, we calculate R_0 by using Next-Generation matrix method (Diekmann, Heesterbeak and Roberts, 2010).

$$R_0 = \frac{\eta\beta}{(\eta+\mu)(\mu+\delta+\gamma)}.$$

Endemic equilibrium point

Disease persists in the population at the endemic equilibrium point but the rate of change of each compartment is zero. Therefore, $I \neq 0$ and $E \neq 0$. To get endemic equilibrium point (*EE*) substituting

$$\lambda = \frac{\mu(-\beta\eta + (\gamma + \delta + \mu)(\eta + \mu))}{\beta(\gamma(\eta + \mu) + \mu(\delta + \eta + \mu))}$$

in equation (2), we get endemic equilibrium point $EE = (S^*, E^*, I^*, R^*) = e_2(\text{say})$ as follows:

$$S^* = \frac{R_0 \Lambda \{\gamma(\eta + \mu) + \mu(\delta + \eta + \mu)\}}{R_0 \mu \{\gamma(\eta + \mu) + \mu(\delta + \eta + \mu)\} + \mu \beta \eta(R_0 - 1)}$$
$$E^* = \frac{\Lambda (\delta + \eta + \mu) (R_0 - 1)}{\eta(\beta - \delta)},$$
$$I^* = \frac{\Lambda(R_0 - 1)}{\beta - \delta},$$

$$R^* = \frac{\Lambda \gamma (R_0 - 1)}{\mu (\beta - \delta)}$$

Here, for $R_0 > 1$, it can be seen that $\beta - \delta > 0$.

For instance, we have $R_0 > 1$

or,
$$\frac{\eta\beta}{(\eta+\mu)(\mu+\delta+\gamma)} > 1$$

or,

$$\beta > \frac{(\eta + \mu)(\mu + \delta + \gamma)}{\eta}$$

 $\beta = \delta = \frac{(\eta + \mu)(\mu + \delta + \gamma)}{\delta} = \delta$

Now,

or,
$$\beta - \delta = \frac{\delta(\eta + \mu) + \mu(\delta + \eta + \mu)}{\eta} > 0$$

or, $\beta - \delta > 0$

$$\therefore \qquad \beta > \delta$$

Thus, for $R_0 > 1$, $S^* > 0$, $E^* > 0$, $I^* > 0$ and $R^* > 0$. Hence, endemic equilibrium point $(S^*, E^*, I^*, R^*) = e_2$ exist.

Local stability analysis

The Jacobean matrix for this system at DFE is given by:

$$J = \begin{pmatrix} -\mu & 0 & -\beta & 0 \\ 0 & -(\eta + \mu) & \beta & 0 \\ 0 & \eta & -(\gamma + \delta + \mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{pmatrix}$$

Let λ be an Eigen values of Jacobian J, then the characteristic equation in λ ,

$$-(\mu + \lambda)[-(\eta + \mu + \lambda)(\gamma + \delta + \mu + \lambda)(\mu + \delta) + \beta\eta(\mu + \lambda)] = 0$$

Then the values of λ , λ_1 , λ_2 , λ_3 and λ_4 . Then clearly

 $\lambda_1 = -\mu$, $\lambda_2 = -\mu$ and other two Eigen values are given by,

$$(\eta + \mu + \lambda)(\gamma + \delta + \mu + \lambda) - \beta \eta = 0$$

or,
$$\lambda_2 + \lambda(\eta + \gamma + \delta + 2\mu) + \frac{\eta\beta}{R_0}(1-R_0) = 0$$

where, R_0 is basic reproduction number. After solving for λ gives

$$\lambda_{3} = \frac{1}{2} \left[-L - \sqrt{L^{2} - 4 \frac{\eta \beta}{R_{0}} (1 - R_{0})} \right], \text{ and}$$
$$\lambda_{4} = \frac{1}{2} \left[-L + \sqrt{L^{2} - 4 \frac{\eta \beta}{R_{0}} (1 - R_{0})} \right]$$

Where, $L = (\eta + \gamma + \delta + 2\mu)$. Clearly, all the Eigen values are negative except λ_4 and clearly $\lambda_4 < 0$ if $R_0 < 1$. So, the disease-free equilibrium point is stable if $R_0 < 1$ otherwise it is unstable.

Data Fitting and Model Validation

Almost all control strategies were lifted from September 17, 2020. We take this as initial time t = 0 for our dynamical system model. We fit the model to the cumulative data given by the solution,

$$L(t) = \int_0^t p \eta E dt$$

by using the principle of least square method that minimizes the following sum of the squared residuals:

$$J(\phi) = \sum_{k=1}^{n} \left[(L(t_k) - \overline{L}(t_k))^2 \right] \qquad \dots (3)$$

where $L(t_k)$, and (t_k) , are the model predicted cumulative cases and those given in the available data and φ is the set of parameter to be estimated and *n* is the number of available data. In our study, all computations were carried out in MATLAB 2020a (The Math Works, Inc.) using its various routines, including "ode45" (ODE solver) and "fmincon" (minimizer).



Figure 2: Model fitting with cumulative number of cases of COVID-19 in Nepal

The model shows the quite good agreement with the data. The estimated state variables and parameters are listed in the table 1.

Results and Discussion

Figure 3: Model prediction: (a) Recovered and (b) Seroprevalence.



Here, we estimated the recovered number of cases and Seroprevalence of COVID-19 in Nepal. We find the total recovered cases (reported and non-reported) at the March 13, 2021 are 7040000. We also estimate the Seroprevalence at the 17th September, 15th October, 2020 and 13th March 2021 which are 3.7%, 12.39% and 26% respectively.

The parameters and their value are given in the table:

Parameter	Description	Value	References
β	Transmission Rate	0.0536 per day	Estimated
h	Rate of becoming infectious	0.192 per day	Estimated
g	Rate of Recovery	0.0588 per day	[Adhikari.et.al,2021]
р	Recorded portion	0.0473	Approximated
δ	Rate of Mortality due to disease	0.004 per day	Calculated
State variable	Description	Value	References
<i>S</i> (0)	Susceptible	25,000,000	Assumed
E(0)	Exposed	100,000	Estimated
I(0)	Infectious	1500,000	Estimated
R(0)	Recovered	1,000,000	Estimated

Table 1: Parameters and their values

Non-pharmaceutical control of COVID-19

The parameter β , the transmission rate is characterized that how fast disease transfers from infectious individuals to susceptible individuals. It can be calculated by multiplying the transmission risk by the average number of contacts per day. Therefore, the value of β can vary by reducing the contact rate by applying various types of control strategies such as quarantine, face mask, social distance, lockdown, isolation, etc. The value of β is approximated 0.0536 for the study period. Reduction of β by 25%, 50%, 75%, reduces reported cumulative cases by the end of 13th March, 2021 by 34.5%, 55.6% and 68.7% respectively.



Figure 4: Effect of reduction of transmission rate on the size of the epidemic

Vaccination and herd immunity

Herd immunity occurs when a large portion of a community (the herd) becomes immune to a disease, making the spread of disease from person to person unlikely.

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As a result, the whole community becomes protected not just those who are immune. Often, a percentage of the population must be capable of getting a disease in order to spread. This is called a threshold proportion. If the proportion of the immune population is greater than this threshold proportion, the spread of the disease will decline. This is known as the herd immunity threshold. The herd immunity level is achieved either through vaccination or immunity developed through the previous infection. It helps to decide what percentages of the population that must be vaccinated in order to stop the spreading of disease. Pathogens with higher R_0 require a higher herd immunity level. The threshold herd immunity level can be calculated as (Fine, 1993):

Proportion of vaccinated population = p

Remaining susceptible population S(t) = (1 - p)N(t)

$$R_s = R_0 \frac{S(t)}{N(t)}$$
$$= R_0 \frac{(1-p) N(t)}{N(t)}$$
$$R_s = R_0 (1-p)$$

For the control of diseases, we must have $R_s < 1$, i.e. $p > \frac{1}{R_0}$.

We found the basic reproduction number of COVID-19 as 1.589, so required herd immunity level in order to terminate the COVID-19 epidemic is $1 - \frac{1}{1.589}$ or 37%. The relationship between basic reproduction number and level of herd immunity is elaborated in figure 5.





Currently, the COVID-19 pandemic is almost everywhere in the world. Nepal is facing it's effect of second wave. The estimated number of cases in this second wave will depends on the Seroprevalence data of Nepal. In this paper we study the estimated Seroprevalence and effect of various level of control strategies on the size of epidemic of COVID-19 in Nepal.

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