

# Model of Transmission Dynamics of COVID-19 including Natural Birth Rate and considering the Difference in Death Rates of Uninfected and Infected Population

R. Lalawmpuii\* and Lalpawimawha<sup>1</sup>

\*Govt. Zirtiri Residential Science College, Aizawl, India.

<sup>1</sup>Pachhunga University College, Aizawl, India.

\*rlalawmpuii@gzrsc.edu.in, <sup>2</sup>raltelalpawimaha08@gmail.com

**Abstract:** We have presented a mathematical model using Ordinary Differential Equations to investigate the transmission of COVID-19. We included the parameter for the natural birth rate of the susceptibles, considering the fact that births take place each day. Taking into account that the infected population would have a higher death rate than the uninfected population, we used different parameters for the death rate of the uninfected and the infected population. We estimated an  $R_0$  of approximately 2.9, meaning that on an average each patient has been spreading the infection to 2.9 other people. The values of the parameters of the model have been estimated based on available data and numerical illustration has been carried out to describe the transmission process.

**Keywords:** Covid-19, SIR model, Ordinary differential equations.

## 1. Introduction

Mathematical modelling has played a major role in understanding the dynamics of infectious diseases and their control. Modelling can be useful for studying epidemiological patterns, evaluating the effectiveness of interventions and forecasting epidemiological patterns [1, 2, 3]. Mathematical models have been used to understand the dynamics of HIV infection [4, 5] and the impact of awareness programs on the spread of HIV/AIDS [6, 7]. Bauchet *al.* [8] gives an overview of all SARS models during the period of the epidemic up to 2 years thereafter. Various routes of transmission of Ebola has also been studied using Mathematical models [20, 21, 22]. Mathematical models have also been used to study the two novel coronaviruses (CoVs), namely, the severe acute respiratory syndrome coronavirus (SARS-CoV) in 2002 that spread to 37 countries and the Middle East respiratory syndrome coronavirus (MERS-Cov) in 2012 that spread to 27 countries [23, 24, 25, 26, 27, 28, 29, 30]. SARS-CoV caused more than 8000 infections and 800 deaths [31] and MERS-CoV infected 2494 individuals and caused 858 deaths [32]. The prevailing COVID-19, by 12 May 2020 has spread to 212 countries and Territories around the world. It has infected 4,088,848 individuals and has caused 283,153 deaths [33].

The evolution and spread of COVID-19 have resulted in an international effort coordinated by the World Health Organization (WHO). On 31<sup>st</sup> December 2019, the WHO China Country Office was informed of cases of pneumonia unknown etiology detected in Wuhan City, Hubei Province of China. On 11<sup>th</sup> and 12<sup>th</sup> January 2020, WHO received further detailed information from the National

Health Commission China that the outbreak is associated with exposures in the seafood market in Wuhan City. The outbreak of the novel coronavirus, 2019-nCoV has been declared a public health emergency of international concern by the WHO on 30<sup>th</sup> January 2020 [35]. On 11<sup>th</sup> February 2020, WHO announced the name COVID-19 for the new coronavirus disease. COVID-19 is a newly discovered infectious disease with a high potential for transmission to close contacts. As of 13<sup>th</sup> April 2020, a total of 747,546 confirmed cases were documented by WHO via case reporting forms received from 113 countries, territories and areas across five different WHO regions and three international conveyances [36]. Control of the disease is mainly on prompt identification of cases and isolation of probable cases and their contacts. The rapid growth in the number of COVID-19 cases set up a strong alarm to the government and people. Public health authorities, physicians and scientists all over the world run awareness campaign through social media to educate the public on COVID-19 prevention. The nationwide lockdown was declared in many countries.

Mathematical models have been used to study the dynamics of COVID-19 [37, 38, 39, 40, 41, 42, 43, 44, 45]. Many authors have studied the early dynamics of the disease in Wuhan [46, 47, 48, 49, 50]. SIR model has been used by many authors to understand the dynamics of the disease. The natural birth rate of the human population has been ignored in most cases [48, 49, 50]. In our model, we have included the natural birth rate of the human population so that the actual decrease in the human population due to the disease may be projected. In most of the models, the death rate of all the populations is represented by one parameter. Here, considering the fact that the death rate of the infected population would be more than the death rate of the uninfected population, we have used separate parameters for the death rate of the uninfected population and the infected population.

## 2. The Mathematical Model

### 2.1. Formulation of the SIR Model:

We propose a SIR model to study the dynamics of COVID-19. In this model, we include the natural inflow of human population considering the fact that babies are born each day. This may give the image of the susceptible population, its decrease due to the disease and as and how the original population may be regained. We divide the human population into three classes, namely, the susceptible population which is denoted by  $S$ , the infected population which is denoted by  $I$  and the recovered population which is denoted by  $R$ . The total human population is denoted by  $N$  and  $N = S + I + R$ .

$$\frac{dS}{dt} = \pi N - \beta \frac{I}{N} S - \mu S \quad (1a)$$

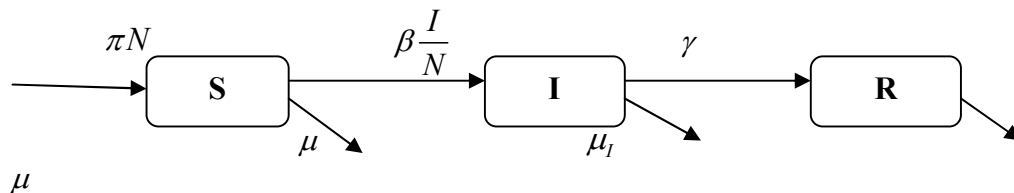
$$\frac{dI}{dt} = \beta \frac{I}{N} S - \gamma I - \mu_1 I \quad (1b)$$

$$\frac{dR}{dt} = \gamma I - \mu R \quad (1c)$$

We assume that except for the infected and recovered individuals, all other individuals are susceptible to the disease. We consider the inflow of the human population at the rate  $\pi$ . We suppose that the disease is transmitted at the rate  $\beta$  and the susceptible population dies at a constant natural death rate  $\mu$ . In most of the SIR models on COVID-

19, the death rate of all the classes of the population is assumed to be the same. In our model, we assume different death rates for the infected population and uninfected population, considering the fact that the death rate of the infected population is higher compared to the death rate of the uninfected population. Thus, we assume that the infected population dies at a rate  $\mu_I$ . Since the recovered individual is free of the disease, we assume that they too die at the natural death rate  $\mu$ . Thus, we assume  $\mu_I > \mu$ . Also, the infected individuals recover at the rate  $\gamma$  and move to the recovered class.

A schematic diagram of the model is provided in Figure 1.



**Figure 1.** A Schematic Representation of the SIR model including Demography. S, I and R represent the Total number of Susceptible, Infected and Recovered individuals in a Population.  $\beta$  is the Rate of Transmission of the disease and  $\gamma$  is the Recovery Rate.  $\mu$  is the Death Rate of the Susceptible and Recovered Population and  $\mu_I$  is the Death Rate of the Infected Population.

**2.2 Determination of the Steady States.**

Consider

$$\pi N - \beta \frac{I}{N} S - \mu S = 0 \tag{2a}$$

$$\beta \frac{I}{N} S - \gamma I - \mu_I I = 0 \tag{2b}$$

$$\gamma I - \mu R = 0 \tag{2c}$$

**Theorem 2.2.1.** The disease-free steady state is  $E(S, 0, 0)$ , where  $S = \frac{\pi N}{\mu}$ .

**Proof.** At the disease-free state,  $I = 0$  and  $R = 0$ . With these in equation (2a), we have

$$S = \frac{\pi N}{\mu}.$$

**Theorem 2.2.2.** The endemic steady state is  $E^*(S^*, I^*, R^*)$ , where  $S^* = \frac{\gamma + \mu_I}{\beta} N$ ,

$$I^* = \frac{\mu N}{\beta} \left[ \frac{\pi \beta}{\mu(\gamma - \mu_I)} - 1 \right] \text{ and } R^* = \frac{\mu N}{\beta} \left[ \frac{\beta - \gamma}{\mu} + \frac{\mu - \mu_I}{\mu} - \frac{\pi \beta}{\mu(\gamma - \mu_I)} \right].$$

**Proof.** From equation (2b), we have  $S^* = \frac{\gamma + \mu_I}{\beta} N$

From equation (2a), we have  $I^* = \frac{\mu N}{\beta} \left[ \frac{\pi\beta}{\mu(\gamma - \mu_I)} - 1 \right]$

Also, since  $N = S^* + I^* + R^*$ , we get

$$R^* = \frac{\mu N}{\beta} \left[ \frac{\beta - \gamma}{\mu} + \frac{\mu - \mu_I}{\mu} - \frac{\pi\beta}{\mu(\gamma - \mu_I)} \right].$$

### 2.3. The Basic Reproduction Number

The basic reproduction number,  $R_0$ , is an important quantity in disease modelling and is generally defined as the average number of secondary infections arising from primary infection in an entirely susceptible population.

For  $I^* > 0$ , that is, for the disease to prevail, we need  $\frac{\pi\beta}{\mu(\gamma - \mu_I)} > 1$ . We take

this ratio as the basic reproduction ratio. Therefore, we define  $R_0 = \frac{\pi\beta}{\mu(\gamma - \mu_I)}$ .

### 2.4. Stability Analysis

**Theorem 2.4.1.** The disease-free state  $E(S, 0, 0)$  is locally asymptotically stable when  $R_0 < 1$ .

**Proof.** The Jacobian matrix of the system at the disease-free state  $E\left(\frac{\pi N}{\mu}, 0, 0\right)$  is

$$J(E) = \begin{bmatrix} \frac{-\beta}{N} & \frac{-\pi\beta}{\mu} & 0 \\ 0 & \frac{\pi\beta}{\mu} - (\gamma + \mu_I) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

$E(S, 0, 0)$  is locally asymptotically stable if  $\frac{\pi\beta}{\mu(\gamma + \mu_I)} < 1$  that is, if  $R_0 < 1$ .

**Theorem 2.4.2.** The endemic steady state  $E^*(S^*, I^*, R^*)$  is locally asymptotically stable when  $R_0 > 1$ .

**Proof.** The Jacobian matrix of the system at the endemic steady state  $E^*(S^*, I^*, R^*)$  is

$$J(E^*) = \begin{bmatrix} -\beta \frac{I^*}{N} - \mu & -\beta \frac{S^*}{N} & 0 \\ \beta \frac{I^*}{N} & \beta \frac{S^*}{N} - (\gamma + \mu_1) & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

The characteristic equation is

$$f(\lambda) = \begin{vmatrix} a_1 - \lambda & a_2 & 0 \\ a_3 & a_4 - \lambda & 0 \\ 0 & a_5 & a_6 - \lambda \end{vmatrix} = 0$$

where  $a_1 = -\left(\beta \frac{I^*}{N} + \mu\right)$ ,  $a_2 = -\beta \frac{S^*}{N}$ ,  $a_3 = \beta \frac{I^*}{N}$ ,  $a_4 = \beta \frac{S^*}{N} - (\gamma + \mu_1)$ ,  $a_5 = \gamma$ ,

$$a_6 = -\mu$$

that is  $\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0$

where  $A_1 = -(a_1 + a_4 + a_6)$

$$A_2 = a_1a_4 + a_1a_6 + a_4a_6 - a_2a_3$$

$$A_3 = a_2a_4a_6 - a_1a_5a_6$$

By Routh-Hurwitz criteria,  $E^*(S^*, I^*, R^*)$  is locally asymptotically stable if  $A_1 > 0$ ,  $A_2 > 0$ ,  $A_3 > 0$  and  $A_1A_2 - A_3 > 0$ .

The above criterion are satisfied under the condition that  $\frac{\pi\beta}{\mu(\gamma + \mu_1)} - 1 > 0$  which gives

$$R_0 > 1.$$

## 2.5. Positivity and Boundedness of solutions.

The SIR model given by the system (1) describes a human population, therefore, it is necessary to prove that the populations (susceptible, infected and recovered) are positive for all time,  $t > 0$ . That is, all solutions of the system (1) with non-negative initial data will remain positive for any  $t > 0$ .

**Theorem 2.5.1.** Let the initial data be  $S(0) = S_0 > 0$ ,  $I(0) = I_0 > 0$  and  $R(0) = 0$ .

Then the components of the solution  $S(t)$ ,  $I(t)$  and  $R(t)$  of system (1) are positive for all time  $t > 0$ .

**Proof.** Let the initial conditions be  $S(0) = S_0 > 0$ ,  $I(0) = I_0 > 0$  and  $R(0) = 0$ .

Suppose for  $S(t) = 0$  for some time  $t > t_0$  and  $I(t) \geq 0$ ,  $R(t) \geq 0$ .

From system (1),

$$\frac{dS}{dt} = \pi N - \beta \frac{I}{N} S - \mu S = 0 \text{ when } S(t) = 0.$$

which shows that the component of the solution  $S(t)$  will be non-negative for all  $t > 0$ . Now, to show that the component  $I(t)$  of the solution will be non-negative for all  $t > 0$ , we assume  $I(t) = 0$  for some time  $t > t_0$ ,  $S(t) \geq 0$ ,  $R(t) \geq 0$  and show that  $\frac{dI}{dt} \geq 0$ .

Considering the system (1)

$$\frac{dI}{dt} = \beta \frac{I}{N} S - \gamma I - \mu_1 I = 0 \text{ when } I(t) = 0$$

which shows that the component of the solution  $I(t)$  will be non-negative for  $t > 0$ .

Finally, to show that the component of the solution  $R(t)$  stays positive for all time, we assume  $R(t) = 0$  for some time  $t > t_0$ ,  $S(t) \geq 0$ ,  $I(t) \geq 0$  and show that  $\frac{dR}{dt} \geq 0$ .

From system (1),

$$\frac{dR}{dt} = \gamma I - \mu R \geq 0 \text{ when } I(t) \geq 0$$

since the constant recovery rate  $\gamma$  is positive, the solution  $R(t)$  will be non-negative for all time  $t > 0$ , which completes the proof.

The boundedness of the components of the solution  $S(t)$ ,  $I(t)$  and  $R(t)$  follows from the fact that  $N(t) = S(t) + I(t) + R(t)$  and that  $S(t)$ ,  $I(t)$  and  $R(t)$  are non-negative for all time  $t > 0$ . Therefore we have that each component of the solution is at most equal to  $N$ . That is  $S(t), I(t), R(t) \leq N(t)$  for all  $t \geq 0$ . Since each component of the solution is non-negative at the outbreak of the disease ( $t = 0$ ). This implies that each component of the solution  $S(t)$ ,  $I(t)$  and  $R(t)$  is bounded between zero and the total population size  $N$ .

### 3. Numerical Simulation

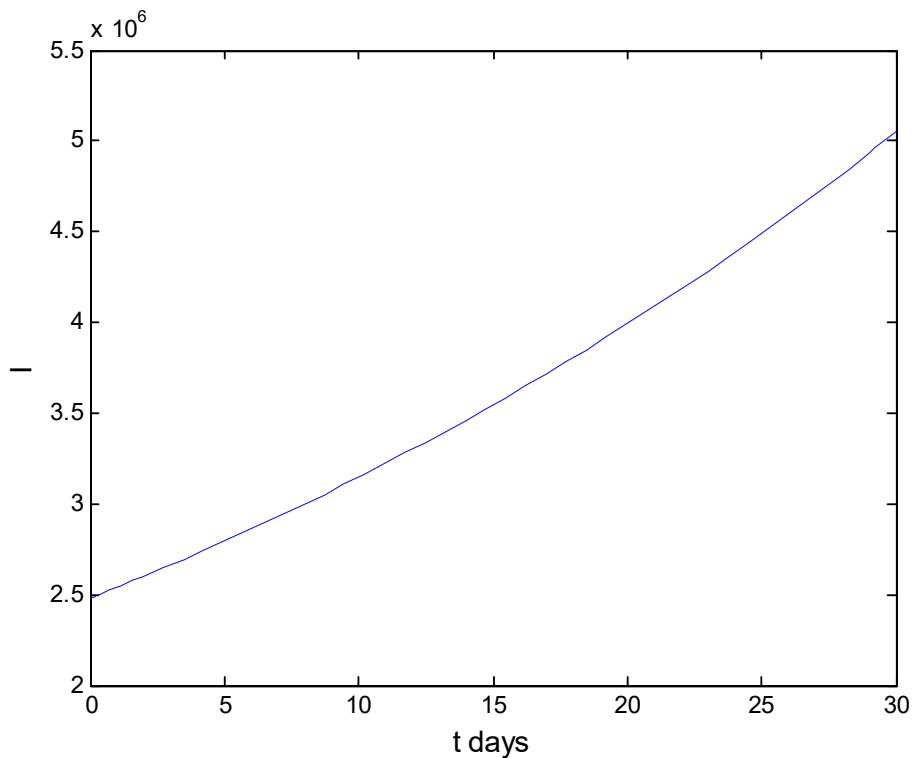
For numerical validation of our model, we take the total population of the world as 759.43 crores [51]. For the initial values of the model variables, we use the data of COVID-19 as on 20<sup>th</sup> April 2020, where the total number of infected,  $I_0 = 2,480,503$ , the number of recovered is  $R_0 = 646,328$  so that the susceptible population,  $S_0 = 7,591,819,000$  [52]. The values of the parameters of the model are given in Table 1.

**Table 1. Values of Parameters for COVID-19 Model**

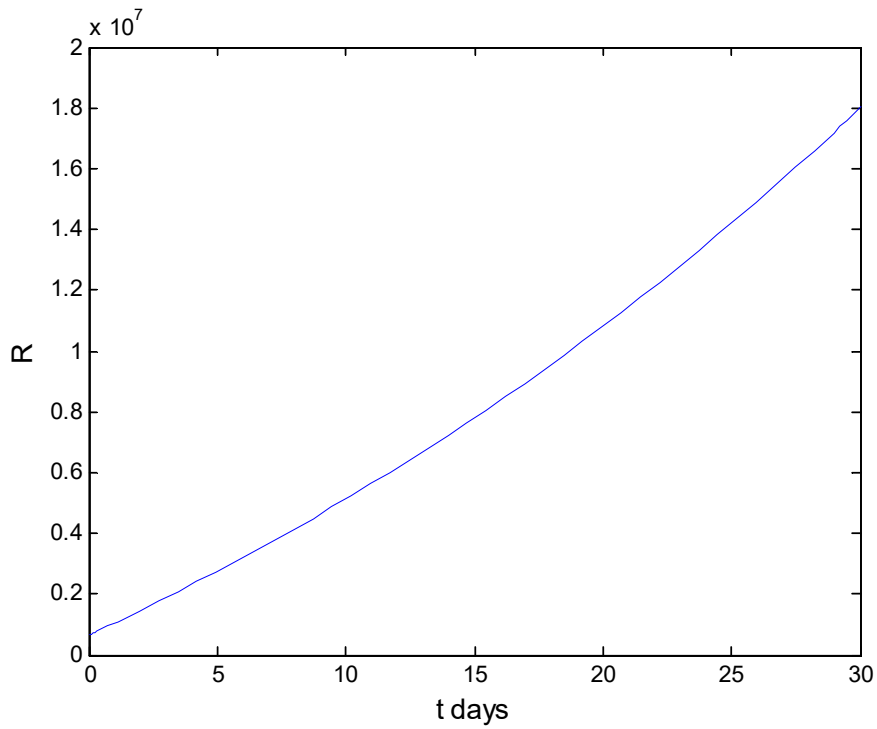
Parameter	Description	Value	Source
$\pi$	Natural birth rate	18.5 per 1000 in a year = 0.00005 $day^{-1}$	[53]
$\mu$	Natural death rate		[53]
$\beta$	Rate of transmission of the disease	7.7 per 1000 in a year = 0.00002 $day^{-1}$	fitted
$\mu_1$	Death rate due to the disease	0.187 $day^{-1}$	[53]

$\gamma$	Recovery rate	$0.067 \text{ day}^{-1}$	[49]
		$0.16 \text{ day}^{-1}$	

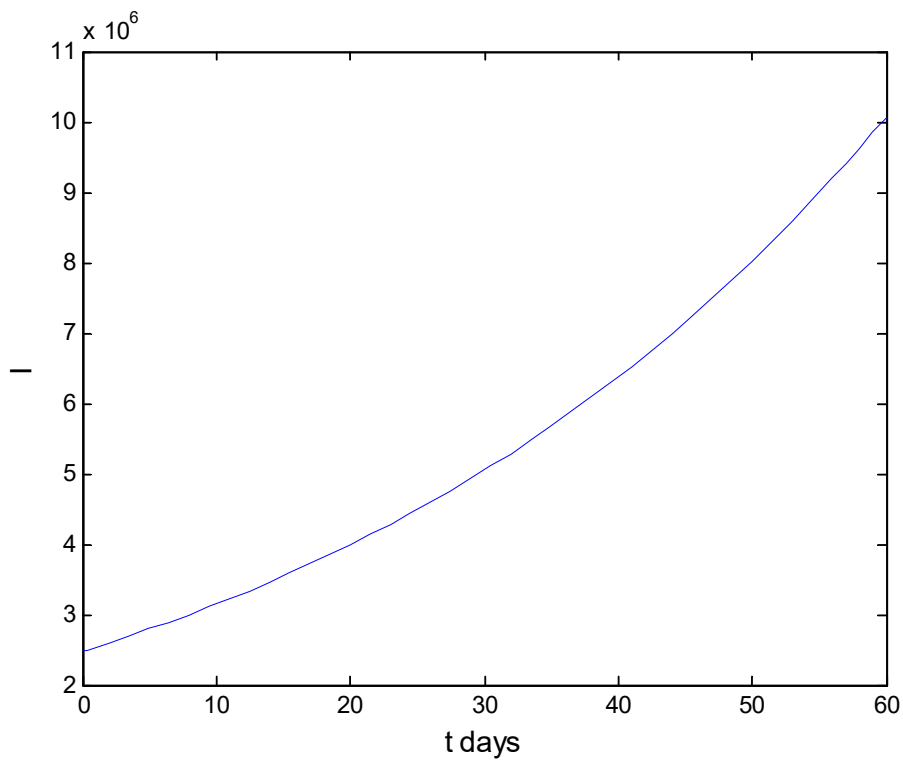
Using these parameter values, we estimated the basic reproduction number  $R_0$  to be 2.8681, which is approximately 2.9, which means that on an average each patient has been spreading the infection to 3 other people. Using the values of the parameters given in Table 1 and taking the data of 20<sup>th</sup> April 2020 as the initial value, we estimated the total number of infected for 30 days. It is shown in figure 2 that this estimation matches with the data of 20<sup>th</sup> may 2020 [52]. Also, we estimated the total number of recovered population for 30 days as is shown in figure 3. As per the Worldometer report of 20<sup>th</sup> May 2020, the total number of infected is 5,076,964 and the total number of recovered is 2,018,814. Figure 4 shows the estimated number of infected population 60 days after 20<sup>th</sup> April 2020.



**Figure 2.** The Dynamics of the Infected Population with  $I_0 = 2,480,503$ .



**Figure 3. The Dynamics of the Recovered Population with  $R_0 = 646,328$ .**



**Figure 4. The Dynamics of the Infected Population taking a duration of 60 days with  $I_0 = 2,480,503$ .**



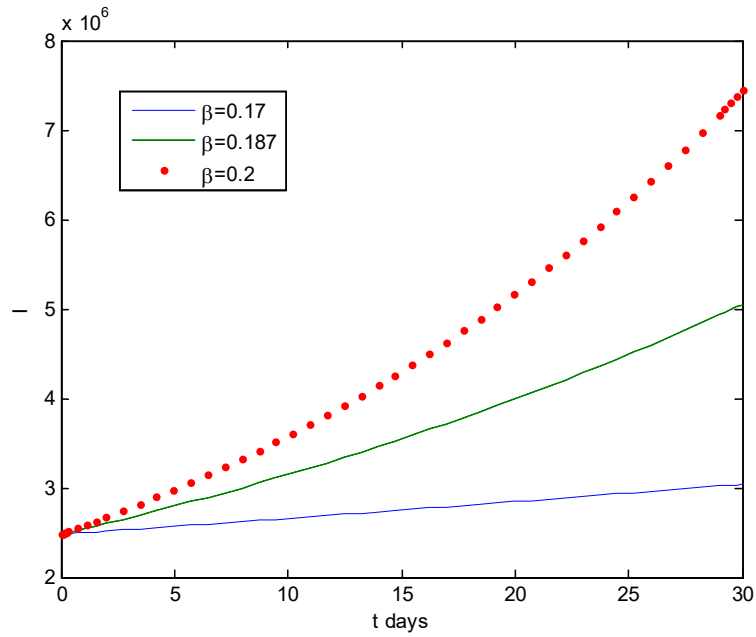


Figure 5. The behavior of the infected population with different values of the rate of transmission  $\beta$ .

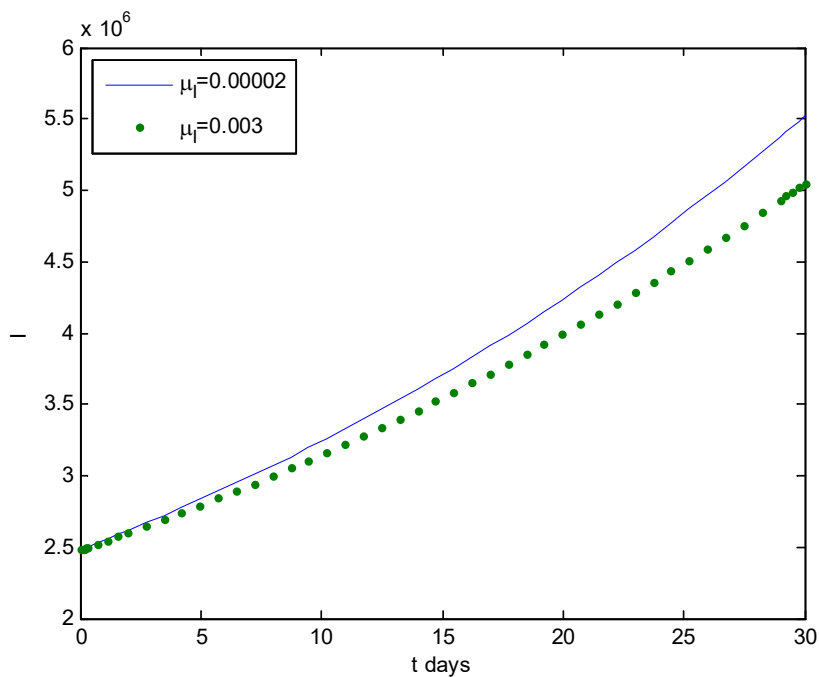
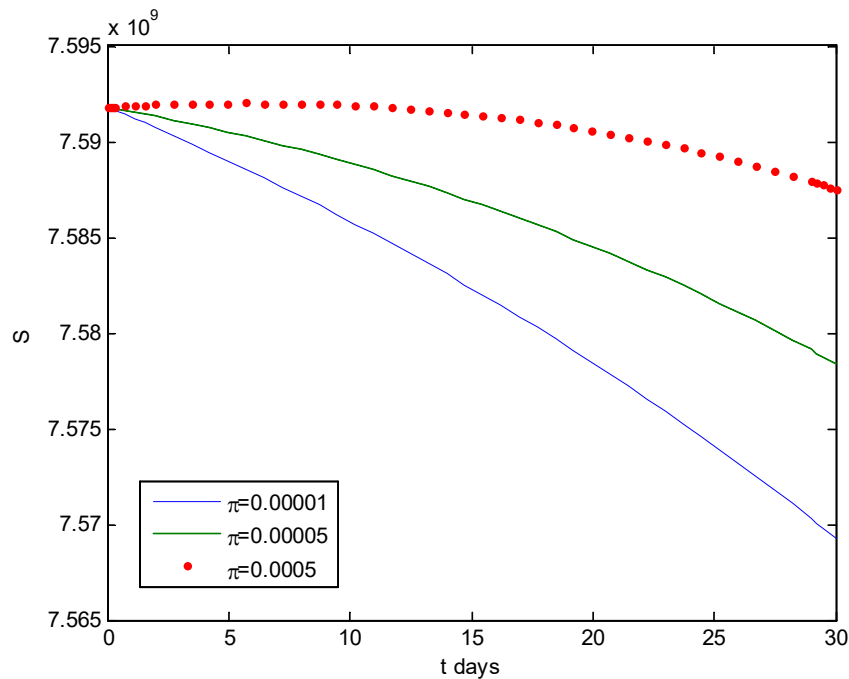


Figure 6. Comparison of the behavior of the infected population with different death rates. It is seen that when the death rate of the infected population is the same as the death rate of the uninfected population, the number of infected population increases.



**Figure 7. Dynamics of the Susceptible Population with different values of Normal Birth Rate. It is seen that if the normal birth rate increases, then the total population of susceptibles would increase.**

#### 4. Conclusion

We presented an SIR mathematical model describing the transmission dynamics of COVID-19. We included the parameter for the natural birth rate of the susceptible considering the fact that births take place each day. Taking into account that the infected population would have a higher death rate than the uninfected population, we used different parameters for the death rate of the uninfected and the infected population. Assuming that once a person recovered, he/she is free of the disease, we have taken the death rate of the recovered population to be the same as the uninfected population. We determined the two steady states, namely, the disease-free steady state and the endemic steady state. We defined the basic reproduction  $R_0$ . We found that the disease-free steady state is locally asymptotically stable when  $R_0 < 1$  and the endemic steady state is locally asymptotically stable when  $R_0 > 1$ . We have shown the positivity and boundedness of the solutions. Numerical simulation of the model is carried out using available resources for estimating the values of the parameters. We have taken the data of 20<sup>th</sup> April 2020 as the initial condition and studied the dynamics of the model. It is seen that the estimated number of infected population and the recovered population after a duration of 30 days is relatively same as the real data of 20<sup>th</sup> May 2020. It is shown that the infected population would decrease as the rate of transmission decreases. This shows that interventions such as social distancing and lock-downs would have great impact on reducing the spread of the disease. We have shown that if the death rate of the infected population is the same as the death rate of the uninfected population, then the number of infected would be more than the real data. Further, it is shown that if the natural birth rate may be increased, then the loss in the human population may be reduced.

## References

1. R.M. Anderson, R.M. May, "Infectious diseases of humans: Dynamics and control", *Oxford University Press*, Oxford (1991).
2. D. Bernoulli, "Essaid'une nouvelle analyse de la mortalité causée par la petite vérole et des avantages de l'inoculation pour la prévenir", *Mem. Math. Phys. Acad. R. Sci.*, (1760), pp. 1-45.
3. R.M. May, R.M. Anderson, "Endemic infections in growing populations", *Mathematical biosciences*, vol. 77, no.1-2, (1985), pp. 141-156.
4. O.M. Ogunlaran, S.C. Noutchie, "Mathematical model for an effective management of HIV infection", *Biomed Research International*(2016).<https://dx.doi.org/10.1155/2016/4217548>
5. D.J. Nokes, R.M. Anderson, "The use of mathematical models in the epidemiological study of infectious diseases and in the design of mass immunization programmes", *Epidemiology and infection*, vol. 101 no.1, (1988) pp. 1-20.
6. P. van den Driessche, "Reproduction numbers of infectious disease models", *Infectious disease modelling*, vol.2, (2017), pp. 288-303.
7. N.C. Grassly, C. Fraser, "Mathematical models of infectious disease transmission", *Nature reviews microbiology*, vol. 6, no. 6, (2008), pp. 477-487.
8. K. Dietz, "The estimation of the basic reproduction number for infectious diseases", *Statistical Methods in Medical Research*, vol. 2, (1993), pp. 23-41.[doi:10.1177/096228029300200103](https://doi.org/10.1177/096228029300200103).
9. G.U. Achi, J.U. Okafor, K. Chimereucheya, "The stability analysis of the mathematical model of tuberculosis transmission dynamics", *International Journal of Science and Research*, vol.2, no. 1, (2013), pp. 643-647.
10. H. Nishiura, D. Klinkenberg, M. Roberts, J. Heesterbeek, "Early epidemiological assessment of the virulence of emerging infectious diseases: A case study of an Influenza pandemic", *PLoS ONE*, vol. 4, no. 8, (2009), e6852.
11. H. Wu, H. Zhu, H. Miao, A. S. Perelson, "Parameter identifiability and estimation of HIV/AIDS dynamic models", *Bulletin of mathematical biology*, vol. 70, (2008), pp. 785-799.
12. A.M. Elaiw, "Global properties of a class of HIV models", *Nonlinear Analysis: Real World Applications*, vol. 11, (2010), pp. 2253-2263.
13. J. Hussain, R. Lalawmpuii, "Modelling the dynamics of CD+T cells with and without delay", *Sci Vis*, vol. 13 no. 4, (2013), pp. 191-199.
14. P. Rani, V.P. Saxena, D.S. Hooda, "Age-structured mathematical model for HIV/AIDS in a two-dimensional heterogeneous population", *Commun. Math. Biol. Neurosci.*, vol. 29, (2015).
15. J.T. Rowley and R.M. Anderson, "Modeling the impact and costeffectiveness of HIV prevention efforts", *AIDS*, vol. 8, no. 4, (1994), pp. 539-548.
16. R. Lalawmpuii, J. Hussain, "Modelling the impact of awareness programs on the spread of HIV/AIDS", *International Journal of Science and Research*, vol. 4 no. 3, (2015), pp. 2260-2266.
17. M.H. Gail, D. Preston and S. Piantadosi, "Disease prevention models of voluntary confidential screening for human immunodeficiency virus (HIV)", *Statistics in Medicine*, vol.8, no. 1,(1989), pp.59-81.
18. J.T. Rowley and R.M. Anderson, "Modeling the impact and costeffectiveness of HIV prevention efforts", *AIDS*, vol. 8, no. 4, (1994), pp. 539-548.

19. C.T. Bauch, J.O. Lloyd Smith, M.P. Coffee, A.P. Galvani, “Dynamically modelling SARS and other newly emerging respiratory illnesses: past, present and future”, *Epidemiology*, vol. 16, (2005), pp. 791-801.
20. A. Camacho, A.J. Kucharski, S. Funk, J. Berman, P. Piot, W.J. Edmunds, “Potential for large outbreaks of Ebola virus disease”, *Epidemics*, vol. 9, (2014), pp. 70-78.
21. O.S. Deepa, G.V.S. Teja, “Mathematical model for transmission of Ebola”, *Procedia Computer Science*, vol. 48, (2015), pp. 741-745.
22. S. Funk, I. Ciglenecki, A. Tiffany *et.al.*, “The impact of control strategies and behavioural changes on the elimination of Ebola from Lofa County, Liberia”, *Phil. Trans. R. Soc. B*, vol. 372, (2016), <http://dx.doi.org/10.1098/rstb.2016.0302>
23. M. Lipsitch, T. Cohen, B. Cooper *et.al.*, “Transmission Dynamics and control of Severe Acute Respiratory Syndrome”, *Science* vol. 300, (2003), pp. 1966-1970.
24. C.T. Bauch, J.O. Lloyd-Smith, M.P. Coffee, A.P. Galvani, “Dynamically modeling SARS and other newly emerging respiratory illnesses”, *Epidemiology* vol. 16, no. 6, (2005), pp. 791-801.
25. K.C. Ang, “A simple model for a SARS epidemic”, *Teaching Mathematics and its Applications* vol. 23, no. 4, (2004), pp. 181-188.
26. C. Castillo-Chavez, A. Yakubu, “Mathematical models of isolation and quarantine”, *Journal of American Medical Association*, vol. 290, no. 21, (2003), pp. 2876-2877.
27. X. Han, S.J. de Vlas, L. Fang, D. Fang, W. Cao, J.F. Habbema, “Mathematical modelling of SARS and other infectious diseases in China: a review”, *Tropical Medicine and International Health*, vol. 14, no. 1, (2009), pp. 92-100. doi:10.1111/j.1365-3156.2009.02244.x
28. Y. Zhou, Z. Ma, A discrete epidemic model for SARS transmission and control in China, *Mathematical and Computer Modelling* 40, 1491-1506 (2004) doi:10.1016/j.mcm.2005.01.007
29. J. Lee, G. Chowell, E. Jung, “A dynamic compartmental model for the Middle East respiratory syndrome outbreak in the Republic of Korea: A retrospective analysis on control interventions and superspreading events”, *Journal of Theoretical Biology* vol. 408, (2016), pp. 118-126. <http://dx.doi.org/10.1016/j.jtbi.2016.08.009>
30. Q. Lin, A. Chiu, S. Zhao, D. He, “Modeling the spread of Middle East respiratory syndrome coronavirus in Saudi Arabia”, *Statistical Methods in Medical Research*, vol. 27, no. 7, (2018), pp. 1968-1978. doi:10.1177/0962280217746442
31. WHO, SARS (Severe Acute Respiratory Syndrome) can be found at <https://www.who.int/ith/diseases/sars/en/>
32. WHO, Middle East respiratory syndrome coronavirus (MERS-CoV) can be found at <https://www.who.int/emergencies/mers-cov/en/>
33. WHO, Novel Coronavirus (2019-nCoV). Situation report-113, (2020), May 12. Available in <https://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports>.
34. WHO, Novel Coronavirus (2019-nCoV). Situation report-1, (2020), January 21.
35. WHO, Novel Coronavirus (2019-nCoV). Situation report-11, (2020), January 31.
36. WHO, Novel Coronavirus (2019-nCoV). Situation report-89, (2020), April 18.
37. A.J. Kucharski, T.W. Russell, C. Diamond, Y. Liu, J. Edmunds, S. Funk, R.M. Eggo, “Early dynamics of transmission and control of COVID-19: a mathematical modelling study”, *Lancet Infect Dis*, (2020), [https://doi.org/10.1016/S1473-3099\(20\)30144-4](https://doi.org/10.1016/S1473-3099(20)30144-4)
38. H. Najafimehr, K.M. Ali, S. Safari, M. Yousefifard, M. Hosseini, “Estimation of basic reproduction number for COVID-19 and the reason for its differences”, *International Journal of Clinical Practice*, (2020) <https://doi.org/10.1111/ijcp.13518>.

39. T. Chen, J. Rui, Q. Wang, Z. Zhao, J. Cui, L. Yin, "A mathematical model for simulating the phase-based transmissibility of a novel coronavirus", *Infectious Diseases of Poverty* vol. 9, no. 24, (2020) <https://doi.org/10.1186/s40249-020-00640-3>.
40. S. Zhang, M. Diao, W. Yu, L. Pei, Z. Lin, D. Chen, Estimation of the reproductive number of novel coronavirus (COVID-19) and the probable outbreak size on the Diamond Princess cruise ship: A data-driven analysis, *International Journal of Infectious Diseases* **93**, 201-204 (2020). <https://doi.org/10.1016/j.ijid.2020.02.033>
41. S.P. Adhikari, S. Meng, Y. Wu, Y. Mao, R. Ye, Q. Wang, C. Sun, S. Sylvia, S. Rozelle, H. Raat, H. Zhou, "Epidemiology, causes, clinical manifestation and diagnosis, prevention and control of coronavirus disease (COVID-19) during the early outbreak period: a scoping review", *Infectious Diseases of Poverty*, vol. 9, no. 29, (2020), <https://doi.org/10.1186/s40249-020-00646-x>
42. J. Arino, S. Portet, "A simple model for COVID-19", *Infectious disease modelling* vol. 5, (2020), pp. 309-315.
43. R. Verity, L.C. Lokell, I. Dorigatiet. al., "Estimates of severity of coronavirus disease 2019: a model based analysis", *Lancet infectious disease*, (2020), [https://doi.org/10.1016/S1473-3099\(20\)30243-7](https://doi.org/10.1016/S1473-3099(20)30243-7).
44. Y. Liu, A.A. Gayle, A. Wilder-Smith, J. Rocklov, "The reproductive number of COVID-19 is higher compared to SARS coronavirus", *Journal of travel medicine*, (2020), pp. 1-4. doi: 10.1093/jtm/taaa021.
45. C. Anastassopoulou, L. Russo, A. Tsakris, C. Siettos, "Data-based analysis, modelling and forecasting of the COVID-19 outbreak", *PloS ONE*, vol. 15, no. 3, (2020), pp. 1-21.
46. J.T. Wu, K. Leung, G.M. Leung, "Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan, China: a modelling study", *The Lancet* (2020) [https://doi.org/10.1016/S0140-6736\(20\)30260-9](https://doi.org/10.1016/S0140-6736(20)30260-9).
47. S. Zhao, Q. Lin, J. Ran, S.S. Musa, G. Yang, W. Wang, Y. Lou, D. Gso, L. Yang, D. He, M.H. Wang, "Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCov) in China, from 2019 to 2020: A data-driven analysis in the early phase of the outbreak", *International journal of infectious diseases* vol. 92, (2020), pp. 214-217.
48. Q. Li, X. Guan, P. Wu, X. Wang, B. Cowling, B. Yang, M. Leung, Z. Feng, "Early transmission dynamics in Wuhan, China of novel coronavirus-infected pneumonia", *The new England journal of medicine (NEJM)*, vol. 382, no. 13, (2020), pp. 1199-1207.
49. Q. Lin, S. Zhao, D. Gao, Y. Lou, S. Yang, S.S. Musa, M.H. Wang, Y. Cai, W. Wang A, L. Yang, D. He, "A conceptual model for the coronavirus disease 2019 (COVID-19) outbreak in Wuhan, China with individual reaction and governmental action", *International journal of infectious diseases*, vol. 93, (2020), pp. 211-216.
50. Yubei Huang, Lei Yang, H. Dai, F. Tian, K. Chen, "Epidemic situation and forecasting oif COVID-19 in and outside China", *Bulletin of WHO*, (2020), March 16.
51. Statista can be found at [www.statista.com](http://www.statista.com) .
52. Worldometer [www.worldometers.info/coronavirus](http://www.worldometers.info/coronavirus) .
53. Wikipedia can be found at <https://en.m.wikipedia.org/wiki> .