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[ijirset@gmail.com](mailto:ijirset@gmail.com)

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# A Mathematical Modelling of COVID-19, Epidemic Based on a Deterministic Model without Removal

Jitendra Kumar

Department of Mathematics, Govt. Degree College Bhojpur, Moradabad, U.P., India

**ABSTRACT:** The most abundant species in nature are viruses; they are parasites, since they cannot replicate themselves. Upon replication, some viruses cause serious infectious diseases in human and/or animals and are medically, socially, and economically important. One of these species is the corona virus. An outbreak of pneumonia caused by a novel corona virus (COVID-19) began (officially) on January 30, 2020, in Kerala (India), and the number of newly reported cases is still increasing. Approximately 149649 casualties are reported in India at the time of writing (January 04, 2021). The serious danger COVID-19 poses is reflected in the high number of cases of transmission to healthcare workers and police in India.

There is a long history of mathematical models in epidemiology, going back to the eighteenth century. Bernoulli used a mathematical method to evaluate the effectiveness of the techniques of variolation against smallpox, with the aim of influencing public health policy. Most of the models are compartmental models, with the population divided into classes and with assumptions being made about the rate of transfer from one class to another. We consider a Simple Deterministic Model to describe the spread of the virus and compute the number of infected.

**KEYWORDS:** Covid-19, Corona Virus, Lockdown, Quarantine, Social Distancing.

## I. INTRODUCTION

In this research, we study the propagation patterns of epidemic diseases such as the COVID-19 corona virus, from a mathematical modelling perspective. The study is based on an extensions of the well-known susceptible-infected-recovered and become susceptible again (SIS) family of compartmental models [1]. It is shown how social measures such as distancing, regional lockdowns, quarantine and global public health vigilance, influence the model parameters, which can eventually change the mortality rates and active contaminated cases over time, in the real world. As with all mathematical models, the predictive ability of the model is limited by the accuracy of the available data and to the so-called used for modelling the problem. In order to provide the broader audience of researchers a better understanding of spreading patterns of epidemic diseases.

## II. ABOUT MODEL AND DIFFERENTIAL EQUATIONS

When no vaccine is available, the isolation of diagnosed infectives and social distancing are the only control measures available. In a given population at time  $t$ ,  $S(t)$  be the number of susceptible, i.e; the number of those who can be infected,  $I(t)$  be the number of infected persons in the population, and  $R(t)$  be the number of those removed from the population by recovery, immunization, death, hospitalization, or by any other means. If  $N(t)$  is the total population size, we have [2]

$$S(t) + I(t) + R(t) = N = \text{constant} \quad (1)$$

Here we first consider simple epidemic models [3,4], i.e., models in which there are no removals. Let  $n$  be initial number of susceptible in the population in which one infected person has been introduced so that [5]

$$S(t) + I(t) = n + 1, S(0) = S_0 = n, I(t) = I_0 = 1 \quad (2)$$

Now, due to infection, the number of susceptible decreases and the number of infected person increases. It is assumed that the rate of decrease of  $S(t)$ , or the rate of increase of  $I(t)$ , is proportional to the product of the number of susceptible and the number of infected so that this model gives [6,7]

$$\frac{dS}{dt} = -\beta SI, \frac{dI}{dt} = \beta SI \quad (3)$$



From (2) and (3),

$$\frac{dS}{dt} = -\beta S(n + 1 - S) \tag{4}$$

Integrating and using (2), we get

$$S = \frac{n(n+1)}{n+e^{(n+1)\beta t}}, \quad I(t) = \frac{(n+1)e^{(n+1)\beta t}}{n+e^{(n+1)\beta t}} \tag{5}$$

so that

$$\lim_{t \rightarrow \infty} S(t) = 0, \quad \lim_{t \rightarrow \infty} I(t) = n + 1 \tag{6}$$

and, ultimately, all persons will be infected.

In practice, public records do not show the number of infected persons; rather they show the number of new cases reported every day. The corresponding curve gives a relation between  $dS/dt$  and  $t$ . This curve is known as the *epidemic curve*, and the equation for this curve is given by

$$\frac{dS}{dt} = -\frac{\beta n(n+1)^2 \exp [(n+1)\beta t]}{[n+\exp \{(n+1)\beta t\}]^2} \tag{7}$$

which is a symmetrical unimodal curve with a maximum at

$$t_0 = \frac{\ln n}{\beta(n+1)} \tag{8}$$

This shows that the rate of appearance of new cases rises rapidly to maximum at a time, depending on  $\beta$  and  $n$ , and then falls to zero.

### III. SIS MODEL

In this model, a susceptible person can become infected at a rate proportional to  $SI$  and an infected person can recover and become susceptible against at a rate  $\gamma I$  so that we get the model

$$\frac{dS}{dt} = -\beta SI + \gamma I, \quad \frac{dI}{dt} = \beta SI - \gamma I \tag{9}$$

which gives

$$S(t) + I(t) = N + S(0) + I(0) = S_0 + I_0 \quad (I_0 \neq 0) \tag{10}$$

From (9) and (10),

$$dI/dt = (\beta N - \gamma)I - \beta I^2 = kI - \beta I^2 \tag{11}$$

Integrating (11), we obtain

$$I(t) = \begin{cases} \frac{\exp(kt)}{\beta[\exp(kt)-1]/k+I_0^{-1}}, & (k \neq 0) \\ \frac{1}{\beta t+I_0^{-1}}, & (k = 0) \end{cases} \tag{12}$$

As  $t \rightarrow \infty$ ,

$$I(t) = \begin{cases} N - \rho & \text{if } N > \rho = \gamma/\beta \\ 0 & \text{if } N \leq \rho = \gamma/\beta \end{cases} \tag{13}$$

#### SIS Model with Specific Rate of Infection as a Function of t

In this case, (11) becomes

$$dI/dt = [\beta(t)N - \gamma]I - \beta(t)I^2 \tag{14}$$

or

$$dJ/dt + [\beta(t)N - \gamma]J = \beta(t) \tag{15}$$

where

$$J(t) = [I(t)]^{-1} \tag{16}$$

Integrating (15), we obtain

$$J(t)[\exp \{ \int_0^t [\beta(u)N - \gamma] du \}] = \int_0^t \beta(u)[\exp \{ \int_0^t (\beta(u)N - \gamma) du \}] du + J(0)$$

After simplifying this equation and using (16), we obtain

$$I(t) = \frac{\exp [N \int_0^t \beta(u) du - \gamma t]}{\int_0^t \beta(v) \exp [N \int_0^t \beta(u) du - \gamma v] dv + I_0^{-1}} \tag{17}$$

**SIS Model with Constant Number of Carriers**

In this model, infection is spread both ways, by infective and a constant number  $C$  of carriers so that equation (9) becomes

$$\frac{dI}{dt} = \beta (I + C)S - \gamma I = \beta CN + \beta(N - C - \rho)I - \beta I^2 \quad (18)$$

Integrating, we obtain

$$I(t) = \frac{1}{\beta} \frac{\alpha_1 (\beta I_0 + \alpha_2) \exp(\alpha_1 t) + \alpha_2 (\beta I_0 - \alpha_1) \exp(-\alpha_2 t)}{(\beta I_0 + \alpha_2) \exp(\alpha_1 t) - (\beta I_0 - \alpha_1) \exp(-\alpha_2 t)} \quad (19)$$

where

$$\alpha_1, \alpha_2 = \frac{1}{2} \beta \left[ \{(N - C - \rho)^2 + 4CN\}^{1/2} \pm (N - C - \rho) \right] \quad (20)$$

$\alpha_1, \alpha_2$  Correspond to the positive and negative signs respectively in this equation. As  $t \rightarrow \infty, I(t) \rightarrow \alpha_1/\beta$

So that  $I(t)$  is asymptotic to a positive constant for all values of  $N$  and  $\rho$ . Thus, with a constant number of carriers,  **$I(t)$  does not tend to zero.**

**IV. CONCLUSION**

A high number of secondary COVID-19 infections can take place when an infected individual is introduced into a community. It is essential to simulate the process of infection (and death) in advance so as to apply adequate control measures and mitigate the risk of virus diffusion. One of the most commonly used mathematical algorithms to describe the diffusion of an epidemic disease is the SIS model, which we have applied to compute the number of infected, recovered, and dead individuals on the basis of the number of contacts, probability of disease transmission, incubation and infectious periods, and disease fatality rate.

A first analysis of the results of the model is based on the parameters of the SARS disease, and we assume that the parameters do not change during the whole epidemic. When the number of contacts is reduced, the peak decreases in intensity but moves to a later time period, although it is wider. Moreover, a larger number of exposed people does not affect the intensity of the peak but precipitates the epidemic. The incubation period also has an impact on the results, with higher values delaying the epidemic. Increasing the infectious period has the same effect as increasing the incubation period. Moreover, the day when isolation starts is important, since a difference of only few days makes a big difference to the number of casualties.

Models can be used to predict and understand how an infectious disease spreads in the world and how various factors affect the dynamics. Even if the predictions are inaccurate, it has been clear to scientists from many decades that quarantine, social distancing, and the adoption of very strict health and safety standards are essential to stop the spread of a virus. Quarantine was even implemented in medieval times to fight the black death before there was knowledge of the existence of viruses. In this sense, this pandemic reveals the failure of policy-makers, since it is well-known from basic modelling results that earlier adoption of those measures can save lakhs of lives and even prevent the pandemic. The interface of science, society, and politics is still uneasy, even in highly developed countries, revealing a disregard for scientific evidence. Moreover, one of the consequences is that some of these countries do not invest sufficiently in R&D and must acquire the new technology from overseas at a much higher cost.

**$I(t)$  does not tend to zero without vaccination. "NO LAXITY UNTILL THERE IS A VACCINE FOR CURE."**

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