

Short Communication

Understanding the second wave of epidemics using the susceptible-infectious-recovered model

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Received: 16 June 2021

Accepted: 13 July 2021

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ABSTRACT

The world is currently reeling under the COVID-19 pandemic and secondary waves of the same are occurring in different countries. In the current paper, the authors try to explain the exact mathematical concept of a second wave based on their analysis of the popular SIR (susceptible-infectious-recovered) model in epidemiology. Effort is made to graphically and mathematically illustrate the natural infection curve, the necessity of austerity measures, the effects of such measures on the infection curve and the possible reasons for a second wave. The risk of quick mutation and need for effective vaccination is also discussed. It is believed that this analysis will be of immense help to scientists, doctors and policy-makers to devise proper strategies to urgently control the current COVID-19 pandemic, especially in countries where virus variants and secondary waves are occurring.

Keywords: COVID-19, SIR model, Second wave

INTRODUCTION

Second wave is a commonly heard term in epidemiology of infectious diseases. A number of mathematical models are used to study the spread of infectious diseases in a population. The SIR model is one of the most widely used ones. This paper assumes that the reader is versed with this model. Still a basic premise is provided.

In this model, the entire population is broken into 3 compartments as shown in Figure 1.

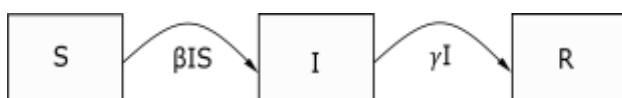


Figure 1: SIR model.

SIR model and herd immunity

S represents those who haven't contracted the disease and are susceptible to it. I represents those who are currently infected with the disease. R represents those who have recovered from the disease. There is in fact another category which represents those who died from the infection, but it is assumed to be negligible compared to the total population size N. Thus we can write:¹

$$S + I + R = N \dots (1)$$

Using standard notations of the SIR model, we know that the reproduction number R of the pathogen may be written as,

$$R = \frac{\beta S}{\gamma} \dots (2)$$

Initially $S \cong N$ and if we assume $N=1$ for the purpose of normalization, we can write,

$$R_0 = \frac{\beta N}{\gamma} = \frac{\beta}{\gamma} \dots (2)$$

We know for SARS-COV-2 virus, R_0 is different in different places. Let's assume $R_0 \cong 2.4$ as reported in some places.² Note that as the epidemic progresses, more and more individuals shift from S to I to R categories. For the rest of the paper, R shall denote the reproduction number of the virus and not the number of recovered individuals, to avoid any confusion. Now as S reduces, so does the effective value of R. When $R=1$, the point of herd immunity is reached. To better appreciate this fact, let us work on the following differential equation which is clear from Figure 1.

$$\frac{dI}{dt} = \beta IS - \gamma I = I(\beta S - \gamma) = (R - 1)\gamma I \dots (3)$$

This differential equation is slightly difficult to solve as R is not constant. However considering R and γ as constant for simplicity, we can solve the equation as,

$$I = I_0 e^{(R-1)t} \dots (4)$$

Now simulating with an R decreasing with time, we get a graph as in Figure 2.

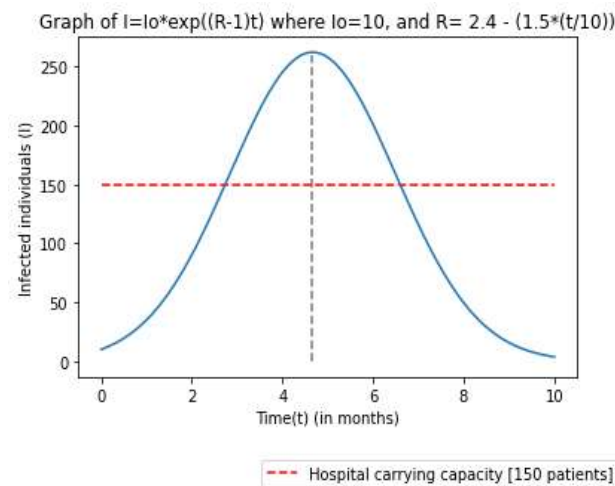


Figure 2: Infection curve without lockdowns/restrictions.

As we can see, one infected individual exponentially infects others until $t=t_r$, whereafter I starts decreasing. Thus t_r may be considered as the point of herd immunity. It is understandable that R keeps decreasing and $R=1$ at $t=t_r$, whereafter the exponent in equation (4) becomes negative.

Now putting $R=1$ in equation (2), we get,

$$S_{herd} = \frac{\gamma}{\beta} = \frac{1}{R_0}$$

Assuming $R_0=2.4$ for a COVID-19 strain, S_{herd} comes to be roughly 40% which means that at least 60% of the susceptible population should have become infected/recovered/dead before herd immunity can set in, whereafter the virus will not be able to spread in any meaningful way, unless it mutates into another strain.³

Effect of lockdown/restrictions

Now let us assume the carrying capacity of hospitals in an area is 150 beds (the red line in Figure 2). Thus to prevent others above the red line from dying without treatment, it is necessary to both reduce the height of the peak as well as delay the peak to buy more time to gear up resources, invent vaccines, drugs. These twin objectives can be simultaneously achieved by reducing the R value, that is, by reducing the β/γ ratio. For this either γ may be increased (through quarantining infected individuals) or β may be decreased (through social distancing and lockdown measures). Let's assume the standard value of β of SARS-COV-2 is β_0 . And let us assume that due to lockdown/restrictions, the population has reduced its effective value to $\beta_{lockdown}$. On just decreasing β , magnitude of $dS/dt = -\beta IS$ also decreases. Thus it takes more time to reach S_{herd} , thus delaying the peak. This increases the X-coordinate of peak, thereby naturally reducing the height of the peak even more as R reduces (refer equation (4)). Thus the twin objectives have been achieved. This is simulated in Figure 3.⁴

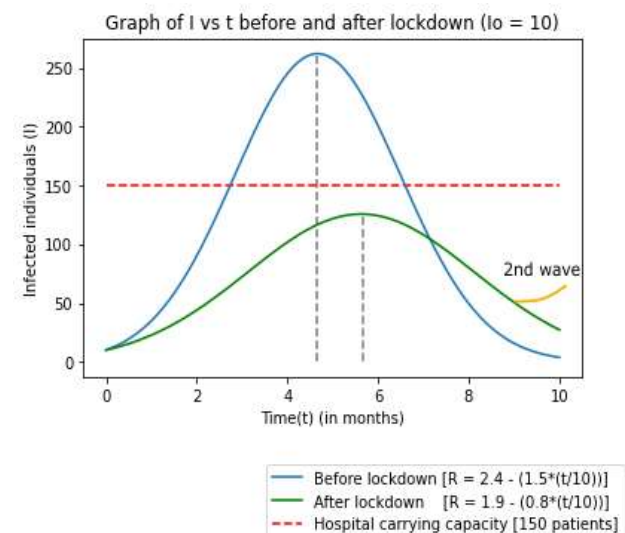


Figure 3: Flattening the curve and 2nd wave.

SECOND WAVE

Now suppose the social distancing/lockdown restrictions are eased at some point P on the downward slope of 'After lockdown' Curve in Figure 3. Say this makes $\beta_{eased} > \beta_{lockdown}$. Now if $[(\beta_{eased}/\gamma) * S_{at \text{ the time of easing}}] = R_{eased}$ exceeds 1, the 'After lockdown' Curve will start exponentially rising again instead of falling. This is called the second wave of the epidemic. However if

$R_{\text{lockdown}} < R_{\text{eased}} < 1$, the curve will still keep decaying albeit more gradually : a case of calibrated easing of lockdown. The same, if done in several phases over a period of time may avoid any second wave altogether as S would soon drop below N/R_0 and β_0 (pre-lockdown behaviour) may be resumed safely.

The aforementioned analysis assumes the virus does not mutate throughout the process. However it often does, in which case S suddenly increases leading to exponential rise of the Infection curve as previous infection provides incomplete immunity against the virus. This may lead to a second wave anytime.

Further if large-scale vaccination⁵ is administered within a reasonably short period of time, S suddenly decreases without passing to the I or R stages, thus leading to an exponential fall in the infection curve after $S < S_{\text{herd}}$.

Furthermore, like any other virus SARS-CoV 2 also has a high propensity to mutate over time. This attribute could possibly be explained by the theory of natural selection proposed by Charles Darwin in 1859⁶. With the advent of a range of vaccines from inactivated virus vaccines, protein based vaccines, viral vector vaccines to RNA and DNA vaccines, the virus is fast mutating. In addition, in thickly populated countries, where vaccination campaigns are slow targeting the middle-aged and elderly population, mutant strains have emerged affecting the young adults. Mutant strains that have cropped up over the past few months include “B.1.1.28” variant, “B.1.351” variant, “B.1.1.7” variant, “N440K” variant, E484Q variant, “B.1.36” variant, B.1.617 (double mutant variant) and the evolving triple mutant variants.⁷

CONCLUSION

It may thus be concluded that any epidemic may be studied as per the SIR model subject to certain conditions. Such studies become difficult if the pathogen mutates effectively and quickly as it leads to rapid rise in S value. The mathematical concepts of herd immunity and second wave were also discussed. Similarly third

wave and other secondary waves may also occur. The aim of various lockdowns/restrictions is to reduce the effective value of the R of the pathogen, so that the rate of spread dI/dt is lowered as well as time can be bought for better preparedness. We believe this study will surely help everyone in better understanding the current COVID-19 pandemic and devising strategies to deal with it.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. *Mathematic Phys Engineer Sci.* 1927;115(772):700-21.
2. Dharmaratne S, Sudaraka S, Abeyagunawardena I, Manchanayake K, Kothalawala M, Gunathunga W. Estimation of the basic reproduction number (R0) for the novel coronavirus disease in Sri Lanka. *Virology J.* 2020;17:144.
3. Randolph HE, Barreiro LB. Herd immunity: understanding COVID-19. *Immunity.* 2020;52(5):737-41.
4. Hunter JD. Matplotlib: a 2D graphics environment. *Comput Sci Engineer.* 2020;9(3):90-5.
5. Anderson RM, May RM. Vaccination and herd immunity to infectious diseases. *Nature.* 1985;318(6044):323-9.
6. Darwin C. *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life* (1859). 1st ed. London: John Murray; 1859.
7. Chakraborty D, Agrawal A, Maiti S. Rapid identification and tracking of SARS-CoV-2 variants of concern. *Lancet.* 2021;397(10282):1346-7.

Cite this article as: Butta R, Butta S, Gupta MK. Understanding the second wave of epidemics using the susceptible-infectious-recovered model. *Int J Res Med Sci* 2021;9:2452-4.