Bifurcation Analysis of an SIR Epidemic Model through Differential Equation Approach

Sumit Kumar Banerjee and John Lanta

Abstract — The well-known SIR models have been around for many years. Under some suitable assumptions, the models provide information about when the epidemic occurs and when it doesn't. The models can be restructured by incorporating birth & death rate, portion of population vaccinated, carrying capacity of population, saturation rate, growth rate, time delay and immunization to analyze the outcome mathematically. In this regard several SIR models including birth, death and immunization as well as bifurcation analysis associated with disease free and epidemic equilibrium have been studied. Findings of this research are with some suitable assumptions how these incorporated parameters as well as bifurcation analysis can play an important role in determining epidemic status in the society in more reliable and convenient way.

Keywords — SIR models, Epidemic, Carrying capacity, Immunization, Equilibrium, Bifurcation.

I. INTRODUCTION

Based on some mathematical assumptions, it is known that epidemics can be modeled mathematically in order to study the severity and prevention mechanism. This model (SIR) is used in epidemiology to compute the number of susceptible, infected, and recovered people in a population at any time. It can be used to explain the change in the number of people needing medical attention during an epidemic. The whole population is divided into three classes, S, the number of susceptible, I, the number of infected and R, the number of recovered during an epidemic. This model assumes that the total population remains the same with closed demography meaning that there is no birth and no natural death. Any disease related death, however, can be included in R. We study the basic SIR model with some reasonable assumptions. Then we include herd immunity, birth and death into the model. The constant vaccination at birth is also considered. The ultimate goal is to model the issue of saturated susceptible population, the time delay of infection to become infectious, the stability of equilibrium solutions and associated bifurcation.

Definition 1: Susceptible individuals are individuals that have never been infected and they are able to catch the disease. Once they have it, they move into the infected compartment. Infected individuals can spread the disease to susceptible individuals. Recovered individuals in the recovered compartment are assumed to be immune for life.

Let S (t) be the number of susceptible individuals I (t) be the number of infected individuals and let R (t) be the number of recovered individuals at time t respectively. It is also assumed that S + I + R = N. Also we normalize this sum by dividing each of the variables by N. We still denote the new variables by the same letters S, I and R.

II. THE SIR MODELS

SIR models have been around for many years, for example [1]-[5] and the references therein. The first one was introduced and published in 1927, in "Contribution to the Mathematical Theory of Epidemics", written by William Kermack and Anderson McKendrick. They introduced the important compartments, which make up the SIR model, S- susceptible, I - infected and R - recovered. They searched for a mathematical answer as to when the epidemic would terminate and observed that, in general, whenever the population of susceptible individuals falls below a threshold value, which depends on several parameters, the epidemic terminates [6], [7].

Published on July 6, 2023.

S. K. Banerjee, PNG University of Technology, Lae, PNG.

⁽corresponding e-mail: sumit.banerjee@pnguot.ac.pg)

J. Lanta, PNG University of Technology, Lae, PNG.

⁽corresponding e-mail: john.lanta@pnguot.ac.pg)

III. THE BASIC MODEL

The population is fixed so S + I + R = 1. The disease spreads through the interaction of susceptible and infected [8]-[11]. We assume that only a fraction of this interaction causes the disease to pass from an individual (I) to a susceptible individual (S) so the rate of change of S is proportional to the product of S and I [3]. We assume that the individuals recover at a rate of β so the period of infection is 1 β days. The only way a person can leave the susceptible group is to become infected. The only way a person can leave the infected group is to recover. Once a person is recovered, the person is no longer susceptible and is immune [12] – [15]. Age, sex, race and social status do not affect the probability of a person being affected. There is no inherited immunity at this time. The people of the population mix homogeneously. Based on the above assumptions the differential equations governing the disease can be modeled as:

$$\frac{ds}{dt} = -\alpha SI$$

$$\frac{dI}{dt} = \alpha SI - \beta I$$

$$\frac{dR}{dt} = \beta I$$
(1)

Remark: Since the total population is assumed to be constant, the third equation can be derived from the first two. Basically we study the first two in detail. It turns out that the epidemic occurs if $\frac{dI}{dt} > 0$ it doesn't if $\frac{dI}{dt} < 0$. So for the epidemic to occur we have to have $\alpha S > \beta$ implying $S > \frac{\beta}{\alpha}$. For the epidemic to terminate the rate of change of I has to be negative, this implies that $S < \frac{\beta}{2}$

Contact Number: Contact number is defined as the average number of adequate contacts of a typical infective during the infection period. It is denoted by σ . Tree diagram for $\sigma = 3$ is as follows:



Fig. 1. Tree diagram for the contact number $\sigma = 3$.

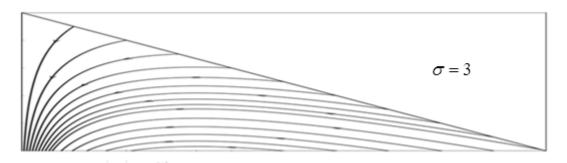


Fig. 2. The phase portrait for the classical SIR epidemic model with contact number $\sigma = 3$.

Definition 2 (Basic Reproductive Number): The basic reproductive number R_0 (the average number of persons infected by one case in a totally susceptible population in absence of interventions aimed at controlling the infection). Since S=1 initially, the ratio $\frac{\alpha S}{\beta} = \frac{\alpha}{\beta} = R_0$.

This is one of the most important parameters in the SIR modeling of any epidemic. R_0 is especially important in this case as it will inform one as to when an epidemic is in progress. So if $R_0 > 1$ an epidemic will occur and if $R_0 < 1$ there will be no epidemic. The values of R_0 are known for various diseases. For example for Covid-19, it is reported to be 1.3-1.6 (in PNG) in (1) The first two equations can be solved for I and S as in (3) The variation of I versus S can be seen from the figure provided (Figure 2). The solutions of I vs. S can be written as (2).

$$I(S) = -S + \frac{1}{R_0} \ln S + 1$$
 (2)

The graphs of this equation (2) are shown for different values of R_0 . The system of equations can be solved for several values of the parameters.

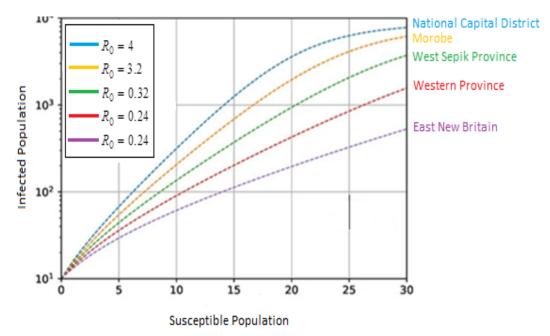


Fig. 3. The graphs of 'I' versus 'S' for different values of R_0 for Covid-19 in different region of PNG. Data collected from PNG National Department of Health (NDOH) and World Health Organization (WHO).

IV. HERD IMMUNITY

For this portion of the model we use p to be the proportion of susceptible population that is immunized before the outbreak of an epidemic and assume the above mentioned conditions, new equations governing the disease can be written as

$$S' = \alpha(1 - p)SI$$

$$I' = \alpha(1 - p)SI - \beta I$$
(3)

An outbreak of the epidemic mathematically means that

$$I' > 0 \Rightarrow \alpha(1-p)SI - \beta I > 0 \Rightarrow \alpha(1-p)S > \beta \Rightarrow \frac{\alpha}{\beta}(1-p)S > 1 \Rightarrow R_0 > \frac{1}{1-p}$$

Note: The value of R_0 is approx. 1.6 (in PNG) for Covid-19. Thus the above inequalities says that at least 38% need to be immunized in order to contain the disease.

V. SIR WITH BIRTH AND DEATH

As a modification to the SIR model we introduce birth and death. We assume that all death is natural. The variable m is used to represent a constant rate of birth and death. The basic reproduction number is now given by $R_0 = \frac{\alpha}{\beta + m}$. Thus the new equations with the consideration of birth and death are:

$$\frac{dS}{dt} = m - \alpha I - mS
\frac{dI}{dt} = \alpha IS - (m + \beta)I$$
 (for disease free)
$$\frac{dS}{dt} = m - \alpha IS - mS
\frac{dI}{dt} = \alpha IS - (m + \beta)I$$
 (for epidemic)
(5)

$$\frac{dS}{dt} = m - \alpha I S - mS$$

$$\frac{dI}{dt} = \alpha I S - (m + \beta)I$$
(for epidemic)
(5)

The system of equations has now two equilibrium solutions. The disease-free equilibrium $(S_1, I_1) = (1,0)$ and the epidemic equilibrium $(S_2, I_2) = \left(\frac{\beta + m}{\alpha}, \frac{m}{\alpha}(R_0 - 1)\right)$

The eigenvalues of the Jacobian matrix reveal the stability of these equilibrium solutions. The Jacobian matrices are computed as follows:

$$J|_{(S_1,I_1)} = \begin{pmatrix} -m & -\alpha \\ 0 & \alpha - \beta - m \end{pmatrix} \quad J|_{(S_2,I_2)} = \begin{pmatrix} -m - (R_0 - 1)m & -\frac{\alpha}{R_0} \\ (R_0 - 1)m & 0 \end{pmatrix}$$

The eigenvalues of $J|_{(S_1,I_1)}$ are -m and $\alpha-\beta-m$. They are both negative if $\alpha-\beta-m<0 \Rightarrow R_0<1$

In this case the eigenvalues of the Jacobian $J|_{(S_2,I_2)}$ are both negative. So the disease free equilibrium is locally stable and the epidemic equilibrium is unstable. Likewise, if $R_0 > 1$ the eigenvalues of $J|_{(S_1,I_1)}$ are of opposite sign and that of $J|_{(S_2,I_2)}$ are both negative. So the epidemic equilibrium is locally stable and the disease free equilibrium is unstable. Authors [4] have mentioned that these locally stable equilibrium are global as well. The value of $R_0 = 1$ thus provides the bifurcation point for the system.

VI. CONSTANT VACCINATION AT BIRTH

For this particular model we introduce certain assumptions that involve a constant vaccination for the newly born, which will enter our population. A proportion p of the new born population has the constant vaccination, while others will enter the population susceptible to infection. We still assume that the population is constant.

$$\frac{dS}{dt} = (1 - p)m - (\alpha I + m)S.$$
 The number of infected is still represented as:
$$\frac{dI}{dt} = \alpha SI - mI - \beta I$$

It has two equilibrium solutions. The disease-free equilibrium $(S_1, I_1) = (1 - p, 0)$ and the epidemic equilibrium $(S_2, I_2) = \left(\frac{\beta + m}{\alpha}, \frac{mR_0(1-p) - m}{\alpha}\right)$. The Jacobian at these equilibrium solutions are computed to be $J|_{(S_1,I_1)} = \begin{pmatrix} -m & -\alpha(1-p) \\ 0 & -\beta - m + \alpha(1-p) \end{pmatrix}$, $J|_{(S_2,I_2)} = \begin{pmatrix} -R_0m(1-p) - 2m & -\beta - m \\ R_0m(1-p) - m & 0 \end{pmatrix}$

be
$$J|_{(S_1,I_1)} = \begin{pmatrix} -m & -\alpha(1-p) \\ 0 & -\beta - m + \alpha(1-p) \end{pmatrix}$$
, $J|_{(S_2,I_2)} = \begin{pmatrix} -R_0 m(1-p) - 2m & -\beta - m \\ R_0 m(1-p) - m & 0 \end{pmatrix}$

It can be seen from the eigenvalues of these matrices that if, $R_0(1-p) < 1$ the disease-free equilibrium is stable while the epidemic equilibrium is unstable. If, $R_0(1-p)>1$ then the disease-free equilibrium is unstable and the epidemic equilibrium is stable. It follows that when, $R_0(1-p)=1$ the bifurcation occurs. This value

of p is called a critical vaccination. So, the critical vaccination, denoted by, p_c is given by $p_c = 1 - \frac{1}{R_0}$

Example. The value of R_0 for Measles is known to be 16–18. So the critical vaccination for this epidemic turns out to be 94.4%. If the new born are vaccinated at a rate higher than 94.4%, then the population will move towards the disease free equilibrium.

VII. SATURATED SUSCEPTIBLE POPULATION

In the case that the birth and death rate are not constant. There are specific assumptions that must be taken into account. These assumptions are that susceptible individuals, S(t) are born at a rate M(S,I,R) which is a function of the densities of the susceptible, infected, and recovered hosts. Susceptible are infected at a given rate given by the product of the densities of susceptible and infected hosts.

$$\frac{dS}{dt} = RS - \frac{RS^2}{K} - \alpha SI$$

The number of infected is still represented as: $\frac{dI}{dt} = \alpha SI - \beta I - MI$

When both host types are well mixed and encounters are random, it is known as mass action kinetics derived from chemical kinetics. Infected hosts recover at a rate β . Susceptible and recovered hosts die at a rate m, which describes the natural death rate due to causes unrelated to the infection. Infected hosts die at a rate, m which includes both natural death and disease induced death. It has two equilibrium solutions. The disease-free equilibrium, $E_0 = (N,0) = (1,0) = \frac{m+\beta}{\alpha}$ and the epidemic equilibrium, $E_1 = (S^*,I^*) = (mS^* + \beta)(R_0 - 1)$

Where $R_0 = \frac{\alpha}{m+\beta}$ is the reproduction number which denotes the number of individuals infected by a single infected individual placed in a totally susceptible population. The Jacobian at these equilibrium solutions are computed to be

$$J|_{(S_1,I_1)(0,0)} = \begin{pmatrix} r & 0 \\ 0 & -\beta - m \end{pmatrix} J|_{(S_2,I_2)(K,0)} = \begin{pmatrix} -r & -\alpha K - m \\ 0 & -\beta + \alpha K - m \end{pmatrix}$$

VIII. MATURATION DELAY

The delayed SIR Epidemic Model makes the assumption that the people in the susceptible group are infectious and carry the disease but only after a certain period of time are they infected.

$$S'(t) = RS(t)\left(1 - \frac{S(t)}{K}\right) - \frac{\beta S(t)I(t-T)}{1+\alpha S(t)} = 1, \quad I'(t) = \frac{\beta S(t)I(t-T)}{1+\alpha S(t)} - \alpha I(t) - \alpha^*I(t) = 0$$

For this project the two equilibrium solutions have been set to 0 and 1 to see if there is an epidemic or a disease free occurrence. We are able to decipher between the two by finding the infection free equilibrium

$$E_{0} = (0,0), E_{1} = (K,0), E_{+} = (S^{*}, I^{*}) = \left(\frac{\alpha + \alpha^{*}}{\beta - \sigma(\alpha + \alpha^{*})}, \frac{rS^{*2}(R_{0} - 1)}{K(\alpha + \alpha^{*})}\right)$$

In conclusion, we see that over a certain period the population is susceptible and infectious, but not everyone is infected at one specific time period. It takes a certain period of time for infection to circulate throughout a population.

IX. CONCLUSION

In conclusion, we reviewed, analyzed, and discussed the continuous SIR epidemic model. In the basic SIR model only rate of infection and recovery rate were imposed, whereas in the restructure model with respect to different reliable important factors say, basic reproduction number (R_0) , portion of population vaccinated (p), carrying capacity (K), constant birth and death rate (m), saturation rate (σ) and growth rate (r) have been incorporated in order to determine the bifurcation point as well as the status of the epidemic in the society in a more reliable and convenient way and as a result that will help the society to adopt the necessary precautionary measures well in advance against the outbreak and save the community from the devastation.

ACKNOWLEDGMENT

The authors would like to express their sincere gratitude to the PNG National Department of Health (NDOH) and World Health Organization (WHO) for their kind cooperation towards providing the genuine data and help them to forecast the explicit results based on the model formulated. The authors would also

ISSN: 2736-5484

like to express their gratitude to the editor and learned reviewers for their valuable comments and suggestions to improve the earlier version of this manuscript. There is no conflict of interest as declared by the authors.

CONFLICT OF INTEREST

Authors declare that they do not have any conflict of interest.

REFERENCES

- [1] Kermack W, McKendrick A. A short history of mathematical population dynamics. Springer-verlag London limited; 2011
- Fraser C, Donnelly CA. Pandemic potential of a strain of influenza A (H1N1). Early findings. Science. 2009; 324(2):1557-1561.
- [3] Banerjee SK, Andrews B. Mathematical Analysis of Regression model Epidemiology. American Academic Scientific Research Journal for Engineering, Technology, and Sciences. 2023; 93(1): 39-49.
- Stephen BH and Steven RC. Designing Clinical Research. Williams and Wilkins. 2017; 7(1): 131-145.
- Herbert H, Dimitri B. Mathematics of Epidemiological Stabilities. SIAM Review. 2017; 8(2): 217-235.
- [6] Brooks-Pollock E, Ken TDE. Pigs didn't fly, but swine flu. Mathematics Today. 2011; 47(1): 36-40.
- [7] Stone L, Shulgin B, Agur Z. Analysis of an SIR epidemic model with pulse vaccination and distributed time delay. Journal of Biomedicine and Biotechnology. 2007; 28(1): 239-254.
- Sharon K, Leo K, Thomas M. Applied Regression Analysis and Multivariate Methods. London: Cole Publishing; 2018.
- [9] Kenneth JR, Sander G. Modern Epidemiology. PA: Lippincott-Raven Publishing; 2018.
- [10] Andrea M and Peter H. Nonlinear seasonally forced epidemiological models. Journal of Mathematical Biology. 2016; 52(2):
- [11] Nicolas B, Herbert H, Dimitri B. Wavering in seasonally forced epidemiological models. Journal of Mathematical Biology. 2018; 89(2):118-148.
- [12] Elyas H, Dimitri B. An SIR epidemic model with inconsistent latency infectious period. Mathematical Biosciences and Engineering. 2017; 3(1): 654-676.
- [13] Herbert H, Lawrence DW. Mathematics of inconsistent incidence rate of infectious diseases. SIAM review. 2017; 10(1): 233-
- [14] Carlos R, Andrea M, Nicolas B. Persistence in seasonally forced epidemiological models. Journal of Mathematical Biology.2012; 64(3): 933-949.
- [15] Elizabeth B, Dimitri B. An SEIR epidemic model with constant latency time and infectious period. Mathematical Biosciences and Engineering. 2018; 8(1): 931-952.



B. Sumit Kumar studied his M.Sc., (2001), M.Phil, (2002), PhD, (2005) and D.Sc, (2008) in Mathematics from University of Hull, England. He has been working as a full professor of Mathematics since 2014 in various organizations and presently associated with PNG University of Technology, Lae, Papua New Guinea. His area of specialization is Mathematical Modeling and stability Analysis. Dr. S.K. Banerjee has published 68 research papers in various SCIE and ESCI indexed International Journals and proceedings of repute and presented 32 papers in various National and International conferences. He has worked as a Research & Development Dean (R&D) as well as a co-principal investigator in two externally funded research projects and supervised 4 Ph.D. scholars so far. He has authored 5 books on Higher Mathematics.



L. John studied his B.Sc., (1992) in Physics and completed his M. Sc, (2010) in Mathematical Science from Queensland University of Technology, Australia. Presently he is pursuing his Ph.D. on Topological algebraic structures. He has worked as a HOD in the Department of Mathematics & Computer Science at Papua New Guinea University of Technology for a considerable number of years. His research interests includes, but not limited to, Differential Equations, Mathematical Modeling and Topology. He is currently active with research work with the Department of Mathematics and Computer Science at the Papua New Guinea University of Technology, Papua New Guinea.