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STABILITY ANALYSIS OF A GENERAL SIR EPIDEMIC MODEL

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Abstract. *A proper structure of mathematical model is required to understand the large size dynamics of the spread of an infectious disease. In this paper, we discuss a general SIR epidemic model which represents the direct transmission of infectious disease. The reproduction number R_0 is determined and the local and global stabilities of both the disease-free and the endemic equilibrium are derived.*
Keywords: Epidemic model; basic reproduction number; global stability.

1. **Introduction.** Infectious diseases are caused by pathogenic microorganisms, such as bacteria, viruses, fungi and parasites. The diseases can spread directly or indirectly from one person to another or from animals/birds to humans. Theses diseases are a leading cause of death worldwide. Despite all the advancement in medicines, infectious disease outbreaks still pose a significant threat to the public health and economy [12-20]. The spread rates of different infectious diseases are rising due to changes in human behavior, inappropriate use of antibiotic drugs, increased trade and travel, larger and denser cities and the emergence of new and resurgent pathogens. Furthermore, the transmission interactions in a community are very complicated and it is hard to understand the large size dynamics of the spread of the disease without a proper structure of mathematical model.

Mathematical modeling has become a valuable tool to understand the dynamics of infectious disease and to support the development of control strategies [2-8]. A lot of mathematical models for different infectious diseases were proposed by several researchers and scientists. Shulgin et al. [17] considered a simple Susceptible-Infected-Recovered (SIR) epidemic model with pulse vaccination. In their work they presented that if certain conditions regarding the magnitude of vaccination proportion together with period of pulses are satisfied then the pulse vaccination leads to epidemic eradication. Kribs-Zaleta and Velasco-Henandez [10] considered a simple two dimensional SIS model with vaccination showing backward bifurcation. Farrington [1] studied the impact of vaccination program on the transmission potential of the infection in large populations and obtained relation between vaccine efficacy against transmission, reproduction number and vaccine coverage.

In this work we discuss the stability analysis of a general Susceptible-Infected-Recovered (SIR) epidemic model of infectious disease. We present both disease-free equilibria and the endemic equilibria of the proposed model. The local dynamics of a general SIR is determined by the basic reproduction number R_0 which depends on the parameter values. For $R_0 \leq 1$ the disease-free equilibrium is locally asymptotically stable while for $R_0 > 1$ the endemic equilibrium exists. By using the theory of Lyapunov function, we present the global asymptotic stability.

The paper is organized as follows. In Section 2, we present a formulation of general epidemic model. In section 3, we show the local and global stability of both disease-free and endemic equilibrium. Numerical results and conclusion of our work are studied in Section

2. **Model frame work.**
In this section, we formulate an epidemic model for the spread of a general infectious disease. We split the total population $N(t)$, into three distinct subclasses which are susceptible $S(t)$, infectious $I(t)$ and recovered $R(t)$. The model can be represented by the following system of differentials equations.

$$\begin{aligned} \frac{dS(t)}{dt} &= \mu - \lambda S(t)I(t) - \mu S(t), \\ \frac{dI(t)}{dt} &= \lambda S(t)I(t) - \gamma_1 I(t) - \mu I(t), \\ \frac{dR(t)}{dt} &= \gamma_1 I(t) - \mu R(t), \end{aligned} \tag{1}$$

with the initial conditions

$$S(0) \geq 0, \quad I(0) \geq 0, \quad R(0) \geq 0. \tag{2}$$

Here μ is the recruitment and natural death rate, λ is the effective contact rate between susceptible and infected individuals and γ is the recovery rate of infected individuals.

By considering the total population density, we have $S(t)+I(t)+R(t) = 1 \Rightarrow R(t) = 1-S(t)+I(t)$.

Therefore it is enough to consider

$$\begin{aligned} \frac{dS(t)}{dt} &= \mu - \lambda S(t)I(t) - \mu S(t), \\ \frac{dI(t)}{dt} &= \lambda S(t)I(t) - \gamma_1 I(t) - \mu I(t). \end{aligned} \tag{3}$$

The feasible region for the above system is

$$\Omega = \{(S(t), I(t)) \in R_+^2, \quad S(t) + I(t) \leq 1\}.$$

Since

$$\begin{aligned} S(t) &= 0 \Rightarrow \tfrac{dS(t)}{dt} = \mu > 0, \\ I(t) &= 0 \Rightarrow \tfrac{dI(t)}{dt} = 0, \\ \tfrac{dS(t)}{dt} + \tfrac{dI(t)}{dt} &= -\gamma I(t) \leq 0. \end{aligned}$$

Thus Ω is positively invariant.

3. **Threshold analysis.** In this section we show the stability analysis. The disease-free equilibrium (DFE) point is $E_0 = (1, 0)$.
To find the endemic equilibrium point $E_1 = (S^*, I^*)$ we set the right hand side of the system (3) equal to zero to get

$$S^* = \frac{\gamma + \mu}{\lambda}, \qquad I^* = \frac{\mu}{\lambda}(R_0 - 1),$$

where

$$R_0 = \frac{\lambda}{\gamma + \mu}.$$

In mathematical epidemiology an important concept is related to the basic reproduction number R_0 as it serves as a threshold parameter that governs the spread of infectious diseases in a population. This is defined as the second expected number produced from just one individual in a susceptible population. For any infectious disease, one of the most important concerns is its ability to invade a population [6]. This can be expressed by a threshold parameter R_0. If $R_0 < 1$, then each infected individual in its entire period of infectivity, will produce less than one infected individual on average. In DFE case the system is locally asymptotically stable, which shows that the disease will be wiped out of the population. If $R_0 > 1$, then the each infected individual in its entire

infective period having contact with susceptible individuals will produce more than one infected individual, which will then lead to the disease invading the susceptible population, and the DFE is unstable [18].

The linearization by Routh Hurwitz criteria around the endemic equilibrium point E_1 is [3] is locally asymptotically stable for $R_0 > 1$.

To show that the proposed system is globally asymptotically stable, we use the Lyapunov function theory for both the disease free and the endemic equilibrium. First we present the global stability of the disease-free equilibrium.

Theorem 3.1. *If $R_0 \leq 1$, then the disease-free equilibrium E_0 of the system is globally asymptotically stable on Ω.*

Proof. *To establish the global stability of the disease free equilibrium E_0, we construct the following Lyapunov function $V : \Omega \to R$:*

$$V(S, I) = I(t)$$

Calculating the time derivative of V along the solution of the proposed system, we obtain

$$V^{'}(t) = \lambda S(t)I(t) - (\gamma + \mu)I(t),$$

$$= (\gamma + \mu)(R_0 S(t) - 1)I(t).$$

We see that $V^{'}(t) \leq 0$ for $R_0 < 1$.

If $R_0 < 1$ then $V^{'}(t) = 0 \Leftrightarrow I(t) = 0$.

If $R_0 = 1$ then $V^{'}(t) = 0 \Leftrightarrow S(t) = 1$.

Hence by LaSalle's invariance principle [11] *the diseases-free equilibrium point E_0 is globally asymptotically stable on Ω.* □

Theorem 3.2. *The endemic equilibrium $E_1 = (S^*, I^*)$ of the system is globally asymptotically stable on Ω.*

Proof. *For the global stability of the endemic equilibrium E_1, we construct the Lyapunov function $L : \Omega_+ \to R, where \quad \Omega_+ = \{(S(t), I(t)) \in \Omega \mid S(t) > 0, \quad I(t) > 0\}$ is given by*

$$L(S, I) = W_1\left[S - S^* \ln\left(\frac{S}{S^*}\right)\right] + W_2\left[I - I^* \ln\left(\frac{I}{I^*}\right)\right].$$

Where W_1 and W_2 are positive constant to be chosen latter. By taking the derivative of the above function, we have

$$\begin{aligned} \frac{dL}{dt} &= W_1(S - S^*)(-\lambda I - \mu + \frac{\mu}{S}) \\ &\quad + W_2(I - I^*)(\lambda S - (\gamma + \mu)). \end{aligned}$$

Considering the equilibrium point, we have $-\mu = \lambda I^* - \frac{\mu}{S}$ and $-(\gamma + \mu) = -\lambda S^*$. So the above equation becomes

$$\frac{dL}{dt} = \lambda(W_2 - W_1)(S - S^*)(I - I^*) - W_1\mu(S - S^*)^2.$$

For $W_1 = W_2 = 1$, we have

$$\frac{dL}{dt} = -\mu\frac{(S - S^*)^2}{SS^*} \leq 0.$$

Also we obtain

$$\frac{dL}{dt} = 0 \Leftrightarrow S = S^*.$$

Hence by LaSalle's invariance principle [11] *the endemic equilibrium point E_1 is globally asymptotically stable on Ω.* □

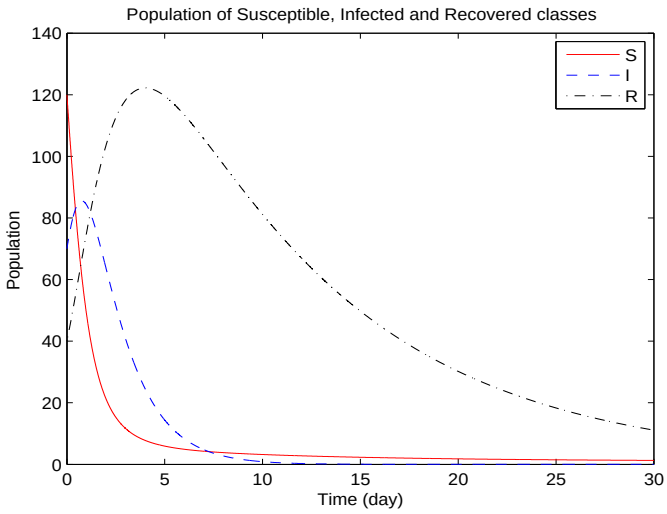

FIGURE 1. The plot shows the population of susceptible, infected and recovered individuals.

4. **Numerical simulation and Conclusion.** In this section we use an iterative method to find the numerical simulation. For numerical simulation we consider the parameter value $\mu = 0.1$, $\lambda = 0.0098$ and $\gamma = 0.5$. By using Runge-Kutta order 4 scheme, we solve our proposed model (1). The plots in Figure 1. shows the population of susceptible, infected and recovered individuals.

We did not consider a mathematical model to represents some special disease in this paper but our main goal was to give idea that the transmission of infection can be easily studied by epidemic models. Analysis of the model showed that there are two equilibria one is disease-free equilibria and the other one is endemic equilibria. The local dynamics of the proposed model are determined by the basic reproduction number R_0 which depends on the parameter values. We also presented that for $R_0 \leq 1$ the disease-free equilibrium is locally asymptotically stable while for $R_0 > 1$ the endemic equilibrium exists.

REFERENCES

[1] Farrington, C. P. (2003). On vaccine efficacy and reproduction numbers. Mathematical biosciences, 185(1), 89-109.
[2] Fenner, F., Henderson, D. A., Arita, I., Jezek, Z., and Ladnyi, I. D. (1988). Smallpox and its eradication, WHO 1998.
[3] Gantmacher,F.R. (1959). The Theory of Matrices. Chelsea Publ. Co., New York, .
[4] Gumel, A. B., Shivakumar, P. N., and Sahai, B. M. (2001). A mathematical model for the dynamics of HIV-1 during the typical course of infection. Nonlinear Analysis-Theory Methods and Applications, 47(3), 1773-1784
[5] Handel, A., Longini, I. M., and Antia, R. (2007). What is the best control strategy for multiple infectious disease outbreaks?. Proceedings of the Royal Society B: Biological Sciences, 274(1611), 833-837.
[6] Heffernan, J. M., Smith, R. J., and Wahl, L. M. (2005). Perspectives on the basic reproductive ratio. Journal of the Royal Society Interface, 2(4), 281-293.
[7] Hethcote, H. W. (2000). The mathematics of infectious diseases. SIAM review, 42(4), 599-653..
[8] Kamien, M. I., and Schwartz, N. L. (1991). Dynamic optimization: the calculus of variations and optimal control in economics and management (Vol. 1, No. 4). New York: North-Holland.
[9] Kar, T. K., and Batabyal, A. (2011). Stability analysis and optimal control of an SIR epidemic model with vaccination. Biosystems, 104(2), 127-135.

[10] Kribs-Zaleta, C. M., and Velasco-Hernndez, J. X. (2000). A simple vaccination model with multiple endemic states. Mathematical biosciences, 164(2), 183-201.
[11] LaSalle, J. P. (1987). The stability of dynamical systems (Vol. 25). Society for Industrial and Applied Mathematics.
[12] Pontriaguine, L. S., Boltnskij, V. G., Gamkrelidze, R. V., and Mienko, E. F. (1962). The mathematical theory of optimal processes. Interscience Publishers.
[13] Riedel, S. (2005). Edward Jenner and the history of smallpox and vaccination. Proceedings (Baylor University. Medical Center), 18(1), 21.
[14] Ullah, R., Zaman, G., and Islam, S. (2012). Prevention of influenza pandemic by multiple control strategies. Journal of Applied Mathematics, 2012. doi:10.1155/2012/294275.
[15] Ullah, R., Zaman, G., and Islam, S. (2012) *Global dynamics of avian-human influenza with horizontal transmission in human population,* Life Sci. J. 9 5747-5753.
[16] Ullah, R., Zaman, G., Islam, S and Ahmad, I, *Dynamical features and vaccination strategies in an SEIR epidemic model,* Res. J. Recent Sciences. (In Press)
[17] Shulgin, B., Stone, L., and Agur, Z. (1998). Pulse vaccination strategy in the SIR epidemic model. Bulletin of Mathematical Biology, 60(6), 1123-1148.
[18] Van den Driessche, P., and Watmough, J. (2008). Further notes on the basic reproduction number. In Mathematical Epidemiology (pp. 159-178). Springer Berlin Heidelberg.
[19] Zaman, G., Han Kang, Y., and Jung, I. H. (2008). Stability analysis and optimal vaccination of an¡ i¿ SIR¡/i¿ epidemic model. BioSystems, 93(3), 240-249.
[20] Zaman, G., Khan, M. A., Islam, S., Chohan, M. I., and Jung, I. H. (2012). Modeling dynamical interactions between Leptospirosis infected vector and human population. Applied Mathematical Sciences, 6(26), 1287-1302