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Research Article

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Stability Analysis of an SIR Model with a Nonlinear Incidence Rate and Latency

Ime Okonna*[1], Mfon Okonna[2]

[1]Dept. of Gen. Studies, Akwa Ibom State Polytechnic, Ikot Osurua, Akwa Ibom State, Nigeria
[2]Dept. of Statistics, Akwa Ibom State Polytechnic, Ikot Osurua, Akwa Ibom State, Nigeria
Email: imeokonna2004@yahoo.com

Abstract In this paper, we consider an SIR epidemic model with nonlinear incidence rate and latency. The local stability conditions of the model proposed are established in the absence of time delay {τ= 0} and in the presence of time delay {τ> 0}. Numerical simulations are done with the DDE suite in Matlab and are found to be in agreement with analytical results obtained and the transient oscillations occasioned by the presence of time delay highlighted.

Keywords Stability Analysis, SIR Model, Nonlinear Incidence Rate, Latency

1. Introduction

In this paper, we consider saturation incidence rate and assume the force of infection in the form $\frac{\varphi S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}$ which is saturated with the infectives. Introduction of the saturation factor makes the model more realistic because the number of effective contacts between infective individuals and susceptible individuals may saturate at high infective levels due to protective measures by the susceptibles e.g Measles. Incidence rate of this type have been widely studied, for example Z. Hu et al in [1], M.E. Alexander and S.M. Moghadas in [2] and S. Ruan and W. Wang in [3]. In our model, a delay term (τ> 0) is introduced to represent time delay describing the latent period of the disease. Systems with nonlinear incidence rate and latency are widely studied (See [4] – [12] and references therein).

Analytically, we derive a threshold value (R_0) and prove that when R_0< 1, the disease free equilibrium point is locally asymptotically stable and unstable otherwise. Numerical simulations support our analytical calculations and also show that we have global asymptotic stability of the disease free equilibrium for R_0< 1 and the endemic equilibrium for R_0> 1. The paper is organized as follows: The model is described in Section 2. The basic reproduction number and relevant results for the stabilities of the disease free and endemic equilibria could be found in Section 3. We have numerical simulations in 4 and conclusion in Section 5.

2. Derivation of the Model

We consider an SIR model with a three dimensional differential equation system. Individuals are assumed to be in one of the following epidemiological states: Susceptibles (S) - at risk of contracting the disease, Infectives (I) - infected and capable of transmitting the disease, and Recovered (R) - population recovered from the infection. All recruitment to the system is into the susceptible class, and occurs at a constant rate β. We consider here a saturated incidence rate and assume the force of infection in the form $\frac{\phi S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}$ which is saturated with the infectives. We present the model as follows:

$$\frac{dS}{dt} = \beta - \mu S - \frac{\phi S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)}$$

$$\frac{dI}{dt} = \frac{\phi S(t-\tau)I(t-\tau)}{1+\alpha I(t-\tau)} - (\mu + \mu_d + \Upsilon)I \quad (1)$$

$$\frac{dR}{dt} = \Upsilon I - \mu R$$

This model has the natural death rate μ and disease induced death rate μ_d. An infected individual has a recovery rate of (Υ) into the recovery compartment. ϕ is the infection rate while α is the saturation factor. Since this model is for human population, we assume that all its state variables and parameters are nonnegative for all t≥ 0. The region biologically relevant is given by

$$\Omega = \left\{(S,I,R) \in \mathcal{R}_+^3 : 0 \le S+I+R \le \frac{\beta}{\mu_m}\right\} \quad (2)$$

The total human population is given by N = $S + I + R$, so that $dN/dt \le \beta - \mu_m N$, thus $N \to \beta/\mu_m$ as $t \to$ □, Where $\mu_m = min\{\mu, \mu_d\}$.

3. Stability Analysis

The basic reproduction number for the model obtained by the first generation matrix approach introduced by Diekmann et al [13] is given as

$$R_0 = \frac{\phi\beta}{\mu(\mu+\mu_d+\Upsilon)} \quad (3)$$

The disease-free equilibrium given by $E^0 = (S^0, I^0, R^0)$ is the only equilibrium for $R_0 \le 1$, where

$$S^0 = \frac{\beta}{\mu}, \quad I^0 = 0, \quad R^0 = 0$$

If $R_0 > 1$, then there is also an endemic equilibrium given by $E^* = (S^*, I^*, R^*)$, where

$$S^* = \frac{(\alpha\beta + \mu + \Upsilon + \mu_d)}{\phi + \mu\alpha}$$

$$I^* = -\frac{(\mu^2 + \mu\Upsilon + \mu\mu_d - \phi\beta)}{(\mu^2\alpha + \mu\alpha\Upsilon + \mu\alpha\mu_d + \mu\phi + \phi\Upsilon + \phi\mu_d}$$

$$R^* = -\frac{\Upsilon(\mu^2 + \mu\Upsilon + \mu\mu_d - \phi\beta)}{\mu(\mu^2\alpha + \mu\alpha\Upsilon + \mu\alpha\mu_d + \mu\phi + \phi\Upsilon + \phi\mu_d}$$

Stability of the Disease Free Equilibrium at $\tau = 0$.

The characteristics equation after linearizing (1) about the disease free equilibrium E^0 gives

$$(\lambda+\mu)^2\left(-\mu\lambda + \phi\beta e^{-\tau\lambda} - \mu\Upsilon - \mu^2 - \mu\mu_d\right) = 0 \quad (4)$$

This gives $\lambda_{1,2} = -\mu$ and the solution to the following transcendental equation

$$\mu\lambda - \phi\beta e^{-\tau\lambda} + \mu\Upsilon + \mu^2 + \mu\mu_d = 0 \quad (5)$$

At $\tau = 0$, equation (5) reduces to

$$\mu\lambda - \phi\beta + \mu\Upsilon + \mu^2 + \mu\mu_d = 0$$

This gives

$$\lambda_3 = (\mu + \mu_d + \Upsilon)\{R_0 - 1\}$$

This is stable if $R_0 < 1$ and marginally stable if $R_0 = 1$, giving us the following theorem.

Theorem 1.

The disease-free equilibrium point of the System (1) is locally asymptotically stable at $\tau = 0$ when $R_0 < 1$, marginally stable when $R_0 = 1$ and unstable when $R_0 > 1$.

Stability of the Disease Free Equilibrium at $\tau > 0$.

At $\tau > 0$, equation (5) gives

$$\mu^2 + \mu\Upsilon + \mu\lambda + \mu\mu_d = \phi\beta e^{-\tau\lambda} \quad (6)$$

Suppose $\lambda = i\omega, (\omega > 0)$ is a root of (6)

$$\mu^2 + \mu\Upsilon + i\mu\omega + \mu\mu_d = \phi\beta e^{-i\tau\omega}$$

Squaring both sides and adding, we have the following

$$\omega^2 + (\mu + \mu_d + \Upsilon)^2\left[1 - {R_0}^2\right] = 0 \quad (7)$$

Equation (7) has no real root for $R_0 < 1$. Hence it is stable for all $\tau > 0$ giving the following theorem

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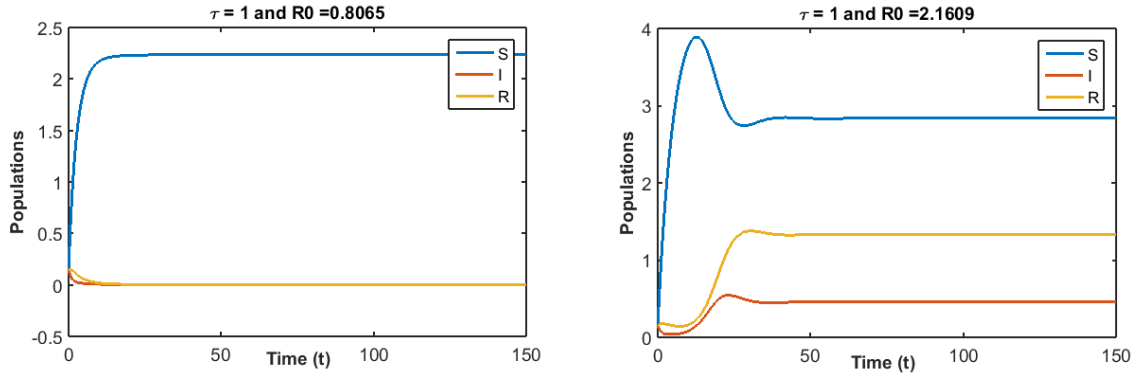
Theorem 2.
The disease free equilibrium point (E^0) of System (1) is locally asymptotically stable at $\tau > 0$, whenever $R_0 < 1$ and unstable when $R_0 > 1$.

4. Numerical Simulation

We show in this section numerically, the established results in earlier sections about the stability of the System (1) as it relates to the time delay (τ), and the basic reproduction number (R_0). We use the dde23 suite in Matlab to simulate the System (1) with the parameters as shown below the figures. The parameters are chosen solely for simulation convenience and do not reflect actual collected data.



(a) $R_0 = 0.8065 < 1$, $\mu = 0.048$ (b) $R_0 = 2.1609 > 1$, $\mu = 0.38$

Figure 1: Other parameters are: $\beta = 0.85$, $\phi = 0.34$, $\alpha = 065$, $\mu_d = 0.038$ and $\Upsilon = 0.525$

Solutions converge to the disease-free equilibrium point for $\tau = 1$ and $R_0 = 0{:}8065 < 1$, Fig 1a and endemic equilibrium point for $\tau = 1$ and $R_0 = 2{:}1609 > 1$, Fig 1b, showing that the dynamics of the system depends only on the basic reproduction number. At $R_0 < 1$, the disease free equilibrium becomes stable which shows that the infection dies out of the population while at $R_0 > 1$, the endemic equilibrium becomes stable meaning that the disease will persist in the population. This simulation agrees with Theorems (1) and (2) in Section 3.
In the next figure, we show the effect of the time delayτon System (1) by plotting the number of infectives for different values of τ.

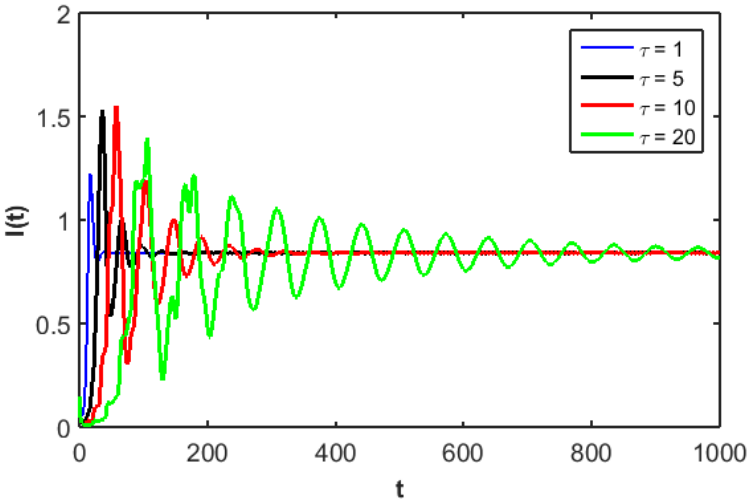


Figure 2: Simulation of the evolution of the number of infected individuals for different values of τ. Other parameters are: $\beta = 0.85$, $\mu = 0.098$, $\varphi = 0.34$, $\mu_d = 0.038$ and $\Upsilon = 0.525$.

In figure 2, at $\tau > 0$, we observe an initial transient oscillations that is prolonged as τ increases, but the endemic equilibrium point of the system is always stable for all time delays.

5. Conclusion

In this paper, we formulated an SIR epidemic model with a nonlinear incidence rate and latency. The threshold value R_0 was found and analytical calculations and numerical simulations show that the local and global dynamics of the System (1) do not depend on the time delay but are completely determined by the values of the threshold number R_0. We found that if $R_0 < 1$, the disease free equilibrium point is always stable for $\tau \geq 0$ and the endemic equilibrium point is always stable for $\tau \geq 0$ if $R_0 > 1$.

The effect of time delay (τ) and the long term behaviour of the system (1), as highlighted in Fig 2, shows that at $\tau > 0$, an initial transient oscillations exist. The oscillations get prolonged as τ increases, but the endemic equilibrium point of the system is always stable for all time delays. The epidemiological interpretation of the oscillations experienced in the system at $\tau > 0$, is that the population will have an initial period of epidemic fluctuation before it finally become endemic.

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