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Analysis of SEIR Epidemic Model Engraft with Incompatible Incidence Rate

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Abstract: A passivity SEIR epidemic model with inconsistent incidence rate embedded with latency period for the imparting dynamics of epidemics is succeed and thoroughly inspected. The problem is constructed by a system of nonlinear ordinary differential equations analyzing the evaluation of susceptible, exposed, infected and removed individuals. The suggested model is established in terms of existence, positivity and boundedness of solutions. Four equilibrium points have been discussed, namely, the disease free equilibrium, endemic equilibrium with respect to strain 1, endemic equilibrium with respect to strain 2 and the terminal endemic equilibrium with respect to both strains. By constructing the suitable stability analysis function the global stability of the disease free equilibrium is proved depending on the basic reproduction number. Furthermore by using other well-known functionals the global stability results of the endemic equilibria are established depending on the strain 1 reproduction number and strain 2 reproduction number. Necessary numerical simulations are performed in order to confirm the theoretical results. Numerical comparison between the model results and clinical data was conducted. The findings of this research includes the model consistence of discordant compartments which are globally asymptotically stable aseptic equilibrium in state have an epidemiological threshold value (also known as basic reproduction rate) less than unity.

Keywords: Epidemic, Incidence, Asymptotically, Threshold, Basic Reproduction Number

1. Introduction

Mathematical models on contagious diseases have been extensively used to gain apprehensively into the spread and control of crop up diseases. The agitation of these models is usually set on by a threshold quantity known as the basic reproduction number. The basic reproduction number is defined as the number of secondary cases produced by completely susceptible populations [1-4, 6]. Categorically when less than unity is a small inundation of infected individuals, will not generate large outburst and the disease will dies out in time. On the other hand when it will exceeds unity, the disease will carry on. A basic epidemic model is supported by at least two equilibrium, namely disease-free and endemic. Several models found in the literature [1, 2, 3, 8,, 12, 14] have been used to show that when crosses the doorway then an asymptotic local stability is transferred from the disease-free to endemic state. In some cases it can be exhibit that the transfer of asymptotic stability is independent of initial conditions, i.e. it is comprehensive [7, 9]. Let $S(t), I(t), N(t)$ denote the number of susceptible, infected individuals and total size of the population at time t respectively. Further let $\beta(N)$ be the average number of contacts that is sufficient to transmit infection. Then the force of infection given by $\frac{\beta(N)I}{N}$ will represents the average number of contacts a susceptible individual makes with infectious individuals per unit time. If $\beta(N)=\beta N$ i.e. contact rate depends on total population then the incidence function $g_1(I)=\beta I$ is called mass action incidence. If $\beta(N)=\beta$ (a constant) then the incidence function $g_2(I)=\frac{\beta I}{N}$ is called standard incidence [4-6]. These two functions are widely used in modeling the transmission dynamics of human diseases [8, 10, 11, 13]. Another widely used incidence function is the Holling type II incidence

function given by $g_3(I)=\dfrac{\beta I}{(1+\omega I)}$ with $\omega>0$

2. Model Formulation

The total population at time t denoted by $N(t)$ is subdivided into five compartments such as susceptible $S(t)$, exposed $E(t)$, uneducated infected individuals $I_u(t)$, educated infected individuals $I_e(t)$ and recovered individuals $R(t)$. Thus we have

$$N(t)=S(t)+E(t)+I_u(t)+I_e(t)+R(t) \tag{1}$$

The susceptible population is increased by the recruitment of individuals into the population at a rate π. Susceptible individuals may acquire infection from following effective contact with infected individuals at a rate $\lambda(t)$, where

$$\lambda(t)=\beta\left(\frac{I_u}{1+\alpha_1 I_u}+\frac{\eta I_e}{1+\alpha_2 I_e}\right) \tag{2}$$

In (2) is the effective contact rate while the modification parameter $0<\eta<1$ accounts for the assumed depletion in disease channeling by educated infected individuals in comparison to uneducated infected individuals in the I_u class. The population of susceptible individuals is further decreased by natural death. Thus the rate of change of the susceptible population is given by

$$\frac{dS}{dt}=\pi-\left[\mu+\lambda(t)\right]S(t) \tag{3}$$

The population of exposed individuals is generated by the infection of susceptible individuals at a rate of $\lambda(t)$. This population is decreased by development of disease symptoms at a rate κ and natural death at a rate μ so that

$$\frac{dE}{dt}=\lambda(t)S(t)-(\kappa+\mu)E(t) \tag{4}$$

The population of uneducated infected individuals is generated at a rate κ. It is decreased by natural recovery at a rate γ_1, education at a rate σ, natural death at a rate μ and disease-induced death at a rate δ_1. This gives

$$\frac{dI_u}{dt}=\kappa E(t)-(\sigma+\gamma_1+\mu+\delta_1)I_u(t) \tag{5}$$

The population of educated infected individuals is generated by the education of infected individuals at a rate of σ. This population is decreased by recovery at a rate γ_2, natural death at a rate μ and disease-induced death at a rate $\delta_2<\delta_1$. It is assumed that the disease persuade mortality rate of educated infected individuals is lower than uneducated infected individuals. Hence the rate of change of this population will be given by

$$\frac{dI_e}{dt}=\sigma I_u(t)-(\gamma_2+\mu+\delta_2)I_e(t) \tag{6}$$

Finally the population of recovered individuals is generated by the recovery of uneducated and educated infected individuals at rates γ_1 and γ_2 respectively.

It is decreased by natural death at a rate μ so that

$$\frac{dR}{dt}=\gamma_1 I_u(t)+\gamma_2 I_e(t)-\mu R(t) \tag{7}$$

Thus the model for the transmission dynamics of an infectious disease in the presence of educated infected individuals is given by the following nonlinear systems of differential equations:

$$\left.\begin{aligned}
\frac{dS}{dt}&=\pi-\left[\mu+\lambda(t)\right]S(t)\\
\frac{dE}{dt}&=\lambda(t)S(t)-\left[\mu+\kappa\right]E(t)\\
\frac{dI_u}{dt}&=\kappa E(t)-\left[\mu+\sigma+\gamma_1+\delta_1\right]I_u(t)\\
\frac{dI_u}{dt}&=\sigma I_u(t)-\left[\mu+\gamma_2+\delta_2\right]I_e(t)\\
\frac{dR}{dt}&=\gamma_1 I_u(t)+\gamma_2 I_e(t)-\mu R(t)
\end{aligned}\right\} \tag{8}$$

2.1. Basic Properties

Since the model (8) monitors human populations, all its associated parameters are nonnegative. Further the following nonnegative result holds.

Theorem 1

The variable of the model (8) are nonnegative for all the time. In other words, solutions of the system (8) with positive initial data will remain positive for all the time $t>0$

Proof. Let $t_1=Sup\{t>0: S>0, E>0, I_u>0, I_e>0, R>0\in[0,t]\}$, thus $t_1>0$

Now it follows from the first equation of the system (8) that

$$\frac{dS}{dt}=\pi-\lambda(t)S(t)-\mu S(t)\geq\pi-(\lambda+\mu)S(t) \tag{9}$$

This can be rewritten as

$$\frac{d}{dt}\left[S(t)\exp\left\{\mu t+\int_0^t\lambda(\tau)d\tau\right\}\right]\geq\pi\exp\left\{\mu t+\int_0^t\lambda(\tau)d\tau\right\} \tag{10}$$

Hence

$$S(t_1)\exp\left\{\mu t_1+\int_0^t\lambda(\tau)d\tau\right\}-S(0)\geq\int_0^{t_1}\pi\exp\left\{\mu y+\int_0^y\lambda(\tau)d\tau\right\}dy \tag{11}$$

So that

$$S(t)\geq S(0)\exp\left\{-\mu t_1-\int_0^{t_1}\lambda(\tau)d\tau\right\}\exp\left\{-\mu t_1-\int_0^t\lambda(\tau)d\tau\right\}\left(\int_0^{t_1}\pi\exp\left\{\mu y+\int_0^y\lambda(\tau)d\tau\right\}dy\right)>0 \tag{12}$$

similarly it can be shown that $E>0, I_u>0, I_e>0$ and $R>0$ for all time $t>0$

2.2. Lemma 1

The closed set

$$\Im=\left\{(S,E,I_u,I_e,R)\in\Re^5{}_+ : S+E+I_u+I_e+R\leq\frac{\pi}{\mu}\right\}$$

Proof. Summing up all the equations of the model (8) gives,

$$\frac{dN}{dt}=\pi-\mu N-(\delta_1 I_u+\delta_2 I_e) \tag{13}$$

Since $\frac{dN}{dt}\leq\pi-\mu N$, it follows that $\frac{dN}{dt}\leq 0$ if $N\geq\frac{\pi}{\mu}$. Thus a standard comparison theorem can be used to show that $N\leq N_0e^{-\mu t}+\left(\frac{\pi}{\mu}\right)\left(1-e^{-\mu t}\right)$. In particular $N(t)\leq\frac{\pi}{\mu}$ if $N(0)\leq\frac{\pi}{\mu}$. Thus the region $\Im$ is positively invariant. Further if $N(0)>\frac{\pi}{\mu}$ then either the solution enter in $\Im$ for infinite time or $N(t)$ approaches $\frac{\pi}{\mu}$ asymptotically. Hence the region $\Im$ attracts all solutions in $\Re^5{}_+$. Since the region $\Im$ is positively invariant, it is sufficient to consider the dynamics of the flow generated by the model (8) in $\Im$.

3. Local Stability of Disease-Free Equilibrium

The disease free equilibrium of the model (8) is given by

$$\varepsilon_0=\left(S^*,E^*,I_u{}^*,I^*{}_e,R^*\right)=\left(\frac{\pi}{\mu},0,0,0,0\right) \tag{14}$$

The local stability of ε_0 will be explored by using the next generation operator method. The nonnegative matrix F of the new infection terms and the M-matrix V of the transition terms associated with the model (8) is given by

$$F=\begin{pmatrix}0 & \frac{\beta\pi}{\mu} & \frac{\eta\beta\pi}{\mu}\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix},V=\begin{pmatrix}\mu+\kappa & 0 & 0\\ -\kappa & \mu+\sigma+\gamma_1+\delta_1 & 0\\ 0 & -\sigma & \mu+\gamma_2+\delta_2\end{pmatrix} \tag{15}$$

respectively.

It follows that the control reproduction number denoted by $\Re_0=\rho\left(FV^{-1}\right)$ where ρ is the spectral radius, is given by

$$\Re_0=\frac{\beta\pi\kappa(k_3+\eta\sigma)}{\mu k_1k_2k_3} \tag{16}$$

Where

$$k_1=\mu+\kappa, k_2=\mu+\sigma+\gamma_1+\delta_1, k_3=\mu+\gamma_2+\delta_2 \tag{17}$$

3.1. Lemma 2

The disease-free equilibrium of the system (8) given by (14) is locally asymptotically stable if $\Re_0<1$

The quantity $\Re_0$ measures the average number of new infections generated by a single infected individual in a population. Lemma 2 implies that the disease can be eliminated from the community $\left(\text{When}\,\Re_0<1\right)$ if the initial sizes of the subpopulations of the model are in the basin of attraction of the disease-free equilibrium $\left(\varepsilon_0\right)$

3.2. Global Stability of Disease-Free Equilibrium

Theorem 2

The disease-free equilibrium of the model (8) given by (14) is globally asymptotically stable in $\Im$ whenever $\Re_0 \le 1$

Proof: Consider the following Lyapunov function:

$$\wp = \left[\frac{\kappa(k_3+\eta\sigma)}{\eta k_1 k_2}\right]E + \left(\frac{k_3+\eta\sigma}{\eta k_2}\right)I_u + I_e = \frac{\kappa(k_3+\eta\sigma)}{\eta k_1 k_2}\left[\beta S\left(\frac{I_u}{1+\alpha_1 I_u}+\frac{\eta I_e}{1+\alpha_2 I_e}\right)-k_1 E\right]$$
$$+\frac{k_3+\eta\sigma}{\eta k_2}\left[\kappa E - k_2 I_u\right]+\sigma I_u - k_3 I_e \le \frac{\kappa(k_3+\eta\sigma)}{\eta k_1 k_2}\left[\frac{\beta\pi}{\mu}\left(I_u+\eta I_e\right)-k_1 E\right]+\frac{k_3+\eta\sigma}{\eta k_2}\left[\kappa E - k_2 I_u\right] \quad (19)$$
$$+\sigma I_u - k_3 I_e = \frac{k_3}{\eta}\left(R_0-1\right)\left(I_u+\eta I_e\right)$$

Since all the parameters and variables of the model (8) are nonnegative, it follows that

$\wp \le 0$ for $\Re_0 \le 1$ with $\wp = 0$ if and only if $E = I_u = I_e = 0$. Hence $\wp$ is a Lyapunov function on $\Im$. Therefore the largest compact invariant subset of the set where $\wp = 0$ is the singleton set $\{(E, I_u, I_e) = (0,0,0)\}$. Thus it follows by the LaSalle's invariance principle that

$$(E, I_u, I_e) \to (0,0,0) \text{ as } t \to \infty \quad (20)$$

Since $\limsup_{t\to\infty} I_u = 0$ and $\limsup_{t\to\infty} I_e = 0$, it follows that for sufficiently small $\omega^* > 0$ there exist constants $M_1.M_2 > 0$ such that $\limsup_{t\to\infty} I_u \le \omega^*$ for all $t > M_1$ and $\limsup_{t\to\infty} I_e \le \omega^*$ for all $t > M_2$. Hence it follows from the last equation of (8) that for $t > Max\{M_1, M_2\}$,

$$R \le \gamma_1\omega^* + \gamma_2\omega^* - \mu R \quad (21)$$

Thus by comparison theorem we have

$$R^{\infty} = \limsup_{t\to\infty} R \le \frac{\gamma_1\omega^* + \gamma_2\omega^*}{\mu} \quad (22)$$

So that by letting

$$\omega^* \to 0, R^{\infty} = \lim_{t\to\infty} SupR \le 0 \quad (23)$$

$$\wp = \left[\frac{\kappa(k_3+\eta\sigma)}{\eta k_1 k_2}\right]E + \left(\frac{k_3+\eta\sigma}{\eta k_2}\right)I_u + I_e \quad (18)$$

With Lyapunov derivative given by

Similarly it can be shown that

$$R_{\infty} = \liminf_{t\to\infty} R \ge 0 \quad (24)$$

Thus it follows from (23) & (24) that

$$R_{\infty} \ge 0 \ge R^{\infty} \quad (25)$$

Hence

$$\lim_{t\to\infty} R = 0 \quad (26)$$

$$\text{similarly } \lim_{t\to\infty} S(t) = \frac{\pi}{\mu} \quad (27)$$

Thus by combining (20), (26) & (27) it follows that every solutions of the equations of the model (8) with initial conditions in $\Im$ approaches to ε_0 as $t \to \infty$ (for $\Re_0 < 1$).

4. Existence and Stability for Endemic Equilibrium Point

In this section the possible existence and stability of endemic equilibrium of the model (8) has been explored. The system (8) is said to be uniformly persistent if there exists a constant c such that any solution of $(S(t), E(t), I_u(t), I_e(t), R(t))$ satisfies

$$\liminf_{t\to\infty} S(t) \ge c, \liminf_{t\to\infty} E(t) \ge c, \liminf_{t\to\infty} I_u(t) \ge c, \liminf_{t\to\infty} I_e(t) \ge c, \liminf_{t\to\infty} R(t) \ge c \quad (28)$$

provided $(S(0), E(0), I_u(0), I_e(0), R(0)) \in \Im$

Theorem 3. System (8) is uniformly persistent in $\Im$ if and only if $\Re_0 > 1$

Proof: The theorem can be proved by applying a uniform persistent result in [15] and noting the fact that the disease-free equilibrium of the model (8) is unstable whenever $\Re_0 > 1$. When $\Re_0 > 1$ it follows from lemma 2 that model (8) is uniformly persistent.

5. Conclusions

In this paper global stability of two strain epidemic Model have been studied with two general incidence functions. Existence, boundedness and positivity of solutions have been studied The disease free equilibrium, endemic equilibrium

with respect to strain 1, endemic equilibrium with respect to strain 2 and endemic equilibrium with respect to both the strains are calculated.

Findings of this research are as follows:

i). The model (8) has a locally stable disease-free equilibrium whenever the associated reproduction number is less than unity.

ii). The disease-free equilibrium of the model (8) is shown to be globally asymptotically stable when $\Re_0 \leq 1$

iii). The model (8) is uniformly persistent in $\Im$ if and only if $\Re_0 > 1$

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