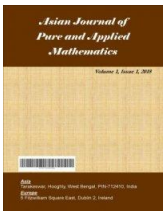

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Mathematical Transmission of SEIR Epidemic Model with Natural Immunity

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Original Research Article

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Abstract

The SEIR mathematical and epidemiological model with natural immunity and treatment rate are explored in this paper. Both local and global stability were analyzed for disease-free equilibrium point. The threshold quantity "Basic Reproduction Number" (R_0) with natural immunity was derived using next generation matrix method (NGM), and it is shown that the disease free equilibrium point is locally and globally asymptotically stable whenever the basic reproduction number is less than unity i.e. ($R_0 < 1$), while endemic whenever ($R_0 > 1$). Numerical simulations show that, strong natural immunity reduces the dynamical spread of epidemic diseases.

Keywords: Epidemic; basic reproduction number; stability; natural immunity; treatment.

1 Introduction

Mathematical epidemiology is the simplest form of describing a communicable disease within susceptible individuals [1]. The modeling of infectious diseases is a powerful tool which has been used to study the dynamical spread of diseases, to predict the future course of an outbreak and to evaluate best control strategies in an epidemic disease(s) [2,3]. It is known that the spread of many infectious diseases can be prevented by the natural immunity of susceptible individuals; some types of infections provide recovering individuals with short or long immunity against re-infection. It means that it is normal to include immunological effects in mathematical models for better representation of dynamics of epidemic spread and prediction of future epidemics [4]. Immunity can be obtained through targeted immunization; It can be acquired naturally after an individual has successfully recovered from the infection, and in some cases the

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mother's antibodies can be passed on to the newborn to provide a certain level of immunity. In each case, the duration of immunity will be different, with some diseases providing almost lifelong immunity while others only give a very short state of insensitivity. Often, the immunity induced by the vaccine requires a boost after a certain period of time, such as the effectiveness of the vaccine diminishes due to lack of exposure to disease. In the case of measles, for example, vaccinated individuals have less immunity from those who have natural immunity [5,6]. The first physician to investigate the dynamical spread of disease using mathematical modeling was Daniel Bernoulli. He formulated a mathematical model to gain insight into the practice of inoculating against smallpox [7,8]. The results from his research showed that universal inoculation against smallpox would increase the life expectancy from 26 years 7 months to 29 years 9 months [8]. The SIR model, first published by Kermack and Mackendrick in 1927, provides and established basics to model the global stability of a class of SIR epidemic models [9]. The SIR model is undoubtedly the most famous mathematical model for the spread of an infectious disease. Here people are characterized into three (3) classes: Susceptible S, Infective I, and Removed R. (Recovered). Removed individuals are no longer susceptible nor infective for whatever reasons; for example, they have recovered been from the diseased and non immune, or they have been vaccinated, or they have been isolated from the rest of the population; or perhaps they have died from the disease [10].

Several scholars had extensively analyzed the dynamical spread of the SIR, SIRS, SEIR and SVEIR epidemic model.

Cooke [11] formulated an SIR epidemic model with bilinear incidence rate and a discrete time delay which takes the form of BSI to investigate the spread of an infectious disease transmitted by a vector e.g. Mosquitoes, rats, flies etc after an incubation time denoting the time during which the infectious agents develop in the vector. This is called the phenomena of time delay effect which now has important biological meanings in epidemic model.

Ma et al. [12] considered a continuous SIR model with a discrete delay and investigated the global asymptotic stability of the equilibrium, while a continuous SIR model with distributed delays also studied in Ma et al. The distributed delays is more appropriate form than the discrete one because it is considered more realistic to assume that the time delay is not a fixed time but distributed parameter which is upper bounded by some positive finite time.

Beretta et al. [13] studied the class of discrete SIR epidemic models which are derived from SIR epidemic models with distributed delay, applying a Lyapunov functional technique. It is shown that the global dynamics of each discrete SIR epidemic model are fully determined by a single threshold parameter and the effect of discrete time delays are harmless for the global stability of the endemic equilibrium of the model.

This study is more realistic because it includes natural immunity that was not given much importance before, hence, the aim of this paper is to propose an epidemic model, to gain insight into natural immunity needed to ensure the disease eradication from the population.

2 Formulation of the Model

In this research, SEIR Epidemic model is constructed and the total population N(t) is sub divided into four epidemiological classes; Susceptible (S), Exposed (E), Infected Infectious (I) and Recovered (R), i.e. The total population is

$$N(t) = S(t) + E(t) + I(t) + R(t)$$

An epidemiological model is presented and analyzed to gain understanding on the importance of natural immunity in the dynamical spread of SEIR epidemic model.

In summary, the dynamics transmission model is given by the following system of non-linear differential equations below;

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \beta SI - \mu S + \theta R + \kappa E \\
\frac{dE}{dt} &= (1-\varepsilon)\beta SI - K_1 E \\
\frac{dI}{dt} &= \varepsilon\beta SI + \sigma E - K_2 I \\
\frac{dR}{dt} &= \omega I - K_3 R
\end{aligned}\right\} \tag{1}$$

Where

$$K_1 = (\mu + \sigma + \kappa)$$
$$K_2 = (\mu + \delta + \omega)$$
$$K_3 = (\mu + \theta)$$

Where π is the recruitment rate either by birth or immigration into the population, β is the effective contact rate, μ is the natural death rate , θ is the recovery rate, κ is the natural immunity, ε is the active infection, σ is the progression rate from exposed to infected individuals, δ is the disease induced death rate and ω is the treatment rate of infected individuals.

Table 1. Description of variables

Variables	Definitions
S	Susceptible individuals
E	Exposed individual
I	Infected individual
R	Recovered individual

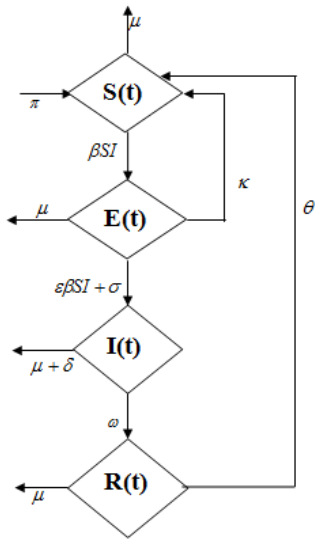


Chart 1. Flow chart

2.1 Positivity of solutions

For the model to be mathematically and epidemiologically meaningful and well posed, we need to prove that all state variables are non-negative for all $t > 0$

$$D = \{(S + E + I + R) \in R^4_+ : N \leq \pi / \mu \tag{2}$$

Lemma 1. The closed set $D = \{(S + E + I + R) \in R^4_+ : N \leq \pi / \mu$ is positively-invariant and attracting with respect to the model (1).

Proof: Consider the biologically-feasible region D, defined above. The rate of change of the total population, obtained by adding all equations of the model (1), is given by

$$\frac{dN}{dt} = \pi - \mu N - \delta \tag{3}$$

It follows that $\frac{dN}{dt} < 0$ whenever $N > \frac{\pi}{\mu}$.

Furthermore,

Since $\frac{dN}{dt} \leq \pi - \mu N$,

it is clear that $N(t) \leq \frac{\pi}{\mu}$ if $N(0) \leq \frac{\pi}{\mu}$.

Therefore, all solutions of the model with initial conditions in D is positively-invariant and attracting. In this region, the model can be considered as been epidemiologically and mathematically well posed.

2.1.1 Existence of disease free and endemic equilibriums

For critical points, we set;

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

At disease free equilibrium, we assumed there is no infection in the population. Hence, the system (1) has a disease free

$$\varepsilon_0 = (S, E, I, R) = \left(\frac{\pi}{\mu}, 0, 0, 0\right) \tag{4}$$

2.1.2 Endemic equilibrium

The endemic equilibrium of the model (1) is given below;

$$\left.\begin{aligned} S^* &= \frac{\pi + \theta R^* + \kappa E^*}{\mu + \beta I^*} \\ E^* &= \frac{(1-\varepsilon)\beta S^* I^*}{K_1} \\ I^* &= \frac{\sigma E^*}{K_2 - \varepsilon\beta S^*} \\ R^* &= \frac{\omega I^*}{K_3} \end{aligned}\right\} \tag{5}$$

2.1.3 Basic reproduction number (R_O)

Let $X = (R, I, E, S)^T$ System (1) can be written as;

$$\frac{dx}{dt} = F(x) - \upsilon(x)$$

Where;

$$F(x) = \begin{pmatrix} -\beta SI \\ (1-\varepsilon)\beta SI \\ \varepsilon\beta SI \\ 0 \end{pmatrix} \tag{6}$$

$$\upsilon(x) = \begin{pmatrix} \mu S - \theta R - \kappa E \\ K_1 E \\ -\sigma E + K_2 I \\ -\omega I + K_3 \end{pmatrix} \tag{7}$$

After taking partial derivatives of F and V, we have

$$F = \begin{pmatrix} 0 & (1-\varepsilon)\dfrac{\beta\pi}{\mu} \\ 0 & \varepsilon\dfrac{\beta\pi}{\mu} \end{pmatrix}$$

$$V = \begin{pmatrix} K_1 & 0 \\ -\sigma & K_2 \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} (1-\varepsilon)\dfrac{\beta\pi\sigma}{\mu K_1 K_2} & (1-\varepsilon)\dfrac{\beta\pi}{\mu K_2} \\ \varepsilon\dfrac{\beta\pi\sigma}{\mu K_1 K_2} & \varepsilon\dfrac{\beta\pi}{\mu K_2} \end{pmatrix}$$ The next generation matrix of system (1) is the spectral radius of FV^{-1} which is;

$$R_0 = \frac{\beta\pi(\sigma + \varepsilon K_1 - \sigma\varepsilon)}{\mu K_1 K_2} \tag{8}$$

The threshold quantity R_0 is the basic reproduction number of the normalized model system (1). It is the average number of new secondary infections generated by a single infected infectious individual in his or her infectious period [14].

2.2 Local stability of disease free equilibrium

Theorem 1: If $R_0 < 1$, then, the disease free equilibrium is locally asymptotically stable and unstable if $R_0 > 1$

Proof: The Jacobian matrix $J(P_0)$ of the model equation (1) evaluated at disease free equilibrium is given by;

$$J(P_o) = \begin{bmatrix} -\mu & \kappa & \dfrac{-\beta\pi}{\mu} & \theta \\ 0 & -K_1 & (1-\varepsilon)\dfrac{\beta\pi}{\mu} & 0 \\ 0 & \sigma & \varepsilon\dfrac{\beta\pi}{\mu} - K_2 & 0 \\ 0 & 0 & \omega & -K_3 \end{bmatrix} \tag{9}$$

The eigenvalues of $J(P_0)$ are $\lambda_1 = -\mu, \lambda_2 = -K_3$ and the remaining sub-matrix is;

$$J_1(P_o) = \begin{bmatrix} -K_1 & (1-\varepsilon)\dfrac{\beta\pi}{\mu} \\ \sigma & \varepsilon\dfrac{\beta\pi}{\mu} - K_2 \end{bmatrix} \tag{10}$$

The characteristics polynomial of equation (10) above is;

$$A_2\lambda^2 + A_1\lambda + A_0 = 0$$

Where;

$$A_2 = 1$$

$$A_1 = K_1 + K_2$$

$$A_0 = \mu K_1 K_2 - \beta\pi(\sigma + \varepsilon K_1 - \sigma\varepsilon)$$

From A_0

$$\mu K_1 K_2 - \beta\pi(\sigma + \varepsilon K_1 - \sigma\varepsilon) > 0$$

$$-\beta\pi(\sigma + \varepsilon K_1 - \sigma\varepsilon) > -\mu K_1 K_2 \tag{11}$$

Divide both sides by the RHS of (11), gives;

$$\frac{\beta\pi(\sigma + \varepsilon K_1 - \sigma\varepsilon)}{\mu K_1 K_2} < 1$$

Hence $R_0 < 1$

It can be seen clearly from the above that $A_2 > 0$, $A_1 > 0$ and that $A_0 > 0$ if $R_0 < 1$, From the above, all the eigen-values of the Jacobian matrix $J(P_O)$ are real and negative when $R_o < 1$, therefore the disease free equilibrium is locally asymptotically stable [15].

2.3 Global stability of disease free equilibrium

Theorem 1: The DFE of the model (1), is Global Asymptotically Stable (GAS) if $R_0 < 1$.

Proof:

$$\begin{pmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dR}{dt} \end{pmatrix} = (G_1 - G_2 - G_3) \begin{pmatrix} E \\ I \\ R \end{pmatrix} \tag{12}$$

Where the matrices G_1, G_2 and G_3 are given by;

$$G_1 = \begin{pmatrix} \frac{\beta\pi}{\mu} & \frac{\beta\pi}{\mu} & 0 \\ \frac{\beta\pi}{\mu} & \frac{\beta\pi}{\mu} & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{13}$$

$$G_2 = \begin{pmatrix} K_1 & 0 & 0 \\ -\sigma & K_2 & 0 \\ 0 & -\omega & K_3 \end{pmatrix} \tag{14}$$

And

$$G_3 = \begin{pmatrix} \lambda & \lambda & \lambda \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{15}$$

Since matrix G_3 is non-negative, thus;

$$\begin{pmatrix} \frac{dE}{dt} \\ \frac{dI}{dt} \\ \frac{dR}{dt} \end{pmatrix} \leq (G_1 - G_2) \begin{pmatrix} E \\ I \\ R \end{pmatrix} \tag{16}$$

If $R_0 < 1$ then $\ell(G_1 G_2^{-1}) < 1$ (from the local stability result given in theorem 1), which is equivalent to $G_1 - G_2$ having all its eigenvalues in the left-half plane [16]. It follows that the linearized differential system (1) is stable whenever $R_0 < 1$. Consequently, by comparison theorem [16], it follows that $(E(t), I(t), R(t) \to (0,0,0)$. Hence, since D is positively-invariant, it follows that DFE is GAS in $D^* \; if \;\; R_0 < 1$.

3 Numerical Simulation

To verify the dynamical behavior of the SEIR epidemic model, the numerical simulations is analyzed by MAPLE 18 software using Runge-Kutta method of order four (4).

4 Discussion and Conclusions

Four (4) compartmental mathematical and epidemiological models were presented to gain insight on importance of natural immunity on the dynamical spread of SEIR epidemic models. The positivity of solution shows that the solution exists and moreover, the system of equation (1) is mathematically and epidemiologically well posed. The threshold quantity "Basic reproduction number" 'R_0' which measures the average number of secondary infection generated by infected infectious individuals in his/her infectious period in the population of susceptible was analyzed using method of next generation matrix, it shows that, the system is stable and the disease dies out whenever the threshold $R_0 < 1$ but unstable (spreads) when $R_0 > 1$. The local stability of disease free equilibrium also confirmed that, disease would die off since $R_0 < 1$.

Numerical simulations of the system (1) show the dynamical behavior of natural immunity most especially when it is high. Fig. 1 shows highly pronounced infected individuals due to low / weak natural immunity, Fig. 2 shows that infected individuals reduced to less than 5500 infected individuals when natural immunity $\kappa = 0.5$, likewise in Fig. 3, infected individuals reduced to 3000 when $\kappa = 0.7$. Fig. 4 shows that, natural immunity plays a vital role in the dynamical control of SEIR epidemic diseases most especially when it is high / strong. Infected individuals reduced to zero when natural immunity rate $\kappa = 1$.

Conclusively, Natural immunity should be targeted by medical practitioners and policy health makers as best control strategy against SEIR epidemic disease(s).

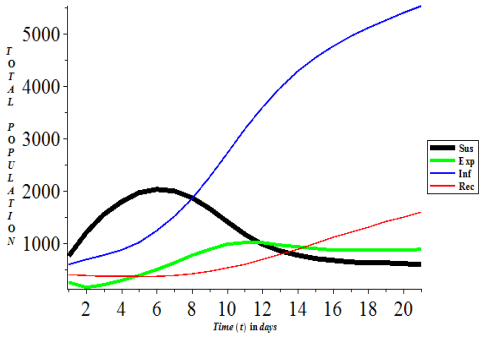


Fig. 1. $\pi = 500,\ \omega = 0.05,\ \sigma = 0.522,\ \beta = 0.5,\ \delta = 0.02, \theta = 0.1,\ \mu = 0.02,\ \varepsilon = 0.2,\ \kappa = 0.2$

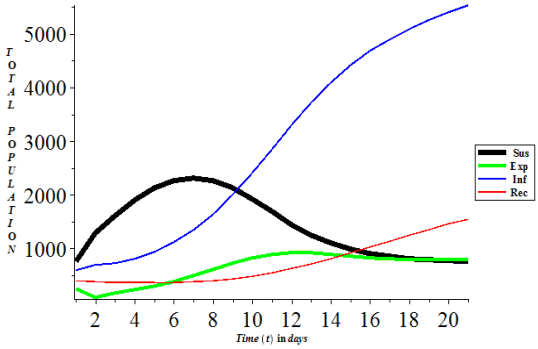
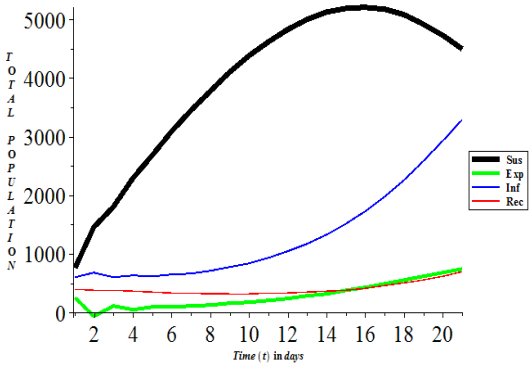


Fig. 2. $\pi = 500,\ \omega = 0.05,\ \sigma = 0.522,\ \beta = 0.5,\ \delta = 0.02, \theta = 0.1,\ \mu = 0.02,\ \varepsilon = 0.2,\ \kappa = 0.5$



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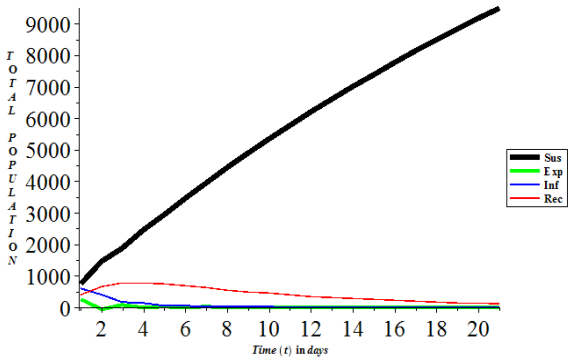

Fig. 4. $\pi = 500,\ \omega = 0.05,\ \sigma = 0.522,\ \beta = 0.5,\ \delta = 0.02, \theta = 0.1,\ \mu = 0.02,\ \varepsilon = 0.2,\ \kappa = 1$

Competing Interests

Authors have declared that no competing interests exist.

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