Journal of the Nigerian Mathematical Society
Vol. 38, Issue 1, pp. 45-54, 2019

A GENERALIZED SEIR MATHEMATICAL MODEL WITH INFECTIVITY IN EXPOSED PERIOD

A. O. SANGOTOLA [1] AND A. A. ONIFADE

ABSTRACT. In this paper, an SEIR model is presented with infectivity in exposed period. Positivity and the boundedness of solutions are established. We also determined the conditions of existence and stability for the disease-free and endemic equilibria. A threshold parameter R_0 exists and the disease can persist if and only if R_0 exceeds 1. Local and global stabilities of the disease free and endemic equilibria were also determined using suitable Lyapunov function. Numerical simulation of the model is also carried out to illustrate the dynamics of the model.

Keywords and phrases: Mathematical model; stability; basic reproduction number; Lyapunov function.

2010 Mathematical Subject Classification: 92B05

1. INTRODUCTION

An infectious disease is a clinically evident illness resulting from the presence of a pathogenic microbial agent. The microbial agent causing the disease can be bacterial, viral, parasitic or fungal, posing a constant threat to human wellness[1]. Every individual on the earth can be affected by a disease and that is why understanding the dynamics of infectious disease is very important since emergence and reemergence of infectious diseases have become a significant worldwide problem. The history of mathematical models in epidemiology dates back to the eighteenth century (Bernoulli 1760). Thereafter, theoretical epidemiology has witnessed numerous developments [3]. The popular epidemic dynamic models constructed by Kermack and Mckendrick in 1927 is being developed by many other bio mathematicians [4]. In epidemiological compartment models of infectious diseases, transmission of infectious agents in the host population is the fundamental process to be described. When a

Received by the editors August 27, 2018; Revised December 05, 2018 ; Accepted: December 23, 2018

www.nigerianmathematicalsociety.org; Journal available online at https://ojs.ictp.it/jnms/

[1]Corresponding author

pathogen appears in a host community, it partitions individuals in the community into categories depending on parasite density inside them and the type of infection. These categories or compartments are represented by standard notation of SEIR after the pioneering work of Kermack and McKendrik. In a simple form they are as follows: the first group consists of the fraction of host population that is Susceptible (S) to infection; then comes the exposed (E) class, the fraction of population whose individuals are infected by the pathogen, but not capable of passing on the infection to others during a latent period. The next is I class or infectious individuals, who give rise to more infected individuals through interaction with the susceptibles. Finally, those individuals who recover from the infection make up the R class [5]. The remaining sessions of this work entails the dynamics and mathematical analysis for the possibility of infectivity in the exposed stage.

2. MODEL FORMULATION

The model divides the total human population into susceptible humans $S(t)$, exposed humans $E(t)$, infectious humans $I(t)$ and recovered humans $R(t)$. Thus we have $N(t) = S(t)+E(t)+I(t)+R(t)$. The dynamics of the model is such that susceptible individual are recruited into the human population by input rate Λ. Every class of human population is decreased by natural death μ except for the infectious class which has a per capita disease induced death rate δ. After treatment, the exposed and infectious humans recover and move to recovered class. However, the recovered humans develops a temporary acquired immunity against the disease and later loses this immunity to become susceptible again at per capita rate ω. The model has the form

$$\frac{dS}{dt} = \Lambda - \beta S(I+E) - \mu S + \omega R \tag{1}$$

$$\frac{dE}{dt} = \beta S(I+E) - (\mu+\sigma+\gamma)E \tag{2}$$

$$\frac{dI}{dt} = \sigma E - (\delta+\mu+\theta)I \tag{3}$$

$$\frac{dR}{dt} = \theta I + \gamma E - (\mu+\omega)R \tag{4}$$

The parameters used in the model (1) - (4) are described in Table 1.

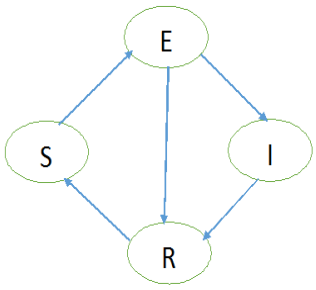

Fig.1. Flow diagram of the model

Table 1. The description of the state variables and parameters of model.

Definition	Symbols
Recruitment term of the susceptible humans	Λ
Progression rate of exposed human to infectious humans	σ
Effective contact rate	β
Effective treatment rate of exposed human	γ
Effective treatment of infectious humans	θ
Disease induced death rate of humans	δ
Per capita transition rate of recovered humans	ω
Natural death rate of humans	μ

3. RESULTS AND DISCUSSIONS

Theorem 1: (Existence and positivity of solution). The feasible region $\mathcal{R}$ defined by $\{S(t), E(t), I(t), R(t) \in \mathbb{R}^4_+ : N(0) \leq N(t) \leq \frac{\Lambda}{\mu}\}$ with initial conditions $S(0) \geq 0, E(0) \geq 0, I0) \geq 0, R(0) \geq 0$ is positive invariant for system $(1) - (4)$.

Proof: If the total population size is given by $N(t) = S(t) + E(t) + I(t) + R(t)$.It is clear from $(1) - (4)$ that

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I \tag{5}$$

$$\frac{dN}{dt} \leq \Lambda - \mu N \tag{6}$$

$$N \leq C\Lambda e^{-\mu t} + \frac{\Lambda}{\mu} \tag{7}$$

As $t \to \infty$,we have

$$N \leq \frac{\Lambda}{\mu} \tag{8}$$

Thus $\mathcal{R}$ is a positivity invariant set under the model described by $(1) - (4)$. Hence it is sufficient to consider the dynamics of model $(1) - (4)$ in region $\mathcal{R}$.

Disease-free equilibrium points is a steady-state solution where there is no infection. Solving the system of equation $(1) - (4)$ in the absence of disease, we obtain the following equilibrium point,

$$\pi_0 = (\frac{\Lambda}{\mu}, 0, 0, 0) \tag{9}$$

The differential of the disease states and transfer states evaluated at the disease free equilibrium $\pi_0 = (\frac{\Lambda}{\mu}, 0, 0, 0)$ respectively give:

$$\mathbf{F} = \begin{pmatrix} \frac{\beta\Lambda}{\mu} & \frac{\beta\Lambda}{\mu} \\ 0 & 0 \end{pmatrix}$$

$$\mathbf{V} = \begin{pmatrix} (\mu + \sigma + \gamma) & 0 \\ -\sigma & (\delta + \mu + \theta) \end{pmatrix}$$

The basic reproduction R_0 is given by $\rho(FV^{-1})$ where ρ is the spectral radius. Thus,

$$R_0 = \frac{\Lambda\beta[(\delta + \mu + \theta + \sigma]}{\mu(\mu + \sigma + \gamma)(\delta + \mu + \theta)} \tag{10}$$

Theorem 2: (Local stability of disease free equilibrium). The disease-free equilibrium for the system $(1) - (4)$ is locally asymptotically stable if $a > 0$ and $R_0 < 1$.

Proof: The Jacobian matrix evaluated at the disease free is given by

$$J(\pi_0) = \begin{pmatrix} -\mu & -\frac{\beta\Lambda}{\mu} & -\frac{\beta\Lambda}{\mu} & \omega \\ 0 & \frac{\beta\Lambda}{\mu} - (\mu + \sigma + \gamma) & \frac{\beta\Lambda}{\mu} & 0 \\ 0 & \sigma & -(\delta + \mu + \theta) & 0 \\ 0 & \gamma & \theta & -(\mu + \omega) \end{pmatrix}$$

The roots of the characteristic equation are $-\mu$ and $-(\mu+\omega)$. The others roots can be obtained from the sub matrix given below.

$$\begin{pmatrix} \frac{\beta\Lambda}{\mu} - (\mu + \sigma + \gamma) & \frac{\beta\Lambda}{\mu} \\ \sigma & -(\delta + \mu + \theta) \end{pmatrix}$$

The characteristic equation of the matrix above is $\lambda^2 + a\lambda + b = 0$ where $a = (2\mu+\sigma+\gamma+\delta+\theta)$ and $b = (\mu+\sigma+\gamma)(\delta+\mu+\theta)(1-R_0)$ According to Routh-Hurwitz criterion, The disease free equilibrium is locally asymptotically stable if $a > 0$ and $R_0 < 1$.

Theorem 3: (Local stability of endemic equilibrium). The model $(1) - (4)$ has no endemic equilibrium when $R_0 < 1, R_e > 1$ and a unique endemic equilibrium exist when $R_0 > 1, R_e < 1$.

Proof: Let $E_e^* = (E^*, I^*, R^*)$ be a non trivial equilibrium of the model $(1) - (4)$ i.e all component of E_e^* are zero. Then the model $(1) - (4)$ at steady state becomes

$$\begin{aligned} E^* &= \frac{BI^*}{\sigma} \\ R^* &= \frac{(\theta + B\gamma)I^*}{\sigma} \\ I^* &= \frac{\sigma\mu(R_0 - 1)}{\beta(\sigma + B)(1 - R_e)} \end{aligned}$$

where

$$A = (\mu + \sigma + \gamma), B = (\delta + \mu + \theta), R_e = \frac{\omega(\theta + B\gamma)}{AB}$$

Theorem 4: (Global stability of disease free equilibrium). The disease-free equilibrium, π_0, of the model $(1) - (4)$, is globally asymptotically stable if $R_0 \leq 1$

Proof: Consider the following linear Lyapunov function

$$V = (\delta + \mu + \theta) + \sigma)E + (\mu + \sigma + \gamma)I \tag{11}$$

$$\dot{V} = (\delta + \mu + \theta) + \sigma)[\beta S(I + E) - (\mu + \sigma + \gamma)E] + (\mu + \sigma + \gamma)$$
$$X[\sigma E - (\delta + \mu + \theta)I] \tag{12}$$

Simplifying gives

$$\dot{V} \leq (\mu + \sigma + \gamma)(\delta + \mu + \theta)I[R_0 - 1] \tag{13}$$

Thus $\dot{V} \leq 0$ for $R_0 < 1$ with equality if and only if $I = 0$

Theorem 5: (Global stability of endemic equilibrium). The unique endemic equilibrium, E_e, of the model $(1) - (4)$ is globally asymptotically stable if $R_0 > 1$.

Proof: Let $R_0 > 1$ so that a unique endemic equilibrium exists and consider the following nonlinear Lyapunov function defined by

$$L = S - S^{**} - S^{**}\ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**}\ln\left(\frac{E}{E^{**}}\right) + \frac{(\mu+\sigma+\gamma)}{\sigma}\left[I - I^{**} - I^{**}\ln\left(\frac{I}{I^{**}}\right)\right] \tag{14}$$

$$\dot{L} = \dot{S} - \frac{S^{**}}{S}\dot{S} + \dot{E} - \frac{E^{**}}{E}\dot{E} + \frac{(\mu+\sigma+\gamma)}{\sigma}\left[\dot{I} - \frac{I^{**}}{I}\dot{I}\right] \tag{15}$$

$$\begin{aligned}\dot{L} = \Lambda - \beta S(I+E) - \mu S - \frac{S^{**}}{S}(\Lambda - \beta S(I+E) - \mu S) \\ + \beta S(I+E) - (\mu+\sigma+\gamma)E - \frac{E^{**}}{E}[\beta S(I+E) \\ - (\mu+\sigma+\gamma)E] + \frac{(\mu+\sigma+\gamma)}{\sigma}[\sigma E - (\delta+\mu+\theta)I \\ - \frac{I^{**}}{I}[\sigma E - (\delta+\mu+\theta)I]]\end{aligned} \tag{16}$$

$$\begin{aligned}\dot{L} = \Lambda\left(1 - \frac{S^{**}}{S}\right) - \mu S\left(1 - \frac{S^{**}}{S}\right) + \beta S^{**}(I+E) \\ - \frac{\beta S(I+E)E^{**}}{E} + (\mu+\sigma+\gamma)E^{**} - \frac{(\mu+\sigma+\gamma)(\delta+\mu+\theta)I}{\sigma} \\ - \frac{(\mu+\sigma+\gamma)EI^{**}}{I} + \frac{(\mu+\sigma+\gamma)(\delta+\mu+\theta)I^{**}}{\sigma}\end{aligned} \tag{17}$$

At the endemic equilibrium, it is seen from $(1)-(4)$ that

$$\left.\begin{array}{l}\Lambda = \beta S^{**}(I^{**}+E^{**}) + \mu S^{**} \\ (\mu+\sigma+\gamma) = \frac{\beta S^{**}(I^{**}+E^{**})}{E^{**}} \\ (\delta+\mu+\theta) = \frac{\sigma E^{**}}{I^{**}}\end{array}\right\} \tag{18}$$

Substituting (18) into (17) gives

$$\begin{aligned}\dot{L} = [\beta S^{**}(I^{**}+E^{**}) + \mu S^{**}]\left(1 - \frac{S^{**}}{S}\right) - \mu S\left(1 - \frac{S^{**}}{S}\right) \\ + \beta S^{**}(I+E) - \frac{\beta S(I+E)E^{**}}{E} + \beta S^{**}(I^{**}+E^{**}) \\ - \frac{\beta S^{**}(I^{**}+E^{**})I}{I^{**}} - \frac{\beta S^{**}(I^{**}+E^{**})EI^{**}}{E^{**}I} + \beta S^{**}(I^{**}+E^{**})\end{aligned} \tag{19}$$

Further algebraic simplification gives

$$\dot{L} = \mu S^{**}\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}}\right)$$
$$+\beta S^{**}(I^{**}+E^{**})\left(4 - \frac{S^{**}}{S} - \frac{EI^{**}}{E^{**}I} - \frac{I(I^{**}+E^{**})}{I^{**}(I+E)} - \frac{SE^{**}(I+E)}{S^{**}E(I^{**}+E^{**})}\right)$$
$$+\beta S^{**}(I^{**}+E^{**})\left(\frac{I+E}{I^{**}+E^{**}} - \frac{I}{I^{**}} + \frac{(I^{**}+E^{**})I}{(I^{+}E)I^{**}} - 1\right) \quad (20)$$

Since the arithmetic mean exceed the geometric mean, we have that $\dot{L} \leq 0$ for $R_0 > 1$. Hence it follows from LaSalle's invariant principle that every solution of the equation of model $(1) - (4)$ approaches unique endemic equlibria of the model as $t \to \infty$ for $R_0 > 1$.

4. NUMERICAL SIMULATION

In this section, we discus the simulation results for dynamics of the model $(1) - (4)$. The model (1) - (4) is simulated using the parameters in Table 1 to illustrate some of the theoretical results established in this study and by considering initial conditions $S(0)$ =100, $E(0) = 20$, $I(0) = 5$, $R(0) = 0$. The following values for the parameters are used: $\Lambda = 0.25, \sigma = 0.08, \beta = 0.12, \gamma = 0.011, \theta = 0.06, \delta = 0.016, \omega = 0.143$ and $\mu = 0.00005$. The numerical simulations are conducted using Maple 17 software and the results are given in Figure 2, 3 ,4 and 5 to illustrate the system's behaviour for different values of the model's parameters. In Figure 2, it is observed that the susceptible population drops significantly as a result of interaction with exposed and infectious class which leads to an initial sharp increase in the exposed class before a steady decline as seen in figure 3. The exposed class population decreases when they progress to infected class or when they moves to recovered class. The infectious class also increases as exposed class progresses to this class as shown in figure 4 before expressing decline due to treatment and death. The decrease in the number of infectious human population contribute to the increase in the number of recovered human population as shown in figure 5.

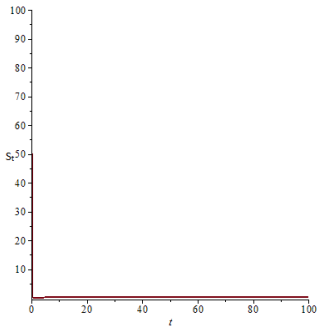

Fig.2. Numerical simulation showing the dynamics of susceptible population

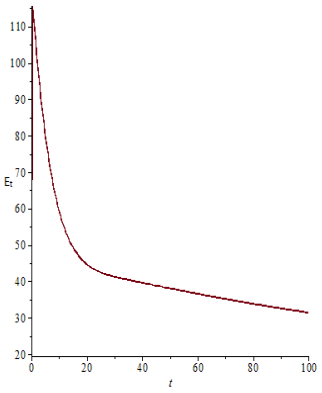

Fig.3. Numerical simulation showing the dynamics of exposed population

5. CONCLUDING REMARKS

In this paper, the basic reproduction number of a generalized SEIR model with infectivity in exposed period is examined. We also investigated the local and global stability of the disease free equilibrium and the endemic equilibrium. The numerical simulation helps to show how the disease progress from one stage to the other.

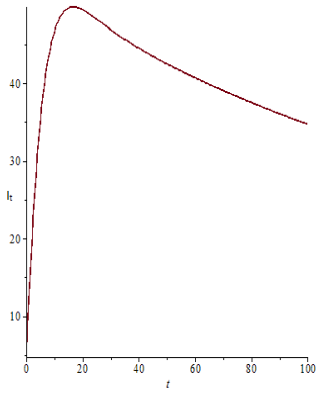

Fig.4. Numerical simulation showing the dynamics of infectious population

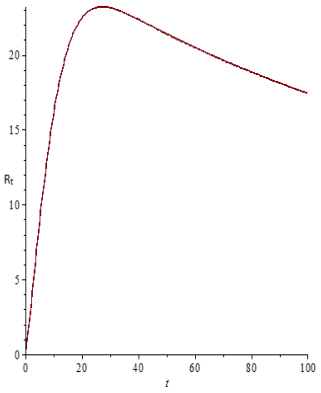

Fig.5. Numerical simulation showing the dynamics of recovered population

References

[1] Maia Martcheva, *An introduction to mathematical epidemiology*, Springer New York, 2015.

[2] M. A. Yau, H.S. Ndakwo and A. M. Umar, *A Global Stability Analysis of a Susceptible-Infected-Removed-Prevented- Controlled Epidemic Model* Applied Mathematics. **5** (1) 1393-1399, 2014.

[3] S. A. Al-Sheikh, *Modeling and Analysis of an SEIR Epidemic Model with a Limited Resource for Treatment* Global Journal of Science Frontier Research. **12** (16), 2012.

[4] Zhien Ma, Yincang Zhou, and Jianhong Wu, *Modelling and dynamics of infected disease*, World Scientific Publishing Co Pte Ltd 5 Toh Tuch Link, Singapore 596224, 2009.
[5] Sandip Mandal, Ram Rup Sarkar and Somdatta Sinha, *Mathematical models of malaria - a review* Malaria Journal. **10** 1-19, 2011.
[6] P.V.Driessche and J. Watmough, *Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission* Mathematical Biosciences. (180) 29-48, 2002.
[7] M.M. Ojo and F.O. Akinpelu, *Lyapunov Functions and Global Properties of SEIR Epidemic Model* International journal of Chemistry, Mathematics and Physics (IJCMP).**1** 11-16, 2017.
[8] Fred Brauer and Carlos Castillo-Chavez, *Mathematical Models in Population Biology and Epidemiology*, Springer New York, 2010.
[9] Fred Brauer and Carlos Castillo-Chavez, *Mathematical Models for Communicable Diseases*, Society for Industrial and Applied Mathematics Philadelphia, 2013.

DEPARTMENT OF PHYSICAL SCIENCES, BELLS UNIVERSITY OF TECHNOLOGY, OTA, NIGERIA
E-mail address: adekunle4000@hotmail.co.uk

DEPARTMENT OF MATHEMATICS, UNIVERSITY OF IBADAN, IBADAN, NIGERIA
E-mail addresses: akiin4all80@gmail.com