

# Lyapunov Functions and Global Properties of SEIR Epidemic Model

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**Abstract**— *The aim of this paper is to analyze an SEIR epidemic model in which prophylactic for the exposed individuals is included. We are interested in finding the basic reproductive number of the model which determines whether the disease dies out or persist in the population. The global attractivity of the disease-free periodic solution is obtained when the basic reproductive number is less than unity and the disease persist in the population whenever the basic reproductive number is greater than unity, i.e. the epidemic will turn out to endemic. The linear and non-linear Lyapunov function of Goh–Volterra type was used to establish the sufficient condition for the global stability of the model.*

**Keywords**—*Epidemic Model; Lyapunov function; Global stability; Basic Reproduction number.*

## I. INTRODUCTION

Infectious diseases have tremendous influence on human life, and millions of people died of various infectious diseases. Controlling infectious diseases has been an increasingly complex issue in recent year Driessche [21]. It has been revealed that many infectious diseases in nature incubate inside the hosts for a period of time before the hosts become infectious.

In a lot of epidemic models Diekmann [5]; Liu [16]; Anderson [3]; Lu [17]; Kermack [10], the total population was divided into three groups: the susceptible, the infectives and the removed individuals. Since Kermack and McKendrick constructed a system of ODE to study epidemiology in 1927, the method of compartment modelling is used until now Driessche [21]. But using the compartmental approach, an assumption that a susceptible

individual first goes through an incubating period (in class E) after infection before becoming infectious can be made.

Adebimpe [1] considered a SIR epidemic model with saturated incidence rate and treatment. Liu [15] discussed the dynamical behavior of an epidemiological model with non-linear incidence rates; also Greenhalgh [2] considered SEIR models that incorporate density dependence in the death rate. Cooke [4] introduced and studied the SEIRS models with delays. Greenhalgh [5] studied Hopf bifurcations in models of the SEIRS type with density dependent contact rate and death rate. Li [12] analyzed the global dynamics of a SEIR model with vertical transmission and a bilinear incidence rate. Korobeinikov [10] considered the global properties of the disease free equilibrium for SEIS and SEIR and also, Li [14] considered the global dynamic of the SEI and SEIR model with infectious force in latent infected and immune period. Research on global properties of epidemic models of SEIR is scarce in the literature.

In this paper, we consider the global properties of an SEIR model using the linear and non-linear Lyapunov function (Goh-Volterra) to establish the global stability of the disease free equilibrium and the endemic equilibrium state respectively.

## II. MODEL FORMULATION

The total population is divided into four compartments namely: Susceptible ( $S$ ), Exposed ( $E$ ), Infectious ( $I$ ) and Recovered ( $R$ ) population. The total size at time  $t$  is denoted by  $N(t)$ , where  $N(t) = S(t) + E(t) + I(t) + R(t)$ . The transfers diagram is depicted in the following figure:

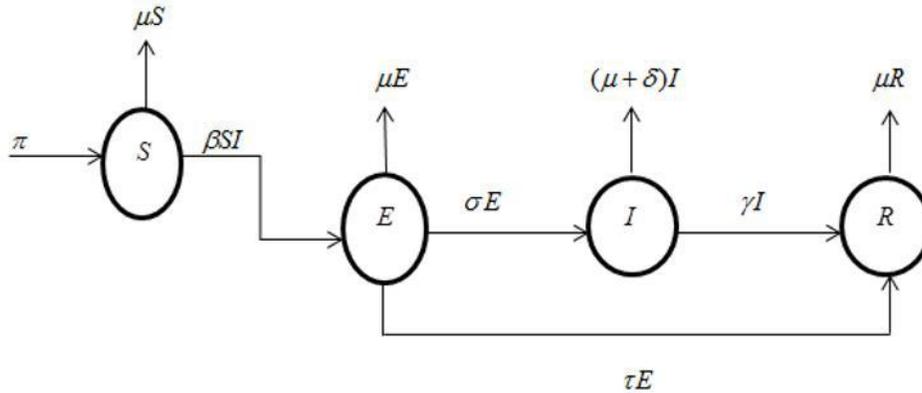


Fig.1: The schematic illustration of the SEIR model

The govern model is given by the system of non-linear ordinary differential equations below:

$$\begin{aligned} \frac{dS}{dt} &= \pi - \beta SI - \mu S \\ \frac{dE}{dt} &= \beta SI - \mu E - \sigma E - \tau E \\ \frac{dI}{dt} &= \sigma E - \delta I - \mu I - \gamma I \\ \frac{dR}{dt} &= \gamma I + \tau E - \mu R \end{aligned} \tag{1}$$

$N = S + E + I + R$  where  $N$  is the total population.

Table.1: Description of the parameters.

Parameters	Description
$\pi$	Recruitment rate
$\beta$	Effective contact rate
$\mu$	Natural death rate
$\sigma$	Progression rate of exposed individuals into infectious population
$\tau$	Recovery rate of the exposed due to prophylactic treatment
$\delta$	Disease-induced death rate due to infection in the diseased infected
$\gamma$	Recovery rate of the exposed due to treatment

### III. ANALYSIS OF THE MODEL

**Lemma 1:** The region  $D^* = \left\{ (S, E, I, R) \in \mathfrak{R}_+^4 : N \leq \frac{\pi}{\mu} \right\}$  is positively-

invariant for the model (1).

Proof: Adding the equations in the model system (1) gives:

$$\frac{dN}{dt} = \pi - \mu N - \delta I \tag{2}$$

Thus, whenever  $N > \frac{\pi}{\mu}$ , then  $\frac{dN}{dt} < 0$ . Hence, since it follows from the right-hand side of the equation (2) that  $\frac{dN}{dt}$  is bounded by  $\pi - \mu N$ , and by the standard comparison theorem [19] it can be shown that:

$$N(t) \leq N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t}) \tag{3}$$

If  $N(0) \leq \frac{\pi}{\mu}$ , then  $N(t) \leq \frac{\pi}{\mu}$ . Thus,  $D^*$  is a positivity invariant set under the flow described by (1) so that no solution path leaves through any boundary of  $D^*$ . Hence, it is sufficient to consider the dynamics of the model (1) in the domain  $D^*$ . In this region, the model can be considered as been mathematically and epidemiologically well-posed [10].

**3.1 Asymptotic stability of Disease free equilibrium (DFE)**

The DFE of the model (1) is given by:

$$\epsilon_0 = (S^*, E^*, I^*, R^*) = \left( \frac{\pi}{\mu}, 0, 0, 0 \right) \tag{4}$$

A very important concern about any infectious disease is its ability to invade a population [20]. The threshold condition known as the basic reproduction number (usually written as  $R_0$ ) is used in determining whether the disease will persist in the population or dies out as time increases; if  $R_0 < 1$ , then the disease free equilibrium (DFE) will be locally asymptotically stable and the disease cannot invade the population while when  $R_0 > 1$ , then the DFE is unstable and invasion is possible which could leads to an endemic equilibrium state [21].

The Local stability of  $\epsilon_0$  can be established using the next generation operator method on (1) [21]. Using the notation in [21], it follows that the matrices  $F$  and  $V$ , for the new infection terms and the remaining transfer terms are respectively given by:

$$F = \begin{pmatrix} 0 & \beta\pi \\ \mu & 0 \\ 0 & 0 \end{pmatrix}, V = \begin{pmatrix} A_1 & 0 \\ -\sigma & A_2 \end{pmatrix} \tag{5}$$

where  $A_1 = (\mu + \sigma + \tau)$ ,  $A_2 = (\delta + \mu + \gamma)$

Hence, it follows from [21] that:

$$R_0 = \rho(FV^{-1}) = \frac{\beta\pi\sigma}{A_1A_2\mu} \tag{6}$$

where  $\rho$  is the spectral radius (maximum eigenvalues). The result below follows from Theorem 2 in [18]:

**Lemma 2:** The DFE ( $\epsilon_0$ ) of model (1) is locally asymptotically stable (LAS) if  $R_0 < 1$  and unstable when basic reproduction number is greater than unity.

The threshold quantity  $R_0$ , is the effective reproduction number of the disease. It represents the average number of secondary cases generated by one infected person in a completely susceptible population [9]. The epidemiological implication of the **Lemma 2** is that when the  $R_0 < 1$ , a small infection of the virus into the population will not generate large infection outbreaks, and this indicates that the disease will dies out in a short period of time.

**3.2. Global asymptotic stability of DFE for  $R_0 < 1$**

**Theorem 1:** The DFE of the model (1) is globally asymptotically stable (GAS) in  $D^*$  whenever  $R_0 < 1$ .

Proof: Considering the following linear Lyapunov function:

$$V = B_1E + B_2I \tag{7}$$

With Lyapunov derivative (where a dot represents differentiation with respect to time)

$$\dot{V} = B_1\dot{E} + B_2\dot{I} \tag{8}$$

Substituting the expression for  $\dot{E}$  and  $\dot{I}$  from (1) into (8), we have:

$$\begin{aligned} \dot{V} &= B_1(\beta SI - A_1E) + B_2(\sigma E - A_2I) \\ &= B_1\beta SI - B_1A_1E + B_2\sigma E - B_2A_2I \end{aligned} \tag{9}$$

Little perturbation from equation (9) with the reproduction number (6) gives:

$$\begin{aligned} B_1 &= \pi\sigma \\ B_2A_2 &= A_1A_2\mu \end{aligned} \tag{10}$$

Substituting the expression of  $B_1, B_2$  obtained from equation (10) we have:

$$\begin{aligned} \dot{V} &= \beta SI\pi\sigma - IA_1A_2\mu \\ &= I[\beta S\pi\sigma - A_1A_2\mu] \\ &= I(A_1A_2\mu) \left[ \frac{\beta S\pi\sigma}{A_1A_2\mu} - 1 \right] \end{aligned}$$

Since  $S = \frac{\pi}{\mu} \leq N$ , it then follows that:

$$\begin{aligned} \dot{V} &\leq I(A_1A_2\mu) \left[ \frac{\beta\pi\sigma}{A_1A_2\mu} - 1 \right] \\ &= I(A_1A_2\mu)(R_0 - 1) \end{aligned}$$

with equality only at  $\varepsilon_0$ . For  $R_0 < 1$ ,

$$\dot{V} \leq 0$$

with equality only when  $I = 0$ . Hence, by LaSalle's extension to Lyapunov's principle [12], the limit set for each solution is contained in the largest invariant set for which  $I = 0$ , this being the singleton  $\varepsilon_0$ .

Hence, the Lyapunov function (7) required for the GAS of the disease free equilibrium for the model in question is given as:

$$\varepsilon_1 = \left\{ \begin{array}{l} S^{**} = \frac{A_1 A_2}{\beta \sigma}, \quad E^{**} = \frac{\pi \beta \sigma - A_1 A_2 \mu}{A_1 \beta \sigma} \\ I^{**} = \frac{\pi \beta \sigma - A_1 A_2 \mu}{A_1 A_2 \beta}, \quad R^{**} = \frac{(\pi \beta \sigma - A_1 A_2 \mu)(\gamma \sigma + A_2 \tau)}{A_1 A_2 \beta \mu \sigma} \end{array} \right\} \quad (12)$$

For which  $\pi \beta \sigma - A_1 A_2 \mu > 0$  to have a positive equilibria in the domain.

where  $A_1 = (\mu + \sigma + \tau)$ ,  $A_2 = (\delta + \mu + \gamma)$ .

### 3.3 Global asymptotic stability of boundary equilibrium, for $R_0 > 1$

**Theorem 2:** The unique boundary equilibrium of the model (1) is globally asymptotically stable (GAS) in  $D^*$  whenever  $R_0 > 1$ .

Proof: Considering the model (1) and  $R_0 > 1$ , so that the associated unique endemic equilibrium  $\varepsilon_1$  of the model exists. We consider the following non-linear Lyapunov function of Goh-Volterra type:

$$\mathbf{Z} = \left( S - S^{**} - S^{**} \log \frac{S}{S^{**}} \right) + \left( E - E^{**} - E^{**} \log \frac{E}{E^{**}} \right) + Q \left( I - I^{**} - I^{**} \log \frac{I}{I^{**}} \right) \quad (13)$$

With Lyapunov derivative (where a dot represents differentiation with respect to time)

$$\dot{\mathbf{Z}} = \left( \dot{S} - \frac{S^{**} \dot{S}}{S} \right) + \left( \dot{E} - \frac{E^{**} \dot{E}}{E} \right) + Q \left( \dot{I} - \frac{I^{**} \dot{I}}{I} \right) \quad (14)$$

Substituting the derivatives  $(\dot{S}, \dot{E}, \dot{I})$  from (1) into (14), we have:

$$\dot{\mathbf{Z}} = \left( \pi - \beta SI - \mu S - \frac{S^{**} \pi}{S} + S^{**} \beta I + S^{**} \mu \right) + \left( \beta SI - A_1 E - \frac{E^{**} \beta SI}{E} + E^{**} A_1 \right) + \left( Q \sigma E - Q A_1 I - \frac{Q I^{**} \sigma E}{I} + Q I^{**} A_2 \right) \quad (15)$$

At steady state from equation (1) we have:

$$\pi = \beta S^{**} I^{**} + \mu S^{**} \quad (16)$$

Substituting equation (16) into (15) gives:

$$\dot{\mathbf{Z}} = \left( \beta S^{**} I^{**} + \mu S^{**} - \beta SI - \mu S - \frac{S^{**} \beta S^{**} I^{**}}{S} - \frac{S^{**} \mu S^{**}}{S} + S^{**} \beta I + S^{**} \mu \right) + \left( \beta SI - A_1 E - \frac{E^{**} \beta SI}{E} + E^{**} A_1 \right) + \left( Q \sigma E - Q A_1 I - \frac{Q I^{**} \sigma E}{I} + Q I^{**} A_2 \right) \quad (17)$$

$$V = \pi \sigma E + A_1 \mu I \quad (11)$$

### 3.2 Existence of Stability of Boundary Equilibria

Besides the disease – free equilibrium point, we shall show that the formulated model (1) has an endemic equilibrium point. The endemic equilibrium point is a positive steady state solution where the disease persists in the population.

Solving the system of equation (1) above at the endemic steady- state resulted into:

Further simplification gives:

$$\dot{\mathbf{Z}} = \left( \beta S^{**} I^{**} + \mu S^{**} - \mu S - \frac{S^{**2} \beta I^{**}}{S} - \frac{S^{**2} \mu}{S} + S^{**} \beta I + S^{**} \mu \right) + \left( -A_1 E - \frac{E^{**} \beta S I}{E} + E^{**} A_1 \right) + \left( Q \sigma E - Q A_1 I - \frac{Q I^{**} \sigma E}{I} + Q I^{**} A_2 \right) \tag{18}$$

Collecting all the infected class without the double star (\*\*) from (18) and equating to zero:

$$S^{**} \beta I - A_1 E + Q \sigma E - Q A_2 I = 0 \tag{19}$$

A little perturbation of steady state from (1) and (19) resulted into:

$$Q = \frac{S^{**} \beta}{A_2}, \quad A_1 = \frac{\beta S^{**} I^{**}}{E^{**}}, \quad \sigma = \frac{A_2 I^{**}}{E^{**}} \tag{20}$$

Substituting the expression from (20) into equation (18) (a several algebraic calculations) gives:

$$\dot{\mathbf{Z}} = \beta S^{**} I^{**} + \mu S^{**} - \mu S - \frac{S^{**2} \beta I^{**}}{S} - \frac{S^{**2} \mu}{S} + S^{**} \mu - \frac{E^{**} \beta S I}{E} + S^{**} I^{**} \beta - \frac{I^{**2} E S^{**} \beta}{I E^{**}} + S^{**} \beta I^{**} \tag{21}$$

Factorizing equation (19) resulted into:

$$\dot{\mathbf{Z}} = \mu S^{**} \left( 2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) + \beta S^{**} I^{**} \left( 3 - \frac{S^{**}}{S} - \frac{I^{**} E}{I E^{**}} - \frac{S E^{**} I}{E} \right) \tag{22}$$

Finally, since the arithmetic mean exceeds the geometric mean, the following inequality from (22) holds:

$$\left( 2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) \leq 0, \quad \left( 3 - \frac{S^{**}}{S} - \frac{I^{**} E}{I E^{**}} - \frac{S E^{**} I}{E} \right) \leq 0$$

Thus,  $\dot{\mathbf{Z}} \leq 0$  for  $R_0 > 1$ . Hence,  $\mathbf{Z}$  is a Lyapunov function in  $D^*$  and it follows by LaSalle’s Invariance Principle [12], that every solution to the equations of the model (1) approaches the associated unique endemic equilibria  $(\mathcal{E}_1)$ , of the model as  $t \rightarrow \infty$  for  $R_0 > 1$ .

#### IV. CONCLUSION

A simple SEIR model with a prophylactic for the exposed individuals was considered and analyzed with an assumption of the disease induce rate. This work has examined the global stability of the equilibria of the model using linear and non-linear Lyapunov function. We have shown that the disease free and the endemic equilibria are globally asymptotically stable whenever the associated reproduction number is less than unity ( $R_0 < 1$ ) and greater than unity ( $R_0 > 1$ ) respectively; for the endemic equilibrium, this implies that if the disease is contained in the population, then the disease will persist in the population.

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