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Mathematical Analysis of Infectious Disease Dynamics

Ekwomadu M. Onyebuchi[1], AzeezA.Waheed[*2] and AkindoyeM. Okedoye[3]
[1] *Department of Mathematics and Computer Science Federal University of Petroleum Resources, Nigeria.*
[2]*Department of Mathematics, Lead City University, Ibadan, Oyo State Nigeria*
[3]*Department of Mathematics and Computer Science Federal University of Petroleum Resources, Nigeria.*
azewah2004@yahoo.com[2], okedoye.akindele@fupre.ng[3]

Abstract:
An S; Ic; I; R epidemic model is developed in this study to aid in the prevention and spread of diseases. The existence of a plausible region in which the disease can spread is demonstrated in this research by a careful analysis and study of the available knowledge. Quantitative study and the application of mathematical modeling techniques demonstrate that the disease has an endemic equilibrium point (EEP) and a disease free equilibrium (DFE).When the basic reproduction number R_0 is less than zero, the DFE was found to be worldwide asymptotically stable, but the EEP was discovered to be globally asymptotically stable when R_0 is greater than zero and the disease is latent when R = 0.The model was numerically simulated using the maple-coded rkf45 ODE solvers, and the results indicated that a combination of immunization, early detection, and separation of infected people from the general population will lead to a faster eradication of the disease.
Key Word*:Epidemic models, global stability, Lyapunov functions, Infectious diseases.*

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I. Introduction

For some infectious diseases, there are humans who are able to transfer their illness but will not show any symptoms. These set of people are called carriers and they play a major role in the transmission of the disease. We can categorize carriers in two different types. Genetic carriers possess the illness on their recessive genes. They can only transfer the disease to their children and are not contagious. The major part of our study focuses on infectious disease carriers. These set of people are asymptomatic and are likely unaware of their conditions, and therefore are more likely to transfer the infection to others. A deadly infectious disease that produces long-term asymptotic carriers is the Typhoid fever caused by the bacteria Salmonella Typhus. The incidents of Mr. N the milkman in England and Typhoid Mary in the US at the turn of the 20th century brought typhoid fever to the attention of the general public. Over the years, as they worked in the food production industry and in private residences, these individuals contaminated hundreds of other people. Even now, typhoid fever kills 200,000 people annually throughout the world and infects 21 million people.[16] Asymptomatic carriers obstruct the use of therapy and vaccine to eradicate typhoid fever, and they are thought to be crucial in the evolution and global transmission of typhus.[16]

Hepatitis B, a liver condition brought on by the Hepatitis B Virus (HBV) of the Hepadnavirus family, is another serious viral disease that can be fatal and results in long-term asymptomatic carriage. The majority of HBV-infected people fully recover and acquire a lifetime immunity to the virus. However, 15–25% of persons with persistent HBV infection will also develop liver disease. This percentage is 5–10%. Jaundice, nausea, weariness, and joint discomfort are among the signs of hepatitis B. According to Riggs et al. (2007), about 30% of those who have the condition are also carriers. The existence of a sizable population of chronic carriers, who are in charge of spreading the majority of new infections, is a significant public health concern in the fight against hepatitis B infection in many nations. Asymptomatic carriers are also known to result from infections with other organisms. One of the most prevalent viruses in humans is the Epstein-Barr virus (EBV), a member of the herpes family. EBV infection commonly causes infectious mononucleosis, also known as glandular fever. Most people infected with EBV are asymptomatic, as it remains dormant in those who have had it for the rest of their lives in the cells of the throat and the immune system. A bacteria called Clostridium diffcile is the source of disorders with such name (CDAD). More than 300,000 instances of acute hospital-acquired diarrhea occur each year in acute-care institutions in the US, with Clostridium diffcile related disorders still the most common cause. In long-term care facilities, asymptomatic carrying rates of up to 30% have been documented. Large outbreaks of CDAD in Europe and North America are thought to have been caused by carriers.[15]

The paper discussed a mathematical model that incorporates a person's sexual orientation to describe the dynamics of HIV transmission. The threshold that determines whether or not the sickness disappears is

provided by the basic reproduction number. The non-linear ODE model demonstrates the presence of distinctive disease-free and disease-persistent equilibria. To quantitatively evaluate the trend of infection among each gender, least squares curve fitting is shown. The findings suggest that the female population is more infectious.[4]

The effects of the HBV vaccination were examined by[18] using a mathematical model for hepatitis B with carriers. Hepatitis B and other diseases are the focus of several other studies that use large-scale computer models with carriers.[2,3,5,12,14.]

The fundamental ideas and practical applications of mathematical modeling of infectious disease were examined and the paper focuses on the use of mathematical modeling of infectious illnesses in epidemiology by health professionals and students. Results from stochastic models are more applicable to transmission dynamics than those from deterministic models, which are non-probabilistic but assist construct a prospection of potential scenarios in epidemiology.[1]

Mathematical analysis of hepatitis B epidemic models with optical control was discussed[8]. The hepatitis B virus (HBV) infection is examined as a global health issue in the research. Numerous control measures were taken into consideration, including immunization, therapy, and education campaigns (awareness). The solution of the disease-free and endemic equilibrium mode was studied for stability and existence. The results of a numerical simulation indicate that the best approach to preventing hepatitis B virus (HBV) infection is to combine treatment, vaccine, and education and campaigning to raise public awareness.[8]

Mathematical Modeling and Analysis of Pneumonia Infection Dynamics One of the main causes of death globally is pneumonia, particularly in young children under 5 years old, seniors over 65 years old, and those with weakened immune systems. Due to the severe toll it took on humanity, it is frequently referred to as the "captain of the men of death." In this study, they used a deterministic SEIR model to mathematically analyze the dynamics of the pneumonia disease. If the fundamental reproduction number is more than one, the pneumonia endemic equilibrium is observed to be globally asymptotically stable in the invariant region, and if it is less than one, the pneumonia free equilibrium is observed to be locally asymptotically stable. The most sensitive factors, according to the sensitivity analysis, were the rates of infection and transmission, and the occurrence of forward bifurcation was discovered by the bifurcation analysis using the center manifold theory.[11]

The Susceptible-Infected-Recovered (SIR) model is investigated using ill-defined biological factors. Here, the model with erroneous data is taken into consideration because there aren't any precise numerical data for the biological parameters. Sometimes it is not possible to gather the numerical data as a fixed value, but it is simple to identify the interval to which it belongs. Because of this, a SIR model is being introduced with interval numbers as its parametric functional form.[7]

By using the variational iteration method the solution of a deterministic mathematical model of typhoid fever was studied. In this study, the typhoid fever model will be solved using the variational iteration method (VIM) for a given constant population. Ordinary differential equations of nonlinear first order are used to explain this mathematical model. First, we use the Variational Iteration Method to determine this model's solution (VIM). We compare the results from VIM and RK4 to demonstrate the effectiveness of the method. Using the traditional fourth-order Runge-Kutta method incorporated within the computer, the validity of the VIM in solving the model is established. We represented the profiles of each compartment's solutions, and based on these, we made the assumption that the VIM and RK4 solutions were in good agreement.[17]

As in most mathematical modeling fields, there is always a trade-off between simple models, which leave out most details and are only intended to highlight general quantitative behavior, and detailed models, which are typically intended for specific situations and include short-term quantitative predictions. Although their strategic relevance may be great, detailed models are typically difficult or impossible to solve analytically, which limits their usefulness for theoretical reasons.[13]

II. Mathematical Model Formulation

Darja and Micheal [6] consider compartment model similar to

$$S - I_c - I - R \quad (1)$$

However, we make a few modifications from the model discussed by Darja and Micheal [6]

Table 1. Parameters in the Model

PARAMETERS	DESCRIPTIONS
b	Rate of influx of susceptible
d_1, d_4	Natural death rates
d_2, d_3	Death rates for Ic and I compartments, respectively, including both natural and disease-caused death
β	Transmission coefficient for the carrier compartment I_c

γ	Transmission coefficient for the symptomatically infected compartment I
α	Rate at which carriers develop symptoms
π	Rate of recovery
P	Probability of a newly infected individual is asymptomatic
θ	Vaccination rate
μ	Rate at which recovered individuals become susceptible again

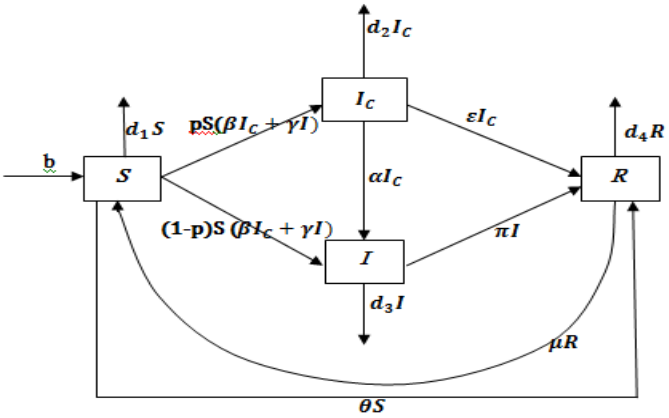

Figure 1: Model diagram [3]

The Propose Model

From the model diagram, the following system of differential equation were gotten

$S^1 = b - d_1S - pS(\beta I_C - \gamma I) - (I - p)S(\beta I_c - \gamma I) - \theta S + \mu R$ (2)

$I_c^1 = pS(\beta I_C - \gamma I) - d_2 I_c - \alpha I_c - \varepsilon I_c$ (3)

$I^1 = (1 - p)S(\beta I_c - \gamma I) - d_3 + \pi)I + \alpha I_c$ (4)

$R^1 = \pi I + \theta S + \epsilon I_c - (d_4 + \mu)R$ (5)

and simplifying the above equations we get the following

$S^1 = b - d_1S - S(\beta I_c - \gamma I) - \theta S + \mu R$ (6)

$I_c^1 = pS(\beta I_C - \gamma I) - (d_2 + \alpha + \varepsilon)I_c$ (7)

$I^1 = (1 - p)S(\beta I_c - \gamma I) - (d_3 + \pi)I + \alpha I_c$ (8)

$R^1 = \pi I + \theta S + \epsilon I_c - (d_4 + \mu)R$ (9)

Equilibrium points

The system's equilibrium points represent a period of time during which the population's rate of change is zero. Therefore, we put $S^1 = I_c = R^1 = 0$ and solve the resulting system of non-linear equations below to achieve our equilibrium positions.

$$b - d_1S - S(\beta I_c - \gamma I) - \theta s + \mu R = 0$$

$$pS(\beta I_c - \gamma I) - (d_2 + \alpha + \varepsilon)I_c \quad (10)$$

$$1 - p)S(\beta I_c - \gamma I) - (d_3 + \pi)I + \alpha I_c = 0$$

$$\pi I + \theta S + \varepsilon I_C - (d_4 + \mu)R = 0$$

III. Local stability of the Disease Free Equilibrium point (DFEP)

The DFE occurs when $I = I_c = 0$. Therefore, applying this to the equations in (10) we have

$S^1 = b - (d_1 + \theta)S + \mu R$ (11)

$R^1 = \theta S - (d_4 + \mu)R = 0$ (12)

Thus solving (11) and (12) we obtain

$S = \frac{b + \mu R}{(d_1 + \theta)}$ and $R = \frac{\theta S}{d_1}$

Then putting R into S in

$S = \frac{b+\mu\left(\frac{\theta S}{d_4+\mu}\right)}{(d_1+\theta)}$ so that

$$S = \frac{S\mu\theta + b\mu + bd_4}{(d_4+\mu)(d_1+\theta)} \Rightarrow S(d_4+\mu)(d_1+\theta) = S\mu\theta + b\mu + bd_4$$

Then $S\big((d_1+\mu)-(d_1+\theta)-\mu\theta\big) = b(\mu+d_1)$ and hence

$S = \frac{b(\mu+d_4)}{d_1(d_1+\theta)+\mu d_1}$ (13)

Putting S from equation (13) into R gives

$R = \frac{\theta\left(\frac{b(\mu+d_1)}{d_4(d_1+\theta)\mu d_1}\right)}{(d_4+\mu)} = \frac{\theta b}{d_4(d_1+\theta)+\mu d_1}$ hence

$$R = \frac{\theta b}{d_4(d_1+\theta)+\mu d_1}$$

And then we have the Diseases free equilibrium point as $p_0 = \{S, I_c, I, R\}$,i.e

$P_0 = \left\{\frac{\theta b}{d_4(d_1+\theta)+\mu d_1}, 0, 0\ \frac{\theta b}{d_4(d_1+\theta)+\mu d_1}\right\}$ (14)

IV. Global Stability Analysis of the Disease Free Equilibrium

Here, we examine the DFE's stability using the Lyapunov functions method described by Darija and Micheal[6] ,demonstrating that the DFEF is globally asymptotically stable under the condition that $R_0 \leq 1$.

Theorem 1: (Global stability of the Disease Free Equilibrium). The DFEP P_0 is globally asymptotically stable in the feasible region provided $R_0 \leq 1$ Darya and Micheal[6]

Proof. Using the method of Lyapunov theorem we define Lyapunov function L given has

$L = \left(\frac{\beta}{d_2+_\alpha+\varepsilon} + \frac{\alpha\gamma}{(d_3+\pi)(d_2+\alpha+\varepsilon)}\right) I_c + \left(\frac{\gamma}{d_3+\pi}\right) I$ (15)

Then

$\frac{\partial L}{\partial t} = \left(\frac{\beta}{d_2+\alpha+\epsilon} + \frac{\alpha\gamma}{(d_3+\pi)(d_2+\alpha+\epsilon)}\right) I_c + \left(\frac{\gamma}{d_3+\pi}\right)\gamma$ (16)

Substituting the values for I_c and I give (6) and (7) into (16) then

$$\frac{\partial L}{\partial t} = \left(\frac{\beta}{d_2+\alpha+\epsilon} + \frac{\alpha\gamma}{(d_3+\pi)(d_2+\alpha+\epsilon)}\right)[pS(\beta I_c + \gamma I) - (d_2+\alpha+\varepsilon)I_c] + \left(\frac{\gamma}{d_2+\pi}\right)[(1-p)S(\beta I_C + \gamma I) - (d_3+\pi)I + \alpha I_c]$$

$$= \left\{\frac{p\beta}{d_2+\alpha+\varepsilon} + \frac{p\alpha\gamma}{(d_3+\pi)(d_2+\alpha+\varepsilon)} + \frac{(1-p)\gamma}{(d_3+\pi)}\right\} S(\beta I_c + \gamma I) - \beta I_c - \frac{p\beta}{(d_3+\pi)} I_c - \gamma I + \frac{\alpha\gamma}{(d_3+\pi)} I_c$$

Therefore

$\frac{\partial L}{\partial t} = \left\{\frac{p\beta}{d_2+\alpha+\varepsilon} + \frac{p\alpha\gamma}{(d_3+\pi)(d_2+\alpha+\varepsilon)} + \frac{(1-p)\gamma}{(d_3+\pi)}\right\} S(\beta I_c + \gamma I) - (\beta I_c + \gamma I)$ (17)

However, from (17)

$\left\{\frac{p\beta}{d_2+\alpha+\varepsilon} + \frac{p\alpha\gamma}{(d_3+\pi)(d_2+\alpha+\varepsilon)} + \frac{(1-p)\gamma}{(d_3+\pi)}\right\} = R_0\left(\frac{d_4(d_1+\theta)+\mu d_1}{d_1(d_4+\mu)}\right)$ (18)

Then putting (17) and (18) we have

$$\frac{\partial L}{\partial t} = R_0\left(\frac{d_4(d_1+\theta)+\mu d_1}{d(d_1+\mu)}\right) S(\beta I_c + \gamma I) - (\beta I_c + \gamma I)$$

and from the feasible region in (12) we know that

$$S \leq \frac{b+\mu R}{d_1+\theta} = \frac{\theta b}{d_4(d_1+\theta)+\mu d_1}$$

Using this results in

$$\frac{\partial L}{\partial t} \leq R_0\left(\frac{d_4(d_1+\theta)+\mu d_1}{d(d_1+\mu)}\right).\left(\frac{\theta b}{d_4(d_1+\theta)+\mu d_1}\right)(\beta I_c + \gamma I) - (\beta I_c + \gamma I)$$

$\frac{\partial L}{\partial t} \leq R_0(\beta I_c + \gamma I) - (\beta I_c + \gamma I)$ and then

$\frac{\partial L}{\partial t} \leq (R_0 - 1) - (\beta I_c + \gamma I)$ (19)

From the above we see that $\frac{\partial L}{\partial t} \leq 0\ provided\ R_0 < 1$

Furthermore $\frac{\partial L}{\partial t} = 0 \Leftrightarrow I_c = I = 0\ or\ R = 1\ and\ S = \frac{\theta b}{d_4(d_1+\theta)+\mu d_1}$. Therefore the set $\{P_0\} = \{S, I_c, I, R\}$ which is the DFEP is the largest variant set in the closure of at which $\frac{\partial L}{\partial t} = 0$ .Therefore by Laselle's invariance principle, Lasall[19]the DFEP is globally asymptotically stable in Ω.

V. Globally Stability of the Endemic Equilibrium P*

Theorem 2: (Globally stability of the Endemic Equilibrium). The EPP P* is globally asymptotically stable on the feasible region Ω provided $R_0 > 1$

Proof.

To study the stability, we make use of the same method applied by [9]. Hence using the Lyapunov function

$V(S, I_c, I) = \alpha_1(S - S^* \ln S) + \alpha_2(I_c - I_c^* \ln I_c) + \alpha_3(I - I_c^* \ln I_c)$ (20)

Where $\alpha_1, \alpha_2, \alpha_3 > 0$ are constants

We wish to show that the derivatives of V are negative definite with respect to the EEP P*

From equation (5) we show that

$b = (d_1 + \theta)S^* + \beta I_c^* S^* + \gamma I^* S^* - \mu R^*$(21)

Differentiating (20) with respect to t gives

$V^1 = \alpha_1\left(S^1 - \frac{S^*}{S}S^*\right) + \alpha_2\left(I_c^1 - \frac{I_c^*}{I_c}\right) + \alpha_3\left(I^1 - \frac{I^*}{I}I^1\right)$ (22)

$V^1 = \alpha_1\left[b - (d_1 + \theta)S - (\beta I_c + \gamma I)S + +\mu R\frac{bS^*}{S} + (d_1 + \theta)S^* - (\beta I_c + \gamma I)S^* - \frac{\mu R S^*}{S}\right]\alpha_2\left[(1-p)(\beta I_c + \gamma IS - d2 + \alpha + \varepsilon Ic1 - pS\beta Ic* - 1 - pS\gamma Ic*Ic + d2 + \alpha + \varepsilon Ica3p\beta Ic + \gamma IS + \alpha Ic - d3 + \pi I - pS\beta IcI*I - p\gamma SI* - \alpha IcI*I + d3 + \pi\right.$ (23)

Putting b from (21) into (22) we obtain

$$V^1 = \alpha_1\left[(d_1 + \theta)S^* - (\beta I_c^* + \gamma I^*)S^* + +\mu R^*\frac{bS^*}{S} + (d_1 + \theta)S^* - (\beta I_c + \gamma I)S^* - (d_1 + \theta)S - (\beta I_c + \gamma I)S + \mu R(d_1 + \theta)\frac{(S^*)^2}{S} - (\beta I_c^* + \gamma I^*)\frac{(S^*)^2}{S} + \frac{\mu R S^*}{S} + (d_1 + \theta)S^* + (\beta I_c + \gamma I)S^* - \frac{\mu R S^*}{S}\right] + \alpha_2\left[(1-p)(\beta I_c + \gamma I)S - (d_2 + \alpha + \varepsilon)I_c - (1-p)S\beta I_c^* - (1-p)S\frac{\gamma I_c^* I}{I_c} + (d_2 + \alpha + \varepsilon)I_c\right] + \alpha_3\left[p(\beta I_c + \gamma I)S + \alpha I_c - (d_3 + \pi)I - pS\frac{\beta I_c I^*}{I} - p\gamma SI^* - \alpha\frac{I_{cI^*}}{I} + (d_3 + \pi)\right]$$

(24)

The constants α_1, α_2 and α_3 are chosen as

$\alpha_1 = 1, \alpha_2 = \frac{(d_3+\pi)\beta S^* + \gamma\alpha S^*}{(d_2+\alpha)(d_1+\pi)}$, $\alpha_3 = \frac{\gamma S^*}{d_3+\pi}$

And they satisfy the equation

$$-\alpha_1 + \alpha_2(1-p) + \alpha_3 p = 0$$
$$\alpha_1\gamma S^* - d_3(d_3 + \pi) = 0$$
$$\alpha_1\beta S^* - \alpha_2(d_2 + \alpha) + \alpha_3\alpha = 0$$

The term V^1 are then regrouped in the form $V^1 = \frac{\pi_3 p\beta S I_c I^*}{I}$ where

$$V_1 = (d_1 + \theta s^*)\left(2 - \frac{S}{S^*} - \frac{S^*}{S}\right)$$
$$V_2 = \alpha_1(\beta I_c^* + \gamma I^*)S^* + \alpha_2(d_2 + \alpha) + \alpha_3(d_3 + \pi)I^*$$
$$V_3 = -\frac{\alpha_1(\beta I_c^* + \gamma I^*)S^*}{S} - \alpha_2(1-p)\beta SI_c^* - \alpha_3 p\gamma SI^* - \frac{\alpha_2(1-p)\gamma ISI_c^*}{I_c} - \frac{\pi_3 p\beta SI_c I^*}{I} - \frac{\pi_3\alpha I_c I^*}{I}$$

And as shown by Darja and Michael[6]both V^1,V_2 and V_3 are all less than zero thus$V^1 \leq 0$ and therefore the EEP P* is gradually asymptotically stable**.**

VI. Numerical Simulations

To see the dynamic behavior of system (2 -5), we solve the system using the maple-coded rkf45 ODE solvers using the following parameters; b = 80,0000 , $d_1 = \frac{1}{70}$, $d_2 = d_1 + 0.003$, , β = 0.398, γ = 0.143, π = 0.75, $d_3 = d_2$, $d_4 = d_1$,μ = 0.4e^{-2},ε = $\frac{1}{100}$, $p = \frac{1}{25}$ with the different values of αand θ. In figure 1,2, We vary the parameter α to see the effects of diagnosis rate at which carriers move into the infected class. We use different values of α and we discovered that the number of symptomatically infected individuals decreases significantly. In figure 3 and 4 we use different values of θ and that though the number of symptomatically infected reduces rapidly, the number of carriers remains high. Increasing θ from 0.1 t0 0.7 slightly alters the

disease dynamics; the number of carriers only shows a moderate decline. This implies that the parameters have a part to play in the eradication of the diseases in the population.

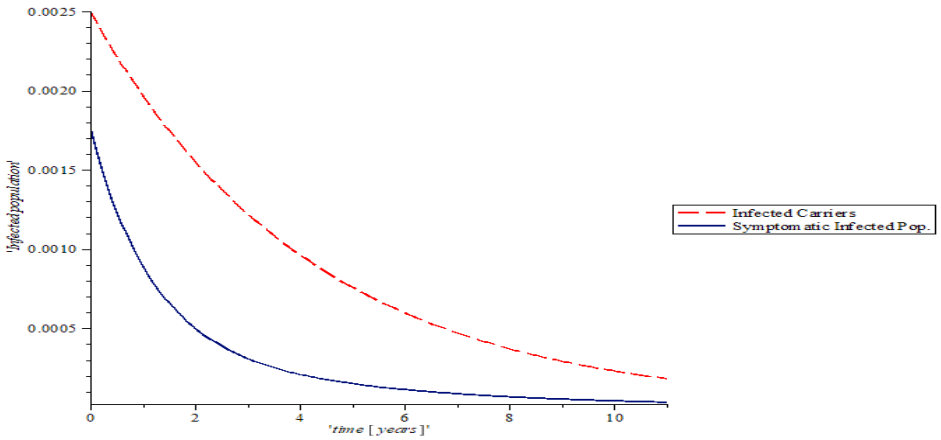

Figure 1: Graph showing the effect of diagnosis when $\alpha = 0.1$

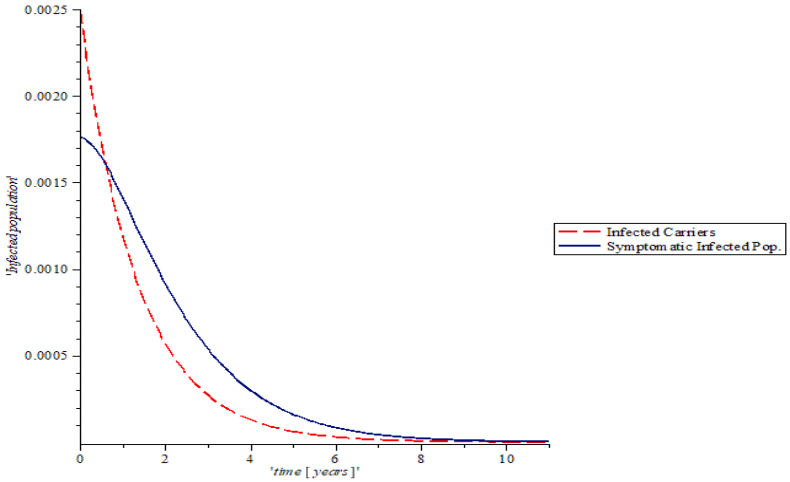

Figure 2: Graph showing the effect of diagnosis when $\alpha = 0.6$

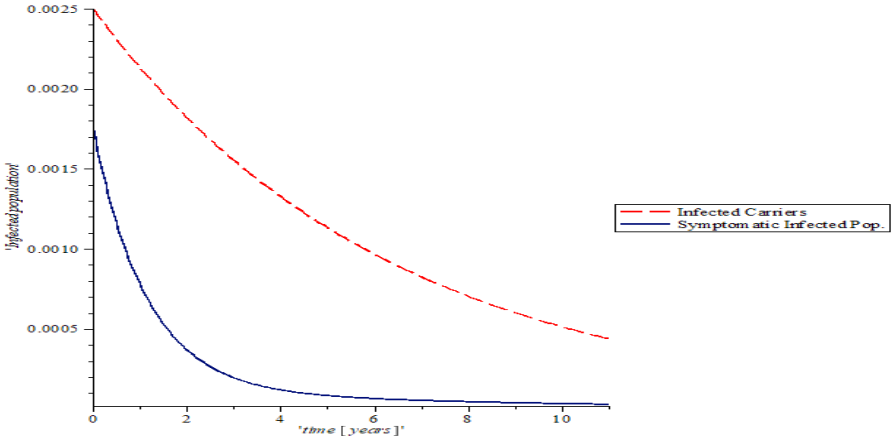

Figure 3a: Graph showing the effect of vaccination rate when $\theta = 0.1$

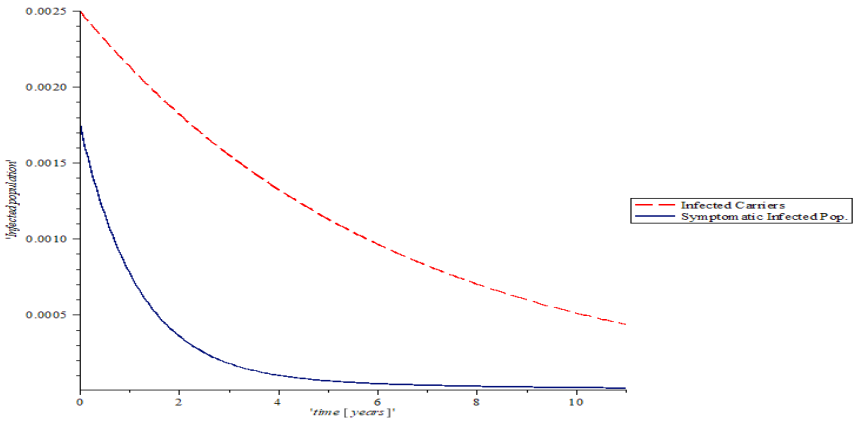

Figure 3b: Graph showing the effect of vaccination rate when $\theta = 0.7$

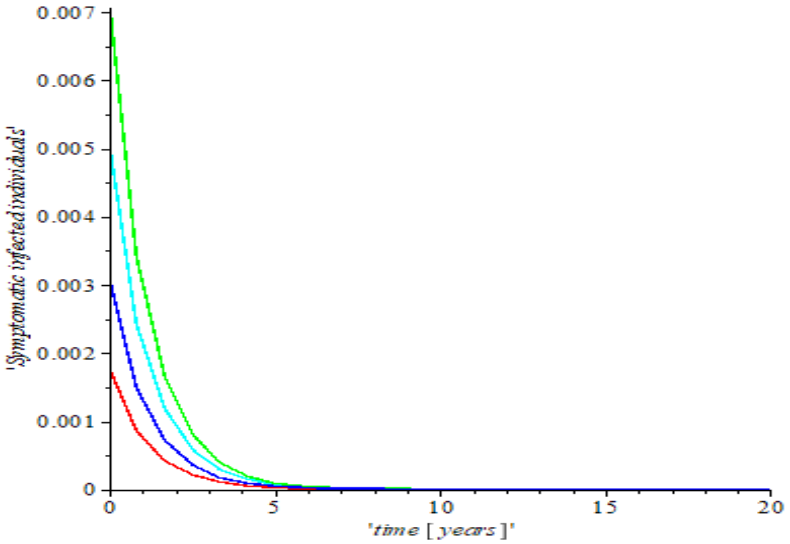

Figure 4: The trajectory of the symptomatic infected compartment when $R_o < 1$ is shown in (a)

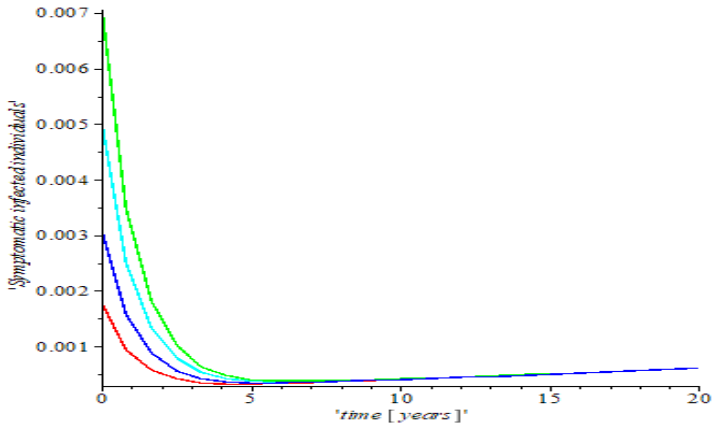

Figure 6: The trajectory of the symptomatic infected compartment when when $R_o > 1$ shown in (b) at different initial conditions of I

VII. Conclusion

In this paper, an SEIRS deterministic model with saturated incidence rate is formulated. Some of the main findings of this study are;

i. The model has locally and globally asymptotically stable disease-free equilibrium whenever the associated reproduction number is less than unity;

ii. The model has a unique endemic equilibrium and the endemic equilibrium is globally asymptotically stable.

iii. Numerical simulations illustrate the importance of the parameter, that measures the effects of sociological, psychological or other mechanisms of the disease.

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