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Stability analysis of SIR holing type II infectious epidemic model with treatment failure rate

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Abstract

In this paper a mathematical model of **SIR** epidemics diseases holing type II with a treatment failure rate on a population of individuals had been introduced and studied so, its dynamical behavior such as stability derived ,first we prove the existence of equilibrium points via Discartes rule of signs ,then local stability demonstrate for the above equilibrium points using Roth Hurwitz criteria , finally the global stability established with assistance of Lyapunov theorem to the equilibrium points above and the results was encouraging and satisfy.

Key words stability , Holling type II , epidemic SIR model .

1.Introduction

It is well common infectious have tremendous influenced on human life and for this controlling these disease is very important issue . Consequently , many epidemic models which used mathematics for describing the evolution of infectious diseases in the populations, are constructed and investigated in the researches and theses , these models are different from each other depending on the type of transmission of disease , latent period , resistance , immigrants , vaccination and many other causes. The existence of infectious disease fractionate the population into many parts depending on the type of disease , such as susceptible (S) ,infected (I) , recovered (R) and others. The well known epidemic model **SIR** which proposed originally by *kermark* and *Mackendrick* in 1927 [1]. In the **SIR** model the susceptible individuals become infective by contact with infected individuals and then the infected individuals may recovered and transfer to removal individuals at a specific rate . In [2] Ahmed A. M. and Hanan K. studied stability of Prey-Predator of SIS model epidemic disease in predator involving Holling type II functional response . In [3] studied Many models for the spread of infectious diseases in populations been analyzed mathematically and applied to specific diseases. the contact number σ, and the replacement number R are reviewed for the classic SIR epidemic and endemic models. Similar results with new expressions for $R0$ are obtained for MSEIR and SEIR endemic models with either continuous age or age groups. Values of $R0$ and σ are estimated for various diseases including measles in Niger and peruses in the United States. Previous models with age structure, heterogeneity, and spatial structure are surveyed .
In [4] David Easley and Jon Kleinberg study the epidemic diseases and the networks that transmit them in there book " Networks, Crowds, and Markets: Reasoning about a Highly Connected World " The patterns by which epidemics spread through groups of people is determined not just by the properties of the pathogen carrying it including its contagiousness, the length of its infectious period, and its severity but also by network structures within the population it is affecting. The social network within a population recording who knows whom determines a lot about how the disease is likely to spread from one person to another. But more generally, the opportunities for a disease to spread are given by a *contact network* : there is a node for each person, and an edge if two people come into contact with each other in a way that makes it possible for the disease to spread from one to the other. In [5] Vijaya L. G. M. R. and others investigates the dynamical complexities of a prey predator model with susceptible and infected (SI) prey with nonlinear feedback . adequately in1965 Holling identified three general categories of functional response that he called Types 1, 2, and 3, Type 1 is the simplest: capture rate increases in direct proportion to prey density until it abruptly saturates. Type 2 is similar in that the rate of capture increases with increasing prey density, but in contrast to the linear increase of Type 1, Type 2 approaches saturation gradually. Type 3 is similar to Type 2 except at low prey density, where the rate of prey capture accelerates the aura of received knowledge. Now designated by roman numerals, Holling's functional responses appear in every introductory ecology text, usually with illustrative examples (e.g., filter feeders are Type I; insects and parasitoids, Type II; vertebrates, Type III), and his classification is commonly employed by

theoretical ecologists when incorporating predation into models of population and community dynamics, see [6]. In our work we study the **SIR** model evolved with Holling type II, depending on the case that the treatment fail in a fixed rate $\boldsymbol{\theta}$, in the next sections we illustrate and demonstrate the dynamic of our model equilibrium points such as asymptotically local stability based on Roth Hurwitz criteria , and asymptotically global stability based on Lyapunove theorem supported with .

2. The mathematical model

From the classical simple **SIR** epidemiological model for a set of people with summation equal to N(t) at time t is breaker to three subsets, the susceptible individuals S(t) , infected individuals I(t) and the removable individuals R(t).
Such model can be represented as a system of nonlinear differential equations in follows :

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \frac{\beta SI}{K1+I} - \mu S \\ \frac{dI}{dt} &= \frac{\beta SI}{K1+I} + \theta R - \psi(1-m)I - (\mu+\alpha)I \\ \frac{dR}{dt} &= \psi(1-m)I - (\theta+\mu)R\end{aligned} \qquad \text{……..(1)}$$

Where Λ>0 is the natural birth rate of the population , β>0 is the incidence rate of the susceptible individuals because of parasitic disease transmitted by contact from the individual to the susceptible , ψ>0 is the recovery rate , m is the failure treatment rate such that $(0 \le m \le 1)$, θ>0 is the loosing immunity rate of the recovered individuals , μ >0 natural death rate , α >0 is the disease related death , K1 >0 the half saturation constant.

Therefore at any point at time ***t*** the total number of the individuals is N(t) = S(t)+I(t)+R(t) is clear , according to a biological concepts and meanings of the variables S(t) , I(t) , and R(t) , system (1) has the domain $\mathbb{R}^3_+ = \{(S,I,R) \in \mathbb{R}^3_+ : S \ge 0, I \ge 0, R \ge 0\}$, This is positively invariant for system (1) Clearly, the interaction functions on the right hand side of system (1) are continuously differentiable. In fact they are Liptschizan function on $\mathbb{R}^3_+$, for this the solutions of system (1) exists and unique , in addition all solutions of system (1) with non-negative initial conditions are uniformly bounded as shown in the following theorem .

Theorem 2.1 All the solutions of system (1), which are initiate in $\mathbb{R}^3_+$, are uniformly bounded.

Proof . let (S(t),I(t),R(t)) be any solution of system (1) , with a non-negative initial conditions (S(0),I(0),R(0)) .

Since N(t) = S(t)+I(t)+R(t) then

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

This gives

$$\frac{dN}{dt} = \Lambda - \mu(S+I+R)$$

$$\frac{dN}{dt} = \Lambda - \mu N$$

$$\frac{dN}{dt} \le \Lambda - \mu N$$

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

Therefore $N(t) \leq \frac{\Lambda}{\mu}$, as $t \to \infty$, hence all the solutions of system (1) that initiate in $\mathbb{R}^3_+$ are confined in the region $\Gamma = \{(S, I, R) \in \mathbb{R}^3_+ : N \leq \frac{\Lambda}{\mu}\}$

Which complete the proof .

3. Existence of equilibrium point of system (1)

The system of differential equations (1) has two equilibrium points say E_0, E_1 and Below we will discuss the conditions to be met to prove the existence of each of the points:

1) If I =0 then the system (1) has an equilibrium point called disease free equilibrium point and denoted by $E_0 = (S_0, 0,0)$ where $S_0 = \frac{\Lambda}{\mu}$.
2) If I ≠0 then the system (1) has an equilibrium point called endemic equilibrium point and denoted by $E_1 = (S_1, I_1, R_1)$ where S_1, I_1 *and* R_1 represent the positive solution of the following set of equations :

$$\Lambda - \frac{\beta S_1 I_1}{K_1 + I_1} - \mu S_1 = 0$$

$$\frac{\beta S_1 I_1}{K_1 + I_1} + \theta R_1 - \psi(1 - m)I_1 - (\mu + \alpha)I_1 = 0 \qquad (2)$$

$$\psi(1 - m)I_1 - (\theta + \mu)R_1 = 0$$

Straightforward computation to solve the above system of equations and from Eqs.(1) and (3) of system (2) gives that:

$$S_1 = \frac{\Lambda(K_1 + I_1)}{\beta I_1 + \mu(K_1 + I_1)} \ ,$$

$$R_1 = \frac{\psi(1-m)I_1}{(\theta + \mu)}$$

Now substituting S_1 and R_1 in Eq. (2) we get I_1 which it is a positive root for the following equation :

$$D_1 I^4 + D_2 I^3 + D_3 I^2 + D_4 I + D_5 = 0 \qquad (3)$$

Here

$$D_1 = -[\,\psi(1 - m)(\theta + \mu)(\beta + \mu) + \mu(\theta + \mu)(\beta + \mu) + \alpha(\theta + \mu)(\beta + \mu)]$$

$$D_2 = \Lambda\beta(\theta + \mu) - \psi K_1(1 - m)(\theta + \mu)(\theta + 2\mu) - \mu K_1(\theta + \mu)(\beta + 2\mu) - \alpha K_1(\theta + \mu)(\beta + 2\mu)$$

$$D_3 = \Lambda\beta K_1(\theta + \mu) + \theta\psi(1 - m)(\beta + \mu) - \psi\mu K_1^2(1 - m)(\theta + \mu) - \mu^2 K_1^2(\theta + \mu) - \alpha\mu K_1^2(\theta + \mu)$$

$$D_4 = \theta\psi K_1(1 - m)(\beta + 2\mu)$$

$$D_5 = \theta\psi\mu K_1^2(1 - m)$$

Clearly by used Descartes rule then Eq. (3) has a unique positive root given by I_1 if and only if one of the bellow conditions satisfied :

$\beta < K_1(\beta + 2\mu)(\psi(1-m) + \mu + \alpha)$ … …(1a)

$(\Lambda\beta K_1 + \psi\theta(1-m)) > \mu K_1^2(\psi(1-m) + \mu + \alpha)$ ……..(2a)

4.Local stability of system (1)

In this section the local stability analysis of the equilibrium points E_0 , E_1 of system (1) studied as shown in the following theorems .

Theorem 4.1: The disease free equilibrium point $E_0 = (S_0, 0,0)$ of system (1) is locally asymptotically stable provided that : $\frac{\beta S_0}{K_1} < \psi(1-m) + \mu + \alpha$ … … ….(3a)

Proof . The Jacobin matrix of system (1) at E_0 can be written as :

$$J(E_0) = \begin{bmatrix} -\mu & -\beta K_1 S_0 & 0 \\ 0 & \frac{\beta S_0}{K_1} - \psi(1-m) - \mu - \alpha & 0 \\ 0 & \psi(1-m) & -(\theta + \mu) \end{bmatrix}$$

we get the characteristic Eq. of $J(E_0)$ can be written by :

$$\left(\left(\frac{\beta S_0}{K_0} - (\psi(1-m) + \mu + \alpha)\right) - \lambda\right)(\lambda^2 - T\lambda + D) = 0$$

Where T (trace) = $-(2\mu + \theta)$, D (determinant) = $\mu(\theta + \mu)$

$\lambda_{SR} = -\frac{T}{2} \mp \frac{\theta}{2}$, $D = 0$

$\lambda_I = \frac{\beta S_0}{K_1} - (\psi(1-m) + \mu + \alpha)$

On the other hand E_0 is saddle node if $\frac{\beta S_0}{K_1} > \psi(1-m) + \mu + \boldsymbol{\alpha}$.

Proof complete

Theorem 4.2. If the endemic equilibrium point $E_1 = (S_1, I_1, R_1)$ of system (1) exist then it is locally asymptotically stable provided that : $K_1\beta S_1 < (\mu + m)(K_1 + I_1)^2$ …….. (4a)

Proof . The Jacobin matrix of system (1) at the endemic equilibrium point E_1 can be written as:

$$J(E_1) = \begin{bmatrix} \frac{-\beta I_1}{K_1 + I_1} - \mu & \frac{-K_1\beta S_1}{(K_1 + I_1)^2} & 0 \\ \frac{\beta I_1}{K_1 + I_1} & \frac{K_1\beta S_1}{(K_1 + I_1)^2} - \mu - \alpha & 0 \\ 0 & 0 & -(\theta + \mu) \end{bmatrix} = [b_{ij}]_{3\times 3}$$

The characteristic equation of Jacobin matrix is given by :

$\lambda^3 + \Omega_1\lambda^2 + \Omega_2\lambda + \Omega_3 = 0$

here $\Omega_1 = -(b_{11} + b_{12} + b_{33})$

Then $\Omega_1 > 0$ iff $K_1\beta S_1 < (\mu + \alpha)(K_1 + I_1)^2$

i.e. condition (3a) hold .

$\Omega_2 = b_{11}b_{21} - b_{12}b_{21} + b_{11}b_{33} + b_{22}b_{33}$

$\Omega_3 = -[b_{11}b_{22}b_{33} - b_{33}b_{12}b_{21}]$

We get $\Omega_3 > 0$ iff condition (3a) satisfied , now to check $\Delta = \Omega_1\Omega_2 - \Omega_3$

$$\Delta = -(b_{11} + b_{33})(b_{22}^2 + b_{33}^2) + b_{12}b_{21}(b_{11} + b_{22}) - b_{11}^2(b_{22} + b_{33}) - 2b_{11}b_{22}b_{33}$$

Clearly Δ is positive if the condition (4a) satisfied , proof complete .

5. Global stability analysis of system (1):

In this section, the global dynamics of system (1) is studied with the help of Lyapunov function as shown in the following theorems :

Theorem 5.1. Assume that the disease free equilibrium point E_0 of system (1) is locally asymptotically stable , then the basin of attraction of E_0 , say $B(E_0) \subset \mathbb{R}_+^3$, satisfy the following condition :

$\frac{\mu}{S}(S - S_0)^2 + (\mu + \alpha)I + \mu R > \frac{\beta S_0 I}{K_1 + I}$ .....(5a)

Proof . consider the following positive function :

$$W_1 = \left(S - S_0 - S_0 \ln\left(\frac{S}{S_0}\right)\right) + I + R$$

$\frac{dW_1}{dt} = (\frac{S - S_0}{S})\frac{ds}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$

$$\frac{dW_1}{dt} = -\frac{\mu}{S}(S - S_0) - (\mu + \alpha) - \mu R + \frac{\beta S_0 I}{K_1 + I}$$

It is clear that $\frac{dW_1}{dt} \leq 0$, for every initial points and then W_1 is a Lyapunov function provided that condition (5a) hold. Thus $E0$ is globally asymptotically stable in the interior of $B(E0)$ which means that $B(E0)$ is the basin of attraction and that complete the proof.

Theorem 5.2. Let the endemic equilibrium point (E_1) of system (1) is locally asymptotically stable , then the basin of attraction of E_1 , say $B(E_1) \subset \mathbb{R}_+^3$, satisfy the following conditions :

$$\psi(1 - m) + \mu + \alpha > \frac{\beta}{I + K_1}\left(S + \frac{K_1 S_1}{K_{1+I}}\right) \ldots\ldots\ldots (6a)$$

$$\big(\theta + \psi(1 - m)\big)(I - I_1) < (\theta + \mu)(R - R_1) \ldots\ldots\ldots (6b)$$

$$2\mu(S - S_1) > \frac{\beta}{K_1 + I}\left(S + \frac{K_1 S_1}{K_1 + I_1}\right)(I - I_1) \ldots\ldots\ldots (6c)$$

Proof. Conceder the following positive definite function :

$$w_2 = \frac{(S-S_1)^2}{2} + \frac{(I-I_1)^2}{2} + \frac{(R-R_1)^2}{2}$$

$$\frac{dW_2}{dt} = (S-S_1)\frac{dS}{dt} + (I-I_1)\frac{dI}{dt} + (R-R_1)\frac{dR}{dt}$$

By simplifying this equation we get:

$$\frac{dW_2}{dt} = (S-S_1)q11 + (I-I_1)q22 + (R-R_1)q33$$

With :

$$q_{11} = -\mu(S-S_1) - \frac{\beta SI}{K_1+I} + \frac{\beta SI_1}{K_1+I} - \frac{\beta S_1 I}{K_1+I} + \frac{\beta S_1 I_1}{K_1+I_1}$$

$$q_{22} = \theta(R-R_1) - \psi(1-m)(I-I_1) - (\mu+\alpha)(I-I_1) + \frac{\beta SI}{K_1+I} + \frac{\beta SI_1}{K_1+I} - \frac{\beta S_1 I}{K_1+I} - \frac{\beta S_1 I_1}{K_1+I_1}$$

$$q_{33} = \psi(1-m)(I-I_1) - (\mu+\alpha)(R-R_1)$$

It is clear that $\frac{dW_2}{dt} \leq 0$, for every initial points and then W_2 is a Lyapunov function provided that condition (6a , 6b , 6c) holds. Thus E_1 is globally asymptotically stable in the interior of $B(E_1)$ which means that $B(E_1)$ is the basin of attraction and that complete the proof.

6. Numerical simulation

In this section, the global dynamics of system (1) is studied numerically .the objective of this study are confirming our obtained analytical results and understand the effect of treatment and the range of its failure against the disease on the dynamics of SIR epidemic model . For the following set of hypothetical, biologically feasible, set of parameters, definitely different set of hypothetical parameters can be chosen also, The system (1) is solved numerically starting in different three initial conditions and (3500,2000,1000) ,(2000,1500.1500) and (500,500,500) and different parameters

Λ=500 , β=0.000001, μ=0.1,$K_1 = 0.3$,ψ= 0.1,m = 0.2 ,α =0.01, θ=0.1 as illustrated in (fig.1) shows the existence of a unique disease free equilibrium point of system (1) which is locally asymptotically stable , while with β= 0.2 i.e. *incidence rate is higher* and Λ=500 , μ=0.1,$K_1 = 0.3$,ψ= 0.1,m = 0.2 here in (fig.2) shows the existence of unique endemic equilibrium point for system (1) which is locally asymptotically stable .

It is obvious from time series trajectories the blue solid line refers to susceptible individuals , the green solid line refers to infected individuals and the red solid line refers to recovered one, in (fig.3 a,b,c) that when the *recovery rate* $\boldsymbol{\psi}$ increases from 0.1 to 0.6 the endemic equilibrium point of system (1) becomes stable point and the trajectory of susceptible individuals increases and the number of the infected individuals decreases while the recovery individuals stay fixed . In (fig. 4 a,b,c) when the *failure rate* ***m*** increases from 0.2 to 1 the disease resistance become at the top stage and the treatment would fail absolutely , therefore the number of susceptible not effect and the recovered individuals decrease while the infected individuals increase .

In (fig.5 a,b,c) when $\boldsymbol{\alpha}$ the *disease related death rate* increase from 0.01 to 1.5 the number of the infected and recovered individuals decrease while the susceptible individuals increase . finally as $\boldsymbol{\theta}$ the *loosing immunity rate* increase from 0.1 to 0.9 the susceptible individuals stay fixed in the other hand the number of infected individuals increase and recovered decrease as shown in (fig.6 a,b,c) .

7. conclusion and discussions

In this paper we study the dynamical behavior of epidemic model **SIR** Holling type II in system (1)when the treatment rate ***m*** become failed against the disease with the loosing of immunity because of it .

The model consists of three non-linear autonomous differential equations that describe the dynamics of three different population's namely first susceptible (S), second infected (I) and recovered (R) . In order to confirm

our analytical results and understand the effect of m in system (1) that has been studied numerically with different set of parameters and the following observation are made:

i. For the three initial conditionse system (1) as we see in fig.1when the treatment rate $m \simeq 0$ the free equilibrium point $\simeq$ (5000,0,0) asymptotically stable point .

ii. When the treatment failed rate m increase to 0.2 and the same three initial conditions in (i) system (1) has endemic equilibrium point $\simeq$ (1750,2250,1000) which it is asymptotically stable point ,as we see in fig.2 this mean the resistance against the disease became so week and this is consistent with the desired objective to research .

iii. In fig.3 (a,b,c) the time series trajectories is observed , when the *recovery rate* ψ increases from 0.1 to 0.6 the endemic equilibrium point of system (1) becomes stable point and the trajectory of susceptible individuals increases and the number of the infected individuals decreases while the recovery individuals stay fixed .

iv. In fig.4 (a,b,c) the time series trajectories is observed , when the *failure rate* m increases from 0.2 to 1 the disease resistance become at the top stage and the treatment would fail absolutely , therefore the number of susceptible not effect and the recovered individuals decrease while the infected individuals increase .

v. In fig.5 (a,b,c) the time series trajectories is observed , when α the *disease related death rate* increase from 0.01 to 1.5 the number of the infected and recovered individuals decrease while the susceptible individuals increase .

vi. Finally in fig.6 (a,b,c) the time series trajectories is observed , as θ the *loosing immunity rate* increase from 0.1 to 0.9 the susceptible individuals stay fixed in the other hand the number of infected individuals increase and recovered decrease .

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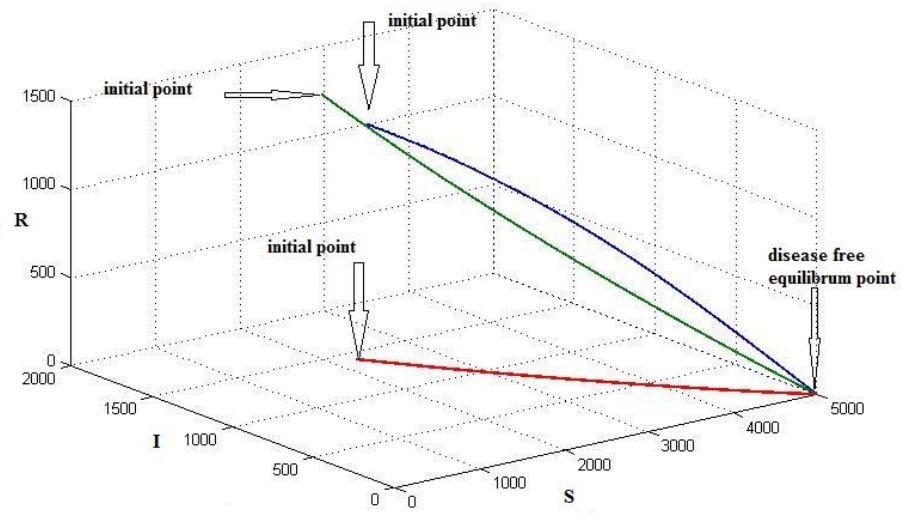


Fig. 1 Disease free equilibrium point of system (1) with Λ=500 , β=0.000001, μ=0.1,$K_1 = 0.3$,ψ= 0.1,m = 0.2 ,α =0.01, θ=0.1and the initial conditions are (3500,2000,1000) ,(2000,1500.1500) and (500,500,500) .

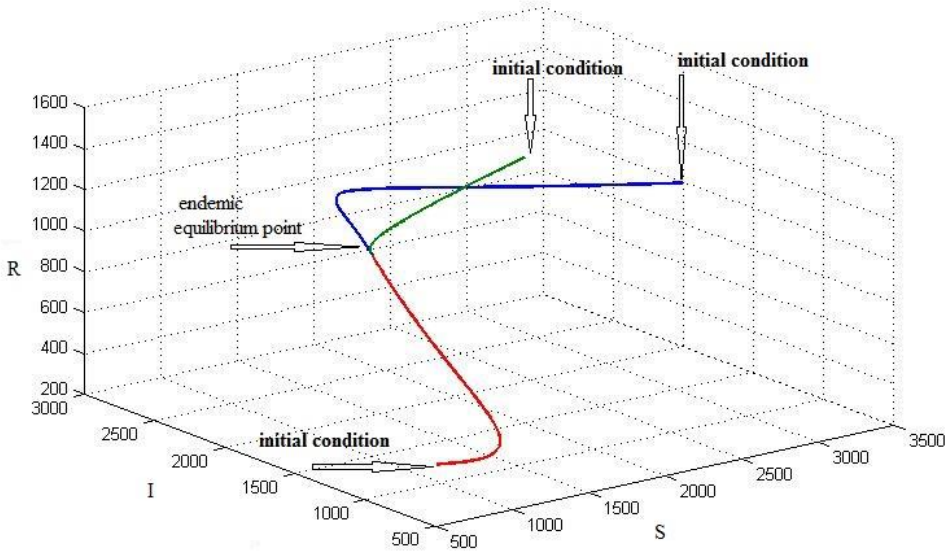
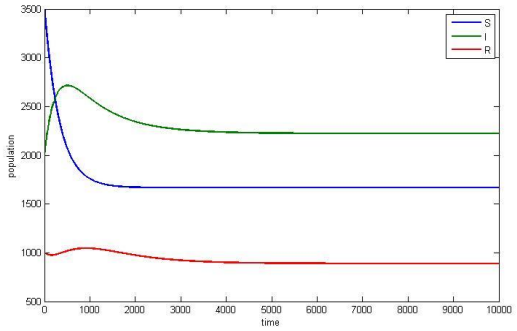
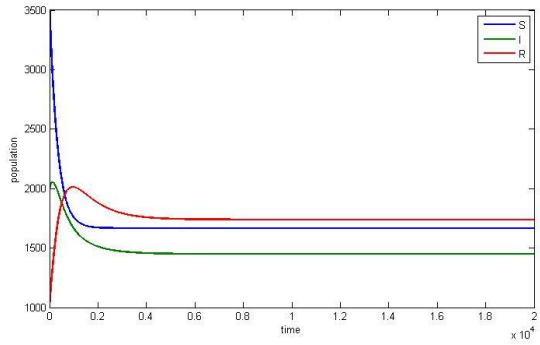


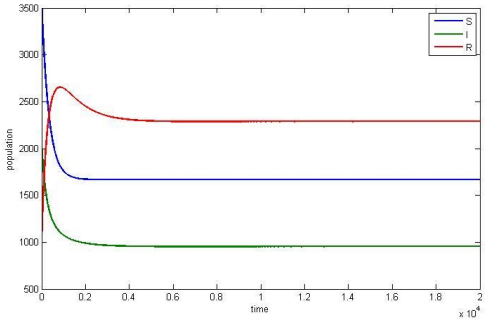
Fig.2 Endemic equilibrium point for system (1) with Λ=500 , μ=0.1,$K_1 = 0.3$,ψ= 0.1,m = 0.2 ,α =0.01, θ=0.1 but β= 0.2 i.e. incidence rate is higher and the initial conditions (3500,2000,1000) ,(2000,1500.1500) and (500,500,500) .



(a) $\psi = 0.1$

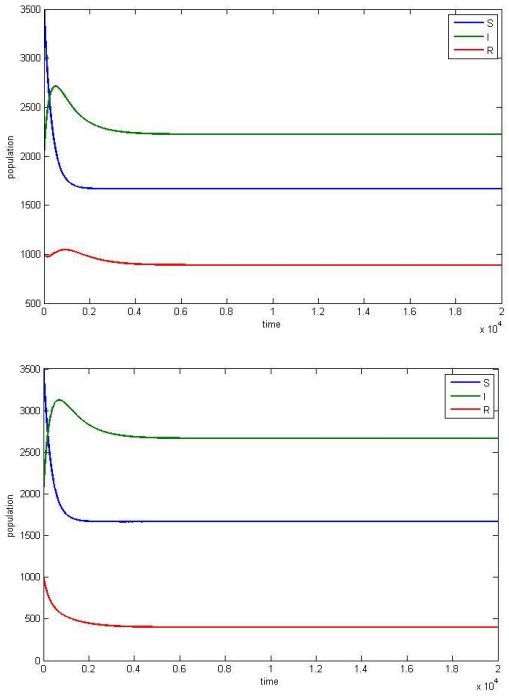


(b) $\psi = 0.3$



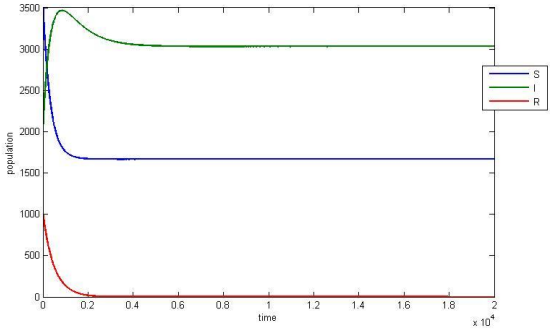
(c) $\psi = 0.6$

Fig. 3 Time series of system (1) when ψ increase from 0.1 to 0.6



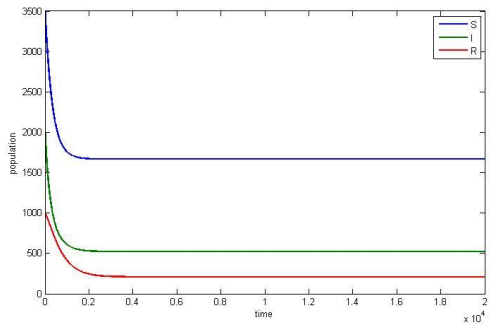
(a) ***m =0.2***

(b) ***m =0.7***

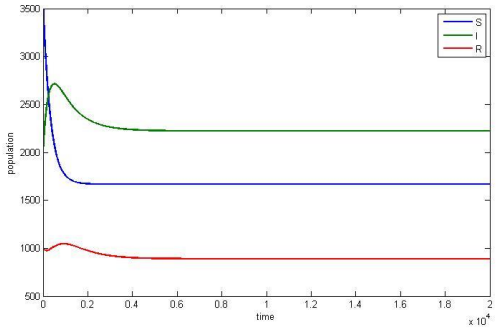


(c) ***m =1***

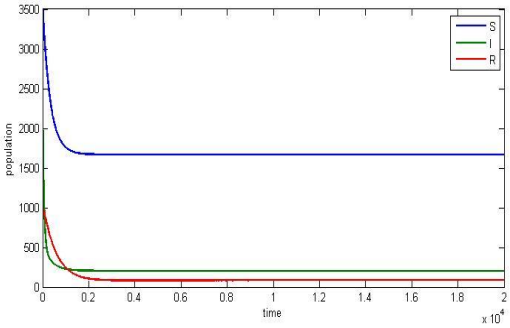
Fig. 4 Time series of system (1) when ***m*** increase from 0.2 to 1 .



(a) α = 0.01

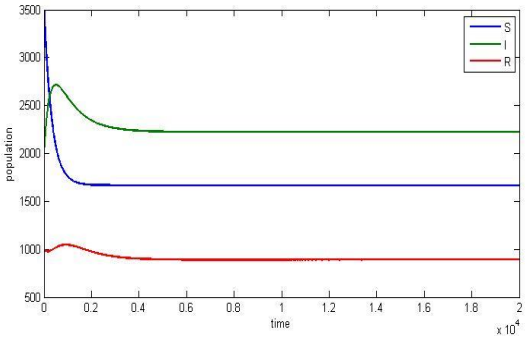


(b) α = 0.5

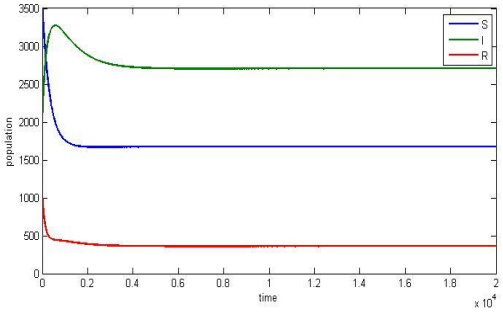


(c) α = 1.5

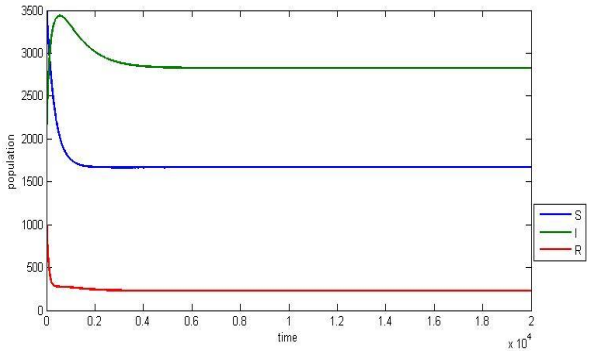
Fig . 5 Time series of system (1) when α increase from 0.01 to 1.5 .



(a) $\theta = 0.1$



(b) $\theta = 0.5$



(c) θ =0.9

Fig . 6 Time series of system (1) when θ increase from 0.1 to 0.9