Global Dynamics of an SIQR Epidemic Model With Specific Non-Linear Incidence Rate

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Abstract

In this paper, an $SIQR$ (Susceptible, Infected, Quarantined, Recovered) epidemic model with a specific non linear incidence rate function is proposed and the dynamics of this model are analyzed by both theoretical and numerical means. At last, some numerical reproductions are introduced to represent the examination results.

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1 Introduction

Mathematical models have become important tools in examining the dissemination and control of infectious diseases. Furthermore, mathematical models now play a key role in policy making, including emergency planning and risk assessment, health economic aspects, control-program evaluation and optimizing various discoveries. Modeling in the field of epidemiology has had its roots in the early twentieth century. Over the most recent couple of years, people have designed different epidemiological models (SIR, $SIER$, $SIERS$, $SIQR$, $SEIV$ and so on.) with various incidence rate to control the spread of diseases [1,2,4,9,12,15,16,19,20,21].

Isolate (Quarantine) is the most immediate control technique for the spread of infectious disease. Mathematical models have been used to study their impact on the dynamics of infectious diseases under isolation and quarantine (I and Q) in order to test the effectiveness of various scenarios (strategies) on the prevention or amelioration of the spread of highly contagious diseases [3,6,8,10,14]. The implementation of quarantine has been assumed to be perfect in the sense that isolated people have been totally separated from the rest of the population, that is, all contacts between isolated infected and susceptible people have been eliminated

with some of these models capable of supporting sustained oscillatory outbreaks. In addition, extensions of the $SIQR$ model that purposely incorporate a class A of asymptomatic people have also been studied by [5,18].

In this work, we introduce and analyze a model that incorporates isolation, under the assumption that only a fraction of individuals in the Q compartment manages to stay totally isolated from the rest of the population. Our objective of this paper is to consider a $SIQR$ model with a non linear Crowley-Martin incidence rate $\frac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)}$ which can be used to interpret the case of varicella (chickenpox) dynamics. Here β, α_1 and α_2 are positive parameters that describe the effects of contact rate, social awareness rate among susceptible and magnitude of interference among infective population, respectively, on the incidence rate.

This manuscript is organized as follows: In ***Sect.*2**, $SIQR$ model is presented. In ***Sect.*3**, basic properties of solutions are discussed. In ***Sect.*4**, we calculate the basic reproduction number then in ***Sect.*5**, we determine all possible equilibria of model. In ***Sect.*6**, we discuss and analyze the local stability of the equilibriums. In ***Sect.*7**, we discuss and analyze the global stability of the equilibriums. We present in ***Sect.*8**, some numerical examples of the dynamics of the model. Finally, in ***Sect.*9**, we discussed the conclusion.

2 Model Formulation

In this section, we formulate a new $SIQR$ epidemic model based on the nonlinear Crowley-Martin incidence rate [7] which can be used to interpret the case of varicella (chickenpox) dynamics. Thus, the resulting model is given as follows:

$$\begin{cases} \dfrac{dS}{dt} = A - dS - \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} \\ \dfrac{dI}{dt} = \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} - (\gamma + \delta + d + d_1)I \\ \dfrac{dQ}{dt} = \delta I - (\mu + d + d_2)Q \\ \dfrac{dR}{dt} = \gamma I + \mu Q - dR \end{cases} \tag{2.1}$$

whose state space is the first quadrant $R_4^+ = \big\{(S,\ I,\ Q,\ R) : S \geq 0,\ I \geq 0,\ Q \geq 0,\ R \geq 0\big\}$ and subject to the initial conditions $S(0) = S_0 \geq 0$, $I(0) = I_0 \geq 0$, $Q(0) = Q_0 \geq 0$, $R(0) = R_0 \geq 0$. It is assumed that all the parameters are positive. From the model, the parameters can be summarized in the following list:
A is the recruitment rate of the population, β is the contact rate due to infected individuals, d is the natural death rate of the population, d_1 is the disease caused mortality of quarantined individuals, d_2 is the total population size at time t, γ is the rate at which individuals recover from compartment I and move to compartment R, β is the contact rate due to infected individuals, δ and μ are the removal rate constants from the compartments Q and R respectively, α_1 and α_2 are the inhibition effect due to susceptible and infected population.

3 Basic Properties of the Model

Summing up the four equations of model (2.1) and denoting

$$N(t) = S(t) + I(t) + Q(t) + R(t),$$

having $N'(t) = A - dN - d_1 I - d_2 Q \leq A - dN$. If disease is not present, then $N'(t) = A - dN$. This shows that population size $N \to \frac{A}{d}$ as $t \to \infty$. It follows that the solutions of model (2.1) exists in the region defined by

$$\Omega = \left\{ (S,\ I,\ Q,\ R) \in R_4^+ : S,\ I,\ Q,\ R \geq 0,\ S + I + Q + R \leq \frac{A}{d} \right\}. \tag{3.1}$$

This gives the following lemma which shows that the solutions of model (2.1) are bounded, continuous for all positive time and lie in a compact set.

Lemma 3.1. The set Ω defined in (3.1) is a positively invariant region for model (2.1). Moreover, every trajectory of model (2.1) is eventually staying in a compact subset of Ω.

4 Basic Reproductive Number

The basic reproduction number, sometimes called basic reproductive rate or basic reproductive ratio, is one of the most useful threshold parameters which characterize mathematical problems concerning infectious diseases. This metric is useful because it helps determine whether or not an infectious disease will spread through a population. In this section, we will calculate the basic reproduction number R_0 of model (2.1) by using the next-generation matrix method described in [17]. For that, we rewrite model (2.1) as $\frac{dx}{dt} = \mathbb{F}(x) - \mathfrak{A}(x)$, where $x = (I, Q, R, S)$

$$\mathbb{F}(x) = \begin{pmatrix} \frac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} \\ 0 \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathfrak{A}(x) = \begin{pmatrix} (\gamma + \delta + d + d_1)I \\ -\delta I + (\mu + d + d_2)Q \\ -\gamma I - \mu Q + dR \\ -A + dS + \frac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} \end{pmatrix}.$$

We calculate the Jacobian matrices for $\mathbb{F}(x)$ and $\mathfrak{A}(x)$ at the disease-free equilibrium $x_0 = (0, 0, 0, A/d\ )$ and denoted by:

$$\mathbf{F} = \begin{pmatrix} \frac{\beta A}{A\alpha_1 + d} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \mathbf{V} = \begin{pmatrix} (\gamma + \delta + d + d_1) & 0 & 0 & 0 \\ -\delta & (\mu + d + d_2) & 0 & 0 \\ -\gamma & -\mu & d & 0 \\ \frac{\beta A}{A\alpha_1 + d} & 0 & 0 & d \end{pmatrix}$$

$\mathbf{FV^{-1}}$ is the next generation matrix for model (2.1). It then follows that the spectral radius of matrix $\mathbf{FV^{-1}}$ is $\rho(\mathbf{FV^{-1}}) = \frac{A\beta}{(A\alpha_1 + d)(\gamma + \delta + d + d_1)}$. Thus, the basic reproduction number of model (2.1) is $R_0 = \frac{A\beta}{(A\alpha_1 + d)(\gamma + \delta + d + d_1)}$.

4.1 Sensitivity analysis of basic reproductive number

The value R_0 is the average number of secondary infections produced by one infected individual during the mean period of infection in a fully susceptible population. It means the average new infections caused by a single infected individual

in a whole susceptible population. The basic reproduction number R_0 depends on six parameters β, α_1, γ, δ, d and d_1. Among those parameters, we cannot control the natural death rate of population d. Therefore, to examine the sensitivity of R_0 to the parameters β, α_1, γ, δ and d_1, normalized forward sensitivity index with respect to each of those parameters are computed as follows:
$\Gamma_{\beta}^{R_0} = \frac{\beta}{R_0}\frac{\partial R_0}{\partial \beta} = \left\{\frac{\beta(A\alpha_1+d)(\gamma+\delta+d+d_1)}{A\beta}\right\}\left\{\frac{A}{(A\alpha_1+d)(\gamma+\delta+d+d_1)}\right\} = 1$, $\Gamma_{\alpha_1}^{R_0} = \frac{-\alpha_1 A}{(A\alpha_1+d)} \Rightarrow \left|\Gamma_{\alpha_1}^{R_0}\right| < 1$, $\Gamma_{\gamma}^{R_0} = \frac{-\gamma}{(\gamma+\delta+d+d_1)} \Rightarrow \left|\Gamma_{\gamma}^{R_0}\right| < 1$, $\Gamma_{\delta}^{R_0} = \frac{-\delta}{(\gamma+\delta+d+d_1)} \Rightarrow \left|\Gamma_{\delta}^{R_0}\right| < 1$ and $\Gamma_{d_1}^{R_0} = \frac{-d_1}{(\gamma+\delta+d+d_1)} \Rightarrow \left|\Gamma_{d_1}^{R_0}\right| < 1$. From the above discussion, it is clear that the basic reproduction number R_0 is most sensitive to changes in β. If β will increase R_0 will increase in the same proportion and if β will decrease R_0 will also decrease in the same proportion. On the other hand α_1, γ, δ and d_1 have an inversely proportional relationship with R_0, i.e., an increase in any of them will cause a decrease in R_0 and a decrease in any of them will cause an increase in R_0.

5 Existence of Equilibria

In this section, we obtain the existence of the disease-free equilibrium E_0 and the endemic equilibrium E^* of model (2.1). Set the right sides of model (2.1) equal zero, that is,

$$\begin{cases} A - dS - \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} = 0 \\ \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} - (\gamma+\delta+d+d_1)I = 0 \\ \delta I - (\mu+d+d_2)Q = 0 \\ \gamma I + \mu Q - dR = 0 \end{cases} \tag{5.1}$$

The model (2.1) always has the disease-free equilibrium point $E_0(A/d\ , 0, 0, 0)$. Solving (5.1) we also get a unique positive, endemic equilibrium point $E^*(S^*, I^*, Q^*, R^*)$ of the model (2.1), where $S^* = \frac{(\gamma+\delta+d+d_1)(1+\alpha_2 I^*)}{\beta-\alpha_1(\gamma+\delta+d+d_1)(1+\alpha_2 I^*)}$, $Q^* = \frac{\delta I^*}{\mu+d+d_2}$, $R^* = \frac{\gamma(\mu+d+d_2)+\mu\delta}{d(\mu+d+d_2)} I^*$, and I^* is given as a root of the quadratic equation $\Omega_1 I^2 + \Omega_2 I + \Omega_3 = 0$, where,

$$\Omega_1 = [\alpha_1\alpha_2\beta(\gamma+\delta+d+d_1)^2],$$

$$\Omega_2 = \beta[\alpha_1(\gamma+\delta+d+d_1) - \alpha_2(A\alpha_1+d) - \beta\}](\gamma+\delta+d+d_1),$$

$$\Omega_3 = [A\beta^2 - \beta(A\alpha_1+d)(\gamma+\delta+d+d_1)].$$

Now,

$$I^* = \frac{-\beta(\gamma+\delta+d+d_1)[\alpha_1(\gamma+\delta+d+d_1) - \alpha_2(A\alpha_1+d) - \beta] + \sqrt{\Delta}}{2\alpha_1\alpha_2\beta(\gamma+\delta+d+d_1)^2}$$

where,

(5.1)
$\Delta^2 = \beta^2(\gamma+\delta+d+d_1)^2[\alpha_1(\gamma+\delta+d+d_1)-\alpha_2(A\alpha_1+d)-\beta]^2 - 4\alpha_1\alpha_2\beta^2(\gamma+\delta+d+d_1)^3$
(5.2)
$(A\alpha_1+d)[R_0-1]$
(5.3)

6 Local Stability Analysis

In this section, we study the local stability of the disease-free equilibrium E_0 and the endemic equilibrium E^* of model (2.1).

Theorem 6.1. If $R_0 < 1$, the disease-free equilibrium E_0 of model (2.1) is locally asymptotically stable. If $R_0 > 1$, the disease-free equilibrium E_0 is unstable.

Proof. The Jacobian matrix of model (2.1) at the disease-free equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -d & \frac{-\beta A}{d+\alpha_1 A} & 0 & 0 \\ 0 & \frac{\beta A}{d+\alpha_1 A} - (\gamma+\delta+d+d_1) & 0 & 0 \\ 0 & \delta & -(\mu+d+d_2) & 0 \\ 0 & \gamma & \mu & -d \end{pmatrix}$$

The characteristic equation of $J(E_0)$ is $(d+\lambda)^2\{\mu+d+d_2+\lambda\}\{\lambda-\frac{\beta A}{d+\alpha_1 A}+(\gamma+\delta+d+d_1)\}=0$.

This equation has the following roots: $\lambda_1=-d$, $\lambda_2=-d$, $\lambda_3=-(\mu+d+d_2)$ and $\lambda_4=\frac{\beta A}{d+\alpha_1 A}-(\gamma+\delta+d+d_1)$, where $\lambda_1,\lambda_2,\lambda_3<0$, while $\lambda_4<0$ for $R_0<1$ and $\lambda_4>0$ for $R_0>1$.

Hence E_0 is locally asymptotically stable for $R_0<1$, while it is unstable for $R_0>1$.

Theorem 6.2. If $R_0>1$, the endemic equilibrium E^* of model (2.1) is locally asymptotically stable.

Proof. Consider

$$J(E^*) = \begin{pmatrix} -V_1-d & -V_2 & 0 & 0 \\ V_1 & V_2-(\gamma+\delta+d+d_1) & 0 & 0 \\ 0 & \delta & -(\mu+d+d_2) & 0 \\ 0 & \gamma & \mu & -d \end{pmatrix}$$

where,

$$V_1 = \frac{\beta I^*}{(1+\alpha_1 S^*)(1+\alpha_2 I^*)} - \frac{\beta S^* I^*(\alpha_1+\alpha_1\alpha_2 I^*)}{[(1+\alpha_1 S^*)(1+\alpha_2 I^*)]^2}$$

$$V_2 = \frac{\beta S^*}{(1+\alpha_1 S^*)(1+\alpha_2 I^*)} - \frac{\beta S^* I^*(\alpha_2+\alpha_1\alpha_2 S^*)}{[(1+\alpha_1 S^*)(1+\alpha_2 I^*)]^2}$$

The characteristic equation of $J(E^*)$ is

$$(d+\lambda)(\mu+d+d_2+\lambda)\{\lambda^2+\lambda(V_1-V_2+2d+\gamma+\delta+d_1)+V_1(\gamma+\delta+d+d_1)+ d(\gamma+\delta+d+d_1-V_2)\}=0$$

Clearly, the two eigenvalues have strictly negative real part other two eigenvalues are given by the quadratic equation

$$\lambda^2+\lambda(V_1+d+\gamma+\delta+d+d_1-V_2)+V_1(\gamma+\delta+d+d_1)+d(\gamma+\delta+d+d_1-V_2)=0$$

or $\lambda^2+\lambda a_1+a_2=0$
where $a_1=(V_1+d)+(\gamma+\delta+d+d_1-V_2)$, $a_2=V_1(\gamma+\delta+d+d_1)+d(\gamma+\delta+d+d_1-V_2)$
By Routh-Hurwitz criteria, we know that the model is stable if $a_1,a_2>0$ and unstable if $a_1,a_2<0$. We obtain $\gamma+\delta+d+d_1>V_2$. Thus all eigenvalues have negative real parts and hence model (2.1) is locally asymptotically stable at endemic equilibrium E^* if $R_0>1$.

7.Global Stability Analysis

In this section, we study the global stability of the disease-free equilibrium E_0 and the endemic equilibrium E^* of model (2.1).

Theorem 7.1. If $R_0<1$, the disease-free equilibrium E_0 of model (2.1) is globally asymptotically stable.

Proof. We prove the global stability of the model (2.1) at the equilibrium E_0 when $R_0<1$. Taking the Lyapunov function

$$V(t)=I(t)$$

Calculating the derivative of $V(t)$ along the positive solution of model (2.1), it follows that

$$\dot{V}(t)=\frac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)}-(\gamma+\delta+d+d_1)I$$

Since the incidence function $\frac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)}\leq\frac{\frac{\beta IA}{d}}{(1+\frac{\alpha_1\beta IA}{d})(1+\alpha_2 I)}=\frac{\beta IA}{(\alpha_1 A+d)(1+\alpha_2 I)}$ for $0\leq S\leq\frac{A}{d}$.

$$\dot{V}(t)\leq\left[\frac{A\beta}{(A\alpha_1+d)}-(\gamma+\delta+d+d_1)\right]I$$

$$=(\gamma+\delta+d+d_1)\left[R_0-1\right]I\leq 0$$

Furthermore, $\dot{V}=0$ only if $I=0$, so the largest invariant set contained in $\{(S,\ I,\ Q,\ R)\in\Omega:\dot{V}=0\}$ is the plane $I=0$. By Lassalle's invariance principle [13], this implies that all solution in Ω approach the plane $I=0$ as $t\to\infty$. On the other hand, solutions of (2.1) contained in such plane satisfy $\frac{dS}{dt}=A-dS$, $\frac{dQ}{dt}=-(\mu+d+d_2)Q$, $\frac{dR}{dt}=\mu Q-dR$, which implies that $S\to\frac{A}{d}$ and $Q\to 0$, $R\to 0$ as $t\to\infty$, that is, all of these solutions approach E_0 is globally asymptotically stable in Ω.

Then, we obtain the limit equations of model (2.1)

$$\begin{cases} \dfrac{dS}{dt} = A - dS - \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} \\ \dfrac{dQ}{dt} = \delta I - (\mu + d + d_2)Q \\ \dfrac{dR}{dt} = \gamma I + \mu Q - dR \end{cases}$$

So, that disease-free equilibrium E_0 is globally attractive in the region Ω. Therefore, the disease-free equilibrium E_0 of model (2.1) is globally asymptotically stable when $R_0 < 1$.

Theorem 7.2. If $R_0 > 1$, the endemic equilibrium E^* of model (2.1) is globally asymptotically stable.

Proof. Since the front two equations of model (2.1) can be independent, we consider the following model

$$\begin{cases} F_1 = \dfrac{dS}{dt} = A - dS - \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} \\ F_2 = \dfrac{dI}{dt} = \dfrac{\beta SI}{(1+\alpha_1 S)(1+\alpha_2 I)} - (\gamma + \delta + d + d_1)I \end{cases}$$

Take a Dulac function $D = \frac{1}{I}$. Notice that

$$\frac{\partial}{\partial S}(DF_1) + \frac{\partial}{\partial I}(DF_2) = -\frac{d}{I} - \frac{\beta}{(1+\alpha_1 S)^2(1+\alpha_2 I)^2}\left[(1+\alpha_2 I) + \alpha_2 S(1+\alpha_1 S)\right] < 0,$$

if $(1+\alpha_2 I) + \alpha_2 S(1+\alpha_1 S) > 0$.
The conclusion follows.

7 Numerical Simulations

In this section, we will give some numerical examples to illustrate our main results by using Milstein's Higher Order Method [11].

Example 8.1. When we choose the parameters $A = 0.3$, $d = 0.2$, $\alpha_1 = 0.01$, $\alpha_2 = 0.05$, $\delta = 0.2$, $\beta = 0.07$, $\gamma = 0.2$, $\mu = 0.1$, $d_1 = 0.1$, $d_2 = 0.2$ then the basic reproduction number $R_0 = 0.14778325 < 1$. In this case, $S(t)$ approaches to its steady state value while $I(t)$, $Q(t)$ and $R(t)$ approaches to zero as $t \to \infty$. Hence the disease disappears and dies out (***Fig.1***).

Example 8.2. When $A = 1.2$, $d = 0.3$, $\alpha_1 = 0.05$, $\alpha_2 = 0.015$, $\delta = 0.2$, $\beta = 0.65$, $\gamma = 0.02$, $\mu = 0.1$, $d_1 = 0.01$, $d_2 = 0.2$ then the basic reproduction number $R_0 = 4.0880503 > 1$. In this case $S(t)$, $I(t)$, $Q(t)$ and $R(t)$all approaches to their steady state values as $t \to \infty$. Hence the disease becomes endemic (***Fig.2***).

Example 8.3. Keeping all parameters fixed of endemic equilibrium and taking different initial conditions in model (2.1), the phase portrait in SQI-space is

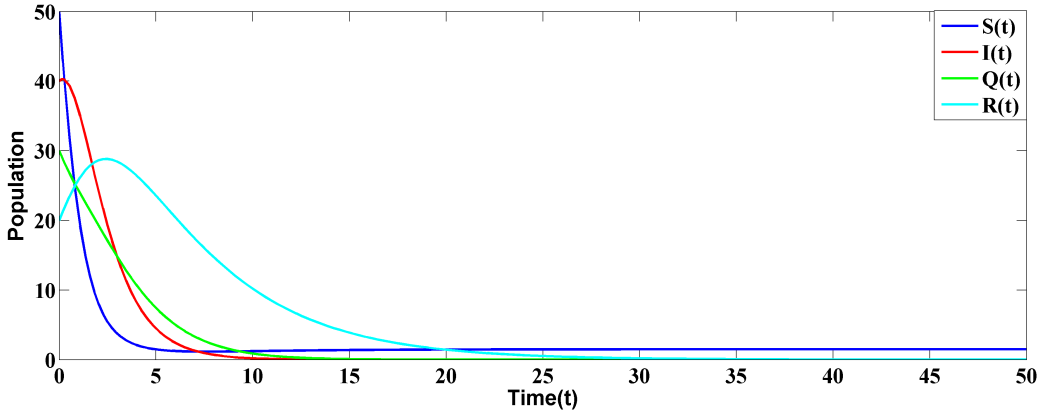

Fig. 1: The figure represents that the disease dies out

displayed. This phase diagram shows that $\lim_{t\to\infty}(S(t), I(t), Q(t)) = (S^*, I^*, Q^*)$ for $R_0 = 4.0880503 > 1$ (***Fig.3***).

8 Discussions and Conclusions

Quarantine has been used to reduce the transmission of diseases for many centuries. In this manuscript, an epidemic $SIQR$ model is proposed and discussed. This model consists of Crowley-Martin type incidence rate. The mathematical analysis shows that the basic reproduction number plays an important role to control the disease. It has been obtained that disease-free equilibrium E_0 is locally and globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Similarly, for the endemic equilibrium E^*, it has been obtained under certain conditions for locally as well as globally asymptotically stable. The phase diagram is demonstrated in ***Fig.3***, at different initial values to validate the global stability. The basic reproduction number R_0 depends on six parameters β, α_1, γ, δ, d and d_1. Among those parameters, we cannot control the natural death rate of population d. Therefore, the sensitivity analysis tells us that efforts to increase prevention are more effective in controlling the spread of disease in population. We leave this model for future work on Coronavirus (COVID-19) infection disease.

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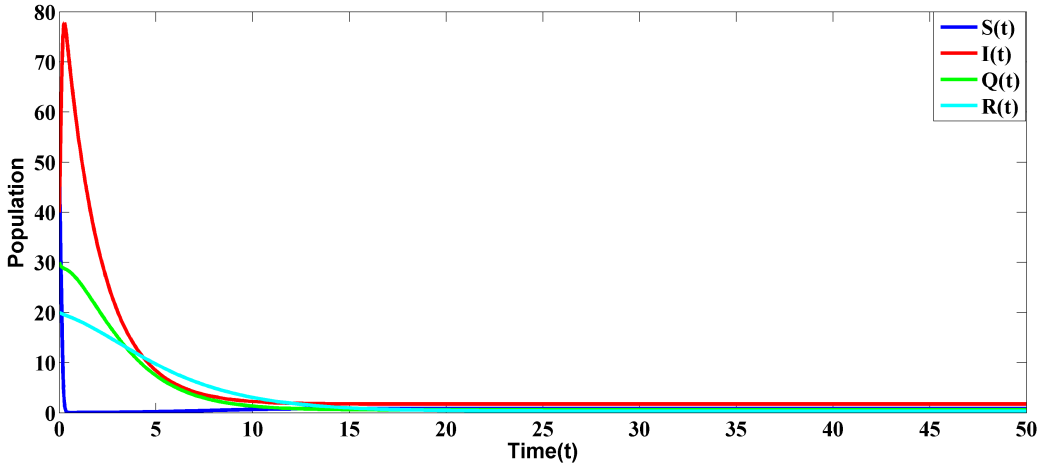

Fig. 2: The figure represents that the disease endemic

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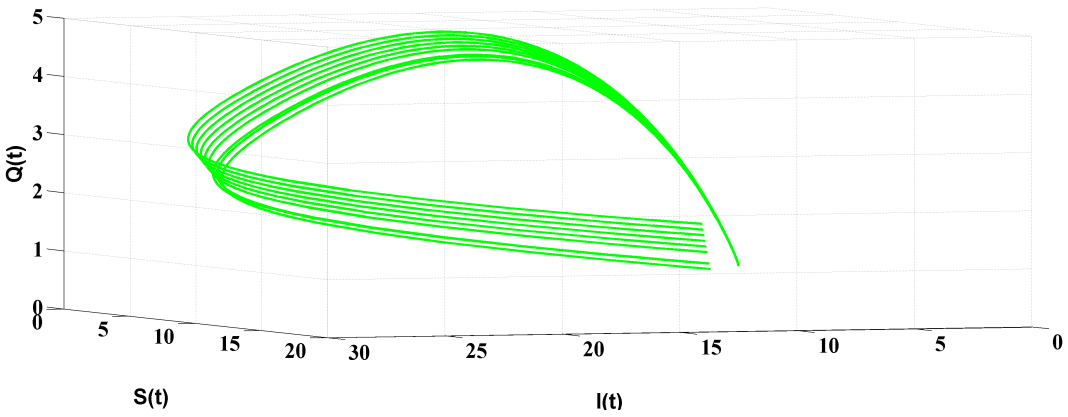

Fig. 3: The phase diagram at different initial values endemic equilibrium

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