A SIQR model with a nonlinear incidence

ALAKES MAITI1, PROSENJIT SEN2 and G. P. SAMANTA3

1Department of Mathematics Vidyasagar Evening College Kolkata – 700006 (India)
2Department of Mathematics Indian Institute of Engineering Science and Technology, Shibpur
Howrah - 711 103, (India)
3Department of Mathematics Indian Institute of Engineering Science and Technology, Shibpur
Howrah - 711 103, (India)
‡Corresponding author Email: g-p-samanta@yahoo.co.uk, gpsamanta@math.iiests.ac.in
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Abstract

In this paper we have studied the dynamical behaviours of an SIQR epidemiological model which takes into account the psychological effect introduced by a nonlinear and non-monotone incidence. Positivity and boundedness of the system have been studied. Stability analysis of the equilibrium points is presented. We have carried out numerical simulations to validate the analytical findings. The biological implications of our analytical and numerical findings are discussed.

Key words : Epidemiology model; Quarantine; Boundedness; Stability.

Mathematics Subject Classification: 34D05, 34C25, 92D30.

1 Introduction

From the time immemorial, diseases have took a fearful toll for human civilization. The reemergence of many diseases and appearance of many new diseases have laid down serious threats to civilization, and a handful of deadly infectious diseases claim millions of lives worldwide every year.

Quarantine has been used as an intervention procedure to control the spread of many infectious diseases over hundred years. The list of such diseases include leprosy, plague, cholera, smallpox, diphtheria, tuberculosis, measles, mumps and many others. Quarantine has also been used for animal diseases such as foot and mouth, psittacosis and rabies6.

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Mathematical modeling of infectious diseases can be traced back to 1760 when Bernoulli used mathematical models for smallpox. The research on infectious diseases, using deterministic mathematical models, actually began in the 20th century. A Nobel laureate physician, Dr. Ross, used a differential equation model to describe the transmissions of malaria between human beings and mosquitoes in 1911, and determined that there exists a threshold of the size of mosquitoes below which the spread of malaria can be controlled. In 1927, Kermack and McKendrick formulated a well-recognized SIR (susceptible-infective-recovered) compartmental model to study the outbreak of Black Death in London during the period of 1665–1666, and the outbreak of plague in Mumbai (Bombay) in 1906. They later, in 1932, formulated an SIS compartmental model and, based on the investigation of this model, formally introduced the concept of thresholds that determines whether a disease spreads in a given population. The Kermack-McKendrick SIR model consists of a system of three coupled nonlinear ordinary differential equations given by

\[
\begin{align*}
    \frac{dS}{dt} &= -\beta SI \\
    \frac{dI}{dt} &= \beta SI - \gamma I \\
    \frac{dR}{dt} &= \gamma I,
\end{align*}
\]

where \( S(t) \) is the number of susceptible individuals, \( I(t) \) is the number of infected individuals, and \( R(t) \) is the number of individuals who have recovered and developed immunity to the infection, \( \beta \) is the infection rate, and \( \gamma \) is the recovery rate. We should note that for the above model, \( S + I + R = \text{constant} \). Thus \( R \) is determined once \( S \) and \( I \) are known, and we can drop the \( R \) equation from the model, leaving the system of two equations:

\[
\begin{align*}
    \frac{dS}{dt} &= -\beta SI \\
    \frac{dI}{dt} &= \beta SI - \gamma I,
\end{align*}
\]

More intensive studies on epidemic dynamics took place after the middle of the 20th century. A remarkable and landmark publication is the book by Bailey. During the past 30 years, a huge number of mathematical models have been formulated to study various infectious diseases (Anderson and May, Murray, Samanta et al., Muntaser et al.). For mathematical works on SIR models, see, and references there in. An SIR model can be modified by introducing a new class \( Q \) of quarantined individuals, who have been removed and isolated either voluntarily or coercive from the infectious class.

The developing countries have increasingly realized the necessity of social consciousness in preventing the diseases. Also different protective measures and policies to reduce infection rate are found to be effective (for real-life evidences, see). That is, for a large number of infective individuals the infection force decreases as the number of infective individuals increases, because in the presence of large number of infective the population tends to reduce the number of contacts per unit time. This phenomenon is termed as psychological effect by Xiao and Ruan. Clearly, the mass-action incidence is incapable of describing such a phenomenon. For this, we need a nonlinear (and also nonmonotone) incidence, which is increasing when number of infectives is small and decreasing when number of infectives is large. Here we have used a nonlinear incidence (originally proposed by Xiao and Ruan), which will incorporate the psychological effect.
In this paper, we have studied the dynamics of a $SIQR$ model with such a nonlinear incidence. The paper is structured as follows. In section 2, we present the mathematical model with basic considerations. In section 3, positivity and boundedness of the model are discussed. The equilibria and their stability are studied in section 4. To illustrate our analytical findings, computer simulations of variety of solutions of the system are performed; and the results are presented in section 5. Section 6 contains the general discussion of the paper and biological significance of our analytical findings.

2 The mathematical model:

First we would like to present a brief sketch of the construction of the model, which will indicate the relevance of the model. Let us divide the total population into four compartments: susceptible, infected, quarantined, and recovered. Let $S$ be the number of individuals in the susceptible class, $I$ the number of individuals who are infectious but not quarantined, $Q$ the number of individuals who are quarantined, and $R$ the number of individuals in the recovered class (with permanent immunity). We make the following assumptions:

1. The infection confers permanent immunity, so that individuals can move from the $I$ and $Q$ classes to the $R$ class.
2. The flow is from the $S$ class to the $I$ class, and then either directly to the $R$ class or to the $Q$ class and then to the $R$ class as shown in Figure 1.

3. Usually, a mass-action incidence $g(I)S$ is considered in epidemic models, where $g(I) = \beta I$ and $\beta$ is the infection rate. It is already mentioned that, as the disease manifests, social awareness (in preventing the diseases) is grown, and different measures are taken against the disease. To incorporate these into the model, we have taken the following nonlinear and nonmotone form for $g(I)\,^{13}$:

$$g(I) = \frac{\beta I}{1 + \alpha I^2},$$

where $\beta I$ measures the infection force of the disease and $1/(1 + \alpha I^2)$ describes the psychological or inhibitory effect from the behavioral change of the susceptible individuals when the number of infective individuals is very large. This is important because the number of effective contacts between infective individuals and susceptible individuals decreases at high infective levels due to the quarantine of infective individuals or due to the protective measures by the susceptible individuals. Here the parameter $\alpha$ measures the psychological or inhibitory effect. The pattern of the function in (2.1) and its dependence on $\alpha$ is shown in Figure 2. Notice that
the case $\alpha = 0$ corresponds to mass-action incidence. The above considerations motivate us to introduce the basic mathematical model under the framework of the following set of nonlinear ordinary differential equations:

$$\frac{dS}{dt} = A - \frac{\beta SI}{1 + \alpha I^2} - dS$$

$$\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha I^2} - (\gamma + \delta + d + \alpha_1)I,$$

$$\frac{dQ}{dt} = \delta I - (\epsilon + d + \alpha_2)Q,$$

$$\frac{dR}{dt} = \gamma I + \epsilon Q - dR.$$  \hspace{1cm} (2.2)

Here the parameter $A$ is the recruitment rate of susceptibles corresponding to births and immigration, $\beta$ is the disease contact rate for the susceptible individuals, $d$ is the per capita natural death rate, and $\alpha$ measures the psychological or inhibitory effect. The constant $\delta$ is the rate constant for individuals leaving the infective compartment $I$ for the quarantine compartment $Q$. $\alpha_1$ and $\alpha_2$ are the disease-related extra death rate constant in compartments $I$ and $Q$, respectively; and $\gamma$ and $\epsilon$ are the removal rate constants from compartments $I$ and $Q$, respectively, to enter into the $R$ compartment. The parameters $A$, $d$ and $\beta$ are positive constants and $\gamma$, $\delta$, $\epsilon$, $\alpha$ are non negative constants.

3 Positivity and boundedness :

In this section, we discuss the positivity and boundedness of the solutions of the model (2.2) which guarantee that the model is biologically well behaved. For positivity of the system (2.2), we have the following theorem.
Theorem 3.1 All solutions of the system (2.2) that start in $\mathbb{R}^4$ remain positive forever.

The proof is simple and therefore it is omitted. The next theorem ensures the boundedness of the system (2.2).

Theorem 3.2 All solutions of the system (2.2) that start in $\mathbb{R}^4$ are uniformly bounded.

Proof. Let $N = S + I + Q + R$. Then we have

$$\frac{dN}{dt} = A - dN - \alpha_1 I - \alpha_2 Q.$$

So, the population size $N$ approaches the carrying capacity $A/d$ when there is no disease. The differential equation for $N$ implies that solutions of (2.2) starting in the positive orthant $\mathbb{R}^4$ either approach, enter, or remain in the subset

$$D = \{(S, I, Q, R) : S \geq 0, I \geq 0, Q \geq 0, R \geq 0, S + I + Q + R \leq A/d\}.$$

4 Equilibrium points and their stability:

First we state the following theorem\textsuperscript{3,6}, which would be very helpful for our analysis of the equilibria of the system (2.2) and the stability.

Lemma 4.1 Consider the following two systems

$$\begin{align*}
\frac{dx}{dt} &= f(t, x), \\
\frac{dy}{dt} &= g(y),
\end{align*}$$

where $x, y \in \mathbb{R}^n$, $f$ and $g$ are continuous, satisfy a local Lipschitz condition in any compact set $X \subset \mathbb{R}^n$, and $f(t, x) \to g(x)$ as $t \to \infty$, so that the second system is the limit system for the first system. Let $\Phi(t, t_0, x_0)$ and $\varphi(t, t_0, y_0)$ be solutions of these systems, respectively. Suppose that $E \in X$ is a locally asymptotically stable equilibrium of the limit system and its attractive region is

$$W(E) = \{y \in X : \varphi(t, t_0, y) \to E, t \to \infty\}.$$

Let $W_\Phi$ be the omega limit set of $\Phi(t, t_0, x_0)$. If $W_\Phi \cap W(E) \neq \emptyset$ then $\lim_{t \to \infty} \Phi(t, t_0, x_0) = E$.

Now find the equilibrium points of the system (2.2) and study their stability. The system (2.2) always has the disease free axial equilibrium point $E_1(A/d, 0, 0, 0)$, which exists unconditionally.

Let the quarantine reproduction number $R_q$ as

$$R_q = \frac{A\beta}{d(\gamma + \delta + d + \alpha_1)}.$$

Then we have the following theorem

Theorem 4.2 The equilibrium $E_1$ is locally asymptotically stable if $R_q < 1$ and unstable if $R_q > 1$.

Proof. The variational matrix $V(E_1)$ at the equilibrium point $E_1$ is given by

$$V(E_1) = \begin{bmatrix}
-d & -A\beta/d & 0 & 0 \\
0 & (\gamma + \delta + d + \alpha_1)(R_q - 1) - (\varepsilon + d + \alpha_2) & 0 & 0 \\
0 & \delta & -d & 0 \\
0 & \gamma & \varepsilon & -d
\end{bmatrix}.$$
The corresponding eigenvalues are 
\[ \lambda_1 = -d, \quad \lambda_2 = (\gamma + \delta + d + \alpha)(R_q - 1), \quad \lambda_3 = -\epsilon + d + \alpha_2, \quad \text{and} \quad \lambda_4 = -d. \]
Clearly \( \lambda_1, \lambda_3, \lambda_4 \) are all negative, and \( \lambda_2 \) is negative or positive according as \( R_q < 1 \) or \( R_q > 1 \). Hence the theorem follows.

**Theorem 4.3** If \( R_q \leq 1 \), then \( E_1 \) is globally stable in \( D \).

**Proof.** Let us consider the positive definite function \( J(S, I, Q, R) \) as follows
\[ J = I. \]
Differentiating \( J \) with respect to \( t \) along the solution of (2.2), we get
\[
\frac{dJ}{dt} = \left[ \frac{\beta S}{1 + \alpha I^2} - (\gamma + \delta + d + \alpha_1) \right] I \\
\leq [\beta S - (\gamma + \delta + d + \alpha_1)] I \\
\leq \left[ \frac{\beta A}{d} - (\gamma + \delta + d + \alpha_1) \right] I \leq 0.
\]
By the Liapunov-Lasalle theorem, solutions in \( D \) approach the largest positively invariant subset of the set where \( \frac{dJ}{dt} = 0 \), which is the set where \( I = 0 \). In this set, we have
\[
\frac{dQ}{dt} = -(\epsilon + d + \alpha_2)Q,
\]
and
\[
\frac{dS}{dt} = A - dS,
\]
which imply that \( Q \to 0 \) and \( S \to A/d \) as \( t \to \infty \).

Then the differential equation for \( R \) is asymptotically equivalent to
\[
\frac{dR}{dt} = -dR,
\]
which implies that \( R \to 0 \) as \( t \to \infty \).

Thus all solutions in the set \( I = 0 \) go to the disease-free equilibrium \( E_1 \). This, in turn, implies that all solutions in \( D \) must also approach \( E_1 \) (by Lemma 4.1).

**Remark.** Here we notice that \( E_1 \) is globally asymptotically stable if \( R_q < 1 \).

Now we consider the existence of the interior equilibrium point \( E^*(S^*, I^*, Q^*, R^*) \).

**Theorem 4.4** The unique interior equilibrium point \( E^*(S^*, I^*, Q^*, R^*) \) of the system (2.2) exists if and only if \( R_q > 1 \). When this condition is satisfied, \( S^*, I^*, Q^*, R^* \) are given by
\[ S^* = \frac{A\beta}{dR_q} \left[ 1 + \frac{\alpha}{2} \left( P - \frac{\beta}{d\alpha} \right)^2 \right], \]
\[ I^* = \frac{1}{2} \left( P - \frac{\beta}{d\alpha} \right), \]
\[ Q^* = \frac{\delta}{2(\varepsilon + d + \alpha_2)} \left( p - \frac{\beta}{d\alpha} \right), \]

\[ R^* = \frac{\gamma \varepsilon + d\gamma + \gamma\alpha_2 + \delta \varepsilon}{2d(\varepsilon + \alpha_2 + d)} \left( p - \frac{\beta}{d\alpha} \right), \]

where

\[ P = \sqrt{\frac{\beta^2}{d^2\alpha^2} + \frac{4}{\alpha}(R_q - 1)}. \]

**Remark.** We notice that the existence of \( E^* \) destabilizes \( E_1 \).

We now study the stability of \( E^* \). The variational matrix of the system (2.2) at \( E^* \) is given by

\[
V(E^*) = \begin{bmatrix}
  v_{11} & v_{12} & 0 & 0 \\
  v_{21} & v_{22} & 0 & 0 \\
  0 & v_{32} & v_{33} & 0 \\
  0 & v_{42} & v_{43} & v_{44}
\end{bmatrix}
\]

where

\[ v_{11} = -\frac{\beta I^*}{1 + \alpha I^*} - d, \]

\[ v_{12} = \frac{\beta S^*(1 - 2\alpha I^*^2)}{(1 + \alpha I^*^2)^2}, \]

\[ v_{21} = \frac{\beta I^*}{1 + \alpha I^*^2}, \]

\[ v_{22} = \frac{2\alpha \beta S^* I^*^2}{(1 + \alpha I^*^2)^2}, \]

\[ v_{32} = \delta, \]

\[ v_{33} = -(\varepsilon + d + \alpha_2), \]

\[ v_{42} = \gamma, \]

\[ v_{43} = \varepsilon, \]

\[ v_{44} = -d. \]

The characteristic equation is

\[ \lambda^4 + A_1 \lambda^3 + A_2 \lambda^2 + A_3 \lambda + A_4 = 0, \]

where

\[ A_1 = -v_{11} - v_{22} - v_{33} - v_{44}. \]

\[ A_2 = v_{22}v_{33} + v_{11} v_{22} + v_{11} v_{33} + v_{11} v_{44} - v_{12} v_{21} + v_{33} v_{44} + v_{22} v_{44}. \]

\[ A_3 = -v_{11} v_{23} v_{44} - v_{11} v_{33} v_{44} - v_{22} v_{33} v_{44} - v_{11} v_{22} v_{33} - v_{12} v_{21} v_{44} + v_{12} v_{21} v_{33}. \]
Let $A = A_1 A_2 A_3 - A_1^2 A_2 - A_3^2 A_4$. Then we have the following theorem on local stability of $E^*$.

**Theorem 4.5** If $A_3 > 0$, $A_4 > 0$ and $\Delta > 0$, then $E^*$ is locally asymptotically stable.

**Proof.** It is easy to notice that $A_1 > 0$. Then, as $A_3 > 0$, $A_4 > 0$ and $\Delta > 0$, the theorem follows from Routh Hurwitz criterion.

The following theorem gives a global result on $E^*$.

**Theorem 4.6** If $E^*$ is locally asymptotically stable, then $\Omega = \{ (S, I, Q, R) : S = 0 \text{ or } I = 0 \}$ is a region for the endemic equilibrium $E^*$.

**Proof.** Let $G = \{ (S, I) : S > 0, I > 0, \text{ and } S + I > A/d \}$. We consider the following $SI$ subsystem of the system (2.2) in $G$:

$$\frac{dS}{dt} = A - \frac{\beta SI}{1 + \alpha I^2} - dS = P(S, I),$$

$$\frac{dI}{dt} = \frac{\beta SI}{1 + \alpha I^2} - (\gamma + \delta + d + \alpha_2) I = Q(S, I).$$

Let us take a Dulac function

$$D(S, I) = \frac{1}{SI}.$$ 

Then we notice that

$$\frac{\partial(DP)}{\partial S} + \frac{\partial(DQ)}{\partial I} = \frac{1}{S^2 I} - \frac{2\alpha\beta I}{(1 + \alpha I^2)^2} < 0.$$ 

Therefore, there is no periodic solution in $G$ by Bendixson-Dulac negative criterion. Thus by the Poincaré-Bendixson theorem, all solutions starting in $G$ approach $(S^*, I^*)$ as $t \to \infty$.

In this case, the differential equation for $Q$ has the limiting equation

$$\frac{dQ}{dt} = \delta I^* - (\epsilon + d + \alpha_2) Q.$$ 

Therefore, by Lemma 4.1, $Q \to Q^*$ as $t \to \infty$.

Similarly, the differential equation for $R$ has the limiting equation

$$\frac{dR}{dt} = \gamma I^* + \epsilon Q^* - dR,$$

so that $R \to R^*$ as $t \to \infty$ (by Lemma 4.1).

Hence $E^*$ is a globally asymptotically stable equilibrium for the system (2.2) in $\Omega$. □

5 Numerical simulation:

We present computer simulations of some solutions of the system (2.2) in this section. These simulations are performed to validate some of the analytical findings of some solutions of the system (2.2) using MATLAB.

First, we take the parameters of the system (2.2) as $A = 0.5$, $\beta = 0.3$, $\alpha = 0.5$, $d = 1$, $\gamma = 0.7$, $\delta = 0.3$, $\alpha_1 = 0.5$, $\epsilon = 0.3$, $\alpha_2 = 0.2$. Then the condition $R_e = 0.06 < 1$ of Theorem 4.2 is satisfied and consequently $E_1(A/d, 0, 0, 0)$ is locally asymptotically stable. The behaviours of $x$, $y$ with $t$ is depicted in Figure 3.
Next we consider the stability of the interior equilibrium. We choose the parameters of the system (2.2) as $A = 15$, $\beta = 3$, $\alpha = 0.5$, $d = 1$, $\gamma = 0.7$, $\delta = 0.3$, $\alpha_1 = 0.5$, $\varepsilon = 0.3$, $\alpha_2 = 0.2$. Then $R_q = 18 > 1$ and so the unique interior equilibrium point $E^*(S^*, I^*, Q^*, R^*)$ exists, where $S^* = 6.1064$, $I^* = 3.5574$, $Q^* = 0.7115$ and $R^* = 2.7039$. Now for the above choice of parameters, conditions of Theorem 4.5 are satisfied, as such $E^*$ is locally asymptotically stable. Figure 4 shows the corresponding behaviours of $x, y$ with $t$. 

**Figure 3:** Behaviour of the system (2.2) as $A = 0.5$, $\beta = 0.3$, $\alpha = 0.5$, $d = 1$, $\gamma = 0.7$, $\delta = 0.3$, $\alpha_1 = 0.5$, $\varepsilon = 0.3$, $\alpha_2 = 0.2$ for $S(0) = 0.35$, $I(0) = 0.35$, $Q(0) = 0.35$, $R(0) = 0.35$, showing that $E_1(A/d, 0, 0, 0)$ is locally asymptotically stable.

**Figure 4:** $A = 15$, $\beta = 3$, $\alpha = 0.5$, $d = 1$, $\gamma = 0.7$, $\delta = 0.3$, $\alpha_1 = 0.5$, $\varepsilon = 0.3$, $\alpha_2 = 0.2$. Phase portrait of the system (2.2) for $S(0) = 3$, $I(0) = 3$, $Q(0) = 3$ and $R(0) = 3$, $E^*(S^*, I^*, Q^*, R^*)$ where $S^* = 6.1064$, $I^* = 3.5574$, $Q^* = 0.7115$ and $R^* = 2.7039$ is locally asymptotically stable.
6 Concluding remarks

This paper aims to study the dynamics of an SIQR model, which takes into account the psychological effect. This has been done by a nonlinear and nonmonotone incidence (originally suggested by Xiao and Ruan\textsuperscript{13}). It is shown (in Theorem 3.1 and Theorem 3.2) that the solutions of the system (2.2) remains non-negative forever, and they are uniformly bounded. These, in turn, imply that the system is well-behaved. The equilibria and their stability analysis is the main recipe of the paper. It is seen that, if the quarantine reproduction number $R_q \leq 1$, then the disease-free equilibrium $E_1$ is globally stable. On the other hand, the existence of the endemic equilibrium destabilizes $E_1$. A criterion for global stability of the endemic equilibrium is also established.

When the disease is endemic, the equilibrium value $I^*$ of the infectives decreases as $\alpha$ increases. This implies that the spread of disease decreases as the social or psychological consciousness increases. In other words, $I^*$ increases as $\alpha$ decreases, which means that decreased social awareness about the disease might cause a rapid spread of diseases. Also one might notice that $I^*$ approaches zero as $\alpha \to \infty$ (although, in reality, it is perhaps beyond expectation that social or psychological protective measures will reach such a level that we might think of $\alpha \to \infty$; still, this theoretical observation might act as a motivation for such measures). These results are in good agreement with those of Xiao and Ruan\textsuperscript{13}, and Pathak et al.\textsuperscript{10}. The scope of future work is developing and analyzing such models which is an open area of research. Of course, this will require more sophisticated mathematical techniques and some more details of experimental findings.

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