Abstract

This paper aims to study the dynamical behaviours of an SIQR epidemiological model. Positivity and boundedness of the system are discussed. Stability analysis of the equilibrium points is presented. Numerical simulations are carried out to validate our analytical findings. Implications of our analytical and numerical findings are discussed critically.

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

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

1. Introduction

From prehistory to the present day, diseases have been a source of fear and superstition. Nowadays, infectious diseases have become a cause for great concern for domestic and global health systems. 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 each year.

Over the centuries, quarantine has been used as an intervention procedure to control the spread of many infectious diseases. 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, Newcastle disease and rabies10.

In the past one hundred years, mathematics has been used to understand and predict the spread
of diseases, relating important public-health questions to basic transmission parameters. A comprehensive picture of disease dynamics requires a variety of mathematical tools, from model creation to solving differential equations to statistical analysis. Although mathematics has been so far done quite well in dealing with epidemiology but there is no denying that there are certain factors which still lack proper mathematical. Almost all mathematical models of diseases start from the same basic premise: that the population can be subdivided into a set of distinct classes, dependent upon their experience with respect to the disease. One line of investigation classifies individuals as one of susceptible, infectious or recovered. Such a model is termed as an SIR model. A detailed history of mathematical epidemiology and basics of SIR epidemic models may be found in the classical books of Bailey\(^3\), Murray\(^13\) and Anderson and May\(^2\). For mathematical works on SIR models, see\(^14\), 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\(^10,12\).

At present, almost all the developing countries have increasingly realized the necessity of social consciousness in preventing the diseases. Also different protective measures against diseases are found to be effective. Therefore, a saturated incidence will be better to describe the disease dynamics rather than the mass action incidence. Such incidence have recently been used by many authors\(^5,7,11,16,18,15\).

In this paper, we have studied the dynamics of a SIQR model with a saturated incidence. The rest of the paper is organized as follows. In section 2, we present the mathematical model with basic considerations. Boundedness and positivity of the solutions of the model are established in section 3. Section 4 deals with all the possible equilibrium points of the model and their stability analysis. 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. Section 7 indicates further scope of future work.

2 The mathematical model:

Before we introduce the basic model, and dip into the depth of the things, 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. 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 a saturated incidence to describe the disease dynamics.

![Figure 1: The transfer diagram for the model.](image-url)
The above considerations motivate us to introduce the basic mathematical model under the framework of the following set of nonlinear ordinary differential equations:

\[ \frac{dS}{dt} = A - \frac{\beta SI}{1 + \alpha I} - dS, \]
\[ \frac{dI}{dt} = \frac{\beta SI}{1 + \alpha I} - (\gamma + \delta + d + \alpha_1) I, \]
\[ \frac{dQ}{dt} = \delta I - (\epsilon + d + \alpha_2) Q, \]
\[ \frac{dR}{dt} = \gamma I + \epsilon Q - dR, \]

(2.1)

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 \( \alpha \) shows the effect of saturated incidence. 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:

Positivity and boundedness of a model guarantee that the model is biologically well behaved. For positivity of the system (2.1), we have the following theorem.

**Theorem 3.1** All solutions of the system (2.1) 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 (2.1).

**Theorem 3.2** All solutions of the system (2.1) that start in \( \mathbb{R}_+^4 \) are uniformly bounded.

**Proof.** Let \( N = S + I + Q + R \). Then we have

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

Therefore, the population size \( N \) approaches the carrying capacity \( A/d \) when there is no disease. The differential equation for \( N \) implies that solutions of (2.1) 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:

Before we find the equilibria of the system (2.1), and dip into the depth of the stability analysis, we state the following theorem, which would be very helpful for our analysis.

**Lemma 4.1** Consider the following two systems

\[ \frac{dx}{dt} = f(t, x), \quad \frac{dy}{dt} = g(y), \]
where \( x, y \in \mathbb{R}^n \), \( f \) and \( g \) are continuous, satisfy a local Lipschitz condition in any compact set \( X \subseteq \mathbb{R}^n \), 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 it is time to find the equilibrium points of the system (2.1) and study their stability. The system (2.1) always has the disease free axial equilibrium point \( E_1(A/d, 0, 0, 0) \), which exists unconditionally.

Let us define 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
\[
V(E_1) = \begin{bmatrix}
-d & -A\beta/d & 0 & 0 \\
0 & (\gamma + \delta + d + \alpha_1)(R_q - 1) & 0 & 0 \\
0 & \delta & -(\epsilon + d + \alpha_2) & 0 \\
0 & \gamma & \epsilon & -d
\end{bmatrix}
\]

The corresponding eigenvalues are \( \lambda_1 = -d, \ \lambda_2 = (\gamma + \delta + d + \alpha_1)(R_q - 1), \ \lambda_3 = -\epsilon + d + \alpha_2, \) and \( \lambda_4 = -d \). 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 a the positive definite function \( J(S, I, Q, R) \) as follows
\[
J = I.
\]

Differentiating \( J \) with respect to \( t \) along the solution of (2.1), we get
\[
\frac{dJ}{dt} = \left[ \frac{\beta S}{1 + \alpha I} - (\gamma + \delta + d + \alpha_1) \right] I
\leq \left[ \beta S - (\gamma + \delta + d + \alpha_1) \right] I
\leq \left[ \frac{\beta A}{d} - (\gamma + \delta + d + \alpha_1) \right] I \leq 0.
\]

By the Liapunov-Lasalle theorem\(^8\), solutions in \( D \) approach the largest positively invariant subset of the set where \( \frac{dI}{dt} = 0 \), which is the set where \( I = 0 \). In this set, we have
\[
\frac{dQ}{dt} = -(\epsilon + d + \alpha_2) Q,
\]

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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.** It is easy to 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 (2.1) exists if and only if \( R_q > 1 \). When this condition is satisfied, \( S' \), \( I' \), \( Q' \), \( R' \) are given by

\[
S^* = \frac{(\gamma + \delta + d + \alpha_1)(A\alpha + \gamma + \delta + d + \alpha_1)R_q}{\beta(\gamma + \delta + d + \alpha_1)R_q + A\alpha},
\]

\[
I^* = \frac{A(R_q - 1)}{(\gamma + \delta + d + \alpha_1)R_q + A\alpha},
\]

\[
Q^* = \frac{A\delta(R_q - 1)}{(\epsilon + d + \alpha_2)(\gamma + \delta + d + \alpha_1)R_q + A\alpha},
\]

\[
R^* = \frac{A(R_q - 1)(\gamma(\epsilon + d + \alpha_2) + \epsilon)}{d(\epsilon + d + \alpha_2)(\gamma + \delta + d + \alpha_1)R_q + A\alpha}.
\]

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

We now study the stability of \( E^* \). The variational matrix of the system (2.1) 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

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

\[
v_{12} = -\frac{\beta S^*}{(1 + \alpha_I^*)^2},
\]
The characteristic equation is
\[ \lambda^4 + A_1 \lambda^3 + A_2 \lambda^2 + A_3 \lambda + A_4 = 0, \]
where
\[ A_1 = -v_{11} - v_{22} - v_{33} - v_{44}, \]
\[ A_2 = v_{22} v_{33} + v_{11} v_{22} + v_{11} v_{33} + v_{11} v_{44} - v_{12} v_{21} + v_{13} v_{44} + v_{22} v_{44}, \]
\[ A_3 = -v_{11} v_{22} v_{44} - v_{11} v_{33} v_{44} - v_{22} v_{33} v_{44} - v_{11} v_{22} v_{33} - v_{12} v_{21} v_{33} + v_{12} v_{21} v_{33}, \]
\[ A_4 = v_{11} v_{22} v_{33} v_{44} - v_{12} v_{21} v_{33} v_{44}. \]

Let \( A = A_1 A_2 A_3 - A_2^2 A_4 \). Then we have the following theorem on local stability of \( E^* \).

**Theorem 4.5** If \( \Delta > 0 \), then \( E^* \) is locally asymptotically stable.

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

The following theorem gives a global result on \( E^* \).

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

**Proof.** Let \( \Gamma = \{(S, I) : S > 0, I > 0, \text{ and } S + I > A/d\} \). We consider the following \( SI \) subsystem of the system (2.1) in \( \Gamma \):

\[ \frac{dS}{dt} = A - \frac{\beta SI}{1 + \alpha I} - dS \equiv P(S, I), \]
\[ \frac{dI}{dt} = \frac{\beta SI}{1 + \alpha I} - (\gamma + \delta + d + \alpha_1) I \equiv Q(S, I), \]

Let us take a Dulac function
\[ D(S, I) = \frac{1}{SI}. \]

Then we notice that
\[ \frac{\partial (DP)}{\partial S} + \frac{\partial (DQ)}{\partial I} = -\frac{1}{S^2 I} - \frac{\alpha \beta}{(1 + \alpha I)^2} < 0. \]
Therefore, by Bendixson-Dulac negative criterion, there is no periodic solution in \( \Gamma \). Thus by the Poincaré-Bendixson theorem, all solutions starting in \( \Gamma \) approach \((S^*, I^*)\) as \( t \to \infty \).

In this case, the differential equation for \( Q \) has the limiting equation

\[
\frac{dQ}{dt} = \delta I^* - (\epsilon + d_2 + \alpha_2)Q.
\]

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 (2.1) in \( \Omega \).

5 Numerical simulation:

In this section, we present computer simulations of some solutions of the system (2.1). These simulations are performed to validate some of the analytical findings of some solutions of the system (2.1) using MATLAB.

First, we take the parameters of the system (2.1) as \( A = 0.5, \quad \beta = 0.3, \quad \alpha = 0.5, \quad d = 1, \quad \gamma = 0.7, \quad \delta = 0.3, \quad \alpha_1 = 0.5, \quad \epsilon = 0.3, \quad \alpha_2 = 0.2 \) in Figure 2. Then the conditions of Theorem 4.2 is satisfied and consequently \( E_1(A/d, 0, 0, 0) \) is locally asymptotically stable.

Next we consider the stability of the interior equilibrium. We choose the parameters of the system (2.1) as \( A = 15, \quad \beta = 3, \quad \alpha = 0.5, \quad d = 1, \quad \gamma = 0.7, \quad \delta = 0.3, \quad \alpha_1 = 0.5, \quad \epsilon = 0.3, \quad \alpha_2 = 0.2 \) in Figure 3. Then there is a unique interior equilibrium point \( E^*(S^*, I^*, Q^*, R^*) \) where \( S^* = 2.8571, \quad I^* = 4.8571, \quad Q^* = 0.9714 \) and \( R^* = 3.6914 \). Hence by Theorem 4.5, \( E^* \) is locally asymptotically stable.

![Figure 2: Behaviour of the system (2.1) as \( A = 0.5, \quad \beta = 0.3, \quad \alpha = 0.5, \quad d = 1, \quad \gamma = 0.7, \quad \delta = 0.3, \quad \alpha_1 = 0.5, \quad \epsilon = 0.3, \quad \alpha_2 = 0.2 \) for \( S(0) = 0.35, \quad I(0) = 0.35, \quad Q(0) = 0.35, \quad R(0) = 0.35, \quad E_1(A/d, 0, 0, 0) \) is locally asymptotically stable.](image)

![Figure 3: \( A = 15, \quad \beta = 3, \quad \alpha = 0.5, \quad d = 1, \quad \gamma = 0.7, \quad \delta = 0.3, \quad \alpha_1 = 0.5, \quad \epsilon = 0.3, \quad \alpha_2 = 0.2 \). Phase portrait of the system (2.1) for \( S(0) = 2, \quad I(0) = 2, \quad Q(0) = 2 \) and \( R(0) = 2, \quad E^*(S^*, I^*, Q^*, R^*) \), where \( S^* = 2.8571, \quad I^* = 4.8571, \quad Q^* = 0.9714 \) and \( R^* = 3.6914 \) is locally asymptotically stable.](image)
6 Concluding remarks

The entire globe is now concerned about the menace of infectious diseases, and these diseases have caused fearful tolls in different communities. However, almost all the developing countries have increasingly realized the necessity of social consciousness in preventing the diseases. Also different protective measures against diseases are found to be effective.

Quarantine has been used to reduce the transmission of diseases for many centuries. In this paper, we have studied the dynamics of an $SIQR$ epidemiological model. A saturated incidence is taken to take into account the effect of social consciousness, etc. It is shown (in Theorem 3.1 and Theorem 3.2) that the solutions of the system (2.1) 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.

It is interesting to note that, when the disease is endemic, the steady-state value $I^*$ of the infectives decreases as $\alpha$ increases. This implies that the spread of disease decreases as the social or psychological protective measures for the infectives 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$^{17}$, and Pathak et al.$^{14}$.

7 Scope of future work :

The system (2.1) in a random environment under pulse vaccination strategy can be formulated as follows:

$$
\frac{dS}{dt} = \frac{A(t)}{1+\alpha(t)I} - \frac{\beta(t)SI}{1+\alpha(t)I} - d(t)S + \sigma_1(t)S(t) \frac{dW_1}{dt}, \; \; t \neq nT,
$$

$$
\frac{dI}{dt} = \frac{\beta(t)SI}{1+\alpha(t)I} - (\gamma(t) + (\delta(t) + d(t) + \alpha(t))I + \sigma_2(t)I(t)) \frac{dW_2}{dt}, \; \; t \neq nT,
$$

$$
\frac{dQ}{dt} = \delta(t)I - (\epsilon(t) + d(t) + \alpha_2(t))Q + \sigma_3(t)Q(t) \frac{dW_3}{dt}, \; \; t \neq nT,
$$

$$
\frac{dR}{dt} = \gamma(t)I + \epsilon(t)Q - d(t)R + \sigma_4(t)R(t) \frac{dW_4}{dt}, \; \; t \neq nT,
$$

$$
S(t^*) = (1-p)S(t), \; \; t = nT, \; n = 1, 2, \ldots
$$

$$
I(t^*) = I(t), \; \; t = nT, \; n = 1, 2, \ldots
$$

$$
Q(t^*) = Q(t), \; \; t = nT, \; n = 1, 2, \ldots
$$

$$
R(t^*) = R(t) + pS(t), \; \; t = nT, \; n = 1, 2, \ldots,
$$

where $A(t)$, $\beta(t)$, $\alpha(t)$, $d(t)$, $\gamma(t)$, $\delta(t)$, $\alpha_2(t)$, $\epsilon(t)$ and $\sigma_i(t)$, $(i = 1, 2, 3, 4)$ are all positive $T$– periodic continuous functions, $T$ is a positive constant; $T$ is the period of pulse vaccination. Here $p(0 < p < 1)$
is the constant fraction of susceptible who are vaccinated successfully at discrete time $t = T, 2T, 3T, \ldots$, which is called impulsive vaccination rate. In this system, $\eta_i = \frac{dW_i}{dt}$, $(i = 1, 2, 3, 4)$ are independent standard zero mean Gaussian white noises characterized by

\[ \langle \eta_i(t) \rangle = 0, \quad \langle \eta_i(t_1) \eta_i(t_2) \rangle = \delta(t_1 - t_2) \text{ and } \langle \eta_i(t_1) \eta_j(t_2) \rangle = 0, \quad (i \neq j), \]

where $\langle \cdot \rangle$ represents the average over the ensemble of the stochastic process and $\delta(t)$ denotes the Dirac delta function.

The dynamical behaviour of this model system can be taken as scope of future work.

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