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Original Article

Global Stability of SEIQR Model with Isolation Compartment

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Abstract *- Isolation is currently an effective measure to reduce exposure. In combination with this control factor, we take the isolation compartment into account in the infectious disease model. We build a system of SEIR with quarantine in the beginning. We calculate the basic reproduction number $\mathcal{R}_0$, which is the threshold for disease extinction. The disease-free equilibrium is globally asymptotically stable for $\mathcal{R}_0$ <1, while is unstable for $\mathcal{R}_0$ >1. Finally, we show that appropriate increasing the intensity of quarantine and vaccination coverage is effective for disease control numerically.*

Keywords *- Epidemic, SEIQR model, Threshold dynamics, Global stability, Autonomous system.*

1. Introduction

For a long time, epidemics endanger human health and affect economic development. Tuberculosis, hepatitis B and AIDS have not yet been eradicated, and new infectious diseases have broken out one after another, such as SARS in 2003, influenza A (H1N1) in 2009 and avian influenza (H7N9) in 2013, which have caused great impact on social economy and human health. We know that prevention is the basic premise. Therefore, we should give full attention to the prevention and control of epidemics.

Epidemiological models play a large role in the process of disease transmission and the transition between different disease states. Their merits can be reflected in many aspects. Epidemiological models are easier to analyze concretely. Many diseases (such as tuberculosis, Hepatitis C, influenza, COVID-19, chicken pox, and so on) are known to have an exposed or latent phase, where individuals are infected but not yet infectious. There have been many epidemic models incorporated latency ([8, 9, 10, 11, 12, 13, 14, 15] and references therein).

We know that prevention is better than cure. [22] considers vaccination in the SEIR model when modelling and analyzing measles disease. In the development of the disease, timely isolation of the infected will slow the further spread of the disease. Epidemics such as SARS and, more recently, COVID-19 have resorted to quarantines. Inspired by disease, in this paper we consider an infectious disease model with quarantine. It is very natural to study the combined effects of quarantine and vaccination. Based on the SEIR epidemic model, the total population is divided into five compartments in this paper.

Next we present the structure of the paper. We introduce an epidemic model with quarantine in the second part. We know that mathematical models are tightly integrated with biological meaning. In part 3 we calculate a threshold. Next we discuss the global nature of the system. We present two numerical examples in the fourth part. Finally a brief discussion is presented in Section 5.

2. Formulation of the Model

In this section, we formulate the epidemic model with quarantine to describe the disease transmission. We denote the total numbers of population by $N(t)$ and divide the population into five subclasses: susceptible, exposed, infectious, quarantined and recovered. Let $S(t)$, $E(t)$, $I(t)$, $Q(t)$ and $R(t)$ denote the numbers of the susceptible, exposed, infectious, quarantined and removed individuals at time t , respectively. Then an epidemic model containing quarantine can be described by the following system

$$\begin{cases} \frac{dS}{dt} = A(1-\rho) - \beta SI - \mu S, \\ \frac{dE}{dt} = \beta SI - \mu E - \sigma E, \\ \frac{dI}{dt} = \sigma E - \mu I - \xi I - \gamma I, \\ \frac{dQ}{dt} = \xi I - \mu Q - dQ, \\ \frac{dR}{dt} = dQ + \gamma I + \rho A - \mu R. \end{cases} \tag{1}$$

Here A is human birth rate; $\mu$is unit of natural mortality; β is the probability of transmission by contact between $S(t)$ and $I(t)$; σmeans probability of transfer of latent individuals from compartment E; ξ is the isolation intensity of an infected individual entering the isolation chamber after taking isolation measures; γ is the recovery rate of infectious individuals; ρ is human vaccination rate. All the parameters are positive constant.

3. Mathematical Analysis

Notice that from the equations in model (1), we have

$$\frac{dN}{dt} = A - \mu N. \tag{2}$$

Letting $\Lambda = \{(S,E,I,Q,R) | S > 0, E \geq 0, I \geq 0, Q \geq 0, R > 0, 0 < S + E + I + Q + R < A/\mu\}.$ According to the biological significance of system (1), we only need to study system (1) in Λ. From the concept of invariant set, we can get that the largest positive invariant set of system (1) is Λ.

Clearly, system (1) always has a disease-free equilibrium $P_0 = (\hat{S}, 0, 0, 0, \hat{R})$, which is reflected in the biological sense indicating the extinction of the disease, where $\hat{S} = A(1-\rho)/\mu$,$\hat{R} = A\rho/\mu$. For threshold dynamics of model (1), we need to compute the threshold. The tools in [20, 23] give us considerable help.

Denote

$$F = \begin{pmatrix} 0 \\ \beta SI \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad V = \begin{pmatrix} A(\rho-1) + \beta SI + \mu S \\ (\mu+\sigma)E \\ -\sigma E + (\mu + \xi + \gamma)I \\ -\xi I + (\mu + d)Q \\ -\rho A - dQ - \gamma I + \mu R \end{pmatrix}.$$

The infection compartments corresponding to model (1) are E and I, and then

$$F' = \begin{pmatrix} 0 & \frac{\beta A(1-\rho)}{\mu} \\ 0 & 0 \end{pmatrix}, \quad V' = \begin{pmatrix} \mu + \sigma & 0 \\ -\sigma & \mu + \xi + \gamma \end{pmatrix}.$$

By using the solution method of inverse matrix, we can get

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu+\sigma} & 0 \\ \frac{\sigma}{(\mu+\sigma)(\mu+\xi+\gamma)} & \frac{1}{\mu+\xi+\gamma} \end{pmatrix},$$

And

$$FV^{-1} = \begin{pmatrix} \frac{\beta\sigma A(1-\rho)}{\mu(\mu+\sigma)(\mu+\xi+\gamma)} & \frac{\beta A(1-\rho)}{\mu(\mu+\xi+\gamma)} \\ 0 & 0 \end{pmatrix},$$

then

$$\Re_0 = \rho(FV^{-1}) = \frac{\beta\sigma A(1-\rho)}{\mu(\mu+\sigma)(\mu+\xi+\gamma)}.$$

Thus we have the following result..

Theorem 3.1. When $\Re_0 \leq 1$, there is only disease-free equilibrium P_0.

We have obtained the existence of P_0, and next we analyze the stability of P_0.

Theorem 3.2. P_0 is locally asymptotically stable when $\Re_0 < 1$ and unstable when $\Re_0 > 1$.

Proof. First, we calculate the Jacobian matrix of model (1) with regard to $P_0 = (\hat{S}, 0,0,0, \hat{R})$

$$J = \begin{pmatrix} -\mu & 0 & -\frac{\beta A(1-\rho)}{\mu} & 0 & 0 \\ 0 & -(\mu+\sigma) & \frac{\beta A(1-\rho)}{\mu} & 0 & 0 \\ 0 & \sigma & -(\mu+\xi+\gamma) & 0 & 0 \\ 0 & 0 & \xi & -(\mu+d) & 0 \\ 0 & 0 & \gamma & d & -\mu \end{pmatrix}.$$

Then the corresponding characteristic equation is given.
On the basic reproduction number $\Re_0$ of model (1), we have

$$(\lambda+\mu)^2(\lambda+\mu+d)[(\lambda+\mu+d)(\lambda+\mu+\xi+\gamma) - \frac{\sigma\beta A(1-\rho)}{\mu}] = 0. \quad (3)$$

From (3) we can figure out that the roots are $-\mu$ and $-(\mu+\sigma)$, where $-\mu$ is a double root. Next we need to solve the following

$$(\lambda+\mu+d)(\lambda+\mu+\xi+\gamma) - \frac{\sigma\beta A(1-\rho)}{\mu} = 0. \quad (4)$$

Simplifying (4) yields

$$\lambda^2 + [(\mu+\sigma)+(\mu+\xi+\gamma)]\lambda + (\mu+\sigma)(\mu+\xi+\gamma) - \frac{\sigma\beta A(1-\rho)}{\mu} = 0 \quad (5)$$

Obviously, $(\mu+\sigma)+(\mu+\xi+\gamma) > 0$.Because of $\Re_0 < 1$, $\sigma\beta A(1-\rho) < \mu(\mu+\sigma)(\mu+\xi+\gamma)$, then $\frac{\sigma\beta A(1-\rho)}{\mu} < (\mu+\sigma)(\mu+\xi+\gamma)$. Therefore, according to the Routh-Hurwitz Criteria, when $\Re_0 < 1$, the eigenroots of equation (3) are all negative roots, that is, P_0 is locally asymptotically stable. When $\Re_0 > 1$, after solving equation (3), we get the eigenroot that has a positive real part. Thus P_0 is unstable.

Theorem 3.3. P_0 is globally asymptotically stable if $\Re_0 \le 1$ and P_0 is unstable if $\Re_0 > 1$.

Proof. Lyapunov function has a wide range of applications. [29] proposes a systematic algebraic method to determine the coefficients and show the negative (semi-) determinism of the derivatives. Consider the Lyapunov function

$$V_1 = E + \frac{\mu+\sigma}{\sigma} I,$$

then we have

$$\frac{dV_1}{dt}\Big|_{(1)} = \beta SI - (\mu+\xi+\gamma)I \le \beta\frac{A(1-\rho)}{\mu}(1-\Re_0)I - \beta(E+I+Q+R)I \le 0.$$

Denote

$$L = \{(S,E,I,Q,R) | V'_{(1)} = 0\} = \{(S,E,I,Q,R) | I = 0\}.$$

Since $L \to \{P_0\}$ as $t \to +\infty$, $\{P_0\}$ is the largest positive invariant subset of L. Thus the global asymptotic stability of P_0 can be obtained by the Lasalle invariant set principle. Together with Theorem 3.2, it follows that Theorem 3.3 holds.

4. Numerical Simulation

In this part we consider the effect of changes in parameters ξ and ρ on $\Re_0$ with the help of the data. We take

$$A = 1\times 10^6, \beta = 1\times 10^{-10}, \mu = 0.00053, \sigma = 2, \gamma = 2, d = 3.$$

Taking $\rho = 0.8216$, we vary ξ to obtain the relationship between $\Re_0$ and ξ, as shown in the left figure below. Taking $\xi = 2$, we let ρ vary to analyze the relationship between $\Re_0$ and ρ, shown in the right panel below.

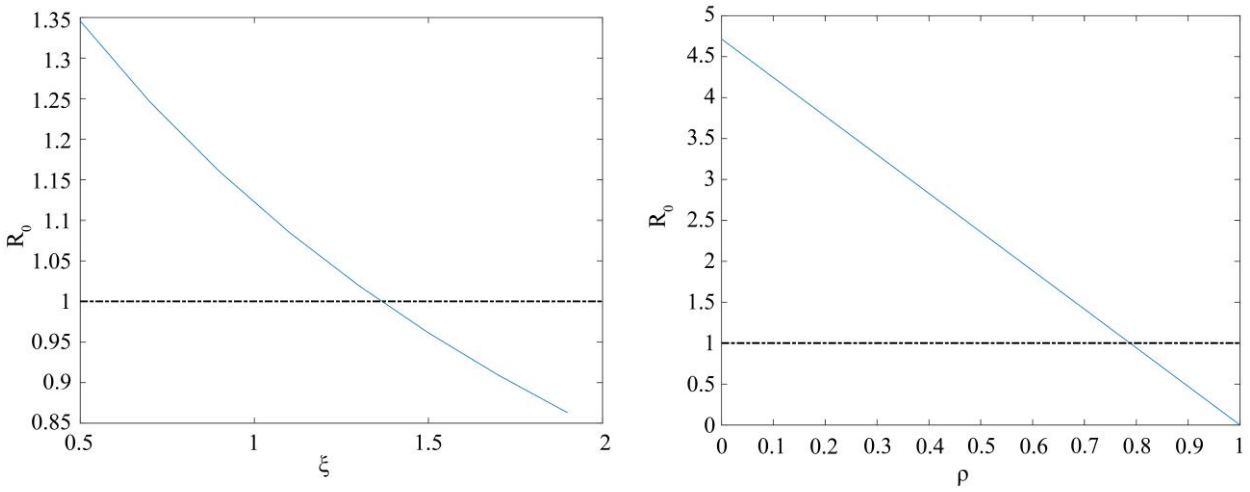

Fig. 1 The relationships between $\Re_0$ and ξ and ρ

The left diagram of Figure 1 shows a very clear relationship between $\Re_0$ and ξ. Going back to infectious diseases, the goal is to keep them from spreading as much as possible. Therefore, increasing the intensity of isolation appropriately is a positive effect on the development of the disease. As shown in the right figure, with the increase of vaccination rate ρ, $\Re_0$ gradually decreases. That is, the higher the vaccination coverage, the more beneficial it is for disease prevention and control.

5. Conclusion

Starting from the development process of the disease, we build the corresponding infectious disease model. Based on the basic reproduction number $\Re_0$, we put the mathematical model into a biological sense. We obtain the condition for disease extinction, that is, $\Re_0 < 1$. In addition, for the problems that cannot be solved by theory, we present them in the fourth section.

This model provides theoretical guidance for the spread of infectious disease models. Moreover, our model can be extended in many directions. For example, we can consider vaccination as a function of age. At present, there are corresponding vaccines for many infectious diseases, but the effectiveness of vaccination varies at different ages. Therefore, considering vaccination as a function of age can better reflect the true effectiveness of disease prevention.

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