Research on Computer Aided Computation of Infectious Disease SIR Model Algorithm Based on Parameter Control

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Abstract. The algorithm proposed in this paper conceives the process of solving optimization problems as a process in which an infectious disease spreads among several individuals in an ecosystem. Its propagation law can be described by the SIR infectious disease model. Infectious diseases attack certain locations in several disease-causing genes of individuals. For different individuals, which disease genes and which sites are attacked are completely random; if an individual is cured, which immune genes and which sites are immunized are also completely random. Because of the problem of determining the growth rate of the number of infectious diseases, and improved SIR model is established, combined with historical data to quantitatively predict the future growth rate of the number of infected people, to formulate corresponding disease prevention and control measures.

Keywords: Parameter control; computer algorithm; SIR model; infectious disease prevention.

1. Introduction
Infectious diseases seriously threaten human health. Diseases such as AIDS, smallpox, avian influenza, and tuberculosis have claimed the lives of thousands of people. It is increasingly important to study the transmission mechanism of infectious diseases and take relevant measures to control infectious diseases. Infectious diseases have been the focus of research in recent years, and basic theories have been applied to the fields of social science, biology, and medicine. When studying infectious diseases in groups, factors such as infection rate, mortality, and immunity cannot be ignored. Infectious Disease Dynamics It is a method for quantitative research on infectious diseases. Based on population characteristics, disease occurrence, and transmission characteristics, a model is established, and the model is
theoretically analyzed to predict the development and changes of the disease, which can effectively control the spread of the disease.

Literature introduces the pulse vaccination theory in the infectious disease model in detail; literature analyzes the SIR epidemic disease model with constant population and compares the different effects of constant vaccination and pulse vaccination; literature The SIR infectious disease model of pulse vaccination was analyzed in detail; literature introduced that when the treatment resources are limited and the number of patients exceeds a certain threshold, even if the guaranteed basic regeneration number is less than 1, the disease will continue. Based on the aforementioned literature, this paper chooses the SIR model as the basic model, and assumes that the treatment resources are sufficient (to avoid backward branching), establishes a corresponding warehouse model for different strategies for controlling infectious diseases, and studies basic regeneration Number and sufficient conditions for stable disease-free equilibrium point, compare the conditions of threshold changes under different strategies, to explore the impact of different strategies on epidemic control. Through comparison, we found that continuous vaccination is more effective than a disease in treating disease, and when vaccination When the period \( T < T_c \), pulse vaccination is more effective for disease control than continuous vaccination [1].

At present, there are mainly four kinds of research on infectious diseases: descriptive research, analytical research, experimental research, and theoretical research. Infectious disease dynamics is an important method for theoretical quantitative research on infectious diseases. It is based on the characteristics of population growth, the occurrence of diseases, and the laws of transmission and development within the population, as well as the social factors related to them, establish a mathematical model that can reflect the dynamic characteristics of infectious diseases. Through the qualitative and quantitative analysis of the dynamic behavior of the model Analysis and numerical simulation to show the development process of the disease, reveal its epidemic law, predict its changing trend, analyze the causes and key factors of the disease epidemic, seek the optimal strategy for its prevention and control, and provide a theoretical basis for people's control decisions And quantitative basis. The thesis is aimed at the number of infectious diseases to determine the growth rate, establish an improved SIR model, and combine historical data to quantitatively predict the growth rate of the number of future infections, to formulate corresponding disease prevention and control measures.

2. SIR model of infectious disease transmission

2.1. Parameter introduction

The SIR warehouse model is to divide the population in the area into the following three categories (ie three warehouses) for a certain type of infectious disease: First, the number of susceptible people is recorded as \( S(t) \), indicating that it is infected at the time but may be affected by this category The number of people infected with the disease. Second, the number of infected persons is recorded as \( I(t) \), indicating the number of people who have been infected as patients at the time and is infectious. Third, the number of removed persons is recorded as \( R(t) \), indicating that they have been infected from the infection at the moment the number of people who moved out of the class. The Kermack-McKendrick model is based on the following three assumptions:

Hypothesis 1: Set the total population to \( N(t) \), then there is \( N(t) = S(t) + R(t) + I(t) \). The population dynamics factors such as infection, death, and migration are not considered, which means that a closed environment is considered and it is assumed that the change of disease with time is more than that of infection and death. The latter can be ignored so that the total population \( N(t) \) of this environment always maintains a constant \( N(t) = S(t) + R(t) + I(t) = C \): KM's SIR model is very simple and rough.

Hypothesis 2: Once a patient comes into contact with a susceptible person, it must have a certain degree of contagion. We assume that the number of susceptible persons a patient can infect per unit time is proportional to the total number of susceptible persons in this environment, \( S(t) \) the proportional
coefficient is \( \beta \) so that the number of people infected by all patients (that is, the number of new patients) in a unit time at a time is \( \beta S(t)R(t) \).

Hypothesis 3: at time \( t \), the number of people removed from the infected person per unit time is proportional to the number of patients, and the proportionality coefficient is \( \gamma \), so the number of people removed per unit time is \( \gamma I(t) \). Is the number of patients removed per unit time the proportion is called the removal rate coefficient, which is also referred to as the removal rate when it is not confusing. When the removed person includes the rehabilitation person, the removal rate is also called the recovery rate coefficient or simply the recovery rate [2]. Under the above assumptions, it’s the process is shown in Figure 1:

**Fig.1** Block diagram of the basic model of SIR infectious diseases

2.2. Establishment of the SIR model of infectious diseases

2.2.1. Model overview. Consider the SIR model with infections and deaths, let \( N \) be the total population, \( S \) is the susceptible class and its number, \( I \) am the infected person and its number, \( R \) is the removed person and its number. Then the total population satisfies the equation

\[
N = S + R + I
\]

Further hypothesis: during the epidemic period, the population’s infection rate coefficient and natural mortality coefficient are equal, and its value is \( \mu \). The mortality rate coefficient due to disease is \( \varepsilon \). The incidence of the disease is bilinear \( \beta SI \). The recovery rate coefficient is \( \gamma \) The infected person is no longer infected after moving out. Figure 2 shows the block diagram of the epidemic of infectious diseases.

**Fig.2** Epidemic block diagram of infectious diseases with death due to disease

2.2.2. Establishment of the SIR epidemic model. From the above analysis, the following SIR warehouse model is obtained:

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta SI - \mu S, \\
\frac{dI}{dt} &= \beta SI - (\mu + \varepsilon + \gamma)I, \\
\frac{dR}{dt} &= \gamma I - \mu R.
\end{align*}
\]

(2)

The first two equations do not contain \( R \), so only the first two equations need to be discussed.
\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta SI - \mu S, \\
\frac{dI}{dt} &= \beta SI - (\mu + \gamma) I
\end{align*}
\] (3)

The basic regeneration number is \( R_0 = \frac{\beta \Lambda}{(\mu + \gamma) \mu} \). Equation (2) always has a disease-free equilibrium point \( E_0 = \left( \frac{\Lambda}{\mu}, 0 \right) \). When \( R_0 > 1 \), there is a positive equilibrium point \( E_* = (S^*, I^*) = \left( \frac{\mu + \gamma}{\beta}, \frac{\beta \Lambda - \mu (\mu + \gamma)}{\beta (\mu + \gamma)} \right) \).

3. Infectious disease SIR model with genetic variation

Infectious diseases are usually caused by viruses or bacteria. The spread of infectious diseases caused by bacteria often conforms to SIS, SIRS, and other infectious disease models, while the spread of infectious diseases caused by viruses often conforms to SI, SIR, and other infectious disease models. There have been a lot of research results. In recent years, people have gradually introduced some prevention and control measures for disease transmission into infectious disease models for analysis and research. International research on the dynamics of infectious diseases has progressed rapidly, and a large number of mathematical models have been used to analyze various infectious diseases. Most of these mathematical models apply to the study of the general laws of various infectious diseases [3].

3.1. Related parameters

The parameters introduced in the infectious disease model with genetic variation are as follows: \( A \) represents the constant input rate to the population. \( d > 0 \) represents the natural mortality rate of the population. \( \rho \geq 0 \) represents the genetic variation rate of infectious diseases of susceptible people. \( \beta \geq 0 \) represents the transmission coefficient of the disease. \( \gamma \geq 0 \) represents the recovery rate of the infected person. \( \alpha \geq 0 \) represents the mortality rate of the infected person. \( Q(t)(t \geq 0) \) represents the probability that the genetic variant of the infectious disease will still have immunity within the period \( t \) before returning to the susceptible class after losing immunity. According to the assumption, there is

\[
0 \leq \int_{0}^{t} S(u) Q(t-u) e^{-\mu (t-u)} du
\] (4)

Where \( V(t) \) represents the number of individuals with genetic variation of infectious diseases that have immunity from time 0 to time \( t \). The function \( V'(t) \) is a non-negative, monotonic, non-increasing piecewise continuous function. If the function \( Q(t) \) is an exponential function, \( Q(t) = \exp(-\mu t)(\mu > 0) \). Then it means that the infectious disease genetic variant moves out of the infectious disease genetic variation class (V) and enters the susceptible class (S) at the rate of \( \epsilon V \), and the infectious disease genetic variation person has an average immune period \( 1/\epsilon \). At this time, the corresponding \( V'(t) = V_0(t) \exp\{-\epsilon(\mu + \gamma)t\} \). Therefore, the integral equation (1) Corresponding to the ordinary differential equation

\[
V' = pS - (d + \epsilon)V
\] (5)

If the function \( Q(t) \) is a step function, that is, \( Q(t) = 1 \) on \([0, \tau)(\tau > 0)\) and \( Q(t) = 1 \) on \([\tau, +\infty)\), where \( \tau \) is the immunization period. This step function \( Q(t) \) means that the individual with genetic variation of the infectious disease at the initial moment no longer has immunity after the moment \( \tau \), Therefore, when \( t \geq \tau \), the integral equation (1) becomes

\[
V(t) = p \int_{0}^{t} S(u) e^{-\epsilon(t-u)} du
\] (6)

Where \( e^{-\epsilon(t-u)} \) represents the probability that an individual with genetic variation of infectious disease at time \( u \) is still alive at time \( t \). To ensure the continuity of \( V(t) \), assume
Then the integral equation (7) is equivalent to the delay differential equation
\[ V'(t) = pS(t) - pS(t - \tau)e^{-d\tau} - dV \]  
(8)

Thus, corresponding to the two forms of \( \hat{Q}(t) \), the SIR-V model with bilinear infection rate is
\[
\begin{align*}
S' &= A - dS - pS - \beta SI + pS(t - \tau)e^{-d\tau} \\
I' &= \beta SI - (d + \alpha + \gamma)I \\
V' &= pS - dV - pS(t - \tau)e^{-d\tau} \\
R &= \gamma I - dR
\end{align*}
\]
(9)

In system (9), the first two equations do not explicitly contain variables \( V(t) \) and \( Q(t) \), so the dynamic behavior of (5) can be determined by the following delay differential system
\[
\begin{align*}
S' &= A - dS - pS - \beta SI + pS(t - \tau)e^{-d\tau} \\
I' &= \beta SI - (d + \alpha + \gamma)I \\
V' &= pS - dV - pS(t - \tau)e^{-d\tau} \\
R &= \gamma I - dR
\end{align*}
\]
(10)

According to the actual background, assume that the initial condition of (9) is
\[ (S(0), I(0)) \in C([-\tau, 0], R_{+}^{2}), \phi(0) > 0, \phi_{2}(0) \geq 0 \]
(11)

It is easy to know that there is a unique solution to \( t \geq 0 \) under the initial conditions (8)

3.2. Stability analysis of an epidemic model with genetic variation

3.2.1. Analysis of differential systems with delay. The Yizhi system (9) has a positively invariant set
\[ D = \{S, I\} : S > 0, I \geq 0, S + I \leq \frac{A}{d} \} \]. At the same time, the following conclusions can be obtained:
\[ R_{1} = \frac{\beta A}{(d + \alpha + \gamma)(d + p(1 - e^{-\tau}))} \]
(12)
The system (9) always has a disease-free balance point
\[ P_{1}(S_{1}, I_{1}) = \left( \frac{A}{d + p(1 - e^{-\tau})}, 0 \right) \]
(13)

There is only one endemic disease balance point when \( R_{1} > 1 \)
\[ P_{2}(S_{2}, I_{2}) = \left( \frac{d + \alpha + \gamma}{\beta}, \frac{A}{d + \alpha + \gamma}(1 - \frac{1}{R_{1}}) \right) \]
(14)

\( P_{1} \) is globally asymptotically stable on \( D \) when \( R_{1} \leq 1 \); \( D \) is globally asymptotically stable on \( P_{2} \) when \( R_{1} > 1 \). For the system (9), the existence of the equilibrium point can be known directly. The global stability of the disease-free equilibrium point \( P_{1} \) is discussed below.
\[ x = S - S_{1} \]
(15)

Then the system (9) becomes
\[
\begin{align*}
x' &= -(d + p)S - \beta Sx - \beta SI + px(t - \tau)e^{-d\tau} \\
I' &= [\beta S_{1} - (d + \alpha + \gamma)]I + \beta Sx
\end{align*}
\]
(16)
definition
\[ W_{1} = \frac{x^{2}}{2} + S_{1}I \]
(17)

Then
\[
\frac{dW_1}{dt} = -(d + p)x^2 - \beta x^2I + pe^{-\xi t}x(t)x(t-\tau)I
\]

\[
W_1 = W_1 + \frac{pe^{-\xi t}}{2} \int_{t-\tau}^{t} x^2(u)du
\]

\[
\frac{dW_1}{dt} \leq -(d + p(1-e^{-\xi t}))x^2 - S_0[(d + \alpha + \gamma) - \beta S_1]I
\]

Note that \( R_i \leq 1 \) is equivalent to \( d + \alpha + \gamma \leq \beta S_i \), so \( \frac{dW_1}{dt} \leq 0 \) when \( R_i \leq 1 \), \( P_i \) is globally asymptotically stable on \( D \) by Lasalle invariant set principle.

### 3.2.2. Stability analysis of local equilibrium points.

The global stability of the endemic disease equilibrium point \( P_i \) is discussed below.

1) Make variable substitution \( x = S - S_2 \),

Then the system (9) becomes

\[
\begin{align*}
\dot{x} &= -(d + p)S - \beta Sx - (d + \alpha + \gamma)(I - I_2) + px(t-\tau)e^{-\xi t} \\
I &= \beta Sx 
\end{align*}
\]

(19)

2) Definition

\[
W_i = \frac{x^2}{2} + \frac{(d + \alpha + \gamma)}{\beta}(I - I_2 - I_1 \ln \frac{I}{I_2})
\]

(20)

Then

\[
\frac{dW_i}{dt} = -(d + p)x^2 - \beta x^2I + pe^{-\xi t}x(t)x(t-\tau)
\]

\[
\leq -(d + p)x^2 + \frac{pe^{-\xi t}}{2}[x^2(t) + x^2(t-\tau)]
\]

(21)

3) Define the Lyapunov functional

\[
W_i = W_1 + \frac{pe^{-\xi t}}{2} \int_{t-\tau}^{t} x^2(u)du
\]

(22)

Then

\[
\frac{dW_i}{dt} \leq -(d + p(1-e^{-\xi t}))x^2 \leq 0
\]

(23)

Therefore, when \( R_i > 1 \) is the endemic disease equilibrium point \( P_i \) is globally asymptotically stable.

### 4. Case analysis

This part takes a sudden outbreak of an infectious disease in a boarding school as an example.

The empirical method of determining the parameters of the SIR model, in a boarding school in the UK, the whole school

The number \( N=763 \). In an outbreak of infectious disease, more than 500 people have been infected in the school within 14 days (no deaths). Infected people will be forced to suspend classes. Because it is a boarding school, the total population is relatively stable. Meet the basic assumptions of the SIR model [4].

| date | Number of patients | Proportion of patients | Proportion of patients |
|------|--------------------|------------------------|------------------------|
| 1    | 3                  | 1/763                  | 0.0013                 |
| 2    | 6                  | 3\*763                 | 0.0039                 |
| 3    | 25                 | 6/763                  | 0.0079                 |
| 4    | 73                 | 25/763                 | 0.0328                 |
| 5    | 21                 | 73/763                 | 0.0957                 |
| 6    | 94                 | 221/763                | 0.2896                 |
The implementation of the algorithm here is also based on the previous parameter assumptions in the article, the parameter vector \( \theta = (\beta, \gamma) \in [0, 10]^2 \), which is accurate to three decimal places when the parameter accuracy is determined; the combination of path update and parameter update adopted in the literature The results obtained are for the control reference group, the model's parameter estimates under this method are \( \hat{\beta} = 1.82, \hat{\gamma} = 0.49 \) the confidence interval for the infection coefficient confidence level is 95% is \([1.63, 2.02]\), and the confidence interval for the recovery coefficient confidence level is 95% is \([0.45, 0.52]\), its estimated basic regeneration rate.

\[
R_0 = \frac{\hat{\beta}}{\hat{\gamma}} = \frac{1.82}{0.49} = 3.7143 \tag{24}
\]

Figure 3 shows a scatterplot of the proportion of patients and a simulation result of the path simulation based on the ODE path simulation. The value corresponding to the horizontal line in the figure is \( \hat{\beta} = 0.27 \), and the value corresponding to the vertical line segment corresponds to the specific time point of the turning point and the turning point. It shows that the proportion of susceptible people has dropped to the level of relative removal rate, and the proportion of infected people has reached the peak at the moment and began to gradually decrease (the above is a simple summary analysis of the reality reflected in the data itself based on the results of existing models):

![Fig.3](image)

**Fig.3** The proportion of affected patients and the effect of ODE path simulation

The following two figures (Figure 4, Figure 5) are the parameter inversion results obtained when the initial population number is \( N = 20, 10 \). In this example, although the data dimension is low and the amount of data information is low, the GA algorithm is still a good parameter estimation result is achieved; when the initial population number is selected as \( N = 20 \), in the actual inversion process, the algorithm can obtain a stable convergence solution \((\hat{\beta}, \hat{\gamma}) = (1.666, 0.448)\) of the parameters when the population has evolved to the 70th to 80th generations; when the initial When the number of populations is selected as \( N = 10 \), in the actual inversion process of the algorithm, the stable convergence solution of the parameters can also be obtained when the populations evolve to the 150th generation or so \((\hat{\beta}, \hat{\gamma}) = (1.666, 0.448)\).

![Fig.4](image)

**Fig.4** Initial population \( N = 20 \)
The inversion values of the parameters obtained in the two cases are consistent, and the estimated results have a certain stability. The obtained parameters are slightly different from the literature, but the basic regeneration rate of the parameters obtained under the algorithm is solved [5].

\[ R_0 = \frac{\hat{\beta}}{\gamma} = \frac{1.666}{0.448} = 3.7187 \]  \hspace{1cm} (25)

It is consistent with the basic regeneration rate of 3.7143 obtained in, and the relative error is about 0.12%. The reference value in the specific control and treatment of the disease is also basically consistent. It is considered that the GA algorithm has achieved very good parameters to Estimate the effect. The table presents the parameter estimation results obtained by the algorithm used in [6] and the algorithm used in this paper:

| Algorithm name                | OLS          | Bayes posterior estimation | GA          |
|-------------------------------|--------------|----------------------------|-------------|
| Achieve results \((\beta, \gamma)\) | (1.67,0.45)  | (1.82,0.49)                | (1.666, 0.448) |
| Confidence interval \(\beta\)  | no           | [1.63,2.02]                | no          |
| Confidence interval \(\gamma\)| no           | [0.45,0.52]                | no          |
| Basic regeneration rate       | 3.7111       | 3.7143                     | 3.7187      |

As can be seen from the table, the estimation results obtained by using the GA algorithm in this paper ((1.666, 0.448) are the same (this is related to the selection of the fitness function in the text), indicating that the GA algorithm also implements the parameter estimation of the SIR model. It has certain stability and practicability; and the algorithm is convenient to get started, even in high-dimensional parameter estimation, it has considerable advantages, so this paper believes that the GA algorithm can be widely applied to similar infectious disease dynamic models Parameter estimation. In the end, it is worth affirming that any model or algorithm is just a simplified simulation of a real problem, limited to various assumptions in the modeling process. We must start from a variety of methods for comprehensive analysis of any problem, the results obtained Sexual parameters give in-depth explanations and multifaceted parameter references, and should not be rushed to be used directly for practical problem guidance [7].

5. Conclusion

This article has also studied a SIR infectious disease model with genetic variation. Most infectious diseases such as smallpox, flu, hepatitis, measles, etc. have strong immunity after being cured, so the people who are cured are not healthy (susceptible Person), not a patient (infected person), they have withdrawn from the Department of Infectious Diseases, and, for many epidemics, susceptible people are vaccinated, but the classic SIR model does not consider this situation. In this regard, a SIR infectious disease model with vaccination was built based on the classic SIR model, and its asymptotic stability was proved through theoretical analysis. The disease-free balance point and endemic disease balance point of the model were discussed. Through the linearization method and Lyapunov function, the following conclusions are obtained: There is a disease-free equilibrium point \( P_1(S, I) = \left( \frac{A}{d + p(1 - e^{-\tau})}, 0 \right) \),

and there is also a unique endemic equilibrium point \( P_2(S, I) = \left( \frac{d + \alpha + \gamma}{\beta}, \frac{A}{d + \alpha + \gamma(1 - \frac{1}{R_0})} \right) \) when \( R_1 > 1 \). When \( R_1 \leq 1 \) is \( P_1 \), it is globally asymptotically stable on \( D \), and when \( R_1 > 1 \) is \( P_2 \), it is globally
asymptotically stable. Compared with traditional statistical methods, this model can better understand some global behaviors in the popular process.

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