A Class of Epidemic Models Based on Ornstein-Uhlenbeck Process

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Abstract: The paper will discuss some classical epidemic models with the Ornstein-Uhlenbeck process. The mechanism of infectious disease models works in different ways. The influential factors involve population, psychology, environment, climate and so on. Especially, the parameters in models follow some certain assumptions, the Ornstein-Uhlenbeck process. In addition, the Ornstein-Uhlenbeck process was introduced into dynamic models to describe the transmission pattern. Finally, the simulation will provide to illustrate the correctness of the theoretical results. The summary of different models will provide the information for further study about the spread and control of epidemic diseases.

Keywords: Epidemic models, infectious diseases, Ornstein-Uhlenbeck process, partial differential equation, parameters

1. Introduction

Throughout history, humans and viruses have had several rounds of confrontations, and each virus outbreak has claimed countless lives. Numerous people died, but the virus never stopped. Infectious diseases are a major threat to human health, and the outbreak of infectious diseases is often accompanied by social panic and economic crisis. Since SARS or even earlier, people have used mathematical models to predict the development of infectious diseases, and have achieved good results. However, the outbreak of COVID-19 at the end of 2019 has caused earth-shattering changes in the world. Therefore, the establishment of infectious disease prediction models can not only effectively control the epidemic and maintain social stability, but also apply the models to other fields with similar transmission methods to infectious diseases, such as network information dissemination, epidemiology and environmental science.

The Ornstein-Uhlenbeck process plays an important role in epidemic and disease models. It has been studied by many scholars at home and abroad. A variety of epidemic models with many special features have also been studied in the literature. They provide us with different perspectives to understand the epidemic model and related innovations. About a century ago, Kermack and Mckendrick laid the foundation for the control and dynamics of epidemic diseases, they proposed the epidemic disease definite model, the SIR model. Korobeinikov and Wake [1] introduced Lyapunov functions for classical SIR, SIRS, and SIS epidemiological models. Li *et al.* [2] established a class of SIRS epidemic models based on psychological effects. In addition, Wang *et al.* [3] put forward a stochastic differential equation SIS epidemic model with the Ornstein-Uhlenbeck process. Fang *et al.* [4] study the spread trend of infectious diseases by a dynamic SEIR model based on the Susceptible Exposed Infectious Removed (SEIR) model. Recently, Olusegun [5] has been

interested in the analysis of multi-strain infection of vaccinated and has recovered population through the epidemic model. Corresponding results can also be found in Sun [6], Gong and Yang [7] and some others.

Since ancient times, humans have been plagued by epidemics. This project mainly studies a kind of stochastic prediction model containing the Ornstein-Uhlebeck process, and the model considers the possibility of multiple factors, such as environment, psychology, population and others. In addition, the author summarizes the application scope and advantages and disadvantages of different models, and makes a comparative analysis of the development trend of infectious diseases, to fully understand the transmission mechanism and disease control theory of infectious diseases. This project achieves the optimal control solution based on computer simulation and understanding of the prediction of infectious disease transmission mechanisms.

Diseases are affected by a variety of factors in the process of infection, such as population density, psychological factors, environmental factors, medical level, etc. Therefore, the uncertainty, limitation and complexity of transmission pathways lead to the diversity of models, so that predictive models and multivariate analysis provide a strong theoretical basis for disease control and prevention [8].

2. The Summary of Epidemic Models

In this section, we will consider several classical epidemic models. Modeling infectious diseases at different stages can capture the rich dynamics relationships that exist in interdependent diseases. The key to modeling infectious diseases is to divide the population into several easily monitored stages according to the stages of infection and to find the dynamic relationship between the parts. This idea of dividing the whole part into several parts to find its dynamic connotation applies to many modeling problems.

Research methods mainly through mathematical model research, mathematical model research is to simulate the trend and development process of disease epidemic through mathematical modeling, especially the analysis of the inflection point and peak period, to provide services for disease prevention and control and the formulation of health policies. Common infectious disease models can be divided into the following types according to the types of infectious diseases: SI, SIS, SIR, SEIR, and according to the transmission mechanism, they can be divided into different types based on the ordinary differential equation, partial differential equation, and network dynamics equation .

These models use mathematical equations to describe the dynamics of disease spread. They can be simple or complex, depending on the level of detail required to capture the specific characteristics of a disease. Epidemic models can be used to estimate parameters such as the basic reproduction number (R_0), which is the average number of secondary infections caused by an infected individual in a fully susceptible population. By adjusting the parameters and assumptions in these models, researchers can simulate different scenarios and evaluate the potential impact of different interventions in controlling an epidemic [9].

The basic reproduction number (R_0) implies the number of secondary infections in an otherwise susceptible population caused by a single initial individual. It is a widely used estimate of the severity of an epidemic outbreak and quantifies the transmission potential of a disease. A R_0 of 1 or less indicates that the outbreak is coming to an end, as it can produce less than its initial number over time. A value greater than one indicates a growing epidemic, producing more than its current number. The infectious disease is dynamic and changes over time, eventually decreasing. Gross estimates of R_0 for COVID-19 ranged from 2.2 to 2.7. The UK strain's R_0 is also higher than that of other strains.

- The basic transmission number of COVID-19 is 5.7, and if R_0 is less than 1, the infection will gradually disappear.
- If $R_0 > 1$, the infection will spread exponentially and become an epidemic. However, it won't last

forever, as the number of people likely to be infected slowly decreases. Some of the population may die from the infection, while others may recover and develop immunity.

- If R_0 = 1, the infectious disease becomes endemic in the population. The higher the R_0 number, the harder it is to control the epidemic. Omicron, a variant of COVID-19 and by far the most transmissible virus, has an estimated mean R_0 value of 9.5, which is considered higher than the delta of 5.08. Measles has the highest base number of infections, between 16 and 18, HIV and Ebola have relatively low R_0, and smallpox is between 5 and 7.

2.1. Model 1 SI

The SI model is the simplest form of all disease models. Individuals are born into the simulation with no susceptible. Once infected and with no treatment, individuals stay infected and infectious throughout their life, and remain in contact with the susceptible population.

$$0 = S'(t) + I'(t) \tag{1}$$

$$S'(t) = -\lambda IS, S(0) = S_0 > 0 \tag{2}$$

$$I'(t) = \lambda IS - \alpha I, I(0) = I_0 \geq 0 \tag{3}$$

where $S(t)$ is the susceptible and $I(t)$ is the infected. But the infected can't be recovered. S_0 and I_0 are initial values. The HIV virus is a typical example of the SI model. The constants λ and α are the transmission rate and removal rate, average values are all greater than 0. Suppose let $\sigma = \frac{\lambda}{\alpha}, \frac{1}{\sigma}$ is the relative removal rate.

The equation cannot be solved for $S(0)$ and $I(0)$, but we can evaluate the different values of λ, α, S_0, and I_0 by Matlab, then we get the trend of the curve for S_0 and I_0. This model is not very widely applicable, because the infected cannot be cured.

- The transmission rate depends on the relative level between the transmission rate of the patient and the cure rate of the patient, and if the cure rate of the patient is greater than the transmission rate of the patient, then the infection will eventually disappear;
- Even if patients are spreading faster than they can be cured, only a fraction of the population will eventually become infected;
- The final proportion of infected people is $1 - 1/\sigma$, $\sigma = \rho/\mu$, σ is the effective contact number of each patient during the entire infection period, which can be understood as the total number of patients in contact with the entire illness period.

The Equilibrium solution is

- Unstable solution: $I(0) = 0, S(0) = P$
- Stable solution: $I_\infty = N, S_\infty = 0$

2.2. Model 2 SIS

The SIS model developed on the basis of the SIR model takes into account that the infected person can be cured and become susceptible. The contact number in the infectious period on the surface of the SIS model is an important indicator of the spread, prevention and control of infectious diseases, which determines whether the infectious diseases will eventually be zero or develop into local long-term diseases. Common cold, and dysentery can be categorized by the SIS model.

$$0 = S'(t) + I'(t) + S'(t) \tag{4}$$

$$S'(t) = \mu P - \frac{\beta SI}{P} + \gamma I - vS, S(0) = S_0 > 0 \quad (5)$$

$$I'(t) = \frac{\beta SI}{P} - \gamma I - vS, I(0) = I_0 \geq 0 \quad (6)$$

If the susceptible person $S(t)$ comes into contact with the infectious person, he becomes the infected person $R(t)$, and the infected person becomes the susceptible person after treatment and recovery. This model is most commonly used for the common cold and dysentery. The infectious rate, β, controls the rate of spread which represents the probability of transmitting disease between a susceptible and an infectious individual. Recovery rate, $\gamma = 1/D$, is determined by the average duration, D, of infection. The birth rate is μ, μP is the newly-born population, $\frac{\beta SI}{P}$ is the number of the infected people [10].

The Equilibrium solution is

- Unstable solution: $I(0)=0, S(0) = P$
- Stable solution: $I_\infty = (1-\frac{\gamma}{\beta})P, S_\infty = \frac{\gamma}{\beta}P$

2.3. Model 3 SIR

Considering the basic and classical epidemic model, SIR model. The SIR model of infectious diseases is more appropriate for epidemics without incubation periods, and is not applicable to the COVID-19 outbreak in early 2020.

$$P(t) = S(t) + I(t) + R(t) \quad (7)$$

Where $S(t)$ is the susceptible, $I(t)$ is the infected, $R(t)$ is the recovered. $P(t)$ is the total number at time t. Model 3 means that the population has been divided into three groups. People who are immune don't play in it. The basic assumption for the SIR model is that $S(t)$, $I(t)$, and $R(t)$ are all continuous and differential functions. The virus has no incubation period, and the disease appears shortly after infection[11].

$$S'(t) + I'(t) + R'(t) = 0 \quad (8)$$

$$S'(t) = -\alpha SI, S(0) = S_0 > 0 \quad (9)$$

$$I'(t) = \alpha \mathrm{SI} - \beta I, I(0) = I_0 \geq 0 \quad (10)$$

$$R'(t) = \beta I(t), R(0) = R^0 = 0 \quad (11)$$

It assumes that individuals progress from susceptibility to infection, and then to recovery with immunity. This model is useful for diseases where recovery confers immunity.

$$I'(t) < 0 \Rightarrow \frac{\alpha S(t)}{\beta} < 1 \quad (12)$$

$$I'(t) > 0 \Rightarrow \frac{\alpha S(t)}{\beta} > 1 \quad (13)$$

$$I'(t) = \alpha S(t) I(t) - \beta I(t) = 0 \quad (14)$$

$$S(t) = \frac{\beta}{\alpha} \quad (15)$$

where $S(t)$ is a monotonically decreasing function. If $I'(t) = 0$, it shows that the infectious disease was at its worst. If $I'(t) < 0$, it shows that the epidemic began to gradually subside. The greater the value of $\frac{\beta}{\alpha}$, the sooner the epidemic will subside. In the lockdown and isolation at the same time, the use of scientific research and intensive care methods to improve the cure rate and will lower the α and β, therefore the value of $\frac{\beta}{\alpha}$ will become greater, the turning point of the epidemic is as early as possible.

The Equilibrium solution is

- Unstable solution: $I(0) = 0, S(0) = P$
- Stable solution: $I_\infty = 0, S_\infty + R_\infty = P$

The following Fig. 1 shows an example simulation of this model.

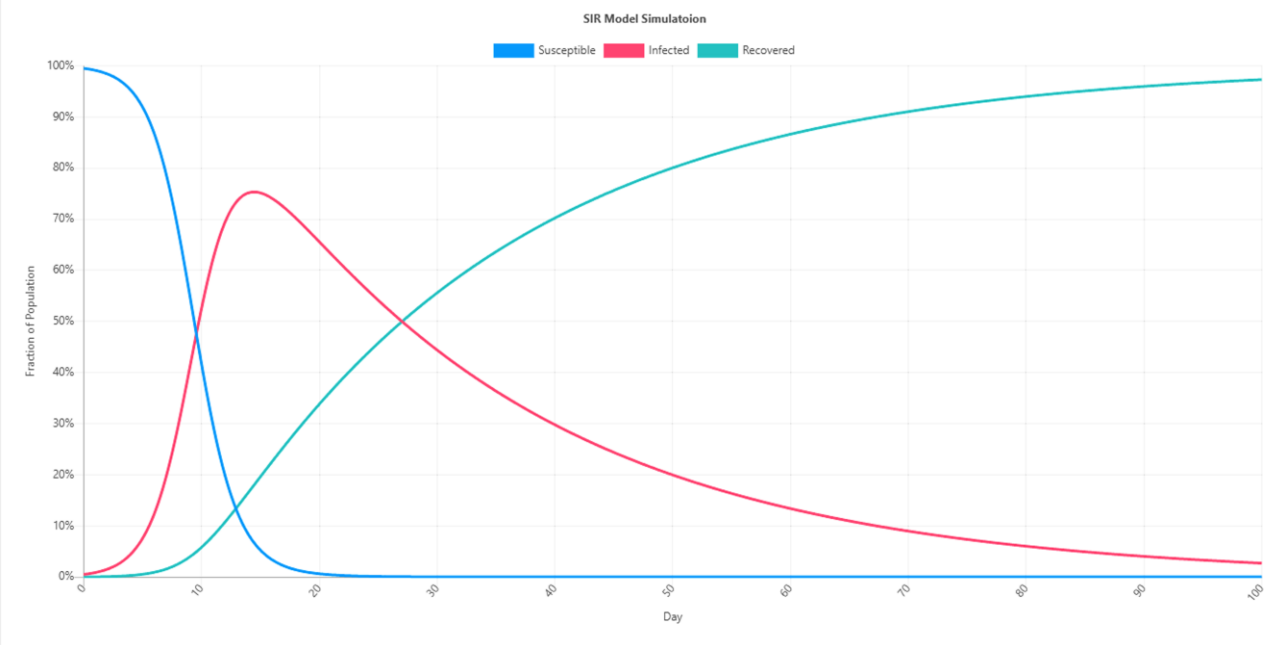

Fig. 1. SIR model.

A typical SIR Model simulated solution. This result is obtained with the following parameters: $\beta = 0.02$, $\alpha = 0.22$, $days = 100$, and the initial values: $I(0) = 0$, $R(0) = 0$.

2.4. Model 4 SEIR

Based on the classic SIR model, the derived SEIR model adds susceptible patients, mainly for patients with incubation period and common infectious diseases, making it more suitable for simulating the new coronavirus

$$P(t) = S(t) + E(t) + I(t) + R(t) \tag{16}$$

where $E(t)$ represents the number of people with an incubation period, SEIR is more appropriate for the COVID-19 outbreak. $P(t)$ represents the number of total population at time t. $S(t)$, $E(t)$, $I(t)$, and $R(t)$ are all continuous and differentiable functions of time t.

$$S'(t) + E'(t) + I'(t) + R'(t) = 0 \tag{17}$$

$$S'(t) = -\beta SI/P + \sigma R \tag{18}$$

$$E'(t) = \beta SI/P - \alpha E \tag{19}$$

$$I'(t) = \alpha E - \gamma I \tag{20}$$

$$R'(t) = \gamma I - \sigma R \tag{21}$$

where the parameter α determines the average retention time of immunity obtained by recovered individuals. The system has two fixed points: $S = P$ ($I = R = 0$) or $S = \gamma/\beta(I/R = \alpha/\gamma)$. The former indicates that the disease is eradicated from the studied area, while the latter represents the epidemic status. The parameter condition for eliminating the epidemic is $\gamma > \beta P$. If this cannot be achieved, it is necessary to minimize α and increase γ as much as possible to enable more people to maintain immunity against the disease.

A typical SEIR Model simulated solution. This result is obtained with the following parameters: $\beta = 0.5$, $\sigma = 0.00549$, $\gamma = 0.14286$, $\alpha = 0.1$, $days = 365$, and the initial values: $I(0) = 0$, $R(0)=0$. Fig. 2 shows that the Basic Reproduction Number, R_0 is 3.5 since Jan. 2020.

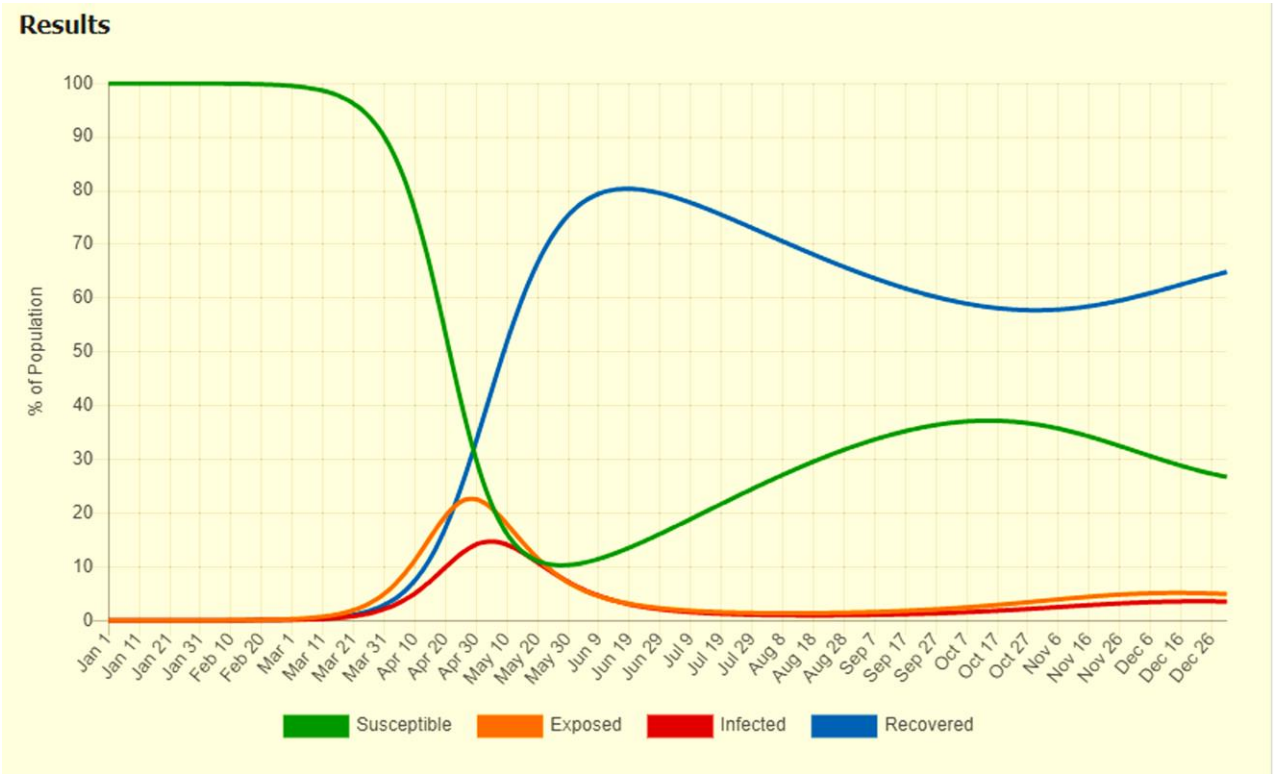

Fig. 2. SEIR graphing.

The SEIR model extends the SIR model by including an exposed (E) compartment for individuals who have been exposed to the disease but are not yet infectious. This is particularly relevant for diseases with an incubation period.

The transitions between compartments in the SEIR model are governed by parameters such as the transmission rate, the rate at which exposed individuals become infectious, and the recovery rate. These parameters are used to describe the dynamics of disease spread and can be adjusted according to the specific characteristics of the disease being modeled.

Overall, the SEIR model is a valuable tool in epidemiology for studying the dynamics of infectious diseases and informs public health interventions to mitigate their impact.

2.5. Model 5 SIRS

The SIRS model applies to susceptible people, sick people and recovered people. Recovered people have only temporary immunity, and those who become susceptible after the unit time are likely to be re-infected again and become ill.

$$P(t) = S(t) + I(t) + R(t) \tag{22}$$

The model assumptions: The total number of population at time t is $P(t)$, which does not take into account for births and deaths of the population, migration and immigration, the total population does not change. $S(t)$ represents the susceptible. $I(t)$ are the infected. $R(t)$ is the recovered. A susceptible person is infected by effective contact with a sick person, becomes sick, can be cured and becomes susceptible again, has temporary immunity, no incubation period.

$$P'(t) = S'(t) + I'(t) + R'(t) = 0 \tag{23}$$

$$S'(t) = -\frac{\beta SI}{P} + \xi R, S(0) = S_0 > 0 \tag{24}$$

$$I'(t) = \frac{\beta SI}{P} - \gamma I, I(0) = I_0 \geq 0 \tag{25}$$

$$R'(t) = \gamma I - \xi R, R(0) = 0 \tag{26}$$

where β controls spreading rates, ξ means recovery rates, γ means infectious rates. When β is larger, the infection speed is greater. When it is larger, the recovery speed is faster, and the epidemic trend of the infectious disease will slow down. When γ is larger, the probability of reinfection of the susceptible people is higher, and the cycle of the infectious disease will be shorter [12].

The SIRS model overly simplifies the complexity of infectious disease transmission and neglects some real situations, such as population mobility, immunity changes and other factors. There are certain difficulties in parameter estimation and data collection in the model, resulting in a certain impact on the accuracy of the simulation results. The SIRS model may have poor application effects on certain specific types of infectious diseases and requires adjustment and improvement according to the actual situation.

2.6. Model 6 SEIRS

Now we will consider more complex disease transmission scenarios can be added to the model by introducing new groups and more complicated flows between them. The SEIRS model will allow us to model important aspects such as birth, death, loss of immunity and age.

$$P(t) = S(t) + E(t) + I(t) + R(t) \tag{27}$$

The SEIRS model with demography. Rates are β (contact), σ (latency), γ (recovery), ξ (loss of immunity), α (infection-induced death), and μ (birth and background death).

$$P'(t) = S'(t) + I'(t) + R'(t) = 0 \tag{28}$$

$$S'(t) = \mu P - \frac{\beta SI}{P} + \xi R - \mu S, S(0) = S_0 \geq 0 \tag{29}$$

$$E'(t) = \frac{\beta SI}{P} - \sigma E - \mu E, E(0) = E_0 \geq 0 \tag{30}$$

$$I'(t) = \sigma E - \gamma I - (\mu + \alpha)I, I(0) = I_0 \geq 0 \tag{31}$$

$$R'(t) = \gamma I - \xi R - \mu R, R(0) = 0 \tag{32}$$

where μP means birth cases, $\frac{\beta SI}{P}$ means infection cases, ξR means lost immunity cases, μS and $(\mu+\alpha)I$ means death cases, σE means latency, and γI means recovery cases.

The six models above are only the basic models of the spread of infectious diseases, and many factors are not considered, such as the birth and death of the population, migration and immigration. There are also more detailed factors that can be considered, such as the speed of population flow, the age distribution of susceptible people, the susceptibility of different groups to the disease, the severity of symptoms of infected people, population density, the level of medical care, the means of inspection and quarantine, the level of government attention, and the psychological factors of the population. These factors have a direct or indirect effect on the number of exposures, morbidity, cure rate, and duration of infection.

3. Main Results

The Ornstein-Uhlenbeck process, named after Leonard Ornstein and George Eugene Uhlenbeck, is a stochastic process that describes the velocity of a particle undergoing Brownian motion subject to a frictional force. It is commonly used in physics, economics, and finance to model mean-reverting behavior.

In some cases, the parameters in the models may satisfy the certain assumptions. The parameters in the infectious disease model satisfies Ornstein-Uhlenbeck process. The Ornstein-Uhlenbeck process is important in many areas, including:

- Statistical mechanics, where it originated,
- Mathematical finance, where it appears in the Vasicek model for the term structure of interest rates.
- It can protect actual systems, such as mechanical arms.

Firstly, the basic model for processes of this type is given by the (linear) stochastic differential equation, the Ornstein-Uhlenbeck Process is given by

$$\gamma_t = \gamma + (\gamma_0 - \gamma)e^{-kt} + \rho \int_0^t e^{-k(t-s)} dB_t \tag{33}$$

Whose solution is called the Ornstein-Uhlenbeck process with k and ρ. $B(t)$ is a Wiener process. The OU-process is time-dependent. The OU-process is time-dependent. The noise of OU-process does not have as large a difference in values between adjacent two steps as Gaussian noise. Instead, it will explore for a certain distance in the positive or negative direction around the mean value, like the fluctuations of commodity prices and interest rates. This is conducive to exploration in one direction[13].

Due to the random variability of the environment, white noise is often introduced to establish the dynamics model of infectious diseases. $r_0 = r(0)$, the expectation and variance of $r(t)$:

$$E(\gamma(t)) = \gamma + (\gamma_0 - \gamma)\, e^{-kt} \tag{34}$$

$$V(\gamma(t)) = \frac{\rho^2}{2k}(1 - e^{-2kt}) \tag{35}$$

Combining Eqs. (33), (34), and (35), $\rho \int_0^t e^{-k(t-s)} dB_t$ follow normal distribution $(0, \frac{\rho^2}{2k}(1 - e^{-2kt}))$, we obtain:

$$\rho \int_0^t e^{-k(t-s)} dB_t = \frac{\rho}{\sqrt{2k}} \sqrt{1 - e^{-2kt}} \frac{dB_t}{dt} \quad a.s. \tag{36}$$

Therefore, Eq. (33) can rewrite as

$$\gamma_t = \gamma + (\gamma_0 - \gamma) e^{-kt} + \sigma_t \frac{dB_t}{dt} \tag{37}$$

where $\sigma_t = \frac{\rho}{\sqrt{2k}} \sqrt{1 - e^{-2kt}}$ and B_t follow the Brownian motion [14].

Due to the randomness and variability of the Environment, we introduce the white noise into the dynamic differential equations. For example, in SIS model, $\gamma \longrightarrow \gamma + \sigma dB(t)$, we have

$$dS(t) = (\mu P - \frac{\beta SI}{P} + \gamma I - vS)dt + \sigma I dB(t) \tag{38}$$

$$dI(t) = (\frac{\beta SI}{P} - \gamma I - vI)dt - \sigma I dB(t) \tag{39}$$

Substituting Eq. (37) into Differential equations, Eqs. (5) and (6),

$$dS(t) = \left(\mu P - \frac{\beta SI}{P} + (\gamma + (\gamma_0 - \gamma)e^{-kt})I - vS\right) dt + \sigma I dB(t) \tag{40}$$

$$dI(t) = (\frac{\beta SI}{P} - (\gamma + (\gamma_0 - \gamma)e^{-kt})I - vI)dt - \sigma I dB(t) \tag{41}$$

where the initial value $I(0) = I_0$, when k goes to ∞, the stochastic model will eventually change to Eqs. (5) and (6).

The Ornstein-Uhlenbeck process with intercepts can be divided into three parts. There are long-term averages, average conversions, and Brownian motion. Among them, β, k, ρ are all constants, representing the rate of mean reversion, the long-term average, and the volatility, respectively. As time goes on, we expect the process to drift towards k, because as r_0 approaches, the damping decreases, and as s approaches t, the Brownian motion term is zero [15].

The process shows mean-reverting behavior as it tends to return to the mean μ over time due to the term $(k(\mu - X_t)dt)$. The term (σdB_t) represents the stochastic component due to randomness in the system.

The Ornstein-Uhlenbeck process has applications in various fields, including physics to model the motion of particles in a medium, finance to model interest rates or asset prices that tend to revert to a long-run average, and neuroscience to model the behavior of neurons that fire in response to stimuli.

4. Conclusion

The key to establishing an infectious disease model is to divide the population into several easily monitored parts according to the stage of infection, and to find the dynamic relationship between the parts. Many scholars at home and abroad have revised and innovated the model according to different research objects, which can be applied to the fields with common characteristics of infectious diseases.

The significance of the infectious disease model lies in the mechanism, prediction, prevention and control guidance. The change of parameters depends on the intensity and measures of epidemic prevention and control in reality.

Limited by the model principle and data, this study has certain limitations. The results of this study confirm that the model is reliable in analyzing of infectious disease situation, which can provide theoretically support for formulating epidemic prevention strategies in the future. However, this paper does not consider

the impact of measures such as prevention and control isolation, vaccination and asymptomatic infected persons on the epidemic situations, which will inevitably differ from reality and will be considered in further studies.

In this study, the basic models of several infectious diseases are established according to the general transmission mechanism. The transmission process of different types of infectious diseases has its own different characteristics, and to clarify these characteristics requires considerable pathological knowledge, so it is impossible to analyze the transmission of various infectious diseases from a medical point of view. The establishment of mathematical models of infectious diseases, the description of the transmission process of infectious diseases, the analysis of changes in the number of infected people, and the exploration of means to stop the spread of infectious diseases has been the subject of concern for experts and officials in various countries.

Conflict of Interest

The author declares no conflict of interest.

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