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Research article

Stochastic dynamical behavior of COVID-19 model based on secondary vaccination

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Abstract: This paper mainly studies the dynamical behavior of a stochastic COVID-19 model. First, the stochastic COVID-19 model is built based on random perturbations, secondary vaccination and bilinear incidence. Second, in the proposed model, we prove the existence and uniqueness of the global positive solution using random Lyapunov function theory, and the sufficient conditions for disease extinction are obtained. It is analyzed that secondary vaccination can effectively control the spread of COVID-19 and the intensity of the random disturbance can promote the extinction of the infected population. Finally, the theoretical results are verified by numerical simulations.

Keywords: stochastic model of COVID-19; brownian motion; global positive solution; disease extinction; secondary vaccination

1. Introduction

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by the severe acute respiratory syndrome coronavirus which can lead to severe sequelae or death due to breathing difficulties, headaches and loss of taste or smell [1, 2]. Since the outbreak of COVID-19 in 2019, it has seriously affected the development of society and people's lives all over the world. In order to control COVID-19 as soon as possible, mathematical researchers have studied the infectious disease by establishing mathematical models for COVID-19 and analyzed their dynamical behavior [3–8].

The mathematical model of infectious disease was first proposed by Kermack and McKendrick [9]. They considered a fixed population with only three parts, i.e., SIR: the susceptible population $S(t)$, the infected population $I(t)$ and the recovery population $R(t)$. Since then, many improved models, such as $SEIR$ and $SIRS$ models, have been used to describe various properties and

laws of disease transmission [10–15]. Batabyal [16] built an $SEIR$ model based on the seasonal transmission nature of the virus, analyzed the stability of the model and obtained the influence of the parameters on the basic regeneration number. Gumel et al. [17] proposed extended models of the infectious disease in which the stability of the equilibrium point and parameter estimation was studied. The global properties of an SIR model with nonlocal disperse and immigration effects were studied in [18]; they evaluated the persistence, extinction of the disease, global stability analysis of the model, existence of the positive equilibrium state and its uniqueness. The above deterministic models have well revealed the mechanism of infectious diseases. In fact, due to the disturbance of uncertain factors such as the environment, temperature, virus variation, prevention and control measures, the COVID-19 model with random factors has gradually attracted more and more attention [19–23]. Sweilam et al. [24] considered environmental noise and fractional calculus in the COVID-19 model which concluded that random factors have a considerable impact on the demise of infection. In a stochastic SIQ model affected by white noise, the sufficient conditions for stable distribution and disease extinction were proved by Din et al. [25]. Khan et al. [26] described a stochastic $SEQIR$ model disturbed by white noise and telegraph noise at the same time; the uniqueness of global positive solutions and random thresholds was obtained. The stochastic coronavirus disease model with jump diffusion has also been researched, and Tesfay et al. [27] found that random disturbance can inhibit the outbreak of disease better than the deterministic model.

As we all know, vaccination can improve the level of immunity of the population and control the spread of the epidemic [28–31]. Djilali and Bentout [32] investigated an $SVIR$ system with distributed delay and drew the conclusion that increasing the number of people vaccinated will reduce the spread of the disease. Zhang et al. [33] proposed a stochastic $SVIR$ model based on one vaccination and got the sufficient conditions for the existence of non-trivial periodic solutions. A basic qualitative analysis of the positivity, invariant region and stability of disease-free equilibrium points was performed for a stochastic $SVITR$ model with vaccination [34]. Wang et al. [35] constructed a stochastic COVID-19 mathematical model with quarantine, isolation and vaccination; they also studied the influence of vaccination rates, vaccine effectiveness and immune loss rates on COVID-19. Omar et al. [36] studied a discrete time-delayed influenza model with two strains and two vaccinations, and then concluded that the vaccination of one strain would affect the disease dynamics of the other strain. Alshaikh [37] generated a fractional order stochastic model based on the secondary vaccination and analyzed various vaccination strategies. With the development of current medical technology and research, more and more of the population has completed a secondary vaccination, even the third vaccination. Based on the above references, although there have been extensive studies and application of COVID-19, it should be pointed out that the complexity modeling of COVID-19 is still not enough, and that the mechanisms of transmission for the complex model are unclear. Based on this, the research on the transmission dynamics characteristics of stochastic COVID-19 epidemics with secondary vaccination is very necessary.

In this paper, a stochastic disease model with secondary vaccination is established in Section 2. In Section 3, we apply random Lyapunov function theory to study the qualitative characteristics. In the last section, numerical simulations are applied to verify the theoretical results.

2. Model building

In this study, we divided the population into seven time-dependent categories and built a mathematical model of COVID-19 with secondary vaccination (see Figure 1) based on the current situation of the epidemic. That is, it includes the susceptible person $S(t)$, exposed person $E(t)$, first vaccinator $V_1(t)$, second vaccinator $V_2(t)$, asymptomatic infected person $A(t)$, symptomatic infected person $I(t)$ and recovering person $R(t)$.

In order to obtain the required results, we present the following assumptions:

1) The recovered population has immunity and will not be infected again.

2) Most people who receive the second vaccination will have immunity and will not be infected again.

3) Vaccinated people will transfer to exposure after being infected.

4) Assuming that the interval between two vaccinations is short, the first vaccinated person will not be infected temporarily.

The model is shown in Eq (2.1):

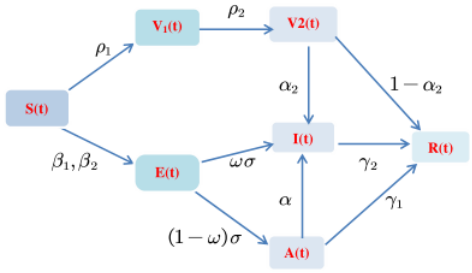


Figure 1. Model of COVID-19 .

$$\begin{cases} \dfrac{dS}{dt} = \Lambda - \beta_1 S A - \beta_2 S I - \mu_1 S - \rho_1 S, \\ \dfrac{dE}{dt} = \beta_1 S A + \beta_2 S I + \alpha_2 \beta_2 V_2 I - \sigma E - \mu_1 E, \\ \dfrac{dV_1}{dt} = \rho_1 S - \rho_2 V_1 - \mu_1 V_1, \\ \dfrac{dV_2}{dt} = \rho_2 V_1 - \mu_1 V_2 - \alpha_2 \beta_2 V_2 I - (1-\alpha_2) V_2, \\ \dfrac{dA}{dt} = (1-\omega)\sigma E - \alpha A - \gamma_1 A - (\mu_1 + \mu_2) A, \\ \dfrac{dI}{dt} = \omega\sigma E + \alpha A - \gamma_2 I - (\mu_1 + \mu_2) I, \\ \dfrac{dR}{dt} = (1-\alpha_2) V_2 + \gamma_1 A + \gamma_2 I - \mu_1 R, \end{cases} \tag{2.1}$$

where Λ is the constant migration rate of the susceptible population, β_1 is the transmission rate of asymptomatic infected persons, β_2 is the transmission rate of symptomatic infected persons, ρ_1 is the first vaccination rate, ρ_2 is the second vaccination rate, σ is the infection rate of the exposed to the

infected, ω is the proportion of infections in symptomatic patients, α is the ratio of asymptomatic infected people to symptomatic infected people and α_2 ($0 < \alpha_2 < 1$) is the ratio of secondary vaccinators to symptomatic infected people, so $1 - \alpha_2$ is the effectiveness of the vaccine. γ_1 and γ_2 respectively represent the recovery rate of asymptomatic and symptomatic infected persons. μ_1 and μ_2 represent the natural mortality rate and disease-related mortality rate respectively. All parameters are positive.

The disease-free equilibrium point of Eq (2.1) can be obtained (E, A and I are all zero)

$$P_0 = (S^*, 0, V_1^*, V_2^*, 0, 0, R^*),$$

where

$$\begin{aligned} S^* &= \frac{\Lambda}{\mu_1 + \rho_1}, \quad V_1^* = \frac{\Lambda\rho_1}{(\mu_1 + \rho_1)(\mu_1 + \rho_2)}, \\ V_2^* &= \frac{\Lambda\rho_1\rho_2}{(\mu_1 + \rho_1)(\mu_1 + \rho_2)(\mu_1 + 1 - \alpha_2)}, \\ R^* &= \frac{\Lambda\rho_1\rho_2(1 - \alpha_2)}{\mu_1(\mu_1 + \rho_1)(\mu_1 + \rho_2)(\mu_1 + 1 - \alpha_2)}. \end{aligned}$$

Using the next generation matrix method [17], the basic regeneration number of Eq (2.1) is

$$\begin{aligned} R_0 =& [\frac{\Lambda\beta_1}{\mu_1 + \rho_1} + \frac{\Lambda\alpha_2\beta_2\rho_1\rho_2}{(\mu_1 + \rho_1)(\mu_1 + \rho_2)(\mu_1 + 1 - \alpha_2)}](\frac{\alpha + \omega\sigma(\gamma_1 + \mu_1 + \mu_2)}{(\sigma + \mu_1)(\alpha + \gamma_1 + \mu_1 + \mu_2)(\gamma_2 + \mu_1 + \mu_2)}) \\ &- \frac{(1 - \omega)\sigma}{(\sigma + \mu_1)(\alpha + \gamma_1 + \mu_1 + \mu_2)}. \end{aligned}$$

As we all know, the following theoretical results on equilibrium stability have been established [1]:

1) when $R_0 < 1$, the disease-free equilibrium point of the infectious disease model is locally asymptotically stable; otherwise, it is unstable;

2) when $R_0 = 1$, the infectious model passes through a transcritical bifurcation near the disease-free equilibrium.

In fact, in order to have a more accurate understanding of the real disease development process, we added the influence of random factors into Eq (2.1). The improved stochastic model SEV_1V_2AIR is as follows:

$$\begin{cases} dS = [\Lambda - \beta_1 SA - \beta_2 SI - (\mu_1 + \rho_1) S] dt + \sigma_1 dB_1(t), \\ dE = [\beta_1 SA + \beta_2 SI + \alpha_2\beta_2 V_2 I - (\sigma + \mu_1) E] dt + \sigma_2 dB_2(t), \\ dV_1 = [\rho_1 S - (\rho_2 + \mu_1) V_1] dt + \sigma_3 dB_3(t), \\ dV_2 = [\rho_2 V_1 - \mu_1 V_2 - \alpha_2\beta_2 V_2 I - (1 - \alpha_2) V_2] dt + \sigma_4 dB_4(t), \\ dA = [(1 - \omega)\sigma E - (\alpha + \gamma_1 + \mu_1 + \mu_2) A] dt + \sigma_5 dB_5(t), \\ dI = [\omega\sigma E + \alpha A - (\gamma_2 + \mu_1 + \mu_2) I] dt + \sigma_6 dB_6(t), \\ dR = [(1 - \alpha_2) V_2 + \gamma_1 A + \gamma_2 I - \mu_1 R] dt + \sigma_7 dB_7(t), \end{cases} \tag{2.2}$$

where $B_i(t), i = 1, 2, 3, \cdots, 7$ is the independent standard Brownian motion which represents small disturbance on each variable, and $\sigma_i \geq 0$ is the intensity of $B_i(t), i = 1, 2, 3, \cdots, 7$.

3. Existence and uniqueness of global positive solutions

In order to study the dynamical behavior of the infectious disease model proposed in this paper, we first need to consider whether the solution is global and non-negative. So in this section, we will prove that there exists a unique global positive solution for Eq (2.2).

Theorem 1. *For any given initial value* $\{S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)\} \in R_+^7$*, Eq* (2.2) *has a unique positive solution* $\{S(t), E(t), V_1(t), V_2(t), A(t), I(t), R(t)\}$ *at* $t \geq 0$*, and this solution will stay in* R_+^7 *with a probability of 1. So, for all* $t \geq 0$*, the solution* $\{S(t), E(t), V_1(t), V_2(t), A(t), I(t), R(t)\} \in R_+^7$ *a.s.*

Proof: Since the coefficients of the equation are Lipschitz continuous, for any given initial value $\{S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)\} \in R_+^7$, there is a unique local solution $\{S(t), E(t), V_1(t), V_2(t), A(t), I(t), R(t)\}, t \in [0, \tau_e)$, where τ_e stands for the explosion time, and if $\tau_e = \infty$, this local solution is global. To do that, we have to make k_0 sufficiently large and $S(t)$, $E(t)$, $V_1(t)$, $V_2(t)$, $A(t)$, $I(t)$, $R(t)$ is in the interval $[\frac{1}{k_0}, k_0]$. For each integer $k \geq k_0$, we define the stopping time as follows:

$$\tau_k = \inf\left\{t \in [0, \tau_e) : S(t) \notin \left(\frac{1}{k}, k\right) \text{or } E(t) \notin \left(\frac{1}{k}, k\right) \text{or } V_1(t) \notin \left(\frac{1}{k}, k\right)\right.$$
$$\left. \text{or } V_2(t) \notin \left(\frac{1}{k}, k\right) \text{or } A(t) \notin \left(\frac{1}{k}, k\right) \text{or } I(t) \notin \left(\frac{1}{k}, k\right) \text{or } R(t) \notin \left(\frac{1}{k}, k\right)\right\}.$$

In this section, we set $inf\emptyset = \infty$ ($\emptyset$ denotes the empty set). It is easy to get τ_k that is increasing as $k \to \infty$. Set $\tau_\infty = lim_{k\to\infty}\tau_k$ which implies $\tau_\infty < \tau_k$ a.s.. If the hypothesis $\tau_\infty < \infty$ is true, then $\tau_e = \infty$ a.s. For all $t \geq 0$, this means

$$\{S(t), E(t), V_1(t), V_2(t), A(t), I(t), R(t)\} \in R_+^7 \ a.s.$$

In other words, we just prove $\tau_e = \infty$ a.s. If this statement is wrong, then there are constants $T > 0$ and $\varepsilon \in (0, 1)$ which make $P\{\tau_\infty < T\} > \varepsilon$. Hence there is an integer $k_1 > k_0$ such that

$$P\{\tau_\infty \leq T\} \geq \varepsilon, \ \forall k > k_1. \tag{3.1}$$

Define a C^2-function $V : R_+^7 \to R_+$ as follows:

$$\begin{aligned} &V(S, E, V_1, V_2, A, I, R) \\ =&(S - 1 - \ln S) + (E - 1 - \ln E) + (V_1 - 1 - \ln V_1) + \\ &(V_2 - 1 - \ln V_2) + (A - 1 - \ln A) + (I - 1 - \ln I) + (R - 1 - \ln R); \end{aligned} \tag{3.2}$$

the non-negativity of Eq (3.2) can be obtained from

$$u - 1 - lnu \geq 0, \ \forall u > 0.$$

Using *Itôs* formula, we can get

$$\begin{aligned} &dV(S, E, V_1, V_2, A, I, R) \\ =&LV(S, E, V_1, V_2, A, I, R)\, dt + \sigma_1(S - 1)dB_1(t) \\ &+ \sigma_2(E - 1)dB_2(t) + \sigma_3(V_1 - 1)\, dB_3(t) \\ &+ \sigma_4(V_2 - 1)\, dB_4(t) + \sigma_5(A - 1)dB_5(t) \\ &+ \sigma_6(I - 1)dB_6(t) + \sigma_7(R - 1)dB_7(t), \end{aligned} \tag{3.3}$$

where $LV : R_+^7 \to R_+$ is defined by

$$\begin{aligned}
&LV(S, E, V_1, V_2, A, I, R)\\
&=\left(1-\frac{1}{S}\right)[\Lambda-\beta_1 SA-\beta_2 SI-(\mu_1+\rho_1)S]\\
&\quad+\left(1-\frac{1}{E}\right)[\beta_1 SA+\beta_2 SI+\alpha_2\beta_2 V_2 I-(\sigma+\mu_1)E]\\
&\quad+\left(1-\frac{1}{V_1}\right)[\rho_1 S-(\rho_2+\mu_1)V_1]\\
&\quad+\left(1-\frac{1}{V_2}\right)[\rho_2 V_1-\mu_1 V_2-\alpha_2\beta_2 V_2 I-(1-\alpha_2)V_2]\\
&\quad+\left(1-\frac{1}{A}\right)[(1-\omega)\sigma E-(\alpha+\gamma_1+\mu_1+\mu_2)A]\\
&\quad+\left(1-\frac{1}{I}\right)[\omega\sigma E+\alpha A-(\gamma_2+\mu_1+\mu_2)I]\\
&\quad+\left(1-\frac{1}{R}\right)[(1-\alpha_2)V_2+\gamma_1 A+\gamma_2 I-\mu R]\\
&\quad+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2+\sigma_3^2+\sigma_4^2+\sigma_5^2+\sigma_6^2+\sigma_7^2\right)\\
&\leqslant\Lambda+\rho_1+\sigma+\rho_2+(1-\alpha_2)+\alpha+\gamma_1+\gamma_2+2\mu_2+7\mu_1+(\beta_2(1-\alpha_2)-\mu_1)I\\
&\quad+(\beta_1-\mu_1)A-\mu_2(A+I)-\mu_1(S+E+V_1+V_2+A+I+R)\\
&\quad+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2+\sigma_3^2+\sigma_4^2+\sigma_5^2+\sigma_6^2+\sigma_7^2\right)\\
&\leqslant\Lambda+\sigma+\rho_1+\rho_2+(1-\alpha_2)+\alpha+\gamma_1+\gamma_2+2\mu_1+7\mu_2\\
&\quad+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2+\sigma_3^2+\sigma_4^2+\sigma_5^2+\sigma_6^2+\sigma_7^2\right)\\
&=: K,
\end{aligned}$$

where $\beta_2 \le max\left\{\frac{\mu_1}{1-\alpha_2}, \mu_1\right\}$ is assumed and K ($K \in N^+$) is a positive constant, which does not rely on S, E, V_1, V_2, A, I, R and t. So, there is

$$\begin{aligned}
dV(S, E, V_1, V_2, A, I, R) \leqslant & Kdt+\sigma_1(S-1)dB_1(t)+\sigma_2(E-1)dB_2(t)\\
&+\sigma_3(V_1-1)dB_3(t)+\sigma_4(V_2-1)dB_4(t)\\
&+\sigma_5(A-1)dB_5(t)+\sigma_6(I-1)dB_6(t)\\
&+\sigma_7(R-1)dB_7(t).
\end{aligned} \tag{3.4}$$

Let us integrate both sides of Eq (3.4) from 0 to $\tau_k \wedge T = min\{\tau_k, T\}$ and then take the expectation; we can get

$$\begin{aligned}
&EV(S(\tau_k\wedge T), E(\tau_k\wedge T), V_1(\tau_k\wedge T), V_2(\tau_k\wedge T), A(\tau_k\wedge T), I(\tau_k\wedge T), R(\tau_k\wedge T))\\
&\leqslant V(S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)) + KE(\tau_k\wedge T)\\
&\leqslant V(S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)) + KT.
\end{aligned} \tag{3.5}$$

Set $\Omega_k = \{\tau_k \leq T\}$, and we obtain $P\{\Omega_k\} \geq \varepsilon$ by Eq (3.1). Now, notice that, for every $\omega \in \Omega_k$, there is at least one of $S(\tau_k, \omega)$, $E(\tau_k, \omega)$, $V_1(\tau_k, \omega)$, $V_2(\tau_k, \omega)$, $A(\tau_k, \omega)$, $I(\tau_k, \omega)$ or $R(\tau_k, \omega)$ that equals to k or $\frac{1}{k}$.

Therefore

$$V\{S(\tau_k, \omega), E(\tau_k, \omega), V_1(\tau_k, \omega), V_2(\tau_k, \omega), A(\tau_k, \omega), I(\tau_k, \omega), R(\tau_k, \omega)\}$$

is no less than

$$k - 1 - lnk \text{ or } \frac{1}{k} - 1 + lnk;$$

thus,

$$\begin{aligned} &V\{S(\tau_k, \omega), E(\tau_k, \omega), V_1(\tau_k, \omega), V_2(\tau_k, \omega), A(\tau_k, \omega), I(\tau_k, \omega), R(\tau_k, \omega)\} \\ &\geq min\left\{k - 1 - lnk, \frac{1}{k} - 1 + lnk\right\}. \end{aligned} \tag{3.6}$$

Substitute Eq (3.5) into Eq (3.6), we have

$$\begin{aligned} &V(S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)) + KT \\ \geqslant &E\left[1_{\Omega_{k_\omega}} \forall V\{S(\tau_k, \omega), E(\tau_k, \omega), V(\tau_k, \omega), V(\tau_k, \omega), A(\tau_k, \omega), I(\tau_k, \omega), R(\tau_k, \omega)\}\right] \\ \geqslant &\varepsilon \min\left\{k - 1 - \ln k, \frac{1}{k} - 1 + \ln k\right\}, \end{aligned} \tag{3.7}$$

where $1_{\Omega_{k_\omega}}$ denotes the indicator function of Ω_k. Letting $k \to \infty$, then

$$\infty > V(S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)) + KT = \infty. \tag{3.8}$$

Equation (3.8) is a contradiction. Therefore we have $\tau_\infty = \infty$. The proof is completed. Therefore, there exists a unique global positive solution to Eq (2.2).

4. Extinction of disease

Lemma 1. *For any initial value*

$$\{S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)\} \in R_+^7,$$

Eq (2.2) always has a unique positive solution

$$\{S(t), E(t), V_1(t), V_2(t), A(t), I(t), R(t)\} \in R_+^7 \text{ at } t \geq 0,$$

$\{S(t), E(t), V_1(t), V_2(t), A(t), I(t), R(t)\}$ *has the following properties:*

$$\begin{aligned} &\lim_{t\to\infty} \frac{S(t)}{t} = 0, \lim_{t\to\infty} \frac{E(t)}{t} = 0, \lim_{t\to\infty} \frac{V_1(t)}{t} = 0, \lim_{t\to\infty} \frac{V_2(t)}{t} = 0, \\ &\lim_{t\to\infty} \frac{A(t)}{t} = 0, \lim_{t\to\infty} \frac{I(t)}{t} = 0, \lim_{t\to\infty} \frac{R(t)}{t} = 0, \end{aligned} \tag{4.1}$$

and

$$\begin{aligned}&\lim_{t\to\infty}\frac{\log S(t)}{t}\leqslant 0,\lim_{t\to\infty}\frac{\log E(t)}{t}\leqslant 0,\lim_{t\to\infty}\frac{\log V_1(t)}{t}\leqslant 0,\lim_{t\to\infty}\frac{\log V_2(t)}{t}\leqslant 0,\\&\lim_{t\to\infty}\frac{\log A(t)}{t}\leqslant 0,\lim_{t\to\infty}\frac{\log I(t)}{t}\leqslant 0,\lim_{t\to\infty}\frac{\log R(t)}{t}\leqslant 0,\end{aligned}\tag{4.2}$$

as well as

$$\begin{aligned}&\lim_{t\to\infty}\frac{\int_0^t S(x)dB_1(x)}{t}=0,\lim_{t\to\infty}\frac{\int_0^t E(x)dB_2(x)}{t}=0,\\&\lim_{t\to\infty}\frac{\int_0^t V_1(x)dB_3(x)}{t}=0,\lim_{t\to\infty}\frac{\int_0^t V_2(x)dB_4(x)}{t}=0,\\&\lim_{t\to\infty}\frac{\int_0^t A(x)dB_5(x)}{t}=0,\lim_{t\to\infty}\frac{\int_0^t I(x)dB_6(x)}{t}=0,\\&\lim_{t\to\infty}\frac{\int_0^t R(x)dB_7(x)}{t}=0.\end{aligned}\tag{4.3}$$

In order to get the conditions of disease extinction, we set Theorem 2.

Theorem 2. *Let* $\{S(t),E(t),V_1(t),V_2(t),A(t),I(t),R(t)\}\in R_+^7$ *be a positive solution for Eq (2.2), and the initial solution of Eq (2.2) is* $\{S(0),E(0),V_1(0),V_2(0),A(0),I(0),R(0)\}$.

If $R_0^S=\frac{(1-\omega)\sigma\Lambda}{\mu_1\left(\alpha+\gamma_1+\mu_1+\mu_2+\frac{\sigma_5^2}{2}\right)}<1$, *then the solution of the system has the following properties:*

$$\lim_{t\to\infty}sup\frac{\log A(t)}{t}<0,\lim_{t\to\infty}sup\frac{\log I(t)}{t}<0.\tag{4.4}$$

Proof: First, integrate Eq (2.2) to obtain

$$\begin{aligned}\frac{S(t)-S(0)}{t}=&\Lambda-\beta_2\langle S(t)\rangle\langle I(t)\rangle-\beta_1\langle S(t)\rangle\langle A(t)\rangle-(\mu_1+\rho_1)\langle S(t)\rangle\\&+\frac{\sigma_1}{t}\int_0^t S\,dB_1(x),\\\frac{E(t)-E(0)}{t}=&\beta_2\langle S(t)\rangle\langle I(t)\rangle+\beta_1\langle S(t)\rangle\langle A(t)\rangle+\alpha_2\beta_2\langle V_2(t)\rangle\langle I(t)\rangle\\&-(\mu_1+\sigma)\langle E(t)\rangle+\frac{\sigma_2}{t}\int_0^t E dB_2(x),\\\frac{V_1(t)-V_1(0)}{t}=&\rho_1\langle S(t)\rangle-(\mu_1+\rho_2)\langle V_1(t)\rangle+\frac{\sigma_3}{t}\int_0^t V_1 dB_3(x),\\\frac{V_2(t)-V_2(0)}{t}=&\rho_2\langle V_1(t)\rangle-\mu_1\langle V_2(t)\rangle-\alpha_2\beta_2\langle V_2(t)\rangle\langle I(t)\rangle\\&-(1-\alpha_2)\langle V_2(t)\rangle+\frac{\sigma_4}{t}\int_0^t V_2 dB_4(x),\\\frac{A(t)-A(0)}{t}=&(1-\omega)\sigma\langle E(t)\rangle-(\alpha+\gamma_1)\langle A(t)\rangle-(\mu_1+\mu_2)\langle A(t)\rangle\\&+\frac{\sigma_5}{t}\int_0^t A dB_5(x),\end{aligned}\tag{4.5}$$

$$\begin{aligned}\frac{I(t)-I(0)}{t} =&\omega\sigma\langle E(t)\rangle + \alpha\langle A(t)\rangle - \gamma_2\langle I((t)\rangle - (\mu_1+\mu_2)\langle I(t)\rangle \\ &+ \frac{\sigma_6}{t}\int_0^t I dB_6(x), \\ \frac{R(t)-R(0)}{t} =&(1-\alpha_2)\langle V_2(t)\rangle + \gamma_1\langle A(t)\rangle + \gamma_2\langle I(t)\rangle - \mu_1\langle R(t)\rangle \\ &+ \frac{\sigma_7}{t}\int_0^t R dB_7(x).\end{aligned}$$

Adding the left and right sides of Eq (4.5) respectively, we obtain

$$\begin{aligned}&\frac{S(t)-S(0)}{t} + \frac{E(t)-E(0)}{t} + \frac{V_1(t)-V_1(0)}{t} + \frac{V_2(t)-V_2(0)}{t} \\ &+ \frac{A(t)-A(0)}{t} + \frac{I(t)-I(0)}{t} + \frac{R(t)-R(0)}{t} \\ =&A - \mu_1[\langle S(t)\rangle + \langle E(t)\rangle + \langle V(t)\rangle + \langle V(t)\rangle + \langle R(t)\rangle] - (\mu_1+\mu_2)[\langle A(t)\rangle + \langle I(t)\rangle] \\ &+ \frac{\sigma_1}{t}\int_0^t S dB_1(x) + \frac{\sigma_2}{t}\int_0^t E dB_2(x) + \frac{\sigma_3}{t}\int_0^t V_1 dB_3(x) + \frac{\sigma_4}{t}\int_0^t V_2 dB_4(x) \\ &+ \frac{\sigma_5}{t}\int_0^t A dB_5(x) + \frac{\sigma_6}{t}\int_0^t I dB_6(x) + \frac{\sigma_7}{t}\int_0^t R dB_7(x).\end{aligned}$$

At this time, we have

$$\begin{aligned}\langle S(t)\rangle =&\frac{A}{\mu_1} - [\langle E(t)\rangle + \langle V_1(t)\rangle + \langle V_2(t)\rangle + \langle R(t)\rangle] \\ &- \frac{(\mu_1+\mu_2)}{\mu_1}[\langle A(t)\rangle + \langle I(t)\rangle] + \phi(t),\end{aligned} \tag{4.6}$$

where

$$\begin{aligned}\phi(t) =&\frac{\sigma_1}{\mu_1 t}\int_0^t S dB_1(x) + \frac{\sigma_2}{\mu_1 t}\int_0^t E dB_2(x) + \frac{\sigma_3}{\mu_1 t}\int_0^t V_1 dB_3(x) + \frac{\sigma_4}{\mu_1 t}\int_0^t V_2 dB_4(x) \\ &+ \frac{\sigma_5}{\mu_1 t}\int_0^t A dB_5(x) + \frac{\sigma_6}{\mu_1 t}\int_0^t I dB_6(x) + \frac{\sigma_7}{\mu_1 t}\int_0^t R dB_7(x) \\ &- \frac{1}{\mu_1}\left[\frac{S(t)-S(0)}{t} + \frac{E(t)-E(0)}{t} + \frac{V_1(t)-V_1(0)}{t} + \frac{V_2(t)-V_2(0)}{t}\right] \\ &- \frac{1}{\mu_1}\left[\frac{A(t)-A(0)}{t} + \frac{I(t)-I(0)}{t} + \frac{R(t)-R(0)}{t}\right].\end{aligned}$$

We can also obtained

$$\begin{aligned}\langle E(t)\rangle =&\frac{A}{\mu_1} - [\langle S(t)\rangle + \langle V_1(t)\rangle + \langle V_2(t)\rangle + \langle R(t)\rangle] \\ &- \frac{(\mu_1+\mu_2)}{\mu_1}[\langle A(t)\rangle + \langle I(t)\rangle] + \phi(t).\end{aligned} \tag{4.7}$$

Then, *Itôs* formula is used for the fifth formula in Eq (2.2), we can get

$$d\log A(t) = \left[(1-\omega)\sigma E - (\alpha+\gamma_1+\mu_1+\mu_2)A\right]\frac{1}{A}dt - \frac{\sigma_5^2}{2}dt + \sigma_5 dB_5(t). \tag{4.8}$$

Integrate Eq (4.8) from 0 to t:

$$\begin{aligned}
&\log A(t) - \log A(0)\\
\leqslant &\int_0^t \left[(1-\omega)\sigma E - \left(\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}\right)\right] dt + \sigma_5 dB_5(t)\\
\leqslant &\left[(1-\omega)\sigma E - \left(\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}\right)\right] t + \sigma_5 B_5(t).
\end{aligned} \tag{4.9}$$

Substitute Eq (4.7) into Eq (4.9) and divide by t, so there is

$$\begin{aligned}
&\frac{\log A(t) - \log A(0)}{t}\\
\leqslant &(1-\omega)\sigma\langle E(t)\rangle - (\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}) + \frac{\sigma_5 B_5(t)}{t}\\
\leqslant &\frac{(1-\omega)\sigma}{\mu_1}\Lambda - \frac{(\mu_1+\mu_2)(1-\omega)\sigma}{\mu_1}\langle A(t)\rangle + (1-\omega)\sigma\phi(t)\\
&- (\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}) + \frac{\sigma_5 B_5(t)}{t}\\
\leqslant &(\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2})(\frac{(1-\omega)\sigma A}{\mu_1\left(\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}\right)} - 1)\\
&- \frac{(\mu_1+\mu_2)(1-\omega)\sigma}{\mu_1}\langle A(t)\rangle + \frac{\sigma_5 B_5(t)}{t} + \varphi(t),
\end{aligned} \tag{4.10}$$

where $\varphi(t) = \sigma(1-\omega)\phi(t)$. R_0^s is the called random threshold and it is shown as

$$R_0^s = \frac{(1-\omega)\sigma A}{\mu_1\left(\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}\right)}.$$

According to the strong number theorem, we have $\lim\limits_{t\to\infty}\frac{B_5(t)}{t} = 0$, i.e., $\lim\limits_{t\to\infty}\frac{\sigma_5 B_5(t)}{t} = 0$. Therefore, take the limit on both sides of Eq (4.10) at the same time; we then get

$$\begin{aligned}
\limsup_{t\to\infty}\frac{\log A(t)}{t} \leqslant &\left(\alpha + \gamma_1 + \mu_1 + \mu_2 + \frac{\sigma_5^2}{2}\right)(R_0^s - 1)\\
&- \frac{(\mu_1+\mu_2)(1-\omega)\sigma}{\mu_1}\langle A(t)\rangle\\
<&0;
\end{aligned}$$

this means that when $R_0^s < 1$

$$\lim_{t\to\infty} A(t) = 0. \tag{4.11}$$

Taking the limit on both sides of Eq (4.6) at the same time, we can obtain

$$\lim_{t\to\infty} S(t) = \frac{\Lambda}{\mu_1}. \tag{4.12}$$

Set $M = \left\{\omega = \Omega : \lim_{t\to\infty} S(t) = \frac{\Lambda}{\mu_1}\right\}$, which means that $P(M) = 1$ at this point.

From Eq (4.7), we can get

$$\lim_{t\to\infty}\langle E(t)\rangle \leqslant \frac{\Lambda}{\mu_1}.$$

For $\forall\omega \in M, t > 0$, we can get $\lim_{t\to\infty} E(\omega, t) = 0,\ \omega \in M$. Considering that $P(M) = 1$,we can obtain

$$\lim_{t\to\infty} E(t) = 0, a.s.. \tag{4.13}$$

It can be seen from Eq (4.5) that

$$\frac{I(t) - I(0)}{t} = \omega\sigma\langle E(t)\rangle + \alpha\langle A(t)\rangle - \gamma_2\langle I((t)\rangle - (\mu_1 + \mu_2)\langle I(t)\rangle + \frac{\sigma_6}{t}\int_0^t IdB_6(x),$$

i.e.

$$\langle I(t)\rangle = \frac{1}{\gamma_2 + \mu_1 + \mu_2}[\omega\sigma\langle E(t)\rangle + \alpha\langle A(t)\rangle - \frac{I(t) - I(0)}{t} + \frac{\sigma_6}{t}\int_0^t IdB_5(t)]. \tag{4.14}$$

It can be concluded that $\lim_{t\to\infty}\langle I(t)\rangle = 0$, a.s.

Therefore, this conclusion is proved.

5. Numerical simulation

In this section, in order to verify the effects of vaccination and random disturbance on controlling the spread of disease, we used the Milstein method [38, 39] to conduct numerical simulations of the stochastic model described by Eq (2.2).

The parameter values of Eq (2.2) are shown in the following table (Table 1).

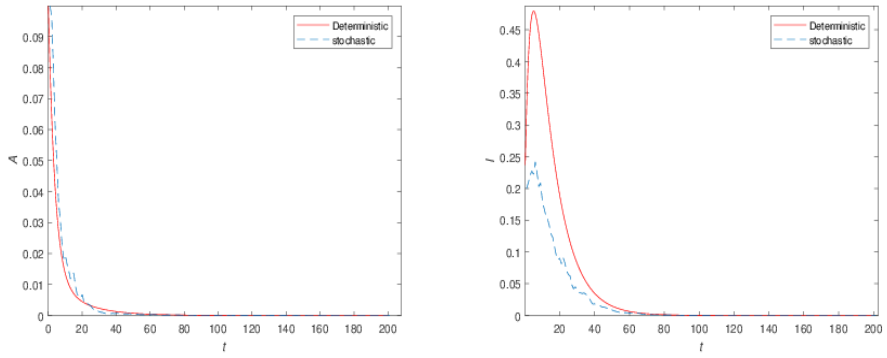
Table 1. Numerical experimental parameter values of Eq (2.2).

Parameter	Value	Source
Λ	0.03	
β_1	1.038	
β_2	0.083	
σ	$\frac{1}{5.2}$	[40]
ω	0.8	[40]
α	0.19	[40]
γ_1	0.3	[41]
γ_2	0.001	[41]
μ_1	0.09	[41]
μ_2	0.001	[41]

Eq (2.2) is discretized into the following format:

$$
\begin{cases}
S_j = S_{j-1} + \left[\Lambda - \left(\beta_1 A_{j-1} + \beta_2 I_{j-1}\right) S_{j-1} - (\mu_1 + \rho_1) S_{j-1}\right] \Delta t \\
\quad + \sigma_1 \left(S_{j-1}\right) \sqrt{\Delta t} \zeta_{j-1} + \frac{\sigma_1^2}{2} \left(S_{j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t, \\
E_j = E_{j-1} + \left[\left(\beta_1 A_{j-1} + \beta_2 I_{j-1}\right) S_{j-1} + \alpha_2 \beta_2 V_{2j-1} I_{j-1} - (\sigma + \mu_1) E_{j-1}\right] \Delta t \\
\quad + \sigma_2 \left(E_{j-1}\right) \sqrt{\Delta t} \zeta_{j-1} + \frac{\sigma_2^2}{2} \left(E_{j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t, \\
V_{1j} = V_{1j-1} + \left[\rho_1 S_{j-1} - (\rho_2 + \mu_1) V_{1j-1}\right] \Delta t + \sigma_3 \left(V_{1j-1}\right) \sqrt{\Delta t} \zeta_{j-1} \\
\quad + \frac{\sigma_3^2}{2} \left(V_{1j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t, \\
V_{2j} = V_{2j-1} + \left[\rho_2 V_{1j-1} - \alpha_2 \beta_2 V_{2j-1} I_{j-1} - (\mu_1 + 1 - \alpha_2) V_{2j-1}\right] \Delta t \\
\quad + \sigma_4 \left(V_{2j-1}\right) \sqrt{\Delta t} \zeta_{j-1} + \frac{\sigma_4^2}{2} \left(V_{2j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t, \\
A_j = A_{j-1} + \left[(1 - \omega) \sigma E_{j-1} - (\alpha + \gamma_1 + \mu_1 + \mu_2) A_{j-1}\right] \Delta t \\
\quad + \sigma_5 \left(A_{j-1}\right) \sqrt{\Delta t} \zeta_{j-1} + \frac{\sigma_5^2}{2} \left(A_{j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t, \\
I_j = I_{j-1} + \left[\omega \sigma E_{j-1} + \alpha A_{j-1} - (\gamma_2 + \mu_1 + \mu_2) I_{j-1}\right] \Delta t \\
\quad + \sigma_6 \left(I_{j-1}\right) \sqrt{\Delta t} \zeta_{j-1} + \frac{\sigma_6^2}{2} \left(I_{j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t, \\
R_j = R_{j-1} + \left[(1 - \alpha_2) V_{2j-1} + \gamma_1 A_{j-1} + \gamma_2 I_{j-1} - \mu_1 R_{j-1}\right] \Delta t \\
\quad + \sigma_7 \left(R_{j-1}\right) \sqrt{\Delta t} \zeta_{j-1} + \frac{\sigma_7^2}{2} \left(R_{j-1}\right) \left(\zeta_{j-1}^2 - 1\right) \Delta t,
\end{cases}
$$

where $\zeta_j (j = 1, 2, \cdots, n)$ are independent Gaussian random variables $N(0, 1)$. Based on the parameters in Table 1, the initial value of the numerical experiment was set as $(S(0), E(0), V_1(0), V_2(0), A(0), I(0), R(0)) = (0.1, 0.1, 0, 0, 0.1, 0.2, 0)$.



(a) (b)

Figure 2. Trends of infected persons for the deterministic and stochastic models.

Based on Table 1, let $\rho_1 = 0.9, \rho_2 = 0.9, \alpha_2 = 0.008$, $\sigma_1 = 0.15$, $\sigma_2 = 0.063$, $\sigma_3 = 0.338$, $\sigma_4 = 0.068$, $\sigma_5 = 0.25$, $\sigma_6 = 0.09$ and $\sigma_7 = 0.023$. The asymptotic behavior of the stochastic model at the disease-free equilibrium point $E_0 = (0.0303, 0, 0.0275, 0.0229, 0, 0, 0.2526)$ can be shown in Figure 2. At this time, the random threshold is $R_0^s = 0.0209 < R_0 = 0.2872 < 1$.

In Figure 2, asymptomatic and symptomatic infections gradually decrease until they disappear, which means that the disease tends to extinction. This is consistent with the previous theoretical results on the extinction of disease. In other words, appropriate noise intensity can promote the extinction of the disease. With the passage of time, the probability of disease extinction becomes 1.

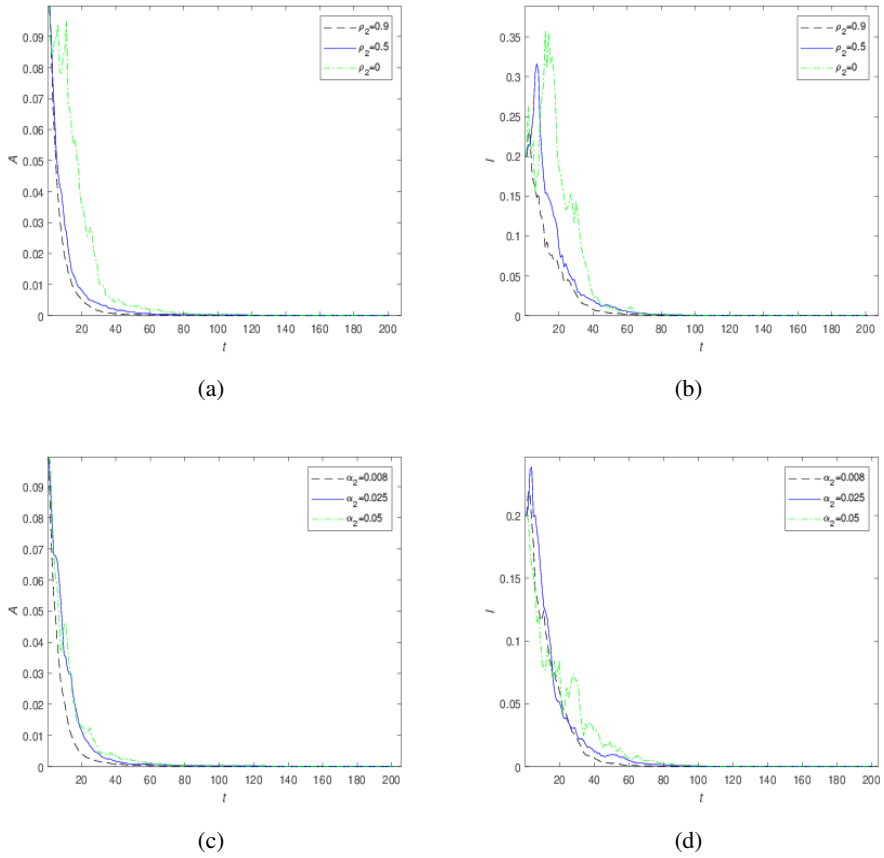


Figure 3. Trends of infected persons affected by secondary vaccination rate and vaccine efficiency.

Based on the parameters given in Table 1, $\rho_1 = 0.9, \alpha_2 = 0.008$ and we let ρ_2 equal to 0, 0.5 and 0.9 respectively, the extinction trends of asymptomatic infected person A and symptomatic infected person I are shown in Figure 3(a) and (b). It is clear from Figure 3 that with the increase of the secondary vaccination rate, disease extinction approaches faster. In addition, when $\rho_1 = 0.9, \rho_2 = 0.9$, the extinction trends of the two infected populations are shown in Figure 3(c) and (d) as α_2 is equal to 0.008, 0.025 and 0.05 respectively. We found that the smaller the value of α_2 and the higher the vaccine efficiency, the faster the disease tends to extinction. So, we can know from Figure 3 that improving the secondary vaccination rate and vaccine efficiency is one of the important measures to effectively control the epidemic.

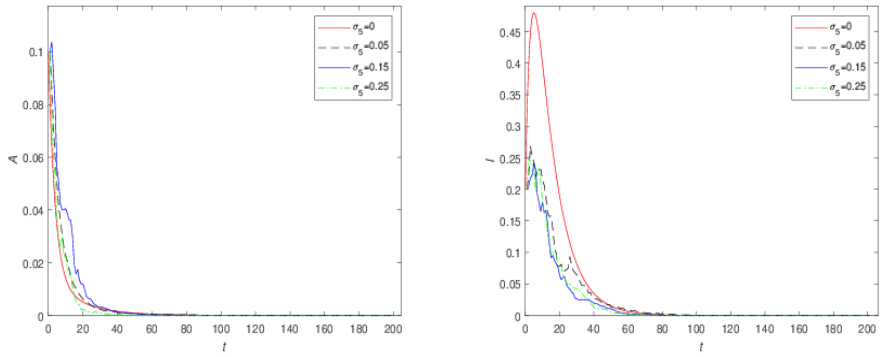


Figure 4. Trends of infected persons in stochastic model under different intensities σ_5.

According to Theorem 2, σ_5 is random intensity acting on asymptomatic infected persons which can directly affect the random threshold R_0^s. This means that σ_5 can directly affect the speed of the extinction of the disease and that other noise intensity types indirectly affects the extinction of the disease. Therefore the influence of σ_5 on the extinction of the disease was mainly investigated. When other noise intensity types are kept constant and σ_5 is set to 0, 0.05, 0.15 and 0.25 respectively, the influence of different noise intensities on disease extinction is shown in Figure 4. It can be seen in Figure 4 that the noise intensity can promote the extinction of the infected population. Under the constraint of a random threshold, the higher the noise intensity σ_5, the faster the disease-free equilibrium point is reached.

In real life, σ_5 represents the disturbance intensity of infected persons under the policies of isolation measures, the effective diagnosis of asymptomatic infected persons and self-protection. From the above analysis, it can be seen that the more complete the intervention policies for infected people, the better the disease prevention and control.

6. Conclusions

At present, the mathematical modeling of infectious diseases plays an important role in providing epidemic prevention and control strategies. In order to control the epidemic in real life, quarantine and other epidemic prevention measures have been proposed, which have become a random disturbance factor affecting the spread of the epidemic in the infectious disease model. So far, vaccination is considered to be one of the most popular and effective methods to alleviate and prevent epidemics. Based on this, the SEV_1V_2AIR model was established by considering random disturbance and secondary vaccination, and then the dynamical analysis of the model was carried out. First, using the stop time theory and Lyapunov analysis method, it was proved that the proposed model has a globally unique positive solution. Then, we obtained the theoretical results about the random threshold R_0^s. When $R_0^S < 1$, the disease tends to become extinct. Finally, numerical simulations were performed to verify the theoretical results.

In this paper, the theoretical and numerical simulations show that vaccination and random disturbance have a great impact on disease dynamics. The intensity of the random disturbance

directly affects the extinction rate of the infected population. In addition, the higher the secondary vaccination rate and vaccine effectiveness, the faster the extinction rate of the disease. In order to control infectious diseases, we can not only reduce the disease transmission rate through isolation, self-protection and other measures, but also encourage susceptible individuals to get vaccinated and improve the effectiveness of the vaccine. These conclusions provide theoretical basis for preventing and controlling the spread of COVID-19.

Furthmore, the impact of some discontinuous factors and vaccine frequency on COVID-19 will be discussed in the future work because of more influential disturbances, such as large-scale human aggregation, the promotion of antiviral drugs and so on.

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Conflict of interest

The authors declare that there is no conflict of interest.

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