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Stability analysis of a novel epidemics model with vaccination and nonlinear infectious rate

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\textbf{Keywords:}
Epidemic model
Vaccination
Growth rate of viruses
Growth behavior
Stability analysis

\textbf{Abstract}

In this paper, by considering pathogen evolution and human interventions behaviors with vaccines or drugs, we build up a novel SEIRW model with the vaccination to the newborn children. The stability of the SEIRW model with time-varying perturbation to predict the evolution tendency of the disease is analyzed. Furthermore, we introduce a time-varying delay into the susceptible and infective stages in the model and give some global exponential stability criteria for the time-varying delay system. Finally, numerical simulations are presented to verify the results.

\section{1. Introduction}

Infectious diseases result in 14.7 million deaths, 26\% of global mortality in accordance with WHO estimates in 2001 [1]. Mathematical analysis and modeling operation of infectious diseases are critical to study virus spreading dynamics which can state clearly the origination and evolution of viruses. In recent years, many mathematical models have been proposed for the transmission dynamics of infectious diseases [2–9] such as, SI (susceptible–infective), SIR (susceptible–infective–removed), SEIR (susceptible–exposure–infective–recovered), SEI (susceptible–exposure–infective), SIRS (susceptible–infective–removed–susceptible), SEIS (susceptible–exposure–infective–susceptible). The development of such models is aimed at exploring the transmission dynamics of epidemic virus, investigating the evolution of resistance to antibiotics and the evolutionary cost of resistance, and designing the programs for disease control. However, a model's ability to achieve the above goals depends greatly on whether the assumptions made in the modeling process are consistent with the actual evolution of the epidemic diseases. Understanding and predicting the actual transmission dynamics of the epidemic diseases is, therefore, an important pursuit in mathematical epidemiology, which is one of our motivations for this work.

At the same time, the stability analysis of epidemic models has also attracted much attention of biologists, mathematicians and ecologists. The underlying reason behind such attention is that through stability analysis for the epidemic models, the tendency of the infectious diseases can be found by the basic reproductive number $R_0$ (the average number of secondary cases produced by a typical primary case in an entirely susceptible population) and the generation time (the average time from symptom onset in a primary case to symptom onset in a secondary case), which determine to a large extent the speed of epidemic outbreaks. Based on the stability principles, the conditions of the infectious diseases persistence or extinction were obtained [10–15].

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0096-3003/$ - see front matter © 2013 Elsevier Inc. All rights reserved.
http://dx.doi.org/10.1016/j.amc.2013.07.016
Huang [3] presented an SEIR model, which is suitable for the eradication of diseases by mass vaccination or the control of diseases by case isolation combined with contact tracing, incorporating the vaccine efficacy or the control efficacy into the model. An SIRC model with a fourth class, i.e., the cross-immune individuals, was proposed [9]. Via bifurcation analysis of the model, they discussed the effect of seasonality on the epidemiological regimes. In this model, the incubation period of virus is not considered, nevertheless, a majority of infectious diseases have incubation period. Buonomo et al. [16] studied the global behavior of a non-linear SIR epidemic model with a non-bilinear feedback mechanism, which describes the influences of information and information-related delays on a vaccination campaign. Wan and Cui [17] proposed an SEIS epidemic model to study the effect of transport-related infection on the spread and control of infectious disease. Furthermore, through stability analysis, they found that it is very essential to strengthen restrictions of passengers once infectious diseases appearance is known. In [18], McCluskey studied the complete global stability for the SIR models with distributed delay and with discrete delay. Lahrouz et al. [19] analyzed the complete global stability for an SIRS epidemic model with generalized non-linear incidence and vaccination. In [20], Kuniya studied the global asymptotic stability for an age-structured multigroup SIR epidemic model with a discretization approach. Muroya et al. [21] investigated the global asymptotic stability of a disease transmission model of SIRS type with latent period and the specific non-monotone incidence rate. The results of these studies, however, do not take into account the whole process of the epidemic diseases such as the exposed fraction and the partial immunity individuals, which are to a great extent owing to deficiency of the genetic variation behavior of the virus and the epidemiology theory. Consequently, they are limited on reflecting the actual transmission dynamics of the epidemic diseases and offering the practical proposal for government to draw up the policy.

On the other hand, human interference behaviors such as enhancing the host's immune system with drugs or vaccines that alter pathogen genetic diversity, carrying out public health educational campaigns and vaccination in adolescents have been incorporated into the epidemic diseases models [22–25]. Mathematical models for the spread of infectious diseases are an important tool for investigating and quantifying such effects of these behaviors. However, few efforts have been made to quantify and capture the effects in a systematic way.

In this paper, to overcome the disadvantages above, by considering more complete processes of the epidemic diseases, we first build a novel susceptible–exposed–infective–recovered–partial immunity (SEIRW) model, which incorporates pathogen evolution and human interventions behaviors with vaccines or drugs that alter pathogen genetic diversity, aiming to reflect the transmission dynamics of infectious diseases more realistically. Our model differs from the existing ones in [17–21] in the following respect: (i) our model additionally takes into account the exposed fraction and the partial immunity individuals; (ii) our model is coupled with evolutionary–epidemic strategy and human interference behaviors such as vaccination to a proportion of all newborn children; (iii) in particular, the force of infection of the infectious disease in the existing work is multiplied by a constant such as survival probability while in this paper, it is governed by a nonlinear time-varying growing curve of pathogens, which reflects the viruses' evolution. Subsequently, we transform the SEIRW model into the ordinary differential equation with time-varying perturbation. Then we carry out a complete stability analysis of the transformed SEIRW model and establish its stability criteria. Furthermore, we introduce the time-varying delay into the susceptible and infective stages of the SEIRW model and provide the globally exponential stability criteria for the SEIRW model with the time-varying delay. Finally, the numerical simulations verify the results. One of our purposes for this work is to investigate the effect of the treatment on the long term dynamics of the disease, and show that the treatment or rational immunization to prolong the susceptible or infective stages can hasten the disease dying out. To the best of our knowledge, this work is the first one that builds a novel SEIRW model by considering pathogen evolution and human interference behaviors, and further establishes sufficient conditions for the asymptotic stability of the model with time-varying perturbation and the global exponential stability of the model with time-varying delay.

The rest of this paper is organized as follows: in Section 2, the infectious rate function of pathogens is obtained. The SEIRW model is provided in Section 3. In Section 4, the stability of the SEIRW model with time-varying perturbation is analyzed. In Section 5, we discuss the global stability of the SEIRW model with time-varying delay by means of suitable Lyapunov functionals. Simulation to verify analysis for the model is gained in Section 6, concluding remarks. Section 7 concludes this work.

2. The infectious rate function of pathogens

Mathematical modeling of the evolution of pathogens responding to the viruses' genetic variation plays an important role in clinical epidemiology and statistics of infectious diseases. In the existing literatures, the mathematical descriptions of the contact rate which implies the infectivity depend on the analysis for the empirical data. Nevertheless, the contact process described by empirical data could be questionable on account of the incomplete statistical methods, e.g., the methods of the investigation based conversational contacts, which describe the spread of respiratory infections, the routine surveys of travel. Furthermore, the contact rate served as the infectious power cannot reflect the evolution of pathogens, especially the viruses' genetic variation in the available studies.

The growing curve of pathogens indicates the speed of viruses spread as it is the coordinate figure of time-viruses number. The one-step growth curves, which reflect pathogen genetic diversity under the condition of interpretation of interventions with drugs or vaccines, have been studied by many biologists [26–29]. Usually, the viruses' growth simulating the viruses' growth curve is expressed by:

\[ y = \frac{k}{1 + be^{-at}}, \quad a, b, k > 0. \]  

(1)
Eq. (1a) is a classical equation which is widely used in life science to describe the growth of the organism such as leaves, bacterium. The parameters \(a, b\) and \(k\) have specific biological meanings and are only positive real numbers in (1a). The unit of time \(t\) is day. Here, the derivation of Eq. (1a) is given by,

\[
g(t) = \frac{ab^k e^{-at}}{(1 + be^{-at})^2}, \quad a, b, k > 0, \tag{1b}
\]

which denotes the infectious rate function of viruses and the speed of the viruses growth. Since the transmission of the viruses among the populations is related with the infectious rate of the viruses; furthermore, it is direct proportion to the infectious rate, therefore, in this paper, the speed of the viruses’ transmission among the populations is expressed by the function \(\omega(t)\) below.

\[
\omega(t) = k_1 g(t) = \frac{k_1kabe^{-at}}{(1 + be^{-at})^2}, \quad a, b, k > 0, \tag{1c}
\]

In (1c), the product of \(k_1\) is constant and it can be represented by \(k\) just as well. Consequently, the infectious rate of transmission among the populations can be given by (2).

\[
\omega(t) = \frac{ab^k e^{-at}}{(1 + be^{-at})^2} \quad \text{is constant and it can be represented } a, b, k > 0. \tag{2}
\]

### 3. The SEIRW epidemic model

A general flow process diagram of the SEIRW epidemic mathematical model is shown in Fig. 1. From an epidemiological point of view, the normal community of people can be divided into five compartments at any time \(t\) for the majority infectious diseases such as influenza A, SARS and AIDS: the susceptible individuals \(S(t)\), who may be infected by the viruses, and do not have specific immune defenses against the infectious disease; the exposed fraction \(E(t)\), which denotes hosts whom viruses have entered into while the viruses are in incubation period; the infective individuals \(I(t)\), who are infected by the specific viruses and are infectious to the susceptible individuals; the recovered fraction \(R(t)\), which denotes those individuals who have recovered from the infection but have partially immune to the specific pathogen. However, after a period of time, a part of the individuals in \(R(t)\) enter the next group \(W(t)\), the partial immunity individuals, because they have only partial immunity to the viruses owing to their variation in the new environment [26], as in the case of SRAS [26], influenza [31] and pertussis [32]. The environment is much important for the survival of intermediate hosts and for the survival of the pathogens outside the host, which can affect the efficiency of transmission. The environmental component of infectiousness can result in an unusually large number of secondary infections, which motivates that the environmental effect should be considered in our model. For example, the ‘superspreading’ event led to various secondary severe acute respiratory syndrome (SARS) infections among residents of the Amoy Gardens estate in Hong Kong in 2003 [26].

In Fig. 1, the susceptible class is generated from individuals at a net birth \(ZN\), where \(N\) denotes the total population size and \(Z\) denotes the birth rate. Similarly, it is assumed that the natural death rate of all the classes is \(\mu\) which is not equal to the birth rate. \(\eta\) denotes the death rate caused by the special infectious diseases. Infectious rate function \(\omega(t)\) represents the viruses’ state of genetic variation. Those from the susceptible individuals to latency ones are proportion to the susceptible, the infective and the infectious rate function. Therefore, the incidence of disease can be considered as \(S(t)I(t)\omega(t)\) per unit time, and \(I(t)\omega(t)\) is often used to represent the force of infection. The rate of transformation from incubation period individuals to infective individuals per unit time is given by \(\varepsilon\). The cure rate (i.e. the rate at which the infective people recover per unit time) is denoted by \(\xi\).

In this model, vaccination strategies are the focus soul and they are the influential measures to control the outbreak or extinction of the infectious diseases by the government. In order to evoke the given immune response with clinical infectious disease rather than natural infection, vaccines can provide a foreign antigen to the immune system and the subsequent immune stimulation to natural infection is gained more rapidly so as to prevent the attack of severe disease viruses. In this study, vaccination is applied to a ratio of all newborn children. Because of newborn children’s highly immature immune systems, it is difficult to generate antibody to the special infectious disease after they have been injected vaccination or drug.

![Fig. 1. The SEIRW model with vaccination and growth rate.](image-url)
Even so, it is the forceful strategies for the government to prevent viruses such as AIDS and influenza A transmission. We assume that the ratio of vaccination to newborn children is $Bv$, and the newborns successfully gain the vaccine and obtain immunity to infection. Then there are an inflow of susceptible individuals $ZN(1 - Bv)$ and an inflow of vaccinated individuals $ZN Bv$ to the vaccinated compartment.

Though the recovery individuals obtain the specific immunity to the infection in the original period, due to the variability and evolution of the viruses, the ability of the hosts to evade natural immune protection is diminished or died away in a new environment. In this study, it is assumed that a proportion $\xi$ of the recovered population enter the partial immunity group $W(t)$ in some given period. After some time, a proportion $\gamma$ of the hosts become incubation period individuals, and a proportion $1 - \gamma$ of the vaccinated hosts become recovered people. $d$ is the contact rate. $\beta$ is the rate from the partial immunity group $W(t)$ to the susceptible individuals $S(t)$. The relationship expression $\xi > \gamma$ is obvious.

Formally, the SEIRW model can be illustrated by the following set of five ordinary differential equations.

$$
\begin{cases}
\frac{dS}{dt} = \phi + \beta W - \mu S - \omega(t)SI,
\frac{dE}{dt} = \omega(t)SI - (\mu + \varepsilon)E + d_{\gamma}W,
\frac{dI}{dt} = \varepsilon E - (\mu + \eta + \alpha)I,
\frac{dR}{dt} = \alpha I - (\mu + \zeta)R + d(1 - \gamma)W,
\frac{dW}{dt} = \xi R - \mu W - d(1 - \gamma)W - d_{\gamma}W - \beta W + \varphi,
\end{cases}
$$

(3)

where $\omega(t) = \frac{akt}{1 + bkt}$, $a, b, k > 0$ is the infectious rate of the viruses, $\phi = ZN(1 - Bv)$, $\varphi = ZNBv$. It is not difficult to see that as $t \in [0, \infty)$, the infectious rate $\omega(t)$ is continuous and uniformly bounded on $[0, ka/4]$ and achieves maximum when the time $t$ equals to $\ln b/a$, which shows that the speed of the viruses growth is bounded and its maximum value is $ka/4$.

4. Stability analysis

4.1. The SEIRW model with time-varying perturbation

Let $x(t) = [S(t), E(t), I(t), R(t), W(t)]^T$, $[\cdot]^T$ denotes the transpose of a matrix and $\mathbb{R}^5$ be the 5-dimensional Euclidean space. For $x(t) \in \mathbb{R}^5$, we define the norm $\| \cdot \| = \sqrt{\cdot^T \cdot}$. For a positive number $r \leq +\infty$, we define $\Omega_r = \{z \in \mathbb{R}^5 : \| z \| \leq r \}$.

Then system (3) can be rewritten in the form of compact matrix as

$$
\dot{x}(t) = Ax(t) + B\omega(t)F(x(t)) + C,
$$

(4)

where

$$
A = 
\begin{bmatrix}
-a_1 & 0 & 0 & 0 & \beta \\
0 & -a_2 & 0 & 0 & d_{\gamma} \\
0 & \varepsilon & -a_3 & 0 & 0 \\
0 & \alpha & -a_4 & 0 & d(1 - \gamma) \\
0 & 0 & \xi & -a_5 & 0
\end{bmatrix},
B = [-1, 1, 0, 0, 0]^T,
\quad a_1 = \mu, \quad a_2 = \mu + \varepsilon,
\quad a_3 = \mu + \eta + \alpha, \quad a_4 = \mu + \zeta,
\quad a_5 = d + \mu + \beta,
\quad C = [\phi, 0, 0, 0, \varphi]^T.
$$

Let $G(t, x) = B\omega(t)F(x(t)) + C$. Since $G(t, 0) \equiv 0$, it is obvious that system (4) can be regarded as the non-zero time-varying perturbation to linear autonomous system. Take the initial condition

$$
x(0) = x_0, \quad x_0 \in \mathbb{R}^5,
$$

(5)

A solution of problem (4) is a function $x : [0, \infty) \rightarrow \mathbb{R}^5$, having a locally bounded derivative and satisfying (5) and (3) for all $t \in [0, \infty)$. Existence of solution is assumed. Furthermore, the following assumptions are satisfied:

$H_5$: There is a real number $\rho > 0$ such that $\|F(u) - F(v)\| \leq \rho \|u - v\|$, $F(0) = 0$ for all $u \in \mathbb{R}^5$ and $v \in \mathbb{R}^5$.

In addition, function $G(t, x)$ can be referred to as a continuous mapping $[0, \infty) \times \Omega_r \rightarrow \mathbb{R}^5$. In order to analyze the stability of system (4), we need show that the following lemma holds.

Lemma 1. For $\forall t \geq 0$, $\forall x \in \Omega_r$, the perturbation function $G(t, x)$ satisfies the following condition:

$$
\|G(t, x)\| \leq \delta \|x\|,
$$

(6)

where $\delta$ is a non-negative constant number, i.e., $\delta = \sqrt{\phi^2 + \varphi^2}$.

Proof. From (4), we have

$$
\|G(t, x)\|^2 = \|B\omega SI + C\|^2 = \|B\omega SI + C\|^T \|B\omega SI + C\| = 2(\omega SI)^2 - 2\phi(\omega SI)^2 + \phi^2 + \varphi^2 = (\omega SI)^2 + (\phi - \omega SI)^2 + \varphi^2.
$$
Because only a proportional of susceptible individuals are infected by the viruses and enter into the eclipse group, the condition below is of existence, \( \mu N(1 - B_2) \geq \omega SI \), i.e., \( \phi - \omega SI \geq 0 \), it is not difficult to obtain that
\[
(\omega SI)^2 + (\phi - \omega SI)^2 \leq (\omega SI + \phi - \omega SI)^2 = \phi^2.
\]
In addition, because \( S \) denotes the size of the susceptible group, i.e., \( S \geq 1 \), we have \( \|x\| \geq 1 \). Therefore, \( \|G(t,x)\|^2 \leq \phi^2 + \phi^2 \leq (\phi^2 + \phi^2)/\|x\|^2 \).

Obviously, there exists a non-negative constant number \( \delta = \sqrt{\phi^2 + \phi^2} \) such that
\[
\|G(t,x)\| \leq \delta\|x\|.
\]
This completes the proof. \( \square \)

4.2. Stability analysis of the SEIRW model

In the first place, we shall investigate the stability of the linear part of system (3):
\[
\dot{x}(t) = Ax(t).
\]
We can reasonably arrive at the following theorem.

Theorem 1. For \( \mu > 0, \varepsilon > 0, \eta > 0, \alpha > 0, \xi > 0 \) and \( \beta > 0 \), the linear system (7) is asymptotically stable.

Proof. It is well known that the trivial solution of the system (7) is asymptotically stable if all roots of the characteristic Eq. (8) have negative real parts, and unstable if one root has positive real part. The characteristic equation of system (7) can be given by
\[
\begin{bmatrix}
-a_1 - \lambda & 0 & 0 & 0 & \beta \\
0 & -a_2 - \lambda & 0 & 0 & d\gamma \\
0 & \varepsilon & -a_3 - \lambda & 0 & 0 \\
0 & 0 & \alpha & -a_4 - \lambda & d(1 - \gamma) \\
0 & 0 & 0 & \xi & -a_5 - \lambda
\end{bmatrix} = 0.
\]

It can be rewritten in the form of polynomial as
\[
(\lambda + a_1)(\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4) = 0.
\]

where
\[
b_1 = a_2 + a_3 + a_4 + a_5, b_2 = (a_2 + a_3)(a_4 + a_5) + a_2a_3 + a_4a_5 - \xi d(1 - \gamma), \\
b_3 = a_2a_4a_5(a_2 + a_3) + a_2a_4a_5 - \xi d(1 - \gamma)(a_2 + a_3), \\
b_4 = a_2a_3a_4a_5 - a_2a_3\xi d(1 - \gamma) - \xi d^2 \alpha \xi, \\
b_5 = a_2a_3a_4a_5 - a_2a_3\xi d(1 - \gamma) - \xi d^2 \alpha \xi.
\]

In order to study the stability of the equilibrium of system (7), we need to investigate the distribution of roots of Eq. (8). For the first term in the left-hand side of (8), we have \( \lambda = -a_1 < 0 \). It is clear that the equilibrium point of system (7) is asymptotically stable. Therefore, we only need to consider the following characteristic equation:
\[
\lambda^4(b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4) = 0.
\]

Since \( a_2a_5 = (\mu + \xi)(d + \mu + \beta) = \mu(d + \mu + \beta) + \xi d + \xi (\mu + \beta) > \xi d + \xi d > \xi d(1 - \gamma) \), it is clear that \( b_1 > 0, b_2 > 0 \) and \( b_3 > 0 \).

Furthermore, we have \( b_4 > 0 \), which is due to
\[
a_2a_3a_4a_5 = a_2a_3\mu d + a_3\xi d(\mu + \beta) + a_2a_3\xi d = a_2a_3\mu d + a_3\xi d(\mu + \beta) + a_2a_3\xi d(1 - \gamma) + a_2a_3\xi d^2 \gamma = a_2a_3\mu d + a_3\xi d(\mu + \beta) + a_2a_3\xi d(1 - \gamma) + a_2a_3\xi d(1 - \gamma) - \xi d^2 \alpha \xi
\]

According to Routh–Hurwitz criterion, all roots of the characteristic Eq. (8) will have negative real parts provided that the following conditions are satisfied:
\[
\Delta_1 = b_1b_2 - b_3 > 0
\]
and
\[
\Delta_2 = b_1(b_2 - b_3) - b_4b_5 > 0.
\]

In the following, we will prove the conditions (9a) and (9b) hold. It follows from (8) that
\[ \begin{align*}
\dot{b}_3(b_1b_2 - b_3) - b_3b_1^2 & \geq b_1(b_1 + a_3)^2(a_4 + a_5)(\mu d + b_4 + a_3) + 2a_3(b_1a_2 + a_3(a_4 + a_5) - b_1(a_2 + a_3)^2(a_4 + a_5)\dot{d}(1 - \gamma) \\
& + [\dot{d}(1 - \gamma)]^2(a_2 + a_3)(a_4 + a_5) + 2\dot{d}(1 - \gamma)(a_2 + a_3)(a_4 + a_5)(a_5 + a_3)
\end{align*} \]

Since \( a_4 + a_5 - d_5 + d_5' = \mu(d + \mu + \beta) + \dot{d}(d + \mu + \beta) > 0 \), we can get \( b_1b_2 - b_3 \geq 0 \). Therefore, the first condition (9a) is satisfied.

It is clear that the condition (9b) holds, i.e., \( \triangle_2 > 0 \). This completes the proof. \( \square \)

**Remark 1.** We can conclude from *Theorem 1* that the value of \( \gamma \) does not affect on the stability of system (7). In other words, the stability of system (7) is unrelated to the proportion of the partial immunity individuals to the exposed ones, or to the recovered ones. The stability criterion of system (7) is much relaxed.

Furthermore, we shall study the stability criteria of the system (4) with non-zero time-varying perturbation term.

**Definition 1.** If a continuous function \( \phi: [0, a) \rightarrow [0, \infty) \) is strictly increasing, and \( \phi(0) = 0 \), then \( \phi \) belongs to a \( K \)-class function. If \( a = \infty \) and \( \phi(r) \rightarrow \infty \) when \( r \rightarrow \infty \), then \( \phi \) belongs to a \( K^\infty \)-class function.

**Definition 2.** For a continuous function \( \varphi: [0, a) \times [0, \infty) \rightarrow [0, \infty) \), if for every fixed \( s \), function \( \varphi(r, s) \) is a \( \kappa \)-class function with regard to \( r \); for every fixed \( r \), function \( \varphi(r, s) \) is a decreasing function with regard to sand \( \varphi(r, s) \rightarrow 0 \) as \( s \rightarrow \infty \), then \( \varphi \) belongs to a \( KL \)-class function.

**Definition 3.** If there exist positive constant \( b \) and \( c \), which are not related with \( t_0 \), for each \( a \in (0, c) \), and a \( T \geq 0 \) being independence of \( t_0 \), such that

\[ ||x(t_0)|| \leq a \Rightarrow ||x(t)|| \leq b, \quad \forall t \geq t_0 + T, \]

then the solutions of system are uniformly ultimately bounded and the ultimate bound is \( b \).

**Lemma 2 33.** Assume \( D \subset \mathbb{R}^a \) is a definition domain including original point, for \( \forall t \geq 0 \) and \( \forall x \in D \), \( V: [0, \infty) \times D \rightarrow \mathbb{R} \) is a continuous differential function such that

\[ \varphi(||x||) \leq V(t, x) \leq \psi(||x||), \quad \text{and} \quad \dot{V}(t, x) \leq -\vartheta(x), \quad \forall ||x|| \geq u, \]

where \( \varphi \) and \( \psi \) are \( K \)-class function, \( \vartheta(x) \) is a continuous positive definition function. Let \( r > 0 \) make \( B_r \subset D \) and assume \( v < \varphi^{-1}(\varphi(r)) \). Then for every initial state \( x(t_0) \) such that \( ||x(t_0)|| \leq \vartheta^{-1}(\vartheta(r)) \), there exists a \( KL \)-class function \( \varphi \) and \( T \geq 0 \), which is not related with \( x(t_0) \) and \( v \), to make the solutions of system (4) satisfy

\[ \begin{align*}
||x(t)|| & \leq \varphi(||x(t_0)||, t - t_0), \quad \forall t \leq t_0 + T, \\
||x(t)|| & \leq \varphi^{-1}(\vartheta(v)), \quad \forall t \geq t_0 + T.
\end{align*} \]

By using the definitions and lemma above, we have the following theorem.

**Theorem 2.** The system (4) is asymptotically stable if there exists a symmetrical positive definite matrix \( P \) such that

\[ A^TP + PA = -I \quad \text{and} \quad \varphi^2 + \varphi^2 < 1/(2\lambda_{\max}(P)^2), \]

where \( I \) is the unit matrix, \( \lambda_{\max}(\cdot) \) and \( \lambda_{\min}(\cdot) \) denote the maximum and the minimum eigenvalues, respectively.

**Proof.** We can choose the candidate Lyapunov function to be

\[ V(t, x) = x^TPx. \]
Then the following relationship holds:
\[ \lambda_{\text{min}}(P)\|x\|^2 \leq V(x) \leq \lambda_{\text{max}}(P)\|x\|^2, \]

which indicates that the Lyapunov function is bounded.

It is not difficult to see that (12) is the function about \( t \) and \( x \). Calculating derivative of this function along the trajectories of system (4) yields
\[ \dot{V}(t,x) = \frac{\partial V}{\partial t} + \frac{\partial V}{\partial x} \dot{x} = \frac{\partial V}{\partial x} A x + \frac{\partial V}{\partial x} G(t,x). \]

The first two terms of the right side of (14) constitute the derivation of the trajectories of the linear part of system (4), and the third term is the result of perturbation.

Taking the derivative of (12) along the linear part of system (4), we have
\[ \dot{V}(t,x) = x^T (AP + PA) x. \]

By comparison with the first two terms of the right side of (14), it is easy to obtain that
\[ \frac{\partial V}{\partial t} = 0, \quad \frac{\partial V}{\partial x} A x = x^T (A^T P + PA) x. \]

We know from Theorem 1 that the matrix \( A \) is Hurwitz matrix and the linear system (7) is asymptotically stable. Thus \( A^T P + PA \) is positive definite. Without loss of generality, we let \( A^T P + PA = -Q \), where \( Q \) is a symmetrical positive definite matrix. Then we can get
\[ \frac{\partial V}{\partial x} A x = -x^T Q x \leq -\lambda_{\text{min}}(Q)\|x\|^2. \]

Moreover, the following relationship can be obtained:
\[ \|\frac{\partial V}{\partial x}\| = \|2x^T P\| \leq 2\|P\|\|x\| = 2\lambda_{\text{max}}(P)\|x\|. \]

According to Lemma 1 and the relationships (15) and (16), it follows that
\[ \dot{V}(t,x) = \frac{\partial V}{\partial t} + \frac{\partial V}{\partial x} A x + \frac{\partial V}{\partial x} G(t,x) 0 < -\lambda_{\text{min}}(Q)\|x\|^2 + \|\frac{\partial V}{\partial x}\|\|G(t,x)\| \leq -\lambda_{\text{min}}(Q)\|x\|^2 + 2\delta\lambda_{\text{max}}(P)\|x\|^2 \]
\[ = (-\lambda_{\text{min}}(Q) + 2\delta\lambda_{\text{max}}(P))\|x\|^2. \]

Thus, if \( \delta < \lambda_{\text{min}}(Q)/(2\lambda_{\text{max}}(P)) \), \( \dot{V}(t,x) < 0 \), i.e., the system (4) is asymptotically stable.

Obviously, the bound of \( \delta \) depends on the choose of \( Q \). Therefore, it is a challenge that how to chose \( Q \) makes \( \lambda_{\text{min}}(Q)/\lambda_{\text{max}}(P) \) maximum. It has been proven in [34] that when \( Q = I \), i.e., \( A^T P + PA = -I \), the goal can be reached.

From Lemma 1, we have that if \( \phi^2 + \rho^2 < 1/(2\lambda_{\text{max}}(P))^2 \), the system (4) is asymptotically stable. This completes the proof. \( \square \)

Remark 2. It can be concluded from Theorem 2 that the system (4) is asymptotically stable if the condition is satisfied:
\[ \rho^2 + \phi^2 < 1/(2\lambda_{\text{max}}(P))^2. \]
The condition implies that if the quadratic sum of the inflow of susceptible individuals and the inflow of vaccinated individuals to the vaccinated compartment, is below a constant value, the disease will tend to be stable, that is to say, the infectious disease will not be diffuse infinitely but will become endemic disease. This means that we can control the value of \( Bv \), the ratio of vaccination to newborn children, to postpone the outbreak of the disease and the epidemic disease becomes disappeared gradually.

In the proof of Lemma 1 and Theorem 2, we use
\[ \|G(t,x)\|^2 \leq \phi^2 + \rho^2 \leq (\phi^2 + \rho^2)\|x\|^2. \]

The right inequality can result in the larger bound of \( \|G(t,x)\| \), which can further induce the conservativeness of stability criterion of Theorem 2. In order to overcome this weakness, we can give the following result by only using
\[ \|G(t,x)\| \leq \sigma = \sqrt{\phi^2 + \rho^2}. \]

Theorem 3. If there exists a symmetrical positive definite matrix \( P \) and a \( T \geq 0 \), and for all \( t \geq 0, \ 0 < \theta < 1 \) and \( x \in \Omega_r = \{ x \in \mathbb{R}^n : \|x\| \leq r \} \), the condition \( \sigma < \frac{\theta k}{2\lambda_{\text{max}}(P)} \) holds, then for all \( \|x(t_0)\| \leq rk \), the system (4) is asymptotically stable, whose solutions are uniformly ultimately bounded and satisfy:
\[ \|x(t)\| \leq \left\{ \begin{array}{ll}
\frac{1}{\theta} \exp(-\zeta(t-t_0))\|x(t_0)\|, & \forall t_0 \leq t \leq t_0 + T, \\
\frac{1}{b}, & \forall t \geq t_0 + T.
\end{array} \right. \]
where \( k = \sqrt{\max_{P \in \mathcal{P}} \frac{1}{\min_{P} \phi}} \) \( \zeta = \frac{1}{\max_{P} \phi} \) and \( b = \frac{2\sigma_{\max}(P)}{d_{k}} \). \( \beta \) denotes the ultimate bound of the solutions of system (4).

**Proof.** Similar to the proof of Theorem 2, we can also choose a candidate Lyapunov function to be
\[
V(t, x) = x^{T}Px
\]
and let \( A^{T}P + PA = -I \), here, \( I \) denotes the unit matrix.

The derivative of this function along the trajectories of system (4) can be given by
\[
\dot{V}(t, x) = \frac{\partial V}{\partial t} + \frac{\partial V}{\partial x}Ax + \frac{\partial V}{\partial x}G(t, x) \leq -\lambda_{\min}(A^{T}P + PA)\|x\|^{2} + \max_{x} \left\| G(t, x) \right\| \leq -\|x\|^{2} + 2\sigma_{\max}(P)\|x\|.
\]

Thus for any \( \|x\| \geq 2\sigma_{\max}(P)/\theta \), we have
\[
\dot{V}(t, x) \leq -(1 - \theta)\|x\|^{2}.
\]

According to Lemma 2, we can get
\[
\dot{z}_{1}(\|x\|) = \lambda_{\min}(P)\|x\|^{2}, \quad \dot{z}_{2}(\|x\|) = \lambda_{\max}(P)\|x\|^{2}, \quad \dot{v}(x) = (1 - \theta)\|x\|^{2} \quad \text{and} \quad \nu = 2\sigma_{\max}(P)/\theta.
\]

Thus it follows that if the conditions (13) and (17) are satisfied, for each initial state \( x(t_{0}) \) such that \( \|x(t_{0})\| \leq \dot{z}_{2}^{-1}(z_{1}(r)) \), there exist a \( K\)-class function \( \varphi \) and a \( T \geq 0 \) to make the solutions of system (4) satisfy
\[
\|x(t)| < \varphi(\|x(t_{0})\|, t - t_{0}, \forall t_{0} \leq t \leq t_{0} + T, \|x(t)\| < \dot{z}_{1}^{-1}(z_{2}(\nu)), \forall t \geq t_{0} + T.
\]

From Lemma 2 and the converse function of \( z_{1} \) and \( z_{2} \), we can get
\[
\dot{z}_{1}^{-1}(z_{2}(\nu)) = \frac{2\sigma_{\max}(P)}{\nu}
\]

Without loss of generality, we can choose \( \varphi(\|x(t_{0})\|, t - t_{0}) = \frac{1}{k} \exp(-\zeta(t - t_{0}))\|x(t_{0})\| \), which obviously belongs to a \( K\)-class function

According to \( \nu < \dot{z}_{1}^{-1}(z_{1}(r)) \), i.e., \( 2\sigma_{\max}(P)/\theta < k \), we can obtain \( \sigma < \frac{\theta k}{2\sigma_{\max}(P)} \). This completes the proof. \( \square \)

**Remark 3.** Compared with the results in Theorem 2, the stability condition obtained in Theorem 3 is much relaxed, i.e., the condition of postponing the outbreak of the disease is easy to be satisfied. More importantly, the attractive region of the feasible solutions is provided. Biologically, the ultimate bound of the ratio of susceptible people, the exposed fraction, the infective individuals and the recovered fraction is given.

5. The SEIRW model with time-varying delay

In this section, by introducing time-varying delay \( \tau(t) \) to the susceptible individuals \( S(t) \) and the infective individuals \( I(t) \) in the SEIRW model (3), we investigate the effect of time delay on the stability of the SEIRW model. \( \tau(t) > 0 \) is assumed to be a variable incubation period during which the infection of the susceptible and clinical manifestation of infective is postponed because of the therapy treatment.

The system (3) can be transformed as follows:
\[
\begin{align*}
\dot{S} &= \phi + \beta W - \mu S - \omega(t)S(t - \tau(t))I(t - \tau(t)), \\
\dot{E} &= \omega(t)S(t - \tau(t))I(t - \tau(t)) - \mu E - d_{1}W, \\
\dot{I} &= \epsilon E - (\mu + \xi)I, \\
\dot{R} &= \lambda I - (\mu + \zeta)R + d(1 - \gamma)W, \\
\dot{W} &= \xi R - \mu W - d(1 - \gamma)W - d_{2}W - \beta W + \varphi,
\end{align*}
\]

which can be rewritten in the form of compact matrix as
\[
\dot{x}(t) = Ax(t) + \omega(t)H(x(t - \tau(t))) + C.
\]
where \( H(x(t - \tau(t))) = BS(t - \tau(t))I(t - \tau(t)) \). Other parameters are the same as in (4).

In this section, we introduce the following assumption:

(S1) Function \( \tau(t) \) is nonnegative, bounded and continuously differentiable defined on \( R_{+} \) and \( \inf_{t \in R_{+}} (1 - \tau(t)) > 0 \), where \( \tau(t) \) denotes the derivative of \( \tau(t) \) with respect to time \( t \).
Theorem 4. Assume that a \( a \in \mathbb{R} \) and \( b \in \mathbb{R} \) are defined and bounded on \( \mathbb{R}^+ \). Then we have the inequality

\[
abla \leq \frac{1}{p} a^p + \frac{1}{q} b^q.
\]

Lemma 3. Assume that \( a \geq 0, b \geq 0, p > 1, q > 1 \) with \( 1/p + 1/q = 1 \). Then we have the inequality

\[
abla \leq \frac{1}{p} a^p + \frac{1}{q} b^q.
\]

Lemma 4. Assume that \( u(t) \) is a differentiable function defined on \( \mathbb{R}^+ \). Then for any \( t \in \mathbb{R} \), the Dini upper right derivative \( D^+ u(t) \) of function \( u(t) \) exists and has the following expression:

\[
D^+ u(t) = \lim_{h \to 0^+} \sup_{h \neq 0} \frac{1}{h} |u(t + h) - u(t)| = \sigma(u(t)) \dot{u}(t),
\]

where

\[
\sigma(u(t)) = \begin{cases} 
1 & \text{if } u(t) > 0 \text{ and } \dot{u}(t) > 0, \\
-1 & \text{if } u(t) < 0 \text{ and } \dot{u}(t) < 0, \\
0 & \text{if } u(t) = 0 \text{ and } \dot{u}(t) = 0.
\end{cases}
\]

5.1. Boundedness of solutions

For model (21) to be mathematically tractable and epidemiologically meaningful, it is important to prove that all the state variables are nonnegative and bounded for all time. We prove that all solutions of system (21) with positive initial data will remain positive for all time \( t \geq 0 \).

Theorem 4. Assume that (S1) and (S2) hold. Then all solutions of system (21) are defined and bounded on \( \mathbb{R}^+ \).

Proof. Let \( x(t) = [x_1(t), \ldots, x_5(t)] \) be any solution of system (21) with the initial functions \( \varphi \in \mathcal{C}^0[-\tau, 0] \) at \( t = 0 \). System (21) can be transformed into the following form

\[
\dot{x}_i(t) = \sum_{j=1}^{5} A_{ij} x_j(t) + \omega(t) H_i(x_i(t - \tau(t))) + C_i,
\]

Calculating the upper right derivative \( D^+[x_i(t)]^n \), \( i = 1, \ldots, 5 \), we have
This is a contradiction. Hence, (22a) holds, which is due to that \( \dot{x}_i(t) \) and \( H(x_i(t - \tau(t))) \) are the \( i \)th elements of vectors \( x(t) \) and \( H(x(t - \tau(t))) \), respectively, that is, both of them are real number. As a result, it is not difficult to obtain that \( x_i(t) \leq |x_i(t)| \) and \( H(x_i(t - \tau(t))) \leq |H_i(x_i(t - \tau(t)))| \). The second inequality in (22a) holds, which is since \( |\omega(t)| \in [0,ka/4] \).

By using Young inequality, we further have

\[
\frac{rka}{4}|x_i(t)|^{\tau-1} |H_i(x_i(t - \tau(t)))| = \frac{rka}{4}|x_i(t)|^{\tau-1} \cdot |H_i(x_i(t - \tau(t)))|^{1/\tau} \leq \frac{ka}{4} \left[ (r - 1)|x_i(t)|^\tau + |H_i(x_i(t - \tau(t)))|^\tau \right].
\]

\[
rC_i|x_i(t)|^{\tau-1} = r(|x_i(t)|^\tau - (C_i)^{\tau-1}) \leq (r - 1)|x_i(t)|^\tau + C_i.
\]

Therefore we get

\[
D^+|x_i(t)|^\tau \leq r \sum_{j=1}^{5} A_{ij}|x_i(t)|^\tau + \frac{ka}{4} [(r - 1)|x_i(t)|^\tau + |H_i(x_i(t - \tau(t)))|^\tau] + (r - 1)|x_i(t)|^\tau + C_i^\tau
\]

\[
\leq \left( r \sum_{j=1}^{5} A_{ij} + r - 1 + \frac{ka}{4} (p + r - 1) + C_i^\tau \right) |x_i(t)|^\tau.
\]

Thus there exists a sufficient large constant \( w \) such that

\[
\max_{t - \tau \leq t \leq t + \tau} |H_i(x_i(t))|^\tau \leq p|x_i(t)|^\tau.
\]

Furthermore, we can obtain

\[
D^+|x_i(t)|^\tau \leq r \sum_{j=1}^{5} A_{ij}|x_i(t)|^\tau + \frac{ka}{4} [(r - 1)|x_i(t)|^\tau + |H_i(x_i(t - \tau(t)))|^\tau] + (r - 1)|x_i(t)|^\tau + C_i^\tau
\]

\[
\leq \left[ r \sum_{j=1}^{5} A_{ij} + r - 1 + \frac{ka}{4} (p + r - 1) + C_i^\tau |x_i(t)|^{-\tau} \right] |x_i(t)|^\tau.
\]

This is a contradiction. Hence, \( |x_i(t)| \leq w \) for all \( t \geq 0 \). Therefore, the solution \( x(t) = [x_1(t), \ldots, x_5(t)]^\tau \) of system (21) is defined and is bounded on \( R_+ \). This completes the proof. \( \square \)

### 5.2. Global exponential stability

In this subsection, by constructing new Lyapunov functional and using the technique of matrix analysis, we will establish new criteria on the global exponential stability of system (21).
Theorem 5. Assume that (S1) and (S2) hold. System (21) is globally exponentially stable if there exist 5-dimensional symmetrical positive definite matrix $P$, diagonal matrix $\alpha = \text{diag}(\alpha_1, \ldots, \alpha_5) > 0$, $F = \text{diag}(f_1, \ldots, f_5)$, and a constant $a > 0$ such that \( \lambda_{\text{min}}(D(t)) \geq a \), for all $t \in R_+$, where

\[
D(t) = -\hat{A}^T P - PA - \frac{1}{1 - \tau(t)} F^T \alpha F - \omega(t) P \alpha^{-1} \omega(t) P.
\]

Proof. Let $x^{(i)}(t) = [x_1(t), \ldots, x_5(t)]^T$, $i = 1, 2$ be any two solutions of system (21) satisfying the initial conditions $x^{(i)}(t) = \phi_i(\theta)$ for all $\theta \in [-\tau, 0]$, where $\phi_i(\theta) = [\phi_{i1}(\theta), \ldots, \phi_{i5}(\theta)] \in C^5[-\tau, 0]$ for $i = 1, 2$.

Let $z(t) = x^{(1)}(t) - x^{(2)}(t) = [z_1(t), \ldots, z_5(t)]$, where $z_1(t) = x_{11}(t) - x_{21}(t)$, $i = 1, \ldots, 5$, then system (21) is transformed into the following form

\[
\dot{z}(t) = Az(t) + \omega(t) H(z(t - \tau(t))),
\]

where $H(z(t - \tau(t))) = [H_1(z_1(t - \tau(t))), \ldots, H_5(z_5(t - \tau(t)))]$, and $H_i(z_i(t - \tau(t))) = H_i(x_{1i}(t - \tau(t))) - H_i(x_{2i}(t - \tau(t)))$. Let $\varepsilon$ be a constant. We construct the Lyapunov functional as follows:

\[
V(t, z) = z^T(t) P z(t) e^{\varepsilon t} + \sum_{i=1}^{5} \int_{t - \tau(t)}^{t} \frac{\alpha_i}{1 - \tau(s)} H_i^2(z_i(s)) e^{\varepsilon(t - s)} ds.
\]

By Theorem 4, $\dot{x}(t)$ is defined for all $t \in R$, and is bounded, which means that $z(t)$ is bounded on $R_+$. Furthermore, we obtain that $V(t, z(t))$ is also bounded on $R_+$.

The derivative of this functional along the solution of system (21) is

\[
\dot{V}(t, z) = e^{\varepsilon t} \left[ 2z^T(t) P z(t) + \varepsilon z^T(t) P z(t) + \sum_{i=1}^{5} \frac{\alpha_i}{1 - \tau(t)} H_i^2(z_i(t)) e^{\varepsilon(t - s)} - \sum_{i=1}^{5} \alpha_i H_i^2(z_i(t - \tau(t))) \right]
\]

Since

\[
2z^T(t) P \omega(t) H(z(t - \tau(t))) - H^T(z(t - \tau(t))) \alpha H(z(t - \tau(t))) \leq z^T(t) P \omega(t) \alpha^{-1} \omega(t) P^T z(t)
\]

and $H^T(z(t)) \alpha H(z(t)) \leq z^T(t) P \alpha^{-1} \omega(t) P^T z(t)$, we further obtain

\[
\dot{V}(t, z) \leq e^{\varepsilon t} \left[ z^T(t) (A^T P + PA) z(t) + \varepsilon z^T(t) P z(t) + \frac{\varepsilon e^{\varepsilon(t)}}{1 - \tau(t)} H^T(z(t)) \alpha H(z(t)) + z^T(t) P \omega(t) \alpha^{-1} \omega(t) P^T z(t) \right]
\]

Let

\[
D_1(t, \varepsilon) = \hat{A}^T P - PA - \varepsilon P - \frac{\varepsilon e^{\varepsilon(t)}}{1 - \tau(t)} F^T \alpha F - P \omega(t) \alpha^{-1} \omega(t) P^T.
\]

Hence, we have $\lim D_1(t, \varepsilon) = D(t)$ uniformly for all $t \in R_+$. It follows that there exists a constant $\varepsilon > 0$ such that $\lambda_{\text{min}} (D_1(t, \varepsilon)) \geq a$, for all $t \in R_+$. Therefore, we finally obtain

\[
\dot{V}(t, z) \leq -a e^{\varepsilon} z^T(t) z(t) \leq 0 \quad \text{for all } t \in R_+,
\]

which implies that

\[
V(t) \leq V(0) \quad \text{for all } t \geq 0.
\]

(25a)

Directly from (24) and assumption (S2) we have

\[
V(t) \geq z^T(t) P z(t) e^{\varepsilon t} \geq \lambda_{\text{min}}(P) e^{\varepsilon t} \sum_{i=1}^{5} z_i^2(t)
\]

for all $t \geq 0$ and

(25b)
\[ V(0) = z^T(0)p_2(0) + \sum_{i=1}^{5} \int_{-\tau(0)}^{0} \frac{z_i}{1 - \tau(s)} H_i^2(z_i(s)) e^{\alpha(s+\tau(s))} ds \]
\[ \leq \lambda_{\text{max}}(P) \sum_{i=1}^{5} \sup_{s_i \in [-\tau, 0]} (\phi_{1i}(s) - \phi_{2i}(s))^2 + \sum_{i=1}^{5} L_i \sup_{s_i \in [-\tau, 0]} (\phi_{1i}(s) - \phi_{2i}(s))^2 \leq M ||\phi_1(s) - \phi_2(s)||^2, \quad (25c) \]

where \( L_i = \sup_{s_i \in [-\tau, 0]} \frac{z_i}{1 - \tau(s)} e^{\alpha(s+\tau(s))} f_i^2(0) \) and \( M = \lambda_{\text{max}}(P) + \max_{i \leq 5} L_i \). Note that the first inequality in (25c) holds which is due to that according to assumption (S2), we have \( H_i^2(z_i(s)) \leq f_i^2 z_i^2(s) \).

Hence, by (25a)-(25c), we finally obtain
\[ \sum_{i=1}^{5} z_i^2(t) \leq M_0 ||\phi_1(s) - \phi_2(s)||^2 e^{-\alpha t}, \quad \text{for all} \ t \geq 0, \]

where \( M_0 = M_0 \lambda_{\text{min}}(P) \geq 1 \) is a constant and is independent of any solution of system (21). We further obtain that system (21) is globally exponentially stable, and the proof is thus completed. \( \square \)

In Theorem 1, if we choose \( \alpha = \sigma E \), where \( \sigma > 0 \) is a constant and \( E \) is a unit matrix, then we have
\[ D(t) = \sigma \left[ -A^TP - AP - \frac{1}{1 - \tau(t)} F^T F - \sigma \omega^2(t) P^2 \right]. \]

Thus we obtain the following corollary as a special case of Theorem 1.

**Corollary 1.** Suppose that (S1) holds. If there exist 5-dimensional symmetrical positive definite matrix \( P \) and constants \( \sigma > 0 \) and \( \omega > 0 \) such that
\[ \lambda_{\text{min}}(-A^TP - AP - \frac{1}{1 - \tau(t)} F^T F - \sigma \omega^2(t) P^2) \geq \sigma. \quad \text{for all} \ t \in R, \]

then system (21) is globally exponentially stable.

Since \( \omega(t) \) belongs to \([0, ka/4]\) and achieves maximum when the time \( t = \ln b/a \), it follows from (25) that
\[ \dot{V}(t, z_i) \leq -e^{\alpha t} z_i^T(t) \left[ -A^TP - PA + eP - e^{\alpha t} \frac{1}{1 - \tau(t)} F^T xF - \frac{ka}{4} P x^{-1} P \right] z_i(t) \]
\[ \leq -e^{\alpha t} z_i^T(t) \left[ -A^TP - PA + eP - e^{\alpha t} \frac{1}{1 - \tau(t)} F^T xF - \left( \frac{ka}{4} \right)^2 P x^{-1} P \right] z_i(t) \]

Let
\[ D_1(t, \varepsilon) = -A^TP - PA - eP - e^{\alpha t} \frac{1}{1 - \tau(t)} F^T xF - \left( \frac{ka}{4} \right)^2 P x^{-1} P. \]

Hence, we have \( \lim_{\varepsilon \to 0} D_1(t, \varepsilon) = D(t) \) uniformly for all \( t \in R \), where
\[ D(t) = -A^TP - PA - \frac{1}{1 - \tau(t)} F^T xF - \left( \frac{ka}{4} \right)^2 P x^{-1} P. \]

Thus we have the following corollary.

**Corollary 2.** Suppose that (S1) holds. If there exist 5-dimensional symmetrical positive definite matrix \( P \), diagonal matrix \( \alpha = \text{diag}(\alpha_1, \ldots, \alpha_5) > 0 \) and a constant \( \sigma > 0 \) such that \( \lambda_{\text{min}}(D(t)) \geq \sigma \), for all \( t \in R \), where
\[ D(t) = -A^TP - PA - \frac{1}{1 - \tau(t)} F^T xF - \left( \frac{ka}{4} \right)^2 P x^{-1} P, \]

then system (21) is globally exponentially stable.

### 6. Simulation results

In this section, we shall verify the correctness and effectiveness of the stability conditions in Theorems 2 and 3. In the first place, we shall show that the system (4) is asymptotically stable when the stability conditions in Theorem 2 are satisfied. According to the biologically practical meanings, without loss of generality, we let \( \mu = 1.5 \times 10^{-3}, \ v = 0.85, \ \eta = 0.65, \ \alpha = 0.88, \ \xi = 0.85, \ d = 0.85, \ \beta = 0.35 \). It is not difficult to obtain that there exists a symmetrical positive definite matrix \( P \) such that \( A^TP + PA = -I \).
whose eigenvalues are 0.2212, 0.3088, 0.4920, 1.4456, and 13.8226. Thus we have $\lambda_{\min}(P) = 0.2212$ and $\lambda_{\max}(P) = 13.8226$. Since the bound of $\omega(t)$ is less than 1, it follows that $ak/4 \leq 1$. In this case, we might as well let $a = 0.45$, $b = 0.25$, $k = 8$, $N = 1000$, $z = 2.0 \times 10^{-4}$. The simulation results are depicted in Fig. 2. From the results of numerical computation, we can see that if the stability conditions in Theorem 2 are satisfied, the system (4) is asymptotically stable.

Furthermore, we shall verify the effectivity of Theorem 3. We assume that $r = 500$, $\theta = 0.65$. Then we have $k = \sqrt{\frac{\lambda_{\min}(P)}{\lambda_{\max}(P)}} = 0.1265$, $\zeta = 0.01266$. The following relations can be obtained:

$$2 \leq \phi^2 + \theta^2 < 2.2112 \quad \text{and} \quad \sqrt{2} < \sigma < 1.487. \quad (20)$$

We further find that the bound of $Bv$ is $0.3375 < Bv < 0.6625$, which implies that we can control the ratio of vaccination to newborn children, to interrupt the outbreak of the disease or to slow the transmission of the infectious disease. According

![Fig. 2. The waveform plot of the variables $S,E,I,R,W$ with respect to time $t$ for system (4) when the stability conditions in Theorem 2 are satisfied.](image1)

![Fig. 3. The waveform plot of the variables $S,E,I,R,W$ with respect to time $t$ for system (4) when the stability conditions in Theorem 3 are satisfied.](image2)
to $\|x(t_0)\| \leq 63.25$, we assume $x(t_0) = [40 \ 20 \ 15 \ 10 \ 20]^T$. It is not difficult to see that the system (4) is asymptotically stable when the stability conditions in Theorem 3 are satisfied.

More importantly, we let $\sigma = 1.425$ and the ultimate bound of the solutions of system (4) can be given by $b = 479.1$, which implies that the value of $b$ works upon the transmission of the infectious disease, that is the growth function of the pathogens has a very close connection with the disease spread. The values of $\sigma$ and $b$ are the conditions of influencing the stability of the system (4) and the ratio of vaccination to newborn children. Practically, government can control $Bv$ by vaccinating the newborn children to delay the outbreak of the disease.

From Fig. 3, we can also see that during the initial stage, owing to vaccination to newborn children, the initial values of $W$ and $S$ do not equal to 0. As time goes on, if the conditions are satisfied, the system will be stable. At last, the infected individuals will achieve minimal number and the disease will be endemic diseases rather than diffused.

It is shown from Fig. 4 that the speed of the viruses’ growth descends faster as parameter $a$ increases while it decreases and trends to zero as time $t$ goes on. Furthermore, we can find that in the case of $0 < a < 1$, when time $t$ is about 2 days, the infectious rate of viruses is a constant, 0.3, which is independent on the parameter choice of $\alpha(t)$. Thus, we have $t = 2$, which implies that if we let $t_0 = 0$, the ultimate bound of the solutions of system (4) is time-varying when time $t$ is less than 2, otherwise, it is a constant. This time point is the best one that the governments control the infectious rate of viruses.

It is shown from Fig. 5 that in the initial time, when $0 < t < 1$, as time $t$ goes on, the individuals in incubation time, $E(t)$, increases rapidly. After $E(t)$ goes to the apex of the curve, it decreases rapidly and trends to zero as time $t$ goes on. On the
point of the apex, the individuals $E(t)$ in incubation period increases with the increase of the parameter $a$. From Fig. 5 we can also conclude that it is the inflection of the curve when time $t$ is about 2 years. $E(t)$ decreases slowly and trends to stability when time $t$ is greater than 2 while $E(t)$ decreases as parameter $a$ increases. We can conclude that the number of infected individuals is related with the value of $a$, the index of the infectious rate function.

In the following, we shall verify the correctness and effectiveness of the stability conditions in Theorem 5 and its corollaries. We let $\tau(t) = 1 + 1/2\sin(t)$, $a = 0.045$, $Z = 3 \times 10^{-3}$ and $F = \text{diag}(Z, Z, Z, Z, Z)$. Other parameters are the same as the verification for Theorem 2. We obtain that $\inf_{t \in R} \{1 - \tau(t)\} \geq 1/2$. Furthermore, it is not difficult to see that the assumptions (S1) and (S2) are satisfied. We choose $\tau(t) = 1/2$, and it follows from Corollary 2 that there exist 5-dimensional symmetrical positive definite matrix $P$, diagonal matrix $\alpha = \text{diag}(2.2773, 2.7832, 3.0514, 1.7118, 2.3170) > 0$ such that $\lambda_{\min}(D(t)) = 0.9130$, where

$$P = \begin{bmatrix}
2.9578 & -0.3050 & -0.3127 & -0.8650 & 0.0003 \\
-0.3050 & 2.3276 & 0.9802 & 1.4294 & 0.5805 \\
-0.3127 & 0.9802 & 2.4207 & 2.5510 & 0.5578 \\
-0.8650 & 1.4294 & 2.5510 & 6.2269 & 1.0987 \\
0.0003 & 0.5805 & 0.5578 & 1.0987 & 1.6651
\end{bmatrix}.$$

Hence, we can see that there exists a constant $\sigma > 0$ such that $\lambda_{\min}(D(t)) \geq \sigma$, for all $t \in R$. This shows that from Corollary 2, system (21) is globally exponentially stable, as shown in Fig. 6, which implies that the treatment or rational immunization for the susceptible or infective individuals may hasten the disease dying out.

7. Discussions and conclusions

In this paper, we present a new epidemic model, SEIRW model, with human interference behaviors such as vaccination and therapy treatment. Furthermore, the stability of the model with variable coefficients in five dimensions is analyzed so as to predict the evolution tendency of the disease. One of our goals is to obtain the relatively accurate conditions to ensure the asymptotic stability of the SEIRW model, in other words, to give the practical conditions that the infectious disease will not diffuse infinitely and disappear gradually. This study can quantify the effects of human interference behaviors like vaccination on the diseases spread. Furthermore, we introduce the time-varying delay into the susceptible individuals and the infective individuals in the SEIRW model and analyze the global exponential stability of the SEIRW model with time-varying delay. Our results show that rational immunization for the susceptible or infective individuals may hasten the disease dying out. Finally, the numerical simulations verify the results.

Therefore, one of the main aims of our research is to activate epidemiological surveys for purpose of deepening current knowledge of vaccinating behavior to the newborn children and perform therapy treatment to the infective. The study takes into account such human interference behaviors and quantifies their effects on the spread of the infectious diseases. Thus, some key efforts might be made to public healthy authorities, for example, rational immunization could be acceptable only in the case where the spontaneous baseline vaccination rate fulfills the reverse of inequality. The challenge to the infectious disease epidemiologist is to resolve the concealed determinants of the human interference behaviors of the transmission of
the infectious disease and discriminate rules that could enable effective controls to be identified and this behavior to be predicted.

Acknowledgment

The work described in this paper was supported by the Grants from the Natural Science Foundation of Chongqing (CSTC, cstcjjA40003).

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