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Research article

Global stability mathematical analysis for virus transmission model with latent age structure

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Abstract: *Background and objective:* Mathematical model is a very important method for the control and prevention of disease transmissing. Based on the communication characteristics of diseases, it is necesssery to add fast and slow process into the model of infectious diseases, which more effectively shows the transmission mechanism of infectious diseases. *Methods:* This paper proposes an age structure epidemic model with fast and slow progression. We analyze the model's dynamic properties by using the stability theory of differential equation under the assumption of constant population size. *Results:* The very important threshold R_0 was calculated. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, whereas if $R_0 > 1$, the Lyapunov function is used to show that endemic equilibrium is globally stable. Through more in-depth analysis for basic reproduction number, we obtain the greater the rate of slow progression of an infectious disease, the fewer the threshold results. In addition, we also provided some numerical simulations to prove our result. *Conclusions:* Vaccines do not provide lifelong immunity, but can reduce the mortality of those infected. By vaccinating, the rate of patients entering slow progression increases and the threshold is correspondingly reduced. Therefore, vaccination can effectively control the transmission of Coronavirus. The theoretical incidence predicted by mathematical model can provide evidence for prevention and controlling the spread of the epidemic.

Keywords: age structure; Lyapunov function; infection equilibrium; global stability; prevention and control

1. Introduction

COVID-19 is an emerging acute infectious disease, which incubation period is 1–14 days, usually 3–7 days. COVID-19 generally have no obvious precursors, the infection will be after the Coronavirus disease early, some patients may also have no obvious symptoms, most common in patients with fever,

dry cough, lack of power as the main performance, but beyond that, due to individual differences, the patient also can appear muscle pain, chest tightness, pharyngeal itching, runny nose. The lack of specificity, which can be seen in many diseases and is not unique to COVID-19, makes it difficult for patients to identify infection by symptoms themselves. Some immunity can be acquired after infection or after the cornavirus vaccine, but the duration is unclear.

The establishing and analyzing of mathematical models play an important role in the control and prevention for disease transmissing. McKendrick first proposed the PDE formulation for the age distribution of a population [1]. Ever since the research results by Kermack and McKendrick [2], Hoppensteadt [3], Iannalli [4] and Webb [5], age structure models have been widely used in the study of transmission dynamics of infectious diseases [6–8]. Recently, Bentout Soufiane et al. considered an alcoholism model for age structure and investigated the glabal behavior [9]. In December 2019, some medical institutions of Wuhan reported some cases of pneumonia of unknown cause. On 11 February 2020, the World Health Organization officially named the pneumonia contracted by the Novel Coronavirus as "COVID-19". Recently many scholars have studied on COVID-19, which all proposed and studied the dynamical model which has helped to control infectious diseases. Glenn Webb proposed a model of a COVID-19 epidemic which is developed to predict the effectiveness of vaccination [10]. B. Tang et al. proposed calculation of the basic reproduction number by virtue of mathematical modeling can help decide the potential and severity of an outbreak and provide critical information for identifying the type of disease interventions and intensity [11]. J. Jiao et al. presents an SEIR epidemic model with infectivity in incubation period and homestead-isolation on the susceptible [12]. Besides, deep learning frameworks [13] also can be used for prediction virus spread and a more reliable model incorporating more parameters input into a neural network based virus transmission predictor may be implemented.

McCluskey assumed that infected individuals can develop disease by either of two pathogenic machanism: fast progression or endogenous reactivation [14]. Generally, acute infectious diseases develop through four stages, among which the incubation period is very important, which refers to the period from pathogen invasion to the onset of clinical symptoms. Take COVID-19 for example which has a certain incubation period. According to current epidemiological statistics, the incubation period is about 1–14 days. Some patients will show symptoms on the day of infection or one or two days later, while some patients will have a longer incubation period of about two weeks. Consequently, it is necessary to consider the factors of fast and slow progress in infection modeling.

Besides, the treatment of disease is proportional due to medical resources. Capasso and Serill [15] introducing saturated incidence in the cholera epidemic model, describes the tingible into infected class average relationship $g(I) = kI/(1+\mu), k, \mu > 0$, which said the disease infection ability, said a crowded or change on the influence of the individual, the increase in the number of infected people, easy to dye more vigilant, lead to easy dyeing behaviour change or when the disease is especially crowded in the environment may be unlimited effective contact.

2. Materials and methods

Zunyou Wu who is the chief expert of epidemiology at the Chinese Center for Disease Control and Prevention, said there are three possible cases of re-positive symptoms for COVID-19: first, false negative or false positive nucleic acid test; second, the virus is active again; third, reinfection. Zijian

Feng who is the deputy director of the Chinese Center for Disease Control and Prevention, said that the re-positive case caused continued transmission is rare and does not play a big role. Recently, some provinces in China have seen new cases of COVID-19 including asymptomatic patients. Therefore an infected individual ows into the exposed class after been treated but not becomes susceptible in our models. Inspired by the above discussions, we consider an SEI epidemic model which introduce the latent age, the nonlinear incidence of reactive unsaturated treatment and the saturation treatment function.

$$\begin{cases} \dfrac{dS(t)}{dt} = \Lambda - \beta S I - \mu S, \\ \dfrac{\partial e(t,a)}{dt} + \dfrac{\partial e(t,a)}{da} = -(\mu + \alpha(a))e(t,a), \\ \dfrac{dI(t)}{dt} = \displaystyle\int_0^{\infty} \alpha(a)e(t,a)da + (1-p)\beta S I - f(I) - (d+\mu)I, \end{cases} \tag{2.1}$$

for boundary and initial values conditions

$$\begin{aligned} &e(t,0) = p\beta S I + f(I), \\ &S(0) = S_0,\ e(0,a) = e_0(a),\ I(0) = I_0. \end{aligned} \tag{2.2}$$

where $S(t), e(t,a)$ and $I(t)$ respectively be the population sizes of susceptible, latent and infective classes. We assume that the population size is changeless. β represents susceptible people in contact with an infected person transmit rate, p represents susceptible people contact with an infected person enter slow propagation process called latent stage which denoted by e(t,a) where individuals are infected with disease but are not yet contagious, where a is called the age of latency progression, which is the duration of the incubation period. We denote $E(t) = \int_0^{+\infty} e(t,a)da$ as the latent individuals' total density. $1-p$ represents susceptible people and the onset of contact after rapid development for the onset of ratio, μ and d represent people natural mortality and mortality due to illness. $f(x) = \gamma x/(1+mx)$ represents the saturation treatment function where the γ is cure rate of the disease.

We define $X = R^+ \times L^1_+(0,+\infty) \times R^+$, equipped with the norm $\|(x_1,x_2,x_3)\|_X = |x_1| + \int_0^{\infty} x_2(a)da + |x_3|$. The initial condition of system (2.1) belongs to the positive cone of X, then can be rewritten as $x_0 = (S_0, e_0(\cdot), I_0) \in X$. We can get a continuous semi-flow associated with system (2.1), that is, $\Theta : R^+ \times X \longrightarrow X$ produced be system (2.1) adopts the following form, $\Theta(t,x_0) = (S(t), e(t,\cdot), I(t)), t \geq 0, x_0 \in X$, with

$$\| \Theta(t,x_0) \|_X = |(S(t)| + \int_0^{+\infty} |e(t,\cdot)|da + |I(t)|. \tag{2.3}$$

For simplicity, let $\varepsilon(s) = u + \alpha(s), \theta = \int_0^{\infty} \alpha(a)e^{-\int_0^a \varepsilon(\tau)d\tau}da, K_0(a) = e^{-\int_0^a \varepsilon(\tau)d\tau}$. The second equation of system (2.1) is solved along $t - a = constant$

$$e(t,a) = \begin{cases} (p\beta S(t-a)I(t-a) + \dfrac{\gamma I(t-a)}{1+mI(t-a)})K_0(a), & t > a \geq 0, \\ e_0(a-t)\dfrac{K_0(a)}{K_0(a-t)}, & a \geq t \geq 0, \end{cases} \tag{2.4}$$

Let $\Omega = \left\{(S(t), e(t,\cdot), I(t)) \in X,\ S(t) + \int_0^{+\infty} e(t,a)da + I(t) \leq \frac{\Lambda}{\mu}\right\}$. The interior of Ω is $\mathring{\Omega}$. we can easily verify the non-negative and positive invariance set of the system (2.1) with the help of article [5].

We note that if $R_0 > 1$, Ω is the positive invariant set for Θ, and it attracts all solutions of the system (2.1) with non-negative initial conditions.

2.1. Reproduction number

Firstly, there is a disease-free equilibrium point $E_0 = (S_0, 0, 0)$ in the system (2.1), where $S_0 = \frac{\Lambda}{\mu}$, and we define the basic reproduction number of the system (2.1) as following

$$R_0 = \frac{p\beta\Lambda\theta + \gamma\mu\theta + (1-p)\beta\Lambda}{(\gamma + d + \mu)\mu}$$

and it is easily known that if $R_0 > 1$, system (2.1) has an only positive endemic equilibrium point $E_*(S^*, e^*(a), I^*)$, where

$$S^* = \frac{\Lambda}{\beta I^* + \mu}, \quad e^*(a) = (p\beta S^* I^* + \frac{\gamma I^*}{1 + mI^*})e^{-\int_0^a \varepsilon(\tau)d\tau}.$$

Define space

$$\begin{aligned} X &:= R^3 \times L^1((0,+\infty), R), \\ X_0 &:= R^2 \times 0 \times L^1((0,+\infty), R), \\ X_+ &:= R^3_+ \times L^1_+((0,+\infty), R), \end{aligned}$$

and $X_{0+} = X_+ \bigcap X_0$. Define operator $T : D(T) \subset X \to X$,

$$T\begin{pmatrix} S \\ I \\ \begin{pmatrix} 0 \\ e \end{pmatrix} \end{pmatrix} = \begin{pmatrix} -\mu \\ -(d+\mu) \\ \begin{pmatrix} -e(0) \\ -e' - (\mu + \alpha(a))e \end{pmatrix} \end{pmatrix}$$

where $D(T) = R \times R \times 0 \times W^{1,1}((0,+\infty), R)$. Consider nonlinear mapping $F : X_0 \to X$, for

$$F\begin{pmatrix} S \\ I \\ \begin{pmatrix} 0 \\ e \end{pmatrix} \end{pmatrix} = \begin{pmatrix} \Lambda - \beta S I \\ \int_0^{\infty} \alpha(a)e(t,a)da + (1-p)\beta S I - f(I) \\ \begin{pmatrix} p\beta S I + f(I) \\ 0_{L^1} \end{pmatrix} \end{pmatrix}$$

define

$$U(t) = \left(S(t), I(t), \begin{pmatrix} 0 \\ e(t,\cdot) \end{pmatrix} \right)^T$$

Therefore systerm (2.1) can be rewritten as an abstract Canchy problem

$$\frac{dU(t)}{dt} = TU(t) + F(U(t)) \ \ t \geq 0, \ \ U(0) = u_0 \in X_{0+}. \tag{2.5}$$

Draw on the results in Magal [16] and Magal and Thiemel [17], there exists an uniquely deterministic semiflow $\{U(t)\}_{t\geq 0}$ on X_{0+} which is bound dissipative and asymptotically smooth, and $\{U(t)\}_{t\geq 0}$ has a global attractor $T \subset X$ which attracts the bounded sets of X.

2.2. Locally asymptotic stability

Theorem 2.1. *If $R_0 \leq 1$, the disease-free equilibrium E_0 is locally asymptotically stable.*

Proof. Let $x_1(t) = S(t) - S_0$, $x_2(t) = e(t,a)$, $x_3(t) = I(t)$, linearizing the system (2.1) at E_0, and considering the exponential solution $x_1(t) = x_1^0 e^{\lambda t}$, $x_2(t,a) = x_2^0 e^{\lambda t}$, $x_3(t) = x_3^0 e^{\lambda t}$, we can derive

$$\begin{cases} (\lambda+\mu)x_1^0 + \beta S_0 x_3^0 = 0, \\ \dfrac{dx_2^0(a)}{da} = -(\lambda+\mu+\alpha(a))x_2^0(a), \\ x_2^0(0) = p\beta S_0 x_3^0 + \gamma x_3^0, \\ \lambda x_3^0 = \displaystyle\int_0^\infty \alpha(a)x_2^0(a)da + (1-p)\beta S_0 x_3^0 - (\mu+d+\gamma)x_3^0. \end{cases} \tag{2.6}$$

Integrating the second equation of system (2.6) from 0 to a, considering the boundary condition, deduces $x_2^0(a) = (p\beta S_0 x_3^0 + \gamma x_3^0)e^{-(\lambda+\mu)a - \int_0^a \alpha(s)ds}$. Substituting $x_2^0(a)$ into the fourth equation of system (2.6), solving it, we get the characteristic equation

$$H(\lambda) = -\lambda + \int_0^\infty \alpha(a)(\gamma + p\beta\frac{\Lambda}{\mu})e^{-(\mu+\lambda)a}e^{-\int_0^a \alpha(\tau)d\tau}da + (1-p)\beta\frac{\Lambda}{\mu} - (d+\mu+\gamma).$$

easily know $H'(\lambda) < 0$ which implies that $H'(\lambda)$ is a decreasing function, and

$$\lim_{\lambda\to+\infty} H(\lambda) = -\infty, \ \lim_{\lambda\to-\infty} H(\lambda) = +\infty, \ H(0) = (d+\mu+\gamma)(R_0-1).$$

Let $\lambda = x + yi$ is an arbitrary complex root of $H(\lambda) = 0$, then

$$\begin{aligned} H(x+yi) = -x + \int_0^\infty \alpha(a)(\gamma + p\beta\frac{\Lambda}{\mu})e^{-(\mu+x)a}e^{-\int_0^a \alpha(\tau)d\tau}cos(ya)da + (1-p)\beta\frac{\Lambda}{\mu} - (d+\mu+\gamma) \\ - (iy + \int_0^\infty \alpha(a)(\gamma + p\beta\frac{\Lambda}{\mu})e^{-(\mu+x)a}e^{-\int_0^a \alpha(\tau)d\tau}isin(ya)da) = 0. \end{aligned}$$

be equivalent to

$$\begin{aligned} -x + \int_0^\infty \alpha(a)(\gamma + p\beta\frac{\Lambda}{\mu})e^{-(\mu+x)a}e^{-\int_0^a \alpha(\tau)d\tau}cos(ya)da + (1-p)\beta\frac{\Lambda}{\mu} - (d+\mu+\gamma) = 0, \\ y + \int_0^\infty \alpha(a)(\gamma + p\beta\frac{\Lambda}{\mu})e^{-(\mu+x)a}e^{-\int_0^a \alpha(\tau)d\tau}sin(ya)da = 0. \end{aligned}$$

And because $0 = -x + \int_0^\infty \alpha(a)(\gamma + p\beta\frac{\Lambda}{\mu})e^{-(\mu+x)a}e^{-\int_0^a \alpha(\tau)d\tau}cos(ya)da + (1-p)\beta\frac{\Lambda}{\mu} - (d+\mu+\gamma) \leq H(x)$ then $0 = H(\lambda) \leq H(x)$ which stands for $x < \lambda^*$, where λ^* is the unique real root of $H(\lambda) = 0$. Thus if and only if $R_0 \leq 1$, all the roots of system (2.6) have negative real part.

Theorem 2.2. *If $R_0 > 1$, the unique endemic equilibrium E_* is locally asymptotically stable.*

Proof. The perturbation variables are as follows

$$y_1(t) = S(t) - S^*, \ \ y_2(t,a) = e(t,a) - e^*(a), \ \ y_3(t) = I(t) - I^*.$$

Linearizing system (2.1) at E_* and assuming the form of solution is as follows

$$y_1(t) = y_1^0 e^{\lambda t}, \;\; y_2(t,a) = y_2^0(a)e^{\lambda t}, \;\; y_3(t) = y_3^0 e^{\lambda t}, \tag{2.7}$$

where $y_1^0, y_2^0(a), y_3^0$ will be determined. we get

$$\begin{cases} 0 = (\lambda + \beta I^* + \mu)y_1^0 + \beta S^* y_3^0, \\ \dfrac{dy_2^0(a)}{da} = -(\lambda + \mu + \alpha(a))y_2^0(a), \\ y_2^0(0) = p\beta y_1^0 I^* + p\beta y_3^0 S^* + \dfrac{\gamma}{(1+mI^*)^2}y_3^0, \\ \lambda y_3^0 = \displaystyle\int_0^\infty \alpha(a)y_2^0(a)da + (1-p)\beta(y_1^0 I^* + y_3^0 S^*) \\ \qquad\qquad -\frac{\gamma}{(1+mI^*)^2}y_3^0 - (d+\mu)y_3^0. \end{cases} \tag{2.8}$$

By calculating, we can get the following eqution

$$\begin{aligned} &[\lambda + \frac{\gamma}{(1+mI^*)^2} + (d+\mu) - (1-p)\beta S^*]y_3^0 - (1-p)\beta I^* y_1^0 \\ &= p\beta I^* y_1^0 W(\lambda) + (p\beta S^* + \frac{\gamma}{(1+mI^*)^2})y_3^0 W(\lambda), \end{aligned} \tag{2.9}$$

where $W(\lambda) = \int_0^\infty \alpha(a)e^{-(\lambda+\mu)a - \int_0^\infty \alpha(\tau)d\tau}da$ satisfies the following properties

$$W(0) = \theta, \quad \lim_{\lambda\to-\infty} W(\lambda) = +\infty, \quad \lim_{\lambda\to+\infty} W(\lambda) = \int_0^\infty \alpha(a)da.$$

From the first equation of system (2.8) and the Eq (2.9), we obtain the following characteristic equation

$$\begin{aligned} (\lambda+\mu+\beta I^*)(\lambda+\mu+d) =&(\lambda+\mu+\beta I^*)\{(W(\lambda)-1)\frac{\gamma}{(1+mI^*)^2} \\ &+ [(1-p)\beta + p\beta W(\lambda)]\frac{\Lambda}{\beta I^*+\mu}\} \\ &- \frac{\beta\Lambda}{\beta I^*+\mu}[(p-1)\beta I^* - p\beta I^* W(\lambda)] \end{aligned} \tag{2.10}$$

Note M is the right side of the Eq (2.10). Assuming $\lambda > 0$, M satisfies the following inequality

$$\mid M \mid \leq R_0(\lambda + \beta I^* + \mu + 1)(\gamma + d + \mu). \tag{2.11}$$

which is equivalent to

$$h(\lambda) = \lambda^2 + P\lambda + Q \leq 0.$$

where

$$\begin{aligned} P &= \beta I^* + 2\mu + d - R_0(\gamma + d + \mu), \\ Q &= (\beta I^* + \mu)(d+\mu) - R_0(\beta I^* + \mu + 1)(\gamma + d + \mu). \end{aligned}$$

Since $\lambda > 0$, therefore

$$\frac{(\beta I^*+\mu)(d+\mu)}{(\beta I^*+\mu+1)(\gamma+d+\mu)} > R_0.$$

Let $A = \frac{(\beta I^*+\mu)(d+\mu)}{(\beta I^*+\mu+1)(\gamma+d+\mu)}$, easily know $A < 1$, i.e. $R_0 < 1$ which is contradict with $R_0 > 1$. then we can get $\lambda < 0$.

2.3. Uniform persistence

Theorem 2.3. *If $R_0 > 1$, there exists $\varepsilon > 0$, such that for each $\forall\ y \in M_0$, $\lim\limits_{t\to+\infty} inf d(U(t)y, \partial M_0) \geq \varepsilon$. Furthermore, there exists a compact subset $H_0 \subset M_0$ which is a global attractor for $\{U(t)\}_{t\geq 0}$ in M_0.*

Proof. Define $M_0 = \{(S, I, 0, e)^T \in X_{0+} : T(t) = I + \int_0^\infty e(a)da > 0\}$, and $\partial M_0 = X_{0+}\backslash M_0$. Firstly, let $(S^0, I_0, 0, e_0) \in M_0$, we can get $T'(t) \geq -max\{d+\mu, \mu+\alpha_{max}\}T(t)$, where $\alpha_{max} = ess\sup_{a\in(0,\infty)}\alpha(a)$, easily know $U(t)M_0 \subset M_0$. Secondly, if $(S^0, I_0, 0, e_0) \in \partial M_0$, with the help of system (2.4), we know if $t \geq 0$, we get $I(t) = 0$ and $\int_0^\infty e(t,a)da \to 0$, for $t \to +\infty$. Therefore $U(t)\partial M_0 \subset \partial M_0$. Let $(S^0, I_0, 0, e_0) \in \partial M_0$, we get

$$\begin{cases} \dfrac{dI(t)}{dt} = \displaystyle\int_0^\infty \alpha(a)e(t,a)da + (1-p)\beta S I - f(I) - (d+\mu)I, \\ \dfrac{\partial e(t,a)}{dt} + \dfrac{\partial e(t,a)}{da} = -(\mu+\alpha(a))e(t,a), \\ e(t,0) = p\beta S I + f(I), \\ I(0) = I_0, e(0,a) = e_0(a) \end{cases}$$

Since $S(t) \leq S^0$ as $t \to +\infty$, we get $I(t) \leq \tilde{I}(t), e(t,a) \leq \tilde{e}(t,a)$, where

$$\begin{cases} \dfrac{d\tilde{I}(t)}{dt} = \displaystyle\int_0^\infty \alpha(a)\tilde{e}(t,a)da + (1-p)\beta S^0\tilde{I} - \gamma\tilde{I} - (d+\mu)\tilde{I}, \\ \dfrac{\partial\tilde{e}(t,a)}{dt} + \dfrac{\partial\tilde{e}(t,a)}{da} = -(\mu+\alpha(a))\tilde{e}(t,a), \\ \tilde{e}(t,0) = p\beta S^0\tilde{I} + \gamma\tilde{I}, \\ \tilde{I}(0) = \tilde{I}_0, \tilde{e}(0,a) = \tilde{e}_0(a) \end{cases} \tag{2.12}$$

By the system (2.4), we have

$$\tilde{e}(t,a) = \begin{cases} (p\beta S^0\tilde{I}(t-a) + \gamma\tilde{I}(t-a))K_0(a), & t > a \geq 0, \\ \tilde{e}_0(a-t)\dfrac{K_0(a)}{K_0(a-t)}, & a \geq t \geq 0, \end{cases} \tag{2.13}$$

where $K_0(a) = e^{-\int_0^a \varepsilon(\tau)d\tau}$. Substituting system (2.13) into system (2.12), we obtain

$$\begin{cases} \dfrac{d\tilde{I}(t)}{dt} = T_1 + T_2 + (1-p)\beta S^0\tilde{I} - \gamma\tilde{I} - (d+\mu)\tilde{I} \\ \tilde{I}(0) = 0 \end{cases} \tag{2.14}$$

where

$$\begin{aligned} T_1 &= \int_0^t \alpha(a)(p\beta S^0\tilde{I}(t-a) + \gamma\tilde{I}(t-a))K_0(a)da, \\ T_2 &= \int_t^{+\infty} \alpha(a)\tilde{e}_0(a-t)\tfrac{K_0(a)}{K_0(a-t)}da. \end{aligned}$$

It's simple to know that if $T_2 = 0$, we get that system (2.14) has an only solution $\tilde{I}(t) = 0$. From the system (2.13), we can get $\tilde{e}(t,0) = 0, t \to +\infty$. Through comparison, we get $(I(t), e(t,a)) \to (0,0)$ for $t \to +\infty$.

Since $E_0 = (S^0, 0, 0, 0_{L^1})$ is globally asymptotically stable in ∂M_0, next we only need to proof that $W^s(E_0) \bigcap M_0 = \Theta$, where $W^s(E_0) = \{z \in X_{0+}, \lim\limits_{t\to+\infty} U(t)z = E_0\}$. Assume there exists $z \in W^s(E_0) \bigcap M_0$,

it follows that there exists $t_0 > 0$ such that $I(t_0) + \int_0^\infty e(t_0, a)da > 0$. Using the method in [18], we have $e(t, a) > 0$ for $(t, a) \in [0, \infty) \times [0, \infty)$, $I(t) > 0$ for $t \geq 0$. By means of the method of Braueretal [19], the following function is defined

$$H(a) = \int_a^\infty \alpha(s) e^{-\int_a^s \varepsilon(\tau)d\tau} ds$$

for $\forall\ a > 0$, $H(a) \geq 0$, and $H(0) = \theta$. Furthermore, for $\forall a \geq 0, H'(a) = -\alpha(a) + \varepsilon(a)H(a)$. Let

$$\Phi(t) = I(t) + \int_0^\infty H(a)e(t, a)da$$

which satisfies

$$\frac{d\Phi(t)}{dt} = \frac{I(d+\mu)}{1+mI}\left(\frac{(\theta p + 1 - p)\beta S(1+mI) + (\theta - 1)\gamma}{(d+\mu)} - 1\right).$$

If $R_0 > 1$, there exists $t_0 > 0$ such that $\Phi(t) \geq \Phi(t_0)$ for all $t \geq t_0$. Since $\Phi(t_0) > 0$, this prevents that $(I(t), e(t, a)) \to (0, 0_{L^1}, 0_{L^1})$, which is contradiction with $S(t)$ converges to S^0 and $I(t)$ converges to 0, for $t \to \infty$. We get the semiflow $\{U(t)\}_{t\geq 0}$ is uniformly persistent with respect to the pair $(\partial M_0, M_0)$. Besides, there exists a compact subset $H_0 \subset M_0$ which is a global attractor for $\{U(t)\}_{t\geq 0}$ in M_0.

2.4. Global asymptotic stability

Theorem 2.4. *Assume $\alpha(a)$ is an bounded function, if $R_0 < 1$, the infection-free equilibrium E_0 is globally asymptotically stable. Note* $\bar{\alpha} = \lim\limits_{a\in[0,+\infty)} \sup \alpha(a)$.

Proof. Consider systems(2.1)–(2.4) we get

$$\begin{aligned}\int_0^\infty \alpha(a)e(t, a)da \leq &\int_0^t \alpha(a)[p\beta S(t-a)I(t-a) + \gamma I(t-a)]K_0(a)da \\ &+ \bar{\alpha} \parallel e \parallel_{L_1} e^{-\mu t}\end{aligned} \tag{2.15}$$

where $\parallel e \parallel_{L_1} = \int_0^{+\infty} e_0(a)da$. Take the limit of both sides of the inequality (Eq 2.15), since $S \leq \frac{\Lambda}{\mu}$ and $\lim\limits_{t\to+\infty} e^{-\mu t} = 0$, we get

$$\begin{aligned}\lim_{t\to+\infty} \sup \int_0^\infty \alpha(a)e(t, a)da \leq &\int_0^{+\infty} \alpha(a)p\beta\frac{\Lambda}{\mu}K_0(a)da \lim_{t\to+\infty} \sup I(t) \\ &+ \int_0^\infty \gamma\alpha(a)K_0(a)da \lim_{t\to+\infty} \sup I(t).\end{aligned} \tag{2.16}$$

With the help of Taylor's formula and the third equation of system (2.1), we get

$$\frac{dI(t)}{dt} \leq \int_0^\infty \alpha(a)e(t, a)da + (1-p)\beta I\frac{\Lambda}{\mu} - (\gamma + d + \mu)I. \tag{2.17}$$

By solving inequality (Eq 2.17) and taking the limit of both sides, we can get

$$\lim_{t\to+\infty} \sup I(t) \leq R_0 \lim_{t\to+\infty} \sup I(t)$$

Therefore, when $R_0 < 1, \lim\limits_{t\to+\infty} \sup I(t) = 0$. By using inequality (Eq 2.16) and system (2.1) respectively, we get $\lim\limits_{t\to+\infty} \sup e(t,a) = 0$ and $\lim\limits_{t\to+\infty} \sup S(t) = \frac{\Lambda}{\mu}$. Therefore, if $R_0 < 1$, the disease-free equilibrium point E_0 is global attraction. Further, with the help of Theorem 2.1, we have the disease-free equilibrium point is global asymptotically stable.

Theorem 2.5. *If $R_0 > 1$, the unique endemic equilibrium E_* is globally asymptotically stable.*

Proof. The Lyapunov functional $\mathbb{V}_*(t)$ is constructed

$$\mathbb{V}_*(t) = W_S + W_e + W_I, \tag{2.18}$$

where

$$W_S(t) = (1-p)S^* g(\frac{S}{S^*}), \quad W_e(t) = \int_0^\infty W(a)e^*(a)g(\frac{e(t,a)}{e^*(a)})da, \quad W_I(t) = I^* g(\frac{I}{I^*}).$$

for $W(a) = \int_a^{+\infty} \alpha(s)e^{-\int_a^s(\mu+\alpha(\tau))d\tau}$, $g(x) = -1 + x - \ln x$, $\frac{de^*(a)}{da} = -\varepsilon(a)e^*(a)$. Calculating the derivative of the $\mathbb{V}_*(t)$ along with the solutions of system (2.18), yields

$$\begin{aligned}
\frac{d\mathbb{V}_*(t)}{dt} =& (1-p)\mu S^*(2 - \frac{S}{S^*} - \frac{S^*}{S}) + (1-p)\beta S^* I^*(1 - \frac{S^*}{S} - \frac{SI}{S^*I^*} + \frac{I}{I^*}) \\
&- W(a)e^*(a)g(\frac{e(t,a)}{e^*(a)})|_\infty + \theta e^*(0)g(\frac{e(t,0)}{e^*(0)}) - \int_0^\infty \alpha(a)e^*(a)g(\frac{e(t,a)}{e^*(a)}) \\
&+ \int_0^\infty \alpha(a)e^*(a)(1 - \frac{I}{I^*} - \frac{I^*e(t,a)}{Ie^*(a)} + \frac{e(t,a)}{e^*(a)})da \\
&+ (1-p)\beta S^* I^*(1 - \frac{S^*}{S} + \frac{SI}{S^*I^*} - \frac{I}{I^*}) \\
=& (1-p)\mu S^*(2 - \frac{S}{S^*} - \frac{S^*}{S}) + (1-p)\beta S^* I^*(2 - \frac{S^*}{S} - \frac{S}{S^*}) - W(a)e^*(a)g(\frac{e(t,a)}{e^*(a)})|_\infty \\
&+ \int_0^\infty \alpha(a)e^*(a)(1 - \frac{I}{I^*} - \frac{I^*e(t,a)}{Ie^*(a)} + \frac{e(t,a)}{e^*(a)} + 1 - \frac{e(t,a)}{e^*(a)} + \ln\frac{e(t,a)}{e^*(a)})da \\
&+ \theta e^*(0)g(\frac{e(t,0)}{e^*(0)}) \\
=& (1-p)\mu S^*(2 - \frac{S}{S^*} - \frac{S^*}{S}) + (1-p)\beta S^* I^*(2 - \frac{S^*}{S} - \frac{S}{S^*}) - W(a)e^*(a)g(\frac{e(t,a)}{e^*(a)})|_\infty \\
&- \int_0^\infty \alpha(a)e^*(a)(g(\frac{I}{I^*}) + g(\frac{I^*e(t,a)}{Ie^*(a)})da + \theta e^*(0)g(\frac{e(t,0)}{e^*(0)})
\end{aligned}$$

if $\frac{I}{I^*} > 1$ and $\frac{S^*}{S} > 1$, or $0 < \frac{I}{I^*} < 1$ and $0 < \frac{S^*}{S} < 1$, we have

$$\theta e^*(0)g(\frac{e(t,0)}{e^*(0)}) = \int_0^\infty \alpha(a)e^*(a)g(\frac{e(t,0)}{e^*(0)}) \le \int_0^\infty \alpha(a)e^*(a)g(\frac{I}{I^*})da$$

Hence,

$$\begin{aligned}\frac{d\mathbb{V}_*(t)}{dt} ==&(1-p)\mu S^*(2-\frac{S}{S^*}-\frac{S^*}{S})+(1-p)\beta S^* I^*(2-\frac{S^*}{S}-\frac{S}{S^*})-W(a)e^*(a)g(\frac{e(t,a)}{e^*(a)})|_\infty\\ &-\int_0^\infty \alpha(a)e^*(a)(g(\frac{I}{I^*})+g(\frac{I^*e(t,a)}{Ie^*(a)})da+\int_0^\infty \alpha(a)e^*(a)g(\frac{I}{I^*})da\\ &=(1-p)\mu S^*(2-\frac{S}{S^*}-\frac{S^*}{S})+(1-p)\beta S^* I^*(2-\frac{S^*}{S}-\frac{S}{S^*})-W(a)e^*(a)g(\frac{e(t,a)}{e^*(a)})|_\infty\\ &-\int_0^\infty \alpha(a)e^*(a)g(\frac{I^*e(t,a)}{Ie^*(a)})da\end{aligned}$$

For $g(x)\geq 0, x\in R$, therefore $\frac{dV_*(t)}{dt}\leq 0$ is always true, furthermore the strict equality holds if and only if $S=S^*$, $e(t,a)=e^*(a)$, $I=I^*$. Therefore when $R_0>1$ the endemic equilibrium E_* is globally asymptotically stable.

3. Simulations and summary of results

In the work, an age structure epidemic SEI model with fast and slow progression is considered. The basic reproduction number R_0 is obtained as $R_0=\frac{p\beta\Lambda\theta+\gamma\mu\theta+(1-p)\beta\Lambda}{(\gamma+d+\mu)\mu}$. We have proved the globally asymptotically stable for disease-free and endemic equilibrium respectively. In the following, we also give some numerical simulations to illustrate the global stability. Let $\Lambda=1;\beta=0.055;\gamma=0.7;\mu=0.063;d=0.04;p=0.8;m=0.02;S(0)=30,e(0,a)=6e^{-0.4a},I(0)=10.$ and

$$\alpha(a)=\begin{cases}0.4, & a\geq\tau\\ 0, & \tau\geq a\geq 0\end{cases}.$$

In Figure 1, we choose $\tau=12$, then $R_0<1$, it can be seen that E_0 is globally asymptotically stable. While in Figure 2, we choose $\tau=1$, then $R_0>1$, it can be seen that E^* is globally asymptotically stable. The figures show the series of $S(t)$ and $I(t)$ which converge to their equilibrium values, in addition the age distribution of $e(t,a)$.

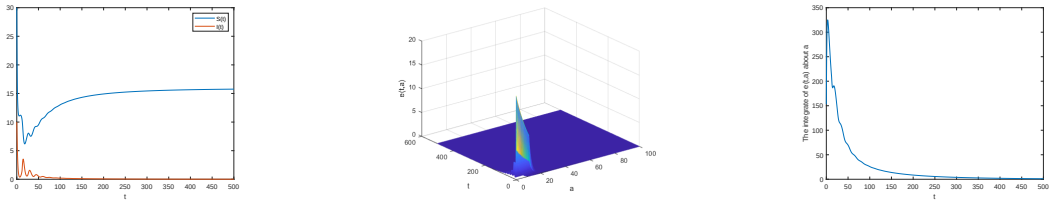

Figure 1. The time series of $S(t)$ and $I(t)$, and the age distributions of $e(t,a)$ when $\tau=12$.

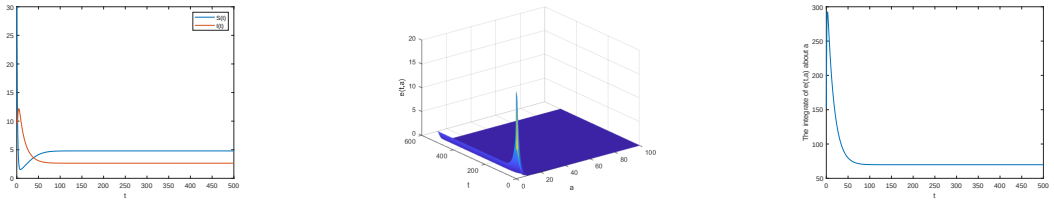

Figure 2. The time series of $S(t)$ and $I(t)$, and the age distributions of $e(t, a)$ when $\tau = 1$.

4. Discussion

COVID-19 has spread rapidly around the world since 2020 with a high fatality rate. Today the epidemic in some countries is still unable to be effectively controlled, and social and economic life has been greatly disrupted. COVID-19 trend prediction has become a major research focus. Current trend prediction methods include epidemic disease prediction model, COVID-19 trend prediction model based on deep learning, etc. These models have effectively assisted medical experts and scientific research institutions to efficiently predict COVID-19. The countermeasures and suggestions for strengthening epidemic prevention and control are put forward, which have a good guiding role for accurate epidemic prevention and control.

The large-scale epidemic of COVID-19 in China has basically ended, but there are still occasional imported cases or local outbreaks caused by cold chain pollution which prevention and control enters a new phase of normalization. Since the outbreak of COVID-19, a large number of researchers have conducted extensive studies on infectious disease dynamics and prevention and control measures through various models and data analysis methods. Many scholars have built traditional dynamics models based on warehouses to explore the development trend of COVID-19 and provide scientific basis for epidemic prevention and control.

5. Conclusions

According to transmission characteristics of infectious diseases, the paper proposed the methods of fast and slow transmission, which more effectively reveals the transmission mechanism for infectious diseases. The global asymptotic stability of the system has analyzed with the help of the principle of dynamics, and abtained the threshold of infectious disease control. The greater the rate of slow progression of an infectious disease, the fewer the threshold results. The world is now being vaccinated which cannot provide lifelong immunity, but can reduce the mortality of those infected. By vaccinating, the rate of patients entering slow progression increases and the threshold is correspondingly reduced.

Therefore, vaccination can effectively control the transmission of Coronavirus.

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Conflict of interest

The authors declare there is no conflict of interest.

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