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MODELLING AND STABILITY OF EPIDEMIC MODEL WITH FREE-LIVING PATHOGENS GROWING IN THE ENVIRONMENT

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Abstract To understand the impact of free-living pathogens (FLP) on the epidemics, an epidemic model with FLP is constructed. The global dynamics of our model are determined by the basic reproduction number R_0. If $R_0 < 1$, the disease free equilibrium is globally asymptotically stable, and if $R_0 > 1$, the endemic equilibrium is globally asymptotically stable. Some numerical simulations are also carried out to illustrate our analytical results.

Keywords Epidemics, free-living pathogen, basic reproduction number, global stability.

MSC(2010) 34K20, 34D23, 92D30.

1. Introduction

Epidemics are diseases caused by various pathogens which can spread between humans. Each infectious disease is caused by its specific pathogen, which can be a microbe or a parasite. Including viruses, bacteria, fungi or parasites, etc. Although the development of medical science has been able to prevent and control many infectious diseases, there are still some outbreaks or epidemics that danger people's health and lives. Some infectious diseases are highly contagious, leading to high disability rates and great harm, for example, there are 1.5 million deaths from respiratory infectious worldwide annually [6]. Therefore, it is of great practical significance to study the process of epidemics.

Following the pioneering of Kermack and Mckendrick [15], many researchers' attention have been attracted by epidemic models. In order to better understand the dynamics of infectious diseases, many different epidemic models have emerged and have been studied in different ways. For example, we can find susceptible-infectious-recovered (SIR) model (see for example [1, 16]), apart from this, there is also susceptible-infectious-susceptible (SIS) model [4, 8, 9, 21]. Some researchers added the exposed compartment E or the treatment T to the model, just like susceptible-exposed-infectious-recovered ($SEIR$) model in [31]; A. Ricardo. [22] proposed a susceptible-exposed-infectious-recovered-treatment ($SEIRT$) model and studied the effects of treatment and exposure on epidemics; in addition, there is a mixture of the above models, like [2, 13, 20, 28–30]. Furthermore, some authors have studied the virus model (see [26]) to study the virus infection dynamics. From the work of S.A. Boone, et al. [6] and A. Gabbuti, et al. [10], we know that vaccination

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against hepatitis B is the most effective measure to control the spread of hepatitis B due to prevention and management of viral disease heavily relies upon vaccines and antiviral medications. So, many researchers incorporated the new compartment, that is, the vaccination V to their model, such as [14,19].

However, vaccines and drugs are not absolutely effective, and there are still no vaccines or drugs for many common viruses [6]. So, avoiding exposure to the virus is more important to prevent infectious disease. Viral transmission is not only depend on interaction with the host, but also interaction with the environment. A contaminated environment such as air, water, food may transmit infection to susceptible hosts [3,7,23]. If we are contacting with infections who is sneezing or coughing, we will be very susceptible to the pathogens. The pathogens in a free-living can survive even growth in the environment [5,18]. Additionally, a free-living pathogen (FLP) in the environment will naturally die and be replenished by the infected people [3].

By taking into account the above mentioned factors, in this paper, we extend a simple $SIRT$ model to an $SIRTP$ model that include the FLP capable of growth and survival in the environment. The organization of this paper is as follows: In section 2, the model is derived under some assumptions. In section 3, we get the basic reproduction number and obtain the stability of all the equilibria. Some numerical simulations are given in section 4 to illustrate our analytical result. Some discussions are given in the last section.

2. The Model

2.1. System description

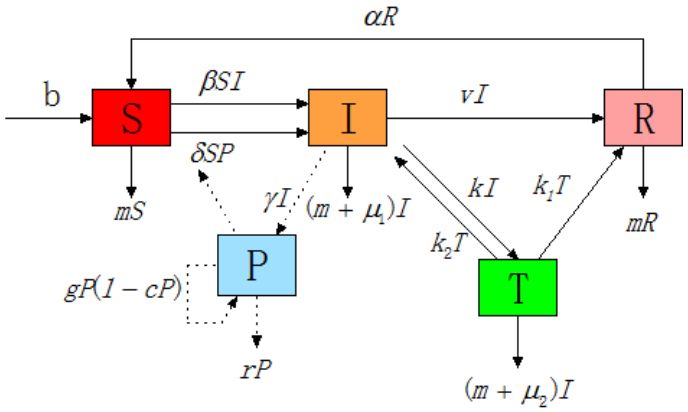


Figure 1. Transfer diagram for the dynamics of epidemic model with FLP.

In this section, a simple model with treatment and FLP is introduced. The total population $N(t)$ is divided into four compartments, namely, $S(t)$ represents the number of susceptible individuals. $I(t)$ represents the number of infectious individuals. Note that some people who are infected with pathogens are able to heal themselves. $R(t)$ represents the number of recovered individuals and the recovered individuals may be infected again. $T(t)$ represent the number of individuals being

treated. In our model, we assume that not all people can be cured completely for some of practical reasons. If the treatment is successful, individuals in compartment T enter into the compartment of R. If the treatment is terminated or failure, people who failed the treatment still carry and transmit the virus. And the compartment $P(t)$ indicates the FLP load in the environment, people who are susceptible to infection can become infected not only by adequate contacts with infectious people but also contacting pathogens in the environment. Hence, the total population is given by

$$N(t) = S(t) + I(t) + R(t) + T(t).$$

The transfer diagram of the model is shown in Figure 1 (in this figure, the dotted line and the solid line represent the dynamics of host and FLP, respectively). According to Figure 1, we have following model:

$$\begin{aligned}
\dot{S} &= b - \beta SI - \delta SP + \alpha R - mS,\\
\dot{I} &= \beta SI + \delta SP - (m + \mu_1 + \nu + k)I + k_2 T,\\
\dot{T} &= kI - (k_2 + k_1 + m + \mu_2)T,\\
\dot{R} &= \nu I - (\alpha + m)R + k_1 T,\\
\dot{P} &= \gamma I + gP(1 - cP) - rP.
\end{aligned} \tag{2.1}$$

We assume that α is non-negative and all other parameters in the model are positive constants. The parameters are described in Tabel 1. In this paper, we assume that the FLP population cannot maintain itself through growth in the environment (i.e. the FLP growth rate g is always less than the FLP decay rate r) [3].

Table 1. Description of parameters

Parameter	Description	Data estimated	Data sources
b	Host birth rate	Variable	Estemate
m	Host natural death rate	0.01 day^{-1}	Estimate
β	Host-to-Host transmission	Variable	Estemate
δ	Environment-to-Host transmission	Variable	Estemate
γ	Pathogen shedding rate	10 $cells\, day^{-1}\, individuals^{-1}$	[3]
r	Pathogen decay rate	0.8 day^{-1}	[3]
$1/c$	Carrying capacity of FLP	10^6 $cells$	[3]
g	Pathogen growth rate	0.3 day^{-1}	[3]
μ_1	The disease-related death of the infectious	0.03 day^{-1}	[3]
μ_2	The disease-related death of being treated	0.005 day^{-1}	Estemate
k	Progression rate to T from I	0.4 day^{-1}	Estemate
k_1	The proportion of successful treatment	Variable	Estemate
k_2	The proportion of failure treatment	Variable	Estemate
$1/v$	Infection period	3 day	[3]
$1/\alpha$	Immune period	Variable	Estemate

2.2. Basic properties

2.2.1. Positive of solutions

For system (2.1), In order to ensure the solutions of the system with positive initial conditions remain positive for all $t > 0$, it is necessary for us to prove that all the variables are non-negative. Therefore, we give the following lemma.

Lemma 2.1. *If $S(0) \geq 0$, $I(0) \geq 0$, $T(0) \geq 0$, $P(0) \geq 0$, $R(0) \geq 0$, the solutions $S(t)$, $I(t)$, $T(t)$, $P(t)$, $R(t)$, of system (2.1) are positive for all $t > 0$.*

Proof. Under the giving initial conditions, it is easy to prove that the solution of the system (2.1) are positive; if not, we assume a condition: that there exists a first time t_1 such that

$$S(t_1) = 0, S'(t_1) < 0, I(t) \geq 0, T(t) \geq 0, P(t) \geq 0, R(t) \geq 0, 0 \leq t \leq t_1, \tag{2.2}$$

there exists a t_2

$$I(t_2) = 0, I'(t_2) < 0, S(t) \geq 0, T(t) \geq 0, P(t) \geq 0, R(t) \geq 0, 0 \leq t \leq t_2, \tag{2.3}$$

there exists a t_3

$$T(t_3) = 0, T'(t_3) < 0, S(t) \geq 0, I(t) \geq 0, P(t) \geq 0, R(t) \geq 0, 0 \leq t \leq t_3, \tag{2.4}$$

there exists a t_4

$$R(t_4) = 0, R'(t_4) < 0, S(t) \geq 0, I(t) \geq 0, P(t) \geq 0, T(t) \geq 0, 0 \leq t \leq t_4, \tag{2.5}$$

there exists a t_5

$$P(t_5) = 0, P'(t_2) < 0, S(t) \geq 0, I(t) \geq 0, R(t) \geq 0, T(t) \geq 0, 0 \leq t \leq t_2. \tag{2.6}$$

In the (2.2) we have

$$S'(t_1) = b + \alpha R(t_1) > 0, \tag{2.7}$$

which is contradictory to the assumption $S'(t_1) < 0$, it means that $S'(t) > 0$, $t \geq 0$. In the second case, we have

$$I'(t_2) = \delta S(t_2) P(t_2) + k_2 T(t_2) \geq 0, \tag{2.8}$$

which is contradictory to the assumption $I'(t_2) < 0$, it means that $I'(t) > 0$, $t \geq 0$. Similarly, it can be shown that $T(t) \geq 0$, $R(t) \geq 0$, $P(t) \geq 0$ for all $t \geq 0$.

Hence, the solutions $S(t)$, $I(t)$, $T(t)$, $P(t)$, $R(t)$ of system (2.1) remain positive for all $t > 0$. □

2.2.2. Invariant regions

Lemma 2.2. *We assume that there is a constant H, then, all feasible solution of the system (2.1) are bounded and enter the region $\Omega = \{(S(t), I(t), T(t), R(t), P(t)) \in R_+^4 \mid 0 \leq S + I + T + R \leq \frac{b}{m}, P \leq H\}$.*

Proof. We assume that (S, I, T, R) is any solution with non-negative initial conditions, adding the first four equations of the system (2.1), we have

$$\begin{aligned} \frac{\mathrm{d}}{\mathrm{d}t}(S + I + T + R) &= b - mS - mI - \mu_1 I - mT - \mu_2 T - mR \\ &= b - m(S + I + T + R) - (\mu_1 I + \mu_2 T) \\ &\leq b - m(S + I + T + R) \\ &= b - mN(t), \end{aligned}$$

where

$$N(t) = S(t) + I(t) + T(t) + R(t).$$

It follows that

$$0 \leq N(t) \leq \frac{b}{m} + N(0)e^{-mt},$$

where $N(0)$ represents initial value of the total population. Thus $0 \leq N(t) \leq \frac{b}{m}$, as $t \to \infty$.

For the last equation of the system (2.1), we have

$$\begin{aligned}\frac{\mathrm{d}P}{\mathrm{d}t} &= \gamma I + gP(1-cP) - rP \\ &= \gamma I - (r-g)P - gcP^2 \\ &\leq \gamma\frac{b}{m} - (r-g)P - gcP^2,\end{aligned}$$

and thus there exists $H > 0$ so that $\limsup_{t\to\infty} P(t) \leq H$. The constant H can be chosen as the unique positive zero of the quadratic polynomial $\gamma\frac{b}{m} - (r-g)x - gcx^2$. Therefore all feasible solutions of system (2.1) enter the region

$$\Omega = \{(S(t), I(t), T(t), R(t), P(t)) \in R_+^4 \mid 0 \leq S + I + T + R \leq \frac{b}{m}, P \leq H\}.$$

□

So we consider dynamic of system (2.1) on the set Ω in this paper.

3. Analysis of the model

3.1. Disease free equilibrium and the reproduction number

It's easy for us to get the disease free equilibrium of the system (2.1)

$$E_0 = (\frac{b}{m}, 0, 0, 0, 0).$$

In the following, the basic reproduction number of system (2.1) will be derived by using the next generation matrix method formulated in [3, 12, 25, 27]. At first, we rearrange system (2.1) as following:

$$\begin{aligned}\dot{I} &= \beta SI + \delta SP - (m + \mu_1 + \nu + k)I + k_2 T, \\ \dot{T} &= kI - (k_2 + k_1 + m + \mu_2)T, \\ \dot{P} &= \gamma I + gP(1-cP) - rP, \\ \dot{S} &= b - \beta SI - \delta SP + \alpha R - mS, \\ \dot{R} &= \nu I - (\alpha + m)R + k_1 T.\end{aligned} \tag{3.1}$$

Then let $x = (I, T, P, S, R)^T$, thus the system (3.1) can be written as:

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \mathscr{F}(x) - \mathscr{V}(x),$$

where

$$\mathscr{F}(x) = \begin{pmatrix} \beta SI + \delta SP \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathscr{V}(x) = \begin{pmatrix} (m+\mu_1+\nu+k)I - k_2T \\ (k_2+k_1+m+\mu_2)T - kI \\ -\gamma I - gP(1-cP) + rP \\ mS - \alpha R + \delta SP + \beta SI - b \\ (\alpha+m)R - k_1T - \nu I \end{pmatrix}.$$

The Jacobian matrices of $\mathscr{F}(x)$ and $\mathscr{V}(x)$ at the disease free equilibrium E_0 are respectively,

$$D\mathscr{F}(E_0) = \begin{pmatrix} F_{3\times 3} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad D\mathscr{V}(E_0) = \begin{pmatrix} V_{3\times 3} & 0 & 0 \\ \beta\frac{b}{m} & 0 & \delta\frac{b}{m}\, m & -\alpha \\ -\nu & -k_1 & 0 & 0 & \alpha+m \end{pmatrix}.$$

Where,

$$F = \begin{pmatrix} \beta\frac{b}{m} & 0 & \delta\frac{b}{m} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} m+\mu_1+\nu+k & -k_2 & 0 \\ -k & k_2+k_1+m+\mu_2 & 0 \\ -\gamma & 0 & r-g \end{pmatrix}. \tag{3.2}$$

The model reproduction number, denoted by R_0 is thus given by

$$\begin{aligned} R_0 &= \rho(FV^{-1}) \\ &= \frac{\beta b(k_2+k_1+m+\mu_2)}{m[(m+\mu_1+\nu+k)(k_2+k_1+m+\mu_2) - kk_2]} \\ &\quad + \frac{\delta b\gamma(k_2+k_1+m+\mu_2)}{m(r-g)[(m+\mu_1+\nu+k)(k_2+k_1+m+\mu_2) - kk_2]}. \end{aligned}$$

Following Theorem 2 of [25], we have the following result on the local stability of E_0.

Theorem 3.1. *The disease free equilibrium E_0 is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.*

3.2. Global stability of E_0

Theorem 3.2. *If $R_0 < 1$, the disease free equilibrium E_0 of system (2.1) is globally asymptotically stable.*

Proof. The comparison theorem can be used to prove the global stability of the disease free equilibrium. The rate of change of the variables (I, T, P) of system (3.1) can be written as

$$\begin{pmatrix} \dot{I} \\ \dot{T} \\ \dot{P} \end{pmatrix} = (F-V)\begin{pmatrix} I \\ T \\ P \end{pmatrix} - (1-\frac{m}{b}S)\begin{pmatrix} \beta\frac{b}{m} & 0 & \delta\frac{b}{m} \\ 0 & 0 & 0 \\ 0 & 0 & \frac{cgP}{1-\frac{m}{b}S} \end{pmatrix}\begin{pmatrix} I \\ T \\ P \end{pmatrix},$$

where F and V are defined in (3.2).

Since $\gamma I + (g-r)P - gcP^2 \leq \gamma I + (g-r)P$ and $S \leq \frac{b}{m}$ for all $t \geq 0$ in Ω, then

$$\begin{pmatrix} \dot{I} \\ \dot{T} \\ \dot{P} \end{pmatrix} \leq (F-V) \begin{pmatrix} I \\ T \\ P \end{pmatrix}.$$

Note that both F and V^{-1} are non-negative. Following the Perron-Frobenius Theorem, let μ^T be a non-negative left eigenvector of $V^{-1}F$ with respect to $\rho(V^{-1}F) = \rho(FV^{-1}) = R_0$, which is, $\mu^T V^{-1}F = R_0\mu^T$. Motivated by [11], give a Lyapunov function

$$L = \mu^T V^{-1}(I, T, P).$$

The derivative of L is given by

$$\begin{aligned} L' &= \mu^T V^{-1}(\dot{I}, \dot{T}, \dot{P}) \\ &\leq \mu^T V^{-1}(F-V)(I, T, P) \\ &= (R_0 - 1)\mu^T(I, T, P). \end{aligned}$$

If $R_0 < 1$, then $L' = 0$ implies that $\mu^T(I, P, T) = 0$, and thus $I = 0$ or $T = 0$ or $P = 0$. It follows from the system (2.1) that the largest invariant set where $L' = 0$ satisfies $\delta SP + k_2 T = 0$ or $kI = 0$ or $\gamma I = 0$; Therefore, $I = T = P = 0$ because of $\delta \geq 0$, $k \geq 0$, $\gamma \geq 0$. The first and the fourth equations of the system (2.1) lead to $S = \frac{b}{m}$ and $R = 0$, respectively, in the above invariant set, which is the singleton $\{(\frac{b}{m}, 0, 0, 0, 0)\}$. By LaSalle's invariance principle [17], the disease free equilibrium E_0 of system (2.1) is globally asymptotically stable in Ω if $R_0 < 1$. □

3.3. Epidemic equilibrium

3.3.1. Existence of the endemic equilibrium

Theorem 3.3. *The system* (2.1) *admits a unique endemic equilibrium* $E^*(S^*, I^*, T^*, R^*, P^*)$ *if and only if* $R_0 > 1$.

Proof. The equilibrium equation of the system (2.1) are given by

$$\begin{aligned} 0 &= b - \beta S^* I^* - \delta S^* P^* + \alpha R^* - mS^*, \\ 0 &= \beta S^* I^* + \delta S^* P^* - (m + \mu_1 + \nu + k)I^* + k_2 T^*, \\ 0 &= kI^* - (k_2 + k_1 + m + \mu_2)T^*, \\ 0 &= \nu I^* - (\alpha + m)R^* + k_1 T^*, \\ 0 &= \gamma I^* + gP^*(1 - cP^*) - rP^*. \end{aligned} \tag{3.3}$$

It follows that the endemic equilibrium satisfies

$$I = \psi(P) := \frac{gc}{\gamma}P^2 + \frac{r-g}{\gamma}P,$$

$$P=\phi(I):=\frac{I}{\delta}\Bigg[\frac{m(m+\mu_1+\nu+k)}{b-(m+\mu_1+\nu+k)I+\frac{(\alpha+m)k_2k+\alpha\nu(k_2+k_1+m+\mu_2)+\alpha kk_1}{(k_2+k_1+m+\mu_2)(\alpha+m)}I} - \frac{mkk_2}{\left(\begin{array}{c} b(k_2+k_1+m+\mu_2)-(m+\mu_1+\nu+k)(k_2+k_1+m+\mu_2)I \\ +\frac{(\alpha+m)k_2k+(k_2+k_1+m+\mu_2)\alpha\nu+\alpha k_1k}{\alpha+m}I \end{array}\right)} - \beta\Bigg].$$

To see if there is a endemic equilibrium solution in the above formulas (3.3), we only need to judge whether the two lines determined by the functions $I=\psi(P)$ and $P=\phi(I)$ have intersections. Because of the P and I are all positive numbers, so we just consider the graphs in the first quadrant, by analyzing these two functions, we can get that the graph of the function $I=\psi(P)$ is a parabola, the function $P=\phi(I)$ has two vertical asymptotes, take $I_0=\frac{b(k_2+k_1+m+\mu_2)(\alpha+m)}{(m+\mu_1+\nu+k)(k_2+k_1+m+\mu_2)(\alpha+m)+kk_2(\alpha+m)+\alpha\nu(k_2+k_1+m+\mu_2)+\alpha kk_1}$ (Because of the range that $P>0$, another vertical asymptote is not considered here). Then, $\lim_{I\to I_0}\phi(I)=+\infty$, so the intersection exists if and only if $\phi'(0)<\frac{1}{\psi'(0)}$, see Figure 2 (This is a figure that reflects the positional relationship between the two functions, in this figure, the real curve and the dashed curve represent the function $I=\psi(P)$ and the function $P=\phi(I)$, respectively, while the curve perpendicular to the I axis represents the asymptote of the function $P=\phi(I)$. Through calculations, we get

$$\phi'(0)=\frac{1}{\delta}\Big(\frac{m(m+\mu_1+\nu+k)}{b}-\frac{mkk_2}{b(k_2+k_1+m+\mu_2)}-\beta\Big),$$

and $\psi'(0)=\frac{r-g}{\gamma}$. Then,

$$\frac{1}{\delta}\Big(\frac{m(m+\mu_1+\nu+k)}{b}-\frac{mkk_2}{b(k_2+k_1+m+\mu_2)}-\beta\Big)<\frac{\gamma}{r-g}.$$

Organize the above inequality to get

$$\frac{\beta b(k_2+k_1+m+\mu_2)}{m[(m+\mu_1+\nu+k)(k_2+k_1+m+\mu_2)-kk_2]} + \frac{\delta b\gamma(k_2+k_1+m+\mu_2)}{m(r-g)[(m+\mu_1+\nu+k)(k_2+k_1+m+\mu_2)-kk_2]}>1$$

□

Thus, the sufficient and necessary condition of a unique endemic equilibrium exists is $R_0>1$.

3.3.2. Global stability of the endemic equilibrium

Theorem 3.4. *Assume that $\alpha=0$, if $R_0>1$ the unique endemic equilibrium E^* of system (2.1) is globally asymptotically stable.*

Proof. If $R_0>1$, by Theorem 3.3, we know that there exist a unique endemic equilibrium $E^*(S^*,I^*,T^*,R^*,P^*)$, where S^*, I^*, T^* , R^*, P^* satisfy the equilibrium equations (3.3). Following [19, 24], we introduce a Lyapunov function V as follows

$$V=S-S^*\ln S+B(I-I^*\ln I)+D(T-T^*\ln T)+E(P-P^*\ln P).$$

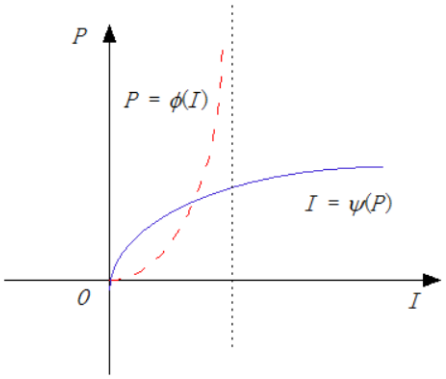


Figure 2. the graphs of functions $I = \psi(P)$ and $P = \phi(I)$.

Where B, D, E are positive constants to be determined later, the derivative of V is given by

$$\begin{aligned}
\dot{V} &= \dot{S}(1-\frac{S^*}{S}) + B\dot{I}(1-\frac{I^*}{I}) + D\dot{T}(1-\frac{T^*}{T}) + E\dot{P}(1-\frac{P^*}{P}) \\
&= (1-\frac{S^*}{S})(b-\beta SI-\delta SP+\alpha R-mS) \\
&\quad + B(1-\frac{I^*}{I})(\beta SI+\delta SP-(m+\mu_1+\nu+k)I+k_2T) \\
&\quad + D(1-\frac{T^*}{T})(kI-(k_2+k_1+m+\mu_2)T) + E(1-\frac{P^*}{P})(\gamma I+gP(1-cP)-rP) \\
&= (1-\frac{S^*}{S})(\beta S^*I^*+\delta S^*P^*+mS^*-\beta SI-\delta SP-mS) \\
&\quad + B(1-\frac{I^*}{I})(\beta SI+\delta SP-\frac{\beta S^*I^*+\delta S^*P^*+k_2T^*}{I^*}I+k_2T) \\
&\quad + D(1-\frac{T^*}{T})(kI-\frac{kI^*}{T^*}T) + E(1-\frac{P^*}{P})(\gamma I+(g-r)P-gcP^2).
\end{aligned}$$

By denoting $\frac{S}{S^*}=x$, $\frac{I}{I^*}=y$, $\frac{T}{T^*}=z$, $\frac{P}{P^*}=u$, we have

$$\begin{aligned}
\dot{V} =& (1-\frac{1}{x})(\beta S^*I^*+\delta S^*P^*+mS^*-\beta S^*I^*xy-\delta S^*P^*xu-mS^*x) \\
&+ B(1-\frac{1}{y})(\beta S^*I^*xy+\delta S^*P^*xu+k_2T^*z-\beta S^*I^*y-\delta S^*P^*y-k_2T^*y) \\
&+ D(1-\frac{1}{z})(kI^*y-kI^*z) + E(1-\frac{1}{u})(\gamma I^*y+(g-r)P^*u-gcP^{*2}u^2) \\
=& -mS^*\frac{(1-x)^2}{x} + \beta S^*I^*(1-xy-\frac{1}{x}+y) + \delta S^*P^*(1-xu-\frac{1}{x}+u) \\
&+ B\beta S^*I^*(1-\frac{1}{y})(xy-y) + B\delta S^*P^*(1-\frac{1}{y})(xu-y) + Bk_2T^*(1-y)(z-y) \\
&+ DkI^*(1-\frac{1}{z})(y-z) + E\gamma I^*(1-\frac{1}{u})(y-u) - EgcP^{*2}(1-u)^2 \\
=& -mS^*\frac{(1-x)^2}{x} - EgcP^{*2}(1-u)^2 + \beta S^*I^*(1-xy-\frac{1}{x}+y)
\end{aligned}$$

$$
\begin{aligned}
&+\delta S^*P^*(1-xu-\frac{1}{x}+u)+B\beta S^*I^*(xy-y-x+1)+B\delta S^*P^*(xu-y-\frac{xu}{y}+1)\\
&+Bk_2T^*(z-y-\frac{z}{y}+1)+DkI^*(y-z-\frac{y}{z}+1)+E\gamma I^*(y-u-\frac{y}{u}+1)\\
=&-mS^*\frac{(1-x)^2}{x}-EgcP^{*2}(1-u)^2\\
&+[\beta S^*I^*+\delta S^*P^*+B\beta S^*I^*+B\delta S^*P^*+Bk_2T^*+DkI^*+E\gamma I^*]\\
&+xy[B\beta S^*I^*-\beta S^*I^*]-\frac{1}{x}[\beta S^*I^*+\delta S^*P^*]\\
&+y[\beta S^*I^*-B\beta S^*I^*-B\delta S^*P^*-Bk_2T^*+DkI^*+E\gamma I^*]\\
&+xu[B\delta S^*P^*-\delta S^*P^*]+u[\delta S^*P^*-E\gamma I^*]+z(Bk_2T^*-DkI^*)\\
&-xB\beta S^*I^*-\frac{xu}{y}B\delta S^*P^*-\frac{z}{y}Bk_2T^*-\frac{y}{z}DkI^*-\frac{y}{u}E\gamma I^*. \qquad (3.4)
\end{aligned}
$$

The variable terms that appear in (3.4) with positive coefficients are xy, y, xu, and u. If all of the coefficients are positive, then there is a possibility that $\dot{V}$ could be positive. Making the coefficients of xy, y, xu, and u equal to zero, we get that

$$
\begin{aligned}
&B\beta S^*I^*-\beta S^*I^*=0,\\
&B\delta S^*P^*-\delta S^*P^*=0,\\
&\delta S^*P^*-E\gamma I^*=0,\\
&Bk_2T^*-DkI^*=0,\\
&\beta S^*I^*-B\beta S^*I^*-B\delta S^*P^*-Bk_2T^*+DkI^*+E\gamma I^*=0. \qquad (3.5)
\end{aligned}
$$

From (3.5), we obtain that

$$
B=1, E=\frac{\delta S^*P^*}{\gamma I^*}, D=\frac{k_2T^*}{kI^*}.
$$

Hence, we have

$$
\begin{aligned}
\dot{V}=&-mS^*\frac{(1-x)^2}{x}-EgcP^{*2}(1-u)^2+\beta S^*I^*(2-x-\frac{1}{x})\\
&+\delta S^*P^*(3-\frac{xu}{y}-\frac{y}{u}-\frac{1}{x})+k_2T^*(2-\frac{z}{y}-\frac{y}{z}).
\end{aligned}
$$

It's easy for us to see that $-mS^*\frac{(1-x)^2}{x}\le 0$ for $x>0$ and $-mS^*\frac{(1-x)^2}{x}=0$ if only if $x=1$; $-EgcP^{*2}(1-u)^2\le 0$ for $u>0$ and $-EgcP^{*2}(1-u)^2=0$ if and only if $u=1$. Since the arithmetical mean is greater than or equal to the geometrical mean, then $2-x-\frac{1}{x}\le 0$ for $x>0$ and $2-x-\frac{1}{x}=0$ if and only if $x=1$; $3-\frac{xu}{y}-\frac{y}{u}-\frac{1}{x}\le 0$ for $x\ge 0, y\ge 0, u\ge 0$ and $3-\frac{xu}{y}-\frac{y}{u}-\frac{1}{x}=0$ if and only if $y=1, x=u$; $2-\frac{z}{y}-\frac{y}{z}\le 0$ for $z\ge 0, y\ge 0$ and $2-\frac{z}{y}-\frac{y}{z}=0$ if and only if $z=y$. Therefore, $\dot{V}\le 0$ for $x,y,z,u\ge 0$ and $\dot{V}=0$ if and only if $x=1, y=1, x=u, z=y$. Substituting relations $S=S^*, I=I^*, T=T^*, P=P^*$ into the first equation of system (2.1) gets $0=b-\beta S^*I^*-\delta S^*P^*+\alpha R-mS^*$, it follows from the first equation of (3.3) that $R=R^*$. The maximum invariant set of system (2.1) on set $\{(x,y,z,u):\dot{V}=0\}$ is the singleton $(1,1,1,1)$. It means that, the largest invariant set where $\dot{V}=0$ is the singleton $\{(S^*,I^*,T^*,P^*,R^*)\}$. Thus, by Laselle's invariance principle [17], for system (2.1), the endemic equilibrium E^* is globally asymptotically stable if $\alpha=0$ and $R_0>1$. □

Remark 3.1. Theorem 3.4 tells us that when $\alpha = 0$ ($1/\alpha$ is immune period) and $R_0 > 1$, the endemic equilibrium E^* is globally asymptotically stable. Note that the global stability of the endemic equilibrium E^* is an open problem when $\alpha > 0$. But, when $\alpha \neq 0$, the same result is validated by numerical simulations (Figure 6).

4. Numerical simulation

In this section, to illustrate the analytic results obtained above, we have presented some simulations of system (2.1) using the parameter values in Table 1. The parameter values are mainly taken from [3].

We choose $b = 0.2, \beta = 2.5 \times 10^6, \delta = 1.07 \times 10^{-7}, k_1 = 0.05, k_2 = 0.01, \alpha = 0.01$, numerical simulation gives $R_0 < 1$, then the disease free equilibrium E_0 is global asymptotically stable (Figure 3). From Figure 3, we can clearly see that $I(t)$ declined sharply and get zero finally. It also illustrate our proof of the existence of E_0 is correct.

We choose $b = 12.05, \beta = 0.003, \delta = 0.0002, k_1 = 0.6, k_2 = 0.3, \alpha = 0$, numerical simulation gives $R_0 = 12.2 > 1$, then the endemic equilibrium E^* is global asymptotically stable (Figure 4). From Figure 4, we can clearly see as time going on, the number of people finally tends to a constant, however, we can't see some details clearly from Figure 4. Change the axis range, we can obtain the partial enlarged imagines as Figure 5, from Figure 5 we can see that four curves reach their equilibrium positions are not zero. It reveals that our proof of the existence of E^* is correct. Figure 6 shows when $\alpha \neq 0$, the E^* is also globally asymptotically stable.

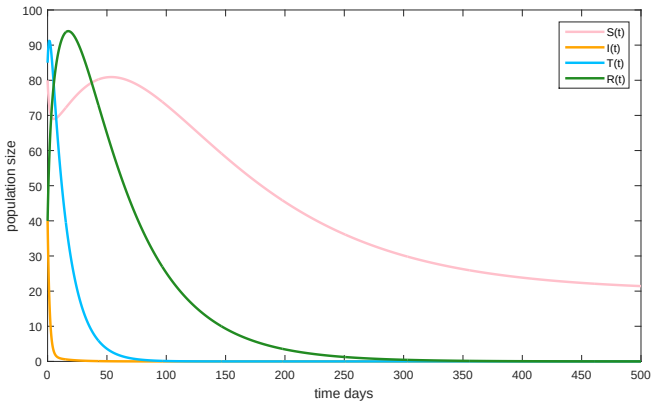


Figure 3. The disease free equilibrium E_0 is globally asymptotically stable.

5. Discussion

In this paper, we have formulated a novel epidemic model, which is incorporated a new compartment, that is, FLP capable of growth and survival in the environment. With the help of the next generation matrix method, we obtained the basic reproduction number R_0 which play a important role, and we derived the globally

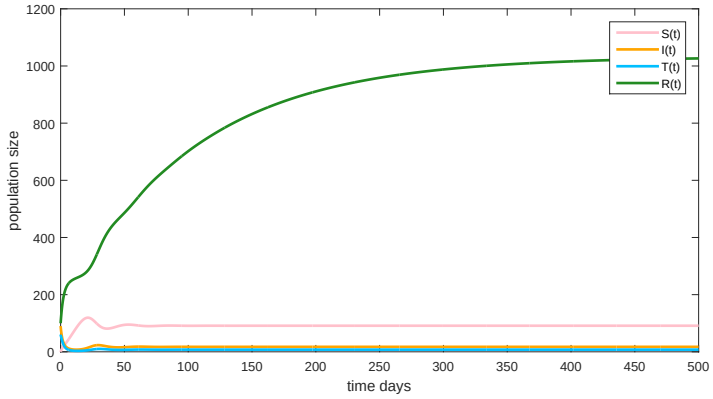


Figure 4. When $\alpha = 0$, the endemic equilibrium E^* is globally asymptotically stable.

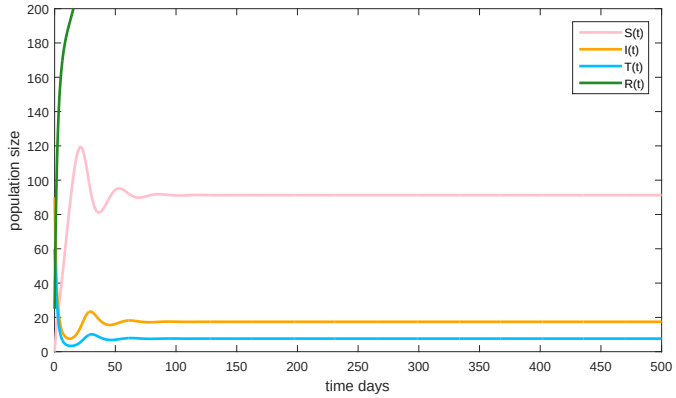


Figure 5. Local amplification of Figure 4

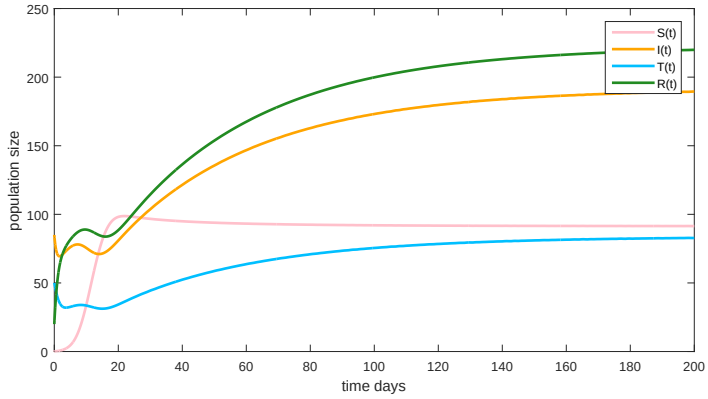
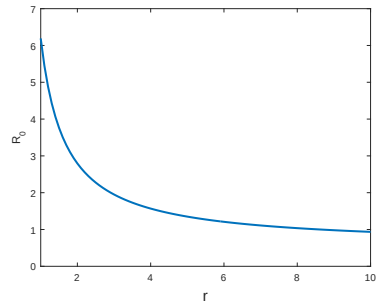
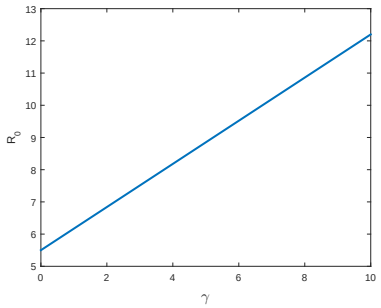


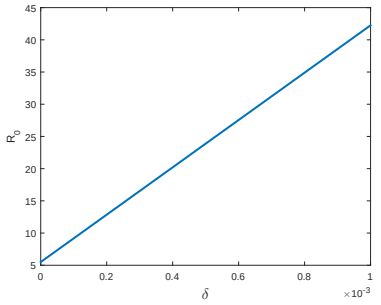
Figure 6. When $\alpha \neq 0$, the endemic equilibrium E^* is globally asymptotically stable.



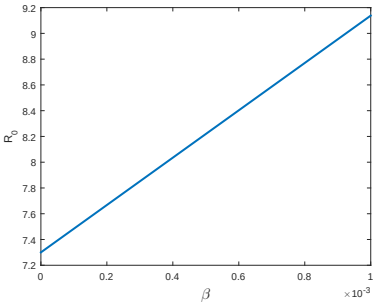
(a) The relationship among R_0 and r



(b) The relationship among R_0 and γ



(c) The relationship among R_0 and δ



(d) The relationship among R_0 and β

Figure 7. The relationship among R_0 and some parameter values

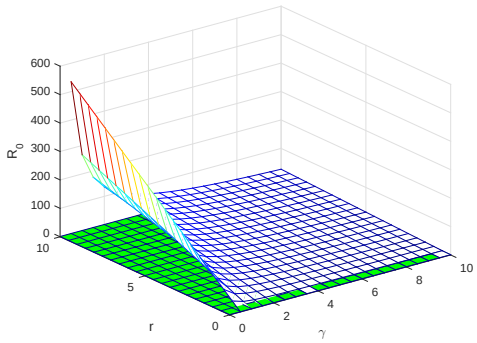


Figure 8. The relationship among R_0, r and γ

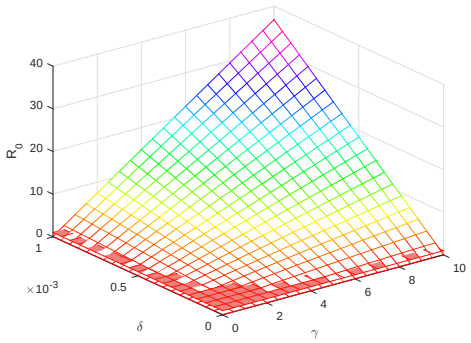


Figure 9. The relationship among R_0, δ and γ

dynamics of the model by constructing Lyapunov function. When the basic reproduction number R_0 is less than unity, the disease free equilibrium is globally asymptotically stable, that is, the disease will be extinct; When the basic reproduction number R_0 is greater than unity, the endemic equilibrium is globally asymptotically stable, which means that the disease will be permanent.

We all know that the basic reproduction number can be used to distinguish

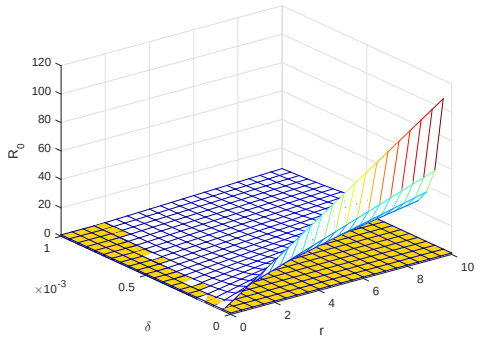


Figure 10. The relationship among R_0, δ and r

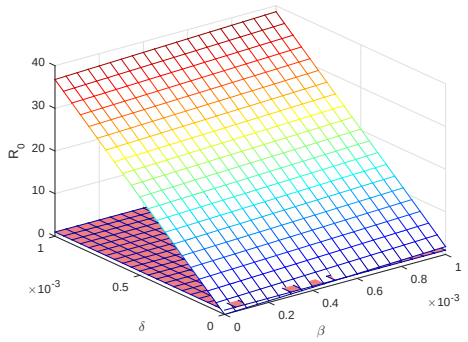


Figure 11. The relationship among R_0, β and δ

whether the disease disappears or not. For system (2.1), r reflects pathogens decay rate, γ reflects pathogens shedding rate while δ and β denote the environment-to-host transmission and the host-to-host transmission, respectively. In the event of an epidemic outbreaks, we can control the disease by reducing contact with pathogens, the measures may including:

(1) more frequent purification of the environment (i.e. increasing the value of r by washing our hands often or disinfecting and so on);

(2) enhance people's physical and antiviral ability by antibiotic treatments (i.e. the shedding rate γ will be decreased);

(3) reducing contacts with infectious host (i.e. the host-to-host transmission β is reduced);

(4) reducing contacts with contaminated environment (i.e. the environment-to-host transmission δ is reduced).

Figure 7 shows the change in the basic reproduction number R_0 due to changes in r, γ, δ, β. All other parameter values are as given in Table 1. Note that when r increase, infectious individuals will decrease, as stated in (1). Moreover, as shown in Figure 7(b)–7(d), R_0 grows with γ, δ, β, it means that if we want to control the disease, we should reduce the γ, δ and β, just like described in $(2)-(4)$. Comparing Figure 7(c) with Figure 7(d), we can see that for some infectious diseases, the environment-to-host transmission is even more serious than the infection between people. Figure 8 shows the relation among the basic reproduction number R_0, r, and γ. Figure 9 shows the relation among the basic reproduction number R_0, δ, and γ. Figure 10 shows the relation among the basic reproduction number R_0, δ, and r. Figure 11 shows the relation among the basic reproduction number R_0, β, and δ. From Figures 8–11, we can also see that if γ, δ and β are increase, then R_0 will increase, and if r increase, then R_0 will decrease.

Our results show that the role of environment-to-host disease transmission is becoming evident, it is necessary and meaningful to take the FLP into account. In addition, in this paper we discuss the global dynamic behaviors of system (2.1), we obtain that when the precondition are $\alpha = 0$ and the basic reproduction number $R_0 > 1$, the endemic equilibrium is globally asymptotically stable. Although when $\alpha \neq 0$, the same result is validated by numerical simulations (Figure 6), we didn't prove by mathematics that it is right. So we may need to conduct further research on this issue in the future.

References

[1] C. N. Angstmann, B. I. Henry and A. V. McGann, *A fractional order recovery SIR model from a stochastic process*, Bulletin of Mathematical Biology, 2016, 78(3), 468–499.

[2] Y. Bai and X. Mu, *Global asymptotic stability of a generalized SIRS epidemic model with transfer from infectious to susceptible*, Journal of Applied Analysis & Computation, 2018, 8(2), 402–412.

[3] M. Bani-Yaghoub, R. Gautam, Z. Shuai et al., *Reproduction numbers for infections with free-living pathogens growing in the environment*, Journal of Biological Dynamics, 2012, 6(2), 923–940.

[4] D. Bichara, Y. Kang, C. Castillo-Chavez et al., *SIS and SIR epidemic models under virtual dispersal*, Bulletin of Mathematical Biology, 2015, 77(11), 2004–2034.

[5] D. J. Bolton, C. M. Byrne, J. J. Sheridan et al., *The survival characteristics of a non-toxigenic strain of escherichia coli O157:H7*, Journal of Applied Microbiology, 1999, 86(3), 407–411.

[6] S. A. Boone and C. P. Gerba, *Significance of fomites in the spread of respiratory and enteric viral disease*, Applied & Environmental Microbiology, 2007, 73(6), 1687–1696.

[7] T. Caraco and I. Wang, *Free-living pathogens: Life-history constraints and strain competition*, Journal of Theoretical Biology, 2008, 250(3), 569–579.

[8] H. A. A. El-Saka, *The fractional-order SIS epidemic model with variable population size*, Journal of the Egyptian Mathematical Society, 2014, 22(1), 50–54.

[9] K. Fushimi, Y. Enatsu and E. Ishiwata, *Global stability of an SIS epidemic model with delays*, Mathematical Methods in the Applied Sciences, 2018, 41(13), 5345–5354.

[10] A. Gabbuti, L. Romanò, P. Blanc et al., *Long-term immunogenicity of hepatitis B vaccination in a cohort of italian healthy adolescents*, Vaccine, 2007, 25(16), 3129–3132.

[11] H. Guo, M. Li and Z. Shuai, *Global stability of the endemic equilibrium of multigroup SIR epidemic models*, Canadian Applied Mathematics Quarterly, 2006, 14(3), 259–284.

[12] H. Huo, F. Cui and H. Xiang, *Dynamics of an SAITS alcoholism model on unweighted and weighted networks*, Physica A: Statistical Mechanics & Its Applications, 2018, 496, 249–262.

[13] H. Huo, P. Yang and H. Xiang, *Dynamics for an SIRS epidemic model with infection age and relapse on a scale-free network*, Journal of the Franklin Institute, 2019.

[14] H. F. Huo and L. X. Feng, *Global stability of an epidemic model with incomplete treatment and vaccination*, Discrete Dynamics in Nature and Society, 2012, 2012(1), 87–88.

[15] W. O. Kermack and A. G. McKendrick, *A contribution to the mathematical theory of epidemics*, Proceedings of The Royal Society of London. Series A. Mathematical, Physical and Engineering Sciences, 1927, 115, 700–721.

[16] T. Kuniya and J. Wang, *Global dynamics of an SIR epidemic model with nonlocal diffusion*, Nonlinear Analysis Real World Applications, 2018, 43, 262–282.

[17] J. P. Lasalle, *The stability of dynamical systems*, Regional Conference Series in Applied Mathematics, SIAM, Philadelphia, 1976. DOI: 10.1007/s13398-014-0173-7.2.

[18] J. T. Lejeune, T. E. Besser and D. D. Hancock, *Cattle water troughs as reservoirs of escherichia coli O157*, Applied and Environmental Microbiology, 2001, 67(7), 3053–3057.

[19] J. Q. Li, Y. L. Yang and Y. C. Zhou, *Global stability of an epidemic model with latent stage and vaccination*, Nonlinear Analysis: Real World Applications, 2011, 12(4), 2163–2173.

[20] S. Liu, L. Zhang and Y. Xing, *Dynamics of a stochastic heroin epidemic model*, Journal of Computational and Applied Mathematics, 2019, 351, 260–269.

[21] S. Liu, L. Zhang, X. Zhang and A. Li, *Dynamics of a stochastic heroin epidemic model with bilinear incidence and varying population size*, International Journal of Biomathematics, 2019, 12(1), 1950005(21 pages).

[22] A. Ricardo, *Analysis of a fractional SEIR model with treatment*, Applied Mathematics Letters, 2018, 84, 56–62.

[23] E. Scott and S. Bloomfield, *The survival and transfer of microbial contamination via cloths, hands and utensils*, Journal of Applied Bacteriology, 1990, 68(3), 271–278.

[24] J. Tien and D. Earn, *Multiple transmission pathways and disease dynamics in a waterborne pathogen model*, Bulletin of Mathematical Biology, 2010, 72(6), 1506–1533.

[25] P. van den Driessche and J. Watmough, *Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission*, Mathematical Biosciences, 2002, 180(1), 29–48.

[26] H. Xiang, Y. L. Tang and H. F. Huo, *A viral model with intracellular delay and humoral immunity*, Bulletin of the Malaysian Mathematical Sciences Society, 2017, 40(3), 1011–1023.

[27] H. Xiang, Y. Wang and H. Huo, *Analysis of the binge drinking models with demographics and nonlinear infectivity on networks*, Journal of Applied Analysis & Computation, 2018, 8(5), 1535–1554.

[28] H. Xiang, M. Zou and H. Huo, *Modeling the effects of health education and early therapy on tuberculosis transmission dynamics*, International Journal of Nonlinear Sciences & Numerical Simulation, 2019, 20, 243–255.

[29] L. Zhang, S. Liu and X. Zhang, *Aaymptonic behavior of a stochastic virus dynamics model with intracellular delay and humoral immunity*, Journal of Applied Analysis and Computation, 2019, 9(4), 1425–1442.

[30] L. Zhang and Y. Xing, *Extremal solutions for nonlinear first-order impulsive integro-differential dynamic equations*, Mathematical Notes, 2019, 105(1), 124–132.

[31] S. X. Zhang and H. Guo, *Global analysis of age-structured multi-stage epidemic models for infectious diseases*, Applied Mathematics and Computation, 2018, 337, 214–233.