In order to study the transmission of rabies epidemics in vampire bats, we propose a mathematical model for vampire bat rabies virus. A threshold $R_0$ is identified which determines the outcome of the disease. If $R_0 < 1$, the disease-free equilibrium is globally asymptotically stable, and if $R_0 > 1$, the endemic equilibrium is globally asymptotically stable with certain conditions. Through the numerical simulation, the correctness of the theoretical results is verified. We carry out the sensitivity analysis of the parameters which provide a theoretical basis for preventing and controlling the transmission of bat rabies.

1. Introduction

Rabies is a zoonotic disease caused by acute infectious viruses. Most rabies models have focused on disease dynamics and control within terrestrial mammals (e.g., dogs and foxes). Hou et al. [1] studied the prevalence and control of canine rabies in Guangdong Province, China. Asamoah et al. [2] studied an optimal control model of rabies transmission dynamics in dogs and the best way of reducing the death rate of rabies in humans. In fact, vampire bats are a host of zoonotic and potential zoonotic pathogens [3] and are also effective vectors for rabies. Most vampire bats transmit the virus through bites via infected saliva, but when the population density is high, they also spread the virus through aerosols. The habits of vampire bats are particularly exceptional. In the case of Mexican vampire bats, they mainly feed on the blood of animals. The unique tooth structure provides conditions for them to open the skin, absorb blood, and diffuse the rabies virus. The latent period of bat rabies is about 21–209 days. The longer the latent period, the more likely infected bats will survive long enough to enter hibernation and provide infectious contacts in the transmission season [4]. The target of bat rabies transmission is mainly large-scale warm-blooded animals such as cattle and sheep [5].

VBRV has been spreading for more than 100 years [5]. Estimating from Brazil alone suggested that across Latin America at least thousands of cattle were infected with rabies each year, which became a major burden for the livestock industry [6]. Furthermore, human deaths related to vampire bats had been on the rise, with the earliest description dating back to the Spanish conquest of the Americas in the 16th century [7]. Chile reported the first case of an insect-eating bat carrying rabies virus infecting a human [8]. Martinez-Burnes et al. [9] experimentally confirmed the presence of rabies virus in vampire bats. Therefore, VBRV attracted close attention and in-depth study by researchers. They had different concerns about vampire bats and conducted different research. Obregón-morales et al. [10] demonstrated that rousette bats exposed to rabies had an innate low susceptibility to bat rabies. Based on five-year observations of large brown bats, George et al. [4] established a bat rabies seasonal transmission model and analyzed key parameters that determine the seasonality and maintenance of bat rabies. Blackwood et al. [11] used longitudinal study data from vampire bats to proposed four bat rabies models and developed a parameterized and evaluated stochastic performance model to assess the determinants of virus persistence in vampire bats. Aguilar-setien et al. [12] studied the saliva excretion of rabies...
virus by vampire bats and indicated that bats that were infected with rabies virus and survived will expel the virus through saliva in the early stage of infection. Kobayashi et al. [13] confirmed that there is a considerable diversity in bat rabies variants through sequence analysis of the Brazilian bat rabies virus, which provided conditions for the epidemic of bat rabies. Bat population was monitored for rabies by fluorescent antibody testing and simplified fluorescent inhibition microtesting by Almeida et al. [14], who indicated an increase in rabies virus circulation among bats and risk of a rabies outbreak. Streicker et al. [15] highlighted the importance of international viral dispersal for shaping the burden of rabies in Costa Rica and suggested a Central American corridor of rabies virus invasions between continents. Although researchers have carried out a lot of studies on bat rabies from different aspects, there are relatively few studies on relevant mathematical analysis from the perspective of the dynamic model. The transmission mechanism of bat rabies cannot be analyzed by sensitivity analysis. In this paper, we will use mathematical derivation to study transmission in vampire bats: here, we formulate the dynamics model of rabies epidemic in vampire bats. The next five sections are devoted to studying the stability of the established model. In Section 3, we derive the threshold \( R_0 \). It is shown that if \( R_0 < 1 \) then the disease-free equilibrium is globally asymptotically stable. In Sections 4–7, the model has a unique endemic equilibrium, which asymptotically attracts all nontrivial solutions under certain conditions. These theoretical results are supported with numerical simulations. Sensitivity analysis of \( R_0 \) on various parameters is carried out at the end of Section 8. The paper concludes with a brief conclusion.

### 2. Model Formulation

According to the transmission mechanism of rabies virus in bat population and the immunity of bats to rabies virus, the transfer diagram of the model is given by Figure 1.

Therefore, we formulate the dynamics model of rabies transmission in vampire bats:

\[
\begin{align*}
\frac{dS}{dt} &= bN + eV - \frac{\beta SI}{N} - \mu S, \\
\frac{dE}{dt} &= \frac{\beta SI}{N} - \mu E - \tau_1 E, \\
\frac{dI}{dt} &= \alpha \tau_1 E - \tau_2 I - \mu I - \delta I, \\
\frac{dV}{dt} &= (1 - a)\tau_1 E + \tau_2 I - \mu V - eV.
\end{align*}
\]

\( \text{Figure 1: The transfer diagram of the SEIVS model.} \)

A population size \( N(t) \) is partitioned into subclasses of individuals who are susceptible, exposed (infected but not yet infectious), infectious, and immune, with sizes denoted by \( S(t) \), \( E(t) \), \( I(t) \), and \( V(t) \), where \( N(t) = S(t) + E(t) + I(t) + V(t) \). The meanings of related parameters in (1) and their values are shown in the Table 1 (time scale is week).

Adding the four equations of system (1),

\[
\frac{dN}{dt} = bN - \mu N - \delta I.
\]

### 3. Threshold and Stability of Disease-Free Equilibrium

Let \( s = (S/N), e = (E/N), i = (I/N), \) and \( v = (V/N) \) denote the fractions of the classes \( S, E, I, \) and \( V \) in the population, respectively. It is easy to verify that \( s, e, i, \) and \( v \) satisfy the system of differential equations:

\[
\begin{align*}
\frac{ds}{dt} &= b + eV - \beta si - bs + \delta si, \\
\frac{de}{dt} &= \beta si - \tau_1 e - be + \delta ei, \\
\frac{di}{dt} &= \alpha \tau_1 e - \tau_2 i - \delta i - bi + \delta i^2, \\
\frac{dv}{dt} &= (1 - a)\tau_1 e + \tau_2 i - ev - bv + \delta vi,
\end{align*}
\]

and obviously \( s + e + i + v = 1 \). Note that the total population size \( N(t) \) does not appear in (3). Also, observe that the variable \( v \) does not appear in the first three equations of (3). This allows us to attack (3) by studying the system:

\[
\begin{align*}
\frac{ds}{dt} &= b + e(1 - s - e - i) - \beta si - bs + \delta si, \\
\frac{de}{dt} &= \beta si - \tau_1 e - be + \delta ei, \\
\frac{di}{dt} &= \alpha \tau_1 e - \tau_2 i - \delta i - bi + \delta i^2.
\end{align*}
\]

From biological considerations, we study (4) in the closed set:

\[
\Gamma = \{ (s, e, i) \in \mathbb{R}_+^3 \mid 0 \leq s, e, i \leq 1, 0 \leq s + e + i \leq 1 \}.
\]
Lemma 1. Let $\Lambda = \{(e, i) \in \mathbb{R}^2_+ \mid 0 \leq e + i \leq 1\}$ and $h(e, i) = (a_1 - b_1) e + (c_1 - b_1) i + b_1$. Therefore, for any positive constants $a_1, b_1,$ and $c_1$, $\max_{(e, i) \in \Lambda} h(e, i) = \max \{a_1, b_1, c_1\}$. Theorem 1. The disease-free equilibrium $E_0 = (1, 0, 0)$ of (4) is globally asymptotically stable in $\Gamma$ if $R_0 < 1$.

Proof. Let $L = a_1 e + (b + \tau_1) i$. Then,

$$L' = a_1 b i + a_1 i e - (b + \tau_1)(b + \tau_2 + \delta) i + (b + \tau_1) i \delta^2$$

$$\leq a_1 b (1 - e - i) i + a_1 i e - (b + \tau_1)(b + \tau_2 + \delta) i + (b + \tau_1) i \delta^2$$

$$= h(e, i) - (b + \tau_1)(b + \tau_2 + \delta),$$

where

$$h(e, i) = a_1 b (1 - e - i) + a_1 i e + (b + \tau_1)$$

$$= (a_1 \delta - a_1 \beta)e + [(b + \tau_1) \delta - a_1 \beta] i + a_1 \beta.$$  

Applying Lemma 1 to $h(e, i)$ leads to

$$L' = \max \{0, \beta \} \leq 0, \text{ if } R_0 < 1.$$  

4. Existence and Uniqueness of Endemic Equilibrium $E^*$

From Theorem 1, system (4) admits a disease-free equilibrium $E_0 = (1, 0, 0)$ if $R_0 < 1$ and $E_0$ is unstable when $R_0 > 1$. Furthermore, we remark that $R_0 > 1$ implies $\beta > \delta$. If $R_0 > 1$, system (4) has a unique endemic equilibrium $E^* (s^*, e^*, i^*)$ and satisfies

$$b + e (1 - s^* - e^* - i^*) - \beta s^* i^* - \delta s^* e^* = 0,$$

$$\beta s^* i^* - (b + \tau_1) e^* + \delta e^* e^* = 0,$$

and thus $s^* > 0, e^* > 0, \text{ and } i^* > 0$. Adding the above-mentioned equations leads to

$$(b + e - \delta^2 S^* + (1 - s^* - e^* - i^*) = \tau_2 i^* + (1 - \alpha) r_1 e^*,$$

which gives the following range of $i^*$:

$$0 < i^* < \min \left\{ \frac{b + e}{\delta} \right\}.$$  

According to the first and third equations of system (12), we have

$$e^* = \frac{(b + \tau_2 + \delta - \delta i^*) i^*}{\alpha s_1},$$

$$s^* = \frac{b + e (1 - i^*) - \delta e^*}{b + e + (\beta - \delta) i^*}.$$  

Using (15), we eliminate $s^*$ and $e^*$ in the second equation of (12) and derive that $i^*$ satisfies

$$L' = \max \{0, \beta \} \leq 0, \text{ if } R_0 < 1.$$
where
\[ a_0 = (\delta - \beta)\delta^2 < 0, \]
\[ a_1 = \delta [\beta \epsilon - (b + \epsilon)\delta + (\beta - \delta)(\tau_1 + \tau_2 + \delta + 2b)], \]
\[ a_2 = \alpha \tau_1 \beta \left( \frac{\delta - \beta}{R_0} - \epsilon \right) + (b + \epsilon)(\tau_1 + \tau_2 + \delta + 2b)\delta - (b + \tau_2 + \delta)\beta \epsilon, \]
\[ a_3 = \alpha \tau_1 \beta (b + \epsilon) \left( 1 - \frac{1}{R_0} \right) > 0. \]

Therefore, \( h(0) = a_1 > 0 \) and \( h(\infty) = -\infty < 0 \). There is at least one positive equilibrium in the interval \([0, \infty)\) for Intermediate Value Theorem.

We see that \( i^* \) satisfies
\[ f(i^*) = g(i^*), \]
where
\[ f(i^*) = \left(1 - \frac{\delta}{\tau_1}\right) \left(1 - \frac{\delta}{b + \tau_2 + \delta}\right) \left(1 + \frac{\beta - \delta}{b + \epsilon}\right), \]
\[ g(i^*) = R_0 + \frac{\epsilon \beta \delta}{(b + \epsilon)(b + \tau_1)(b + \tau_2 + \delta)} i^2 - \frac{\epsilon \beta (\alpha \tau_1 + \tau_2 + \delta + b)}{(b + \epsilon)(b + \tau_1)(b + \tau_2 + \delta)} i^*. \]

When \( R_0 > 1 \), assume that \( b > \delta \), discussing the number of intersections of \( f(i^*) \) and \( g(i^*) \) at \([0, 1]\). Obviously, \( g(0) = R_0 > 1 = f(0) \), and the symmetry axis of \( g(i^*) \) is greater than 1. Suppose that \( f(1) < g(1) \), that is,
\[ \frac{(b + \tau_2)(\beta \epsilon + \delta^2 + \tau_1^2 + \tau_1 b)}{\alpha \tau_1 \beta b + (b + \tau_2) \beta \delta} > 1. \]

Then, \( f(i^*) \) and \( g(i^*) \) have only one intersection in \([0, 1]\). Therefore, we see that (4) has a unique endemic equilibrium of \( E^* \) at \([0, 1]\) (see Figure 2).

5. Local Asymptotic Stability of the Endemic Equilibrium \( E^* \)

To show the asymptotic stability of the endemic equilibrium \( E^* \), we use Routh–Hurvitz conditions to proof it.

**Theorem 2.** If \( R_0 > 1, b > \delta, \) and \( ((\tau_2 + b)(\beta \epsilon + \delta^2 + \tau_1^2 + \tau_1 b)/\alpha \tau_1 \beta b + (\tau_2 + b) \beta \delta) > 1, \) then (4) has a unique endemic equilibrium \( E^* \) in \( \Gamma \) and \( E^* \) is locally asymptotically stable.

**Proof.** The Jacobian matrix of (4) at the endemic equilibrium \( E^* = (s^*, e^*, i^*) \in \Gamma \) is
\[ h(i^*) = a_0 i^3 + a_1 i^2 + a_2 i + a_3 = 0, \]

Figure 2: The existence and uniqueness of \( i^* \) in the interval \([0, 1]\).

\[ I = \begin{pmatrix} -b - \epsilon - \beta i^* + \delta i \epsilon & -\epsilon & -\epsilon - \beta \delta i^* + \delta i \epsilon \\ \beta i^* & -\tau_1 - b + \delta i & \beta i^* + \delta i \\ 0 & \alpha \tau_1 & -\tau_2 - \delta - b + 2 \delta i \end{pmatrix}. \]

6. Uniform Persistence

To prove the global stability of \( E^* \), in this section, we investigate the uniform persistence of (4) with the uniform persistence theory of Hale and Waltman [24].

**Definition 1.** Let \( X \) be a complete metric space. Suppose that \( X^0 \) is an open set in \( X, \partial X^0 \subset X, X^0 \cap \partial X^0 = \emptyset. \) Assume that \( T(t) \) is a \( C_0 \)-semigroup of \( X \) satisfying
\[ T(t): X^0 \rightarrow X^0, \]
\[ T(t): \partial X^0 \rightarrow \partial X^0. \]

Let \( T\beta(t) = T(t)|_{\partial X^0}, \) and let \( A_0 \) be the global attractor for \( T\beta(t). \)

**Definition 2.** The semigroup \( T(t) \) is said to be point dissipative in \( X \) if there is a bound nonempty set \( B \) in \( X \) such that, for any \( x \in X, \) there is \( t_0 = t_0(x, B) \) such that \( T(t)x \in B \) for \( t \geq t_0. \)
Definition 3. A nonempty invariant subset $M$ of $X$ is called an isolated invariant set if it is the maximal invariant set of a neighborhood of itself.

The orbit $\gamma^+(x)$ through $x$ is defined as $\gamma^+ = \bigcup_{t\geq 0}[T(t)x]$.

The $\omega$-limit set is defined as
\[
\omega(x) = \bigcap_{t \geq 0} C \cup \{T(t)x\},
\]
where $C$ is a closure.

The stable set of a compact invariant set $A$ is denoted by $W^s$ and is defined as
\[
W^s(A) = \{x \mid x \in X, \omega(x) \neq \emptyset, \omega(x) \subset A\}.
\]

The particular invariant set of interest is $A_0 = \bigcup_{x \in A_0} \omega(x)$.

Definition 4. $A_0$ is isolated if there exists a covering $M_k$ of $A_0$ by pairwise disjoint, compact, and isolated invariant sets $M_1, M_2, \ldots, M_n$ for $T_0$ such that each $M_i$ is also an isolated invariant set for $T$. $M$ is called an isolated covering.

Definition 5. $A_0$ will be called acyclic if there exists some isolated covering $M = \bigcup_{i=1}^n M_i$ of $A_0$ such that no subset of $M_i$'s forms a cycle.

Lemma 2. Suppose that $T(t)$ satisfies (4) and that the following conditions are valid:

(i) There is $t_0 > 0$ such that $T(t)$ is compact for $t > t_0$.

(ii) $T(t)$ is point dissipative in $\mathcal{X}$.

(iii) $\omega(x)$ is isolated and has an acyclic covering $M$, where $M = \bigcup_{i=1}^n M_i$.

(iv) $W^s(M_i) \cap X^0 = \emptyset, i = 1, 2, \ldots, n$.

Then, $T(t)$ is uniformly persistent.

Theorem 3. If $R_0 > 1$, system (4) is uniformly persistent.

Proof. Let $C([-\tau, 0], \mathbb{R}^3)$ be a Banach space which consists of $[-\tau, 0]$ to $\mathbb{R}^3$ continuous functions and let $\mathcal{C} = \{(\varphi_1, \varphi_2, \varphi_3) \in [-\tau, 0], \mathbb{R}^3], \varphi_1(0) \neq 0, \varphi_2(0) = \varphi_3(0) = 0, \theta \in [-\tau, 0])\}$. We assume $X^0 = C([-\tau, 0], \mathbb{R}^3), \partial X^0 = \mathcal{C}$. Then, (4) has a constant solution, such that $E \subset C$, where $E = \{(\varphi_1, \varphi_2, \varphi_3) \in [-\tau, 0], \mathbb{R}^3], \varphi_1(0) = (b + \sigma)(1 - s), \varphi_2(0) = \varphi_3(0) = 0, \theta \in [-\tau, 0])\}.

Define $(b + \sigma)(1 - s) = \tau_0$. Using Definition 1, (4) satisfies conditions (i) and (ii) of Lemma 2. Furthermore, $X^0$ and $\partial X^0$ are invariant sets; then, (24) is also established.

From the second equation of system (4), we know that $e' > \beta(s_0 - \epsilon)i(t) + \beta s(t)i(t) - (\tau_1 + b)f(t)$.

Thus, we can construct the following auxiliary system:
\[
\begin{align*}
\dot{m}_1(t) &= \beta(s_0 - \epsilon)i(t) + \beta m_1(t) - (\tau_1 + b)m_1(t), \\
\dot{m}_2(t) &= \alpha \tau_1 m_1(t) + \beta m_2(t) - (\tau_1 + \beta + b)m_2(t), \\
\dot{m}_3(t) &= e(t), m_2(t) = i(t), t \in [\eta, \eta + \sigma],
\end{align*}
\]
which obtains $e(t) \geq m_1(t)$ and $i(t) \geq m_2(t)$, using comparison principle for any $t \geq \eta$. If $R_0 > 1$, when $b > \delta$, the unique endemnic equilibrium $(m^*_1, m^*_2)$ of the auxiliary system has
\[
\begin{align*}
\lim_{t \to \infty} m_1(t) &= \frac{(Q_1 \delta + Q_2)(s_0 - \epsilon)\beta}{(2b + 2\tau_1 - Q_1 \delta - Q_2)\delta} = m^*_1 > \sigma, \\
\lim_{t \to \infty} m_2(t) &= Q_1 \delta + Q_2 = m^*_2 > \sigma,
\end{align*}
\]
where $Q_1 = \tau_1 + \tau_2 + \delta + 2b$ and $Q_2 = [Q_1 \delta^2 + 4\alpha \tau_1 \beta \delta^2][s_0 - \epsilon - (1/R_0)]^{1/2}$. Thus, $\lim E_{t \to \infty} e(t) \geq m^*_1 > \sigma$ and $\lim E_{t \to \infty} i(t) \geq m^*_2 > \sigma$ for $t > \eta$. This result contradicts the boundedness of the solution. Then, all of the conditions of Lemma 2 are valid. This establishes the theorem.

7. Global Asymptotic Stability of Endemic Equilibrium $E^*$

Theorem 4. If $R_0 > 1$, suppose that $((\tau_2 + b)(\beta e + \delta^2 + \tau_1^2 + \tau_1, b)/(\alpha e + \beta b + (\tau_2 + b)\beta) > 1$ and $b > \delta$: there is a unique endemic equilibrium $E^*$. When $2\delta - b < \epsilon < \min[\delta, (\tau_1/2)], E^*$ is globally asymptotically stable.

Proof. Let $P(e, \epsilon, i) = \text{diag}(1, (s_0/i), (s_0/i))$, then $P = C$ and nonsingular in $\Gamma$. Let $f$ denote the vector of system (4); then, $P^{-1} = \text{diag}(0, (e/\epsilon) - (i/i), (e/\epsilon) - (i/i))$. The second compound matrix $[25] A^{[2]}$ associated with a general solution $x(t) = (s(t), e(t), i(t))$ of system (4) takes the form...
\[ A^{[2]} = \begin{pmatrix} -2b - \tau_1 - \epsilon - \beta i + 2\delta i & \beta s + \delta e & e + \beta s - \delta s \\ \alpha \tau_1 & -2b - \epsilon - \tau_2 - \beta i + 3\delta i & -\epsilon \\ 0 & -\beta i & -\tau_2 - 2b - \delta + 3\delta i \end{pmatrix} \]  
\[ (32) \]

Then,

\[ P_f P^{-1} + PA^{[2]} P^{-1} = \begin{pmatrix} X + 2\delta i & \beta s + \delta e & (e + \beta s - \delta s) i \epsilon \\ \frac{\alpha \tau_1 e i}{e} & Y + 3\delta i + \frac{e}{e - i} \left( \frac{\tau}{i} \right) & -\epsilon \\ 0 & \beta i & Z + 3\delta i + \frac{e}{e - i} \left( \frac{\tau}{i} \right) \end{pmatrix}, \]
\[ (33) \]

where \( X = -2b - \epsilon - \tau_1 - \beta i, Y = -2b - \epsilon - \tau_2 - \delta - 2b \). Let

\[ B = P_f P^{-1} + PA^{[2]} P^{-1}, \]
\[ (34) \]

that is, \( B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix} \), where \( B_{11} = \frac{\alpha \tau_1 e i}{e} \), and

\[ B_{21} = \begin{pmatrix} \frac{\alpha \tau_1 e i}{e} \\ 0 \end{pmatrix}, \]

\[ B_{22} = \begin{pmatrix} Y + 3\delta i + \frac{e}{e - i} \left( \frac{\tau}{i} \right) & -\epsilon \\ \beta i & Z + 3\delta i + \frac{e}{e - i} \left( \frac{\tau}{i} \right) \end{pmatrix}. \]
\[ (35) \]

Let \( (u, v, w) \) be a vector in \( \mathbb{R}^3 \); we can define a vector norm in \( \mathbb{R}^3 \) as

\[ |(u, v, w)| = \max|u|, |v|, |w| \].
\[ (36) \]

Let \( \mu \) be the L"{o}zinskii measure with respect to this norm; then, as described in [26], we choose

\[ \mu(B) \leq \sup \{ g_1, g_2 \}, \]
\[ (37) \]

where

\[ g_1 = \mu(B_{11}) + |B_{12}|, \]
\[ g_2 = |B_{21}| + \mu(B_{22}). \]
\[ (38) \]

\( |B_{12}| \) and \( |B_{21}| \) are matrix norms with respect to the \( L^1 \) vector norm and \( \mu \) denotes the L"{o}zinskii measure with respect to the \( L^1 \) norm. Then,

\[ |B_{21}| = \frac{\alpha \tau_1 e i}{e}, \]
\[ \mu_1(B_{11}) = -2b - \epsilon - \tau_1 - \beta i + 2\delta i, \]
\[ \mu_1(B_{12}) = -2b - \epsilon - \tau_2 - \delta + 3\delta i \]
\[ (39) \]

Therefore,

\[ g_1 = -2b - \epsilon - \tau_1 - \beta i + 2\delta i - \max \left\{ (\beta s + \delta e) i \epsilon, (e + \beta s - \delta s) i \epsilon \right\}, \]
\[ g_2 = -2b - \epsilon - \tau_2 - \delta + 3\delta i - \max \left\{ \frac{\alpha \tau_1 e i}{e}, \frac{e}{e - i} \left( \frac{\tau}{i} \right) \right\} \]
\[ (40) \]

From system (4), it follows that \( (\epsilon r/e) = (\beta s i/e) - \tau_1 - b + \delta i \) and \( (\alpha r i/e) = (\tau r i) - \tau_2 + \beta + \delta - i \). We have

\[ g_1 = \begin{cases} \frac{\epsilon r}{e} - b - \epsilon - \beta i + 2\delta i + \left[ e - \delta (s + e) \right] i \epsilon, & \epsilon \geq \delta, \\ \frac{\epsilon r}{e} - b - \epsilon - \beta i + 2\delta i, & \epsilon < \delta. \end{cases} \]
\[ (41) \]

When \( 2\delta - b < \epsilon < \min\{ \delta, (\tau r)/2 \} \), according to the expression of \( g_1 \) and \( g_2 \), \( g_1 < g_2 \) is verified; that is,
Figure 3: Time series diagram of $s$, $e$, and $i$. (a) $R_0 < 1$. (b) $R_0 < 1$.

Figure 4: Continued.
Figure 4: The curve-trend diagram of $i(t)$ with time, when the parameters $\alpha$, $\beta$, $\tau_1$, and $\tau_2$ take different values.

Figure 5: Continued.
Figure 5: The curve-trend diagram of $i(t)$ with time, when the parameters $\alpha$, $\beta$, $\tau_1$, and $\tau_2$ take different values.

Figure 6: Continued.
and \( \tau \) variables. It is found that if Figure 3(a) shows the time-variation diagram of system (4) state \( \alpha \) parameters compare the sizes of \( \beta \), \( \delta \), \( \tau_1 \), and \( \tau_2 \) for \( R_0 \). Unfortunately, we only prove one case. It is difficult to compare the sizes of \( g_1 \) and \( g_2 \). Hence, other cases are not discussed in this paper.

\[
\mu(B) \leq \sup_{t_{0}} \{ g_1, g_2 \} = \frac{\sigma_t}{\epsilon} - b - \epsilon + 2\delta t. \tag{42}
\]

Therefore, we have
\[
\lim_{t_{0} \to +\infty} \sup_{t_{0} \to +\infty} \frac{1}{t} \int_{t_{0}}^{t} \mu(B) ds \leq \frac{1}{t} \left[ \ln e(t) \left( \ln e(0) + (b + \epsilon)(i - 1) \right) \right] < 0, \tag{43}
\]

with Mean Value Theorems for Integrals. This completes the proof.

8. Numerical Simulations

To verify the theoretical results, we perform numerical simulations of system (4). We assume \( \beta = 0.2, \delta = 0.06 \), and \( \tau_2 = 0.1 \). The values of other parameters are shown in Table 1. Figure 3(a) shows the time-variation diagram of system (4) state variables. It is found that if \( R_0 < 1 \), the curve tends to be stable with time, and the disease-free equilibrium \( E_0 \) is globally asymptotically stable. Moreover, we take \( \beta = 0.0106, \delta = 0.007, \) and \( \tau_2 = 0.015 \). As shown in Figure 3(b), it is found that if \( R_0 > 1 \), the state variables of system (4) change with time, and the endemic equilibrium \( E^* \) is globally asymptotically stable.

Figure 4 shows \( i(t) \) will increase with the increase of the parameters \( \alpha, \beta, \tau_1 \), and \( \tau_2 \) if \( R_0 < 1 \). But the change of the parameter \( \tau_1 \) does not change significantly for the proportion of infected individuals. The larger the parameter \( \tau_2 \) is, the shorter the time for the proportion of infected individuals to reach stable state is. Figure 5 shows \( i(t) \) will increase with the increase of the parameters \( \alpha, \beta, \tau_1 \), and \( \tau_2 \) if \( R_0 > 1 \). We can see that when the parameters values of \( \alpha, \beta, \tau_1 \), and \( \tau_2 \) change slightly, this will cause a large change in \( i(t) \).

We further simulate the effect of two different parameters on the threshold \( R_0 \) (Figure 6). When two parameters are higher simultaneously, the threshold \( R_0 \) also increases. For two different parameters, the threshold changes differently.

9. Conclusion

This paper considers the propagation of bat rabies with latent period and establishes a dynamic model. The threshold of the model is calculated as \( R_0 \), which proves that when \( R_0 < 1 \), disease-free equilibrium \( E_0 \) is globally asymptotically stable; when \( R_0 > 1 \), under certain conditions, the system has a unique endemic equilibrium \( E^* \). Under the restrictions, we use the geometric method [27] to prove the global stability of \( E^* \). Finally, numerical simulations and parameter sensitivity analysis are carried out to verify the correctness of the theoretical results. This is consistent with [4]. That is, bats have an adequate latent period and a very low disease-related mortality rate of \( \delta \), which further leads to the maintenance of the virus and continuous transmission. Obviously, our results do not reflect changes of the seasonal transmission compared to [4]. In subsequent studies, we will consider the effects of bat population density constraints, seasonal birth impulses, and hibernation on virus transmission.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

This research is partially supported by the National Science Foundation of China (Grant nos. 11971278, 61873154, and
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