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MATHEMATICAL ANALYSIS OF A GENERALIZED REACTION-DIFFUSION MODEL OF EBOLA TRANSMISSION IN BATS

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Abstract. Bats of the Pteropodidae family are believed to be the natural hosts of the Ebola virus (EV). These bats often have extensive home ranges, which can span large areas, including across countries and regions. We propose in this work to consider the mobility effect by studying a new generalized reaction-diffusion spatiotemporal system that emphasizes the transmission of Ebola virus disease (EVD) among bats. Besides transmission through direct contact with infectious bats, the model also considers infection via a contaminated environment. This transmission mechanism is characterized by two general incidence functions, encompassing various types of incidence rates. We provide evidence of the uniqueness, non-negativity, and boundedness of solutions considering the Neumann boundary conditions, indicating that the flux is zero at the boundary, and positive initial data. The stability behavior of the equilibria is demonstrated theoretically by using appropriate Lyapunov functionals and the linearization method, and numerically via some numerical simulations.

Keywords: Ebola; bats; general incidence rate; PDEs; global stability.

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1. INTRODUCTION

EVD was initially identified in the Democratic Republic of Congo in 1976 and had caused 280 deaths out of 318 reported cases. In the same year, Sudan experienced another outbreak, causing 156 deaths. Since its discovery, EVD has reappeared in several countries in West Africa. The largest outbreak was in 2014-2016. It started in Guinea and spreads to other countries such as Liberia and Sierra Leone, killing more than 11000 people [1]. The second deadliest outbreak was declared in the Democratic Republic of Congo in August 2018 and caused the death of about 2299 persons as of 3^{th} July 2020 [2].

The frequent resurgence of the EV has raised suspicions as to the existence of a natural reservoir of the virus. Bats, particularly fruit bats from the Pteropodidae family, have been identified as potential natural reservoirs of the EV. These bats can harbor the virus without showing signs of illness, making them key players in the virus's ecology [3, 4, 5]. These findings suggest that bats can carry the virus asymptomatically, contributing to its persistence in nature.

The virus can be spread from bats to other animals such as monkeys, apes, and antilopes through direct contact with bat saliva, urine, feces, or partially eaten fruits dropped by bats. Humans can contract the EV through direct exposure to the bodily fluids or tissues of infected animals. This often occurs during hunting, butchering, or preparing bushmeat. Additionally, bats roost in large colonies, often in close proximity to human settlements, increasing the likelihood of virus spillover to humans and domestic animals. Once a human is infected, the virus can spread from person to person by coming into direct contact with organs, secretions, blood or other bodily fluids of infected individuals, or through contact with materials and surfaces contaminated with these fluids.

In the literature, there are few mathematical models dealing with the spread of the virus in the bat population. For example, in [6], the authors used a combination of statistical and computational models to predict the spatial distribution of infected bats. This study presents a framework for predicting the spatial distribution of bats infected with filoviruses, which include viruses like Ebola and Marburg. The framework aims to identify regions with a higher likelihood of infection, thereby aiding in disease monitoring and control efforts. In [7], the bat-to-bat EV transmission model proposed by Berge et al. described the EV transmission to

the susceptible bats from the infectious bats and the contaminated environment. The authors utilized bilinear incidence functions and supposed that bats never recover from Ebola.

On the other hand, the unique immune system of bats and their ability to coexist with various pathogens, including the EV, has been the subject of several scientific studies. Bats have evolved immune responses that allow them to tolerate and coexist with pathogens that can be deadly to other species. For example, in [8], the authors explore the immune system of bats, focusing on their ability to host a variety of viruses, including Ebola, without developing disease symptoms. The review [9] discusses how bats can host viruses and the mechanisms that allow them to persist without causing disease. The paper [10] presents the idea of disease tolerance in bats, highlighting their immune system's ability to control infections without severe disease symptoms. The work [11] delves into the immune responses of bats and how these responses contribute to their ability to harbor viruses without significant health impacts. This ability does not necessarily mean that bats carry the virus for their entire lives, but it does suggest they can manage these infections without succumbing to them.

Rhoubari et al. [12] introduced a cure rate and extended the epizootic model presented in [7] by modeling the process transmission via two general incidence functions that comprise a variety of incidence rates presented in the literature such as the classical bilinear incidence, the saturated incidence, the Beddington-DeAngelis functional response, the Crowley-Martin functional response and the Hattaf-Yousfi functional response. A particular case of the model [12] was studied in [13] by considering the effect of memory and two saturated incidence functions.

It is important to note that bats are highly mobile creatures, capable of long-distance migrations and daily foraging trips that span significant distances. Their mobility is a critical factor in the spread of zoonotic diseases like Ebola. Migration patterns vary among species, with some bats traveling hundreds of kilometers during seasonal migrations. For instance, the Eidolon helvum, or African straw-colored fruit bat, migrates across Africa, covering vast distances and potentially spreading pathogens over large areas [14, 15, 16]. Moreover, this extensive mobility facilitates the mixing of bat populations and the potential exchange of viruses.

Motivated by the above mathematical and ecological reasons, and in order to give an improved picture of the disease diffusion between bats, we propose to study the following reaction-diffusion problem which is built by partial differential equations (PDEs) as follows:

(1)
$$\begin{cases} \dfrac{\partial P_1}{\partial t} = d_1\Delta P_1 + \beta - mP_1(x,t) - \zeta(P_1(x,t),P_2(x,t))P_2(x,t) - \Gamma(P_1(x,t),Q(x,t))Q(x,t), \\ \dfrac{\partial P_2}{\partial t} = d_2\Delta P_2 + \zeta(P_1(x,t),P_2(x,t))P_2(x,t) + \Gamma(P_1(x,t),Q(x,t))Q(x,t) - (m+r)P_2(x,t), \\ \dfrac{\partial P_3}{\partial t} = d_3\Delta P_3 + rP_2(x,t) - mP_3(x,t), \\ \dfrac{\partial Q}{\partial t} = d_Q\Delta Q + \delta P_2(x,t) - eQ(x,t). \end{cases}$$

where $P_1(x,t)$, $P_2(x,t)$ and $P_3(x,t)$ represent the concentrations of susceptible, infectious and recovered bats, respectively at location $x \in \Theta$ and time t. Additionally, $Q(x,t)$ denotes the concentration of EV in the environment at time t and location $x \in \Theta$. d_1, d_2, d_3 and d_Q are the diffusion coefficients and Δ represent the Laplacien operator. Θ is bounded within $\mathbf{R}^n$ and has a smooth boundary, $\partial\Theta$.

Moreover, the susceptible population of bats grows due to recruitment at rate β, which includes immigration or births, and declines at the natural rate of mortality m. Additionally, this population decreases and shifts to the infectious category through direct contact with infectious bats at rate $\zeta(P_1,P_2)P_2$ or through contact with a contaminated environment at rate $\Gamma(P_1,Q)Q$. Therefore, the overall infection rate for the susceptible bat population is $\zeta(P_1,P_2)P_2 + \Gamma(P_1,Q)Q$. Infectious bats recover from Ebola at rate r and die solely at the natural mortality rate m. δ represents the deposition rate of EV into the environment by infectious bats, while e denotes the decay rate of EV in the environment.

Since the compartment P_3 does not affect the others, this permits us to reduce the system (1) to

(2)
$$\begin{cases} \dfrac{\partial P_1}{\partial t} = d_1\Delta P_1 + \beta - mP_1(x,t) - \zeta(P_1(x,t),P_2(x,t))P_2(x,t) - \Gamma(P_1(x,t),Q(x,t))Q(x,t), \\ \dfrac{\partial P_2}{\partial t} = d_2\Delta P_2 + \zeta(P_1(x,t),P_2(x,t))P_2(x,t) + \Gamma(P_1(x,t),Q(x,t))Q(x,t) - (m+r)P_2(x,t), \\ \dfrac{\partial Q}{\partial t} = d_Q\Delta Q + \delta P_2(x,t) - eQ(x,t). \end{cases}$$

Furthermore, we accept that the three sub-populations do not migrate across $\partial\Theta$. Consequently, we analyze system (2) with the homogeneous Neumann boundary conditions

$$\frac{\partial P_1}{\partial n} = \frac{\partial P_2}{\partial n} = \frac{\partial Q}{\partial n} = 0, \text{ on } \partial\Theta \times (0, +\infty), \tag{3}$$

and initial conditions

$$P_1(x,0) = \gamma_1(x) \geq 0, \;\; P_2(x,0) = \gamma_2(x) \geq 0, \;\; Q(x,0) = \gamma_3(x) \geq 0, \;\; x \in \bar{\Theta}. \tag{4}$$

Note that $\dfrac{\partial}{\partial n}$ is the derivative along the outward normal on $\partial\Theta$.

As in [12, 17, 18], we assume that the functions ζ and Γ are continuously differentiable in $\mathbf{R}^2_+$ and meet the following criteria:

(H_1) $\zeta(0,P_2) = 0$, $\dfrac{\partial \zeta}{\partial P_1}(P_1,P_2) > 0$, $\dfrac{\partial \zeta}{\partial P_2}(P_1,P_2) \leq 0$ for all $P_1, P_2 \geq 0$.

(H_2) $\Gamma(0,Q) = 0$, $\dfrac{\partial \Gamma}{\partial P_1}(P_1,Q) > 0$, $\dfrac{\partial \Gamma}{\partial Q}(P_1,Q) \leq 0$ for all $P_1, Q \geq 0$.

From a biological standpoint, the two hypotheses are both reasonable and align with the observed reality. The first hypothesis indicates that the incidence rate by infectious is zero when there are no susceptible bats. Morever, this incidence rate increases as the number of susceptible bats rises, even when the number of infectious bats remains constant. And the more the amount of infectious bats, the less the average number of susceptibles which are infected by each infectious bat in the unit time will occur. Analogically, we explain the biological significance of the second hypothesis for the incidence rate by contamined environment.

The remainder of the paper is structured as follows. The next section examines that the model (2)-(4) is mathematically and biologically meaningful by showing the existence, boundedness, and positivity of solutions. In Section 3, we demonstrate the global stability of equilibria. Section 4 validates our theoretical findings through numerical simulations. Finally, we conclude the paper in Section 5.

2. Model Validity and Equilibria

Firstly, we prove that the problem (2)-(4) is mathematically and biologically significant by investigating the existence, positivity, boundedness and uniqueness of solutions.

Theorem 2.1. *Let $s_0 = (\gamma_1, \gamma_2, \gamma_3)^T \in \mathscr{B} = [C(\bar{\Theta})]^3$ be any initial condition satisfying (4). There is a unique solution of problem (2)-(4) defined on $[0, +\infty)$ and this solution remains positive and bounded for all positive t.*

Proof. System (2)-(4) can be expressed in an abstract form within the Banach space $\mathscr{B}$ as follows

$$\begin{aligned} s'(t) &= As(t) + W(s(t)), \quad t > 0, \\ s(0) &= s_0 \in \mathscr{B}, \end{aligned} \tag{5}$$

where $s = (P_1, P_2, Q)^T$, $As = (d_1 \triangle P_1, d_2 \triangle P_2, d_Q \triangle Q)^T$ and

$$W(s) = \begin{pmatrix} \beta - mP_1 - \zeta(P_1, P_2)P_2 - \Gamma(P_1, Q)Q \\ \zeta(P_1, P_2)P_2 + \Gamma(P_1, Q)Q - (m+r)P_2 \\ \delta P_2 - eQ \end{pmatrix}.$$

It is apparent that W is locally Lipschitz in $\mathscr{B}$. According to [19], we infer that the problem (5) possesses a unique local solution on $[0, T_m)$, where T_m indicates the maximal existence time for solution of problem (5).

In addition, system (2) can be rewritten in the form

$$\begin{cases} \dfrac{\partial P_1}{\partial t} - d_1 \Delta P_1 = K_1(P_1, P_2, Q), \\ \dfrac{\partial P_2}{\partial t} - d_2 \Delta P_2 = K_2(P_1, P_2, Q), \\ \dfrac{\partial Q}{\partial t} - d_Q \Delta Q = K_3(P_1, P_2, Q). \end{cases} \tag{6}$$

Obviously, $K_1(P_1, P_2, Q)$, $K_2(P_1, P_2, Q)$ and $K_3(P_1, P_2, Q)$ are continuously differentiable with $K_1(0, P_2, Q) = \beta \geq 0$, $K_2(P_1, 0, Q) = \Gamma(P_1, Q)Q \geq 0$, and $K_3(P_1, P_2, 0) = \delta P_2 \geq 0$. Moreover, the initial data of system (2) are non-negative. Therefore, using the maximum principle as discussed in Smoller's book [20], we deduce that the local solution is positive.

Now, we examine that the solution is bounded. From (2)-(4), we have

$$\begin{cases} \dfrac{\partial P_1}{\partial t} - d_1 \triangle P_1 \leq \beta - mP_1, \\ \dfrac{\partial P_1}{\partial n} = 0, \\ P_1(x, 0) = \gamma_1(x) \leq \|\gamma_1\|_\infty = \max\limits_{x \in \bar{\Theta}} \gamma_1(x), \end{cases} \tag{7}$$

Utilizing Lemma 1 as presented by Hattaf in [21], we obtain

$$P_1(x,t) \leq \max\{\frac{\beta}{m}, \|\gamma_1\|_\infty\},\ \forall (x,t) \in \bar{\Theta} \times [0, T_m).$$

Based on Theorem 2 in [22], the L^1 uniform boundedness of $P_2(x,t)$ leads to the L^∞ uniform boundedness of $P_2(x,t)$.

Using $\dfrac{\partial P_1}{\partial n} = \dfrac{\partial P_2}{\partial n} = 0$ and

$$\frac{\partial}{\partial t}\big(P_1 + P_2\big) - \triangle\big(d_1 P_1 + d_2 P_2\big) \leq \beta - m\big(P_1 + P_2\big),$$

we get

$$\frac{\partial}{\partial t}\big(\int_\Theta (P_1 + P_2)dx\big) \leq \text{mes}(\Theta)\beta - m\big(\int_\Theta (P_1 + P_2)dx\big).$$

Hence,

$$\int_\Theta (P_1 + P_2)dx \leq \text{mes}(\Theta) \max\{\frac{\beta}{m}, \|\gamma_1 + \gamma_2\|_\infty\},$$

which implies, $\sup_{t\geq 0} \int_\Theta P_2(x,t)dx \leq C := \text{mes}(\Theta) \max\{\dfrac{\beta}{m}, \|\gamma_1 + \gamma_2\|_\infty\}$.

Using Theorem 3.1 in [22], we derive that there exists a positive constant C^* depending on C and on $\|\gamma_1 + \gamma_2\|_\infty$ such that

$$\sup_{t\geq 0} \|P_2(.,t)\|_\infty \leq C^*.$$

From the above, we deduce the L^∞ boundedness of $P_1(x,t)$ and $P_2(x,t)$ on $\bar{\Theta} \times [0, T_m)$. On the other hand, since P_2 is bounded and from the system (2)-(4), we get

$$\text{(8)} \qquad \begin{cases} \dfrac{\partial Q}{\partial t} - d_Q \triangle Q \leq \delta K - eQ, \\ \dfrac{\partial Q}{\partial n} = 0, \\ Q(x,0) = \gamma_3(x) \leq \|\gamma_3\|_\infty = \max\limits_{x\in\overline{\Theta}} \gamma_3(x), \end{cases}$$

where $K = \max\{P_2(x,t), (x,t) \in \Theta \times [0, T_m)\}$.

Thus, we obtain

$$Q(x,t) \leq \max\{\frac{\delta}{e}K, \|\gamma_3\|_\infty\},\ \forall (x,t) \in \bar{\Theta} \times [0, T_m).$$

Therefore, we achieve the L^∞ boundedness of all compartments of model (2) on $\bar{\Theta} \times [0, T_m)$. Utilizing the standard theory for semilinear parabolic systems [23], we obtain that $T_m = +\infty$. This finishes the proof. ■

On the other hand, based on the work of Rhoubari in [12], the basic reproduction number of Ebola in bats when the spatial effect is not considered is

$$\mathscr{R}_0 = \frac{e\zeta\left(\frac{\beta}{m},0\right)+\delta\Gamma\left(\frac{\beta}{m},0\right)}{e(m+r)}. \tag{9}$$

Moreover, system (2) has consistently an EVD free equilibrium (EFE) of the form $E_1(\frac{\beta}{m},0,0)$. And if $\mathscr{R}_0$ exceeds 1, system (2) has an endemic equilibrium $E_2(P_1^*,P_2^*,Q^*)$ with $P_1^* \in (0,\frac{\beta}{m})$, $P_2^* = \dfrac{\beta - mP_1^*}{m+r}$ and $Q^* = \dfrac{\delta}{e}P_2^* = \dfrac{\delta(\beta - mP_1^*)}{e(m+r)}$. Next, we analyze the equilibria stability.

3. Analysis of Equilibria Stability

We have the main following result concerning the stability of the EFE E_1.

Theorem 3.1. *The EFE of model (2) is globally asymptotically stable if $\mathscr{R}_0 \leq 1$. However, if $\mathscr{R}_0$ exceeds 1, this equilibrium becomes unstable.*

Proof. In the absence of the spatial component, Rhoubari et al. [12] presented the following Lyapunov functional:

$$G(t) = eP_2(t) + \Gamma(\frac{\beta}{m},0)Q(t).$$

By employing the method of Hattaf and Yousfi described in [24], we build the following Lyapunov functional for the PDEs problem (2) at E_1 as

$$\mathscr{G} = \int_\Theta G(P_1(x,t),P_2(x,t),Q(x,t))dx.$$

By calculating the time derivative of $\mathscr{G}$ along the model's (2) solution, we get

$$\begin{aligned}\frac{d\mathscr{G}}{dt} &= \int_\Theta \left\{ \left(e\zeta(P_1,P_2) - e(m+r) + \delta\Gamma(\frac{\beta}{m},0) \right) P_2 \right. \\ &\quad \left. + e\left(\Gamma(P_1,Q) - \Gamma(\frac{\beta}{m},0) \right) Q \right\} dx.\end{aligned}$$

Using (7) and based on Lemma 1 of [21], we get

$$\limsup_{t\to\infty} \left(P_1(x,t)\right) \leq \frac{\beta}{m}.$$

Thus, all ω-limit points satisfy $P_1(x,t) \leq \dfrac{\beta}{m}$. Then, It suffices to take into account solutions for which $P_1(x,t) \leq \dfrac{\beta}{m}$. By the $\mathscr{R}_0$ formula given in (9) and under the hypotheses $(H_1)-(H_2)$, we derive

$$\begin{aligned}\frac{d\mathscr{G}}{dt} &\leq \int_\Theta \left\{ \left(e\zeta(\frac{\beta}{m},0) - e(m+r) + \delta\Gamma(\frac{\beta}{m},0) \right) P_1 \right. \\ &\leq \int_\Theta \left\{ e(m+r)\big(\mathscr{R}_0 - 1\big) P_1 \right\} dx.\end{aligned}$$

Since $\mathscr{R}_0 \leq 1$, we get $\dfrac{d\mathscr{G}}{dt} \leq 0$. And by a simple verification, we find that the singleton $\{E_1\}$ is the largest invariant set within $\{(P_1,P_2,Q) | \dfrac{d\mathscr{G}}{dt} = 0\}$. Thanks to LaSalle invariance principle [25], we confirm the global stability of E_1 when $\mathscr{R}_0 \leq 1$.

Now, we check that the EFE E_1 loses its stability when $\mathscr{R}_0 > 1$.

Denote $0 = \varepsilon_1 < \varepsilon_2 < ... < \varepsilon_n < ...$ the eigenvalues of the Laplace operator $-\triangle$ in a bounded domain with homogeneous Neumann boundary conditions and $\psi(\varepsilon_i)$ the eigenfunction space related to the eigenvalue ε_i in $C^1(\Theta)$.

Let $\left\{ \varphi_{ij} : j = 1,2,...,\dim\psi(\varepsilon_i) \right\}$ be an orthonormal basis of $\psi(\varepsilon_i)$, $\mathbb{V} = [C^1(\Theta)]^3$ and $\mathbb{V}_{ij} = \left\{ \mathbf{v}\varphi_{ij} : \mathbf{v} \in \mathbf{R}^3 \right\}$. Then we have

$$\mathbb{V} = \bigoplus_{i=1}^{\infty} \mathbb{V}_i \qquad \text{and} \qquad \mathbb{V}_i = \bigoplus_{j=1}^{\dim\psi(\varepsilon_i)} \mathbb{V}_{ij}$$

Let $\tilde{E} = \left\{ \tilde{P}_1, \tilde{P}_2, \tilde{Q} \right\}$ be any given homogeneous equilibrium according to space of the system (2)-(4). Carry out a variable change:

$S_1(x,t) = P_1(x,t) - \tilde{P}_1$, $S_2(x,t) = P_2(x,t) - \tilde{P}_2$, and $S_3(x,t) = Q(x,t) - \tilde{Q}$. Then system (2) is linearized at $\tilde{E}$ as follows

$$\frac{\partial S}{\partial t} = \mathscr{D}\triangle S + \mathscr{J}\, S(x,t), \tag{10}$$

where

$$\mathscr{J} = \left[\ \mathscr{J}_1, \mathscr{J}_2, \mathscr{J}_Q \ \right],$$

with

$$\mathscr{J}_1 = \left(-m - \tilde{P}_2 \frac{\partial\zeta}{\partial P_1} - \tilde{Q}\frac{\partial\Gamma}{\partial P_1},\ \tilde{P}_2\frac{\partial\zeta}{\partial\tilde{P}_1} + \tilde{Q}\frac{\partial\Gamma}{\partial P_1}, 0 \right)^T,$$

$$\mathscr{J}_2 = \left(-\zeta(\tilde{P_1},\tilde{P_2}) - \tilde{P_2}\frac{\partial \zeta}{\partial P_2},\ \tilde{P_2}\frac{\partial \zeta}{\partial P_2} + \zeta(\tilde{P_1},\ \tilde{P_2}) - (m+r), \delta \right)^T,$$

$$\mathscr{J}_Q = \left(-\tilde{Q}\frac{\partial \Gamma}{\partial Q} - \Gamma(\tilde{P_1},\tilde{Q}),\ \tilde{Q}\frac{\partial \Gamma}{\partial Q} + \Gamma(\tilde{P_1},\tilde{Q}), -e \right)^T,$$

$\mathscr{D} = \text{diag}(d_1, d_2, d_Q)$ and $S = (S_1, S_2, S_3)^T$.

Let $\mathscr{L}S = \mathscr{D}\triangle S + \mathscr{J}S(x,t)$. For any $i \geq 1$, $\mathbb{V}_i$ remains invariant under the operator $\mathscr{L}$. Additionaly, ξ is an eigenvalue of $\mathscr{L}$ signifies that it is a root of the equation

$$\det(\mathscr{J} - \varepsilon_i \mathscr{D} - \xi \mathbb{I}_3) = 0, \tag{11}$$

for some $i \geq 1$, for which an eigenvector exists in $\mathbb{V}_i$.

Now, we determine the characteristic equation (11) at the EFE E_1 as

$$(m + \varepsilon_i d_1 + \xi) g_i(\xi) \ = \ 0, \tag{12}$$

where

$$\begin{aligned} g_i(\xi) \ = \ & \xi^2 + \left(\varepsilon_i(d_2 + d_Q) + m + r + e - \zeta(\frac{\beta}{m}, 0) \right)\xi + \Big[\varepsilon_i^2 (d_2 d_Q) \\ & + \varepsilon_i \left(e d_2 + d_Q(m+r) - d_Q \zeta(\frac{\beta}{m}, 0) \right) + e(m+r)(1 - \mathscr{R}_0) \Big] \end{aligned}$$

It is evident that $\xi = -m - \varepsilon_i d_1$ is a negative root of the equation (12). The solutions of the equation $g_i(\xi) = 0$ represent the other remaining roots. Since $\varepsilon_1 = 0$ and $\mathscr{R}_0$ is greater than 1, we have $g_1(0) = e(m+r)(1-\mathscr{R}_0) < 0$. Moreover, we have $\lim_{\xi \to +\infty} g_i(\xi) = +\infty$. Therefore, we deduce the existence of a positive real root ξ^* of the equation $g_1(\xi) = 0$. This leads to conclude that the equilibrium E_1 is unstable when $\mathscr{R}_0$ exceeds 1. This finnishes the proof ■

For the global stability of the second equilibrium E_2, we assume that $\mathscr{R}_0 > 1$ and the functions ζ and Γ satisfy, for all $P_1, P_2, Q > 0$, the following hypothesis

$$(H_3) \qquad \begin{array}{l} \left(1 - \dfrac{\zeta(P_1,P_2)}{\zeta(P_1,P_2^*)}\right)\left(\dfrac{\zeta(P_1,P_2^*)}{\zeta(P_1,P_2)} - \dfrac{P_2}{P_2^*}\right) \leq 0, \\ \left(1 - \dfrac{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}\right)\left(\dfrac{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)} - \dfrac{Q}{Q^*}\right) \leq 0. \end{array}$$

Theorem 3.2. *Assuming $\mathscr{R}_0 > 1$ and hypothesis (H_3) is valid, then the endemic equilibrium E_2 achieves global asymptotic stability.*

Proof. Using the same method as above and the Lyapunov functional (13) from the ordinary differential equations (ODEs) model in [12], we construct the Lyapunov functional of PDEs model for the endemic state E_2 as follows

$$\mathscr{H} = \int_\Theta H(P_1(x,t), P_2(x,t), Q(x,t))dx.$$

with

$$(13) H(t) = P_1(t) - P_1^* - \int_{P_1^*}^{P_1(t)} \frac{\zeta(P_1^*, P_2^*)}{\zeta(X, P_2^*)} dX + P_2^* \Phi\left(\frac{P_2(t)}{P_2^*}\right) + \frac{\Gamma(P_1^*, Q^*)}{e} Q^* \Phi\left(\frac{Q(t)}{Q^*}\right),$$

and $\Phi(Y) = Y - 1 - \ln Y$, $Y > 0$. By a quick study of the variations of Φ, we show that it attains its strict global minimum at 1 and $\Phi(1) = 0$. Then $\Phi(Y) \geq 0$ and the functional H is non-negative.

Calculating the time derivative of $\mathscr{H}$ along the solution of model (2), we have

$$\begin{aligned}
\dot{\mathscr{H}}(t)|_{(2)} = & \int_\Theta \left\{ mP_1^* \left(1 - \frac{P_1}{P_1^*}\right)\left(1 - \frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)}\right) \right. \\
& + \zeta(P_1^*, P_2^*) P_2^* \left(2 - \frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)} - \frac{\zeta(P_1, P_2)}{\zeta(P_1^*, P_2^*)} + \frac{\zeta(P_1, P_2) P_2}{\zeta(P_1, P_2^*) P_2^*} - \frac{P_2}{P_2^*}\right) \\
& + \Gamma(P_1^*, Q^*) Q^* \left(3 - \frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)} + \frac{\zeta(P_1^*, P_2^*)\Gamma(P_1, Q) Q}{\zeta(P_1, P_2^*)\Gamma(P_1^*, Q^*) Q^*} - \frac{Q}{Q^*}\right. \\
& \left. - \frac{\Gamma(P_1, Q) Q P_2^*}{\Gamma(P_1^*, Q^*) Q^* P_2} - \frac{Q^* P_2}{Q P_2^*}\right) + \left(1 - \frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)}\right) d_1 \triangle P_1 + \left(1 - \frac{P_2^*}{P_2}\right) d_2 \triangle P_2 \\
& \left. + \frac{\Gamma(P_1^*, Q^*)}{e}\left(1 - \frac{Q^*}{Q}\right) d_Q \triangle Q \right\} dx.
\end{aligned}$$

Introducing Φ, we get

$$\begin{aligned}
\dot{\mathscr{H}}(t)|_{(2)} = & \int_\Theta \left\{ mP_1^* \left(1 - \frac{P_1}{P_1^*}\right)\left(1 - \frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)}\right) \right. \\
& + \zeta(P_1^*, P_2^*) P_2^* \left(-1 - \frac{P_2}{P_2^*} + \frac{\zeta(P_1, P_2^*)}{\zeta(P_1, P_2)} + \frac{\zeta(P_1, P_2) P_2}{\zeta(P_1, P_2^*) P_2^*}\right) \\
& + \Gamma(P_1^*, Q^*) Q^* \left(-1 - \frac{Q}{Q^*} + \frac{\zeta(P_1, P_2^*)\Gamma(P_1^*, Q^*)}{\zeta(P_1^*, P_2^*)\Gamma(P_1, Q)} + \frac{\zeta(P_1^*, P_2^*)\Gamma(P_1, Q) Q}{\zeta(P_1, P_2^*)\Gamma(P_1^*, Q^*) Q^*}\right) \\
& - \zeta(P_1^*, P_2^*) P_2^* \left[\Phi\left(\frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)}\right) + \Phi\left(\frac{\zeta(P_1, P_2)}{\zeta(P_1^*, P_2^*)}\right) + \Phi\left(\frac{\zeta(P_1, P_2^*)}{\zeta(P_1, P_2)}\right)\right] \\
& - \Gamma(P_1^*, Q^*) Q^* \left[\Phi\left(\frac{\zeta(P_1^*, P_2^*)}{\zeta(P_1, P_2^*)}\right) + \Phi\left(\frac{Q^* P_2}{Q P_2^*}\right) + \Phi\left(\frac{\Gamma(P_1, Q) Q P_2^*}{\Gamma(P_1^*, Q^*) Q^* P_2}\right)\right.
\end{aligned}$$

$$+\Phi\left(\frac{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}\right)\Big]+\left(1-\frac{\zeta(P_1^*,P_2^*)}{\zeta(P_1,P_2^*)}\right)d_1\triangle P_1+\left(1-\frac{P_2^*}{P_2}\right)d_2\triangle P_2$$
$$+\frac{\Gamma(P_1^*,Q^*)}{e}\left(1-\frac{Q^*}{Q}\right)d_Q\triangle Q\Big\}dx.$$

By applying Green's theorem and considering the homogeneous Neumann boundary conditions (4), we have:

$$\int_\Theta\left(1-\frac{\zeta(P_1,P_2^*)}{\zeta(P_1^*,P_2^*)}\right)\triangle P_1dx=\int_{\partial\Theta}\left(1-\frac{\zeta(P_1,P_2^*)}{\zeta(P_1^*,P_2^*)}\right)\frac{\partial P_1}{\partial n}ds-\int_\Theta\nabla\left(1-\frac{\zeta(P_1,P_2^*)}{\zeta(P_1^*,P_2^*)}\right)\cdot\nabla P_1dx$$

with ds is the differential arc length element along the boundary $\partial\Theta$.

The boundary term vanishes. Morever, since

$$\nabla\left(1-\frac{\zeta(P_1,P_2^*)}{\zeta(P_1^*,P_2^*)}\right)=-\frac{\partial}{\partial P_1}\left(\frac{\zeta(P_1,P_2^*)}{\zeta(P_1^*,P_2^*)}\right)\nabla P_1$$

We are left with the term

$$\int_\Theta\left(1-\frac{\zeta(P_1,P_2^*)}{\zeta(P_1^*,P_2^*)}\right)d_1\triangle P_1dx=-d_1\int_\Theta\frac{\zeta(P_1,P_2^*)}{\left(\zeta(P_1^*,P_2^*)\right)^2}\frac{\partial\zeta(P_1,P_2^*)}{\partial P_1}\|\nabla P_1\|^2dx.$$

In addition, we easily verify that

$$\int_\Theta\left(1-\frac{P_2^*}{P_2}\right)d_2\triangle P_2dx=-d_2P_2^*\int_\Theta\frac{\|\nabla P_2\|^2}{P_2^2}dx,$$

and

$$\int_\Theta\left(1-\frac{Q^*}{Q}\right)d_Q\triangle Qdx=-d_QQ^*\int_\Theta\frac{\|\nabla Q\|^2}{Q^2}dx.$$

Substituting this into the formula of the time derivative of $\mathscr{H}$, we get:

$$\begin{aligned}\dot{\mathscr{H}}(t)|_{(2)} &= \int_\Theta\Big\{mP_1^*\left(1-\frac{P_1}{P_1^*}\right)\left(1-\frac{\zeta(P_1^*,P_2^*)}{\zeta(P_1,P_2^*)}\right)\\
&+\zeta(P_1^*,P_2^*)P_2^*\left(-1-\frac{P_2}{P_2^*}+\frac{\zeta(P_1,P_2^*)}{\zeta(P_1,P_2)}+\frac{\zeta(P_1,P_2)P_2}{\zeta(P_1,P_2^*)P_2^*}\right)\\
&+\Gamma(P_1^*,Q^*)Q^*\left(-1-\frac{Q}{Q^*}+\frac{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}+\frac{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)Q}{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)Q^*}\right)\\
&-\zeta(P_1^*,P_2^*)P_2^*\left[\Phi\left(\frac{\zeta(P_1^*,P_2^*)}{\zeta(P_1,P_2^*)}\right)+\Phi\left(\frac{\zeta(P_1,P_2)}{\zeta(P_1^*,P_2^*)}\right)+\Phi\left(\frac{\zeta(P_1,P_2^*)}{\zeta(P_1,P_2)}\right)\right]\\
&-\Gamma(P_1^*,Q^*)Q^*\left[\Phi\left(\frac{\zeta(P_1^*,P_2^*)}{\zeta(P_1,P_2^*)}\right)+\Phi\left(\frac{Q^*P_2}{QP_2^*}\right)+\Phi\left(\frac{\Gamma(P_1,Q)QP_2^*}{\Gamma(P_1^*,Q^*)Q^*P_2}\right)\right.\\
&\left.+\Phi\left(\frac{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}\right)\right]dx\Big\}-d_1\zeta(P_1^*,P_2^*)\int_\Theta\frac{\partial\zeta(P_1,P_2^*)}{\partial P_1}\frac{\|\nabla P_1\|^2}{\left(\zeta(P_1,P_2^*)\right)^2}dx\end{aligned}$$

$$-d_2P_2^*\int_\Theta \frac{\|\nabla P_2\|^2}{P_2^2}dx - d_Q\left(\frac{\Gamma(P_1^*,Q^*)}{e}\right)Q^*\int_\Theta \frac{\|\nabla Q\|^2}{Q^2}dx.$$

Now, we verify the sign of the functional $\mathscr{H}$.

Since the function $\zeta(P_1,P_2)$ is monotonically strictly increasing with respect to P_1, we have

$$\left(1-\frac{P_1}{P_1^*}\right)\left(1-\frac{\zeta(P_1^*,P_2^*)}{\zeta(P_1,P_2^*)}\right)\leq 0.$$

From (H_3), we obtain

$$-1-\frac{P_2}{P_2^*}+\frac{\zeta(P_1,P_2^*)}{\zeta(P_1,P_2)}+\frac{\zeta(P_1,P_2)P_2}{\zeta(P_1,P_2^*)P_2^*}=\left(1-\frac{\zeta(P_1,P_2)}{\zeta(P_1,P_2^*)}\right)\left(\frac{\zeta(P_1,P_2^*)}{\zeta(P_1,P_2)}-\frac{P_2}{P_2^*}\right)\leq 0$$

and

$$\begin{aligned}&-1-\frac{Q}{Q^*}+\frac{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}+\frac{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)Q}{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)Q^*}\\ =\ &\left(1-\frac{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}\right)\left(\frac{\zeta(P_1,P_2^*)\Gamma(P_1^*,Q^*)}{\zeta(P_1^*,P_2^*)\Gamma(P_1,Q)}-\frac{Q}{Q^*}\right)\leq 0.\end{aligned}$$

Since $\Phi(Y)\geq 0$ for $Y>0$, we have $\dot{\mathscr{H}}(t)|_{(2)}\leq 0$ with equality if and only if $P_1=P_1^*$, $P_2=P_2^*$ and $Q=Q^*$. As a result, and by [25], we conclude that E_2 is globally asymptotically stable. ∎

4. Numerical Simulations

This section aims mainly to confirm Theorems 3.1 and 3.2 numerically. For this reason, we apply the primary results mentioned above to the example below.

$$\begin{cases}\dfrac{\partial P_1}{\partial t}=d_1\Delta P_1+\beta-mP_1(x,t)-\dfrac{q_1P_1(x,t)P_2(x,t)}{1+o_1P_2(x,t)}-\dfrac{q_2P_1(x,t)Q(x,t)}{1+o_2Q(x,t)},\\ \dfrac{\partial P_2}{\partial t}=d_2\Delta P_2+\dfrac{q_1P_1(x,t)P_2(x,t)}{1+o_1P_2(x,t)}+\dfrac{q_2P_1(x,t)Q(x,t)}{1+o_2Q(x,t)}-(m+r)P_2(x,t),\\ \dfrac{\partial Q}{\partial t}=d_Q\Delta Q+\delta P_2(x,t)-eQ(x,t),\end{cases}\tag{14}$$

with Neumann boundary conditions

$$\frac{\partial P_1}{\partial n}=\frac{\partial P_2}{\partial n}=\frac{\partial Q}{\partial n}=0,\ on\ \partial\Theta\times(0,+\infty),\tag{15}$$

and initial conditions

(16)

$$P_1(x,0)=\gamma_1(x)=100\geq 0,\ \ P_2(x,0)=\gamma_2(x)=20\geq 0,\ \ Q(x,0)=\gamma_3(x)=2\geq 0,\ \ x\in\bar{\Theta}.$$

q_1 and q_2 present the infection rates attributed to infectious bats and contaminated environment, respectively. The positive constants o_1 and o_2 describe the effect of saturation.

By replacing ζ and Γ by their expressions, the basic reproduction number $\tilde{\mathscr{R}}_0$ of model (14) is given by

$$\tilde{\mathscr{R}}_0 = \frac{\beta}{me(m+r)}\Big(eq_1 + \delta q_2\Big). \tag{17}$$

In addition, the functions ζ and Γ verify the hypotheses $(H_1)-(H_3)$. Based on Theorems 3.1 and 3.2, we derive the following corollary.

Corollary 4.1.

(i): *Provided that $\tilde{\mathscr{R}}_0 \leq 1$, the EFE E_1 of model (14)-(16) is globally asymptotically stable.*

(ii): *If $\tilde{\mathscr{R}}_0 > 1$, then E_1 loses its stability and the equilibrium E_2 of model (14)-(16) is globally asymptotically stable.*

We choose $d_1 = 0.1$, $d_2 = 0.5$,and $d_Q = 0.001$. The other parameter values are $\beta = 50$, $m = 0.5$, $q_1 = 0.005$, $q_2 = 0.001$, $o_1 = 0.01$, $o_2 = 0.01$, $r = 0.06$, $\delta = 0.02$, and $e = 0.8$. Then $\tilde{\mathscr{R}}_0 = 0.8973$ and model (14) has one EFE $E_1(100,0,0)$ which is globally asymptotically stable. This means that EVD is eradicated from bats. This result is visualized in Figure 1.

Now, we verify the other case when $\tilde{\mathscr{R}}_0$ exceeds one. In this case, we replace the value of q_1 by 0.008 and get $\tilde{\mathscr{R}}_0 = 1.433 > 1$. Thus, model (14) has a unique endemic equilibrium $E_2(81.3567, 16.6458, 0.4161)$ and it is globally asymptotically stable. This simulation result is presented in Figure 2 and means that the EVD is still active in the bat population.

In addition, Figure 3 illustrates how $\tilde{\mathscr{R}}_0$ changes as q_1 varies from 0.005 to 0.008. Certainly, a $\tilde{\mathscr{R}}_0$ value greater than 1 implies that the infection will spread in the bat population, as each an infectious bat will, on average, infect more than one other bat. For q_1 values resulting in $\tilde{\mathscr{R}}_0$ values below 1, the infection will likely die out over time, as each an infectious bat will infect less than one other bat on average.

Figure 4 represents the contour plot of $\tilde{\mathscr{R}}_0$ with respect to q_1 and q_2 by keeping the same values of the other parameters. Warm colors (reds and yellows) represent higher values of $\tilde{\mathscr{R}}_0$. These regions indicate combinations of q_1 and q_2 where the infection spread is slower or less likely. The contour plot represents how the basic reproduction number $\tilde{\mathscr{R}}_0$ varies with changes in the

infection rates q_1 and q_2. This helps in understanding the dynamics of EVD spread between bats and planning effective control measures.

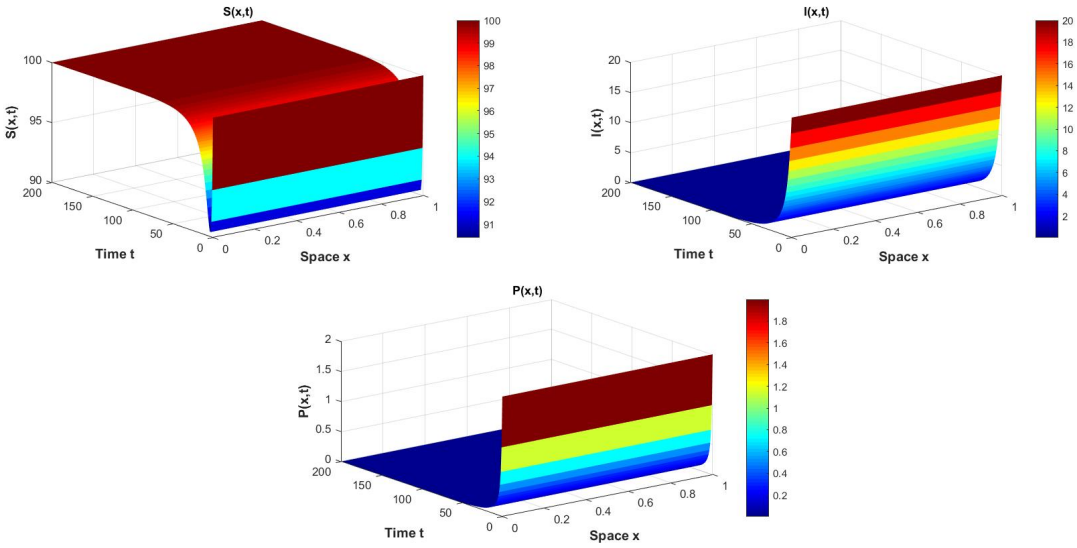

FIGURE 1. Visualizing the global behavior of the EFE E_1 of problem (14)-(16) in the case of $\tilde{\mathscr{R}}_0 = 0.8973 \leq 1$.

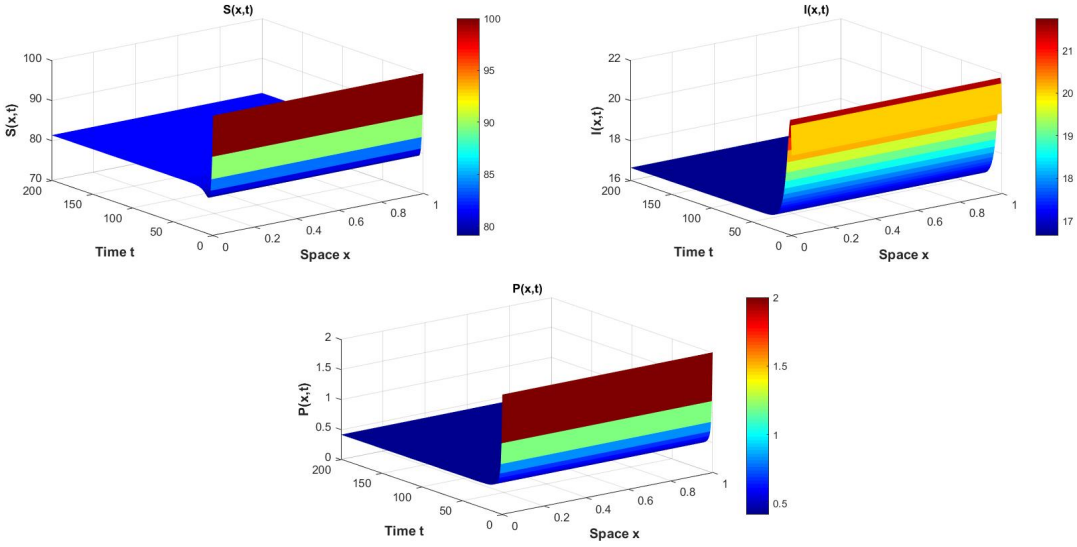

FIGURE 2. Visualizing the global behavior of the endemic EVD equilibrium E_2 of model (14)-(16) for $\tilde{\mathscr{R}}_0 = 1.433 > 1$.

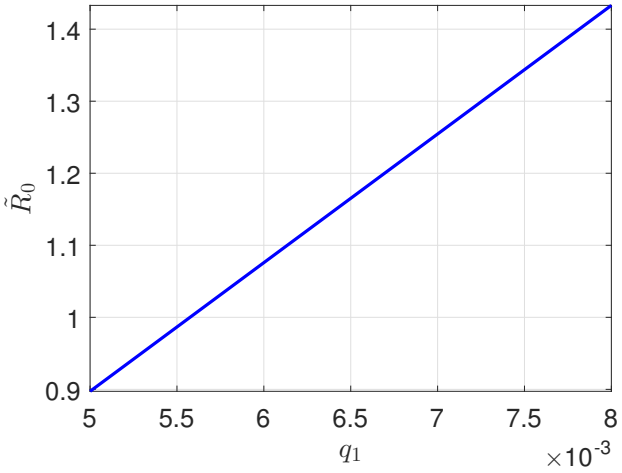

FIGURE 3. Variation of $\tilde{\mathscr{R}}_0$ with respect to q_1 for fixed parameters $q_2 = 0.001$, $\beta = 50$, $m = 0.5$, $r = 0.06$, $\delta = 0.02$, and $e = 0.8$.

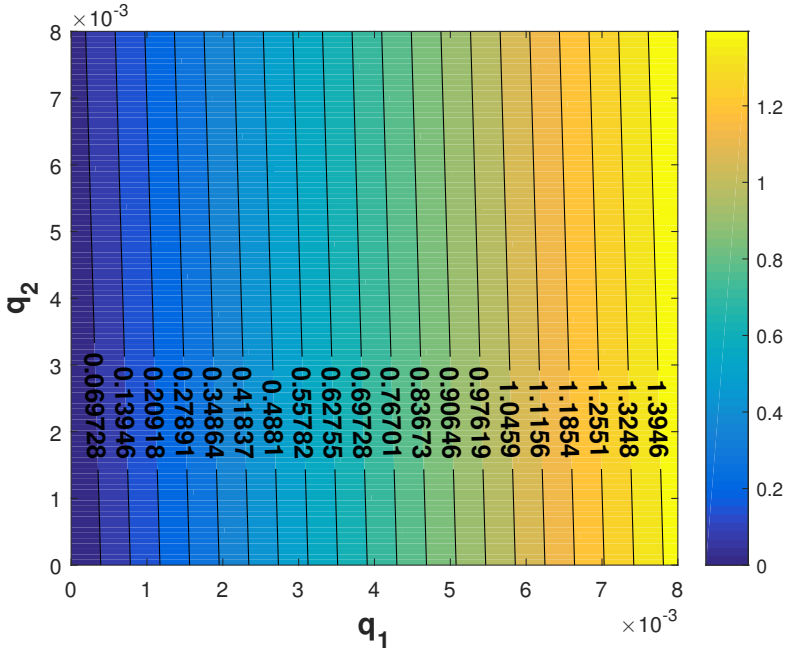

FIGURE 4. Contour Plot of $\tilde{\mathscr{R}}_0$ with respect to the infection rates q_1 and q_2 for $\beta = 50$, $m = 0.5$, $r = 0.06$, $\delta = 0.02$, and $e = 0.8$.

5. CONCLUSION

Mathematical modeling is a powerful tool in the fight against infectious diseases, offering a comprehensive framework for understanding, predicting, and controlling outbreaks. It bridges the gap between theoretical research and practical public health applications, ultimately contributing to better health outcomes worldwide. Due to their high mobility, bats can carry pathogens like the EV over large geographic areas. This can potentially introduce the virus to new regions and populations, increasing the risk of outbreaks. To account for the mobility of bats, we have proposed a generalized reaction-diffusion model governed by PDEs for the transmission of EVD within bat populations.

Bats become infected either directly through contact with other infectious bats or indirectly through a contaminated environment. This double transmission mechanism is described by two general incidence functions, encompassing various incidence rates found in the literature, such as the classical bilinear incidence and the Hattaf-Yousfi functional response.

From a theoretical standpoint, we investigate a meticulous qualitative analysis, including uniqueness, boundedness and positivity of solutions. In addition, we examine the stability behavior of equilibria.

Under certain conditions on these general incidence functions, it has been shown that the stability behavior of the proposed model is governed by the basic reproduction number $\mathscr{R}_0$. Specifically, if $\mathscr{R}_0 \leq 1$, the EFE is globally asymptotically stable, indicating that Ebola has been eradicated from the bat population. Conversely, if $\mathscr{R}_0 > 1$, the EFE becomes unstable, and the model exhibits an endemic equilibrium that is globally asymptotically stable, meaning the disease persists in the bat population. Moreover, Numerical simulations confirmed the existence of a unique positive global stable equilibrium for the PDEs problem in the two cases of $\mathscr{R}_0$ greater than or less than unity.

Compared to the results given in [12], we observe that spatial diffusion under Neumann boundary conditions and constant space coefficients has no impact on the behavior of equilibria stability.

Spatiotemporal modeling of Ebola in bats provides a comprehensive framework for understanding the complex interactions between bats, the virus, and the environment. It is a powerful tool for predicting and preventing Ebola outbreaks, guiding public health strategies, and fostering collaboration between ecologists, epidemiologists, and public health officials. It is also essential for understanding the virus's ecology, transmission dynamics, and potential for spillover to humans. This spillover event from bats to humans will be considered in our future work.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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