J. Math. Computer Sci., 39 (2025), 338–347

Online: ISSN 2008-949X

Journal of Mathematics and Computer Science

Journal Homepage: www.isr-publications.com/jmcs

Global analysis of mathematical model of infection by bacteriophages and bacteria with a mechanism of protection

Miller Cerón Gómez[a,*], Eduardo Ibarguen Mondragón[a], Eddy Patricia Lopez Molano[b]

[a]Research Group in Mathematical Biology and Applied Mathematics (GIBIMMA), University of Nariño, Nariño, Colombia.

[b]Departament of Biology, University of Nariño, Nariño, Colombia.

Abstract

We present a mathematical model of pathogenic bacteria and bacteriophages, incorporating an abortive infection as a mechanism of bacterial protection against viral infection. We identified three equilibria: the extinction equilibrium point, the bacteriophages extinction equilibrium, and the coexistence equilibrium. The stability of these equilibria is determined by thresholds. We found that the extinction equilibrium point is always unstable, while the bacteriophages extinction equilibrium is globally asymptotically stable. The stability of the coexistence equilibrium varies, being unstable, locally asymptotically stable, or globally asymptotically stable depending on certain thresholds. We conclude that bacterial extinction is not possible, possibly due to the inclusion of the abortive infection, but it is feasible to maintain a low level of pathogenic bacteria.

Keywords: Abortive infection, Dulac function, bacteria-virus model, bacteriophages.

2020 MSC: 34D23, 37N25, 92D25.

1. Introduction

Most bacteria are beneficial to humans, but there are some that are pathogenic and can cause serious damage to human health. Fortunately, to counteract this, there are antibiotics that can control the damage caused by these pathogenic bacteria. However, the indiscriminate use of antibiotics has led many bacteria to develop resistance, creating a significant problem in both health and economic impact worldwide. Currently, to address antibiotic resistance, bacteriophage therapies are being used. Bacteriophages (phages) are viruses that attack bacteria and lyse them. On the other hand, bacteria have defense mechanisms against phage infection. One of these mechanisms is Abortive Infection (Abi), a phage resistance strategy in which the infected cell commits suicide before the phage can complete its replication cycle. Abi prevents the phage epidemic from spreading to nearby cells, thus protecting the bacterial colony [1, 4, 12, 15].

In this paper, we formulate a model considering pathogenic bacteria with bacteriophages, incorporating the Abi effect, to determine the levels at which we could control the bacterial population or in what conditions bacteria might survive despite bacteriophage attacks. To do this, we denote the bacterial population by $B(t)$ and the bacteriophage population by $P(t)$. We assume that the bacteria grow following the

*Corresponding author
Email address: millercg@udenar.edu.co (Miller Cerón Gómez)

doi: 10.22436/jmcs.039.03.03

Received: 2024-05-27 *Revised:* 2024-11-30 *Accepted:* 2025-01-13

logistic growth equation, i.e., $kB\left(1-\frac{B}{N}\right)$, where k is the growth rate and N is the carrying capacity. The control of pathogenic bacteria occurs due to the action of bacteriophages at a rate $\alpha\frac{BP}{a+B}$, where α is the infection rate of bacteria by bacteriophages and a is the half-saturation bacteria density. Additionally, we consider that in some cases, when a bacterium is infected with a phage, it dies, and the infecting phage is lost; this is the protection mechanism by Abi and is included in the model as δBP.

For the bacteriophages, their growth occurs at a rate $\theta\alpha\frac{BP}{a+B}$, where θ is the burst size for infected bacteria, and they decay at a rate rP. Therefore, the two compartments are described dynamically by the following set of ordinary differential equations:

$$\frac{dB}{dt} = kB\left(1-\frac{B}{N}\right) - \alpha\frac{BP}{a+B} - \delta BP, \quad \frac{dP}{dt} = \theta\alpha\frac{BP}{a+B} - rP. \tag{1.1}$$

This is a simplistic model, but it captures many characteristics of the dynamics between bacteria and bacteriophages. There are models considering these interactions; for example, in [2], a mathematical model of interaction between bacteriophages and bacteria is presented, which includes temperature and pH as parameters affecting bacterial growth. In [10], the authors explore a mathematical model to evaluate the spread of antimicrobial resistance by phages. Many other models consider viral infection by phages and bacteria (see [3, 5, 6, 8, 9, 11, 16]). Our model includes the abortive infection of bacteria and considers bacterial infection as a predator-prey process with a saturation effect, which the others do not.

The organization of this paper is as follows. In Section 2, the local stability analysis of the extinction equilibrium point, bacteriophage extinction equilibrium, and coexistence equilibrium is proved. In Section 3, the global stability of equilibrium points is proved. In Section 4, numerical simulations were performed to corroborate our analytical results. In Section 5, we discuss our results.

2. Local stability analysis

The equilibrium points are: $E_0 = (0,0)$, $E_1 = (N,0)$, and $E_2 = (B^*, P^*)$, where $B^* = \frac{ra}{\theta\alpha - r}$ and $P^* = \frac{ka\theta}{(\delta a\theta + \theta\alpha - r)}\left(1-\frac{ra}{N(\theta\alpha - r)}\right)$. For the existence of E_2, we need $\theta\alpha > r$ and $Q_0 = \frac{N(\theta\alpha - r)}{ra} > 1$ (equivalently, $Q_0 = \frac{N}{B^*} > 1$ or $B^* < N$). We call these points E_0 the extinction equilibrium, E_1 the bacteriophage extinction equilibrium, and E_2 the coexistence equilibrium.

Since we are modeling microbiological species, we take the set $\Omega = \{(B,P) : P \geqslant 0, 0 \leqslant B \leqslant N\}$ as the set of biological interest (it is easy to show that this set is positively invariant with respect to the system (1.1)).

We are going to study the asymptotic stability of the system (1.1) at the extinction equilibrium $E_0 = (0,0)$, the bacteriophage extinction equilibrium $E_1 = (N,0)$, and the coexistence equilibrium point $E_2 = (B^*, P^*)$. The linearization of this system around an equilibrium E is given by $x' = J(E)x$, where $x = (B,P)^T$, and the matrix J is the Jacobian matrix of the system (1.1) evaluated at E, which is

$$J(E) = \begin{pmatrix} k - \frac{2kB}{N} - \frac{\alpha aP}{(a+B)^2} - \delta P & -\frac{\alpha B}{(a+B)} - \delta B \\ \frac{\theta\alpha aP}{(a+B)^2} & \frac{\theta\alpha B}{(a+B)} - r \end{pmatrix}.$$

It is easy to check that the eigenvalues of the Jacobian matrix evaluated at E_0 are k and $-r$. In this way, this point is always unstable. Now we check the point E_1, and the Jacobian matrix evaluated at this point is

$$J(E_1) = \begin{pmatrix} -k & -\frac{\alpha N}{(a+N)} - \delta N \\ 0 & N(\theta\alpha - r)\left(1-\frac{1}{Q_0}\right) \end{pmatrix}.$$

The eigenvalues are $-k$ and $N(\theta\alpha - r)\left(1-\frac{1}{Q_0}\right)$. The last eigenvalue can be negative, zero, or positive, leading to the following result.

Theorem 2.1. *The equilibrium E_1 is locally asymptotically stable if $Q_0 < 1$ and unstable if $Q_0 > 1$.*

Remark 2.2. Observe that when $Q_0 = 1$, the Jacobian matrix has a zero eigenvalue, so we cannot conclude the stability in this case. We will discuss this in the next section.

Theorem 2.3. *The coexistence equilibrium* E_2 *is*

1. *locally asymptotically stable if* $1 < Q_0 \leqslant 2$ *(equivalently* $\frac{N}{2} \leqslant B^* < N$*);*
2. *locally asymptotically stable if* $Q_0 > 2$ $(B^* < N/2)$ *and* $Q_1 = \frac{(\theta\alpha - r)^2(N\theta\alpha + ra)}{2ra\theta\alpha(\theta\alpha - r) + ra^2\alpha\theta^2\delta + N(\theta\alpha - r)^3} < 1$*;*
3. *unstable if* $Q_0 > 2$ $(B^* < N/2)$ *and* $Q_1 > 1$*;*
4. *a Hopf bifurcation exists if* $Q_0 > 2$ $(B^* < N/2)$ *and* $Q_1 = 1$.

Proof. The Jacobian matrix evaluated at E_2 is

$$J(E_2) = \begin{pmatrix} k - \frac{2kB^*}{N} - \frac{\alpha aP^*}{(a+B^*)^2} - \delta P^* & -\frac{\alpha B^*}{(a+B^*)} - \delta B^* \\ \frac{\theta\alpha aP^*}{(a+B^*)^2} & \frac{\theta\alpha B^*}{(a+B^*)} - r \end{pmatrix} = \begin{pmatrix} k - \frac{2kB^*}{N} - \frac{\alpha aP^*}{(a+B^*)^2} - \delta P^* & -\frac{\alpha B^*}{(a+B^*)} - \delta B^* \\ \frac{\theta\alpha aP^*}{(a+B^*)^2} & 0 \end{pmatrix},$$

the characteristic polynomial is $\det(J(E_2) - \lambda I) = \lambda^2 - \mathrm{Tr}(J(E_2))\lambda + \det J(E_2) = 0$, where $\mathrm{Tr}(J(E_2))$ is the trace of $J(E_2)$ and $\det J(E_2)$ is the determinant of $J(E_2)$. If $\mathrm{Tr}(J(E_2)) < 0$ and $\det J(E_2) > 0$, we will have that the roots of this polynomial have negative real part. In fact,

$$\det J(E_2) = \frac{\theta\alpha aP^*}{(a+B^*)^2}\left(\frac{\alpha B^*}{(a+B^*)} + \delta B^*\right)$$

is positive and

$$\mathrm{Tr}(J(E_2)) = k - \frac{2kB^*}{N} - \frac{\alpha aP^*}{(a+B^*)^2} - \delta P^* = k\left(1 - \frac{2B^*}{N}\right) - \frac{\alpha aP^*}{(a+B^*)^2} - \delta P^*. \tag{2.1}$$

Then, if the inequalities in item 1 are satisfied, we conclude that the trace is negative, and in this way, we check 1. For the other items, we proceed as follows: first, we assume that $B^* < N/2$, as this is the only scenario where the trace can be positive or zero. Second, we substitute the values of B^* and P^* in (2.1) to obtain:

$$\begin{aligned}
\mathrm{Tr}(J(E_2)) &= k\left(1 - \frac{2}{N}\frac{ra}{\theta\alpha - r}\right) - \frac{\alpha a}{(a + \frac{ra}{\theta\alpha - r})^2}\frac{ka\theta}{(\delta a\theta + \theta\alpha - r)}\left(1 - \frac{ra}{N(\theta\alpha - r)}\right) \\
&\quad - \delta\frac{ka\theta}{(\delta a\theta + \theta\alpha - r)}\left(1 - \frac{ra}{N(\theta\alpha - r)}\right) \\
&= k\left(1 - \frac{2}{N}\frac{ra}{\theta\alpha - r}\right) - \frac{k(\theta\alpha - r)^2}{\theta\alpha(\delta a\theta + \theta\alpha - r)}\left(1 - \frac{ra}{N(\theta\alpha - r)}\right) - \delta\frac{ka\theta}{(\delta a\theta + \theta\alpha - r)}\left(1 - \frac{ra}{N(\theta\alpha - r)}\right) \\
&= \frac{k}{N(\theta\alpha - r)\theta\alpha(\delta a\theta + \theta\alpha - r)}\{N(\theta\alpha - r)\theta\alpha(\delta a\theta + \theta\alpha - r) - 2ra\theta\alpha(\delta a\theta + \theta\alpha - r) \\
&\quad - \left[(\theta\alpha - r)^2 + \delta a\theta^2\alpha\right]\left[N(\theta\alpha - r) - ra\right]\} \\
&= \frac{k}{N(\theta\alpha - r)\theta\alpha(\delta a\theta + \theta\alpha - r)} \\
&\quad \times \left\{N(\theta\alpha - r)^2\theta\alpha - 2ra\theta\alpha(\theta\alpha - r) - ra^2\alpha\theta^2\delta - N(\theta\alpha - r)^3 + (\theta\alpha - r)^2 ra\right\} \\
&= \frac{k\left[2ra\theta\alpha(\theta\alpha - r) + ra^2\alpha\theta^2\delta + N(\theta\alpha - r)^3\right]}{N(\theta\alpha - r)\theta\alpha(\delta a\theta + \theta\alpha - r)}\left[\frac{(\theta\alpha - r)^2(N\theta\alpha + ra)}{2ra\theta\alpha(\theta\alpha - r) + ra^2\alpha\theta^2\delta + N(\theta\alpha - r)^3} - 1\right] \\
&= \frac{k\left[2ra\theta\alpha(\theta\alpha - r) + ra^2\alpha\theta^2\delta + N(\theta\alpha - r)^3\right]}{N(\theta\alpha - r)\theta\alpha(\delta a\theta + \theta\alpha - r)}(Q_1 - 1).
\end{aligned}$$

From this we have $\mathrm{Tr}(J(E_2)) < 0$ if $Q_1 < 1$, $\mathrm{Tr}(J(E_2)) > 0$ if $Q_1 > 1$, and $\mathrm{Tr}(J(E_2)) = 0$ if $Q_1 = 1$, from which the items 2, 3, and 4 will follow. □

3. Global stability analysis

We are going to prove that the bacteriophage extinction equilibrium E_1 and coexistence equilibrium E_2 are globally asymptotically stable.

Theorem 3.1. *If $Q_0 \leqslant 1$, the equilibrium E_1 is globally asymptotically stable.*

Proof. $P \geqslant 0$ and $0 < B \leqslant N$. According to the first equation of (1.1), we have

$$\frac{dB}{dt} < kB\left(1-\frac{B}{N}\right) \implies B < \frac{N}{e^{-kt}+1} \implies \limsup_{t\to\infty} B \leqslant N.$$

We start analyzing the case when $Q_0 = 1$, i.e., $N(\theta\alpha - r) = ra$. According to the second equation of (1.1), we get

$$\begin{aligned}\frac{dP}{dt} = \frac{P}{a+B}(\theta\alpha B - ra - rB) &= \frac{P}{a+B}(B(\theta\alpha - r) - ra)\\ &= \frac{P}{a+B}(B(\theta\alpha - r) - N(\theta\alpha - r)) = \frac{P}{a+B}[(B-N)(\theta\alpha - r)].\end{aligned}$$

Then for $P \geqslant 0$ and $0 < B \leqslant N$, we have $\frac{dP}{dt} \leqslant 0$, therefore, $P(t) \to 0$ as $t \to \infty$. Now, we consider the case $Q_0 < 1$. From the second equation of (1.1), we have

$$\begin{aligned}\frac{dP}{dt} = \frac{P}{a+B}(\theta\alpha B - ra - rB) &< \frac{P}{a}(\theta\alpha B - ra - rB)\\ &= \frac{P}{a}(B(\theta\alpha - r) - ra)\\ &< rP\left(\frac{N(\theta\alpha - r)}{ra} - 1\right) \implies P < c_2 e^{r\left(\frac{N(\theta\alpha-r)}{ra}-1\right)t} = c_2 e^{r(Q_0-1)t}.\end{aligned}$$

Since $Q_0 < 1$ and $P \geqslant 0$, then $\lim_{t\to\infty} P = 0$. Therefore, for all $\epsilon \in (0,1)$, there exists T such that if $t \geqslant T$, then $0 < P < \epsilon$. According to the first equation of (1.1), we get:

$$\frac{dB}{dt} > kB\left(1-\frac{B}{N}\right) - \frac{\alpha}{a}B\epsilon - \delta B\epsilon \implies B > \frac{N(k - \frac{\alpha\epsilon}{a} - \delta\epsilon)}{e^{-(k-\frac{\alpha\epsilon}{a}-\delta\epsilon)t}+1} \implies \liminf_{t\to\infty} B \geqslant N.$$

Therefore, $\lim_{t\to\infty} B = N$. In other words, the equilibrium E_1 is globally asymptotically stable if $Q_0 \leqslant 1$. □

Theorem 3.2. *The set Ω_1 defined by*

$$\Omega_1 = \left\{(B,P) : \frac{N}{2} \leqslant B < N, P > 0\right\}$$

is positively invariant with respect to the solutions of the system (1.1), when $1 < Q_0 \leqslant 2$ (see Figure 1).

Proof. We start with $P > 0$, $B = N$. Then, in the equations of system (1.1), we have:

$$\begin{aligned}\frac{dB}{dt} &= -\alpha\frac{NP}{a+N} - \delta NP < 0,\\ \frac{dP}{dt} &= \theta\alpha\frac{NP}{a+N} - rP = P\left(\frac{N(\theta\alpha - r) - ra}{a+N}\right) = raP\left(\frac{Q_0 - 1}{a+N}\right) > 0.\end{aligned}$$

This means that any solution with initial conditions $P > 0$, $B = N$, tends to move toward the set Ω_1. Now, we consider $P > 0$, $B = \frac{N}{2}$, and we will prove that all the solutions get inside the set Ω_1. In fact,

$$\frac{dP}{dt} = \theta\alpha\frac{\frac{N}{2}P}{a+\frac{N}{2}} - rP = P\left(\frac{\frac{N}{2}(\theta\alpha - r) - ra}{a+\frac{N}{2}}\right) = raP\left(\frac{\frac{Q_0}{2} - 1}{a+\frac{N}{2}}\right) \implies P = e^{ra\left(\frac{\frac{Q_0}{2}-1}{a+\frac{N}{2}}\right)t} > 0,$$

which shows that P decays exponentially provided $1 < Q_0 \leqslant 2$. Therefore, $P(t) > 0$. From this, we have that there exists $\epsilon > 0$ such that $0 < P < \epsilon$ and

$$\frac{dB}{dt} = kB\left(1-\frac{B}{N}\right) - \alpha\frac{BP}{a+B} - \delta BP > kB\left(1-\frac{B}{N}\right) - \alpha\frac{B\epsilon}{a+B} - \delta B\epsilon > 0.$$

This means that starting at $B = \frac{N}{2}$, the function B starts to increase, i.e., the solution of the system enters inside of Ω_1. Now we take a point $P > 0$ and $\frac{N}{2} < B < N$, and we have

$$\frac{P}{a+\frac{N}{2}}\left(\frac{N}{2}(\theta\alpha - r) - ra\right) < \frac{dP}{dt} = \frac{P}{a+B}\left(B(\theta\alpha - r) - ra\right) < \frac{P}{a+N}\left(N(\theta\alpha - r) - ra\right).$$

This implies that

$$0 < c_1 e^{\frac{t}{a+\frac{N}{2}}\left(\frac{N}{2}(\theta\alpha - r) - ra\right)} < P < c_2 e^{\frac{t}{a+N}(N(\theta\alpha - r) - ra)} \quad \text{or} \quad 0 < c_1 e^{\frac{rat}{a+\frac{N}{2}}\left(\frac{Q_0}{2} - 1\right)} < P < c_2 e^{\frac{rat}{a+N}(Q_0 - 1)}.$$

Using a similar argument as before, we have that there exists $\upsilon > 0$ such that

$$kB\left(1-\frac{B}{N}\right) - \alpha\frac{B\upsilon}{a+B} - \delta B\upsilon < \frac{dB}{dt} < kB\left(1-\frac{B}{N}\right),$$

which implies that $\frac{N}{2} < B < N$. This means that any initial condition starting with $P > 0$ and $\frac{N}{2} < B < N$ will stay in Ω_1. In this way, the set Ω_1 is positively invariant. □

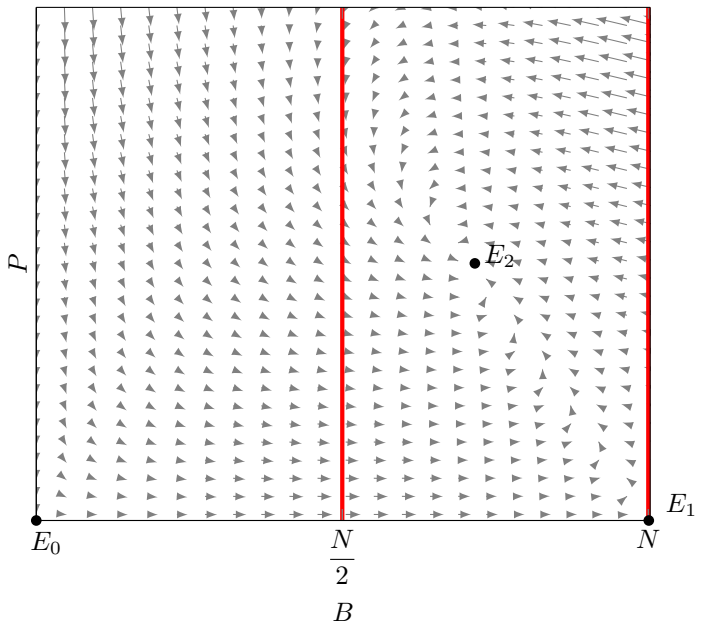

Figure 1: The figure illustrates the set Ω_1 and phase plane when $1 < Q_0 \leqslant 2$.

Theorem 3.3. *If* $1 < Q_0 \leqslant 2$ *(equivalently* $\frac{N}{2} \leqslant B^* < N$*), the coexistence equilibrium* E_2 *is globally asymptotically stable.*

Proof. We are going to show that this system has no periodic orbits in the region Ω_1. Denote the right-hand side of (1.1) by $(Q(B,P), R(B,P))$, i.e.,

$$Q(B,P) = kB\left(1-\frac{B}{N}\right) - \alpha\frac{BP}{a+B} - \delta BP, \quad R(B,P) = \theta\alpha\frac{BP}{a+B} - rP.$$

We will show that the function $\phi(B,P)=\frac{1}{P}$ is a Dulac function by showing that $\frac{\partial}{dB}\left(\frac{Q}{P}\right)+\frac{\partial}{dP}\left(\frac{R}{P}\right)<0$ for all $B>0$ and $P>0$ in Ω_1. In fact,

$$\frac{\partial}{dB}\left(\frac{Q}{P}\right)+\frac{\partial}{dP}\left(\frac{R}{P}\right)=\frac{k}{P}\left(1-\frac{2B}{N}\right)-\frac{\alpha a}{(a+B)^2}-\delta.$$

By the condition $B\geqslant\frac{N}{2}$, we have

$$\frac{\partial}{dB}\left(\frac{Q}{P}\right)+\frac{\partial}{dP}\left(\frac{R}{P}\right)<0,$$

which is negative for all $B>0$ and $P>0$ in Ω_1. Thus, the system (1.1) does not have periodic orbits in the set Ω_1. Since the equilibria E_0 and E_1 are unstable, E_2 becomes the only stable attractor of the system when $1<Q_0\leqslant 2$, i.e., the interior of Ω_1 becomes the basin of attraction of E_2. Consequently, E_2 is globally asymptotically stable. □

A summary of the dynamics is presented in the next image.

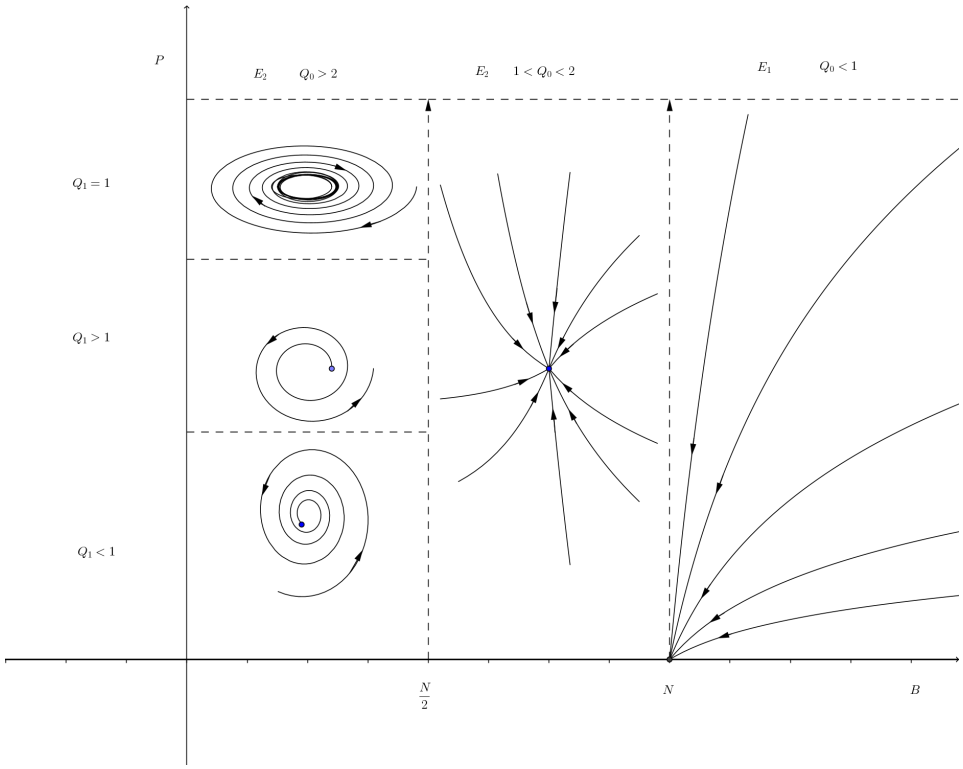

Figure 2: The dynamics of the equilibrium points E_1 and E_2

4. Numerical simulations

For the numerical simulations, we used the parameters listed in Table 1. In Figure 3, the global asymptotic stability when $Q_0\leqslant 1$ is shown. The interpretation of this result is that the Abi protection mechanism was successful against bacteriophage infection [7, 14]. In Figure 4, the global asymptotic stability when $1<Q_0\leqslant 2$ is shown. Note that in this case, the condition $1<Q_0\leqslant 2$ implies $\frac{N}{2}\leqslant B^*<N$. If B^* is very close to N, P^* will be approximately zero due to its definition, which is a similar situation to the previous one, i.e., the Abi protection mechanism was again successful. In Figure 5, the local stability is shown when $Q_0>2$ and $Q_1<1$. Observe that this implies $B^*<\frac{N}{2}$, and by the equation of P^*, if B^* is very close to zero, P^* is close to its maximum value $\frac{k a\theta}{\delta a\theta+\theta\alpha-r}$. This is the best scenario because the level of bacteria can be kept below a threshold where it is no longer harmful [9]. Meanwhile, in Figures 6 and

7, we show the instability of point E_2, which indicates that we cannot predict the concentration of bacteria and phages [8, 13].

Table 1: Model parameters.

Parameter	Definition	Value
k	Growth rate for bacteria	0.056
N	Carrying capacity	3.2
α	α is infection rate of bacteria by bacteriophages	1.002* 0.18♦ 0.518★ 0.8♠♣
a	Half-saturation bacteria density	10.63*♦★ 0.63♠ 2.63♣
r	Decay rate of bacteriophages	0.03*♦★ 0.2♠ 0.001♣
δ	Abortive infection rate	0.01* 0.05♦★ 0.00006♠ 0.055775♣
θ	Burst size for infected bacteria	0.1* 0.8♦★♠ 0.09♣

* For E_1
For E_2 in Theorem 2.3: Item 1 ♦, Item 2 ★, Item 3 ♠, Item 4 ♣

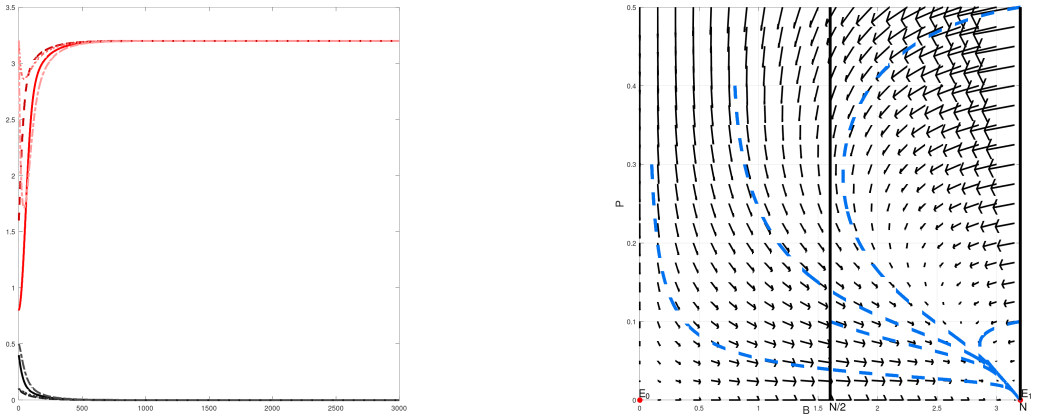

Figure 3: Dynamics of phage extinction equilibrium E_1 when ($Q_0 \leqslant 1$). In the left figure, the red lines correspond to E. Coli, and the black tones correspond to phages.

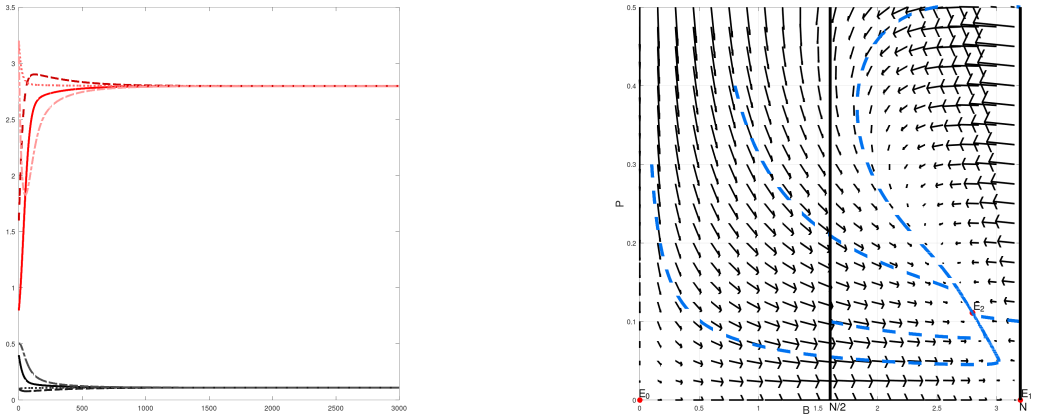

Figure 4: Stability of the coexistence equilibrium when $1 < Q_0 \leqslant 2$ (Case 1 from Theorem 2.3 and Theorem 3.3). In the left figure, the red lines correspond to E. Coli, and the black tones correspond to phages.

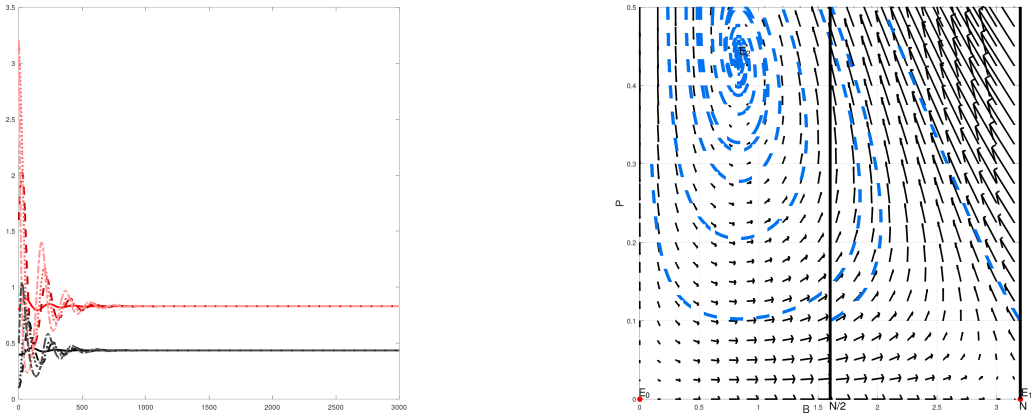

Figure 5: Stability of the coexistence equilibrium when $Q_0 > 2$ and $Q_1 < 1$ (Case 2 from Theorem 2.3). In the left figure, the red lines correspond to E. Coli, and the black tones correspond to phages.

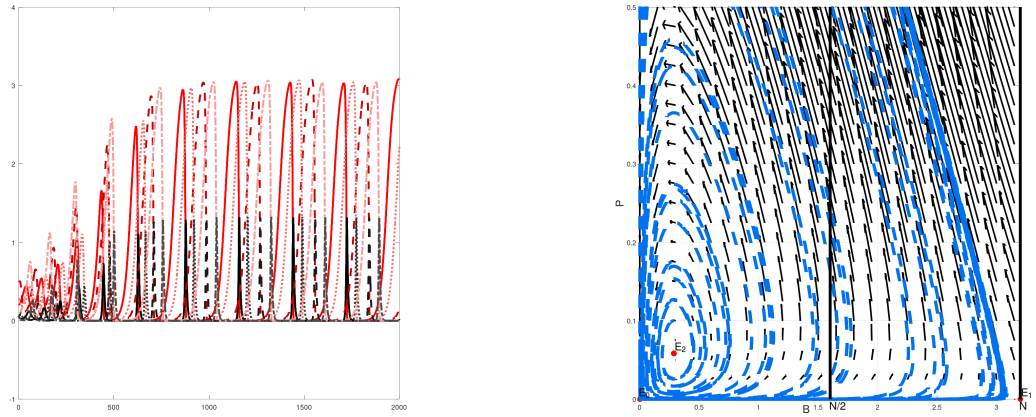

Figure 6: Instability of the coexistence equilibrium E_2 when $Q_0 > 2$ and $Q_1 > 1$ (Case 3 from Theorem 2.3). In the left figure, the red lines correspond to E. Coli, and the black tones correspond to phages.

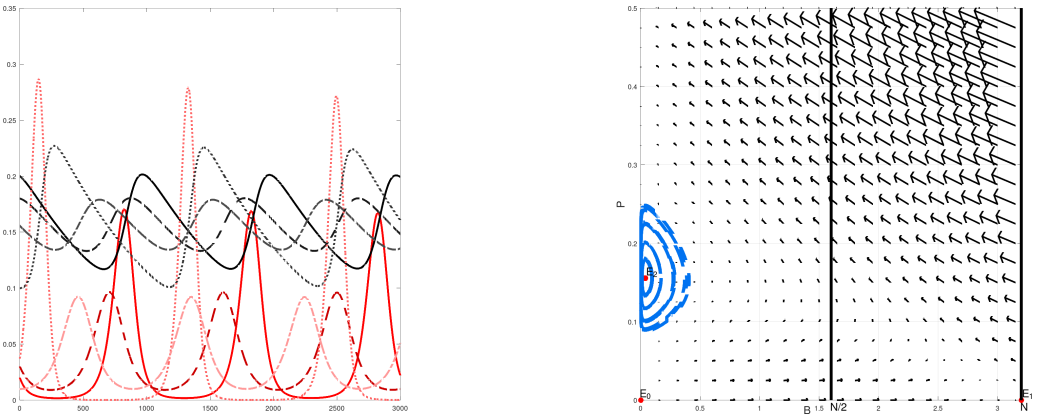

Figure 7: Instability of the coexistence equilibrium when $Q_0 > 2$ and $Q_1 = 1$. We have a limit cycle when $Q_1 = 1$ (Case 4 from Theorem 2.3). In the left figure, the red lines correspond to E. Coli, and the black tones correspond to phages.

5. Discussion

In the realm of epidemiology or virus-host models, the condition $Q_0 < 1$ denotes the potential for disease elimination or the elimination of the causative virus. However, in mathematical models concerning phage-pathogen bacteria interactions, this scenario is deemed unfavorable, as the primary objective is the control or suppression of pathogenic bacteria. Within our model, the equilibrium of phage extinction is globally asymptotically stable under the condition $Q_0 \leqslant 1$. This observation indicates that, irrespective of the initial viral load of phages, bacteria demonstrate the capacity to eradicate them through abortive mechanisms or alternative pathways [14]. Consequently, this circumstance amplifies the probability of bacterial resistance against such phages. To address this issue effectively, the exploration of diverse phages exhibiting heightened lytic potential becomes imperative for control purposes.

When the condition $Q_0 > 1$ is met, the existence of a coexistence equilibrium is observed. This equilibrium can be globally asymptotically stable under the condition $1 < Q_0 \leqslant 2$, or locally asymptotically stable under the condition $Q_0 > 2$ and $Q_1 < 1$, and unstable for $Q_0 > 2$ and $Q_1 > 1$ or $Q_0 = 1$. Consequently, within the framework of our model, complete eradication of the pathogenic bacteria is not attainable; rather, it is feasible to maintain its presence at a level where its potential danger is mitigated.

In conclusion, our model analysis indicates that the dynamics of phage-pathogen bacteria interactions are not markedly influenced by the initial viral load, but rather by the thresholds Q_0 and Q_1. The potential for virus elimination or the control of pathogenic bacteria is determined by these parameters, with $Q_0 < 1$ denoting the potential for phage elimination and $Q_0 > 1$ indicating the coexistence of phages and bacteria. The worst scenarios are when $Q_0 \leqslant 1$ and $1 < Q_0 \leqslant 2$, because the bacteria are very close to or equal to the maximum level N, while the phages are near extinction or zero, which means that the abortive effect was successful [7, 14]. The best scenario is when $Q_0 > 2$ and $Q_1 < 1$, because we can have a very low amount of bacteria, where it is no longer harmful [9]. On the other hand, when $Q_0 > 2$ and $Q_1 > 1$, or $Q_0 = 1$, we can have an outbreak of bacteria or a periodic occurrence of bacteria [8, 13].

It is evident that the need for exploring and utilizing phages with heightened lytic potential is crucial for the effective control and suppression of pathogenic bacteria. Our model highlights the complexity of these interactions and emphasizes the importance of considering various scenarios to devise strategies for mitigating potential dangers posed by pathogenic bacteria. However, further refinement and integration of empirical data are necessary to enhance the model's predictive capabilities and inform the development of effective phage-based control strategies against antibiotic-resistant bacteria.

Acknowledgment

Miller Cerón Gómez, Eduardo Ibarguen mondragón, and Eddy Patricia Lopez Molano thank financial support, project No. 2817/10/2023 from Vicerrectoria de Investigación e interacción social (Vipri-Udenar).

References

[1] N. Aframian, A. Eldar, *Abortive infection antiphage defense systems: separating mechanism and phenotype*, Trends Microbiol., **31** (2023), 1003–1012. 1

[2] G. Beke, M. Stano, L. Klucar, *Modelling the interaction between bacteriophages and their bacterial hosts*, Math. Biosci., **279** (2016), 27–32. 1

[3] B. Boldin, *The importance of ecological dynamics in evolutionary processes: A host-bacteriophage model revisited*, J. Theoret. Biol., **539** (2022), 10 pages. 1

[4] J. E. Egido, A. R. Costa, C. Aparicio-Maldonado, P. J. Haas, S. J. J. Brouns, *Mechanisms and clinical importance of bacteriophage resistance*, FEMS Microbiol. Rev., **46** (2022), 16 pages. 1

[5] H. I. Egilmez, A. Y. Morozov, E. E. Galyov, *Modelling the spatiotemporal complexity of interactions between pathogenic bacteria and a phage with a temperature-dependent life cycle switch*, Sci. Rep., **11** (2021), 13 pages. 1

[6] M. C. Gómez, E. Ibarguen Mondragon, E. L. Molano, A. Hidalgo-Troya, M. A. Mármol-Martínez, D. L. Guerrero-Ceballos, M. A. Pantoja, C. Paz-García, J. Gómez-Arrieta, M. Burbano-Rosero, *Mathematical model of interaction Escherichia coli and Coliphages*, Math. Biosci. Eng., **20** (2023), 9712–9727. 1

[7] M. Hasan, J. Ahn, *Evolutionary dynamics between phages and bacteria as a possible approach for designing effective phage therapies against antibiotic-resistant bacteria*, Antibiotics, **11** (2022), 20 pages. 4, 5

[8] E. E. Kyaw , H. Zheng, J. Wang, *Stability and hopf bifurcation analysis for a phage therapy model with and without time delay*, Axioms, **12** (2023), 27 pages. 1, 4, 5
[9] Q. J. Leclerc, J. A. Lindsay, G. M. Knight, *Modelling the synergistic effect of bacteriophage and antibiotics on bacteria: Killers and drivers of resistance evolution*, PLoS Comput. Biol., **18** (2022), 26 pages. 1, 4, 5
[10] Q. J. Leclerc, J. Wildfire, A. Gupta, J. A. Lindsay, G. M. Knight, *Growth-dependent predation and generalized transduction of antimicrobial resistance by bacteriophage*, Msystems, **7** (2022), 22 pages. 1
[11] X. Li, R. Huang, M. He, *Dynamics model analysis of bacteriophage infection of bacteria*, Adv. Differ. Equ. **2021** (2021), 11 pages. 1
[12] A. Millman, S. Melamed, A. Leavitt, S. Doron, A. Bernheim, J. Ho¨ r, J. Garb, N. Bechon, A. Brandis, A. Lopatina, G. Ofir, D. Hochhauser, A. Stokar-Avihail, N. Tal, S. Sharir, M. Voichek, Z. Erez, J. L. M. Ferrer, D. Dar, A. Kacen, G. Amitai, R. Sorek, *An expanded arsenal of immune systems that protect bacteria from phages*, Cell Host & Microbe **30** (2022), 1556–1569. 1
[13] H. M. Ndongmo Teytsa, B. Tsanou, S. Bowong, J. M.-S. Lubuma, *Bifurcation analysis of a phage-bacteria interaction model with prophage induction*, Math. Med. Biol., **38** (2021), 28–58. 4, 5
[14] E. Rodríguez-Román, J. A. Manuel, D. Goldberg, B. R. Levin, *The contribution of abortive infection to preventing populations of Lactococcus lactis from succumbing to infections with bacteriophage*, PLoS ONE, **19** (2024), 18 pages. 4, 5
[15] A. D. Teklemariam, R. R. Al-Hindi, I. Qadri, M. G. Alharbi, W. S. Ramadan, J. Ayubu, A. M. Al-Hejin, R. F. Hakim, F. F. Hakim, R. F. Hakim, L. I. Alseraihi, T. Alamri, S. Harakeh, *The battle between bacteria and bacteriophages: a conundrum to their immune system*, Antibiotics, **12** (2023), 17 pages. 1
[16] V. V. Volkova, Z. Lu, T. Besser, Y. T. Gröhn, *Modeling the infection dynamics of bacteriophages in enteric escherichia coli: estimating the contribution of transduction to antimicrobial gene spread*, Appl. Environ. Microbiol., **80** (2014), 4350–4362. 1