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ANALYSIS OF A CHEMOSTAT IN THE PRESENCE OF VIRAL INFECTION EXPRESSED BY A GENERAL INFECTION RATE

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Abstract. Viral are present greater abundance than bacteria in marine and lake environments. In order to analyze the viral infection on the dynamics of the bioprocess, the paper deals with the problem of a substrate-biomass interaction chemostat model where biomass is divided into uninfected and infected groups. The general nutrient uptake functions and infection rate function are taken into account. Conditions for infected biomass extinction are derived by taking the dilution rate as a control parameter The results show that when the dilution rate is kept in a proper level, the uninfected biomass can be sustained while the infected biomass is eliminated. It is also observed that the three component system may persist for a certain range of dilution rate.

Keywords: Chemostat; Uninfected biomass; Infected biomass; Coexistence; Stability

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1. Introduction

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Viruses are evidently the most abundant entities in marine and lake environments, which have significant impact on the aquatic populations [1, 2]. In a letter to Nature, Bergh et al. stated that "... virus infection may be an important factor in the ecological control of planktonic microorganisms ..." and indicated that studying the role of viruses in aquatic environments should not be neglected [3]. Quite a good number of researches focus on studying the role of viral infection in the population [4, 5, 6]. Among these studies, Beltrami and Carroll [4] proposed and analyzed a predator-prey system in which some of the susceptible phytoplankton cells were infected by viral contamination and formed an infected group. Chattopadhyay and Pal [5] modified the model equations of Beltrami and Carroll and observed that there is a possibility for the coexistence of the system when the contact rate follows the law of mass action rate. But if the contact rate follows the law of standard incidence rate then only a minute amount of infection can destabilize the system. And followed, Chattopadhyay et al [6] introduced the virus infection in phytoplankton populations and analyzed Nutrient-phytoplankton model. Different threshold values of spread of infection have been calculated. The results also show that if both the susceptible and infected phytoplankton consume nutrient, then the coexistence of both uninfected and infected population is not feasible.

The chemostat is a laboratory apparatus used for the continuous culture of microorganisms, which has the advantage that certain of the biological parameters presumed to influence competitive outcome can be controlled by the experimenter. The basic chemostat model predicts that coexistence of two or more microbial populations competing for a single non-reproducing nutrient is not possible [7, 8, 9]. To answer the question "Can the incorporation of a virus induce the stable coexistence of bacterial competitors in a chemostat-like environment?", Northcott *et al.* [10] derived a model of competition between two populations of bacteria for a single limiting nutrient in a chemostat where a virus is present. It was proved that the persistence of both the uninfected and the resistant populations (provided the disease is endemic) can either be in the form of convergence to an asymptotically stable steady state or in the form of sustained oscillatory behavior. Other researchers' studies that consider the effect of a virus on competing species in a basic chemostat include Mestivier *et al.* [11] and Weitz *et al.* [12]. In Mestivier *et al.* [11] simulations show that coexistence between two bacterial populations can be induced

by the addition of a virulent virus. In Weitz *et al.* [12] it was shown that for a reasonable choice of parameters, the system possesses a coexistence steady state.

The current research aims at providing a further analysis of the viral infection on the aquatic population and the coexistence of uninfected and infected population in a chemostat. The general nutrient uptake functions and infection rate function are taken into account. In Section 2, a mathematical model consisting of concentration of substrate, uninfected biomass and infected biomass with substrate uptake rate and infection rate as general continuous functional form is proposed. In Section 3, the boundedness, local stability and persistence of the system are studied. The analysis leads to different thresholds, which are expressible in terms of the model parameters and determine the existence and stability of various equilibrium states of the system. In Section 4, an applied instance and numerical simulations are provided to verify the theoretical results. Section 5 contains the general conclusion of the paper.

2. Model formulation

Let $S(t)$ be the substrate concentration in the bioreactor medium at time t. Let $X_S(t)$ and $X_I(t)$ be the uninfected biomass concentration and infected biomass concentration, respectively at time t. Let S_F be the constant input of limiting substrate concentration; D, the dilution rate; δ_{X_S}, mortality rate of uninfected biomass and δ_{X_I}, mortality rate of infected biomass, where $\delta_{X_S} \leq \delta_{X_I}$. The maximum specific growth rate for the uninfected biomass and infected biomass is denoted by $\mu_{\max}^{X_S}$ and $\nu_{\max}^{X_I}$, respectively. The biomass yield coefficient for the uninfected biomass and infected biomass is denoted by $Y_{X_S/S}$ and $Y_{X_I/S}$, respectively, and it is assumed that the uninfected biomass and infected biomass have the same biomass yield constant, i.e. $Y_{X_S/S} = Y_{X_I/S} = 1/\gamma$. The capability for the infected biomass to consume the substrate is denoted by κ ($0 \leq \kappa \leq 1$), where $\kappa = 1$ represents the full capability and $\kappa = 0$ represents that the infected biomass is incapable of substrate consumption. The mathematical model can be formulated as

$$
\left\{
\begin{array}{l}
\dfrac{\mathrm{d}S}{\mathrm{d}t} = D(S_F - S) - \gamma\mu(S)X_S - \kappa\gamma\nu(S)X_I, \\
\dfrac{\mathrm{d}X_S}{\mathrm{d}t} = \mu(S)X_S - D_{X_S}X_S - \alpha(X_S, X_I), \\
\dfrac{\mathrm{d}X_I}{\mathrm{d}t} = \kappa\nu(S)X_I + \alpha(X_S, X_I) - D_{X_I}X_I,
\end{array}
\right. \tag{1}
$$

with initial conditions

$$S(0) \geq 0, X_S(0) \geq 0, X_I(0) \geq 0,$$

where $D_{X_S} := D + \delta_{X_S}$ and $D_{X_I} := D + \delta_{X_I}$. The functions $\mu(S)$ and $\nu(S)$ describe the substrate uptake rates of the uninfected and infected biomass, respectively; $\alpha(X_S, X_I)$ represents the infection rate between the uninfected and infected biomass. We assume the following hypotheses on the substrate uptake functions and infection function:

H1) $\mu(S)$ and $\nu(S)$ are continuously differentiable, increasing on $[0,\infty)$, and $\alpha(X_S, X_I)$ is continuously differentiable on $[0,\infty) \times [0,\infty)$;

H2) $\mu(0) = 0$, $\mathrm{d}\mu(S)/\mathrm{d}t > 0$, and $\lim_{S\to\infty} \mu(S) = \mu_{\max}^{X_S}$;

H3) $\nu(0) = 0$, $\mathrm{d}\nu(S)/\mathrm{d}t > 0$, and $\lim_{S\to\infty} \nu(S) = \nu_{\max}^{X_I}$;

H4) $\mu(S) \geq \nu(S)$ for all $S \geq 0$.

H5) $X_S X_I = 0 \Rightarrow \alpha(X_S, X_I) = 0$; $X_S X_I > 0 \Rightarrow 0 < \alpha_u(X_S, X_I) \leq (\alpha/u)(X_S, X_I) < 1$, $u = X_S, X_I$.

3. Dynamics analysis

Firstly, it is shown the boundedness of the system (1).

Theorem 3.1. *System (1) has a unique and bounded solution with initial value* $(S(0), X_S(0), X_I(0)) \in \Gamma_0 := \{(S, X_S, X_I) \in R_+^3 : S \leq S_F\}$. *Moreover, the compact set*

$$\Gamma_1 := \{(S, X_S, X_I) \in \Omega : S \leq S_F,\ S + \gamma X_S + \gamma X_I \leq S_F + \varepsilon, \forall \varepsilon > 0\} \tag{2}$$

attracts all positive solutions in Γ_0.

Proof. Define a new variable $\Sigma := S + \gamma X_S + \gamma X_I$. Taking the time derivative of Σ along the solutions of system (1) yields that

$$\begin{aligned} \frac{\mathrm{d}\Sigma}{\mathrm{d}t} &= \frac{\mathrm{d}S}{\mathrm{d}t} + \gamma\frac{\mathrm{d}X_S}{\mathrm{d}t} + \gamma\frac{\mathrm{d}X_I}{\mathrm{d}t} \\ &= D(S_F - S) - D_{X_S}\gamma X_S - D_{X_I}\gamma X_I \\ &\leq DS_F - D\Sigma, \end{aligned}$$

which implies that

$$\Sigma(t) \leq S_F - [S_F - \Sigma(0)]e^{-Dt} = S_F(1 - e^{-Dt}) + \Sigma(0)e^{-Dt}. \tag{3}$$

From Eq. (3) we get that

$$\Sigma = S + \gamma X_S + \gamma X_I \leq \max\{\Sigma(0), S_F\}, \forall t \geq 0$$

and for $t \to \infty$, there is $Sup(N + \gamma X_S + \gamma X_I) \leq S_F$. Hence all the solutions of system (1) which initiate in Γ_0 are eventually confined in the region Γ. This completes the proof.

Lemma 3.2. *If $D_{MID} < 1$ and $D > D_{MID}$ hold, where*

$$D_{MID} := \max_{S+\gamma X_S+\gamma X_I \leq S_F} \{\kappa\nu(S) + (\alpha/X_I)(X_S, X_I)\} - \delta_{X_I},$$

then there is $X_I \to 0$ as $t \to \infty$.

Lemma 3.3. *If $D > D_{MAX}$, then there is $X_S \to 0$ as $t \to \infty$.*

3.1. Infected biomass extinction dynamics

It can be observed that if $X_I(0) = 0$, then $X_I(t) = 0$ for all $t \geq 0$. Thus, the plane $S - X_S$ is an invariant set of the system (1). In this case, system (1) is reduced to

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = D(S_F - S) - \gamma\mu(S)X_S, \\ \dfrac{\mathrm{d}X_S}{\mathrm{d}t} = \mu(S)X_S - D_{X_S}X_S. \end{cases} \tag{4}$$

Define $D_{MAX} := \mu(S_F) - \delta_{X_S}$. Then the following result for the system (4) holds.

Theorem 3.4. *If $D > D_{MAX}$, then the biomass extinction equilibrium $\widetilde{E_0}(S_F, 0)$ of the system (4) is global asymptotically stable; if $0 < D < D_{MAX}$, then $\widetilde{E_0}(S_F, 0)$ is unstable and the positive equilibrium $\widetilde{E_1}(S_1, X_S^1)$ exists and is global asymptotically stable in the $S - X_S$ plane, where $0 < S_1 = \mu^{-1}(D_{X_S}) < S_F$ and $0 < X_S^1 = D(S_F - S_1)/\gamma D_{X_S}$.*

Proof. The biomass extinction equilibrium $\widetilde{E_0}(S_F, 0)$ is locally stable since the eigenvalues of the variational matrix of subsystem (4) are negative if $D > D_{MAX}$. In addition, for any $X_S(0) > 0$, there is $X_S \leq X_S(0)e^{(D_{MAX}-D)t} \to 0$ when $D > D_{MAX}$, which implies that $\widetilde{E_0}(S_F, 0)$ is global asymptotically stable. When $0 < D < D_{MAX}$, there exists a unique $0 < S_1 < S_F$ such that $\mu(S^1) = D_{X_S}$. In this case, the positive equilibrium $\widetilde{E_1}(S_1, X_S^1)$ exists, where $S_1 = \mu^{-1}(D_{X_S})$ and $X_S^1 = D(S_F - S_1)/\gamma D_{X_S}$. The eigenvalues of the variational matrix of subsystem (4) at $\widetilde{E_1}$

are the roots of the equation

$$\lambda^2 + (D + \gamma\mu'(S_1)X_S^1)\lambda + \gamma\mu(S_1)\mu'(S_1)X_S^1 = 0,$$

which have negative real parts, and leads to the result of locally stable of $\widetilde{E_1}$. To investigate the global asymptotic stability of $\widetilde{E_1}$, let us define a Liapunov function

$$V_1(S,X_S) := \int_{S_1}^{S} \frac{\mu(\sigma)-\mu(S_1)}{\mu(\sigma)}\mathrm{d}\sigma + \gamma\int_{X_S^1}^{X_S} \frac{\sigma - X_S^1}{\sigma}\mathrm{d}\sigma.$$

Then $V_1(S,X_S) \geq 0$ in the $S-X_S$ plane and $V_1(S,X_S) = 0$ if and only if $S = S_1$, $X_S = X_S^1$. Taking the time derivative of V_1 along the solutions of subsystem (4) yields that

$$\begin{aligned}
\frac{\mathrm{d}V_1}{\mathrm{d}t} &= \frac{\partial V}{\partial S}\frac{\mathrm{d}S}{\mathrm{d}t} + \gamma\frac{\partial V}{\partial X_S}\frac{\mathrm{d}X_S}{\mathrm{d}t} \\
&= \frac{\mu(S)-\mu(S_1)}{\mu(S)}\frac{\mathrm{d}S}{\mathrm{d}t} + \gamma\frac{X_S - X_S^1}{X_S}\frac{\mathrm{d}X_S}{\mathrm{d}t} \\
&= [\mu(S)-\mu(S_1)]\left[\frac{D(S_F-S)}{\mu(S)} - \gamma X_S\right] + \gamma(X_S - X_S^1)\left[\mu(S) - D_{X_S}\right] \\
&= [\mu(S)-\mu(S_1)]\left[\frac{D(S_F-S)}{\mu(S)} - \frac{D(S_F-S_1)}{\mu(S_1)}\right] \\
&= [\mu(S)-\mu(S_1)]\left[\varphi(S) - \varphi(S_1)\right]
\end{aligned}$$

where $\varphi(S) = D(S_F - S)/\mu(S)$. Since $\varphi'(S) = -D(\mu(S) + \mu'(S)(S_F - S))/\mu^2(S) < 0$ and $\mu'(S) > 0$, then $\mathrm{d}V_1/\mathrm{d}t \leq 0$ and $\mathrm{d}V_1/\mathrm{d}t = 0$ if and only if $S = S_1$. By LaSalle's invariance principle [13], $\widetilde{E_1}(S_1, X_S^1)$ is global asymptotically stable in the $S-X_S$ plane and attracts all feasible solutions. This completes the proof.

3.2. Uninfected biomass extinction dynamics

It is noted that when $X_S(0) = 0$, $X_S(t) = 0$ for all $t \geq 0$. So, the plane $S-X_I$ is also an invariant set of the system (1). In this case, system (1) is reduced to

$$\left\{\begin{array}{l}
\dfrac{\mathrm{d}S}{\mathrm{d}t} = D(S_F - S) - \kappa\gamma\nu(S)X_I, \\
\dfrac{\mathrm{d}X_I}{\mathrm{d}t} = \kappa\nu(S)X_I - D_{X_I}X_I.
\end{array}\right. \tag{5}$$

Define $D_{MIN} := \max\{\kappa\nu(S_F) - \delta_{X_I}, 0\}$. Then similar to Theorem 3.4, the following result for the system (5) holds.

Theorem 3.5. *If $D > D_{MIN}$, then the unique biomass extinction equilibrium $\widehat{E_0}(S_F,0)$ of the system (5) is global asymptotically stable; if $0 < D < D_{MIN}$, then $\widehat{E_0}(S_F,0)$ is unstable and the positive equilibrium $\widehat{E_2}(S_2,X_I^2)$ exists and is global asymptotically stable in the $S-X_I$ plane, where $0 < S_2 = \nu^{-1}(D_{X_I}/\kappa) < S_F$ and $0 < X_I^2 = D(S_F - S_2)/\gamma D_{X_I}$.*

3.3. The full dynamics of the system (1)

In this section, the dynamics of the system (1) for $X_S(0) > 0$ and $X_I(0) > 0$ are discussed. At the steady state, (S, X_S, X_I) satisfies that

$$\begin{pmatrix} \mu(S) - D_{X_S} - \alpha/X_S & 0 \\ \mu(S) - D_{X_S} & \kappa\nu(S) - D_{X_I} \\ \gamma D_{X_S} & \gamma D_{X_I} \end{pmatrix} \begin{pmatrix} X_S \\ X_I \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ D(S_F - S) \end{pmatrix}. \tag{6}$$

Obviously, the biomass extinction equilibrium $E_0(S_F,0,0)$ always exists. By (H4), there is $D_{MIN} \le D_{MAX}$. Thus if $D > D_{MAX}$ holds, E_0 is the unique steady state, and its local stability can be determined by the eigenvalues of the variational matrix of the system (1) at E_0, i.e.

$$J_{E_0} = \begin{pmatrix} -D & -\gamma\mu(S_F) & -\kappa\gamma\nu(S_F) \\ 0 & \mu(S_F) - D_{X_S} & 0 \\ 0 & 0 & \kappa\nu(S_F) - D_{X_I} \end{pmatrix}.$$

Theorem 3.6. *If $D > D_{MAX}$ holds, then the system (1) has a unique biomass extinction equilibrium $E_0(S_F,0,0)$, which is global asymptotically stable.*

Proof. For the case of $D > D_{MAX}$, the eigenvalues of the variational matrix J_{E_0} are all negative, which means that E_0 is locally asymptotically stable. Besides, by Theorem 3.4, if $D > D_{MAX}$, then $X_S \to 0$ when $t \to \infty$, which means that system (1) has a limiting system (5). Then by Theorem 3.5 that $\widehat{E_0}$ is global asymptotically stable in the plane $S - X_I$. Therefore, E_0 is global asymptotically stable and attracts all the feasible solutions.

For the case of $0 < D < D_{MAX}$, defined

$$\Theta_1 := \frac{\kappa\nu(S_1) + \alpha_{X_I}(X_S^1, 0)}{D_{X_I}}.$$

Theorem 3.7. *If $D_{MIN} < D < D_{MAX}$, then the uninfected biomass extinction equilibrium doesn't exist and the system (1) has an infected biomass extinction equilibrium $E_1(S_1, X_S^1, 0)$, which is locally asymptotically stable if $\Theta_1 < 1$ and unstable when $\Theta_1 > 1$.*

Proof. Similar to the discussion in Theorem 3.6, the biomass extinction equilibrium $E_0(S_F, 0, 0)$ is unstable in the direction orthogonal to $S - X_I$ plane in the case of $D_{MIN} < D < D_{MAX}$. The infected biomass extinction equilibrium $E_1(S_1, X_S^1, 0)$ exists and the variational matrix of the system (1) at E_1 is

$$J_{E_1} = \begin{pmatrix} -D - \mu'(S_1)X_S^1 & -\gamma\mu(S_1) & -\kappa\gamma\nu(S_1) \\ \mu'(S_1)X_S^1 & 0 & \alpha_{X_I}(X_S^1, 0) \\ 0 & 0 & \kappa\nu(S_1) - D_{X_I} + \alpha_{X_I}(X_S^1, 0) \end{pmatrix}.$$

The eigenvalues of the variational matrix J_{E_1} are the roots of the equation

$$[\lambda - (\kappa\nu(S_1) + \alpha_{X_I}(X_S^1, 0) - D_{X_I})][\lambda^2 + \lambda(\mu'(S_1)X_S^1 + D) + \gamma\mu(S_1)\mu'(S_1)X_S^1] = 0. \tag{7}$$

If $\Theta_1 < 1$, then all the eigenvalues have negative real parts, which means that E_1 is locally asymptotically stable. When $\Theta_1 > 1$, one eigenvalue is positive, then E_1 is unstable in the direction orthogonal to $S - X_S$ coordinate plane.

Remark. *Θ_1 is a threshold for the stability of $E_1(S_1, X_S^1, 0)$, which is the ratio of the sum of maximal uptake rate of the infected biomass and the infection rate to its mortality rate at the steady state E_1. By H5, $\Theta_1 > 1$ means that $\kappa\nu(S) - D_S + (\alpha/X_I)(X_S, X_I) > 0$, i.e. $dX_I/dt > 0$ for $X_I > 0$ and $S \geq S_1$, which leads to the instability of E_1.*

Theorem 3.8. *For the case of $D_{MIN} = 0$ and $D < D_{MAX}$, if $\Theta_1 > 1$, the system (1) has a positive equilibrium $E^*(S^*, X_S^*, X_I^*)$ with $S_1 < S^* < S_F$. The steady state E^* is locally asymptotically stable if $\Delta < 0$ and unstable when $\Delta > 0$, where Δ is defined in Eq. (10). Especially, when $\kappa = 0$, i.e. the infected biomass is incapable of substrate consumption, E^* is locally asymptotically stable if $\Delta_1 \leq 0$ where*

$$\Delta_1 := [(\alpha_{X_I} - \alpha/X_I)(\alpha_{X_S} - \alpha/X_S) - \alpha_{X_S}\alpha_{X_I}](X_S^*, X_I^*).$$

Proof. By Eq. (6), it can be obtained that

$$\begin{pmatrix} X_S^* \\ X_I^* \end{pmatrix} = \frac{D(S_F - S^*)}{\gamma D_{X_I}(\mu(S^*) - D_{X_S}) - \gamma D_{X_S}(\kappa\nu(S^*) - D_{X_I})} \begin{pmatrix} D_{X_I} - \kappa\nu(S^*) \\ \mu(S^*) - D_{X_S} \end{pmatrix}.$$

Substituting X_S^* and X_I^* into Eq. (6) yields that

$$F(S^*) =: \mu(S^*) - D_{X_S} - (\alpha/X_S)(X_S^*, X_I^*) = 0 \tag{8}$$

and

$$G(S^*) =: \kappa\nu(S^*) - D_{X_I} + (\alpha/X_I)(X_S^*, X_I^*) = 0. \tag{9}$$

Define $H(S) := F(S) - G(S)$. Then Eqs. (8)-(9) mean that $S = S^*$ is a root of the equation $H(S) = 0$. It is noted that $H(S_1) = -G(S_1)$ due to $F(S_1) = 0$, and then by $\Theta_1 > 1$, it follows that $G(S_1) > D_{X_S}(\Theta_1 - 1) > 0$, i.e. $H(S_1) < 0$. On the other hand, $H(S_F) = F(S_F) - G(S_F) > 0$, then by the intermediate value theorem of continuity function H, there exists $S_1 < S^* < S_F$ such that $H(S^*) = 0$, i.e. the positive equilibrium $E^*(S^*, X_S^*, X_I^*)$ exists. The variational matrix of the system (1) at the positive equilibrium E^* is

$$J_{E^*} = \begin{pmatrix} m_{11} & -\gamma\mu(S^*) & -\kappa\gamma\nu(S^*) \\ \mu'(S^*)X_S^* & m_{22} & -\alpha_{X_I}(X_S^*, X_I^*) \\ \kappa\nu'(S^*)X_I^* & \alpha_{X_S}(X_S^*, X_I^*) & m_{33} \end{pmatrix},$$

where $m_{11} := -D - \gamma X_S^*\mu'(S^*) - \kappa\gamma X_I^*\nu'(S^*) < 0$, $m_{22} := \alpha(X_S^*, X_I^*)/X_S^* - \alpha_{X_S}(X_S^*, X_I^*) \geq 0$ and $m_{33} := \alpha_{X_I}(X_S^*, X_I^*) - \alpha(X_S^*, X_I^*)/X_I^* \leq 0$. By using the elementary operations, the matrix J_{E^*} is equivalently changed to J_1, where

$$J_1 = \begin{pmatrix} m_{11} & -\gamma\mu(S^*) & -\kappa\gamma\nu(S^*) \\ 0 & m'_{22} & m'_{23} \\ 0 & m'_{32} & m'_{33} \end{pmatrix}$$

where $m'_{22} = m_{22} + \gamma\mu(S^*)\mu'(S^*)X_S^*/m_{11}$, $m'_{23} = \kappa\gamma\nu(S^*)\mu'(S^*)X_S^*/m_{11} - \alpha_{X_I}(X_S^*, X_I^*) < 0$, $m'_{32} = \alpha_{X_S}(X_S^*, X_I^*) + \kappa\gamma\mu(S^*)\nu'(S^*)X_I^*/m_{11}$ and $m'_{33} = m_{33} + \kappa^2\gamma\nu(S^*)\nu'(S^*)X_I^*/m_{11} < 0$. In

a similar way, the matrix J_1 can be equivalently changed to J_2, where

$$J_2 = \begin{pmatrix} m_{11} & -\gamma\mu(S^*) & -\kappa\gamma\nu(S^*) \\ 0 & m''_{22} & 0 \\ 0 & m'_{32} & m'_{33} \end{pmatrix}$$

where $m''_{22} = m'_{22} - m'_{23}m'_{32}/m'_{33}$. Define

$$\Delta = m'_{23}m'_{32} - m'_{22}m'_{33}. \tag{10}$$

Clearly, m''_{22} has the same sign with Δ. The eigenvalues of the matrix J_2 are $\lambda_1 = m_{11} < 0$, $\lambda_2 = m''_{22}$ and $\lambda_3 = m'_{33} < 0$. Since the matrix J_{E^*} and J_2 are equivalent and the operations are elementary, the eigenvalues of J_{E^*} have the same signs with the eigenvalues of J_2, i.e. if $\Delta < 0$, then the eigenvalues of J_{E^*} are all negative, which means that E^* is locally asymptotically stable. Otherwise, when $\Delta > 0$, E^* is unstable. When $\kappa = 0$, i.e. the infected biomass is incapable of substrate consumption, there is $m'_{23} = -\alpha_{X_I}(X_S^*, X_I^*)$, $m'_{32} = \alpha_{X_S}(X_S^*, X_I^*)$, $m'_{33} = \alpha_{X_I}(X_S^*, X_I^*) - (\alpha/X_I)(X_S^*, X_I^*)$, $m'_{22} < (\alpha/X_S)(X_S^*, X_I^*) - \alpha_{X_I}(X_S^*, X_I^*)$. Therefore,

$$\Delta < \left[(\alpha_{X_I} - \alpha/X_I)(\alpha_{X_S} - \alpha/X_S) - \alpha_{X_S}\alpha_{X_I}\right](X_S^*, X_I^*) := \Delta_1,$$

so when $\Delta_1 \leq 0$, there is $\Delta < 0$, i.e. E^* is locally asymptotically stable. For the case of $D_{MIN} > 0$ and $D < D_{MIN}$, define

$$\Theta_2 := \frac{\mu(S_2)}{D_{X_S} + \alpha_{X_S}(0, X_I^2)}.$$

Theorem 3.9. *For the case of $D_{MIN} > 0$ and $D < D_{MIN}$, the system (1) has a uninfected biomass extinction equilibrium $E_2(S_2, 0, X_I^2)$ and an infected biomass extinction equilibrium $E_1(S_2, X_S^1, 0)$. Furthermore, E_1 is locally asymptotically stable if $\Theta_1 < 1$ and unstable when $\Theta_1 > 1$; E_2 is locally asymptotically stable if $\Theta_2 < 1$ and unstable when $\Theta_2 > 1$.*

Proof. For the case of $D_{MIN} > 0$ and $D < D_{MIN}$, two eigenvalues of the variational matrix J_{E_0} are positive, the biomass extinction equilibrium E_0 is unstable. In this case, the system (1) has an infected biomass extinction equilibrium $E_1(S_2, X_S^1, 0)$ and a uninfected biomass extinction equilibrium $E_2(S_2, 0, X_I^2)$. By Theorem 3.7, E_1 is locally asymptotically stable if $\Theta_1 < 1$ and

unstable when $\Theta_1 > 1$. The variational matrix of the system (1) at E_2 is

$$J_{E_2} = \begin{pmatrix} -D - \kappa\gamma\nu'(S_2)X_I^2 & -\lambda\mu(S_2) & -\kappa\gamma\nu(S_2) \\ 0 & \mu(S_2) - D_{X_S} - \alpha_{X_S}(0, X_I^2) & 0 \\ \kappa\nu'(S_2) & \alpha_{X_S}(0, X_I^2) & 0 \end{pmatrix}.$$

The eigenvalues of the variational matrix J_{E_2} are the roots of the equation

$$[\lambda - (\mu(S_2) - D_{X_S} - \alpha_{X_S}(0, X_I^2))][\lambda^2 + \lambda(D + \kappa\gamma\nu'(S_2)X_I^2) + \kappa^2\gamma\nu(S_2)\nu'(S_2)] = 0.$$

If $\Theta_2 < 1$, then $E_2(S_2, 0, X_I^2)$ is locally asymptotically stable. When $\Theta_2 > 1$, then $E_2(S_2, 0, X_I^2)$ is unstable.

Theorem 3.10. *For the case of $D_{MIN} > 0$ and $D < D_{MIN}$, if $\Theta_1 > 1$ and $\Theta_2 > 1$, then the system (1) has a positive equilibrium $E^*(S^*, X_S^*, X_I^*)$ with $S_1 < S^* < S_2$. Furthermore, E^* is locally asymptotically stable if $\Delta < 0$ and unstable when $\Delta > 0$, where Δ is defined in Eq. (10).*

Proof. The proof is similar to that of Theorem 3.8 and the existence of the positive equilibrium $E^*(S^*, X_S^*, X_I^*)$ can be determined as follows: $F(S_1) = G(S_2) = 0$. Thus, $H(S_1) = -G(S_1)$ and $H(S_2) = F(S_2)$. By $\Theta_1 > 1$, it follows that $G(S_1) > D_{X_S}(\Theta_1 - 1) > 0$. And by $\Theta_2 > 1$, it follows that $F(S_2) > 0$. Therefore, $H(S_1) < 0$ and $H(S_2) > 0$. By the intermediate value theorem of continuity function H, there exists $S_1 < S^* < S_2$ such that $H(S^*) = 0$, i.e. the positive equilibrium $E^*(S^*, X_S^*, X_I^*)$ exists.

4. Applied instance and computer simulations

The substrate uptakes are chosen as Holling type-II functional form, i.e.

$$\mu(S) = \frac{\mu_{\max}^{X_S} S}{K_{X_S} + S}, \quad \nu(S) = \frac{\nu_{\max}^{X_I} S}{K_{X_I} + S}, \tag{11}$$

with $\mu_{\max}/\nu_{\max} \geq \max\{1, K_{X_S}/K_{X_I}\}$. The infection rate is chosen as

$$\alpha(X_S, X_I) = \frac{\rho X_S X_I}{g(X_S, X_I)}. \tag{12}$$

The dilution rate D can be treated as a control parameter in the following discussion.

4.1. Case of $g(X_S, X_I) = X_S + X_I$ and $0 < \kappa \leq 1$

In this case, system (1) takes the following form:

$$
(13)\qquad \begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = D(S_F - S) - \gamma \mu_{\max}^{X_S} \dfrac{SX_S}{K_{X_S} + S} - \kappa\gamma \nu_{\max}^{X_I} \dfrac{SX_I}{K_{X_I} + S}, \\ \dfrac{\mathrm{d}X_S}{\mathrm{d}t} = \mu_{\max}^{X_S} \dfrac{SX_S}{K_{X_S} + S} - D_{X_S} X_S - \dfrac{\rho X_S X_I}{X_S + X_I}, \\ \dfrac{\mathrm{d}X_I}{\mathrm{d}t} = \kappa \nu_{\max}^{X_I} \dfrac{SX_I}{K_{X_I} + S} + \dfrac{\rho X_S X_I}{X_S + X_I} - D_{X_I} X_I. \end{cases}
$$

The dynamics of the system (13) can be depicted as follows:

(i) The system always has a biomass extinction steady state $E_0(S_F, 0, 0)$. This steady state is global asymptotically stable when

$$
D > D_{\max} := \frac{\mu_{\max}^{X_S} S_F}{K_{X_S} + S_F} - \delta_{X_S},
$$

as shown in Figure 1, where $\mu_{\max} = 0.8$, $\nu_{\max} = 0.6$, $K_{X_S} = 8$; $K_{X_I} = 10$, $\rho = \rho_1 = 0.115$, $\delta_S = 0.05$, $\delta_I = 0.1$, $\kappa = 1$, $\gamma = 2$, $S_F = 60$ and $D = 0.7 > D_{\max} \simeq 0.656$. The initial conditions are $S(0) = 40$, $X_S(0) = 20$ and $X_I(0) = 6$. In this case, E_0 attracts all feasible solutions.

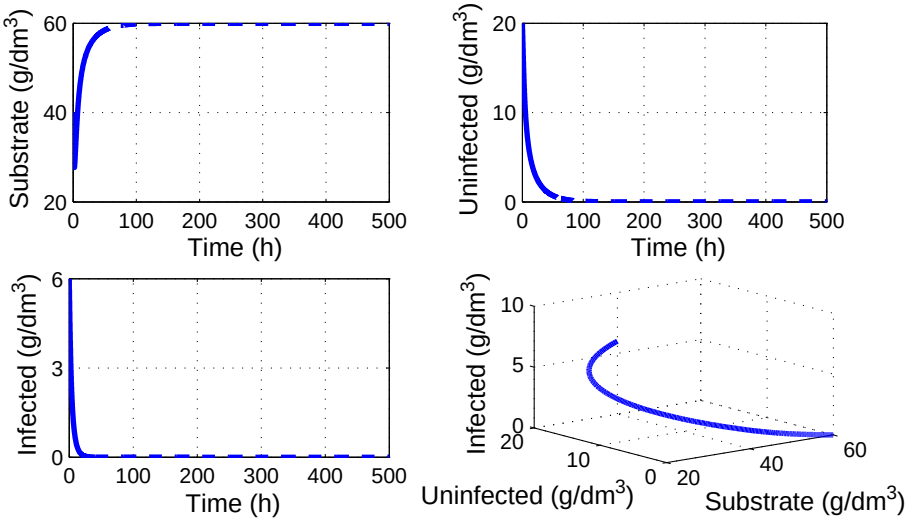

FIGURE 1. Time series and phase portrait of the system (1) for $D > D_{\max}$, where E_0 is asymptotically stable and global attractor.

(ii) For $D_{MIN} < D < D_{MAX}$, the biomass extinction steady state E_0 is unstable in the direction orthogonal to $S - X_S$ coordinate plane, and there exists an infected biomass extinction steady state $E_1(S_1, X_S^1, 0)$, where

$$S_1 = \frac{D_S K_{X_S}}{\mu_{\max}^{X_S} - D_{X_S}},\ X_S^1 = \frac{D(K_{X_S} + S_F)}{\gamma D_{X_S}} \frac{D_{MAX} - D}{\mu_{\max}^{X_S} - D_{X_S}},$$

which is locally asymptotically stable when

$$\Theta_1 = \frac{\nu_{\max}^{X_I} S_1 + \rho(K_{X_I} + S_1)}{D_{X_I}(K_{X_I} + S_1)} < 1,$$

and unstable when $\Theta_1 > 1$. For the parameters used in (i), when D is decreased to 0.6, which is smaller than D_{MAX} and greater than $D_{MID} = 0.414$, the solutions are presented in Figure 2. In this case, it can be easily calculated that $\Theta_1 = 0.83$, i.e. E_1 is locally asymptotically stable. Meanwhile, by Theorem 3.5, in the $S - X_I$ plane, $\widehat{E_0}(S_F, 0)$ is global asymptotically stable, as illustrated in Figure 3, which indicates that E_1 is not globally attractive. When ρ is increased to $\rho_2 = 0.25$, then Θ_1 will be increased to 1.02, which means that E_1 is unstable, as shown in Figure 4.

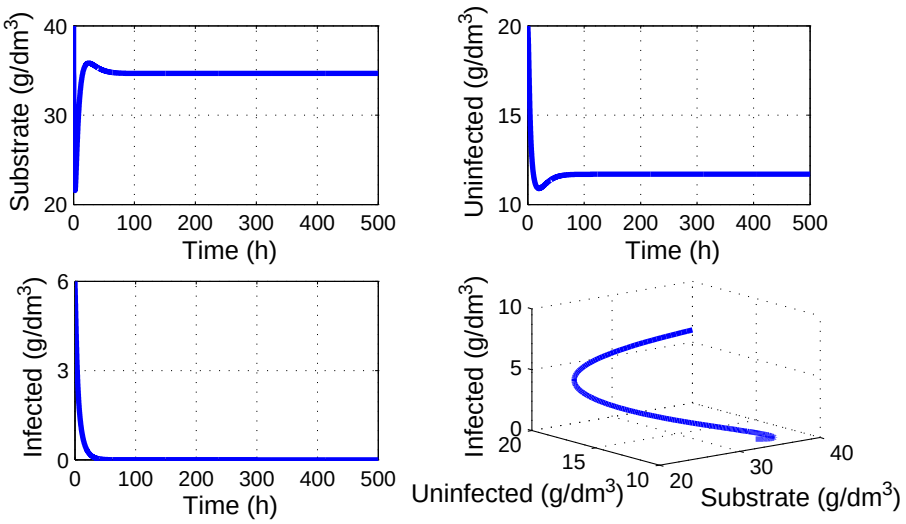

FIGURE 2. Time series and phase portrait of the system (1) for $D_{MIN} < D < D_{\max}$, where E_1 is locally asymptotically stable.

(iii) For the case of $D < D_{MIN}$, E_0 is unstable, and E_1 and $E_2(S_2, 0, X_I^2)$ coexist, where

$$S_2 = \frac{D_{X_I} K_{X_I}}{\nu_{\max}^{X_I} - D_{X_I}},\ X_I^2 = \frac{D(K_{X_I} + S_F)}{\gamma D_{X_I}} \frac{D_{MIN} - D}{\nu_{\max}^{X_I} - D_{X_I}}.$$

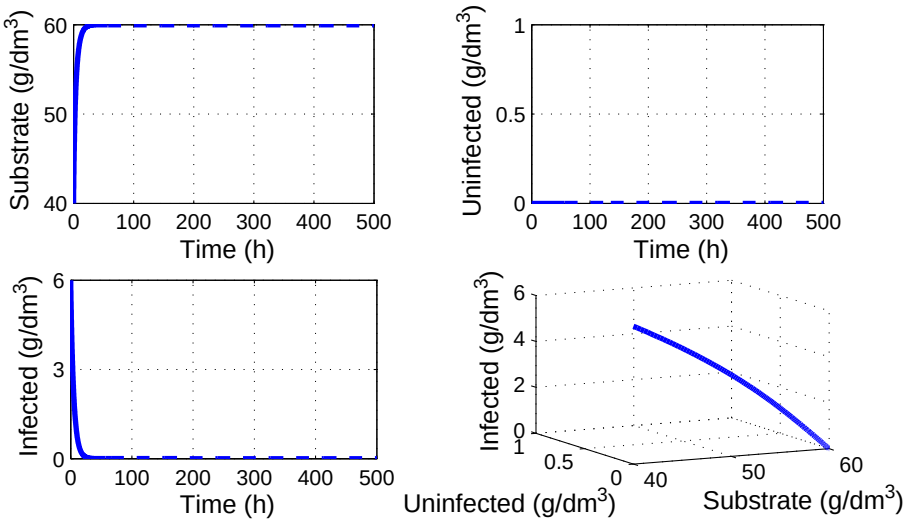

FIGURE 3. Time series and phase portrait of the system (1) for $D_{MIN} < D < D_{\max}$ in the $S - X_I$ plane, where $\widehat{E_0}$ is stable.

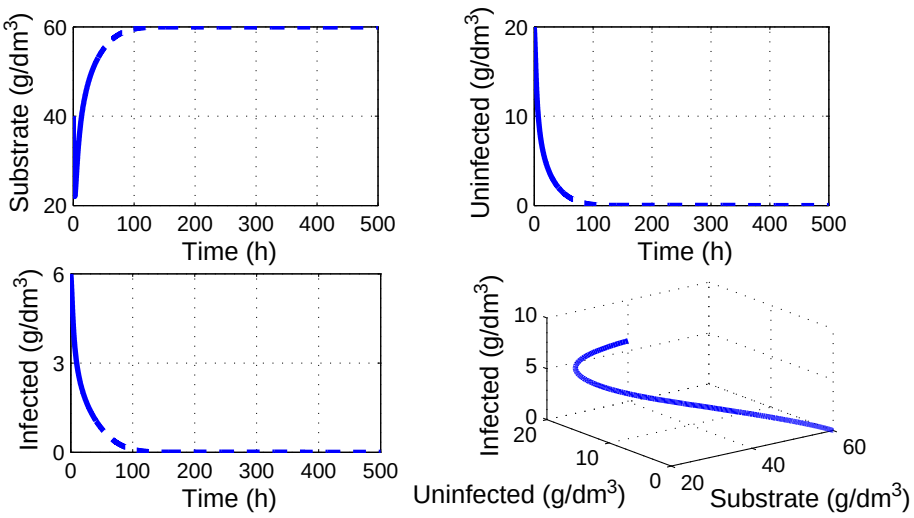

FIGURE 4. Time series and phase portrait of the system (1) for $D_{MIN} < D < D_{\max}$, where E_1 is unstable.

Meanwhile, by (ii), E_1 is locally asymptotically stable if $\Theta_1 < 1$ and unstable when $\Theta_1 > 1$. The steady state E_2 is locally asymptotically stable if

$$\Theta_2 = \frac{\mu_{\max}^{X_S} K_{X_I} D_{X_I}}{(D_{X_S} + \rho)(K_1 \nu_{\max}^{X_I} + (K_{X_I} - K_{X_S}) D_{X_I})} < 1,$$

and unstable when $\Theta_2 > 1$. For the parameters used in (i), when D is decreased to 0.4, which is smaller than D_{MIN}, the solutions are presented in Figure 5. In this case,

$\Theta_1 = 0.84$ and $\Theta_2 = 1.22$, so E_1 is locally stable and E_2 is unstable. When ρ is increased to $\rho_2 = 0.25$, $\Theta_1 = 1.1$ and $\Theta_2 = 0.985$, so E_2 is locally stable and E_1 is unstable, as illustrated in Figure 6. When ρ lies between ρ_1 and ρ_2, for example $\rho = 0.2$, then $\Theta_1 = 1.008$ and $\Theta_2 = 1.06$, and thus E_1 and E_2 are unstable, and there exists a coexistence steady state $E^*(11.28, 19.51, 1.93)$, as shown in Figure 7, in this case, $\Delta = -0.0012 < 0$, which means that E^* is locally asymptotically stable. It is noted that the stability of E^* is determined by the model parameters, i.e. the sign of Δ, when some parameters changed, for example, $v_{\max}$ is increased from 0.6 to 0.64, and the other parameters are the same as those in Figure 7, then there is $\Delta = 0.021$, which implies that E^* is unstable.

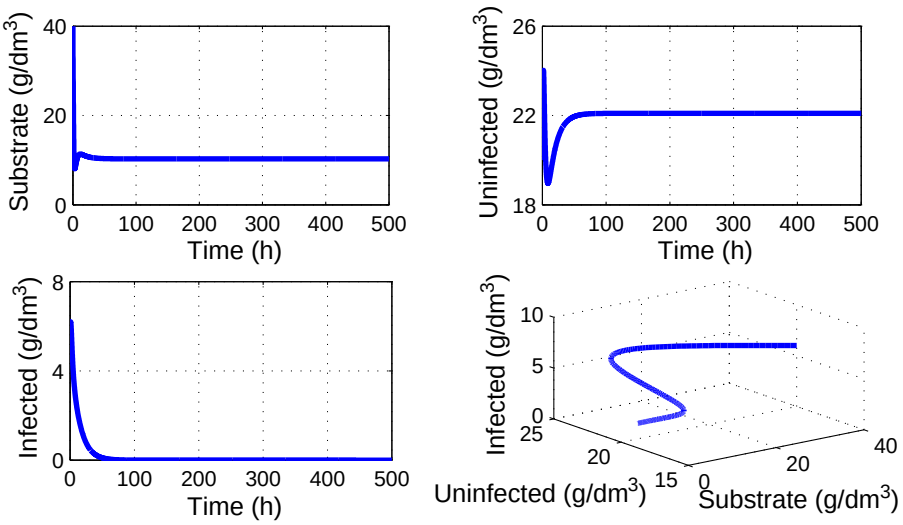

FIGURE 5. Time series and phase portrait of the system (1) for $D < D_{MIN}$, where E_1 is locally asymptotically stable and E_2 is unstable.

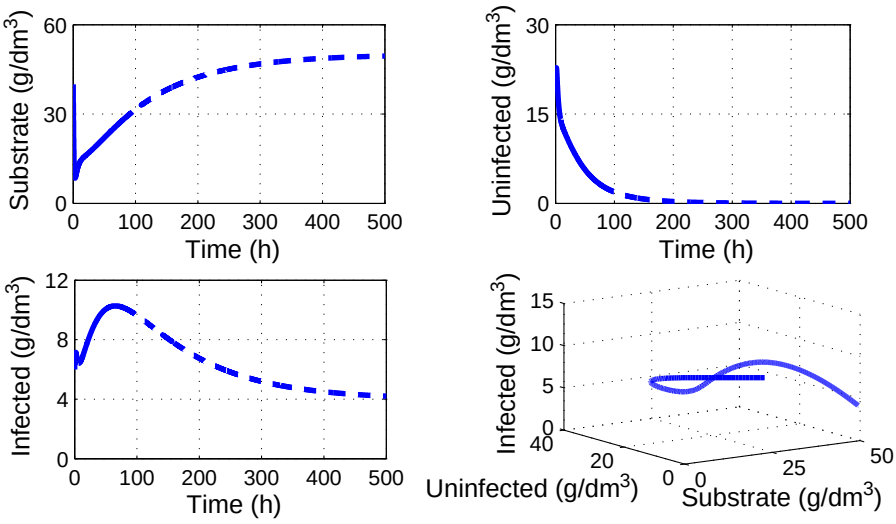

FIGURE 6. Time series and phase portrait of the system (1) for $D < D_{MIN}$, where E_2 is locally asymptotically stable and E_1 is unstable.

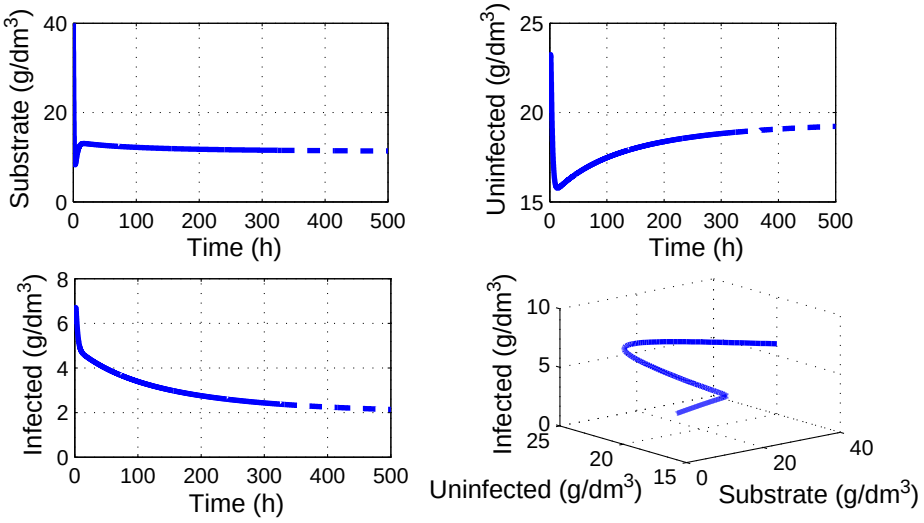

FIGURE 7. Time series and phase portrait of the system (1) for $D < D_{MIN}$, where E_1 and E_2 are unstable, E^* exists and is locally asymptotically stable.

4.2. Case of $g(X_S, X_I) = C$, where $C \geq S_F/\gamma$ and $\kappa = 0$

In this case, the system (1) takes the following form:

$$\begin{cases} \dfrac{dS}{dt} = D(S_F - S) - \gamma \mu_{\max}^{X_S} \dfrac{SX_S}{K_{X_S} + S} \\ \dfrac{dX_S}{dt} = \mu_{\max}^{X_S} \dfrac{SX_S}{K_{X_S} + S} - D_{X_S} X_S - \overline{\rho} X_S X_I \\ \dfrac{dX_I}{dt} = \overline{\rho} X_S X_I - D_{X_I} X_I \end{cases} \tag{14}$$

where $\overline{\rho} := \rho / C$. The dynamics of the system (14) are depicted as follows:

(i) If $D > D_{MAX}$, then the uninfected biomass and hence the infected biomass would be eventually extinct, i.e. the biomass extinction steady state E_0 is global asymptotically stable. In this case, E_0 attracts all feasible solutions.

(ii) When $D < D_{MAX}$, the biomass extinction steady state E_0 is unstable and there exists a feasible infected biomass extinction steady state $E_1(S_1, X_S^1, 0)$, where

$$S_1 = \frac{D_S K_{X_S}}{\mu_{\max}^{X_S} - D_{X_S}}, \; X_S^1 = \frac{D(K_{X_S} + S_F)}{\gamma D_{X_S}} \frac{D_{MAX} - D}{\mu_{\max}^{X_S} - D_{X_S}},$$

and E_1 is locally asymptotically stable when $\Theta_1 = \rho X_S^1 / D_{X_I} < 1$, as shown in Figure 8, where $\mu_{\max} = 0.8$, $K_{X_S} = 8$, $\rho = 0.05$, $\delta_S = 0.05$, $\delta_I = 0.1$, $\gamma = 2$, $S_F = 60$, $C = 30$ and $D = 0.6 < D_{\max} \simeq 0.656$. The initial conditions are $S(0) = 40$, $X_S(0) = 20$ and $X_I(0) = 6$.

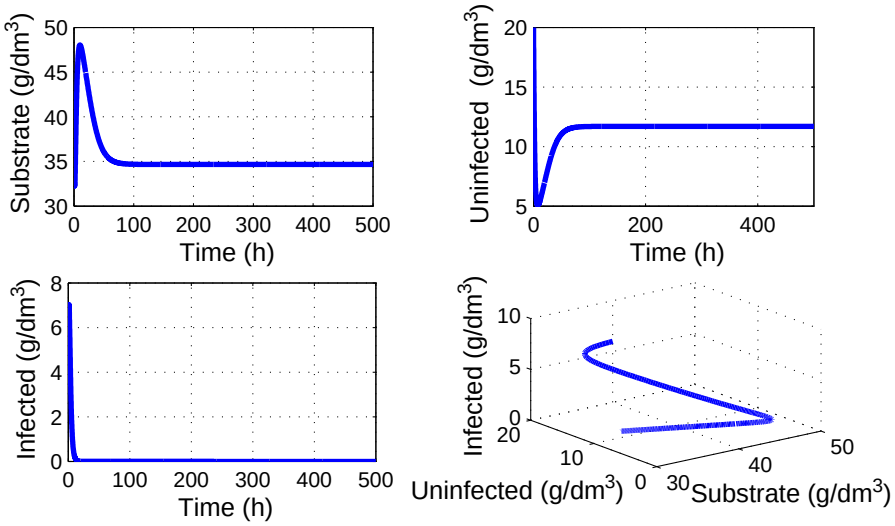

FIGURE 8. Time series and phase portrait of the system (1) for $D < D_{\max}$, where E_1 is locally asymptotically stable.

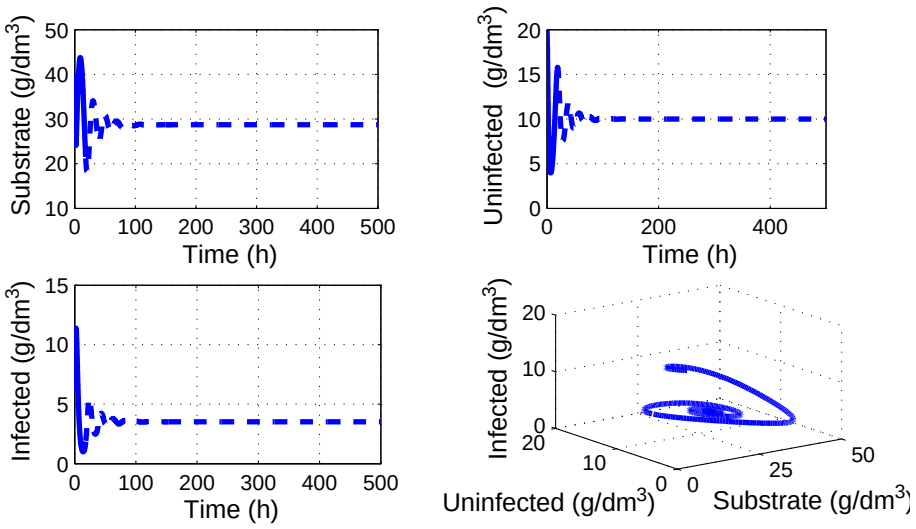

FIGURE 9. Time series and phase portrait of the system (1) for $D < D_{\max}$, where E_* is locally asymptotically stable.

(iii) When $\Theta_1 > 1$, the infected biomass extinction steady state E_1 is unstable in the direction orthogonal to the $S - X_S$ plane, which implies the persistence of the system (14). The system (14) possesses a positive interior steady state $E^*(S^*, X_S^*, X_I^*)$ with

$$S^* = \frac{\rho D(S_F - K_{X_S}) - \gamma \mu_{\max}^{X_S} D_{X_I} + \sqrt{(\rho D(S_F - K_{X_S}) - \gamma \mu_{\max}^{X_S} D_{X_I})^2 + 4\rho D K_{X_S} S_F}}{2\rho D},$$

$$X_S^* = \frac{D_{X_I}}{\rho}, X_I^* = \frac{D(S_F - S^*) - \gamma D_{X_S} X_S^*}{\gamma D_{X_I}}.$$

The variational matrix of system (14) around the positive equilibrium E^* is

$$J_{E^*} = \begin{pmatrix} -D - \gamma \mu_{\max}^{X_S} K_{X_S} X_S^* / (K_{X_S} + S^*)^2 & -\gamma \mu_{\max}^{X_S} S^* / (K_{X_S} + S^*) & 0 \\ \mu_{\max}^{X_S} K_{X_S} X_S^* / (K_{X_S} + S^*)^2 & 0 & -X_S^* \\ 0 & X_I^* & 0 \end{pmatrix}.$$

Since $\Delta_1 = 0$ for $\alpha = \rho X_S X_I$, then by Theorem 3.8, the positive equilibrium E^* is locally asymptotically stable, as illustrated in Figure 9, where $D = 0.4$.

In Figures. 6-7, 9 the general behaviors of the bioprocesses after infection are presented. It is visible that high concentration of the infected biomass lasts for a very long time. Moreover, the uninfected biomass concentration presented in Figures 1, 3-4 drops to zero. In this article the

theoretical aspects of these cases have been signaled, but in practice these processes should be interrupted as soon as possible.

5. Conclusion

In this work, a three component chemostat model consisting of substrate, uninfected biomass and infected biomass concentrations are presented. The stability behavior of the system around the feasible steady states with general infection rate function and general nutrient uptake function both for uninfected and infected biomass are discussed in detail. By taking the dilution rate as a control parameter, conditions for infected biomass extinction are obtained. The results indicated that when the dilution rate is kept at a proper level, the uninfected biomass can be sustained while the infected biomass is eliminated from the system. Meanwhile, there exists a certain range of the dilution rate such that the three component system persists.

Conflict of Interests

The authors declare that there is no conflict of interests.

Acknowledgement

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