MATHEMATICAL BIOSCIENCES
AND ENGINEERING
Volume **9**, Number **2**, April **2012**

doi:10.3934/mbe.2012.9.445

pp. **445–459**

ANALYSIS OF A MODEL FOR THE EFFECTS OF AN EXTERNAL TOXIN ON ANAEROBIC DIGESTION

Marion Weedermann

Dominican University
7900 W Division St, River Forest, IL 60305, USA

(Communicated by Yang Kuang)

Abstract. Anaerobic digestion has been modeled as a two-stage process using coupled chemostat models with non-monotone growth functions, [9]. This study incorporates the effects of an external toxin. After reducing the model to a 3-dimensional system, global stability of boundary and interior equilibria is proved using differential inequalities and comparisons to the corresponding toxin-free model. Conditions are given under which the behavior of the toxin-free model is preserved. Introduction of the toxin results in additional patterns such as bistabilities of coexistence steady states or of a periodic orbit and an interior steady state.

1. **Introduction.** Anaerobic digestion is a natural process during which cohorts of micro-organisms break down organic matter in the absence of oxygen. The resulting biogas consists of methane, carbon dioxide, and trace gases. Recently, there has been an increased interest in the commercial utilization of anaerobic digestion for its environmental and economic benefits. Anaerobic digestion is used in waste treatment facilities, especially for the treatment of sewage sludge; the biogas is captured before it can escape into the atmosphere and can be used as renewable energy either by combusting the gas to produce electrical energy or by extracting the methane and using it as a natural gas fuel. While anaerobic digestion is a naturally occurring bioprocess, the process appears to be unstable and difficult to control in industrial settings. Anaerobic digestion is an extremely complex process that involves a large number of strains of bacteria and is not yet completely understood. In particular, biologists' understanding of the microbiology of the organisms involved is still incomplete. Mathematical models can provide insight into the process and the microbiology, and help to establish guidelines for the control and stabilization of large scale installations.

Numerous comprehensive models for anaerobic digestion processes have been developed, see [2, 5, 13] and the references therein. Among these, the most comprehensive model was developed in [2]. This high-dimensional model describes many of the biological, chemical, and kinetic processes of anaerobic digestion and can be used to calibrate individual installations. However, the complexity of this model makes a qualitative analysis very difficult. A different, more macroscopic approach to modeling anaerobic digestion was taken in [8, 9, 11, 17]. We are continuing this

2000 *Mathematics Subject Classification.* Primary: 34D23, 92D25; Secondary: 34D23.

Key words and phrases. Anaerobic digestion, chemostat, non-monotone growth, global stability, bistability.

effort in an attempt to gain a better understanding of the process and how the bacteria interact.

Anaerobic digestion is a 4-phase process consisting of hydrolysis, acidogenesis, acetogenesis, and methanogenesis. In the first phase, glucose, long chain fatty acids, amino acids are formed. During acidogenesis these are converted to ethanol, volatile fatty acids, acetate, hydrogen, and carbon dioxide. Hydrogen-producing heteroacetogens convert alcohols and short fatty acids into acetate, hydrogen, and carbon dioxide through acetogenic dehydrogenation. Hydrogen-consuming homoacetogens convert hydrogen and carbon dioxide into acetate. In the final stage, aceticlastic methanogens convert acetic acid into methane and carbon dioxide through acetate decarboxylation and hydrogenotrophic methanogens combine carbon dioxide and hydrogen into methane and water. In most biogas installations, about 70% of the methane is produced through the conversion of acetate to methane. By focusing on this main path and separating hydrolysis from the overall process one can model anaerobic digestion as a two-stage process consisting of (1) acidogenesis/acetogenesis to volatile fatty acids/acetate and (2) aceticlastic methanogenesis as was done in [11, 17, 9]. The models considered in these studies consist equations involving two substrates and two micro-organisms. In [11], monotone growth of the bacteria was assumed and the existence of a unique interior equilibrium was shown.

Methanogens belong to the group of Archaebacteria. They are strict anaerobes and can only survive within pH-range of about 6.5 - 8. A high concentration of acids lowers the pH and inhibits the growth of methanogenic archae and the production of methane. To model the impact of acid concentration on methanogens, their growth is described by a non-monotone growth function as was done in [9, 17]. In [9] it was shown that for some parameter conditions a unique, globally stable interior equilibrium exists while for other parameters the system exhibits the bistability of an interior equilibrium and a boundary equilibrium. The boundary equilibrium is such that methanogens responsible for the second phase are no longer present in the system and corresponds to a frequently observed scenario in industrial installation described as *acid accumulation* under which all methane production ceases.

In [8], the role of hydrogen on the acetogenesis and methanogenesis phases of anaerobic digestion were considered. The impact of acid on bacteria growth was ignored. This model can be considered a modification of the model studied in [11] where one of the substrates acts as an internally allocated inhibitor. The analysis gives conditions for the existence of a unique, globally stable interior equilibrium.

In this paper we perturb the model considered in [9, 11] to study the effects of an externally introduced toxin. We wish to know under what conditions an external toxin has limited to no effects on the limiting behavior of the system and whether a toxin can alter the limiting behavior to patterns different from steady state.

External toxins can act in different ways; they can affect both micro-organisms, or they can affect only one of the micro-organisms. If none of the micro-organisms are able to break-down the toxin, thus reducing its toxicity, the effects of the toxin can be studied by changing the parameters. However, if the micro-organisms can reduce the toxicity, then the toxin has to be modeled as a state variable. Here we assume that the toxin affects only one microorganism and that the other microorganisms breaks down the toxin or is able to decrease its toxicity. In the context of anaerobic digestion, well-known toxins are heavy metals, [7]. In separate studies [3, 15, 16] it has been shown methanogens are able to reduce heavy metal toxicity by converting methylmercury to an oxidation stage with increased solubility,

therefore decreasing the toxicity of methylmercury. While methane production has been reported in methylmercury decomposition, it is unclear whether it contributes to bacteria growth. In this study it is assumed that the reduction of toxin does not result in bacteria growth. We show that while many of the features of the inhibition-free system studied in [9] are preserved, including such a toxin increases the complexity of the system and may lead to the bistabilities of two coexistence equilibria or of a coexistence equilibrium and a periodic solution. Previous models for anaerobic digestion did not possess periodic solutions. Thus, an observed periodic pattern in biogas production data can be indicative of the presence of a toxin in the system.

The paper is organized as follows. We first summarize the results of [9] and provide a different but equivalent analysis in the appendix. In section 3 we give a local and global analysis of the modified model with an externally introduced toxin. The results give conditions under which the behavior of the toxin-free model is preserved. We conclude with a numerical study that shows the existence of stable periodic solution which coexists with a locally stable steady state. The periodic solutions are the result of a toxin-induced supercritical Hopf bifurcation.

2. **Two-stage model for methanogenesis.** We consider anaerobic digestion as a two-stage process. During the first stage, acid-forming bacteria (X_1) convert sugars and volatile fatty acids (S_1) into acetic acid (S_2). Methanogens (X_2) then transform acetic acid into methane and carbon dioxide. The nutrient supplied to the chemostat, S_1, is growth limiting for X_1. The intermediate product S_2 is growth limiting to X_2. However, if the concentration of S_2 is large, the conditions in the chemostat are altered leading to growth inhibiting conditions for X_2.

$$S_1 \xrightarrow{X_1} S_2 \xrightarrow{X_2} M$$

The interaction between the species can be described by the following system of differential equations.

$$\begin{aligned} \dot{S}_1 &= DS_1^{(0)} - DS_1 - \frac{1}{c_1} g_1(S_1)X_1 \\ \dot{X}_1 &= -DX_1 + p_1 g_1(S_1)X_1 \\ \dot{S}_2 &= -DS_2 + \frac{p_2}{c_{12}} g_1(S_1)X_1 - \frac{1}{c_2} g_2(S_2)X_2 \\ \dot{X}_2 &= -DX_2 + g_2(S_2)X_2 \end{aligned} \tag{1}$$

with $S_i(0) \geq 0$ and $X_i(0) \geq 0$. Here D is the dilution rate, $S_1^{(0)}$ the concentration of nutrient S_1 in the inflow, c_1, c_{12}, c_2 are yield coefficients, p_1 is the fraction of substrate consumption dedicated to bacteria growth, and p_2 is the fraction of substrate consumption used to form the second nutrient S_2. We assume that g_i are non-negative with $g_i(0) = 0$ and continuously differentiable. Furthermore, g_1 is monotone increasing, and g_2 is non-monotone, i.e., there is an $s_m > 0$ such that $g_2'(s) > 0$ for $s < s_m$ and $g_2'(s) < 0$ for $s > s_m$.

A similar model was given in [11] where both g_1 and g_2 were of Monod-type. The analysis given here also applies in that case if we allow for $s_m = \infty$ and $g_2'(s) > 0$ for $s > 0$. In [9], (1) was considered with the inclusion of an inflow term $DS_2^{(0)}$ in the equation for S_2 and under the assumption that g_1 is of Monod-type. Since (S_1, X_1) evolve independently of (S_2, X_2), the results for the global behavior of (1) are very

similar to that given in [9]. We will first derive a dimensionless version of (1) and then give a summary of the global behavior of the scaled system in Proposition 1. The analysis (which is different from the one given in [9] and foreshadows the techniques used in section 3) is given in the appendix.

Using the scaling $\hat{t} = tD$, $\hat{S}_i = S_i/S_1^{(0)}$, $\hat{X}_i = X_i/(c_i S_1^{(0)})$, and letting $\hat{g}_i(s) = g_i\left(sS_1^{(0)}\right)/D$, $(i = 1, 2)$, (1) becomes (after leaving off the $\hat{}$)

$$\begin{array}{lcll} \dot{S}_1 &=& 1 - S_1 - g_1(S_1)X_1 & S_1(0) \geq 0 \\ \dot{X}_1 &=& -X_1 + \gamma_1 g_1(S_1)X_1 & X_1(0) \geq 0 \\ \dot{S}_2 &=& -S_2 + \gamma_2 g_1(S_1)X_1 - g_2(S_2)X_2 & S_2(0) \geq 0 \\ \dot{X}_2 &=& -X_2 + g_2(S_2)X_2 & X_2(0) \geq 0 \end{array} \tag{2}$$

where $\gamma_1 = p_1$ and $\gamma_2 = c_1 p_2/c_{12}$. The following two assumptions are needed to ensure the existence of an interior equilibrium and are standard in the theory of the chemostat, [18].

(H1) there exist a unique $s = \lambda_o > 0$ such that $\gamma_1 g_1(s) = 1$
(H2) there exist two $s = \sigma_i$ with $0 < \sigma_1 < \sigma_2$ so that $g_2(s) = 1$

We will refer to λ_o as toxin-free break-even concentration and note that (H2) implies that $g_2(s) < 1$ for $s \notin [\sigma_1, \sigma_2]$ and $g_2(s) > 1$ for $s \in (\sigma_1, \sigma_2)$.

Proposition 1. *Assume that (H1) and (H2) hold. Then* $E_o = (1, 0, 0, 0)$, $E_1 = (\lambda_o, \gamma_1(1-\lambda_o), \gamma_2(1-\lambda_o), 0)$, *and* $E_c^i = (\lambda_o, \gamma_1(1-\lambda_o), \sigma_i, \gamma_2(1-\lambda_o)-\sigma_i)$, $(i = 1, 2)$, *are the equilibria of (2). Assuming* $S_i(0) > 0$ *and* $X_i(0) > 0$, *the behavior of the solution is as follows:*

(P1) If $\lambda_o > 1$, *then all solutions of (2) approach* E_o.
(P2) If $\lambda_o < 1$, *and* $\gamma_2(1 - \lambda_o) < \sigma_1$, *then all solutions of (2) approach* E_1.
(P3) If $\lambda_o < 1$, *and* $\gamma_2(1 - \lambda_o) \in (\sigma_1, \sigma_2)$, *then all solutions of (2) approach* E_c^1.
(P4) If $\lambda_o < 1$, *and* $\gamma_2(1 - \lambda_o) > \sigma_2$, *then all equilibria of (2) lie in* $\mathbb{R}^4_+$, *and* E_1 *and* E_c^1 *are locally asymptotically stable, while* E_o *and* E_c^2 *are unstable.*

The following lemma is a direct consequence of Proposition 1 and will be used in later proofs of global stability.

Lemma 2.1. *Let* $\beta > 0$ *and assume that* $\gamma_1 \beta g_1(s) = 1$ *has a solution* λ_β. *Consider*

$$\begin{array}{ll} \dot{u} = u\left(\gamma_1 \beta g_1\left(1 - \frac{1}{\gamma_1}u\right) - 1\right) & u(0) \geq 0 \\ \dot{v} = v\left(g_2\left(\frac{\gamma_2}{\gamma_1}u - v\right) - 1\right) & v(0) \geq 0 \end{array} \tag{3}$$

(i) If $\lambda_\beta > 1$, *then* $\lim_{t\to\infty} u(t) = 0$ *and* $\lim_{t\to\infty} v(t) = 0$.
(ii) If $\lambda_\beta < 1$, *then* $\lim_{t\to\infty} u(t) = \gamma_1(1 - \lambda_\beta)$. *If, in addition,* $\gamma_2(1 - \lambda_\beta) < \sigma_1$, *then* $\lim_{t\to\infty} v(t) = 0$. *If* $\gamma_2(1 - \lambda_\beta) \in (\sigma_1, \sigma_2)$, *then* $\lim_{t\to\infty} v(t) = \gamma_2(1 - \lambda_\beta) - \sigma_1$.

3. **Non-lethal external inhibition.** System (1) describes how the micro-organism X_2 depends on the activities of micro-organism X_1. Assume that a toxin T is introduced into the environment that inhibits the growth of the organism X_1 while X_2 has the ability to break down the toxin.

$$\begin{aligned}
\dot{S}_1 &= qS_1^{(0)} - qS_1 - \frac{1}{c_1}\, g_1(S_1) f(T) X_1 \\
\dot{X}_1 &= -qX_1 + (1-p)\, g_1(S_1) f(T) X_1 \\
\dot{S}_2 &= -qS_2 + p\, g_1(S_1) f(T) X_1 - \frac{1}{c_2}\, g_2(S_2) X_2 \\
\dot{X}_2 &= -qX_2 + g_2(S_2) X_2 \\
\dot{T} &= qT^{(0)} - qT - g_3(T) X_2
\end{aligned} \tag{4}$$

$$S_i(0) \geq 0, X_i(0) \geq 0, T(0) \geq 0,$$

where f and g_3 are continuously differentiable, f is decreasing and g_3 is decreasing, $f(T) > 0$ for $T \geq 0$, $f(0) = 1$ and $g(0) = 0$. Using the same scaling as in the previous section with the addition of $\hat{T} = T/T^{(0)}$, $\hat{f}(T) = f(TT^{(0)})$ and $\hat{g}_3(T) = c_2 \dfrac{g_3(TT^{(0)})}{qT^{(0)}}$, (4) can be written as

$$\begin{aligned}
\dot{S}_1 &= 1 - S_1 - g_1(S_1)\ f(T) X_1 \\
\dot{X}_1 &= -X_1 + \gamma_1 g_1(S_1)\ f(T) X_1 \\
\dot{S}_2 &= -S_2 + \gamma_2 g_1(S_1)\ f(T) X_1 - g_2(S_2) X_2 \\
\dot{X}_2 &= -X_2 + g_2(S_2) X_2 \\
\dot{T} &= 1 - T - g_3(T) X_2
\end{aligned} \tag{5}$$

$$S_i(0) \geq 0, X_i(0) \geq 0, T(0) \geq 0$$

Proposition 2. *The solutions of (5) are bounded.*

Proof. From the equations for X_i we can deduce directly that $X_i(t) \geq 0$, $(i = 1, 2)$. Thus, $\dot{T} \leq 1 - T$, $T(0) \geq 0$, which proves the boundedness of T. $\dot{S}_i|_{S_i=0} > 0$ implies that $S_i(t) \geq 0$. The boundedness of (S_1, X_1) follows from $\frac{d}{dt}\left(S_1 + \frac{1}{\gamma_1} X_1\right) = 1 - S_1 - \frac{1}{\gamma_1} X_1$. With (S_1, X_1) bounded, $\frac{d}{dt}(S_2 + X_2) \leq -(S_2 + X_2) + M$ for some $M > 0$. Therefore, (S_2, X_2) is also bounded. □

The theory of autonomous asymptotic systems developed in [14, 18, 19] can be used to obtain results for the global stability of (5). Let

$$\Sigma_1 = 1 - S_1 - \frac{1}{\gamma_1} X_1 \quad \text{and} \quad \Sigma_2 = -S_2 - X_2 + \frac{\gamma_2}{\gamma_1} X_1$$

Then $\Sigma_1' = -\Sigma_1$ and $\Sigma_1(0) \geq 0$ if $S_1(0) + \frac{1}{\gamma_1} X_1(0) \leq 1$. $\lim_{t\to\infty} \Sigma_1(t) = 0$, and $0 \leq \Sigma_1(t) \leq \Sigma_1(0)$ gives $S_1(0) + \frac{1}{\gamma_1} X_1(0) \leq S_1(t) + \frac{1}{\gamma_1} X_1(t) \leq 1$ for $t \geq 0$. Similarly, $\Sigma_2' = -\Sigma_2$ and $\Sigma_2(0) \geq 0$ if $S_2(0) + \frac{1}{\gamma_1} X_2(0) \leq \frac{\gamma_2}{\gamma_1} X_1(0)$. $\lim_{t\to\infty} \Sigma_2(t) = 0$, and $\Sigma_2(t) \geq 0$ together with earlier observations gives $0 \leq S_2(t) + X_2(t) \leq \frac{\gamma_2}{\gamma_1} X_1(t)$ for $t \geq 0$.

Writing (5) in terms of Σ_1 and Σ_2 gives

$$\begin{aligned}
\dot{\Sigma}_1 &= -\Sigma_1 \\
\dot{\Sigma}_2 &= -\Sigma_2 \\
\dot{X}_1 &= -X_1 + \gamma_1 g_1\left(1 - \Sigma_1 - \frac{1}{\gamma_1}X_1\right) f(T)X_1 \\
\dot{X}_2 &= -X_2 + g_2\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2 - \Sigma_2\right)X_2 \\
\dot{T} &= 1 - T - g_3(T)X_2
\end{aligned} \tag{6}$$

with $D = \Big\{(\Sigma_1, \Sigma_2, X_1, X_2, T) \mid X_i \geq 0, \Sigma_i \geq 0, \Sigma_1 + \frac{1}{\gamma_1}X_1 \leq 1, \Sigma_2 + X_2 \leq \frac{\gamma_2}{\gamma_1}X_1, 0 \leq T \leq 1\Big\}$. Note that D is positively invariant for (6). Letting $\Sigma_1 = \Sigma_2 = 0$ results in the reduced system

$$\begin{aligned}
\dot{X}_1 &= -X_1 + \gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right) f(T)X_1 \\
\dot{X}_2 &= -X_2 + g_2\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)X_2 \\
\dot{T} &= 1 - T - g_3(T)X_2
\end{aligned} \tag{7}$$

$$\Omega = \left\{(X_1, X_2, T) \mid 0 \leq X_1 \leq \gamma_1, 0 \leq X_2 \leq \frac{\gamma_2}{\gamma_1}X_1, 0 \leq T \leq 1\right\}$$

The existence of interior equilibria is dependent on T and the following condition is necessary.

(H3) there exists a unique $s = \lambda_T$ so that $\gamma_1 f(1) g_1(s) = 1$

We will refer to λ_T as the maximum inhibition break-even concentration. (H3) implies (H1) and $\lambda_o < \lambda_T$. Furthermore, (H3) implies that $\gamma_1 \beta g_1(s) = 1$ has unique solution λ_β for all $f(1) < \beta \leq 1$ with $\lambda_\beta|_{\beta=1} = \lambda_o$, $\lambda_\beta|\beta = f(1) = \lambda_T$, an $\mathrm{d}\lambda'(\beta) < 0$.

The Jacobian of (7) is

$$J = \begin{pmatrix} J_{11} & 0 & J_{13} \\ J_{21} & J_{22} & 0 \\ 0 & J_{32} & J_{33} \end{pmatrix}$$

with $J_{11} = -1 + g_1'\left(1 - \frac{1}{\gamma_1}X_1\right)X_1 f(T) + \gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right) f(T)$, $J_{13} = \gamma_1 g_1\Big(1 - \frac{1}{\gamma_1}X_1\Big)X_1 f'(T)$, $J_{21} = \frac{\gamma_2}{\gamma_1}g_2'\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)X_2$, $J_{22} = -1 - g_2'\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)X_2 + g_2\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)$, $J_{32} = -g_3(T)$, and $J_{33} = -1 - g_3'(T)X_2$.

3.1. **Equilibria on the boundary of Ω.** Since $X_1 = 0$ implies $X_2 = 0$, which implies $T = 1$, there exist two equilibrium on the boundary of Ω, $R_o = (0, 0, 1)$ and, provided that $\lambda_T < 1$, $R_1 = (\bar{X}_1, 0, 1)$, where $\bar{X}_1 = \gamma_1(1 - \lambda_T)$.

At R_o, $J_{13} = J_{21} = 0$ and the eigenvalues of J are on the main diagonal with $J_{11} = \gamma_1 g_1(1) f(1) - 1$ and $J_{22} = J_{33} = -1$. Hence, if $\lambda_T > 1$, all eigenvalues are negative, and R_o is locally asymptotically stable. If $\lambda_T < 1$, then R_o is a saddle with $dim W^s(R_o) = 2$ corresponding to the (X_2, T) plane.

Assuming that $\lambda_T < 1$, we find that at R_1, $J_{21} = 0$ and the eigenvalues are given by the main diagonal of J, $J_{11} = -g_1'\left(1 - \frac{1}{\gamma_1}\bar{X}_1\right)\bar{X}_1 f(1) < 0$, $J_{33} = -1$, and $J_{22} = g_2\left(\frac{\gamma_2}{\gamma_1}\bar{X}_1\right) - 1$. Thus, R_1 is locally asymptotically stable if either $g_2(s) < 1$ for all $s \geq 0$ or $\frac{\gamma_2}{\gamma_1}\bar{X}_1 \notin (\sigma_1, \sigma_2)$. If $\frac{\gamma_2}{\gamma_1}\bar{X}_1 \in (\sigma_1, \sigma_2)$, R_1 is a saddle with $dim W^s(R_1) = 2$ corresponding to the (X_1, T) plane.

Theorem 3.1. *(Global stability of R_o) Assume (H1), (H2), and (H3) hold and either $\lambda_o > 1$ or $1 - \frac{\sigma_1}{\gamma_2} < \lambda_o < 1 < \lambda_T$. Then $\lim_{t\to\infty} X_1(t) = \lim_{t\to\infty} X_2(t) = 0$ and $\lim_{t\to\infty} T(t) = 1$.*

Proof. Since f is decreasing and $0 \leq T \leq 1$, $f(1) \leq f(T) \leq 1$. Consequently, $\dot{X}_1 \leq X_1\left(\gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right) - 1\right)$. If $\lambda_o > 1$, then $\lim_{t\to\infty} X_1(t) = 0$. Since $0 \leq X_2(t) \leq \frac{\gamma_2}{\gamma_1}X_1(t)$, $\lim_{t\to\infty} X_2(t) = 0$. This implies $\lim_{t\to\infty} T(t) = 1$.

If $\gamma_1 m_1 f(1) > 1$ and $1 - \frac{\sigma_1}{\gamma_2} < \lambda_o < 1 < \lambda_T$, then from Lemma 2.1 it follows that $\limsup_{t\to\infty} X_1(t) \leq \gamma_1(1 - \lambda_o)$ and $\lim_{t\to\infty} X_2(t) = 0$. Therefore, $\lim_{t\to\infty} T(t) = 1$. $\lambda_T > 1$ implies $\frac{1}{\gamma_1 f(1)} > g_1(1)$. There exists an $\epsilon > 0$ so that $\frac{1}{\gamma_1 f(1-\epsilon)} > g_1(1)$. For t sufficiently large, $T(t) > 1 - \epsilon$ and $f(T(t)) < f(1 - \epsilon)$. This implies $\dot{X}_1 \leq X_1\left(\gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right) f(1-\epsilon) - 1\right)$. Let $\lambda(\epsilon)$ be the solution to $g_1(s) = \frac{1}{\gamma_1 f(1-\epsilon)}$. Then $\lambda(\epsilon) > 1$, and $\lim_{t\to\infty} X_1(t) = 0$ follows directly from Lemma 2.1. □

Next, we see that the behavior of the inhibition-free model is preserved if both break-even concentrations λ_o and λ_T satisfy the conditions given in (P2).

Theorem 3.2. *(Global stability of R_1) Assume (H1), (H2), and (H3) hold. If $1 - \frac{\sigma_1}{\gamma_2} < \lambda_o < \lambda_T < 1$, then $\lim_{t\to\infty} X_1(t) = \gamma_1(1 - \lambda_T)$, $\lim_{t\to\infty} X_2(t) = 0$ and $\lim_{t\to\infty} T(t) = 1$.*

Proof. As before $f(T) \leq 1$ gives $\dot{X}_1 \leq X_1\left(\gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right) - 1\right)$. Lemma 2.1 implies $\limsup_{t\to\infty} X_1(t) \leq \gamma_1(1 - \lambda_o)$ and $\lim_{t\to\infty} X_2(t) = 0$, and thus $\lim_{t\to\infty} T(t) = 1$. Since $\lambda_T < 1$, $\gamma_1 g_1(1) f(1) > 1$, there is an $\epsilon > 0$ so that for all $0 < \eta < \epsilon$, $\gamma_1 g_1(1) f(1 - \eta) > 1$. For t sufficiently large, $T(t) > 1 - \eta$ and $f(T(t)) < f(1 - \eta)$. Thus,

$$\dot{X}_1 \leq X_1\left(\gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right) f(1-\eta) - 1\right)$$

This implies, $\limsup X_1(t) \leq \gamma_1(1 - \lambda(\eta))$, where $g_1(\lambda(\eta)) = \frac{1}{\gamma_1 f(1-\eta)}$. Clearly, $\lim_{\eta\to 0} \lambda(\eta) = \lambda_T$. Thus, $\limsup_{t\to\infty} X_1(t) \leq \gamma_1(1 - \lambda_T)$. On the other hand, $\dot{X}_1 \geq X_1\left(\gamma_1 f(1) g_1\left(1 - \frac{1}{\gamma_1}X_1\right) - 1\right)$. Therefore, $\liminf_{t\to\infty} X_1(t) \geq \gamma_1(1 - \lambda_T)$. □

3.2. **Equilibria in the interior of Ω.** Define $F(T) = (\gamma_1 f(T))^{-1}$ and $G_i(T) = g_1\left(1 - \frac{1}{\gamma_2}\left(\sigma_i + \frac{1-T}{g_3(T)}\right)\right)$, $i = 1, 2$. Any interior equilibrium $R_c^i = (X_1, X_2, T)$ has to satisfy the equations

$$G_i(T) = F(T) \qquad X_2 = \frac{1 - T}{g_3(T)} \qquad X_1 = \frac{\gamma_1}{\gamma_2}(\sigma_i + X_2) \tag{8}$$

It is easy to see that $G_i'(T) > 0$ and $G_i''(T) > 0$ for $T \in (0,1)$. $G_i(T) = 0$ for $\frac{1-T}{g_3(T)} = \gamma_2 - \sigma_i$. Since $h(T) = \frac{1-T}{g_3(T)}$ is decreasing in $(0,1)$, $h(1) = 0$, and $\lim_{T\to 0^+} h(T) = \infty$, $h(T) = \gamma_2 - \sigma_i$ has a unique solution $\tau_i \in (0,1)$ provided that $\gamma_2 - \sigma_i > 0$. Consequently, any solution $T \in (0,1)$ of $G_i(T) = F(T)$ must lie in $(\tau_i, 1)$. Assume that

(H4) $f''(T)f(T) < 2[f'(T)]^2$ for $T \in (0,1)$

This condition is satisfied by exponential functions $f(T) = exp(-\mu T)$ with $\mu > 0$, which are frequently used to describe inhibition. (H4) ensures that $F(T)$ is concave up in $(0,1)$. Since G_i is concave down, equation $G_i(T) = F(T)$ can have at most two solutions in $(\tau_i, 1)$. The solutions and corresponding equilibria will be denoted $R_c^{(i,*)}$ and $R_c^{(i,**)}$, with the understanding that $T^{(i,\star)} < T^{(i,\star\star)}$.

Lemma 3.3. *Assume (H1)-(H4) hold. If* $1 - \frac{\sigma_2}{\gamma_2} < \lambda_o < \lambda_T < 1 - \frac{\sigma_1}{\gamma_2}$, *then* $G_1(T) = F(T)$ *has a unique solution* $T^{(1,\star)}$ *in* $(\tau_1, 1)$ *while* $G_2(T) = F(T)$ *has no solution in* $(0,1)$.

Proof. Since $\frac{1}{\gamma_1} < F(T) < \frac{1}{\gamma_1 f(1)}$ for $0 < T < 1$, any solution of $G_i(T) = F(T)$ must be such that $\lambda_o < 1 - \frac{1}{\gamma_2}(\sigma_i + h(T)) < \lambda_T$ or

$$\gamma_2(1-\lambda_T) - \sigma_i < h(T) \overset{(*)}{<} \gamma_2(1-\lambda_o) - \sigma_i \tag{9}$$

$1 - \frac{\sigma_2}{\gamma_2} < \lambda_o$ is equivalent to $\gamma_2(1-\lambda_o) - \sigma_2 < 0$. Since $h(T) > 0$ for $T \in (0,1)$, $(*)$ in (9) does not hold for $i = 2$ and $G_2(T) = F(T)$ has no solution.

$\lambda_T < 1 - \frac{\sigma_1}{\gamma_2}$ implies $\gamma_2 - \sigma_1 > 0$. Thus $h(T) = \gamma_2 - \sigma_1$ has a unique solution $\tau_1 \in (0,1)$, for which $F(\tau_1) > 0 = G_1(\tau_1)$. On the other hand, $G_1(1) = g_1(1 - \frac{\sigma_1}{\gamma_2}) > g_1(\lambda_T) = F(1)$. Since both G_1 and F are continuous on $(\tau_1, 1)$, $G_1(T) = F(T)$ has at least one solution in $(\tau_1, 1)$. The uniqueness of the solution follows from the concavity of F and G_1. □

Theorem 3.4. *(Global stability of* $R_c^{(1,\star)}$*) Assume that (H1)-(H4) hold. If* $1 - \frac{\sigma_2}{\gamma_2} < \lambda_o < \lambda_T < 1 - \frac{\sigma_1}{\gamma_2}$, *then (7) has a unique interior equilibrium* $R_c^{(1,\star)}$ *in* Ω *and any solution of (7) converges to* $R_c^{(1,\star)}$.

Proof. Lemma 3.3 guarantees the existence of a unique solution $T^{(1,\star)}$ for $G_1(T) = F(T)$ only. Let $X_2^{(1,\star)} = h(T^{(1,\star)})$ and $X_1^{(1,\star)} = \frac{\gamma_1}{\gamma_2}(\sigma_1 + h(T^{(1,\star)}))$.

Assume that $(X_1(t), X_2(t), T(t))$ is a solution of (7). It suffices to show that $\lim_{t\to\infty} T(t) = T^{(1,\star)}$. Since $f(1) < f(T) < 1$, Lemma 2.1 assures that $\gamma_2(1-\lambda_T) - \sigma_1 \le \liminf X_2 \le \limsup X_2 \le \gamma_2(1-\lambda_o) - \sigma_1$. For $\epsilon > 0$ there is an $s_1(\epsilon) > 0$ such that for all $t > s_1$, $\alpha_1 \le X_2(t) \le \beta_1$, where $\alpha_1 = \gamma_2(1-\lambda_T) - \sigma_1 - \epsilon > 0$ and $\beta_1 = \gamma_2(1-\lambda_o) - \sigma_1 + \epsilon$. Let μ_1 and ν_1 be the solutions to $h(T) = \alpha_1$ and $h(T) = \beta_1$, respectively. Since for $t > s_1$, $1 - T - g_3(T)\beta_1 \le \dot{T} \le 1 - T - g_3(T)\alpha_1$, we obtain that $\nu_1 \le \liminf T(t) \le \limsup T(t) \le \mu_1$. Hence, for $\eta > 0$ there is a $t_1(\eta) > 0$ such that for all $t > t_1$, $0 < \nu_1 - \eta \le T(t) \le \mu_1 + \eta < 1$. Define $\zeta_1 = \mu_1 + \eta$ and $\xi_1 = \nu_1 - \eta$, and denote the solutions to $g_1(s) = F(\zeta_1)$ and $g_1(s) = F(\xi_1)$ by $\lambda(\zeta_1)$ and $\lambda(\xi_1)$, respectively. Then $\lambda_o < \lambda(\xi_1) < \lambda(\zeta_1) < \lambda_T$.

For $n > 1$, let $\alpha_n = \gamma_2(1 - \lambda(\zeta_{n-1})) - \sigma_1 - \epsilon$, $\beta_n = \gamma_2(1 - \lambda(\xi_{n-1})) - \sigma_1 + \epsilon$, μ_n such that $h(\mu_n) = \alpha_n$, ν_n so that $h(\nu_n) = \beta_n$, $\zeta_n = \mu_n + \eta$ and $\xi_n = \nu_n - \eta$, and label the solutions to $g_1(s) = F(\zeta_n)$ and $g_1(s) = F(\xi_n)$ by $\lambda(\zeta_n)$ and $\lambda(\xi_n)$,

respectively.

Claim 1: $\xi_n \le T(t) \le \zeta_n$ for t sufficiently large.
Assume that $\xi_{n-1} \le T(t) \le \zeta_{n-1}$ for $n > 1$. Then

$$\begin{aligned} X_1(\gamma_1 g_1(1-\tfrac{1}{\gamma_2}X_1)f(\zeta_{n-1})-1) &\le \dot{X}_1 \le X_1(\gamma_1 g_1(1-\tfrac{1}{\gamma_2}X_1)f(\xi_{n-1})-1) \\ \dot{X}_2 &= X_2(g_2(\tfrac{\gamma_2}{\gamma_1}X_1 - X_2)-1) \end{aligned}$$

According to Lemma 2.1 there is an $s_n > 0$ such that for all $t > s_n$, $\alpha_n \le X_2(t) \le \beta_n$. As a result, for $t > s_n$, $1 - T - g_3(T)\beta_n \le \dot{T} \le 1 - T - g_3(T)\alpha_n$. Hence, there is a $t_n > 0$ so that for $t > t_n$, $\xi_n \le T(t) \le \zeta_n$.

Claim 2: The sequences $\{\zeta_n\}$ and $\{\xi_n\}$ are monotone and bounded, and thus convergent.
From the definition of ζ_n, μ_n, α_n, and $\lambda(\zeta_n)$, we see that $\zeta_n > \zeta_{n+1}$ iff $\mu_n > \mu_{n+1}$ iff $\alpha_{n+1} > \alpha_n$ iff $\lambda(\zeta_{n-1}) > \lambda(\zeta_n)$ where $\lambda(\zeta_0) = \lambda_T$. Thus, $\zeta_1 > \zeta_2$ follows from $\lambda_T > \lambda(\zeta_1)$. Assume that $\zeta_{n-1} > \zeta_n$. Then $F(\zeta_{n-1}) > F(\zeta_n)$ which leads to $\lambda(\zeta_{n-1}) > \lambda(\zeta_n)$. The boundedness follows from $\zeta_1 < 1$ and $\zeta_n > 0$. The monotonicity and boundedness of $\{\xi_n\}$ can be deduced similarly.

Denote the limits of $\{\zeta_n\}$ and $\{\xi_n\}$ by ζ and ξ, respectively. Then,

$$\begin{aligned} G_1(\zeta) &= g_1\left(1-\frac{1}{\gamma_2}(\sigma_1+h(\zeta))\right) = g_1\left(1-\frac{1}{\gamma_2}\left(\sigma_1+\lim_{n\to\infty}h(\mu_n)+O(\eta)\right)\right) \\ &= \lim_{n\to\infty} g_1\left(1-\frac{1}{\gamma_2}(\sigma_1+\alpha_n+O(\eta))\right) \end{aligned}$$

On the other hand,

$$\begin{aligned} F(\zeta) &= \lim_{n\to\infty} g_1(\lambda(\zeta_n)) = \lim_{n\to\infty} g_1\left(1-\frac{1}{\gamma_2}(\sigma_1+\alpha_{n+1})-\epsilon\right) \\ &= \lim_{n\to\infty} g_1\left(1-\frac{1}{\gamma_2}(\sigma_1+\alpha_n)-\epsilon\right) \end{aligned}$$

Since ϵ and η were arbitrary, we may let $\epsilon \to 0$ and $\eta \to 0$, and see that $G_1(\zeta) = F(\zeta)$. Similarly, $G_1(\xi) = F(\xi)$. Since the solution of $G_1(T) = F(T)$ is unique, we conclude that $\zeta = \xi = T^{(1,\star)}$. As a result, $\lim_{t\to\infty} T(t) = T^{(1,\star)}$. □

Other interior equilibria may exist. Necessary conditions for the existence of an interior equilibrium are $\gamma_2 - \sigma_i > 0$ and $F(0) < G_i(1)$ which is equivalent to $\lambda_o < 1 - \frac{\sigma_i}{\gamma_2}$. If this holds, then $G_i(T) = F(T)$ has a unique solution provided that $G_i(1) > F(1)$. This is equivalent to $\lambda_T < 1 - \frac{\sigma_i}{\gamma_2}$. If $G_i(1) < F(1)$, then $G_i(T) = F(T)$ may have no, one (degenerate), or two solutions, as illustrated in figure 2. The existence of interior equilibria and global stability results are summarized in table 1.

Theorem 3.5. *(i) If $R_c^{(1,\star)}$ exists, it is locally asymptotically stable.*
(ii) If $R_c^{(1,\star\star)}$ exists, it is unstable.
(iii) If $R_c^{(2,\star)}$ exists, it is unstable.
(iv) If $R_c^{(2,\star\star)}$ exists, it is locally asymptotically stable provided that

$$1 + g_1'\left(1-\frac{X_1^{(2,\star\star)}}{\gamma_1}\right)X_1^{(2,\star\star)} f(T^{2,\star\star}) + g_3'(T^{2,\star\star})X_2^{(2,\star\star)} > -g_2'(\sigma_2)X_2^{(2,\star\star)} \quad (10)$$

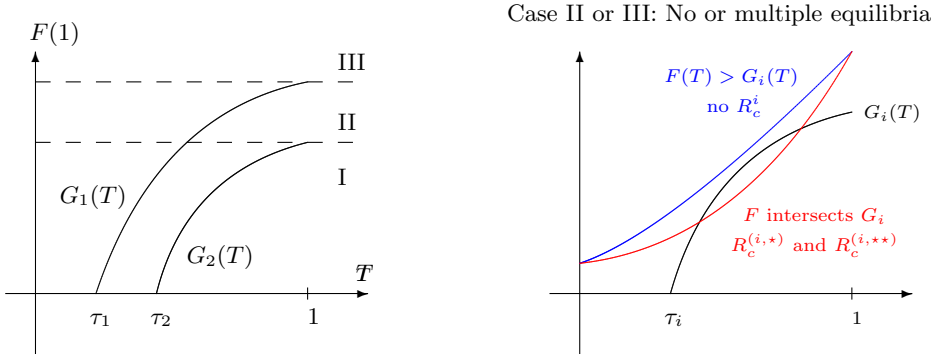

FIGURE 1. Case I: For $\lambda_T < 1 - \frac{\sigma_2}{\gamma_2}$ there exist two interior equilibria, in case II: $1 - \frac{\sigma_2}{\gamma_2} < \lambda_T < 1 - \frac{\sigma_1}{\gamma_2}$ or case III: $\lambda_T > 1 - \frac{\sigma_1}{\gamma_2}$ multiple equilibria may exist

	λ_T			
λ_o	I_1	I_2	I_3	I_4
I_1	$R_c^{(1,\star)}$ $R_c^{(2,\star)}$	$R_c^{(1,\star)}$ potential $R_c^{(2,\star)}$, $R_c^{(2,\star\star)}$	potential $R_c^{(1,\star)}$, $R_c^{(1,\star\star)}$ potential $R_c^{(2,\star)}$, $R_c^{(2,\star\star)}$	
I_2		$R_c^{(1,\star)}$ globally stable	potential $R_c^{(1,\star)}$, $R_c^{(1,\star\star)}$	
I_3			R_1 globally stable	R_o globally stable
I_4				R_o globally stable

TABLE 1. Existence of interior equilibria and global stability, where $I_1 = \left(0, 1 - \frac{\sigma_2}{\gamma_2}\right)$, $I_2 = \left(1 - \frac{\sigma_2}{\gamma_2}, 1 - \frac{\sigma_1}{\gamma_2}\right)$, $I_3 = \left(1 - \frac{\sigma_1}{\gamma_2}, 1\right)$, and $I_4 = (1, \infty)$

Proof. The Jacobian J is such that $J_{11} = -g_1'\left(1 - \frac{1}{\gamma_1}X_1\right)X_1 f(T) < 0$, $J_{13} = \gamma_1 g_1\left(1 - \frac{1}{\gamma_1}X_1\right)X_1 f'(T) < 0$, $J_{21} = \frac{\gamma_2}{\gamma_1}g_2'\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)X_2$, $J_{22} = -g_2'\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)X_2$, $J_{32} = -g_3(T) < 0$, and $J_{33} = -1 - g_3'(T)X_2 < 0$.

The characteristic polynomial is $Q(z) = z^3 + a_1 z^2 + a_2 z + a_3$ where $a_1 = -(J_{11} + J_{22} + J_{33})$, $a_2 = J_{11}J_{22} + J_{11}J_{33} + J_{22}J_{33}$, $a_3 = -(J_{11}J_{22}J_{33} + J_{13}J_{21}J_{32})$. The Routh-Hurwitz criterion states that all solutions of $Q(z) = 0$ have negative real parts if and only if $a_1 > 0$, $a_3 > 0$, and $a_1 a_2 > a_3$.

For $i = 1$, condition $a_3 > 0$ is equivalent to $G_1'(T) > -\frac{f'(T)}{\gamma_1[f(T)]^2}$. This only holds for $R_c^{(1,*)}$. Consequently, $R_c^{(1,**)}$ is unstable. For $R_c^{(1)}$, $J_{22} < 0$, which gives $a_1 > 0$. The remaining condition, $a_1 a_2 > a_3$, is $a_1 a_2 - a_3 = a_1 a_2 + J_{11}J_{22}J_{33} + J_{13}J_{21}J_{32} > 0$ because $a_1 a_2 + J_{11}J_{22}J_{33} > 0$ and $J_{13}J_{21}J_{32} > 0$. Thus, if $R_c^{(1,*)}$ exists, it is locally asymptotically stable while $R_c^{(1,**)}$ is unstable.

For $i = 2$, $a_3 > 0$ is equivalent to $G_1'(T) < -\frac{f'(T)}{\gamma_1[f(T)]^2}$. This only holds for $R_c^{(2,**)}$. $a_1 > 0$ is equivalent to (10). If $a_1 > 0$ and $a_3 > 0$, $a_1 a_2 > a_3$ holds. □

The possibility for bi-stability is preserved after the inclusion of inhibition. More precisely, if the inhibition-free system exhibited bi-stability, then so will system (5) if the maximum inhibition break-even concentration λ_T is sufficiently small.

Corollary 1. *If $0 < \lambda_o < \lambda_T < 1 - \frac{\sigma_2}{\gamma_2}$, then (7) has two interior equilibria, $R_c^{(1,\star)}$ and $R_c^{(2,\star)}$. $R_c^{(1,\star)}$ and R_1 are locally asymptotically stable while R_o and $R_c^{(2,\star)}$ are unstable.*

Proof. Since $1 - \frac{\sigma_2}{\gamma_2} < 1 - \frac{\sigma_1}{\gamma_2}$, the existence of $R_c^{(1,\star)}$ follows from Lemma 3.3. The existence of $E_c^{(2,\star)}$ can be shown as in Lemma 3.3, replacing σ_1 by σ_2. $G_i(1) > F(1)$ implies that there are no other interior equilibria. The local stability of the various equilibria was given in Theorem 3.5 and in section 3.1. □

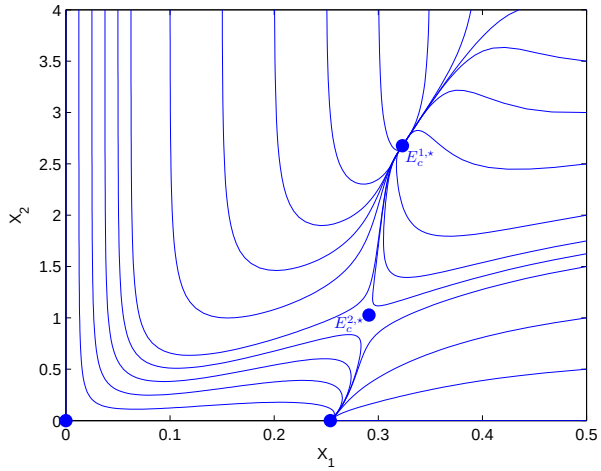

FIGURE 2. Bistable attractors for system (7). Functions and parameter values used: $g_1(x) = \frac{10x}{1+x}$, $g_2(x) = \frac{150x}{50+x} exp(-0.9x)$, $g_3(x) = \frac{0.5x}{1+x}$, $f(T) = exp(-0.5T)$, $\gamma_1 = 0.5$, $\gamma_2 = 5$.

3.3. **Existence of limit cycles.** Assume $f(T) = exp(-\mu T)$, $\mu > 0$ and $\lambda_o < 1 - \sigma_2/\gamma_2$. For $\lambda_T < 1 - \sigma_2/\gamma_2$, $G_1(T) = F(T)$ has one solution $T^{1,\star}$ in $(\tau_1, 1)$ and $G_2(T) = F(T)$ has one solution $T^{2,\star}$ in $(\tau_2, 1)$. Each equation has a second solution which is greater than 1.

As μ increases, λ_T increases, and there are two possibilities. Either $G_2(T) = F(T)$ has no solutions for μ sufficiently large and $G_1(T) = F(T)$ eventually has two solutions, or, an increase in μ causes the second solution of $G_2(T) = F(T)$ to move into $(\tau_2, 1)$ and that of $G_1(T) = F(T)$ into $(\tau_1, 1)$. With μ increasing further, the two solutions of either equation would merge and vanish.

Numerical results show that when the second solution of $G_2(T) = F(T)$ enters $(\tau_2, 1)$, the corresponding equilibrium $E_c^{(2,\star\star)}$ is first locally asymptotically stable,

but becomes unstable as μ increases. This loss of stability appears to be accompanied by a Hopf bifurcation and the existence of a stable limit cycle. In this case, system (5) exhibits the bi-stability of an interior equilibrium and a limit cycle (Fig. 3).

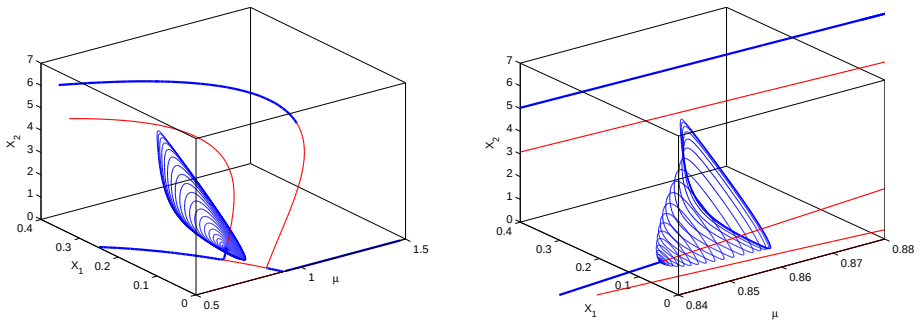

FIGURE 3. Bifurcation diagram for (7) for bifurcation parameter μ showing several bifurcations including a Hopf-bifurcation and bistabilities (thick lines correspond to stable equilibria while thin lines correspond to unstable equilibria, the periodic orbits are stable). Functions and parameter values as in Fig. 2 with $f(T) = exp(-\mu T)$ for μ ranging from 0.5 to 1.5.

4. **Conclusion.** The inhibition-free model exhibits three basic types of behavior: complete wash-out of all bacteria and accumulation of substrate in the reactor, accumulation of acids in the reactor so that one organism X_2 (methanogens) goes extinct, or coexistence of both cohorts of micro-organisms. The later is the behavior most desired in practical settings as it results in continuous biogas production.

In industrial settings, system failure is usually associated with hydraulic and/or organic overload, [13]. In terms of the constants in the unscaled model (1), the break-even concentration $\bar{\lambda}_o$ is the solution to $p_1 g_1(sS^{(0)}) = D$. Thus $\bar{\lambda}_o$ increases with D and decreases with $S^{(o)}$. A complete wash-out occurs when the hydraulic loading rate D is so large that the acid forming bacteria cannot grow fast enough to absorb the incoming material (no $\bar{\lambda}_o$ exists or it is greater that $S^{(o)}$.) A high hydraulic loading rate can also result in the acidification of the reactor. In our notation, the equilibria E_1 and R_1 correspond to an acidification scenario. Condition (P2) is equivalent to $\frac{c_1 p_2}{c_{12}}(S_1^{(0)} - \bar{\lambda}_o) < \bar{\sigma}_1$, where $\bar{\sigma}_1$ solves $g_2(s) = D$. This condition can hold for large values of D. Thus, a hydraulic overload causes either a complete washout of all bacteria or acidification.

Organic overload on the other hand can cause bistable conditions as described in (P4) or in Corollary 1. For $g_2(\bar{\sigma}_2) = D$, condition (P4) becomes $\bar{\sigma}_2 < c_1 c_2 p(S_1^{(0)} - \bar{\lambda}_o)$ which holds for large values of $S_1^{(0)}$. In this case, the system may either stabilize around the acidification state or around the state of coexistence. In practical setting with such operating conditions, a one-time addition of the micro-organism X_2 to the system may prevent acidification.

We have seen that it is possible to preserve the behavior of the toxin-free system when a toxin is introduced externally (theorems 3.1 - 3.4 and corollary 1). However,

if strong enough, the toxin may cause complete wash-out of bacteria, fluctuations (limit cycles) or additional bistabilities. This is similar to what has been seen for other chemostat models with inhibitors, [4, 10, 21]. The effects of a strong toxin are such that the system can have two stable coexistence steady states. In this case the long-term behavior of the system depends on the initial conditions of the system. For very strong toxins, the system exhibits the presence of a stable periodic solution and a stable coexistence steady state.

Previous models for anaerobic digestion have not shown periodic solutions. Thus, if data for biogas production show fluctuations with a periodic pattern, this might indicate the presence of a toxin in the system. Our numerical results show that a stable periodic solution can only exist in conjunction with a stable coexistence equilibrium. This implies that even if a toxin is present, altering the initial conditions can stabilize the system at the more desirable coexistence steady state.

Acknowledgments. The author would like to thank Gail Wolkowicz for her feedback on this work. This work was supported in part by a Faculty Development Grant from Dominican University.

REFERENCES

[1] "Agricultural Biogas Casebook," Great Lakes Regional Biomass Energy Program. Available from: http://www.cglg.org/biomass/pub/AgriculturalBiogasCasebook.pdf.

[2] D. J. Batstone, J. Keller, I. Angelidaki, S. Kalyhuzhnyi, S. G. Pavlosthathis, A. Rozzi, W. Sanders, H. Siegrist and V. Vavilin, (IWA Task Group on Modeling Anaerobic Digestion Processes), "Anaerobic Digestion Model No.1 (ADM1)," IWA Publishing, London, UK, 2002.

[3] T. Barkay and I. Wagner-Döbler, *Microbial transformations of mercury: Potentials, challenges, and achievements in controlling mercury toxicity in the environment*, Adv. Appl. Microbiol., **57** (2005), 1–40.

[4] J. P. Braselton and P. Waltman, *A competition model with dynamically allocated inhibitor production*, Math. Biosci., **173** (2001), 55–84.

[5] J. D. Bryers, *Structured modeling of anaerobic digestion of biomass particulates*, Biotechn. Bioeng., **27** (1984), 638–649.

[6] G. J. Butler and G. S. K. Wolkowicz, *A mathematical model of the chemostat with a general class of functions describing nutrient uptake*, SIAM J. Appl. Math., **45** (1985), 138–151.

[7] M. Gerardi, "The Microbiology of Anaerobic Digesters," Wiley Interscience, Hoboken, 2003.

[8] M. El Hajji, F. Mazenc and J. Harmand, *A mathematical study of syntrophic relationship of a model of anaerobic digestion process*, Math. Biosci. Eng., **7** (2010), 641–656.

[9] J. Hess and O. Bernard, *Design and study of a risk management criterion for an unstable wastewater treatment process*, J. of Process Control, **18** (2008), 71–79.

[10] S.-B. Hsu and P. Waltman, *A survey of mathematical models of competition with an inhibitor*, Math. Biosci., **187** (2004), 53–91.

[11] S. Jeyaseelan, *A simple mathematical model for anaerobic digestion process*, Wat. Sci. Tech., **35** (1997), 185–191.

[12] B. Li, *Global asymptotic behaviour of the chemostat: General response function and differential removal rates*, SIAM J. Appl. Math., **59** (1999), 411–422.

[13] G. Lyberatos and I. V. Skiadas, *Modelling of anaerobic digestion–a review*, Global Nest: The Int. J., **1** (1999), 63–76.

[14] L. Markus, *Asymptotically autonomous differential systems*, in "Contributions to the Theory of Nonlinear Oscillations," Princeton University Press, **3**, Princeton, NJ, (1953), 17–29.

[15] R. S. Oremland, C. W. Culbertson and M. Winfrey, *Methylmercurcy decomposition in sediments and bacterial cultures: Involvement of methanogens and sulfate reducers in oxidative demethylation*, Appl. Env. Microbiology, **57** (1991), 130–137.

[16] K. Pak and R. Bartha, *Products of mercury demethylation by sulfidogens and methanogens*, Bull. Environ. Contam. Toxicol., **61** (1998), 690–694.

[17] G. E. Powell, *Stable coexistence of syntrophic associations in continuous culture*, J. Chem. Tech. Biotechnol. B, **35** (1985), 46–50.
[18] H. Smith and P. Waltman, "The Theory of the Chemostat: Dynamics of Microbial Competition," Cambridge Studies in Mathematical Biology, **13**, Cambridge University Press, Cambridge, 1995.
[19] H. Thieme, *Convergence results and a Poincaré-Bendixson trichotomy for asymptotically autonomous differential equations*, J. of Math. Biology, **30** (1992), 755–763.
[20] G. S. K. Wolkowicz and Z. Lu, *Global Dynamics of a mathematical model of competition in the chemostat: General response functions and differential death rates*, SIAM J. Appl. Math., **52** (1992), 222–233.
[21] L. Zhu, X. Huang and H. Su, *Bifurcation for a functional yield chemostat when one competitor produces a toxin*, J. Math. Anal. Appl., **329** (2007), 891–903.

Appendix A. **Analysis of toxin-free model (2).** Consider system (2) replicated below and assume that (H1) and (H2) hold.

$$\begin{array}{lcll} \dot{S}_1 &=& 1 - S_1 - g_1(S_1)X_1 & S_1(0) \geq 0 \\ \dot{X}_1 &=& -X_1 + \gamma_1 g_1(S_1)X_1 & X_1(0) \geq 0 \\ \dot{S}_2 &=& -S_2 + \gamma_2 g_1(S_1)X_1 - g_2(S_2)X_2 & S_2(0) \geq 0 \\ \dot{X}_2 &=& -X_2 + g_2(S_2)X_2 & X_2(0) \geq 0 \end{array} \tag{2}$$

We see immediately that $X_i(t) \geq 0$ and can deduce $S_i(t) \geq 0$ from $\dot{S}_i|_{S_i=0} > 0$. The boundedness of solutions is guaranteed because for $\Sigma_1 = 1 - S_1 - \frac{1}{\gamma_1}X_1$ and $\Sigma_2 = -S_2 - X_2 + \frac{\gamma_2}{\gamma_1}X_1$ we obtain $\dot{\Sigma}_i = -\Sigma_i$. Also, $D = \{(S_1, X_1, S_2, X_2) |\ S_i \geq 0,\ X_i \geq 0, S_1 + \frac{1}{\gamma_1}X_1 \leq 1, S_2 + X_2 \leq \frac{\gamma_2}{\gamma_1}X_1\}$ is positively invariant for (2).

Proposition 1 can be seen intuitively by recognizing that the equations for (S_1, X_1) decouple from (S_2, X_2). The equations for (S_1, X_1) correspond to a basic chemostat model for which it is well known that if $\lambda_o > 1$, then $\lim_{t\to\infty} S_1(t) = 1$ and $\lim_{t\to\infty} X_1(t) = 0$. On the other hand, if $\lambda_o < 1$, then $\lim_{t\to\infty} S_1(t) = \lambda_o$ and $\lim_{t\to\infty} X_1(t) = \gamma_1(1 - \lambda_o)$. Assuming that $\lim_{t\to\infty} S_1(t) = \alpha$ and $\lim_{t\to\infty} X_1(t) = \beta$, and setting $S_1 = \alpha$ and $X_1 = \beta$ in (2), gives

$$\begin{aligned} \dot{S}_2 &= \Pi - S_2 - g_2(S_2)X_2 \\ \dot{X}_2 &= -X_2 + g_2(S_2)X_2 \end{aligned}$$

where $\Pi = \gamma_2 g_1(\alpha)\beta$. Chemostat equations with non-monotone response functions have been considered for much more general equations involving multiple species and substrates [6, 12, 20]). Under assumption (H2), the system has three equilibria in $\mathbb{R}^2_+$, $M_1 = (\Pi, 0)$, $M_c^1 = (\sigma_1, \Pi - \sigma_1)$, and $M_c^2 = (\sigma_2, \Pi - \sigma_2)$, where M_c^1 is always stable and M_c^2 is always stable. Thus one can see why Proposition 1 would hold.

A more formal analysis is given below using the theory of asymptotic autonomous differential equations, which was also used in section 3.

Proof. Proof of Proposition 1.

Replacing S_i by Σ_i in (2) and setting $\Sigma_i = 0$ reduces (2) to

$$\dot{X}_1 = -X_1\left(1-\gamma_1 g_1\left(1-\frac{1}{\gamma_1}X_1\right)\right) \tag{11}$$

$$\dot{X}_2 = -X_2\left(1-g_2\left(\frac{\gamma_2}{\gamma_1}X_1 - X_2\right)\right) \tag{12}$$

The region $\Omega = \left\{(X_1,X_2)|\ 0\le X_1\le\gamma_1,\ 0\le X_2\le\frac{\gamma_2}{\gamma_1}X_1\right\}$ is positively invariant for (11, 12).

This system can have up to four equilibria, $F_o = (0,0)$, $F_1 = (\gamma_1(1-\lambda_o),0)$ and $F_c^i = (\gamma_1(1-\lambda_o),\gamma_2(1-\lambda_o)-\sigma_i)$, where F_1 lies in Ω only if $\lambda_o < 1$ and $F_c^i \in \Omega$ provided that $\lambda_o < 1-\sigma_i/\gamma_2$. The local stability analysis of these equilibria immediately transfers to that of the corresponding equilibria of (2). Any global stability results only transfer if the conditions from the theory of asymptotic autonomous differential equations are satisfied. Among those one has to guarantee that no polycycle exists between the equilibria, [18, 19], which is reasoned below.

Equilibrium	Existence	Local stability
F_o	always	$\lambda_o > 1$
F_1	$\lambda_o < 1$	$\lambda_o > 1-\sigma_1/\gamma_2$ or $\lambda_o < 1-\sigma_2/\gamma_2$
F_c^1	$\lambda_o < 1-\sigma_1/\gamma_2$	always
F_c^2	$\lambda_o < 1-\sigma_2/\gamma_2$	never

TABLE 2. Conditions for existence and local stability of the equilibria of (11,12)

Since equation (11) is independent of X_2, $X_1(t)$ cannot be a periodic solution. This in turn implies that (11,12) can have no periodic solution. The set $\{(X_1,X_2)|\ X_1>0,\ X_2=0\}$ is positively invariant. Thus there can be no polycycle connecting F_o and F_1, or F_o, F_1 and F_c^2. Since F_c^1 is always locally asymptotically stable, it cannot be part of a polycycle. The Poincaré-Bendixson Theorem implies that when only one locally asymptotically equilibrium exists in Ω, it must be globally stable. This means that the above conditions for existence and local stability also give global stability as summarized here.

- $\lambda_o > 1$ implies that F_o is globally stable.
- If $1-\sigma_1/\gamma_2 < \lambda_o < 1$, F_1 is globally stable.
- If $1-\sigma_2/\gamma_2 < \lambda_o < 1-\sigma_1/\gamma_2$, F_c^1 is globally stable.

As a result, the corresponding equilibria of system (2) share the same stability properties. □

Received November 17, 2010; Accepted August 9, 2011.

E-mail address: mweederm@dom.edu