Mathematical Biosciences 296 (2018) 71–81

Contents lists available at ScienceDirect

Mathematical Biosciences

journal homepage: www.elsevier.com/locate/mbs

# On the intrinsic dynamics of bacteria in waterborne infections

Chayu Yang, Jin Wang*

*Department of Mathematics, University of Tennessee at Chattanooga, Chattanooga, TN 37403, USA*

ARTICLE INFO

*Keywords:*
Logistic growth
Allee effects
Compartment models

ABSTRACT

The intrinsic dynamics of bacteria often play an important role in the transmission and spread of waterborne infectious diseases. In this paper, we construct mathematical models for waterborne infections and analyze two types of nontrivial bacterial dynamics: logistic growth, and growth with Allee effects. For the model with logistic growth, we find that regular threshold dynamics take place, and the basic reproduction number can be used to characterize disease extinction and persistence. In contrast, the model with Allee effects exhibits much more complex dynamics, including the existence of multiple endemic equilibria and the presence of backward bifurcation and forward hysteresis.

## 1. Introduction

Waterborne infectious diseases remain a significant threat to public health throughout the world [3,28]. Common waterborne infections include cholera, typhoid fever, cryptosporidiosis, giardiasis, and many others; these can be caused by a variety of pathogenic microbes (bacteria, protozoa, etc.) in contaminated water. The World Health Organization (WHO) [46] estimates that such infections account for 3.6% of the total global burden of diseases, and lead to about 1.5 million human deaths each year. Even the most developed countries, such as the US, are occasionally plagued by these infections. For example, in 1993, a waterborne cryptosporidiosis outbreak in Milwaukee, Wisconsin led to an estimated 403,000 infected individuals, including 4400 people hospitalized. On a worldwide scale, the best known waterborne disease is perhaps cholera, caused by virulent strains of the Gram-negative bacterium *Vibrio cholerae* (*V. cholerae*). The past decade witnessed an increasing number of cholera outbreaks, including one of the largest cholera epidemics in modern history that took place in Haiti from 2010 to 2012 with more than 530,000 reported cases and over 7000 deaths [45].

Mathematical modeling, analysis, and simulation have long provided useful insight into epidemiology. In particular, a large number of mathematical models have been published for the dynamics of waterborne diseases [6,12,18,25,31–33,37,40,41,43,44]. Many of these models included both direct and indirect transmission pathways in order to better characterize the transmission pattern of waterborne infections. One major limitation of current modeling studies in waterborne diseases, however, is that the intrinsic dynamics of the waterborne pathogens are poorly addressed, leading to incomplete, and often, inadequate, understanding of the pathogen evolution and its impact on disease transmission and spread. For example, a standard assumption in the majority of cholera models, based on an early theory in cholera ecology [14], is that the Vibrios (i.e., *V. cholerae*) cannot sustain themselves in the absence of human contribution; e.g., shedding from infected individuals and inflow from contaminated sewage. The assumption allows a simple, often linear, representation of the rate of change for the bacterial density: a positive contribution from the infected human population, and a negative contribution due to natural death of the Vibrios. Such a representation considerably simplifies the mathematical analysis. Unfortunately, there have been strong evidences in recent ecological studies that the Vibrios can independently survive and multiply in various aquatic environments, including freshwater, estuaries and seawater [4,7,11,13]. Other waterborne pathogens, such as the bacterium *Escherichia coli*, have also been found to be capable of independently surviving in the aquatic environment [20]. These ecological findings demand new modeling efforts toward better understanding the intrinsic dynamics of waterborne pathogens (especially bacteria) and the connection between their environmental persistence and disease outbreaks.

The present paper aims to shed light on this important aspect of waterborne disease epidemiology, using mathematical modeling and analysis based on differential equations. To that end, we will incorporate nonlinear dynamics terms into the pathogen evolution equation, and we will focus on two types of intrinsic bacterial dynamics: quadratic growth, and cubic growth.

The quadratic growth, more commonly referred to as the logistic growth [19], is probably the most popular model to describe population changes, ranging from macroscopic to microscopic organisms.

* Corresponding author.
*E-mail address:* Jin-Wang02@utc.edu (J. Wang).

https://doi.org/10.1016/j.mbs.2017.12.005
Received 24 June 2017; Received in revised form 2 December 2017; Accepted 26 December 2017
Available online 29 December 2017
0025-5564/ 

Essentially, this model introduces a threshold for the total population, known as the carrying capacity, so that a population would exponentially grow initially but then stabilize at the carrying capacity. The model could reflect realistic constrains (such as lack of resources) on the population growth.

A significant departure from the logistic growth pattern, the cubic growth model is known to introduce Allee effects. An Allee effect refers to a correlation between individual fitness and population density [1,2]. Particularly, a strong Allee effect describes a population that exhibits positive growth at intermediate population density but declines when the population density is either too low or too high.

Allee effects have been well documented and extensively studied for the growth dynamics among animal populations [1,8,26]; related mathematical modeling work includes, for example, [9,10,15,23,39]. There are relatively few studies for Allee effects in populations of microorganisms such as bacteria and parasites [24]. Kadam and Velicer [22] reported laboratory measurements of the bacterium *Myxococcus xanthus* and found that it sporulates less efficiently at lower population densities and produces no spores at all below a minimum threshold density. Li et al. [30] demonstrated that bacterial populations in high-density biofilms are better able to generate a coordinated protective response against highly acidic conditions than are populations at low density, thus promoting the survival of the microbial species. Ji et al. [21] observed that a minimum population density is typically needed for some pathogenic microbes to initiate the expression of virulence factors necessary for the establishment of successful infections. In addition, Smith et al. [38] investigated Allee effects on the bacterial spread and survival through the engineered bacterium *E. coli*. In all these studies, it is observed that a minimal density, known as the Allee threshold, is required to initiate positive population development.

Given the significance of waterborne infections, the complications of their related dynamics, and the experimental evidence of the nontrivial growth patterns of waterborne pathogens, it is worthwhile to mathematically explore the details of the intrinsic bacterial dynamics and the impact on waterborne disease transmission. To that end, we organize the remainder of this paper as follows. In Section 2, we describe and analyze a waterborne disease model where the bacteria go through logistic growth. In Section 3, we modify the waterborne disease model by replacing the logistic growth with the cubic growth (i.e., growth with Allee effects) for the bacteria, and investigate the dynamics using both mathematical analysis and numerical simulation. In Section 4, we conclude the paper with some discussion.

## 2. Logistic growth

We consider the following equations that describe the transmission dynamics of a waterborne bacterial infection. We incorporate both direct (i.e., human-to-human) and indirect (i.e., environment-to-human) transmission pathways, each represented by a bilinear incidence. Our focus is the intrinsic dynamics of the bacteria in this process, represented by a logistic growth model here.

$$\begin{cases} \dot{S}(t) = \mu N - (\alpha I + \beta B)S - \mu S, \\ \dot{I}(t) = (\alpha I + \beta B)S - \mu I - \delta I, \\ \dot{R}(t) = \delta I - \mu R, \\ \dot{B}(t) = rB\left(1 - \dfrac{B}{k}\right) - \tau B + \xi I. \end{cases} \tag{1}$$

The parameter $N = S + I + R$ is the total population size of the host individuals with a constant birth and death rate $\mu$. The variables $S$, $I$ and $R$ represent the susceptible, infected and recovered individuals, respectively, whereas $B$ represents the concentration of the bacteria in the contaminated water. $\alpha$ and $\beta$ denote the direct and indirect transmission rates, respectively, $\delta$ is the recovery rate, $r$ is the bacterial intrinsic growth rate, $k$ is the carrying capacity, $\tau$ is the bacterial removal rate, and $\xi$ is the rate of contribution from an infected individual to the bacterial population in the environment (e.g., through shedding). We neglect the disease related mortality here. All these parameters are assumed to be positive.

It can be easily verified that the domain of biological interest

$$\Gamma = \{(S, I, R, B) \in \mathbb{R}^4_+ : \ S + I + R = N\}$$

is positively invariant with respect to the model (1).

There is a unique disease free equilibrium (DFE) at $X_0 = (N, 0, 0, 0)$. We proceed to determine the basic reproduction number $\mathscr{R}_0$ for this model. The compartments $I$ and $B$ are directly related to the disease. Using the notions in [42], the non-negative matrix $F$ that denotes the generation of new infections and the non-singular matrix $V$ that denotes the transfer among infectious compartments, are respectively given by

$$F = \begin{bmatrix} \alpha N & \beta N \\ \xi & r \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \mu + \delta & \\ & \tau \end{bmatrix}, \tag{2}$$

which then lead to the next-generation matrix

$$FV^{-1} = \begin{bmatrix} \dfrac{\alpha N}{\mu + \delta} & \dfrac{\beta N}{\tau} \\ \dfrac{\xi}{\mu + \delta} & \dfrac{r}{\tau} \end{bmatrix}. \tag{3}$$

It follows from (3) that the basic reproduction number can be represented by

$$\mathscr{R}_0 = \rho(FV^{-1}) = \frac{1}{2}\left(\frac{\alpha N}{\mu + \delta} + \frac{r}{\tau} + \sqrt{\left(\frac{\alpha N}{\mu + \delta} - \frac{r}{\tau}\right)^2 + \frac{4\xi\beta N}{\tau(\mu + \delta)}}\right), \tag{4}$$

where ρ denotes the spectral radius of the matrix $FV^{-1}$.

We comment here that the expression for the basic reproduction number is not unique [5,42]. For example, in the above we derived $\mathscr{R}_0$ by treating the intrinsic bacterial growth and host shedding as generation of new infections in $B$. If, instead, we treat these as transfer of infections and put them in the matrix $V$, then we obtain another basic reproduction number

$$\widetilde{\mathscr{R}_0} = \frac{\alpha N}{\mu + \delta} + \frac{\xi\beta N}{(\mu + \delta)(\tau - r)}$$

where we assume $\tau \neq r$. Through simple algebraic manipulation, we can show that $\mathscr{R}_0 > (=, <) 1$ if and only if $\widetilde{\mathscr{R}_0} > (=, <) 1$. That is, the two reproduction numbers are equivalent in characterizing disease risks.

### 2.1. Nontrivial equilibrium

A nontrivial equilibrium $X = (S, I, R, B)$ for system (1) satisfies

$$\mu N = (\alpha I + \beta B)S + \mu S, \tag{5}$$

$$(\alpha I + \beta B)S = (\mu + \delta)I, \tag{6}$$

$$\delta I = \mu R, \tag{7}$$

$$\xi I = \frac{r}{k}B^2 + (\tau - r)B. \tag{8}$$

Let $\theta = \frac{\mu N}{\mu + \delta}$ and cancel $S$ from (5) and (6), we obtain

$$\alpha I^2 + (\mu + \beta B - \alpha\theta)I - \beta\theta B = 0. \tag{9}$$

Since $I$ is nonnegative, we have $I = g(B)$, where

$$g(B) = \frac{\sqrt{(\mu - \alpha\theta + \beta B)^2 + 4\alpha\beta\theta B} - (\mu - \alpha\theta + \beta B)}{2\alpha}, \quad B \geq 0. \tag{10}$$

In addition, we have $B = p(I)$ from (9), where