

Stability Analysis of a Waterborne Infectious Disease Model with Infectious Saturation Effect on Bacterial Disease Transmission

Cemil Büyükdalı

Department of Mathematics, Faculty of Science, Van Yuzuncu Yil University, 65080 Van, Turkey

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Abstract: This paper considers a waterborne infectious disease model with infectious saturation effect on bacterial disease transmission. For this model we find sufficient conditions for global stability of disease free equilibrium. We verify the result using appropriate numerical simulations.

Keywords: Compartmental models, waterborne infectious diseases, infectious saturation effect, global stability.

1 Introduction

Waterborne diseases are the major health problem for many developing countries around the world. Cholera is an infectious disease caused by the bacterium *Vibrio Cholerae* that can be transmitted indirectly through environment to human contact (i.e. via ingesting food or water contaminated by cholera) or directly human to human contact. This bacterium releases an enterotoxin that can cause a watery diarrhea and severe dehydration, and it can kill infected humans unless treated. Fundamental treatments such as oral rehydration solutions or intravenous fluids are highly effective, but cholera cases have been increasing globally since 2005 [1] and this disease is still active in Africa, Southeast Asia, Haiti and Latin America. Recent significant cholera outbreaks took place in South Africa (2000-2001) [2], Angola (2006) [3], Zimbabwe (2008-2009) [4], Haiti (2010-2012) [5], South Sudan (2014). There were 1.3-4 millions cases of cholera, and 21.000-143.000 of deaths annually between 2008 and 2012 [1]. These severe cholera outbreaks have shown inadequacy of our knowledge.

Mathematical modeling of an infectious disease plays an important role to predict asymptotic behavior of epidemic dynamics and control of the disease. Capasso and Paveri-Fontana in 1979 first proposed an epidemic model [6] to study the 1973 cholera epidemic in the

European Mediterranean region. The model consists of two compartments: the concentration of the bacteria in the water and the density of the infected humans. Following this work, Codeço in 2001 proposed an epidemic model [7] by adding an environmental component B i.e. the *Vibrio Cholerae* concentration in the water supply into a regular Susceptible - Infectious - Recovered (SIR) compartmental model with saturation effect $\lambda(B) = B/(1+B)$ on the transmission of disease caused by waterborne bacteria. Hartley, Morris and Smith in 2006 extended Codeço's model based on laboratory observations by including highly infective *Vibrio Cholerae* [8]. Also, in 2011 Mukandavire et al. [9] simplified Hartley's model to study the 2008-2009 cholera outbreak in Zimbabwe by considering both human-to-human and environment-to-human transmission pathways. Moreover, in 2010 Tien and Earn [10] proposed a waterborne disease model with multiple transmission pathways without considering any saturation effect. In 2011 Wang and Modnak [11] proposed a cholera epidemiological model with control measures. Many researchers have proposed and investigated various waterborne epidemiological models. Some of the important models can be found, for example, in [12]-[28]. However, there is no work, as far as we are aware of, that has been devoted to the saturation effect of infectious population on the transmission of disease caused by waterborne bacteria.

* Corresponding author e-mail: cbuyukadali@yyu.edu.tr

In this paper we propose a waterborne bacterial infection model with saturation effect of infectious population on the transmission of disease caused by waterborne bacteria. The total population size $N(t)$ is divided into two compartments: susceptible $S(t)$ and infectious with symptoms $I(t)$ at time $t \geq 0$. Furthermore, we consider a compartment $B(t)$ that reflects the bacterial concentration at time t . We assume positive natural death rate μ . Susceptible individuals can become infected by contact with infected individuals at rate α . Susceptible individuals can become infected with cholera by contact with contaminated sources at rate $\beta B(t)/(1+I(t))$ where $\beta > 0$ is the ingestion rate of the bacteria through contaminated sources. We assume nonnegative death rate δ caused by infection. Each infected individual contributes to the increase of the bacterial concentration at rate ξ . On the other hand, the bacterial concentration can decrease at mortality rate γ . With these assumption we have the following mathematical model:

$$\begin{aligned} S'(t) &= \Lambda - \left(\alpha I + \frac{\beta B}{1+I}\right)S - \mu S, \\ I'(t) &= \left(\alpha I + \frac{\beta B}{1+I}\right)S - \delta I - \mu I, \\ B'(t) &= \xi I - \gamma B. \end{aligned} \quad (1)$$

Let us emphasize that the total population size $N(t) = S(t) + I(t)$ satisfies the differential equation

$$N'(t) = \Lambda - \mu N(t) - \delta I.$$

One can see easily that for sufficiently large $t \geq 0$ we have $N(t) \leq \Lambda/\mu$ and $B(t) \leq \xi\Lambda/(\mu\gamma)$. Thus the set

$$\Omega = \left\{ (S, I, B) \in \mathbb{R}_+^3 : S + I \leq \frac{\Lambda}{\mu}, 0 \leq B \leq \frac{\xi\Lambda}{\mu\gamma} \right\}$$

is positively invariant region.

The organization of the paper is as follows: In Section 2, we find equilibriums and basic reproduction number of system (1). In Section 3, we show the global stability of the disease-free equilibrium. In Section 4, we verify the result using appropriate numerical simulations.

2 Equilibriums and Reproduction Number

In this section we find equilibriums and basic reproduction number of system (1).

The equilibrium points of system (1) satisfy the following equations

$$\begin{aligned} \text{(i)} \quad & \Lambda - \left(\alpha I + \frac{\beta B}{1+I}\right)S - \mu S = 0, \\ \text{(ii)} \quad & \left(\alpha I + \frac{\beta B}{1+I}\right)S - \delta I - \mu I = 0, \\ \text{(iii)} \quad & \xi I - \gamma B = 0. \end{aligned} \quad (2)$$

From these equations we obtain

$$B = \frac{\xi I}{\gamma}, \quad S = \frac{\Lambda}{\mu} - \frac{(\delta + \mu)I}{\mu}. \quad (3)$$

If $I = 0$, then it can be seen easily that the only disease-free equilibrium of system (1) is $E_0 = (S_0, I_0, B_0) = (\Lambda/\mu, 0, 0)$.

Following the paper by van den Driessche and Watmough [29], it is easy to obtain that

$$F = \begin{bmatrix} (\alpha\Lambda/\mu) & (\beta\Lambda/\mu) \\ 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} (\delta + \mu) & 0 \\ -\xi & \gamma \end{bmatrix}$$

It follows that:

$$V^{-1} = \begin{bmatrix} 1/(\delta + \mu) & 0 \\ 1/(\gamma(\delta + \mu)) & 1/\gamma \end{bmatrix}$$

Hence,

$$FV^{-1} = \begin{bmatrix} \frac{\alpha\Lambda\gamma + \beta\xi\Lambda}{\mu\gamma(\delta + \mu)} & \frac{\beta\Lambda}{\mu\gamma} \\ 0 & 0 \end{bmatrix}$$

The spectrum radius of FV^{-1} gives the basic reproduction number

$$R_0 = \frac{\left(\alpha + \frac{\beta\xi}{\gamma}\right)\Lambda}{\mu(\delta + \mu)}.$$

Suppose $I \neq 0$. If we put (3) into (2)(ii), we obtain a second order polynomial of I of the form

$$I^2 + a_1 I + a_2 = 0,$$

where

$$\begin{aligned} a_1 &= -\frac{\Lambda}{\mu} + \left(1 + \frac{\beta\xi}{\alpha\gamma}\right) + \frac{\mu}{\alpha} = -\frac{\Lambda}{\mu} + \frac{\mu}{\alpha} + \frac{(\delta + \mu)\mu}{\alpha\Lambda} \\ a_2 &= \frac{\mu}{\alpha}(1 - R_0) \end{aligned}$$

Hence, by Descartes rule of signs one can easily see that there is exactly one positive real endemic equilibrium whenever $R_0 > 1$ which is $E_* = (S_*, I_*, B_*)$ where

$$I_* = \frac{-a_1 + \sqrt{a_1^2 - 4a_2}}{2} \quad (4)$$

and S_* and B_* can be found directly using (3).

3 Global stability of the disease free equilibrium

Let us first introduce the local stability of disease free equilibrium E_0 of system (1).

Theorem 3.1. Suppose $R_0 < 1$. Then disease-free equilibrium E_0 of system (1) is locally asymptotically stable.

Proof. Jacobian matrix of system (1) at $E_0 = (\Lambda/\mu, 0, 0)$ is

$$J(E_0) = \begin{bmatrix} -\mu & -\alpha\Lambda/\mu & -\beta\Lambda/\mu \\ 0 & \left(\frac{\alpha\Lambda}{\mu} - \mu - \delta\right) & \beta\Lambda/\mu \\ 0 & \xi & -\gamma \end{bmatrix}$$

which has eigenvalues $\lambda_1 = -\mu < 0$,

$$\lambda_{2,3} = -\frac{q}{2} \pm \frac{\sqrt{q^2 - 4\gamma(\mu + \delta)(1 - R_0)}}{2} < 0,$$

where $q = (\gamma + \mu + \delta - \alpha\Lambda/\mu)$ whenever $R_0 < 1$. Hence, all eigenvalues are negative and the disease-free equilibrium E_0 of system (1) is locally asymptotically stable. The proof is complete. \square

Now we can establish global stability of disease-free equilibrium of system (1).

Theorem 3.2. Suppose $R_0 \leq 1$. Then disease-free equilibrium E_0 of system (1) is globally asymptotically stable on Ω .

Proof. Let us define the Lyapunov function

$$L(S, I, B) = \gamma I + \beta \frac{\Lambda}{\mu} B.$$

Then

$$\begin{aligned} \frac{dL}{dt} &= \gamma \frac{dI}{dt} + \beta \frac{\Lambda}{\mu} \frac{dB}{dt} \\ &= \gamma \left[\left(\alpha I + \frac{\beta B}{1+I} \right) S - \delta I - \mu I \right] + \beta \frac{\Lambda}{\mu} [\xi I - \gamma B] \\ &\leq \gamma \left[\left(\alpha I + \frac{\beta B}{1+I} \right) \left(\frac{\Lambda}{\mu} - I \right) - \delta I - \mu I \right] \\ &\quad + \beta \frac{\Lambda}{\mu} [\xi I - \gamma B] \\ &= \left((\alpha\gamma + \beta\xi) \frac{\Lambda}{\mu} - \gamma(\delta + \mu) \right) I - \alpha\gamma I^2 - \frac{\gamma\beta BI}{1+I} \\ &\quad - \gamma\beta \frac{\Lambda}{\mu} \left(1 - \frac{1}{1+I} \right) B \\ &\leq -\gamma(\delta + \mu)(1 - R_0)I - \alpha\gamma I^2 \leq 0 \end{aligned}$$

in Ω . Moreover, $dL/dt = 0$ is satisfied only if $I = 0$ where the only positively invariant set is $\{E_0\}$. Therefore, by LaSalle invariance principle [30], E_0 is globally asymptotically stable on Ω . \square

4 Simulations

Let us consider several situations to verify the result. In Figure 1 and Figure 2 we verify that when $R_0 \leq 1$ the disease-free equilibrium E_0 is asymptotically stable as in Theorem 3.2 while in Figure 3 we see that the endemic equilibrium E_* is asymptotically stable if $R_0 > 1$.

5 Conclusion

We consider waterborne infectious disease model with infectious saturation effect on bacterial disease transmission in system (1). For this model we find the global stability of disease free equilibrium E_0 using

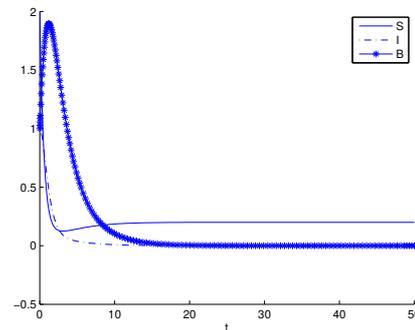


Fig. 1: We have stability of disease-free equilibrium $E_0 = (S_0, I_0, B_0) = (0.2, 0, 0)$ for $\mu = 1, \Lambda = 0.2, \alpha = 0.5, \beta = 0.5, \delta = 0.5, \gamma = 0.5, \xi = 2, R_0 = 0.333 < 1$.

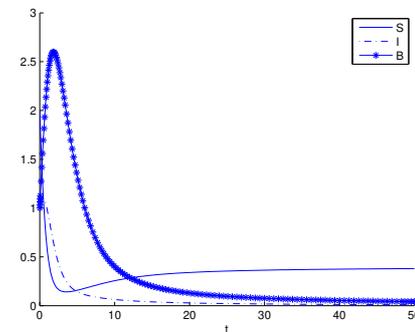


Fig. 2: We have stability of disease-free equilibrium $E_0 = (S_0, I_0, B_0) = (0.4, 0, 0)$ for $\mu = 0.5, \Lambda = 0.2, \alpha = 0.5, \beta = 0.5, \delta = 0.5, \gamma = 0.5, \xi = 2, R_0 = 1$.

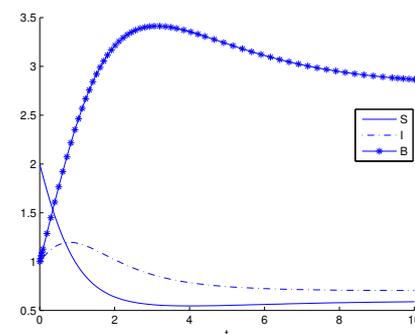


Fig. 3: We have stability of endemic equilibrium $E_* = (S_*, I_*, B_*) = (0.5969, 0.7016, 2.8062)$ for $\mu = 0.5, \Lambda = 1, \alpha = 0.5, \beta = 0.5, \delta = 0.5, \gamma = 0.5, \xi = 2, R_0 = 5 > 1$.

Lyapunov method and LaSalle invariance principle in Theorem 3.2. Figure 1 and Figure 2 verify this theorem whenever $R_0 \leq 1$ and Figure 3 shows the asymptotic stability of endemic equilibrium using numerical simulation if $R_0 > 1$.

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Cemil Büyükadalı received the PhD degree in Mathematics at Middle East Technical University. His research interests are in the areas of applied mathematics and mathematical biology. He is referee of mathematical journals.