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Global dynamics of an age-structured cholera model with both human-to-human and environment-to-human transmissions and saturation incidence

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ABSTRACT

In this paper, an age-structured cholera model with both human-to-human and environment-to-human transmissions and saturation incidence is proposed. In the model, we consider the infection age of infectious individuals and the biological age of pathogen in the environment. It is verified that the global dynamics of the model is completely determined by the basic reproduction number. Asymptotic smoothness is verified as a necessary argument. By analyzing corresponding characteristic equations, we discuss the local stability of each of feasible steady states. Uniform persistence is shown by using the persistence theory for infinite dimensional dynamical system. The global stability of each of feasible steady states is established by using suitable Lyapunov functionals and LaSalle's invariance principle. Numerical simulations are carried out to illustrate the theoretical results.

1. Introduction

Cholera is a common infectious disease caused by the bacterium Vibrio cholerae, which can give rise to acute diarrhea and vomit. The World Health Organization (WHO) estimates that there are 1.3–4 million cholera cases per year with about 21,000–143,000 deaths all over the world [1]. Beginning in April 2017, a major cholera epidemic occurred in Yemen, with about 500,000 reported cases and 2000 deaths. Due to the deterioration of health systems and its associated infrastructure, cholera spreads more seriously. The WHO claims that as many as 30,000 health care workers are devoted to the treatment in Yemen. In August 2017, the epidemic situation of major cities including Sanaa, Hajja, and Amran, is under control, while, the reported cases and deaths of cholera keeps rising [1]. In order to understand the transmission dynamics of cholera and provide some valuable insights on the prevention and control, some cholera models of ordinary differential equations (ODEs) have been proposed (see, for example, [4]). Nelson et al. [2] and Clemens et al. [3] found that cholera can be transmitted directly to humans by human-to-human contact or indirectly to humans via contaminated water. In [4], Tien and Earn considered the following cholera model with both human-to-human (direct) and environment-to-human (indirect)

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Table 1
Definitions of frequently used symbols.

Symbols	Description
a	Infection age, i.e., the time that has lapsed since the cholera pathogen has invaded human small intestines and produced exotoxin
b	Biological age, i.e., the time that has lapsed since the cholera pathogen has penetrated into the aquatic environment
$S(t)$	The density of susceptible individuals at time t
$i(a, t)$	The density of infected individuals with infection age a at time t
$p(b, t)$	The concentration of cholera pathogen with biological age b at time t
$\beta(a)$	The transmission coefficient of infected individuals with infection age a
$\beta_e(b)$	The transmission coefficient for per concentration of cholera pathogen with biological age b
$\theta(a)$	The change rate of infected individuals, including natural death rate, recovery rate and disease-induced death rate
$\delta(b)$	The removal rate of cholera pathogen with biological age b
$\xi(a)$	The pathogen production rate of an infected individual with infection age a

transmissions:

$$\begin{aligned}
\frac{dS(t)}{dt} &= A - \mu S(t) - \beta S(t)I(t) - \beta_e S(t)P(t),\\
\frac{dI(t)}{dt} &= \beta S(t)I(t) + \beta_e S(t)P(t) - \gamma I(t) - \mu I(t),\\
\frac{dP(t)}{dt} &= \xi I(t) - \delta P(t),\\
\frac{dR(t)}{dt} &= \gamma I(t) - \mu R(t),
\end{aligned} \tag{1.1}$$

where $S(t)$, $I(t)$ and $R(t)$ denote the density of susceptible, infected and recovered individuals, respectively; $P(t)$ denotes the concentration of cholera pathogen in contaminated water. In system (1.1), all parameters are positive constants, where A is the recruitment rate of susceptible individuals; μ is per capita natural death rate; β is the direct transmission coefficient of infected individuals; β_e is the indirect transmission coefficient for per concentration of cholera pathogen; $1/\gamma$ is the mean infectious period; ξ is the pathogen production rate of an infected individual; δ is the removal rate of cholera pathogen.

However, the models mentioned above assumed that direct and indirect transmission coefficients, disease-induced death rate, the production and removal rates of cholera pathogen are invariant with time and ignored that the infectivity in both human-to-human and environment-to-human transmissions varies with infection age of infected individuals and biological age of pathogen [2,5]. Experimental investigations [6] showed that the infectivity of Vibrio cholerae existing outside the host decays in time. In [7], Brauer et al. considered the following age-structured cholera model:

$$\begin{aligned}
&\frac{dS(t)}{dt} = A - \mu S(t) - S(t)\int_0^\infty \beta(a)i(a,t)da - S(t)\int_0^\infty \beta_e(b)p(b,t)db,\\
&\frac{\partial i(a,t)}{\partial t} + \frac{\partial i(a,t)}{\partial a} = -\theta(a)i(a,t), \quad a > 0,\\
&\frac{\partial p(b,t)}{\partial t} + \frac{\partial p(b,t)}{\partial b} = -\delta(b)p(b,t), \quad b > 0,
\end{aligned} \tag{1.2}$$

with boundary conditions

$$\begin{aligned}
i(0,t) &= S(t)\int_0^\infty \beta(a)i(a,t)da + S(t)\int_0^\infty \beta_e(b)p(b,t)db, \quad t > 0,\\
p(0,t) &= \int_0^\infty \xi(a)i(a,t)da, \quad t > 0.
\end{aligned} \tag{1.3}$$

Here, A and μ are the same as those in system (1.1). $S(t)$, $i(a, t)$, $p(b, t)$ and other parameters are described in Table 1. System (1.2) with the boundary conditions (1.3) were further investigated by Yang et al. [8] and Wang et al. [9].

Noting that, incidence rates in systems (1.1) and (1.2) are bilinear, which regards the infection rate per infected individual or per concentration of pathogen as a constant. Actually, incidence rate is influenced by the inhibition effect from behavioral change of susceptible individuals and the crowding effect of infective individuals. In [10], Capasso and Serio introduced a saturated incidence rate $\beta I/(1+\alpha I)$, where βI measures the infection force of the disease and $1/(1+\alpha I)$ measures the inhibition effect and crowding effect. There have been several works on cholera models with saturation incidence in the literature (see, for example, [11,12]). As for the infection rate for per concentration of cholera pathogen, experimental studies [13] indicated that the probability of infection depends on the concentration of V. cholerae in the contaminated water and only enough inoculum of V. cholerae can lead to cholera. In [14], Codeço introduced a new form $\beta_e P/(k+P)$ to measure the