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Influence of human behavior on cholera dynamics

Xueying Wang [a], Daozhou Gao [b,*], Jin Wang [c]

[a] Department of Mathematics, Washington State University, Pullman, WA 99164, United States
[b] Francis I. Proctor Foundation, University of California, San Francisco, San Francisco, CA 94143, United States
[c] Department of Mathematics, University of Tennessee at Chattanooga, Chattanooga, TN 37403, United States

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ABSTRACT

This paper is devoted to studying the impact of human behavior on cholera infection. We start with a cholera ordinary differential equation (ODE) model that incorporates human behavior via modeling disease prevalence dependent contact rates for direct and indirect transmissions and infectious host shedding. Local and global dynamics of the model are analyzed with respect to the basic reproduction number. We then extend the ODE model to a reaction–convection–diffusion partial differential equation (PDE) model that accounts for the movement of both human hosts and bacteria. Particularly, we investigate the cholera spreading speed by analyzing the traveling wave solutions of the PDE model, and disease threshold dynamics by numerically evaluating the basic reproduction number of the PDE model. Our results show that human behavior can reduce (a) the endemic and epidemic levels, (b) cholera spreading speeds and (c) the risk of infection (characterized by the basic reproduction number).

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1. Introduction

Mathematical modeling, analysis and simulation for infectious diseases have long provided useful insight into disease dynamics that could guide public health administration for designing effective prevention and control measures against epidemics. Over the past few decades, compartmental models such as SIR (susceptible-infected-recovered) and SEIR (susceptible-exposed-infected-recovered) and their threshold dynamics have been established as the standard framework in mathematical epidemiology (see review [33] and references therein). Meanwhile, numerous extensions of these basic mathematical models have been proposed that incorporate more detailed biological, ecological, demographic, and geographical information, such as spatial heterogeneities, age-structures, seasonal variations, and others, with significant advances in almost all of these directions.

The mechanisms of disease transmission and spread are usually complex and possibly involve social, economic and psychological factors in addition to the intrinsic disease biology and ecology. In particular, human behavior could have significant influence on disease transmission and vice versa. For example, individuals avoid close contact with obviously sick persons to protect themselves and therefore the frequency and strength of contacts between uninfected and infected people generally are reduced. In case of severe disease outbreaks, people will attempt to change their routine schedules (including, but not limited to, work, recreation, and travel), wash hands often with soap and clean water, receive vaccines or preventive treatment if available, so as to minimize their risk of infection. Nowadays, the fast growth of information technology allows prompt and up-to-date reports on the details of disease outbreaks from internet (especially those popular social networking sites), newspaper, television and radio stations, and government announcements. Consequently, these media coverage and health education will, to a large extent, affect human behavior which can lead to a significant reduction in outbreak morbidity and mortality.

It is clear that human behavior could play an important role in shaping the complex epidemic and endemic pattern of a disease [3,26]. There are an increasing number of studies on the mathematical epidemiological modeling of human behavior [13]. Funk et al. [14] classified epidemic models under the impact of behavioral changes into belief-based and prevalence-based. Cui et al. [11] proposed a simple SIS model that incorporated the effects of media coverage. Gao and Ruan [16] extended the work in [11] to a patch model with non-constant transmission coefficients. Liu et al. [25] investigated the psychological impact on disease dynamics that involve multiple outbreaks and sustained infections. Collinson and Heffernan [10] found that the outcome of an epidemic model with the effects of mass

* Corresponding author. Tel.: +1 415 502 0275; fax: +1 415 476 0527.
E-mail addresses: xueying@math.wsu.edu (X. Wang), daozhou.gao@ucsf.edu (D. Gao), jin-wang02@utc.edu (J. Wang).

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media is strongly affected by the choice of media function. Recently, Chowell et al. [9] fitted logistic growth models to the cumulative reported number of Ebola cases to reflect changes in population behavior and interventions. In addition, Mummert and Weiss [27] modeled and analyzed the social distancing strategies in limiting disease transmission and spread, particularly for short-term outbreaks.

A goal of this paper is to improve our quantitative understanding of the impact of human behavior on disease dynamics. Particularly, we will incorporate human behavior into mathematical modeling of cholera, a severe water-borne disease caused by the bacterium *Vibrio cholerae*. There have been many studies published in recent years on cholera modeling and analysis (see, e.g., [5–7,17,28,29,34,36–38,41–44]), yet, to our knowledge, few of these have specifically taken human behavior into consideration (see Capasso [5,6], Al-Arydah et al. [1], and Carpenter [7]). In the present paper, we will modify the cholera model proposed by Mukandavire et al. [28] to explicitly include disease prevalence dependent contact rates (for both the direct and indirect transmissions) and host shedding rate, and analyze the resulting dynamics. Particularly, we will show that the reduction of contact rates due to human behavior leads to reduced epidemic and endemic sizes. We will then extend the ODE system to a reaction–convection–diffusion PDE system to investigate the interaction among human behavior, host and pathogen movement, and the disease intrinsic transmission dynamics. We will pay special attention to the traveling wave solutions and threshold dynamics of the PDE model. Our study regarding cholera spatial dynamics is different from the work of Bertuzzo et al. [4,31]. Our PDE model formulation is more general in terms of inclusion of multiple transmission pathways. Specifically, our model incorporates both direct (or, human-to-human) and indirect (or, environment-to-human) transmission pathways whereas their model has considered only indirect transmission route. The scope of our work is also different from that in [4,31] as our focus is on the impact of human behavior on cholera transmission.

We organize the remainder of the paper as follows. In Section 2 we introduce the ODE cholera model that incorporates human behavior, with relevant notations and assumptions. We then conduct a thorough epidemic and endemic analysis of the model in Section 3, for both local and global dynamics. In Section 4 we present the PDE model and investigate its traveling wave solutions under the impact of human behavior, followed by a threshold dynamics analysis in Section 5. We conclude the paper in Section 6 with discussion.

2. Model formulation

The cholera model proposed in [28] incorporates both the environment-to-human (or, indirect) and human-to-human (or, direct) infection routes, and all the model parameters take constant values. The model has standard SIR (susceptible-infected-recovered) compartments, with an additional compartment B that denotes the concentration of the bacteria *V. cholerae* in the contaminated water. We now extend this model by assuming that the direct and indirect transmission rates and the bacterial shedding rate are all dependent on the number of infectives, representing the influence of human behavior change due to health education, hygiene and sanitation practices. In addition, we assume that recovered individuals become susceptible to cholera again after a certain period of time, taking into account the immunity loss in the real life. The new model takes the form

$$\begin{aligned}
\frac{dS}{dt} &= \mu N - \beta_1(I)SI - \beta_2(I)\frac{SB}{B+K} - \mu S + \sigma R,\\
\frac{dI}{dt} &= \beta_1(I)SI + \beta_2(I)\frac{SB}{B+K} - (\gamma+\mu)I,\\
\frac{dR}{dt} &= \gamma I - (\mu+\sigma)R,\\
\frac{dB}{dt} &= \beta_3(I)I - \delta B. \qquad (2.1)
\end{aligned}$$

The total population, $N = S + I + R$, is fixed. The definition and base values of the model parameters are provided in Appendix A, Table A.1.

The most important feature of our model is the incorporation of disease prevalence dependent contact rates and host shedding rate. For $i = 1, 2, 3$, we formulate that

$$\beta_i(I) = a_i - b_i m_i(I),$$

where a_i is the usual contact rate (or shedding rate) without considering the influence of human behavior, b_i is the maximum reduced contact rate due to behavior change, and $m_i(I)$ is a saturation function. These functions satisfy

$$\begin{aligned}
&a_i > b_i \geq 0,\ m_i(I) \in C^1([0, I_u]) \text{ with } m_i'(I) \geq 0,\\
&m_i(0) = 0,\ 0 < m_i(I_u) \leq 1,
\end{aligned}$$

where $I_u \in (0, N]$ is an upper bound of the solution $\{I(t): t \geq 0\}$. Some typical examples of $m(I)$ with such properties are $1 - k/(k + I^n)$ with $k > 0$ and $n > 0$, $1 - e^{-kI}$ with $k > 0$, and I/I_u [16].

One can easily verify that the disease-free equilibrium is given by $(N, 0, 0, 0)$. Let F denote the matrix characterizing the generation of secondary infection, and V denote the matrix depicting transition rates between compartments. Based on the standard next-generation matrix technique [12,40] and our assumptions, matrices F and V can be written as:

$$F = \begin{bmatrix} a_1 N & a_2 N/K \\ a_3 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \mu+\gamma & 0 \\ 0 & \delta \end{bmatrix}.$$

The next generation matrix is

$$\boldsymbol{M} = FV^{-1} = \begin{bmatrix} \dfrac{a_1 N}{\mu+\gamma} & \dfrac{a_2 N}{\delta K} \\ \dfrac{a_3}{\mu+\gamma} & 0 \end{bmatrix}.$$

Hence, the basic reproduction number $\mathcal{R}_0$ of model (2.1) is given by

$$\begin{aligned}
\mathcal{R}_0 &= \mathcal{R}_0^{ODE} = \rho(\boldsymbol{M})\\
&= \frac{1}{2}\left[\frac{a_1 N}{\mu+\gamma} + \sqrt{\left(\frac{a_1 N}{\mu+\gamma}\right)^2 + 4\frac{a_2 a_3 N}{\delta(\mu+\gamma)K}}\right].
\end{aligned}$$

Here ρ denotes the spectral radius. Note that the basic reproduction number $\mathcal{R}_0$ is independent of b_i for $i = 1, 2, 3$. This is due to our model assumption that behavior change only starts when the disease has already started and $\mathcal{R}_0$ is calculated at the disease-free state. An implication is that behavior change alone is usually not sufficient to terminate an outbreak. Nevertheless, previous studies have shown that it can significantly reduce the burden of an endemic disease [16]. We will demonstrate this for our cholera model in the next section.

Meanwhile, if disease control is targeted at a particular host type, a useful threshold is known as the *type reproduction number, T*. The type reproduction number defines the expected number of secondary infective cases of a particular population type caused by a typical primary case in a completely susceptible population [18,32]. It is an extension of the basic reproduction number $\mathcal{R}_0$. Particularly, the type reproduction number T_1 for control of infection among humans is defined in the references [18,32] as

$$T_1 = \boldsymbol{e}_1^T \boldsymbol{M}(\boldsymbol{I} - (\boldsymbol{I} - \boldsymbol{P}_1)\boldsymbol{M})^{-1}\boldsymbol{e}_1,$$

provided the spectral radius of matrix $(\boldsymbol{I} - \boldsymbol{P}_1)\boldsymbol{M}$ is less than one, i.e., $\rho\big((\boldsymbol{I} - \boldsymbol{P}_1)\boldsymbol{M}\big) < 1$. Here $\boldsymbol{I}$ is the 2×2 identity matrix, vectors $\boldsymbol{e}_1 = (1, 0)^T$, $\boldsymbol{M}$ is the next generation matrix, and $\boldsymbol{P}_1$ is the 2×2 projection matrix with all zero entries except that the (1,1) entry is 1. Write

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