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Global stability of a multiple infected compartments model for waterborne diseases

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ABSTRACT

In this paper, mathematical analysis is carried out for a multiple infected compartments model for waterborne diseases, such as cholera, giardia, and rotavirus. The model accounts for both person-to-person and water-to-person transmission routes. Global stability of the equilibria is studied. In terms of the basic reproduction number R_0, we prove that, if $R_0 \leqslant 1$, then the disease-free equilibrium is globally asymptotically stable and the infection always disappears; whereas if $R_0 > 1$, there exists a unique endemic equilibrium which is globally asymptotically stable for the corresponding fast–slow system. Numerical simulations verify our theoretical results and present that the decay rate of waterborne pathogens has a significant impact on the epidemic growth rate. Also, we observe numerically that the unique endemic equilibrium is globally asymptotically stable for the whole system. This statement indicates that the present method need to be improved by other techniques.

1. Introduction

Nowadays, waterborne diseases, including cholera, giardia, campylobacter, norovirus and rotavirus, are becoming a major public health concern to scientists and health agencies, and they have caused severe cholera outbreaks in parts of Zimbabwe, resulting in more than 98,000 cases and 4200 deaths from August 2008 to July 2009 [1]. Worldwide, about 3.5 billion people died of the waterborne diseases [2] according to WHO estimates. To avoid ambiguity, waterborne diseases refer to any disease that can be transmitted through water [3–7], which is mainly via pathogen ingestion (e.g. fecal-oral route). However, many other potential transmission routes, such as eating food touched by an individual with soiled hands, drinking sewage-contaminated water, or contacting an infected individual during treatment in a hospital, may account for the spread of waterborne diseases. Even for the same disease outbreaks, the relative contribution of different transmission routes may vary greatly. For example, cholera propagation is frequently considered to occur through drinking contaminated water, but direct person to person transmission might be responsible for an outbreak in a Singapore psychiatric hospital [8]. Giardia is typically communicated via drinking contaminated water, but direct transmission among individuals is also a determined risk factor [9]. Moreover, hepatitis A transmission appears typically by person to person contact, with contaminated water providing a additional transmission pathway.

In the last two decades, several mathematical models have been widely used in the literature to investigate transmission dynamics of waterborne diseases in various regions. A number of different approaches have been adopted to understand

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mechanisms of waterborne diseases transmission. Eisenberg et al. [10] proposed a stochastic model to quantify the secondary infections produced by infected individuals both within a household through direct person to person contact, and between households by shedding pathogen into a common water source. King et al. [11] formulated stochastic differential equation models of cholera to incorporate both person to person transmission and environmental reservoir transmission, i.e. water to person transmission, without considering feedback mechanism from infected individuals to environmental reservoir. Some other studies [12–14] presented compartmental ordinary differential equation (ODE) models to consider only person–water–person transmission (infected individuals release pathogen into environmental reservoir from which susceptible individuals then drink), and ignored person to person transmission. Koelle et al. [15,16] studied waterborne diseases models in the standard *SIR*/*SIRS* framework. Tien and Earn [17] considered a feedback from infected individuals to environmental reservoir by adding a water compartment W, a simple extension of the traditional *SIR* model.

Tien and Earn [17] primarily focused on a simple extension of the *SIR* framework, i.e. *SIWR* model, and derived fundamental quantities in mathematical epidemiology, such as the basic reproduction number, the epidemic growth rate, and the final outbreak size when birth and death rates were zero. However, for disease that progress through a long infectious period, infectivity or infectiousness of individuals may vary differently in time. For example, it can take 8 to 10 years before an infected individual has clinical syndrome (AIDS) in the progression of a typical HIV infection. During the first few weeks after infection, HIV-infected individuals are highly infectious, then enter into an asymptotic stage of low infectiousness for many years and become gradually more infectious with immune system collapse and they progress to AIDS [18]. Multiple infected compartments are frequently used to model diseases with latent period, different stages of disease progression [18,19]. Tien et al. also extended the *SIWR* model to include multiple infected compartments (see Appendix in [17]) and only obtained the final outbreak size when the birth and death rates were zero. But they did not study (global) stability of the equilibria. This is partly because the system is high-dimensional and investigation of global behavior for such system is challenging. For high-dimensional compartmental epidemic models, some results concerning global stability of the equilibria exist, for example, see [20–24]. But in fact, compared to the existing high-dimensional epidemic models, results on (global) stability of high-dimensional epidemic models are scarce.

The aim of present paper, on the contrary, is to show analytically global asymptotic behavior of the equilibria, which is completely determined by the basic reproduction number R_0. The remaining part is organized as follows. In Section 2, we first describe briefly the multiple infected compartments model and establish the existence and the number of equilibria. The following two sections are devoted to investigating (global) stability of the equilibria. In Section 3, global stability of the disease-free equilibrium is investigated and the basic reproduction number R_0 is derived explicitly. It is shown that if $R_0 \leqslant 1$ then the disease-free equilibrium is globally asymptotically stable; while if $R_0 > 1$ then the disease-free equilibrium is unstable and the system is uniformly persistent. Next, in Section 4, we show that if $R_0 > 1$, the endemic equilibrium for fast–slow system is globally asymptotically stable. All the theoretical results are verified through numerical simulations and some new insights are presented in Section 5. We conclude the paper in Section 6.

2. Model description and the equilibria

To formulate a multiple infected compartments model, the total host population is partitioned into the following compartments: a susceptible compartment S, infectious compartment I_k, whose members are in the kth stage of disease progression, $k = 1, 2, \ldots, n$, and a recovered compartment R. A compartment W, which measures the pathogen concentration in a water source, is added to incorporate feedback mechanism from water. At each time step, susceptible individuals are infected both by direct person to person contact and by the pathogen in contaminated water shed by infected individuals; on the contrary, infected individuals can contaminate the water compartment by releasing the pathogen into W. Transfer diagram for the multiple infected compartments model is depicted in Fig. 1.

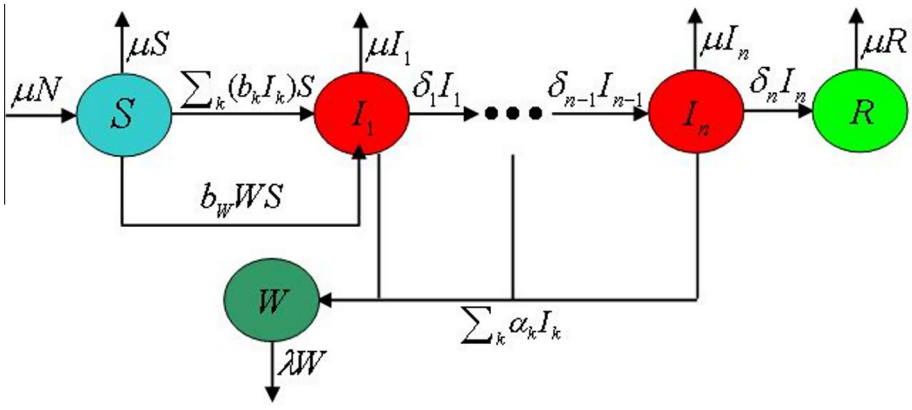

Fig. 1. Transfer diagram for the multiple infected compartments model.