



Incorporating heterogeneity into the transmission dynamics of a waterborne disease model



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HIGHLIGHTS

- Devised new model for the evolution of water-borne diseases incorporating heterogeneity.
- Incorporated multiple water sources for the first time.
- Fitted model to real data from Haiti.

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ABSTRACT

We formulate a mathematical model that captures the essential dynamics of waterborne disease transmission to study the effects of heterogeneity on the spread of the disease. The effects of heterogeneity on some important mathematical features of the model such as the basic reproduction number, type reproduction number and final outbreak size are analysed accordingly. We conduct a real-world application of this model by using it to investigate the heterogeneity in transmission in the recent cholera outbreak in Haiti. By evaluating the measure of heterogeneity between the administrative departments in Haiti, we discover a significant difference in the dynamics of the cholera outbreak between the departments.

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1. Introduction

Waterborne diseases can be transmitted via person–water–person contact. This means that an infected individual will first shed pathogens into the water source and susceptible individuals can then contact the disease when they drink contaminated water. In reality, the transmission rate and the shedding rate vary from one individual to another, hence leading to heterogeneity in the transmission of waterborne diseases. Even though, in some of the theoretical studies on the dynamics and control intervention strategies (Tien and Earn, 2010; Zhou et al., 2012; Mwsa and Tchuente, 2011; Liao and Wang, 2011; Capasso and Paveri-Fontana, 1979; Pourabbas et al., 2001; Codeco, 2001; Ghosh et al., 2004; Hartley et al., 2006; King et al., 2008; Eisenberg et al., 2003; Mukandavire et al., 2011a, 2011b) this is not taken into account, heterogeneity is crucial to understand the dynamics of waterborne disease and how best to reduce the spread of the infection. Since most of the factors affecting the spread of

waterborne diseases vary within and across a population, it is expected that most of the important mathematical features of waterborne disease models such as the basic reproduction number, the type reproduction number and the final outbreak size will also vary. Understanding the behaviour of each of these mathematical features is very important in defining better control intervention strategies that will reduce the spread of the disease. It is our interest in this study to explore the effects of heterogeneity on each of the mathematical feature of waterborne disease model which is necessary for defining better control strategies that will reduce the spread of the disease.

Waterborne disease can be transmitted through contaminated environmental water sources such as lake, river as well as through contaminated household water sources like pond, private water reservoir, etc. (Huq et al., 2005). It is important to know that some individuals can be exposed to more than one contaminated water source thus adding more heterogeneity in disease transmission. To take this into account, it is necessary to consider a situation whereby individuals are exposed to multiple contaminated water sources.

Consider a community where individuals are exposed to multiple contaminated water sources. Despite the fact that individuals are exposed to contaminated water sources, studies have shown

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that some groups of individuals (especially children) are more vulnerable to infection. Some of the reasons for this differences might be due to hygienic practices of the individuals (like boiling water before drinking, washing hands after going to the toilet, proper washing of dishes and food before eating) and the level of the immune system of the individuals. Understanding the dynamics of waterborne diseases for such a community is complicated as homogeneous models cannot explain such situations. As a result we resort to a multi-group model where a number of environmental, biological and socio-economic factors are used to categorise a group or sub population.

Tuite et al. (2011) constructed a mathematical model of cholera epidemic dynamics for the ten departments in Haiti that is based on both population and distance (a “gravity” model) between the departments. They used the model to predict the sequence and timing of regional cholera epidemics in Haiti and explore the potential effects of disease-control strategies. Bertuzzo et al. (2011) formulated a mathematical model which describes the epidemiological dynamics and pathogen transport and use it to determine the timing and the magnitude of the epidemic in the ten Haitian departments. Mukandavire et al. (2013) formulated a system of coupled stochastic differential equations and use it to estimate the reproductive numbers and vaccination coverage for the cholera outbreak in Haiti. Robertson et al. (2013) extended the Tien and Earn (2010) model to an n -patch waterborne disease model in networks with a common water source to investigate the effect of heterogeneity in dual transmission pathways on the spread of the disease. Other works being done on spatially explicit models of waterborne diseases include those by Bertuzzo et al. (2010), Mari et al. (2011) and Gatto et al. (2012).

There is no doubt that the above studies have contributed immensely towards understanding the dynamics and control of waterborne diseases particularly for the recent cholera outbreak in Haiti. To the best of our knowledge, the heterogeneity in the transmission dynamics of waterborne disease has not yet been explored neither have it been investigated for the recent cholera outbreak in Haiti. The objectives of this paper is to develop and analyse a mathematical model in order to improve the understanding of the transmission dynamics of waterborne disease. We do this by investigating the heterogeneity in the transmission dynamics of the model and consequently use the model to investigate the heterogeneity in the recent cholera outbreak in Haiti.

The remaining part of this work is organized as follows: the model we are going to discuss is formulated in Section 2 and its qualitative analyses are carried out in Section 3. In Section 4, we apply our model to investigate heterogeneity in the recent cholera outbreak in Haiti. We conclude the paper by discussing our results in Section 5.

2. Model formulation

To formulate the model, we consider a total human population N where individuals are exposed to m multiple contaminated water sources. We partition the population into n distinct sub populations or groups based on the activity level. These groups when combined together form the total population model in which secondary infections can be generated both within a given group and between groups. The secondary infections within a group occur when an individual from a group sheds pathogens into water sources with which susceptible individuals from the same group subsequently come into contact. However, if the susceptible individuals that come in contact with the pathogens shed from an individual are from different groups, we say that secondary infections between groups have occurred.

We partition N , the total human population of a community at risk for waterborne disease infections, into n groups or homogeneous sub populations of size N_j such that each group is made up of susceptible $S_j(t)$, infected $I_j(t)$ and recovered $R_j(t)$, individuals. The compartment W_k measures pathogen concentration in water reservoir k . In this study, we assume that there is no person to person transmission and only consider transmission through contact with contaminated water, as it is often considered to be the main driver of waterborne disease outbreaks (Mukandavire et al., 2011b; Sanches et al., 2011). Susceptible individuals $S_j(t)$ become infected through contact with the contaminated water sources W_k at rate b_{jk} . Infected individuals $I_j(t)$ can contaminate the water source k by shedding pathogen into it at rate θ_{jk} . The $I_j(t)$ can recover naturally at rate γ_j . Pathogens in the contaminated water source k grow naturally at rate α_k and decay at rate ξ_k . We assume that $\sigma_k = -(\alpha_k - \xi_k) < 0$ is the net decay rate of pathogens in the k th water reservoir. Natural death occurs in all the groups at rate μ . Note that $j = 1, 2, \dots, n$ and $k = 1, 2, \dots, m$. Putting these assumptions together, we obtain the model

$$\begin{aligned} \dot{S}_1(t) &= \mu N_1(t) - S_1(t) \sum_{k=1}^m b_{1k} W_k(t) - \mu S_1(t), \\ \dot{I}_1(t) &= S_1(t) \sum_{k=1}^m b_{1k} W_k(t) - (\mu + \gamma_1) I_1(t), \\ \dot{R}_1(t) &= \gamma_1 I_1(t) - \mu R_1(t). \\ \dot{S}_2(t) &= \mu N_2(t) - S_2(t) \sum_{k=1}^m b_{2k} W_k(t) - \mu S_2(t), \\ \dot{I}_2(t) &= S_2(t) \sum_{k=1}^m b_{2k} W_k(t) - (\mu + \gamma_2) I_2(t), \\ \dot{R}_2(t) &= \gamma_2 I_2(t) - \mu R_2(t), \\ &\vdots \\ \dot{S}_n(t) &= \mu N_n(t) - S_n(t) \sum_{k=1}^m b_{nk} W_k(t) - \mu S_n(t), \\ \dot{I}_n(t) &= S_n(t) \sum_{k=1}^m b_{nk} W_k(t) - (\mu + \gamma_n) I_n(t), \\ \dot{R}_n(t) &= \gamma_n I_n(t) - \mu R_n(t), \\ \dot{W}_1(t) &= \sum_{j=1}^n \theta_{j1} I_j(t) - \sigma_1 W_1(t), \\ \dot{W}_2(t) &= \sum_{j=1}^n \theta_{j2} I_j(t) - \sigma_2 W_2(t), \\ &\vdots \\ \dot{W}_m(t) &= \sum_{j=1}^n \theta_{jm} I_j(t) - \sigma_m W_m(t). \end{aligned} \tag{1}$$

A pictorial illustration of model (2) showing all the possible transmission dynamics that resulted in heterogeneity is given in Fig. 1. The model (1) can be written in compact form as

$$\begin{aligned} \dot{S}_j(t) &= \mu N_j(t) - S_j(t) \sum_{k=1}^m b_{jk} W_k(t) - \mu S_j(t), \\ \dot{I}_j(t) &= S_j(t) \sum_{k=1}^m b_{jk} W_k(t) - (\mu + \gamma_j) I_j(t), \\ \dot{W}_k(t) &= \sum_{j=1}^n \theta_{jk} I_j(t) - \sigma_k W_k(t), \\ \dot{R}_j(t) &= \gamma_j I_j(t) - \mu R_j(t), \end{aligned} \tag{2}$$

where $j = 1, 2, \dots, n$ and $k = 1, 2, \dots, m$. Variables and parameters of the model (2) with their meaning are given in Table 1. The force of infection in patch j is given by the linear term $\sum_{k=1}^m b_{jk} W_k$ (Guo, 2012; Lloyd and May, 1996). Since our interest is on heterogeneity in transmission dynamics of the waterborne disease which can be generated due to differences in contact rates and shedding rates, we will not consider explicit movement of individuals from one

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