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Research paper

Mathematical assessment of the role of environmental factors on the dynamical transmission of cholera

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ABSTRACT

In this paper, we investigate the impact of environmental factors on the dynamical transmission of cholera within a human community. We propose a mathematical model for the dynamical transmission of cholera that incorporates the virulence of bacteria and the commensalism relationship between bacteria and the aquatic reservoirs on the persistence of the disease. We provide a theoretical study of the model. We derive the basic reproduction number $\mathcal{R}_0$ which determines the extinction and the persistence of the infection. We show that the disease-free equilibrium is globally asymptotically stable whenever $\mathcal{R}_0 \leq 1$, while when $\mathcal{R}_0 > 1$, the disease-free equilibrium is unstable and there exists a unique endemic equilibrium point which is locally asymptotically stable on a positively invariant region of the positive orthant. The sensitivity analysis of the model has been performed in order to determine the impact of related parameters on outbreak severity. Theoretical results are supported by numerical simulations, which further suggest the necessity to implement sanitation campaigns of aquatic environments by using suitable products against the bacteria during the periods of growth of aquatic reservoirs.

1. Introduction

Cholera was largely eliminated from industrialized countries by water and sewage treatment over a century ago. Today, it remains a significant cause of morbidity and mortality in developing countries, where it is a marker for inadequate drinking water and sanitation infrastructure. After several years of steady increase from 2007, the number of cholera cases reported by the World Health Organization (WHO), as well as the number of countries which reported cholera cases, showed a considerable decrease [1]. Yet, the disease is still a threat to many countries. For instance in 2012 alone, a cumulative total of 245,393 cases, including 3034 deaths with a case-fatality rate of 1.2%, were reported by WHO from all continents. This involves 48 countries among which, 27 from Africa, 12 from Asia, 6 from Americas and 3 from Europe and Oceania. Furthermore, the recent cholera outbreaks in the following countries led to a large number of infectious and deaths [1]:

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Angola (2012), Cameroon (2010–2012), Congo (2008, 2012), Haiti (2010–2011), India (2007), Iraq (2008, 2012), Kenya (2010), Nigeria (2010), Philippines (2012), UK (2012), Vietnam (2009) and Zimbabwe (2008–2009).

Vibrio cholerae (*V. cholerae*) is a Gram-negative, comma-shaped bacterium that causes cholera in humans. Cholera is an acute intestinal infection caused by the ingestion of contaminated foods and water with *V. cholerae* bacterium. Among the 200 serogroups of *V. cholerae*, only *V. cholerae* O1 and O139 are responsible of cholera disease [2]. The etiological agent passes through and survives the gastric acid barrier of the stomach and then penetrates the mucus lining that coats the epithelium [3]. Once they colonise the intestinal gut, they produce enterotoxin (which stimulates water and electrolyte secretion by the endothelial cells of the small intestine) that leads to watery diarrhea. If left untreated, it leads to death within hours. In human volunteer studies, the infection dose was determined to be $10^8 - 10^{11}$ cells [4]. Cholera is characterized, in its most severe form, by the sudden onset of acute watery diarrhea that can lead to death by severe dehydration. *V. cholerae* can stay in faeces without losing its infectious ability for 7–14 days and shed back to the environment. The main reservoirs of *V. cholerae* are people and aquatic sources.

It has been discovered that environmental aquatic bacteria such as *V. Cholerae* O1 and *V. cholerae* non-O1 have ability to survive to the stress caused by the variation of some environmental factors, such as temperature, pH or the lack of nutritional resources [5,6]. The adaptation of these bacteria to their environment will lead to metabolic and phenotypic changes that will condition their survival; what can be compared to a phenomenon of dormancy. Cells are considered “viable but non-culturable ” (VNC) because the main effect of this change is the loss of the ability to be cultivated on a bacteriological culture medium [7]. This dormancy state has been considered for many species of bacteria as a survival strategy in the natural environment [5,6,8–10]. The state change to the cultivable state is possible particularly if the factors causing stress become favorable to the development and growth of the bacterial population. This phenomenon implies to reconsider the thinking concerning the survival of pathogenic bacteria scattered into the environment and its dynamics in the aquatic ecosystem. This cell viability (VNC) is considered as a possible hypothesis at the origin of “disappearance” of the bacteria of the aquatic ecosystem during the colder months. Also, in the aquatic environment, *V. cholerae* has been reported to be associated with a variety of living organisms, including animals with an exoskeleton of chitin, aquatic plants, protozoa, bivalves, waterbirds, as well as abiotic substrates (e.g. sediments). Most of these are well-known or putative environmental reservoirs for the bacterium, defined as places where the pathogen lives over time, with the potential to be released and to cause human infection. Thus, the bacteria are strongly associated with the population of phytoplankton and zooplankton organisms forming commensal, antagonism, parasitism, competition, or symbiotic relationships. In this work, we will focus on the commensalism relationship between phytoplankton and *V. cholerae*. This commensalism relationship greatly enhances the bacterium’s ability to survive in an aquatic environment, as the exoskeleton provides the bacterium with an abundant source of carbon and nitrogen.

The dynamics of cholera are complex due to the multiple interactions between the human host and the pathogen in the water environment [8–19], which contributes to both direct and indirect transmission pathways. Many studies supported that V. cholerae O1 and O139 are commensal to crustacean zooplankton, notably copepods, which are present both in their gut and in biofilms on their chitinous surfaces [10–17]. Furthermore, V. cholerae is present throughout the year in and on its zooplankton host, and V. cholerae serogroup O1 has been shown to attach preferentially to zooplankton, but also to some species of phytoplankton in waters [16]. Its commensal existence provides protection from grazing by heterotrophic nanoflagellates and also from toxic chemicals, including those used to disinfect drinking water, such as alum and chlorine [17]. V. cholerae, like all Vibrio species, produces chitinase(s), with chitin serving as a nutrient source [18]. Also, Kirschner et al. demonstrated that association with zooplankton is important for V. cholerae non-O1/non-O139 serogroup isolates endemic in Neusiedler See, a large, shallow, moderately saline-alkaline lake in Central Europe [14]. A significant correlation was observed between the seasonal pattern in frequency of occurrence of V. cholerae and increased zooplankton biomass [14]. A deep understanding of the disease dynamics would have a significant impact on the effective prevention and control strategies [18,19]. Mathematical modeling and numerical simulations have the potential, and offer a promising way, to achieve this. Many efforts have been and are still being devoted to the modeling of this disease. For a chronological history of the modeling of cholera, we refer the reader to the work [21] which mentions the first mathematical model developed in [20–25]. Some theoretical studies have been carried out on the mathematical modeling of cholera transmission dynamics [26–29]. To our best knowledge, none of these mentioned works on cholera models have considered the change of metabolism of bacteria and the commensal relationships between bacteria and the population of phytoplankton and zooplankton.

In this paper, we explore the impact of environmental factors on the dynamical transmission of cholera within a human community. We formulate a mathematical model for cholera disease, which incorporates some key epidemiological and biological features of the disease such as the waning of recovery-induced immunity of recovered individuals, the virulence of bacteria and the commensal relationships between bacteria and the population of phytoplankton and zooplankton. We present the theoretical analysis of the model. We compute the disease-free equilibrium and derive the basic reproduction number $\mathcal{R}_0$ that depends on the rate of appearance and loss of virulence of bacteria and the carrying capacity of the population of phytoplankton and zooplankton. We do an in-depth analysis of the global asymptotic stability of the disease-free equilibrium and the local asymptotic asymptotical stability of the endemic equilibrium. The sensitivity analysis of the model is carried out to identify the most influential parameters on the model output variables, that is the most robust estimations that are required. Numerical simulations are presented to support the theory and to get insight on the role of the virulence of bacteria and the commensal relationship between bacteria and the population of phytoplankton and zooplankton on the