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Mathematical analysis and numerical simulation of an age-structured model of cholera with vaccination and demographic movements

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ABSTRACT

In this paper, we formulate a deterministic, nonlinear model of cholera with age structure which integrates the direct transmission and the indirect transmission of the disease. The vaccination and the demographic movements are also taken into account in this model. The propounded model is an initial/boundary-value problem constituted of four partial differential equations of first order describing the transmission dynamics of human hosts and of two ordinary differential equations representing the bacterial dynamics in the environment. We conduct a rigorous mathematical analysis of this model and we prove that it admits a unique positive bounded solution. The existence of a unique equilibrium which is infection-free in the absence of the transmission disease and endemic in the presence of the transmission disease is also established. We determine a threshold parameter $\mathfrak{R}_0$ such that this equilibrium is locally asymptotically stable when $\mathfrak{R}_0 < 1$ and unstable when $\mathfrak{R}_0 > 1$. Also, a parameter $\mathfrak{R}_0^\star$ is determined such that when $\mathfrak{R}_0 > 1$ and $\mathfrak{R}_0^\star < 1$, the number of the infected individuals of the equilibrium becomes less than 1. At the end, we use Wendland's Compactly Supported Radial Basis Functions (CSRBFs) method to find the numerical solution of the formulated model. This numerical solution is used to conduct the numerical simulation allowing us thus to check our theoretical results.

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1. Introduction

The cholera is an acute intestinal infection which is the result of the absorption, by ingestion, of the vibrio choleraic finding oneself in water or in food, but can also be the result of a contamination of a person to an another starting from the pathological products (excrements, vomiting, sweat). The experiences show

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that the vibrios ingested in the food are most liable to be the cause of an infection than those ingested in the water. The infectious dose determined experimentally is of the order of 10^8 to 10^{11} bacteria. The gastric acidity is little propitious to the survival of the bacterium in the stomach. After the crossing of the gastric barrier, the vibrios settle down in the nearness part of the small intestine, going through the mucus layer and secrete the choleraic toxin. This toxin modifies the exchanges of the water and of the electrolytes by preventing the penetration of the sodium inside the cell. This induces a crossing in the light of the digestive tract of a big quantity of water being able to reach 15 l per day, causing a severe dehydration of the ill individual [1].

The World Health Organization evaluates of 3 to 5 million cases of cholera per year with more than 100,000 deceases allocated between 40 and 50 countries [2]. It is well known that in the African context, the disease causes many difficulties, particularly in the regions where the poor economic factors do not permit an appropriate treatment of the contaminated streams. For example, the recent outbreaks of the epidemic in Haiti [3], Zimbabwe [4] and Angola [5] have led to a large number of infections and hence have received worldwide attention.

The developed common types of the cholera spreading models are time dependent models which conduct at ordinary differential equations (ODEs) systems [6–11]. For humans living in crowded conditions with a poor sanitation, the common type of model used to express the spread of an infectious disease is a SIR model, of which the name results of the fact that the population is subdivided into three disjoined classes: susceptible, infected and recovered [12]. To model the pathway of the infection more precisely, Hartley et al. [13] have proposed a model which takes account of the role of the hyperinfective vibrios introduced into the water reserves by the infected individuals. This new model explains the explosive nature of the disease more clearly as seen in the historical accounts of the cholera epidemics. This feature of the hyperinfective vibrios, which brings out the increase of the transmission, is included in the model studied in [14]. To see the biological sequencing background of the vibrio choleraic, we refer the interested reader to [15]. Let us notice that King et al. [10] have proposed a two-patch cholera ODEs model including classes for 'mild' infections and the feature of waning immunity. In their work, Miller Neilan et al. [8], have investigated the optimal control of three strategies to slow the spread of the disease in an ODE model with hyperinfective vibrios, asymptomatics infected and waning immunity. There is another modeling approach with a compartment that tracks pathogen in the water, this approach gives a SIWR system of four ODEs and have been used to simulate the cholera epidemic at 19th century to London [9].

In this work, we consider an extension of Cai et al.'s model (see [16]), which is also an extension of the Hartley et al. SIR model (see [13]) . The cholera such that we know it is a waterborne disease, but several studies (see for example [4,9,17]) have shown that the direct transmission (i.e. transmission of a human to another human) also plays an important role in the shaping of the epidemics. In this paper, a PDEs–ODEs cholera model which incorporates simultaneously the direct transmission and the indirect transmission of the disease is formulated. Let us notice that there exists in the literature of other epidemics, models which take account of both direct and indirect transmissions of the disease (see for example [18,19]). It must be noted that the considered model here is an extension of the model proposed by Cai et al. (see [16]), though Cai et al. have only admitted the existence and uniqueness of a solution of the PDEs system, suggesting that the proof could be elaborated similarly as in [13], i.e. by using the fixed point argument and then they have derived numerical simulation which is not an easy task.

Our goal is to attempt to extend Cai et al.'s results (see [16]) in the case where both direct and indirect transmissions of the disease are used. In order to do such extension, we need to obtain more rigorous mathematical results on the existence, uniqueness, boundedness and positivity of the solution of the formulated model. Let us notice that most of these results especially boundedness and positivity, are not generally built in the topic's literature when the model is a PDEs system. Furthermore, we need to give some connections between the mathematical results and the biological interpretations and to propose an

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