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A metapopulation model for cholera transmission dynamics between communities linked by migration

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ABSTRACT

A metapopulation model is developed to describe the spread of cholera between two communities connected by migratory movement. Disease threshold ratios specific to the communities are given, considering a case when the communities are isolated and when the communities are connected. The connection of the threshold ratios to disease spread and stability is discussed. The disease free equilibrium is globally stable whenever, the corresponding community specific disease threshold ratios are less than one and unstable otherwise. Community specific endemic equilibrium points are unique, locally asymptotically stable, and only exist when the corresponding disease thresholds are greater than unit. Disease spread is explosive in nature at the beginning of the outbreak but more severe in a community with poor facilities relative to a community with more improved facilities. In isolated communities, in the case of endemic cholera, the infection is characterised by a big outbreak, followed by a small episode of the infection. Only one typically big outbreak is observed in the community with improved facilities with no recurrence of the epidemic. In connected communities, movement of individuals across communities not only influences persistence of the infection but also results in more pronounced outbreak in a relatively well facilitated community in the long term. Synchronous fluctuation of the population is observed when there is unrestricted movement of both immunologically naive and infected individuals across the communities. Our results suggest that during times of cholera, movement to and fro cholera endemic areas should be avoided if the outbreak is to be contained. Otherwise, with continued migration, the infection may potentially worsen even in communities with relatively good facilities.

1. Introduction

Similar to ecological studies of persistence and extinction of species, there is great interest in ascertaining why infections persist in the population. This concern has shaped the history of epidemiological research as well as population dynamics studies. For a long time now, homogeneity of the population in dynamics of infectious diseases has always been assumed. It however does not account for vital heterogeneous aspects related to spatial structure. When spatial information is required, or when spatial homogeneity does not adequately account for the observed behaviour or disease transmission, then spatial modelling becomes handy in accounting for spatially distinct individual characteristics. Some of these characteristics include differences in mixing behaviour as well as migration which results into heterogeneity [1]. Spatial structure

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and the spatial scale roughly operate in three broad classes of heterogeneities which are; (1) environmental (covering geography and space including climate and hydrological factors), (2) contact (which involves contact patterns between hosts and pathogens including movement of hosts) and (3) host/pathogen heterogeneity (related to genetic factors and resistance to the disease) [2]. It is for this reason that spatial modelling has been widely used in the study of disease dynamics while accounting for the degree of uniqueness in one patch relative to another. Different approaches used in spatial modelling range from the use of systems of reaction–diffusion equations (RDEs) [3] to metapopulation model framework. The use of RDEs is often faced with challenges of complexity and are based on an assumption of continuous movement of organisms in space in a random walk fashion, which may not be entirely realistic [1]. Metapopulation (patch) models are however, based on the assumption of homogeneity within a patch and spatial heterogeneity is accounted for by migration between patches. This assumption is plausible in epidemiological studies since humans often frequent a home range, as opposed to random walk which is assumed in RDEs. We note that the above two mentioned approaches may not be the only ways in which to account for spatial heterogeneity. For instance, Lloyd and May [4] in their multi-patch model accounted for heterogeneity through mixing of individuals from separate patches without explicitly considering migration.

Populations in spatially disjunct locations exhibit spatial synchrony if they fluctuate in a similar manner [5]. Synchronous fluctuation is characterised by coincidental changes in the abundance, population cycles or other varying characteristics of geographically adjunct populations. These fluctuations in populations may be predominantly quantitative or qualitative. According to Matter [6], populations or local populations within a metapopulation often vary together in population size. In addition, in theoretical assessment of synchrony, identical ability of populations to absorb and produce dispersing individuals is assumed. In ecological studies, spatial synchrony is attributed to dispersal (migration) or environmental stochasticity (“Moran effect”). In accordance with the Moran effect, “if two populations have the same density-dependent structure, then correlated density-dependent structures can bring fluctuations in populations into synchrony” [7]. Therefore, the dynamics of the population may congruently depend on some asynchronous exogenous factors which may include temperature and rainfall among others. These factors are important drivers of the cholera epidemic most especially in areas where it is endemic. Migration has also been observed as a potential mechanism by which an infection may be transmitted from one community to another. However, there has been no explicit study on the would be anticipated consequences of migration between communities in times of cholera epidemic. Our study is motivated by the recent major cholera outbreak in South Africa during 2000–2002, that spread to eight out of the nine provinces [8] in the country possibly through migration. The provinces are not all connected by a common river network and neither did the epidemic start at the same time in all the affected provinces. The first cases were reported in the KwaZulu Natal province and later in other regions. Since the disease is water-borne but the different provinces are not all connected by a hydrologic network, then the mechanism by which it spread becomes a puzzle. On the other hand, human-to-human transmission is know as a potential route, this raises speculations that human movement may have played a significant role in disease transmission. In some recent work by Bertuzzo et al. [9], a spatial transmission model developed and analysed indicated hydrologic networks and human mobility as drivers of the dispersal of the pathogen.

The work in this paper is motivated by the desire to understand the possible effect of migration between communities on the spread of cholera. Although substantial work has been done on the study of cholera transmission dynamics, no explicit consideration of metapopulation study has been previously done. We consider an SIR metapopulations model for cholera describing disease transmission between two communities (patches) connected by transport network. In the cholera transmission dynamics, we assume that only those infected with no symptoms are capable of moving from one community to another, most especially if the separating distance is large. The communities considered in the model are assumed to differ in the level of living conditions. This vital difference is depicted in the parameter values chosen from literature and simulations, where the evolution of populations can be traced. In general the transmission dynamics of the *vibrios* in humans may be complex due to the local and long-range movements of individuals. In addition, the non human source of the *vibrios* i.e. the aquatic reservoir is also complex. This is dependent on the rate at which the water flows, the volume which is related to the dilution of the pathogen concentration, its virulence and the salinity of the aquatic reservoir. In the same way it is important to note that the movement between populations depends on the density of the population and the distance separating the adjunct communities considered.

This manuscript is organised as follow; in the next section, we develop the mathematical model and give a comprehensive mathematical analysis. The analysis in this section is related to positivity and boundedness of solutions, evaluating the community specific disease threshold values for the case of isolated and non-isolated communities. In Section 5, numerical results are given and discussed with emphasis sensitivity analysis of the model output to the input parameters and evolution of population, and in Section 6 we conclude the manuscript.

2. The mathematical model

In the model formulation, the general population considered is divided in two main communities and each community divided into three compartments with reference to *vibrio* transmission and disease states of the individuals. The compartmentalisations in a single community involves individuals who are immunologically naive (Susceptible population) S, those infected I and those individuals who have recovered R but with temporary immunity. Within a community, the subpopulations within a compartment are assumed to be homogeneous and thus mix homogeneously. We note that in