



Spatially explicit cholera model: effects of population, water resources and health conditions distributions.

E. Bertuzzo (1), R. Casagrandi (2), M. Gatto (2), I. Rodriguez-Iturbe (3), and A. Rinaldo (1)

(1) Laboratory of Ecohydrology ECHO/ISTE/ENAC, Ecole Polytechnique Federale Lausanne, Lausanne, Switzerland, (2) Dipartimento di Elettronica e Informazione, Politecnico di Milano, Milano, Italy., (3) Department of Civil and Environmental Engineering, Princeton University, Princeton, NJ, USA.

Cholera epidemics are still a major public health concern to date in many areas of the world. In order to understand and forecast cholera outbreaks, one of the most important factors is the role played by the environmental matrix in which the disease spreads. The environmental matrix is constituted by different human communities and their inter-connections. Each community is characterized by its spatial position, population size, water resources availability and hygiene conditions. By implementing a spatially explicit cholera model we seek the effects on epidemic dynamics of: i) the topology and metrics of the pathogens pathways that connect different communities; ii) the spatial distribution of the population size; and iii) the spatial distributions of water resources and public health conditions, and how they vary with population size. We further extend the model by deriving the speed of propagation of traveling fronts in the case of uniformly distributed systems for different topologies: one and two dimensional lattices and river networks. The derivation of the spreading celerity proves instrumental in establishing the overall conditions for the relevance of spatially explicit models. The conditions are sought by comparison between spreading and disease timescales. Consider a cholera epidemic that starts from a point and spreads throughout a finite size system, it is possible to identify two different timescales: i) the spreading timescale, that is the time needed for the disease to spread and involve all the communities in the system; and ii) the epidemic timescale, defined by the duration of the epidemic in a single community. While the latter mainly depends on biological factors, the former is controlled also by the geometry of the environmental matrix. If the epidemics timescales are comparable or larger than pathogens' spreading timescales, one expects that the spatial variability does not play a role and the system may be approximated by a well-mixed reactor. When the spreading timescales are larger than the local epidemics timescales, the overall epidemic patterns are controlled by the spatial spreading and one expects notable differences between spatially implicit and explicit models.