# Simulation of Cholera Diffusion to compare transmission mechanisms

Ellen-Wien Augustijn-Beckers<sup>1</sup>, Juliana Useya<sup>1</sup>, Raul Zurita-Milla<sup>1</sup>, Frank Osei<sup>2</sup>

<sup>1</sup>University of Twente, Faculty of Geo-Information Science and Earth Observation (ITC), Hengelosestraat 99, 7500 AA Enschede, The Netherlands Telephone: +31 (0)53 4874 414

Fax: +31 (0)53 4874335 Email: augustijn@itc.nl

<sup>2</sup>Kwame Nkrumah University of Science and Technology (KNUST), Department of Geomatic Engineering, Kumasi, Ghana

### 1. Introduction

Since the initial transmission mechanism of Cholera was revealed by John Snow in 1854, the cause and spread of this disease has been under continuous research. Snow's study showed how disease incidences can be linked to a source based on the spatial distribution of the patients. However, Snow's work did not address the question of diffusion mechanisms. The predominant transmission mechanism of Cholera is via the fecal-oral route but in recent years several scientists have pointed toward a number of other transmission mechanisms that might contribute to the prevalence of the disease.

Cholera risk factors vary and stem from multiple transmissions including interactions between human hosts, pathogen and environment leading to person to person transmission (secondary transmission) and transmission via the environment (primary transmission) (Hartley et al., 2006). It is possible for the toxigenic *V. cholerae* to survive in surface water for up to several years (Codeco, 2001). Especially oceans and brackish water seem to function as a long term biotic reservoir for cholera (Emch et al., 2008). Driving factors for the ecology of *V. cholerae* are meteorological and climate variation. Where environmental forcing plays an important role at the macro level, secondary transmission is more related to local environmental variation.

Examples of local variation that influences the spread of cholera are water sources for household activities (pipe – well water), food control (seafood, fish and contamination during preparation) (Said, 2006) and sanitation (Emch et al., 2008). Fotedar (2001) provided evidence that houseflies (Musca domestica) are able to carry *V. cholerae*. Osei & Duker (2008) related cholera transmission to the mechanisms of filth breeding flies and flood water contamination. Spatial dependency of cholera infection on the proximity to and density of refuse dumps was shown by Osei et al. (2010) indicating that runoff from dump sites carry fecal materials to local rivers, creating a pathway for fecal contamination of surface water. Hartley et al. (2006) investigated the relative importance of the transmission factors and found a dependency on sanitation, population density and hygiene. We continue this work by investigating the relative importance of micro level transmission mechanisms by means of an agent-based simulation model.

There are relatively few mathematical Cholera models, perhaps because of the complex transmission mechanisms. A model was developed by Codeco (2001), who extended an existing model by Capasso for an Italian cholera outbreak. This model allows for long term dynamics incorporating an environmental reservoir of *V. cholerae*. A line of spatially explicit mathematical models was developed based on hydrology-driven cholera spreading (Bertuzzo et al., 2008, Bertuzzo et al., 2009, Righetto et al., 2010), and an age structured model was developed by Agheksanterian & Gobbert (2007).

## 2. Cholera model

The model presented in this research is a geographically explicit agent-based Cholera simulation. It is a micro scale, hydrology-driven model that differs from already existing ones in that it:

- Includes the spread of Cholera from dumpsites by the housefly (*M. domestica*)
- Includes runoff from dumpsites as a pathway of bacteria and feces to rivers
- Includes human to human transmission of cholera
- Is based on a synthetic population representing age categories, income levels and other population dynamics like hygiene levels and access to pipe water.

The proposed model consists of four different sub-models: (i) a hydrological model for the transport of the *V. cholerae* pathogen (ii) an epidemic model (iii) a house fly model for modeling flies as disease carriers (iv) a human interaction model.

#### 2.1 Hydrological sub-model

The hydrological model consists of three elements, an elevation raster, line elements representing the river branches and rainfall particles. Rainfall particles will flow downhill according to the elevation surface and can be transferred into carriers of feces or carriers of feces with pathogen. The model assumes constant flow of water along the river branches. Changes in river water volume and speed of flow are not taken into account. Growth rate of free-living bacteria (in water) is normally negative (Bertuzzo et al., 2008). Because of the small area included in the simulation no "bacterial mortality" is implemented.

#### 2.2 Epidemic sub-model

The model is based on the cholera transmission model from Hartley (2006) including hyper-infectivity. Hyper-infectivity is the fact that *V. cholerae* when passed through the gastrointestinal tract (via a human being) transfers into a short-lived hyper-infectious state (Hartley et al., 2006). The existence of this hyper-infectivity is associated with the explosive nature of some cholera outbreaks. Chance of developing the disease after exposure to hyper infective *V. cholerae* is very high. After recovery people become immune and this immunity lasts for at least two years (Koelle et al., 2005). For the time span of this simulation the immunity is permanent and no waning is considered. Currently the severity of the disease is not modeled although some cases are known to be asymptomatic.

#### 2.3 House Fly sub-model

House Fly density maps were generated around the locations known to be exposed to human excreta (refuse dumps and rivers). For this model, density layers were regarded to be static. Assumption is made that flies are able to carry *V. cholerae* from open dumpsites to surrounding areas within a critical buffer distance of 500 meters (Osei and Duker, 2008). Within this buffer distance transmission can occur.

#### 2.4 Human interaction sub-model

Agent unit is the individual person. Individuals are grouped into families. Prior to the start of the simulation families are distributed over houses, with multiple families living in a single building. Important attributes of families include the income level and the level of hygiene. Composition of families is based on Census data. Individuals are age specific and their behavior is based on age grouping. Individual behavior includes all activities that can lead to disease exposure including drinking/eating, caring for diseased relatives, dumping of feces and playing at dumpsites.



Figure 1. The Interface of the model

## 3. Case study

Study area is Kumasi, capital city of Ashanti Region located in south central Ghana. Kumasi has a population of approximately 3.5 million. The study area is located in the north-eastern part of the city. A severe outbreak of cholera occurred in this area in 2005 (data: Kumasi Metropolitan Disease Control Unit).

In the paper we will present the conceptual design and the initial findings of the model. Findings include the comparison of different transmission mechanisms. Importance of an agent-based cholera model is that the heterogeneity of the population is accounted for and that experiments can be conducted with intervention and changes in behavior of population. In future, we will continue to include behavioral changes of agents into this model.

## 4. Acknowledgements

Base data for this project was partially obtained from data obtained via the Planet Action Program (Spot Image).

## 5. References

- Agheksanterian, A. & Gobbert, M. K. 2007. Modeling the spread of epidemic cholera: an age-structured model. Department of Mathematics and statistics, University of Maryland, Baltimor County.
- Bertuzzo, E., Azaele, S., Maritan, A., Gatto, M., Rodriguez-Iturbe, I. & Rinaldo, A. 2008, On the spacetime evolution of a cholera epidemic. *Water Resources Research*, 44.
- Bertuzzo, E., Casagrandi, R., Gatto, M., Rodriguez-Iturbe, I. & Rinaldo, A. 2009, On spatially explicit models of Cholear epidemics. *Journal of the Royal Society Interface*, 7, 321-333.
- Codeco, C. 2001, Endemic and epidemic dynamics of cholera: the role of the aquatic reservoir. BMC Infectious Diseases, 1, 1.
- Emch, M., Feldacker, C., Islam, M. S. & Ali, M. 2008, Seasonality of cholera from 1974 to 2005: a review of global patterns. *International Journal of Health Geographics*, 7, 1-13.
- Fotedar, R. 2001, Vector potential of houseflies (Musca domestica) in the transmission of Vibrio cholerae in India. *Acta Tropica*, 78, 31-34.
- Hartley, D. M., Morris, J. G. & Smtih, D. L. 2006, Hyperinfectivity: A Critical Element in the Ability of V. cholerae to cause epidemics? *PLoS Med*, *3*, 63-69.
- Koelle, K., Rodo, X., Pascual, M., Yunus, M. & Mostafa, G. 2005, Refractory periods and climate forcing in cholera dynamics. *Nature*, 436, 696-700.
- Osei, F. & Duker, A. 2008, Spatial dependency of V. cholera prevalence on open space refuse dumps in Kumasi, Ghana: a spatial statistical modelling. *International Journal of Health Geographics*, 7, 62.
- Osei, F. B., Duker, A. A., Augustijn, E.-W. & Stein, A. 2010, Spatial dependency of cholera prevalence on potential cholera reservoirs in an urban area, Kumasi, Ghana. *International Journal of Applied Earth Observation and Geoinformation*, 12 331-339.
- Righetto, L., Bertuzzo, E., Casagrandi, R., Gatto, M., Rodriguez-Iturbe, I. & Rinaldo, A. 2010, Modelling human movement in cholera spreading along fluvial systems. *Ecohydrology*.
- Said, M. D. 2006, *Epidemic cholera in KwaZulu-Natal: The role of the natural and social environment.* PhD, University of Pretoria.