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The spatial spread of schistosomiasis: A multidimensional network model applied to Saint-Louis region, Senegal

Manuela Ciddio[a,*], Lorenzo Mari[a], Susanne H. Sokolow[b,c], Giulio A. De Leo[b], Renato Casagrandi[a,*], Marino Gatto[a]

[a] Dipartimento di Elettronica, Informazione e Bioingegneria, Politecnico di Milano, 20133 Milano, Italy
[b] Hopkins Marine Station, Stanford University, Pacific Grove, CA 93950, United States
[c] Marine Science Institute, University of California, Santa Barbara, CA 93106, United States

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ABSTRACT

Schistosomiasis is a parasitic, water-related disease that is prevalent in tropical and subtropical areas of the world, causing severe and chronic consequences especially among children. Here we study the spatial spread of this disease within a network of connected villages in the endemic region of the Lower Basin of the Senegal River, in Senegal. The analysis is performed by means of a spatially explicit metapopulation model that couples local-scale eco-epidemiological dynamics with spatial mechanisms related to human mobility (estimated from anonymized mobile phone records), snail dispersal and hydrological transport of schistosome larvae along the main water bodies of the region. Results show that the model produces epidemiological patterns consistent with field observations, and point out the key role of spatial connectivity on the spread of the disease. These findings underline the importance of considering different transport pathways in order to elaborate disease control strategies that can be effective within a network of connected populations.

1. Introduction

Schistosomiasis is an acute and chronic water-related disease caused by parasitic worms that affects about 250 million individuals worldwide (WHO Expert Committee, 2002). As one of the commonest and most devastating parasitic diseases, it is second only to malaria, inducing severe consequences to 20 million people (Kheir et al., 1999) and being directly responsible for 12,000 deaths yearly (Lozano et al., 2012). With an estimated burden of 4.5 million disability-adjusted life years (Fenwick, 2012; WHO Expert Committee, 2002), schistosomiasis is prevalent in tropical and subtropical areas, especially in poor communities without access to safe drinking water and adequate sanitation. It is estimated that at least 90% of those requiring treatment for schistosomiasis live in Africa (WHO Expert Committee, 2002).

Water has a key role in schistosomiasis transmission and spread. Human-to-environment transmission occurs when infected people contaminate freshwater bodies with their excreta containing parasite eggs. Environment-to-human transmission occurs when people are exposed to infested water during routine activities, ranging from agricultural to domestic and from occupational to recreational. Therefore, the disease is especially prevalent in rural communities. Lack of hygiene and certain play habits make school-aged children particularly vulnerable to infection, an aspect which must be regarded with care, because schistosomiasis may induce severe health consequences in absence of adequate treatments.

There are two major forms of schistosomiasis – intestinal and urogenital – caused by six species of blood flukes belonging to the genus *Schistosoma*, of which *S. haematobium*, *S. mansoni* and *S. japonicum* are the three most important ones (Colley et al., 2014). People become infected when larval forms of the parasite penetrate their skin during contact with infested water. The freely swimming, short-lived larval stages of the parasites are known as cercariae and are shed by some species of freshwater snails belonging to the genus *Bulinus* (for *S. haematobium*), *Biomphalaria* (for *S. mansoni*) or *Oncomelania* (for *S. japonicum*), which serve as species-specific obligate intermediate hosts for the parasites. Within the human body, the larvae need 5–7 weeks to develop into sexually mature adult schistosomes (Colley et al., 2014). Adult worms can live for a few years in the human blood vessels, where the females produce hundreds to thousands of fertilized eggs daily. Some of the eggs become trapped in body tissues, causing immune reactions and progressive damage to internal organs (e.g. liver),

* Corresponding authors.
E-mail addresses: manuela.ciddio@polimi.it (M. Ciddio), renato.casagrandi@polimi.it (R. Casagrandi).

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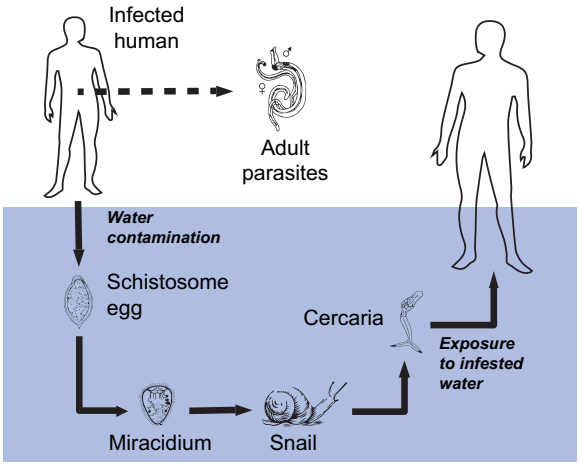

Fig. 1. Schistosoma life cycle. Adult schistosomes within infected human hosts produce eggs, which are shed in the environment through excreta. The eggs that reach freshwater can hatch into miracidia and infect species-specific intermediate snail hosts. Infective snails shed free-swimming cercariae that can penetrate human skin and eventually develop into reproductive worms. See text for further details about transmission.

other leave the human host by being shed in the environment through the feces (*S. mansoni* or *S. japonicum*) or urine (*S. haematobium*) to continue the parasite's life-cycle. The eggs released out of the human body that reach freshwater can hatch into a second short-lived larval form of the parasite, the miracidia, that are infectious for snails. In the snail, miracidia undergo asexual replication for 4–6 weeks (the so-called prepatent period; Colley et al., 2014), then the snail becomes infective and starts shedding hundreds of cercariae per day into water. A sketched scheme of the parasite life cycle is shown in Fig. 1.

The analysis of the coupled dynamics of human, parasite and snail populations, together with the free-living stages involved in the parasite's life cycle, are fundamental to describe and understand the transmission mechanisms of schistosomiasis. Previous studies have already shown that disease dynamics not only depend upon interactions between infectious agents and the hosts, but also that they are strongly affected by environmental factors (Gurarie and Seto, 2009; Perez-Saez et al., 2015). In addition, large-scale dynamics are better described by metapopulation models, which proved to be a powerful tool in order to understand disease persistence and infection intensity in human societies (Grenfell and Harwood, 1997; Hagenaars et al., 2004). In the case of schistosomiasis, the movement of infectious agents can occur via various transport processes involving hosts and pathogens, including human mobility, larval transport along canals and streams, and snails dispersal through hydrological interconnections. The spread of the disease under study is thus the result of the interplay between various mechanisms acting at different spatial and temporal scales. On the human host side, social connections provide a pathway for adult parasite transport while people travel between endemic and non-endemic areas. This movement can involve very large spatial scales in ways that are often difficult to predict (Remais, 2010), and constitutes an effective transmission mechanism provided that disease-transmitting snails live in the visited areas. On the snail and parasite side, connectivity via physical processes (hydrological transport and animal dispersal) increases the risk of larval and snail propagation over shorter spatial scales. As an example, all over the world, an estimated 63 million people at risk for schistosomiasis live in irrigated environments, with an increased relative risk of urinary and intestinal schistosomiasis of 1.1 and 4.7, respectively, compared with non-irrigated environments (Steinmann et al., 2006).

Here we explore a spatially realistic metapopulation model (Section 2), in which schistosomiasis spreads within a network of connected villages. The model is applied to the area of the Lower Basin of the Senegal River, in the northern part of Senegal (for more details, see Section 3). Social and environmental interconnections link villages through human mobility (direct transport of parasites by humans) and hydrology (a pathway for larvae and snails). Results are presented in Section 4, while a set of concluding remarks closes the paper (Section 5).

2. The model

The basis of our analysis is a spatially explicit nonlinear model that accounts for local epidemiological dynamics, human mobility, snail dispersal and hydrological transport of schistosome larvae. At the local scale, the model extends the work presented in Ciddio et al. (2015) by including the dynamics of the larval stages of parasites. The system of differential equations is expressed in terms of the human population size (N_v) and the total number of parasites (P_v) within human hosts living in each village (subscript v), the density of susceptible, exposed, and infectious snails (S_w, E_w, I_w) in the freshwater point (subscript w), and the concentration of cercariae (C_w) and miracidia (M_w) in the freshwater body. Early studies on the heterogeneity of schistosomiasis transmission (Barbour, 1978) already introduced a partitioning between human and animal host populations, but did not consider physical connectivity through the environment, an approach followed also in later works (Gurarie and King, 2005; Gurarie et al., 2010; Woolhouse et al., 1998; 1991). On the other hand, other studies (Gurarie and Seto, 2009; Perez-Saez et al., 2015; Xu et al., 2006) did consider the role of environmental connectivity (typically through larval dispersal alone), while at the same time neglecting the possible spatial mismatch between villages and water contact points (but see Remais, 2010, in which, however, snail dispersal is neglected). In our work, instead, n_v villages and n_w water points constitute two distinct sets of nodes of a fully coupled, multi-layered (multidimensional, sensu Boccaletti et al., 2014), spatially explicit network. Epidemiological dynamics can be described by the following set of $(2n_v + 5n_w)$ ordinary differential equations:

$$\begin{cases} \dot{N}_v = \mu_H(H_v - N_v) - \alpha P_v \\ \dot{P}_v = \mathcal{F}_v N_v - (\mu_H + \mu_P + \alpha)P_v - \alpha\dfrac{k+1}{k}\dfrac{P_v^2}{N_v} \\ \dot{S}_w = \nu S_w[1 - \gamma_w(S_w + E_w + I_w)] - \mu_S S_w - \rho M_w S_w + \mathcal{D}_w^S \\ \dot{E}_w = \rho M_w S_w - (\mu_S + \eta)E_w - \delta E_w + \mathcal{D}_w^E \\ \dot{I}_w = \delta E_w - (\mu_S + \eta)I_w + \mathcal{D}_w^I \\ \dot{C}_w = \zeta I_w - \mu_C C_w + \mathcal{L}_w^C \\ \dot{M}_w = \mathcal{G}_w - \mu_M M_w + \mathcal{L}_w^M. \end{cases} \quad (1)$$

At each village v, human hosts are characterized by a constant recruitment $\mu_H H_v$ (with H_v being the community size and μ_H being the per capita natality rate), and two loss contributions due to non-schistosomiasis-related deaths (with rate μ_H) and mortality induced by parasites (with α being a constant determining the pathogenicity of the parasite to the human host; Anderson and May, 1978). The human-to-schistosome interaction is modeled as a macroparasitic infection (Anderson and May, 1992), assuming that parasites are unevenly distributed among human hosts according to a negative binomial distribution with clumping parameter k (Feng et al., 2002). Parasite acquisition by human hosts is determined by contact with cercariae at infested water points, as described by the force of infection $\mathcal{F}_v$, expressed as

$$\mathcal{F}_v = \beta \sum_{j=1}^{n_w} \Omega_{vw} C_w \quad (2)$$