Endemic persistence and percolation transition on small-world networks

N R Bernardino, A Nunes & M M Telo da Gama

Centro de Física Teórica e Computacional & Faculdade de Ciências, U Lisboa, Av Prof Gama Pinto 2, 1649-003 Lisboa Codex, Portugal

1. Introduction

Stochastic models of epidemic spread and persistence can be mapped on percolation models [1]. In particular, the transition between extinction and persistence is a dynamical phase transition, from an absorbing to a fluctuating phase (endemic state) [2].

On regular lattices these transitions are well known: they belong to the isotropic percolation class [3] if the sites cannot be re-infected and to directed percolation if re-infection (birth of susceptibles, mutations of infectious agents, etc) occurs [4].

On small-world networks the transitions are mean-field percolation transitions. The scaling region, however, depends on the small-world parameter, p, and vanishes in the limit of p=0. The threshold depends on the model parameters: infection rate, small-world probability, infectious time and birth rate [5].

Here we address the problem of using this mapping on percolation approach to extract relevant epidemiological information for disease persistence on dynamic complex networks. We consider a class of diseases with the general characteristics of childhood infections: an infectious period several orders of magnitude smaller than the average life time and lifelong immunity upon recovery.

2. The model

In the basic epidemiological model, SIR [7], a community of N, constant, individuals is divided in three classes: susceptibles, S, infectious, I and recovered R. In terms of the densities s and i the equations are:

$$\frac{\mathrm{d}s}{\mathrm{d}t} = -\beta si - \mu s + \mu \tag{1}$$

$$\frac{\mathrm{d}i}{\mathrm{d}t} = \beta si - (\gamma + \mu)\, i \tag{2}$$

Where constant infection, β, recovery, γ and birth/death, μ rates are assumed. Endemic equilibrium occurs at $i_0 = \mu(1/(\gamma+\mu) - 1/\beta)$ and $s_0 = (\gamma+\mu)/\beta$.

The stochastic version of the model considers a discrete population and quantized jumps among the classes given by the probabilities:

$$Pr\{(S,I) \rightarrow (S-1, I+1)\} = \frac{\beta}{N} SI\Delta t \tag{3}$$

$$Pr\{(S,I) \rightarrow (S, I-1)\} = \gamma I\Delta t + \mu I\Delta t \tag{4}$$

$$Pr\{(S,I) \rightarrow (S+1, I)\} = \mu N\Delta t - \mu S\Delta t \tag{5}$$

In order to include spatial correlations a cellular automaton version of the stochastic model on a small-world network with an underlying square lattice and 12 neighbors was considered in [6]. A deterministic recovery time was used.

3. The persistence transition

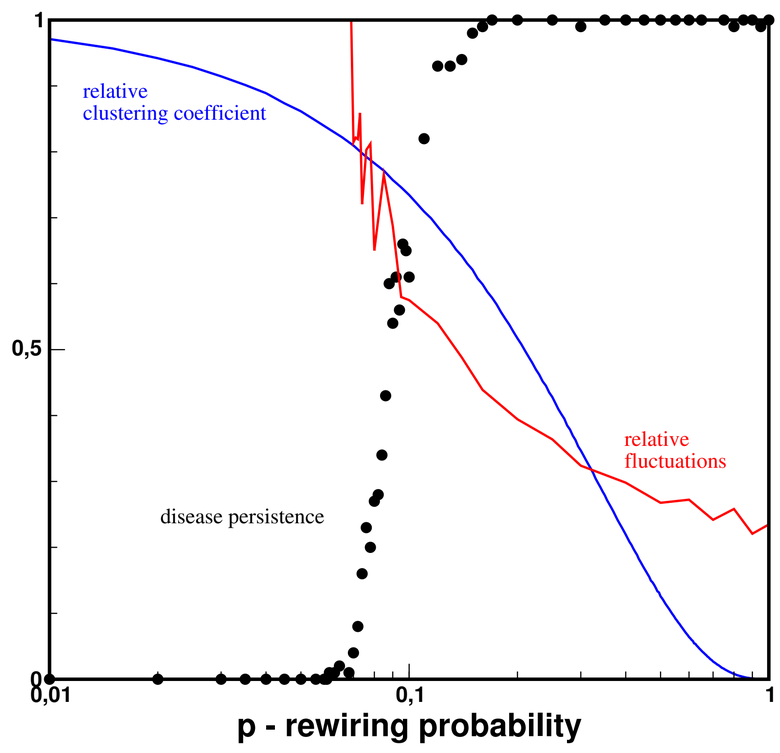

Figure 1. Persistence (fractions of the simulations surviving a given number of time steps) as a function of the small-world parameter p for $N = 250000$, $\mu = 0.0006$ day^{-1}, $\gamma = 0.0625 = 1/16$ day^{-1} and $\beta = 0.66$ day^{-1}. From [6].

In [6] it was observed that the transition from disease extinction to epidemic persistence occurs over a narrow range of p. When the clustering is large, fluctuations dominate and stochastic extinction occurs even for large N.

When $p = 1$ the fluctuations are gaussian and the epidemic persists for sufficiently large N. The transition occurs in the region of intermediate clustering, i.e., at the edge of the small-world regime.

4. Percolation on an effective model

We wish to compare the behaviour of realistic epidemiological models with finite birth rates with standard models of percolation, when both models are implemented on small-world networks.

For $\mu = 0$ and $N = \infty$, all the relevant quantities were calculated with the methods of [8]. However, in open and finite populations, there is no alternative to a direct simulation of the epidemiological model.

The basic assumption is that for small values of μ and times of the order of the epidemiological records, the main effect of the birth rate is sustaining the value of the average value of s, s^*, which plays the role of a site percolation probability.

We consider site and bond percolation on a small world network with $N = 250000$ nodes. We calculated the critical line $s^*(p)$, where p is the small world probability. The other parameter p_{bond} is fixed through the infectiousness of the disease, β (Figure 2).

In the same figure we plot $s^*(p)$ obtained from the simulations of a small world cellular automaton (CA) implementation of the stochastic model (eqs. 3-5). The averages were taken after an appropriate transient, as p is decreased from 1 down to the value where extinction occurs.

The steady state behaviour of the CA lies within the "critical region" of the percolation model and extinction occurs close to the percolation threshold for that value of s^*.

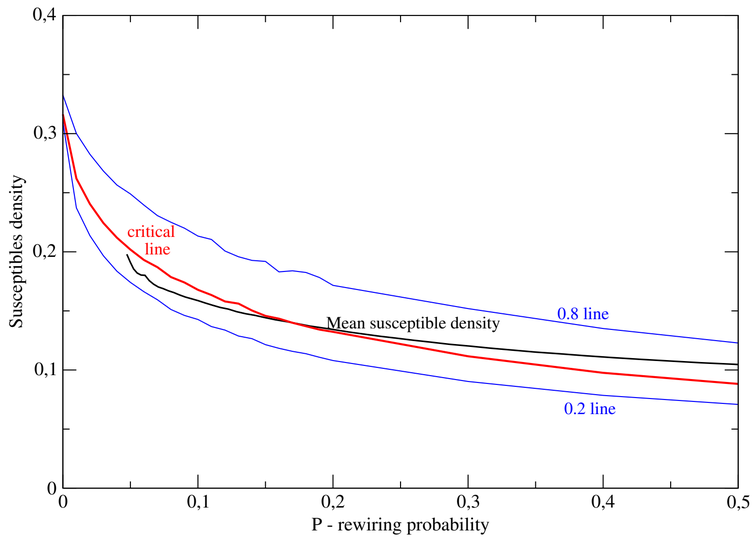

Figure 2. The percolation critical line and the extinction point. The critical line is calculated as the point at which the mean fraction of infected nodes in an epidemic is 0.5. The 0.2 and 0.8 lines are the points at which these fraction are infected and are a measure of the critical region. All parameters as in Figure 1.

We find that for models with $\mu \neq 0$ the persistence transition occurs close to the critical line of the percolation model (Figure 3).

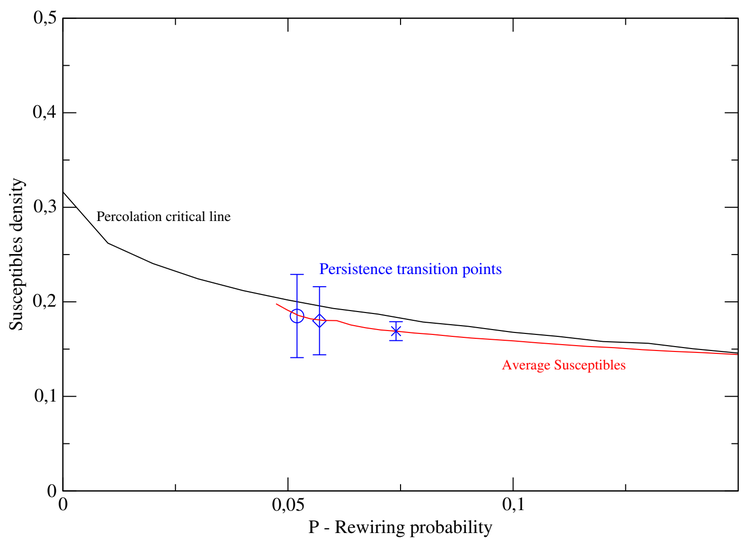

Figure 3. The percolation critical line and the persistence transition points for three values of μ: circle $= 1.2 \times 10^{-3}$, diamond $= 10^{-3}$ and cross $= 6 \times 10^{-4}$.

5. Conclusions

It is well known that in the mapping on percolation approach, the parameters of a closed model for epidemic spread are mapped on the bond percolation probability. For models with realistic birth rates, or models with a small rate of infection, our results show that the persistence threshold for the small-world parameter is close to that of site and bond percolation on a small-world network.

The results suggest that for realistic values of μ, one order of magnitude smaller than the ones used here, the percolation model is a sufficiently good approximation to calculate the persistence threshold for any finite population.

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Acknowledgements

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