

ECONOMIC AND SOCIAL FACTORS IN DESIGNING DISEASE CONTROL STRATEGIES FOR EPIDEMICS ON NETWORKS*

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Models for control of epidemics on local, global and small-world networks are considered, with only partial information accessible about the status of individuals and their connections. The main goal of an effective control measure is to stop the epidemic at a lowest possible cost, including treatment and cost necessary to track the disease spread. We show that delay in detection of infectious individuals and presence of long-range links are the most important factors determining the cost. However, the details of long-range links are usually the least-known element of the social interactions due to their occasional character and potentially short life-span. We show that under some conditions on the probability of disease spread, it is advisable to attempt to track those links, even if this involves additional costs. Thus, collecting some additional knowledge about the network structure might be beneficial to ensure a successful and cost-effective control.

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1. Introduction

One of the main goals of epidemiological modeling is to provide guidelines for controlling disease outbreaks. Traditionally this has been understood in terms of reducing the number of infected individuals. With a cheap vaccination available, “blind” vaccination of a large proportion of individuals

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might be a simple and yet optimal solution [1]. However, in many cases the epidemic must be stopped at a manageable cost and with potentially limited resources, leading to a mixture of preventive and responsive measures. In the simplest case the goal of a successful prevention and eradication programme is to minimize a number of individuals who have either been treated or have been through the infection.

In a series of previous papers [1,2] we have studied the suitability of local control strategies for stopping spread of diseases on networks with a mixture of local and global links. These include “small-world” networks [3], with a majority of contacts between nearest neighbors and a small number of global links. By a local strategy we understand control measures limited to some neighborhood of an infected individual. We have proposed a strategy that is a mixture of responsive and preventive actions. A control event is triggered by an appearance of a symptomatic individual (responsive measure) and spans not only this individual but also its immediate neighbors on a certain control network (preventive measure).

The preventive control (analogous to a ring-vaccination strategy) is necessary because of the delay between the onset of infectiousness of an individual and the onset of symptoms. Thus, there is a possibility of pre-symptomatic yet infectious individuals to be located in the neighborhood of the observed disease case. The preventive local control strategy attempts to treat such potential cases. The crucial assumption in our paper is that the network that defines the control neighborhood is only a subset of the network on which the disease spreads and in particular contains only a subset of long-range links. This reflects the limited ability of medical authorities to track and follow contacts between individuals leading to spread of the disease. In particular, we ask the following question: how detailed should our knowledge be of the network structure to be able to stop the disease at the lowest possible cost? We compare different strategies by looking at the final size of the epidemics including individuals who have been through the disease as well as those treated [2]. We also include an additional cost associated with tracking of long-range links.

2. Model

The model of epidemic spread and the associated control must take into account the topology of the network on which the epidemic spreads, the topology of the sub-network which is used for control, the state of each individual and transitions between different states. We consider two basic topological structures, a 1-dimensional small world topology (SW1D) [3] and 2-dimensional small world topology (SW2D). The disease spreads on the full network, including local and global links. The control measures can

only follow a subset of those links and in particular for the SW1D and SW2D topologies we assume that the subset contains all local links and a subset of additional shortcuts. This approach is caused by the fact that it is much easier to track local interactions, interactions with surrounding individuals, fields and farms, than long-range interactions, which might be caused by geographical, technical, cultural or economical factors.

The epidemiological part of the model is based on an SIR model [4] modified so that it includes pre- and post-symptomatic individuals (who can both contribute to the spread of the infection) and recovered as well as treated individuals.

2.1. Topology

SW2D topology is constructed from a regular lattice, with periodic boundary conditions, to which a given number of additional random shortcuts is added. Thus, every individual placed on the SW2D topology interacts with its four nearest neighbors and some other individuals via additional shortcuts (Fig. 1). The SW1D topology is constructed in a similar way, by adding long-range links to a one-dimensional ring. For compatibility between SW2D and SW1D topology every node of the initial ring has 4 first order neighbors, 2 of them located on the left-hand side and 2 on the right-hand side of the given node.

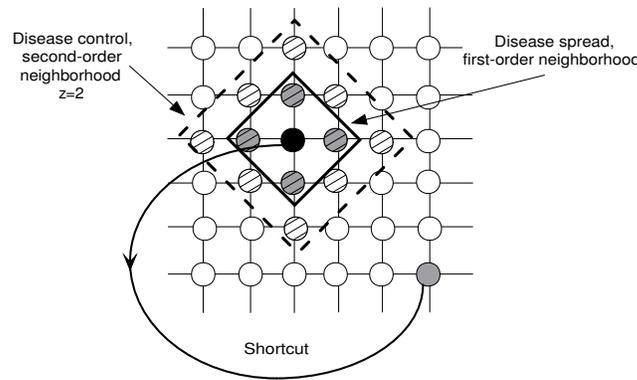


Fig.1. SW2D topology: In this example, a detected individual (black circle) is in contact with its four nearest-neighbors on the disease network and to one node connected by a shortcut (gray circles to indicate non-symptomatic infected individuals). The control might then be applied locally, limited to the eight second-order neighbors and individual itself on a treatment network (marked by a square). In general, a given ratio of additional shortcuts can be incorporated in the disease control neighborhood making control more efficient.

In the first instance, the control network contains the regular (local) part of the infection network. In addition, we assume that a certain number of long-range links is included in the control network. This reflects an additional effort that a government or health authority put into disease tracking. A control neighborhood of given order, z , is constructed in an iterative way. Starting from the infected node the first order control neighbors are localized. The second order neighbors are then found as first order neighbors of the previous group. The whole procedure is repeated iteratively z times. A single control action involves all individuals in the control neighborhood of order z .

2.2. Individuals and transitions

Individuals are placed on a given topology and can be in one of the following states:

1. **S** – susceptible (or healthy), which can be infected with probability p by any infectious or detected individual in its epidemic neighborhood;
2. **I** – infectious (infected but pre-symptomatic); can infect other nodes from its epidemic neighborhood but cannot trigger a control measure. In addition, with probability q it can spontaneously move to the detected class, *i.e.* symptoms become observable;
3. **D** – detected (infected and symptomatic), can infect other nodes from its epidemic spread neighborhood. In addition, it can spontaneously move to the recovered class (with the probability r) or can trigger a treatment measure with the probability v that includes all individuals within its control neighborhood;
4. **R** – recovered. This class includes individuals that have been through the disease, can be treated but cannot become re-infected;
5. **V** – vaccinated (treated). Individuals in this class have been in a control neighborhood of a detected individual when the treatment event was triggered. They cannot become re-infected.

We assume that all nodes in the network are occupied. The initial state is a mixture of a majority of susceptible individuals with an addition of few (0.1%, 0.5% or 5%) infectious (symptomatic) individuals. We denote the total number of nodes by N and the number of susceptible nodes by S , infected by I , detected by D , recovered by R and treated (vaccinated) by V .

2.3. Simulations

Details of the simulations are given in [1, 2]. The model was updated synchronously and the simulation loop was performed until the number of infected individuals was equal to zero, *i.e.* until T_{\max} such that $I(T_{\max}) +$

$D(T_{\max}) = 0$. In every iteration, spontaneous transitions from $I \rightarrow D$, $D \rightarrow R$ and state-dependent transitions $S \rightarrow I$, $D \rightarrow V$ were performed.

We consider three treatment strategies, random vaccination, local vaccination and a mixed strategy combining local vaccination with tracking of long-range links. In the random “blind” vaccination, the given ratio of randomly chosen individuals is vaccinated shortly after the first detection of the disease. For local treatment all individuals up to a given order z surrounding and including the detected infected individual, are vaccinated regardless of their current disease status. For the mixed strategy, a certain proportion of long-range links is also tracked and individuals to which the detected individual is linked are treated as well.

For a given set of parameters the simulation was averaged over 50 realizations for the total number of nodes equal to 2500 (*i.e.* the SW2D topology is created from the square 50×50 lattice), with or without addition of a fixed number of 1023 long-range links. Larger sizes of the networks and larger number of realizations were explored as well, but they did not improve or change the results.

3. Results

Simulation results were analyzed to extract information that is relevant for the design of an optimal control strategy. In particular, we look at a severity index, a combined number of treated and recovered individuals, $X \equiv R(\infty) + V(\infty)$ at the end of an epidemic. This quantity represents the combined severity of an untreated epidemic, $R(\infty)$, and the costs of treating it, $V(\infty)$. In this paper, we mainly focus on effects of a network structure (including shortcuts) and probability of spread, p , on the severity index, X , of the epidemic and the optimal extent of a control neighborhood z_c . z_c is defined as such a diameter of control neighborhood for which $X(z_c)$ is minimal. All other parameters, except r which was set to 0.01, take all possible values from the allowed domains and $z \in \{1, 2, \dots, 15\}$. In addition, we vary the structure of the control network by changing the proportion ($T_L = \{0\%, 10\%, 20\%, \dots, 100\%\}$) of long-range links (shortcuts) that are tracked and included in the control neighborhood.

We first consider a “blind” vaccination strategy, Fig. 2 and assess the effect of different proportions of vaccinated individuals on the impact of disease. This strategy is effective when applied early and the number of non-local links is small, see Fig. 2. Addition of long-range links or delaying the application of the “blind” treatment renders it ineffective, *cf.* Fig. 2. In addition, from the social point of view, such a strategy is difficult to accept, because it is purely preventive and control measures are focused only on initial vaccination of randomly chosen individuals without any further actions during the outbreak.

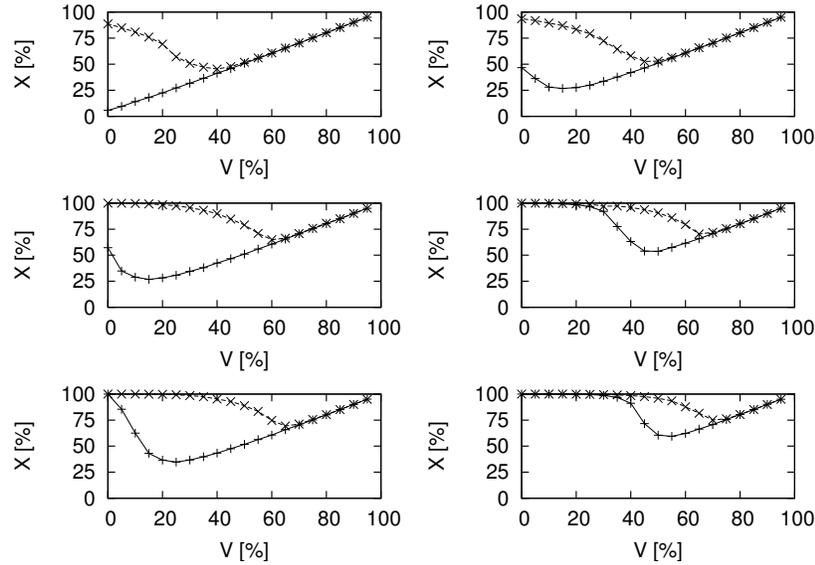


Fig. 2. $X = R(\infty) + V(\infty)$ as a function of proportion of initially vaccinated individuals for SW1D topology (left panel) and SW2D topology (right panel) for various values of infection probability, p : $p = 0.01$ (top panel), $p = 0.05$ (middle panel) and $p = 0.5$ (lower panel). Other parameters: $q = 0.5$, $r = 0.01$. Different symbols correspond to various numbers of additional shortcuts: “+” 0 shortcuts, “x” 1023 shortcuts. Initially, at $t = 0$, 0.5% of all individuals were in the symptomatic class.

The next group of possible control strategies is characterized by a mixture of responsive and preventive actions. As new foci of the disease are formed and spread, they trigger control measures that are applied in a broader neighborhood of detected symptomatic individuals. The extended control neighborhood compensates for the lack of our knowledge about the exact state of individuals and the exact structure of interactions. The severity index $X \equiv V(\infty) + R(\infty)$ is plotted in Fig. 3 as a function of infection probability p and the control neighborhood size, z . For each value of p , there exist an optimal value z_c for which the control measures are most efficient. If $z < z_c$ the disease escapes the control, while for $z > z_c$ too many individuals are vaccinated. The exact shape of the surface depends on network properties and epidemic parameters. Nonlocal interactions make minima less pronounced; nevertheless, purely local strategies are capable of stopping epidemics even in the presence of long-range links [2].

Epidemics can spread not only to the nearest neighbors but also, via non-local shortcuts, to distant part of the network. On the one hand, long-range links are crucial for the spread of the outbreak. On the other hand

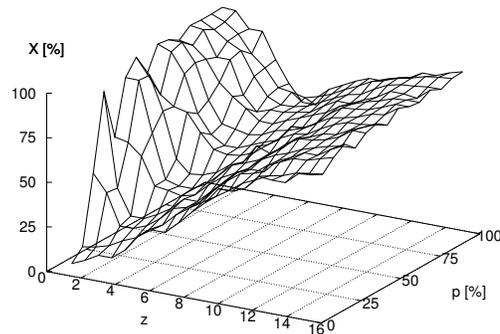


Fig. 3. $X \equiv R(\infty) + V(\infty)$ as a function of the infection probability p and diameter of the vaccination z for SW2D network with 63 additional shortcuts. Other parameters: $q = 0.5$, $v = 0.1$ and $r = 0.01$. Initially, at $t = 0$, 0.5% of all individuals were in the symptomatic class.

they are hard to identify and their identification requires an additional cost. Therefore, for knowledge oriented strategies, more general cost functions need to be considered. We propose $X \equiv V(\infty) + R(\infty) + \alpha L_T$, where L_T represents the ratio of identified to the total number of shortcuts and α is the cost associated with contact tracking.

Figs. 4–5 show ratio of tracked links (top panel), the number of vaccinated individuals (middle panel) and cost function X (lower panel), corresponding to the optimal solutions. In the following we examine the influence of incubation time, controlled by q , and effectiveness of the vaccination, v , on the optimal strategy.

For the parameters used in this paper, the cost associated with an optimal strategy is generated mainly by vaccination and links tracking. The relative importance of these two factors depends on the cost of tracking a single long-range link, $\alpha' = \alpha/1023$. When links tracking is cheap, it is optimal to track all shortcuts, see Figs. 4–5 (top panel). When disease incubation time is long (small q) and vaccination is inefficient (small v) detailed contact tracking is less important and costs are largely influenced by treatment, *cf.* Fig. 4. The combined effect of the long incubation time and low effectiveness of vaccination decrease the effect the additional knowledge about long range links has on the control. When the incubation time is long, epidemics can infect large proportion of individuals before they are detected. For short incubation times (large q) and more effective treatment (large v), there is a clear distinction between strategies applying contact recognition and purely local strategies, *cf.* Fig. 5. The recognition of short-

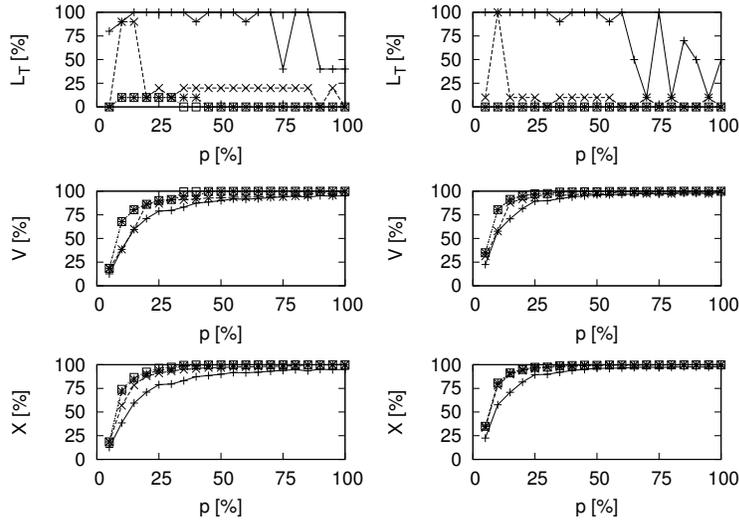


Fig. 4. Ratio of the tracked links L_T (top panel), proportion of the vaccinated individuals $V(\infty)$ (middle panel) and $X \equiv R(\infty) + V(\infty) + \alpha L_T$ (bottom panel) as a function of the infection probability p for SW1D topology (left panel) and SW2D topology (right panel). Other parameters: $q = 0.1$, $v = 0.1$ and $r = 0.01$. Initially, at $t = 0$, 0.5% of all individuals were in the symptomatic class. Different symbols correspond to various cost of a single non-local link tracking α' : “+” 0 , “x” 0.5, “*” 1.0, “□” 1.5.

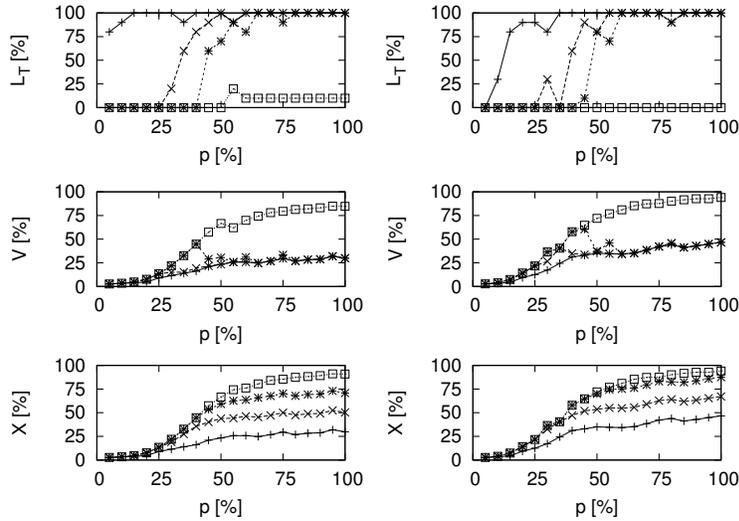


Fig. 5. The same as in Fig. 4 for $q = 0.5$, $v = 0.5$.

cuts, despite the associated costs, can significantly decrease the number of individuals that need to be vaccinated to eradicate epidemics. Furthermore, such strategies lead to smaller value of the severity index X than purely local strategies, *cf.* middle and bottom panels of Fig. 5.

4. Discussion

Designing control strategies for networks incorporating long-range links is complicated. In the simplest case, we envisage treating infected and/or susceptible individuals so that the disease progress is slowed down or even stopped. Examples of such treatment include preventive vaccination, culling of animals and quarantine of fields or individuals. For networks with only short-range interactions the spread of a disease is geographically limited and can therefore, be contained locally [2]. For non-local networks there is always a possibility of infection jumping to another location to form a new focus. In designing control strategies for such networks it is necessary to know not only the geographical location of new cases (so that they and their immediate neighbors can be treated) but also all possible connections that can span the whole population. Obtaining this information can be very expensive and time consuming. With the authorities faced by a large-scale epidemic the collection of such data might be difficult and might lead to many inappropriate decisions. It is thus imperative to use epidemiological models to explore the possibilities of simplifying the control strategies.

Most models of disease spread used to predict its advance and to design efficient control measures assume a perfect knowledge of both the status of each individual (healthy *versus* infectious) and the network structure (who acquires the disease from whom [4, 5]). Among the epidemiological parameters, the difference between the onset of infectiousness and the earliest detectability of the disease is the key issue for controlling the disease. For most diseases an individual can be infectious even though the infection cannot be detected and controlled. Such an individual can be a source of further infections for a relatively long time until the source is identified and controlled by isolation or treatment. In many cases, even post-symptomatic individuals cannot be treated straight after the detection, which further adds to a spread of the epidemic. Control strategies should aim at decreasing the time until control measures are applied by increasing detectability and speeding up control.

We have shown that long-range links dramatically reduce the effectiveness of local control measures. Our results show that in some cases it is possible to control epidemics with only limited knowledge about interactions between individuals. If this is not possible, our model gives guidance on conditions under which it is advisable to attempt to track long-range

links, despite the high costs associated with such a strategy. From the economic point of view, contact tracking is important when disease incubation time is short and vaccination is efficient. Furthermore, if the epidemic is highly infectious, knowledge oriented strategies lead to a significant decrease in the severity index characterizing the costs of disease eradication.

There is a clear distinction between the case when the control measure works and when it does not. If the control neighborhood is too small, or we track insufficient numbers of long-range links, the disease keeps escaping the treatment and as a result we need to treat practically the whole population. Making the ring of control even a fraction larger might lead to a dramatic increase in the efficiency of the control strategy. Similarly, incorporating more long-range links might improve the effectiveness of the control measures.

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REFERENCES

- [1] B. Dybiec, A. Kleczkowski, C.A. Gilligan, *Acta Phys. Pol. B* **36**, 1509 (2005).
- [2] B. Dybiec, A. Kleczkowski, C.A. Gilligan, *Phys. Rev.* **E70**, 066145 (2004).
- [3] D.J. Watts, S.H. Strogatz, *Nature* (London) **393**, 440 (1998); D.J. Watts, *Small Worlds*, Princeton University Press, Princeton NJ 1999; A. Kleczkowski, B.T. Grenfell, *Physica A* **274**, 355 (1999); C. Moore, M.E.J. Newman, *Phys. Rev.* **E62**, 7059 (2000).
- [4] R.M. Anderson, R.M. May, *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, Oxford 1991.
- [5] R. Pastor-Satorras, A. Vespignani, *Phys. Rev.* **E65**, 036104 (2002); Z. Dezső, A.L. Barabási, *Phys. Rev.* **E65**, 055103 (2002).