OPTIMISING CONTROL OF DISEASE SPREAD ON NETWORKS*

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Dedicated to Professor Andrzej Fuliński on the occasion of his 70th birthday

We consider models for control of epidemics on local, global, small-world and scale-free networks, with only partial information accessible about the status of individuals and their connections. The effectiveness of local (*e.g.* ring vaccination or culling) *vs* global (*e.g.* random vaccination) control measures is evaluated, with the aim of minimising the total cost of an epidemic. The costs include direct costs of treating infected individuals as well as costs of treatment. We first consider a random (global) vaccination strategy designed to stop any potential outbreak. We show that if the costs of the preventive vaccination are ignored, the optimal strategy is to vaccinate the whole population, although most of the resources are wasted on preventing a small number of cases. If the vaccination costs are included, or if a local strategy (within a certain neighbourhood of a symptomatic individual) is chosen, there is an optimum number of treated individuals. Inclusion of non-local contacts ('small-worlds' or scale-free networks) increases the levels of preventive (random) vaccination and radius of local treatment necessary for stopping the outbreak at a minimal cost. The number of initial foci also influences our choice of optimal strategy. The size of epidemics and the number of treated individuals increase for outbreaks that are initiated from a larger number of initial foci, but the optimal radius of local control actually decreases. The results are important for designing control strategies based on cost effectiveness.

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1. Introduction

One of the main goals of epidemiological modelling is to provide guidelines for controlling disease outbreaks [1]. Traditionally this has been understood in terms of reducing the number of infected individuals. Recent outbreaks of large-scale epidemics like AIDS, malaria, foot-and-mouth, avian influenza or rhizomania as well as a possibility of a smallpox pandemic have brought a new dimension to the studies. Not only must the epidemic be stopped as quickly as possible but also at manageable cost and with potentially limited resources. The problem of minimising the number of infected individuals becomes only a part of an optimisation problem in which infection on one side and control measures on the other side generate costs.

Typically two cases of disease spread are considered, with appropriate control measures. Thus, for a purely local case, local measures like ring culling, vaccination or quarantine are suggested [2]. At the other extreme, when any individual can be in contact with any other individual in the population (even if the number of contacts is limited), global measures like a mass vaccination [1] seems to be a better option. More recently, attention has been given to another mode of disease spread, in which the contact structure presents a mixture of local and global links. Termed 'small-world' networks [3], the underlying structure of contacts potentially leading to a disease advance, present an interesting and challenging case for the control of diseases. While the choice between local and global control measures seems to be clear for local and random networks, it is not clear how to design control for 'small-worlds'.

The modelling and prediction is made even more complicated by the fact that not all information about disease dynamics and about the underlying network structure is available. Among the epidemiological parameters and processes, the key problem lies in a difference between the onset of infectiousness and the earliest detectability of the disease and then application of control measures. For the network structure, long-range links are notoriously difficult to identify and to follow.

In this paper we compare the suitability of various control measures designed for the spread of diseases on networks with local, global and 'small-world' connectivity. We have recently shown [4] that under some conditions it is possible to control disease spread by using purely local methods applied in a neighbourhood centred around a detected infectious individual, even for networks with non-local interactions. We have shown that if all costs of disease are included, there exists an optimal radius for such a control neighbourhood leading to the lowest severity of the epidemic in terms of economic costs associated with disease and treatment [5]. The efficiency of such a local control strategy is very sensitive to the choice of the radius.

However, many unresolved issues remain. In this paper we extend the analysis in [4] in several directions. First, in addition to local control strategies, we also include a preventive random vaccination, in which susceptible individuals are treated *before* any potential outbreak. Traditional modelling concentrated on the potential risks and costs associated with the outbreak, characterised by the total number of individuals who have been through the disease progress by the end of the outbreak. However, in many cases vaccination is in itself a source of costs and any potential strategy should included this as well. In this paper we show that inclusion of vaccination costs leads to an optimal control strategy that is different to the 'traditional' one, based on minimising disease risks.

Second, we study the effect of initial conditions on the spread and control of disease outbreaks on lattices including both local and non-local interactions. In particular, we show that the size of potential outbreaks, the number of treated individuals and the optimal radius of a local control, depend in a non-trivial way on the number of initial foci.

Finally, we formulate the detailed mechanism responsible for a large difference between regular networks and scale-free networks in the behaviour of the control of epidemics at the optimum strategy. For scale-free networks [6] that involve a lot of long-range contacts, it is necessary to treat almost everybody in the population, even at the optimum [4]. This stands in a contrast to local networks and small-world networks with a small number of links. In this paper we show that this behaviour is caused directly by a structure of the scale-free networks, whereby even a single neighbourhood of an order larger than one, covers on average a large proportion of individuals on the whole network.

Although our work deals mainly with the theory of networks, it has important biological applications. Notable among these are SARS [7], seal distemper virus (SDV) [8], foot-and-mouth disease (FMD) [2], Dutch Elm disease [9], citrus canker [10], sudden oak death [11] and rhizomania [12]. The nodes on the networks are formed by individual people or groups of people (SARS), animals or farms (seal distemper virus, FMD), fields or farms growing sugar beet (rhizomania) or trees (Dutch Elm disease, citrus canker and sudden oak death). The structure of network edges is dictated by spread of infectious agents, either by a direct contact (SARS, SDV), by sharing farm machinery (rhizomania, FMD), or by rain, wind or insect vectors (Dutch Elm disease, sudden oak death, citrus canker).

2. Model

2.1. Network structure

The structure of interactions between individuals in a population can be captured by a network topology that includes short- and long-range links [4]. We consider two types of topologies, networks with short-range links only and networks with a mixture of short- and long-range links. Most of the time individuals are in a direct contact with their geographical neighbours, leading to the following topological structures: a 1-dimensional chain with periodic boundary conditions (ring) and a 2-dimensional regular lattice with periodic boundary conditions. In addition to these local contacts, individuals sporadically interact on longer distances. Thus, more realistic interactions are implemented by adding a given number of shortcuts to these structures. Addition of shortcuts results in the 1-dimensional small world topology (SW1D) and the 2-dimensional small world topology (SW2D) respectively [3]. Finally, we consider the scale-free topology (SF), characterised by preferential attachment [6] whereby some individuals are very highly connected, while most have only few links. The scale-free network is constructed by adding nodes to the initially fully connected core, in such a way that the probability that a new node is attached to a given node is proportional to the number of nodes already attached to that node.

We also distinguish between an epidemic network and control network, with the latter being a subset of the former, reflecting limited knowledge about long-range contacts. For the SW1D topology, the disease spreads on a ring with addition of shortcuts, whereas the basic control topology encompasses the ring only, Fig. 1. The same mechanism is applied for the SW2D topology, Fig. 2, where the control topology corresponds to a square lattice with 4 nearest neighbours. For the SF network, a more complicated algorithm is needed to reflect various levels of knowledge about the contact structure. In this case, for nodes outside a fully connected core of size C, only the first C_{I} or C (whichever is smaller) links were chosen. This mechanism reflects our limited ability to track contacts. For nodes from the core, every node is connected to up to C_{I} or $C-1$ (whichever is smaller) older nodes. Due to the fact that links are not directed, such a mechanism guarantees that resulting control network is not disconnected and with increasing C_{I} a larger ratio of links from the full topology is included in the control topology, Fig. 3. For $C_{\mathrm{I}} = C$, the control topology is equivalent to the disease spread topology.

The local control is performed onto another topology, which forms only a subset of a full topology on which disease spreads. In our approach this reflects our limited knowledge about interactions between individuals.

2.2. Disease dynamics

Disease spreads on networks, changing status of individuals which are placed on the nodes. The individuals can be in one of five exclusive states:

1. ***S*** — susceptible, can be infected with probability p by any infectious or detected individual that is in its epidemic neighbourhood;

2. ***I*** — infectious (infected but pre-symptomatic), can infect other nodes within its epidemic neighbourhood but cannot trigger a control measure; can spontaneously move with probability q to the detected class, when symptoms become observable;

3. ***D*** — detected (infected and symptomatic), spreads disease in a similar way as infectious nodes. Furthermore, it can spontaneously move to the recovered class (with probability r) or can trigger a treatment measure with probability v;

4. ***R*** — recovered. Individuals in this class can be treated but cannot become re-infected, *i.e.* they do not return to ***S*** class;

5. ***V*** — vaccinated (treated). Individuals in this class are in a control neighbourhood of a detected individual when the treatment event is triggered. They cannot become re-infected, *i.e.* they do not return to ***S*** class again.

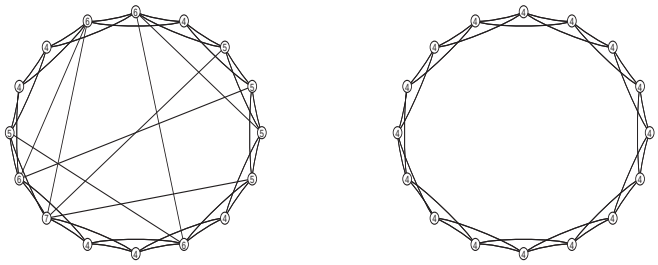

Fig. 1. SW1D network $N = 16$ with additional 8 shortcuts (left panel) and corresponding control network (right panel). The pictures were created by use of [13].

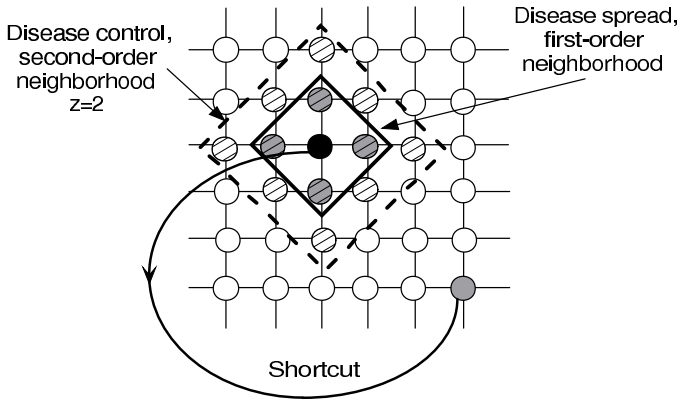

Fig. 2. SW2D topology: here a detected individual (black circle) is in contact with its four nearest-neighbours on the disease network and to one node connected by a shortcut (grey circles). The control might then be applied locally, *i.e.* in that case all additional shortcuts are excluded from the control neighbourhood, limited to the eight second-order neighbours ($z = 2$) and individual itself on a treatment network. Different colours represent individuals in various states: white — $\boldsymbol{S}$, grey — $\boldsymbol{I}$ and black — $\boldsymbol{D}$.

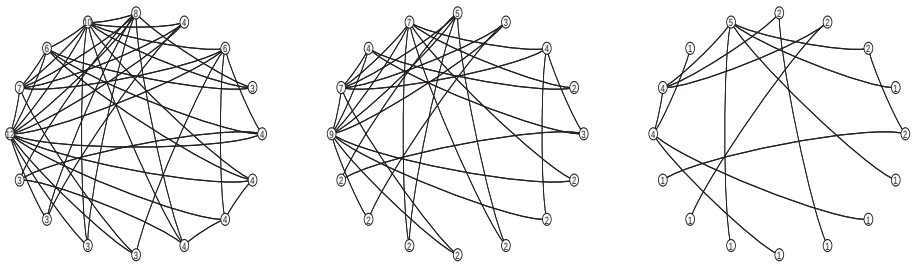

Fig. 3. SF network $N = 16, C = 3$ (left panel) and corresponding control networks $C_{\mathrm{I}} = 2$ (middle panel) and $C_{\mathrm{I}} = 1$ (right panel), number at nodes represent nodes' degree, *i.e.* the number of links attached to the node. The pictures were created by use of [13].

The model is a generalisation of the classical SIR models [1]. The following diagram lists all model transitions and probabilities:

$$\begin{array}{ccccccc} & & & & & & \boldsymbol{R} \\ & & & & & \overset{r}{\nearrow} & \\ \boldsymbol{S} & \overset{p}{\rightarrow} & \boldsymbol{I} & \overset{q}{\rightarrow} & \boldsymbol{D} & & . \\ & & & & & \underset{v}{\searrow} & \\ & & & & & & \boldsymbol{V} \end{array} \tag{1}$$

2.3. Control strategies

We consider a range of control schemes, broadly divided into non-local and local strategies. The simplest example of a non-local strategy is a random vaccination of a given ratio of individuals in the population. The main advantage of this strategy is that no information about social contacts and state of individuals is required. This strategy can also be applied preventively before epidemics outbreak. However, for highly infectious diseases, a large proportion of the individuals needs to be vaccinated [1], and so it is only optimal if the vaccination is cheap. We will discuss the choice of optimal strategies in the next section.

The alternative group of strategies assumes that we possess some knowledge about a location and timing of disease cases, at least for symptomatic individuals. In our approach, an appearance of a symptomatic individual may trigger a treatment 'event' with a certain probability v, and for local strategies we assume that treatment is limited to the control neighbourhood of the symptomatic individual, on a control network constructed as above. The control neighbourhood of a given order, z, is constructed in an iterative way. Starting from the infected (and symptomatic) individual all the first order neighbours on its control network are allocated. Subsequently, starting from the first order neighbours, the same procedure is applied in order to find the second order neighbours. The whole procedure is repeated z times. In a single control event, all (or only some) individuals in a control neighbourhood of a given range are treated, with the neighbourhood centred on the symptomatic individual, Fig. 2. The diameter of vaccination neighbourhood, z, is a parameter in the simulation.

2.4. Simulations

Simulations were performed for each topology consisting of $N = 2500$ individuals (for SW2D topology the starting point was a 2-dimensional square lattice 50×50 with periodic boundary conditions). We also studied individual disease progress curves (see the next section) and used a larger system (300×300) to decrease variability. All results were averaged over 50 realisations. At $t = 0$, the system with a given proportion (0.005%, 0.1%, 0.5% or 5% of the total population) of infectious symptomatic individuals ($\boldsymbol{D}$) is generated. The system is updated in a synchronous way till the time when epidemic die out due to lack of infectious individuals, *i.e.* for $t < T_{\max}$ such that $I(T_{\max}) + D(T_{\max}) \equiv 0$. In every iteration the status of each node is checked and all admissible transitions described by Eq. (1) performed.

2.5. Optimal strategy

In our paper we consider the existence and form of an optimal strategy that allows to stop epidemics at a manageable cost. The economics of disease and its control are summarised here by introducing a severity index $X = a\,R(\infty) + b\,V(\infty)$. Thus, the severity index depends linearly on the number of individuals that have been through the disease $R(\infty)$, with an individual cost of each case a. The cost might include hospitalisation, drug treatment and loss of work. The second term represents individuals that have been treated preventively $V(\infty)$, for example by vaccination, with an individual cost of b. In general, more sophisticated cost functions are possible, including non-linear terms. Our approach differs from the 'usual' one used in epidemiology in designing vaccination strategies, in that we intend to minimise X rather than $R(\infty)$.

3. Results

3.1. Epidemic outbreaks without control

The parameters for the disease spread are chosen in such a way that this always leads to a major outbreak in the absence of any control measure. The course of an epidemic typically follows a rise-and-fall curve for infected ($\boldsymbol{I}$) and detected ($\boldsymbol{D}$) individuals, and a sigmoidal curve for the number of recovered individuals ($\boldsymbol{R}$), Fig. 4 ($z = 0$, top panel). We show results for SW2D only; the results for other topologies are similar. The left panel of Fig. 4 corresponds to the epidemics outbreak on the regular lattice (SW2D topology without additional shortcuts) while the right panel to the regular lattice with 1000 additional shortcuts. The lattice size is 300×300 and initially 0.005% (*i.e.* about 5 from 9×10^4) of individuals were in the symptomatic state ($\boldsymbol{D}$). When local control is applied, the number of infected ($\boldsymbol{I}$), recovered ($\boldsymbol{R}$) and detected ($\boldsymbol{D}$) individuals becomes very small, as most of the detected nodes are quickly treated, Fig. 4, middle and bottom row. If the size of the control neighbourhood is small ($z = 1$, middle panel), the number of treated individuals ($\boldsymbol{V}$) is comparable with the number of infected individuals in the absence of any control. However, in this case the outbreak is finished much earlier than when no control is applied (compare top and middle panels). Even a small increase of z (from $z = 1$ to $z = 4$) arrests the epidemics sooner, requiring treatment of a smaller number of individuals (middle *vs* bottom panel). Addition of non-local interactions changes the character of the epidemics outbreak, and not surprisingly increases the prevalence and makes the local control mechanisms less efficient (compare the right column with the left column in Fig. 4).

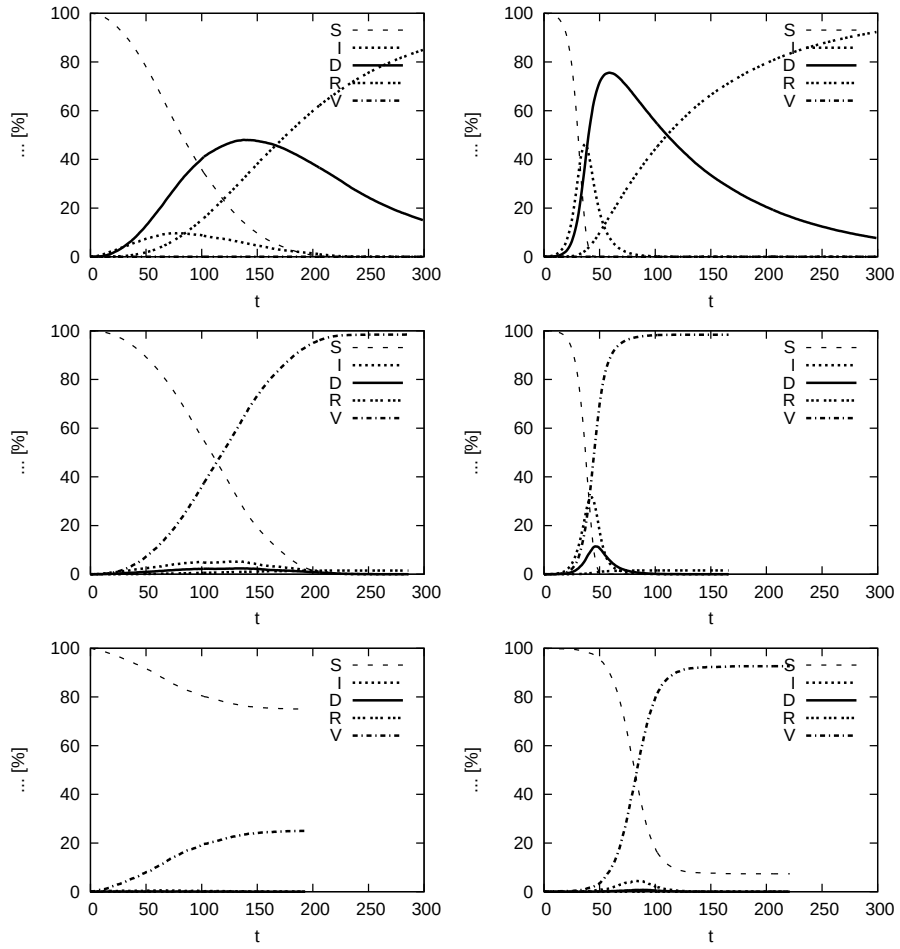

Fig. 4. The epidemics outbreak for SW2D 300×300 topology without additional shortcuts (left panel) and with 1000 shortcuts (right panel). In the top panel no control action is taken ($z = 0$). In the middle panel $z = 1$ and $z = 4$ in the bottom panel. Values of the other parameters $p = 0.5$, $q = 0.09$, $r = 0.01$, $v = 0.1$. Initially, 0.005% of population was in the symptomatic state.

3.2. Preventive random blind vaccination

In this strategy, a given proportion of randomly chosen individuals is vaccinated and afterwards no control mechanisms are applied. In Fig. 5 the severity index $X = R(\infty) + V(0)$ is plotted for various topologies and for various infection probabilities ($V(\infty) = V(0)$ in this case). With the increasing numbers of additional random shortcuts, the costs of the control

mechanisms increase and a larger number of individuals need to be vaccinated preventively. The same effect is caused by the increasing infection probability, p. There is an optimal vaccination proportion, which increases with an infection probability and with an addition of shortcuts.

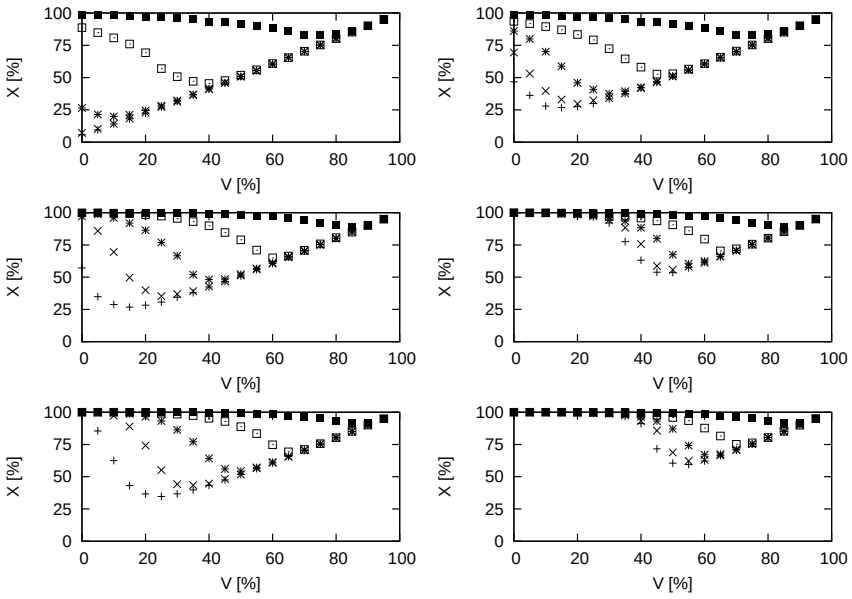

Fig. 5. $X = R(\infty) + V(\infty)$ as a function of proportion of initially vaccinated individuals for SW1D topology (left panel) and SW2D topology (right panel) for various values of infection probability, p: $p = 0.01$ (top panel), $p = 0.05$ (middle panel) and $p = 0.5$ (lower panel). Other parameters: $q = 0.5$, $r = 0.01$. Different symbols correspond to various numbers of additional shortcuts: $+$ — 0 shortcuts, $\times$ — 63 shortcuts, $*$ — 255 shortcuts, $\square$ — 1023 shortcuts and $\blacksquare$ for the SF topology. Initially, at $t = 0$, 0.5% of all individuals were in the symptomatic class. Because the treatment does not change in time, $V(\infty) \equiv V(0)$.

It is also easier to prevent the epidemic in the 1-dimensional case, even though the number of neighbours in the first order epidemic neighbourhood is the same (four nearest neighbours). These results are in contrast to the 'usual' approach in designing optimal vaccination strategies, where the final size of an epidemic $R(\infty)$ is only being minimised (see Fig. 6). If $X = R(\infty)$ only, there is no discernible minimum in the severity index (reflecting total costs of the epidemic and its attempted control), so — formally speaking — the optimal strategy in this case is to vaccinate the whole population. However, in most cases, $R(\infty)$ is initially high, but drops rapidly to almost zero at a certain critical proportion of vaccinated individuals. This

drop corresponds to a classical critical vaccination proportion, well known in epidemiology [1, 14]. Thus, when this critical level of preventive treatment is reached, increasing vaccination levels only marginally increases the effectiveness of the control strategy (Fig. 6).

The difference is particularly clear for the SF topology. When the vaccination costs are included ($X = R(\infty) + V(0)$), there is a clear optimal vaccination strategy leading to a reduction in the severity index (Fig. 5). However, no such threshold appears for the case when vaccination is assumed to have no costs, *i.e.* $X = R(\infty)$, (Fig. 6).

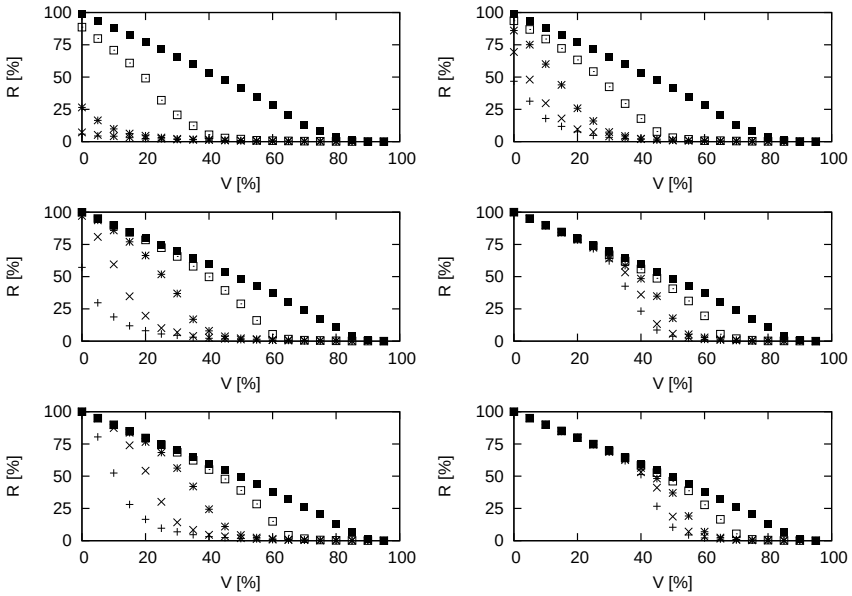

Fig. 6. $X = R(\infty)$ as a function of proportion of initially vaccinated individuals for SW1D topology (left panel) and SW2D topology (right panel) for various values of infection probability, p: $p = 0.01$ (top panel), $p = 0.05$ (middle panel) and $p = 0.5$ (lower panel). Other parameters as in Fig. 5. Different symbols correspond to various numbers of additional shortcuts: $+$ — 0 shortcuts, $\times$ — 63 shortcuts, $*$ — 255 shortcuts, $\square$ — 1023 shortcuts and $\blacksquare$ for the SF topology. Initially, at $t = 0$, 0.5% of all individuals were in the symptomatic class.

3.3. Vaccination of all individuals in a given neighbourhood

This strategy assumes that each symptomatic individual ($\boldsymbol{D}$) triggers a treatment 'event' with probability v. However, because it is only symptomatic individuals that trigger such 'events', the disease can spread undetected for a substantial time. How far it can proliferate, depends on the

probability of detection q and the probability of treating once detected v. To compensate for this cryptic spread, we extend the control neighbourhood beyond the epidemic one, and assume that all individuals within the given radius need to be treated. Fig. 7 shows that for all networks there is an optimal control neighbourhood that minimises $X = R(\infty) + V(\infty)$.

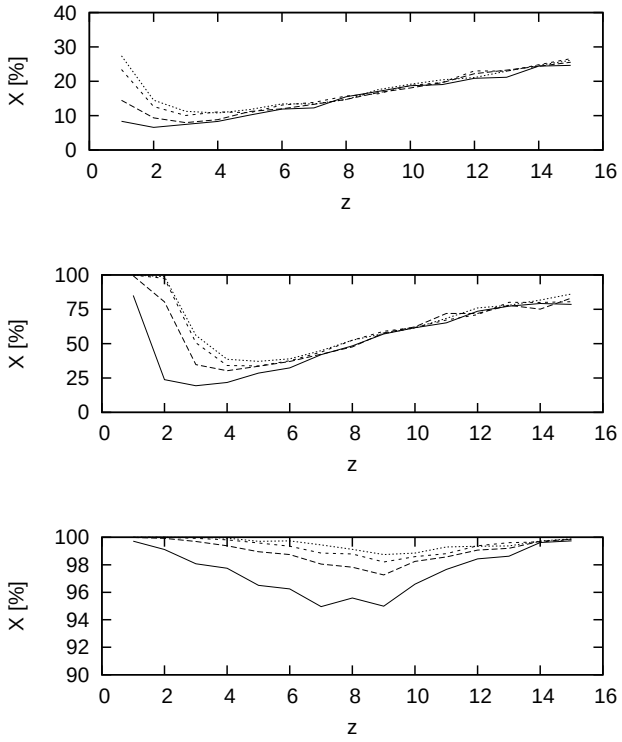

Fig. 7. Severity index ($X = R(\infty) + V(\infty)$) as a function of the treatment neighbourhood, z, for different values of the probability of infection, p, for (from top to bottom) the SW1D, SW2D and SF topologies (averaged over 50 replicates). There were no shortcuts for the SW1D and SW2D networks. $p = 0.25$, 0.5, 0.75 and 1 for curves from the bottom to the top. Other parameters are: $q = 0.5, r = 0.01, v = 0.1, C = 5, C_{\mathrm{I}} = 1$.

We also examined the influence of additional random shortcuts on the severity index X for SW1D and SW2D topologies. Simulation were performed for various numbers of additional shortcuts $\{0, 3, 15, 63, 255, 1023\}$. The addition of random shortcuts increases the duration of the epidemic, epidemic severity (as measured by $X = R(\infty) + V(\infty)$) and also the optimal range of vaccination z_c (Fig. 8). Increasing the number of shortcuts for SW1D or SW2D topologies produces results that approach those obtained

for the SF topology (Fig. 8). This convergence is faster in the case when the cryptic period is longer (smaller q) or when the control mechanisms are applied later (higher ratio of initially infected individuals).

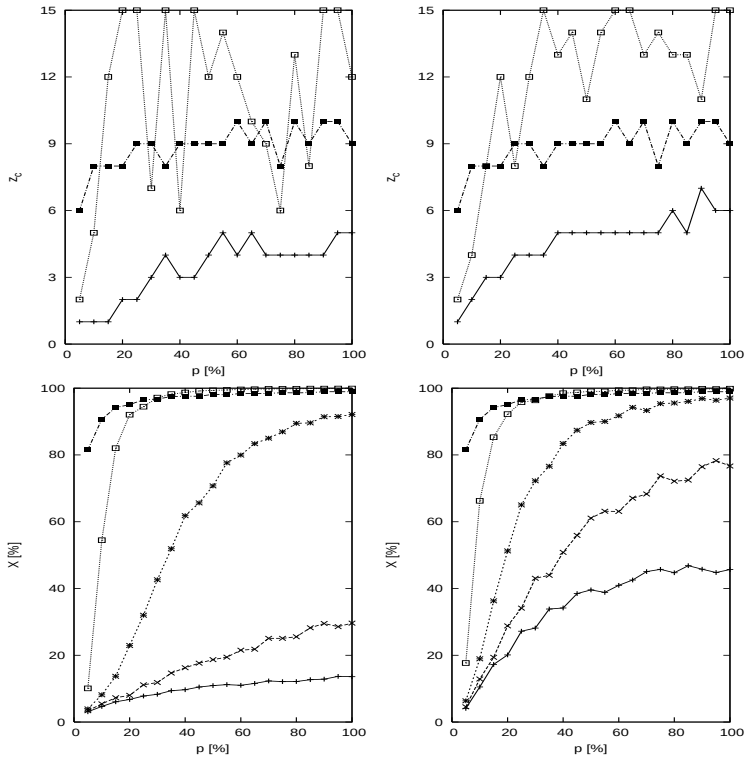

Fig. 8. Optimal range of vaccination z_c and severity index $X = R(\infty) + V(\infty)$ for SW1D topology (left panel) and SW2D topology (right panel) for $q = 0.2, v = 0.1$ and ratio of initially detected individuals 0.5%. Different symbols represent various numbers of additional shortcuts: $+$ — 0, $\times$ — 63, $*$ — 255, and $\square$ — 1023 additional shortcuts. The control strategy does not utilise any information about non-local links. Results for SF topology are marked with ■. Values of the severity index X correspond to the optimal range of vaccination z_c.

Higher ratio of initially detected individuals increase severity of the epidemic as measured by X, but at the same time decrease the optimal range of vaccination (compare Fig. 8 with Fig. 9). In general, epidemics are more severe on the SF topology than on SW2D and SW1D topologies and control measures have to be applied in larger neighbourhoods (larger z_c), even in the case of SW1D and SW2D with shortcuts. However, for networks with as many as 1023 shortcuts in a system size of 2500 (corresponding to about

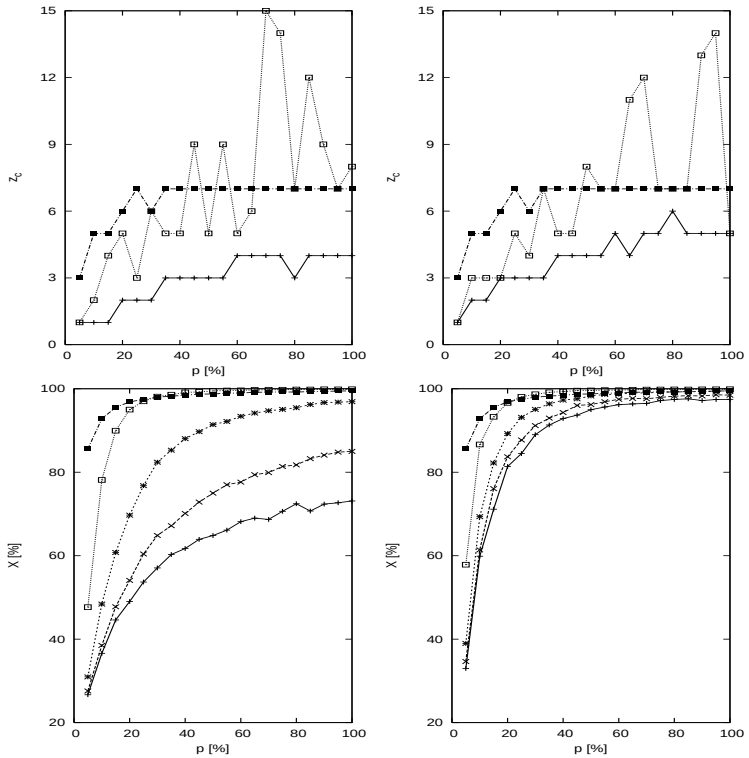

Fig. 9. The same as in Fig. 8. All parameters as in Fig. 8, except the ratio of initially detected individuals is now 5%.

10% increase in the number of links), the optimal range is smaller than for SF topology. The link between the severity of epidemics, the optimal range of control and the neighbourhood size for different network topologies is explored below.

3.4. Neighbourhood size

The addition of long-range interactions generally renders the local control less effective. The natural way to compensate for this is to increase the size of the control neighbourhood. This leads, however, to a fast increase in the number of individuals treated in a single 'event'. In our model, the neighbourhood order z is a measure of how far the disease can spread (for the epidemic neighbourhood), or how far the control measures can extend in a single 'event' (for the control neighbourhood). Two factors are of importance here, the number of the individuals that are added to the neighbourhood

if the order increases by 1 (the size of a 'shell') and the total size of the neighbourhood of a given order. Tables I, II and III list both characteristics in absolute numbers or as the ratio of the total population size. Results for the SW1D and SW2D topologies were calculated analytically, while for the SF topology we used a Monte Carlo method [15].

TABLE I

Number of individuals and percentage of the whole population in neighbourhoods of order z and in the neighbourhood up to order z for the SW1D topology with 2500 nodes.

z	in z		up to z	
	# indv.	% of pop.	# indv.	% of pop.
1	4	0.2	4	0.2
2	4	0.2	8	0.3
3	4	0.2	12	0.5
4	4	0.2	16	0.7
5	4	0.2	20	0.8
6	4	0.2	24	1.0
7	4	0.2	28	1.2
8	4	0.2	32	1.3
≥ 9	2468	98.7	2500	100

TABLE II

Number of individuals and percentage of the whole population in neighbourhoods of order z and in the neighbourhood up to order z for the SW2D topology with 2500 nodes.

z	in z		up to z	
	# indv.	% of pop.	# indv.	% of pop.
1	4	0.2	4	0.2
2	8	0.3	12	0.5
3	12	0.5	24	1.0
4	16	0.6	40	1.6
5	20	0.8	60	2.4
6	24	1	84	3.4
7	28	1.1	112	4.5
8	32	1.3	144	5.8
≥ 9	2356	94.2	2500	100

TABLE III

Average number of individuals and percentage of the whole population in neighbourhoods of order z and in the neighbourhood up to order z for the SF topology with 2500 nodes, $C = 5$ and $C_{\mathrm{I}} = 1$.

z	in z		up to z	
	# indv.	% of pop.	# indv.	% of pop.
1	1.9	0.1	1.9	0.1
2	8.8	0.4	10.7	0.4
3	31.0	1.2	41.8	1.7
4	77.2	3.1	119.0	4.8
5	148.6	5.9	267.6	10.7
6	235.3	9.4	502.9	20.2
7	322.2	12.9	825.1	33.0
8	375.0	15.0	1200.0	48.0
≥ 9	1300.0	52.0	2500	100

For the SW1D topology (Table I), the number of individuals in the 'shell' is constant and equal to four. It is caused by the fact that SW1D is a 1D ring. For the other topologies, the number of individuals in the 'shell' is an increasing function of the neighbourhood order z. Due to the clustered character of the SF topology (Table III), the size of the neighbourhood grows very fast and quickly extends to the whole population.

4. Discussion

In this paper we have analysed a spatially-extended, modified SIR model for disease spread with varying network complexity and with different spatial control strategies. The goal is to find an optimal strategy, leading to a minimal combined cost of treatment and disease [4]. Search for such a strategy is complicated because of uncertainty caused by our limited knowledge about the status of individuals (whether infectious or not) and their interactions (not all contacts are known). We considered two contrasting cases. In the first one, the population is treated to a random vaccination applied before any potential outbreak. In the second case, treatment is applied locally around each individual that has been detected at the onset of symptoms. We have shown that an optimal strategy exists in both cases, and studied its dependence on a range of factors. We found that the proportion of treated individuals (for the global strategy) and the radius of treatment neighbourhood (for the local strategy) increase with the probability of disease transmission and the number of shortcuts.

However, increased number of initial foci increases the number of treated individuals, but decreases the optimal range of treatment. Decrease in the optimal range of vaccination reflects two possible ways in which an optimal strategy can be constructed. For a small number of initial foci, it is more efficient to apply control within a larger neighbourhood, and less frequently. For a large number of initial foci, when the disease has already spread very far, control can be applied within a smaller neighbourhood, but has to be applied in many initial places.

Interesting difference in designing control strategies have been noticed for the random vaccination applied in order to prevent any potential outbreak. The traditional approach is to try to minimise the final size of the epidemic [1, 14]. However, this leads to a strategy in which a large proportion of individuals is treated. If we define an optimal strategy as the one that *minimises* the total size of an outbreak, the whole population must be treated regardless of the disease parameters. In this approach, most of the resources are spent on an attempt to prevent disease spreading to very few individuals. However, when the cost of the treatment is included, it is preferable to lower the vaccination level, even though this will allow some limited disease spread, in order to minimise the total costs of containing the outbreak.

There are several ways in which the search for improved control strategies can be extended. One possibility is to vaccinate up to a given number of randomly chosen individuals in a given control neighbourhood, or to treat individuals at the perimeter of the control neighbourhood only (ring vaccination). This kind of strategy can be applied when the number of individuals in a single control neighbourhood is large and it is impossible to treat all of them, due to lack of time or resources. Nevertheless, such strategies, due to the fact that not all information about contacts between individuals is recognised, are less efficient that a treatment of all individuals in the local control neighbourhood.

We have found that small-world shortcuts have a major impact on the size and duration of epidemics and on the effectiveness of control strategies, both local and global. It is possible to extend control strategies to include contact tracking [16] and to include some of long-range links in the control network. For the scale-free networks, control targeted at highly connected individuals has been shown to be a promising strategy [17].

Our approach is based on a linear relationship between a number of infected or treated individuals and associated costs. This might not be always true, and nonlinear relationships might change the particular choice of an optimal strategy.

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