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On social percolation and small world network

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Abstract. The social percolation model is generalized to include the propagation of two mutually exclusive competing effects on a one-dimensional ring and a two-dimensional square lattice. It is shown that the result depends significantly on which effect propagates first *i.e.* it is a non-commutative phenomenon. Then the propagation of one effect is studied on a small network. It generalizes the work of Moore and Newman of a disease spread to the case where the susceptibility of the population is random. Three variants of the Domany-Kinzel model are given. One of them (delayed) does not have a chaotic region for some value of the delay weight.

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1 Social percolation

Social percolation [1,2] is a model to relate a social trend to percolation theory [3]. Consider the propagation of a social effect such as say going to a movie. The society members are assumed to occupy the sites of a lattice. Everyone can affect the opinion of his (her) nearest neighbors (n.n.). Every site has its own threshold $p(i)$ and also the effect (movie) is given a value say q (a real number). Initially a small number of sites are informed about the movie. Then the propagation rule is: If an individual at site i has not gone to the movie and if one of his (hers) n.n. has seen it then if $q > p(i)$ then agent i will go to the movie. On a regular lattice this is a classical percolation problem *i.e.* the effect will spread if $q \geq p_c$, where p_c is the critical concentration of the lattice.

2 Two mutually exclusive effects on a regular lattice

The situation becomes more interesting when two mutually exclusive effects (*e.g.* two movies but everyone has to go only to one of them) compete to spread on the lattice. As a corollary an individual will have to stick to its choice. We assumed $q_2 = 2q_1$ hence one might intuitively expect that the second effect will occupy twice as much as the first one provided that the initial number of agents of each effect is the same. However the result depends significantly on the order of spread *i.e.* which effect spreads first. Two rules of propagation can be followed in this problem.

The first is, if an individual at site i is not yet affected and if one of his (her) n.n is of type one (*e.g.* has seen the movie) and if $q_1 \geq p(i)$ then site i becomes of type one. Else if one of its n.n. is of type two and if $q_2 \geq p(i)$ then site i is of type two.

The second propagation rule is, if site i is not affected and if one of its n.n. is of type two and $q_2 \geq p(i)$ then site i becomes of type two. Else if one of its n.n. is of type one and $q_1 \geq p(i)$ then it becomes of type one. Consequently this propagation has the order reversed compared to the first rule.

In all our one-dimensional simulations we have 500 agents. Let $q_1 = 0.4$, $q_2 = 0.8$, and initially 10% of the sites were informed about each effect *i.e.* at the beginning a total of 20% of the lattice was already affected. When the first rule is applied we obtained the average result $\rho_1 = 0.271$, $\rho_2 = 0.188$ but in the case of the second rule we obtained $\rho_1 = 0.156$, $\rho_2 = 0.419$. where $\rho_1(\rho_2)$ is the final fraction of sites of type 1(2).

In two dimensions we have an 80×80 square lattice and the results are $\rho_1 = 0.411$, $\rho_2 = 0.34$ when the first rule is followed while the second rule gave $\rho_1 = 0.186$, $\rho_2 = 0.640$.

This confirms that this propagation process is non-commutative and shows the importance of reaching first to the potential agents. This result depends also on the assumption that the effects are mutually exclusive.

3 Small world network and social percolation

Small world networks [4–6] is a model proposed for social networks. It is a one-dimensional ring plus shortcuts joining some random sites. Here we consider shortcuts with length $k = 1$ as seen in Figure 1. Let ϕ be the average number of shortcuts per bond on the lattice, hence for a large number (L) of sites in the lattice, the probability that two random sites are connected by a shortcut

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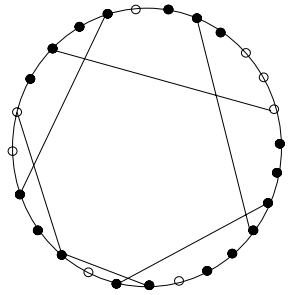

Fig. 1. An example of a small-world graph with $L = 26$ sites (black sites are occupied and white sites are empty) and 6 shortcuts with length $k = 1$.

is $\Psi \simeq 2k\phi/L$. Naturally the critical concentration of this graph p_c is smaller than the one for the ring $p_c = 1$. To derive the new p_c the work of Moore and Newman will be used. Build the lattice starting form a connected local cluster then follow shortcuts. Let v_i be the probability that a local cluster of length i is included, hence in the next step in the cluster building one has

$$v_i' = \sum_j M_{ij} v_j, \qquad M_{ij} \simeq ij\Psi N_i, \tag{1}$$

for full derivation see [7]. If the maximum eigenvalue of M_{ij} is less than one then the propagation process will eventually stop otherwise it will propagate throughout the cluster. Thus the critical concentration corresponds to the eigenvalue one *i.e.*

$$\sum_j M_{ij} v_j = \lambda v_i \Longrightarrow \lambda = \Psi \sum_j j^2 N_j \implies \lambda = 2\phi p \frac{1+p}{1-p}\,. \tag{2}$$

So the critical concentration is

$$p_c = \frac{\sqrt{4\phi^2 + 12\phi + 1} - 2\phi - 1}{4\phi}\,. \tag{3}$$

For small ϕ, we can write,

$$p_c \simeq 1 - 4\phi. \tag{4}$$

Now consider the propagation across a small world network. We consider the spread of an epidemic in a population with random susceptibility thus our work generalizes that of Moore and Newman. In our simulations $\phi = 0.05$ and shortcuts are assigned randomly from the beginning.

The propagation rule is: if site i is not affected and if one of its n.n. or its shortcut neighbor (if exists) is affected and if $q \geq p(i)$ then site i will be affected. This model corresponds to the spread of an epidemic with force of infection q in a population with random susceptibilities $p(i)$. For $q = 0.4$ we found that the disease infected 19% of the population if they live on a ring. Adding shortcuts increased the infection to 23%. Similarly for $q = 0.9$ the infected percentage increased form 69% in the case of a ring to 75% to the case of a small world network. Recall that without shortcuts the disease cannot spread throughout the lattice except only at $q = 1$.

It is relevant to find an estimation of the number of infected persons in the simple susceptible-infected (SI) model studied above as a function of time [8]. Assume that the speed of disease spread is unity and that the persons occupy the vertices of a SWN, let ϕ be the fraction of shortcuts hence 2ϕ is the density of shortcut-ends in the graph. Setting $p(i) = 0 \quad \forall i$ hence the number of infected persons will grow initially as a sphere with surface $\Gamma_d t^{d-1}$ where $\Gamma_1 = 2$, $\Gamma_2 = 2\pi$, $\Gamma_3 = 4\pi$ and so on. This is called the primary sphere. Once a shortcut is reached (the probability of such an event is $2\phi\Gamma_d t^{d-1}$ per unit time) a secondary sphere forms and so on. Hence the total number of infected persons is given by

$$V(t) = \Gamma_d \int_0^t \tau^{d-1}\{1 + 2\phi V(t-\tau)\}\mathrm{d}\tau. \tag{5}$$

Defining $V' = 2\phi V$, $t' = t[2\phi\Gamma_d(d-1)!]^{1/d}$ and differentiating d times with respect to t' one gets $\partial^d V'/\partial t'^d = 1 + V'$ whose solution is

$$V'(\,t') \;=\; \sum_{i=1}^{\infty} \frac{t'^{di}}{(di)!}\,. \tag{6}$$

In one dimension one has $V'(\,t') = \exp(t') - 1$, in two dimensions $V'(t') = \cosh(t') - 1$. For $t' < 1$ the number of infected persons grow as a power law $t'^d/d!$ while for $t' > 1$ it grows exponentially. The transition occurs at $t' = 1$ *i.e.* at $t = [2\phi\Gamma_d(d-1)!]$. This has an important effect on vaccination policies since it implies that vaccination should be administered as early as possible and with the highest possible ability to avoid reaching the exponential phase. Also immunizing sites with shortcuts is more efficient than immunizing ordinary sites. This idea of target immunization and target disease resistance has been proposed in more realistic situations *e.g.* schistosomiasis.

Now we study SIRS (susceptible-infected-recovered-susceptible) [9] on a small world network. The study on an ordinary lattice has been done in [10]. A state $s(i)$ is assigned to each vertex i at time t. $s(i) = 0$ for susceptible, $s(i) = -1$ for infected and infecting, and $s(i) = 1$ for recovered, where $i = 1, 2, ..., n \quad (n = 500)$. The graph is a small world network as defined by Newman and Watts and the automata rules are:

i) if $s(i) = -1$ then $s1(i) = 1$;
ii) if $s(i) = 1$ then $s1(i) = 0$ with probability $q2$;
iii) if $s(i) = 0$ and $s(i+1) = -1$ or $s(i-1) = -1$ or $s(\mathrm{sc}(i)) = -1$ then $s1(i) = -1$ with probability q where $\mathrm{sc}(i)$ is the shortcut neighbor of site i (if existent). We obtained that the boundary separating the regions where the disease persists (region II) or disappears (region I) is given by the following set of points in the $(q, q2)$ plane

$$\{(0.66, 1), (0.7, 0.87), (0.8, 0.5), (0.9, 0.27), (1, 0.18)\}. \tag{7}$$

As expected the region where the disease persists has increased in the SWN case compared to the regular graph case [10], as seen in Figure 2.

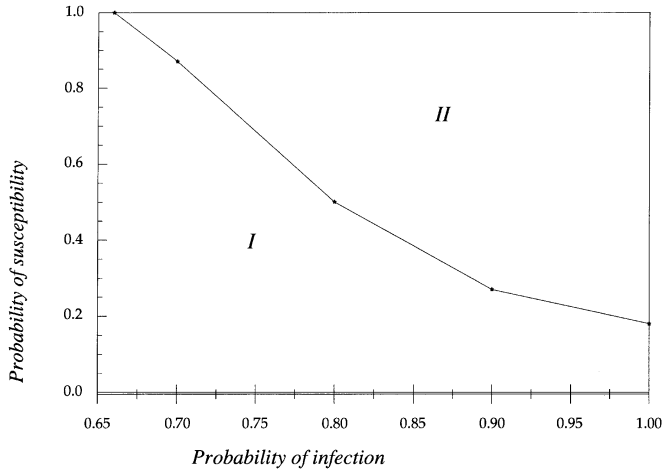

Fig. 2. The phase space of two distinct regions. In the first (region I), the disease disappears while in the second (region II) it persists.

In simulating a small world network one usually fixes the sites connected by shortcuts beforehand. However, it may be easier in simulation if one determines them randomly (with probability ϕ) during simulation. We call this network a random small world network RSWN. Presumably this will not significantly alter the simulation results. Anyway, all the simulations done here are on the standard small world network SWN.

4 Domany-Kinzel model on SWN

The Domany-Kinzel (DK) model [11,12] is an interesting realization of directed percolation. Here it is generalized using ideas form social percolation and SWN.

The first version has every site in a one-dimensional lattice endowed with a random number $p(i)$. Then the evolution rules are:

i) if $s(i) = 0$ and $s(i+1) = 0$ then $s1(i) = 0$;
ii) if $s(i) + s(i+1) = 1$ then if $p_1 \geq p(i)$ then $s1(i) = 1$ else $s1(i) = 0$;
iii) if $s(i) + s(i+1) = 2$ then if $p_2 \geq p(i)$ then $s1(i) = 1$;

where $s1(i)$ is $s(i)$ in the next run. We studied only the case $p_2 = 0$ and found that for $0 \leq p_1 \leq 0.6$ the results were 2-cycle, for $0.6 \leq p_1 \leq 0.8$ the number of active sites ($s(i) = 1$) was a 4-cycle and for $p_1 \geq 0.8$ the number changes chaotically with time.

The second version is to model the Domany-Kinzel model on a SWN. In this case we set $\phi = 0.05$ and $p_2 = 0$. Only a slight change in the critical value of $p_1 = 0.79$ instead of the standard $p_1 = 0.8$ for DK model on the ring. The full phase diagram of the modified DK model will be reported elsewhere.

A third version which is applicable on any graph is the delayed DK model. It is given by the rules:

i) if sum $= 0$ then $s1(i) = 0$;
ii) if sum $= 1$ then $s1(i) = 1$ with probability p_1 else $s1(i) = 0$;
iii) if sum $= 2$ then $s1(i) = 1$ with probability p_2 else $s1(i) = 0$;

where sum is defined by

$$\text{sum} = \text{Int}\{\omega[s(i,t) + s(i+1,t)] + (1-\omega)[s(i,t-1) + s(i+1,t-1)] + 0.5\}, \quad (8)$$

where $1 \geq \omega > 0$ is the delay weight and $\text{Int}[x]$ is the integer part of x. Our simulations have shown that, on a regular graph the introduction of delay reduces the chaotic region and that for the value $\omega = 0.5$ the chaotic region disappears.

5 Conclusions

In conclusion, in the first part of this work, the social percolation problem has been studied. In the second part, the social percolation model is generalized to include the propagation of two mutually exclusive competing effects on a one-dimensional ring and a two-dimensional square lattice. It is noticed that the propagation process is a non-commutative phenomenon.

In the third part, the propagation of one effect is studied on a small world network and the work of Moore and Newman of a disease spread is generalized to the case where the susceptibility of the population is random. We found that the region where the disease persists has increased in the small world network case compared to the regular graph case.

Finally, three variants of the Domany-Kinzel model are given and generalized by using the ideas of social percolation and small world network. Our simulations have shown that, on a regular graph the introduction of delay reduces the chaotic region and that for some value of the delay weight, the chaotic region disappears.

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