



# SHIFT OF PERCOLATION THRESHOLDS FOR EPIDEMIC SPREAD BETWEEN STATIC AND DYNAMIC SMALL-WORLD NETWORKS

J. Ochab<sup>†</sup> and P.F. Góra<sup>‡</sup>

Institute of Physics<sup>†‡</sup> and Mark Kac Complex Systems Research Centre<sup>‡</sup>,  
Jagiellonian University, Cracow, Poland



## INTRODUCTION

While dynamic network models have been accepted and applied in recent research (e.g. epidemic spread on dynamic small-worlds [1]), it seems that we lack **comparative study on how the dynamics of the network influences the process taking place on it**. Hence, our aim is to compare epidemic spread for static and dynamic small-world networks numerically and check it against analytical studies.

## NETWORK

We adopt **Watts-Strogatz model of a small-world network** [2]: first we take a 2-dimensional square lattice with  $L^2 = N$  nodes and  $2N$  undirected edges. To avoid some finite-size effects we impose periodic boundary conditions for the lattice. Then, we add a number of undirected edges between random nodes. The number of additional edges ('shortcuts') is set as  $2\phi N$ , hence  $\phi$  is shortcut probability per underlying bond. Network with  $\phi = 0$  is just a **regular lattice**. For nonzero  $\phi$  we call the network a **static small-world**.

In the **dynamic small-world network** we choose  $2\phi N$  nodes randomly, and keep them fixed for the whole run of epidemics. In every time step we randomly launch shortcuts anchored in these nodes, so the dynamics consists in rewiring the shortcuts. This construction of the source nodes launching shortcuts allows for easier interpretation of the network: the fixed nodes could correspond to centers of activity that can be identified as in the real world networks.

## EPIDEMICS

The SIR (**Susceptible-Infected-Removed**) model is adopted, where  $\mathbf{p}$  is the **probability of infecting** a susceptible node by an infectious neighbour during one time step. The **latency time  $l$**  of the disease is measured in discrete time units (we take  $l = 3 - 4$ ), i.e. the infectious node can infect others with probability  $p$  for  $l$  turns, and after that time it is removed.

Grassberger [3] related the probability of infection to the probability  $\mathbf{T}$  in **bond percolation** through  $T = \sum_{t=1}^l p(1-p)^{t-1} = 1 - (1-p)^l$ , where  $T$  is the so called "transmissibility" (it is the total probability of a node infecting one of its neighbours during the whole latency time). In the case of 2-dimensional square lattice the bond percolation threshold is  $T_c = 0.5$ .

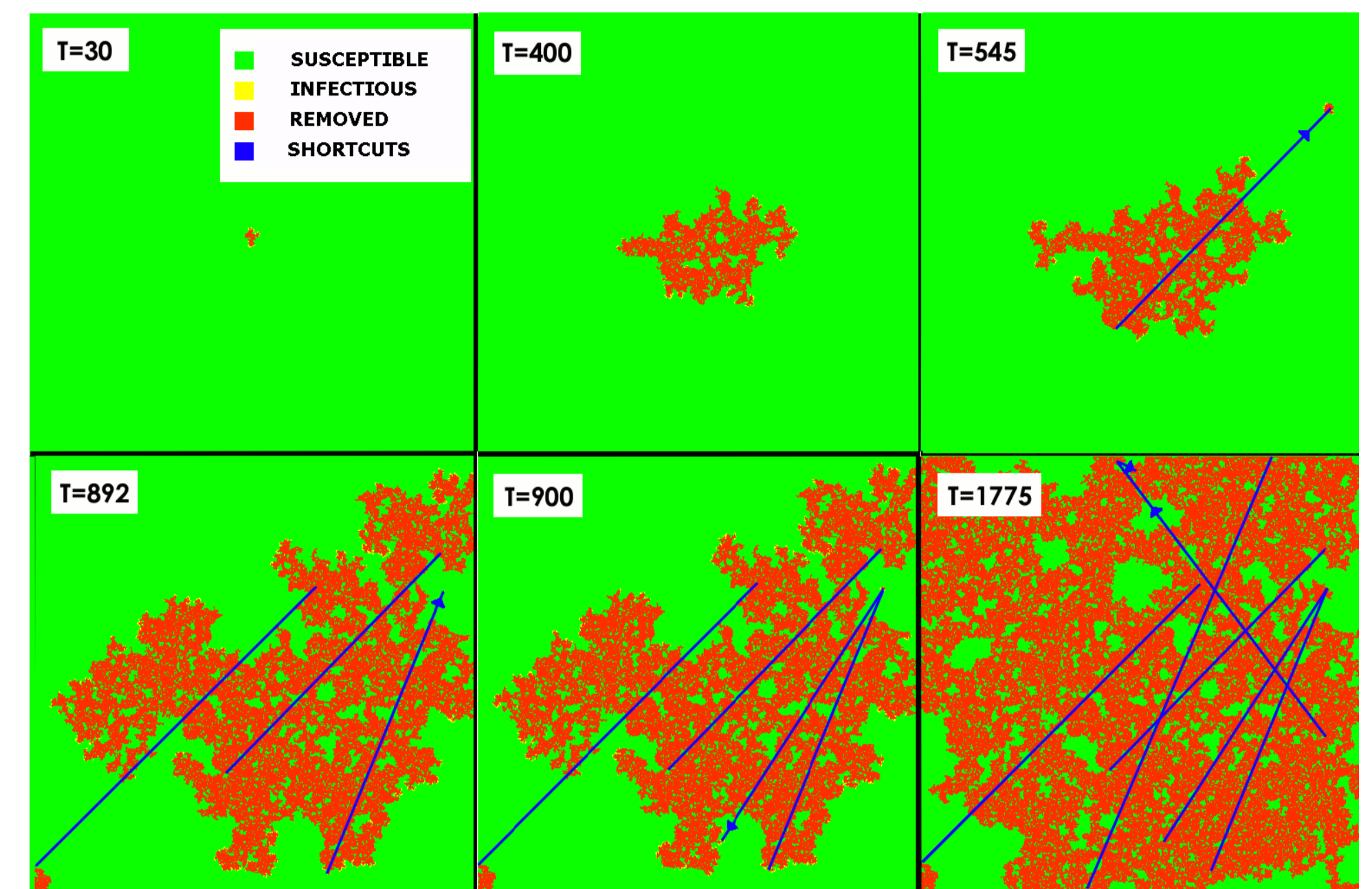


FIGURE 1: Snapshots of epidemic spread slightly above percolation threshold.  $L = 512$ , the number of shortcuts is 10 (which gives  $\phi = 2 \cdot 10^{-5}$ ).  $T$  gives epidemics' time steps. The snapshots in the lower row show a dynamic infection (the two joined blue lines appear).

## THEORETICAL CORRECTIONS

We can account for the change between static and dynamic networks analytically using the model known for static small-world network [4]. One can estimate the number of nodes infected through shortcuts by

$$N_{stat} = \phi_{stat} N \cdot T = \phi_{stat} N \cdot \sum_{t=1}^l p(1-p)^{t-1}, \quad (1)$$

i.e. the number of shortcuts in the static network multiplied by the total probability of infecting a neighbouring node. The analogous expression for the dynamic network is found easily (the first term corresponds to Fig.3b, the second to Fig.3c)

$$N_{dyn} = \phi_{dyn} N / 2 \cdot lp + \phi_{dyn} N / 2 \cdot (l+1)p. \quad (2)$$

We assume that  $N_{dyn} = N_{stat}$  if epidemics on both networks should have the same percolation threshold. Thus, we can obtain the ratio of the two shortcuts' densities

$$r(p, l) = \phi_{stat} / \phi_{dyn} = \frac{p(l+1/2)}{T} = \frac{p(l+1/2)}{1 - (1-p)^l}, \quad (3)$$

which tells us, **how many more static shortcuts are needed to have the same effect as the fewer dynamic ones**. Now, we can calculate  $T_c(r\phi)$  numerically (the lower solid line in Fig.2), just as the fitted  $T_c((1+x)\phi)$  in the Fig.2.

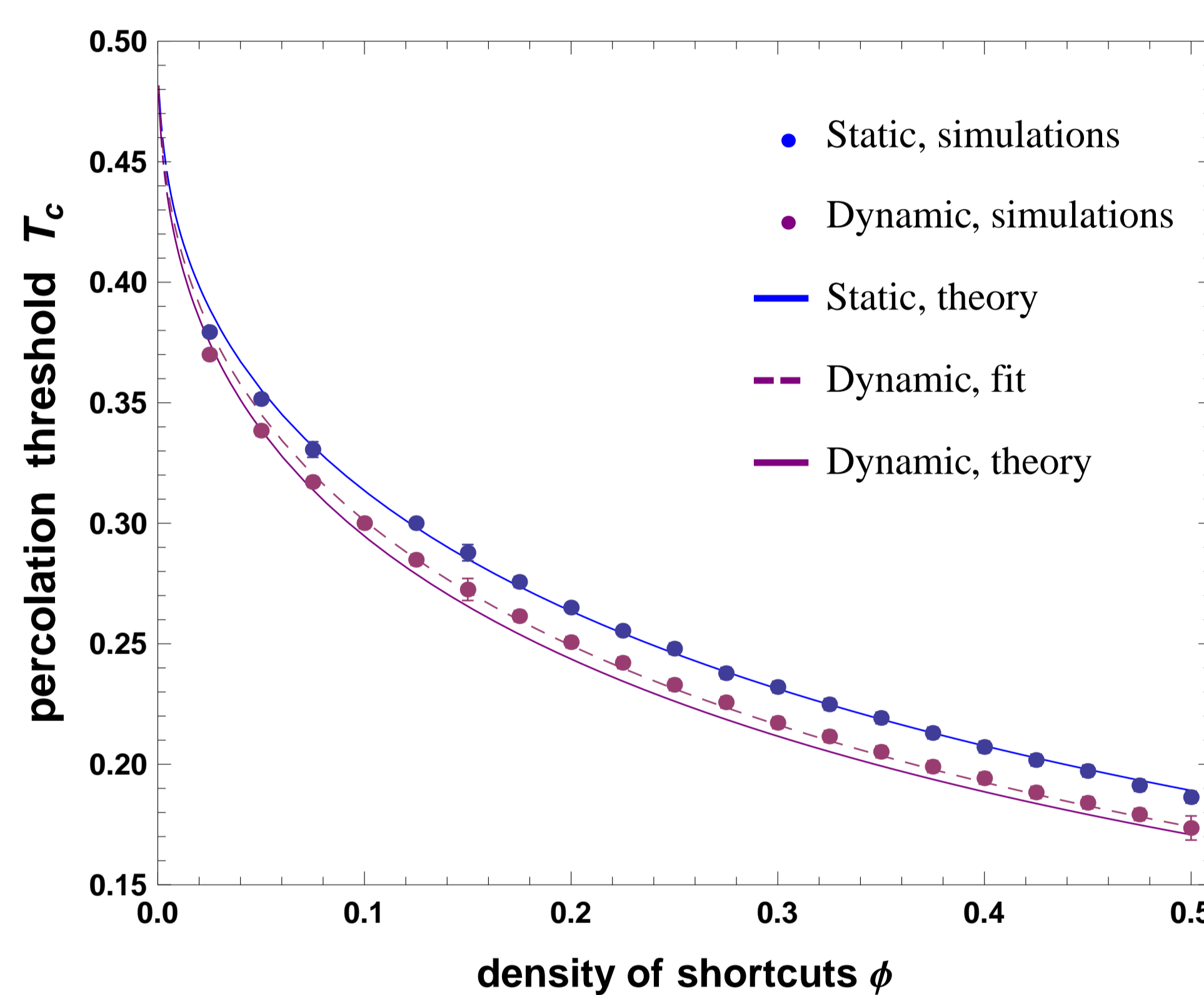


FIGURE 2: Blue (upper) dataset - static small-world. Purple (lower) dataset - dynamic network. The solid blue line (upper) is the analytic approximation [4] for  $T_c(\phi)$  and the dashed line gives  $T_c((1+x)\phi)$ , with the fit parameter  $x = 0.200 \pm 0.013$ . The solid purple line (lower) represents theoretical approximation from Eq.3.

## CALCULATING PERCOLATION THRESHOLD

In the study of epidemic spread on networks, **we stick to the percolation theory as a reference point**. Here, we calculate thresholds from average epidemics' size (average over a number of reruns for different shortcut drawings). We define percolation threshold  $T_c$  as the point at which the average epidemics' size divided by  $N$  rises above a certain value (here, set to 0.15). As we can perform simulations only for finite sizes, we take the results for a relatively large network of  $\sqrt{N} = 500$ .

## NUMERICAL RESULTS

In Fig.2 the resulting data points for static small-world network agree with the analytical approximation [4]. The lower dataset marks the effect of network dynamics. The difference between the two is systematic and significant. The dashed line is a fit of the analytical model for the static network. It follows from the fit that **percolation thresholds for dynamic network are lower as if the shortcut density were greater by  $x = 20.0 \pm 1.3\%$** . However, **qualitatively the epidemics behaves in the same way** on dynamic small world as on the static network for the given range of parameters ( $\phi = 0.5$  means that on average every node in the network has two additional links).

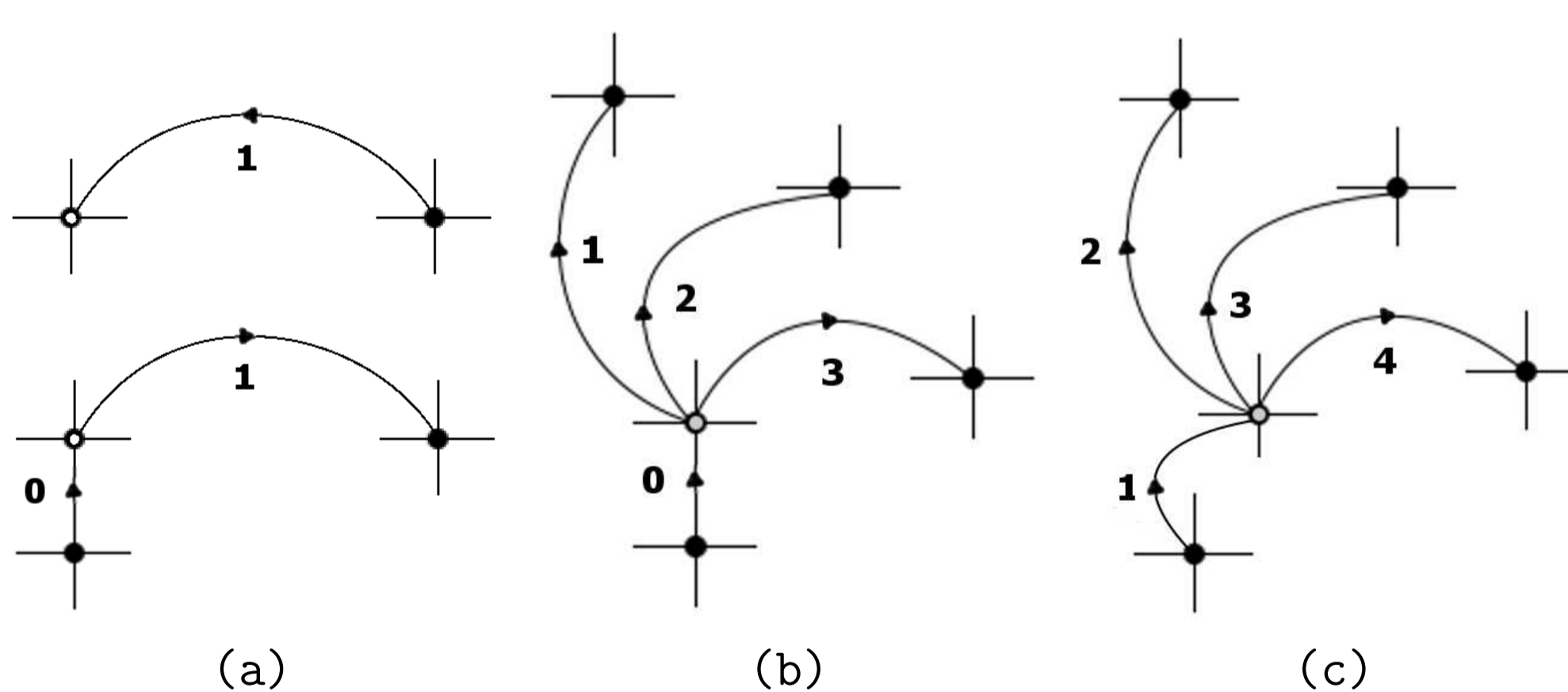


FIGURE 3: (a) Infections through static shortcuts are symmetric. (b) Infection of the dynamic links' source through regular lattice. (c) Infection of the dynamic links' source through a shortcut.

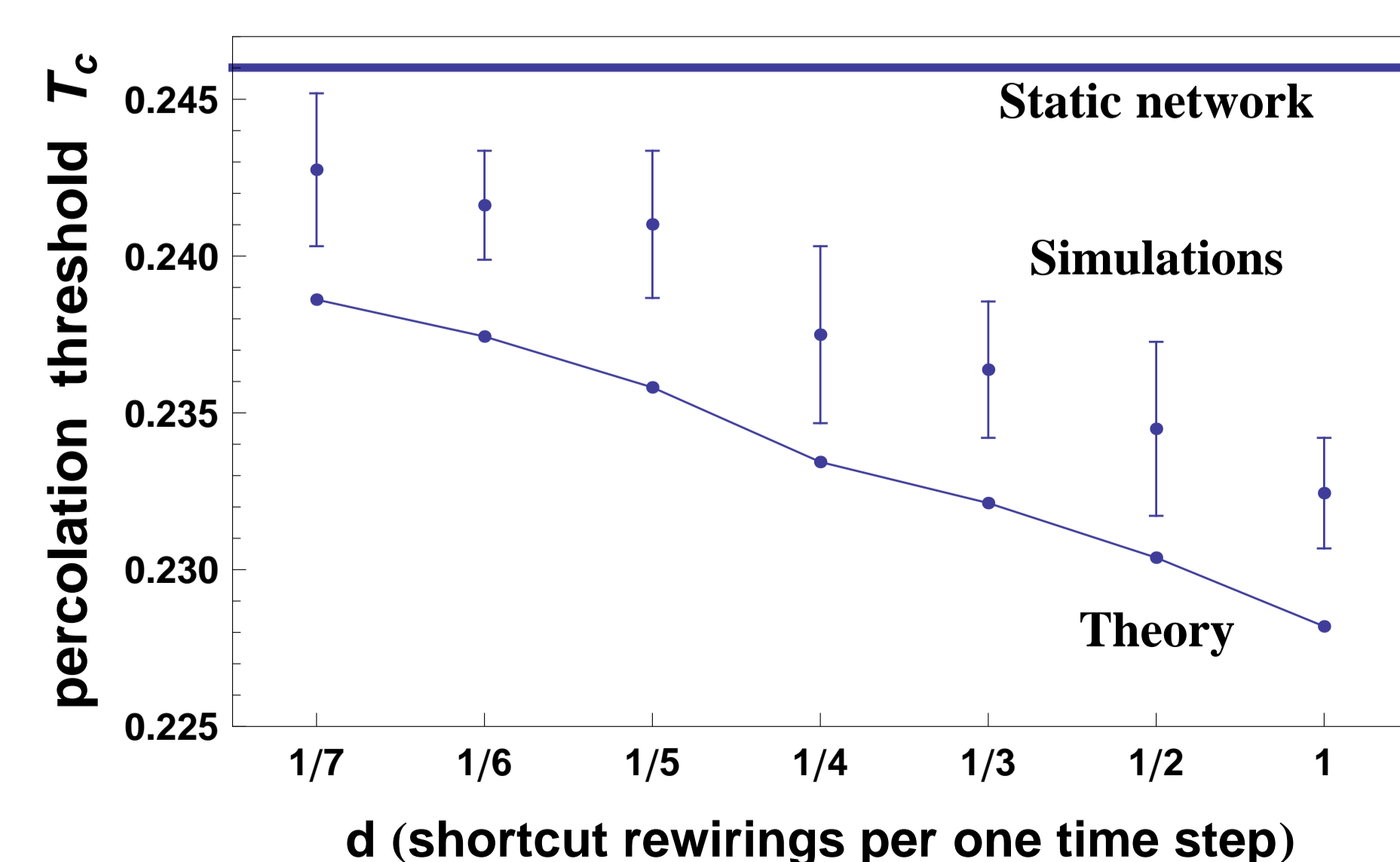


FIGURE 4: Dependence on dynamics for  $\phi = 0.25$ , latency  $l = 4$ .

## DEPENDENCE ON THE RATE OF DYNAMICS

The analysis can be generalised for various rates of dynamics. Let us notice that **there are two time scales in the model**: the latency time  $l$  of the infection and the duration  $1/d$  between consecutive rewirings of dynamic links. As the choice of latency  $l$  only rescales the total probability  $T(p, l)$  of infection, we can dispose of it, and the crucial parameter  $ld$  that accounts for the shift of percolation thresholds is defined as the number of shortcut movements during latency time. Obviously, for a static network we get  $d = 0$ , while for all the above analysis of dynamic network we have  $ld = 3$  ( $l = 3$  and the rewiring was performed every turn, so  $d = 1$ ).

Now, one can easily obtain expressions for  $N_{dyn}$  for any  $d = 1/i$ ,  $i \in \mathbb{Z}$ . Below we give only the general expression for  $1/d \geq l$  where  $l = 4$ :

$$N_{dyn} = \frac{\phi_{dyn} N}{2} \cdot \left( (2A_1(l) + A_2(l) + (1/d + 1 - l)A_0(l)) + (2A_1(l+1) + 2A_2(l+1) + (1/d - l)A_0(l)) \right) \quad (4)$$

where

$$\begin{aligned} A_0(p, l) &= T(p, l) = 1 - (1-p)^l, \quad l \geq 1 \\ A_1(p, l) &= T(p, 1)(1 - T(p, l-1)) + (1 - T(p, 1))T(p, l-1) + 2T(p, 1)T(p, l-1) \\ A_2(p, l) &= T(p, 2)(1 - T(p, l-2)) + (1 - T(p, 2))T(p, l-2) + 2T(p, 2)T(p, l-2) \end{aligned} \quad (5)$$

The first term in the brackets in Eq.4 corresponds to Fig.3b and the second to Fig.3c. The result is plotted against simulated data in Fig.4.

## CONCLUSIONS

The results prove that network dynamics lowers percolation thresholds for epidemics, however the overall dependence on number of shortcuts stays the same. The result should be taken into account in any calculations of epidemic risk or cost analysis for the given network structure.

It should be noted that the shift of percolation thresholds depends on the relative measure of dynamics of the network with respect to the process on the network (rewiring rate and latency time, respectively). Any analytical calculation or simulation must include this quantity as an important parameter, to be estimated for a particular disease and social (or other) network.

## ACKNOWLEDGMENTS

Project operated within the Foundation for Polish Science International Ph.D. Projects Programme co-financed by the European Regional Development Fund covering, under the agreement no. MPD/2009/6, the Jagiellonian University International Ph.D. Studies in Physics of Complex Systems.

## REFERENCES

- [1] J. Saramäki, Kimmo Kaski, J. Theor. Biol. **234**, 413 (2005).
- [2] D.J. Watts and S.H. Strogatz, Nature (London) **393**, 440 (1998).
- [3] P. Grassberger, Math. Biosci. **63**, 157 (1983).
- [4] M.E.J. Newman, I. Jensen, R.M. Ziff, Phys. Rev. E **65**, 021904 (2002).