

# The impact of structure of local networks on epidemic size in a multi-level system

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## 1 Introduction

Traditional compartmental models in epidemiology assume perfect, homogeneous mixing in the population. More recently, a network science perspective has been integrated into epidemiology acknowledging that structure plays a critical role in spreading behavior on single networks. For instance, shorter average path-length and skewed degree distributions increase spreading potential, while local clustering increases local spreading but decreases global cascade [6]. However, in the real-world, epidemics do not occur on single, isolated networks, but involve networks which exist at vastly different scales. For instance, within a single town or city, infections often occur through brief, day-to-day interactions. Yet people tend to travel (via air, land, or sea transportation networks), spending significant amounts of time outside their home city. Consequently, cities are connected through a larger network through which disease can spread on a much larger scale. Clearly, the type of interactions that result in infection within and between cities are fundamentally different, but the feedback among these scales is relatively poorly understood. While previous work has highlighted the importance of network structure in single scale networks[5], other work has suggested that the structure of local networks in multi-level systems has little impact on global cascades [1]. Networked metapopulation models, such as [2], typically do not take into account the structure of the subpopulations. Furthermore, metapopulation models that do investigate the effect of local structure on similar processes have found those effects to be negligible[4]. In this paper, we study the impact of the local structure in a multi-level system. We find that, in contrast with previous work on metapopulations, the structure of local networks can have a substantial impact on global pandemic size and threshold.

## 2 Model design

We created a multi-level agent-based model using NetLogo[8] LevelSpace[3] based on the traditional Susceptible-Exposed-Infected-Removed (SEIR) compartmental model.<sup>6</sup>

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<sup>6</sup>An earlier version of the model that focused on the interactions between just two cities was presented in [7].

When a susceptible agent interacts with either an exposed or infected agent, they may become exposed. Exposed individuals become infected and infected individuals become removed over time. The model consists of two levels of networks: an intercity network, representing a transportation network between cities, and many intracity networks (one for each city), in which the nodes are people and links represent possible interactions.

The intracity model is based on [6]. Each tick of the simulation, each agent will have a number (based on a rate parameter) of interactions with other agents that they are connected to in their city. To guarantee that differences in spreading behavior are due to differences in network structure rather than numbers of interactions, the number of interactions is made independent of degree.

For intercity behavior, each tick, agents have a small probability (0.2%) of traveling to a city connected to their city. To this end, they are removed from their home city's network and are attached to the destination city's network. The attachment is done at random, but in such a way that the destination city's network topology is respected. For instance, if the destination city has a scale-free network, the agent will form connections preferentially based on degree. The visiting agent will then remain in the city for an average of 10 ticks, and then will return to their original city, reconnecting with their original neighbors. Only susceptible and exposed individuals travel from their home city.

*Experimental design* The intercity network was set to be a ring network consisting of 25 cities, each with a degree of 4. The intracity networks were set to be complete, Erdős-Rényi, scale-free, small-world, or ring networks with 200 nodes and an average degree of 10 (except for complete). In each run, each city had the same network topology, but different network structures. As an initial seed, 10% of the population was infected with a disease in a single city. The level of infectiousness was varied by setting  $R_0$  from 1 to 5.<sup>7</sup> Each scenario was repeated 200 times.

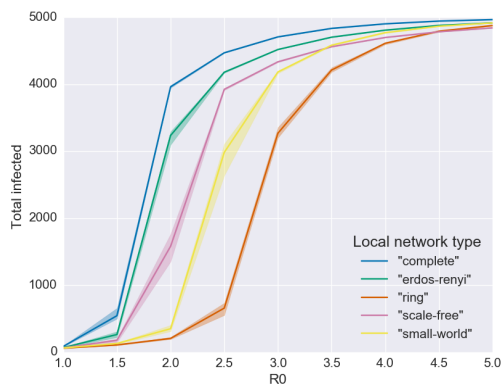
### 3 Results and discussion

The results (Figure 1) show that for  $R_0$ s from 1.5 to 4.0, the intracity network topology has a large and significant effect on the number of people infected. For  $R_0$ s great than 4.0, the impact of local network topology remains significant, but as the vast majority of the agents are affected, the effect size is relatively small in this range. We find that for the biggest part of the  $R_0$  range, a fully connected intracity topology yields the highest rates of disease spread, followed by Erdős-Rényi, scale-free, small-world, and ring respectively.

Our results demonstrate the relevance of a multi-level agent-based modeling approach. Intracity topology not only modifies the size of the global epidemic, but changes the critical  $R_0$  at which global cascade occurs. Much in line with previous work on the effects of network structure we find that the more constraining a network structure is, in

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<sup>7</sup> $R_0$  is the standard measurement of how contagious a disease is. It indicates the average number of people an infected person will infect in an otherwise uninfected, perfectly mixed population.



**Fig. 1.** The median number of individuals infected throughout the simulation for varying  $R_0$ s for each intracity network type. The colored bands around each line are the 95% confidence intervals for the medians.

terms of having more redundancy in local connections, the more constrained the global spread will be.

*Acknowledgements* Research reported in this publication was supported by the National Institute On Drug Abuse of the National Institutes of Health under Award Number P30DA027828, the National Science Foundation under Award Number NSF IIS-1441552, and the Northwestern Institute on Complex Systems.

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