The effects of local network structure on disease spread in coupled networks

W. Vermeer, B. Head and U. Wilensky

Abstract Epidemiology has long used human interaction patterns to understand spreading dynamics. Recently network scientists have embraced the notion that these pattern are best described using a complex multi-layered system, a network of networks, yielding a stream of literature focused on understanding spreading in such coupled systems. Adding this macro level perspective to disease spreading, focusing on the interaction among systems, has shifted focus away from the role of local (within-system) structure. In this paper, using a multi-level Agent-based model, we highlight the importance of the local structure in determining spreading dynamics in coupled settings. We show that the local dynamics in both the focal and neighboring networks, play a significant role in determining focal dynamics. As both are driven by the local structure this highlights a need for incorporating structural details across all levels for accurate modeling of disease spreading dynamics.

1 Introduction

Understanding the spread of disease in populations has long been a focus of the field of epidemics. The inherent difficulty of measuring disease spread has resulted in a tendency to rely on modeling to gain insight into epidemics. Traditional epidemic models assumed a compartmentalization of the population into different states

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(Susceptible, Infected, Removed) and assumed homogenous mixing of such compartments. A vast body of work created since has incorporated a network perspective in modeling of epidemics (eg. [17, 18]). The underlying assumption in these studies is that the network structure serves as the infrastructure for propagation and therefore bounds the dynamics that can occur. Adoption of such a network perspective has yielded an increased understanding of disease spreading behavior.

The notion that spreading phenomena are based on more complex interaction patterns has more recently gained traction in network science. Resulting in studies of cascades in inter-dependent [5, 6], multi-layered [2, 4, 7, 14], and multiplex networks [11]. Specifically in the field of physics, considerable progress has been made in modeling and in understanding how coupling between networks affects the dynamics in multi-layered systems [4, 12]. This body of work has highlighted that the inter-layer connections –both in terms of structure [10] and strength [8, 11]– strongly impacts the spreading dynamics [7], highlighting the importance of adopting a coupled system perspective for spreading phenomena.

While previous examples are all part of the set of coupled system studies, capturing the idea that spread occurs in systems which consist of multiple coupled systems, the way in which the system is described varies strongly across studies. For example, a multiplex network setting assumes a single set of nodes (agents or actors) connected by multiple types of ties, whereas multi-layered and inter-dependent settings assume two (or more) systems, each with a set of separate nodes and ties that are (partially) connected by an inter-system layer.

Especially in social contexts, which are based on the behavior of people, the multi-layered perspective seems to naturally fit. People have a variety of drivers for multiple types of interactions, and mobility patterns (and thus interaction patterns) that are strongly bound by geographical constraints. It is easy to interact with those that are geographically proximate, e.g. within a city of residence. Although long geographical jumps are possible (for example by air travel) such jumps are often much less likely. Therefore, the human interaction system is both fundamentally multiplex (many types of interactions) and multi-layered (mobility on different scales). In this system, locally dense networks across the globe are coupled by means of occasional long jumps. The inherent structure of this system makes any propagation process based on the human interaction a prime example of a phenomena that should be studied using a coupled networks approach.

In line with this reasoning, [2] is a prime example of adopting a coupled system approach in epidemiology, and the model presented is a big step forward from the single system model. It should be noted that, albeit being multi-layered, this is not a model of coupled networks as the local layer consists of a gravity model rather than a network model. While this might have been a modeling choice, as network data with this granularity is hard to obtain, it is indicative of a general issue that applies to most coupled network research. As the scope shifts from a single networked system towards a system of coupled networks, the focus shifts from characteristics of the single network towards the characteristics of layer that connects the networks; from the local structure towards the structure of inter-system layer. In doing so the lessons learned from the local structure seem to be more and more forgotten and/or ignored.

There are many studies that have shown that, in single network settings, the network structure is a critical factor if one wants to understand, predict, and steer spreading dynamics. For example, it is known that shorter average path lengths greatly increase spreading potential [21], skewed degree distributions allow for even faster and more widespread disease cascades [1, 18] and that local clustering improves local spreading but hampers widespread disease cascades [19]. Yet in coupled network studies these local influences are commonly oversimplified, receive little attention, and are by no means systematically addressed. This raises the question whether, in the context of coupled networks, the local structure indeed plays no role (as suggested by [15]), or whether this role is falsely being ignored.

2 Methodology

Exploring the role of local network structure on disease spread in a coupled setting requires a model consisting of two main components; a system consisting of coupled network structures, and a disease spreading mechanism. We incorporate these two elements in an agent-based model (ABM) in NetLogo [22], and using LevelSpace [13] we adopt a multi-level modeling approach [16] for the coupled network scenarios.

2.1 The structure of the system

Building on the notions put forward by [2]) we create a system that consists of two types of layers: the "within-city" layer and the "between-city" layer. The within-city layer describes the structure of a single city which consists of a population of 1000 individuals which are connected in a fixed network structure. The network structure is one of the classical network topologies; Erdös-Rényi [9], scale-free [3], small-world [21] with a rewire probability of 0.05, or a regular ring lattice. The between-city layer consists of a model that captures the effects of coupling, each within-city layer is modeled separately and is connected by means of the between-city layer. Therefore the between-city layer acts as a bridge between the within-city models, effectively making this a multilevel model.

In this study we are interested in the effects of the local structure, the structure of the within-city layers, on disease spread dynamics in coupled settings. We know from previous literature that the inter-system (between-city) structure and strength are critical factors that influence the local dynamics, therefore we aim to reduce the impact of this layer as much as possible. We do so by simplifying the between-city model in three ways. First, we assume that there are only two coupled cities. Second, we assume that any between-city interaction will occur randomly. Both assumptions reduce the complexity of the between layer structure, of which a schematic representation can be found in Figure 1. Third, we assume that the spreading dynamics within and between cities are the same. More details on the dynamics can be found in the next section. Note that the third assumption implicates that the type of ties within and between cities are the same. Therefore one could model this as single

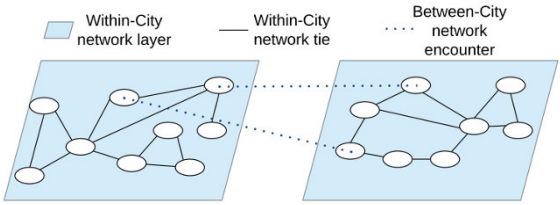


Fig. 1: In our model two within-city networks (n = 1000) with a fixed structure are coupled by randomly occurring encounters across the layers

giant network, where every individual in one cluster (city) is connected to every individual in the other cluster (be it with lower weights). Note that the resulting model would have orders of magnitude more links than the multi-level approach adopted in our study. For our parameter-set, in which cities are relatively small, the number of links in a single network would increase from 10,000 (5000 links in each city), to 1,010,000. This growth in the number of links would significantly increase the computational resources required, indicating that a multilevel modeling approach is far more powerful and scalable in coupled network settings.

2.2 description of disease spreading rules

In line with traditional compartment models, we assume individuals can be in one of four states: **S**usceptible, **E**xposed, **I**nfected, or **R**emoved (SEIR). All individuals are by default in the susceptible state. At the beginning of the simulation, two individuals in the focal city are exposed to the disease, effectively seeding the disease to 0.2% of that city. By interacting with susceptible and infected alters, individuals can then move from Susceptible $\rightarrow$ Exposed $\rightarrow$ Infected $\rightarrow$ Removed states.

We assume that disease spread is caused by interactions (encounters) rather than the network structure itself. One can imagine the network structure as describing the structure of friendships, this structure provides the infrastructure of interactions. This means that having a friend that is sick does not put one directly at risk, however, interacting with that friend does. It is therefore the encounters in the network which drive the spread of disease, not the structure itself. We assume that during each time-step (tick) of the simulation, each Exposed and Infected individual has a certain number of encounters with its network neighbors. The number of such encounters is drawn from a Poisson distribution with a mean that is conditional on the state of the actor which can be varied in our model. Exposed individuals have a mean encounter rate of c_{ES} while infected individuals have a mean encounter rate of c_{IS}. We assume that the social activity (number of encounters) of individuals depends on how how sick they are, hence Exposed (asymptomatic) individuals will have a higher number of encounters than Infected (symptomatic) ones.

The neighbors encountered are chosen randomly and independently; a neighbor

may be encountered multiple times in a single tick. Note that this means that the number of encounters an individual has is completely independent of their degree. This ensures that varying degree does not directly influence the rate at which the disease spreads. When comparing different network structures, keeping the encounter rate independent of degree ensures that any differences we observe are a result of the different network structures rather than different distributions of encounter rates. An example to illustrate: if encounter rates were proportional to degree, almost all individuals in the scale-free network would have a very low encounter rate (due to their low degree) while all individuals in the ring network would have the same, mid-sized encounter rate. This would make it impossible to distinguish if the observed effects are caused by variations in network structure or encounter rate.
When an Exposed (or Infected) individual encounters susceptible neighbors they become exposed with a given probability, which depends on the state of the individual that encountered them (whether the source is exposed (i_{ES}) or infected (i_{IS}). Exposed individuals automatically become infected after a certain duration which is drawn from an exponential distribution with mean $1/\delta$, and infected individuals become removed after a certain duration also drawn from an exponential distribution with mean $1/\delta$.
In line with [19] all experiments use the following parameters:

- mean degree (for all network types): 10
- c_{ES} – mean number of encounters for exposed: 4
- c_{IS} – mean number of encounters for infected: 1.25
- i_{ES} – probability of infection from exposed: 0.05
- i_{IS} – probability of infection from infected: 0.06
- $1/\varepsilon$ – mean duration of exposed: 15
- $1/\delta$ – mean duration of infected: 15

As stated prior, disease dynamics follow the same logic in both layers (between-city and within-city). Rather than adding ties and increasing the pool from which encounters are pulled, the between-city model will redirect a certain percentage of the within-city encounters to be with individuals in the neighboring city. The reasoning behind redirection rather than addition is that adding between-city encounters would effectively change the rate at which disease can spread, which would make comparison across scenarios invalid. In our simulations 1% of the within-city encounters are redirected to the other city, meaning that within-city encounters are reduced to 99% of their initial rate in the single non-coupled city scenarios.
Selection of between-city encounters occurs completely random and independently, where any individual in one city can encounter any individual in the other city. For the purpose of this paper this way of modeling the between-city network is most applicable, yet, future work should be performed that compares different methods of connecting cities in order to understand interaction effects between the local (within-city) and the inter-system (between-city) structures.

2.3 Differential equation model

To create a base-line of disease spreading behavior we compare the within-city Agent-based model (ABM) with the classic SEIR compartmental model based on differential equations (DE). Similar to the ABM, in the DE model the population is divided into four segments: susceptible (S), exposed (E), infected (I), and removed (R). Also similar to the ABM, the susceptible population becomes exposed at a rate based on the infection rate and encounter rate of the exposed and infected populations. The differential equations encoding these relationships are given in the following equations:

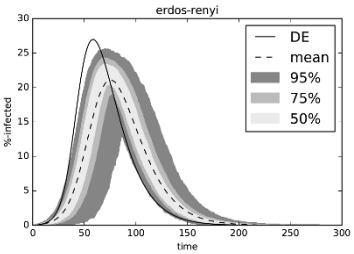
$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= -(c_{ES}i_{ES}E + c_{IS}i_{IS}I)S \\
\frac{\mathrm{d}E}{\mathrm{d}t} &= (c_{ES}i_{ES}E + c_{IS}i_{IS}I)S - \varepsilon E \\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \varepsilon E - \delta I \\
\frac{\mathrm{d}R}{\mathrm{d}t} &= \delta I
\end{aligned}$$

3 Results

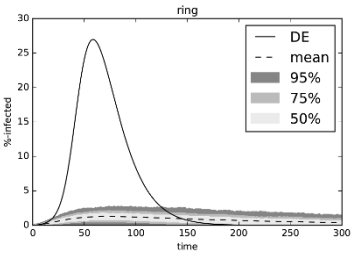
To see if the simulation model behaves as intended, we start our analysis by reproducing the study conducted in [19] in a single network setting. We find that, in comparison, disease dynamics in our model (Figure 2) are stretched out over a longer period of time but follow corresponding trends across various structures. The observed delay is to be expected given our cities are 5x larger than those in the original work. This makes it more time consuming for the disease to reach saturation, which is indeed what we observe. As our disease spread dynamics are in line with [19], this serves as a sign that the agent-based simulation model is behaving as intended.

The single city results show that the spreading dynamics in the ABM differ significantly from those of a Differential Equation (DE) model; the peak load is much lower and occurs much later. Note that, even though the DE model effectively allows any individual to come into contact with any other individual, the number of encounters in the network model is fixed to be the same as in the DE. Therefore these difference do not stem from a reduced number of encounters in the network settings. Instead, the observed differences in spreading speed arise from localized connections and local clustering. The higher clustering increases the chance of inefficient encounters —from sick to sick—, reducing the effective spreading rate [19].

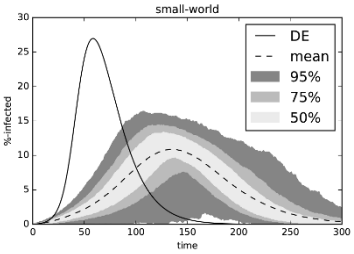
We continue the analysis by using the ABM to study the effects of coupling of within-city networks. While adding inter-city ties effectively adds a second mode of spreading (not only within but also between cities) we correct for the potential effects of such an increase in connectivity by keeping the rate at which individuals encounter others equal across all scenarios. The results (shown in Figure 3) reveal that the effect of coupling on the focal city dynamics is strongly conditional on the structure of the focal city. On the one hand, in cities with Scale-free and Erdös-Rényi networks, coupling does not result in any observable effect on disease spread



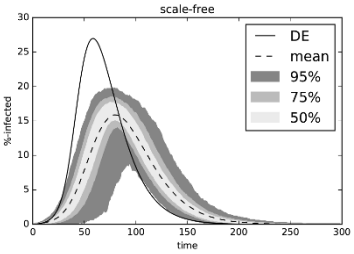
(a) Disease spreading dynamics in a single **Erdös-Rényi** network



(b) Disease spreading dynamics in a single **Ring** network



(c) Disease spreading dynamics in a single **Small-world** network



(d) Disease spreading dynamics in a single **Scale-free** network

Fig. 2: This figure shows the disease spreading dynamics in cities with varying within-city networks. The top (and bottom) percentiles are depicted in greyscale for a total of 1000 simulation runs in the Agent-based model. The dynamics of the differential equation of the same disease are plotted in blue.

dynamics. On the other, in cities with a small-world or ring networks, the spreading seems to be improved due to coupling. This is in line with previous work claiming coupled networks can suffer from increased volatility [20]. These results suggest that the effect of coupling on the focal city's dynamics is strongly dependent on the within-city network structure of the focal city.

It is interesting to note that the focal cities affected by coupling are those that have structures with otherwise highly localized, and thus slow, spreading dynamics. This might suggest that random pathways facilitated by the between-city layer (individuals encountered in the neighboring city are chosen randomly) allow for long jumps which are otherwise unavailable in the focal network structure. This suggests that coupling effectively reduces the diameter of the focal city network via the between-city layer. A more intuitive explanation is that due to the slow spread within the focal network there is enough time for a second order spread —from the focal city to the neighboring city and back to the focal city— to occur before the within-city dynamics have saturated the focal city. The ring network (Figure 3b) clearly shows a second peak of spreading after the initial peak seems to flatten. This suggests the presence of the latter described second order spreading, in which the neighboring city causes

reseeding in the focal city.

These results indicate that the timing of epidemics across coupled networks seems to play a crucial role in the effects of such coupling. As the timing of an epidemic is directly related to where a disease starts, the seed becomes a critical aspect in our simulation. Seeding the focal city, as has been the case in previous analysis, causes the epidemic in the neighboring city to occur with a lag. This lagging reduces the potential impact of the neighboring city on the focal city and consequently the effects of coupling will likely be dominated by the epidemic dynamics within the focal city. To increase the potential effects of coupling we adjust our seeding location and repeat the previous analysis. Now, rather than seeding the focal city, the neighboring city will be seeded. The results (Figure 4) show that when the disease originates from the neighboring city the effects of coupling become much more apparent, resulting in a variety of dynamics in the focal city. When the focal city's epidemic is lagging behind those of the origin city —the city which was seeded with disease— the opportunities for secondary infections increase substantially, but the extent to which they occur depends on the disease growth rate in the the origin city. As we know this growth rate is determined by the local structure (see Figure 2) the observed variance in coupling effects should be attributed to the within-city structure in the origin city.

4 Discussion

Previous research has identified that both network structure and coupling of networks as drivers which can have significant effects on the local dynamics of disease spread. The focus on understanding the effects of coupling has shifted the attention away from the local structure as a driver, resulting in little systematic connection between these two bodies of work. Consequently, the effects of local network structure seem to be poorly integrated in the coupled network literature, both in terms of describing the structure of the local layers of interaction as well as the interaction of such local structures with the inter-layer structure [10]. While both could be addressed using the methodology presented in this paper, the scope of this paper is on highlighting the role of local structure in a coupled network setting.

By means of an Agent-Based Model of two coupled cities we have shown that local growth dynamics, caused by the local within-city structure, plays a crucial role in understanding if and how coupling will affect the focal disease spreading dynamics. While the relevance of the local (within-city) structure of the focal city has been identified in both single [17] as well as in coupled network settings [10], we find that the local (within-city) dynamics of the neighboring city also impacts the focal spreading dynamics. This indicates that simply knowing the focal city's structure and the way in which it is coupled to other cities is not sufficient for understanding spreading behavior. We find that the dynamics in neighboring cities, which depend on the neighboring city's local structure and the dynamics in the neighbor's neighbors, play a critical role in focal city's spreading dynamics. The feedback among cities not only indicates that the structural details in each of the local (within-city) layers matters, but also that dynamics of the focal city cannot be accurately considered without incorporating the coupled perspective.

Our results further emphasize the critical role of the effectiveness of the between-city layer. We find that a sufficient amount of time is needed for the coupling to become effective. This amount is conditional on both the focal growth rate (driven by within-city structure) and neighboring growth rate (driven by neighboring within-city structure). When the focal city's disease load is saturated it will not likely be affected by anything from the outside, making coupling a less important factor. This draws the attention to path dependence as a driver of spreading in coupled networks. If enough time is available, coupling can become efficient and has a strong effect on focal spreading dynamics. This observation is in line with previous work that identifies coupling strength as a key driver for coupling effects [8, 11].

While our model is conceptual in nature, there are interesting implications for health policy that can be devised from it. A comparison among seeding locations (the comparative plots are not included in this paper but can be done by comparing Figure 3 to Figure 4) indicates that for structures with relatively slow disease spreading (Small-world, Ring) a scenario that has a seed outside the focal city results in earlier and higher peak loads in the focal city, compared to the same scenario in which the focal city is seeded. Therefore, outside infections provide a higher risk for the focal population. In concrete terms, our results suggest that reducing disease load within a city (or country) is best achieved by preventing coupling, and this indeed seems to be a strategy implemented to prevent global pandemics like the 2014 Ebola

spread. However, as very small coupling probabilities have significant effects and complete decoupling seems infeasible, the effectiveness of such strategies will be limited, especially as global travel increases over time. When complete uncoupling is not an option it seems that reducing outbreaks in neighboring cities is more critical for controlling the dynamics in the focal city.

This is somewhat in conflict with the current way in which health policy is implemented; based on local agencies (be it the city, state, country) with local data and dynamics. Our results suggest a different approach with global coordination, in which the coupling of networks is considered and a global intervention strategy is implemented, not only because it is socially desired, but because it is in each local network's own self interest.

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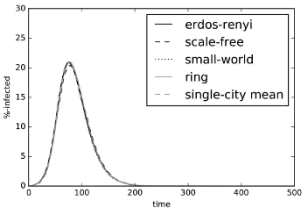
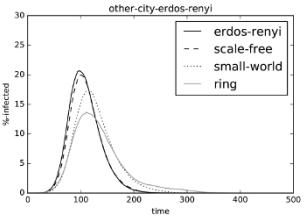


(a) Mean disease spread for a focal city with a **Erdös-Rényi** network, for various structures in the neighboring city



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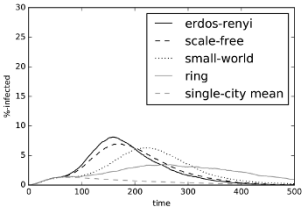
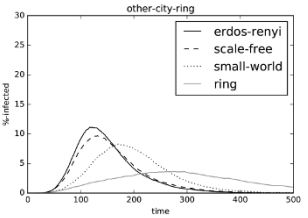


(b) Mean disease spread for a focal city with a **Ring** network, for various structures in the neighboring city



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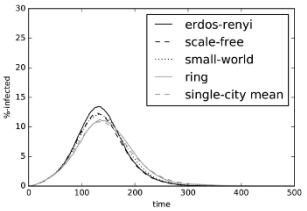
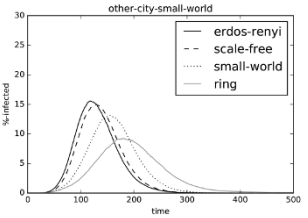


(c) Mean disease spread for a focal city with a **Small-world** network, for various structures in the neighboring city



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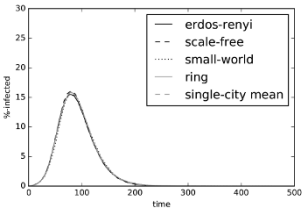
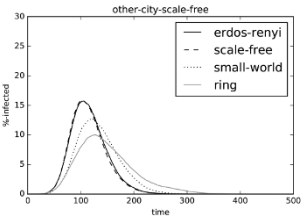


(d) Mean disease spread for a focal city with a **Scale-free** network, for various structures in the neighboring city



(d) Mean disease spread for a focal city with a **Scale-free** network, for various structures in the neighboring city

Fig. 3: This figure compares the disease spread dynamics in the focal city in scenarios in which the **focal** city is seeded with disease, while the network structure of the neighboring city is varied.

Fig. 4: This figure compares the disease spread dynamics in the focal city while the **neighboring** city is seeded with disease, while the network structure of the neighboring city is varied.