

An Agent-Based Model of COVID-19

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Many simple models of disease spread assume a homogeneous population (or population groups) with a uniform basic reproduction number (R_0). The goal here is to develop and analyze an agent-based model of disease that models: (1) variability of interaction rates between agents; and (2) the structure of the in-person contact network.

Keywords: epidemiology; COVID-19; agent-based modeling; network models

1. Introduction

We start by specifying the model and observing its most basic properties, and then spend considerable time investigating its detailed behavior in many different scenarios. By the end, the conclusions will include:

- Unsurprisingly, there is a robust critical point as we vary the mean interaction rate, which determines whether a large fraction of the population is infected, or a small fraction. (This critical point corresponds with when $R_0 \approx 1$ in models that have a uniform R_0 .)
- The structure of the contact network significantly affects disease spread (even after controlling for parameters like graph density).
- It is better to have many small meetings than a few large ones.
- The heterogeneity of interaction rates significantly affects disease spread. In other words, you need to model not just the average amount of interaction, but the distribution of interaction rates from agent to agent.

1.1 Existing Work

There are a number of existing agent-based and network models of disease spread [1]. Some are simple [2], while others can be very complex [3–5].

Many of the more complicated models consider the parameters age, work status, preexisting immunity and so on. However, it is often unclear what effect these added parameters have. It is clear that

they have some effect, but which parameters are important, and what changes to the input parameters correspond with what changes in outcomes?

The goal of this paper is to introduce and analyze a simple but flexible model of disease spread that will allow us to study the effects of different modeling choices. In particular, we investigate the influence of distributions. How does the distribution of recovery times affect disease spread? What about the distribution of interaction rates in a heterogeneous population? Or the graph distribution from which the contact network is sampled? These distributions are often assumed or only dealt with implicitly. We will make them explicit so we can better understand their impact on our model.

2. Making the Agent-Based Model

2.1 Outline

We will start with a graph representing the contact network of the population. Each vertex will represent an agent and edges will connect pairs of agents who might interact. Each agent will be marked as susceptible, infected or recovered. Initially, a few agents will be marked as infected, while all others will be marked as susceptible. At each step, every agent will pick a random subset of its neighbors in the contact network, and it will “meet” (interact) with them. If a susceptible agent meets an infected one, it becomes infected as well. Meetings are considered symmetric, so it does not matter which agent initiates the meeting. Finally, infected agents will become recovered after some period. We run these steps repeatedly until there are no infected agents left, at which point the disease has run its course.

There are many parameters here to investigate. For example, we can look at how the structure of the contact network influences disease spread. We can also look at the importance of the distribution determining the number of meetings each agent initiates, as well as the distribution of recovery times. We will start by implementing the general model, and then we will explore the parameter space.

2.2 Implementation

We start by getting a social network to work with. Later we will use something larger and more realistic, but for the purposes of illustration we will start with a very small example network of Florentine families (Figure 1).

At each step of the simulation, each agent will pick some subset of its neighbors to meet with. We need to specify the distribution that determines how many neighbors each agent will meet with at each

step. Later we will explore which properties of these distributions are important, but for now we will somewhat arbitrarily choose an exponential distribution.

Next, we need to model the recovery time. That is, we need to pick the distribution that determines the number of steps an agent must wait to be marked recovered after being infected. Many susceptible-infectious-recovered (SIR) models implicitly use an exponential distribution, so we will start with that here.

We will start by marking some initial agents as infected (Figure 2).

We then have agents randomly meet their neighbors, marking susceptible agents that meet infected ones (Figure 3).

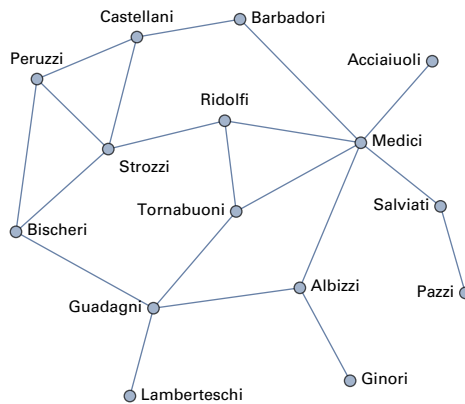


Figure 1. The social network of Florentine families.

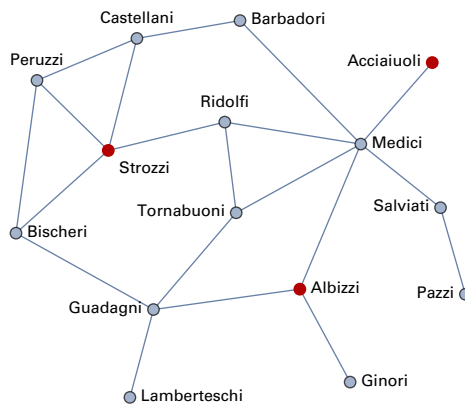


Figure 2. The social network with infected agents highlighted. At the beginning of the simulation, a few random agents are marked as infected.

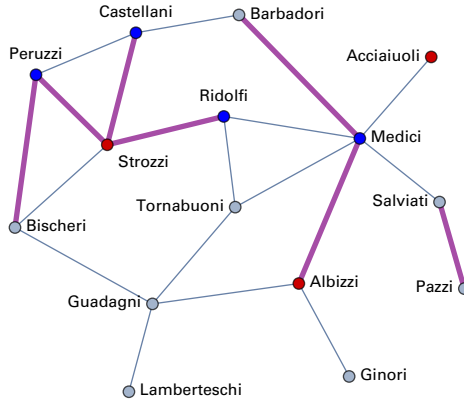


Figure 3. The social network with initially infected agents marked in red, random meetings between agents marked in purple and newly infected agents in blue.

Susceptible agents that meet with infected agents will be marked as infected, and the simulation will continue.

2.3 Simple Example

We will start by running a simple example to test this model. First, we need a contact network to run on. We will look at different graph distributions later, but we will start by using the Watts–Strogatz [2] graph distribution (Figure 4), which is often used to approximate real social networks.

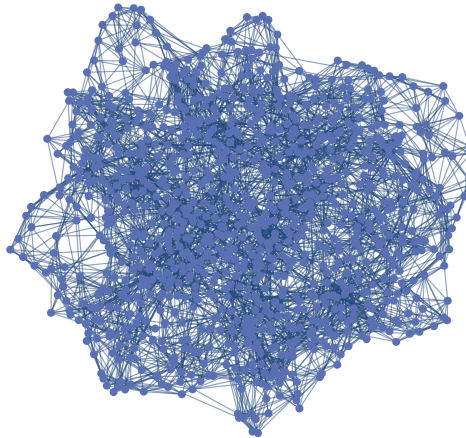


Figure 4. A random graph sampled from the Watts–Strogatz graph distribution with 1000 vertices and rewiring probability 10%.

Next, we run a simulation on this network, with an exponential distribution with mean 0.4 modeling the number of outgoing meetings for every agent, an exponential distribution with mean 7 modeling the recovery time and 10 random agents initially infected. Figure 5 shows the fraction of agents susceptible, infected and recovered over time, in a way similar to common visualizations of SIR models [1, 6, 7].

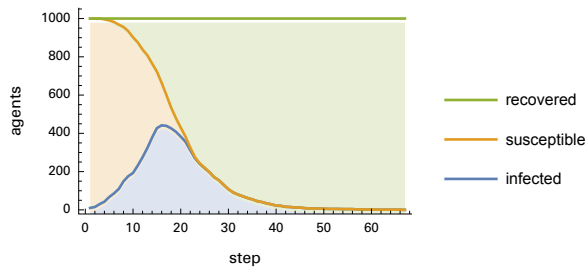


Figure 5. Fraction of agents susceptible, infected and recovered over time (with time measured in steps).

At the beginning, almost all agents are susceptible. Then, an (approximately) exponentially increasing number of agents become infected, while previously infected agents recover. By the end, there are a handful of susceptible agents left who were never infected, while the rest were infected and recovered. This is essentially the output we would expect from a simple SIR model.

2.4 Phase Plots and Basic Properties

We can also visualize the evolution of this simulation as a phase plot (Figure 6) with a curve through three-dimensional space, with axes for the number susceptible, infected and recovered. However, because the number of agents remains fixed ($S + I + R$ is constant), we can convey the same information by just picking any two dimensions.

We can expand this into a vector field showing us where the critical points and attractors are (Figure 7). However, we have to change distribution of recovery times to be geometric. The problem with other distributions is that this phase plot collapses some important dimensions, like how long the infected agents must wait before recovering. The only distribution where this information is not needed is a geometric distribution, because a geometric distribution models the recovery time if there is a constant probability of recovering at each step, which does not require any hidden state.

Using a geometric distribution of recovery times, we get the phase plot shown in Figure 7.

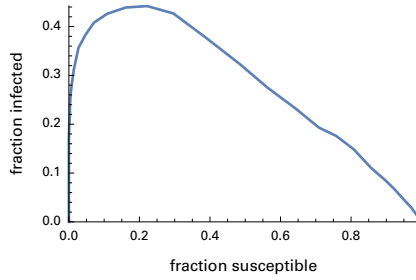


Figure 6. A phase plot of a single simulation. In the initial state, all but a few agents were susceptible, while only a few were infected (the bottom right). With each step, the fraction of agents susceptible decreased, while the fraction infected increased. The infected agents eventually recovered, ending the simulation with very few agents susceptible and all others recovered (the bottom left).

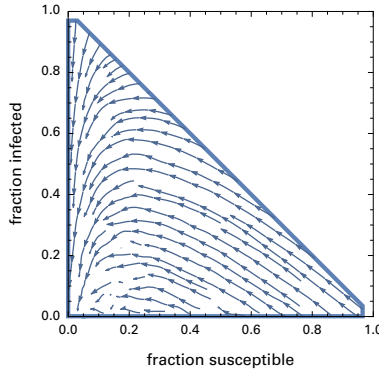


Figure 7. A phase plot showing the tendency toward all agents being marked as recovered (the bottom left).

We can see that there is an attractor at $\{0, 0\}$, suggesting that with these parameters, everyone will become infected (because nobody susceptible will be left). Later we will see how the parameters of our simulation affect this phase plot.

3. Exploring the Parameter Space

There is a huge parameter space for this model, so we will not be able to go through everything. There is the structure of the underlying contact network. There is the distribution of recovery times. There are the distributions that determine the number of meetings that take place (as well as the distribution of those distributions). And so on.

For practical purposes, there are two outputs for any simulation that seem important: the total fraction of agents who are infected and the peak fraction of agents who are infected at any given time (the latter of which is important for “flatten the curve” strategies).

We will experiment with the input parameters to try to see their relationship to these two outputs, and to simulate mitigation strategies.

3.1 Critical Points

We start by investigating the dynamics of the total infected and peak infected variables. We might guess that one of the most important variables is the mean number of meetings per agent per step, which is closely related to R_0 . We can control this by altering the distribution of meeting counts. For now, we will give every agent the same distribution and only change the mean, simulating a homogeneous population (Figure 8).

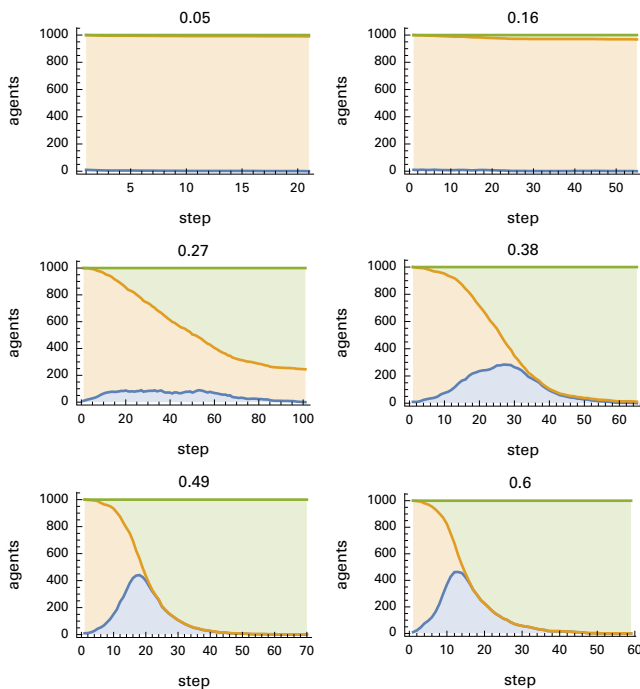


Figure 8. (continues)

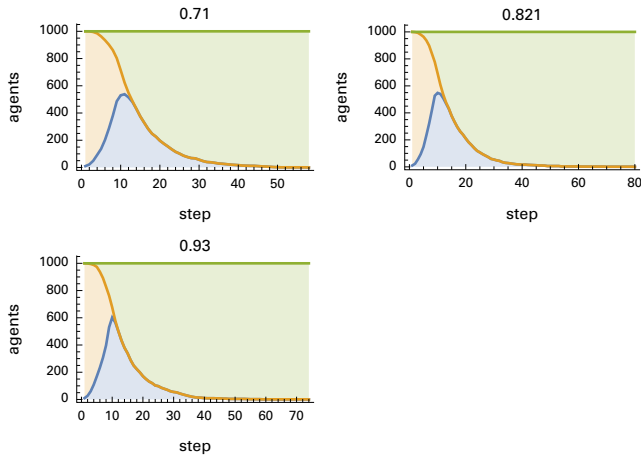


Figure 8. The number of agents susceptible (orange), infected (blue) and recovered (green) over time for different mean meeting counts per agent per step (labeled above each plot).

We can see that as the number of meetings increases, it passes a critical point that determines whether almost everyone gets infected or almost nobody. We can see this critical point especially well when plotting the peak and total infection rates as a function of the mean number of meetings (Figure 9).

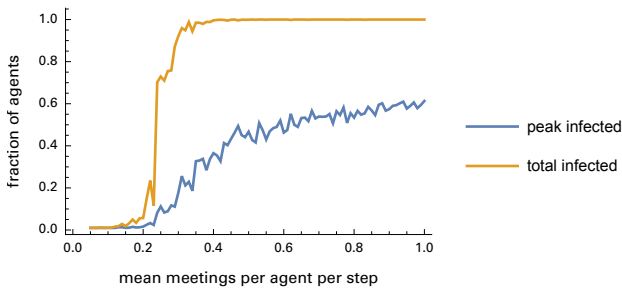


Figure 9. Peak and total infection rates as a function of the mean meeting count. There is a critical point around 0.2 where the total infection jumps up to nearly 100%.

We can clearly see the critical point around 0.2. Interestingly, while the total infection rate jumps almost immediately to 100%, the peak infection rate is much more gradual. This suggests that incremental progress in reducing interaction can have an effect on the peak infection rate, while the total infection rate requires more radical intervention (the former of which is essentially the flatten the curve strategy).

We can also visualize this transition with phase plots (Figure 10).

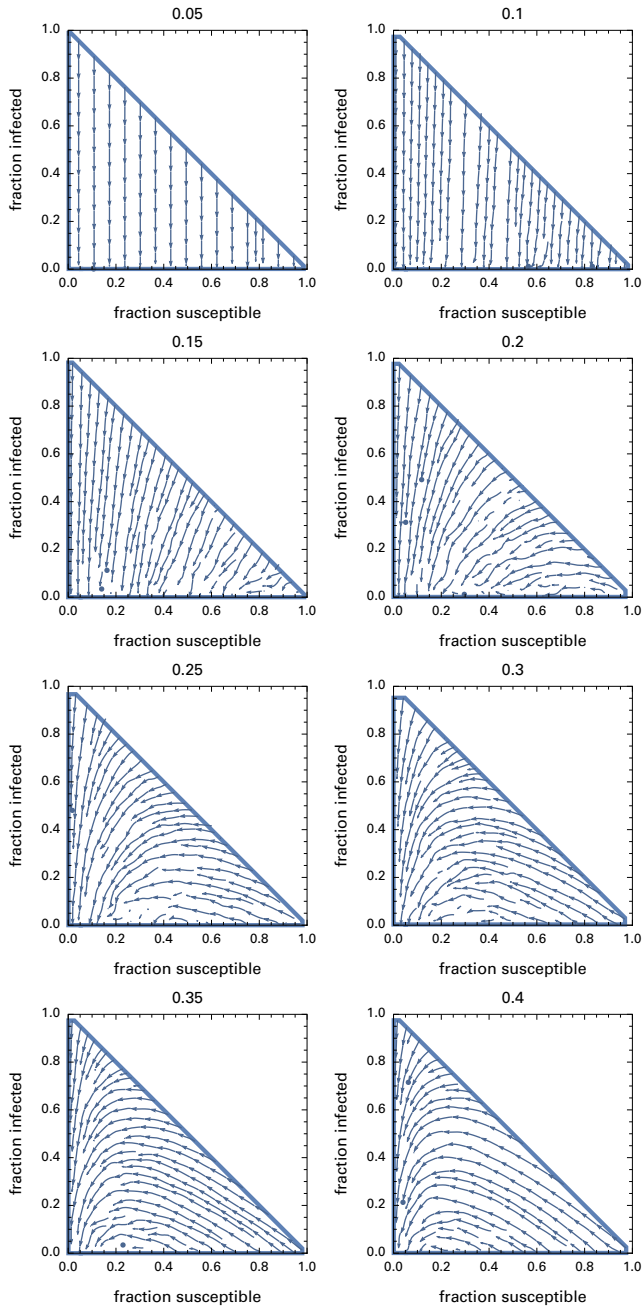


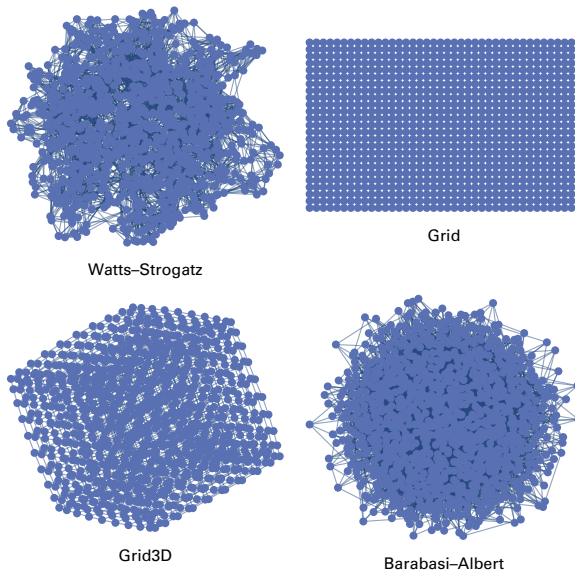
Figure 10. Phase plots similar to Figure 7 with different mean meeting rates.

Here we see that when there is limited interaction, there is an attractor along the bottom edge (where nobody is infected), but as the number of meetings goes up, that attractor is pushed to the bottom-left corner (where nobody is infected or susceptible). We can see that there is a small leftward push for any mean meeting rate, but that the downward push (the recovery rate) is so fast that the number of infected goes to 0 before there can be leftward drift. As the number of meetings goes up, each vector gets more “lift,” which allows it to travel farther to the left.

■ 3.2 Contact Networks

So far we have been using the same Watts–Strogatz graph for our contact network. We could, however, run this model on any graph. Watts–Strogatz is often used to simulate social networks because it generates small-world networks, but there are other graph distributions that simulate various other aspects of social or contact networks. More generally, we do not know yet whether the structure of the contact network matters at all, so we will start by examining that question.

Figure 11 shows a few graphs that we should try.



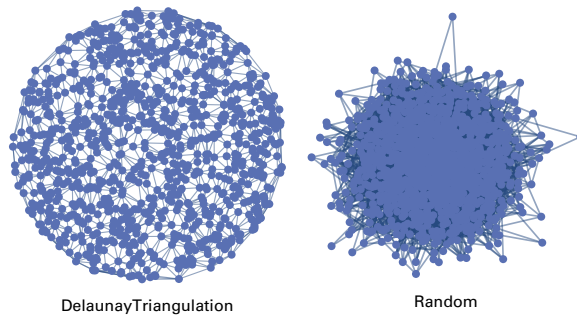


Figure 11. Graph types on which we will run simulations in order to see the importance of the structure of the contact network.

We start with Watts–Strogatz for the reasons mentioned above. Next, we look at the Barabási–Albert graph distribution [8], which is also often used to simulate social networks. The tradeoff between Watts–Strogatz and Barabási–Albert is that Watts–Strogatz has more realistic clustering and an unrealistic degree distribution. (Barabási–Albert has a power law degree distribution [8], which could be good for modeling how there are a few people who have contact with many different people, like cashiers, while most people interact with a smaller consistent set.)

Next we use a grid graph because it is analogous to how many simple spatial SIR models work. It is also not a small-world network, which might yield different results. Grid3D is interesting because it is structurally similar to the grid graph but it has a higher graph dimension. We also use the Delaunay triangulation of random points to generate a less-structured planar graph.

Finally, Figure 12 (left) shows a random graph with approximately the same density as the small-world networks.

The results are shown in Figure 12 (right). The planar networks saw much slower growth. The random network and the Barabási–Albert network were very similar and showed very fast growth. Watts–Strogatz gave an intermediate result.

This ordering seems to roughly coincide with the graph dimension (which models how many vertices are covered by a ball of a given radius).

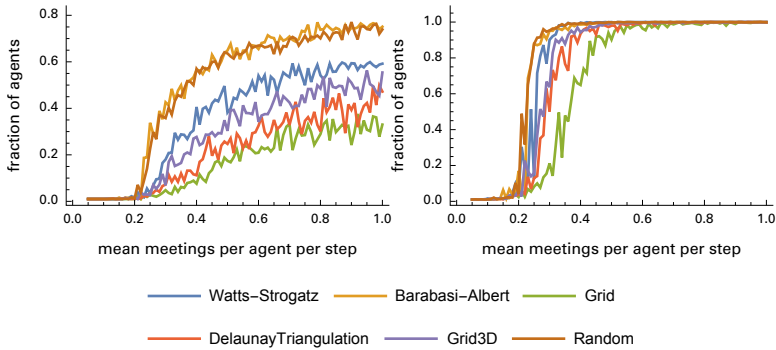


Figure 12. (left) Peak and total infection rates as a function of the mean meeting rate for different graphs. (right) While the critical point at 0.2 stays the same, the behavior for higher interaction rates is significantly different for different graphs.

The main conclusion here is that even if the number of meetings is held the same, agents with a larger number of neighbors who they could potentially meet cause faster spread, demonstrating one way in which the structure of the contact network is important independent of the amount of interaction.

(It should also be noted that this is not just because there are “wasted meetings” where the number of meetings is greater than the number of neighbors. Empirically, this is a rare occurrence even for a high mean number of meetings per agent per step.)

We can also try generating several graphs from the same distribution but with different densities. Figure 13 shows the peak and total infection rates as a function of the mean meeting rate on graphs sampled from the Watts–Strogatz graph distribution with increasing density.

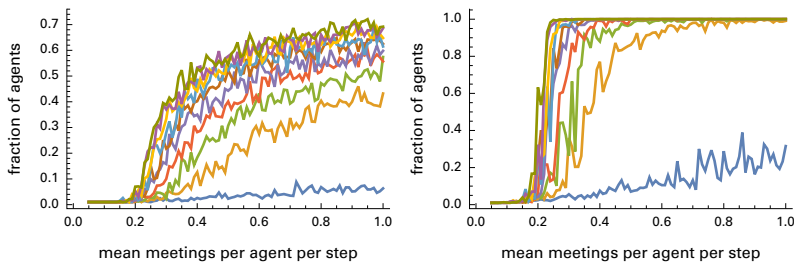


Figure 13. The (left) peak and (right) total infection rates as a function of the mean meeting rate on graphs of increasing density. The lower curves correspond with sparser graphs, while the higher curves correspond with denser graphs.

We can see that the very sparse graphs have softer curves and slow spread, while the denser networks show faster spread. We still have not changed the mean interaction rate, so this again shows that the structure of the contact network matters.

3.2.1 Joined Networks

While social networks are generally considered to be small-world, it is unclear if this is also true of in-person contact networks. People who are geographically close likely have more in-person contact than people who are not. Somebody who lives thousands of miles away might be a friend, but they are many steps away on the network of person-to-person contact. It is not immediately clear what this contact network should look like, but we will attempt to make something that might approximate it.

We start by making a “supergraph” (Figure 14) made up of smaller clusters. Each cluster will be a small-world network, modeling a local community, which could be like a town or city, while the network of clusters will be some planar graph representing the connectivity between these geographic entities.

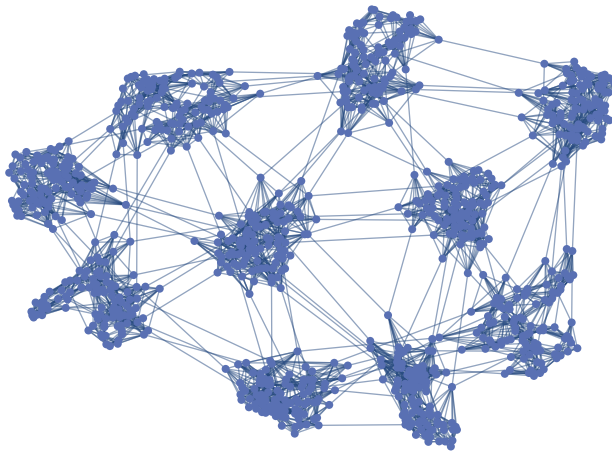


Figure 14. An example of a “supergraph” with 10 communities, each containing 100 vertices and with five edges connecting adjacent communities.

For our purposes, we will use Watts–Strogatz for the clusters (communities) and the Delaunay triangulation of random points for the large-scale structure (the contact between communities). We can vary how many edges connect adjacent clusters to change how interconnected the communities are.

We can now run simulations on these graphs (Figure 15) just like before, except we will force all the initially infected to start in one cluster (so the disease is forced to spread from cluster to cluster).

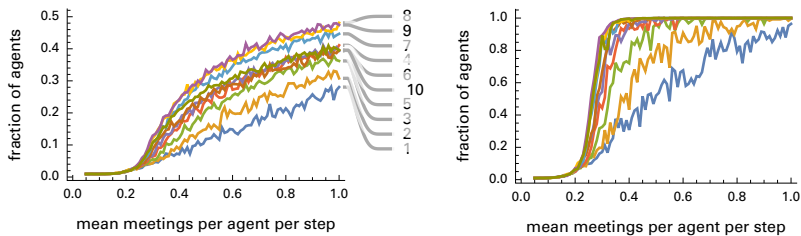


Figure 15. The (left) peak and (right) total infection rates as a function of the mean meeting rate for 10 runs with different numbers of edges connecting adjacent clusters.

In both of these cases, we see that when clusters are only weakly connected, the disease unsurprisingly spreads slower. In particular, we see that the total fraction infected does not transition as quickly from close to 0% to close to 100%, but instead ramps up slowly. We get this because while disease spreads quickly inside the small-world clusters, it does not spread as fast on the large-scale planar graph, meaning that not all clusters get infected. As the clusters become more interconnected, however, it limits the curve we get with a giant small-world network. Interestingly, however, the curve we get when clusters are only weakly connected seems different from the curve we get on planar networks of this type, suggesting that there is modeling value by simulating interactions within the clusters.

In other words, reducing interaction between communities can be more effective than reducing interaction within communities.

3.3 Interaction Rates

Many interventions (like social distancing) involve changing the frequency or size of meetings, so we should look at how the distribution of interaction rates (meeting counts) changes the peak and total infection rates.

We have already looked at what happens when we change the mean meeting count (there is a critical point where the disease switches from dying out to infecting everyone). However, so far we have only used exponential distributions to model the number of meetings per agent. We begin by trying different distributions (Figure 16).

The Pareto distribution produced similar results to the exponential distribution. Otherwise, the higher the standard deviation, the softer

the curve, and the earlier the critical point. In other words, it is on average worse to have some agents meeting lots of people and others meeting nobody than it is to have every agent meeting an average number of people. That is, it is better to have many small meetings than a few large ones.

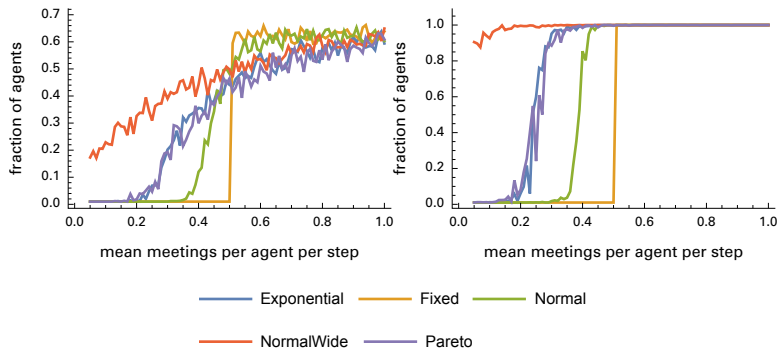


Figure 16. The (left) peak and (right) total infection rates as a function of the mean meeting rate for different distributions of meeting rates.

So far every agent has been the same, with the same distribution determining how many meetings they initiate. However, we could also model a heterogeneous population by giving different agents different distributions of meetings per step.

We will simulate homogeneous and heterogeneous populations. In the homogeneous population, every agent has a normal distribution that determines the number of meetings they will make at each step. In contrast, every agent in the heterogeneous population makes a constant number of meetings at each step, but that constant number is different between agents and modeled by the same normal distribution as in the homogeneous population. In other words, the mean and variance of the number of meetings per agent per step are the same, but in the homogeneous population we sample at each step and in the heterogeneous one we only sample once (Figure 17).

Interestingly, these produce slightly different results, with the heterogeneous population generally seeing slower disease spread. In the heterogeneous population there might be a few small neighborhoods where the local interaction rate is below the critical point, while in the homogeneous population all agents are uniformly on one side of the critical point or the other. More generally, this shows that we cannot assume that a homogeneous population will behave the same as a heterogeneous one.

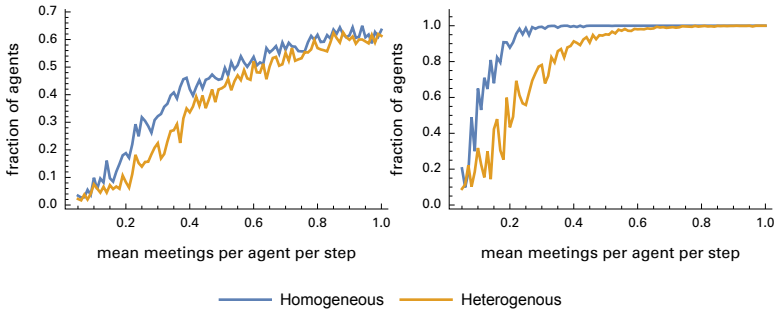


Figure 17. The (left) peak and (right) total infection rate as a function of the mean meeting rate for homogeneous populations, in which every agent has the same distribution of meetings per step, and for heterogeneous populations, in which every agent has a random fixed number of meetings per step.

3.4 Recovery Times

So far we have been using a geometric distribution for recovery times because it makes it possible to generate phase plots. However, actual recovery times are probably modeled by something else (for example, many SIR models implicitly use an exponential distribution).

We can also modify the recovery time distribution to simulate quarantines because a recovered agent no longer infects its neighbors, and so a quarantined agent behaves like a recovered one in our model. However, this trick only works when looking at the total fraction of the population infected because the peak infection rate will be misleading since all of the “infected” agents who are in quarantine will be marked as recovered.

First, we will try different geometric distributions with different means (Figure 18).

We can see that as the mean recovery time gets shorter, the peak fraction of agents infected decreases (which is not surprising because we are just reducing the mean time that each agent spends infected). We also see that the critical point between nobody being infected and the whole population being infected is shifted right. In other words, quarantines increase the amount of interaction required for the disease to spread.

Next, we will look at the effect of choosing different distributions by running simulations with a list of plausible distributions, all with the same mean value (Figure 19).

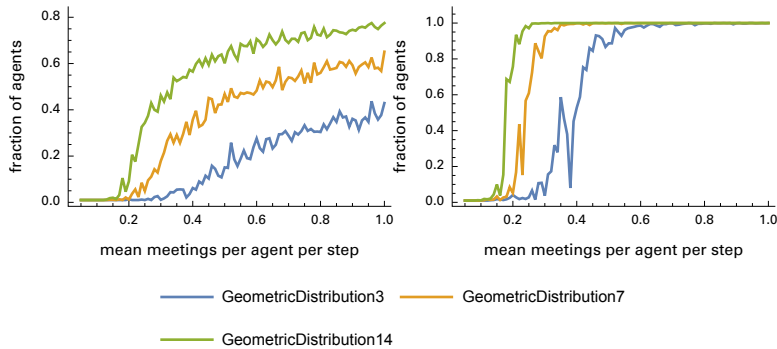


Figure 18. The (left) peak and (right) total infection rates as a function of the mean meeting rate for different recovery time distributions. In this case, we look at recovery times modeled by geometric distributions with means of 3, 7 and 14 steps.

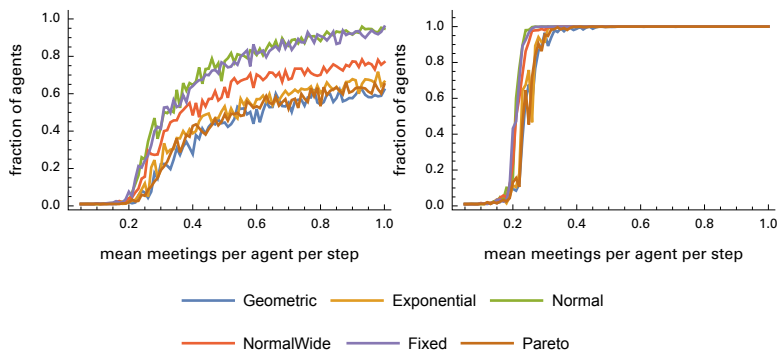


Figure 19. The (left) peak and (right) total infection rates as a function of the mean meeting rate for different distributions with the same mean.

Here we see that the total fraction infected is not significantly affected by the distribution of recovery times. The peak fraction infected, however, is affected by the distribution of recovery times, and the fixed and normal distributions lead to a higher peak. Interestingly, the normal distribution with a higher standard deviation leads to a lower peak than the one with a lower standard deviation.

4. Conclusions

The main purpose of this project was to develop this model and investigate its properties. As with many agent-based models, its behavior is complicated and the relationship between the inputs and outputs is often hard to model. For computational details see [9]. Below is a partial list of findings:

- There is a critical point in the amount of interaction that determines whether everybody gets sick or nobody does. This corresponds approximately to $R_0 \approx 1$.
- The structure of the contact network matters. Even with the same number of vertices, edges and other settings, different contact networks produce different results. Barabási–Albert had very fast spread, Watts–Strogatz had intermediate spread, and the “supergraph,” which was a planar graph made up of Watts–Strogatz clusters, had slow spread when the clusters were only weakly connected.
- Reducing interaction between communities increases the uncertainty in the outcome, but flattens the curve and reduces the average total infection rate.
- It is better to have many small meetings than a few large ones.
- Changing the recovery time (which is equivalent to having quarantines in this model) changes the position of the critical point between everybody getting infected and nobody getting infected.
- The heterogeneity of the agents is important, and the distribution determining how much individual agents interact changes the outcomes. That is, the mean rate of interaction is not enough to model disease spread, and the distribution of interaction rates is needed.

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