3 Q3 - A global bug: Epidemonic

3.1 Description of the model

We study the spread of a virus on a two dimensional regular grid network[3], where each node has a state corresponding to one of the compartments S (susceptible), I (infected), and R (recovered). A node can change its state (each day) in the following ways

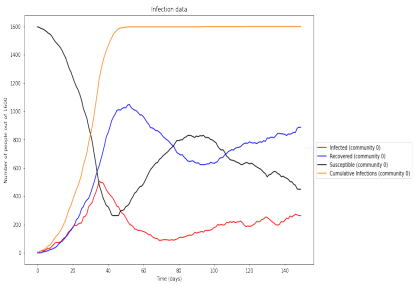
1. A susceptible node becomes infected with a probability $1-(1-p_i)^k$, where k is the number of infected neighbours the node has, and p_i is infection probability
2. A infected node becomes recovered with probability p_r (recovery probability)
3. A recovered node becomes susceptible again with probability p_w (waning immunity probability)

Ten times each day a random pair of nodes are selected to have the state updated, and the selection is without replacement in such a way that all nodes are updated exactly once each day - this was done to mimic the different interactions people have throughout the day.
By default, the infection probability is $p_i = 0.3$, the recovery probability is $p_r = 1/7$, and the waning immunity probability is $p_w = 1/30$. These values were chosen such that the recovery time of an infected individual is geometrically distributed with mean recovery time one week, and the waning immunity time was also a geometric random variable with mean one month - inspired by data observed in Covid patients.
We individually vary the parameters p_i, p_r, and p_w (while keeping all other values default) and generate statistics (mean, and standard deviation for uncertainties) for the following data points:

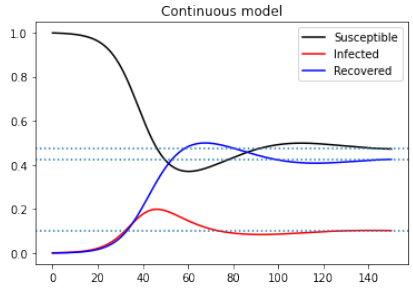
1. Peak infection number (how damaging virus would be to healthcare system)
2. Cumulative number of infection (how far virus spreads)
3. Characteristic time scale - the time when greater than 36% of the population has been infected (speed of virus spread)
4. Equilibrium number of susceptible, infected, and diseased individuals (endemic state of virus)

We will then extend the simulation to involve four sub populations - disjoint 2D grids in the same network - where each day two nodes are randomly selected from two random communities, and with a probability given by the migration

[3] We choose periodic boundary conditions to mitigate boundary effects of a finite sized grid



(a) 40x40 grid with parameter values (p_i, p_r, p_w)=(0.3, $\frac{1}{7}$, $\frac{1}{30}$)



(b) Continuous model with parameters (β, γ, λ)= (0.3, $\frac{1}{7}$, $\frac{1}{30}$)

Figure 12: Comparison of random network model, versus homogeneous mixing continuous model

probability p_m the states of the two nodes are swapped. We will then vary the same parameters and measure the same data points using the total population. In addition, we will also see effect of varying migration probability, as well as measuring the new data point - the time before first infection in each new community.

3.2 Initial observations of the model

3.2.1 Comparing with continuous model

We begin by comparing our model to the continuous SIRS model. A continuous SIRS model can be modeled by the system of differential equations

$$\begin{cases} \frac{dS}{dt} = -\beta IS/N + \lambda R \\ \frac{dI}{dt} = \beta IS/N - \gamma I \\ \frac{dR}{dt} = \gamma I - \lambda R \end{cases} \tag{8}$$

Where $S+I+R = N$ is the total population (assumed constant over timescale). β is the transmission rate, γ is the recovery rate, and λ is the waning immunity rate.

As shown in figure 12, the models have many of the same qualitative features. Note that the continuous model - after some transience - reaches an equilibrium state where number of infected, recovered, and susceptible remain constant. The network models analogous state is an oscillation about an equilibrium state - as shown in figure 13. Note that besides some random influence, at sufficiently large time, the three compartments oscillate[4] about an equilibrium value - these values define the endemic state of the virus.

[4]Interestingly, it appears that the three compartments show phase synchronization, with susceptible and recovered compartments appear off by a constant phase difference of $\frac{\pi}{2}$.

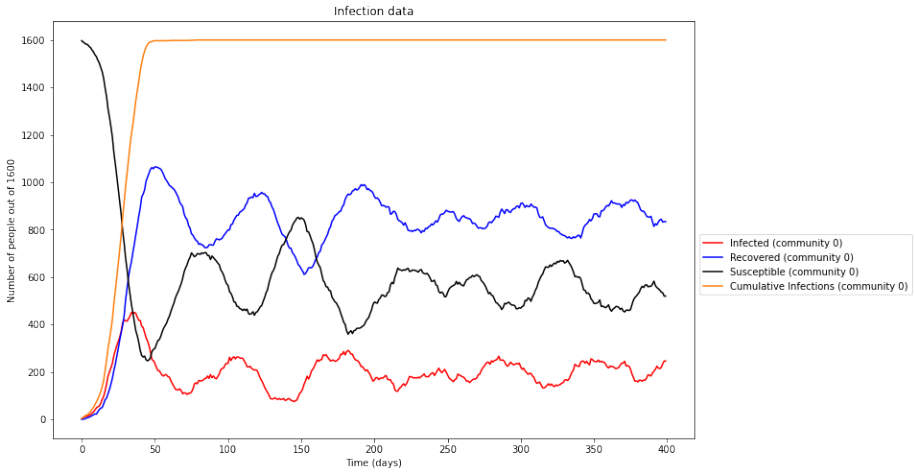


Figure 13: Grid model oscillates about equilibrium state

3.2.2 Observing effect of adding communities

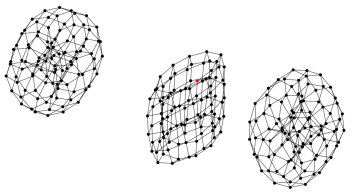
An example of the disease spreading between communities is shown in figure 14. Notice that after each community gets its first infected node, the dynamics of the community is very similar to that of a single community system. In particular, each community evolves to oscillate about the same steady state - as show in figure 15.

3.3 Results

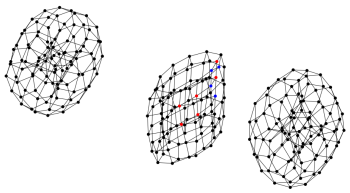
3.3.1 One community statistics

Figure 16 summarises the data gathered for one community. Note that the values plotted are averages over 200 simulation for each parameters, and the error bars indicate standard deviations of the data. Note that some data points are missing, this corresponds to the fact that for the given parameter values, the population died out every time before the average could be taken (disease free state). For example, figure 17f shows the average characteristic time as p_r is varied, but cuts off around p_r=0.65. This means that for $p_r \geq 0.65$ the disease dies out before it reaches a cumulative number of infections greater than 36% of the whole population - this makes sense since infected individuals recover so fast that they can not infect enough people to keep the disease spreading.
Notice the left hand column of figure 16 shows the equilibrium values of each of the compartments, with each figure showing the effect of varying one of p_i, p_r, or p_w. Most of these results are straight-forward to interpret. For example, increasing infection probability results in

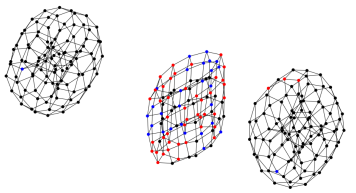
1. A decrease in equilibrium number of susceptible, since higher probability susceptible individuals will be infected and leave S compartment.



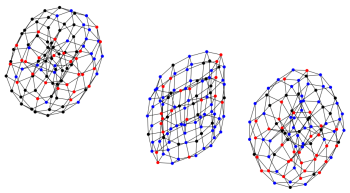
(a) t=1



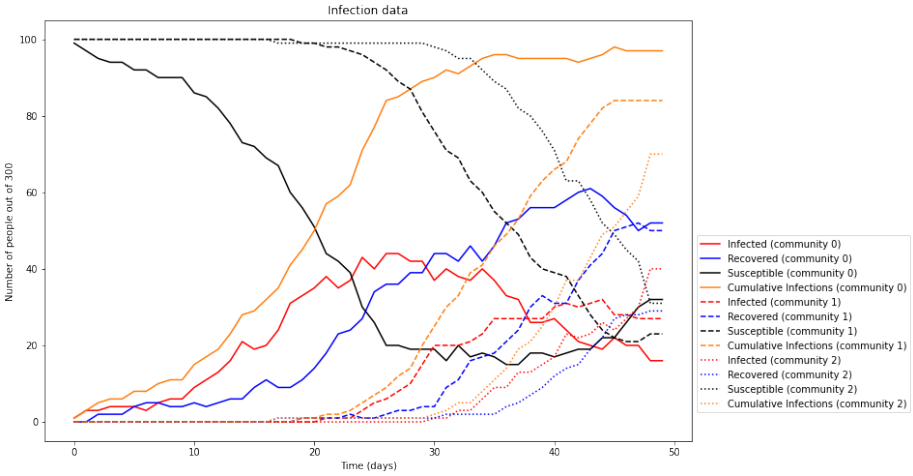
(b) t=10



(c) t=25



(d) t=50



(e) Plotting infection data from t=0 to t=50. (p_i, p_r, p_w)=(0.15, 0.1, 1/30

Figure 14: Virus spread through 3 communities of grid size 10x10. Black nodes show susceptible, red infected, and blue recovered

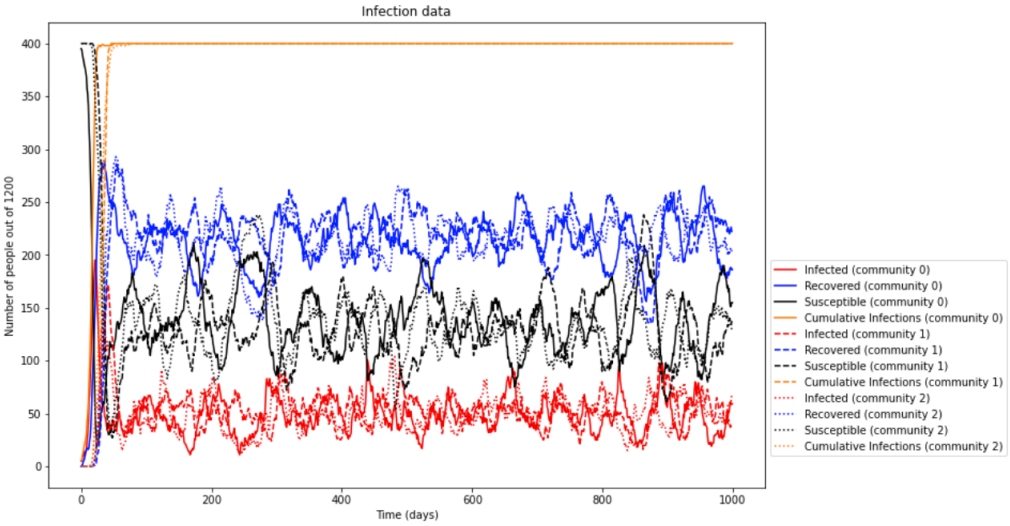


Figure 15: All communities reach same equilibrium after some time period

2. A increase in number of infected, opposite effect to S compartment.

3. A increase in number of recovered, since more infected individuals mean more nodes will have to pass through the recovered state (which is the longest of all the compartments by default, hence the large increase).

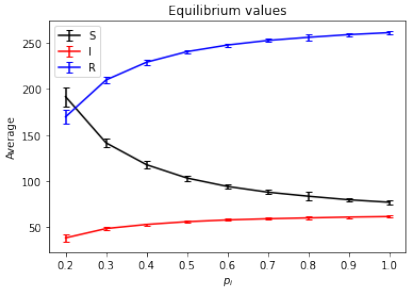
Each figure in the right column of 16 shows how a single statistic varies for each of the parameters p_i, p_r, and p_w. Note that these statistics do not have as good convergence as the equilibrium values, and the standard deviations tend to be higher - showing that there is lots of variation between simulations (unlike equilibrium numbers which stay pretty much the same each simulation). Again, most of these statistics are easily understandable.
For example, the peak number of infected individuals

1. Increases as infection probability increases

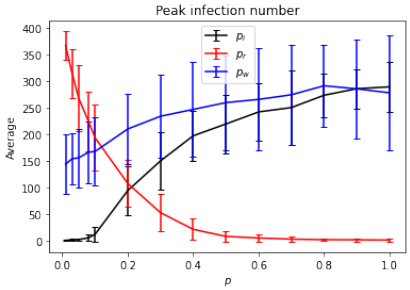
2. Decreases with recovery rate, since many become recovered before peak infection time

3. Increases (moderately) with waning infection probability, since more individuals have time to go through a cycle of $S \to I \to R \to S \to I$ before peak infection time

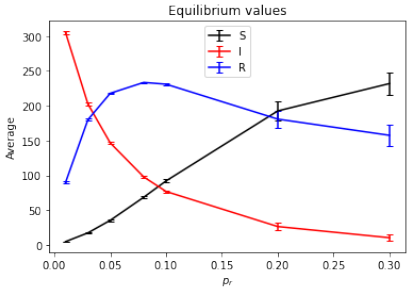
The most interesting observation from these results, is the sharp jump in cumulative number of infections as infection probability is increase beyond $p_i = 0.1$. This suggests $p_i = 0.1$ is some critical value, after which point some kind of percolation occurs where the disease dynamics undergoes a transition from a state where the disease does not spread far in the network before it dies out, to a



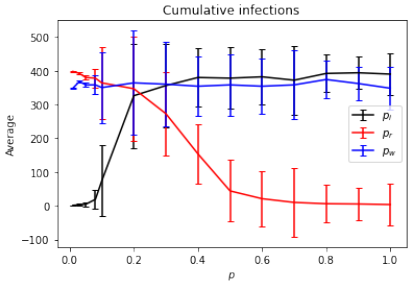
(a) Varying infection probability



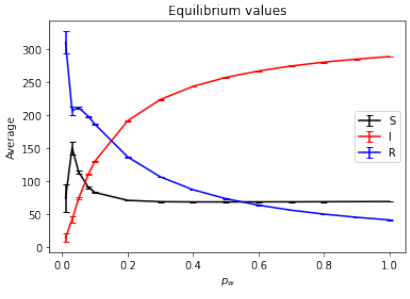
(b) Peak number of infections



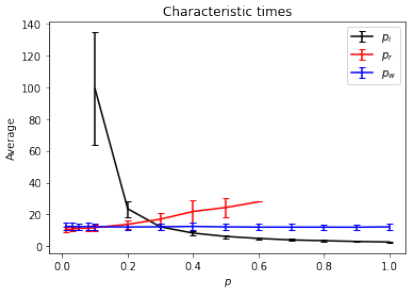
(c) Varying recovery probability



(d) Cumulative infections



(e) Varying waning immunity probability



(f) Characteristic times

Figure 16: Infection data for one community

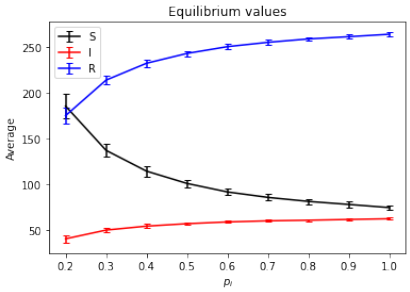
state where the disease widely spreads infecting the majority of the network. In section 4.1 of the appendix, we use concepts from percolation theory to provide a theoretical justification for this behavior.

3.3.2 Four community statistics

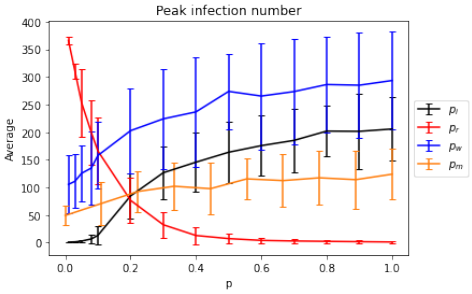
Figure 17 summarises the data collected for four communities, where the population as a whole is considered - and the additional parameter p_m (migration probability) is varied. In addition, figure 18 shows the data where each community was tracked separately.
Unsurprisingly, the equilibrium behavior is almost identical for the whole population as it was for a single community (as predicted), and the the other statistics measured for one community also match for the considered parameters.
The result of varying migration rate is qualitatively very similar to the effect of increasing infection probability. This makes sense, more migration means more chance for infected nodes to migrate from a region of many infected nodes to a node with few infected neighbours - causing an increase in chance of newly infected individuals.

3.3.3 Time when each community becomes infected

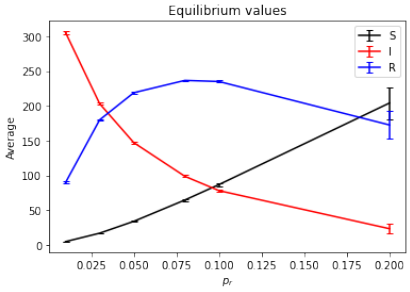
Figure 18 shows the effect of varying each of the parameters on the time when each successive community first becomes infected. As either migration rate or infection probability is increased, the time when each successive community becomes infected decreases (seemingly as an exponential decay), varying waning immunity rate has little effect. The results of varying recovery rate appear wrong on first inspection, where we might expect that increasing p_r will always increase time between when communities first become infected. I believe the problem is my code only accounts for simulations where a community actually becomes infected at least once - but as p_r becomes large the virus dies out most of the time, leading to a biased sample where the virus spreads quickly through the network.
Strangely, the simulation shows that the time between the first community being infected (at t=0), to the next community being infected, is (approximately) the same as the time between the second community becoming infected and the third (ditto between third and fourth). This is really strange and seems wrong, but I was unable to detect anything wrong with the simulation. Although, the high variance on the fourth community shows that there is a high variation between each simulation, this could explain the counter-intuitive results.



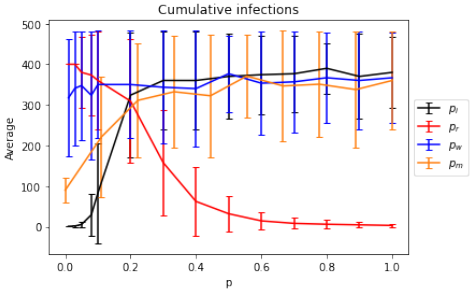
(a) Varying infection probability



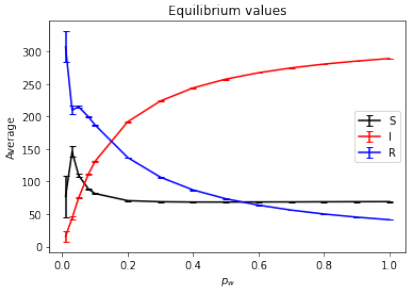
(b) Peak number of infections



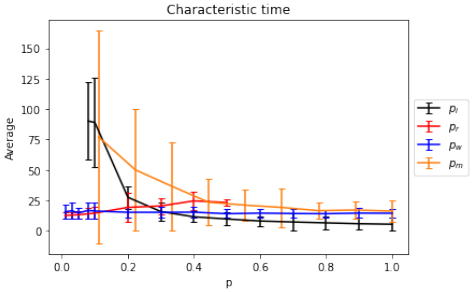
(c) Varying recovery probability



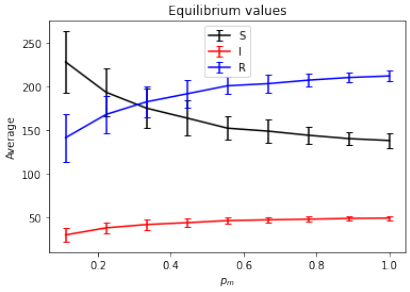
(d) Cumulative infections



(e) Varying waning immunity probability

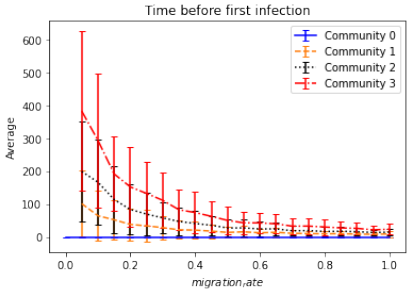


(f) Characteristic times

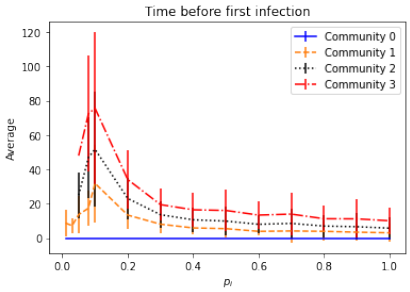


(g) Varying migration probability

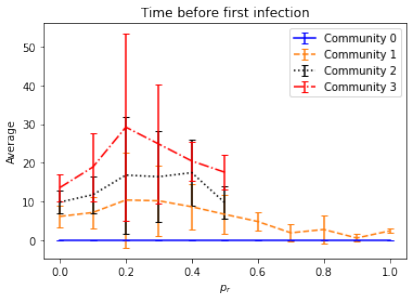
Figure 17: Infection data for four communities



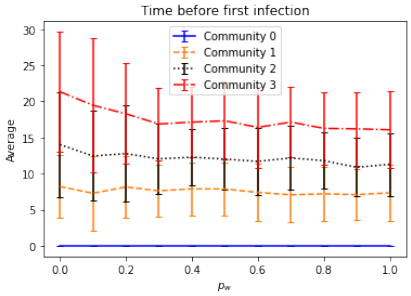
(a) Varying migration rate



(b) Varying infection probability



(c) Varying recovery probability



(d) Varying waning immunity probability

Figure 18: Time before each community gets infected - as a function of migration probability, infection probability, recovery probability, and waning immunity probability

4 Appendix

4.1 Percolation

We can theoretically justify the observed percolation behavior in the spreading of the disease in the network by reinterpreting how diseases spreads on a network. Instead of thinking of the disease spreading through the network in time, with infected node i spreading disease to susceptible node j at time t with some probability $p = p(p_i, p_r)$, consider a static situation where disease spread through the network is determined probabilistically at time t=0. Determining whether node i can infect j (given i becomes infected, and j is not already infected) can be determined at t=0, and we represent node i infecting node j with an edge connecting node i to node j - this creates a network where walks along connected nodes represents disease flow. If there is a path from the initially infected node to a given node, that node will be infected at some point in the simulation - so the cumulative number of infections within the network can be determined immediately by tracing the connected component the initial node is apart of. From this point of view we can borrow concepts from percolation theory to determine how much of the network will be infected - and determine the critical infection probability at which the cumulative infection number over the spread of the disease will dramatically increase.
It is well known that the (bond) percolation threshold on a square lattice - which is the network the model is running on - is $p_c = \frac{1}{2}$ [1]. So if we can find the probability p a given infected node infected a given susceptible neighbour, we can find the critical value of p_i at which the disease begins rapidly spreading through the network.
Each day the probability the infected node i infects a neighbouring susceptible node j is p_i, over the n days i is infected the probability becomes $1-(1-p_i)^n$, the expected number of days i remains infected is related to the recovery probability by $\frac{1}{p_i}$. Thus we approximate probability $p = p(p_i, p_r)$ that the node i infects j (excluding possible infections from other nodes) as

$$p(p_i, p_r) = 1 - (1 - p_i)^{\frac{1}{p_r}} \tag{9}$$

Setting equation (9) equal to the critical value for bond percolation in a square lattice $p_c = \frac{1}{2}$ we arrive at the relation

$$p_i = 1 - 2^{-p_r} \tag{10}$$

Using the default value $p_r = 1/7$ we find the critical value for p_i to be approximately $p_i \approx 0.1$. This percolation can be seen in figure 19, where at the point $p_i = 0.1$ the graph abruptly undergoes a massive increase in number of cumulative infections. Thus, $p_i = 0.1$ represents a transition point in the dynamics of the disease spread, from when the disease usually quickly dies out, to when the disease spreads throughout the whole network and infects the majority of the nodes.

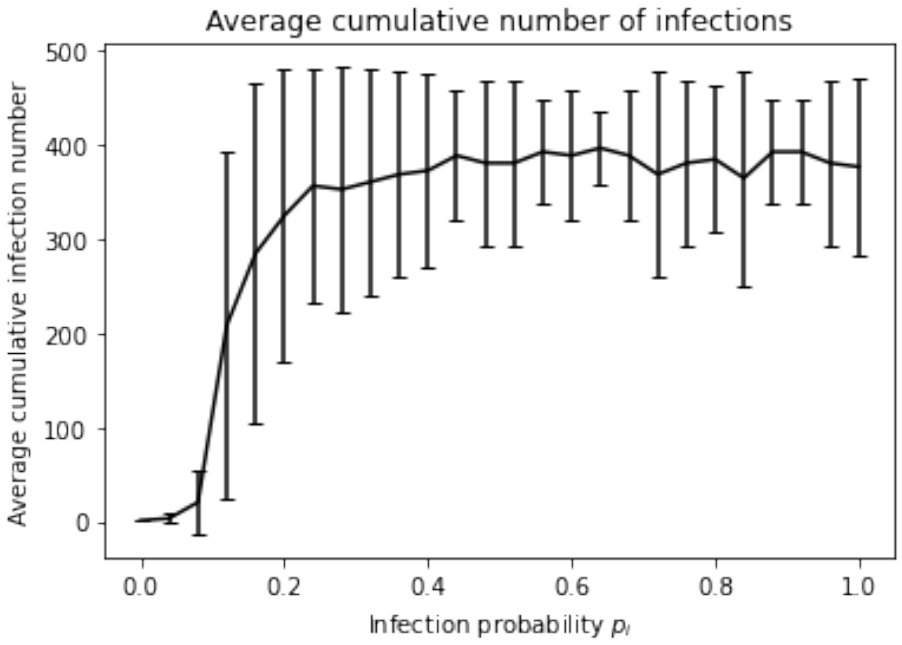


Figure 19: Demonstrating percolation threshold of $p_i = 1/2$. Notice sharp jump in Average cumulative number of infections at $p_i = 1/2$.

References

[1] Harry Kesten et al. The critical probability of bond percolation on the square lattice equals 1/2. *Communications in mathematical physics*, 74(1):41–59, 1980.

[2] Arthur AB Pessa and Haroldo V Ribeiro. ordpy: A python package for data analysis with permutation entropy and ordinal network methods. *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 31(6):063110, 2021.

[3] Steven H Strogatz. *Nonlinear dynamics and chaos: with applications to physics, biology, chemistry, and engineering*. CRC press, 2018.