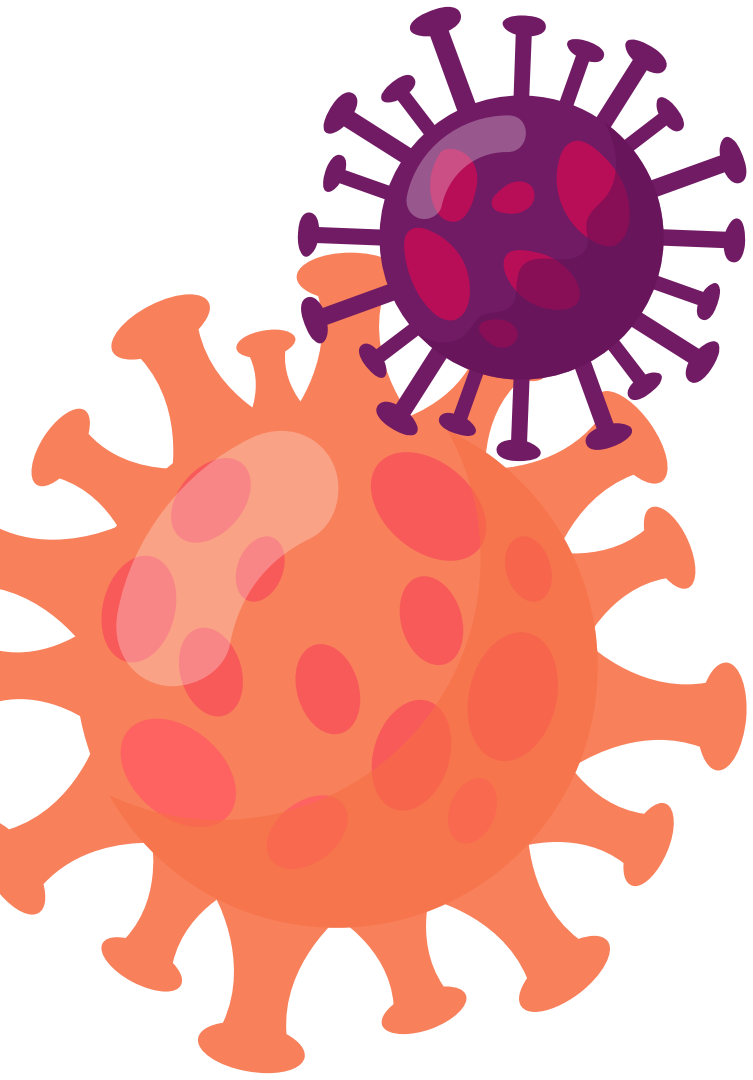
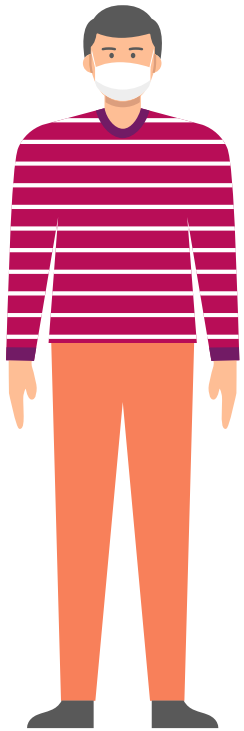


How Local Interactions Impact the Dynamics of an Epidemic

By Grant Durand, Stephen Shell, and Eashan Soni

Outline



Introduction

Models

Results

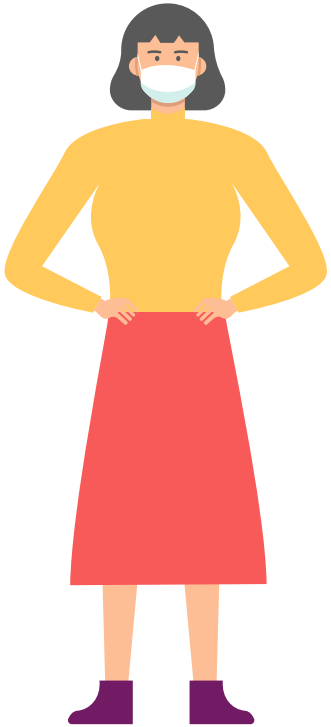
Discussion

1

2

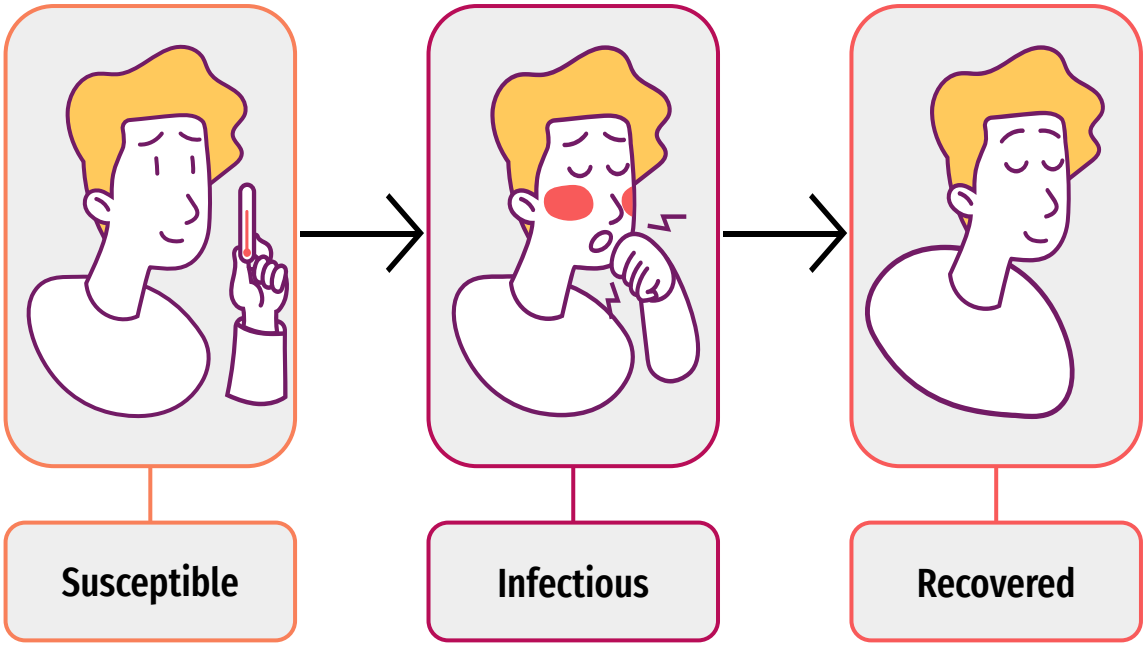
3

4



1

Basic SIR Modeling



An SIR model is a mathematical way of modeling disease spread.

In a basic SIR model, anyone can infect anyone.

STEPHEN

The “Mean-Field Assumption”

In other words, SIR models **assume** that everyone has the same **probability** of interacting with everyone as if they were mingling around the Wii Mii Plaza with **no spatial** or **social structure**!

STEPHEN

How does locality affect disease spread?

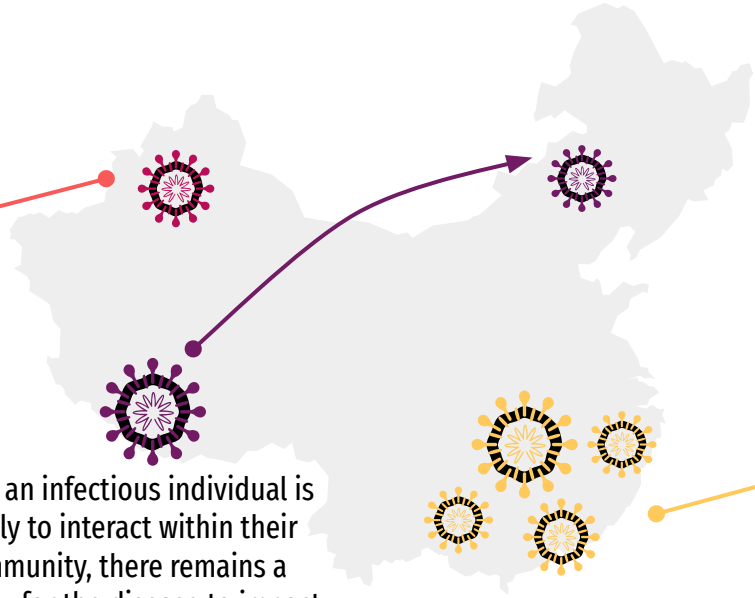
In reality, the probability of being exposed is dependent on many factors:

Quarantining

Infectious individuals are likely to quarantine or cluster with other infectious individuals, decreasing spread.

Travel

Although an infectious individual is most likely to interact within their local community, there remains a possibility for the disease to impact other communities.



Population Density

The population density of an area can drastically affect the number of interactions between infectious and susceptible individuals.

STEPHEN

Our Aim

First, we want to incorporate **locality** into the SIR model, considering **local** and **global** interactions, and **all points in between**.

Then, we want to **simulate SIR models** with locality considered.

Finally, we want to discuss ways we can practically **limit disease spread** based on our results.

2 Mean-Field Model

Since the mean-field model does not account for locality, all individuals in the population are either susceptible (S), infectious (I), or recovered (R), and we assume a constant population (no births/deaths), then the total population, N, can be written:

$$N = S + I + R$$

If we know N, S, and I, then R can be discovered. N is a constant, and we will track S and I with the following equations:

$$\frac{dS}{dt} = -\beta SI \qquad \frac{dI}{dt} = \beta SI - \gamma I$$

Here, β controls transmission rate, γ controls recovery rate, and immunity is permanent. Additionally, β is “density dependent”, which we will discuss later.

Locality

Our key modification on the mean-field model is introducing **L**, which determines the proportion of transmission that occurs **locally** or **globally**.

L ranges from 0 to 1.

As L approaches **0**, interactions become more **global**.

As L approaches **1**, interactions become more **local**.

Since L represents “locality,” 1-L is its remainder, so 1-L represents “globality.”

Density

While locality represents to the “closeness” of interactions, **density** describes the probability of an individual being either susceptible, infectious, or recovered. We will refer to these probabilities as “**singlet densities**.”

Since disease transmission requires an interaction between two individuals, we will track “**pair densities**.” Pair densities are dependent on the singlet densities of each individual involved in a given interaction.

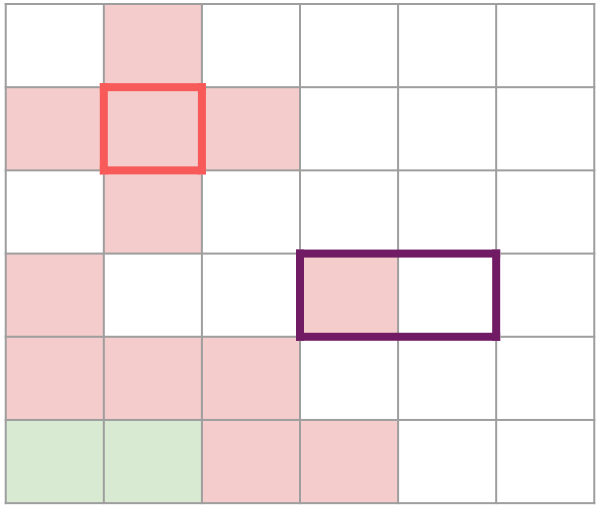
Individuals vs. Interactions

Say we have a square lattice where each cell is an individual

White = Susceptible, Red = Infected, Green = Recovered

Every **single** cell has a "**singlet density**":

P_S , P_I, and P_R



Similarly, every **pair** of **neighboring** cells has a "**pair density**":

P_{SS} , P_{SI} , P_{SR}

P_{II} , P_{IR} , and P_{RR}

Pair Approximation: singlet densities

Each cell within the square lattice has three separate probabilities which sum to 1, so we have the following equation for density:

$$P_R = 1 - P_S - P_I$$

For locality, we are interested in how close susceptible individuals are to infectious individuals, so we have these equations:

$$\text{global: } (1 - L)\beta P_S P_I$$

$$\text{local: } L\beta q_{S/I} P_I .$$

When added together, all points of locality between local and global are considered. We see that when L=1, interactions are entirely local. When L=0, interactions are entirely global.

Pair Approximation: singlet densities (cont)

This method of modeling is called a pair approximation, where all of our pair densities are dependent on our singlet densities, which are represented by the following equations:

P_S represents the probability that a site is occupied by a **susceptible** individual:

$$\frac{\mathrm{d}P_S}{\mathrm{d}t} = -\beta\left[Lq_{S/I} + (1-L)P_S\right]P_I$$

Then P_I represents the probability that a site is occupied by an **infectious** individual:

$$\frac{\mathrm{d}P_I}{\mathrm{d}t} = \beta\left[Lq_{S/I} + (1-L)P_S\right]P_I - \gamma P_I$$

Recall that these are based on the SIR equations but now with locality and density considered:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI \qquad \frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \gamma I$$

Pair Approximation: pair densities

P_{SI} Represents the probability that a randomly chosen pair of **neighbouring** cells are susceptible as well as infectious, yielding this equation:

$$\frac{\mathrm{d}P_{SI}}{\mathrm{d}t} = -\beta(L((1/4)+(3/4)q_{I/SI})+(1-L)P_I)P_{SI} - \gamma P_{SI}$$
$$+\beta(L(3/4)q_{I/SS}+(1-L)P_I)P_{SS}$$

Probability of both susceptible:

$$\frac{\mathrm{d}P_{SS}}{\mathrm{d}t} = -2\beta(L(3/4)q_{I/SS}+(1-L)P_I)P_{SS}$$

Both infectious:

$$\frac{\mathrm{d}P_{II}}{\mathrm{d}t} = -2\gamma P_{II} + 2\beta(L((1/4)+(3/4)q_{I/SI}+(1-L)P_I)P_{SI}$$

Pair Approximation: pair densities (cont)

Susceptible and recovered:

$$\frac{\mathrm{d}P_{SR}}{\mathrm{d}t} = -\beta(L(3/4)q_{I/SR} + (1-L)P_I)P_{SR} + \gamma P_{SI}$$

Infected and recovered:

$$\frac{\mathrm{d}P_{IR}}{\mathrm{d}t} = -\gamma P_{IR} + \beta(L(3/4)q_{I/SR} + (1-L)P_I)P_{SR} + \gamma P_{II}$$

Finally, the probability of neighboring cells both being recovered is represented by:

$$P_{RR} = 1 - P_{SS} - P_{II} - 2P_{SI} - 2P_{SR} - 2P_{IR}$$

As you can see, some are doubled, because, for example, an SI pair is the same as an IS pair, and we must account for every possible pair of S, I, and R.

Basic Reproductive Ratio

The reproductive ratio, R_0, represents the average number of susceptible individuals that get the disease from an infectious individual.

$R_{0,t}$	The total basic reproductive ratio of the entire SIR model.
$R_{0,l}$	The basic reproductive ratio when the interactions are fully local.
R_0	The basic reproductive ratio when the interactions are fully global.

In summary, this equation is saying: The total basic reproductive ratio is the locality factor times the fully local basic reproductive ratio in addition to the globality factor times the fully global reproductive ratio.

$$R_{0,t} = LR_{0,l} + (1 - L)R_0$$

Stochastic Simulations

Looking back on our equations, we can see our model has **constant variables** such as population size, transmission rate, and recovery rate.

But our model also has **random variables**, such as where the initially infecteds are placed within the square lattice.

This is where a stochastic simulation comes in. We used python code to **plug in random values** into these random variables, thus getting different results with every simulation repetition.

We repeated our simulation **100 times** and used only the most **average results** to construct our graphs and infer our results.

SKIP

Stochastic Simulations (cont)

We constructed a **matrix** in python to be our “square lattice.” We set the matrix rows and columns to 25, thus representing a 625 person population. We then set the number of **initial infecteds** to 5, **randomly seeding** them into the matrix.

One function **locates** the infectious individuals in the matrix and then “**infects**” the surrounding individuals. Another function **tracks** how many days have passed in the simulation and **updates** the data accordingly. For example, if the recovery rate was set to 1/14, then the code would update an “infectious” individual to “recovered” after 14 days.

The code tracks the number of infected individuals in an array and records the day of the epidemic’s **peak**. Then the code constructs **graphs** based on all the data.

SKIP

Stochastic Simulations (cont)

Main function:

- Loops through 11 values of L -> 0, 0.1, ..., 0.9, 1.0
  - Loops through 100 simulations
    - Initializes the square lattice / matrix with a preset number of randomly placed infected individuals
    - Simulates 300 days of an epidemic

```
for ll in range (0,11):     # First run sets L=LLow,
    L=ll*0.1
    print(L)
    peaks=[] # For storing peak no. of infecteds
    tots=[] # For storing total no. infected
    days=[]
    for reps in range(0,REPS):
        # Set initial conditions
        init_inf=I0
        grid=np.zeros((N,N))
        for i in range(0,init_inf):
            grid[randint(0,N-1),randint(0,N-1)]=1

        tsteps=[0]
        infecteds=[count_type(1)]
        current_t=0

        # Main run
        while current_t<300:
            # Find tau-leap
            scale=findscale()
            dt = -np.log(random()) / scale
            current_t=tsteps[-1]
```

3 Results

The following three graphs are created through finding the most central 50% of the 100 simulated curves. Below, R_0 equals 2 in graph (a), 5 in (b), and 10 in (c), drastically affecting the curves.

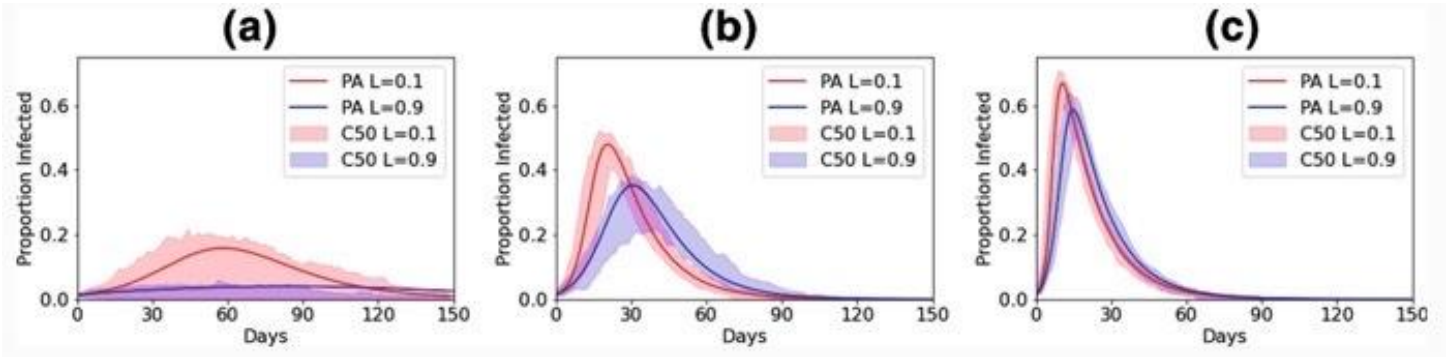
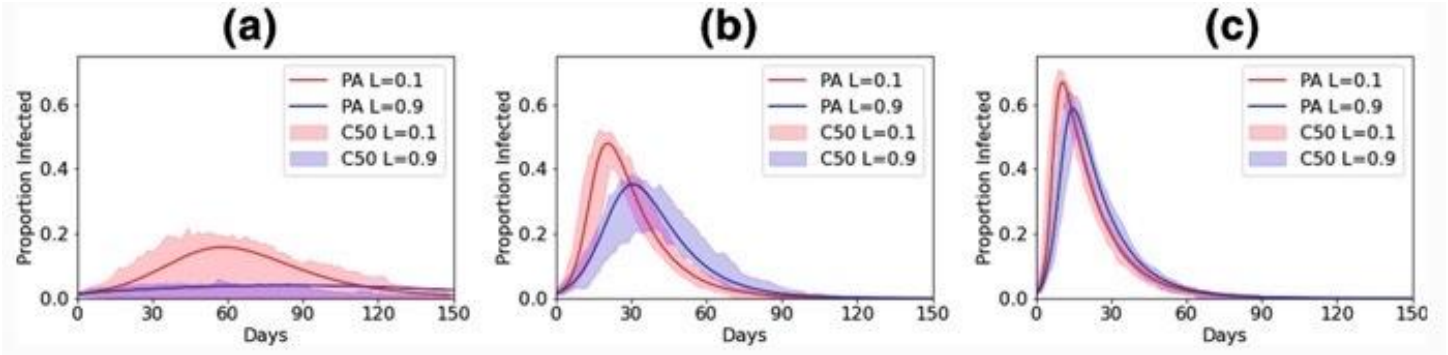


Figure 1

Results (cont)

Larger L values yield a lower and later infection peak, which means that restricting global interactions should slow down and limiting the spread of an epidemic.

Large values for R_0, on the other hand, not only yields a higher and sooner infection peak, but it also makes it more difficult to distinguish the local curve from the global curve.



Results (cont)

Row 1: percentage of population infected at the epidemic's peak

Row 2: the day of the peak

Row 3: percentage of population infected overall

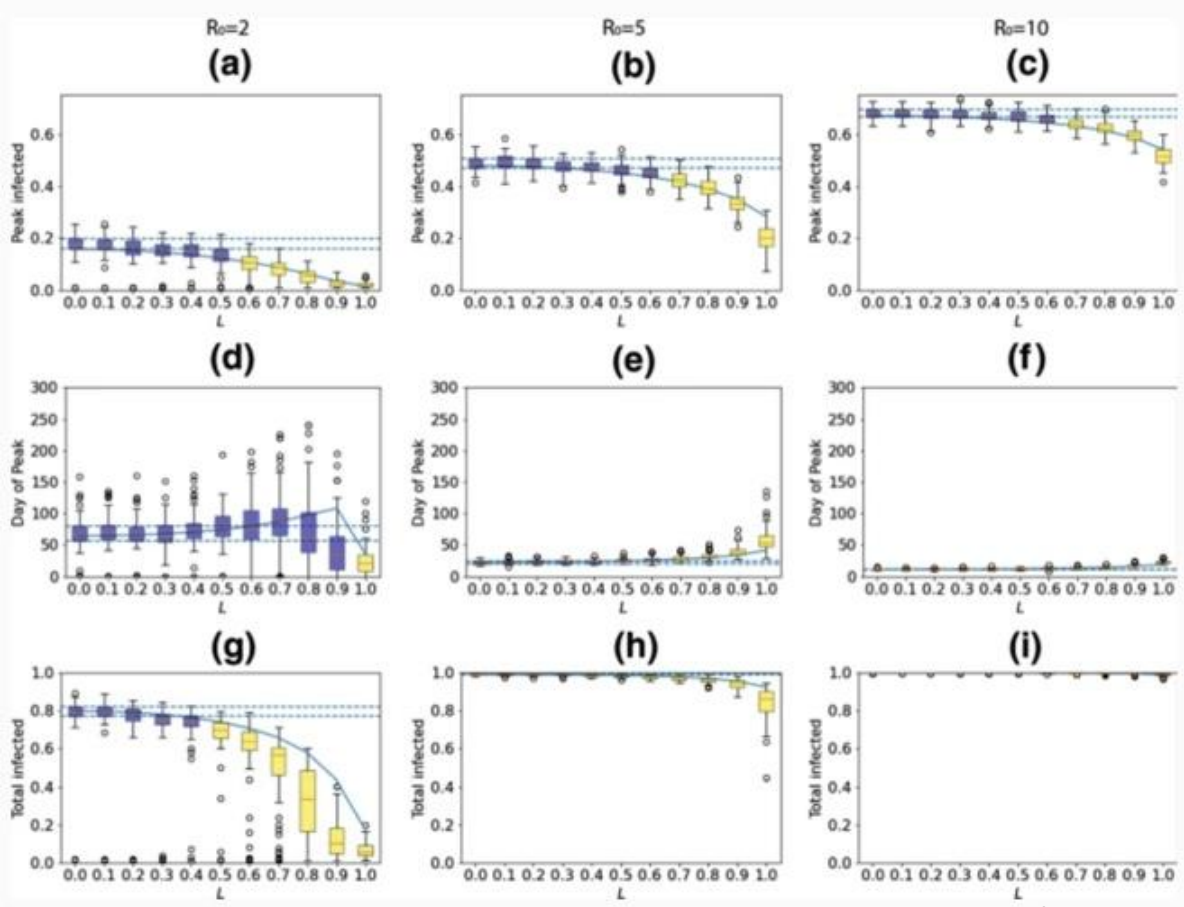
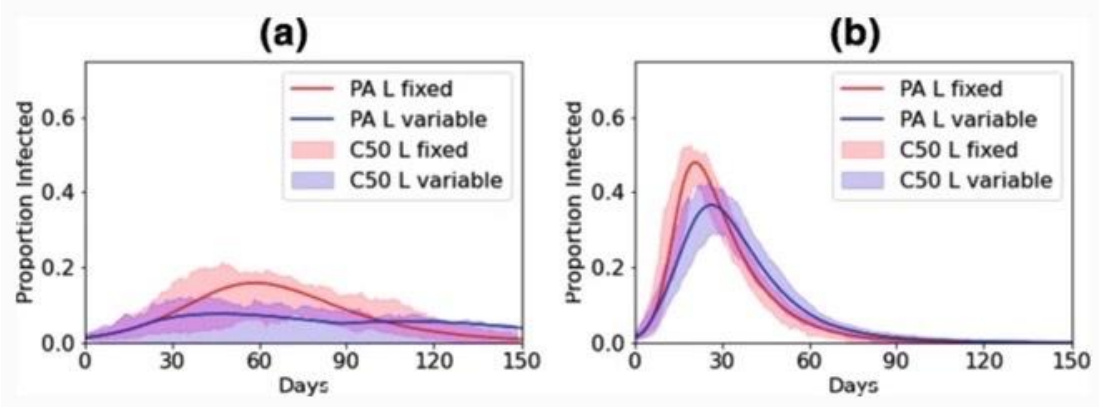


Figure 2

Results (cont)

If we are to restrict movement were able to create a more localised interaction meaning that L is > .5, such as if there were a restriction imposed on a population.

In figure 3 we see what exactly happens to an epidemic when we restrict movement such that the red represents a predominantly global whereas the blue represents when movement is restricted.



Results (cont)

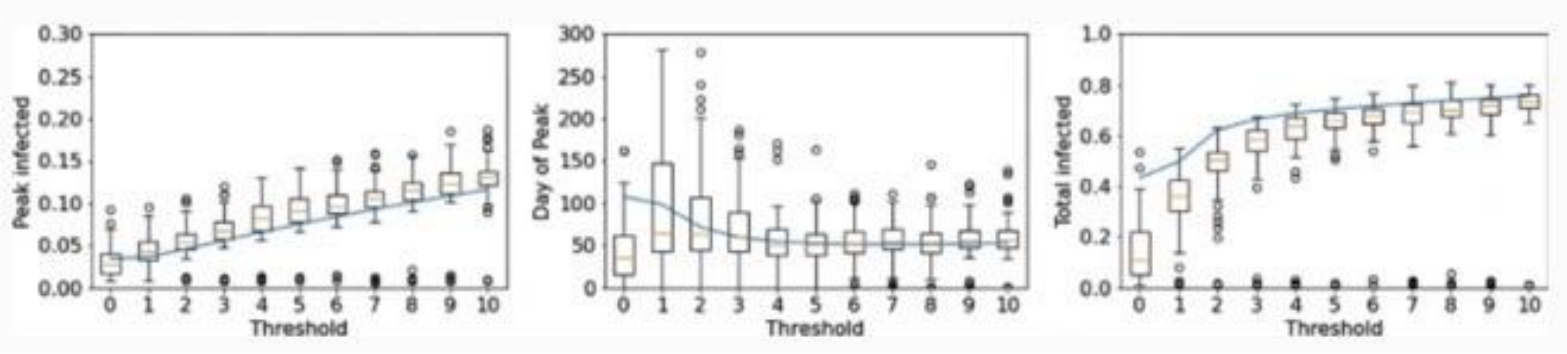


Figure 4

When we change the threshold of interactions from when L=.1 to L=.9 when R_0 is 2, we can see that impact greatly changes the total infected.

Results (cont)

To represent how the correlation between S and I sites on the lattice we've created the formula:

$$C_{SI} = \frac{P_{SI}}{P_S P_I} = \frac{q_{S/I}}{P_S}$$

This formula represents how likely it is to find S sites next to I sites.

When interactions are local we created a formula for the correlation between S and I sites which approach an quasi-equilibrium:

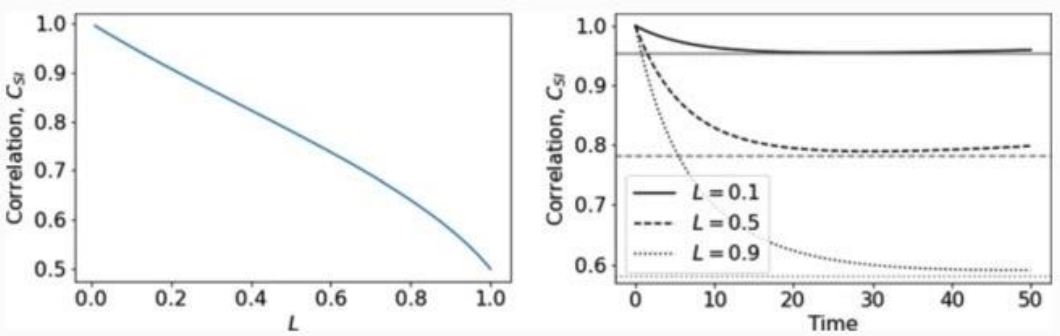
$$\hat{C}_{SI} = \frac{3L - 2 + \sqrt{-7L^2 + 4L + 4}}{4L}$$

Results (cont)

In the two graphs we see that when we increase L it leads to a much stronger S-I correlation, which means that I individuals are more likely to be located near other I individuals.

If an epidemic were to occur in the predominantly local interactions the lattice would quickly become correlated and the local clusters of infection falls as the availability of people to be infected is lessened.

Figure 5



STEPHEN

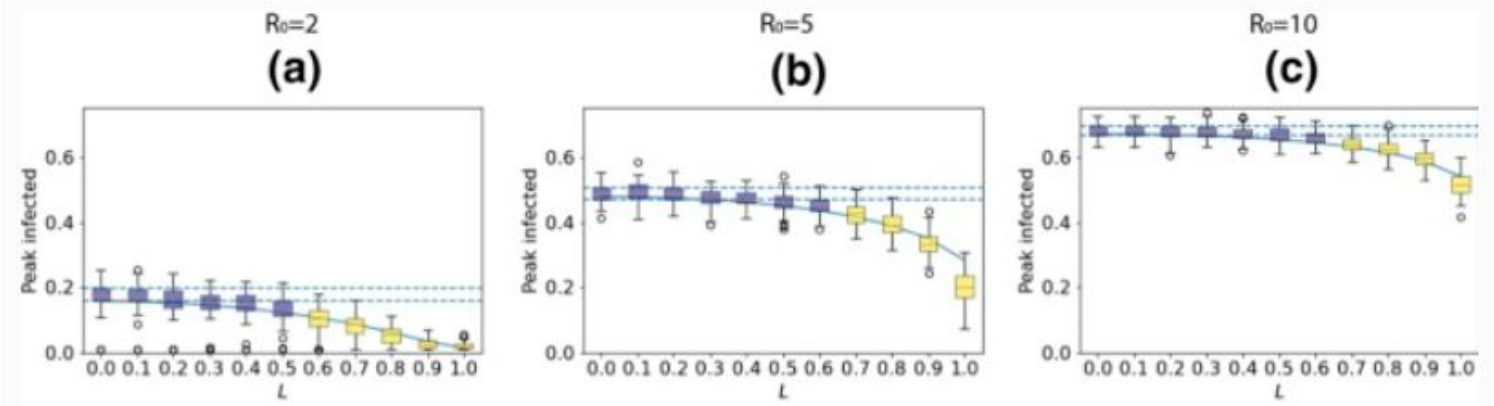
4 Discussion

The goal of the models was to explore just how epidemics over short timescales can be restricted by different degrees of local interactions.

Our most obvious discovery was that predominantly local interactions will result in fewer infections than predominantly global interactions, so limiting global interactions is the key to limiting disease spread.

Discussion (cont)

Our most interesting discovery was that the peak infection did not differ much between values of **L less than 0.5**. Even at relatively low proportions of global interactions, enough long-range infections can occur in the early stages of an epidemic to **seed large numbers of local epidemics**, allowing the infection to spread throughout the population.



Application

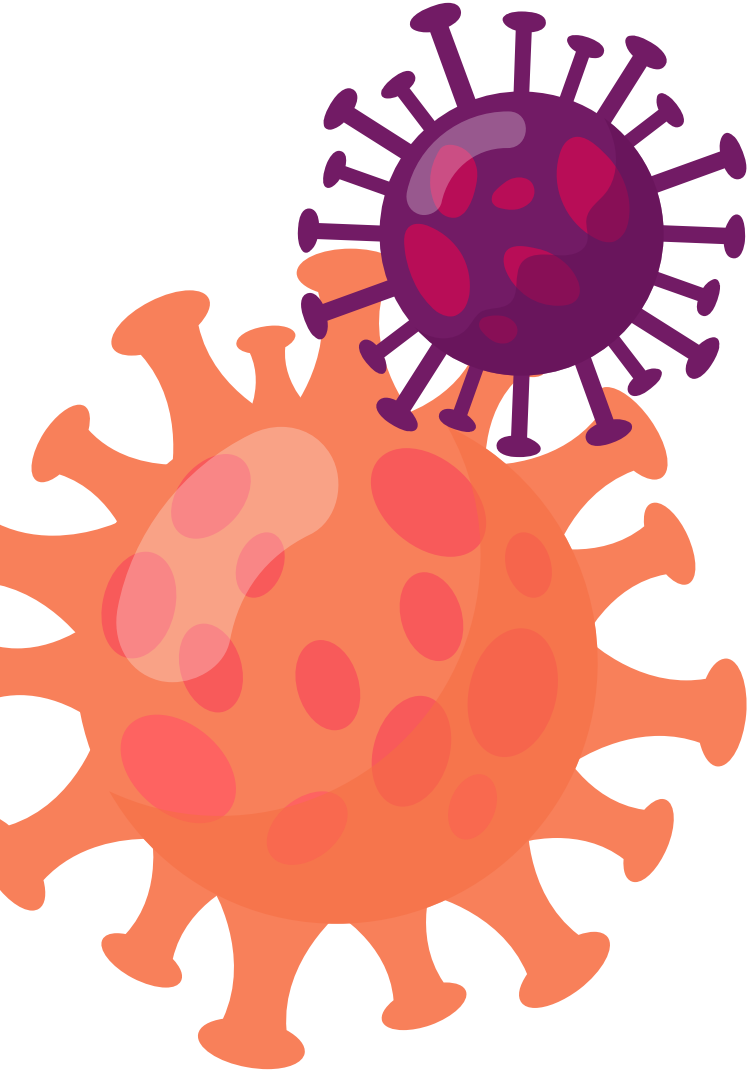
If we know that a population starts by having predominantly **global** interactions, movement restrictions must be imposed as **early** as possible, or else later movement restrictions will have minimal effect.

In this case, the disease has already spread randomly throughout a population, seeding a large number of local outbreaks, so movement restrictions will only protect the few remaining uninfected local communities.

Conclusion

After all the findings we are able to draw clear and abstract conclusions about what can happen in short term epidemic models, but we should understand that we cannot use this data and findings to be an accurately predictive model for a particular epidemic.

In our paper we plan to extend our findings by investigating simulations with different values for population size, transmission and recovery rates, and initial infecteds.



Q’s?

Thanks for listening!

Sources

Article: https://link.springer.com/article/10.1007/s11538-021-00961-w#citeas

Code: https://github.com/abestshef/latticeSIR

Results (cont)

In *Figure 2*, we see that in graphs (d)-(f), the number of days until the peak increases as we increase L, since less global interactions slow down the pace of the epidemic.

However, there is an exception in (d) when R_0 is 2 and L is large. This is because a disease with a low reproductive ratio and predominantly local interactions, will be very contained and end very quickly. Thus, the peak will be low and early.

Results (cont)

In figure 3 you can see a slight second wave which represents when restrictions are lifted, as it was determined that these restrictions wouldn't allow much more positive results compared to a restrictionless population.

In both graphs we can see that when there is a restriction of movement the epidemic isn't as bad when there aren't restrictions, this allows for a better ability to control the epidemic.