

Modeling the Spread of Epidemics Thorough Network Simulation

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This manuscript was compiled on May 28, 2020

In this article, we simulate and examine the spread of infection on random networks as well as a real life social network of interactions in a school, while adjusting and accounting for disease spread parameters like infection rate, contact rate, and mortality. Our general method of simulation utilizes a breadth-first algorithm that cause someone to possibly transmit the disease to their neighbors. We also incorporated variables such as nodes "quarantining" themselves and not interacting with their neighbors. *Put conclusions, results, data, analysis, etc... after we actually run significant simulations.*

simulation | networks | epidemic | disease transmission |

The spread of epidemics can be modeled through simulations on various networks. These models are valuable tools in assessing the appropriate responses and countermeasures to a pandemic. To give a simple view of how a disease can spread, we simulate an arbitrary epidemic over a social network of a school as well as a random network. We concentrate on the impact of a proportion of people quarantining themselves. We also simulate the spread over a number of days to analyze both short term and long term effects that our variables can have.

Results

Seed selection based on Centrality. We observed total infected populations and deaths starting at seed nodes of differing centrality in order to compare infection outcomes. These simulations were conducted on various quarantine rates and averaged over 15 realizations. In our school network (1), there are 236 total nodes.

For non-trivial quarantine rates, we observe that starting the infection with a seed (patient zero) of higher centrality will lead to a greater number of total infections as well as a greater number of deaths.

Table 1. Comparison of infected populations and deaths based on closeness centrality

$N = 236$ Quarantine Rate ψ	Total Deaths			Total Infections		
	Minimum Closeness	Median Closeness	Max Closeness	Minimum Closeness	Median Closeness	Max Closeness
0.0	97.5333	97.2	97.3333	236	236	236
0.25	26.8	38.4667	50.1333	53.8	106.3333	118.7333
0.5	3.8667	6.9333	16.6667	8.9333	21.1333	33.8667
0.75	1.8667	3.7333	5.6	3.4667	7.7333	14.4

Significance Statement

The global pandemic of COVID-19 has sparked a public discussion on the spread of epidemics and "flattening the curve". We can draw some important conclusions from modeling the spread of such diseases through network simulations. These simulations can also provide us with insights into the importance of disease parameters as well as various interventions such as social distancing and self-isolation, and how these variables affect the rate at which a disease can spread. With these results, we can better inform the reasoning behind some of the drastic social policies implemented during this situation.

Include

Table 2. Comparison of infected populations and deaths based on betweenness centrality

$N = 236$ Quarantine Rate ψ	Total Deaths			Total Infections		
	Minimum Bet.	Median Bet.	Max Bet.	Minimum Bet.	Median Bet.	Max Bet.
0.0	98.4	96.7333	100.4667	236	236	236
0.25	23.8	33.4667	49.6667	53.8	106.3333	118.73333
0.5	3.3333	8.8	18.2	32.2665	21.0667	8.0667
0.75	2	4.2	7	14.4667	6.9333	4

Table 3. Comparison of infected populations and deaths based on PageRank (PR) centrality

$N = 236$ Quarantine Rate ψ	Total Deaths			Total Infections		
	Minimum PageRank	Median PageRank	Max PageRank	Minimum PageRank	Median PageRank	Max PageRank
0.0	100.1333	98.2667	98.8	236	236	236
0.25	17.1333	38.1333	50.2	59.8667	85.8	123.7333
0.5	2.6	7.6	17.8	6.8	20.4667	38.9333
0.75	1.4667	3.1333	5.5333	3.6	8.4	13.8667

We can also proceed to observe trajectories over time of the compartments we considered in our model.

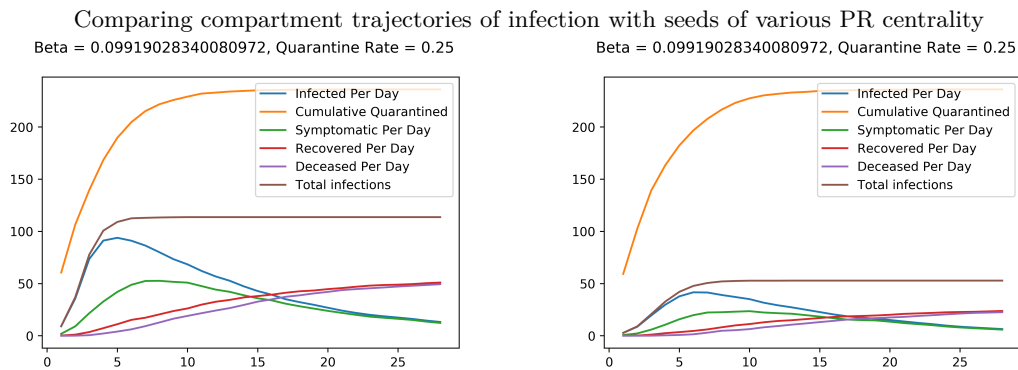


Fig. 1. The graphs above depict compartmental trajectories of infection spread for the maximum (left) and minimum (right) PageRank centrality seed nodes with a quarantine rate of 0.25. We see a significantly larger spike in number of infections and deaths in the maximum centrality infection compared to the minimum centrality infection.

Optimal parameters for Cost Function.

Discussion

Centrality of patient zero influences infection spread.

In cases with non-trivial quarantine rates, we found that if the disease progression begins with a node of higher centrality, the disease will have a higher toll in terms of deaths and total infections. This aligns with theories from current research describing "super-spreaders", or individuals who have greater contact with others, and their increased propensity to pass the disease on (2).

It is important to note that for the cases with no quarantine ($\psi = 0$), the impact of the different centralities of patient zeroes is not as pronounced. Given the relatively large timescale of this configuration on a network of this size, the distinction between infections with low and high centrality seed nodes is obscured by the rapid and unimpeded spread of the virus. If we were to look at a smaller timescale, the significance of the different centralities would once again be more visible.

Understanding node centralities, or colloquially, the importance of an individual in a social network, can be a predictor that can give an early glimpse into how an infection may progress throughout a population.

Additionally, observing that with no quarantine deaths and infections are at their highest points suggests that quarantining to some degree is important. We explore this phenomenon in our parameter scanning analysis.

Optimal parameters for Cost Function. We are currently working on this part of our project.

Methods

The Model.

In considering the status of each node, we have created compartments that each node can be classified into; these compartments include: susceptible, infected but asymptomatic, infected and symptomatic, quarantined, recovered, and deceased.

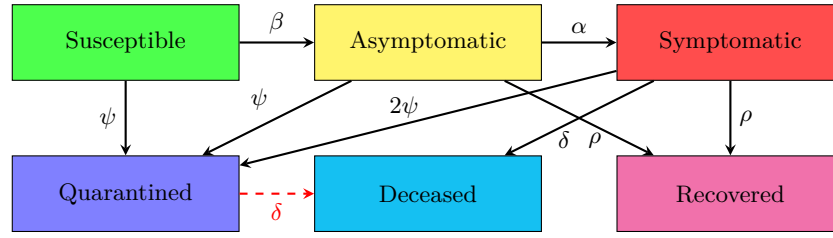


Fig. 2. Schematic diagram of our compartmental model and probabilities of flows between compartments.

We simulate the spread of the disease using a modified breadth-first traversal from every infected node (initially from the Patient Zero). Each infected node infects its susceptible neighbors with rate β . The infection rate is identical between symptomatic and asymptomatic nodes, as each edge represents an interaction that would be sufficient for the possible transmission. This is further compensated by the fact that symptomatic nodes have a higher rate of self-quarantining, and it is also consistent with the network design in *High-Resolution Measurements of Face-to-Face Contact Patterns in a Primary School.*, where they maintained a threshold time for an interaction to have infectious potential. (1).

Every node has a chance of quarantining themselves at a rate ψ , or if they're symptomatic, 2ψ . Quarantined nodes are removed from the network, they cannot pass the disease or get infected - but if they were already infected by the time they went into quarantine, they still have a chance to develop symptoms, recover, or die. Similarly, deceased nodes are ignored from the network. Recovered nodes remain in quarantine, as we did not want to make assumptions about immunity or infectiousness or recovered patients.

Every node is assigned a symptom onset, recovery, and death rate randomly chosen from Gamma distributions $s \sim \Gamma(5.81, 0.95)$, $r \sim \Gamma(8.16, 0.33)$, $d \sim \Gamma(4.94, 0.26)$ respectively (3, 4). These rates were approximated using early COVID-19 data, and represent average times for symptom onset (6.11 days), recovery (24.7 days), and death (18.8 days). These randomly assigned rates are meant to reflect the different experiences of dealing with the virus across health, and age ranges.

Algorithm 1 Daily infection crawl through the network

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At the start of each day, the infection starts spreading from all the infected people in the queue while queue is not empty do
  For each neighbor of the node at the top of the queue if this neighbor is alive, has never been infected, and is not quarantined then
    | Infect them with probability  $\beta$  and if infected, add them to the queue
  end
end

At the end of each day for every node in the network do
  | Quarantine the node with probability  $\psi$  or  $2\psi$  if they're symptomatic
end

for every infected node do
  if Asymptomatic then
    | recover or develop symptoms at a unique rate
  else
    | recover or die at a unique rate
  end
  if Alive, still infected, and not quarantined then
    | put them back in the queue
  end
end

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Seed selection for centrality analysis.

For this analysis, we used an infection rate $\beta = 0.099$, which was calculated using empirical data regarding COVID-19's spread (5). We evaluated an estimate for β as $R_0(\tau)^{-1}$ where τ was the average recovery time.

We retrieved values for three different centrality measures on our network: closeness centrality, betweenness centrality, and PageRank. These values were then sorted, and we selected the nodes with the minimum, median, and maximum centrality values to be seed nodes (patient zero) for our simulations.

Every simulation was run for 15 iterations, and results were averaged.

Parameter scanning and cost function.

We're currently working on this part of our project. As a rough idea, we plan on creating a cost function in which every node will contribute some sort of value. Death of a node, or infected quarantine would remove or contribute negative value to our network. Ultimately, we would like to determine, for a given infection and recovery rate, what is the optimal rate of quarantine so as to maximize network value and minimize death and infection.

ACKNOWLEDGMENTS. We would like to acknowledge Dr. Heather Zinn-Brooks for her guidance throughout this project as well as Dr. Leif Zinn-Bjorkman for teaching some of the authors about ordinary differential equations.

1. J Stehlé, et al., High-resolution measurements of face-to-face contact patterns in a primary school. *PLOS ONE* **6**, e23176 (2011).
2. A Gómez-Carballa, X Bello, J Pardo-Seco, F Martínón-Torres, A Salas, The impact of super-spreaders in COVID-19: mapping genome variation worldwide. , 2020.05.19.097410 (year?) Publisher: Cold Spring Harbor Laboratory Section: New Results.
3. SA Lauer, et al., The incubation period of coronavirus disease 2019 (COVID-19) from publicly reported confirmed cases: Estimation and application. **172**, 577–582 (year?) Publisher: American College of Physicians.
4. R Verity, et al., Estimates of the severity of coronavirus disease 2019: a model-based analysis. **0** (year?) Publisher: Elsevier.
5. S Sanche, et al., Early release - high contagiousness and rapid spread of severe acute respiratory syndrome coronavirus 2 - volume 26, number 7—july 2020 - emerging infectious diseases journal - CDC. (year?).