

Supplementary material to accompany:

Heterogeneity and Network Structure in the Dynamics of Diffusion: Comparing Agent-Based and Differential Equation Models

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This supplement documents the DE and AB models and results, including the construction of the different networks for the AB simulations, sensitivity analysis, the calibration process and results, and instructions for simulating the model and replicating the results. The model, implemented in the Anylogic simulation environment, is available with the online material:

<http://web.mit.edu/hazhir/www/research.html>

Formulation for the Agent-Based SEIR Model

We develop the agent based SEIR model and derive the classical differential equation model from it. Exhibit 1 shows the state transition chart for individual j . Individuals progress from Susceptible to Exposed (asymptomatic infectious) to Infectious (symptomatic) to Recovered (immune to reinfection). In the classical epidemiology literature the recovered state is often termed “Removed” to denote both recovery and cumulative mortality.

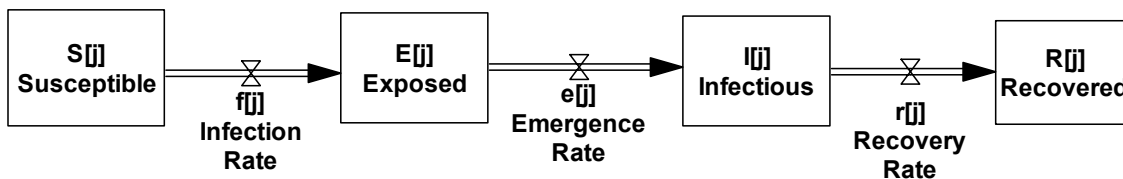


Exhibit 1. State Transitions for the AB SEIR model

The states SEIR are mutually exclusive; each agent in the AB model can only be in one of the states. We first derive the individual Infection Rate $f[k]$ (the hazard of infection for a susceptible individual), then the Emergence and Recovery Rates $e[j]$ and $r[j]$ for individuals in the exposed and infectious states.

Infection Rate: For infection to occur Susceptible individuals must come into contact with either an Infectious or Exposed individual. Some of these contacts result in disease transmission.

Disease transmission (the transition from S to E) for susceptible individual k occurs if, first, the individual comes into contact with an individual j in either the E or I state, and second, that contact results in transmission of the disease. The probability of contact between individual j and k in a short interval of time, dt, is denoted $C_{dt}[j,k]$. The probability of transmission from j to k given such a contact (infectivity) is denoted i_j .

Contacts between individuals occur with probabilities conditioned by the network of relationships in the population. Assuming contacts within the relationship network are independent events, the expected individual Infection Rate, $f[k]$, can be found by summing the probabilities of contact with each individual in states E and I and finding the infectious contact hazard by taking the limit of the sum of infectious contact probabilities as the time interval dt approaches zero:

$$f[k] = \lim_{dt \rightarrow 0} \left(\sum_{j \in E \cup I} C_{dt}[j,k] \cdot i_j \right) / dt = \sum_{j \in E \cup I} \left(\lim_{dt \rightarrow 0} (C_{dt}[j,k] \cdot i_j / dt) \right) = \sum_{j \in E \cup I} (Z[j,k]) \quad (1)$$

In the following section we find the expected infectious contact rate between individuals j and k, $Z[j,k]$, by deriving the formulations for its components based on the implementation of the AB model in the paper.

In the agent-based model, contacts between each E or I individual and other individuals in the population occur through the network of relationships between them. Infection can only occur when these contacts are with an individual in state S. Therefore, $C_{dt}[j,k]$ is composed of two components: (1) The event that an individual j in the state E or I ($R_{dt,n}[j]$) has (n) contact(s), within dt, and (2) The probability that any of these (n) contacts is with individual k in the state S ($P[j,k]$):

$$C_{dt}[j,k] = \sum_{n=1}^{\infty} R_{dt,n}[j] \cdot (1 - (1 - P[j,k])^n) \quad (2)$$

Contacts for an E or I individual are assumed independent and therefore have a Poisson distribution with parameter $L[j]$:

$$R_{dt,n}[j] = \frac{(L[j] \cdot dt)^n \cdot e^{(-L[j] \cdot dt)}}{n!} \quad (3)$$

$$L[j] = c_j \cdot H[j] \quad (4)$$

where c_j , the base contact rate, equals c_{IS} or c_{ES} depending on whether individual j is in the E or I state, and $H[j]$ is the heterogeneity factor for individual j (defined below).

When contact occurs, the probability individual j contacts an individual selected randomly from the $N-1$ others is:

$$\text{Probability}(j \text{ contacting } k | j \text{ has a contact}) = P[j,k] = \frac{\lambda[k] \cdot NW[k, j] / K[k]^\tau}{\sum_N \lambda[i] \cdot NW[i, j] / K[i]^\tau} \quad (5)$$

where:

$\lambda[j]$: The individual heterogeneity factor. Set to 1 in the homogeneous scenarios. In the heterogeneous cases $\lambda[j]$ is drawn from a uniform distribution $U[0.25-1.75]$.

$K[j]$: The number of links individual j has in his or her network.

$NW[i,j]$, the network link, is 1 if there is a link between individuals i and j and 0 if there is none, or if $i=j$. N is the set of all individuals in the population.

The parameter τ captures the time constraint on contacts. In the homogeneous scenarios $\tau = 1$. The total time for contacts with others is fixed, so that individuals with more links have fewer chances of contact with each link, compensating for the heterogeneity created by differing degree distributions in different networks. In the heterogeneous scenarios $\tau = 0$ so that the probability of contact through all links is constant: those with more links contact more people (on average).

The formulation ensures that contacts of the E or I individuals are distributed among other population members depending on their heterogeneity factor and their connectedness.

The overall heterogeneity factor for individual j , $H[j]$, is defined to ensure (1) that contact frequencies capture heterogeneity among individuals, represented by $\lambda[j]$; (2) that individual contact frequencies depend on the number of links to each individual in the heterogeneous

condition but are independent of link density in the homogeneous condition; and (3) that the mean contact frequency across the entire population remains (in expectation) equal to that in the differential equation model. Consequently:

$$H[j] = \frac{\lambda[j] \cdot W[j] \cdot N}{K[j]^\tau \cdot W_N} \quad (6)$$

where:

$$W[j] \text{ is the weighted sum of links to individual } j: \sum_N \lambda[i] \cdot NW[i, j] / K[i]^\tau, \quad (7)$$

W_N , the population level weighted sum of W , normalizes $H[j]$ so that the expected value of mean contacts equals the value in the DE model. Hence

$$W_N = \sum_N \frac{\lambda[i] \cdot W[i]}{K[i]^\tau}. \quad (8)$$

Finally, i_j , probability of infection given a contact between j and k , equals i_{ES} or i_{IS} depending on whether individual j is in the E or I state.

Therefore we can find the infectious contact rate between an individual k in state S and agent j in state E or I, $Z[j, k]$, by substituting and taking the limit:

$$Z[j, k] = \lim_{dt \rightarrow 0} ((C_{dt}[j, k] \cdot i_j) / dt) = \lim_{dt \rightarrow 0} ((\sum_{n=1}^{\infty} R_{dt, n}[j] \cdot (1 - (1 - P[j, k])^n) \cdot i_j) / dt) \quad (9)$$

$$Z[j, k] = \left(\frac{N}{\sum_{i, l} \frac{NW[i, l] \cdot \lambda[i] \cdot \lambda[l]}{(K[i] \cdot K[l])^\tau}} \right) \frac{NW[j, k] \cdot \lambda[j] \cdot \lambda[k]}{(K[j] \cdot K[k])^\tau} \cdot c_j \cdot i_j \quad (10)$$

Therefore the infection hazard for individual k in the state S is:

$$f[k] = \sum_{j \in E \cup I} Z[j, k] \quad (11)$$

Emergence and Recovery: The DE SEIR model assumes populations within each state are well mixed. Consider the recovery process (emergence is analogous). Perfect mixing implies that the hazard rate of recovery for an infectious individual (the transition from I to R) depends only on the expected duration of the infectious phase, δ , and is independent of how long that

particular individual has been in the I state. Consequently residence times for infectious individuals are distributed exponentially. In the AB model, the duration of the infectious stage for infectious individual j is randomly drawn from the exponential distribution with mean duration δ . Hence the hazard rate for recovery is $r[j] = 1/\delta$. Emergence (the transition from E to I) is formulated analogously, so $e[j] = 1/\varepsilon$, where ε is the mean emergence time.

Deriving the mean-field differential equation model:

Exhibit 2 shows the structure of the mean field DE model.

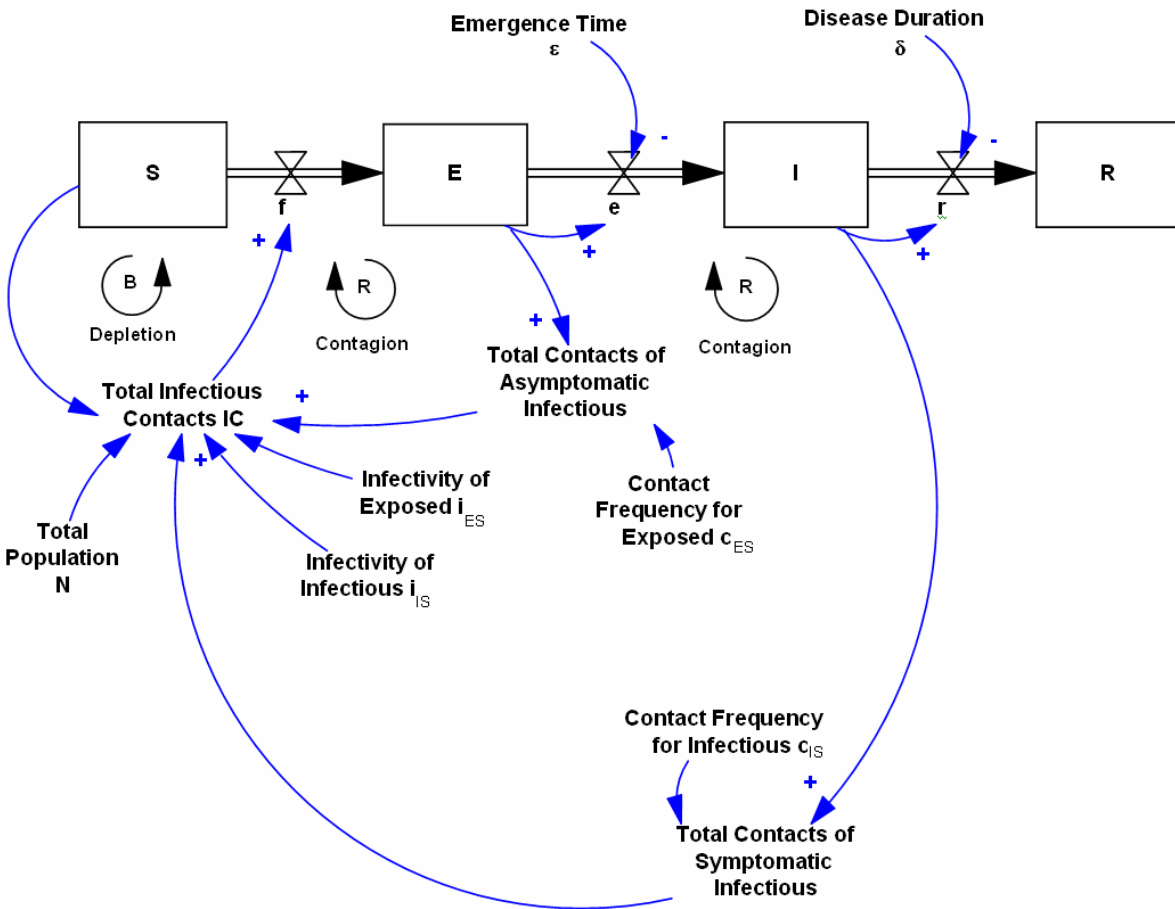


Exhibit 2. The structure of the SEIR differential equation model.

The mean-field model can be derived from the underlying stochastic agent-based model by assuming (1) homogeneity of all agents; (2) perfect mixing within compartments; and (3) all transitions take their expected value.

To derive the DE formulation for the infection rate, f_{DE} , sum the individual infection rates $f[j]$:

$$f_{DE} = \sum_J f[j] = \sum_J \sum_K Z[j,k] \quad (12)$$

Homogeneity implies all individuals have the same number of links, $K[j] = K[k] = K$, and the same propensity to use their links, $\lambda[j] = \lambda[k] = 1$. Therefore:

$$Z[j,k] = \frac{c_j \cdot i_j \cdot N \cdot NW[j,k]}{\sum_{k,j} NW[j,k]} \quad (13)$$

$NW[j,k]$ depends on the network structure. However, in the DE model individuals are assumed to be well mixed, implying everyone is linked to everyone else. Therefore

$$NW[j,k] / \sum_{k,j} NW[j,k] = 1/N^2 \quad (14)$$

yielding

$$Z[j,k] = \frac{c_j \cdot i_j}{N} \quad (15)$$

Noting that homogeneity implies contact frequencies and infectivities are equal within states ($c_j = c_{ES}$ and $i_j = i_{ES}$ for all E and c_{IS} and $i_j = i_{IS}$ for all I) and substituting into the equation for the infection rate yields:

$$f_{DE} = \sum_{k \in S} \sum_{j \in E \cup I} \frac{c_j \cdot i_j}{N} = \frac{1}{N} \sum_{k \in S} \left(\sum_{j \in E} c_{ES} \cdot i_{ES} + \sum_{j \in I} c_{IS} \cdot i_{IS} \right) \quad (16)$$

which simplifies into the formulation for the infection rate in the DE model:

$$f_{DE} = (c_{ES} \cdot i_{ES} \cdot E + c_{IS} \cdot i_{IS} \cdot I) \cdot (S/N) \quad (17)$$

The aggregate emergence and recovery rates in the DE are the sum of the expected individual emergence and recovery rates:

$$e_{DE} = \sum_{j \in E} e[j] = \sum_{j \in E} 1/\varepsilon = E/\varepsilon \quad \text{and} \quad r_{DE} = \sum_{j \in E} r[j] = \sum_{j \in E} 1/\delta = I/\delta \quad (18)$$

which is the formulation for a first-order exponential delay with time constants ε and δ , respectively. The full SEIR model is thus:

$$\frac{dS}{dt} = -f, \quad \frac{dE}{dt} = f - e, \quad \frac{dI}{dt} = e - r \quad (19)$$

$$f = (c_{ES} \cdot i_{ES} \cdot E + c_{IS} \cdot i_{IS} \cdot I) \cdot (S/N) \quad (20)$$

$$e = E/\varepsilon \quad (21)$$

$$r = I/\delta \quad (22)$$

Network Structure:

The connected and lattice networks are deterministic and hence the same in each AB simulation. The random, scale free, and small world networks are stochastic. Each AB simulation of these networks uses a different realization drawn from the appropriate network distribution. We assume the structure of each network remains unchanged over the time horizon of the simulations. The details of construction for each network follow.

Connected: every node is connected to every other node.

Random: The probability of a connection between nodes i and j is fixed, $p = k/(N-1)$, where k is the average number of links per person. In the base case $k = 10$ and $N = 200$. In the sensitivity analysis over population size, $k = 6$ and 18 when $N = 50$ and 800 , respectively.

Scale-free: Barabasi and Albert (1999) outline the preferential attachment algorithm to grow scale-free networks. The algorithm starts with m_0 initial nodes and adds new nodes one at a time. Each new node is connected to previous nodes through m new links, where the probability each existing node j receives one of the new links is $m \cdot K[j] / \sum K[j]$ where $K[j]$ is the number of links into node j . The probability that node j has connectivity smaller than k after t time steps is then:

$$P(K[j]_t < k) = 1 - m^2 t / (k^2 (t + m_0)) \quad (23)$$

We use the same procedure with $m = m_0 = k$ (the average number of links per person). Following the original paper (Albert 2005, personal communication), we start with m_0 individuals connected to each other in a ring, and then add the rest of the $N - m_0$ individuals to the structure. The model accompanying the online material includes the Java code used to implement all networks.

Small World: Following Watts and Strogatz (1998), we build the small world network by ordering all individuals into a one-dimensional ring lattice in which each individual is connected to the $k/2$ closest neighbors. Then we rewire these links to a randomly chosen member of the population with probability $p = 0.05$

Ring Lattice: In the ring lattice each node is linked to the $k/2$ nearest neighbors on each side of the node (equivalent to the small world case with zero probability of long-range links.)

Histogram of Final Size for each network and heterogeneity condition

Histograms for the fraction of the population ultimately infected (final size, F) are shown in exhibit 3 for each network and heterogeneity condition. The heterogeneous cases are shown in blue and homogeneous cases in red. The x-axis is redacted in the middle for the Connected, Random, and Scale-free networks since no simulations had values of F in the redacted range for these cases. The small-world and ring lattice networks include the complete range. The Final Size for the DE model with base case parameters (0.983) is shown in the picture as an arrow.

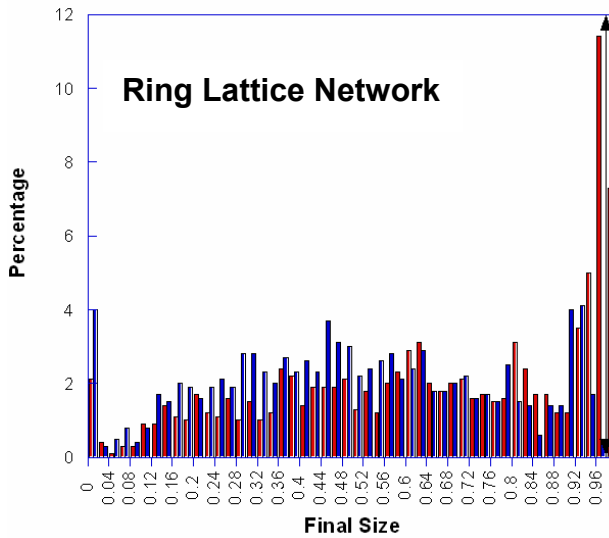
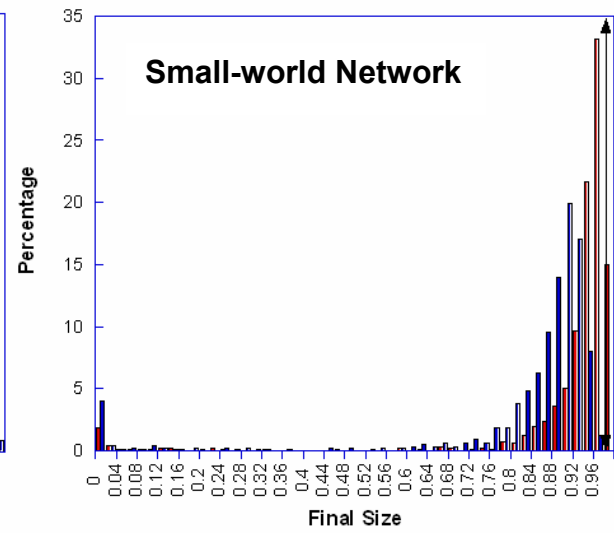
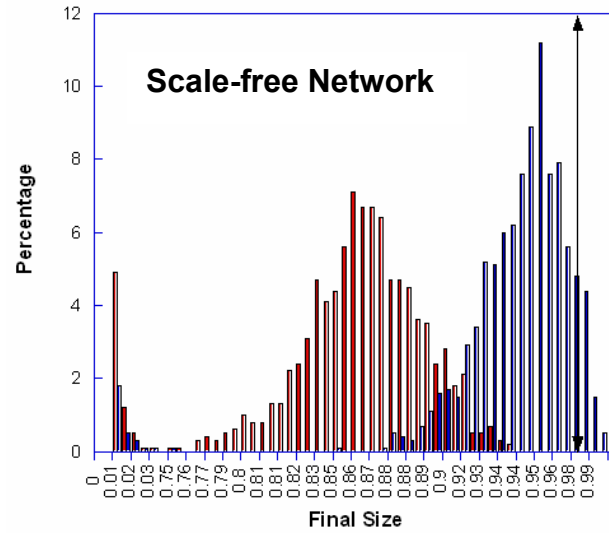
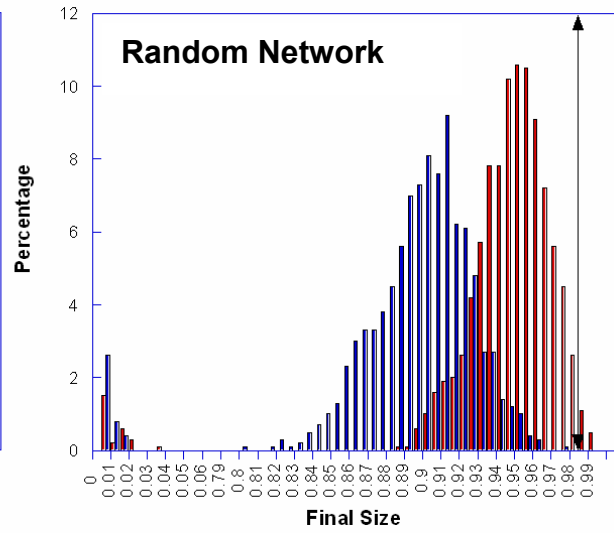
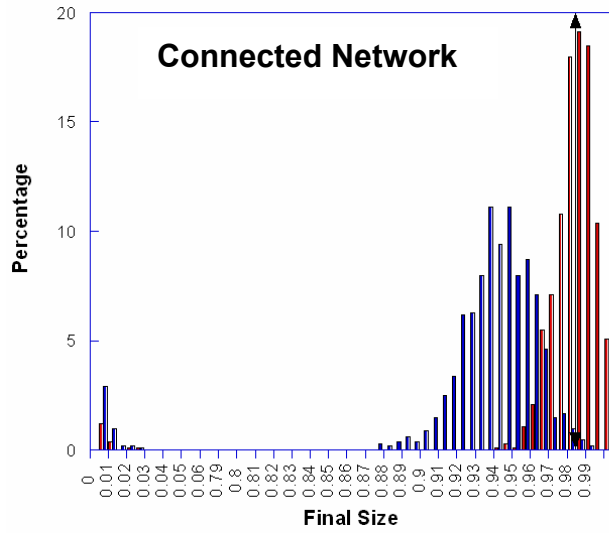


Exhibit 3 Histograms for final size F.

Note the different bins used to create

histograms for different network

settings. Red bars= homogeneous case;

Blue bars= heterogeneous case

The Fitted DE Model

We calibrate the DE model to the trajectory of the infectious population in 200 randomly selected simulations from each of the ten AB scenarios (5 networks x 2 heterogeneity conditions). The best-fit parameters for both infectivities (i_{ES} and i_{IS}) and for the duration of the incubation phase (ε) are estimated by minimizing the sum of squared error between the recovered population in the AB simulation, R_{AB} , and the recovered population in the DE model, R_{DE} , from the start of the simulation through time T , subject to the DE model structure:

$$\min_{i_{ES}, i_{IS}, \varepsilon, \delta} \sum_{t=0}^T [R_{DE}(t) - R_{AB}(t)]^2 \quad (24)$$

We use the Vensim™ software optimization engine for the estimation.

The interval T is selected to capture the full lifecycle of the epidemic. Diffusion is faster in the connected, random, and scale-free cases, so $T = 300$ days. Diffusion is slower in the small world and lattice networks, so $T = 500$ days. The estimated parameters are constrained such that $0 \leq i_{ES}, i_{IS}$ and $0 \leq \varepsilon \leq 30$ days.

Exhibit 4 shows the calibration results, including the median and standard deviation for each parameter, the implied value of R_0 , and the goodness of fit (R^2) for each of the ten scenarios. Exhibit 5 shows how well the trajectory of the calibrated DE model matches the AB models for the three key metrics, including final fraction infected, peak time, and peak prevalence. The calibrated DE models generally fit the AB dynamics extremely well in all network and heterogeneity conditions and for all metrics.

| Parameter | | Connected | | Random | | Scale-free | | Small World | | Lattice | |
|--|----------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|
| | | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ |
| Infectivity of Exposed i_{ES} | Median | 0.045 | 0.053 | 0.044 | 0.049 | 0.034 | 0.065 | 0.025 | 0.026 | 0.025 | 0.040 |
| | σ | 0.021 | 0.088 | 0.023 | 0.171 | 0.093 | 0.245 | 0.055 | 0.135 | 0.280 | 0.310 |
| Infectivity of Infectious i_{IS} | Median | 0.100 | 0.025 | 0.047 | 0.072 | 0.077 | 0.065 | 0.054 | 0.011 | 0.023 | 0.015 |
| | σ | 0.115 | 0.067 | 0.068 | 0.058 | 0.068 | 0.050 | 0.076 | 0.060 | 0.037 | 0.032 |
| Average Incubation Time ε | Median | 14.47 | 10.87 | 12.14 | 6.79 | 12.38 | 3.61 | 21.05 | 16.30 | 6.61 | 3.56 |
| | σ | 4.567 | 5.139 | 4.881 | 5.323 | 5.35 | 5.35 | 7.813 | 8.527 | 13.05 | 10.70 |
| Implied $R_0 = C_{ES}i_{ES}\varepsilon + C_{IS}i_{IS}\delta$ | Median | 4.19 | 2.96 | 3.09 | 2.55 | 3.12 | 2.28 | 3.33 | 2.52 | 1.56 | 1.35 |
| | σ | 1.712 | 0.622 | 0.576 | 0.524 | 0.70 | 0.73 | 0.880 | 0.658 | 0.812 | 0.550 |
| R^2 | Median | 0.999 | 0.999 | 0.999 | 0.999 | 0.999 | 0.999 | 0.998 | 0.998 | 0.985 | 0.987 |
| | σ | 0.025 | 0.049 | 0.017 | 0.050 | 0.019 | 0.038 | 0.040 | 0.059 | 0.056 | 0.043 |

Exhibit 4. Median and standard deviation, σ , of estimated parameters for the calibrated DE model. Reports results of 200 randomly selected runs of the AB model for each cell of the experimental design. Compare to base case parameters $i_{ES} = 0.05$, $i_{IS} = 0.06$, $\varepsilon = 15$ days. The median and standard deviation of the basic reproduction number, $R_0 = C_{ES}i_{ES}\varepsilon + C_{IS}i_{IS}\delta$, computed from the estimated parameters, is also shown.

| Metric | Conf. Bound | Connected | | Random | | Scale-free | | Small World | | Lattice | |
|----------------------|-------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|
| | | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ |
| Final Size | 95% | 99.5 | 94 | 96 | 95.5 | 90 | 87.5 | 97.5 | 91.5 | 96 | 95 |
| | 90% | 95.5 | 89.5 | 88.5 | 89 | 87 | 84.5 | 89.5 | 87.5 | 90 | 89.5 |
| Peak Time, T_p | 95% | 97 | 93.5 | 95.5 | 91.5 | 93.5 | 93.5 | 96 | 93.5 | 89.5 | 93 |
| | 90% | 95 | 88.5 | 90 | 86.5 | 91.5 | 89.5 | 94.5 | 87.5 | 85.5 | 79.5 |
| Peak Prev, I_{max} | 95% | 97.5 | 96.5 | 97.5 | 95.5 | 97.5 | 98.5 | 97 | 98 | 94 | 95.5 |
| | 90% | 92.5 | 93.5 | 89.5 | 83.5 | 86 | 93.5 | 92 | 91.5 | 75.5 | 85 |

Exhibit 5: The percentage of the 200 fitted DE simulations falling inside the 95% and 90% confidence intervals defined by the ensemble of AB simulations, for each of the three metrics of Final Size, Peak Time, and Peak Prevalence, under each network and heterogeneity condition.

Sensitivity to population size:

To investigate the sensitivity of the results to population size, we repeat the analysis for populations of 50 and 800 individuals (\pm a factor of 4 from the base case). For each population we run 1000 simulations for each network and heterogeneity condition. Exhibit 6 reports mean and standard deviation for the four key metrics for each population size. Values of each metric in the base case DE model are shown in parentheses under each metric name (left column). The fraction inside the envelope of AB simulations is calculated from the start of the epidemic through the time when the final recovered population settles within 2% of its final value in the base DE, which is 76 days for $N=800$ and 57 days for $N=50$. ******* indicates the DE simulation falls outside the 95/99% confidence bound defined by the ensemble of AB simulations.

| Population N=800 | | Connected | | Random | | Scale-free | | Small World | | Lattice | |
|---------------------------------------|-----------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|
| | | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ |
| Final Size (0.983) | Mean | 0.95 | 0.90** | 0.94* | 0.88** | 0.94** | 0.82** | 0.95 | 0.89** | 0.82 | 0.64** |
| | σ | 0.17 | 0.18 | 0.15 | 0.17 | 0.16 | 0.20 | 0.14 | 0.18 | 0.24 | 0.27 |
| | % F < 0.1 | 3.1 | 3.9 | 2.4 | 3.5 | 2.7 | 5.6 | 2.1 | 3.9 | 2.2 | 4.8 |
| Peak Time, T _p (57.2) | Mean | 58.2 | 51.9 | 61.4 | 55.6 | 66.4 | 46 | 99.6* | 87.6 | 153.8 | 130 |
| | σ | 12.4 | 12.4 | 13.1 | 12.9 | 14.2 | 13.4 | 22.4 | 23.4 | 123.7 | 101.9 |
| Peak Prev., I _{max} (27%) | Mean | 27 | 25.9 | 26 | 24.6 | 24.9 | 23.5 | 18.8** | 18.2** | 5.4** | 5.0** |
| | σ | 5 | 5.4 | 4.3 | 4.9 | 4.1 | 5.9 | 3.4 | 4.1 | 1.6 | 1.7 |
| Fraction Inside Envelope | | 1 | 1 | 1 | 1 | 1 | 0.88 | 0.35 | 0.42 | 0.30 | 0.32 |

| Population N=50 | | Connected | | Random | | Scale-free | | Small World | | Lattice | |
|--|-----------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|
| | | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ | H ₌ | H _≠ |
| Final Size (0.983) | Mean | 0.96 | 0.90 | 0.89 | 0.81* | 0.85 | 0.80* | 0.80 | 0.70* | 0.70 | 0.60* |
| | σ | 0.14 | 0.17 | 0.17 | 0.21 | 0.22 | 0.20 | 0.25 | 0.27 | 0.28 | 0.27 |
| | % F < 0.1 | 2.1 | 3.5 | 3.2 | 5.5 | 3.3 | 5.8 | 2.8 | 4.7 | 3 | 3.8 |
| Peak Time, T _p (37.8) | Mean | 39.92 | 37.89 | 42.99 | 40.36 | 48.9 | 38.5 | 51.26 | 47.99 | 48.52 | 46.86 |
| | σ | 12.43 | 13.17 | 16.13 | 16.98 | 21.1 | 16.9 | 26.71 | 27.2 | 28.68 | 30.05 |
| Peak Prev., I _{max} (27.5 ¹) | Mean | 33.2 | 31.4 | 29.3 | 27 | 25.9 | 27 | 22.1 | 20.3 | 18.9 | 17.3 |
| | σ | 7.29 | 7.85 | 7.47 | 8.18 | 8.21 | 7.99 | 7.85 | 7.94 | 7.11 | 6.75 |
| Fraction Inside Envelope | | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0.82 | 0.54 |

Exhibit 6. Results for populations of 50 and 800.

Policy Analysis

We assume that the quarantine policy is initiated when the cumulative number of confirmed cases detected rises above some threshold. Typically, the scope of quarantine policies—the number of people affected, and the scope of restrictions on their movements—increases with cumulative confirmed cases, as documented for the SARS epidemic (Wallinga and Teunis 2004): As the extent of the epidemic grows, the broader will be the mandate for quarantine government

¹ Note that values of T_p and I_{max} in the DE depend on population N. The course of the epidemic depends on the initial fraction of the population susceptible to infection. All simulations begin with 2 randomly selected individuals in the emergence phase, and therefore N – 2 susceptibles. Hence the susceptible fraction of the population is initially 96% (48/50) when N = 50 and 99.75% (798/800) when N = 800.

officials will propose and society will accept. Further, the propensity of individuals to self-quarantine by staying away from work, school and other mixing sites increases with the perceived threat from the disease, thus reducing contact frequencies. We model quarantine as a reduction in the frequency of contacts between infectious individuals j and susceptibles s , c_{js} . Specifically, contact frequencies fall linearly from initial levels c_{js}^* to the minimum rates achieved under quarantine, c_{js}^q , as cumulative confirmed cases rise, according to equations (6-7) in the paper:

$$c_{js} = (1 - q)c_{js}^* + qc_{js}^q$$

$$q = \text{MIN}[1, \text{MAX}(0, (P - P_0)/(P_q - P_0))]$$

where the intensity of the quarantine policy, q , rises linearly from zero to one as the number of confirmed cases (cumulative prevalence, $P = E + I$), rises from the quarantine implementation threshold, P_0 , to the level at which quarantine is fully deployed, P_q . We set $P_0 = 2$ and $P_q = 10$ cases. Because quarantine is never perfect (some contacts inevitably arise from infectious individuals who are not quarantined or who violate quarantine, and from contact between the quarantined and health providers), the minimum contact frequency does not fall to zero. In the simulations here we set $c_{js}^q = 0.15c_{js}^*$.

Exhibit 7 compares the results of the quarantine policy to the no-quarantine base case for the DE model. Exhibit 8 shows the first 90 days in more detail, showing that the quarantine is initiated around day 8 and reaches full implementation around day 18. As expected, the quarantine policy dramatically shortens the epidemic and reduces cumulative cases. In the base DE model, F falls from 98% infected to 19%. The epidemic peaks after 31 days, compared to 48 days without quarantine, and with peak prevalence of infectious individuals of 4.4%, compared to 27% in the base case. Due to the incubation time the peak of the infectious population lags full implementation of quarantine.

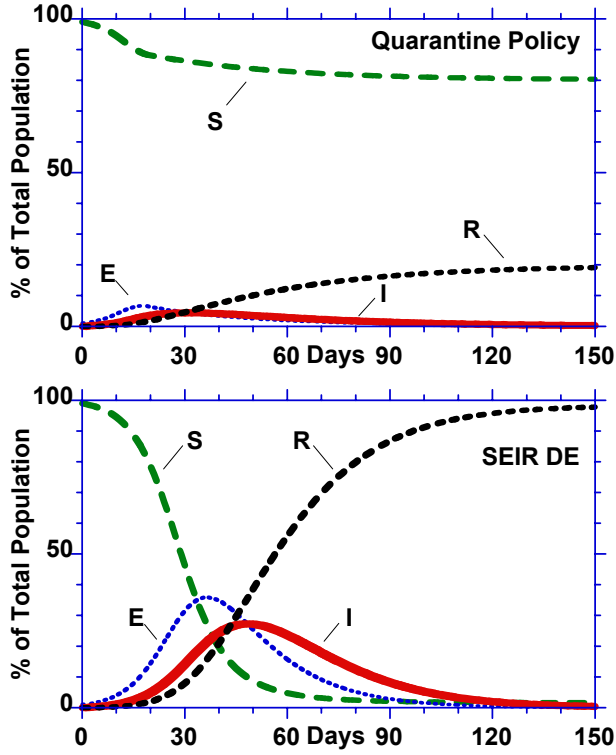


Exhibit 7. DE model behavior under quarantine (top) compared to base case (bottom).

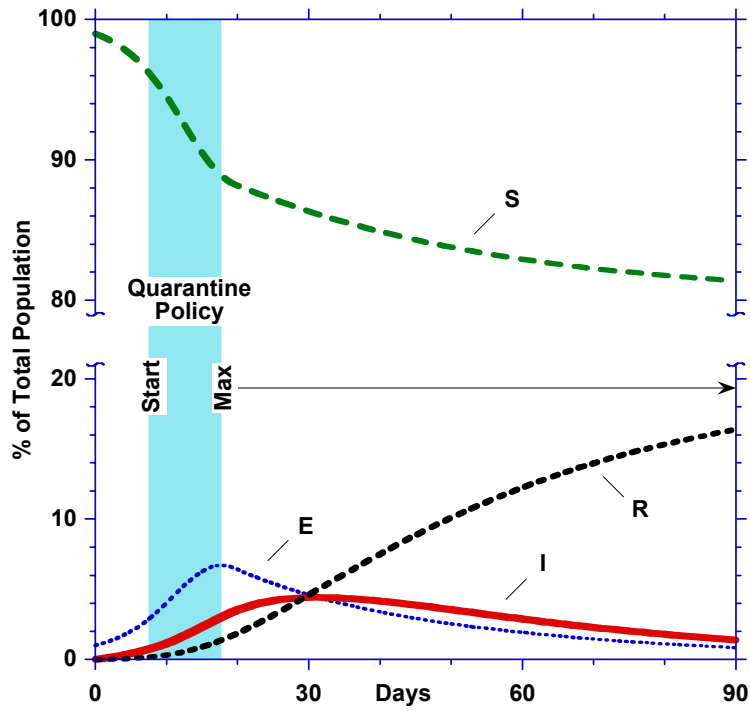


Exhibit 8. First 90 days of the quarantine case in the DE model, showing when the quarantine policy is initiated (around day 8) and when it reaches full implementation (around day 18).

Note that the E and I populations decay quite slowly after full quarantine implementation. The slow decay is due to the assumption that quarantine is imperfect. In the simulation R_0 falls to a minimum of about 0.6 at full implementation. Consequently new cases continue to be generated, lengthening the effective time constant for the decay of the infectious population.

Table 6 in the paper (repeated here as Exhibit 9) compares the key public health metrics in the AB models to the base DE case. Like the original results, the means of the key public health metrics F , T_p and I_{max} are not significantly different from the values of the DE model, with two exceptions: F in the DE model falls outside the 95% confidence band for both the H_+ and H_- cases of the lattice. The DE results are not significantly different from those of the AB model for all other metrics in all other network and heterogeneity conditions.

The impact of the policy on the key public health metrics is large compared to the variability in the realizations of the individual AB simulations and in the variability of the metrics across network and heterogeneity conditions.

| Metric | | Connected | | Random | | Scale-free | | Small World | | Lattice | |
|---------------------|----------|-----------|-------|--------|-------|------------|-------|-------------|-------|---------|--------|
| | | H_+ | H_- | H_+ | H_- | H_+ | H_- | H_+ | H_- | H_+ | H_- |
| Final Size F | μ | 0.215 | 0.249 | 0.157 | 0.201 | 0.148 | 0.247 | 0.112 | 0.117 | 0.102* | 0.099* |
| | σ | 0.084 | 0.091 | 0.064 | 0.088 | 0.062 | 0.105 | 0.044 | 0.048 | 0.037 | 0.035 |
| Peak Time T_p | μ | 35.0 | 36.1 | 33.1 | 34.6 | 34.1 | 34.9 | 30.3 | 30.5 | 29.4 | 30.4 |
| | σ | 15.3 | 15.9 | 14.8 | 16.7 | 15.7 | 18.1 | 13.5 | 14.2 | 13.5 | 14.6 |
| Peak Prev I_{max} | μ | 6.42 | 7.28 | 5.17 | 6.15 | 4.98 | 7.43 | 4.17 | 4.35 | 3.97 | 3.89 |
| | σ | 2.40 | 2.66 | 1.99 | 2.55 | 1.98 | 3.23 | 1.58 | 1.67 | 1.52 | 1.42 |

Exhibit 9. Key public health metrics for simulations of the quarantine policy. */** indicates the value of the metric in the DE model falls outside the 95/99% confidence bound defined by the ensemble of AB simulations. The results for the DE model under quarantine are $F = 0.190$, $T_p = 31.3$ days, and $I_{max} = 4.43\%$.

Simulating the model

Two versions of the model are posted with the online material. Both are developed in the Anylogic™ software. Anylogic supports both agent-based and differential equation models. One interface is a stand-alone Java applet and runs under any Java-enabled browser with no need to install additional software. The second includes the source code and equations of the model and can be used for detailed inspection of model implementation and for simulation, once the Anylogic software is installed.

To open the stand-alone applet, unzip “ABDE-Contagion-applet.zip” into one folder (all three files need be in the same folder). You can then open the “Dynamics of Contagion_Apr05_Paper Applet.html” file in any web browser (e.g. Internet Explorer). The interface is self-explanatory and includes a help file, which can be opened by clicking the [?] button.

To open the Anylogic model source code, first download and install the Anylogic software (a free 15-day trial version is available at <http://www.xjtek.com/download/>). Unzip the attached file “ABDE-Contagion-code.zip” in one folder. Open the file “Dynamics of Contagion_Apr05_Paper.alp” in Anylogic. The list of all objects in the agent-based model is displayed in the left hand column; select any object to inspect its formulation. Run the model by clicking on the run button (or pressing F5), which compiles the model and brings up an interface similar to the Java applet. You can inspect the behavior of the model both through the interface accompanying the model and applet, or by browsing different variables and graphing them in Anylogic run mode. To do the latter, go to “root” tab in run time mode, where you can see all model variables and can inspect their runtime behavior.

The implemented interface has the following capabilities, available through both the applet and source code versions of the model:

1. Run the model for different scenarios in single or multiple runs.

2. Inspect the behavior of the AB model, including the total number of people in each state, state transition rates and user-specified confidence intervals for the behavior of the infected population in the AB model.
3. Visually inspect disease diffusion in the network. Different nodes and links between them are shown. You can choose among 4 different representations of the network structure to show the progression of the epidemic through the network.
4. Observe the metrics of peak value, peak time, and final size.
5. Change model parameters, including the incubation and recovery periods, infectivities, and contact rates (note that mean contact rates for exposed and infected individuals are expressed as fractions of the contact rate for healthy individuals). You can also set the size of the population, network type, number of links per person, and probability of long-range links in small-world network, and select the heterogeneous or homogeneous condition.
6. Save the results into a text file for subsequent analysis.

References

- Barabasi, A. L. and R. Albert. 1999. Emergence of scaling in random networks. *Science* **286**(5439): 509-512.
- Wallinga, J. and P. Teunis. 2004. Different epidemic curves for severe acute respiratory syndrome reveal similar impacts of control measures. *American Journal of Epidemiology* **160**(6): 509-516.
- Watts, D. J. and S. H. Strogatz. 1998. Collective dynamics of "small-world" networks. *Nature* **393**(4 June): 440-442.