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

When is it better to use agent-based (AB) models, and when should differential equation (DE) models be used? Where DE models assume homogeneity and perfect mixing within compartments, AB models can capture heterogeneity in agent attributes and in the network of interactions among them. The costs and benefits of such disaggregation should guide the choice of model type. AB models may enhance realism but entail computational and cognitive costs that may limit sensitivity analysis and model scope. Using contagious disease as an example, we contrast the dynamics of AB models with those of the analogous mean-field DE model. We examine agent heterogeneity and the impact of different network topologies, including fully connected, random, Watts-Strogatz small world, scale-free, and lattice networks. Surprisingly, in many conditions differences between the DE and AB dynamics are not statistically significant for key metrics relevant to public health, including diffusion speed, peak load on health services infrastructure and total disease burden. We discuss implications for the choice between AB and DE models, level of aggregation, and model boundary. The results apply beyond epidemiology: from innovation adoption to financial panics, many important social phenomena involve analogous processes of diffusion and social contagion.

Keywords: Agent Based Models, Networks, Scale free, Small world, Heterogeneity, Epidemiology, Simulation, System Dynamics, Complex Adaptive Systems, SEIR model

* We thank Reka Albert, Rosanna Garcia, Ed Kaplan, David Krackhardt, Marc Lipsitch, Nelson Repenning, Perwez Shahabuddin, Steve Strogatz, Duncan Watts, Larry Wein, anonymous referees, and seminar participants at MIT, the 2004 NAACSOS conference and 2004 International System Dynamics Conference for helpful comments. Ventana Systems and XJ Technologies generously provided their simulation software and technical support. Financial support provided by the Project on Innovation in Markets and Organizations at the MIT Sloan School.
Spurred by growing computational power, agent-based modeling (AB) is increasingly applied to physical, biological, social, and economic problems previously modeled with nonlinear differential equations (DE). Both approaches have yielded important insights. In the social sciences, agent models explore phenomena from the emergence of segregation to organizational evolution to market dynamics (Schelling 1978; Levinthal and March 1981; Carley 1992; Axelrod 1997; Lomi and Larsen 2001; Axtell, Epstein et al. 2002; Epstein 2002; Tesfatsion 2002). Differential and difference equation models, also known as compartmental models, have an even longer history in social science, including innovation diffusion (Mahajan, Muller et al. 2000), biology and epidemiology (Anderson and May 1991), and economics (Black and Scholes 1973).

When should AB models be used, and when are DE models most appropriate? Each method has strengths and weaknesses. Nonlinear DE models often have a broad boundary encompassing a wide range of feedback effects, but typically aggregate agents into a relatively small number of states (compartments). For example, models of innovation diffusion may aggregate the population into categories including unaware, aware, in the market, recent adopters, and former adopters (Urban, Houser et al. 1990; Mahajan, Muller et al. 2000). However, the agents within each compartment are assumed to be homogeneous and well mixed; the transitions among states are modeled as their expected value, possibly perturbed by random events. In contrast, AB models can readily include heterogeneity in agent attributes and in the network structure of their interactions; like DE models, these interactions can be deterministic or stochastic.

The granularity of AB models comes at some cost. First, the extra complexity significantly increases computational requirements, constraining the ability to conduct sensitivity analysis to investigate the robustness of results. A second cost of agent-level detail is the cognitive burden of understanding model behavior. Linking the behavior of a model to its structure becomes more difficult as model complexity grows. A third tradeoff concerns the communication and implementation: the more complex a model, the harder it is to communicate the basis for its results to stakeholders and decision makers, and thus have an impact on policy. Finally, limited time and resources force modelers to trade off disaggregate detail and the scope of the model boundary. Model boundary here stands for the richness of the feedback structure captured endogenously in the model (Meadows and Robinson 1985, Sterman 2000). For example, an agent-
based demographic model may portray each individual in a population and capture differences in age, sex, and zip code, but assume exogenous fertility and mortality; such a model has a narrow boundary. In contrast, an aggregate model may lump the entire population into a single compartment, but model fertility and mortality as functions of food per capita, health care, pollution, norms for family size, etc., each of which, in turn, are modeled endogenously; such a model has a broad boundary. DE and AB models may in principle fall anywhere on these dimensions of disaggregation and scope. In practice, however, where time, budget, and computational resources are limited, modelers must trade off aggregate detail and breadth of boundary. Understanding where the agent-based approach yields additional insight and where such detail is unimportant is central in selecting appropriate methods for any problem.

The stakes are large. Consider potential bioterror attacks. Kaplan, Craft, and Wein (2002) used a deterministic nonlinear DE model to examine smallpox attack in a large city, comparing mass vaccination (MV), in which essentially all people are vaccinated after an attack, to targeted vaccination (TV), in which health officials trace and immunize those contacted by potentially infectious individuals. Capturing vaccination capacity and logistics explicitly, they conclude MV significantly reduces casualties relative to TV. In contrast, Eubank et al. (2004) and Halloran et al. (2002), using different AB models, conclude TV is superior, while Epstein et al. (2004), using an AB model, favor a hybrid strategy. The many differences among these models make it difficult to determine whether the conflicting conclusions arise from relaxing the perfect mixing and homogeneity assumptions of the DE (as argued by Halloran et al. 2002) or from other assumptions such as the size of the population (ranging from 10 million for the DE model to 2000 in Halloran et al. to 800 in Epstein et al.), other parameters, or boundary differences such as capacity constraints on immunization (Koopman 2002; Ferguson, Keeling et al. 2003; Kaplan and Wein 2003). Kaplan and Wein (2003) show that their deterministic DE model closely replicates the Halloran et al. AB results when simulated with the Halloran et al. parameters, including scaled down population and initial attack size, indicating that differences in the logistics of MV and TV are responsible for the different conclusions, not differences in mixing and homogeneity. Halloran and Longini (2003) respond that the equivalence may not hold when population is scaled up to city size due to imperfect mixing and agent heterogeneity, but do not test this proposition because simulation of
their AB model with 10 million agents is computationally prohibitive.

Here we carry out controlled experiments to compare AB and DE models in the context of contagious disease. We choose disease diffusion for four reasons. First, the effects of different network topologies for contacts among individuals are important in the diffusion process (Davis 1991; Watts and Strogatz 1998; Barabasi 2002; Rogers 2003), providing a strong test for differences between the two approaches. Second, the dynamics of contagion involve many important characteristics of complex systems, including positive and negative feedbacks, time delays, nonlinearities, stochastic events, and agent heterogeneity. Third, there is a rich literature on epidemic modeling. The DE paradigm is well developed in epidemiology (for reviews see Anderson and May 1991 and Hethcote 2000), and AB models with explicit network structures are on the rise (for reviews see Newman 2002; 2003 and Watts 2004).

Finally, diffusion is a fundamental process in diverse physical, biological, social, and economic settings. Many diffusion phenomena in human systems involve processes of social contagion analogous to infectious disease, including word of mouth, imitation, and network externalities. From the diffusion of innovations, ideas, and new technologies within and across organizations, to rumors, financial panics and riots, contagion-like dynamics, and formal models of them, have a rich history in the social sciences (Bass 1969; Watts and Strogatz 1998; Mahajan, Muller et al. 2000; Barabasi 2002; Rogers 2003). Insights into the advantages and disadvantages of AB and DE models in epidemiology can inform understanding of diffusion in many domains of concern to social scientists and managers.

Our procedure is as follows. We develop an AB version of the classic SEIR model, a widely used nonlinear deterministic DE model. The simple DE version divides the population into four compartments: Susceptible (S), Exposed (E), Infected (I), and Recovered (R)\(^1\). In the AB model, each individual is separately represented and must be in one of the four states. The same

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\(^1\) More realistic DE models disaggregate a population into additional compartments to capture some of the heterogeneity in real situations. As additional compartments are added the differences between the DE and AB model will diminish. We use the simplest DE model to explore whether results for the DE and AB approaches diverge for the case of maximal differences in aggregation.
parameters are used for both AB and DE models. Therefore any differences in outcomes arise only from the relaxation of the mean-field assumptions of the DE model. We run the AB model under five widely used network topologies (fully connected, random, small world, scale-free, and lattice) and test each with homogeneous and heterogeneous agent attributes such as the rate of contacts between agents. We compare the DE and AB dynamics on a variety of key metrics relevant to public health, including cumulative cases, peak prevalence, and the speed the disease spreads.

As expected, different network structures alter the timing of the epidemic. The behavior of the AB model under the connected and random networks closely matches the DE model, as these networks approximate the perfect mixing assumption. Also as expected, the stochastic AB models generate a distribution of outcomes, including some in which, due to random events, the epidemic never takes off. The deterministic DE model cannot generate this mode of behavior.

Surprisingly, however, even in conditions that violate the perfect mixing and homogeneity assumptions of the DE, the differences between the DE and AB models are not statistically significant for many of the key public health metrics (with the exception of the lattice network, where all contacts are local and diffusion is slow compared to the DE). The dynamics of different networks are close to the DE: even a few long-range contacts or highly connected hubs seed the epidemic at multiple points in the network, enabling it to spread rapidly (Watts and Strogatz 1998).

We also examine the ability of the DE model to capture the dynamics of each network structure in the realistic situation where key parameters are poorly constrained by biological and clinical data. Epidemiologists often estimate potential diffusion, for both novel and established pathogens, by fitting models to the aggregate data as an outbreak unfolds (Dye and Gay 2003; Lipsitch, Cohen et al. 2003; Riley, Fraser et al. 2003). Calibration of innovation diffusion and new product marketing models is similar (Mahajan et al. 2000). We mimic this practice by treating the AB simulations as the “real world” and fitting the DE model to them. Surprisingly, the fitted model closely matches the behavior of the AB model under all network topologies and heterogeneity conditions tested. Departures from homogeneity and perfect mixing are incorporated into the best-fit values of parameters.

The concordance between DE and AB model suggests DE models remain useful and appropriate in many situations, even when mixing is imperfect and agents are heterogeneous. Since
time and resources are always limited, modelers must trade off the data requirements and the computational burden of disaggregation against the breadth of the model boundary and the ability to do sensitivity analysis. The detail possible in AB models is likely to be most helpful where the contact network is highly clustered, known, and stable, where results depend delicately on agent heterogeneity or stochastic events, where the relevant population is small enough to be computationally feasible, and where time and budget permit sensitivity testing. DE models will be most appropriate where network structure is unknown, labile, or moderately clustered, where results hinge on the incorporation of a wide range of feedbacks among system elements (a broad model boundary), where large populations must be treated, and where fast turnaround or extensive sensitivity analysis are required.

The next section reviews the literature comparing AB and DE approaches. We then describe the structure of the epidemic models, the design of the simulation experiments, and results, closing with implications and directions for future work.

**A spectrum of aggregation assumptions:** AB and DE models should be viewed as regions in a space of modeling assumptions, not as incompatible modeling paradigms. Aggregation is one of the key dimensions of that space. Models can range from lumped deterministic differential equations (also called deterministic compartmental models), to stochastic compartmental models, in which the continuous variables of the DE are replaced by discrete individuals, to event history models, where the states of individuals are tracked but their network of relationships is ignored, to models where networks of individual interactions are explicit (e.g., Koopman et al. 2001).

A few studies compare AB and DE models. Axtell et al. (1996) call for “model alignment” or “docking” and illustrate with the Sugarscape model. Edwards et al. (2003) contrast an AB model of innovation diffusion with an aggregate model, finding that the two can diverge when multiple attractors exist in the deterministic model. In epidemiology, Jacquez and colleagues (Jacquez and O’Neill 1991; Jacquez and Simon 1993) analyzed the effects of stochasticity and discrete agents in the SIS and SI models, finding some differences for very small populations. However, the differences practically vanish for populations above 100. Heterogeneity has also been explored in models that assume different mixing sites for population members. Anderson and May (1991, Chapter 12) show that the immunization fraction required to quench an epidemic rises
with heterogeneity if immunization is implemented uniformly but falls if those most likely to transmit the disease are the focus of immunization. Ball et al. (1997) and Koopman, Chick et al. (2002) find expressions for cumulative cases and global epidemic thresholds in stochastic SIR and SIS models with global and local contacts, concluding that the behavior of deterministic and stochastic DE models can diverge for small populations. Keeling (1999) formulates a DE model that approximates the effects of spatial structure even when contact networks are highly clustered. Chen et al. (2004) develop AB models of smallpox, finding the dynamics generally consistent with DE models. In sum, AB and DE models of the same phenomenon sometimes agree and sometimes diverge, especially for smaller populations.

**Model Structure:** The SEIR model is a deterministic nonlinear differential equation model in which all members of a population are in one of four states—Susceptible, Exposed, Infected, and Recovered. Infected (symptomatic) individuals can transmit the disease to susceptibles before they are “removed” (i.e., recover or die). The exposed compartment captures latency between infection and the emergence of symptoms. Depending on the disease, exposed individuals may become infectious before symptoms emerge, and can be called early stage infectious. Typically, the exposed have more contacts than the infectious because they are healthier and are often unaware that they are potentially contagious, while the infectious are ill and often self-quarantined.

SEIR models have been successfully applied to many diseases. Additional compartments are often introduced to capture more complex disease lifecycles, diagnostic categories, therapeutic protocols, population heterogeneity, assortative mixing, birth/recruitment of new susceptibles, loss of immunity, etc. (Anderson and May 1991 and Murray 2002 provide comprehensive discussion of the SEIR model and its extensions). In this study we maintain the boundary assumptions of the traditional SEIR model (four stages, fixed population, permanent immunity). The DE implementation of the model imposes several additional assumptions, including perfect mixing and homogeneity of individuals within each compartment and mean field aggregation (the flows between compartments equal the expected value of the sum of the underlying probabilistic rates for individual agents). To derive the differential equations, consider the rate at which each infectious individual generates new cases:

\[ c_{is} \times \text{Prob}(\text{Contact with Susceptible}) \times \text{Prob}(\text{Transmission|Contact with Susceptible}) \]  

(1)
where the contact frequency $c_{is}$ is the expected number of contacts between infectious individual $i$ and susceptible individual $s$ per time period; homogeneity implies $c_{is}$ is a constant, denoted $c_{IS}$, for all individuals $i$ and $s$. If the population is well mixed, the probability of contacting a susceptible individual is simply the proportion of susceptibles in the total population, $S/N$. Denoting the probability of transmission given contact between individuals $i$ and $s$, or infectivity, as $i_{is}$ (which, due to homogeneity, equals $i_{IS}$ for all $i, s$), and summing over the infectious population yields the total flow of new cases generated by contacts between $I$ and $S$ populations, $c_{IS}i_{IS}*I*(S/N)$. The number of new cases generated by contacts between the exposed and susceptibles is formulated analogously, yielding the total Infection Rate, $f$,

$$f = (c_{ES}i_{ES}E + c_{IS}i_{IS}I)*(S/N).$$

(2)

To model emergence and recovery, consider these to be Markov processes with certain transition probabilities. In the classic SEIR model each compartment is assumed to be well mixed so that the probability of emergence (or recovery) is independent of how long an individual has been in the $E$ (or $I$) state. Denoting the mean emergence time and disease duration $\varepsilon$ and $\delta$, the individual hazard rate of emergence and recovery is then $1/\varepsilon$ and $1/\delta$, respectively. Summing over the $E$ and $I$ populations and taking expected values yields the flows of emergence and recovery:

$$e = E/\varepsilon \text{ and } r = I/\delta.$$  

(3)

The full model is thus: \[ \frac{dS}{dt} = -f, \quad \frac{dE}{dt} = f - e, \quad \frac{dI}{dt} = e - r, \quad \frac{dR}{dt} = r. \]  

(4)

Specifying the $E$ and $I$ stages as single compartments with first-order outflows as in eq (3) implies the probabilities of emergence and recovery are independent of how long an individual has been in the $E$ or $I$ states, respectively, and results in exponential distributions for the residence times in these states. Exponential residence times are not realistic for most diseases, where the probability of emergence and recovery is initially low, then rises, peaks and falls. Note, however, that any lag distribution can be captured through the use of partial differential equations, approximated in the

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2 Due to data limitations the parameters $c$, $i$, and $N$ are often lumped together as $\beta = c*i/N$; $\beta$ is estimated from aggregate data such as reported incidence and prevalence. We distinguish these parameters since they are not necessarily equal across individuals in the AB implementation.
ODE paradigm by adding additional compartments within the exposed and infectious categories (Jacquez and Simon 2002). For example, disaggregating the infectious stage serially into $n$ compartments, each with mean residence time $\delta/n$, yields an $n^{th}$ order Erlang distribution for the recovery probability. Through parallel as well as serial disaggregation any distribution for state transitions can be achieved. Here we maintain the single-compartment structure of the classic SEIR model, noting that the exponential distribution has greater variance than higher-order distributions, maximizing the potential divergence between the DE and AB models.

The AB model relaxes the perfect mixing and homogeneity assumptions. Each individual $j$ is in one of the four states $S[j]$, $E[j]$, $I[j]$, and $R[j]$ for $j \in (1, \ldots, N)$. State transitions depend on the agent’s state, on agent-specific attributes such as contact frequencies, and on the chances of interactions with other agents as specified by the contact network among them. The aggregate rates $f$, $e$, and $r$ are the sum of the individual transitions $f[j]$, $e[j]$, and $r[j]$. The supplementary material details the formulation of the AB model and shows how the DE model can be derived from it by assuming homogeneous agents and applying the mean-field approximation.

The key parameter in epidemic models is the basic reproduction number, $R_0$, the expected number of new cases each contagious individual generates before recovering, assuming all others are susceptible. We use parameters roughly similar to smallpox (Halloran, Longini et al. 2002; Kaplan, Craft et al. 2002; Ferguson, Keeling et al. 2003). Smallpox has a mid-range reproduction number, $R_0 \approx 3–6$ (Gani and Leach 2001), and therefore is a good choice to observe potential differences between DE and AB models: diseases with $R_0 < 1$ die out quickly, while those with $R_0 >> 1$, e.g. chickenpox and measles, generate a severe epidemic in (nearly) any network topology. We set $R_0 \approx 4$ for the DE (Table 1). The AB models use the same values for the infectivities and expected residence times, and we set individual contact frequencies so that the mean contact rate in each network and heterogeneity condition is the same as the DE model. We set the population $N = 200$, all susceptible except for two randomly chosen exposed individuals. While small compared to settings of interest in policy design, e.g., cities, the effects of random events and network

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3 Smallpox-specific models often add a prodromal period between the incubation and infectious stages (e.g. Eubank et al. 2004); for generality we maintain the SEIR structure.
structure are likely to be more pronounced in small populations (Kaplan and Wein 2003), providing a stronger test for differences between the DE and AB models. A small population also reduces computation time, allowing more extensive sensitivity analysis. The DE model has 4 state variables; computation time is negligible for all population sizes. The AB model has 4*N states, and must also track interactions among the N individuals. Depending on the density of the contact network, the AB model can grow at rates up to O(N^2). We report sensitivity analysis of population size below. The supplement includes the models and full documentation.

**Experimental design:** We vary both the network structure of contacts among individuals and the degree of agent heterogeneity in the AB model and compare the resulting dynamics to the DE version. We implement a full factorial design with five network structures and two heterogeneity conditions. In each of the ten conditions we generate an ensemble of 1000 simulations of the AB model, each with different realizations of the random variables determining contacts, emergence, and recovery. Since the expected values of parameters in each simulation are identical to the DE model, differences in outcomes can only be due to differences in network topology, agent heterogeneity, or the discrete, stochastic treatment of individuals in the AB model.

**Network topology:** The DE model assumes perfect mixing, implying anyone can meet anyone else with equal probability. Realistic networks are far more sparse, with most people contacting only a small fraction of the population. Further, most contacts are clustered among neighbors with physical proximity. We explore five different network structures: fully connected (each person can meet everybody else), random (Erdos and Renyi 1960), small-world (Watts and Strogatz 1998), scale-free (Barabasi and Albert 1999), and lattice (where contact only occurs between neighbors on a ring). We parameterize the model so that all networks (other than the fully connected case) have the same mean number of links per node, k = 10 (Watts 1999).

The fully connected network corresponds to the perfect mixing assumption of the DE model. The random network is similar except people are linked with equal probability to a subset

\[ (1/N) \sum_i 2n_i / [k_i(k_i - 1)] \]

where \( n_i \) is the total number of links among all nodes linked to \( i \) and \( k_i \) is the number of links of the \( i \)th node (Watts and Strogatz 1998).
of the population. To test the network most different from the perfect mixing case, we also model a one-dimensional ring lattice with no long-range contacts. With $k = 10$ each person contacts only the five neighbors on each side. The small world and scale-free networks are intermediate cases with many local and some long-distance links. These widely used networks characterize a number of real systems (Watts 1999; Barabasi 2002). We set the probability of long-range links in the small world networks to 0.05, in the range used by Watts (1999). We build the scale-free networks using the preferential attachment algorithm (Barabasi and Albert 1999) in which the probability a new node links to existing nodes is proportional to the number of links each node already has. Preferential attachment yields a power law for the probability that a node has $k$ links, $\text{Prob}(k) = \alpha k^{-\gamma}$. Empirically $\gamma$ typically falls between 2 and 3; the mean value of $\gamma$ in our experiments is 2.60. Scale free networks contain a few highly connected “hubs” and many nodes with few links.

Two of the networks are deterministic (the fully connected and lattice) so every AB simulation of these cases has the same network structure governing contacts among individuals. The Erdos-Renyi, small world, and scale-free cases are random networks. Each simulation of these cases uses a different realization of the network structure; within each simulation the network structure does not change. The supplement details the construction of each network.

Agent Heterogeneity: Each individual has four relevant characteristics: expected contact rate, infectivity, emergence time, and disease duration. In the homogeneous condition ($H_\text{H}$) each agent is identical with parameters set to the values of the DE model. In the heterogeneous condition ($H_\text{H}$) we vary individual contact frequencies. Focusing on contact frequencies reduces the dimensionality of required sensitivity analysis at little cost. First, only the product of the contact rate and infectivity matters to $R_0$ (see note 2). Second, the exponential distributions for $\varepsilon$ and $\delta$ introduce significant variation in the realizations of individual incubation and disease durations even when the expected value is equal for all individuals.

Heterogeneity in contacts is modeled as follows. Given that two people are linked (that they can come into contact), the frequency of contact between them depends on two factors. First, how often does each use their links, on average: some people are gregarious; others shy. Second, time constraints may limit contacts. At one extreme, the frequency of link use may be constant, so that people with more links have more total contacts per day, a reasonable approximation for some
airborne diseases and easily communicated ideas: a professor may transmit an airborne virus or simple concept to many people with a single sneeze or comment, (roughly) independent of class size. At the other extreme, if the time available to contact people is fixed, the chance of using a link is inversely proportional to the number of links, a reasonable assumption when transmission requires extended personal contact: the professor can only tutor a limited number of people each day. We capture these effects by assigning individuals different propensities to use their links, \( \lambda[j] \), yielding the expected contact frequency for the link between individuals i and j, \( c[i,j] \):  

\[
  c[i,j] = \kappa \lambda[i] \lambda[j] / (k[i] k[j]) ^ \tau
\]

where \( k[j] \) is the total number of links individual j has, \( \tau \) captures the time constraint on contacts, and \( \kappa \) is a constant chosen to ensure that the expected contact frequency for all individuals equals the mean value used in the DE model. In the homogeneous condition \( \tau = 1 \) and \( \lambda[j] = 1 \) for all j so that in expectation all contact frequencies are equal, independent of how many links each individual has. In the heterogeneous condition, those with more links have more contacts per day (\( \tau = 0 \)), and individuals have different propensities to use their links. We use a uniform distribution with a large range, \( \lambda[j] \sim U[0.25, 1.75] \).

*Calibrating the DE Model:* In real world applications the parameters determining \( R_0 \) are often poorly constrained by biological and clinical data. For emerging diseases such as vCJD, BSE and avian flu data are not available until the epidemic has already spread. Parameters are usually estimated by fitting models to aggregate data as an outbreak unfolds; SARS provides a typical example (Dye and Gay 2003; Lipsitch, Cohen et al. 2003; Riley, Fraser et al. 2003). Because \( R_0 \) also depends on contact networks that are often poorly known, models of established diseases are commonly estimated the same way (e.g., Gani and Leach 2001). To mimic this protocol we treat the AB model as the “real world” and estimate the parameters of the DE to yield the best fit to the cumulative number of cases. We estimate the infectivities (\( i_{ES} \) and \( i_{IS} \)), and incubation time (\( \epsilon \)) by nonlinear least squares in 200 AB simulations randomly selected from the full set of 1000 in each network and heterogeneity condition, a total of 2000 calibrations (see the supplement). Results assess whether a calibrated DE model can capture the stochastic behavior of heterogeneous individuals in realistic settings with different contact networks.
**Results:** For each of the ten experimental conditions we compare the simulated patterns of diffusion on four measures relevant to public health. The maximum symptomatic infectious population (*peak prevalence*, $I_{\text{max}}$) indicates the peak load on public health infrastructure including health workers, immunization resources, hospitals and quarantine facilities. The time from initial exposure to the maximum of the infected population (the *peak time*, $T_p$) measures how quickly the epidemic spreads and therefore how long officials have to deploy those resources. The fraction of the population ultimately infected (the *final size*, $F$) measures the total burden of morbidity and mortality. Finally, we calculate the fraction of the epidemic duration in which the infectious population in the DE model falls within the envelope encompassing 95% of the AB simulations (the *envelopment fraction*, $E_{95}$). The envelopment fraction indicates what fraction of the time the trajectory of the DE falls significantly outside the range of outcomes caused by stochastic variations in the behavior of individual agents.\(^5\) To illustrate, figure 1 compares the base case DE model with a typical simulation of the AB model (in the heterogeneous scale-free case). The sample scale-free epidemic grows faster than the DE ($T_p = 37$ vs. 48 days), has similar peak prevalence ($I_{\text{max}} = 27\%$), and ultimately afflicts fewer people ($F = 85\%$ vs. 98\%).

Figure 2 shows the symptomatic infectious population, $I$, in 1000 AB simulations for each network and heterogeneity condition. Also shown are the mean of the ensemble and the trajectory of the base case DE model. Table 2 reports the results for the fitted DE models; Tables 3-5 report $T_p$, $I_{\text{max}}$, $F$ and $E_{95}$ for each condition, and compare them to the base and fitted DE models. Except for the lattice, the AB dynamics are qualitatively similar to the DE model. Initial diffusion is driven by the positive contagion feedbacks as infectious individuals spread the infection, further increasing prevalence. The epidemic peaks when the susceptible population is sufficiently depleted that the (mean) number of new cases generated by contagious individuals is less than the rate at which they are removed from the contagious population.

Departures from the DE model, while small, increase from the connected to the random, 

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\(^5\) We calculate $E_{95}$ for 125 days, when the recovered population (cumulative cases) in the base DE model settles within 2% of its final value. Longer periods further reduce the discrepancy between the two models.
scale free, small world, and lattice structures (Figure 2; tables 3-5). The degree of clustering explains these variations. In the fully connected and random networks the chance of contacts in distal regions is the same as for neighbors. The positive contagion feedback is strongest in the connected network because an infectious individual can contact everyone else, minimizing local contact overlap. In contrast, the lattice has maximal clustering. When contacts are localized in a small region of the network, an infectious individual repeatedly contacts the same neighbors. As these people become infected the chance of contacting a susceptible and generating a new case declines, slowing diffusion, even if the total susceptible population remains high.

In the deterministic DE model there is always an epidemic if $R_0 > 1$. Due to the stochastic nature of interactions in the AB model, it is possible that no epidemic occurs or that it ends early if, by chance, the few initially contagious individuals recover before generating new cases. As a measure of early burnout, table 3 reports the fraction of cases where cumulative cases remain below 10%. Early burnout ranges from 1.8% in the homogeneous connected case to 5.9% in the heterogeneous lattice. Heterogeneity raises the incidence of early burnout in each network since there is a higher chance that the first patients will have low contact rates and recover before spreading the disease. Network structure also affects early burnout. Greater contact clustering increases the probability that the epidemic burns out in a local patch of the network before it can jump to other regions, slowing diffusion and increasing the probability of global quenching.

The impact of heterogeneity is generally small, despite a factor of seven difference between the lowest and highest contact frequencies. Heterogeneity yields a slightly smaller final size, $F$, in all conditions: the mean reduction over all ten conditions is 0.09, compared to a mean standard deviation in $F$ across all conditions of 0.19—the expected difference in $F$ is small relative to the expected variation in $F$ from differences due to stochastic events during the epidemic. Similarly, heterogeneity slightly reduces $T_p$ in all conditions (by a mean of 4.5 days, compared to a mean standard deviation of 25 days). Maximum prevalence ($I_{\max}$) also falls in all conditions (by 1.5%, compared to a mean standard deviation of 5%). In heterogeneous cases the more active

6 Except for the lattice, the results are not sensitive to the 10% cutoff. The online appendix shows the histogram of final size for each network and heterogeneity condition.
individuals tend to contract the disease sooner, causing a faster take-off compared to the homogeneous case (hence earlier peak times). These more active individuals also recover sooner, reducing the mean contact frequency and hence the reproduction rate among those who remain compared to the homogeneous case. Subsequent diffusion is slower, peak prevalence is smaller, and the epidemic ends sooner, yielding fewer cumulative cases. However, these effects are small relative to the variation from simulation to simulation within each network and heterogeneity condition caused by the underlying stochastic processes for contact, emergence and recovery.

Consider now the differences between the DE and AB cases by network type.

*Fully Connected:* The fully connected network corresponds closely to the perfect mixing assumption of the DE. As expected, the base DE model closely tracks the mean of the AB simulations, never falling outside the envelope capturing 95% of the AB realizations ($E_{95} = 100\%$ for both $H = H_\epsilon$ and $H \neq H_\epsilon$). The differences in peak time and peak prevalence between the base DE and AB model are small and insignificant in both $H = H_\epsilon$ and $H \neq H_\epsilon$ conditions. Final size is not significantly different in the $H = H_\epsilon$ condition. In the $H \neq H_\epsilon$ condition $F$ is significantly smaller ($p < 0.05$), but the difference, 8%, is small relative to the 19% standard deviation of $F$ within the AB ensemble.

*Random:* Results for the random network are similar to the connected case. The trajectory of the base DE model never falls outside the 95% confidence interval defined by the AB simulations. Differences in $T_p$ and $I_{\text{max}}$ are not significant. The difference in cumulative cases is statistically significant for both heterogeneity conditions, but small relative to the variation in $F$ within each condition. The incidence of early burnout rises somewhat because the random network is sparse relative to the fully connected case, increasing contact overlap.

*Scale-Free:* Surprisingly, diffusion in the scale free network is also quite similar to the base DE, even though the scale free network departs significantly from perfect mixing. The envelopment fraction $E_{95}$ is 100% for both $H = H_\epsilon$ and $H \neq H_\epsilon$. Peak prevalence and peak time are not significantly different.

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7 The reported significance levels are based on the actual distribution of 1000 AB simulations in each experimental condition. These distributions are typically not normal (e.g., $F$ is bounded by 100%). Hence some differences in key metrics are significant even though they are small relative to the standard deviation for the AB simulations.
different from the base DE values. Though most nodes have few links, once the infection reaches a hub it quickly spreads throughout the population. However, as the hubs are removed from the infectious pool, the remaining infectious nodes have lower average contact rates, causing the epidemic to burn out at lower levels of diffusion. Yet while final size is significantly lower than the DE in both heterogeneity conditions, the differences are small relative to the standard deviation in $F$ caused by random variations in agent behavior within the ensemble of AB simulations.

*Small World:* Small world networks are highly clustered—most contacts are with immediate neighbors, and they lack highly connected hubs. Consequently diffusion is slower compared to the DE and the connected, random, and scale-free networks. Slower diffusion relative to the DE causes the envelopment fraction to fall to about 70%, and peak prevalence is significantly lower than the DE. Nevertheless, the few long-range links are sufficient to seed the epidemic throughout the population (Watts and Strogatz 1998). Thus in the $H_-$ condition, cumulative cases and peak time are not significantly different from the values of the base DE model. The main impact of heterogeneity is greater dispersion and a reduction in final size.

*Lattice:* In the one-dimensional ring lattice individuals can only contact their $k$ immediate neighbors. Hence diffusion is far slower than in the DE ($E_{95}$ is only about 55%). The epidemic advances roughly linearly in a well-defined wave-front of new cases trailed by symptomatic and then recovered individuals. Such waves are observed in the spread of plant pathogens, where transmission is mostly local, though in two dimensions more complex patterns are common (Bjornstad, Peltonen et al. 2002; Murray 2002). Because the epidemic wave front reaches a stochastic steady state in which recovery balances new cases, the probability of burnout is roughly constant over time. In the other networks early burnout quickly becomes rare as long-range links seed the epidemic in multiple locations (the supplement shows distributions of final size). Final size is much lower in the lattice than the base DE, though interestingly, the variance is higher as well, so that in the $H_-$ condition the differences in $F$ and $T_p$ are not significant ($I_{max}$ is significantly lower in both heterogeneity conditions).

*Calibrated DE Model:* In practice key parameters such as $R_0$ and incubation times are poorly constrained and are estimated by fitting models to aggregate data. Table 2 summarizes the results
of fitting the DE model to 200 randomly selected AB simulations in each experimental condition. The differences between the AB and best-fit DE models are very small. The median $R^2$ for the fit to the recovered population exceeds 0.985 in all scenarios. The infectious population nearly always remains inside the envelope containing 95% of the AB simulations (Table 5). Across all ten experimental conditions the values of $F$, $T_p$, and $I_{\text{max}}$ in the fitted models are not statistically different from the means of the AB simulations in over 90% of the cases. The DE model fits extremely well even though it is clearly mis-specified in all but the homogeneous fully connected network. As the network becomes increasingly clustered and diffusion slows, the estimated parameters adjust accordingly. As expected from the relationship between $R_0$ and final size\(^8\), for more heterogeneous, and more clustered, networks, where final size is smaller, the estimated $R_0$ is also smaller. For example, the fitted parameters for small-world networks show longer incubation times ($\epsilon$) and lower infectivity ($i_{\text{ES}}$) compared to the base DE values, yielding slower diffusion, a smaller $R_0$ and smaller final size than the base DE values.

Sensitivity to Population Size: To examine the robustness of the results to population size we repeated the analysis for $N = 50$ and 800; results are reported in the supplement.\(^9\) The results change little over a factor of 16. For most conditions, the rate of early burnout falls in larger populations, so the final fraction of the population infected is slightly larger (and therefore closer to the value of the DE). Population size has little impact on other key metrics.

Policy Analysis: Although key public health metrics generally do not differ significantly between the DE compartment model and the AB models, the response of the AB models to policies might differ. While comprehensive policy analysis is beyond the scope of this paper, we illustrate by examining the impact of quarantine (a reduction in contact rates). In the 2003 SARS epidemic both mandatory and self quarantine (where individuals who fear infection voluntarily reduce their contacts with others) increased as more cases of the disease were confirmed (Wallinga and Teunis

\(^8\) $R_0 = -\ln(1 - F)/F$ for SEIR models (Anderson and May 1991).

\(^9\) Mean links per node ($k$) must be scaled along with population. We use the scaling scheme that preserves the average distance between nodes in the random network (Newman 2003), yielding $k=6$ and $k=18$ for $N=50$ and $N=800$. 

---
For simplicity we assume contact rates fall linearly to a minimum value as the total number of confirmed cases (cumulative prevalence $P = I + R$) rises. Specifically, we model the contact frequency $c_{js}$ between infectious persons, $j \in \{E, I\}$, and susceptibles, $s \in \{S\}$, as a weighted average of the initial frequency, $c_{js}^*$, and the minimum achieved under quarantine, $c_{js}^q$:

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

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

The intensity of quarantine, $q$, rises linearly from zero to one as cumulative prevalence, $P$, 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. Quarantine is never perfect; we set the minimum contact frequency, $c_{js}^q = 0.15c_{js}^*$. This value gradually reduces $R_0$ in the DE model from 4.125 to $\approx 0.6$, similar to the reduction Wallinga and Teunis (2004) estimate for the SARS epidemic.

As expected, quarantine quenches the epidemic much earlier and greatly reduces its final size. In the DE model, quarantine cuts cumulative cases from 98% to 19% of the population. The epidemic peaks sooner and maximum prevalence of infectious individuals falls from 27% to 4.4%, greatly easing the burden on public health resources. More importantly, the impact of the policy in the AB models is similar. Table 6 shows that, except for the lattice, the means of final epidemic size ($F$) under the quarantine policy are not significantly different from the DE model. The AB and DE results are not significantly different for all other metrics in all other network and heterogeneity conditions. Except for the lattice, the differences between the base and quarantine results are similar in magnitude to the DE model and large relative to the standard deviation in the ensemble of AB simulations for each network and heterogeneity condition.

**Discussion:** Agent-based simulation adds an important new method to the modeling toolkit. Yet computational and cognitive limits require all modelers to make tradeoffs. Agent-based models allow detailed representation of individual attributes and networks of relationships, but increase computational requirements. Traditional deterministic compartment models are computationally efficient, but assume perfect mixing and homogeneity within compartments. By explicitly contrasting agent-based and deterministic differential equation models of epidemic diffusion we test the importance of relaxing the perfect mixing and agent homogeneity assumptions. With the
exception of the ring lattice, where there are no long-range contacts, there are few statistically significant differences in key public health metrics between the deterministic DE model and the mean of the AB trajectories. Mean peak times, determining how long public health officials have to respond to an epidemic, are not significantly different from the base case DE in any network or heterogeneity condition. Similarly, mean peak prevalence, determining the maximum load on health infrastructure, does not differ significantly from the base DE for the connected, random, and scale free networks. The difference in final epidemic size between the mean of the AB simulations and the base DE model is less than one standard deviation in all but the lattice. (While a pure lattice is unrealistic in modeling human diseases due to the high mobility of modern society, it may be appropriate in other contexts such as the spread of diseases among immobile plant populations.)

The AB representation does offer insight unavailable in the DE: the epidemic fizzles out in a small fraction of cases even though the underlying parameters yield an expected value for the basic reproduction rate greater than one. The more highly clustered the network, the greater the incidence of early burnout. The deterministic DE model cannot generate such behavior.

Overall, however, differences between the DE and the mean trajectory of the AB simulations are small relative to the variation in realizations of the AB model with a given topology and heterogeneity characteristics. While clustering increases the departure of the mean trajectory of the AB model from the DE, it also increases the variability of outcomes. Intuitively, the location of infectious individuals makes no difference in the fully connected homogeneous case, but matters more in highly clustered networks with greater heterogeneity. The increase in variability with clustering and heterogeneity means that the ability to identify network topology from a given trajectory does not grow as fast as the differences in means might suggest.

The differences between the DE and means of the AB simulations are also small relative to the uncertainty in key parameters. For example, $R_0$ for smallpox is estimated to be in the range 3 – 6 (Gani and Leach 2001). Varying $R_0$ over that range in the DE (by scaling both contact frequencies, $c_{ES}$ and $c_{IS}$, proportionately) causes $F$ to vary from 94.1% to 99.7%, peak prevalence to vary from 21.7% to 31.8%, and peak time to vary from 36 to 64 days. Variations in epidemic diffusion due to parameter uncertainty are comparable to the differences caused by relaxing the perfect mixing and homogeneity assumptions.
Compared to the homogeneous case, heterogeneity in individual contact rates causes slightly earlier peak times as high-contact individuals rapidly seed the epidemic, followed by lower diffusion levels as the high-contact individuals are removed, leaving those with lower average transmission probability and a smaller reproduction rate. These results are consistent with the analysis of heterogeneity for SI and SIS models (Veliov 2005). Such dynamics were also observed in the HIV epidemic, where initial diffusion was rapid in subpopulations with high contact rates. Such heterogeneity is often captured in DE models by adding additional compartments to distinguish the highly connected and hermit types.

Since parameters characterizing emerging (and some established) diseases are poorly constrained, epidemiologists typically fit models to aggregate data. We tested the impact of this protocol by treating the realizations of the AB model as the “real world” and fitting the DE model to them. Surprisingly, the deterministic DE model replicates the behavior of the stochastic agent-based simulations very well, even when contact networks are highly clustered and agents highly heterogeneous. The calibration results also highlight an important methodological issue. The parameter values obtained by fitting the aggregate model to the data from an AB simulation (and therefore from the real world) do not necessarily equal the mean of the individual-level parameters governing the micro-level interactions among agents. Aggregate parameter estimates not only capture the mean of individual attributes such as contact rates but also the impact of network structure and agent heterogeneity. Modelers often use both micro and aggregate data to parameterize both AB and DE models; the results suggest caution must be exercised in doing so, and in comparing parameter values across different models (Fahse, Wissel et al. 1998).

The results inform phenomena beyond epidemics. Analogous processes of social contagion (imitation, word of mouth, etc.) play important roles in many social and economic phenomena, from innovation diffusion to crowd behavior to marketing (Strang and Soule 1998; Rogers 2003). Moreover, modelers tackling policy issues related to innovation and product diffusion face tradeoffs on choice of modeling assumptions similar to those studying epidemics (Gibbons 2004). Therefore, the results may provide insight into the costs and benefits of agent-level disaggregation in modeling of social contagion.

How, then, should modelers choose among AB and compartment models (either
deterministic or stochastic)? The purpose, situation, time, and resources available condition the choice. If the network structure is known, stable, and unaffected by the diffusion dynamics themselves, AB models enable analysts to examine the effect of creating and removing nodes and links to simulate random failures or targeted attacks. AB models can help us understand the emergence of aggregate behavior from interaction of the elements of the system (e.g. Reynolds 1987), highlight behaviors not observed in simpler DE representations (e.g. Shnerb, Louzoun, et al. 2000) and allow for more realistic representation and analysis of stochastic behavior in a population. Such questions are harder to answer with aggregate models. Similarly, deterministic models cannot estimate the probability distribution of epidemic take-off when a population is challenged by a small number of infectious individuals; doing so requires models that explicitly capture the stochastic character of contacts. AB models are suited to that purpose, as are less computationally intensive stochastic compartmental models.

Data availability significantly affects model choice. Data on contact networks and the distribution of agent attributes are often hard to obtain and highly uncertain, requiring extensive sensitivity analysis to ensure robust results. Disaggregating the model into an AB representation without data to support the choice of network structure and agent heterogeneity increases the computational cost and dimensionality of sensitivity analysis but for many cases adds little value.

The purpose of the model also determines the risks of different methods. The costs of avoidable morbidity and mortality due to inadequate public health response are high. Some researchers therefore suggest it is prudent to use the well-mixed DE model, where diffusion is fastest, as a worst case limit (Kaplan and Lee 1990; Kaplan 1991). The same argument may suggest greater attention to network structure in studies of innovation diffusion and new product launch, where policymakers seek to promote, rather than suppress, diffusion.

Model complexity can be expanded in different directions. Modelers can add detail, disaggregating populations by location, individual attributes, and relationship networks. Alternatively they can expand the model boundary to include feedbacks with other subsystems. For example, the results reported here assume fixed parameters including network structure, contact rates and infectivities. All are actually endogenous. As prevalence increases, people change their behavior. Self quarantine and the use of safe practices disrupts contact networks, cuts
contact frequencies, and reduces the probability of transmission. From staying home, increased hand washing, and use of masks (for SARS) to abstinence, condom use, and needle exchange (for HIV), endogenous behavior change lowers $R_0$ and can have large effects on disease diffusion (Blower, Gershengorn et al. 2000). Alternatively, behavior change may amplify the epidemic: people fleeing a bioterror attack make contact tracing more difficult and may seed outbreaks in remote areas; development of more effective drugs increases risky behavior among HIV patients (Lightfoot, Swendeman et al. 2005). Such feedback effects may swamp the impact of network structure, heterogeneity, and random events and should not be excluded in favor of greater detail.

The policy test above illustrates: the reduction in $R_0$ with cumulative cases can be interpreted as endogenous self-quarantine. This feedback has a large impact on model behavior compared to the differences between the DE and AB models. Expanding the boundary of a model can have effects much larger than those introduced by disaggregation from compartments to explicit agents.

Before concluding, we consider limitations and extensions. The experiments considered the classic SEIR model. Further work should consider the robustness of results to common elaborations such as loss of immunity, nonexponential distributions for emergence and recovery, recruitment of new susceptibles, non-human disease reservoirs and vectors, etc. Note, however, that by using the classic SEIR model, with only four compartments, we maximize the difference between the aggregation assumptions of the DE and AB representations. In practical applications DE models often disaggregate the population more finely to account for heterogeneity arising from, e.g., sex, age, behavior, location, contact frequencies, assortative mixing and other attributes that vary across population segments and cause clustering in contact networks. Such disaggregation further reduces the differences between the DE and AB representations. A major challenge, however, is choosing the number and definitions of additional compartments optimally. AB simulations may be used effectively to design compartment models that capture important heterogeneity and network effects using the fewest additional compartments—if the network structure is reasonably well known and stable.10

While we examined a wide range of network structures and agent attributes, the agent models contain many parameters that could be subject to additional sensitivity analysis, including

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10 We acknowledge an anonymous reviewer for this suggestion.
the mean number of links per node, the probability of long-range links (in the small world network), and the scaling exponent (in the scale-free case). Other dimensions of heterogeneity and other networks could be examined, including networks derived from field study (Ahuja and Carley 1999). The boundary could be expanded to include endogenously the many effects that alter contact rates and network structure as an epidemic progresses, e.g. through panic or policies such as quarantine and immunization (e.g., Kaplan et al. 2003). The robustness of mitigation policies other than quarantine with respect to network type, agent heterogeneity and model boundary should be examined. Finally, while models of diffusion in other contexts such as new products and innovation adoption are similar to the SEIR family, most notably the Bass (1969) diffusion model and its extensions (e.g. Mahajan, Muller et al. 2000), we do not explicitly address the differences between AB and DE models in these contexts.

**Conclusion:** The declining cost of computation has led to an explosion of agent-based simulations, offering modelers the ability to capture individual differences in attributes, behavior, and networks of relationships. Still, no matter how powerful computers become, limited time, budget, cognitive capabilities, and client attention mean modelers must always choose whether to disaggregate to capture the attributes of individual agents, expand the model boundary to capture additional feedback processes, or keep the model simple so that it can be analyzed thoroughly. When should agent-based models be used, and when should traditional models be used? We compared agent-based and differential equation models in the context of contagious disease. From SARS to HIV to the possibility of bioterrorism, understanding the diffusion of infectious diseases is critical to public health and security. Further, analogous processes of social contagion play important roles in many social and economic phenomena, from innovation diffusion to crowd behavior.

We first compared the classic SEIR model to equivalent agent-based models with identical (mean) parameters. We tested network structures spanning a wide range of clustering, including fully connected, random, scale-free, small world, and lattice networks, each tested with homogeneous and heterogeneous agents. Surprisingly, the DE model often captures the dynamics well, even in networks that diverge significantly from perfect mixing and homogeneity. The presence of even a few long-range links or highly connected hubs can seed an epidemic throughout the population, leading to diffusion patterns that, in most cases, are not significantly different from
the dynamics of the differential equation model.

Second, in practical applications, parameters governing diffusion are poorly constrained by data, for both emerging and established diseases (and for new products). Hence model parameters are typically estimated from data on incidence and prevalence as diffusion occurs. We assessed this practice by treating individual realizations of the agent-based simulations as the “real world” and estimating key parameters of the DE model from these “data”. The calibrated DE model captures the dynamics extremely well in all network and heterogeneity conditions tested. Overall, the differences between the agent-based and differential equation models are small relative to the uncertainty in parameters, and relative to the variation in outcomes caused by stochastic events. The results suggest extensive disaggregation may not be warranted unless detailed data characterizing network structure are available, that structure is stable, and the computational burden does not limit sensitivity analysis or the inclusion of other key feedbacks that may condition the dynamics.

References:


Table 1. Base case parameters. The supplementary material documents the construction of each network used in the AB simulations.

<table>
<thead>
<tr>
<th>Parameter (dimensionless)</th>
<th>Parameter</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectivity, Exposed</td>
<td>$i_{ES}$</td>
<td>0.05</td>
</tr>
<tr>
<td>Infectivity, Infectious</td>
<td>$i_{IS}$</td>
<td>0.06</td>
</tr>
<tr>
<td>Basic reproduction rate</td>
<td>$R_0$</td>
<td>4.125</td>
</tr>
<tr>
<td>Average links per node</td>
<td>$k$</td>
<td>10</td>
</tr>
<tr>
<td>Prob of long-range links (SW)</td>
<td>$p_{sw}$</td>
<td>0.05</td>
</tr>
<tr>
<td>Scaling exponent (scale-free)</td>
<td>$\gamma$</td>
<td>2.60</td>
</tr>
<tr>
<td>Total population</td>
<td>$N$</td>
<td>200 Person</td>
</tr>
<tr>
<td>Contact rate, Exposed</td>
<td>$c_{ES}$</td>
<td>4 1/Day</td>
</tr>
<tr>
<td>Contact rate, Infectious</td>
<td>$c_{IS}$</td>
<td>1.25 1/Day</td>
</tr>
<tr>
<td>Average incubation time</td>
<td>$\kappa$</td>
<td>15 Day</td>
</tr>
<tr>
<td>Average duration of illness</td>
<td>$\delta$</td>
<td>15 Day</td>
</tr>
</tbody>
</table>

Table 2. Median estimated basic reproduction number, $R_0 = c_{ES} i_{ES} \kappa + c_{IS} i_{IS} \delta$, for the DE model fit to the recovered population in 200 randomly selected runs of the AB model for each cell of the experimental design.

<table>
<thead>
<tr>
<th>Parameter</th>
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<th>Random</th>
<th>Scale-free</th>
<th>Small World</th>
<th>Lattice</th>
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</thead>
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<td>$H_s$</td>
<td></td>
<td></td>
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<td>$H_r$</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Median</td>
<td>4.19</td>
<td>2.96</td>
<td>3.09</td>
<td>2.55</td>
<td>3.12</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>1.71</td>
<td>0.62</td>
<td>0.58</td>
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<td>0.70</td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.999</td>
<td>0.999</td>
<td>0.999</td>
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</tr>
<tr>
<td>$\sigma$</td>
<td>0.025</td>
<td>0.049</td>
<td>0.017</td>
<td>0.050</td>
<td>0.019</td>
</tr>
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</table>

Table 3. Mean and standard deviation of Final Size (F) for the AB simulations. */** indicates the base DE value of F (0.98) falls outside the 95/99% confidence bound defined by the ensemble of AB simulations. These distributions are typically nonnormal (e.g., F is bounded by 100%). Assuming normality would further reduce the significance of the differences between the AB and DE models. (2) The percentage of AB simulations with $F < 0.10$ indicates the prevalence of early burnout. (3) Mean and standard deviation of F for the fitted DE models.

<table>
<thead>
<tr>
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<th>Scale-free</th>
<th>Small World</th>
<th>Lattice</th>
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<td>$H_s$</td>
<td>$H_a$</td>
<td>$H_r$</td>
<td>$H_s$</td>
<td>$H_a$</td>
</tr>
<tr>
<td>1 AB Mean ($\sigma$)</td>
<td>0.97</td>
<td>(0.13)</td>
<td>0.90*</td>
<td>(0.19)</td>
<td>0.92*</td>
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<tr>
<td>2 % $F &lt; 0.10$</td>
<td>1.8</td>
<td>4.4</td>
<td>2.7</td>
<td>3.8</td>
<td>2.9</td>
</tr>
<tr>
<td>3 Fitted DE $\mu$ ($\sigma$)</td>
<td>0.98</td>
<td>(0.07)</td>
<td>0.91</td>
<td>(0.17)</td>
<td>0.92</td>
</tr>
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</table>
**Table 4.** Mean and standard deviation of peak time $T_p$ and peak prevalence $I_{\text{max}}$ in 1000 simulations of the AB model for each experimental condition. In the base DE model, */** indicates the values of in the base DE model ($T_p = 48$ days and $I_{\text{max}} = 27.1\%$) fall outside the 95/99% confidence bounds defined by the ensemble of AB simulations. Also reported are the mean and standard deviation of $T_p$ and $I_{\text{max}}$ for the fitted DE models.

<table>
<thead>
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<tr>
<td>Peak Time, $T_p$ (Days)</td>
<td>$H_s$</td>
<td>$H_s$</td>
<td>$H_s$</td>
<td>$H_s$</td>
<td>$H_s$</td>
</tr>
<tr>
<td>AB $\mu$</td>
<td>49.8</td>
<td>44.9</td>
<td>52.8</td>
<td>49.5</td>
<td>60.6</td>
</tr>
<tr>
<td>AB $\sigma$</td>
<td>10.8</td>
<td>12.4</td>
<td>13.4</td>
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<tr>
<td>Fitted DE $\mu$</td>
<td>51.3</td>
<td>49.6</td>
<td>56.6</td>
<td>58</td>
<td>62.9</td>
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<tr>
<td>Fitted DE $\sigma$</td>
<td>9</td>
<td>21.3</td>
<td>21.3</td>
<td>37.7</td>
<td>23.2</td>
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<tr>
<td>Peak Prev $I_{\text{max}}$ (%)</td>
<td>$H_s$</td>
<td>$H_s$</td>
<td>$H_s$</td>
<td>$H_s$</td>
<td>$H_s$</td>
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<tr>
<td>AB $\mu$</td>
<td>29.1</td>
<td>27.1</td>
<td>26.5</td>
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<tr>
<td>AB $\sigma$</td>
<td>4.9</td>
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<td>5.2</td>
<td>5.6</td>
<td>5.9</td>
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<tr>
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<td>26.7</td>
<td>24.6</td>
<td>24.2</td>
<td>24.6</td>
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<tr>
<td>Fitted DE $\sigma$</td>
<td>2.8</td>
<td>13.2</td>
<td>9.6</td>
<td>18.1</td>
<td>5.2</td>
</tr>
</tbody>
</table>

**Table 5.** Envelopment fraction $E_{95}$. The fraction of the first 125 days (the time required for cumulative cases to settle within 2% of its final value in the base DE) in which the infectious population falls within the 95% confidence interval created by the ensemble of AB simulations in each network and heterogeneity scenario. Reported for both the base and fitted DE cases.

<table>
<thead>
<tr>
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<tr>
<td>Base DE</td>
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<td>$H_s$</td>
<td>$H_s$</td>
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<tr>
<td></td>
<td>1.00</td>
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<td>1.00</td>
<td>1.00</td>
<td>0.71</td>
</tr>
<tr>
<td>Fitted DE $\sigma$</td>
<td>(0.07)</td>
<td>(0.09)</td>
<td>(0.04)</td>
<td>(0.05)</td>
<td>(0.04)</td>
</tr>
</tbody>
</table>
Table 6. Key public health metrics for simulations of the quarantine policy. */** indicates the DE simulation 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 $I_{\text{max}} = 4.43\%$.

<table>
<thead>
<tr>
<th>Metric</th>
<th>Connected</th>
<th>Random</th>
<th>Scale-free</th>
<th>Small World</th>
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<tr>
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<td>$H_s$</td>
</tr>
<tr>
<td>Final Size $F$</td>
<td>$\mu$ 0.215</td>
<td>0.249</td>
<td>0.157</td>
<td>0.201</td>
<td>0.148</td>
</tr>
<tr>
<td></td>
<td>$\sigma$ 0.084</td>
<td>0.091</td>
<td>0.064</td>
<td>0.088</td>
<td>0.062</td>
</tr>
<tr>
<td>Peak Time $T_p$</td>
<td>$\mu$ 35.0</td>
<td>36.1</td>
<td>33.1</td>
<td>34.6</td>
<td>34.9</td>
</tr>
<tr>
<td></td>
<td>$\sigma$ 15.3</td>
<td>15.9</td>
<td>14.8</td>
<td>16.7</td>
<td>15.7</td>
</tr>
<tr>
<td>Peak Prev $I_{\text{max}}$</td>
<td>$\mu$ 6.42</td>
<td>7.28</td>
<td>5.17</td>
<td>6.15</td>
<td>4.98</td>
</tr>
<tr>
<td></td>
<td>$\sigma$ 2.40</td>
<td>2.66</td>
<td>1.99</td>
<td>2.55</td>
<td>1.98</td>
</tr>
</tbody>
</table>

Figure 1. Left: DE model with base parameters (Table 1), showing prevalence for each stage. Right: Typical simulation of the equivalent AB model (the heterogeneous condition of the scale free network).

Figure 2 (next page): Comparison of the DE SEIR model to the AB model. Graphs show symptomatic cases as % of population ($I/N$). Each panel shows 1000 simulations of the AB model for each network and heterogeneity condition. The thin black line is the base case DE model with parameters as in Table 1. The thick red lines show the mean of the AB simulations. Also shown are the envelopes encompassing 50%, 75% and 95% of the AB simulations.