AM227 Final Project

Nicole Swartwood
Christian Testa

December 2018

1 Abstract

For our final project we investigated what impact adding stochasticity and spatial dimensions have in the simulation of a basic epidemiological model. We found that the addition of stochasticity or spatial dimensions to the model resulted in simulated epidemics which often took on different trajectories than the base model.

2 Background

Epidemiological data is mired with features that are difficult to model including jagged, seemingly unpredictable trends over time. Further, such data is confounded by many factors such as the natural environment in which the disease spreads and human behavioral patterns which help or hinder the disease in propagation. These features of the data often make it difficult to fit models to the data.

In our project, we will take one of the oldest and most foundational approaches in epidemiology, that of compartmental modeling, and update the approach by adding stochasticity and spatiality.

We added stochasticity to our model because while the average transmission probability may inform something about the overall trajectory of an epidemic, the real world is noisy and often includes large perturbations from the average, especially when it comes to human behavior. Moreover, there are scientists who hypothesize that stochastic resonance may play a significant part in disease dynamics.

The reason we chose to investigate adding spatiality was that we felt there was room for application of the moral of the Lattice-Boltzmann schema we learned about in class: By statistically describing the microscopic interactions of systems with many individual agents, one can achieve a significantly more accurate simulation for less computational cost compared to many other simulation approaches.

Most epidemiological models are either individual based or compartmental. Either they simulate each individual agent and all of their behaviors, often

making for a computational nightmare at large scale, or they lump the population into compartments representative of their disease status, demographics, or other broad-scale descriptors, ignoring any microscopic dynamics. By making a model divided into spatial sub-compartments, we hope that we can capture more of the population heterogeneity originating at the individual-interaction scale, while still saving computational cost by not modeling each individual agent.

2.1 Basic SIRS Model

One of the most basic epidemiological models is a box model that consists of three primary compartments: the susceptible S, the infected I, and the recovered R populations. These populations lend the model its name: the Susceptible-Infected-Recovered-Susceptible, or SIRS, model. This model assumes that the population is closed, that is, there are no in- (births, immigration) or out- (death, emigration) flows. The sum of the compartments must be equal to the total population, N.

The transitions between these compartments are dictated by three rates: infection rate λ, which describes the probability of infection from a direct contact with an individual with the disease; the recovery rate ρ, which describes cure of disease; and the rate of waning immunity γ, which describes the loss of the temporary immunity granted from infection. Applying these rates we have the following system of coupled equations.

$$\dot{S} = -\lambda \frac{SI}{N} + \gamma R \tag{1}$$

$$\dot{I} = \lambda \frac{SI}{N} - \rho I \tag{2}$$

$$\dot{R} = \rho I - \gamma R \tag{3}$$

3 Methods

To estimate the impact of adding spatial subcompartments and stochasticity, we created four experimental models for comparison: stochastic and deterministic 0-dimensional models and stochastic and deterministic 2-dimensional models. We refer hereafter to the basic SIRS model as 0-dimensional because each of the population states is a constant number at a given point in time whereas the 2-dimensional model expands each of these compartments with respect to space.

In the case of the 2D models, we created a 20x20 lattice initialized with 10% of individuals in each of these grid-cells. In order to operationalize the spatiality in the model the individuals in a grid point were not only exposed to the level of infection in their specific grid point, but also the 8 surrounding grid points. To operationalize the stochasticity, we added a random process ξ to λ so that the equations become $\dot{S} = -(\lambda + \xi)\frac{SI}{N} + \gamma R$, $\dot{I} = (\lambda + \xi)\frac{SI}{N} - \rho I$, and $\dot{R}$ remains

unchanged. In the 2D stochastic model, we not only added this term to each of the grid-cell's corresponding stochastic equation, but also scaled the noise in all grid cells to have the value of another random process in time (i.e. $\sum_{i,j} \xi = \zeta$). This was done to reflect the fact that in human behavior there are not only local deviations from the average, but also global deviations.

In the 2-dimensional models, we used fixed boundary conditions which always contain a totally susceptible population with no infected or recovered individuals.

Each experimental model ran 500 time-steps after initialization with the goal of reaching a quasi-steady state. For each model comparison, we initialized each model with the same parameter values. In the spatial (2D) models the infected population is randomly distributed so that the total infected population is ten percent of the total population (N).

To compare the results of the 0-dimensional and the 2-dimensional models we summed the total number of infections in all grid-cells and similarly calculated the total population size in all grid-cells to compute the infected fraction of the total population at a given time. This metric was then plotted for each of the model simulations, with confidence intervals calculated from 100 simulations for those models with stochasticity.

4 Data Results

In using our models to simulate the trajectories of epidemics with various parameters, we discovered that while often these models have similar behaviors, there are some scenarios in which they become quite dissimilar.

4.1 Model Comparison 1

First we present here an example where the simulated epidemics are more or less the same throughout all four models. It is interesting to note that the noise is strongly dampened in the 2D model, although we will see that this is not always the case.

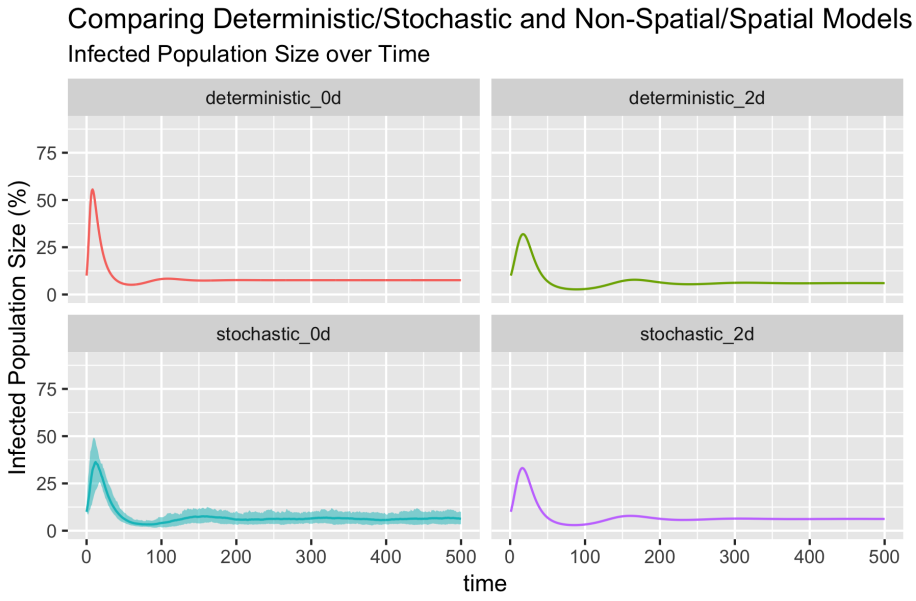

Figure 1: Model Solution Trajectories

λ	0.3	Transmission Rate
ρ	0.1	Removal/Recovery Rate
γ	0.01	End of Temporary Immunity

4.2 Model Comparison 2

Here we have an example where the 2-dimensional models' solutions are significantly different from those of the 0-dimensional models. While it looks like the 2-dimensional models do have a rapidly decaying level of infection, it appears that the steady-state they reach is not 0. Rather, they equilibriate with a very low level of disease prevalence.

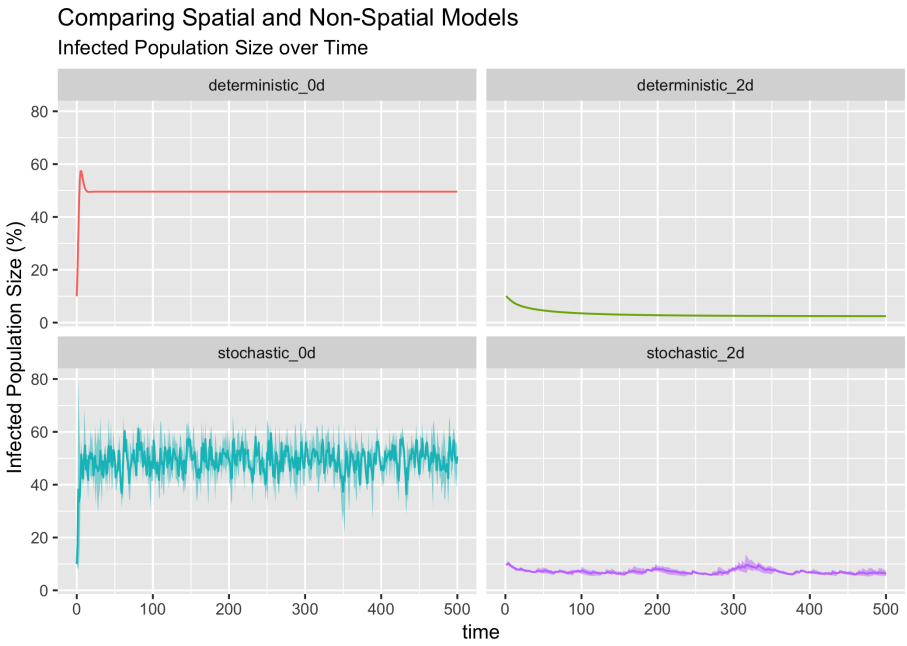

Figure 2: Model Solution Trajectories

λ	0.2	Transmission Rate
ρ	0.19	Removal/Recovery Rate
γ	0.3	End of Temporary Immunity

4.3 Model Comparison 3

In this final set of model simulations, we see that there are even parameters for which neither the stochastic 0D or the deterministic 2D model resemble the deterministic 0D model, and yet the deterministic 0D model and the stochastic 2D model are quite similar in their trajectories.

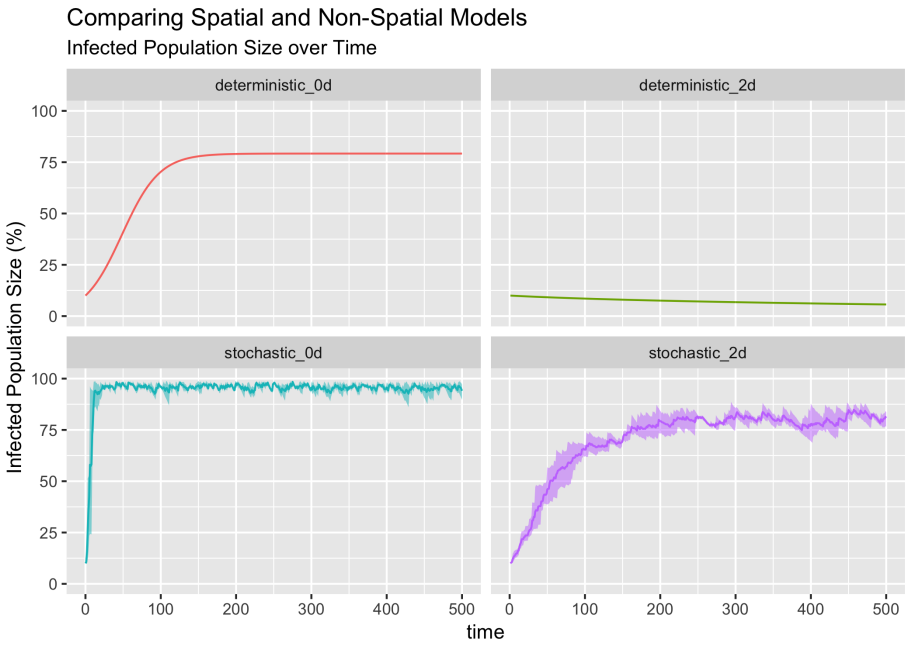

Figure 3: Model Solution Trajectories

λ	0.1	Transmission Rate
ρ	0.1	Removal/Recovery Rate
γ	0.99	End of Temporary Immunity

4.4 Analyzing the Impact of Initial Distributions

Given that the 2-dimensional models can be initialized with many different starting conditions which are all equivalent with respect to the fraction of the population which is infected, we thought it was worthwhile to demonstrate the utility of the 2-dimensional models by showing that the trajectory of a simulated epidemic can be strongly affected by the initial starting conditions.

These are the number of infected individuals in epidemics with an initial distribution of infected individuals in a uniform distribution (flat), a concentrated distribution in the center, a checkerboard pattern, a normal distribution (gaussian), and with infections starting from the corners of the simulation.

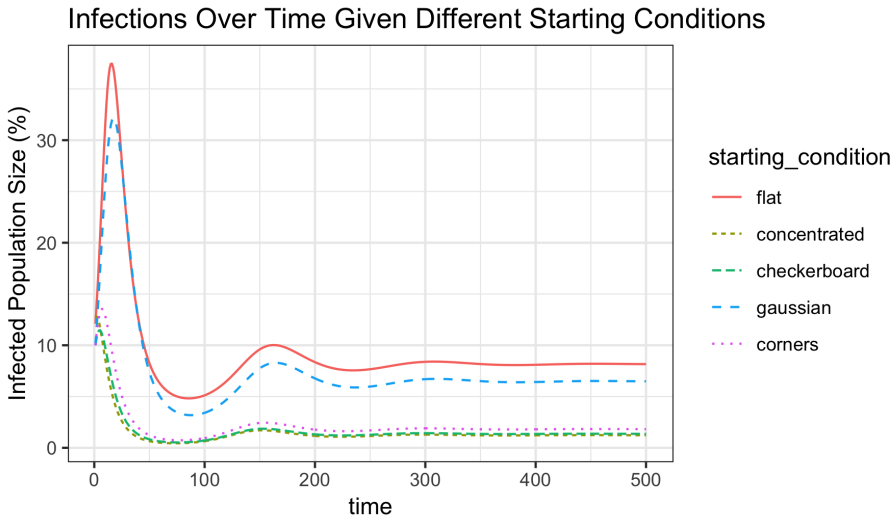

Figure 4: The impact of different initial distributions of infection

5 Conclusions

Figures 1-3 show the average and a 95% confidence interval from each of the experimental models for 3 different comparison exercises. In Figure 1, the confidence intervals in the bottom left plot (the stochastic 0D model) are clearly visible, while the confidence intervals in the bottom right plot (the stochastic 2D model) are not at all visible. There is even greater variability in Figure 2 in the stochastic 0D model, but still the noise is barely visible in the stochastic 2d

model. Finally, in Figure 3 it becomes clear that noise can and does affect the trajectory of the stochastic 2D significantly. Upon further analysis, it appears that the degree to which noise affects the stochastic 2D model is particularly correlated with how quickly the temporary immunity is lost.

Secondarily, we find that stochasticity affects the results most strongly when the transmission rate and the recovery rate are very similar. This is evidenced in Figures 2 and 3. In Figure 2, we see that once the stochastic 0D model settles into the 40-60% region, it continues to oscillate rapidly and jaggedly around. In Figure 3 we see that while the stochastic 0-dimensional model takes some time to equilibriate, the added stochasticity quickly increases the disease prevalence in the population to nearly 100%.

In Figure 4 we present the differences in the modeled epidemic trajectory due to differences in the initial spatial distribution of infections which cannot be represented in 0-dimensional models. We find that some initial conditions yield relatively tame epidemics, only increasing from their initial conditions slightly, while others increase rapidly to more than 3-fold their initial infection level and equilibriate to a steady state with a significantly higher fraction of the population infected.

From our model comparison, we conclude that stochasticity and spatiality can play a significant and large role in the trajectory of an epidemic which is often overlooked in simplistic models.

6 Discussion

Model parsimony and the principle of Occam's razor would indicate that in the absence of conflicting data, the simplest model which can fit the data should be considered the best. However, it is also clear that when a model makes simplifications about the underlying dynamics of a system, relying on such a model's predictions may be ill-advised. Given that the simplifications that the deterministic 0-dimensional model presented makes about disease dynamics yield such a stark difference when updated with slightly more realistic assumptions, we believe that it is worthwhile to pursue further modeling studies with stochasticity and spatiality features considered carefully. Moreover, further investigation is needed into when a simpler model without stochasticity or spatiality could be sufficient, and when stochasticity or spatiality are critically necessary for a modeling study.

These alternative models also suggest practical implications in the field of emergency preparedness. Not only are the predicted experimental epidemics often of a different magnitude, but they also span different lengths of time, recurring in different patterns. These changes have important implications for resource allocation and management. A useful expansion of this project would be to explicitly model the outflow of resources and their restock via various production means.

The current study explored the usefulness of these approaches in an abstract setting. An application of these methods using model parameters from true

disease dynamics, such as influenza, which is both influenced by temporal and spatial trends should be done to realize the full applicability of our method.

Lastly, we conclude that the approach and application of statistical physics is worth investigating further in the context of epidemiological modeling. Infectious disease trends span many scales, including individual interactions, transportation across continents, and airborne particles which disperse in the atmosphere. With sophisticated modeling approaches and physics inspired statistics perhaps disease dynamics occurring at such impossibly different scales can be more fully understood.