Climate 410: Lab 1 Report
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Introduction

The purpose of this lab was to create a Python model that can be applied to different natural phenomena, specifically, the spread of wildfire and the spread of disease. Wildfire models are helpful in predicting how quickly wildfire will spread, and which areas are in the most danger. Similarly disease models can tell us how much of a threat to the population a disease may present and the effect early vaccination will have. In both cases, a simplified model can be created by representing an area using a grid where fire or disease, indicated by a numerical value representing the status of the square, can spread to adjacent squares. By creating such a model, I sought to answer two questions regarding wildfire spread: how does the spread of wildfire depend on the probability that the fire will spread at each location and how does it depend on initial forest density? I then modified the model slightly to represent the spread of disease in order to answer the following two questions: how is disease spread affected by the mortality rate of the disease and how is it affected by early vaccine rates (immunity)?

Methods

In order to create a model that can represent the spread of wildfire and disease, I created a three-dimensional array representing an area of a specified size at multiple different timesteps. For the forest model, each element in the array can have a value of 1, meaning bare, 2, meaning forest, or 3, meaning burning. At time 0, all of the squares are initialized to forest. A certain number of squares will then be set to bare based on the probability of a square starting bare, specified by p_bare. Then, a certain number of squares will be set to burning based on the probability of a square starting on fire, specified by p_start. For the next timestep, squares that were previously bare remain bare, forest squares that were previously adjacent to squares on fire may catch fire, depending on p_spread, and squares that were previously on fire are set to bare. The model will continue to run, plotting the forest grid after each timestep, until there is no more fire or the maximum number of timesteps, specified by nstep, is reached.

To test the model, I started with a simple case of wildfire spread across a 3x3 gridded area. In this test, all squares started at forest, except for the center square, which was set to burning. P_spread was set to 1.0, indicating 100% chance of spread. Figure 1 shows that the model output from this test at iteration 0, 1, and 2 display the behavior that is expected for this test. A second test was conducted with the same initial conditions, except with a 3x5 grid. Figure 2 shows that the model output from this test at iteration 0, 1, and 2 also display correct behavior.

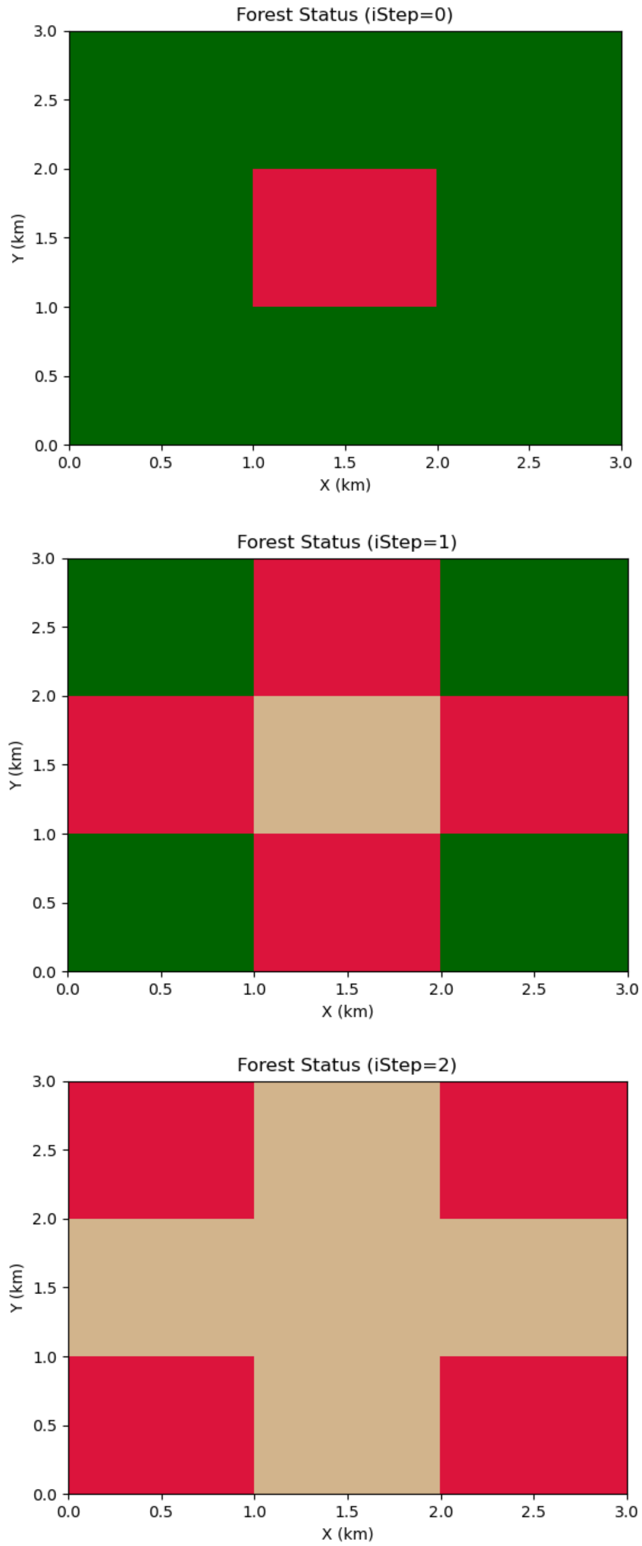

Figure 1: 3x3 Wildfire Model Iteration 0-2

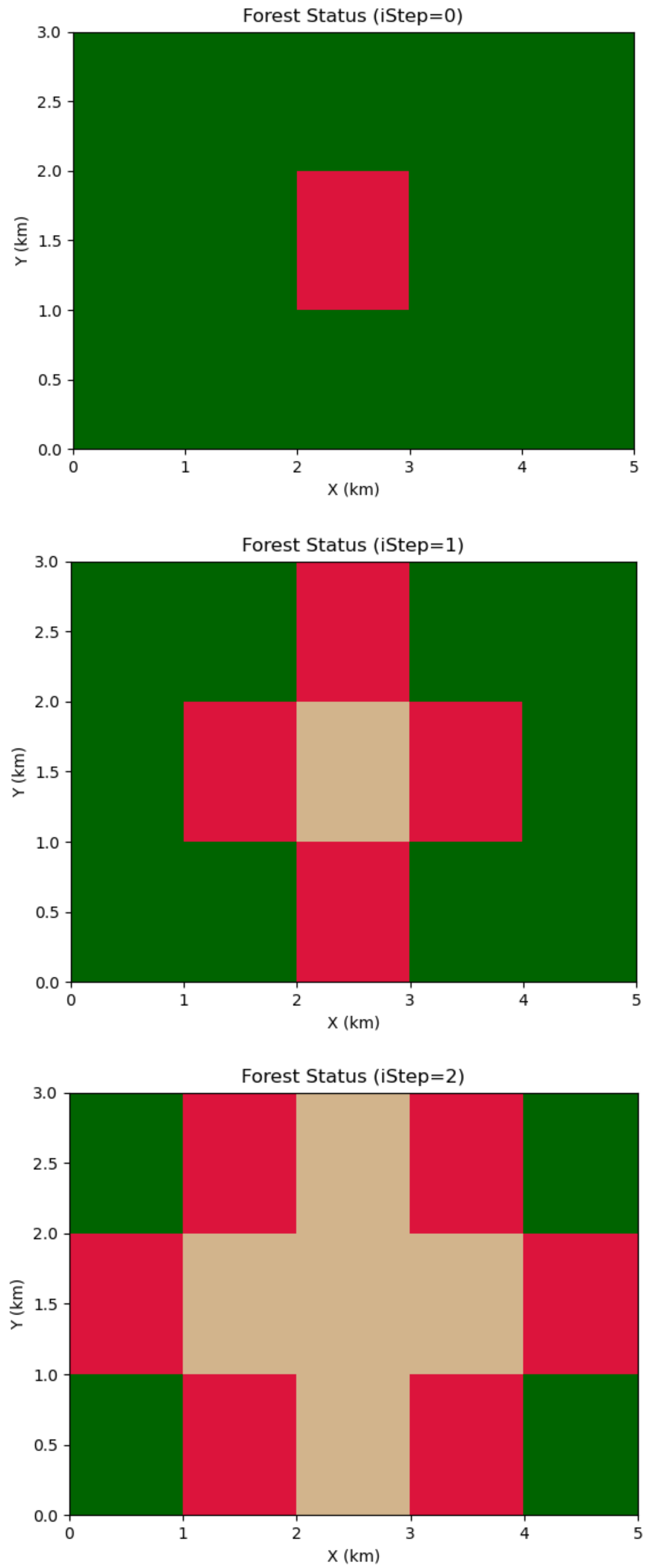

Figure 2: 3x5 Wildfire Model Iteration 0-2

To determine the relationship between wildfire spread and the probability that the fire will spread to a given square, I ran a set of experiments with a 100x100 grid. In choosing a grid size, the benefit of more squares in lowering the impact of outliers had to be weighed against the cost of increased size and memory. I found that 10,000 squares was a sufficient amount to achieve repeatable results without overwhelming my computer. A p_start of 0.001 was chosen so that an average of 10 squares in the model would begin on fire. This p_start was high enough that at least a few squares began on fire in each run, but low enough to emphasize the spread of the fire in the results over the number of squares beginning on fire. P_bare was set to 0.0, so that the effect of varying p_spread could be examined without being affected by forest density. The model was run with a p_spread ranging from 0 to 1 in 0.1 size steps. The number of timesteps for each run to reach a state where no fire was present and the percent of the final grid that was left bare was recorded for each run. The same setup was used to determine the relationship between wildfire spread and initial forest density. However, instead of varying p_spread, p_spread was fixed at 1.0 and p_bare was varied from 0 to 1 in 0.1 size timesteps. The same two quantities were recorded for each run.

To adapt the model for the spread of disease, I used the same values as the wildfire model, with different interpretations: 1 means immune, 2 means healthy, and 3 means sick. I also added a new status with a value of 0 to indicate that someone had died from the disease. In this model, p_start represents the probability that someone will start with the disease, p_spread represents the probability of spread for the disease, and p_bare, which is interpreted as p_immune, represents the probability someone will start with immunity. The model is run exactly the same as the wildfire model except that p_fatal can also be specified to determine the probability that someone will die after being infected with the disease rather than become immune.

Another set of experiments was run to test the relationship between disease spread and disease mortality rate. Once again, a 100x100 grid and p_start of 0.001 were used. P_spread was fixed at 1.0 and p_immune (same as p_bare from forest model) was fixed at 0.0 to isolate the effect of p_fatal, which was varied from 0 to 1 in 0.1 size steps. The number of timesteps for each run to reach a state where no disease was present, the percent of the final grid that ended immune, and the percent that ended dead was recorded for each run. The same setup was used to test the relationship between disease spread and early vaccination, except that p_immune was varied from 0 to 1 in 0.1 size steps to represent early vaccination rates. P_fatal was fixed at 0.5, so that the number of fatalities could be compared to the number that achieved immunity. The same three quantities were measured as the previous disease model experiment.

Results

Figures 3 and 4 show the results of the first set of tests on the wildfire model, aiming to determine the relationship between wildfire spread and probability of spread. Figure 3 shows that between a p_spread of 0.0 and 0.5, an increase in the probability of spread of wildfire relates to the fire taking more time to die out. Above a p_spread of 0.5, the number of timesteps is quite variable with increasing p_spread, finally approaching 50 as p_spread approaches 1. Increases in p_spread are shown in Figure 4 to relate to increases in the percent of the forest that ends up bare. From a p_spread of 0.4 to p_spread of around 0.6 is where the greatest rate of increase in percent bare can be seen. Below 0.4, the percent bare remains at or near 0%, while above 0.6, the percent bare reaches 100%.

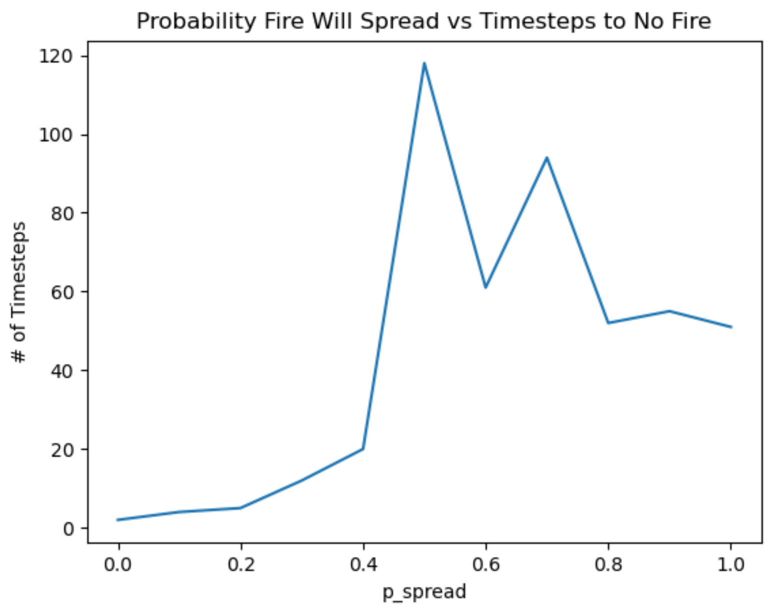

Figure 3: Effect of Probability of Wildfire Spread on Time Until Fire Burns Out

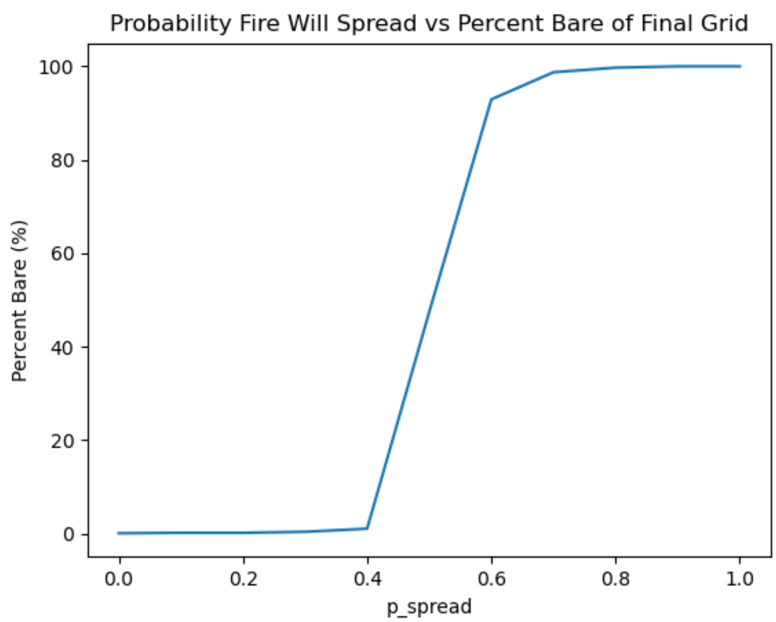

Figure 4: Effect of Probability of Wildfire Spread on Final Forest Density

Figures 5 and 6 show the results of the set of tests which aim to determine the relationship between wildfire spread and initial forest density. Figure 5 shows that an increase in the probability that a square starts bare generally relates to an increase in the number of timesteps for p_bare between 0.0 and 0.4, but a decrease in the number of timesteps for p_bare 0.4 and 1.0. Furthermore, the final percent of bare squares decreases with increasing p_bare for p_bare between 0.0 and 0.5 according to Figure 6. For p_bare between 0.5 and 1.0, it is shown to be increasing with increasing p_bare.

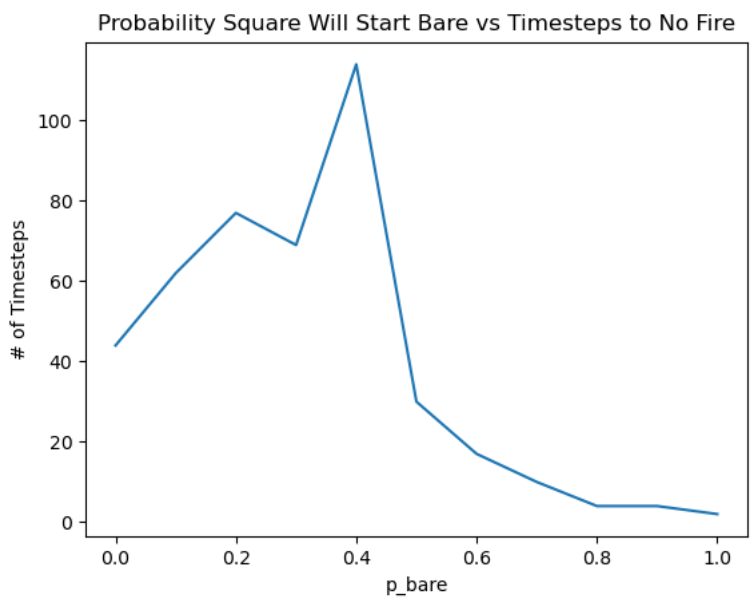

Figure 5: Effect of Initial Forest Density on Time Until Fire Burns Out

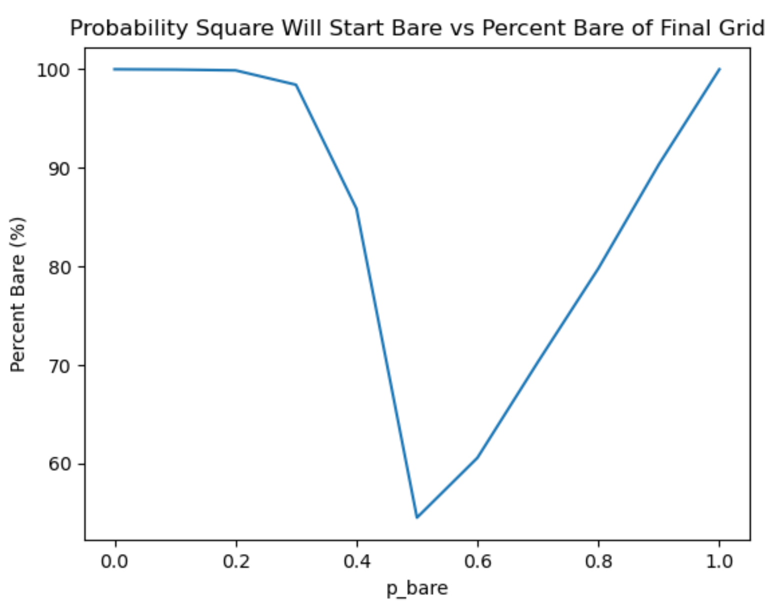

Figure 6: Effect of Initial Forest Density on Final Forest Density

Figures 7, 8, and 9 show the results of the first set of tests on the disease model which aim to determine the effect of disease's mortality rate on its spread. The probability that the disease is fatal does not appear to be correlated with the amount of time it takes for the disease to be eradicated. This is shown by Figure in how the number of timesteps increases and decreases seemingly randomly with increasing p_fatal. Figures 7 and 8 show that as p_fatal increases, the percentage of the final population that dies increases linearly while the percentage that becomes immune decreases linearly at the same rate.

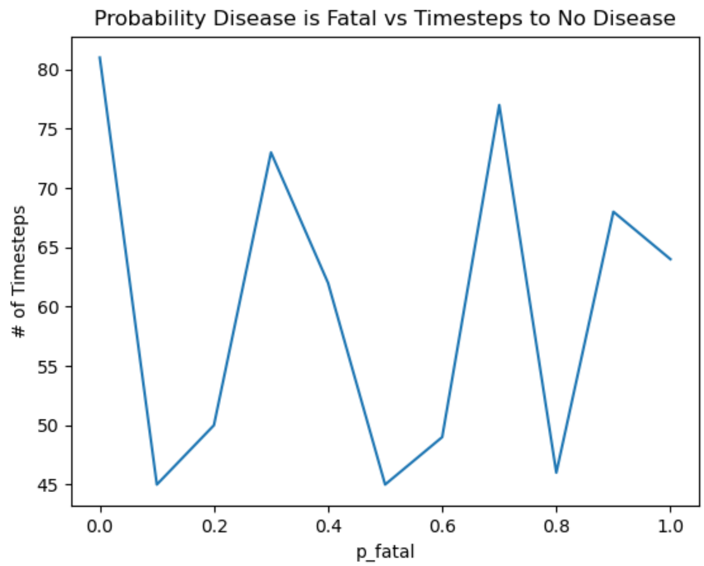

Figure 7: Effect of Disease Mortality Rate on Time Until Disease is Eradicated

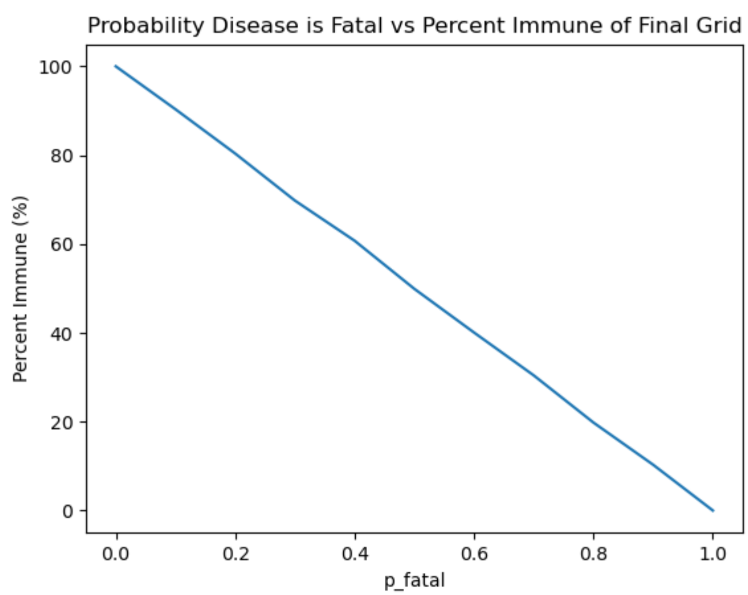

Figure 8: Effect of Disease Mortality Rate on Percent Immunity of Final Population

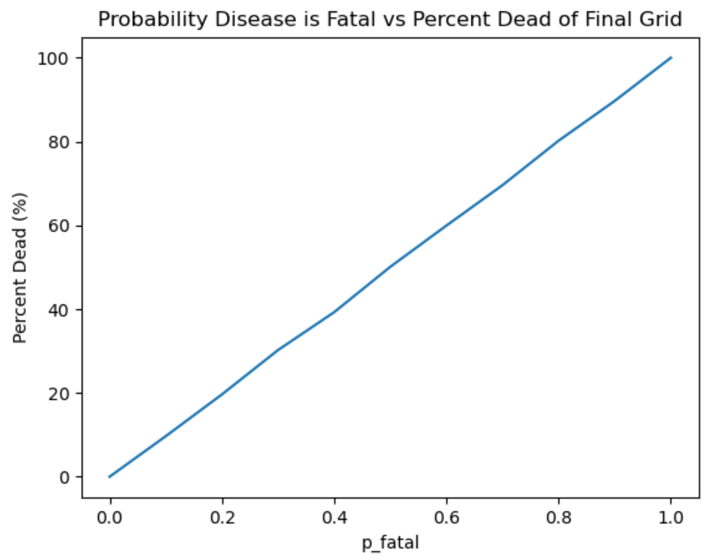

Figure 8: Effect of Disease Mortality Rate on Percent Dead of Final Population

Figures 10, 11, and 12 show the results of the set of tests which aim to determine the effect of early vaccination on disease spread. Figure 10 shows that below a probability of initial immunity of 0.4, the time until the disease disappears peaks at a p_immune of 0.4, and then decreases with increasing p_immune above 0.4. While, according to Figure 11, the percent of the final population with immunity appears to be generally increasing with p_immune, there is an exception between p_immune of 0.4 and 0.5 where the final immune percentage decreases. The percentage of the population that dies is shown in Figure 12 to be always decreasing with increasing p_immune until reaching 0% at a p_immune of 0.7.

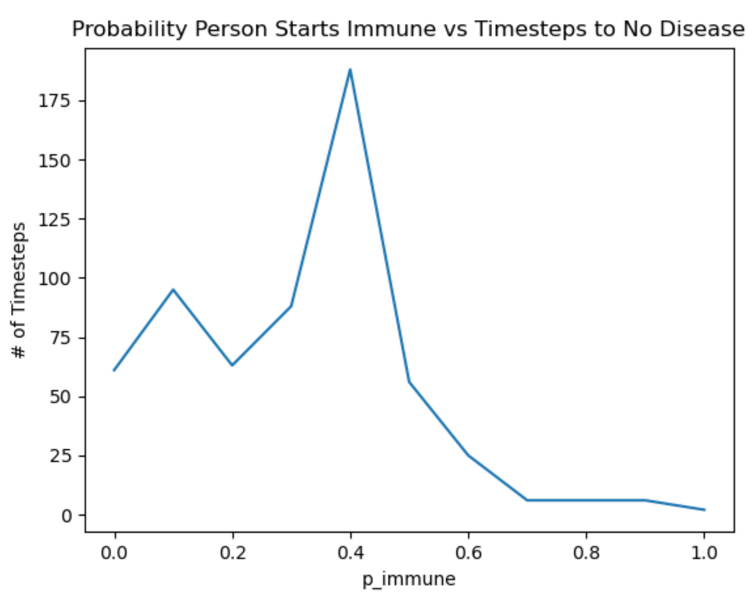

Figure 10: Effect of Initial Immunity on Time Until Disease is Eradicated

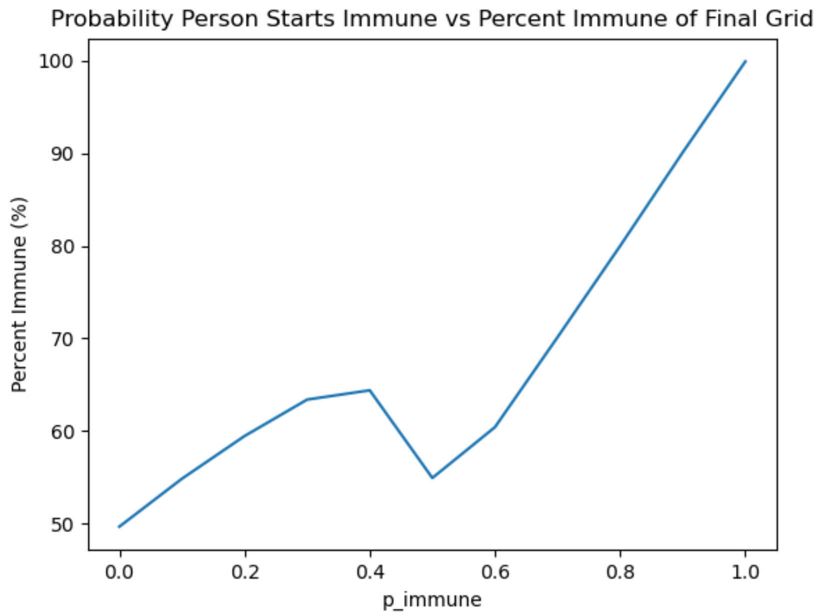

Figure 11: Effect of Initial Immunity on on Percent Immunity of Final Population

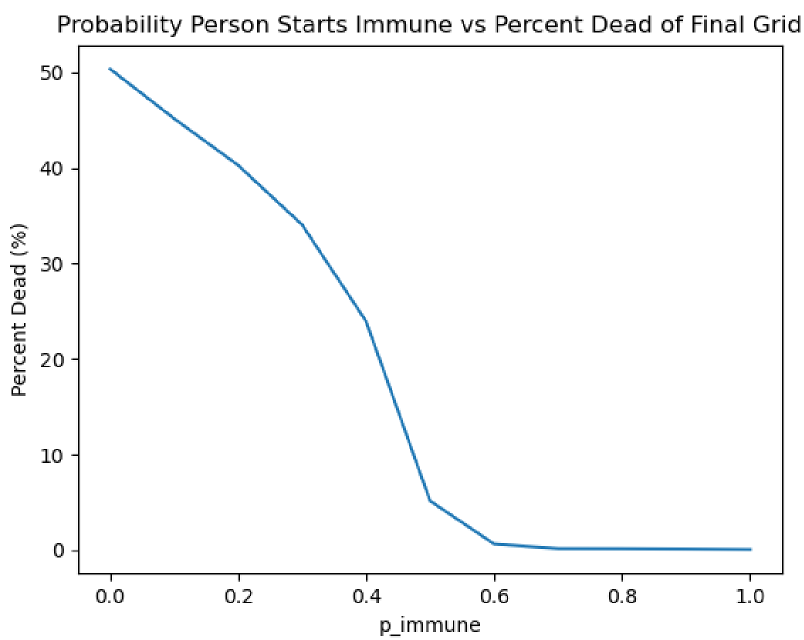

Figure 12: Effect of Initial Immunity on on Percent Dead of Final Population

Discussion and Conclusions

The first wildfire model experiment, which was conducted to determine the relationship between wildfire spread and the probability of spread, revealed that increasing the probability of spread causes more burnt squares in the final grid. This is because the fire has a greater chance of spreading when p_spread is higher. When the fire does not spread much, it burns out quickly. This explains why for p_spread less than 0.5, the fire burns out quicker as p_spread decreases. The number of timesteps reaches a maximum when p_spread is 0.5 because the chance of spreading is high enough that the fire will continue to spread until it reaches nearly all of the squares, but low enough that it will take a long time to do so.

The next wildfire model experiment sought to determine the relationship between initial forest density and wildfire spread. This experiment revealed that low forest density hinders the spread of wildfire. When most of the squares are bare, the fire has nowhere to burn, and will therefore die out. Thus, the model ran more quickly when p_bare was high. The model took the longest when p_bare was 0.4 because this resulted in enough forest squares for the fire to keep burning, but could only spread to very few squares at a time. The p_bare that resulted in the fewest bare squares at the end was 0.5 because this was the smallest bare percentage that was still large enough to put out the fire fairly quickly.

The first experiment conducted with the disease model sought to determine the relationship between the mortality rate of a disease and its spread. The probability that the disease was fatal did not have an effect on the runtime of the model because the disease would spread the same whether the host lived or died. Future experiments could be run where the probability of spread is affected by whether the person is killed by the disease in order to establish a more nuanced relationship. The mortality rate of the disease did affect the ratio of deaths to immunity, which was to be expected.

The final disease model experiment was conducted to establish the relationship between early vaccination rates and disease spread. The results of this experiment suggest that early vaccination makes the disease spread slower. At a probability of initial immunity of 0.5, the spread of the disease becomes significantly hindered. This is shown in that at a p_immune 0.5 or greater, the final immune percentage is very close to the initial. Furthermore, the time it takes for the model to run goes down, as the disease cannot spread as much, and therefore disappears quickly.

To expand upon the results found in this lab, more experiments could be run with different values for the probabilities that were fixed in each experiment, such as p_start. Additionally, more complex relationships between the quantities of p_fatal and p_spread could be defined to represent how more fatal diseases generally do not spread as quickly.