Simulation Final Project -- Agent-based Simulation with Disease

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Running

The project uses the Streams API, so Java 8 or greater is required to build and run the project.

To run the simulation, first build the project using `javac -d build/production/Sim_Final_Proj -classpath "squintV2.19.jar" *.java` Then, run the project using

Windows: `java -classpath "build/production/Sim_Final_Proj;squintV2.19.jar" SimulationManager`

Linux/Mac: `java -classpath "build/production/Sim_Final_Proj:squintV2.19.jar" SimulationManager`

Alternative way to run on Linux/Mac: First, make sure you are in the directory that contains the files. Then run `javac -classpath squintV2.19.jar *.java` to compile. Once compiled, run `java -cp squintV2.19.jar:Sim_Final_proj/*:. SimulationManager` to execute the program.

Changing Parameters

- Number of Diseases - `SimulationManager:18` - Currently set to 12
- Disease Genome Length - `SimulationManager:20` - A lambda function which can return integers. Currently returns a random uniform integer 1-11.
- Disease Metabolic Penalty - `SimulationManager:23` - A lambda function which returns doubles. Currently returns a random uniform double 1-2.
- Display the Current Cell Resource Level or Max Resource Level - `SimulationManager:28` - Initially true, shows the current level. Set to false to show the maximum level.
- Frequency of Random Immune Mutation - `SimulationManager:31` - Currently random uniform double from 3-7.
- Frequency of Immune Disease Response - `SimulationManager:34` - Currently a random normal with mean of 1. `Math.abs` is used to make all times in the future.

Our Experiments

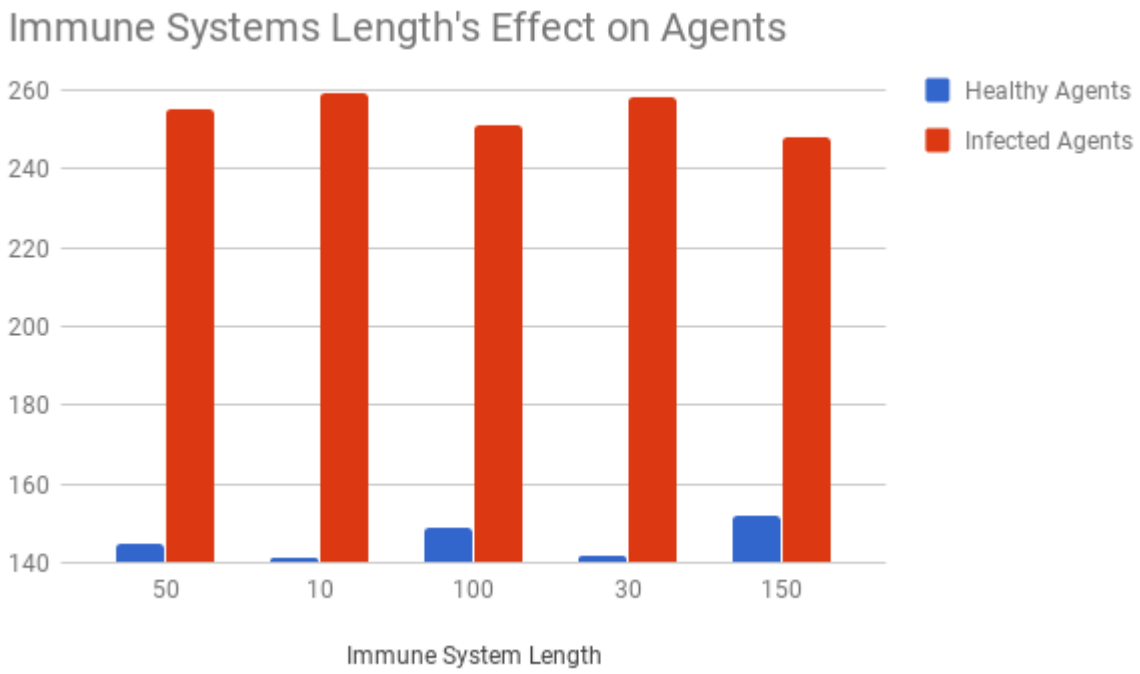
There are many options that we are able to tweak to get different results, these include the length of an agent's immune system, length of disease genome, metabolic penalty of a disease, how often an agent can have an immune response to a disease, and how often the agent's immune system randomly mutates. The sample size for all of the average number of healthy and infected agents found and printed below was 50 experiments with a maximum time of 100 seconds for each experiment.

While holding the length of the disease genome constant as well as the other variables, the length of the agent's immune system can impact how likely the agent will already be immune to the disease. The longer the agent's immune system, the greater the chance that the agent is already immune to the disease. There is also

a greater chance of having a closer match to a disease genome, which could lead to becoming immune to diseases faster than with a shorter agent's immune system. With a greater length of the agents' immune systems, the initial grid of agents will contain more healthy, blue agents than when setting the length of the agents' immune systems to being a smaller length.

In testing these assumptions, we held the genome length constant at 10, the metabolic penalty at 0.5, the mutation rate at 5, the immune response rate at 1, the number of diseases at 12. The following numbers are some of the output we found for the number of healthy and infected agents on average when changing the length of the agent's immune system:

Immune Length	Healthy	Infected
10	141	259
30	142	258
50	145	255
100	149	251
150	152	248



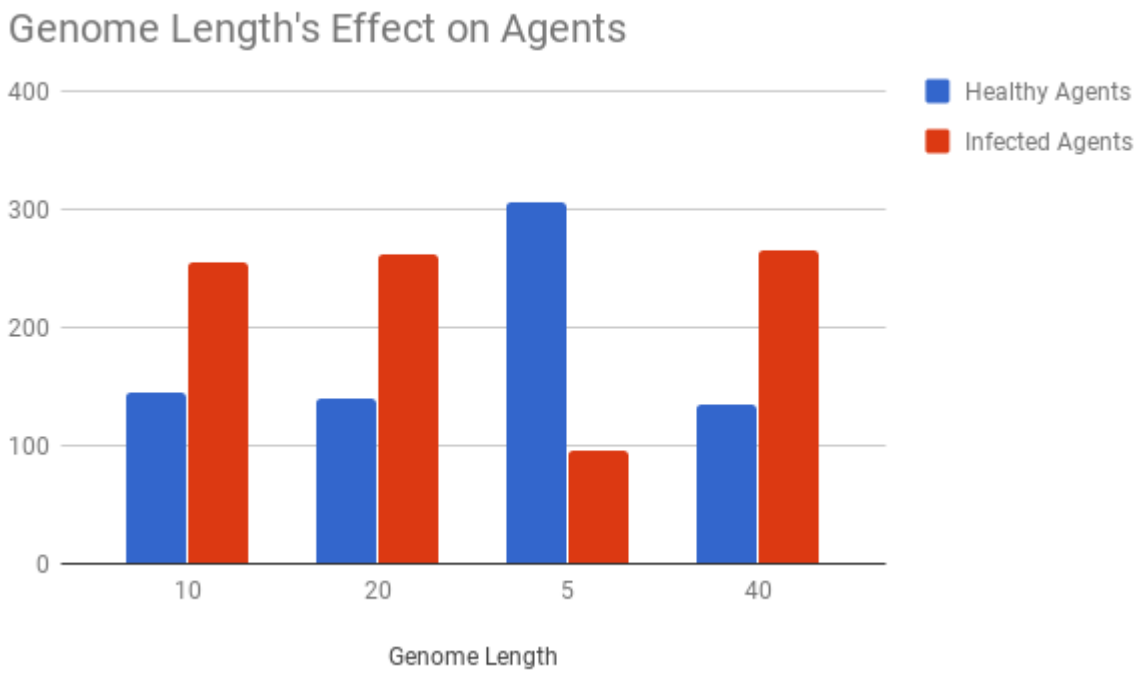
This output confirms the assumptions that were made above that as the immune system length increased, the number of healthy agents in the system also increased. While the numbers were not as drastic as we initially anticipated, the numbers still support our theory.

The shorter the length of a disease genome, the higher the chance that an agent might already be immune to the disease. This will also allow agents to become immune to the disease faster. When the length of the disease genome is shorter, the grid of agents will start with more agents being blue and healthy and more quickly turn into a grid of blue, healthy agents instead of red, infected agents. Oppositely, the longer the length of a disease genome, the less chance that an agent will be already immune to the disease. Longer disease genomes correspond to agents being infected for a longer period of time, because the immune

response will take longer to appropriately flip each digit to have developed an immunity to the disease. When the grid is initially populated, the majority of agents will be red and infected. However, as time progresses, the grid will begin to reflect a combination of blue, healthy agents on the outsides with more red, infected agents near the cells with the most resources. There will be some mixed in blues with the red agents from the replacement of dead agents with new, healthy agents. However, the majority of healthy agents will be found on the outside, which is because while there are less resources, infected agents with higher metabolic rates cannot survive in this environment for very long.

In testing these assumptions, we held the immune system length constant at 50, the metabolic penalty at 0.5, the mutation rate at 5, the immune response rate at 1, the number of diseases at 12. The following numbers are some of the output we found for the number of healthy and infected agents on average when changing the length of the disease's genome length:

Genome Length	Healthy	Infected
5	305	95
10	145	255
20	139	261
40	135	265

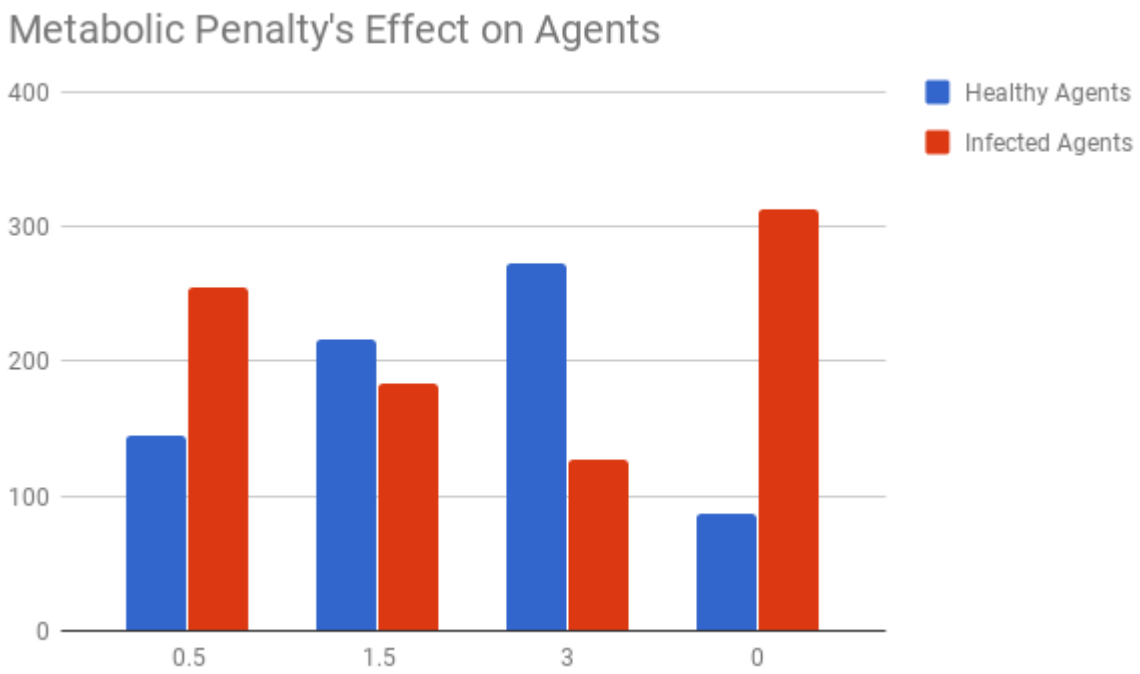


This output confirms the assumptions that were made above that as the disease's genome length increases, the number of healthy agents in the system decreases because the chance that an agent is already immune to a disease decreases.

Increasing the metabolic penalty of a disease results in agents dying off faster. Since every agent that dies is replaced by a new healthy agent, the number of blue agents quickly overtakes the number of sick agents. And if left to run long enough, all the agents would eventually be blue as the sick agents are dying and being replaced at a higher rate than they are moving and infecting their healthy neighboring agents.

In testing the claims made above, we held the immune system length constant at 50, the disease genome length at 10, the mutation rate at 5, the immune response rate at 1, the number of diseases at 12. The following numbers are some of the output we found for the number of healthy and infected agents on average when changing the metabolic penalty for an agent being infected:

Metabolic Penalty	Healthy	Infected
0.1	87	313
0.5	145	255
1.5	216	184
3	273	127

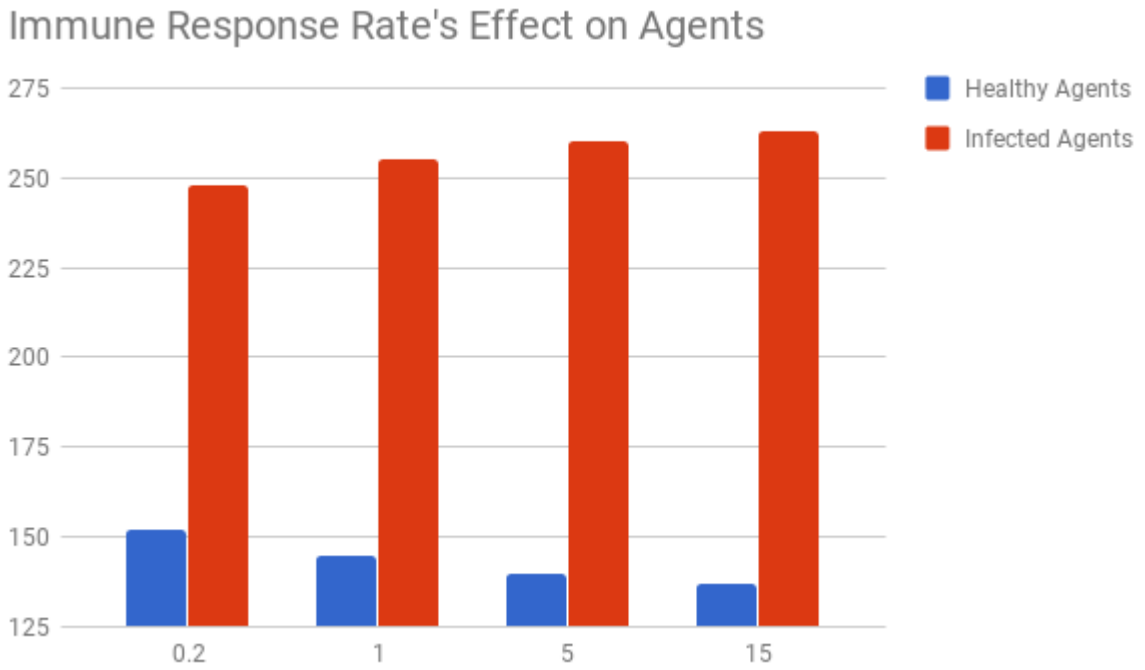


This output confirms the statements that were made above that as the metabolic penalty increases, the number of healthy agents in the system will increase. Initially, this seemed strange. However, when considering that when an agent dies in our landscape, they are replaced with a healthy agent, this makes sense. Having a higher number of healthy agents as the metabolic penalty increases means that agents are in fact dying more quickly and thus more frequently being replaced with healthy agents.

When experimenting with the agent's immune response to a disease, we noticed how an increase in how often an agent can respond to a disease corresponds to an increase in how quickly agents are able to fight off the diseases and thus become healthy more quickly. If an agent's immune system could not respond to a disease frequently, the agent would remain infected for longer and die more quickly because of the increased metabolic rate associated with being infected, especially if the agent is not near a high resource cell.

In testing these assumptions, we held the immune system length constant at 50, the disease genome length at 10, the metabolic penalty at 0.5, the mutation rate at 5, and the number of diseases at 12. The following numbers are some of the output we found for the number of healthy and infected agents on average when changing the agent's immune response rate:

Immune Response Rate	Healthy	Infected
0.2	152	248
1	145	255
5	140	260
15	137	263



This output shows that as the immune response rate is increased, the number of healthy agents in the landscape decreases. This is probably because the agents are able to cure themselves and stick around longer. Whereas, others die and are reborn as healthy.

In addition to agents attacking their diseases, their immune systems also randomly mutate, flip one bit in their immune system, with an even drawn from a uniform 3, 7. We decided that random mutations should be truly random, so the time is drawn from a uniform distribution. We then selected the bounds of the mutation such that over the average agent lifetime there would be, on average, 20 such mutations. Because the mutations are completely random they can aid the agent in attacking a disease, or they may hinder the progress. Increasing the event rate causes noticeably more healthy agents, especially around the high sugar areas which previously had even more disease.

Given that this is how we decided our mutate function to run, the numbers of healthy and infected agents did not change dramatically as the mutation rate increased, which can be seen in the numbers below:

Mutation Rate	Healthy	Infected
0.5	143	257
2	146	254
5	145	255

Mutation Rate	Healthy	Infected
15	146	254