

CA4024 Building Complex Computational Models - ABM Assignment - Simulating Viral Infections

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[GitLab Repo](#)

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1 Introduction

There has been an increased interest in modelling and simulating the spread of viruses through groups of agents in recent years, brought on by the pandemic caused by SARS-CoV-2. The interactions between the agents and passing of infections is difficult to predict, making this a suitable scenario for agent-based modelling. In this way, we attempt to understand the emergent properties of the system when we consider different factors that we know should have an influence on it and its state over time. For virus modelling, the factors we are concerned with are the vaccination rate of the population, the probability of infection when exposed to the virus and the probability of death for an agent that contracts the virus. We believe that simulating the effects of these will lead to an understanding of what level of vaccination is needed to wipe out viruses with different properties.

We first consider a base implementation of virus simulation, following standard virus models that define agent states as susceptible, infected, and removed (deceased or recovered). Susceptible agents have a chance to be infected if in the neighbourhood of an infected agent. The infected agent has a chance to die every time-step and recover after a time period. The recovered agents are immune and the agents that die are removed from the simulation. We propose several changes to this model to better simulate the system. Firstly, agents can be carriers of the virus before they show symptoms and infect other agents. Also, the chance of agents transferring the virus is not a static probability, but a dynamic one that increases as agents become closer. Finally, when an agent is vaccinated or recovered and becomes immune, they can still be infected and die, but the probability of this is much lower as they have built up a defence against the virus. We believe these changes, along with some other minor changes outlined in our method, will result in a simulation more representative of the real system.

The aim of this simulation is to understand how influential the vaccination rate is as a measure to control the spread of the virus. We will measure how effective the vaccination rate is by looking at the number of infections over time, and the number of fatalities. These metrics will be explained in detail in our methodology. The simulation will come to an end when there are no agents that are infected or carrying the virus and thus the virus has been eradicated.

2 Methodology

In this section, we will discuss the different components of the simulation and motivate our choices when deciding what to include and exclude. Our simulation on a high-level is a representation of how a virus would spread through a small, closed community.

2.1 Environment

The environment of our simulation is a 2D-space consisting of a 1x1 tile that agents move around on. We decided to use a speed of 0.05 for the agents, and they can move this distance on both the x and y axes, allowing them to move to any point in a square around them of length 0.1 on each side. This is suitable for our implementation as we are not concerned with the specifics of movements that the agents make and considering the distance moved as a radius would add computation that adds little value for our use.

The environment has no attributes as our primary focus is on the interactions between the agents. We discussed possible additions to the environment such as areas that are well-ventilated (for airborne viruses) that would reduce the risk of infection. We decided to leave this out as it does not work towards solving the main problem we wish to address; how does the vaccination rate affect the spread and fatalities of a virus outbreak?

2.2 Agents

Our viral simulation will use an S-C-I-R model, with modifications from the S-I-S model. The model will have classes for susceptible agents, carriers, and infected. Recovered agents will gain some immunity, but may become susceptible again with a small chance. The model will be a S-C-I-S model, with mild immunity for recovered or vaccinated agents.

2.2.1 Susceptible

"Susceptible" is the initial state agents take. Every time they are updated they find the closest agents that are infected and determine what the infection rate would be considering the distance between the agents (this method will be discussed in detail and motivated later). If by chance the susceptible agent is infected they become a carrier.

2.2.2 Carrier

A carrier is an agent who has been infected with the virus but is yet to develop symptoms. They can still spread the disease to susceptible agents but they cannot die as they are yet to develop symptoms. After a sampled carrier period from a defined uniform distribution, this agent is converted from carrier to infected. The "carrier" state was added into our simulation as it's consistent with what we know about viral spread during outbreaks (See Section 2.6).

2.2.3 Infected

Infected agents are showing symptoms of the virus and have a sufficient viral load to infect susceptible agents. At every timestep, there is a chance that they die, which increases as the virus worsens and decreases as they move towards recovery. The function used to capture this behaviour is discussed later in this section.

2.2.4 Mild Immune (Susceptible)

This covers agents who have become immune, through recovery or vaccination. In our simulation immunity is an attribute of an agent that affects the infection rate and death rate. The motivation for this is that recovered and vaccinated individuals are less likely to become infected or die, but there is still a chance they could. Agents that die are removed from the simulation.

2.3 Infection Rate

The basic implementation made use of a static infection rate that applied when two agents were within a set distance. This is a good implementation for simplicity, but it does not capture the increase one would expect when agents become closer. To account for this we introduced the following function that scales the infection rate based on the distance between agents.

$$\text{Max}(0, \text{Infection Rate}(1 - \frac{\text{Distance between agents}}{\text{Max infection distance}}))$$

We believe that this will better capture the interaction between agents with respect to the infection rate, making our simulation more representative of the real system.

2.4 Death Rate

Due to the nature of our agent-based model, the agents are updated asynchronously and every time step. This means that there is a probability that an infected agent will die at every time step. In an attempt to keep this true to the real system, we consider the probability to rise as the agent's illness worsens and then decrease as they move towards recovery. This forms an arc that rises and falls over the course of the infection. After deliberation, we decided to make use

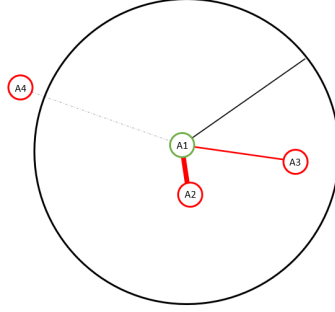


Figure 1: The infection rate is based on the distance to the infected agent. If an agent is outside the max range there is no chance of infection.

of a geometric distribution as it captures the behaviour we want, a change in probability dependent on what stage in the infection we are in. The only issue is that the probabilities decreased as time goes on in the distribution, which is the inverse of what we want. To remedy this we want to move backwards through the distribution. This will allow the resultant probabilities from the distribution to increase.

Another consideration we had was how we can ensure that the probabilities we use over the time steps add up to the case fatality rate after we reach the mean recovery time. This is to say that the probability an agent has died by this time is equal to the fraction of fatalities from the virus over the total cases. This would allow our simulation to have roughly the same case fatality rate as that of the actual virus. To ensure the sum of probabilities from the start of infection to the mean recovery period we made use of the cumulative distribution function (CDF) of the geometric distribution, flipped vertically, as we are moving backwards through the distribution, from right to left. If we mirror the probabilities from the start of infection to the end around the mean recovery period we get something similar to the arc we wanted originally.

This will allow us to model the probability of death as we understand it to be, dynamically changing over the course of the infection. We found that this additional computation is not expensive and adds little overhead as the value for p in the probability density function (PDF) is found at the start and the dynamic death rate is found when evaluating an infected agent's outcome. Overall, we are confident that the model captures many different factors that play a role in how agents interact and how a virus would spread through the population.

2.5 Metrics

2.5.1 Infections / Confirmed Cases

Infections measures the number of agents that become infected with the virus. This is broken down into 2 different levels, a daily and a total. The aim of this

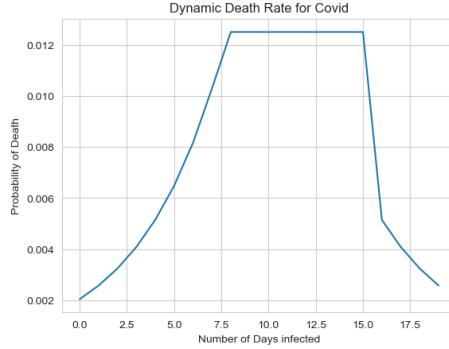


Figure 2: Lineplot of the probability of death at each time step for an agent infected with Covid.

metric is to understand how well the virus spreads and at what points in the simulation. This will help us to understand the impact that the vaccination rate has on the spread of the virus through the population.

2.5.2 Fatalities / Confirmed Deaths

Fatalities measures the number of agents that die as a result of the infection. As with the infection rate, the fatalities are tracked on both daily and total levels. This helps us to get insight into what stages in the simulation death occurs at and understand how deadly the virus is. We expect that we will be able to see the impact of vaccination rates on the number of dead agents in the simulation.

2.6 Virus Selection

We will now motivate the selection of viruses for our simulation.

2.6.1 COVID-19

We have decided to select Covid as one of our viruses due to the impact that it had globally over the period of 2020-2022. Covid had a case fatality rate of roughly 2.5% [1] when the vaccine was introduced, so we decided to use this figure for our simulation. Individuals infected with Covid are carriers for about 4-6 days on average [2], and the active illness can last 1-2 weeks [3]. This leaves us with our carrier period, recovery period and case fatality. There is no exact figure for how infectious covid is, but we have decided to make use of a relatively high figure as Covid spread rapidly during the pandemic.

2.6.2 Marburg Virus Disease (MVD)

MVD was selected due to its high fatality rate and lack of a vaccine. MVD is a severe virus causing haemorrhagic fever in humans. A particular fruit bat

species, the *Rousettus aegyptiacus*, are recognised as the natural hosts of MVD [4]. It is transmitted from fruit bats to humans and then transmits amongst humans through contact with bodily fluids [5]. There have been numerous recorded outbreaks of MVD in the last century, most of which were short and sporadic. But in 2005 there was an outbreak in Angola resulting in 374 cases and 88% case fatality rate. Using simulation, we try to quantify, the impact that early vaccination would have in such an outbreak. Carrier time is typically between 5 and 10 days, and recovery time ranges from a couple of weeks to a month. Although, many recovered people have reported long-term symptoms [5].

2.7 Experiment Design

To analyse the effect of vaccination rate on death and infection rates, we simulated each virus with varying vaccination rates. Vaccination rate is varied in the range 0-1 with increments of 0.1. Each vaccination rate is only run once due to computation constraints on our local machines, but the random seed ensures the reproducibility of our results. The remaining parameters are set depending on the virus we're investigating. The full code implementation in Python can be found [here](#). We saved videos for the simulations at each vaccination rate, which can be found on [Gitlab](#). See the README for where to locate these, and instructions on how to run the simulation, with the option of varying the parameters. Each simulation stops when there are no infected remaining.

3 Results

3.1 Covid

The fatality numbers for different VR against Covid were analyzed. [Figure 3\(a\)](#) shows the cumulative fatalities at each simulation time step, with each line representing a simulation and the VR indicated by colour. The simulation duration varies based on when the termination condition is reached. [Figure 3\(b\)](#) shows the total fatalities at the end of each outbreak. The data suggests a roughly linear decrease in fatalities with increasing VR, with outbreaks becoming almost non-existent at around 80% VR.

The cumulative infections are depicted in [Figure 4](#). A decrease in the infection rate is observed with higher VR. However, plot (b) shows that the relationship is not linear, but rather an exponential decrease in infected up to 80% VR, after which infections bottom out. The cumulative infection curves show a sharp increase in infections, followed by stabilization due to fewer susceptible agents in the environment. This rise becomes less sharp for higher VR. The findings suggest that herd immunity is achieved at approximately 80% VR in the Covid simulation.

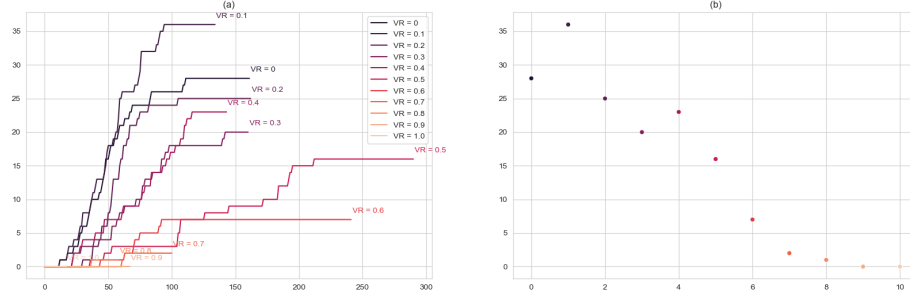


Figure 3: (a): Total Covid fatalities over time, by VR (b): total fatalities after the virus is eradicated, by VR

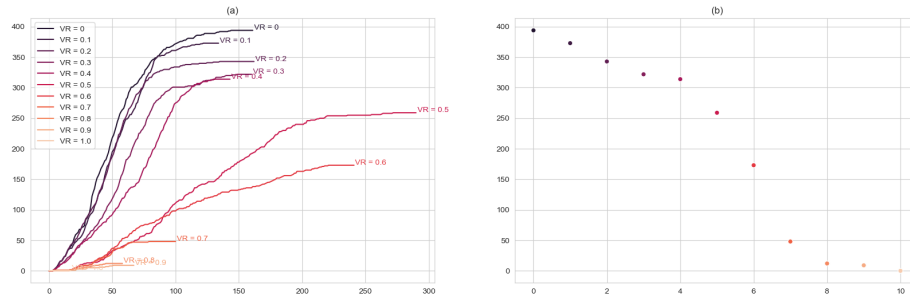


Figure 4: (a): Total Covid infections over time, by VR (b): total infections after the virus is eradicated by VR

3.2 MVD

According to Figure 5, the cumulative number of MVD fatalities decreases linearly with the vaccination rate. At 80% VR, the duration of the outbreak is significantly reduced, and there are less than 5 fatalities per outbreak, compared to the initial 160 fatalities with 0% VR. Based on our simulation, to prevent an outbreak like Angola in 2005, we would suggest that once a vaccine is developed, vaccinating 80% of the population is sufficient for herd immunity. It should be noted that this is an extrapolation based on simplifying assumptions and a smaller number of agents in our simulation than the population of Angola.

Finally, Figure 6 shows MVD infections decrease exponentially with increases in VR, bottoming out at 80% VR. This is due to the higher likelihood of death when infected compared to Covid.

4 Conclusion

In this work, we explored the effect of vaccination rates on the number of infections and deaths caused by two viruses. Namely, Covid-19 and Marburg Virus

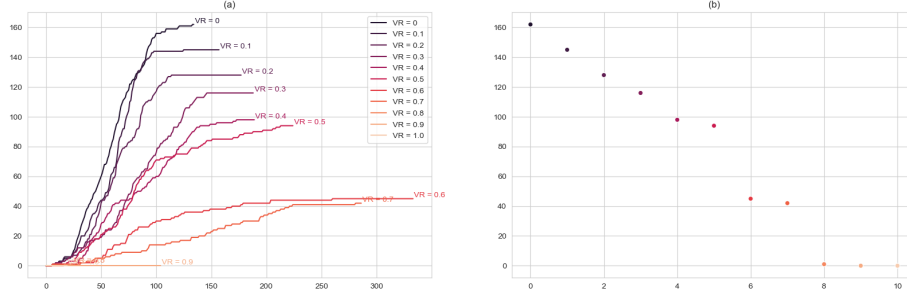


Figure 5: (a): Total MVD fatalities over time, by VR (b): total deaths after the virus is eradicated by VR

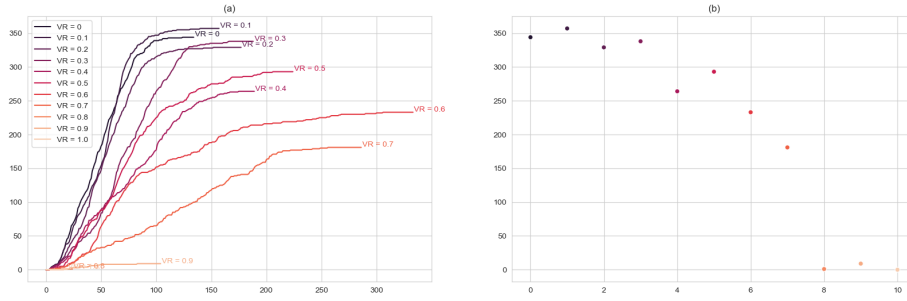


Figure 6: (a): Total MVD infections over time, by VR (b): total infections after the virus is eradicated by VR

Disease. The aim of the simulations was to help understand the causal impact of the vaccination as a measure to control the spread of the viruses. We proposed several improvements to the standard S-C-I-R viral infection simulation in order to better reflect reality. These included dynamic infection probabilities based on distance from an infected agent, dynamic death probability, and the introduction of mild immunity, resulting in the S-C-I-S model. We found that a vaccination rate near 80% helps to prevent outbreaks in the case of both viruses.

References

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