Group 2: Apoorv Saraogee, Braden Wellman

MSDS 460 Section 58, Spring Quarter

Case Study: Epidemiology with agent-based models

This project aims to present a computational simulation of disease transmission using agent based modeling techniques implemented in Python using the Mesa package. By developing a custom disease model, we investigate the effects of various factors such as infection rates, recovery types, age and vaccination availability on the dynamics of an infectious disease. The findings from our simulation aim to contribute to the broader understanding of disease transmission dynamics, which has been a major focus in various fields over the last several years. More specifically, this project exemplifies the power of agent based modeling in capturing complex phenomena, demonstrating to researchers how to explore and evaluate various scenarios in a controlled and computationally efficient manner, as well as informing public health interventions. Overall, this project demonstrates the successful application of agent based modeling techniques using the Mesa package in Python for simulating disease transmission. Through the utilization of various classes, the analysis of simulation outputs, and the exploration of different modeling methods, we aim to contribute to the growing body of knowledge in the field of data-driven epidemiology and provide valuable insights into disease dynamics.

Epidemiology and modeling

Epidemiology is defined as the study of the distribution and determinants of health related events among a specified population (Porta 2014). There are two categories for epidemiology models - aggregate models and agent-based models. Aggregate models have been the most common and use differential equations and compartments for the population. The most traditional compartment model is of SIR type with **S**usceptible, **I**nfectious and **R**emoved (Immune and

Dead) individuals (Kermack and McKendrick, A.G. 1927). Other models include the SIS and SIRV types which don't have Immune populations due to high mutation rate or have an additional vaccinated disease class respectively. These models are more suitable in the initial stages of a pandemic because they require fewer parameters than agent-based models and have lower variability in estimates. Indeed, aggregate compartment models have been made using Markov processes (Miller 2022; Tuckwell and Williams 2007). However, a key drawback of aggregate models compared to agent-based models is the lack of insight into heterogeneity in a population such as child, adult and elder and the stochastic nature of recovery time. Agent-based models, while computationally expensive, have provided results that are comparable to aggregate models by others (Kunwar et al. 2022; Ozmen et al. 2016; Rahmandad and Sterman 2008).

Epidemiological modeling has been prominent during the COVID-19 crisis and while modeling has failed to provide accurate quantitative estimates during the COVID-19 crisis (Ioannidis, Cripps, and Tanner 2022), models have been useful in estimating the relative risk of different interventions in reducing disease burden (Iranzo and Pérez-González 2021). Initial alarming quantitative estimates for the death rate were using a symptomatic infection rate of 65% (Miller 2022) which was later shown to be 15% during seroprevalence testing (Subramanian, He, and Pascual 2021). Consequently, this study investigates symptomatic infection rates of both 15% and 65% used during the COVID-19 crisis. In addition to symptomatic infection rate, this study investigates the impact of the probability of transition between 10% and 30% to model the impact of transmission rates on disease dynamics.

Test Section

We chose to use Mesa in Python in order to implement our agent based disease model. Mesa is an incredibly dynamic, multifaceted package that is specifically designed for agent based

modeling, as opposed to other popular simulation packages like simpy. In preparation for the project, we used ‘A simple agent based infection model with Mesa and Bokeh’ by Farrell (2020) as a reference point when developing our code (Farrell 2020).

The first step in creating our agent based model was implementing different classes, such as the disease class - which contained parameters such as transmission rate, recovery type, number of agents and number of steps - and agent class, which represented individual agents within the simulation and their associated characteristics: infection status, recovery time and age. We derived our fatality rate values - differing by age - from Miller’s “Model of an Epidemic” (Miller 2022). These two classes comprised the bulk of our code - and time spent on the project - and helped facilitate the representation of human interactions, as well as the transmission of the disease.

Next we created a faux geospatial environment where the agents could navigate, interact and infect nearby individuals. We used a random activation scheduler to orchestrate the interactions between the individuals agents and ensure that the simulation followed a stochastic process. Finally, we included a data collector that empowered us to gather extensive data at both the agent level and the disease level, enabling comprehensive analysis of the entire simulation.

The core of our implementation relied on the ‘step’ function that was embedded in our disease class, which performed a sequence of actions in each step of the simulation. The operations included things like updating agent statuses, managing movement and simulating contact interactions in order to capture the dynamics of the epidemic. By executing the step function for a specified number of iterations, we were able to meticulously track the aggregate numbers of susceptible, infected, immune and deceased individuals - as well as the specific events associated with each agent.

Insights

Our study tested the agent-based model by varying parameters such as symptomatic infection rate, probability of transmission and type of model with a constant population size of 1000 people over 100 timesteps. We tracked the number of deaths at the last timestep in Table 1 of the Appendix. Here, we can see that the symptomatic infection rate (inherent pathology factor) and SIS model assumptions had the largest impact on the number of deaths. We also deduce that the probability of transition and SIRV model assumptions had a lower impact on the number of deaths but delayed infections and reduced the number of infections respectively. Our model 10 of SIR type had 9 deaths, similar to the Markovian Model 0 based on Miller (2022) with 1 death.

Disease dynamics are plotted for all possible classes (susceptible, infected, immune, dead) in Figures 1, 2, 3 and 4 of the Appendix for the Markovian Model 0, SIS type model 1, SIR type model 10, SIRV type model 12 respectively. The disease dynamics initially look similar in Figures 1 and 3 for the Markovian Model 0 and SIR type Model 10 respectively. However, the agent-based models diverge with a second wave of infections as immune individuals can become susceptible at a rate of 1% in our agent-based simulation. In Figure 2, also our worst case scenario with a high symptomatic infection rate and elusive recovery from disease, we see a steady increase in deaths over time with most people always infected or susceptible.

Further, we explored the heterogeneity of our population using event graphs following the state of an agent at each timestep in Figures 5 and 6 of the Appendix for the SIS type Model 1 and SIR type Model 10 respectively. It was encouraging to see multiple reinfections for each agent as well as differences in death rates depending on factors such as age in the event graphs. We would incorporate the probability of transmission similar to fatality rates into the agent class to gain further insight into the heterogeneity that is a trademark of agent-based models.

References

Farrell, Damien. 2020. “Bioinformatics and Other Bits - A Simple Agent Based Infection Model with Mesa and Bokeh.” April 14, 2020. https://dmnfarrell.github.io/bioinformatics/abm-mesa-python.

Ioannidis, John P.A., Sally Cripps, and Martin A. Tanner. 2022. “Forecasting for COVID-19 Has Failed.” *International Journal of Forecasting* 38 (2): 423–38. https://doi.org/10.1016/j.ijforecast.2020.08.004.

Iranzo, Valeriano, and Saúl Pérez-González. 2021. “Epidemiological Models and COVID-19: A Comparative View.” *History and Philosophy of the Life Sciences* 43 (3): 104. https://doi.org/10.1007/s40656-021-00457-9.

Kermack, W.O., and McKendrick, A.G. 1927. “A Contribution to the Mathematical Theory of Epidemics.” *Royal Society of London* 115 (772): 700–721. https://doi.org/10.1098/rspa.1927.0118.

Kunwar, Prateek, Oleksandr Markovichenko, Monique Chyba, Yuriy Mileyko, Alice Koniges, and Thomas Lee. 2022. “A Study of Computational and Conceptual Complexities of Compartment and Agent Based Models.” *Networks and Heterogeneous Media* 17 (3): 359–84. https://doi.org/10.3934/nhm.2022011.

Miller, Thomas. 2022. “Model of an Epidemic.” *Data Science Quarterly* 1 (1/2).

Ozmen, Ozgur, James Nutaro, Laura Pullum, and Arvind Ramanathan. 2016. “Analyzing the Impact of Modeling Choices and Assumptions in Compartmental Epidemiological Models.” 2016. https://journals.sagepub.com/doi/10.1177/0037549716640877.

Porta, Miquel. 2014. “Epidemiology.” In *A Dictionary of Epidemiology*, edited by Miquel Porta. Oxford University Press. https://www.oxfordreference.com/display/10.1093/acref/9780195314496.001.0001/acref-9780195314496-e-651.

Rahmandad, Hazhir, and John Sterman. 2008. “Heterogeneity and Network Structure in the Dynamics of Diffusion: Comparing Agent-Based and Differential Equation Models.” *Management Science* 54 (5): 998–1014.

Subramanian, Rahul, Qixin He, and Mercedes Pascual. 2021. “Quantifying Asymptomatic Infection and Transmission of COVID-19 in New York City Using Observed Cases, Serology, and Testing Capacity.” *Proceedings of the National Academy of Sciences* 118 (9): e2019716118. https://doi.org/10.1073/pnas.2019716118.

Tuckwell, Henry C., and Ruth J. Williams. 2007. “Some Properties of a Simple Stochastic Epidemic Model of SIR Type.” *Mathematical Biosciences* 208 (1): 76–97. https://doi.org/10.1016/j.mbs.2006.09.018.

Appendix

Table 1. Model characteristics and death rate at 100th step for 1000 people. Model 0 is the Markovian Model based on Miller 2022. Models 1-12 are agent-based models

Model #	Symptomatic infection rate	Probability of transition	Type of Model	Deaths/1000 at the 100th step
0	0.15	0.1*	SIR	1
1	0.65	0.3	SIS	181
2	0.15	0.3	SIS	56
3	0.65	0.3	SIR	43
4	0.15	0.3	SIR	8
5	0.65	0.3	SIRV	41
6	0.15	0.3	SIRV	7
7	0.65	0.1	SIS	43
8	0.15	0.1	SIS	45
9	0.65	0.1	SIR	41
10	0.15	0.1	SIR	9
11	0.65	0.1	SIRV	29
12	0.15	0.1	SIRV	6

*This is the contagion rate for the Markov model.

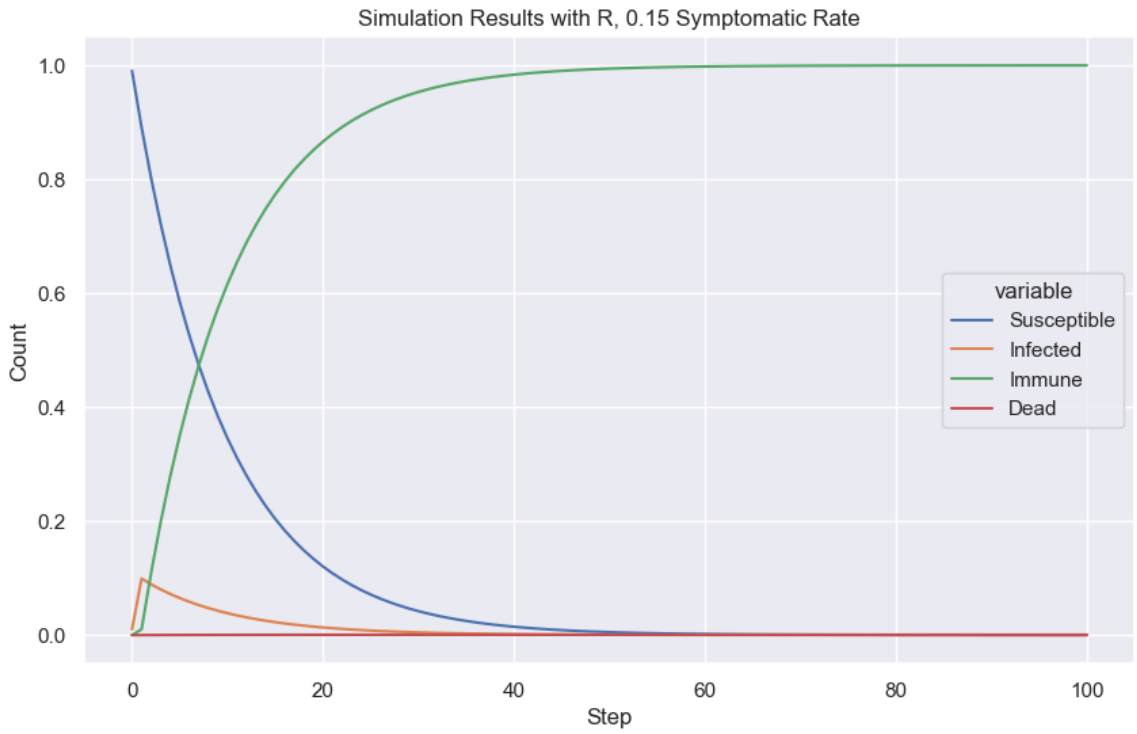

Figure 1. Markov Model 0 aggregate plot. Model from Miller 2022 implemented with 15% symptomatic rate shows basic dynamics of compartments.

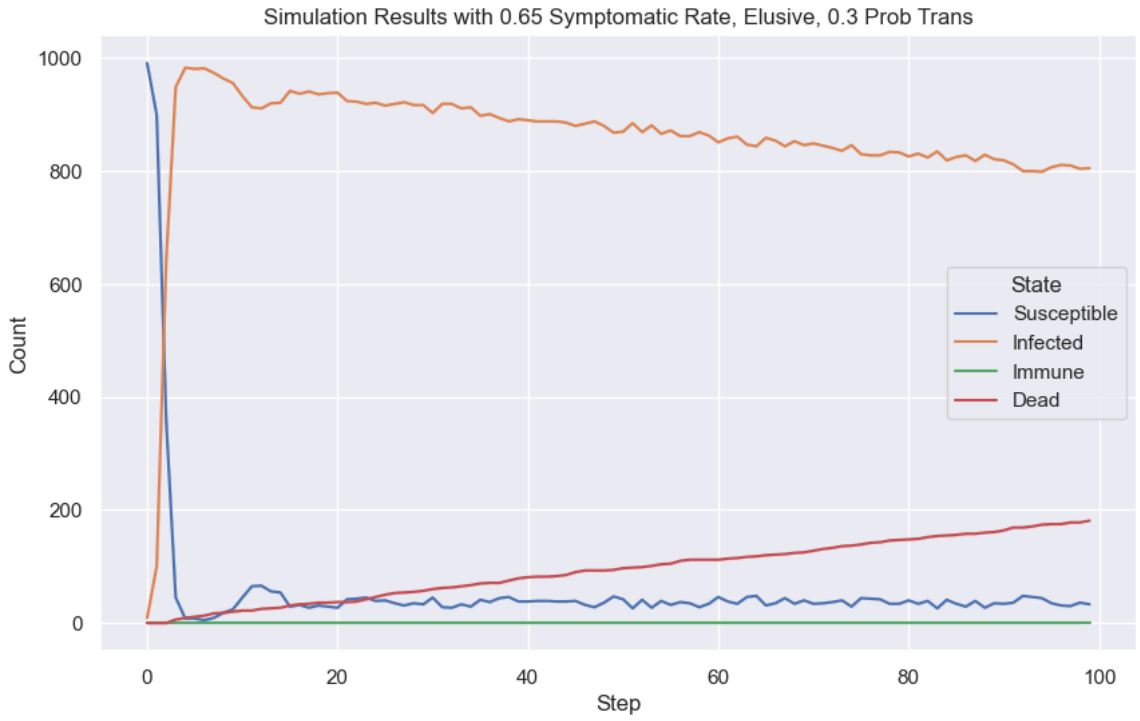

Figure 2. Model 1 aggregate plot. Worst-case scenario plot from the agent-based model of SIS type with 65% symptomatic rate and 30% probability of transition. A steady increase in death rate is seen.

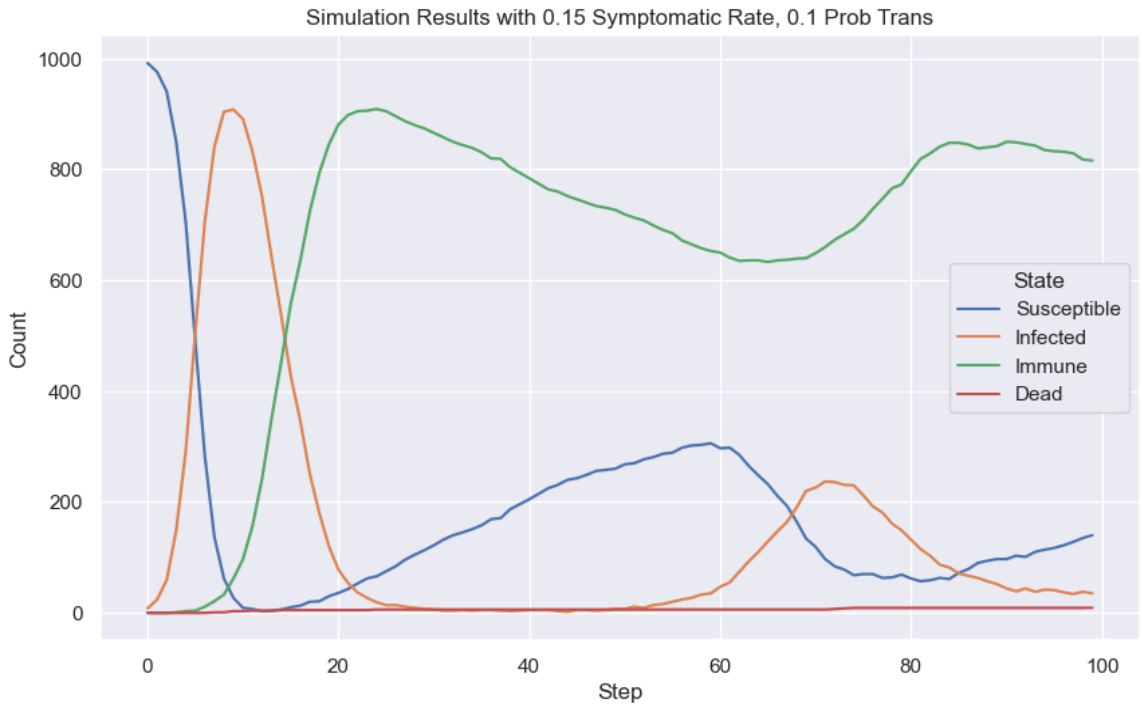

Figure 3. Model 10 aggregate plot. Agent-based model of SIR type with 15% symptomatic rate and 10% probability of transition. We see complex dynamics with a second infection wave.

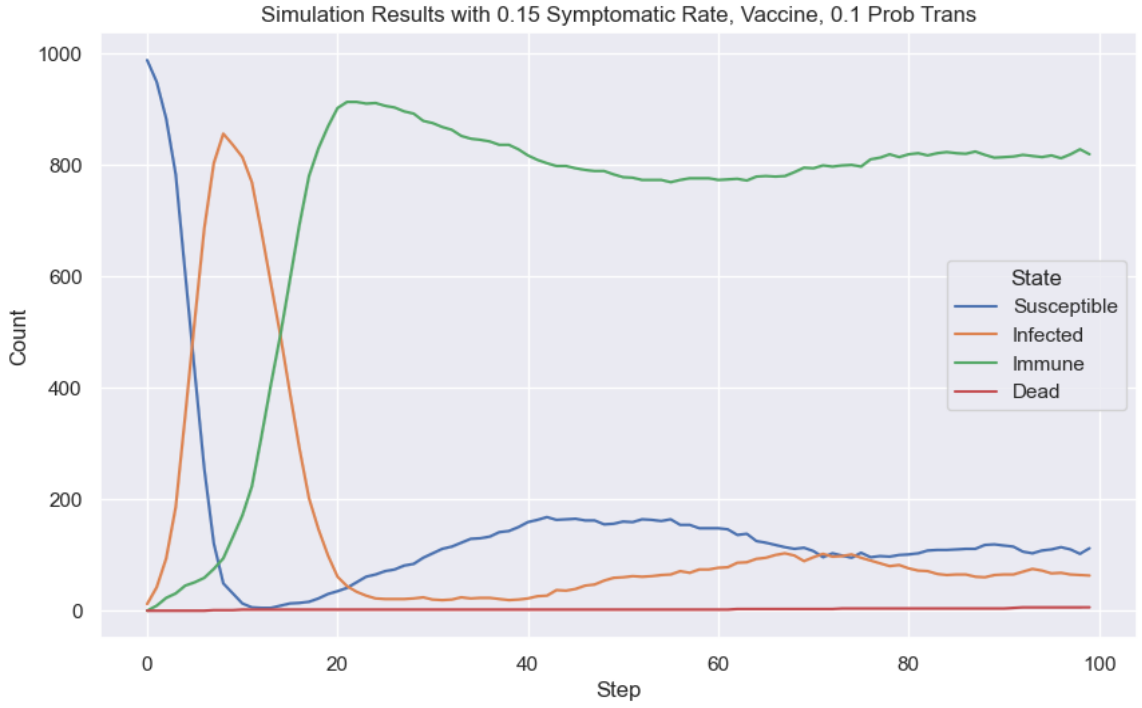

Figure 4. Model 12 aggregate plot. Agent-based model of SIRV type with 15% symptomatic rate and 10% probability of transition. We see the peaks of the infections is smaller than the SIR type plot in Figure 3.

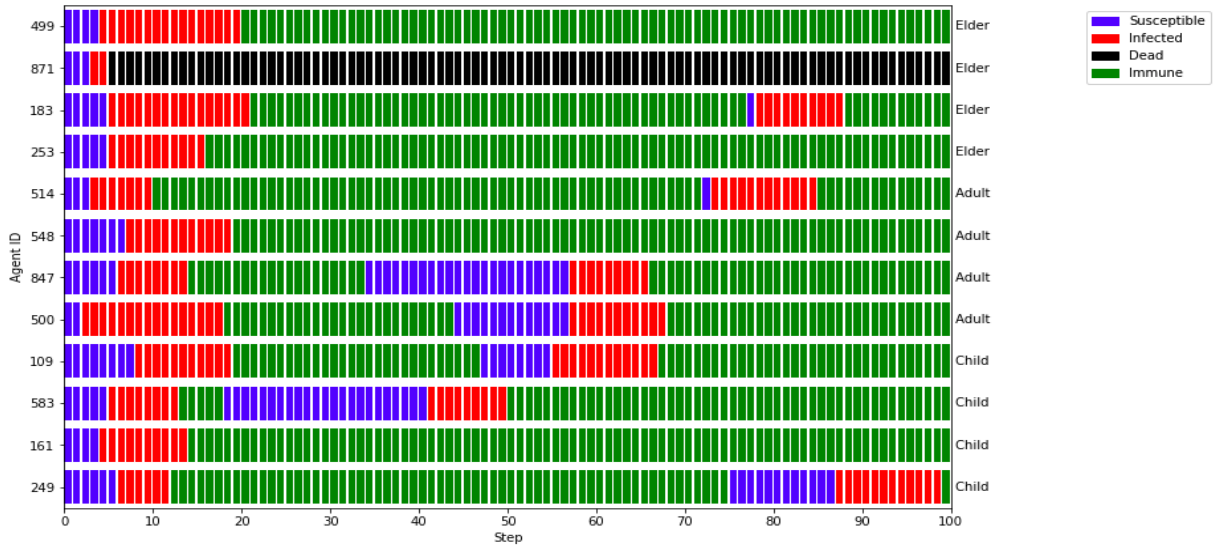

Figure 5. Model 10 agent event plot. Agent-based model of SIR type with 15% symptomatic infection rate and 10% probability of transition. We can see differences among individuals with age category on the right and AgentID on the left.

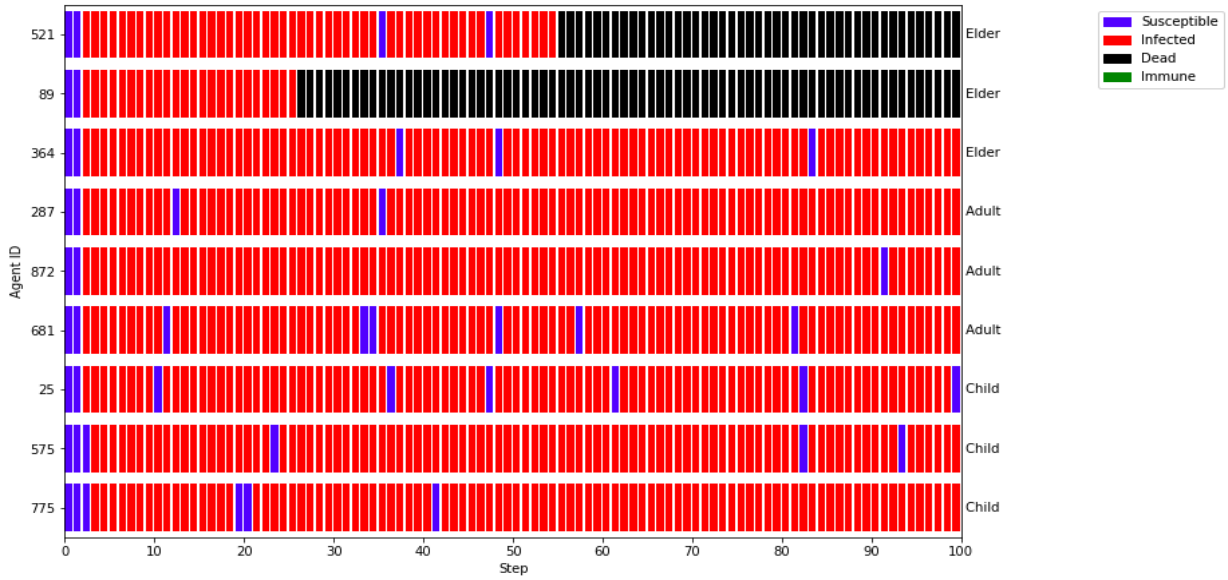

Figure 6. Model 1 agent event plot. Agent-based model of SIS type with 65% symptomatic infection rate and 30% probability of transition. We see that the population spends most of its time in the infected state.