

Agent-Based Simulation Model of COVID-19

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Problem Statement

Background of the Chosen Case and Motivation

SIR models have long been important in simulating the spread of diseases, which provides valuable ideas for transmission patterns and public health interventions. With the global reach and profound impact of COVID-19, it serves as a particularly relevant real-world case study for our analysis. The pandemic presents an array of unique challenges, underscoring the need to deeply understand the dynamics of transmission and the effectiveness of various public health responses.

In this study, COVID-19 was chosen as the topic not only because of its contemporary significance but also because the large amount of data available allowed us to validate our model with a high degree of accuracy.

Historically, SIR models have been pivotal in advancing our understanding of infectious diseases. From the foundational work by Kermack and McKendrick in 1927 to more recent studies, like that of He et al. (2020), these models have proven indispensable in effecting public health decisions. They provide a favourable framework for analysing and predicting the course of disease development, which helps to enable an effective public sector response in times of crisis.

Suitability for Agent-Based Modelling

Agent-based modelling is well suited to simulate SIR models because it allows us to model the behaviour of individuals in a population. Unlike traditional models that assume uniform behaviour, ABM treats each agent with unique interactions. This flexibility is essential for understanding how COVID-19 is transmitted, especially when accounting for factors such as asymptomatic carriers and varying exposure rates.

There are studies showing that ABM is effective in pandemic simulations. The Annals of Operations Research and Epstein (2009) have shown that ABM can be used to predict outbreaks and inform public health decisions. These examples show the model's ability to reflect the complexity of real-world disease transmission.

Complexity of the Case for Simulation

The complexity of SIR models simulating COVID-19 stems from several factors. First, individual differences, such as age, health status, and infection risk, may all contribute to differences in transmission and recovery. Modelling these differences ensures more accurate predictions of the spread of COVID-19 in the real world, and provides a realistic picture of how COVID-19 affects different populations (Ferguson et al., 2020).

A key challenge is asymptomatic transmission, where individuals unknowingly spread the virus without symptoms, which adds uncertainty to the model. Another important aspect is the behaviour of symptomatic individuals. Symptomatic individuals stop or reduce interactions with others, either voluntarily or through isolation, and these behaviours significantly alter transmission dynamics. The effectiveness of quarantine measures and adherence to public health guidelines further influence infection rates. Incorporating these behaviours adds another layer of complexity.

Moreover, government interventions such as quarantines, travel restrictions, and social distancing impact people's movement and interactions. With the simulations of these factors, and

interventions like mask-wearing and vaccination, it play an important role in understanding real-world outcomes (Ferguson et al., 2020).

Model Design

Our agent-based model simulates the spread and progression of COVID-19 by categorising each agent into one of seven specific health states: unexposed people, asymptomatic with contagious, symptomatic with contagious, symptomatic but no longer contagious, post-COVID immunity, people with naturally immune, and death status. These states help the model reflect the complete progression of the disease, from initial exposure to the virus, through the different stages of infection, to recovery or death. In the beginning of the simulation, every agent is assigned one of these health statuses.

Additionally, agents are categorised into different age groups based on real-world population distribution data, which plays a significant role in determining their mortality rate. Each age group is assigned different mortality rates, reflecting the varying levels of risk experienced by different demographics during the pandemic (Ritchie & Roser, 2024; CDC, n.d.). The mortality rate is hypothetical but informed by real data.

Each agent is equipped with a **biostate countdown**, which tracks how long the agent stays in a particular health state before transitioning to the next one. The transition between states is controlled by the **BioTransition matrix**, which is an $n \times n$ matrix (n is number of states) that defines the probability of moving from one state to another. The probabilities in each row of the matrix sum to one, ensuring a coherent state transition system. This design simulates variability in disease progression. For example, the asymptomatic and contagious state lasts between 6 and 10 days, while the Symptomatic and contagious state lasts between 3 and 6 days (Byrne et al., 2020). This randomness reflects variability in infection progression between individuals.

To simulate agent state transitions, the function **setAgentState** updates an agent's current health state based on the transition probabilities from the **BioTransition matrix**. The **updateAgent** function decrements the **biostate countdown** and triggers a state transition when the countdown reaches zero. Additionally, this function contains the simulation of a small probability of immunity loss in the post-COVID immune state, representing rare cases of reinfection.

Complexity Reflection

The model effectively captures the complexity of COVID-19 transmission and disease progression by incorporating several key mechanisms. First, the inclusion of age as a critical factor in determining mortality acknowledges the higher risks faced by older individuals. Agents over 65 have mortality rates as high as 60% (CDC, n.d.), which aligns with epidemiological data showing that older populations are particularly vulnerable.

Moreover, randomness in agent interactions and virus transmission is built into the model to enhance realism. Each agent engages in a set number of daily interactions (an average of 10 per day), with a transmission probability set at 0.05 per interaction. This randomness mirrors real-world human behaviour, where some interactions lead to infections while others do not. It also reflects the unpredictable nature of virus transmission, particularly in high-density environments.

A structured social network is also introduced using the Barabási–Albert (BA) model, which simulates how individuals tend to interact within their social circles rather than randomly. This structure allows the model to replicate super-spreader events, where a few highly connected individuals infect a disproportionately large number of others, a phenomenon observed in real-world outbreaks (Barabási & Albert, 1999).

Additionally, the model also considers behavioural and governmental interventions, such as quarantine periods and lockdowns. For example, during a simulated quarantine period between days 10 and 25, the number of daily interactions is reduced from 10 to 3. After the quarantine, interaction levels return to normal, enabling us to observe the temporary effects of such interventions. This mimics real-world policies, where lockdowns "flatten the curve" but may also prolong the pandemic by keeping portions of the population susceptible to future infections (Kucharski et al., 2020).

Finally, the model incorporates the impact of super-spreaders: people with higher interaction rates and greater probability of spreading the virus as vectors. These individuals can drive rapid spikes in infection rates, much like real-world superspreader events. Taken together, these features: age-dependent mortality, structural interactions, stochasticity of transmission, and policy intervention, all ensure that the model more accurately reflects the complexity of COVID-19 spread and progression in the population.

Simulation Results and Analysis

Visualisation

This simulation provides valuable insights into the progression of COVID-19 by visualising changes in agents' health states over time. Agents are classified into seven health states, including Unexposed, Asymptomatic and contagious, Symptomatic and contagious, and Post-COVID immune. These states are visualised through graphs that track the distribution of health states over 50 days.

In the first visualisation, a line graph illustrates the overall pandemic progression. Initially, the majority of agents are Unexposed, but as the simulation progresses, the number of infected (asymptomatic and symptomatic) agents increases, peaking around day 20. After this peak, the number of recovered (immune) agents rises, while the number of new infections declines. A smaller proportion of the population transitions to the Deceased state, reflecting COVID-19-related fatalities. The graph provides a clear view of the rise and fall of infection levels and the eventual stabilisation as more agents recover.

Another visualisation focuses on the classic SIR model, showing the number of Susceptible, Infected, and Recovered agents throughout the simulation. The epidemic curve follows a typical pattern: infections rise quickly, peak, and then decrease as individuals recover. By the end of the simulation, the Recovered population dominates, while the death rate remains low but steady. These visualisations effectively demonstrate the dynamics of the pandemic, revealing how changes in key parameters, such as infection rates and interaction frequency, shape the progression of COVID-19.

Quantitative Analysis

In this COVID-19 simulation, the key quantitative measure is the number of infected individuals over time, including both asymptomatic and symptomatic cases. This value reflects the rate at which the

virus spreads within the population, the timing of peak infection, and how the pandemic eventually subsides. Analysing this data helps us understand the critical points in the progression of the pandemic and evaluate the effectiveness of various interventions.

We used the SIR model to illustrate the changes in the number of susceptible, infected and recovered individuals during the simulation. The curve of infected individuals plays a crucial role in depicting the evolution of the pandemic. Specifically, the changes in the number of infected individuals visually reveal the virus's transmission speed and the timing of the peak. For example, in the simulation, we observe that the number of infected individuals peaks around day 20, after which it gradually declines and stabilises around day 40. This quantitative analysis helps us identify the critical turning points of the pandemic and assess the effectiveness of public health interventions.

Another important metric is the death rate and recovery rate, representing the number of individuals who transitioned to the death state and those who acquired post-COVID immunity. These data allow us to assess the virus's lethality and the healthcare system's effectiveness during the treatment process. By comparing the ratio of recovered individuals to the number of deaths, we can better understand the impact of medical interventions and vaccination campaigns in curbing the virus's spread. The simulation shows that while the number of recoveries steadily increases, the growth in deaths during the pandemic remains a critical issue, especially among high-risk groups, highlighting the importance of allocating healthcare resources effectively.

Parameter Impact

In the simulation, the infection probability and social interaction frequency are two key parameters that directly impact the infection curve. First, infection probability determines the likelihood of the virus being transmitted during each interaction. We adjust this parameter to simulate different pandemic scenarios. For example, when the infection probability increases, the number of infected individuals rises sharply and reaches a higher peak at an earlier time point. This mirrors real-life situations where the virus's transmission capacity increases or preventive measures weaken. Conversely, lowering the infection probability flattens the infection curve, delays the peak, and reduces the total number of infections. This demonstrates that when effective protective measures or widespread vaccinations are implemented, a reduction in infection probability can effectively slow the pandemic.

Social interaction frequency also plays a crucial role in virus transmission. In the simulation, when the number of daily interactions between individuals increases, such as when there are no lockdowns or restrictions, the virus spreads faster and causes a rapid increase in the number of infections and a peak in a shorter period. When the number of daily interactions decreases, such as during lockdowns or social distancing measures, the rate of infections slows significantly, and the peak is delayed. This mirrors the real-world effects of lockdowns or social distancing policies, showing the importance of reducing social contact to control virus transmission.

By adjusting these parameters, the simulation not only demonstrates how the pandemic evolves under different conditions but also provides valuable insights for policymakers on how to optimise their strategies. For example, by limiting daily interactions and reducing infection probability, the pandemic can be controlled over a longer period while avoiding the overwhelming of healthcare systems.

Real-World Reflection

The simulation results provide valuable insights into our understanding of pandemic behaviour, particularly in the context of COVID-19. By simulating the spread of the virus in a population, the model shows how individual behaviour, government intervention, and biological factors can work together to influence the progress of an outbreak. A key finding is that social networks and behavioural responses play a key role in transmission patterns. In real life, interactions between people are not random; Instead, they usually move in more fixed social circles. Some people are known as "super-spreaders" because their contacts are far wider than the average person's, making their impact particularly significant in accelerating the spread of the virus. This aligns with real-world observations, such as that certain individuals or events (such as large gatherings) tend to have a disproportionately large impact on the spread of COVID-19.

Introducing policy interventions like lockdowns into the simulation further enhances its reflection of real-world scenarios. Reducing daily social contact can effectively slow the spread of the virus and help flatten the infection curve, but it may also prolong the duration of the pandemic. This fits in with the real world, where such interventions do reduce pressure on healthcare systems, but they must also be balanced with economic and social costs. Additionally, the simulation shows that once restrictions are lifted, cases often resurge, which is exactly what many countries have experienced when dealing with successive waves of COVID-19 infections.

Finally, the simulation highlights the role of herd immunity—whether through natural infection or vaccination—in controlling virus transmission. When infection rates are high, the total number of infections decreases, consistent with real-world observations. The interaction between immunity levels, transmission rates, and behavioural factors in the simulation reflects the delicate balance required in managing pandemics. As such, this simulation provides valuable insights into how key factors interact to shape the course of pandemics, offering lessons for future public health strategies.

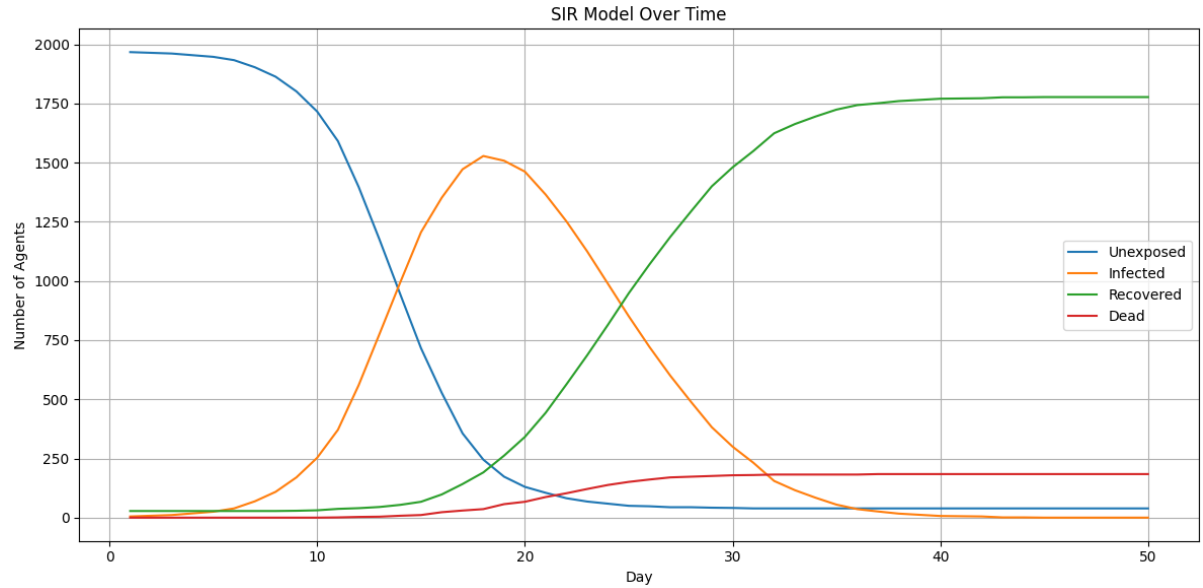
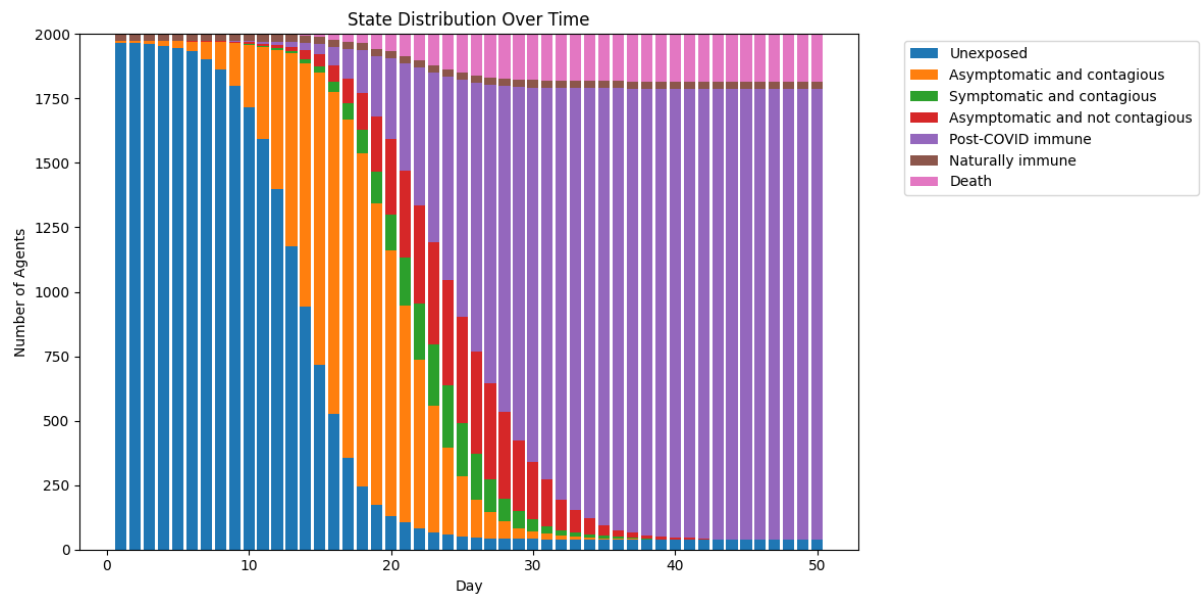
Conclusion

In summary, this project developed an agent-based model to simulate the spread of COVID-19, incorporating key factors such as individual behaviours, age, mortality rates, and the effects of lockdowns. Our simulations show that reducing the probability of infection and the frequency of social interactions can delay the peak of infections and flatten the epidemic curve. However, public health measures that are perceived to be effective might extend the duration of the pandemic. The model does have limitations, including simplified assumptions about individual behaviours and uniform compliance with public health measures, which may not fully capture real-world variability. Additionally, the model does not account for diverse virus variants or limitations in healthcare system capacity, both of which can significantly influence disease outcomes. For future work, the model can be expanded to include more complex agent behaviours, virus mutation dynamics, and healthcare system capacity. Furthermore, simulating the effects of vaccination strategies and their interaction with virus variants will enhance the model's relevance to managing an evolving pandemic. These improvements will provide deeper insights for public health policy.

Reference

1. Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character*, 115(772), 700-721. <https://doi.org/10.1098/rspa.1927.0118>
2. He, S., Tang, S., & Rong, L. (2020). A discrete stochastic model of the COVID-19 outbreak: Forecast and control. *Mathematical Biosciences and Engineering*, 17(4), 2792-2804. <https://doi.org/10.3934/mbe.2020153>
3. Epstein, J. M. (2009). Modelling to contain pandemics. *Nature*, 460(7256), 687-687. <https://doi.org/10.1038/460687a>
4. GitHub. (2020). SEIR Squire Model - Open-source COVID-19 model. *GitHub Repository*. Retrieved from <https://github.com/mrc-ide/squire>
5. SpringerLink. (2020). COVID-19 modeling using SEIR and ABM frameworks. *Springer Annals of Operations Research*. Retrieved from <https://link.springer.com>
6. Ferguson, N. M., Laydon, D., Nedjati-Gilani, G., Imai, N., Ainslie, K., & Baguelin, M. (2020). Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand. *Imperial College COVID-19 Response Team*, 1-20. <https://doi.org/10.25561/77482>
7. Ritchie, H., & Roser, M. (2024, February). Age structure. Our World in Data. <https://ourworldindata.org/age-structure>
8. Centers for Disease Control and Prevention. (n.d.). Risk for COVID-19 infection, hospitalization, and death by age group. U.S. Department of Health and Human Services. <https://www.cdc.gov/coronavirus/2019-ncov/covid-data/investigations-discovery/hospitalization-death-by-age.html>
9. Byrne, A. W., McEvoy, D., Collins, Á. B., Hunt, K., Casey, M., Barber, A., Butler, F., Griffin, J., Lane, E. A., McAloon, C., O'Brien, K., Wall, P., Walsh, K. A., & More, S. J. (2020). Inferred duration of infectious period of SARS-CoV-2: Rapid scoping review and analysis of available evidence for asymptomatic and symptomatic COVID-19 cases. *BMJ Open*, 10(8), e039856. <https://doi.org/10.1136/bmjopen-2020-039856>
10. Barabási, A. L., & Albert, R. (1999). Emergence of scaling in random networks. *Science*, 286(5439), 509-512. <https://doi.org/10.1126/science.286.5439.509>
11. Kucharski, A. J., Russell, T. W., Diamond, C., Liu, Y., Edmunds, J., Funk, S., & Eggo, R. M. (2020). Early dynamics of transmission and control of COVID-19: A mathematical modelling study. *The Lancet Infectious Diseases*, 20(5), 553-558. [https://doi.org/10.1016/S1473-3099\(20\)30144-4](https://doi.org/10.1016/S1473-3099(20)30144-4)

Appendix



Social Network

