Simulation of Epidemic Spread Using Cellular Automata

Ernandes Costa(ernandes.scluiz@ufrpe.br)

Federal Rural University of Pernambuco, Department of Statistics and Informatics.

Abstract

This study explores how cellular automata can be used to simulate the spread of infectious diseases in a population. By using a grid-based model, we analyze the effects of different infection rates and recovery times on disease progression. Our findings highlight how spatial interactions influence outbreaks, offering insights into possible intervention strategies. This approach provides a valuable tool for understanding disease dynamics in a way that complements traditional mathematical models.

Introduction

Understanding how diseases spread is essential for public health planning and response. Traditional epidemic models, such as the widely used SIR (Susceptible-Infected-Recovered) framework, rely on equations to describe the flow of individuals between different health states. However, these models often assume that individuals interact randomly, which does not always reflect real-world patterns of disease transmission.

Cellular automata (CA) provide an alternative way to model epidemics, focusing on local interactions. In a CA model, individuals are represented as cells in a grid, each following simple rules that determine whether they become infected, recover, or remain susceptible. This structure allows for the emergence of complex behaviors, such as the formation of infection clusters, which are not easily captured by traditional models. By simulating the spread of a disease through a CA, we can gain insights into how spatial factors affect outbreaks and how different intervention strategies might work in practice.

Methods

Our model represents a population as a two-dimensional grid, where each cell can be in one of three states: Susceptible (S), Infected (I), or Recovered (R). The simulation follows these basic rules:

- 1. If a susceptible cell has an infected neighbor, it has a probability of becoming infected.
- 2. Infected cells remain in that state for a fixed number of time steps before recovering.
- 3. Recovered cells do not get infected again.

The simulation starts with a small number of infected individuals randomly distributed across the grid. Each time step, the model updates the state of all cells based on their neighbors. The results are visualized through animations and heatmaps, allowing us to observe how the infection spreads over time.

Results and Discussion

The simulations show that the spread of infection depends on factors such as the probability of transmission and the density of the population. Higher transmission probabilities lead to faster outbreaks, while lower probabilities result in slower, more localized infections. The duration of the infection in individuals also plays a role in how long an outbreak lasts before stabilizing.

One key observation is that infections tend to spread in clusters rather than evenly across the grid. This happens because individuals interact mostly with their immediate neighbors, rather than mixing randomly with the entire population. This behavior is closer to how diseases actually spread in real-world settings, where interactions are often limited to families, workplaces, and communities.

Another interesting finding is that simple changes in the rules of the model can simulate different public health interventions. For example, reducing the number of contacts between individuals (by blocking certain connections in the grid) can slow down the spread, mimicking the effects of social distancing or localized lockdowns.

Conclusion

Cellular automata provide a flexible and visually intuitive way to model disease spread. Unlike traditional mathematical models that assume uniform mixing, CA models capture spatial interactions and allow for emergent behaviors. The insights gained from these simulations can help researchers and policymakers better understand epidemic dynamics and evaluate different intervention strategies. Future work could extend this approach by incorporating mobility patterns or adding more complexity to individual behaviors, making the model even more realistic.

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