

# Epidemiological Simulation Using Cellular Automata: A Computational Approach to the SIR Model

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## Abstract

This paper presents an epidemiological simulation based on the Susceptible-Infected-Recovered (SIR) model using cellular automata. The simulation occurs on a two-dimensional grid where cells represent individuals and follow local rules for disease propagation. The model considers a fixed probability of infection and a recovery time for each infected individual. The results illustrate the spread dynamics of an epidemic and the impact of recovery time on disease progression. This study demonstrates how computational models can be used to understand and predict epidemic behaviors, providing insights for decision-making in public health.

**Keywords:** Epidemiological Simulation, SIR Model, Cellular Automata, Disease Propagation, Computational Modeling.

## Introduction

Epidemiological modeling is a fundamental tool in understanding the spread of infectious diseases. Traditional mathematical models, such as the SIR model, offer valuable insights into epidemic dynamics. However, these models often rely on differential equations that assume homogeneous mixing of the population. In contrast, cellular automata-based models provide a spatially explicit approach, capturing local interactions and heterogeneity within populations.

This study implements an SIR-based simulation using cellular automata to explore disease propagation dynamics. The simulation takes place in a discrete two-dimensional grid where individuals interact with their immediate neighbors. Each individual transitions through three states (susceptible, infected, and recovered) according to predefined rules governing infection probability and recovery time. The main objective is to analyze how an infection spreads within a confined population and observe emergent patterns over multiple time steps.

## Methods

The simulation was developed using Python, employing the NumPy and Matplotlib libraries for efficient data handling and visualization. The methodology follows these key steps:

- **Grid Initialization:** A 50×50 matrix represents the population, where each cell corresponds to an individual.
- **State Definitions:** Each individual can be in one of three states:
  - **Susceptible (S):** Healthy individuals who may contract the infection.
  - **Infected (I):** Individuals who can spread the disease to their susceptible neighbors.
  - **Recovered (R):** Individuals who have overcome the infection and are immune.
- **Infection Dynamics:**
  - At the start, one randomly selected cell is infected.
  - Susceptible individuals become infected with a probability of 30% if they have at least one infected neighbor.
  - Infected individuals recover after 10 time steps and transition to the recovered state.
- **Simulation Execution:**
  - The grid updates at each time step according to the transition rules.
  - Visualization is provided through a color-coded representation (white for susceptible, red for infected, and green for recovered individuals).

## Results

The simulation was run for 50 time steps, and the following observations were made:

- **Initial Spread:** The infection quickly propagates to neighboring cells, forming localized clusters of infection.
- **Peak Infection Period:** Around time step 20, the number of infected individuals reaches its peak before declining as more individuals recover.
- **Disease Saturation:** By time step 40, most of the grid has transitioned to the recovered state, indicating that the epidemic has run its course.
- **Effect of Recovery Time:** A shorter recovery time results in a faster decline of the epidemic, whereas a longer recovery period prolongs disease spread.

## Discussion

The results of the simulation highlight the significance of local interactions in the spread of an epidemic. Unlike traditional compartmental models that assume well-mixed populations, the cellular automata approach allows for spatial heterogeneity, leading to more realistic epidemic dynamics. The formation of infection clusters, the non-uniform spread of the disease, and the emergence of immune zones after recovery are important aspects observed in this study.

Further refinements to the model could include factors such as mobility, vaccination strategies, and external interventions to assess their impact on epidemic control. Additionally, incorporating stochastic variations in recovery time and immunity duration could provide a more nuanced understanding of real-world disease spread.

## Conclusion

This study demonstrates how cellular automata can effectively model epidemic spread using local interaction rules. The findings highlight the importance of infection probability and recovery time in shaping epidemic dynamics. Compared to traditional SIR models, cellular automata offer a more granular approach, allowing for spatial variations in disease propagation. Future work could explore additional factors such as reinfection, vaccination strategies, and mobility patterns to enhance model realism.

## References

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