A Cellular Automata-Based Approach for Simulating Epidemic Spread

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ABSTRACT

Mathematical models of epidemic spread are crucial for understanding disease dynamics and informing public health interventions. Cellular automata (CA) offer a flexible and computationally efficient framework for simulating spatially explicit disease transmission. In this study, we present a CA-based epidemic model incorporating the Susceptible-Infected-Recovered (SIR) paradigm. Our simulation framework captures spatial heterogeneities and stochastic interactions between individuals, providing insights into the spread and control of infectious diseases. This model can be used for educational purposes, epidemiological forecasting, and decision-support systems.

Introduction

The study of infectious disease dynamics has long relied on mathematical models, such as compartmental SIR models, to understand transmission patterns and evaluate mitigation strategies. However, classical differential equation-based models often assume homogeneous mixing, which does not reflect real-world spatial heterogeneity. Cellular automata provide an alternative approach, modeling individuals as discrete entities that interact with their local neighbors. This enables a more realistic representation of spatial and stochastic processes in disease spread.

This paper presents a computational framework using a two-dimensional CA model to simulate an epidemic outbreak, demonstrating its effectiveness in capturing spatial transmission dynamics. The model is particularly useful for investigating localized outbreaks, assessing the impact of control measures, and evaluating various transmission scenarios. Moreover, cellular automata facilitate high-resolution modeling, making them valuable for understanding complex epidemic dynamics beyond classical mathematical models.

Methods

We implemented a two-dimensional CA model where each cell represents an individual in a population. The states of the cells follow the SIR framework: Susceptible (S), Infected (I), and Recovered (R). The transition probabilities between these states are determined by infection (τ) and recovery (γ) rates. Disease transmission occurs through local interactions, defined by a Moore neighborhood. The simulation runs iteratively over a predefined number of time steps, updating the state of each cell based on its neighbors.

The model parameters were set as follows: grid size = 50x50, initial infection rate = 0.02, $\tau = 0.3$, and $\gamma = 0.1$. The boundary conditions were assumed to be periodic to eliminate edge effects. The implementation was carried out using Python and Matplotlib for visualization, ensuring an intuitive representation of infection propagation. To analyze the results, we recorded the number of susceptible, infected, and recovered individuals over time, plotting their respective curves. We also performed sensitivity analyses by varying τ and γ, allowing us to explore how different scenarios impact epidemic progression.

Results and Discussion

The simulation successfully reproduced key epidemiological phenomena, including localized outbreak clusters and wave-like propagation patterns. The role of spatial structure was evident, as transmission dynamics varied depending on population density and neighborhood connectivity. The epidemic curve followed classical trends observed in SIR models, showing an initial increase in infections followed by a peak and subsequent decline as individuals recovered.

Further analysis indicated that altering τ and γ significantly influenced epidemic outcomes. Higher infection rates led to rapid outbreaks, whereas increased recovery rates curtailed transmission. The introduction of mobility patterns or external perturbations, such as vaccination campaigns, demonstrated that spatial interventions effectively reduced infection spread.

Additionally, we analyzed the impact of different neighborhood structures (von Neumann vs. Moore) on disease propagation. The results showed that using a Moore neighborhood led to more widespread infections due to the higher number of neighboring contacts per individual, whereas von Neumann neighborhoods resulted in slower propagation rates. These findings suggest that spatial topology significantly affects epidemic dynamics and should be carefully considered in public health models.

To validate the simulation, we compared our results with existing epidemiological studies, noting strong qualitative agreement. This suggests that cellular automata models can serve as valuable tools for policy evaluation, particularly in scenarios where traditional differential equation models may not capture spatial dependencies.

Conclusion

This study highlights the utility of cellular automata models for simulating epidemic spread in a spatially explicit manner. The proposed approach provides a flexible and computationally efficient tool for investigating infectious disease dynamics, complementing traditional mathematical models. Future work may integrate real-world mobility data, contact networks, and adaptive interventions to enhance model realism.

The results underscore the importance of spatial considerations in epidemic modeling, which is critical for designing effective public health strategies. The integration of cellular automata with empirical epidemiological data could further improve predictive capabilities, aiding in decision-making processes for outbreak mitigation and disease control. Expanding the model to include agent-based interactions and heterogeneous population distributions could enhance its applicability in real-world epidemiological studies.

Acknowledgements

The author would like to acknowledge the contributions of computational epidemiology researchers whose work inspired this study.

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