Modeling Disease Spread and Health Behavior Diffusion through Clustering and Social Network Exposure

HSN-619 (Project Assignment)

2024-25 Autumn

OUTLINE

- INTRODUCTION
- SUMMARY OF BASE PAPERS
- LITERATURE REVIEW
- DATA AND MEASURES
- THEORETICAL FRAMEWORKS
- RESULTS
- PUBLIC HEALTH IMPLICATIONS
- CONCLUSION
- REFERENCES

INTRODUCTION

The dynamics of infectious disease spread and the prevalence of chronic conditions are multifaceted issues influenced by a variety of factors, including socioeconomic status, social networks, and individual health behaviors.

This report presents a comprehensive exploration of these dynamics through four distinct analyses, each addressing a critical aspect of disease transmission.

The traditional models, such as the SIR (Susceptible-Infectious-Recovered) framework, often fail to account for the complexity of social networks that influence not only the rate of transmission but also the reach of health behaviors across socioeconomic classes.

The motivation behind this study is to extend these classical models by incorporating Social Network Exposure (SNE) and economic connectedness into the analysis of disease transmission and health behavior diffusion.

SUMMARY OF FIRST BASE PAPER

The first paper is Social Capital I: Measurement and Associations with Economic Mobility by Raj Chetty et al.

Defining Social Capital

Economic Connectedness (EC): This measures the extent to which people from low and high socioeconomic statuses(SES) are connected. It's calculated as the share of high-SES friends among low-SES individuals, normalized by the overall share of high-SES individuals.

Network Cohesiveness: This focuses on the structure of social networks, particularly the presence of cliques and the extent to which friendships are reciprocated. It is measured by clustering coefficient, support ratio, and spectral homophily.

Civic Engagement: Measured using indicators like volunteering rates, participation in civic organizations, and levels of trust.

Key Findings

- **Geographical Variation:** All three measures of social capital show significant variation across US counties.
- **Economic Connectedness and Mobility:** The study finds a strong positive association between EC and upward income mobility. Children from low-income families who grow up in areas with higher EC tend to have higher incomes in adulthood. The study estimates that if children with low-SES parents grew up in counties with EC comparable to that of the average child with high-SES parents, their adult incomes would increase by 20% on average.
- **Other Social Capital Measures:** Network cohesiveness and civic engagement measures show weaker and less consistent correlations with economic mobility. For example, while civic engagement tends to be higher in rural areas, it is more weakly correlated with economic mobility once the data is weighted to account for the greater concentration of low-income families in urban communities.

SUMMARY OF SECOND BASE PAPER

The second paper is Social Capital II: Determinants of Economic Connectedness by Raj Chetty et al.

Decomposing Economic Connectedness

Exposure: The extent to which individuals from different SES backgrounds are present in the same social settings(e.g., schools, religious organizations, workplaces). The study finds that approximately half of the difference between low and high-SES people is due to differences in exposure.

Friending Bias: The tendency for individuals to form friendships with others from similar SES backgrounds, even when controlling for the level of exposure to people from different SES groups within a given setting. The study finds that friending bias is more pronounced in larger, more diverse high schools.

Key Findings

Exposure and Bias Contribute Equally: Both exposure and friending bias play significant roles in shaping EC, each explaining roughly half of the variation in the share of high-SES friends between low- and high-SES individuals.

Context Matters: The level of friending bias varies substantially across different social settings. For instance, friend-ing bias tends to be lower in religious organizations compared to schools or workplaces.

Policy Implications

Integration for Low Bias: In settings with low friending bias, increasing socioeconomic integration (exposure) can effectively boost EC.

Addressing Bias Directly: In settings with high friending bias, interventions should focus on reducing bias within existing groups to promote cross-SES friendships.

Literature Review

Year	Title	Author(s)	Summary
2023	Spatially Explicit Effective Reproduction Numbers from Incidence and Mobility Data	Cristiano Trevisin et al.	Proposes a method to estimate spatially explicit reproduction numbers Rk(t)R_k(t)Rk(t), incorporating mobility data across interconnected communities. The method improves understanding of disease transmission dynamics by accounting for mobility fluxes.
2021	Cryptic Transmission of SARS-CoV-2 and the First COVID-19 Wave in Europe and the United States	Jessica T. Davis et al.	Uses a global metapopulation model to show the undetected spread of SARS-CoV-2 during the early pandemic due to limited testing. The study emphasizes the role of international travel in spreading the virus.
2021	Modeling the Interplay Between Demography, Social Contact Patterns, and SARS-CoV-2 Transmission in the South-West Shewa Zone of Oromia Region, Ethiopia	Filippo Trentini et al.	Simulates COVID-19 spread in Ethiopia using social contact data from various settings. The study shows higher infection rates in urban areas but more severe disease in rural regions, influenced by demographic and social factors.
2021	On a Discrete-Time Network SIS Model with Opinion Dynamics	Weihao Xuan et al.	Introduces a discrete-time SIS model integrated with opinion dynamics to explore how individuals' perceptions of disease severity affect transmission within a network. Simulation results highlight the interaction between health perceptions and infection rates.
2019	Reactive School Closure Weakens the Network of Social Interactions and Reduces the Spread of Influenza	Maria Litvinova et al.	Investigates the impact of school-closure policies on influenza spread by analyzing social interactions during school closures in Russia. The study used contact diaries and a hybrid survey-modeling framework to demonstrate significant reductions in contacts and infection rates.
2018	Measurability of the Epidemic Reproduction Number in Data-Driven Contact Networks	Quan-Hui Liu et al.	Explores the limitations of the basic reproduction number, R0R_0R0, in heterogeneous real-world networks, proposing the time-varying reproduction number R(t)R(t)R(t) to better capture epidemic dynamics using multiplex networks in Italian and Dutch populations.
2017	Spread of Zika Virus in the Americas	Qian Zhang et al.	Analyzes the spatial and temporal dynamics of the Zika virus using the GLEAM model, integrating demographic and mobility data. Findings reveal slow growth and heterogeneity in the epidemic due to vector dynamics and human movement.
2016	How Social Structures, Space, and Behaviors Shape the Spread of Infectious Diseases Using Chikungunya as a Case Study	Henrik Salje et al.	Uses the chikungunya outbreak in Bangladesh to assess the impact of social interactions and spatial proximity on disease transmission, highlighting the role of household and neighborhood factors in shaping the epidemic.
2014	The Representativeness of a European Multi-Center Network for Influenza-Like Illness Participatory Surveillance	Pietro Cantarelli et al.	Evaluates the representativeness of Influenzanet, a European online participatory surveillance system for influenza, identifying demographic biases and underrepresentation of certain population groups such as smokers and the elderly.
2009	Percolation and Epidemics in Random Clustered Networks	Joel C. Miller	Examines percolation and epidemic dynamics in clustered networks, showing that clustering increases the epidemic threshold and reduces component sizes compared to un-clustered networks.

Data used in the Base Papers

- **Dataset Overview**:
- 21 billion friendship links between Facebook users aged 25-44 in the U.S.
- Focus on this age group due to high Facebook usage (80%) and comparability with American Community Survey (ACS) demographic data.

- **User Information**:
- Facebook profile data includes age, gender, ZIP code, county, relationship status, education, donations, phone model, mobile carrier, device usage, familial ties, and group memberships.
- Socioeconomic status (SES) is constructed using this data.

- **Parental SES and Group-Level Data**:
- Family SES during childhood is derived from self-reported family connections, names, posts, and life events.
- Friendships are categorized by shared affiliations, such as schools, employers, neighborhoods, and recreational groups.

- **External Datasets**:
- **American Community Survey (ACS)**: Median incomes, racial/ethnic demographics, and community characteristics.
- **Opportunity Atlas**: Data on income mobility, graduation rates, birth rates.
- **NCES and CRDC**: School-level variables.
- **IPEDS**: College-level data.
- **Census Data**: Used for determining the population with below-national-median parental income for weighting.

Statistical Analysis and Measures

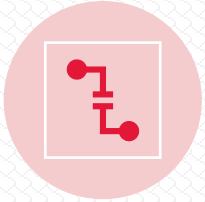
This report primarily focuses on theoretical frameworks to understand the dynamics of infectious disease transmission. Simulations have been conducted to illustrate the model outcomes and validate the theoretical constructs. The simulations were implemented using Python, with the accompanying code provided in the attached folder. This computational approach allows for the exploration of various scenarios and the assessment of the impact of different variables on disease dynamics. The authors calculated the exposure and clustering coefficient, which are utilized in this paper

Infection Population: Measured as the number of individuals currently infected at a given time, expressed per 1,000 individuals in the population.

Susceptible Population: Defined as the number of individuals who are not infected and are at risk of contracting the disease.

Latent Period: The duration during which individuals are infected but not yet infectious. This period is crucial for understanding the dynamics of disease spread.

Social Network Exposure (SNE): Quantified based on the density of social ties within communities, measured through network analysis techniques. The SNE ranges from 0 to 1, where higher values indicate greater social andeconomic connectedness.



Clustering Coefficient: Calculated to assess the degree of interconnectedness among individuals within social networks. Like SNE, the clustering coefficient ranges from 0 to 1, with higher values reflecting more tightly-knit social groups.

THEORETICAL FRAMEWORKS

Analysis 1: Economic Connectedness and Disease Spread

Existing Theory: The Strength of Weak Ties

Granovetter's (1973) theory emphasizes that weak ties, or connections between individuals from different socioeconomic backgrounds, play a critical role in spreading information. Applied to disease transmission, these cross-class ties enable broader and faster infection spread by linking otherwise isolated social groups. In contrast, strong homophily (friending bias) limits cross-group transmission, leading to more contained outbreaks.

Extension: Social Network Exposure (SNE) in the SIR Model

To model economic connectedness, we introduce Social Network Exposure (SNE) into the SIR (Susceptible-Infectious-Recovered) model. SNE quantifies cross-class interactions, influencing infection rates. Higher SNE values reflect more frequent interactions between socioeconomic groups, accelerating disease spread, while lower SNE indicates stronger friending bias, limiting cross-group infections.

1. **Modified Susceptible Population**:

$$\frac{ds(t)}{dt} = -\beta \cdot k \cdot \text{SNE} \cdot i(t) \cdot [1 - r(t) - i(t)]$$

where:

- $s(t)$: Proportion of the population that is susceptible at time t.
- β: Infection rate, the probability of transmission per contact.
- k: Average number of contacts an individual has.
- $i(t)$: Proportion of the population that is infected at time t.
- $r(t)$: Proportion of the population that is recovered.

The SNE modifies the infection rate based on the degree of cross-class interactions, increasing transmission as economic connectedness rises.

2. **Modified Infected Population**:

$$\frac{di(t)}{dt} = \beta \cdot k \cdot \text{SNE} \cdot i(t) \cdot [1 - r(t) - i(t)] - \mu \cdot i(t)$$

where:

- μ: Recovery rate, the rate at which infected individuals recover.

The infection term now reflects the influence of SNE, where higher cross-class interactions boost the probability of infected individuals contracting the disease [20].

3. **Recovered Population**:

$$\frac{dr(t)}{dt} = \mu \cdot i(t)$$

where:

- $r(t)$: Proportion of the population that has recovered at time t.

Analysis 2: Age-Structured Models for Understanding Infectious Disease Dynamics

Age-structured models are essential for studying infectious diseases as they account for variations in transmission rates, susceptibility, and recovery across different age groups. By segmenting populations by age, these models provide a nuanced understanding of disease dynamics and facilitate the design of targeted interventions.

1.Population Segmentation:

1. Infection Rates: Younger individuals often have higher contact rates (e.g., through schools), increasing transmission potential, while older individuals may face more severe outcomes.
2. Recovery Rates: Recovery varies by age, with younger individuals generally recovering faster due to differences in immune response.
3. Contact Patterns: The model considers interaction frequencies, such as children having more close contacts with peers, while older adults may engage in fewer but higher-risk interactions.

2.Dynamic Interactions: These models simulate interactions between age groups, enabling the study of disease spread over time based on differing infection and recovery rates.

Equations for Age-Structured Models

1. **Susceptible Population:**

$$\frac{dS_i}{dt} = -\beta_i S_i \sum_j \left(\frac{I_j}{N_j}\right) \cdot C_{ij}$$

- S_i: The number of susceptible individuals in age group A_i. - β_i: The transmission rate for age group A_i. - I_j: The number of infected individuals in age group A_j. - C_{ij}: The contact matrix element quantifying the interaction rates between age groups A_i and A_j.

$$C_{ij} = SNE_{ij}$$

where: - SNE_{ij}: The Social Network Exposure factor that quantifies the level of cross-class interaction between age groups A_i and A_j. Higher values indicate more interaction and greater economic connectedness.

2. **Infectious Population:**

$$\frac{dI_i}{dt} = \beta_i S_i \sum_j \left(\frac{I_j}{N_j}\right) \cdot C_{ij} - \gamma_i I_i$$

- I_i: The number of infectious individuals in age group A_i. - γ_i: The recovery rate for age group A_i.

3. **Recovered Population:**

$$\frac{dR_i}{dt} = \gamma_i I_i$$

- R_i: The number of recovered individuals in age group A_i.

These equations describe the transitions between susceptible, infected, and recovered states within each age group, while the contact matrix C captures the interactions between different age groups, influencing the spread of the disease.

Matrix Representation of the Model

In matrix form, the system of equations can be represented as:

$$\mathbf{X} = \begin{bmatrix} S_1 \\ S_2 \\ \vdots \\ S_n \\ I_1 \\ I_2 \\ \vdots \\ I_n \\ R_1 \\ R_2 \\ \vdots \\ R_n \end{bmatrix} \quad \text{and} \quad \mathbf{C} = \begin{bmatrix} C_{11} & C_{12} & \dots & C_{1n} \\ C_{21} & C_{22} & \dots & C_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ C_{n1} & C_{n2} & \dots & C_{nn} \end{bmatrix}$$

The contact matrix $\mathbf{C}$ captures the interaction rates between the different age groups, which drive the spread of the disease. The system of equations can be summarized as:

$$\frac{d\mathbf{X}}{dt} = \mathbf{F}(\mathbf{X}) - \mathbf{G}(\mathbf{X})$$

Where $\mathbf{F}$ represents the infection dynamics, and $\mathbf{G}$ represents the recovery dynamics.

Analysis 3: SLIAR Model with Social Network Exposure (SNE) for Infectious Disease Dynamics

The SLIAR model is an extension of the traditional SIR (Susceptible-Infectious-Recovered) framework, adding compartments to capture latent and asymptomatic stages of infection.

1. Compartment Definitions:

•S (Susceptible): Individuals who are not infected and can become infected.

•L (Latent): Infected individuals who are not yet infectious and cannot transmit the disease.

•I (Infectious Symptomatic): Infected individuals displaying symptoms who can spread the disease.

•A (Infectious Asymptomatic): Infected individuals without symptoms who can still transmit the disease.

•R (Removed): Individuals who have recovered with immunity or have died, thus unable to spread the disease.

2. Incorporation of Social Network Exposure (SNE): To enhance the SLIAR model, we modify the equations to include Social Network Exposure (SNE), reflecting the impact of cross-class interactions on disease transmission dynamics. The SNE variable influences infection rates based on social interactions within the community.

Modified SLIAR Model Equations:

1. **Susceptible Population:**

$$\frac{dS}{dt} = -\beta S\,(I + \delta A) \cdot SNE, \quad S(0) = S_0 > 0$$

- SNE: A factor that quantifies social network exposure. Higher values indicate more interaction and thus a higher chance of infection. [20] - β: Transmission rate. - I: Infectious symptomatic individuals. - A: Infectious asymptomatic individuals. - δ: A factor representing the increased risk of infection from asymptomatic individuals.

2. **Latent Population:**

$$\frac{dL}{dt} = \beta S\,(I + \delta A) \cdot SNE - \kappa L, \quad L(0) = L_0 \geq 0$$

- Here, the latent individuals' infection dynamics are also influenced by the SNE factor. - κ: Rate at which individuals progress from the latent stage to the infectious stage.

3. **Infectious Symptomatic Population:**

$$\frac{dI}{dt} = p\kappa L - \alpha I, \quad I(0) = I_0 \geq 0$$

- p: Proportion of latent individuals who become symptomatic. - α: Recovery rate for symptomatic individuals. - This equation remains unchanged since the symptomatic individuals' progression is not directly influenced by SNE.

4. **Infectious Asymptomatic Population:**

$$\frac{dA}{dt} = (1 - p)\kappa L - \eta A, \quad A(0) = A_0 \geq 0$$

- η: Recovery rate for asymptomatic individuals. - This equation remains unchanged as well.

5. **Removed Population:**

$$\frac{dR}{dt} = f\alpha I + \eta A, \quad R(0) = R_0 \geq 0$$

- f: A factor representing the fraction of symptomatic individuals that contribute to the removed class through recovery or death. - This equation remains unchanged as well.

6. **Total Population:**

$$N(t) = S(t) + L(t) + I(t) + A(t) + R(t)$$

- This equation remains unchanged as well.

The inclusion of SNE modifies the transmission dynamics for both the Susceptible and Latent compartments. By capturing the effect of varying degrees of social interactions and economic connectedness, the model better reflects real-world disease dynamics.

Analysis 4: Clustered Network Epidemics via Incorporating Social Interactions

This approach is particularly relevant in studying how social interactions influence the spread of infectious diseases among closely-knit groups. Here, the clustering coefficient is used, which is calculated to assess the degree of interconnectedness among individuals within social networks.

1. Infection Probability in a Triangle (Social Group)

The model treats nodes u, v, and w as members of a triangular social group, representing tight-knit relationships such as family, friends, or coworkers. This configuration captures how infections spread among interconnected individuals.

Infection Probabilities: - **Both Neighbors Infected**:

$$P(\text{infect both}) = C \cdot (3T^2 - 2T^3)$$

- **Exactly One Neighbor Infected**:

$$P(\text{infect one}) = 2T(1-T)^2$$

- **Neither Neighbor Infected**:

$$P(\text{infect none}) = (1-T)^2$$

Here, T is the transmissibility, reflecting the probability of infection occurring along an edge. The clustering coefficient C enhances the likelihood of simultaneous infections within these close-knit groups.

Expected Number of Infections: The expected number of infections per triangle can be calculated as follows:

$$E[\text{Infections}] = 2T(1-T)^2 + 2C(3T^2 - 2T^3) = 2T + 4T^2 - 4T^3 + 6CT^2 - 4CT^3$$

This equation illustrates the dependence of infection spread on both transmissibility T and the clustering coefficient C.

2. Next-Generation Matrix for Social Network Exposure (SNE)

To capture the transmission dynamics through different pathways—independent edges and triangle edges—a next-generation matrix is defined:

$$\begin{bmatrix} n_I(s+1) \\ n_\Delta(s+1) \end{bmatrix} = \begin{bmatrix} c_{II} & c_{I\Delta} \\ c_{\Delta I} & c_{\Delta\Delta} \end{bmatrix} \begin{bmatrix} n_I(s) \\ n_\Delta(s) \end{bmatrix}$$

Where: - $n_I(s)$: Number of nodes infected through independent edges. - $n_\Delta(s)$: Number of nodes infected through triangle edges.

Calculating the Matrix Elements: 1. Infection along Independent Edges:

$$c_{II} = T \cdot \frac{\langle K_I^2 - K_I \rangle}{\langle K_I \rangle}$$

2. Infection along Triangle Edges from Independent Edges:

$$c_{I\Delta} = (2T + 4T^2 - 4T^3 + 6CT^2 - 4CT^3) \cdot \frac{\langle K_I K_\Delta \rangle}{\langle K_I \rangle}$$

3. Infection along Independent Edges from Triangle Edges:

$$c_{\Delta I} = T \cdot \frac{\langle K_I K_\Delta \rangle}{\langle K_\Delta \rangle}$$

4. Infection along Triangle Edges:

$$c_{\Delta\Delta} = (2T + 4T^2 - 4T^3 + 6CT^2 - 4CT^3) \cdot \frac{\langle K_\Delta^2 - K_\Delta \rangle}{\langle K_\Delta \rangle}$$

These matrix elements capture the pathways through which infections spread and highlight the importance of both individual and clustered interactions in the overall transmission dynamics.

3. Basic Reproduction Number R_0 and Critical Transmissibility T_c

The basic reproduction number R_0 is derived from the dominant eigenvalue of the next-generation matrix:

$$R_0 = \text{Dominant Eigenvalue of the Next-Generation Matrix}$$

- If $R_0 > 1$, the infection can spread; if $R_0 < 1$, the infection will die out.

RESULTS

Analysis 1: Economic Connectedness and Disease Spread

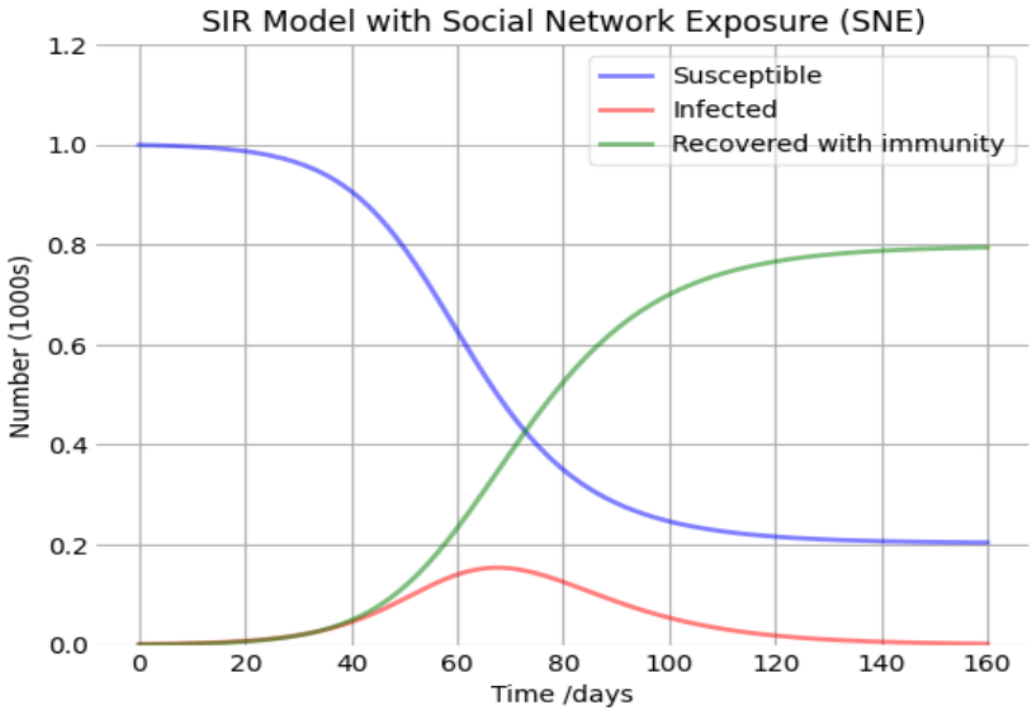


Figure 1: SIR Model with Social Network Exposure (SNE) =1

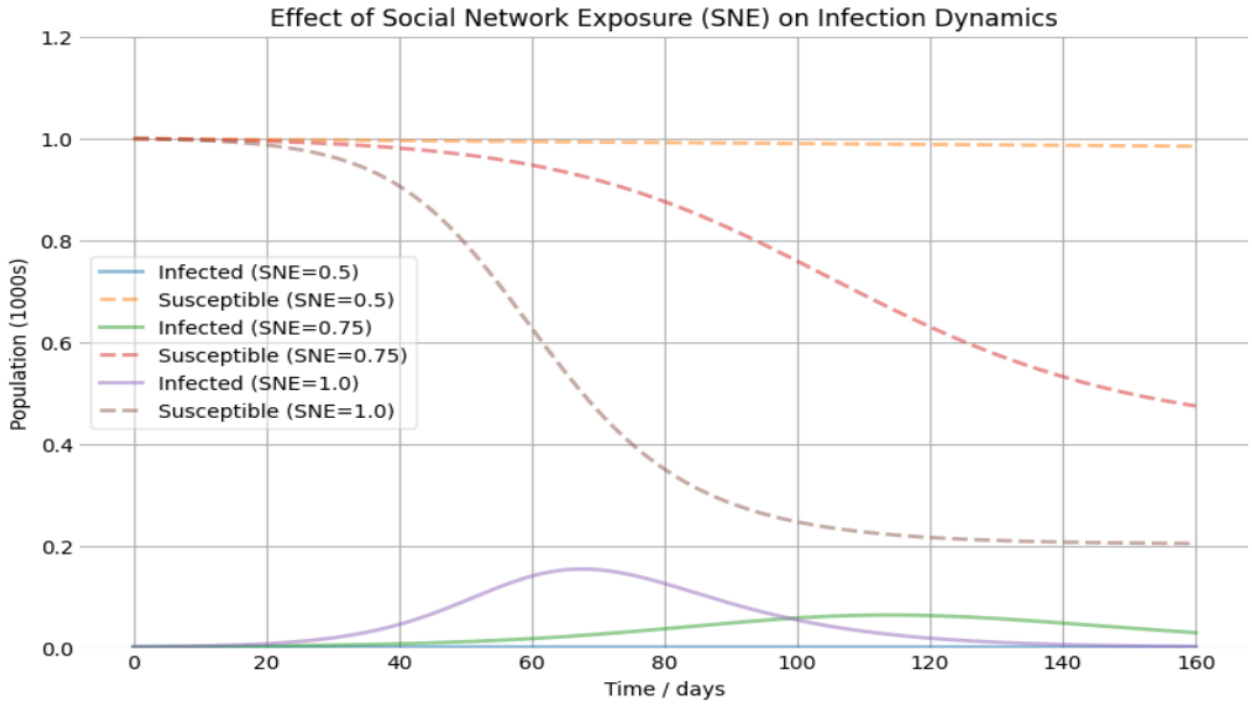
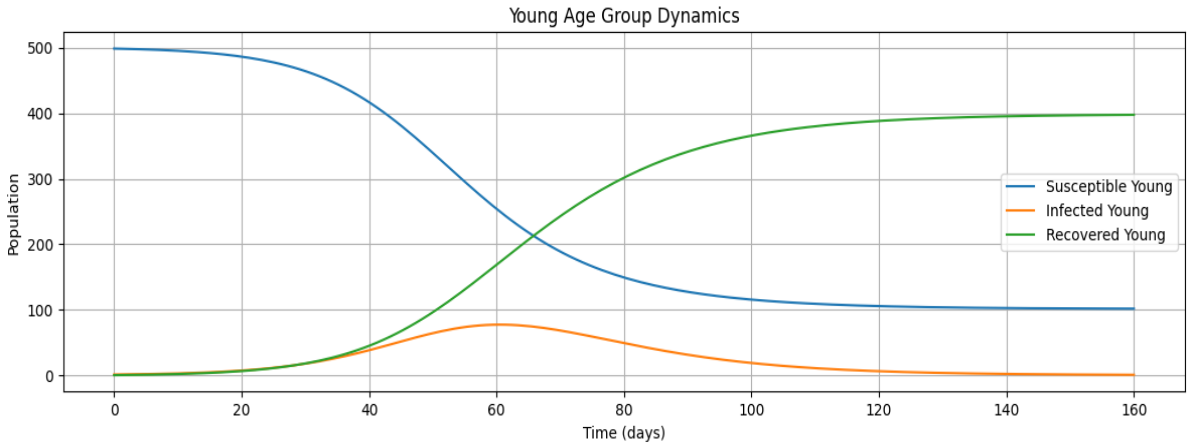


Figure 2: Effect of Social Network Exposure (SNE) on Infection Dynamics

In regions with high economic connectedness (low friending bias), disease spreads rapidly across socioeconomic groups, leading to an earlier peak in infections and a faster decline in susceptible individuals, as shown in Figure 2. This accelerated transmission strains healthcare resources sooner, requiring prompt interventions. Conversely, in areas with high friending bias (low economic connectedness), outbreaks are more localized within socioeconomic groups, resulting in slower, stratified transmission. This delays the infection peak and causes a more gradual decline in susceptible individuals.

Analysis 2: Age-Structured Models for Understanding Infectious Disease Dynamics



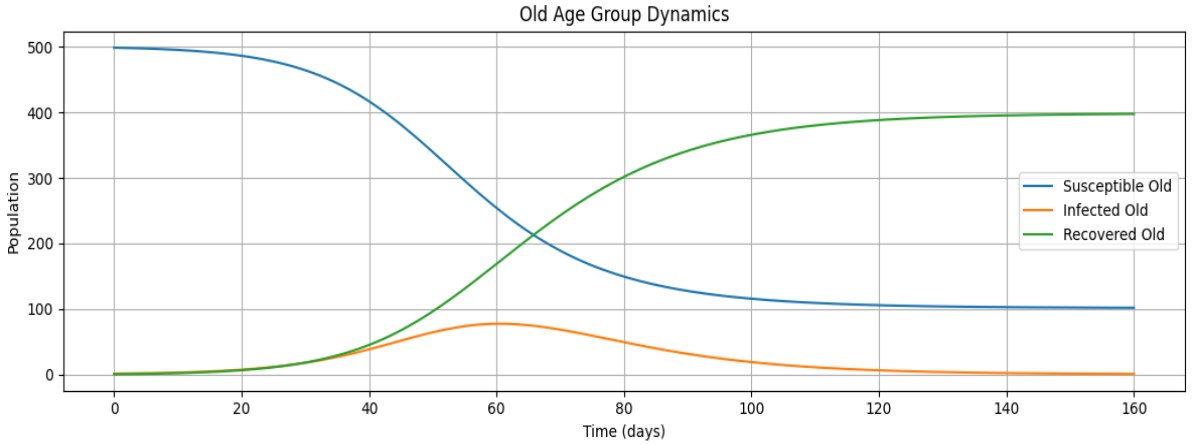
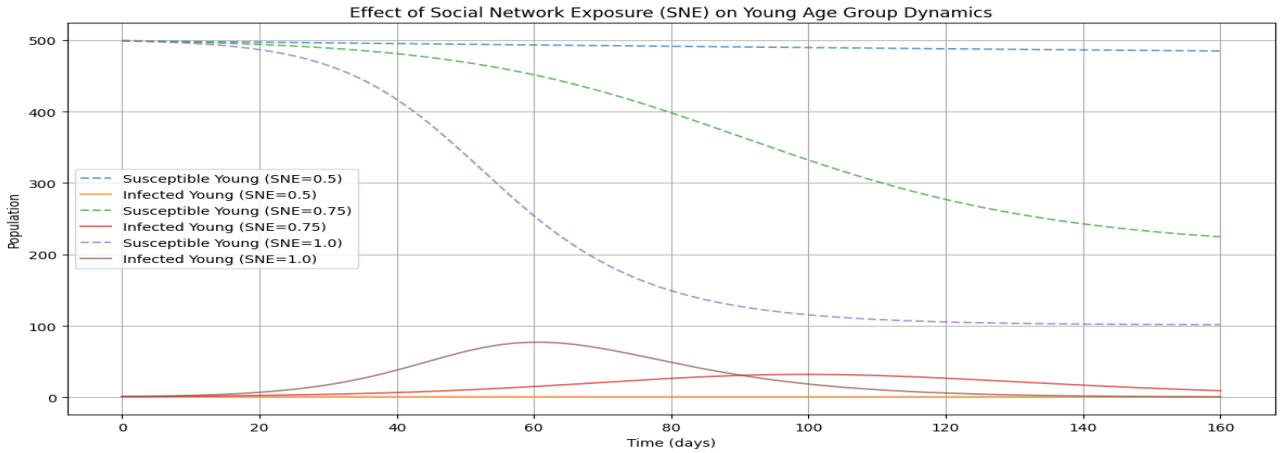


Figure 3: SIR Model with Young and Old Age Group Dynamics with SNE =1



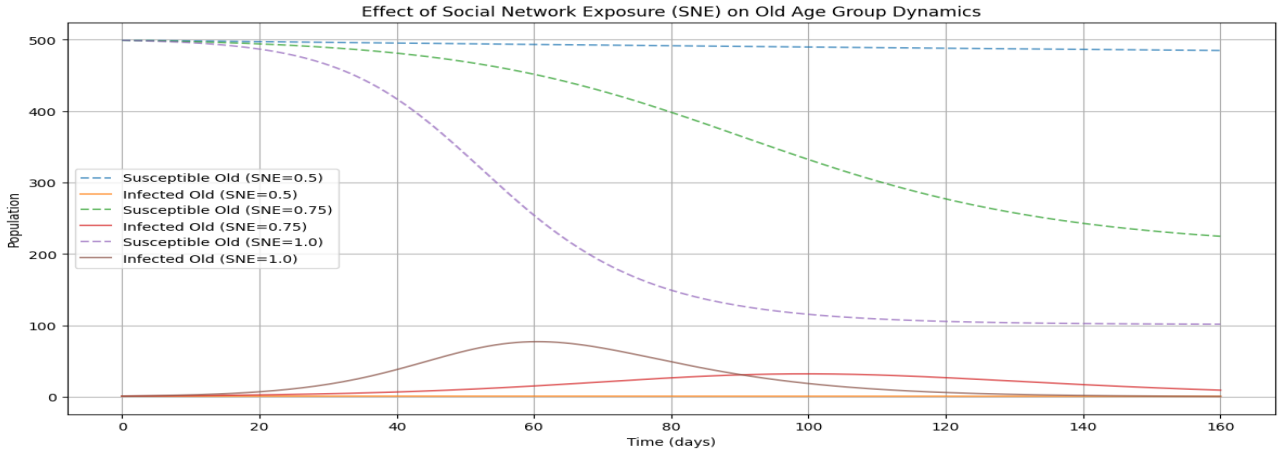


Figure 4: Effect of SNE on Infection Dynamics in Young and Old Age Group SIR Model

Higher Social Network Exposure (SNE) increases cross-class interactions, leading to faster disease transmission and earlier infection peaks across age groups, as shown in Figure 4. This acceleration of transmission is particularly evident when younger individuals, often in schools or community settings, have more frequent contact with older adults, heightening the risk of rapid disease spread. As a result, public health systems must prepare for surges in cases earlier than anticipated, requiring swift response measures. Additionally, changes in SNE can highlight vulnerabilities in certain age groups, especially older adults who may be exposed to younger, asymptomatic carriers. The distribution of risk shifts accordingly, necessitating targeted monitoring and intervention.

Analysis 3: SLIAR Model with Social Network Exposure (SNE) for Infectious Disease Dynamics

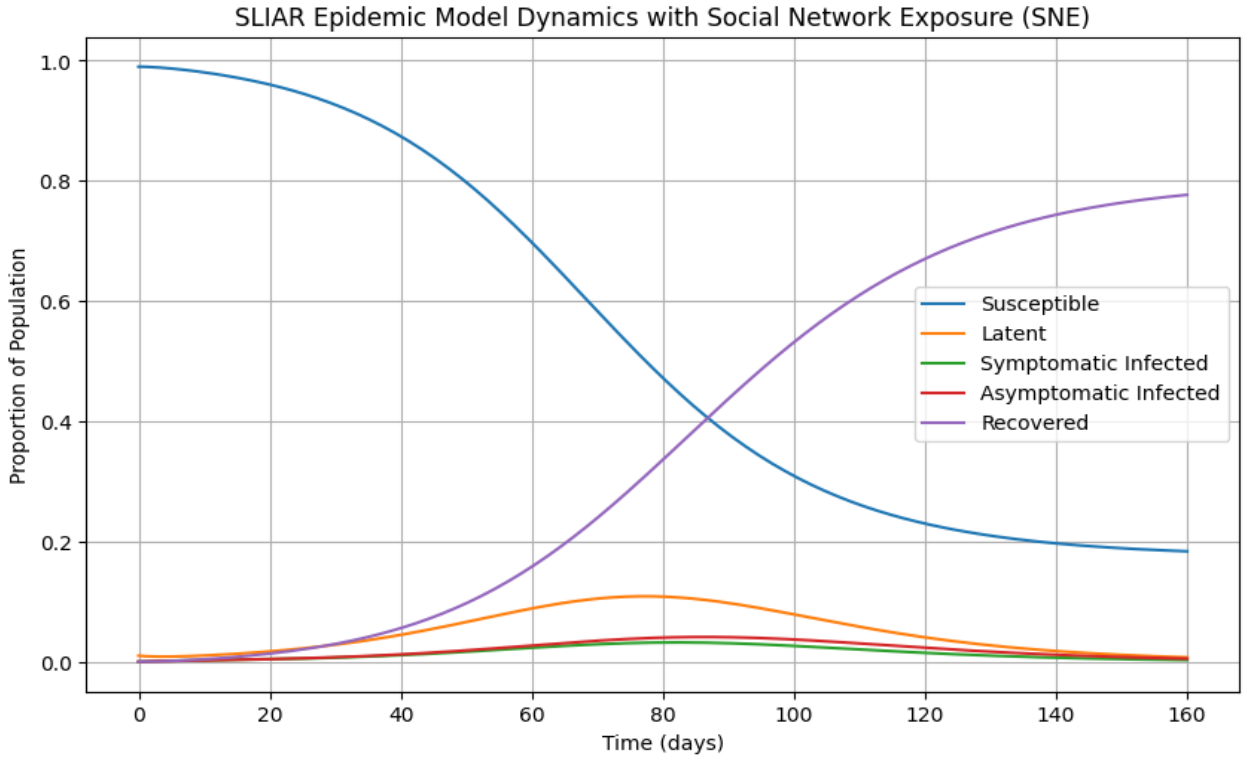


Figure 5: SLIAR Epidemic Model Dynamics with SNE = 1

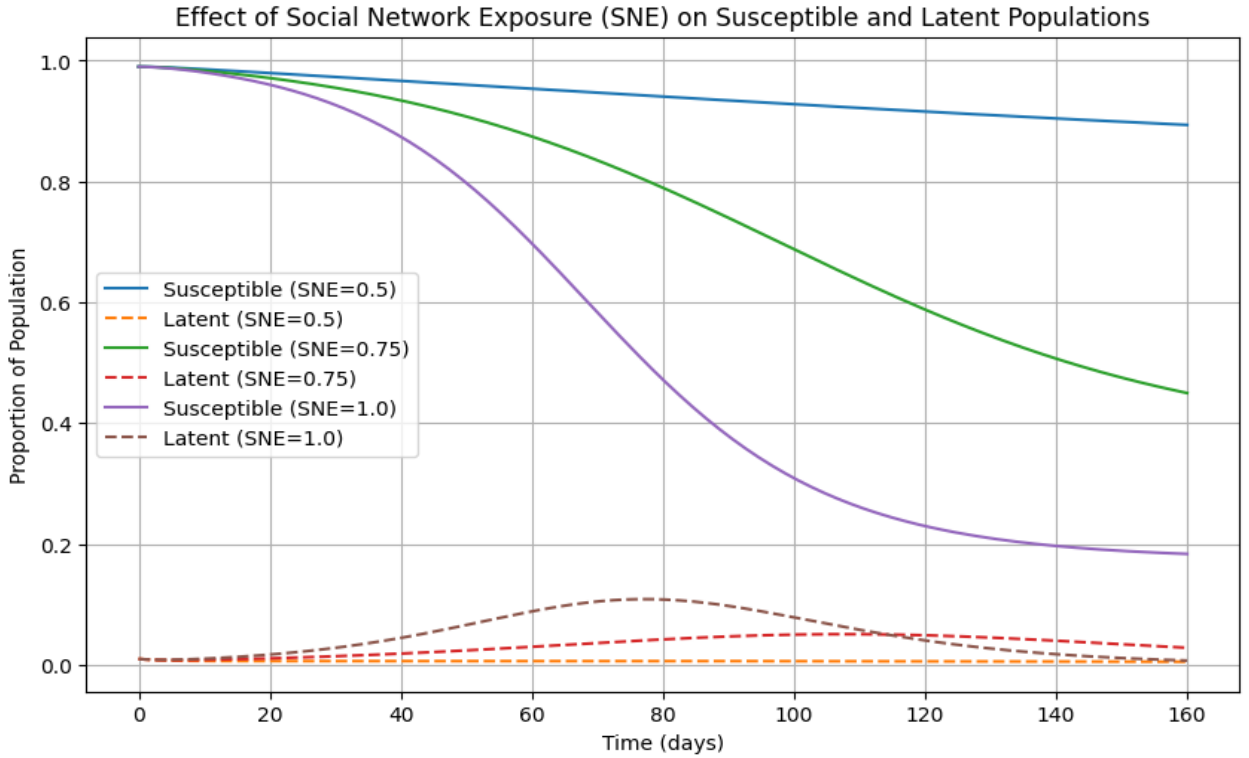


Figure 6: Effect of Social Network Exposure (SNE) on SLIAR Epidemic Model Dynamics

Higher Social Network Exposure (SNE) leads to more frequent interactions across socioeconomic groups, resulting in increased infection rates as individuals rapidly move from the susceptible (S) to latent (L) and infectious (I, A) compartments, as shown in Figure 6. This accelerates disease transmission, particularly among asymptomatic carriers, and drives an earlier peak in infections, putting additional pressure on healthcare systems. Shortened latent and infectious periods further speed up the spread, while asymptomatic individuals contribute significantly to undetected transmission. Different age groups face varying vulnerabilities, with younger populations becoming key transmitters. High SNE also leads to localized outbreaks across socioeconomic groups, complicating containment efforts, whereas low SNE confines outbreaks within specific classes.

Analysis 4: Clustered Network Epidemics via Incorporating Social Interactions

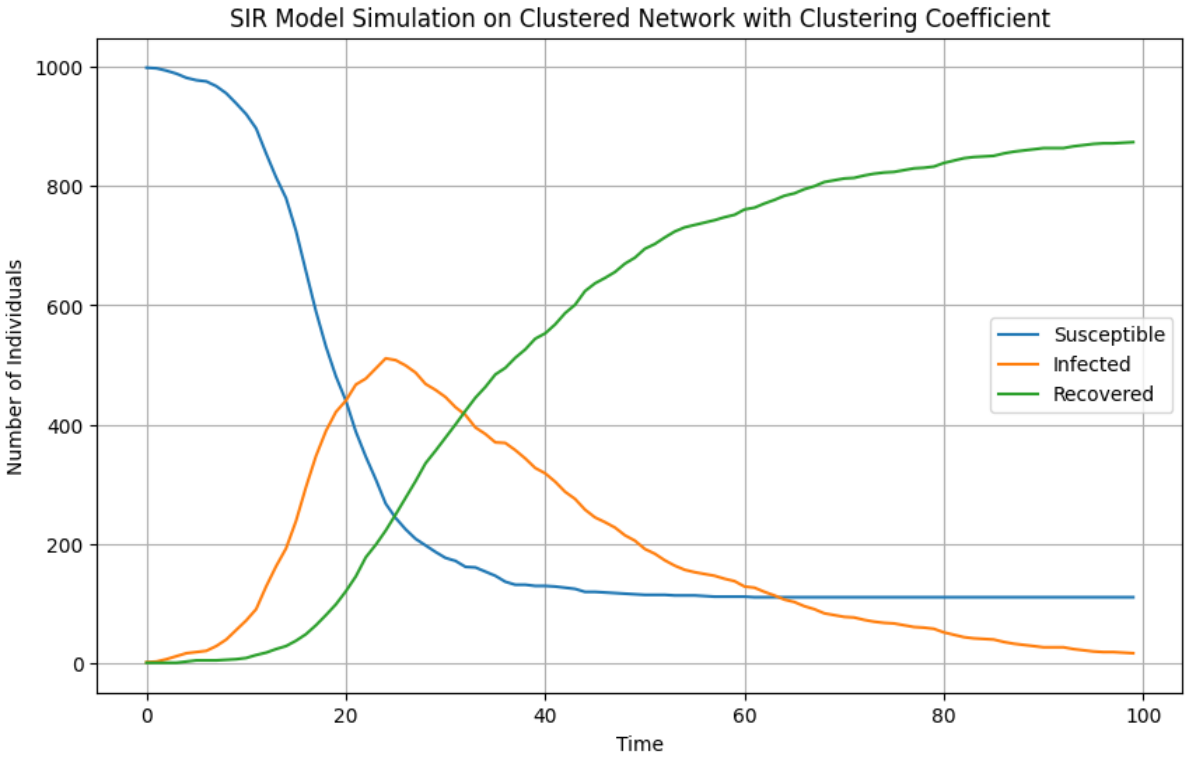


Figure 7: SIR Model Simulation on Clustered Network with Clustering Coefficient

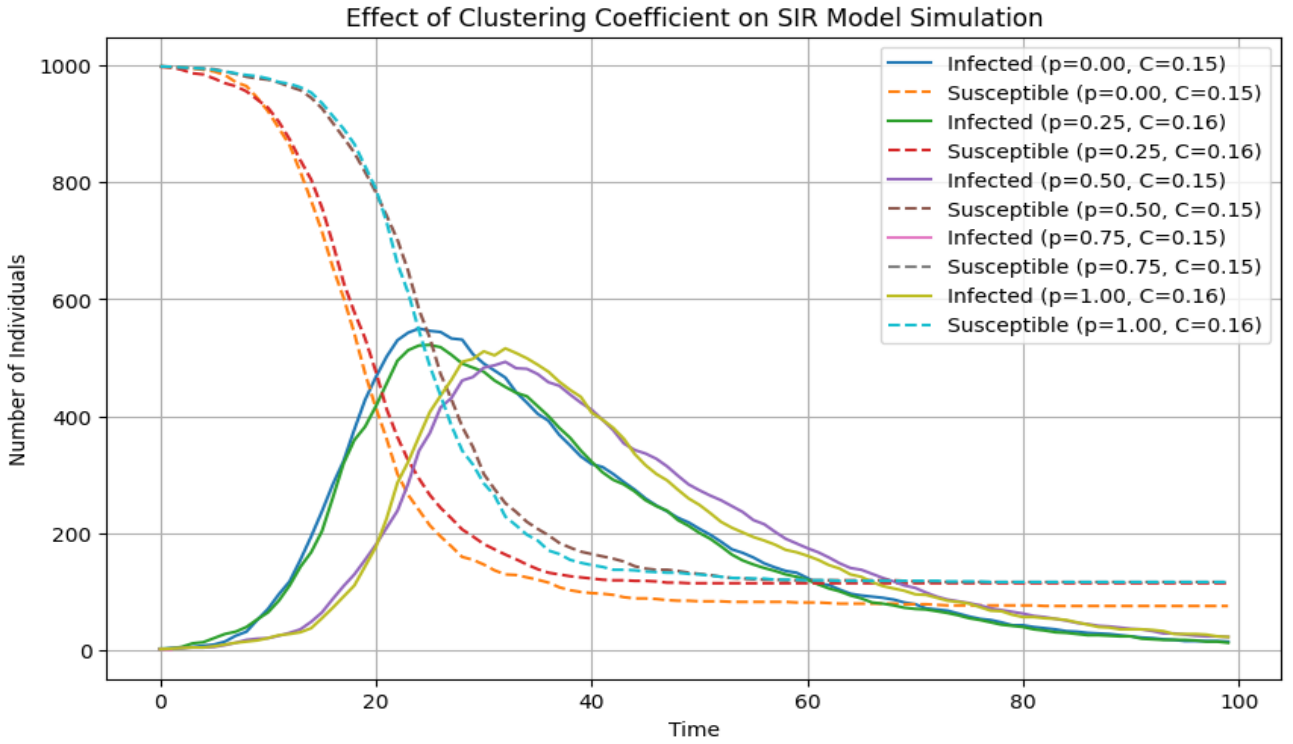


Figure 8: Effect of Clustering Coefficient on SIR Model Simulation of Clustered Network

The Clustering Effect on Infection Spread shows that at p = 0.00, infections quickly die out. As p increases (e.g., 0.25, 0.50, 0.75, 1.00), infections peak before declining, indicating that higher infection probabilities lead to faster, more widespread transmission (Figure 8). A larger clustering coefficient (C = 0.16) prolongs the infection duration due to more efficient spread within closely connected social groups, while lower clustering results in quicker but shorter epidemics. Social Interactions play a crucial role—higher clustering keeps infections active longer, while higher infection probabilities p increase the epidemic's peak magnitude.

Public Health Implications

Targeted Interventions: Public health efforts should prioritize vaccination for high-risk groups identified through Social Network Exposure (SNE) analysis, particularly younger populations acting as key transmitters. Tailored health education campaigns should raise awareness about inter-age group risks and promote preventive measures in high-SNE settings.

Flexible Response Strategies: Public health responses must be adaptive to changes in SNE, implementing temporary restrictions or enhanced measures during community events. Real-time monitoring of SNE can allow for more proactive strategies, enabling health officials to respond dynamically to evolving conditions.

Community Engagement: Encouraging cross-class interactions can strengthen community resilience to outbreaks. Public policies should support initiatives that promote inter-socioeconomic interactions, alongside providing essential support services like transportation for older adults to healthcare facilities.

Resource Allocation: Healthcare resources should be allocated based on age-specific risk and SNE patterns. High-SNE areas may require increased medical support due to faster transmission rates, while strategic healthcare infrastructure planning should account for potential spikes in infections among populations frequently interacting across age groups.

Conclusion

1. The research presented in this paper highlights the critical role of social network exposure (SNE), connectedness, clustering coefficient, and age-structured dynamics in understanding and mitigating the spread of infectious diseases.

2. By extending traditional models such as SIR and SLIAR to incorporate SNE, this study demonstrates how cross-class and inter-age interactions accelerate disease transmission, leading to different public health outcomes in various socioeconomic contexts.

3. The simulations of clustered network epidemics further emphasize the influence of social interactions and clustering on the spread of infections, providing insights into how closely connected groups may sustain transmission longer.

4. The findings underscore need for adaptive and targeted public health strategies, with interventions tailored to specific population dynamics & network structures. These insights have significant implications for vaccine distribution, resource allocation, & community engagement initiatives.

5. Future research should focus on refining models with real-world data to validate the findings & explore additional factors, like mobility patterns and health behaviors, A deeper understanding of how social structures impact epidemic spread can inform more efficient public health policies.

References

[1] L. F. Berkman and T. Glass, “Social integration, social networks, social support, and health,” in Social Epidemiology. Oxford University Press, 2000.
[2] M. Marmot, “Social determinants of health inequalities,” The Lancet, vol. 365, no. 9464, pp. 1099–1104, 2005. [Online]. Available: https://doi.org/10.1016/S0140-6736(05)71146-6
[3] A. V. Diez Roux, “Investigating neighborhood and area effects on health,” American Journal of Public Health, vol. 91, no. 11, pp. 1783–1789, 2001. [Online]. Available: https://doi.org/10.2105/AJPH.91.11.1783
[4] W. O. Kermack and A. G. McKendrick, “A contribution to the mathematical theory of epidemics,” Proceedings of the Royal Society A: Mathematical, Physical and Engineering Sciences, vol. 115, no. 772, pp. 700–721, 1927. [Online]. Available: https://doi.org/10.1098/rspa.1927.0118
[5] M. S. Granovetter, “The strength of weak ties,” American Journal of Sociology, vol. 78, no. 6, pp. 1360–1380, 1973. [Online]. Available: https://doi.org/10.1086/225469
[6] O. Diekmann, H. A. P. Heesterbeek, and T. Britton, Mathematical Tools for Understanding Infectious Disease Dynamics. Princeton University Press, 2013.
[7] M. Ajelli and A. Vespignani, “The sliar model: a framework for modeling the dynamics of infectious diseases with latent and asymptomatic infections,” Journal of Theoretical Biology, vol. 266, no. 1, pp. 121–130, 2010.
[8] J. C. Miller, “Percolation and epidemics in random clustered networks,” Physical Review E, vol. 80, no. 2, p. 020901, 2009. [Online]. Available: https://doi.org/10.1103/PhysRevE.80.020901
[9] R. Chetty, M. O. Jackson, T. Kuchler, J. Stroebel, N. Hendren, R. B. Fluegge, S. Gong, F. Gonzalez, A. Grondin, M. Jacob, D. Johnston, M. Koenen, E. Laguna-Muggenburg, F. Mudekereza, T. Rutter, N. Thor, W. Townsend, R. Zhang, M. Bailey, P. Barbera, M. Bhole, and ´ N. Wernerfelt, “Social capital i: measurement and associations with economic mobility,” Nature, vol. 608, 2022. [Online]. Available: https://doi.org/10.1038/s41586-022-04996-4
[10] R. Chetty, M. O. Jackson, T. Kuchler, J. Stroebel, N. Hendren, R. B. Fluegge, S. Gong, F. Gonzalez, A. Grondin, M. Jacob, D. Johnston, M. Koenen, E. Laguna-Muggenburg, F. Mudekereza, T. Rutter, N. Thor, W. Townsend, R. Zhang, M. Bailey, P. Barber, M. Bhole, and N. Wernerfelt, “Social capital ii: determinants of economic connectedness,” Nature, vol. 608, 2022. [Online]. Available: https://doi.org/10.1038/s41586-022-04997-3

THANK YOU

Divya Aggarwal
BS-MS Economics
21322011