Modelling Impact of Positive and Negative Information on SEIRD-like Epidemics in Multiplex Networks

Abstract

This study investigates the influence of positive and negative information dissemination on SEIRD-like epidemics within multiplex networks, employing the UPN-SEIRD model. By considering distinct transmission and recovery rates for positive and negative information, we gain insight into the complex interplay between information and disease spread. Experimental setups, utilizing Barabási–Albert (BA) and Monte Carlo (MMC) simulations, validate our model's predictions. Our findings highlight the significant role of information in shaping epidemic outcomes, providing valuable insights for public health strategies and epidemic control.

Keywords - SEIRD model, multiplex networks, positive information, negative information, epidemic dynamics, information dissemination

1. Introduction

The study of epidemic spreading is of utmost importance, especially given the intricate nature of real-world interactions. Traditional models, such as the Susceptible-Exposed-Infected-Recovered-Deceased (SEIRD) framework, offer valuable insights into infectious disease dynamics. However, they often neglect the impact of information dissemination. Information, whether promoting preventive measures (positive) or spreading misinformation (negative), significantly influences public behavior and epidemic outcomes. This study aims to address this gap by utilizing the UPN-SEIRD model to investigate the coevolution of information and disease spread in multiplex networks.

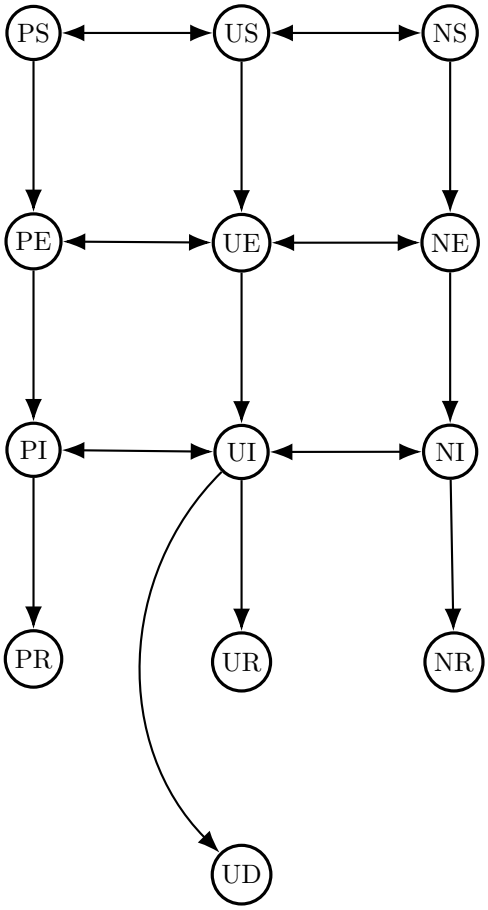
2. Model Description

2.1 UPN-SEIRD Model Overview

The UPN-SEIRD model is a comprehensive framework that captures the interplay between disease spread and information dissemination. It consists of two interconnected layers: the physical contact layer and the virtual communication layer. The physical contact layer represents face-to-face interactions where the disease spreads, while the virtual communication layer simulates the spread of information through online or mass media channels.

1 UPN-SEIRD

1.1 State Transition



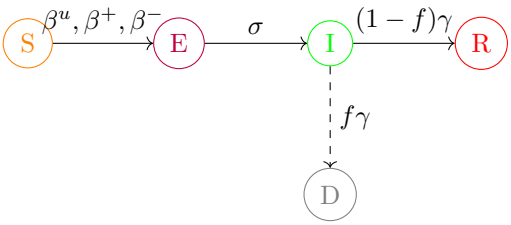
2.2 Compartments and Transitions

The model includes eight compartments: Unaware (U), Positive information spreader (P), Negative information spreader (N), Susceptible (S), Exposed (E), Infected (I), Recovered (R), and Deceased (D). The transitions between compartments depend on the information state of individuals, with different rates for unaware, positive, and negative spreaders.

2.3 Epidemic Layer (SEIRD Model)

The SEIRD model describes disease propagation in the physical contact layer: - Susceptible to Exposed (S $\rightarrow$ E): Individuals become exposed through contact with infected individuals at rates u, +, or - depending on their information state. - Exposed to Infected (E $\rightarrow$ I): Exposed individuals transition to the infected state at a rate of . - Infected to Recovered (I $\rightarrow$ R): Infected individuals recover at a rate of (1-f) . - Infected to Deceased (I $\rightarrow$ D): Infected individuals may die at a rate of f , where f is the mortality rate.

1.1.1 Epidemic Layer Transition



2.4 Information Layer (UPN Model)

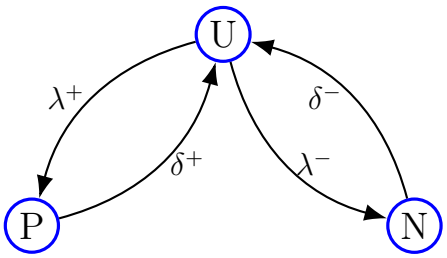
The information layer captures the spread of positive and negative information:

- Unaware to Positive/Negative Spreader (U $\rightarrow$ P/N): Unaware individuals become positive or negative spreaders influenced by aware neighbors at rates + or -. And when individuals already infected, these rates are κ^+or κ^-.

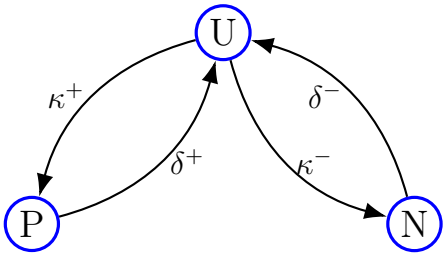
- Positive/Negative Spreader to Unaware (P/N $\rightarrow$ U): Positive or negative spreaders may forget and transition back to the unaware state at rates + or -.

1.2 Model Schematic

1.2.1 aware neighbor



1.2.2 already infected



3. Coupled Dynamics

The dynamics between the epidemic layer and the information layer are coupled, with the state of an individual in one layer influencing their behavior in the other. This is reflected in the transition rates between compartments, creating a complex system where disease spread and information dissemination influence each other.

Heterogeneous Mean-Field Theory

We formulate a heterogeneous mean-field theory of UPN-SEIRD dynamics. We use $x_j y_k \equiv x_j y_k(t)$ ($x \in \{u, p, n\}, y \in \{s, e, i, r, d\}$) to denote the proportion of nodes in state $X_j Y_k$ ($X \in \{U, A\}, Y \in \{S, E, I, R, D\}$) with degrees j and k in the IL and EL at time t, respectively. For example, $u_{js}^k \equiv u_{js}^k(t)$ denotes the proportion of unaware and susceptible nodes with degrees j and k in the IL and EL at time t, respectively. Henceforth, we will not explicitly include the time dependence in the notation $x_j y_k$ for the sake of notational brevity.

The proportions of susceptible, exposed, infected, recovered, and deceased nodes are

$$s_k = \sum_{j=1}^{J} (u_j s_k + p_j s_k + n_j s_k), \tag{1}$$

$$e_k = \sum_{j=1}^{J} (u_j e_k + p_j e_k + n_j e_k), \tag{2}$$

$$i_k = \sum_{j=1}^{J} (u_j i_k + p_j i_k + n_j i_k), \tag{3}$$

$$r_k = \sum_{j=1}^{J} (u_j r_k + p_j r_k + n_j r_k), \tag{4}$$

$$d_k = \sum_{j=1}^{J} u_j d_k, \tag{5}$$

where J is the maximum (or cutoff) degree in the IL. Since dead individuals in the EL cannot contribute to propagating awareness in the IL, the states (P, D) and (N, D) are discarded from Eq. (5). Similarly, we find that the proportions of unaware and positive and negative aware nodes are

$$u_j = \sum_{k=1}^{K} (u_j s_k + u_j e_k + u_j i_k + u_j r_k + d_k), \tag{6}$$

$$p_j = \sum_{k=1}^{K} (p_j s_k + p_j e_k + p_j i_k + p_j r_k), \tag{7}$$

$$n_j = \sum_{k=1}^{K} (n_j s_k + n_j e_k + n_j i_k + n_j r_k), \tag{8}$$

where K is the maximum (or cutoff) degree in the EL. These quantities

satisfy the normalization conditions

$$\sum_{k=1}^{K}(s_k + e_k + i_k + r_k + d_k) = 1, \tag{9}$$

$$\sum_{j=1}^{J}(u_j + p_j + n_j) = 1. \tag{10}$$

2 Equations

1.US

$$\frac{d(u_j s_k)}{dt} = -\lambda^+ j u_j s_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} - \lambda^- j u_j s_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} - \beta^u k u_j s_k \frac{\sum_{k'} k' i_{k'}}{\langle k \rangle} + \delta^+ p_j s_k + \delta^- n_j s_k \tag{11}$$

2.PS

$$\frac{d(p_j s_k)}{dt} = \lambda^+ j u_j s_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} - \beta^+ k p_j s_k \frac{\sum_{k'} k' i_{k'}}{\langle k \rangle} - \delta^+ p_j s_k \tag{12}$$

3.NS

$$\frac{d(n_j s_k)}{dt} = \lambda^- j u_j s_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} - \beta^- k n_j s_k \frac{\sum_{k'} k' i_{k'}}{\langle k \rangle} - \delta^- n_j s_k \tag{13}$$

4.UE

$$\frac{d(u_j e_k)}{dt} = -\lambda^+ j u_j e_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} - \lambda^- j u_j e_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} + \beta^u k u_j s_k \frac{\sum_{k'} k' i_{k'}}{\langle k \rangle} - \sigma u_j e_k + \delta^+ p_j s_k + \delta^- n_j s_k \tag{14}$$

5.PE

$$\frac{d(p_j e_k)}{dt} = \lambda^+ j u_j e_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} - \beta^+ k p_j s_k \frac{\sum_{k'} k' i_{k'}}{\langle k \rangle} - \sigma p_j e_k - \delta^+ p_j e_k \tag{15}$$

6.NE

$$\frac{d(n_j e_k)}{dt} = \lambda^- j u_j e_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} - \beta^- k n_j s_k \frac{\sum_{k'} k' i_{k'}}{\langle k \rangle} - \sigma n_j e_k - \delta^- n_j e_k \tag{16}$$

7.UI

$$\begin{aligned}\frac{d(u_j i_k)}{dt} =& - \lambda^+ j u_j i_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} - \lambda^- j u_j i_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} + \sigma u_j e_k - \gamma u_j i_k \\ & - \kappa^+ u_j i_k - \kappa^+ u_j i_k + \delta^+ p_j i_k + \delta^- n_j i_k\end{aligned} \tag{17}$$

8.PI

$$\frac{d(p_j s_k)}{dt} = \lambda^+ j u_j s_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} + \sigma p_j e_k - \gamma p_j i_k - \kappa^+ u_j i_k - \delta^+ p_j i_k \tag{18}$$

9.NI

$$\frac{d(n_j s_k)}{dt} = \lambda^- j u_j s_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} + \sigma u_j e_k - \gamma u_j i_k - \kappa^+ u_j i_k - \delta^- n_j i_k \tag{19}$$

10.UR

$$\begin{aligned}\frac{d(u_j r_k)}{dt} =& - \lambda^+ j u_j r_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} - \lambda^- j u_j r_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} + (1-f) \gamma u_j r_k \\ & + \delta^+ p_j r_k + \delta^- n_j r_k\end{aligned} \tag{20}$$

11.PR

$$\frac{d(p_j r_k)}{dt} = \lambda^+ j u_j r_k \frac{\sum_{j'} j' p_{j'}}{\langle \tilde{k} \rangle} + (1-f) \gamma p_j r_k - \delta^+ p_j r_k \tag{21}$$

12.NR

$$\frac{d(n_j r_k)}{dt} = \lambda^- j u_j r_k \frac{\sum_{j'} j' n_{j'}}{\langle \tilde{k} \rangle} + (1-f) \gamma n_j r_k + \delta^- n_j r_k \tag{22}$$

13.UD

$$\frac{d(u_j d_k)}{dt} = f\gamma(u_j + p_j + n_j)i_k \tag{23}$$

3 4. Experimental Setup

4.1 Network Models We employ Barabási–Albert (BA) networks and Monte Carlo (MMC) simulations to validate our model. BA networks mimic the scale-free nature of real-world social networks, while MMC simulations allow us to explore dynamics across different network structures.

4.2 Simulation Parameters

The simulation parameters are carefully chosen to represent realistic scenarios, including infection rates, latent, recovery, and mortality rates for the epidemic layer, and awareness and forgetting rates for the information layer.

4.3 Initial Conditions and Simulation Procedure We initiate our simulations with a small number of infectious and aware individuals in a network of 10,000 nodes. The Gillespie algorithm is used to simulate the spread of the disease and information over a period of 150 days.

4.4 Analysis Framework Our analysis focuses on the epidemic threshold, infection dynamics, and information spread patterns. We compare the baseline scenario (uninterrupted information flow) with random and targeted edge removal scenarios, simulating disruptions in information dissemination.

5. Results 5.1 Epidemic Threshold

The presence of positive and negative information influences the epidemic threshold, with varying rates of information dissemination leading to distinct thresholds. MMC networks exhibit a higher threshold compared to BA networks, indicating the impact of network structure.

5.2 Infection Dynamics

The infection dynamics differ between the baseline and edge removal scenarios. The rate of infection spread is influenced by the disruption of information flow, with targeted disruptions having a more pronounced effect. BA networks reach a higher peak prevalence compared to MMC networks.

5.3 Information Spread

The spread of positive and negative information varies over time, with awareness and forgetting rates playing a crucial role. The correlation between information dissemination and infection dynamics reveals interesting patterns, highlighting the complex interplay between the two layers.

6. Discussion

Our findings underscore the significant impact of information on epidemic control. The spread of positive information promoting preventive measures can effectively reduce infection rates, while negative misinformation can hinder epidemic mitigation efforts. The comparison with similar studies reinforces the importance of considering information dissemination in epidemic models.

7. Conclusion

This study highlights the critical role of positive and negative information in shaping SEIRD-like epidemics in multiplex networks. By utilizing the UPN-SEIRD model, we gain valuable insights into the coevolution of information and disease spread. Our results have important implications for public health strategies, emphasizing the need to consider information dissemination in epidemic preparedness and response. Future research may extend this framework to incorporate additional factors and apply it to real-world epidemic scenarios.

4 References

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