Emotional Contagion of Mental Health Issues in Networks Paper

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Abstract

Emotional contagion is the phenomenon by which individuals' emotional states influence and spread to others, and plays a critical role in shaping mental health dynamics within social networks. This paper explores the interplay between emotional contagion and mental health awareness through the lens of evolutionary game theory. By extending the classical SIR framework to a SIRA (Susceptible–Infected–Recovered–Active) model, we simulate how emotional states and awareness behaviors propagate in both fully connected and network-constrained populations. Our model incorporates differential equations and a discrete network-based simulation modeling roles of peer support, natural recovery, and susceptibility to distress. Through over 300 simulations, we analyze how key parameters, including support effectiveness, self-recovery, and network structure influence emotional outcomes. Findings suggest that strong recovery and support mechanisms significantly buffer against emotional contagion, even when exposure is high, whereas weak support structures lead to persistent emotional distress and systemic strain. This research offers a useful framework for understanding the spread of emotional states in social systems and provides actionable insights for designing interventions that promote emotional resilience, mental health awareness, and sustainable support networks.

I Introduction

Mental health challenges, particularly anxiety, have long been recognized as major contributors to global morbidity, mortality, and disability. While traditionally viewed through a clinical or individual lens, an expanding body of research reveals that these conditions are not only internal experiences but also socially embedded phenomena. Emotions like distress or emotional well-being do not exist in a vacuum; rather, they ripple across relationships, communities, and increasingly, digital platforms (Coviello et al., 2014). Emotional

contagion, the process by which individuals' emotions influence and spread to others, offers a powerful framework for understanding the social dynamics of mental health.

Emerging research shows that emotional states can propagate through social networks in measurable, often predictable ways. Individuals with close connections to someone experiencing anxiety are significantly more likely to experience anxious symptoms themselves, with effects strongest within three degrees of separation (Rosenquist, Fowler, Christakis, 2010). Similarly, loneliness, stress, and emotional exhaustion are influenced by peer dynamics, especially in environments like classrooms, college campuses, or online communities. Notably, women are found to be more influential in transmitting emotional states, possibly due to differences in emotional expression (Cacioppo, Fowler, Christakis, 2009). In tightly connected settings, both positive and negative emotional experiences can cascade outward, influencing the mental health outcomes of entire subgroups. Alongside emotional states, mental health awareness itself may also spread through networks, affecting not only individuals' perceptions of psychological well-being, but their willingness to seek support, adopt coping behaviors, or engage with stigma-reduction efforts. However, this awareness is not uniformly protective. Co-evolutionary models suggest that when awareness is poorly framed or untimely, it may amplify vulnerability to distress rather than mitigate it (Nesse and Stein, 2023). Conversely, strategic awareness efforts can delay or disrupt the outbreak of harmful emotional contagion. The framing, timing, and channel of information, all influenced by network structure, determine whether awareness acts as a protective barrier or a catalyst for further emotional distress (Scata, Stefano, Corte, and Lio, 2018). To examine these dynamics, we turn to evolutionary game theory as a framework for understanding how individuals adapt to the emotional landscape of their social environment. Originally designed to model rational decision-making, game theory now offers tools to explore how emotional signals, coping strategies, and awareness behaviors evolve within social systems (Szolnoki, Xie, Ye, and Perc, 2013). Evolutionary game theory allows us to investigate how mental health behaviors may spread, stabilize, or die out within populations based on adaptive feedback, peer influence, and perceived payoffs. This paper investigates the interplay between emotional contagion and mental health awareness in structured networks. Drawing on precursory research, agent-based simulations, and behavioral models, we explore how distress and awareness circulate within social systems: how emotional vulnerability is transmitted, amplified, or buffered by peers, and how the shape and structure of networks modulate this process. While we focus on induction, where emotions directly influence others, we acknowledge that this is only one of several mechanisms through which emotional states may appear to spread. Emotional contagion can also arise from homophily (the tendency for individuals with similar emotional states to connect) and confounding (shared exposures or environments that independently affect emotional outcomes) (Rosenquist, Fowler, Christakis, 2010). Although our model assumes induction as the primary driver of emotional transmission, we recognize that disentangling these mechanisms remains a major methodological challenge in the field. Prior studies

(e.g., Rosenquist et al., 2010; Cacioppo et al., 2009) have provided empirical support for induction, demonstrating that emotional influence persists even when controlling for homophily and confounding. Nevertheless, future extensions of our work could explicitly test these alternative explanations. Although prior research supports the existence of true emotional influence even when controlling for these factors, disentangling them remains a significant methodological challenge. Future extensions of this work could explicitly incorporate and test these alternative pathways. We address this limitation further in the conclusion. Ultimately, this paper aims to illuminate how emotional risk and resilience propagate through social networks, with implications for the design of awareness campaigns, targeted interventions, and systems-level public health strategies.

II Method

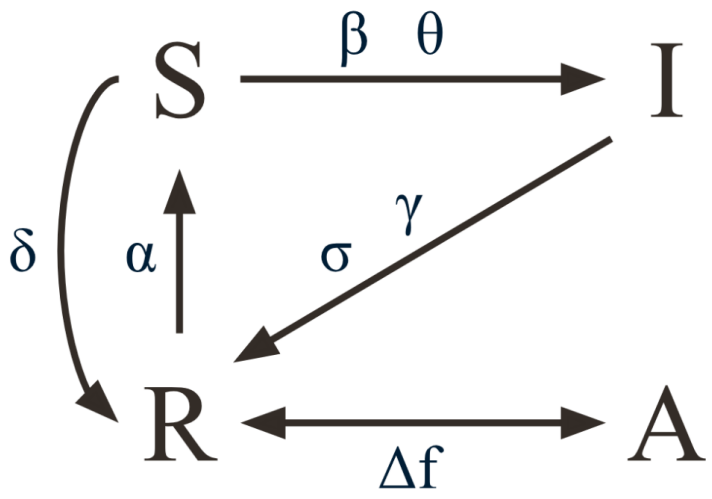

Fig. 1: Diagram of SIRA transitions and the constants affecting transition rates

Our project builds on the classical SIR (Susceptible–Infected–Recovered) epidemiological framework by introducing a fourth category, Active (A), to better capture the dynamics of emotional contagion and mental health awareness within social systems. The Active state represents individuals in the recovered population who are not only uninfected but also actively helping the infected recover, such as through emotional support, advocacy, or awareness-building. This addition transforms the SIR framework into a SIRA model, allowing us to simulate altruistic behaviors within a networked environment and interpret them through the lens of a public goods game (PGG), where emotional support is framed as a costly yet socially beneficial contribution.

II.I Differential Equation Model

$$\frac{dS}{dt} = -\frac{\beta SI}{N} - \frac{\sigma AS}{N} + \alpha R - \theta S$$
$$\frac{dI}{dt} = \frac{\beta IS}{N} - \gamma I - \frac{\delta IA}{N} + \theta S$$
$$\frac{dR}{dt} = \frac{\sigma SA}{N} + \gamma I + \frac{\delta IA}{N} - AR(f_A - f_{NA}) - \alpha R$$
$$\frac{dA}{dt} = AR(f_A - f_{NA})$$

We first formalized the SIRA model using a set of differential equations to describe the rate of change between each state in a fully connected population. We assume each person interacts with every other person in the population once every unit of time. Then, we assign probabilities of individuals changing states, denoted by the Greek letters α, β, θ, δ, γ, and σ. β (beta): controls how a person who can catch an illness interacts with someone who is infected.

- α (alpha): Chance that a recovered person becomes vulnerable again.
- δ (delta): Governs the interaction between a susceptible individual and an active supporter, capturing how likely support leads to protection.
- σ (sigma): Governs interaction between an active and an infected individual, reflecting the impact of support on the infected's likelihood of recovery.
- θ (theta): Stochastic probability that a susceptible individual becomes infected, accounting for random emotional shocks or external influences.
- γ (gamma): Stochastic probability that an infected individual recovers over time.
- $\delta f(I, A)$ (difference in payoff): Governs the transition rate between recovered and active individuals, based on the perceived payoff of each strategy.
  - Active individuals pay a cost $c(I)$ for being active, proportional to the number of infected and susceptible people they assist.
  - They receive a reward $r(I, A)$, proportional to the number of infected and inversely proportional to the number of active people.

These parameters can be interpreted socially. For example, a high δ could represent strong peer-based coping mechanisms, while a high β could indicate environments with poor emotional boundaries or burnout contagion. One direction for future work is to examine how these values can be manipulated through intervention, such as targeted awareness campaigns or peer support structures.

Figure 2: Example of SIRA dynamics over time in the differential equations model

II.II Potential Issues with the Differential Equations Model

This approach assumes an infinite population where every individual in the population is equally connected to every other. This assumption, while analytically convenient, fails to capture the complexity of real-world social systems. Under this model, the system consistently converged to a static distribution of S, I, R, and A over time. While mathematically stable, this equilibrium proved unrealistic in the context of emotional contagion, which is inherently dynamic and sensitive to social context.

The convergence implied that, regardless of initial conditions, the proportions of individuals in each state eventually stabilized, failing to reflect the emotional volatility and feedback loops observed in social groups such as college campuses, online communities, or friend networks. Furthermore, the assumption of homogenous connectivity negated the influence of peer variability, social influence, and information asymmetry, all of which are critical in shaping how mental health issues spread and are responded to.

II.III Transition to a Network-Based Model

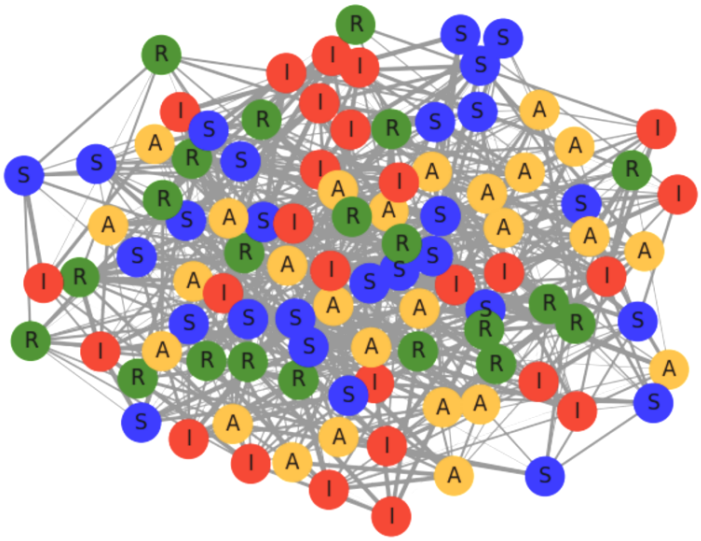

Fig. 2: A prototype of our network model with 100 nodes(population size) and 500 edges (connections)

To more accurately capture the dynamics of emotional contagion, we transitioned from the continuous differential equation model to a discrete network-based simulation. We retained the same payoff structures from the public goods framework but embedded the agents in a non-uniform, limited connectivity network, where each individual (node) interacts only with

a finite set of others. Every connection (edge) was weighted differently, acknowledging that emotional influence, support capacity, and communication frequency differ from one relationship to another. Additionally, each node had a different susceptibility, reflecting how some individuals may be more or less prone to stress. Specifically, our simulation model had 200 nodes, with a total of 1000 edges, thus averaging around 10 connections per node. Each edge had a weight ranging from 0.0 to 1.0, and each node had a susceptibility between 0.5 and 1.0, with a uniform probability density. To see the effect of our various parameters, a parameter sweep was run for 300+ combinations of parameters. σ Values = [0.05, 0.075, 0.1, 0.15] δ Values = [0.25] α Values = [0.15, 0.1, 0.2, 0.3, 0.35] γ Values = [0.05, 0.1, 0.15, 0.2] β Values = [0.7, 0.8, 0.9, 0.95] θ Values = [0.1]

This model allows each individual to engage with only a subset of the population, making the simulation more realistic in terms of how people interact with their immediate peers rather than an abstract collective. The incorporation of network topology introduced a level of heterogeneity and local feedback that was missing in the differential approach. In particular, the simulation accounted for asymmetric relationships, clustering effects, and bottlenecks, factors that profoundly shape emotional contagion and support behavior in real-world contexts.

III Data

For this section , we present 4 of the 320 simulations that we conduct with different variations of the aforementioned parameters. These four in particular were selected because they not only vary largely in terms of the population distributions, but the set of parameters also present opportunities for rich analysis and reflect real world situations.

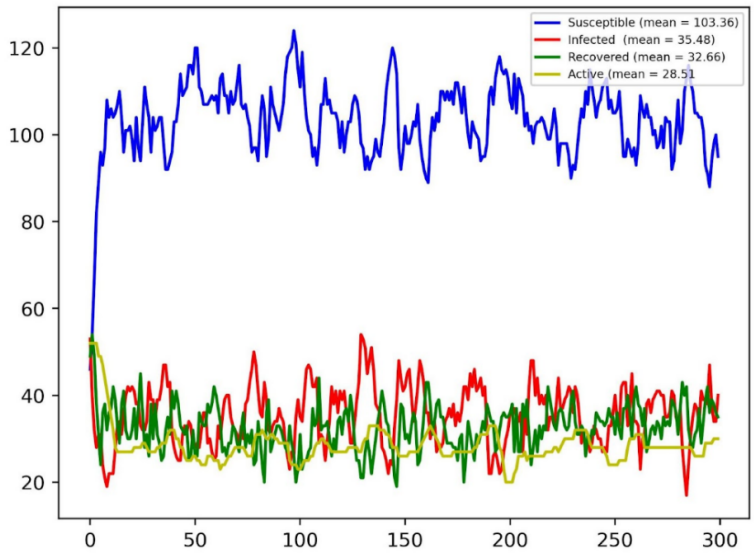

Fig. 3: Network model with parameters $\alpha = 0.35$, $\sigma = 0.15$, $\beta = 0.95$, $\theta = 0.1$, $\delta = 0.25$, $\gamma = 0.2$

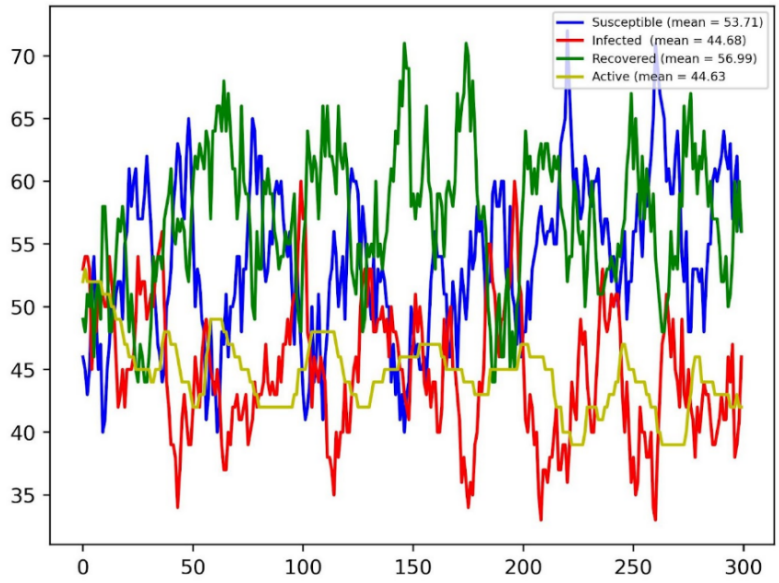

Fig. 4: Network model with parameters $\alpha = 0.1$, $\sigma = 0.05$, $\beta = 0.95$, $\theta = 0.1$, $\delta = 0.25$, $\gamma = 0.05$

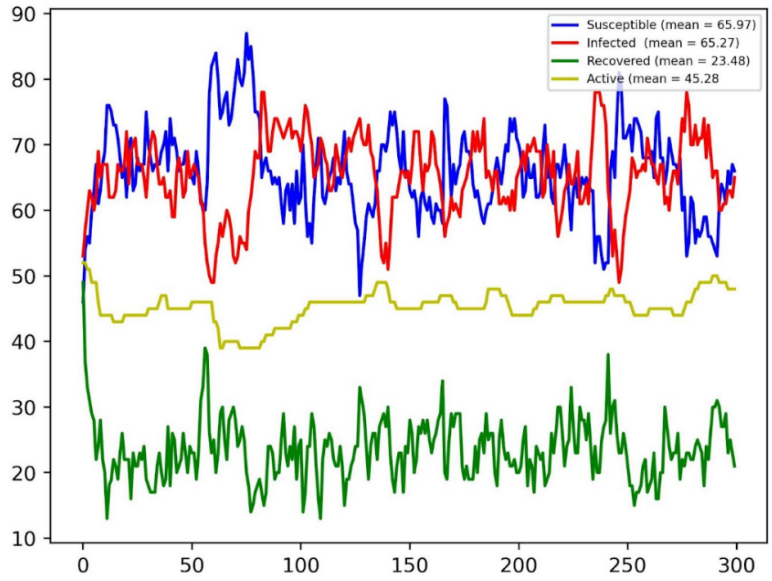

Fig. 5: Network model with parameters $\alpha = 0.35$, $\sigma = 0.05$, $\beta = 0.95$, $\theta = 0.1$, $\delta = 0.25$, $\gamma = 0.05$

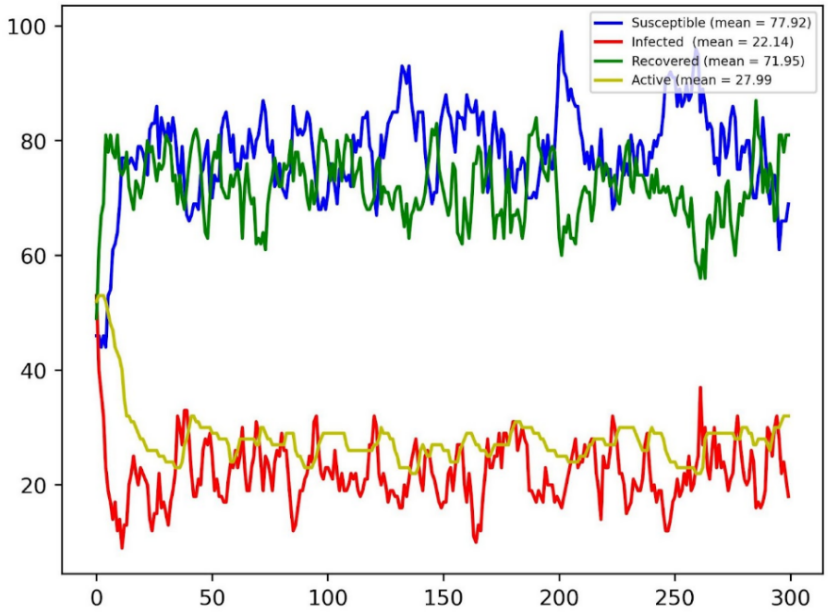

Fig. 6: Network model with parameters $\alpha = 0.1$, $\sigma = 0.15$, $\beta = 0.7$, $\theta = 0.1$, $\delta = 0.25$, $\gamma = 0.2$

IV Analysis

Figure 3 presents a network simulation with parameters $\alpha = 0.35$, $\sigma = 0.15$, $\beta = 0.95$, $\theta = 0.1$, $\delta = 0.25$, and $\gamma = 0.2$. In this setting, both recovery and support mechanisms are relatively strong, as indicated by the moderately high values of γ and σ. However, the high α value reflects a system in which individuals frequently revert to a vulnerable state even after recovering. The results align with this dynamic: the susceptible population dominates, with a mean of 103.36, while infected (mean = 35.48), recovered (mean = 32.66), and active (mean = 28.51) groups remain smaller and fluctuate within a contained range. The stochastic component θ is held constant at 0.1 across all simulations, suggesting that anxiety triggered by random or internal factors is present but not a primary driver

This situation reflects how higher educational institutions can adopt good institutional support systems, particularly during stressful times such as finals week, where emotional contagion, particularly anxiety, spreads rapidly due to dense peer networks and shared stressors (reflected in high β). However, the presence of institutional or structural buffers can raise γ, the probability of natural recovery. For instance, a few days of breaks between exams, supportive climates, or reassurances from teachers/professors may reduce sustained stress, making spontaneous emotional recovery more likely. Together with peer-led support systems (captured by σ), these mechanisms prevent emotional distress from spiraling. Although students frequently become susceptible again due to ongoing and fast academic cycles (high α), the combined effect of natural recovery and active support ensures that anxiety does not dominate the network. Even under intense emotional exposure, strong recovery dynamics and timely interventions allow most individuals to remain stable and susceptible rather than overwhelmed.

In Figure 4, we reduce several key recovery-related parameters: the probability of becoming susceptible after recovery (α) is lowered to 0.1, indicating that once individuals recover, they are less likely to relapse. However, the probability of natural recovery (γ) is also decreased to 0.05, and the effectiveness of support (σ) is similarly reduced to 0.05, while the transmission rate remains high at $\beta = 0.95$. This combination of weak healing and high exposure significantly alters the system's dynamics. The infected population rises to a mean of 44.68, while both the recovered and active groups increase to approximately 56.99 and 44.63, respectively. Meanwhile, the susceptible population drops sharply to 53.71. Despite the weakened healing mechanisms, the lowered α means that those who do recover are more likely to remain in the recovered state, contributing to the sustained increase in that group. However, because γ and σ are so low, recovery is rare, either through natural processes or peer support, leading to a backlog of individuals in the infected state. This forces more people into the active group, attempting to intervene. But with limited support effectiveness, these efforts are insufficient to contain or mute spread. The result is a system marked by persistent emotional burden, where few remain unaffected and many are caught in cycles of illness, intervention, and prolonged recovery

This dynamic somewhat resembles health-

care environments during prolonged crises, such as a pandemic surge. In such scenarios, patients (infected individuals) accumulate as emotional and physical distress spreads rapidly through the system (high β), while opportunities for natural recovery (low γ) or effective intervention (low σ) are scarce. As the crisis amplifies, more healthcare workers (active individuals) are mobilized to provide support, leading to a noticeable rise in the active population. However, despite this increase, their interventions remain largely ineffective due to limited systemic capacity and burnout, which represents the low σ value. Meanwhile, those who do recover are likely to remain in the recovered state (low α).This may illustrate a psychological resilience shaped by gratitude or relief: individuals who have recovered might consciously distance themselves from triggers or reframe stressors, making them less prone to falling back into the same level of emotional distress. The result of this dynamic is a network under sustained emotional pressure, where the rise in active supporters reflects desperation rather than success. The workforce expands in response to the crisis, yet is unable to fully contain the persistent spread of distress

In Figure 5, we hold susceptibility (α) steady at 0.35 but reduce support effectiveness (σ) and external recovery (γ) to 0.05. The results reveal a sharp increase in both infected (mean = 65.27) and susceptible (mean = 65.97) populations, while the recovered group declines significantly to a mean of 23.48. The active population rises modestly to 45.28. The high susceptibility rate, combined with weakened recovery and support mechanisms and a high transmission rate ($\beta = 0.95$), drives rapid infection spread and allows the infected population to dominate the network. This suggests individuals are frequently exposed to emotional distress and repeatedly fall into infected states, with insufficient peer or external support to facilitate effective recovery. The system thus becomes saturated with infection, placing increased strain on the active group, whose efforts to intervene are overwhelmed by the lack of robust structural healing.

This dynamic may resemble situations such as workplace environments during prolonged organizational crises or restructurings, where employees (susceptible individuals) face ongoing stress and uncertainty (high α), but support systems like counseling, management assistance, or peer support groups (σ and γ) are minimal or ineffective. Despite some individuals actively supporting and consoling each other (active group), the lack of adequate recovery pathways results in sustained emotional distress and burnout (high infected population), which spreads across the organization. Without stronger structural interventions, the emotional burden intensifies, undermining both individual well-being and collective productivity, sustaining the pattern of emotional stress. Such dynamics have been widely observed in recent years within healthcare systems during the COVID-19 pandemic, as well as in sectors like education and social services, where prolonged pressures and limited support have led to widespread burnout and emotional exhaustion. This scenario somewhat resembles the one presented in Figure 3; however, the absence of institutional support mechanisms here makes recovery far more difficult, allowing emotional contagion to take deeper root across the network.

Figure 6 represents one of the scenarios with the lowest levels of susceptibility and

transmission in our study, with $\alpha = 0.1$ and $\beta = 0.7$, combined with relatively high recovery and support rates ($\sigma = 0.15$ and $\gamma = 0.2$). This results in a low average number of infected individuals (mean = 22.94), while the susceptible (mean = 77.92) and recovered (mean = 71.95) populations remain high. The number of active individuals is also relatively low (mean = 27.99), indicating that fewer people need to provide support at any given time. This suggests a more sustainable emotional environment, where strong recovery and support reduce infection spread and lessen the burden on those offering help over the long term. This scenario could reflect a workplace with effective mental health policies and resources in place. This could include regular counseling services, wellness programs, and flexible work schedules that help employees recover quickly from stress and prevent emotional distress from spreading widely. Because susceptibility and transmission rates are low, most employees remain either unaffected or quickly recover after experiencing stress. The moderate number of active individuals represents peer supporters or wellness champions who provide occasional help without becoming overwhelmed. This balance creates a healthier, more sustainable work environment where emotional strain is managed effectively, reducing burnout and promoting overall well-being.

Overall, these four simulations show how variations in recovery rate, support effectiveness, and transmission probability directly shape emotional dynamics in a networked population. Strong recovery and peer support mechanisms, as seen in Figure 3, can keep infection low even when exposure is high, highlighting effective emotional resilience. Figures 4 and 5 show weakened recovery and support, leading to higher infection rates and greater emotional strain, with active individuals increasing as they attempt to compensate which may ultimately lead to them facing burnout due to insufficient institutional support. Figure 6 presents a more balanced scenario with low susceptibility and transmission alongside strong recovery and support, resulting in one of the lowest infected populations and a manageable active group, exemplifying a sustainable environment where emotional contagion is limited and support efforts remain effective over time. Together, these models highlight the critical role of recovery and support mechanisms in preventing persistent emotional distress and sustaining well-being within social networks.

V Results

Our simulations show that emotional contagion dynamics in a networked population are primarily shaped by three parameters: susceptibility (α), support effectiveness (σ), and self recovery (γ). While transmission rate (β) influences the spread of distress, the ability to recover (either individually or with support) plays a more critical role in determining overall emotional stability. In Figure 3, relatively high recovery and support rates (γ and σ) help maintain low infection levels and emotional stability, even with moderate susceptibility. The system remains balanced, with infection kept under control by strong peer and

institutional recovery mechanisms. In Figure 4, all recovery mechanisms are weakened. Although the infection rate is still high ($\beta = 0.95$), the lack of effective support and low γ values result in a system that fluctuates between infected and recovered states without achieving lasting stability. The active group expands temporarily to provide support, but their efforts are insufficient, and emotional distress continues to circulate. Figure 5 presents a more extreme breakdown. The combination of high β and weak recovery and support mechanisms leads to sustained infection levels, with a large proportion of the network remaining emotionally distressed. Active individuals increase in number but have limited impact due to low support effectiveness. Recovery is minimal, and emotional volatility persists. In Figure 6, both susceptibility (α) and transmission (β) are low, while σ and γ are improved. This leads to one of the lowest proportions of infected individuals across all models. A large portion of the population remains susceptible or recovered, and the burden on active individuals is comparatively low. While the system still experiences emotional circulation, the overall strain is more manageable, making this a more stable configuration over time. Across all simulations, we observe that recovery and support mechanisms, not just exposure, determine the system's resilience. When σ and γ are strong, even high β values don't result in widespread contagion. But when support is weak, distress spreads more easily and lingers longer. These findings highlight the importance of designing interventions that enhance both institutional recovery pathways and peer support networks. Unlike fully connected models, our network-based simulation captures the localized nature of emotional contagion, allowing for feedback loops, clustering, and variability in outcomes. Rather than settling into a single steady state, the system responds dynamically to small changes in parameters. This provides a more nuanced and realistic picture of how emotional states propagate and persist in complex social environments.

VI Conclusion

Our study demonstrates that emotional contagion and mental health dynamics in social networks can be more accurately modeled by extending the classical SIR framework to a SIRA model embedded within a network-based simulation. This approach reveals that the effectiveness of recovery mechanisms (γ) and peer support (σ) often outweigh the impact of exposure alone in shaping emotional stability. Strong recovery systems can contain emotional contagion even when transmission rates are high, while weak support structures may allow even minor stressors to create persistent distress. In addition to introducing this new framework, we present simulations with parameter sets reflecting plausible real-world scenarios, highlighting the model's flexibility and generalizability. With only slight changes to inputs, the framework can capture a wide range of network behaviors and emotional outcomes, making it adaptable to various social settings. Looking ahead, several directions offer promising extensions. Future models could explore how different network structures such

as scale-free or small-world topologies influence emotional spread and recovery. Incorporating dynamic networks, where relationships change in response to emotional states, would better mirror real social systems. Our study is also limited to first degree connections. Future simulations could also test whether emotional contagion diminishes or intensifies across second and third degrees of separation, offering insight into how far-reaching the influence of distress or support can be in structured networks. Further complexity could be added by accounting for digital interactions, institutional policies, or group-based interventions. Empirical validation is a critical next step. Calibrating the model with real-world data from schools, workplaces, or online communities would test the realism of parameter estimates and the model's predictive power. Additionally, future simulations could evaluate interventions such as awareness campaigns or peer support training by adjusting parameters like δ or σ to assess their systemic impact. Ultimately, refining and testing this framework can contribute to a more predictive, intervention-oriented understanding of emotional contagion. Such insights have the potential to inform public health strategies and promote emotional resilience across diverse populations.

VII Acknowledgments

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