

**INTEGRATING COGNITIVE FACTORS IN NETWORK MODELS OF
EPIDEMIOLOGY WITH APPLICATIONS TO DISEASE CONTROL**

CONGJIE SHI

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Abstract

Understanding the interplay between information dissemination, behavioural responses, and disease dynamics remains a critical challenge in network-based epidemiological modelling. While network models offer a powerful framework for capturing individual-level interactions across both physical and virtual spaces, important knowledge gaps persist—particularly in how misinformation and behavioural adaptation jointly shape epidemic outcomes. This dissertation addresses these gaps by developing a novel three-layer network model that integrates information diffusion, cognitive processing, and epidemic transmission.

In the first part, we show that protective behaviours driven by information-based decision-making are significantly more effective at suppressing disease spread than imitation-based strategies. We also find that educating and warning individuals to counter misinformation is more effective than network-based sanctions, such as suspending gossip spreaders.

The second part explores the structural complexity of the information network, focusing on higher-order interactions represented through hyper-edge topologies. We demonstrate that scale-free information structures sustain prolonged and periodic waves of misinformation, in contrast to the more transient dynamics observed in small-world networks.

In the third part, we extend our analysis to vaccination behaviour. Our results highlight

the importance of timely misinformation correction in enhancing vaccine uptake and reducing disease burden. We also show that preemptive vaccination strategies significantly improve coverage and mitigate attack rates, even in environments saturated with disinformation. Notably, targeted vaccination approaches, which prioritise highly connected individuals (hubs), consistently outperform random strategies in reducing infections and severe disease outcomes.

Together, this dissertation offers a comprehensive framework for examining how complex information-behaviour-epidemic feedbacks shape public health outcomes, and provides actionable insights for designing robust interventions against misinformation and infectious disease spread.

Dedication

To my beloved parents.

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To conclude, I hope to share a quote from Albert-László Barabási: "Complexity often bewilders us, yet life as we know it would not be possible without it."

We will continue to learn – through unexpected edges in our networks and serendipitous links in our lives.

List of Publications

Part of the dissertation is based on the following published papers.

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Chapter 1

Introduction

The comprehensive framework that network science offers for understanding complex systems is beneficial in numerous domains, including biology, sociology, computer science, and physics [1]. This paves the way to examine the structure and function of complex systems, ranging from biological cells to social interactions [1].

For a complex system, the idea of “connectedness” encompasses not only structural ties (which individuals are linked) but also behavioural impacts, where individual actions reverberate throughout the system [2]. Such systems considers both its structure and how interactions unfold within them [2]. In these settings, activities are not isolated events but rather catalysts that elicit systemic responses, revealing cause-and-effect relationships [2].

Infectious diseases account for 43% of the worldwide disease burden, measured by the number of healthy years lost [3, 4]. However, drugs and vaccines alone often fall short in halting these diseases. Understanding how pathogens evolve and propagate across populations is crucial, and this spread may be modelled using contact networks for airborne diseases like influenza and SARS [3].

Social networks are important tools for tracking the dissemination of behaviours, ru-

mours, and information [3]. The diffusion of information and behaviours within these networks depends on the type of disease, with complex contagions requiring social reinforcement and depending heavily on community structure [5]. Models of collective behaviour, which focus on “thresholds,” illustrate how individuals’ decisions are influenced by the actions of others [6]. The distribution of these thresholds is critical in determining group outcomes; thus, even similar average preferences can lead to different results [6]. Understanding the spreading patterns of information and its impact on behaviour change, such as public health interventions, is crucial [7]. The diffusion of behaviour through a population is often modulated by the structure of social networks [7]. For instance, behaviours are more likely to spread when individuals are exposed to many others who have already adopted them, particularly in tightly-knit communities where friends are interconnected [7].

During the initial stages of an emerging infectious disease when no vaccine is yet available, it is important to rely on short-term strategies such as wearing masks, social distancing, restriction on gathering. Therefore, part of this research is dedicated to studying the impact of misinformation on short-term and reversible intervention measures. Vaccines often become available after a few months, and they are an important control strategy for public health policymakers. Thus, a subsequent part of this research focuses on how misinformation and vaccine behaviours interact.

By constructing network-based frameworks, we can gain deeper insights into pathogen transmission, access information spreading patterns, and develop strategies to mitigate or control their impact.

In this dissertation, we first provide some context on network model approaches and identify the knowledge gaps (Chapter 1). Then, we provide a review of the current literature on information, cognition, and disease using network models (Chapter 2). Next, we present a few basic concepts and important terminologies in network structure (Chapter 3). We set the foundation for how network models are represented in existing literature. We also

provide a brief overview of multilayer networks that are frequently used in recent studies.

The goal of Chapter 4 and 5 was to develop a multi-layer network model and investigate the complex interactions within and between the layers of information, cognition, and epidemics (ICE). Rumours spread in the information layer independently of cognition and epidemic layers, while taking into account higher-order interactions (Figure 4.1). An intermediary cognition layer, where individuals switch between protective and unprotected behaviours based on information and cognition layers, modulates the infection rates of the epidemic layer. We investigated the effect of misinformation on disease transmission, focusing on immediate interventions such as mask-wearing and social distancing. Our analysis addresses the characteristics of misinformation spread within hyper-edge structures and identifies strategies to mitigate its propagation. The findings suggest that educating individuals within an information network is more effective in curbing disease spread than the suspension of gossip spreaders from the information network. Our results highlight the relative importance and impact of different strategies for correcting misinformation.

The objective of Chapter 6 is to extend the analysis on the ICE model to understand heterogeneity. We investigate network architecture and higher-order information layer interactions to determine how heterogeneities impact protective decision-making and disease transmission. Our findings indicate that disinformation initiated from low-degree node enhances protective measures, while hyperedge group topologies facilitate misinformation propagation regardless of the size of the source node. Scale-free networks feature sustained epidemics with prolonged gossip peaks, unlike small-world networks. In heterogeneous networks with power-law-distributed hyperedge sizes, knowledge-driven decisions reduce disease transmission more effectively than neighbourhood imitation.

In Chapter 7, we extend our investigation by exploring the interplay between misinformation and vaccination behaviours. Specifically, we assess how varying levels of misinformation correction efforts influence the prevalence of gossip, vaccine uptake, and

disease spread over time. Our findings demonstrate that even modest efforts to correct misinformation can considerably boost vaccination uptake, and in turn, substantially reduce disease transmission. In addition, we show that preemptive vaccination campaigns can effectively mitigate the detrimental impact of persistent misinformation on attack rate. Notably, in highly heterogeneous networks characterised by higher-order interactions, prioritising vaccination of hubs (high-degree nodes) proves especially effective in limiting overall infection and reducing the incidence of severe disease.

Chapters 8 and 9 present synthesis and discussion of our results. The approaches developed in this thesis provide a comprehensive framework for understanding the dynamic interplay between information dissemination, cognition processing, and behavioural responses in public health contexts. For future work, we aim to investigate the role of community structure, network connectedness, and hub dynamics more deeply, especially within data-driven network topologies. We also plan to extend our current model to incorporate time delays, with a focus on how lags between misinformation exposure and individual behavioural responses influence epidemic outcomes.

Chapter 2

Literature Review

Infectious diseases continue to inflict significant health and socioeconomic burdens globally [8, 9]. Mitigating their impact is often influenced not just by the intensity and timeliness of interventions, but also by the degree of adherence to these interventions, which can be affected by various factors such as information dissemination, risk perception, disease outcomes, and behavioural responses during an outbreak [10, 11]. Understanding the interplay between information diffusion and behavioural changes is therefore critical for devising effective control strategies [12, 13, 14].

Transmission dynamic models are vital tools for characterising disease dynamics at both individual and population levels [15, 16, 17, 18]. Conventional models often rely on the simplified assumption of homogeneous mixing among individuals, which is insufficient to capture the real contact patterns and possible effects of exogenous factors such as disease-related information [19, 15, 20]. In contrast, network models improve upon this by incorporating specific network structures that better reflect actual interactions, thereby enhancing predictions on disease transmission and the impact of interventions [21, 3, 22, 23]. Networks can exhibit similar organizational principles despite having different

connectivity structures that represent social interactions and transient separations [3, 24]. Nodes and edges within these networks can symbolize various connections, including familial, occupational, and social ties [25]. Multilayer networks, which integrate both virtual and real connections [26], offer important insights into how information and disease simultaneously propagate along the connected nodes.

2.1 Multilayer networks

Recently, there has been a surge of interest in creating realistic network models to explore how information and behavioural responses influence epidemic dynamics [27, 28, 29, 30, 31, 32, 33]. Despite expanding efforts to study two-layer interactions [34, 35, 36, 37], our understanding of the interplay between information flow, behavioural adjustment, and disease dynamics remains nascent. One common approach to quantifying the relationship between two layers entails a coupled Unaware-Aware-Unaware and Susceptible-Infected-Recovered (SIR) model, which integrates aspects of both disease and perception [38, 39, 40, 41]. In this type of model, the information layer tracks individuals' awareness status, categorising them as either aware or unaware [38, 39, 40, 41], while the disease transmission layer represents their epidemiological status [38, 39, 40, 41]. Extending this approach, a three-layer network model can simulate the effects of knowledge bias, vaccine behaviour dispersion, and epidemic propagation [42]. An alternative strategy is to implement a behaviour layer, which incorporates transient choices such as self-protection [43].

Despite numerous studies on complex contagion and collective behaviour, an important gap in current literature is the lack of investigation into group cognition phenomena regarding the acceptance and behavioural impact of information diffusion on various social media platforms. The role of social networks and peer influence in the spread of information and behavioural changes within a group is crucial, rendering isolated

encounters inadequate to reflect individual decisions under collective effects and group interactions [44, 7]. Complex contagion models, characterised by the involvement of several infected hosts in each transmission event, provide a more realistic representation of the spreading phenomenon in social networks, particularly in the context of online information dissemination [44, 45]. In a basic contagion process, susceptible nodes may become infected *via* separate interactions with contagious neighbours [44]. In a threshold model, the status of a node changes when the proportion of its neighbouring nodes that acquire infection exceeds a certain threshold [46]. Models with higher-order network structures can provide a more accurate description of real-world complexities that extend beyond one-to-one interaction, while allowing for simultaneous impacts from different sources with collective dynamics [47, 48]. The simplicial complex is one established and direct approach to investigate group spreading but falls short in capturing the variations in acceptance of an opinion, whereas nonlinear hyper-graphs extend on this idea to study the nuanced dynamics and effects of collective behaviour [44, 49]. As research efforts on the development of contagion architecture continue [50, 51, 52], our hyper-edge model provides a general framework to integrate key aspects of both the threshold and simplicial complex models.

2.2 Vaccine timeliness, acceptance, and information challenges

Immunisation strategies are plans for the distribution of vaccines as well as therapies [3]. There are a number of factors that preclude universal coverage, including affordability, accessibility, and fears about adverse effects. Vaccine distribution tactics that take into account network topology are the focus of our study, with the goal of reducing the spread

of disinformation and epidemics.

Research on vaccine use and delay has identified many key factors. Economic and psychological variables combine to influence vaccine hesitancy, which is the failure to receive vaccines despite being offered [53]. Individual health beliefs play a crucial part in understanding this phenomenon, as explained by the Health Belief Model (HBM), which varies with susceptibility, disease severity, and confidence in vaccination [53, 54]. Societal norms, fear of negative effects, social media rumours, knowledge, attitudes, and risk perceptions are other factors that contribute to delayed adoption [55, 56, 57, 58, 59].

Historically, anti-vax attitudes (i.e., the refusal of vaccination) has posed formidable barriers to vaccine acceptance before widespread COVID-19 vaccine availability [60]. Since the success of vaccination campaigns depends on broad societal involvement, crafting compelling messages becomes paramount to foster vaccine acceptance [61]. In addition to the issue of health beliefs, vaccine shortages and uneven distribution of vaccines continue to present a challenging problem, especially in underdeveloped regions [62]. Efficient vaccine manufacturing processes are crucial to facilitate rapid development and deployment [63].

Public health efforts must safeguard both individuals and communities. Doubts about vaccine safety might hinder attempts to increase immunisation rates for community protection [64]. Vaccine availability affects public willingness to vaccinate, with evolving safety and efficacy data influencing individual choices over time [65].

Herd immunity denotes the phenomenon whereby immunising a certain portion of the population lowers the risk of wide-spread disease transmission [66]. Often, vaccination campaigns overlook the need to reach these disease eradication benchmarks [67]. Developing herd immunity is often difficult due to the limited duration of protection and logistical constraints [68]. The interplay between vaccine deployment and efficacy suggest that rapid increase in population coverage, even with moderately effective vaccines, during outbreaks may be critical to curb disease spread [69]. Rapid vaccination, even if herd immunity

cannot be achieved, would provide quick partial protection to a larger population while potentially reducing hospitalisations and deaths. [70]. Prioritizing vaccines during disease outbreaks is a complex and challenging endeavour that requires balancing transmission reduction and mortality prevention across various age groups [71].

Chapter 3

Overview of Network Models

Complex systems are intriguing due to their unpredictable and emergent collective behaviours. They are woven into sophisticated networks that capture the essence of their interactions. The elegance of these networks lies in their universal organizing principles, even though their specific connections vary [3]. By leveraging graph theory, we can map these systems' elements as vertices and their interactions as edges, uncovering shared properties such as small-world and scale-free phenomena. This chapter provides a concise overview of the fundamental concepts in complex networks [3], laying the foundation for understanding how the structural intricacies of diverse networks influence their collective dynamics.

3.0.1 Network terminologies

A network is solely characterised by its connections. One effective way to depict a network is through an adjacency matrix, abbreviated as A_{ij} . The rows and columns of the square matrix symbolize the nodes in a static network with N nodes. The undirected link between nodes i and j is denoted by the matrix element A_{ij} , which is set to 0 for unconnected nodes

and 1 for connected ones.

A node's *degree* k_i is its total number of links, obtained through:

$$k_i = \sum_{j=1}^N A_{ij}.$$

The total number of undirected *links* L can be represented with:

$$L = \sum_{i=1}^N k_i = \frac{1}{2} \sum_{ij} A_{ij}.$$

The *average degree* of a network is defined as:

$$\langle k \rangle = \frac{1}{N} \sum_{i=1}^N k_i = \frac{2L}{N}.$$

3.0.2 Degree distribution and n -th moment

The *degree distribution* $P(k)$ represents the probability that a randomly chosen vertex in a network has a degree of k . If N_k is the count of vertices with degree k in a network of N total vertices, then we denote $P(k)$ as:

$$P(k) = \frac{N_k}{N} \tag{3.1}$$

The n -th *moment* of the degree distribution is given by:

$$\langle k^n \rangle = \sum_k k^n P(k), \tag{3.2}$$

where the *first moment* represents the average degree, and the *second moment* is related to the variance, defined as $\sigma^2 = \langle k^2 \rangle - \langle k \rangle^2$.

3.0.3 Network types

Several empirical networks are marked by a degree distribution with heavy tails that are well represented by *power-law distributions* in the form $P(k) \sim k^{-\gamma}$, where γ is the *degree exponent*. We denote the minimum and maximum degrees of a network by k_0 and k_c , respectively. *Hubs* refer to the nodes with a high degree (many neighbours) in a network. Networks characterised by a power-law degree distribution can be classified into three distinct types based on the *degree exponent* γ :

1. *Scale-free networks*: These networks have a degree exponent in the range $2 < \gamma < 3$. In such networks, the first moment of the degree distribution is finite, while the second moment diverges as the network size approaches infinity. This divergence reflects the dominance of hubs, which significantly reduce the average distance between nodes.
2. *Critical Networks*: When $\gamma = 3$, the network exists in a transitional phase, balancing features of both random and scale-free networks. This critical point represents a unique structural state where the network exhibits mixed properties.
3. *Small-world networks*: For networks with $\gamma > 3$, the degree distribution does not exhibit divergent variance. While hubs are present, they are relatively few and insufficiently connected to strongly influence the distances between nodes.

The idea of *scale-free* networks originates from the British physicist Derek de Solla Price in the 1960s, when he observed that citation links to scientific papers followed a heavy-tailed power-law distribution [72]. The scale-free property describes networks where a small number of nodes have a disproportionately high number of connections, with the variance of connections theoretically extending to infinity. This concept is particularly relevant in social networks (especially online platforms), as there is no upper bound to

the number of connections one can establish. Therefore, the scale-free property is a key distinction between virtual networks and physical contact networks.

3.0.4 Model assumptions

In this research, we used synthetic networks to elucidate concepts for various spreading phenomena. Numerous models exist to construct complex networks, such as the Erdős–Rényi model for random small-world graphs [73] and the Barabási–Albert model for power-law degree distribution graphs [74]. We applied the uncorrelated configuration model [75], which generates undirected, synthetic, and unweighted networks based on a predetermined degree distribution $P(k)$. Initially, each vertex is assigned a degree k by sampling from the degree distribution [75]. The unconnected half-edges or stubs are then randomly paired, ensuring that no self-connections or multiple connections are created [75].

3.1 Representation and generation of networks

3.1.1 Adjacency matrix and adjacency lists

There are many ways to represent networks. One way is known as the adjacency matrix A_{ij} . For a network with N nodes, the matrix would have size $N \times N$. If an edge exists between two nodes i and j , then the matrix element in i th row and j th column is $A_{ij} = 1$. Otherwise, if the nodes are not connected, the matrix entry would be $A_{ij} = 0$. This method is simple but not efficient. The degree of a node k_i is then the sum of the edges connected to the node and can be represented by: $k_i = \sum_{j=1}^N A_{ij}$.

A more efficient way to store networks is using adjacency lists [76]. There is a main list with each node followed by its neighbors. There is also a supplementary index list, that stores the starting position of each node’s neighbour group.

3.1.2 The configuration model

To generate networks, a common approach is the configuration model. With a configuration model, we start with a degree sequence, which specifies many neighbors each node should have. Then we assign half-edges to the nodes based on their degrees, and randomly pair nodes to form edges. The popular Erdős-Rényi Model (with Poisson distribution) and the Barabási-Albert Model (with power-law distribution) are both configuration models that follow a statistical distribution but not an exact degree sequence [77]. There are also models that use a specific degree sequence without introducing randomness into the network structure, such as the Chung-Lu Model and the Edge-Swapping Model [78, 79].

3.2 Misinformation and social media platforms

Misinformation is erroneous or inaccurate information that may be shared accidentally. Its rapid diffusion poses key risks to public health efforts. On social media platforms, the spread of misinformation relies on their social connections. When two users are connected, denoted by an edge in network terms, they can affect each other by potentially transmitting gossip between them.

Social media platforms serves as a vital source of misinformation, particularly through two channels. First, viral rumours grow when individuals unintentionally like, comment on, or share false content, which increases its popularity [80]. Second, echo chambers, which are tight-knit online groups, encourage and sustain incorrect views by frequently exposing members to the same incorrect assertions [81]. Misinformation spreads quickly owing to social media, which allows people to effortlessly share content with strangers as well as their own connections.

3.3 Multilayer networks

Multilayer networks are graph structures in which nodes are connected across multiple levels, representing various interactions and permitting inter- and intra-layer connections. Multilayer networks can effectively simulate real-world connectivity and dynamics by capturing nuanced subsystem interactions [25]. To better understand and evaluate complex systems, classical network theory can be extended to unify disparate ideas and facilitate connections across layers and processes [25]. Recent developments in multilayer network theory have transformed network research by including time-varying and context-specific interactions, as well as dynamic network topological features, enabling a more comprehensive understanding of the systems' structural and dynamical attributes [82].

Multilayer networks are important for capturing dynamics in multiscale systems. Each layer depicts separate processes that can influence others. These coupling mechanisms connecting layers often involve higher-order connections beyond pairwise links. Studies have explored how information propagates across time frames under particular structural conditions [83]. Multilayer networks have proven to be useful for modelling multidimensional interactions, explaining emergent behaviours in complex systems such as biological communication and environmental relationships [83].

3.4 Compartmental network models

3.4.1 Disease compartments

Classic network models for infectious disease spread usually employ compartmental methods to describe the systems [84]. In these models, individuals are often divided into three states: susceptible (S), infected (I), and recovered (R). There are two variations: (1) the SIR

model assumes that recovery grants immunity; (2) the SIS model assumes that infected individuals return to become vulnerable again [85, 86]. When agent propagation is modelled in the network, contact between susceptible and infected people results in disease transmission, while recovery occurs independently. More complex variations include models such as the SIRS and SEIR models that account for waning immunity or the occurrence of an exposed stage [87].

3.4.2 Information compartments

Similar concepts of compartments also apply to information dissemination. One method is through a competitive information spread model, examining how positive/negative material competes while including local and global impacts on user behaviour [88]. For example, the SIIHR model takes into account the susceptible, the positively or negatively infected (people may get exposure to correct or false information), hesitant, and recovered classes for spreading knowledge [88]. Another common approach to classifying information status is separating individuals into aware (A) and unaware (U) groups [30]. In this type of model, people in the aware group have knowledge about a disease, while information regarding the disease spreads during the epidemic [30].

3.5 From pairwise to higher-order networks

Conventional networks have connections entirely between pairs of nodes, referred to as pairwise networks. The paired framework limits the depiction of group interactions as those interactions must be split into many binary connections.

Higher-order networks refer to networks that can capture multi-node interactions beyond pairwise connections [89]. Their structure, composed of simplices or hypercubes, provides

a richer framework for analyzing how network topology influences system dynamics [89].

For systems in which the number of participating nodes non-linearly influences interactions, the pairwise strategy falls short. Higher-order networks have been developed to address this constraint, hence going beyond paired links to capture concurrent interactions over many nodes [47, 49]. The higher-order system allows for a more precise and flexible way to depict complex group dynamics [47, 49].

3.5.1 Advances in higher-order processes

A recent study investigated the contagion process in social networks in which the transmission depends on contact with multiple sources [90]. Their findings showed that with overlapping contacts, outbreak dynamics and system stability rely on both pairwise and three-way interactions [90]. Their results also highlighted the effect of reduced infection thresholds, offering a fresh perspective on how group-level architecture affects spreading events outside of pairwise processes [90].

Network topology and contact pattern are crucial elements in diffusion processes [91]. Since the network structure is different for higher-order networks compared to pairwise networks, omitting higher-order effects may distort the dynamics: a discontinuous transition could appear continuous in pairwise approximations [52].

A key theme in network research has been knowing how the spreading processes develop throughout linked networks. The area of network spreading process covers many different applications, ranging from modelling disease transmission in epidemiology to studying how information or ideas spread via social networks [92]. There have been numerous contributions in this field which improve our understanding of opinion diffusion in complicated systems, providing insights on collective motion and behavioural dynamics within communities [92].

Chapter 4

Multilayer Network Model

In this chapter, we developed a multilayer network model to investigate the complex interactions within and between the layers of information, cognition, and epidemics (ICE). We also examined the dynamics of the model's layers to establish a formula for the reproduction number under the assumption of homogeneous mixing.

4.1 Information-cognition-epidemic (ICE) model

We developed a three-layer network model where nodes represent the individuals while the interactions are modelled according to the characteristics of virtual versus in-person networks. Information propagates in the top layer (Figure 4.1), altering the dynamics of behavioural responses in the cognition (middle) layer. Finally, the disease spreads through the physical contact network in the bottom layer where infection rates depend on the cognitive status defined in the cognition layer.

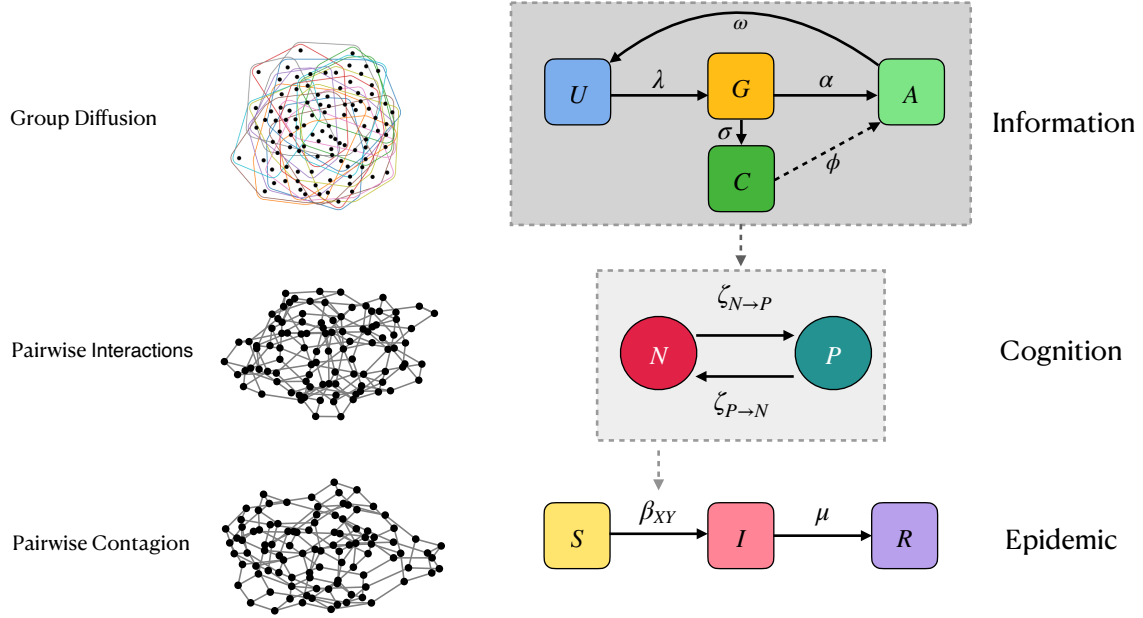


Figure 4.1: Schematic representation of the information, cognition, and epidemic (ICE) model dynamics. The information layer follows a Daley-Kendall [93] rumour-spreading model with higher-order (group) interactions where individuals can be in one of four states: the uninformed (U), the gossip spreader (G), the aware stifter (A), and the corrected (C). Transitions between information states are depicted in the top line. In the cognition layer, individuals can alternate between states that are non-protected (N) and protected (P) against infection with transition rates $\zeta_{N \rightarrow P}$ and $\zeta_{P \rightarrow N}$, respectively, depending on the proportion of gossip spreaders and the perceived level of protection in their neighbourhood. In the epidemic layer, disease transmission occurs as a result of pairwise interactions between susceptible (S) and infectious (I) individuals at a rate β_{XY} , $X, Y \in (N, P)$, which is influenced by the cognitive state of the interacting peers. Infected individuals recover (R) at a rate μ . The pairwise networks were constructed and visualized with the Python library 'NetworkX' [94].

4.1.1 Information Layer

Our approach employs hyper-edge complex contagion to model the spread of misinformation *via* group interactions. Information diffusion is often conceptualized using an SIR-type framework known as the Daley-Kendall model, analogous to infectious disease transmis-

sion [95, 93], generalized into a basic rumour model with an Ignorant-Spreader-Stifler architecture [96, 97, 98]. The distinct stages in our hyper-edge rumour diffusion model include the uninformed (U) who is unaware of the rumour, the gossip spreader (G), the aware stifler (A) who is informed of the rumour but does not transmit it while suppressing spreading upon contact with spreaders, and the corrected (C) who receives correction and is removed from the information network (Figure 4.1). We considered five potential events: (i) the transmission of gossip (when an uninformed encounters a spreader) with rate λ ; (ii) the suppression of misinformation (when a spreader meets a stifler or a spreader) with rate α ; (iii) the renewal of interest to new rumour with rate ω ; (iv) the correction of rumour with rate σ ; and (v) the relapse rate of ϕ after correction (Figure 4.1). In real context, information networks face a continuous flow of new rumours of a similar nature. Therefore, the renewal of interest considering a single rumour simplifies the complexity arising from multiple rumours coexisting within the information networks. The correction, on the other hand, serves as an interventions aimed at curbing misinformation by targeting and penalizing spreaders. State transitions and intervention scenarios are summarised in Table 4.1.

In the baseline scenario of our model, we assumed that there is no relapse after correction, that is $\phi = 0$, which may describe the context for correcting misinformation on a single topic. In additional analyses, we evaluated the effect of relapse, representing the presence of gossip on multiple issues. Misinformation correction is generally contingent on the information structure of different online platforms. For example, banning individuals from the information network to mitigate the effect of misinformation is possible, albeit rare and temporary. For secondary analyses, we considered this mechanism for correction of misinformation by suspending individuals from the network, which can occur in $\mathbb{X}$ (formerly Twitter) and Instagram. This can be modelled by a transition $G \xrightarrow{\sigma} C$ with a relapse rate of $\phi > 0$. This mechanism can represent two scenarios: (i) individuals in G are reported by others and thus suspended from participating in the information network for an

Table 4.1: Description of state transitions and misinformation intervention scenarios in the information layer of the ICE model presented in Figure 4.1.

State transition	Transmission type	Intervention	Real-world example
$U \rightarrow G$	Interaction-based with threshold	Gossip transmission	An uninformed individual encounters a spreader, and the proportion of spreaders in the hyper-edge group exceeds the threshold to spread misinformation
$G \rightarrow A$	Interaction-based	Misinformation suppression	A spreader meets a stifler or another spreader
$G \rightarrow C$	Spontaneous	User suspension	An account is blocked after detection of gossip activities on Facebook
$G \rightarrow C$	Interaction-based	Warning or educating peers	An individual learns from friends in a WhatsApp group and stops spreading misinformation
$C \rightarrow A$	Spontaneous	Relapse after correction	An individual may become stifler of misinformation after correction
$A \rightarrow U$	Spontaneous	Renewal of interest to new rumour	An individual may be exposed to new type of misinformation

average duration of $1/\phi$; (ii) individuals in G correct themselves by learning the falsehood of the information.

We also evaluated another mechanism for correction by warning individuals within the information network, a practice common in WhatsApp groups and Facebook communities. Corrected (or educated) individuals can halt the spread of gossip by warning individuals in G within a group. In this context, the corrected (educated) population can be modelled using a spontaneous transition from G to C due to external influences, and a stimulated transition from G to C upon interactions with corrected individuals, both with a rate of σ . A spontaneous transition occurs independently and does not require interaction with another individual in the information network.

In our model, a hyper-graph with a power-law distribution of contact numbers is constructed through an algorithm adapted from a configuration model for pairwise random

networks [3, 99, 75, 100]. The hyper-edge model can be generated with the desired number of nodes, number of hyper-edges, degree exponent, and minimum degree (Figure 4.2A). We assigned misinformation acceptance threshold values to nodes in the information network based on a normal distribution of mean 0.1 and standard deviation 0.05. The selection of a heterogeneous threshold value is grounded in the complex contagion model, which describes the individual decision-making process based on collective behaviours. Previous work demonstrated the benefits of using a simple threshold rule that allows for local dependencies within neighbourhoods, fractional cutoffs across different nodes, and heterogeneity in node connectivity [46]. Watts’ theoretical analysis investigated diverse mean thresholds for both uniform and scale-free random graphs, with standard deviations of 0.05 and 0.1 [46].

To mirror social media network architecture, we assumed the information contact network follows a scale-free distribution with $2 < \gamma_i < 3$, where γ_i denotes the power-law exponent for the information contact network (Figure 4.1). Virtual networks are generally scale-free because a few nodes have exceptionally high connectivity while the majority of nodes have limited links. This topology has been observed in a variety of systems, including the WWW and citation graphs [3]. Furthermore, high-degree nodes in online social networks, such as influencers, frequently facilitate quick information dissemination.

A hyper-edge is an extension of a conventional edge that may connect more than two nodes concurrently. It is used to represent intricate connections involving multiple entities at once. To implement the threshold paradigm with hyper-edge for spreading misinformation, we first selected a spreader at random according to a pre-defined degree distribution. Next, a hyper-edge that contains the spreader was chosen randomly. For each unaware neighbour in the selected hyper-edge, we assessed whether the proportion of its spreading neighbours met the threshold for accepting misinformation. If the threshold was met, the unaware neighbour became a spreader. An example of the misinformation spreading process is

shown in Figure 4.2B.

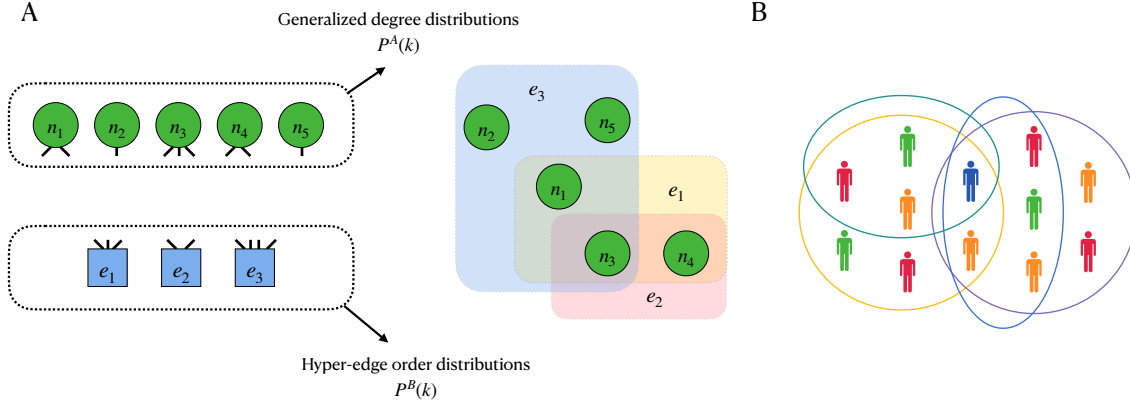


Figure 4.2: (A) The hyper-edge structure of the information layer is based on bipartite configuration model [49]. This configuration includes a degree sequence, represented by the corresponding number of stubs (half-edges) associated with each node (circles in panel A), and a sequence of hyper-edge sizes, represented by the corresponding stubs associated with each hyper-edge (squares). The number of stubs in both nodes and hyper-edges must be equal. Furthermore, the stubs are connected in a bipartite format through random pairing. The resulting network was constructed and visualized with the Python library ‘HyperNetX’ [101]. (B) A schematic diagram for the hyper-edge threshold rumour diffusion process. A spreader node (blue) is randomly selected based on the degree distribution. Then a random hyper-edge containing the spreader node is chosen. Every ignorant (orange) individual in the chosen hyper-edge becomes a spreader if the proportion of spreaders (red) in the hyper-edge exceeds the corresponding threshold.

4.1.2 Cognition Layer

In the cognition layer, there are two behavioural states: protected (P) and unprotected (N) (Figure 4.1). Individuals’ protective decisions are influenced by both the physical and virtual environments in which they dwell and interact [102]. Factors that affect such decisions may include the epidemiological risk from direct interactions and the social risk presented by virtual communications [102]. Thus, a state switch can be triggered by either

the surroundings in the information layer or the neighbouring patterns in the cognition layer.

A *hyper-edge network* is a network where multiple nodes are connected at the same time. A *bipartite graph* is a network such that there exists two disjoint sets of nodes while there is not edge between nodes in the same set. The term k_{info} denotes the node degree (or the total number of neighbours of a node) on the information layer. Similarly, the term k_{cog} denotes the node degree on the cognition layer.

The transition rates between the states P and N depend on the proportions of gossip spreaders in the neighbourhood in the information layer and of protected neighbours in the cognition layer given by $\rho_G = n_G/k_{\text{info}}$ and $\rho_P = n_P/k_{\text{cog}}$, respectively (Figure 4.1), and modulated by mass information, which depends on the overall disease prevalence, ρ_I . The relationships can be expressed by:

$$\zeta_{P \rightarrow N} = \zeta_1 \rho_G [1 - \tanh(b_1 \rho_I)] + \zeta_3 (1 - \rho_P), \quad (4.1)$$

and

$$\zeta_{N \rightarrow P} = \zeta_2 (1 - \rho_G) \tanh(b_2 \rho_I) + \zeta_4 \rho_P, \quad (4.2)$$

for the transitions between protected and unprotected states. The rates ζ_i ($i = 1, 2, 3, 4$) control the behavioural responses to information. The relation between misinformation and risk perception is straightforward: a higher number of spreaders in the information network leads to faster abandonment and slower adoption of protective measures, controlled by rates ζ_1 and ζ_2 , respectively. The influence of the proportion of protected neighbours is reversed, with inhibition and encouragement of protection being governed by parameters ζ_3 and ζ_4 , respectively. To integrate feedback from global disease spread into the cognition layer, we employed the hyperbolic tangent function applied to the density of the infected population,

adjusted by fixed parameters b_1 and b_2 representing global communication intensity. The impact of transition rates on the interaction between information and cognition layers will be explored through simulations.

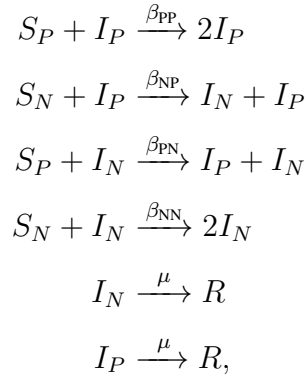
The overall factors controlling information communication, such as mass media, are adjusted by functions that either increase or decrease monotonically with the overall disease prevalence. These functions promote or reduce protective behaviours and are regulated by parameters b_1 and b_2 , respectively. A large b_i value indicates a high level of external intervention and policy enforcement. For example, during the COVID-19 pandemic, China had strict policies on protective measures, which can be described by large b_i values [103], while Italy and Spain had less restrictive policies at the onset of the pandemic [104, 105], which correspond to smaller b_i values. The limit cases $(b_1, b_2) = (0, \infty)$ and $(b_1, b_2) = (\infty, 0)$ correspond to a lack of global information and perfect intervention, respectively. In the present work, we fixed $b_1 = b_2 = 3$ to capture the effect of information diffusion even at a low disease prevalence.

To mimic real-world scenarios, we configured the cognition network with a degree distribution exponent of $\gamma_c = 3$, resulting in a degree of heterogeneity that lies between those of the information and epidemic layers (Figure 4.1). This topology possesses a hybrid structure that combines scale-free and small-world qualities, placing it near a critical point — a regime ideal for adaptation and phase transitions. It can show both hub-like organisation, like scale-free networks, and significant clustering, like small-world networks.

4.1.3 Epidemics Layer

In the epidemics layer, the epidemiological states include susceptible (S_P and S_N), infected (I_P and I_N), and recovered (R) individuals (Figure 4.1). The subscripts P and N represent the protective (for example, wearing masks to reduce viral shedding) and unprotected

behaviours of individuals, respectively. The disease spreads in a pairwise manner, which is implemented using the optimised Gillespie algorithm [106]. In order to link the network layers, we considered distinct transition rates according to the cognitive state [107], and defined state transitions based on the individuals' protection statuses, along with a single recovery process (Figure 4.1). The transitions, and corresponding rates, can be schematically summarized by:



where the infection rates are constrained to $\beta_{PP} \leq \beta_{NP} \leq \beta_{PN} \leq \beta_{NN}$, representing an increase in the risk of infection as protection is reduced. Since disease spreads on physical networks, for which scale-free networks are unrealistic, we assumed a non-scale-free degree distribution with a power-law degree exponent of $\gamma_e = 4$, which still presents some appreciable degree of heterogeneity (Figure 4.1). For the sake of comparison, we also investigated homogeneous counterparts where all nodes have the same degree given by the average degree of the heterogeneous case.

4.1.4 Homogeneous mixing approximation

While the heterogeneity is a key factor of our model, under the assumption of homogeneous mixing, local and global densities are equivalent, and the dynamical system dimension

is significantly reduced, allowing an analytical treatment. The dynamics in the cognition layer in our model depend solely on the individual states of gossip spreaders in the top information layer and the number of protected and unprotected individuals in the cognition layer. To derive the equation for the temporal evolution of $\rho_N = 1 - \rho_P$, representing the proportion of unprotected individuals, we assumed that ρ_G is stationary and, thus, time-independent. In the absence of infected individuals ($\rho_I = 0$), equations (4.1) and (4.2) become $\zeta_{P \rightarrow N} = \zeta_1 \rho_G + \zeta_3 \rho_N$ and $\zeta_{N \rightarrow P} = \zeta_4(1 - \rho_N)$, respectively. Thus, the evolution of the unprotected population under these simplifications is given by

$$\frac{d\rho_N}{dt} = [\zeta_1 \rho_G + (\zeta_3 - \zeta_4) \rho_N](1 - \rho_N). \quad (4.3)$$

Stationary solutions of (4.3) are $\rho_N^* = 1$ and

$$\rho_N^* = \frac{\zeta_1 \rho_G}{\zeta_4 - \zeta_3}, \quad (4.4)$$

where (4.4) represents a biologically acceptable solution for $0 \leq \rho_N^* \leq 1$ only if $\zeta_4 - \zeta_3 > \zeta_1 \rho_G > 0$. The local stability of fixed points is given by the sign of the one-dimensional Jacobian [108]. The Jacobian of (4.3) is given by

$$J(\rho_N^*) = -\zeta_1 \rho_G - (\zeta_4 - \zeta_3) + 2(\zeta_4 - \zeta_3) \rho_N^*. \quad (4.5)$$

When (4.4) is a valid solution, we have $J(1) = \zeta_4 - \zeta_3 - \zeta_1 \rho_G > 0$ and $J(\zeta_1 \rho_G / (\zeta_4 - \zeta_3)) = \zeta_1 \rho_G - (\zeta_4 - \zeta_3) < 0$, showing that the $\rho_N^* = 1$ is unstable, and $\rho_N^* < 1$ represents a locally stable stationary solution. When $\rho_N^* = 1$ is the only biologically valid solution, it is stable. Thus, any initial condition $\rho_N \in (0, 1)$ belongs to the basin of attraction of the stable fixed points.

For the SIR dynamics in the epidemic layer, a temporal evolution equation can be written

for each of the six compartments: $S_N, S_P, I_N, I_P, R_N, R_P$ forming a system of differential equations, given by

$$\begin{aligned}
\frac{dS_N}{dt} &= -\beta_{NN}\langle k \rangle S_N \frac{I_N}{N} - \beta_{PN}\langle k \rangle S_N \frac{I_P}{N} + \tilde{\zeta}_1 \rho_G S_P + \zeta_3 \rho_N S_P \\
&\quad - \tilde{\zeta}_2(1 - \rho_G) S_N - \zeta_4(1 - \rho_N) S_N, \\
\frac{dS_P}{dt} &= -\beta_{NP}\langle k \rangle S_P \frac{I_N}{N} - \beta_{PP}\langle k \rangle S_P \frac{I_P}{N} - \tilde{\zeta}_1 \rho_G S_P - \zeta_3 \rho_N S_P \\
&\quad + \tilde{\zeta}_2(1 - \rho_G) S_N + \zeta_4(1 - \rho_N) S_N, \\
\frac{dI_N}{dt} &= \beta_{NN}\langle k \rangle S_N \frac{I_N}{N} + \beta_{PN}\langle k \rangle S_N \frac{I_P}{N} - \mu I_N + \tilde{\zeta}_1 \rho_G I_P + \zeta_3 \rho_N I_P \\
&\quad - \tilde{\zeta}_2(1 - \rho_G) I_N - \zeta_4(1 - \rho_N) I_N, \\
\frac{dI_P}{dt} &= \beta_{NP}\langle k \rangle S_P \frac{I_N}{N} + \beta_{PP}\langle k \rangle S_P \frac{I_P}{N} - \mu I_P - \tilde{\zeta}_1 \rho_G I_P - \zeta_3 \rho_N I_P \\
&\quad + \tilde{\zeta}_2(1 - \rho_G) I_N + \zeta_4(1 - \rho_N) I_N, \\
\frac{dR_N}{dt} &= \mu I_N + \tilde{\zeta}_1 \rho_G R_P + \zeta_3 \rho_N R_P - \tilde{\zeta}_2(1 - \rho_G) R_N - \zeta_4(1 - \rho_N) R_N, \\
\frac{dR_P}{dt} &= \mu I_P - \tilde{\zeta}_1 \rho_G R_P - \zeta_3 \rho_N R_P + \tilde{\zeta}_2(1 - \rho_G) R_N + \zeta_4(1 - \rho_N) R_N,
\end{aligned}$$

where $\tilde{\zeta}_1 = \zeta_1[1 - \tanh(b_1 \rho_I)]$ and $\tilde{\zeta}_2 = \zeta_2[\tanh(b_2 \rho_I)]$ are the protection parameters modulated by the overall communication, which is a function of $\rho_I = (I_P + I_N)/N$.

4.1.5 Reproduction numbers for homogeneous mixing

The basic reproduction number (R_0) quantifies the number of secondary infections generated by an infected individual during the course of the infectious period in the absence of any interventions [109, 110]. When interventions are implemented, a similar quantity can be defined as the control reproduction number (R_c). To obtain the reproduction numbers with and without protective measures, we first analysed the dynamics of the proportion of

susceptible individuals who are unprotected at time t , denoted by $\rho_N(t)$. We note that the layer influence flows downwards only; thus the cognition layer dynamics rely solely on the state of gossip spreaders in the top information layer and the number of unprotected and protected individuals in the middle cognition layer.

We applied the next-generation matrix (NGM) approach [111] to derive the expression for R_c . Denoting $I'_x = \mathcal{F}_x - \mathcal{V}_x$ such that $x = N$ or $x = P$, $\mathcal{F}_x$ contains all terms that involve the infection rate and $\mathcal{V}_x$ contains the other terms. Hence, we can write

$$\mathcal{F} = \langle k \rangle \begin{bmatrix} \beta_{NN} S_N \frac{I_N}{N} + \beta_{PN} S_N \frac{I_P}{N} \\ \beta_{NP} S_P \frac{I_N}{N} + \beta_{PP} S_P \frac{I_P}{N} \end{bmatrix},$$

and

$$\mathcal{V} = \begin{bmatrix} \mu I_N - \zeta_1 \rho_G I_P - \zeta_3 \rho_N I_P + \tilde{\zeta}_2 (1 - \rho_G) I_N + \zeta_4 (1 - \rho_N) I_N \\ \mu I_P + \tilde{\zeta}_1 \rho_G I_P + \zeta_3 \rho_N I_P - \tilde{\zeta}_2 (1 - \rho_G) I_N - \zeta_4 (1 - \rho_N) I_N \end{bmatrix}.$$

Defining $F = \frac{\partial \mathcal{F}_x}{\partial I_y}$ and $V = \frac{\partial \mathcal{V}_x}{\partial I_y}$, at the disease-free state we get:

$$F = \langle k \rangle \begin{bmatrix} \beta_{NN} \rho_N^* & \beta_{PN} \rho_N^* \\ \beta_{NP} (1 - \rho_N^*) & \beta_{PP} (1 - \rho_N^*) \end{bmatrix},$$

and

$$V = \begin{bmatrix} \mu + \zeta_4 (1 - \rho_N^*) & -\zeta_1 \rho_G - \zeta_3 \rho_N^* \\ -\zeta_4 (1 - \rho_N^*) & \mu + \zeta_1 \rho_G + \zeta_3 \rho_N^* \end{bmatrix},$$

where $\tilde{\zeta}_1 = \zeta_1$ and $\tilde{\zeta}_2 = 0$ at the disease-free state. Let $K = FV^{-1}$. For convenience, we

define

$$K = \frac{\langle k \rangle}{\det(V)} G, \quad (4.6)$$

where

$$\det(V) = \mu[\mu + \zeta_4 + \zeta_1\rho_G + (\zeta_3 - \zeta_4)\rho_N^*],$$

and the matrix elements of G are

$$\begin{aligned} g_{11} &= \rho_N^*[\beta_{NN}(\mu + \zeta_1\rho_G + \zeta_3\rho_N^*) + \beta_{PN}\zeta_4(1 - \rho_N^*)], \\ g_{12} &= \rho_N^*[\beta_{NN}(\zeta_1\rho_G + \zeta_3\rho_N^*) + \beta_{PN}(\mu + \zeta_4(1 - \rho_N^*))], \\ g_{21} &= (1 - \rho_N^*)[\beta_{NP}(\mu + \zeta_1\rho_G + \zeta_3\rho_N^*) + \beta_{PP}\zeta_4(1 - \rho_N^*)], \\ g_{22} &= (1 - \rho_N^*)[\beta_{NP}(\zeta_1\rho_G + \zeta_3\rho_N^*) + \beta_{PP}(\mu + \zeta_4(1 - \rho_N^*))]. \end{aligned}$$

Since ρ_N has a stationary state $\rho_N^* < 1$, which is asymptotically stable, the NGM approach can be used to calculate the control reproduction number (R_c) when protective measures are used. At the disease-free state when $\rho_N = \rho_N^*$, from the spectral radius (the largest eigenvalue modulus) of the matrix K , we obtain the expression:

$$R_c = \frac{\langle k \rangle}{\det(V)} \frac{\tau + \sqrt{\tau^2 - 4\Delta}}{2}, \quad (4.7)$$

where $\langle k \rangle$ is the average degree of the network, and

$$\tau = \text{Tr}(G) = g_{11} + g_{22},$$

$$\Delta = \det(G) = g_{11}g_{22} - g_{12}g_{21}.$$

4.1.6 Basic reproduction number

For a network system, the homogeneous mixing assumption should be modified in two aspects. First, the node degree should be explicitly considered in the heterogeneous mean-field (HMF) theory [112]. Second, the effective degree of infection should be reduced by 1, since the infected node has already been infected by one of its neighbours which cannot be reinfected in SIR dynamics [113]. The full HMF theory for the control reproduction number is beyond the scope of the present work; therefore we considered the basic reproduction number derived using the HMF theory [113, 114, 115, 19]:

$$R_0 = \frac{\beta_{\text{NN}}}{\mu} \cdot \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle}. \quad (4.8)$$

In the absence of any protective measures, the system without interventions reduces to

$$\begin{aligned} \frac{dS_N}{dt} &= -\beta_{\text{NN}} \langle k \rangle S_N \frac{I_N}{N}, \\ \frac{dI_N}{dt} &= \beta_{\text{NN}} \langle k \rangle S_N \frac{I_N}{N} - \mu I_N, \\ \frac{dR_N}{dt} &= \mu I_N, \end{aligned}$$

which results in the basic reproduction number of $R_0 = \beta_{\text{NN}} \langle k \rangle / \mu$. If the network is completely homogeneous [113], (4.8) reduces to $R_0 = \beta_{\text{NN}} (\langle k \rangle - 1) / \mu$. We note that the correction with respect to well-mixed theory, represented by $\langle k \rangle - 1$ replacing $\langle k \rangle$ in the R_0 expression, is corroborated by numerical simulations of the SIR on networks [114, 116].

In our model, the baseline transmission rate is $\beta_{\text{NN}} = 0.16$, assuming $R_0 = 4.0$ in the absence of protective measures, and calculated using (4.8) for given parameters of the network structure (Table 5.1). This reproduction number falls within the estimated range of 1.4–6.7 for the original strain of SARS-CoV-2 causing the COVID-19 pandemic [117, 118].

We assumed a recovery rate of $\mu = 0.2$ (corresponding to an average infectious period of 5 days), a network degree of $\langle k \rangle = 4.5$, a second moment of $\langle k^2 \rangle = 27$, and a power-law degree exponent of $\gamma_e = 4.0$. For the transmission rates with protective behaviours (Table 5.1), we note that their values may be low when considering some measures, such as mask-wearing, potentially overestimating their effect. However, for some interventions (e.g., isolation and quarantine), these values would be reasonable [119].

4.2 Experimental design

4.2.1 Parameterisation

For the structural parameters, each network layer is generated based on a degree sequence defined by the minimum degree ($k_{\min}$) and the power-law degree exponent (γ) (Table 5.1). The power-law exponents for the information, cognition, and epidemic layers are set to 2.5, 3.0, and 4.0, respectively, representing scale-free, critical point, and small-world networks (Table 5.1). The protection-free transmission rate (β_{NN}) (Table 5.1) is derived from (4.8), assuming $R_0 = 4.0$. The transmission rate under protection behaviour is defined as a fraction of β_{NN} to account for the mitigating effect of protection measures (Table 5.1). We used plausible values for rate parameters pertaining to information layer dynamics (Table 5.1). For example, the renewal interest rate (ω) ranges from 0 to 2, capturing the rapid turnover of misinformation on timescales from days to hours.

4.2.2 Computational algorithm

We employed the optimised Gillespie algorithm to simulate distinct events in continuous time [106, 120]. This method accommodates variable and random time steps, where the time (τ) until the next event follows an exponential distribution, given by $\tau = -\ln(u)/L$,

with u drawn from a uniform distribution on $(0,1]$ and L representing the total rate of all events, $L = \sum_j r_j$. The probability of transitioning to each event state is determined by the ratio of its rate to the total rates, expressed as

$$p_j = \frac{r_j}{\sum_j r_j}.$$

During each iteration, a random time increment τ is first obtained. Subsequently, the next event is chosen by comparing its cumulative rate fraction against a uniformly drawn number between 0 and 1. The algorithm then updates the network's node states and advances time to $t + \tau$. Because time intervals are exponentially distributed and event probabilities are proportional to their rates, the Gillespie algorithm ensures exactness in simulation, effectively capturing the current network states.

An important idea in the optimised Gillespie algorithm is considering the “phantom process” where the system state does not change but the time advances [106]. This approach is especially useful in simulating continuous-time epidemic spreading on large heterogeneous networks [106].

Chapter 5

Interplay Between Information Dissemination and Behavioural Responses

In this chapter, we assess the influence of disinformation on disease transmission, particularly in terms of immediate and reversible measures like mask-wearing and social distancing. To study short-term solutions, we use the three-layer ICE modelling framework introduced in Chapter 4, encompassing information, cognition, and epidemics. We investigate the characteristics of misinformation transmission within hyper-edge structures and approaches for reducing its spread. In addition, we identify the short-term consequences of misinformation and protective behaviour and develop ways to optimally restrict disease spread.

First, we extend our analysis on the theoretical derivation of the control reproduction number (R_c) and investigate the effects of protection adoption and removal parameters ($\zeta_1, \zeta_2, \zeta_3, \zeta_4$). Second, we focus solely on the information-spreading layer, comparing

rumour-spreading outcomes for hyper-edge versus pairwise network architectures. We further investigate the impact of gossip acceptance criteria in both structures. We then conduct a quick examination of the misinformation propagation dynamics. Next, we assess the protective adoption variables for their impact on attack rates. Finally, we compare several misinformation correction scenarios, centring on how rumour interest renewal rate, relapse rate, and correction rate influence attack rate.

5.1 Results

5.1.1 Effect of protective behaviours on R_c

The control reproduction number R_c quantifies the impact of public health interventions on disease dynamics, such as reduced transmission rates or behavioural adjustments. We simulated changes in R_c (given by (4.7)) under the homogenous mixing approximation resulting from varying protection adoption rates, influenced by neighbourhood interactions or virtual information exchanges. We observed a rapid increase in R_c as a function of the removal rate of neighbourhood-based protection ζ_3 (Figure 5.1A), which depends on the density of unprotected individuals (ρ_N). Conversely, a rapid decline in R_c occurs when the rate of protection adoption ζ_4 increases (Figure 5.1B). The analysis of R_c indicates that the attack rate is sensitive to ζ_1 only under a high density of gossip spreaders ρ_G , as illustrated in Figure 5.1C.

5.1.2 Hyper-edge misinformation spread

Spreading phenomena through group interactions can be characterized by the sudden rise of prevalence as compared with the pairwise interactions [49]. We verified this by analyzing the rumour spreading in the information layer with either hyper-edge or

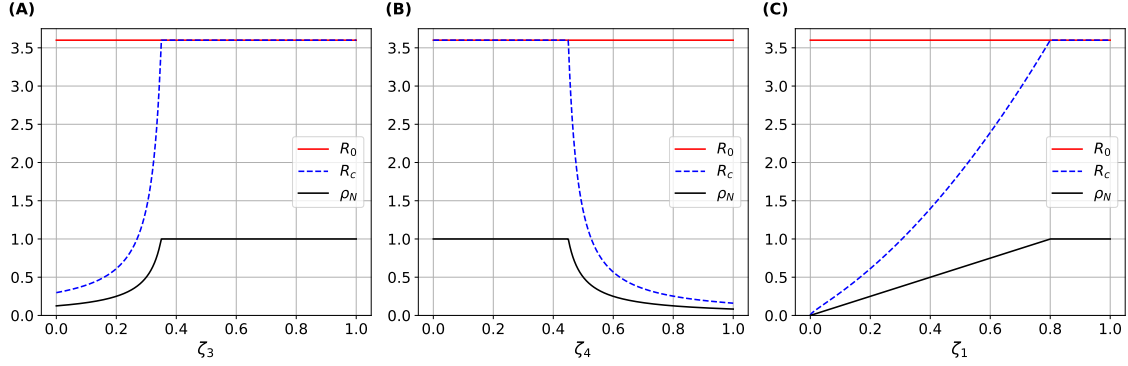


Figure 5.1: Control reproduction number dependence on information and neighbourhood behavioural parameters under the homogeneous mixing approach. Panels (A) and (B) have the same density of gossip spreaders $\rho_G = 0.1$, the same level of information-based protection removal rate $\zeta_1 = 0.5$, with fixed $\zeta_4 = 0.4$ and $\zeta_3 = 0.4$, respectively. For panel (C), the removal and adoption rates of neighbourhood-based protection are $\zeta_3 = 0.2$ and $\zeta_4 = 0.4$, and the density of gossip spreaders is $\rho_G = 0.25$. Other parameters are provided in Table 5.1.

degree distributions following power-laws with exponent $\gamma_i = 2.5$ (scale-free). At various information thresholds (ϵ) for a single rumour ($\omega = 0$) and no correction ($\sigma = 0$), we assessed stifter density at the end of information spreading (when number of spreaders is zero) as a function of the misinformation spreading rate (λ) for pairwise and group-wise scenarios (Figure 5.2). Note that misinformation continues to spread according to the rules of virtual contact until the number of gossip spreaders diminishes to zero due to the stifling effect and waning interest. In the group-wise scenario, the stifter density grows rapidly towards a high density steady-state as the spreading rate increases from $\lambda = 0$ to $\lambda = 0.25$, contrasting a comparatively more gradual increase for the pairwise spreading, corroborating the importance of considering group interactions in modelling information spreading.

Incorporating a threshold for misinformation acceptance is essential, as it dictates the point at which an individual becomes influenced and begins spreading a rumour. A higher threshold indicates greater resistance to misinformation, reducing the likelihood of

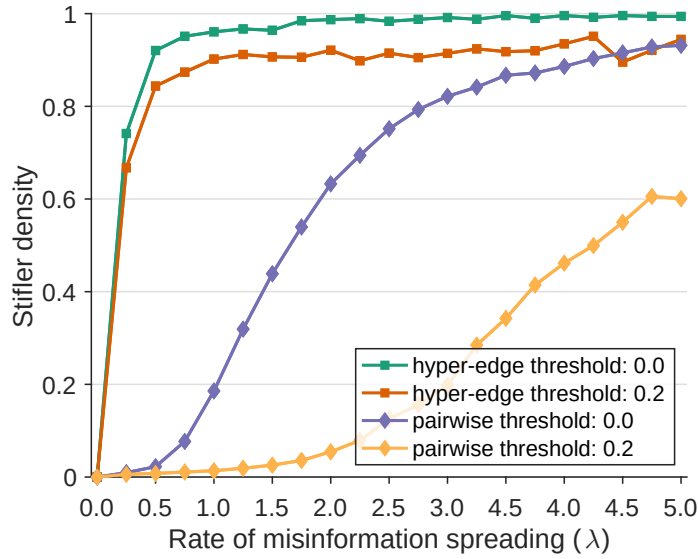


Figure 5.2: Stifler density with either pairwise or hyper-edge misinformation spreading structure when a single rumour vanishes due to the stifling process without correction interventions. The degree exponent is $\gamma_i = 2.5$, which represents a scale-free network. A single rumour ($\omega = 0$) and no correction ($\sigma = 0$) are considered with $\lambda \in [0, 5]$ and a stifling rate of $\alpha = 1$. Other parameters are provided in Table 5.1.

rumour spread. From a modelling perspective, including a threshold—rather than allowing automatic gossip transmission—can result in a slightly lower final number of stiflers and a reduced impact on both the cognitive and epidemic layers.

It should be noted that a single rumour has minimal impact on shaping disease-related behaviours because rumours tend to spread rapidly before the disease affects a significant portion of the population. However, when renewed interest in spreading gossip is considered, bursts of misinformation activity emerge, amplifying the impact on behavioural responses. To underscore the importance of interest renewal in gossip spreading, we examined how the dynamics of the information layer vary with different rumour interest renewal rates (Figure 5.3). Three scenarios were considered, with correction occurring on average every 20 days ($\sigma = 0.05$): (1) a single rumour, (2) a new rumour every 5 days, and (3) a new

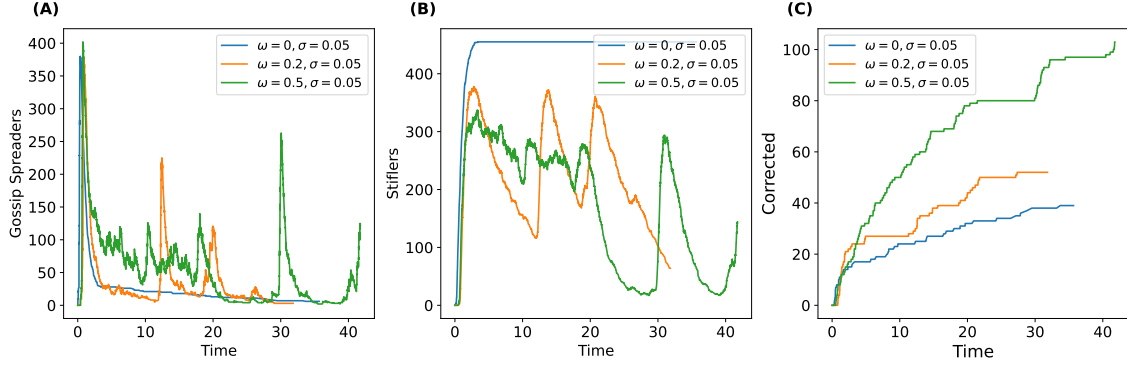


Figure 5.3: Trajectory of gossip spreaders, stiflers, and corrected individuals over time with different values of ω . Other parameters are provided in Table 5.1 with $\lambda = 1/3$, $\alpha = 1/3$, $\zeta_1 = \zeta_2 = 0.2$, and $\zeta_3 = \zeta_4 = 0$. Scenarios correspond to a single rumour ($\omega = 0$), a new rumour every 5 days ($\omega = 0.2$), and a new rumour every 2 days ($\omega = 0.5$).

rumour every 2 days. We observed oscillatory behaviours in gossip spreaders and stiflers in scenarios (2) and (3).

5.1.3 Information-cognition layers coupling

We systematically varied rates for adopting protection based on information (ζ_2) and neighbourhood behaviour (ζ_4) to assess their impact on disease spread. For instance, when $\zeta_2 = 0.2$ or $\zeta_4 = 0.2$, it indicates that individuals transition from an unprotected state to a protected state within an average span of 5 days in an ideal scenario without gossipers ($\rho_G = 0$), perfect global information ($b_2 = \infty$), and full protection perception in the neighbourhood. Deviations from the perfect scenario increase the time to adopt protection.

5.1.3.1 Heterogeneous versus homogeneous networks

With a power-law degree exponent of $\gamma_e = 4.0$, the attack rate, when neither information- nor neighbourhood-based protection is applied ($\zeta_2 = \zeta_4 = 0$), is approximately 20%

Table 5.1: Description of the model parameters and their values used for simulations for the ICE model. The average node degree of a network is $\langle k \rangle = \frac{\gamma-1}{\gamma-2} \cdot k_{\min}$. For example, the average degree for the epidemic network is $\langle k \rangle = \frac{4-1}{4-2} \cdot 3 = 4.5$. All rates are per day.

Symbol	Description	Value
R_0	Basic reproduction number	4
β_{NN}	Transmission rate with no protection	0.16
β_{PN}	Transmission rate with protection behaviour of only susceptible individuals	$0.2\beta_{\text{NN}}$
β_{NP}	Transmission rate with protection behaviour of only infected individuals	$0.1\beta_{\text{NN}}$
β_{PP}	Transmission rate with protection behaviour of both susceptible and infected individuals	0.0
μ	Recovery rate	0.2
λ	Rate of misinformation spreading	Varied
σ	Correction rate	Varied
α	Rate of misinformation suppression	Varied
ϕ	Relapse rate	Varied
ω	Rate of renewal interest	Varied
ζ_1	Rate of protection removal by information	Varied
ζ_2	Rate of protection adoption by information	Varied
ζ_3	Rate of protection removal by mimicking neighborhood behaviour	Varied
ζ_4	Rate of protection adoption by mimicking neighborhood behaviour	Varied
$k_{\min}$	Minimum network degree	3
γ_i	Power-law degree exponent in the information layer	2.5
γ_c	Power-law degree exponent in the cognition layer	3.0
γ_e	Power-law degree exponent in the epidemic layer	4.0
n	Number of nodes in the network	500
$n_{\text{hyperedge}}$	Number of hyper-edges in the information layer	450
b_1, b_2	Global communication factors	3
ρ_{P}	Initial proportion of population protected	0.05

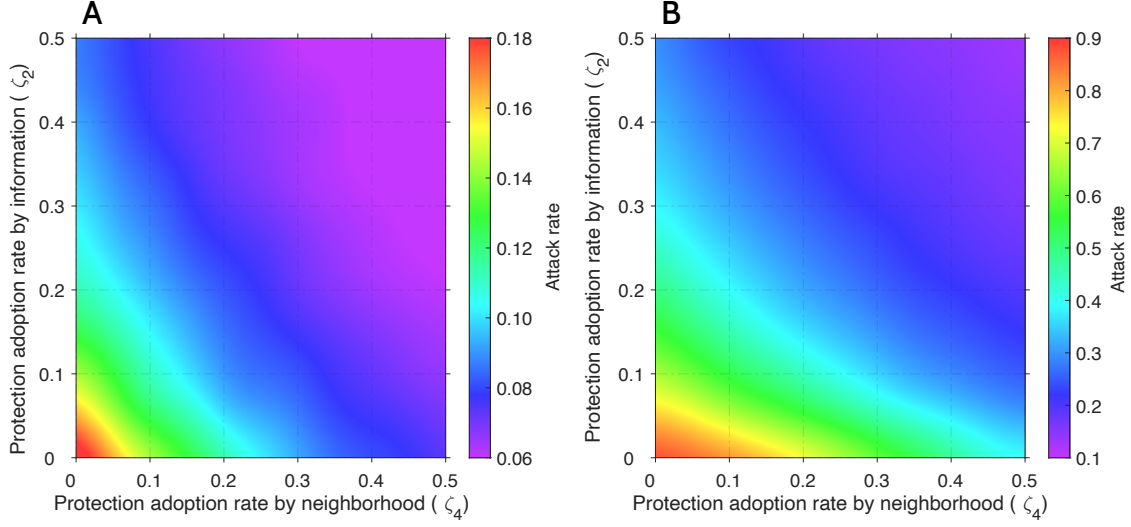


Figure 5.4: Attack rate as a function of the rates of adopting protective measures based on information or neighbourhood behaviour in (A) a heterogeneous network and (B) a homogeneous network with $\langle k \rangle = 4.5$ and $\beta_{NN} = 0.228$. Other parameters are given in Table 5.1, with the misinformation spreading rate of $\lambda = 1/3$, stifling rate of $\alpha = 1/3$, gossip interest renewal rate of $\omega = 1/5$, and correction rate of $\sigma = 0$. The rates of withdrawing from protective behaviours are $\zeta_1 = 0.2$ in the information layer and $\zeta_3 = 0$ in the cognition layer.

(Figure 5.4). When the protection rate through information dissemination is increased to $\zeta_2 = 0.5$, the attack rate decreases to approximately 9%. When the protection rate by following neighbourhood behaviour is increased to $\zeta_4 = 0.5$, the attack rate further decreases to about 7%. Increasing both protection rates to 0.5 reduces the attack rate to approximately 5% (Figure 5.4). These results suggest that enhancing protection mechanisms at the neighbourhood level to influence individual behaviour is slightly more effective in controlling disease spread compared to interfering with disease-related misinformation, while both strategies are important and complementary.

To highlight the importance of a heterogeneous network in disease spread, we also show the outcome for a homogeneous network with the same average degree and basic

reproduction number $R_0 = 4.0$. For a homogeneous network, with neither information- nor neighbourhood-based protection adoption, the attack rate is over 91% (Figure 5.4B). For the neighbourhood-based protection adoption rate of $\zeta_4 = 0.5$, the attack rate drops to about 41% (Figure 5.4B). With the information-based protection adoption rate of $\zeta_2 = 0.5$, the attack rate is further reduced to approximately 30%. The higher attack rate on homogeneous networks, compared to heterogeneous networks with the same reproduction number, can be explained by the significant degree fluctuations of the latter [113]. In the heterogeneous case, an epidemic spreads rapidly across nodes with a degree larger than $\langle k \rangle$, which governs the basic reproduction number. However, the spread decelerates among nodes with a degree smaller than $\langle k \rangle$, which are more abundant but less capable of transmitting the infection to their neighbouring nodes. In the real-world situation, homogeneous assumptions fail to capture the diversity in contact patterns [112].

5.1.3.2 Heterogeneous network with high transmission rates

Matching the transmission rates in the heterogeneous power-law network with those in the homogeneous network with $\zeta_2 = \zeta_4 = 0$, the attack rate was approximately 46% (Figure 5.5A). When protection adoption is made within a week (i.e., $\zeta_2 = 0.15$) based on information, the attack rate decreased to 21%. When $\zeta_4 = 0.15$, the attack reduced to 24.39%. (Figure 5.5A). We observed a lower protection-adoption-free attack rate in the heterogeneous with the same transmission rate compared to the homogeneous network, although the reproduction number was higher.

Now, considering a higher infection rate $\beta_{NN} = 0.4$ ($R_0 = 10$), when $\zeta_2 = \zeta_4 = 0$, the attack rate is about 84% (Figure 5.5B). Setting $\zeta_2 = 0.15$ for protection adoption, the attack rate reduces 42.18%. However, the reduction in attack rate was significantly lower for $\zeta_4 = 0.15$, decreasing to 69%. These results suggest that protection adoption through

information dissemination is considerably more effective in controlling disease spread compared to protection conferred by mimicking the neighbourhood behaviour.

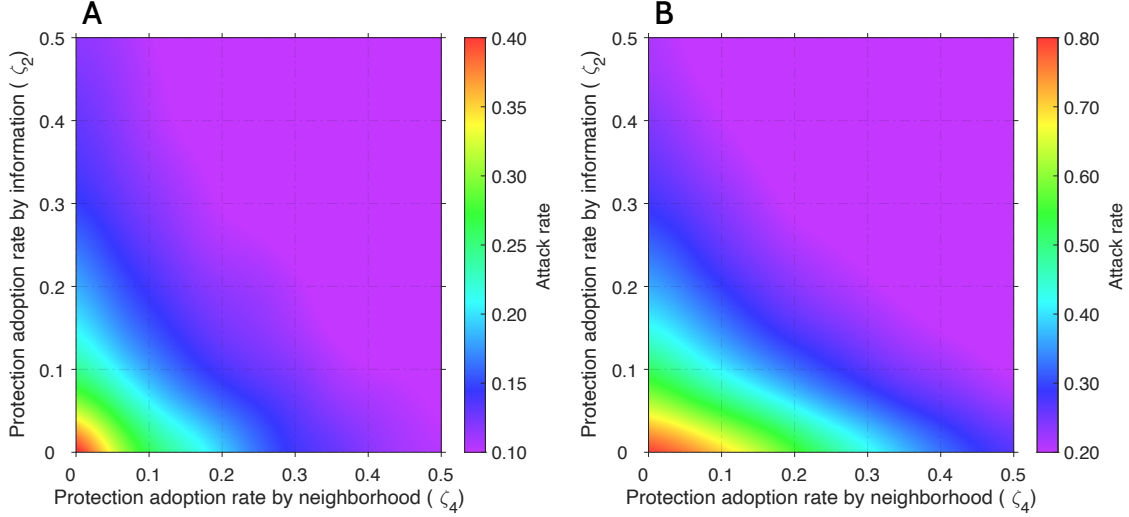


Figure 5.5: Attack rate as a function of information-based and neighbourhood-based rates of adopting protective behaviours. In panel (A), a power-law network matches $\beta_{NN} = 0.228$ for the homogeneous network shown in Figure 5.4, with $R_0 = 5.7$. In panel (B), a power-law network has a high transmission rate $\beta_{NN} = 0.4$, with $R_0 = 10$. Other parameters are provided in Table 5.1, with the misinformation spreading rate of $\lambda = 1/3$, stifling rate of $\alpha = 1/3$, gossip interest renewal rate of $\omega = 1/5$, correction rate of $\sigma = 0$, and rates of $\zeta_1 = 0.2$ and $\zeta_3 = 0$ for withdrawing from protective behaviours.

At higher attack rates in homogeneous networks, as shown in Figure 5.4B, the protection conferred by information is more effective than that triggered by the cognitive perception of the neighbourhood, indicating that controlling misinformation is essential to mitigate an outbreak effectively. Considering heterogeneous networks, we observed a similar pattern for the effect of adoption rates in reducing attack rates with different transmission rates matching those of a homogeneous network (Figure 5.5).

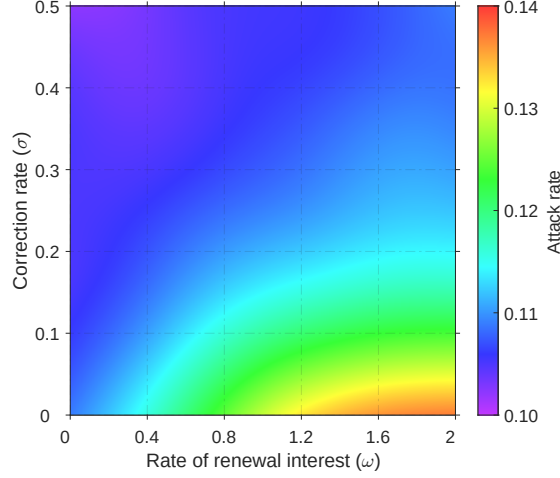


Figure 5.6: Attack rate as a function of the renewal interest rate (ω) and misinformation correction rate (σ). Other parameters are provided in Table 5.1 with the misinformation spreading rate of $\lambda = 1/3$, and stifling rate of $\alpha = 1/3$. The rate of withdrawing from protective behaviours in the cognition layer is $\zeta_1 = 0.2$, and the rate of adopting protective behaviours is $\zeta_2 = 0.2$.

5.1.4 Correcting misinformation

5.1.4.1 Baseline scenario

We evaluated the effect of correcting misinformation to mitigate its negative impact on disease spread while varying the renewal interest rate (ω) of becoming susceptible to gossip spreading. We found that the effect of increasing ω saturates rapidly with minimal variation on the attack rate in the range of 10%–14% in the absence of correction ($\sigma = 0$). Not surprisingly, a higher rate of correction is needed to offset the effect of increased ω on the attack rate (Figure 5.6). For example, if the misinformation is never corrected ($\sigma = 0.0$), the mean attack rate would be approximately 14%, whereas correction within 5 days (i.e., $\sigma = 0.2$) reduces the attack rate to 11%. In the context of our multi-layer model, the spreading process is governed by higher-order interactions. Thus, a single rumour

cannot significantly alter the epidemic course, as the behavioural effects dissipate before a substantial spread of disease. Consequently, our analysis focuses on the renewal rate (ω) as a key parameter.

5.1.4.2 Secondary analyses

We investigated the two additional correction mechanisms following the correction rules outlined in Section 4.1.1.

Suspension of individuals from the information network. We note that gossip spreaders can be banned or corrected in a blog-type network, both of which can be temporary. In the absence of such correction mechanisms ($\sigma = 0$), the attack rate in our simulations ranged from 11% to 14% when the renewal interest rate varied from 0 to 2 (Figure 5.7A). The effect of correction on the attack rate through targeting individual gossip-spreader nodes in the network is more gradual compared to the correction by group contact. For example, at a relatively high rate of gossip renewal ($\omega = 1.4$), rapid correction efforts within 3 days ($\sigma \simeq 0.33$) would be needed to reduce the attack rate from approximately 14% to 12%. Fixing the renewal rate at $\omega = 1.4$, our simulations indicate that the relapse rate (ϕ) has a minimal effect on reducing the attack rate for a given correction rate (Figure 5.7C). This suggest that, even under prolonged suspension of individuals from the information network (corresponding to small values for ϕ), the renewal interest can have a more pronounced impact, providing a conduit for continual spread of misinformation.

Warning and educating individuals within the information network. The effect of correcting peers in the network is more pronounced compared to banning gossip spreaders. For example, with the renewal rate of $\omega = 1.4$, reducing the attack rate from nearly 14% to 12% can be achieved with an over two-fold smaller correction rate of $\sigma = 0.15$ compared to the scenario with suspension of individuals from the information network (Figure 5.7B).

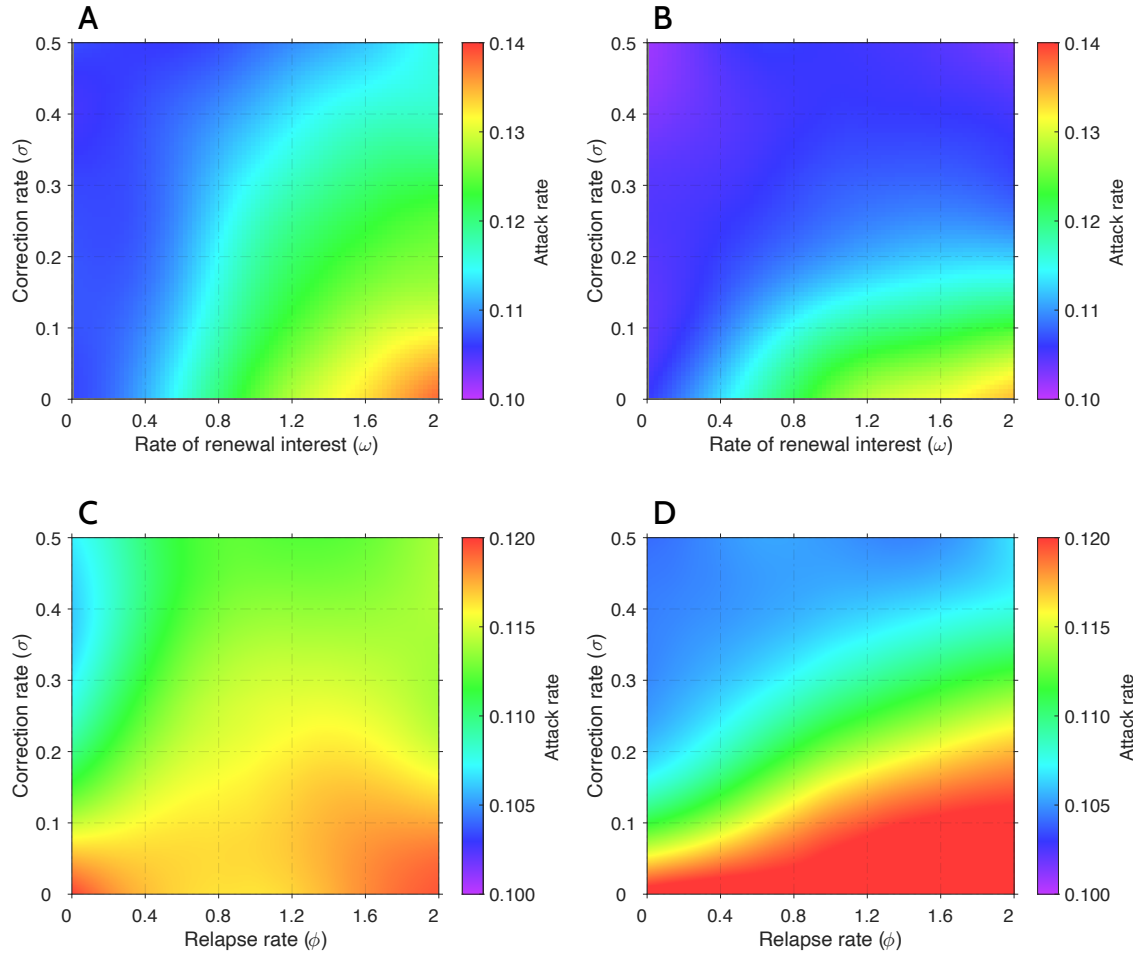


Figure 5.7: Attack rate as a function of the renewal interest rate (ω) and correction rate of misinformation (σ) with two types of correction strategies: (A) suspension of individuals from the information network, and (B) warning and/or educating individuals within the information network. The relapse rate in (A) and (B) is set to $\phi = 0.2$. Fixing $\omega = 1.4$, panels (C) and (D) show the attack rate as a function of the relapse rate (ϕ) and the correction rate (σ) for the two correction strategies, respectively. In all scenarios, the spreading rate of misinformation is $\lambda = 1/3$, stifling rate is $\alpha = 1/3$, the rate of withdrawing from protective behaviours in the cognition layer is $\zeta_1 = 0.2$, and the rate of adopting protective behaviours is $\zeta_2 = 0.2$. Other parameters are provided in Table 5.1.

The patterns in this group-based warning strategy with relapse ($\phi = 0.20$) are similar to those observed for correction without relapse ($\phi = 0$) in Figure 5.6.

Similar to the strategy of suspending individuals from the information network, we found that the relapse rate has a minimal impact on the overall attack rate with a fixed renewal interest rate (Figure 5.7D). However, achieving even a small reduction in attack rate for a given relapse rate would require a substantial increase in the correction rate. For example, with $\phi = 1.6$ and $\omega = 1.4$, the overall attack rate is approximately 12% at a correction rate of $\sigma = 0.1$. Reducing the attack to 10.5% requires at least a 5-fold increase in the correction rate (Figure 5.7D).

For the computational execution of the secondary analysis, we treat the two intervention scenarios differently. For the suspension intervention, we automatically shift people from gossip spreaders to corrected status based on a specified suspension rate. For the warning and education intervention, we allow interactions between gossip spreaders and corrected individuals via hyper-edge group links, permitting state transitions from gossip spreader to corrected status upon contact with corrected persons.

Chapter 6

Heterogeneity in Information Diffusion

Complex networks are well-suited to capturing contact heterogeneity, making them powerful tools for modelling both physical and virtual connections, such as disease transmission, information dissemination, and behavioural patterns. While prior research has investigated how information flow affect epidemic spread, the interdependent dynamics among information diffusion, behavioural responses, and disease transmission remain insufficiently understood.

This chapter begins with a concise overview of key concepts in higher-order network structures, including the construction of hyper-edge groups and the distinction between simplex-based and hyper-edge-based representations. We illustrate how group-level interactions diverge from traditional pairwise models, particularly in the context of information propagation and behavioural contagion.

Building on this foundation, we advance the analysis of the ICE model framework to to explore how network heterogeneity influences information diffusion, protection adoption, and subsequent disease transmission. We focus on the structure of higher-order interactions within the information layer and their behavioural implications. Our findings reveal that

misinformation originating from low-degree nodes leads to only slightly higher level of protective adoption, and the topology of hyper-edge groups exerts a stronger influence than the initial source of gossip. Notably, heterogeneous group structures accelerate the spread of misinformation from smaller to larger groups, amplifying its impact.

We further demonstrate that scale-free information networks sustain recurrent and long-lasting misinformation cycles, in contrast to the short-lived peaks observed in small-world networks—patterns that significantly influence epidemic trajectories. Finally, we find that knowledge-based decision-making consistently outperforms imitative behaviour, especially in networks where hyper-edge group sizes follow a power-law distribution, reinforcing the importance of cognitive-driven strategies in epidemic control.

6.1 Introduction

The spread of COVID-19 and the proliferation of disinformation highlight the critical importance of understanding the mechanisms behind the transmission of diseases, information, and behaviours [52]. Higher-order networks offer fresh perspectives and uncover unique spreading dynamics by capturing interactions beyond simple pairwise connections [52]. These networks often share underlying mathematical patterns and physical principles [52], bridging diverse disciplines and revealing shared characteristics. This growing topic connects many fields and uncovers commonalities. Advances in higher-order dynamics contribute significantly to our understanding of complex systems and their behaviour.

Hypergraphs are a prime example of higher-order networks, extending beyond traditional pairwise interactions to represent group-level dynamics [121]. These models offer a more nuanced view of complex systems, enabling the study of collective behaviours in social, biological, and artificial contexts. They are particularly well-suited to modelling phenomena such as group-based spreading processes and collective responses involving

multiple individuals.

Research in higher-order networks has explored their relevance in scenarios characterised by close-contact interactions, such as households, workplaces, and other environments where people frequently converge [47]. These studies have informed policy decisions on epidemic control, including vaccination strategies and lockdown measures [47]. Beyond epidemic dynamics, investigations have focused on understanding network organisation and architecture. For example, studies of complex systems like neural or transportation networks have revealed insights into clustering and connectivity patterns [122].

The heterogeneity of networks significantly affects models of epidemics and information diffusion. In particular, scale-free networks, characterised by highly variable node connectivity, can amplify outbreaks even in the case of diseases with low transmissibility [123]. Highly connected nodes, or hubs, play a critical role in such dynamics, emphasizing the importance of targeted interventions such as vaccinating these nodes or mitigating their role in spreading misinformation [123]. Understanding the heterogeneous topology of networks is thus crucial for managing the spread of diseases and misinformation in complex biological and social systems.

Despite the significance of heterogeneity in network dynamics, its incorporation into high-order network models remain underexplored [124]. Recent studies highlight the importance of heterogeneity within group structures for effective disease control [125], and demonstrate that accounting for information heterogeneity in networks can enhance prevention strategies [126].

This study aims to address some of the knowledge gaps concerning heterogeneity in higher-order networks, focusing on the role of information diffusion and its implications for behaviour and disease propagation. Specifically, we investigate how factors such as the placement of spreading seeds, variations in network topology, and the distribution of hyper-edge group sizes influence propagation dynamics across interconnected layers of

information, behaviour, and disease.

6.2 Methods

6.2.1 Higher order graphs

The term “higher-order dynamics” refers to group-based, synchronous, non-linear interactions that are fundamentally different from simple pairwise interactions [127]. In network studies, this entails using hyper-edges to model more complex connections between groups of different sizes, in addition to the usual pairwise links. Many real-world systems, such as information dissemination on social media or viral transmission, rely substantially on group interactions [127]. Higher-order dynamics may yield phenomena that are not present in simpler models, such as sudden activation and hybrid transitions [128, 129].

In this work, we propose an approach to represent spreading processes ranging from pairwise interactions to higher-order networks, using a modified bipartite configuration model [130] to build networks with different node degrees and group size distributions. In Figure 6.1, given a node degree distribution and a hyper-edge group size distribution, we pair the half-edges to form hyper-edge groups. Within each hyper-edge group, information spreads simultaneously among all members, in contrast to traditional one-to-one information sharing [128].

Rumour propagation and information dissemination often occur on a large scale within groups [131]. Similarly, gatherings and group settings such as households, schools, and workplaces play a significant role in the spread of epidemics [132, 133]. Traditional networks depend only on connections between pairs of agents. This limits group interactions to a basic collection of paired linkages. This approach fails to capture the complexity of systems in which interactions increase non-linearly with the number of participating nodes.

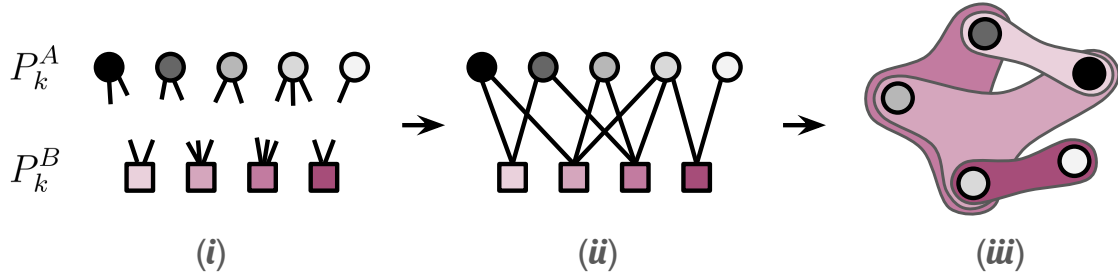


Figure 6.1: A diagram illustrating the process of forming hyper-edge groups. Initially, we define a node degree distribution and a group size distribution. Next, half-edges (or stubs) are connected. Finally, each hyper edge is depicted as a bubble where all nodes are interconnected.

To allow for capacity to effectively predict collective behaviours, group-based structures are needed.

To highlight the difference between pairwise links and group-based interactions, we provide a schematic comparison in Figure 6.2. Here, we show that a traditional simplex represents a fully connected sub-graph or clique, with all pairs of nodes connected, thereby facilitating pairwise interactions. Both hyper-edges and simplicial complex structures spread through collective group setting and simultaneous interactions. However, a simplicial complex with 4 nodes necessarily includes all possible structures of 3 or 2 nodes, while hyper-edge structures do not have these restrictions. The characteristics and behaviours of a hyper-edge group should be analysed as a whole, rather than broken down into individual links and nodes.

Table 6.1: Description of the model parameters and their values used for simulations for the heterogeneity analysis in information diffusion. All rates are per day.

Symbol	Description	Value
R_0	Basic reproduction number	4
β_{NN}	Transmission rate with no protection	0.16
β_{PN}	Transmission rate with protection behaviour of only susceptible individuals	$0.2\beta_{\text{NN}}$
β_{NP}	Transmission rate with protection behaviour of only infected individuals	$0.1\beta_{\text{NN}}$
β_{PP}	Transmission rate with protection behaviour of both susceptible and infected individuals	0.0
μ	Recovery rate	0.2
λ	Rate of misinformation spreading	1/3
σ	Correction rate	0
α	Rate of misinformation suppression	1/3
ϕ	Relapse rate	0
ω	Rate of renewal interest	1/5
ζ_1	Rate of protection removal by information	0.2
ζ_2	Rate of protection adoption by information	Varied
ζ_3	Rate of protection removal by mimicking neighborhood behaviour	0
ζ_4	Rate of protection adoption by mimicking neighborhood behaviour	Varied
k_{min}	Minimum network degree	3
γ_i	Power-law degree exponent in the information layer	2.5
γ_c	Power-law degree exponent in the cognition layer	3.0
γ_e	Power-law degree exponent in the epidemic layer	4.0
n	Number of nodes in the network	500
$n_{\text{hyperedge}}$	Number of hyper-edges in the information layer	450
b_1, b_2	Global communication factors	3
ρ_{P}	Initial proportion of population protected	0.05

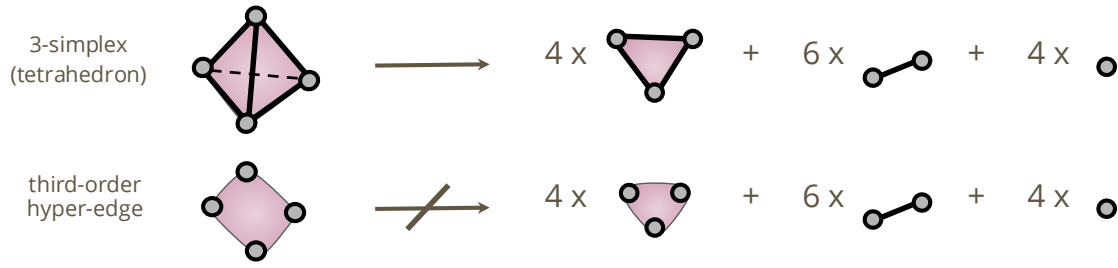


Figure 6.2: A diagram comparing the traditional simplex model and our new hyper-edge representation. In both models, all nodes within a group are connected. However, the hyper-edge model allows for the partial connectivity within a selected set of nodes, while the simplex model mandates complete connectivity between all nodes in the group.

6.3 Results

6.3.1 Protection adoption with different gossip seeds

An important metric in studying behaviour diffusion in social networks is the temporal dynamics of its spread [7]. Figure 6.3 shows the effects of varying gossip-spreading sources on long-term protection adoption. Specifically, we compared scenarios where gossip spreading begins from the 10 highest-degree nodes in the information layer to those initiated from the 10 lowest-degree nodes. Around 40 days into the spreading process, we observe a relatively stable level of protection. Interestingly, gossip spreading from lower-degree sources results in slightly higher long-term protection compared to that from higher-degree sources. The influence of gossip sources appears minimal, likely due to the hyper-edge network topology. In such networks, groups are densely interconnected, allowing gossip originating in smaller groups to quickly reach larger ones, thereby facilitating widespread propagation.

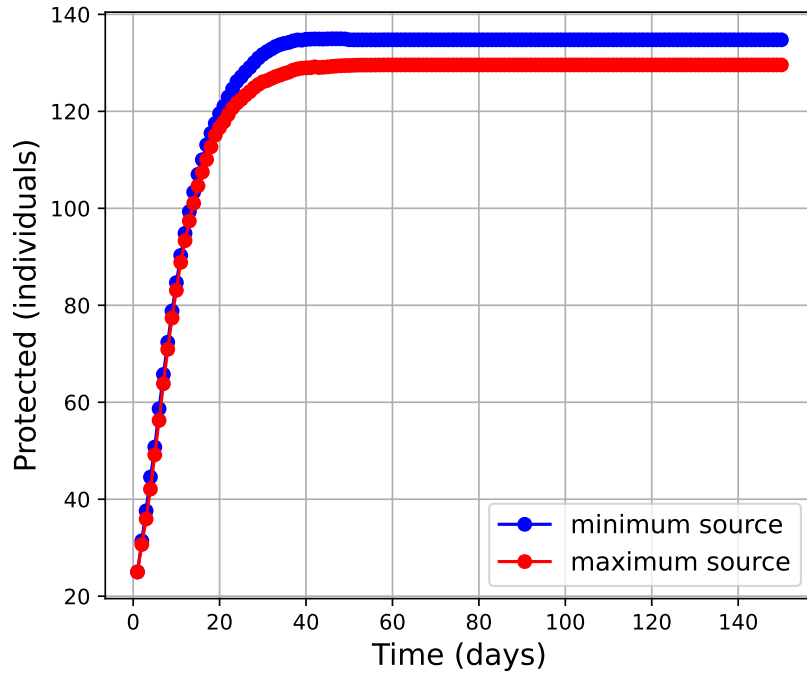


Figure 6.3: Protection adoption levels over time with different misinformation seeds scenarios, averaged over 100 realisations. Protection shift parameters are set to $\zeta_1 = \zeta_2 = 0.2$ and $\zeta_3 = \zeta_4 = 0$. Other parameters are provided in Table 5.1.

6.3.2 Heterogeneity on the information layer

We observed that the scale-free structure of information diffusion generates periodic peaks in gossip spreading (Figure 6.4). While similar peaks can also occur in small-world networks, the effect is not sustained. To better capture the persistence influence of misinformation over time and more accurately reflect its impact on disease prevalence in real-world scenarios, higher heterogeneity—characterised by a scale-free network with a low γ_i value—is preferred.

The densely connected core formed by numerous superspreaders in scale-free networks leads to multiple bursts of misinformation spread, ensuring its ongoing influence on disease dynamics. In contrast, small-world networks feature fewer, sparsely distributed hubs, making repeated bursts from interconnected hubs unsustainable. The impact of misinformation on the outbreak size is evident in Figure 6.4(B), where the outbreak duration extends to 80 days in a scale-free information layer compared to approximately 40 days in a small-world network. This finding, tied to the heterogeneity of higher-order interactions—often underestimated in the literature—raises significant concerns, particularly since real-world information networks tend to follow a scale-free structure [134, 135].

6.3.3 Hyper-edge group size distribution

We also investigated the impact of hyper-edge group size distribution—another feature that has received limited attention in the literature—on disease spread within the hyper-edge spreading information layer setup. Figure 6.5 highlights the critical role of group size distribution. Although the average group size remains consistent across scenarios, the dynamics differ significantly based on the distribution type. In panel (A), where the hyper-edge sizes follow a uniform distribution, making small or large groups equally likely, protection behaviour through neighbour imitation proves more effective. In contrast, panel

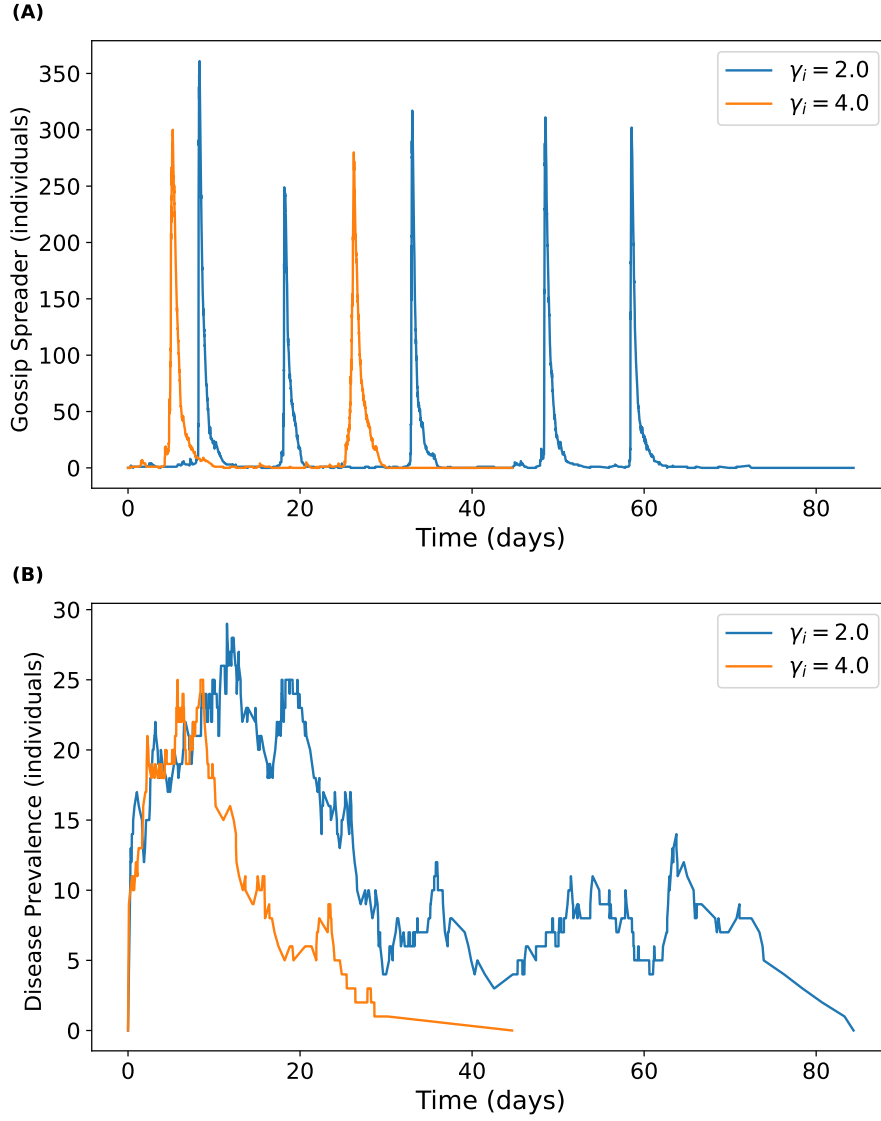


Figure 6.4: Comparison of dynamics across layers: (A) gossip spreader over time, and (B) disease prevalence over time in a small-world network structure ($\gamma_i = 4$) versus a scale-free network structure ($\gamma_i = 2$) on the information layer. The cognition layer is modelled as a critical point network ($\gamma_c = 3$), and the epidemic layer follows a small-world network structure ($\gamma_e = 4.5$). The protection adoption rates from information and neighbourhood mimicking behaviour are set to $\zeta_2 = \zeta_4 = 0$. Other parameters are provided in Table 5.1.

(B) shows that when the hyper-edge sizes follow a power-law distribution, with smaller groups smaller coexisting alongside larger ones, both protection approaches have similar effect while the adoption of protection via the information layer becomes slightly more effective in reducing the overall attack rate. This finding underscores the importance of accounting for group size heterogeneity in modelling and mitigating disease spread.

In general, incorporating a hyper-graph structure with smaller groups amplifies the effectiveness of misinformation control in disease propagation. This outcome highlights the critical role of information dissemination within small groups, where tight-knit interactions can accelerate behaviour adoption. Such structures enhance the potential for protective measures to propagate effectively, demonstrating the significant influence of group size heterogeneity on managing outbreaks and controlling misinformation.

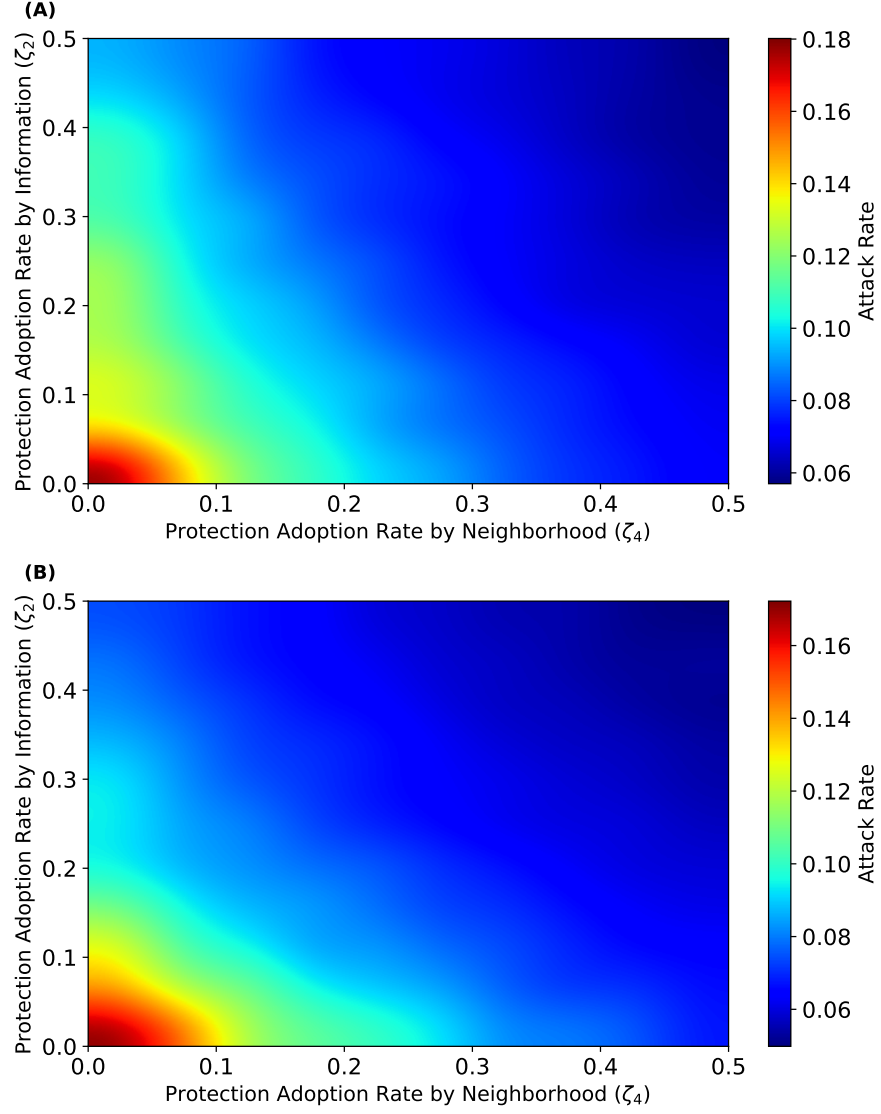


Figure 6.5: Attack rates for different hyper-edge group size distributions: (A) uniform distribution, and (B) power-law distribution ($\gamma = 3.0$). Other parameters are provided in Table 5.1.

Chapter 7

Impact of Information Diffusion on Vaccine Uptake

In this chapter, we examine how misinformation control affects vaccination coverage using an extended ICE network model that combines hyper-edge threshold spread on the disinformation layer with typical pairwise spread on both cognition and epidemic layers. We investigate how misinformation correction affects overall vaccination uptake.

We investigate the negative delay effect of vaccination. We consider two preventative vaccination program cases: (i) vaccinate prior to the onset of gossip and disease; (ii) vaccinate and spread gossip prior to the beginning of disease spread. Our aim is to quantify how preventative vaccination can counteract the negative effects of continuous rumour influx. Furthermore, we compare gossip mitigation techniques that target hubs (high-degree nodes) to those that do not account for degree heterogeneity. This comparison of vaccination strategies is conducted with both a baseline model and an extended model incorporating a severe symptoms compartment.

Our analysis focuses on examining the extended model framework incorporating vacci-

nation from the perspectives of gossip correction, timing and delay, and network heterogeneity. This approach aims to provide strategic guidance for misinformation control and vaccination to alleviate infectious disease burden.

7.1 Background and theories for vaccine network

A Science study found that Facebook users encountered more sensationalized headlines about COVID-19 vaccines from trusted outlets than outright deceptive stories, which were labelled and restricted by the social media platform [136]. Although individual misleading information may not solely determine vaccine decisions, multiple sources can collectively influence vaccine intentions [136]. Another key difficulty is estimating the effect of a belief based on misinformation on the change in behaviour [136]. The global population, especially in developing countries, is also not well represented in the existing literature for fighting misinformation, because many studies were centred on the U.S. and Europe [136].

For complex systems, the idea of "connectedness" refers to not only structural ties (which individuals are linked) but also behavioural impacts, which occur when individual actions reverberate throughout the system [2]. A complex system considers both its structure and how interactions unfold within it [2]. Activities within complex systems are catalysts that elicit systemic responses, revealing cause-and-effect relationships [2].

Social networks are vital for tracking behaviours and information dissemination [3]. Contagions differ in how they propagate. Complex spreading phenomena often rely on social reinforcement and community structure [5]. Collective behaviour models, emphasizing "thresholds," illustrate how others' actions affect personal choices [6]. The distribution of thresholds plays an important role in shaping group outcomes, with even identical average preferences producing distinct results [6]. The structure of social networks may significantly influence the dispersion of behaviour among a population [7]. Social network structures

profoundly influence behaviour dispersion, particularly in closely-knit communities where interconnected friends amplify behaviour spread [7, 137].

There has been ongoing modelling efforts to examine vaccination behaviour during infectious disease outbreaks. Studies have shown that heterogeneous behavioural responses are more accurate in prediction compared to homogeneous approaches, as vaccination impact and collective efficacy in the entire population may differ due to age, income, and risk perception [138, 139, 140]. Studies have also emphasized how past studies tend to overlook the endogenous (people's voluntary) behavioural changes and its effect on disease transmission [138, 141]. Amid high infodemic levels on social media, vaccination willingness fluctuates based on personal knowledge and risk perceptions, highlighting the role of endogenous transmission rate changes in our modelling framework.

To optimise vaccination dynamics and enhance community-wide protection, a modelling framework is essential for assessing cognitive thresholds that influence individual decisions on immunisation strategies under varying levels of anti-vax rumours. Estimating vaccination acceptability involves examining information dissemination effect, rumour debunking, and community engagement.

7.2 Vaccination network models

Despite widespread vaccine availability and real-time updates, online misinformation has consistently outpaced public health efforts. Social media often amplifies the negative impact of vaccine misinformation [142]. According to study on $\times$ (formerly Twitter) notes, the most common topics discouraging vaccination are adverse events and conspiracy theories [142]. Another study highlights the influence of local cultural factors on individual's beliefs and fears [143]. In addition to censored or restricted false content, vague and unfiltered misinformation on social media also undermines public confidence in vaccination [144].

Interestingly, according to Neely and Witkowski’s study, many people—including those already vaccinated—continue to believe in misinformation, which reduces their willingness to receive booster doses [145].

Traditional strategies to increase vaccination uptake include mandates, incentives, and public campaigns [146]. They are supplemented by newer social media-based interventions such as warning individuals, pre-bunking, and debunking disinformation [146]. Although these strategies reduce the spread of false information and influence public opinion, their direct effect on actual vaccination uptake remains uncertain [146].

Many existing studies use two-layer network structures to investigate the interaction between information (virtual) and disease (physical). Studies that isolate a third behavioural layer—separate from information and infection status—to examine individual cognitive decisions (e.g., protection-seeking or vaccination) are still in their infancy. In most published literature, vaccination is embedded within the epidemic layer, and protection is implicitly modelled through awareness of the disease.

Network models are widely used to investigate vaccination behaviours. For example, a case study in France evaluated school policies and targeted vaccination by simulating the transmission of disease among families, schools, and communities using a network model [147]. The study found that diagnosing and vaccinating large families can effectively reduce transmission, and that school-based spread played a significant role in epidemic dynamics [147].

According to a study in Hong Kong, individual beliefs, social dynamics, anxieties, and broader contextual factors all play a role in vaccine reluctance [148]. More specifically, key influences include personal perception of risk, confidence in authority, current health, social context, vaccine safety, and intensity of the epidemic [148]. A recent vaccine game model by Wang et al. demonstrated that vaccine efficacy significantly shapes public perception and influences uptake behaviour [149].

Another study laid the foundation for the co-evolution of separate networks for information, vaccination, and epidemic [150]. It developed a UAU-WVW-SIS model with aware and unaware states on the information layer, vaccinated and unvaccinated on the behaviour layer, and susceptible and infected on the disease layer [150]. A key novelty in their model framework was the integration of both local and global disease severity [150]. Their results highlight that local disease status exerts a strong influence on vaccine uptake compared to global disease status [150].

Some studies have used networks to target the uneven allocation of vaccination. One study highlights the homophily in vaccine uptake and emphasises the challenges in identifying contributing factors and improving vaccination coverage in a community with many unvaccinated individuals [151]. Another study acknowledges the role of population heterogeneity and summarises that to deploy more efficient vaccine strategies, we will need to consider network topology metrics such as node degrees or centrality indices [152].

The idea of node degree heterogeneity in transmission has been a key theme in various studies. For instance, Tuschhoff and Kennedy worked with heterogeneity in susceptibility with contact tracing data to gain further insights into vaccination thresholds [153]. Yin et al.'s research highlighted the importance of degree heterogeneity in vaccine rumour dissemination through a model using Chinese Sina Microblog [154]. Wang et al.'s study further confirmed the benefit of implementing asymmetric vaccination and quarantine measures based on the node degree [155]. A common way vaccination is treated in existing literature is by adding a vaccinated class to the traditional SIR. For instance, the model by Bao and Wu used a Susceptible-Vaccinated-Infected-Recovered (SVIR) network and varied transmission rates by node degree [156]. Using the same SVIR framework idea, Tanwar et al. analysed vaccine intervention networks and emphasized the importance of network topology. Their findings suggest that small-world structures—characterized by relatively homogeneous node degrees and short average path lengths—enhance the

effectiveness of vaccination when compared to scale-free or random networks, which display high heterogeneity and a broader range of node degrees [157].

In addition to the idea of node degree, other studies have shown the role of network structure by incorporating different age groups. For example, one article by González-Parra et al. demonstrated that the optimal strategy to prioritise vaccines to reduce infection and prevent death is to vaccinate the elderly age groups (40-59 and 60-69) first, as supported by their network model [158]. The work by Pant and Gumel also incorporated hospitalisation and used a similar age-based network setup, and confirms the conclusion that prioritising the elderly (65 and older) is an effective vaccination strategy [159]. The study by Nie et al. employed a similar age-structured method with more age groups and revealed slightly different results [160]. They concluded that children have higher vaccine uptake, infants and the elderly have lower vaccination rates, and adults' vaccine willingness depends on the cost considerations [160].

Swinford and Zadeh summarized that there are three main strategies for mitigating rumours during their spread: targeting the source, enhancing the social media control, and educating the audience [161]. Similarly, a recent review also identified some methods to prevent misinformation harm: improving digital health literacy, broad prebunking, debunking, community engagement, and social media regulation [162]. When the coupling with vaccine behaviour is considered, another strategy to combat rumour involves implementing periodic vaccination [163]. Liu et al. investigated both pairwise and hypergraph gossip spreading with vaccine interventions and demonstrated the effectiveness of periodic vaccination in countering the rapid spread of group gossip [163]. A review paper identified the majority of studies focused on assessing the knowledge outcomes, and only a few studies centred their evaluations on actual health behaviours [164].

7.3 Methods

Individual's understanding on vaccine policies often depends on community vaccine uptake and their awareness of disease. Whether people participate in vaccination is strongly influenced by their perception of vaccination, which is often shaped by information circulating within their social networks. To reduce the damage caused by misinformation, efforts should focus on implementing effective control strategies and timely corrective interventions.

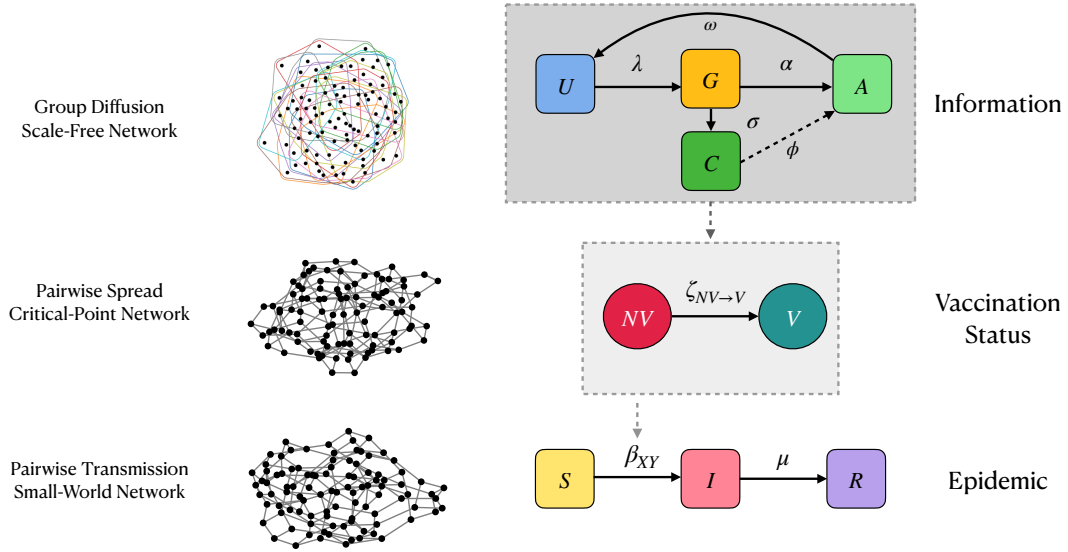


Figure 7.1: Schematic representation of a three-layer network model capturing the interactions between misinformation, cognitive vaccine behaviour, and disease transmission.

7.3.1 The information layer

This vaccination model is an extension of our earlier ICE network model framework, therefore, we adopt the same information layer structure as in our previous research

(Chapters 4 and 5) [165]. The information layer consists of the following states: uninformed (U), misinformed spreader (G), stifler (A), and corrected (C). On the information layer, we consider the following events (Figure 7.1): (i) uninformed (U) individuals become misinformed spreaders (G) upon encountering other misinformed spreaders, at rate λ_M ; (ii) misinformed (G) turn into stifler (A) after losing interest in spreading, at rate α ; (iii) misinformed (G) are corrected and transition to the corrected state (C), at rate σ_M ; (iv) individuals in the corrected state (C) may relapse into the stifler state (A), at rate ϕ_M ; (v) stifler (A) can renew their interest in gossip and revert to the uninformed state (U), at rate ω . We model the information layer as a scale-free network.

7.3.2 The cognition layer

The cognition layer consists of two vaccination status: non-vaccinated (NV) and vaccinated (V). Vaccination is assumed to be irreversible, with vaccination rate $\zeta_{NV \rightarrow V}$. The transition rates between vaccination status, vaccinated (V) and non-vaccinated (NV), depend on the local densities of misinformed (G) gossip spreaders, represented by $\rho_G = n_G/k_{\text{info}}$ and the local densities of corrected (C), given by $\rho_C = n_C/k_{\text{info}}$. A higher number of misinformed or discouraged neighbours reduces vaccine willingness, whereas a greater number of corrected neighbours decreases vaccine hesitancy. The level of vaccine-relevant misinformation can significantly influence an individual's decision to receive vaccination. The rates also rely on vaccinated neighbours in the cognition layer, given by $\rho_V = n_V/k_{\text{cog}}$, and are further influenced by mass information campaigns that reflect the overall disease prevalence, ρ_I . The parameter b is a smoothing coefficient that governs how sharply the transition occurs. For example, $b = 3$ yields a relatively gradual transition, while $b = 30$ produces a much sharper transition. The correction influence coefficient, η , determines the impact of corrected neighbours on vaccination behaviour. The following expression

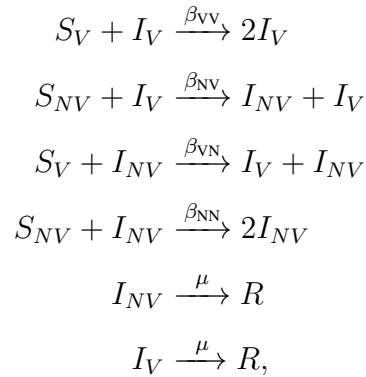
describes the transition from non-vaccinated to vaccinated states:

$$\zeta_{NV \rightarrow V} = \zeta_M(1 - \rho_G + \eta\rho_C) \tanh(b\rho_I) + \zeta_V\rho_V$$

We model the vaccination status layer as having a critical-point structure ($\gamma_c = 3.0$).

7.3.3 The epidemic layer

We model the epidemic layer using a small-world network structure. This layer encompasses three states: susceptible (S) infected (I), and recovered (R). On this layer, we consider the following events (Figure 7.1): (i) susceptible individuals (S) may become infected upon contact with infected individuals (I), at rate β_{XY} ; (ii) infected individuals (I) recover (R) at rate μ . A single recovery process and vaccination-dependent state transition dynamics are used to connect the network layers [107]. Figure 7.1 shows the corresponding cognitive states. A simplified representation of the state transitions and their rates is:



where subscripts V and NV denote vaccinated and unvaccinated individuals, and the infection rates satisfy the relationship $\beta_{VV} \leq \beta_{NV} \leq \beta_{VN} \leq \beta_{NN}$, indicating that the risk of infection depends on the vaccination status of interacting individuals. We assume that

vaccination reduces the transmission rate. For example, assuming an average of 80% vaccine protection against infection, the transmission rate between vaccinated susceptible and non-vaccinated infected individuals (β_{VN}) would be 0.2 times the transmission rate between two unvaccinated individuals (β_{NN}). Vaccination is assumed to reduce both susceptibility and transmissibility, including among breakthrough infections.

In the following results section, we present an analysis to evaluate the impact of prioritising highly connected nodes (referred to as hubs) during vaccination on the overall attack rate. To support this analysis, we extend our baseline model by introducing an additional compartment representing individuals who develop severe symptomatic disease following infection, denoted by I_{severe} . In this extended framework, a fixed proportion of infected individuals progress to this severe state, while the original infected compartment (I) now specifically represents those with non-severe symptomatic infections. This modification allows us to assess not only the effect of hub-targeted vaccination on disease spread but also its potential to reduce the burden of severe cases.

We assume the same transmission rate for both symptomatic (non-severe and severe) states. That is, vaccinated individuals who become infected, whether developing non-severe or severe disease, are equally likely to transmit the disease to susceptible individuals. Transmissibility in our framework depends solely on vaccination status and not on symptom severity. Upon infection, a proportion of susceptible individuals develop severe disease. This probability is denoted by κ_1 in the unvaccinated population and by κ_2 among vaccinated individuals (Table 7.1).

To simulate the network for vaccination and spreading misinformation, we used parameter values listed in Table 7.1. Similar to the basic ICE model described in Chapter 4, we designed the structure of each network layer based on a degree sequence, as presented in Section 4.2.1. We then applied the same optimised Gillespie algorithm for computational implementation as the one described in Section 4.2.2.

Table 7.1: Description of the model parameters and their values used for simulations in the misinformation and vaccine model. All rates are per day.

Symbol	Description	Value
ζ_M	Rate of vaccination based on misinformation	varied
ζ_V	Rate of vaccination based on vaccination	varied
β_{NN}	Transmission rate with susceptible and infected individuals both non-vaccinated	0.26
β_{VN}	Transmission rate with vaccinated susceptible and non-vaccinated infected individuals	$0.2\beta_{NN}$
β_{NV}	Transmission rate with non-vaccinated susceptible and vaccinated infected individuals	$0.1\beta_{NN}$
β_{VV}	Transmission rate with susceptible and infected individuals both vaccinated	$0\beta_{NN}$
R_0	Basic reproduction number	4.0
μ	Recovery rate of infected	0.2
κ_1	Probability of developing severe disease without vaccination	20%
κ_2	Probability of developing severe disease with vaccination	1%
λ_M	Rate of misinformation spreading	1/3
α	Rate of misinformation suppression	1/5
ω	Rate of renewal interest	1/3
σ_M	Correction rate of misinformation	varied
ϕ_M	Relapse rate of misinformation	0
b	Global misinformation coefficient	0.5
η	Correction influence coefficient	2
$k_{\min}$	Minimum network degree	3
γ_i	Power-law degree exponent in the information layer	2.5
γ_c	Power-law degree exponent in the cognition layer	3.0
γ_e	Power-law degree exponent in the epidemic layer	6.0
n	Number of nodes in the network	500

7.4 Results

7.4.1 Effect of rumour correction

Research suggests that the presence and persistence of misinformation alters vaccine belief and uptake, highlighting the importance of quantifying the impact of correcting false information [166, 167]. Thus, our first research goal is to evaluate the effect of rumour correction on vaccine coverage and the resulting disease spread in the context of higher-order networks. Specifically, we assess three scenarios where the correction rates correspond to zero ($\sigma = 0$, no correction), moderate correction ($\sigma = 0.2$), and intense correction ($\sigma = 0.5$). We investigate the dynamics of gossip spreaders, vaccinated individuals, and recovered cases over time under these misinformation correction scenarios.

If the misinformation is left uncontrolled (Figure 7.2 panels A, D, G), the proportion of gossip spreaders stabilizes around 32.8%, with roughly 7.2% of the population vaccinated and an overall attack rate of 44.6%. Implementing moderate correction within 5 days ($\sigma = 0.2$), reduces the proportion of gossip spreaders to 4.0% within 20 days, leading to 6.0% vaccination coverage and a lower attack rate of 38.4%. With rapid and intense correction ($\sigma = 0.5$) initiated within 2 days, gossip spreaders decline further to 1.2% in day 20, vaccination coverage increases to 6.8%, and the attack rate drops to 34%. In both moderate and intense correction scenarios, misinformation is nearly eliminated by day 60. Notably, prompt correction significantly shortens the duration of both misinformation and disease spread. Overall, these findings demonstrate that swift and effective rumour correction enhances vaccine uptake and substantially reduces preventable disease transmission.

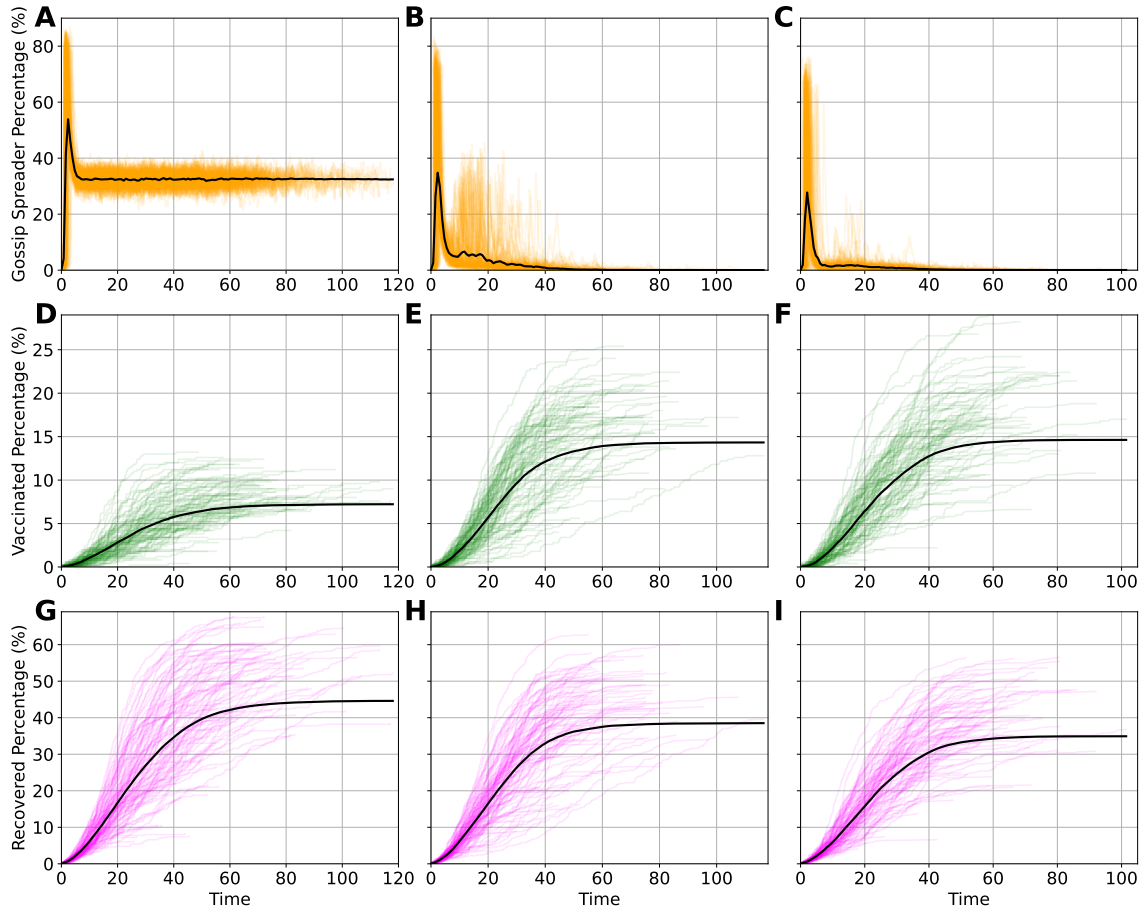


Figure 7.2: (A–C) Percentage of gossip spreaders, (D–F) percentage of vaccinated individuals, and (G–I) percentage of recovered individuals over time under different misinformation correction rates: $\sigma = 0$ (A, D, G), $\sigma = 0.2$ (B, E, H), and $\sigma = 0.5$ (C, F, I). Black lines represent the average of 100 stochastic simulations. The baseline vaccination rate influenced by the information layer is $\zeta_M = 0.10$, while the imitation-based vaccination rate is set to zero ($\zeta_V = 0$). Gossip acceptance thresholds are drawn from a normal distribution with mean of 0.10 and standard deviation of 0.05.

7.4.2 Effect of preemptive vaccination

Studies also indicate that many infectious diseases exhibit seasonal patterns influenced by environmental factors, underscoring the need for early, preventive vaccine strategies [168, 169]. Similarly, evidence shows that the timing of vaccination plays a critical

role in shaping population-wide immunity [170]. Thus, our second research goal is to investigate the effect of preemptive vaccination, or negative vaccination delay (Here, a “negative delay” refers to vaccine deployment prior to the onset of both the epidemic and misinformation spread), on disease spread under ongoing misinformation pressure. We examined a vaccination campaign initiated before disease emergence, and compared it a baseline scenario without delay. Specifically, we analysed three scenarios: (i) no preemptive vaccination; (ii) preemptive vaccination initiated 7 days in advance; and (iii) preemptive vaccination initiated 14 days in advance.

When we pre-vaccinate the population two weeks in advance (Figure 7.3), the epidemic duration is reduced by nearly half, with 2.6% in attack rate compared to $\sim 50.3\%$ in the absence of preemptive vaccination. Vaccine uptake also increases significantly, reaching 50.2%, as opposed to just 4.4% when vaccination starts with the onset of outbreak. Initiating vaccination one week before disease onset lowers the attack rate to $\sim 6.1\%$, with vaccine uptake 30.1%. These results suggest that early vaccination in the absence of an active epidemic proves highly effective in both mitigating attack rates and shortening the epidemic duration. Note that the impact of this intervention is dramatic, which is likely due to the relatively homogeneous vaccine status layer structure and epidemic layer structure. In real-world scenario, we expect to have more heterogeneity in physical contacts.

Next, we explored a scenario in which rumour propagation coincides with preemptive vaccination efforts, both preceding the onset of disease transmission. As before, we find that a two-week preemptive vaccine initiation nearly halves the epidemic duration. As shown in Figure 7.4, if vaccination starts with the onset of outbreak, overall vaccine uptake remains low at 3.9%. In contrast, initiating vaccination one week in advance increases coverage to 20.6%, while a two-week lead boosts it to 37.8%. The overall attack rate drops sharply from 46.6% (without preemptive vaccination) to 9.6% and 4.2% under one- and two-week scenarios, respectively.

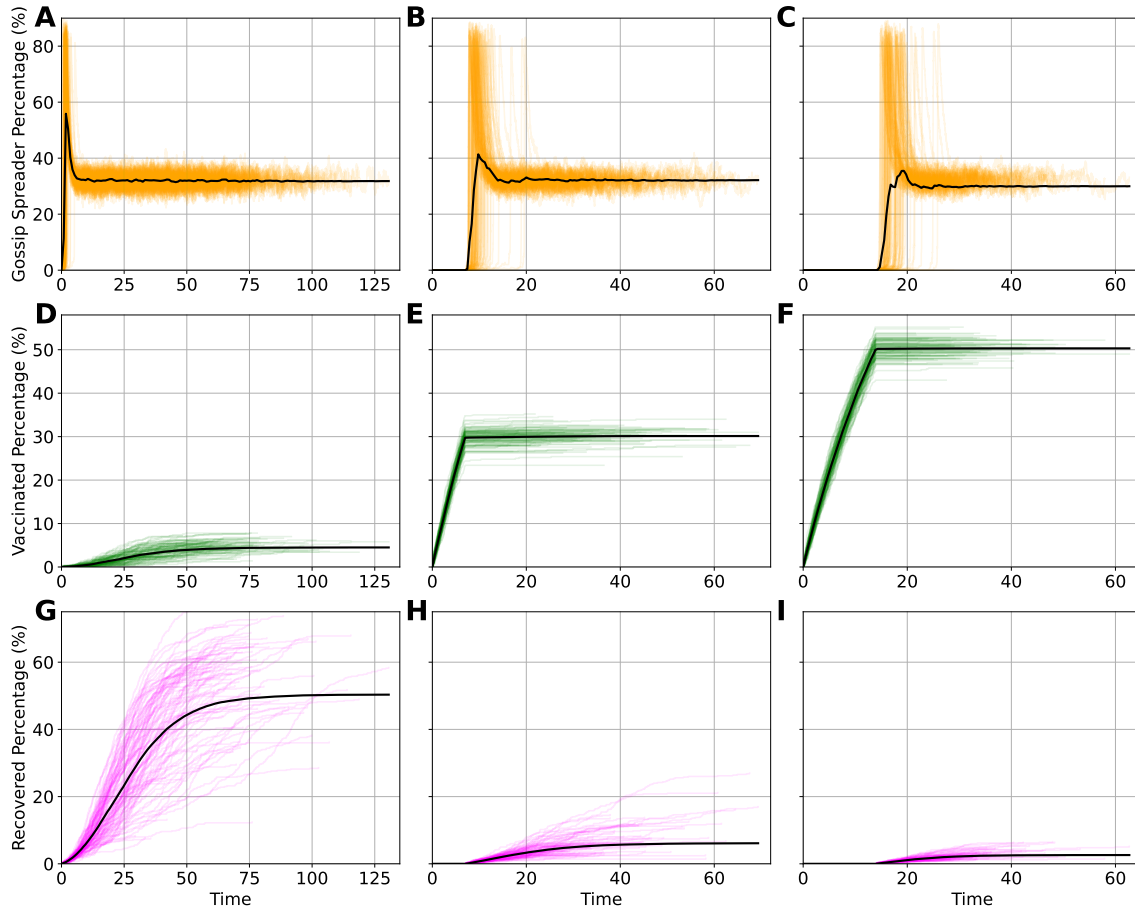


Figure 7.3: (A–C) Percentage of gossip spreaders, (D–F) percentage of vaccinated individuals, and (G–I) percentage of recovered individuals over time for different pre-vaccination durations (negative delays) $\tau = 0$ days (A, D, G), $\tau = 7$ days (B, E, H), and $\tau = 14$ days (C, F, I), with vaccination occurring only during the pre-vaccination period. Black lines represent averages over 100 simulations. The baseline vaccination rate influenced by the information layer is $\zeta_M = 0.05$, while the neighbourhood-based imitation effect is disabled ($\zeta_V = 0$). Gossip acceptance thresholds follow a normal distribution with mean of 0.10 and standard deviation of 0.05.

Even a single week of early vaccination results in a fourfold increase in vaccine uptake and over a 70% reduction in disease spread. The benefits are even more pronounced with a two-week lead. These findings highlight the importance of preemptive vaccination

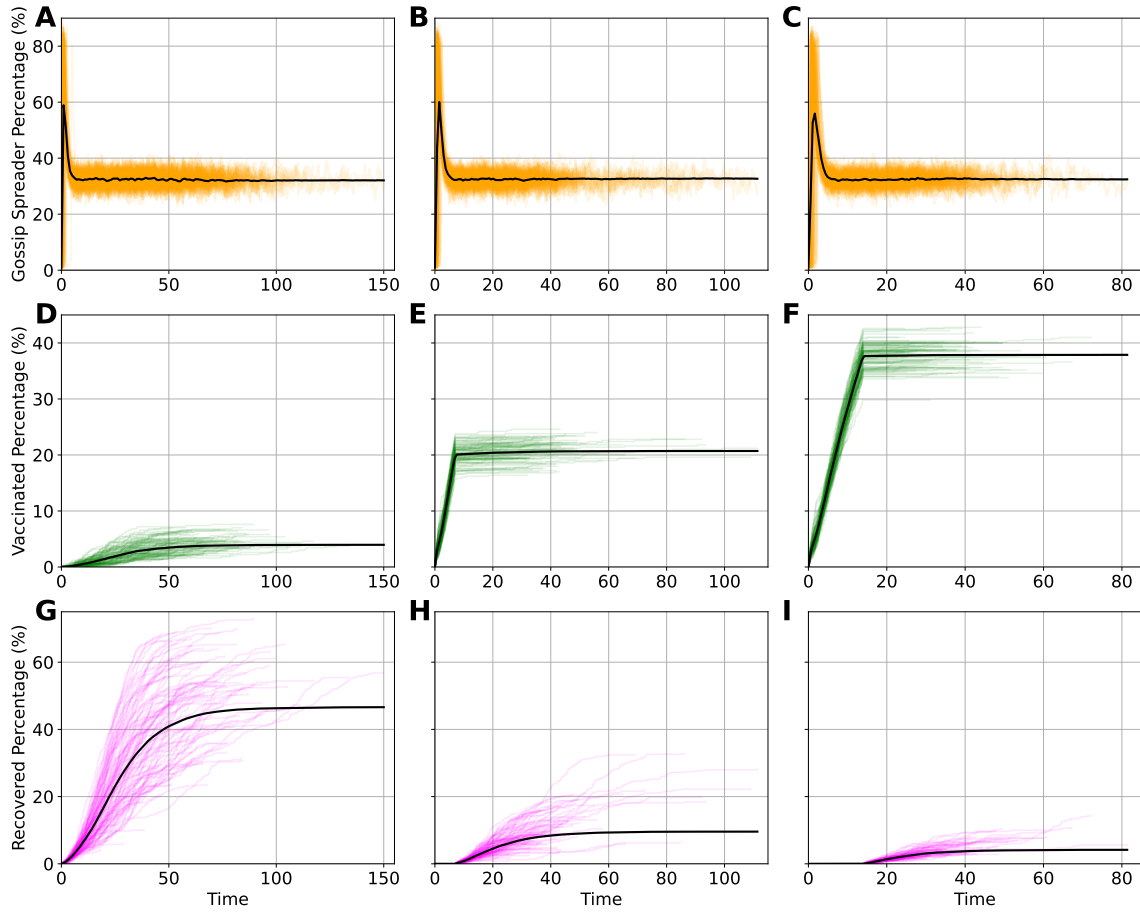


Figure 7.4: (A–C) Gossip spreader percentage, (D–F) vaccinated percentage, and (G–I) recovered percentage over time for different pre-vaccination durations (negative delays) $\tau = 0$ days (A, D, G), $\tau = 7$ days (B, E, H), and $\tau = 14$ days (C, F, I), where both vaccination and gossip dissemination begin during the pre-vaccination phase. Black lines indicate averages over 100 simulations. The baseline vaccination rate driven by the information layer is $\zeta_M = 0.05$, with no influence from neighbourhood imitation ($\zeta_V = 0$). Gossip acceptance thresholds are drawn from a normal distribution with mean of 0.10 and standard deviation of 0.05.

campaigns not only in reducing epidemic impact, but also in enhancing vaccine acceptance, even when misinformation is actively circulating. Preemptive vaccination mitigates the influence of misinformation during an epidemic by establishing immunity ahead of disease

transmission, effectively curbing the epidemic’s spread from the outset.

7.4.3 Effect of hub-prioritised vaccination

Previous studies have shown that network topology and degree distribution are critical factors in optimising vaccination strategies within network-based epidemic models [171, 172]. Building on this foundation, our third research objective is to evaluate the effectiveness of degree-based (hub-prioritised) vaccination in the context of persistent disinformation.

Using the baseline model without additional compartments, our analysis reveals that prioritising highly connected individuals (hubs) for vaccination (at each vaccination event, we select the node with the maximum degree to vaccinate) has limited effect on reducing the overall attack rate. As shown in Figure 7.5, the epidemic curves for random vaccination and hub-prioritised vaccination are nearly indistinguishable. In both cases, with an overall vaccine coverage of approximately 12%, the attack rate remains steady at around 40%. These findings suggest that in the presence of widespread misinformation, degree-based vaccination alone may be insufficient to curb disease spread, underscoring the need for complementary strategies such as misinformation correction or targeted behavioural interventions.

Finally, we extended our analysis introducing a new compartment for severe symptomatic cases, denoted as I_{severe} . We assume that, in the absence of vaccination, 20% of infected individuals develop severe symptoms, whereas this proportion is reduced to 1% among vaccinated individuals.

In a small-world network with moderate degree heterogeneity ($\gamma_e = 6.0$), our results indicate that hub-prioritised vaccination yields minimal advantage over random vaccination in reducing either overall disease burden or severe outcomes Figure 7.6. However, when the degree heterogeneity is increased ($\gamma_e = 4.0$), representing a more heterogeneous

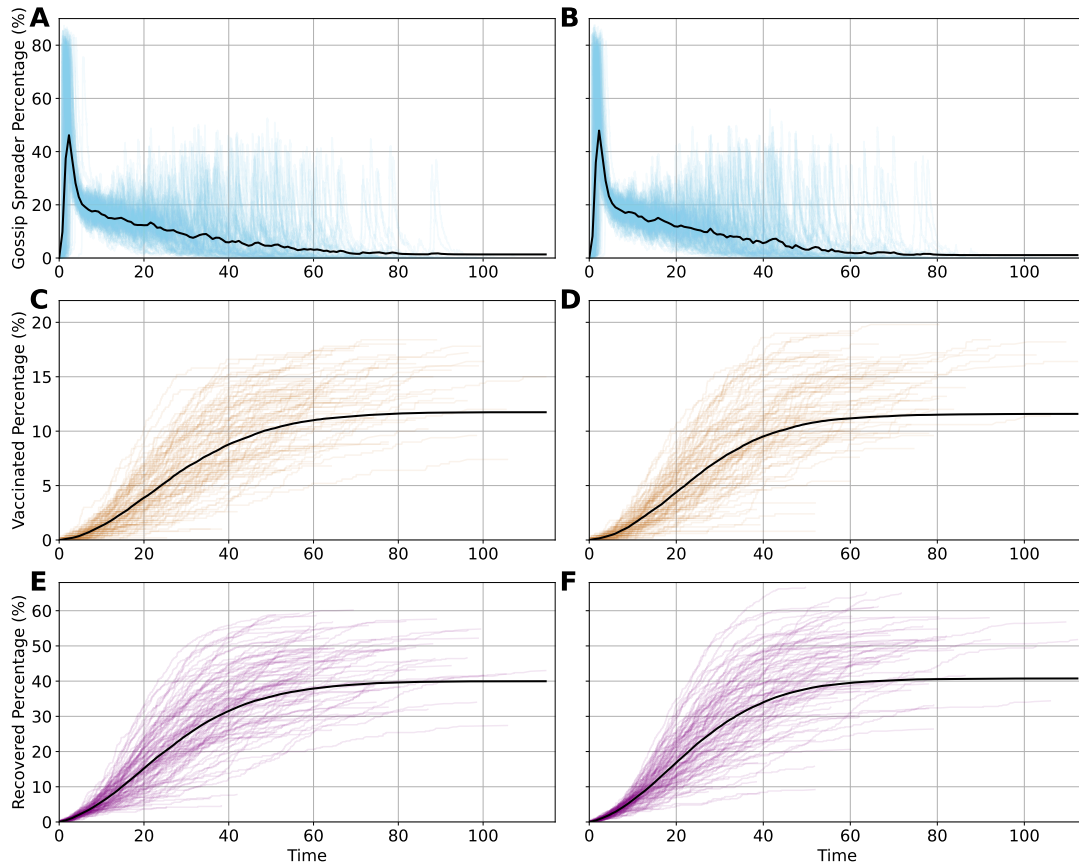


Figure 7.5: Comparison of random versus hub-prioritised vaccination in the baseline model. Panels (A, C, E) show results for random vaccination, while panels (B, D, F) correspond to hub-prioritised vaccination. Specifically, (A–B) show the gossip spreader percentage, (C–D) the vaccinated percentage, and (E–F) the recovered percentage over time, under a misinformation correction rate of $\sigma = 0.05$. Black lines represent averages over 100 simulations. The baseline vaccination rate influenced by the information layer is $\zeta_M = 0.10$, with no contribution from neighbourhood imitation ($\zeta_V = 0$). Gossip acceptance thresholds are drawn from a normal distribution with mean of 0.10 and standard deviation of 0.05.

small-world structure (Figure 7.7), the benefits of hub prioritisation become more evident. Specifically, the attack rate drops from 17.4% under random vaccination to 11.6% with hub prioritisation. Likewise, the proportion of severe symptom cases declines from 4.0% to 2.4%.

These outcomes reflect a relative reduction of approximately 33.3% in attack rate and 40.0% in severe symptom cases when hubs are prioritised for vaccination. In summary, our findings suggest that the effectiveness of degree-based vaccination strategies is significantly amplified in networks with high degree heterogeneity, compared to those with more uniform connectivity patterns.

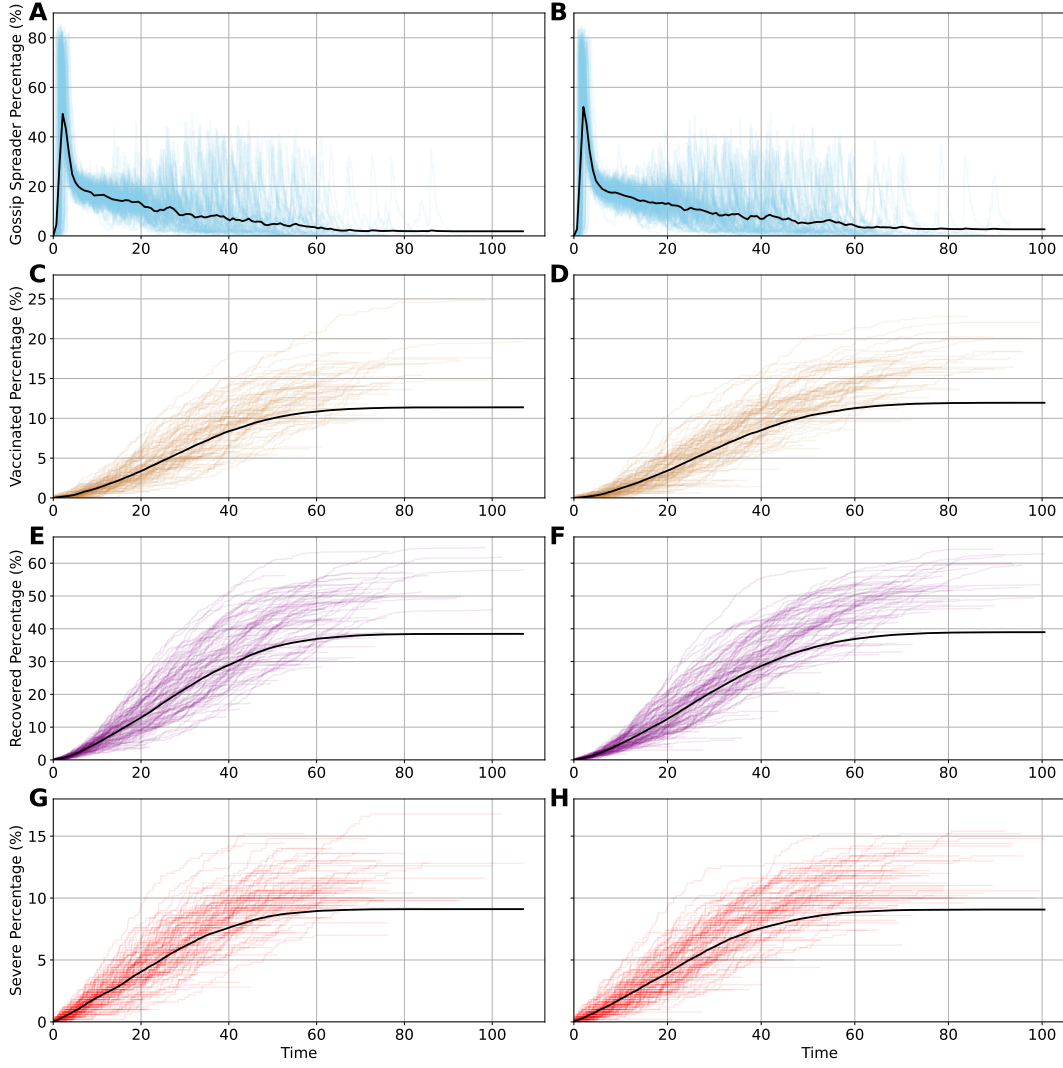


Figure 7.6: Random versus hub-prioritised vaccination in the severe symptoms model with moderate network heterogeneity ($\gamma_e = 6.0$). Panels (A, C, E, G) correspond to random vaccination, while panels (B, D, F, H) show results for hub-prioritised vaccination. Specifically, (A–B) show the gossip spreader percentage, (C–D) the vaccinated percentage, (E–F) the recovered percentage, and (G–H) the percentage of individuals with severe symptoms over time, under a misinformation correction rate of $\sigma = 0.05$. Black lines represent averages over 100 simulations. The baseline vaccination rate influenced by the information layer is $\zeta_M = 0.10$, with no contribution from neighbourhood imitation ($\zeta_V = 0$). Gossip acceptance thresholds are drawn from a normal distribution with mean of 0.10 and standard deviation of 0.05.

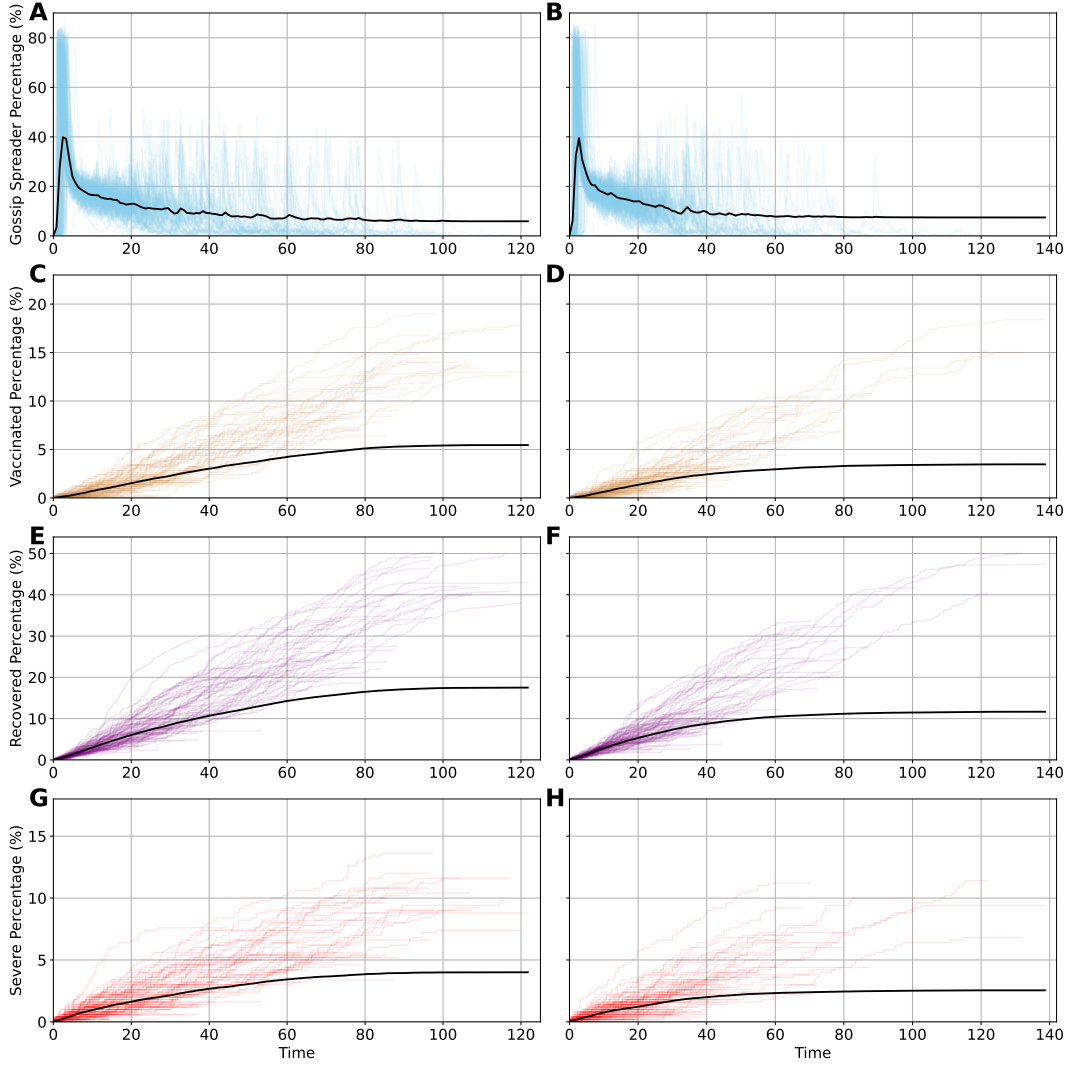


Figure 7.7: Random versus hub-prioritised vaccination in the severe symptoms model with high network heterogeneity ($\gamma_e = 4.0$). Panels (A, C, E, G) correspond to random vaccination, while panels (B, D, F, H) show results for hub-prioritised vaccination. Specifically, (A–B) display the percentage of gossip spreaders, (C–D) vaccinated individuals, (E–F) recovered individuals, and (G–H) individuals with severe symptoms over time, under a misinformation correction rate of $\sigma = 0.05$. The epidemic layer has a degree exponent of $\gamma_e = 4.0$. Black lines represent averages over 100 simulations. The baseline vaccination rate influenced by the information layer is $\zeta_M = 0.10$, with no contribution from neighbourhood imitation ($\zeta_V = 0$). Gossip acceptance thresholds are drawn from a normal distribution with mean of 0.10 and standard deviation of 0.05.

Chapter 8

Discussion

This chapter synthesises key findings from Chapters 5, 6, and 7. We begin by situating our results on misinformation correction (Chapter 5) within the broader literature, highlighting critical model parameters and the unique contributions of our network-based framework. We then examine how network heterogeneity (Chapter 6) shapes the dynamics of the ICE model. Finally, we reflect on insights from Chapter 7 by comparing our findings on vaccine rumours with existing research, emphasizing practical strategies to improve vaccine uptake and reduce both infection rates and severe outcomes.

8.1 Impact of misinformation and correction

Understanding the interplay between information dissemination and the behavioural responses of individuals is critical for designing mitigation strategies that limit the spread of disease in the population. In this study, we developed a multi-layered, hyper-edge model that distinguishes individual decisions based on information exposure and local perception across interconnected layers, with a particular emphasis on the cognition layer. While

most two-layer network models in the literature primarily examine awareness and disease status [173, 174, 175, 26], our framework extends previous work by incorporating both local and global information networks, and modelling perceived risk in physical networks that influence individual protective behaviours [176, 177, 178, 179, 180].

Our comparison of hyper-edge and basic pairwise diffusion models revealed that misinformation has more severe consequences when spread occurs within group-based (hyper-edge) structures. Increasing the acceptance threshold for misinformation leads to a higher density of stiflers. Our network model generalises classical simplicial complex models, offering more flexibility and control over dissemination rules. Importantly, we found that rapid correction of misinformation is crucial. Specifically, warning or educating individuals within the information network was more effective in reducing the overall attack rate than simply removing gossip spreaders from the network. We also observed that relapse after correction has limited influence on the overall attack rate, but meaningful reductions in attack rate still require substantial increases in the correction rate.

There are two levels of super-spreading, corresponding to virtual and physical networks, that shape disease dynamics. On the virtual level, some studies have addressed rumour correction by simultaneously propagating true information with a competitive cascade approach as a countermeasure for misinformation [181, 182]. Our approach differs by considering strategies to directly address misinformation rather than relying on a parallel process for spreading anti-rumours. Incorporating scientific knowledge into the information network has also been proposed to examine its effect on rumour control [183]. This method, which employs external interventions (e.g., communications by the government to provide evidence and enhance public knowledge for countering rumours and misinformation), also differs from our approach that targets internal strategies for mitigating rumour spread within the information network structure (e.g., nodes can self-correct or warn peers and gossip spreaders). Because of the structural complexity of the existing model framework, we

concentrated on misinformation in our framework and did not investigate the scenario with simultaneous true and incorrect information. However, future research could expand our model to include the simultaneous spread of true information.

A key practical challenge is the unequal distribution of disease-related information, especially among diverse and marginalised groups [184, 185]. Since socio-demographic variables may influence information spread, one potential future direction is to extend our existing model framework by incorporating a demographic layer. This would allow the calibration of spreading rates to account for demographic differences in intervention responses. Another approach to addressing this issue is to tailor dissemination strategies to local disease risks, as done during the Omicron wave in Hong Kong [186]. Promoting health equity is also essential, particularly for vulnerable individuals populations [187]. When data is available, integrating social, economic, or geographical factors into existing models can further enhance targeted interventions. Another barrier is public acceptance of health measures. People often prefer less restrictive interventions, making it essential to emphasise the benefits of proposed measures and leverage peer influence to improve the effectiveness of health communication [188].

For physical interactions, studies have evaluated various interventions such as limiting superspreading events and reducing transmission from highly connected nodes [189, 190, 191]. However, implementing similar strategies in virtual information networks is more challenging, especially for users with broad online connectivity on multiple platforms. Despite this complexity, understanding the interplay between virtual and physical layers is vital for designing comprehensive intervention strategies. Our framework offers a generalised platform to investigate how cognition and behavioural feedback loops influence both information diffusion and disease spread *via* physical interactions.

Our model is designed for both flexibility and applicability, allowing adaptation to a variety of network structures and datasets. For instance, the hyper-edge model can be cus-

tomised by adjusting the power-law exponent, the number of nodes and size of groups, and higher-order structures to explore various scenarios. While our current implementation is intentionally simple and general, it is readily extendable to incorporate community-specific structures, social media and datasets, or demographic layers. Additional compartments, such as age groups, information types, or disease outcomes such as hospitalisation and isolation, can also be included. For modelling different diseases, compartment definitions can be tailored to reflect epidemiological characteristics and transmission dynamics.

Nonetheless, there are limitation to consider. We did not use empirical data to derive the hyper-edge distributions, and did not explicitly model community structures within the information layer [192, 193]. Within the epidemic layer, our approach maintains a level of generality by focusing solely on the principles of susceptible-infected-recovered framework, without considering other important epidemiological statuses or disease outcomes [194, 195]. Nevertheless, our model can be expanded to incorporate additional disease states when examining a particular infectious disease. While our work adopts an approach to generalize protection against infection, it is important to distinguish between short- and long-term protective behaviours. For example, mask-wearing and other non-pharmaceutical measures provide immediate (partial) protection, and their effects are only realized during adherence without changing the susceptibility of individuals to infection. Vaccination, on the other hand, could have a long-term effect by reducing the risk of infection or severe outcomes. Our study considered transient protective behaviours that are reversible, but the framework provided here can be adapted to examine virtual and physical networks for irreversible protective behaviours with longer-term effects such as vaccination. Finally, our model does not account for full complexity of information dynamics, which may involve various levels of information spreading with the coexistence of positive, neutral, and negative information states [196, 197].

8.2 Role of information network structure

We examined the influence of gossip-spreading sources on the long-term adoption of protective behaviours within a network. Our analysis revealed that gossip originating from low-degree nodes results in a slightly higher number of protected individuals over time compared to gossip spread by high-degree nodes. This finding suggests that the long-term protective outcomes are largely insensitive to whether misinformation originates from highly or sparsely connected individuals. While previous studies have highlighted the critical role of source nodes in information diffusion [198, 199, 200], our work advances this perspective by incorporating a behavioural response layer, showing that the topology of hyper-edge groups can exert a more pronounced influence on diffusion dynamics than the individual sources themselves.

Our findings also demonstrate that scale-free networks exhibit sustained, periodic peaks in gossip propagation, driven by the high connectivity of hub nodes. This dynamic contrasts with small-world networks, where gossip peaks are short-lived and less persistent. To realistically capture the recurrent and enduring nature of disinformation, scale-free topologies are more appropriate for modelling long-term impacts.

Previous research has shown that in hypergraphs with heterogeneous group sizes, contagion tends to spread through larger groups, particularly under nonlinear transmission dynamics [201]. This behaviour reduces bistability, making it more challenging for infection to either die out completely or dominate the system [201]. Large groups serve as stable reservoirs, sustaining the spread. Our results corroborate these findings, confirming the importance of group size heterogeneity, and extend the analysis by linking behavioural strategies to additional network layers.

However, several limitations must be acknowledged. First, we did not investigate specific community structures and their role in shaping disease-related behaviours. Second,

we did not include additional compartments in the epidemic layer, such as hospitalised individuals, which are often useful for quantifying behavioural changes [202]. Furthermore, our model does not incorporate real-world data or individual behaviour patterns [203], which could enhance model calibration and realism. These elements represent promising directions for future work.

8.3 Combating misinformation and improving vaccination

Bautista et al. demonstrated the benefits of correction of vaccine misinformation through an online statistical experiment [204], showing that rebuttals can improve public understanding. Similarly, Porter et al. found that providing accurate information can reduce belief in vaccine misinformation [205]. However, much of the existing literature explores the impact of misinformation on vaccination and disease spread using pairwise transmission models, without incorporating a distinct cognition or behaviour layer [206, 156, 207]. Only a few studies have introduced separate behavioural layers, and these typically focus on global-level interventions without addressing higher-order group dynamics or localised information effects [208, 209]. Our results offers a novel perspective by leveraging higher-order network structures, demonstrating that rapid correction of misinformation can significantly increase vaccine uptake and reduce attack rates in group-based spreading scenarios.

In high-attack-rate scenarios, our results suggest that prioritising hubs for vaccination yields less reduction in overall attack rate than in low-prevalence settings. We also observe that vaccination exert limited influence on the attack rate in these high-attack contexts, which is consistent with the role of the vaccine in our model—primarily mitigating severe outcomes rather than blocking transmission entirely. Moreover, the timing of vaccination plays a critical role. Because vaccine rollout coincides with disease emergence in our model, many individuals become infected before immunity can develop, diminishing the

population-wide impact of vaccination.

Kulsum et al. emphasised the importance of early vaccination in curbing disease spread compared to delayed interventions [210]. Our analysis supports this finding, showing that early vaccine deployment facilitates faster epidemic containment. If vaccination occurs prior to misinformation spread and epidemic outset, transmission can be substantially reduced, and infection nearly eliminated. Furthermore, early-stage vaccination can generate a strong herd immunity effect, as coverage rapidly transitions from minimal to substantial levels.

Previous studies have highlighted the strategic value of vaccinating highly connected individuals. Blanco-Rodríguez et al. showed the importance of hubs in vaccine optimisation [211], and Cheng et al. demonstrated that targeting high-degree nodes reduces hospitalisation burden in network-based models [212]. Our findings confirm these observations in the context of higher-order networks, indicating that targeting hubs within a highly heterogeneous structure reduces severe disease burden more significantly compared to uniform random vaccination. However, it is important to note that this strategy is effective primarily in networks with high heterogeneity, where the degrees disparities between hubs and low-degree nodes are pronounced.

Despite the flexibility of this structure for expansion to study other network scenarios, limitations exist that should be considered in future work. While we do not explore community-level modularity or social clustering in vaccination, these factors could be integrated in the model. Although our framework is designed to be general, it can be adapted to incorporate region-specific data, such as vaccination rates, demographic distributions, and disease incidence for localised analysis. We also do not include age structure in the current model, and our focus is on the network interplay between misinformation, vaccine behaviour, and disease spread. However, future research could tailor the compartmental structure to study the differential impacts of vaccine misinformation on vulnerable

populations, such as the elderly or school-aged children.

Another noteworthy limitation of our current general framework is its inability to capture the time-evolving character of real-world networks. In real life, physical relationships between people can appear and fade over time, whereas virtual links can also vary owing to social contagion. One potential addition that could better reflect these dynamic topologies is network rewiring. When we rewire a network, we change the edges while preserving the global network features like connectivity or degree distribution [213]. Alternatively, we may use modular architectures to modify edges inside and across communities over time.

Furthermore, it is important to note that our current modelling framework lacks empirical validation for thresholds (normal distribution for misinformation acceptance). To calibrate for the misinformation acceptance threshold, we can collect and analyse social media data such as tweets. Specifically, we can evaluate the probability of an individual reposting or sharing misinformation to their own network after engaging with content through viewing, liking, or commenting on the platform.

In addition, we started with the homogeneous mixing assumptions to derive some of our theoretical results. Homogeneous assumptions help us understand and simplify complex systems, giving important theoretical insights while being mathematically solvable. We use them to obtain general principles and to isolate the effects of heterogeneity. Our heterogeneous network results rely on the heterogeneous mean field (HMF) theory, which addresses heterogeneity while remaining analytically tractable.

Moreover, it is important to note the size of our network. When the size of the network grows, system behaviour can change. Since diffusion processes are impacted by the network's architecture, we may observe change of behaviour as the network size increases. To ensure that our simulations mimic real-world scenarios in scale, it is critical to prioritise the efficiency of our algorithm and optimise the computation in follow-up work. In addition, fluctuations are important in smaller systems. Stochastic effects may result in

rapid eradication of gossip in a small network. However, larger systems are more stable and are unlikely to be dominated by fluctuation. Another main distinction for networks of different sizes is the role of heterogeneity. Large networks are characterised by outliers and high-contact nodes, which are rare in small systems. The gossip spreading dynamics can change significantly with the system size, especially for scale-free networks.

Chapter 9

Conclusion and Future Direction

In this chapter, we summarize the key findings of this thesis, discuss their broader implications, and outline directions for future research that build on the modelling framework developed herein.

9.1 Misinformation correction strategies

This thesis presents an initial investigation into the network-driven collective dynamics and higher-order cognitive processes involved in the spread and control of misinformation, laying the groundwork for future research. Among the corrective strategies evaluated, we find that rapid intervention—especially through warning and educational efforts—offers more effective mitigation than short-term punitive measures such as temporarily banning misinformation spreaders. While not explicitly modelled here, further exploration is warranted to understand the impact of such strategies on longer-term protective behaviours, such as sustained vaccine acceptance. Additionally, the role of community structure and node centrality within the information layer merits deeper theoretical and empirical

investigation, especially in relation to misinformation resilience.

9.2 Information layer heterogeneity

Our findings reveal that gossip sources alone exert limited influence on overall protective behaviour due to the dense interconnectivity inherent in hyper-edge (group-based) networks. Through temporal analysis of gossip prevalence and epidemic trajectories, we demonstrate that higher heterogeneity—characteristic of scale-free networks—better captures the persistent influence of misinformation. In such networks, misinformation can continuously percolate through influential nodes and communities. Consequently, in the presence of power-law group size distributions, it becomes imperative to prioritise information-based interventions to effectively suppress attack rates and support behavioural protection.

9.3 Vaccination in an infodemic

The success of vaccination campaigns during an infodemic is tightly coupled with the management of misinformation. Our analysis suggests that corrective action within a narrow time window—ideally within two days—can substantially increase vaccine uptake and reduce the epidemic attack rate. Preemptive vaccination campaigns are shown to be critical in both reducing infection spread and preventing severe outcomes. Moreover, the benefit of targeting highly connected individuals (network hubs) for vaccination depends on the structural heterogeneity of the disease transmission network. In networks with high degree heterogeneity, hub-prioritised vaccination strategies yield significantly better outcomes, highlighting the importance of tailoring interventions to the underlying contact topology.

9.4 Future work

This thesis has intentionally adopted a general modelling framework to explore the coupled dynamics of misinformation, behavioural response, and disease transmission. However, numerous extensions are possible.

One important direction involves incorporating demographic and community-level heterogeneity. Prior work has shown that vaccine acceptance can vary across communities due to differences in social norms, access to healthcare, and socioeconomic conditions [214, 215]. Future work will integrate regional datasets and empirically derived contact matrices to better capture community structure, modularity, and inequality in vaccination behaviour, particularly within higher-order group interactions.

A second area of expansion lies in addressing temporal delays between the spread of information, behavioural adaptation, and disease transmission. Most existing models, including those used in this study, rely on systems of ordinary differential equations with immediate responses. Although some literature addresses time delays in disease spread with resource constraints [216, 217, 218], few have incorporated delays in information-behaviour-disease feedback loops. While we have explored some timing aspects through computational simulations, a more robust analytical framework using delay differential equations would enrich theoretical understanding.

Lastly, higher-order dynamics in network systems remain an emerging and largely underexplored area [52, 219, 89]. As social platforms evolve rapidly, so too do the mechanisms by which misinformation and behavioural cues are transmitted. These platforms provide fertile ground for modelling interactions via hypergraphs and simplicial complexes, which better reflect real-world group dynamics. Advancing tools for higher-order structural analysis will not only improve epidemic models, but also enhance our ability to predict and manage collective behaviour in complex social systems.

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