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Behavioral Analysis in Complex Networks Using Network Science Algorithms

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ABSTRACT

Complex networks contain important structures to represent individuals' connections. This Research aims to analyze the patterns of complex contagion by considering various factors in network science and standard networks. Influential factors in a network, such as the degree of nodes, network structures, and hubs in scale-free networks, are being analyzed. The novelty of this paper lies in the analysis of the mentioned factors when a behavioral diffusion occurs, regardless of the kind of behavior spreading, and using only the factors in the network. The findings of this research demonstrate that the behavior of each network during the diffusion, considering the factors above, can be different. Random networks show a disciplined behavior of the changes in the number of infected nodes in each timeframe. However, scale-free networks show this property with the number of infected neighbors of each node at the time of infection of that node.

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1 Introduction

In everyday human life, various forms of communication exist. These interactions can generally be categorized into two main types: physical (real) and virtual. Each type of interaction can be represented as a graph, which can be analyzed using the algorithms of network science. One of the key phenomena observed in any social network is diffusion or spreading. Broadly speaking, depending on the nature of what is propagating through the network and how it affects different nodes, diffusion can be classified into two types: simple contagion and complex contagion. In this context, a node with the potential to be affected is referred to as susceptible, while a node that has already been influenced is called infected.

When nodes transition from the susceptible state to the infected state without requiring a high number of infected neighbors-often triggered by contact with only a single infected neighbor-the spreading phenomenon within the network is classified as simple contagion. One of the most common examples of simple contagion is the spread of viruses, both in human populations and through computer networks via the internet.

Various models have been developed to simulate the dynamics of simple contagion. One of the most basic and widely used models is the SI[1] model. In this model, individuals are divided into two categories: susceptible and infected. Initially, all nodes are in the susceptible state. Once a node becomes infected, it begins to spread the virus to its neighbors. Eventually, as all individuals in the network become infected, no further state transitions occur, and the process reaches a steady state. As observed in simple contagion, a node can become infected even after contact with a single infected neighbor. However, this is not necessarily the case in complex contagion. Complex contagion is about the spread of behavioral traits, norms, or habits across a network. As such, the adoption of these behaviors involves greater challenges and complexities compared to simple contagions. In this kind of diffusion, individuals typically do not adopt the spreading behavior upon their first exposure to it. Instead, adoption in complex contagion requires reinforcement through repeated exposure to the contagion from multiple sources.

Nevertheless, mere frequency of exposure may not be sufficient in complex contagion. Factors such as the presence of communities, the degree of closeness between neighbors, the network topology, and even the nature of the behavior being propagated can all significantly influence the likelihood of adoption.

Complex contagion has been extensively studied across a wide range of applications. In the context of public health, it relates to the diffusion of both healthy and unhealthy behaviors within a network. Similarly, the spread of innovation is also a prominent topic within this domain. One of the most significant applications of complex contagion lies in the analysis of interpersonal networks on social media platforms.

The study of complex contagion in social networks is not limited to information dissemination. With the rapid growth of the internet, many of the aforementioned applications are increasingly relevant in online social environments. Specifically, the spread of information that is inherently tied to these networks-such as news, hashtags, and socially driven discussions-is a key area of investigation. Moreover, complex contagion plays a crucial role in advertising, where it can significantly impact marketing strategies and the success of businesses and enterprises.

[1] Susceptible-Infected

2 Related Works

In 2014, a study was conducted in Philadelphia, USA, to examine attendance in fitness classes [25]. Participants were randomly assigned to one of four experimental conditions, which varied in terms of competition and social support, delivered either individually or in groups. The findings revealed that attendance was significantly higher in the competitive and combined conditions (competitive and supportive) compared to the supportive condition. A key takeaway from this study is the critical role of the nature of a network's interpersonal relationships in enhancing behavioral diffusion.

The role of interpersonal relationships has also been investigated in another study focused on the factors influencing weight loss through social networks [5]. This research examined how social ties, such as network structure, network size, relationship types, and social support, affect weight reduction. Contrary to expectations that stronger social ties would facilitate behavior diffusion, the results indicated that individuals with fewer connections achieved greater weight loss. However, participants who experienced high-quality social support within smaller networks showed a positive impact on behavior diffusion. The study involved 387 participants enrolled in a 16-week behavioral weight-loss program.

Another study investigated the diffusion of smoking behavior using a bi-threshold model [11]. This study also highlighted the critical role of hubs in complex contagion. Simulation results demonstrated that when the state of hub nodes remains fixed, their influence can significantly drive behavioral diffusion across the entire network. In another study, researchers examined how the number and types of relationships within a network influence smoking behavior [13]. Social ties in this research were categorized into three groups: family, friends, and virtual friends. One of the key findings was the prominent role of close virtual friends in providing effective social support and influence in smoking cessation.

A study addressing anti-vaccination behaviors during the COVID-19 pandemic issues aimed to identify optimal network parameters that could enhance public trust in vaccination [4]. The study's key finding highlighted that maximizing the spread of pro-vaccination sentiment can be achieved most effectively by utilizing hub nodes as central agents of diffusion. Furthermore, the influence of opinion dynamics related to this topic has been investigated through exponential random graph analysis in another study [21]. This research explored the emergence of the echo chamber effect and its role in the dissemination of health-related misinformation. The authors examined this phenomenon using a social media network formed around discussions on a viral video concerning COVID-19 vaccination.

With the rapid growth of social media usage and the expansion of virtual communication, behaviors and social norms are increasingly being disseminated through these networks. Users' behaviors on platforms such as social networks, and their susceptibility to behavioral contagion, are emerging as critical factors in understanding the dynamics of social diffusion. A study in this context simulated user behavior on the social media platform X[2] using both simple and complex contagion models. The results showed that user adoption in such environments tends to follow patterns of complex contagion [18].

Another study [7] investigated the diffusion of services, such as product or online service

[2]Formerly known as Twitter, rebranded as X in 2023

advertisements, by analyzing mechanisms and network factors influencing adoption among individuals. To study the adoption behavior, the authors examined the purchase of a paid service over an 89-month period. Initially, they applied the Watts threshold model [24], and subsequently proposed an extended version. This generalized model incorporates two new features: the presence of immune nodes-individuals who will never adopt the service regardless of exposure-and spontaneous adopters-susceptible individuals who may adopt independently at the outset of the diffusion process.

In parallel to classical simple contagion models such as SI, SIS, and SIR, the work by [17] introduces several complex contagion models tailored for social network applications. Among them, the ISR model[3] was developed to simulate information diffusion, while the Sethi model was adapted for advertising dynamics. A key innovation is that interactions among spreaders may lead them to collectively halt further dissemination.

Given the scale-free nature of many real-world networks, one of the most critical assumptions in complex contagion is the influential role of hub nodes. A recent study [26] introduced an algorithm to identify individuals with the greatest potential influence in the context of complex contagion. The study introduces a set of relational functions to formalize the connection between node influence and susceptibility within this diffusion-specific topology.

The study by [12] also examined social relationships and susceptibility in social networks using simple contagion models. The methodology of this study consists of two main stages: initially, each individual temporarily adopts the spreading information or decides to return to their original state. Subsequently, in the second stage, individuals make a final and definitive decision regarding acceptance or rejection of the information.

The study [14] analyzed the diffusion of information and its relationship with users exhibiting higher-than-average activity during the 2019 Polish parliamentary elections. Although these users showed increased responsiveness to political leaders and influential users in political content, their overall activity level was not significantly higher than that of ordinary users.

To prevent and detect early propagation of offensive content, the study [22] examined the diffusion of such content on social media by considering follower networks, structural features, user interactions, and cognitive content characteristics. Their results were compared with tweet diffusion patterns in other content domains. Notably, violent tweets tend to become popular through large chains of users with relatively few followers, which differs from other domains and complicates prediction. Moreover, the research [20] studied the spread of offensive content on social networks by exploring four behavioral adoption mechanisms among users: generalized reciprocity, direct reciprocity, leader-mimicry, and peer imitation.

Some studies have examined hybrid models that combine simple and complex contagion processes. One such study, focusing on the spread of viruses and epidemics alongside information diffusion, divided the network into two layers to separately analyze epidemic and informational spread [3]. Additionally, in [15], a transmission model incorporating both simple and complex contagion was introduced. In this model, individuals' adoption states are categorized into simple contagion, complex contagion, and a hybrid of the two. A parameter θ is defined to represent the contagion threshold for each node, computed from a distribution $Q(\theta)$. If $Q(\theta) = 1$, the node can

[3]Ignorant-Spreader-Recovered

be influenced by a single infected neighbor, corresponding to simple contagion. If $Q(\theta) > 1$, the node requires multiple infected neighbors to become infected, representing complex contagion. In the context of spreading various phenomena through social networks, the study [1] proposed a hybrid model for the diffusion of beliefs. Simulations performed on different network structures demonstrate that the spread of beliefs can be classified into simple or complex contagion depending on the nature of the beliefs involved.

The branching process is a type of stochastic process widely used for modeling proliferation phenomena. The study by [8] investigates complex contagion utilizing a multitype branching process (MTBP). Their subsequent work extended the analysis to more general networks [9]. Given the binary nature and threshold-model dependency in analyzing complex contagion, [6] introduced a newer, more general model independent of threshold constraints. Their model is based on a leader-follower consensus (LFC) framework, analyzing system behavior according to nodes' adherence to the leader, influenced by network features such as clustering coefficient. The network used in their study was a Watts–Strogatz model with 240 nodes.

Considering real-world networks' tendency to exhibit scale-free properties, a potential issue arises regarding diffusion. One stage in scale-free network growth is incremental attachment, which can induce polarization by creating minority and majority nodes. Consequently, minority nodes have reduced access to information, potentially limiting contagion among them. This issue is addressed in [23], which examines different network growth methods and types of contagion between groups. Their simulations are based on synthetic networks, initially modeled as Erdős–Rényi random graphs, then transformed into various models by different growth mechanisms. The main finding relates to how network properties and contagion type affect equal information access across individuals-for example, equality is less in simple contagion than in complex contagion. Given the structural importance of networks in contagion processes, ensuring equitable information access could be beneficial for social network development, enabling all individuals to access information fairly.

One well-known problem in contagion studies is Influence Maximization, introduced in 2003 [10]. The main goal is to identify and select k nodes in a graph that maximize influence spread among other nodes. The influence of a node set, such as A, is denoted $\sigma(A)$. Although influence maximization is NP-hard, approximation algorithms have been proposed. The approximation factor achieved in the original study is $(1 - 1/e - \epsilon)$. Their approach uses a greedy hill-climbing algorithm. This problem is relevant in any social network where diffusion occurs, including applications like advertising, information dissemination, and simple contagion-related issues. For example, it applies to epidemic control via mass vaccination. The study [16] introduced an algorithm using multi-level optimization and parallelization to estimate this problem in vaccination contexts. They defined EPICONTROL, derived from influence maximization, for vaccination strategies. Utilizing the submodularity property, they extended the greedy algorithm. It was proven in [10] that if the cost function f, which measures selected nodes' influence, is submodular, a $(1 - 1/e)$ approximation is achievable. Furthermore, [16] demonstrated that if the underlying graph is a rooted tree, the vaccinated nodes and consequently the saved nodes form a submodular set. Their data included social relationship networks analyzed for public health, as well as networks beyond epidemic applications. Their results showed about a 98% reduction in infections at urban scales. The study [19] examined optimal initial node selection in

random graph models, focusing on nonsubmodular models, unlike the Linear Threshold Model and Independent Cascade Model. Their model utilized the Contagion r-Complex and Fixed Threshold Models. Their findings indicate that if the network is not too dense, their strategy identifies optimal nodes within a community.
Another NP-hard problem concerns simultaneous diffusion of two contagions in a social network. In [2], the authors address minimizing the total number of new infections under a limited vaccination budget. Their approach combines linear programming to find an optimal solution and heuristic methods to generalize the set cover problem. They used datasets including Jazz, FB-Politicians, Wiki, Astroph Emails Enron, Epinions, and 0811Slashdot. Initially, 20 nodes were infected in one of three states, with the rest susceptible (state zero).
The summary of related works of complex contagion and behavior analysis with respect to their application is demonstrated at table 1

Table 1: Studies on various applications of complex contagion

Contagion Topic	**Year**	**Reference**
Weight loss	2024	[5]
Smoking behavior	2011	[11]
Smoking behavior	2024	[13]
Medical information dissemination	2023	[4]
Medical information dissemination	2022	[21]
Service adoption	2018	[7]
Information and advertising diffusion	2019	[17]
Information dissemination	2021	[12]
Information dissemination	2023	[14]
Spread of offensive behaviors	2022	[22]
Spread of offensive behaviors	2022	[20]
Information spread concurrent with epidemic	2022	[3]
Spread of beliefs	2024	[1]

3 Proposed Method

Based on the previous studies, most existing research has primarily focused on analyzing the influential factors during the spread of complex contagion.
One of the most critical factors influencing contagion is the degree of each node. The number

of individuals a person is connected to can have either a direct or inverse effect on the contagion dynamics. In this regard, the number of infected neighbors of a given node also plays a significant role in determining whether the node becomes infected.
Another fundamental factor in contagion dynamics is the transmission probability. In simple contagion, this probability can often be defined based on the nature of the virus and environmental conditions. However, in complex contagion, the mechanisms underlying diffusion cannot be easily reduced to a fixed probabilistic parameter. Instead, this type of contagion is shaped by more intricate dynamics and is more profoundly influenced by the underlying network structure and the relational characteristics of individuals. Therefore, to conduct simulations and analyze how contagion unfolds across different networks, we consider various forms of transmission probability.

3.1 Transmition Probabilities

In the simplest form of transmission, we may assume that all nodes have equal and uniform influence. In a network with N nodes, the probability of transmission can be defined as shown in Equation 1:

$$p_{transmition} = \frac{1}{N} \tag{1}$$

In the next case, one of the most important structural features of the network-node degree-is incorporated into the contagion model. The number of neighbors a node has can have both positive and negative effects on transmission. Various scenarios can be investigated under this condition. Specifically, when the transmission probability varies proportionally with the node degree, high-degree nodes contribute more significantly to the spread. This assumption is particularly relevant in online social networks such as X, where users with a large number of followers tend to have greater influence and acceptability. Assuming that node i has degree k_i, the probability of transmission is computed using Equation 2, where L denotes the total number of links in the network. In this case, a smaller L results in a higher transmission probability for individual nodes, since fewer connections imply a more concentrated influence per node. For example, in a star topology, only one node acts as the central influencer and thus should carry a much higher transmission potential. However, due to the inherent complexity of this type of contagion, setting the probability of transmission to 1 is neither realistic nor desirable. Therefore, we introduce a hyperparameter denoted as bias to prevent this issue. This hyperparameter can be adjusted depending on the specific application.
An alternative scenario involves an inverse relationship between transmission probability and node degree. This assumption can be more realistic in human social dynamics, as individuals with numerous relationships must invest more time and resources to influence others, potentially decreasing their effective transmission impact. The corresponding transmission probability for this case is defined in Equation 3.

$$p_{transmition} = \frac{k_i}{L} * (1 - bias) \tag{2}$$

$$p_{transmition} = \frac{1}{k_i} * (1 - bias) \tag{3}$$

Another influential factor we incorporate into transmission probability is betweenness centrality. This network feature captures the importance of a node in bridging separate components of the graph. For example, a node that acts as a bridge between two disconnected modules can play a pivotal role in facilitating cross-component contagion. The value of this probability ranges from 0 to 1. To avoid the possibility of setting the transmission probability to 1, we again apply the bias hyperparameter. The final probability model using betweenness centrality is provided in Equation 4:

$$p_{transmition} = C_B(v) * (1 - bias) \tag{4}$$

4 Results

The results observed in this study demonstrate the heterogeneous and multifaceted impact of various network factors on complex contagion across different network structures and application domains. The effects of the influcnece of network properties during contagion with respect to the formulated transition probabilities on various networks are demonstrated in this chapter.

4.1 Node Degree Effects

One of the critical factors influencing complex contagion is the node degree and the infection degree over time. Across different network structures-and even within the same model but on different networks-node behavior varied significantly. Except for the Erdős–Rényi (ER) random network which is shown in figure 1, node degree alone does not significantly influence the contagion process. Temporal variations in infection degree showed no consistent pattern in most networks, with the exception of the ER random network and the dense small-world network (Figure 2), which exhibited an increasing trend.

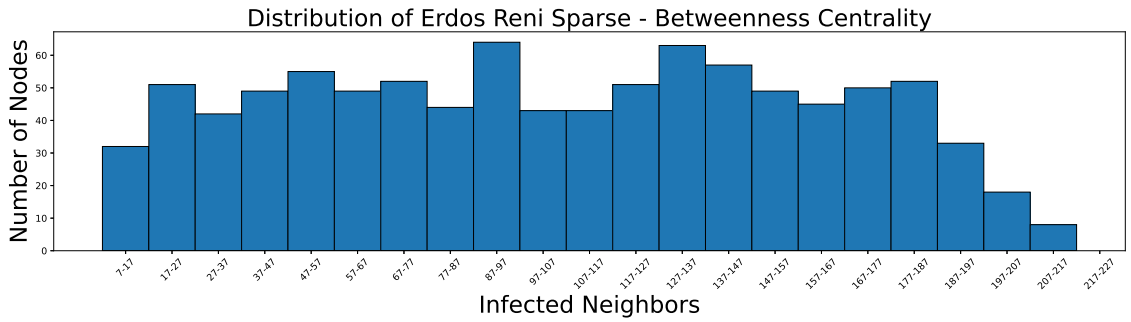


Figure 1: Contagion using direct effect with betweenness centrality

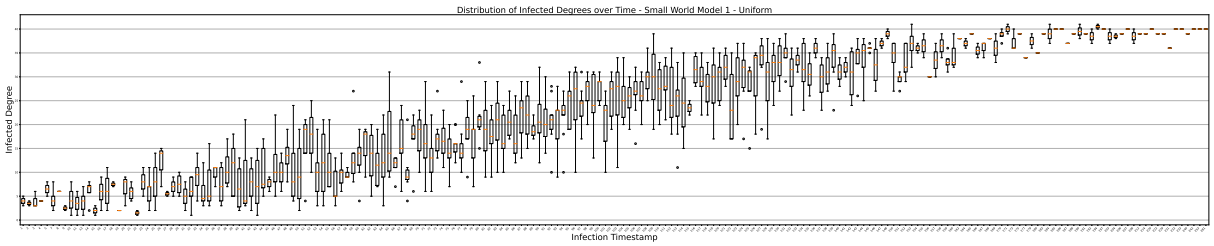


Figure 2: Contagion on small world network with uniform distribution

In contrast, the number of infected neighbors of each node at the time of infection displayed a more structured pattern. These changes are summarized in Table 2.

Network Type	**Infection Degree Trend**
Dense and Sparse Erdős–Rényi	Uniform
Small-world	Uniform
Barabási–Albert	Decreasing (In some cases Normal Distribution)
Price Network	Increasing
Real Networks (Erdos, Enron, Pol)	Decreasing

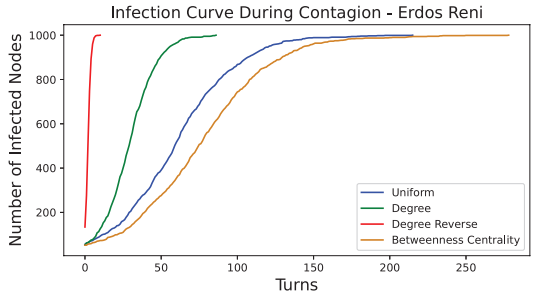
Table 2: Summary of infection degree trends at the time of infection

4.2 Network Structure Effects

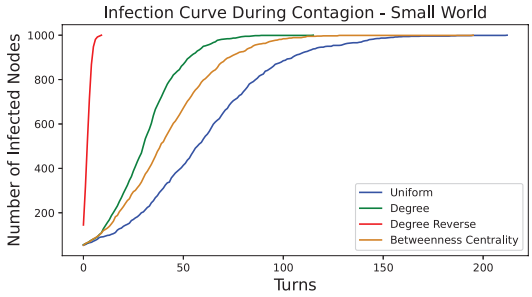
Network structure plays a pivotal role in determining the dynamics of contagion. In random networks, contagion progresses with a lower growth rate compared to scale-free networks. Small-world networks exhibit behavior similar to random networks; however, when transmission is directly influenced by betweenness centrality, the rate of change in node states is higher than that of random networks.

In scale-free networks, the impact of betweenness centrality on contagion is more pronounced than in random networks. Additionally, introducing directionality into scale-free networks significantly slows down the contagion process.

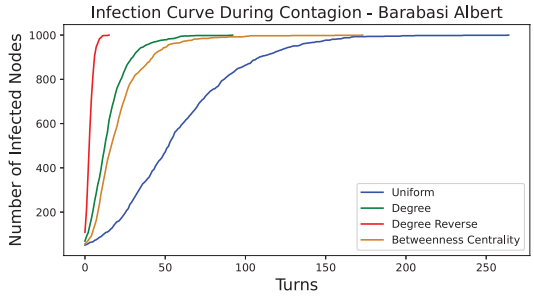
The trend of infection degree variation is more regular in random networks compared to scale-free networks. Moreover, in small-world networks, increasing the average degree results in infection degree trends resembling those of random networks. Random networks do not exhibit



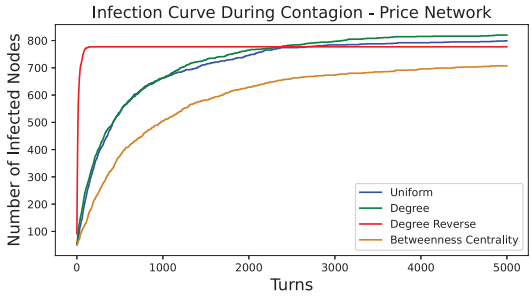
(a) Erdős–Rényi random network



(b) Small-world network



(c) Barabási–Albert scale-free network



(d) Price scale-free network

Figure 3: Infection progression curves across different network structures

a specific pattern of infection degree variation relative to the number of nodes, regardless of time or overall contagion progression. In contrast, scale-free networks display decreasing, increasing, or normally distributed patterns of infection degree.

To enable a more controlled comparison of how network structure affects the contagion process, networks based on the Erdős–Rényi, Watts–Strogatz (small-world), Barabási–Albert, and Price models have been generated-ensuring all networks have an equal number of links. Then their contagion dynamics have been compared.

Figure 3 shows the infection progression over time for each of the four networks and across different contagion models. The order of contagion modes leading to full infection (i.e., when all nodes become infected) is consistent across all networks. However, the slopes of their infection curves differ. The transition of nodes from susceptible to infected states in the Erdős–Rényi random network occurs with a gentler slope, likely due to the more uniform distribution of nodes. The small-world network also exhibits a mild infection curve, but when transmission depends on betweenness centrality, its slope is steeper than that of the random network.

In contrast, the scale-free networks-Barabási–Albert and Price-exhibit steeper infection growth in early stages, with a slowing slope in later stages. Moreover, betweenness centrality has a more significant effect on contagion in the Barabási–Albert network than in the others, emphasizing the role of central nodes in scale-free topologies. Finally, the Price network does not reach full infection and demonstrates the lowest contagion speed among the four network types.

4.3 Hub Effects

The final factor examined in this study is the role of hubs. We investigated the influence of hubs using three different scenarios. Results indicate that, in the Barabási–Albert network, the presence of hubs in the initial infected population does not significantly accelerate contagion. This influence is minimized in the dense variant of the BA network.
However, when all initially infected individuals are hubs, the contagion spreads significantly faster compared to the case with randomly selected initial infections. In contrast, hubs were found to have negligible effect in the Price network, and no specific pattern was observed in contagion dynamics as a result of their presence.
In this study, hubs are defined as the top 1% of nodes with the highest degree. The simulation procedure follows the same setup as in previous sections, and the analyzed networks include two scale-free structures: the Barabási–Albert and Price networks. The structural properties of these networks remain unchanged throughout the analysis.
Initially, for each simulation, the number of hubs that also belong to the group of infectious hubs[4] have been identified. Since the total number of nodes in each network is fixed at 1000, there are exactly 10 hubs and 10 infectious hubs in each case. The number of shared nodes between these two groups across four simulated scale-free networks is reported in Table 3.
According to the results, hubs have the strongest influence in scenarios where the contagion depends on betweenness centrality, and the least influence when there is an inverse relationship between infection probability and node degree. In the Price networks, hubs had no impact on the simulated contagion process, and the infectious hubs were entirely disjoint from the network hubs.

5 Conclusion

The results of this study highlight the broad influence of network-related factors on complex contagion dynamics. The number of infected neighbors for each node was examined in two ways: (1) over time during the diffusion process, and (2) at the moment each node becomes infected.
In random networks, the number of infected neighbors over time shows an increasing trend, whereas in scale-free networks, the pattern becomes irregular and inconsistent. When examining the number of infected neighbors at the point of infection for each node in scale-free networks, the values change without displaying a distinct pattern. Furthermore, in these networks, the changes of the number of infected neighbors with respect to all infected neighbors at the moment of infection, exhibits various behaviors-such as decreasing trends or alignment with a normal distribution. However, when tracked over time during the contagion process, these values fluctuate erratically, without any clear trend.
Contagion spreads more slowly in random networks, while scale-free networks facilitate faster propagation. However, in directed scale-free networks, the diffusion process is significantly slower, and often fails to reach full infection of all nodes.

[4]Infectious hubs are defined as the top 1% of nodes that have infected the most other nodes.

Network Type	**Contagion Mechanism**	**Shared Nodes**
Barabási–Albert (Dense)	Uniform Distribution	8
	Direct Relation with Degree	9
	Inverse Relation with Degree	4
	Direct Relation with Betweenness Centrality	10
Barabási–Albert (Sparse)	Uniform Distribution	6
	Direct Relation with Degree	8
	Inverse Relation with Degree	1
	Direct Relation with Betweenness Centrality	10
Price Network	Uniform Distribution	0
	Direct Relation with Degree	0
	Inverse Relation with Degree	0
	Direct Relation with Betweenness Centrality	0

Table 3: Number of shared nodes between structural hubs and infectious hubs in standard complex contagion simulations

The influence of hubs in scale-free networks also varies. In directed networks, hubs do not significantly contribute to the contagion process. However, in undirected scale-free networks, when all initially infected nodes are hubs, the spread is accelerated, indicating their potential role in boosting the speed of contagion under specific conditions.

6 Future Works

To overcome the limitations of the current study and to improve the understanding of complex contagion, additional factors can be considered. The factors influencing contagion may be individual-specific, depending on each person's prior information and experiences. Furthermore, the type and strength of interactions between individuals play a crucial role, which can be incorporated by assigning weights to network edges. To achieve a more realistic simulation of contagion dynamics, temporal networks may be employed, capturing the evolution of connections over time. Moreover, future studies can consider multi-state contagion processes, where individuals may not be simply classified as susceptible or infected, but may simultaneously be exposed to competing ideas or beliefs, such as the spread of two opposing opinions vying for dominance within the same network.

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