Competing Complex Contagions on Small-World Networks

GROUP-25

1. Introduction

The spread of behaviors, ideas, and innovations through a population—known as **social contagion**—is shaped by both individual decision-making and network structure. While traditional models like the SIR or independent cascade models assume that a single exposure is often enough to trigger adoption (**simple contagion**), many real-world behaviors—such as joining a protest or adopting a new technology—require **multiple reinforcing exposures**. These are captured by **complex contagion models**, where individuals adopt only after a threshold number or fraction of their contacts have already adopted.

Network topology plays a critical role in contagion dynamics. The **Watts-Strogatz small-world model** is widely used to study such processes because it captures both **clustering** and **short path lengths**, features typical of real-world social networks. While long ties accelerate simple contagions, **Centola and Macy (2007)** showed that they can **hinder complex contagions**, since clustered neighborhoods provide stronger reinforcement.

Recent studies have explored **competitive contagion**, where multiple ideas or behaviors compete for adoption. **Melnik et al. (2013)** introduced multi-stage models for complex diffusion, and **Weng et al. (2013)** examined how network modularity impacts meme competition. **Karsai et al. (2014)** and **Gleeson et al. (2014)** further examined how structural and temporal dynamics affect adoption thresholds and collective behavior.

Despite this progress, the interaction between **competing complex contagions** in **small-world networks** remains underexplored—particularly under different adoption rules. Our project addresses this gap by simulating two contagions (A and B) spreading over a small-world network, using both **exclusive** (only one behavior can be adopted) and **non-exclusive** (both can be adopted) scenarios. We analyze how **thresholds** and **rewiring probabilities** affect the competition, dominance, or coexistence of the two contagions.

2. Methodology

2.1 Overview

To investigate how two competing complex contagions spread across a network, we designed an agent-based simulation. The model builds upon Centola and Macy's (2007) framework, extending it to allow simultaneous diffusion of two contagions (A and B) over a Watts–Strogatz small-world network. We vary parameters such as rewiring probability, contagion thresholds, and exclusivity of adoption to understand how these influence cascade dynamics.

2.2 Network Topology

We generate a **Watts–Strogatz network** with **N** = 1000 nodes:

- **k** = 10 (each node initially connected to 10 nearest neighbors)
- **p** ∈ [0, 0.3] (rewiring probability controlling randomness)

This network allows us to smoothly transition from a regular lattice (high clustering) to a random graph (many long ties), which is essential for testing structural effects on complex contagions.

2.3 Agent Behavior and Contagion Rules

Each agent can adopt contagion A and/or B. Adoption depends on a **threshold rule**:

- A node adopts contagion A if ≥ θ_A fraction of its neighbors have adopted A.
- A node adopts contagion B if ≥ θ_B fraction of its neighbors have adopted B.
- Thresholds are fixed globally: θ_A, θ_B ∈ [0.1, 0.5].

We consider two adoption scenarios:

- **Exclusive adoption**: a node can adopt only one contagion (whichever reaches threshold first).
- **Non-exclusive adoption**: a node may adopt both if each threshold is met.

The model uses **synchronous updates**, where all nodes evaluate their status and update simultaneously at each timestep.

2.4 Initialization

- Initially, a small number of nodes (default: 10 each) are randomly seeded with contagion A and contagion B.
- The seeds for A and B are **disjoint** to ensure fairness.
- Simulations are run until one of the following:
  - Both contagions saturate (no further adoptions)
  - Maximum number of steps (default: 100) is reached

2.5 Simulation Procedure

For each value of rewiring probability p, we repeat the following steps:

1. Generate a Watts–Strogatz network with given p.
2. Randomly assign initial seeds for A and B.
3. Simulate contagion spread step-by-step using the threshold rules.
4. Record:
   - Fraction of nodes infected with A and B over time
   - Final adoption rate for each contagion
   - Total steps to convergence

Each simulation is repeated **5 times** per p to reduce noise, and we compute the **mean** and **standard deviation** of adoption outcomes.

2.6 Evaluation Metrics

We analyze results using:

- **Adoption curves**: percentage of nodes with A/B over time
- **Final adoption fraction**: percentage of nodes infected with A or B at the end
- **Dominance**: which contagion reaches more of the population
- **Time to saturation**: number of steps to reach stability

These metrics allow us to assess the **efficiency**, **reach**, and **competitive advantage** of each contagion under different network structures.

2.7 Experiment Design:

We'll repeat batch simulation:

- Same network size and `p ∈ [0, 0.3]`
- Same thresholds (θ_A=0.2, θ_B=0.3)
- **Only difference**: set `exclusive=False`

This lets us directly compare:

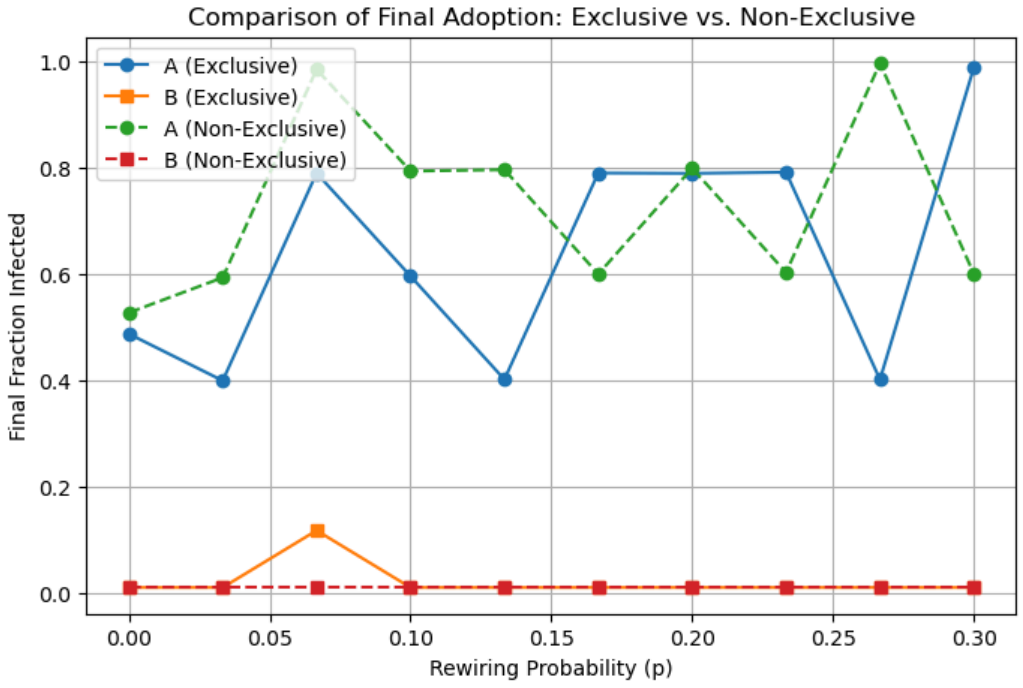
Adoption Mode	**Expected Dynamic**
Exclusive	One contagion often dominates (winner-takes-all)
Non-exclusive	Both contagions may coexist or overlap

3. Results

We present the outcomes of simulations comparing the spread of two complex contagions (A and B) on small-world networks with varying rewiring probabilities. The key variables were:

- **Rewiring probability** p∈[0.0,0.3]p \in [0.0, 0.3]p∈[0.0,0.3]
- **Contagion thresholds** θA=0.2,θB=0.3\theta_A = 0.2, \theta_B = 0.3θA=0.2,θB=0.3
- Two adoption rules: **exclusive** (one contagion per node) and **non-exclusive** (both allowed)

Each configuration was run five times to reduce stochastic noise, and final adoption fractions were averaged.



3.1 Adoption Under Exclusive Rule

When nodes could adopt **only one** contagion, the results showed a competitive dynamic. The simulation outcomes indicate:

- **Contagion A**, with the lower threshold (θ = 0.2), consistently outperformed contagion B.
- At **low rewiring** (p ≈ 0), both contagions spread slowly and adoption remains limited.
- As **rewiring increases**, contagion A benefits from faster global spreading due to lower reinforcement requirements and takes over the network.
- Contagion B, requiring more neighbors to adopt it, often fails to reach critical mass in high-randomness networks.

This supports the idea that **lower-threshold contagions dominate** in competitive settings, especially when long ties reduce local clustering.

3.2 Adoption Under Non-Exclusive Rule

Allowing nodes to adopt **both contagions** significantly changed the dynamics:

- Both A and B reached **higher adoption levels** compared to the exclusive case.
- Contagion A still maintained a lead due to its lower threshold, but **contagion B improved its reach** in all rewiring conditions.
- The competition became less zero-sum: **coexistence became common**, especially in the intermediate rewiring regime (p ≈ 0.1–0.2).
- Saturation occurred earlier (fewer steps), suggesting that **parallel adoption accelerates diffusion**.

This suggests that **relaxing exclusivity leads to richer and more inclusive diffusion patterns**, and reduces the winner-takes-all effect.

3.3 Effect of Rewiring Probability

The rewiring parameter p shaped contagion dynamics in both adoption modes:

Rewiring Level	Observation
Low p (~0.0)	Spread is slow; high clustering impedes contagion growth.
Medium p (~0.1–0.2)	Balanced: allows for local reinforcement and some long-range shortcuts. Best for B under non-exclusive mode.
High p (>0.25)	Contagion A dominates due to fast reach and low threshold; contagion B suffers from lack of reinforcement.

4. Discussion

Our results highlight how network structure and adoption rules jointly shape the spread of competing complex contagions. By simulating two contagions with different adoption thresholds across small-world networks, we uncover several key insights into competitive diffusion dynamics.

4.1 Thresholds Determine Competitive Advantage

The contagion with the **lower threshold** (Contagion A) consistently outperformed the one with the higher threshold (Contagion B). This confirms findings by **Gleeson et al. (2014)**, who showed that lower adoption barriers confer a decisive advantage, especially in less clustered networks. The threshold asymmetry created a **first-mover effect**: Contagion A spread early and broadly, often saturating the network before Contagion B could take hold.

4.2 Network Structure Moderates Contagion Spread

We observed a clear influence of the **rewiring probability ppp** on both speed and reach of contagion spread:

- At low ppp, local clustering limits reach, especially for Contagion B, which requires neighbor reinforcement.
- At high ppp, long ties benefit the lower-threshold contagion (A), enabling fast, global spread with minimal reinforcement.
- **Intermediate values** of ppp (e.g., 0.1–0.2) provided a sweet spot for **coexistence** under the non-exclusive scenario — confirming **Centola & Macy (2007)**'s observation that **too much randomness disrupts complex contagion**.

Thus, **network randomness modulates the reinforcement environment**: it helps simple contagions, but hurts complex ones needing confirmation from multiple neighbors.

4.3 Relaxing Exclusivity Enables Coexistence

A major contribution of our project is the comparison between **exclusive** and **non-exclusive** adoption rules. In the exclusive case, contagion A often "wins" entirely, suppressing B. But under non-exclusive rules:

- Contagion B gains more ground, especially at moderate ppp.
- Final adoption rates of both contagions increase.
- Competition becomes **non-zero-sum**, allowing both behaviors to coexist in the network.

This supports ideas from **Weng et al. (2013)** and **Karsai et al. (2014)**: that real-world social influence is **not strictly competitive**, and agents may adopt **multiple overlapping behaviors**.

4.4 Implications

These findings suggest that in designing interventions (e.g., public health, marketing, or social movements):

- Lower-threshold ideas are likely to dominate in highly connected or noisy networks.

- Structural design (like clustering and community reinforcement) can be **used strategically** to promote high-threshold behaviors.
- **Allowing coexistence** (instead of enforcing binary choices) may improve overall adoption in populations.

5. Limitations

While our model reveals important insights about competing contagions, it also has several limitations that should be acknowledged:

5.1 Simplified Agent Behavior

Agents in the model follow deterministic threshold rules and make synchronous decisions. In reality, human behavior is **heterogeneous and stochastic**: adoption may depend on timing, memory, uncertainty, peer influence strength, or external signals.

Future work could incorporate probabilistic adoption functions, asynchronous updates, or heterogeneous thresholds across agents.

5.2 Fixed Network Topology

We use static Watts–Strogatz networks to model social structure. However, real social networks are often:

- **Dynamic** (connections change over time)
- **Heterogeneous** (scale-free, with hubs)
- **Layered or multiplex** (e.g., online vs offline ties)

An extension could study competing contagions on dynamic or scale-free networks, or in multilayer models where contagions spread differently across layers.

5.3 Two-Contagion Limit

Our model considers only two contagions. In real systems, individuals are exposed to **many competing behaviors**, often interacting in more complex ways (e.g., reinforcement, inhibition, synergy).

Future models could test multiple contagions with network-based priority, spreading order, or mutation effects.

5.4 No Empirical Calibration

The model is theoretical and does not use real-world behavioral or network data. While the mechanisms are plausible, calibration against actual adoption patterns or network traces could improve realism.

Using empirical data (e.g., Twitter cascades or social app adoption) could validate and fine-tune the model.

6. Conclusion

This project explored how two competing complex contagions spread on small-world networks, extending the threshold-based model by Centola and Macy (2007). We showed that:

- **Lower-threshold contagions** consistently dominate under exclusive adoption rules.
- **Rewiring probability** affects which contagion prevails and how fast the system saturates.
- Allowing **non-exclusive adoption** facilitates **coexistence** and increases adoption for both contagions.
- **Intermediate network randomness** supports more balanced diffusion outcomes.

Our work contributes to the growing literature on **competitive diffusion in structured populations**, offering a modular simulation tool to study interaction effects under different assumptions. While simplified, the model provides insight into how thresholds, structure, and exclusivity interact to shape the social spread of competing ideas.

Notes

All code available on our git repository for reproduction.
https://github.com/m-naveed68/Final-Project-Competing-Complex-Contagions-on-Small-World-Networks-

There, you find this report and the jupyter notebook.

With the jupyter notebook you can run and reproduce our results with a single click.

References

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