Diffusion on Sparse Temporal Network

(Dated: March 14, 2024)

This project investigates the dynamics of simple diffusion on Erdos-Renyi temporal networks with memory. The network's temporal nature allows for the exploration of how past interactions influence future interactions on the network. I employ a model that incorporates memory into the network's evolution, where edges remember past interactions and use this information to guide formation of future connections. I simulate diffusion processes on these temporal networks, comparing them to static Erdos-Renyi networks with the same network density. My results reveal insights into the role of memory in shaping network dynamics and offer implications for real-world systems, such as interpersonal interaction, where memory plays a significant role in process dissemination.

I. INTRODUCTION

Temporal networks serve as prevalent models for illustrating the structure of complex systems [1]. These networks are characterized by a dynamic architecture that undergoes changes throughout time, reflecting the evolving nature of interactions within the system. A prominent feature of systems undergoing temporal evolution is their memory, which signifies the degree to which the system's current state and future development rely on its historical states [2, 3].

In temporal networks, the state of a node or an edge may depend not only on its own historical states but also on the states of other nodes or edges, respectively. This observation introduces the concept of network memory, denoting the reliance of a temporal network's structure on its past interactions and states. Recent studies indicate that network memory plays a significant role in phenomena such as diffusion and epidemics, influencing how information spreads and how diseases propagate within these networks [2, 4].

Understanding the impact of network memory on diffusion processes is crucial for various applications, including the modeling of social networks, the spread of information in online platforms, and the containment of epidemics. This paper aims to investigate the dynamics of simple diffusion on Erdos-Renyi temporal networks with memory, exploring how memory affects the spread of a disease and the overall structure of the network.

II. THEORY

In this section, we briefly present the foundational concepts necessary for understanding our approach to modeling diffusion on temporal networks. We begin with an overview of random networks, highlighting their role as a benchmark for more complex network models. We then delve into epidemic modeling, discussing the Susceptible-Infectious (SI) model and its relevance to understanding the spread of diseases on networks. Next, we introduce temporal network representations, which capture the time-varying nature of interactions between edges. Finally, we touch upon information theoretic measures, which provide a framework for quantifying the uncertainty associated with predicting future values in stochastic temporal networks. Together, these concepts form the basis for our analysis of diffusion dynamics on temporal networks.

A. Erdos-Renyi Network

The Erdos-Renyi (ER) model is a fundamental simple random graph model that provides a theoretical framework for understanding the structure of networks [5]. In an Erdos-Renyi network, nodes are connected randomly to form edges, leading to a network where the presence of an edge between any pair of nodes is determined by a probability parameter.

Formally, an Erdos-Renyi network, $\mathcal{G}(N, p)$, is defined by two parameters: the number of nodes, denoted as N, and the probability of an edge existing between any pair of nodes, denoted as p. Equivalently, the probability of generating a graph with N nodes and M edges is given by $p^M(1-p)^{\binom{N}{2}-M}$.

Erdos-Renyi networks exhibit several key characteristics. First, as the probability parameter p increases, the network becomes denser, with more edges connecting nodes. Second, Erdos-Renyi networks are characterized by a Poisson degree distribution, where the number of edges incident on a node follows a Poisson distribution. Finally, Erdos-Renyi networks are homogeneous, meaning that all nodes have a similar degree of connectivity on average. The distribution of the degree of any nodes follows a binomial distribution:

$$P(k) = \binom{N-1}{k} p^k (1-p)^{N-1-k}, \tag{1}$$

where N is the total number of nodes in the graph. As N tends to infinity, and $Np = \lambda$ remains constant, this distribution approaches a Poisson distribution:

$$P(k) = \frac{\lambda^k e^{-\lambda}}{k!} \tag{2}$$

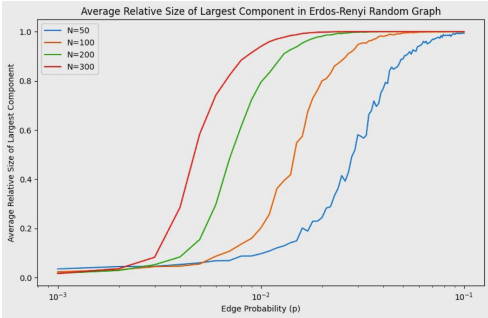


FIG. 1: Size of the largest component as a function of N and p in simple random networks. This is the average size of the largest component averaged over 50 iterations.

The distribution of connected components and the presence of a giant component are key factors in understanding diffusion on networks, especially in Erdős–Rényi networks. These components represent clusters of nodes that can influence each other's behavior. The critical probability p_c in Erdős–Rényi networks signifies the threshold at which a giant component emerges. Below p_c, the network consists of small isolated components, limiting diffusion. Above p_c, a giant component emerges, facilitating efficient diffusion. Thus, $p_c = \frac{\ln N}{N}$ is a sharp threshold for the connectedness of $G(n, p)$ model.

Despite its simplicity, the Erdos-Renyi model has been instrumental in the study of complex networks, serving as a benchmark for comparing more intricate network models. In this study, we focus on temporal dynamics of Erdos-Renyi networks with a connection probability below the critical value p_c. By examining networks below p_c, we gain a deeper understanding of how random shuffling in network structure impacts diffusion processes, offering valuable insights for modeling and predicting diffusion in real-world scenarios.

B. Diffusion Modeling

Diffusion modeling plays a crucial role in understanding how information, behaviors, or diseases spread through networks[6, 7]. It involves studying the dynamics of these processes and predicting their outcomes based on the structure of the underlying network. Various models exist to describe diffusion, each with its assumptions and applicability[8]. For example, the classic SIR (Susceptible-Infectious-Recovered) model is commonly used to study disease spread, where individuals transition between these states based on certain probabilities. Susceptible are healthy individuals who have not yet been exposed to the pathogen, Infectious individuals have been exposed and can therefore spread the pathogen, and Recovered individuals have previously been infected but have since recovered and are no longer infectious.

In this paper, we simplify the diffusion model by adopting a two-state approach known as the Susceptible-Infected (SI) model. In this model, individuals are classified as either susceptible or infected, with no assumption of recovery. This simplification allows us to focus on the initial stages of diffusion, where the emphasis is on the spread of the infection rather than recovery or other complex dynamics.

Given the SI model, there is a closed form solution for the spreading phenomenon. I follow calculation proposed here [9]. Let $S(t)$ represent the number of individuals susceptible at time t, and $I(t)$ denote the number of individuals already infected. We assume that an average individual has $\langle k \rangle$ contacts in our network. This is an approximation for a general network, but in the case of a simple random network and due to homogeneity, we know that $\langle k \rangle = (N-1)p$. We also assume that the probability of disease transmission from an infected to a susceptible individual in a unit time is β, infectivity rate. If a single node becomes infected at time $t = 0$, we can calculate how many individuals will be infected at some later time t.

The likelihood of the infected node encountering a susceptible individual is given by the ratio $S(t)/N$. Accordingly, the infected person comes into contact with $\langle k \rangle S(t)/N$ susceptible nodes in a unit time. Given that there are $I(t)$ infected individuals transmitting the pathogen at a rate of β, the average number of new infections $dI(t)$ during a timeframe dt is

$$\frac{dI(t)}{dt} = \beta \langle k \rangle \frac{S(t)I(t)}{N} \tag{3}$$

Replacing $s(t) = S(t)/N$, $i(t) = I(t)/N$ to obtain the fractions, we will have:

$$\frac{di}{dt} = \beta \langle k \rangle si = \beta \langle k \rangle i(1-i) \tag{4}$$

Integrating, we obtain:

$$i(t) = \frac{i_0 e^{\beta \langle k \rangle t}}{1 - i_0 + i_0 e^{\beta \langle k \rangle t}} \tag{5}$$

With the initial condition $i_0 = i(t = 0)$, representing the fraction of infected node at the beginning of the process.

Initially, the fraction of infected individuals grows exponentially. This rapid increase is due to infected individuals encountering primarily susceptible individuals, facilitating easy spread. Increasing either the link

density $\langle k \rangle$ or the transmission rate β enhances the pathogen's speed and reduces the saturation time. As time progresses, an infected individual encounters fewer susceptible individuals, leading to a slower growth of i for large t. The epidemic concludes when everyone has been infected.

In this article, the focus lies on temporal networks in the regime where $\langle k \rangle \approx 1$, indicating a sparse network where few connected components exist, and transitions occur between different possible configurations over time. It is intriguing that in this scenario, we observe an S-shaped diagram similar to spread on static networks, as predicted by Eq. 5. This phenomenon highlights the complex interplay between network structure and temporal dynamics, suggesting that even in sparse temporal networks, diffusion processes can exhibit behavior reminiscent of more connected static networks.

C. Temporal Network

I employ an edge-centric framework to incorporate temporal dynamics into networks. Additionally, I assume that network temporal dynamics can be represented through a collection of discrete-time stochastic process. Accordingly a temporal network $\mathcal{G}(t)$ with fixed N nodes, can be conceptualized as a collection of m discrete-time stochastic processes, represented as $\mathcal{G}(t) = \{X_t^\alpha\}_{t=0,1,2,\dots}^{\alpha=1,\cdots,m}$, where $m \leq N(N-1)/2$ denotes the number of distinct pairs of nodes, potential edges, that can be linked over the evolution of network.

It is assumed that each X_t^α represents the stochastic process governing the dynamics of edge α. For simplicity, we assume all connected edges have equal weight, denoted as 1, so x_t^α takes the value of 1 if link α is connected at time t, and 0 otherwise. Thus, the stochastic temporal dynamics of each edge can be represented by a time series or a sequence of 1s and 0s. In general, we can assume that edge dynamics are not independent of each other. Therefore, if we aim to predict the value of an edge, such as edge α at time t, we not only require the history of the dynamics of edge α but also potentially the history of its immediate or even more distant neighbors. This interdependence arises from the complex interactions and dependencies between edges in the network.

However, to further simplify our analysis, we initially assume independence among distinct edges. In this scenario, X_t is generally non-Markovian and relies solely on its own memory. This means that the presence of an edge at time t_2 can be influenced by historical patterns of activity of X_t up to time t_2. To put simply one can argue that if historical patterns show that two nodes interacts frequently, we might predict the an imminent interaction between them. To formalize this, we model X_t as the output of a Hidden Markov Model (HMM) with the observable set $\mathcal{O} \equiv \{0, 1\}$ [10]. To capture the historical context of the edge at time t, we define the L-block history as $\vec{X}^L = [X_t, X_{t-1}, \dots, X_{t-L+1}]$, combining the state of the edge at time t with its preceding $L-1$ states.

With this historical information, we can predict future states of the edge, acknowledging a certain level of uncertainty due to its stochastic nature. For a stochastic process with a memory of length p, the optimal description, by definition, can be expressed as a function of the past p-block observation:

$$\mathbb{P}(X_{t+1}|X_t, \cdots, X_{t=0}) = \mathbb{P}(X_{t+1}|X_t, \cdots, X_{t-p+1}) \quad (6)$$

This implies that an analysis of the preceding p observations of all edges in the network can comprehensively describe the temporal evolution of the network. In other words, to optimally predict the properties and structures of the network at time t, we need access to the preceding p observations of all edges to make the best prediction.

D. Information Theoretic Measures To Describe Stochastic Process

In this section, I introduce fundamental concepts in information theory for the characterization of temporal networks. For a comprehensive exploration, There is an in-depth treatment here [11]. Let X_t denote a random variable governing edge dynamics, where $\mathcal{X}_t$ represents its finite set of possible values (here edge weight). The probability of X_t assuming a particular value x is denoted by $\Pr(x)$. The formal definition of the Shannon entropy of X_t is as follows:

$$H(X) = -\sum_{x \in \mathcal{X}} \Pr(x) \log_2 \Pr(x) \quad (7)$$

It quantifies the uncertainty linked with the random variable X_t. For example, if an edge is always connected with value 1, then there is no uncertainty linked with predicting its future values. Likewise, Y represents another random variable. The conditional entropy is then defined as:

$$H(X|Y) = -\sum_{(x,y) \in \mathcal{X} \times \mathcal{Y}} P(x, y) \log_2 P(x|y) \quad (8)$$

which quantifies the average uncertainty associated with variable X, given that we know Y. This can be the uncertainty associated with predicting future values of edge X given that we know the historical values of a

neighboring edge. The mutual information between X and Y is defined as:

$$I(X;Y) = H(X) - H(X|Y) \tag{9}$$

The mutual information is the average reduction of uncertainty of one edge due to knowledge of another.

Similarly, the total Shannon entropy for sequences of L observations, namely $\vec{X}^L$, is defined as:

$$H(L) = -\sum_{\vec{X}^L} \Pr(\vec{X}^L) \log_2 \Pr(\vec{X}^L) \tag{10}$$

where the summation is taken over all possible blocks of L consecutive observations. Last but not least, the source *entropy rate* is the rate of increase with respect to L of the total Shannon entropy in the large L limit:

$$h_\mu = \lim_{L\to\infty} \frac{H(X^L)}{L} \tag{11}$$

This basically quantify how stochastic is inherent to the edge formation process itself given the fact that we have all the information from its infinite past.

III. EXAMPLES & SIMULATIONS

In this section, we examine a couple of illustrative examples employing the Susceptible-Infectious (SI) model, characterized by an infectivity rate denoted as β. Our focus centers on sparse networks, as diffusion in temporal networks reveals distinctive behaviors within the confines of sparsity. In these regimes, spread of infected nodes may encounter occasional delays as a result of being isolated or not transferred due to infectivity rate, necessitating multiple steps before an edge connects their current node to another node.

Furthermore, there are two distinct time scales involved in the overall process. One pertains to network evolution, while the other relates to the random walker's time scale. We operate within the mixed regime where these two time scales are comparable. Consequently, in each iteration, we update the network structure and configuration, followed by an attempted jump in the random walker's trajectory.

A. Bias Coin - No Memory

This class, illustrated in FIG.2, represents a memoryless process involving coin flips, where the bias of the coin determines the probabilities of an edge in the network being connected or not. Each coin flip is regarded as independent of past flips. Consequently, the probabilities of heads or tails, remain constant and unaffected by the sequence of previous outcomes. To construct the structure of the network in the next time step, we need to toss the coin for $N(N-1)/2$ iterations.

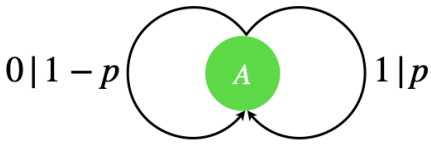


FIG. 2: Biased Coins HMM

It is evident that the average degree $\langle k \rangle$ in a network generated by independently biased coins with N nodes, follows the expression $\langle k \rangle = (N-1)p$. The distribution of the degree of any particular node is given by $\Pr(k) = \binom{N-1}{k} p^k (1-p)^{N-1-k}$. The entropy rate of a biased coin, denoted as h_μ, is given by $H(p)$. Consequently, the overall network's entropy rate can be expressed as:

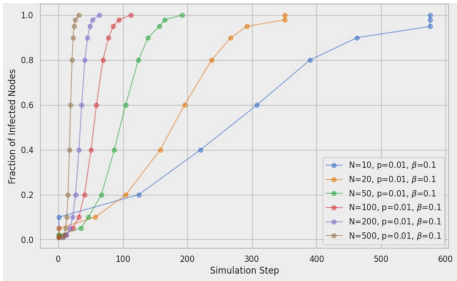
$$h_\mu(\mathcal{G}) = \binom{N}{2} H(p) \tag{12}$$

$$= -\binom{N}{2} [p \log p + (1-p)\log(1-p)] \tag{13}$$

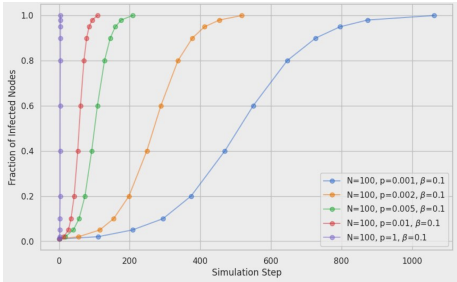
which formalize how unpredictable future network is given its current structure.

In network dynamics, the probability of observing L blocks of consecutive 1s signifies edge persistence, fostering diffusion. In our model, $\Pr(1^L) = p^L$ encapsulates the likelihood of a single edge persisting over a sequence of L time steps. Alternative patterns of activation, such as the sequences $10101010\cdots$ or $110110110\cdots$, among others, present intriguing avenues for exploration in understanding diverse classes of social behavior.

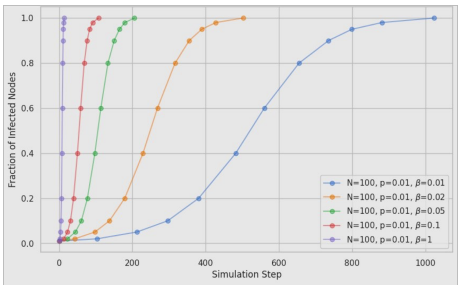
The findings derived from the Susceptible-Infectious (SI) model simulations for different parameters are depicted in Fig. 3. Interestingly, the S-shape derived for spread on static network is also reconstructed for the temporal network. Our analysis is particularly centered on small values of $p \ll 1$, focusing on sparse networks to discern patterns and behaviors unique to this classes of dynamics. Increasing the network size, the probability of edge existence, or the infectivity rate accelerates the infection process. However, further investigation is needed to comprehensively and formally characterize how the speed of propagation depends on these model parameters.



(a) Impact of the size of the network as denoted by N.



(b) Impact of the probability of the edge existence as denoed by p.



(c) Impact of the infectivity rate as denoted by β.

FIG. 3: Simulation results for diffusion over temporal networks generated by an independent biased coin process. For each parameter, we simulated 50 repetitions and plotted the average values

B. Memory 1 network

The Golden Mean Process, illustrated in FIG. 4, is characterized as an order-1 Markov process. In this stochastic model, each coin toss is linked to its antecedent flip. Specifically, a toss of a bias coin(p) is initiated following an outcome of 0, while an outcome of 1 results in the assignment of the value 0.

The average degree, denoted as $\langle k \rangle$, in a network generated by independently Golden Mean, is given by the expression $\langle k \rangle = (N-1)\Pr(X_t = 1)$. By solving the

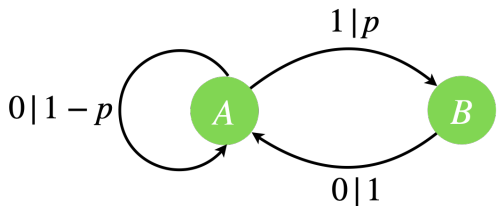


FIG. 4: Golden Mean HMM

stationary state equation $T\vec{\pi} = \vec{\pi}$ for the golden mean process, we can obtain the stationary probabilities of observing a 1 or a 0, where

$$\vec{\pi} = (\pi_A, \pi_B) = (\frac{1}{1+p}, \frac{p}{1+p}) \tag{14}$$

Consequently, $\Pr(X_t = 1) = \pi_B = \frac{p}{1+p}$, and thus $\langle k \rangle = (N-1)\frac{p}{1+p}$. The distribution of the degree of any particular node is given by

$$\Pr(k) = \binom{N-1}{k} \Pr(X_t = 1)^k (1 - \Pr(X_t = 1))^{N-1-k} \tag{15}$$

$$= \binom{N-1}{k} (\frac{p}{1+p})^k (1 - \frac{p}{1+p})^{N-1-k} \tag{16}$$

As $N \to \infty$, binomial distribution approaches a Poisson distribution:

$$\Pr(k) = \frac{e^{-\lambda}\lambda^k}{k!}, \tag{17}$$

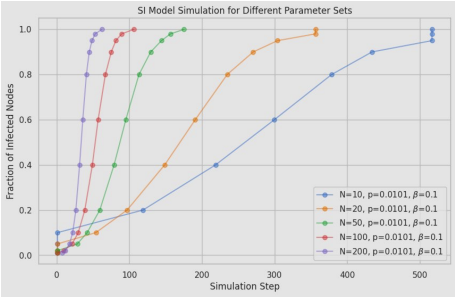
where, $\lambda = \frac{Np}{1+p}$. The entropy rate of a network generated by Golden Mean process can be expressed as:

$$h_\mu(\mathcal{G}) = \binom{N}{2} H(\frac{p}{1+p}) \tag{18}$$

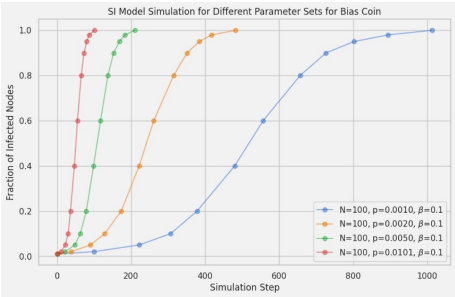
The outcomes of an SI model, as depicted in FIG. **??**, illustrate the temporal evolution of infected nodes in networks generated by Golden Mean process.

The findings derived from the Susceptible-Infectious (SI) model simulations for different parameters are depicted in Fig. 5. Interestingly, the S-shape of the static network is also reconstructed for the this temporal network with unit memory.

We kept average density of network equal to what we had for the previous section so that they can be compared. Although the underlying process is different what we can observe is that the spreading process speed and dynamics closely mimic the process without a memory. However, further investigation is needed to comprehensively and formally characterize how the speed of propagation depends on these model parameters including the memory.



(a) Impact of the size of the network as denoted by N.



(b) Impact of the probability of the edge existence as denoted by $p/(1+p)$.

FIG. 5: Simulation results for diffusion over temporal networks generated by independent golden mean process. For each parameter, we simulated 50 repetitions and plotted the average values.

C. Dependent Edge Interactions

In this section, we aim to relax the assumption of independence in edge processes and introduce a method to integrate dependence into our model. This simple step could make the model more realistic and better able to accommodate real-world phenomena.

We couple edges interactions in two different ways, which we refer to as *local coupling* and *non − local coupling*. For local coupling, we randomly couple each edge in the network with another edge in the network, conditioned on them having one node in common. In the context of social interactions, this can be interpreted as one actor's interactions with another actor influencing the behavior of the first actor in future interactions with a different actor. In other words, if we observe node A forming a connection with a neighboring node B, we might expect that node A will also interact with neighboring node C in the future since we have coupled edge A-C to the history of edge A-B in our network. For non-local coupling, we randomly pair each edge with another edge in the network without any specific condition or requirement. This means that edges can be coupled regardless of whether they share a common node. This can be interpreted such that a regular actor copy another actor patterns of interactions, regardless of being or not being connected to that node.

To determine the state of an edge (connected or not connected) at time t, denoted by X_t^i which is also coupled to X_t^j, we follow a Bernoulli process. With a probability $p_{\text{Bernoulli}}$, we decide whether to replicate one of the previous states of the coupled edge, denoted by $X_{t-\delta t}^j$, or to establish the edge's presence through continuation of its own coin tossing process. Our model includes a memory parameter, denoted as $max\ mem$ (maximum memory length), which determines the length of memory we retain from the edge dynamics. When deciding to copy from memory, we uniformly sample a value from edge X_t^j from the window time starting from $t-1$ to $t - max\ mem$.

$$X_{t+1}^i = Q_t X_{t-\delta t_{ij}}^j + (1-Q_t)Y_t \tag{19}$$

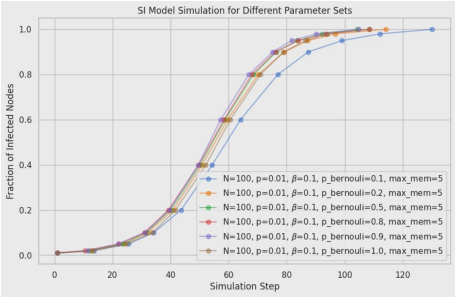
In exploring the network's entropy, an intriguing question arises: Can we analytically express the network's entropy, particularly in cases where edges are paired with each other's history? This seems to be a hard problem given the both complex nature of networks and also the interdependence of edges on each other.

The findings derived from the Susceptible-Infectious (SI) model simulations for different parameters over the network with local edge dependency are depicted in Fig. 6. Interestingly, the S-shape of the static network is also reconstructed here. We kept density of average density of network equal to what we had for the previous section so that they can be compared. We can see that increasing the length of memory does not led to a consistent change in the speed of diffusion, while higher copying from other edges slows down the spread.

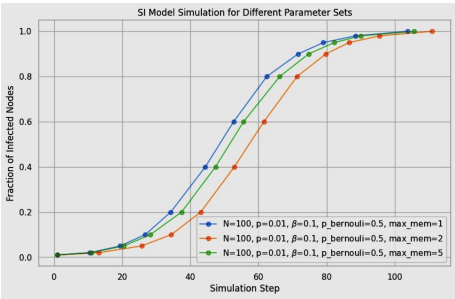
The findings derived from the Susceptible-Infectious (SI) model simulations for different parameters over the network with non-local edge dependency are depicted in Fig. 7. Interestingly, the S-shape of the static network is also reconstructed here. We kept density of average density of network equal to what we had for the previous section so that they can be compared. We see that the impact of copying is much amplified in the non-local interactions rather than the local interaction scenario.

IV. DISCUSSION

The findings of this project shed light on the dynamics of diffusion on temporal networks with memory, particularly in the context of simple network structures and memory effects. By employing an edge-centric approach



(a) Impact of the dependence of edges on each other's history as denoted by $p_{bernouli}$ on local coupling scenario.



(b) Impact of the maximum memory length as denoted by max-mem on local coupling scenario.

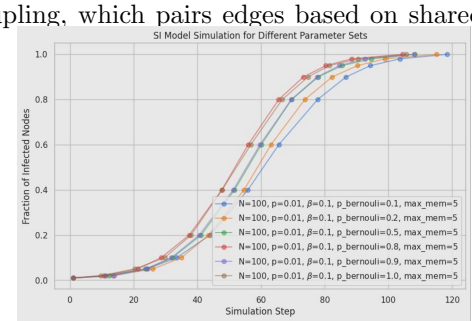
FIG. 6: Simulation results for diffusion over temporal networks generated by locally dependent bias coin process. For each parameter, we simulated 50 repetitions and plotted the average values.

and introducing memory parameters, we were able to simulate the diffusion process and observe distinct behaviors.

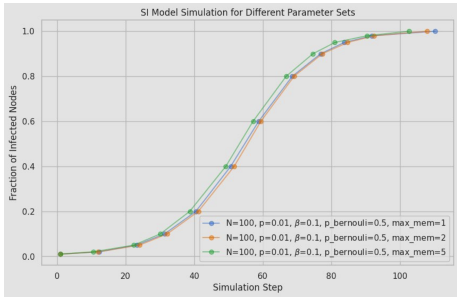
One key observation is the S-shaped pattern in the diffusion process, similar to static networks, as network size, edge probability, or infectivity rate increases. This suggests that these parameters significantly influence the speed and extent of information propagation in temporal networks.

The introduction of memory in edge dynamics further complicates the diffusion process, as edges are no longer independent. This dependence requires considering the historical values of neighboring edges to predict future edge values accurately. We saw some deviations amplified better in local interactions, while others where more significant in non-local interactions.

Our investigation into local and non-local coupling of edges revealed interesting insights into how past interactions between edges influence future interactions. Local coupling, which pairs edges based on shared nodes,



(a) Impact of the dependence of edges on each other's history as denoted by $p_{bernouli}$ on non local coupling scenario.



(b) Impact of the maximum memory length as denoted by max-mem on non local coupling scenario.

FIG. 7: Simulation results for diffusion over temporal networks generated by non-locally dependent bias coin process. For each parameter, we simulated 50 repetitions and plotted the average values.

mimics real-world scenarios where interactions between individuals influence future interactions within the same social circle. Non-local coupling, on the other hand, introduces randomness into edge pairing, representing interactions that are less influenced by past interactions.

Overall, these findings contribute to a better understanding of how temporal dynamics and memory affect diffusion processes in networks. Future research could delve deeper into more complex network structures and investigate how different memory models impact diffusion dynamics.

[1] P. Holme and J. Saramäki, Physics reports **519**, 97 (2012).

[2] M. Tizzani, S. Lenti, E. Ubaldi, A. Vezzani, C. Castel-

lano, and R. Burioni, Physical Review E **98**, 062315 (2018).
[3] N. Masuda and R. Lambiotte, *A guide to temporal networks* (World Scientific, 2016).
[4] O. E. Williams, L. Lacasa, A. P. Millán, and V. Latora, Nature communications **13**, 499 (2022).
[5] P. Erdős, A. Rényi, *et al.*, Publ. math. inst. hung. acad. sci **5**, 17 (1960).
[6] R. M. Bond, C. J. Fariss, J. J. Jones, A. D. Kramer, C. Marlow, J. E. Settle, and J. H. Fowler, Nature **489**, 295 (2012).
[7] D. Centola and M. Macy, American journal of Sociology **113**, 702 (2007).
[8] S. P. Borgatti, Social networks **27**, 55 (2005).
[9] A.-L. Barabási, Philosophical Transactions of the Royal Society A: Mathematical, Physical and Engineering Sciences **371**, 20120375 (2013).
[10] S. R. Eddy, Nature biotechnology **22**, 1315 (2004).
[11] T. M. Cover, *Elements of information theory* (John Wiley & Sons, 1999).

V. APPENDIX