Simplicial Activity Driven Model

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Many complex systems find a convenient representation in terms of networks: structures made by pairwise interactions (links) of elements (nodes). For many biological and social systems, elementary interactions involve however more than two elements, and simplicial complexes are more adequate to describe such phenomena. Moreover, these interactions often change over time. Here, we propose a framework to model such an evolution: the Simplicial Activity Driven (SAD) model, in which the building block is a simplex of nodes representing a multi-agent interaction. We show analytically and numerically that the use of simplicial structures leads to crucial differences in the outcome of paradigmatic processes modelling disease propagation or social contagion, with respect to the activity-driven (AD) model, a paradigmatic temporal network model involving only binary interactions. In particular, fluctuations in the number of nodes involved in the interactions can affect the outcome of models of simple contagion processes, contrarily to what happens in the AD model. Moreover, social contagion models such as cascading processes present a much richer phenomenology and can become extremely slow when occurring on evolving simplicial complexes.

The use of a network representation has become commonplace for describing and studying a large number of complex systems: the elements of the systems are seen as nodes, and links between nodes represent pairwise interactions\textsuperscript{[1,2]}. However, in many contexts, representing interactions as pairwise does not tell the whole story. Examples include collaborations among groups of actors in movies\textsuperscript{[3]}, spiking neuron populations\textsuperscript{[4,5]} and co-authorships in scientific publications\textsuperscript{[6]}.

Let us consider the latter for illustration purposes: In a network representation, a paper co-authored by \( n \) scientists yields a clique of \( n(n - 1)/2 \) links, which is however treated in the same way as \( n(n - 1)/2 \) papers authored by pairs of scientists (or any other combination of subgroups among these \( n \) scientists leading to the same number of links). While this is equivalent for \( n = 2 \), the number of co-authors of a scientific paper is often larger than 2. For instance, data (see Supplemental Material) show that the average number of authors of APS papers has steadily increased from 2 to 6 between the 1940s and now. In such cases (\( n > 2 \)), simplicial representations are more apt to preserve the information observed in data. Formally, a \( (d - 1) \)-dimensional simplex \( \sigma \) is defined as the set of \( d \) vertices \( \sigma = \{ x_0, x_1, \ldots, x_{d-1} \} \). A collection of simplices is a simplicial complex \( K \) if for each simplex \( \sigma \) all its possible surfaces (defined as subsets of \( \sigma \)) are themselves contained in \( K \). In the case of group interactions, this requirement can be considered trivially satisfied, as each group interaction implies all the possible sub-interactions.

To take this issue into account, simplicial descriptions have been recently adopted in models of emerging geometry\textsuperscript{[7,8]}, null models for higher order interactions\textsuperscript{[9,10]}, network inference\textsuperscript{[11]}, brain structure and dynamics\textsuperscript{[12,13]}.

Networks and interactions moreover evolve in time, and the field of temporal networks has indeed recently attracted a lot of attention\textsuperscript{[14,15]}. In particular, several models of time-evolving networks have been put forward, based on microscopic rules for the establishment and end of interactions between pairs of nodes\textsuperscript{[16,18]}. Among these, the activity driven (AD) temporal network model\textsuperscript{[17]} has attracted a lot of attention. In this model, each agent (node) is assigned an activity potential that determines at each time its probability to create pairwise interactions with other agents selected at random. The AD model and its extensions\textsuperscript{[19,21]} have become a paradigm of temporal networks and have been used to study the impact of the network’s temporal evolution on dynamical processes occurring on top of it\textsuperscript{[17,22]}.

Models of temporally evolving simplicial interactions are however still missing. Here we bridge this gap by proposing a general modeling framework for temporal group activation data: the Simplicial Activity Driven model. Our aim is to provide a simple framework that can be used as a basis for richer temporal models taking into account the simplicial nature of interactions, and on which dynamical processes can be studied by analytical and numerical means to shed light on the impact of both simplicial and temporally evolving interactions.

In its simplest version, the model considers \( N \) nodes, whose interactions change over time as follows:

(i) Each node \( i \) is endowed with an activity rate \( a_i \) taken from a predefined distribution \( F \);

(ii) At each time step \( \Delta t \), each node \( i \) fires with probability \( a_i \Delta t \); when it fires, it creates an \( (s - 1) \)-simplex (in networks’ terms, a clique of size \( s \)) with \( s - 1 \) other nodes chosen uniformly at random. Each activation hence contributes \( s(s - 1)/2 \) interactions to the network;

(iii) At the following time step, the existing simplices are erased and the process starts anew.
In the framework of collaborations, nodes can represent scientists, and the activity \( a_i \), their propensity to create collaborations: step (ii) corresponds to the creation of a collaboration of \( s \) scientists resulting in the co-authorship of a paper. It is worth noting here the main difference with the AD model (see Fig. 1): in the AD model, each active node creates a set of binary interactions with the chosen nodes (in the language of collaborations, \( s - 1 \) papers with each 2 authors), while, in the SAD model, nodes that are not active but are targeted by an active node obtain links to all the other nodes in the simplex, hence creating a coherent activation unit. The parameter \( s \) defines the size of the collaborations and can either be fixed or a random variable extracted at each activation from a distribution \( p(s) \). As for the AD model, the SAD model can moreover be refined by adding memory or community effects.

In the following, we study this model both from a structural point of view and through the analysis of paradigmatic dynamical processes. Since the AD model has been widely studied in the literature as a paradigm for temporally evolving networks, we underline in each case how the introduction of coherent units of \( s \) nodes as building blocks, instead of sets of binary interactions, results in networks displaying radically different properties and impacts the properties of dynamical processes. In particular, we will show how fluctuations in \( s \) impacts the outcome of disease spreading models for the SAD model (while it does not for the AD model) and how simplicial structures can dramatically slow down social contagion models.

![FIG. 1. SAD model. a) At each timestep, a node \( i \) activates with probability \( a_i \Delta t \). Upon activation it creates a coherent unit of \( s \) nodes (an \( (s-1) \)-simplex, or \( s \)-clique), creating links between all pairs. b) In contrast, in the standard AD model (nAD) only the \( s - 1 \) edges stemming from the activated node are added. c) In the eAD model instead \( \binom{s}{2} \) links stem from the activated node, conserving at each interaction the number of links of the SAD model.

It is worth noting here that the comparison with the activity-driven model can be done in two ways: we can indeed consider AD models designed to involve either the same number of nodes (node-matched AD model, nAD) or the same number of interactions (edge-matched AD model, eAD) as the SAD model at each time step (see Fig. 1). In the former, for each activation with group size \( s \) in the SAD model, we consider an AD activation with size \( m = s - 1 \), i.e., the activated node creates interactions with \( s - 1 \) other nodes chosen at random: this leads to the same total number of contacted nodes per activation in the nAD and in the SAD model. In the eAD, for each activation with group size \( s \), we consider instead an AD activation with \( m = \binom{s}{2} \), hence preserving the total number of interactions of each activation.

**Structure** We first focus on the structural properties of the SAD model, once aggregated over a fixed number \( T \) of timesteps. In such an aggregated network, each node \( i \) is linked by an edge to all the nodes with whom it had interacted at least once during the aggregation time-window. The degree of \( i \) in this aggregated network corresponds to the number of distinct nodes with whom \( i \) has interacted; in the interpretation of a scientific collaboration network, it gives the total number of distinct collaborators of a scientist.

We denote the expected value of the degree at time \( T \) of a node \( i \) with activity \( a_i \) by \( \kappa_T(i) \) and we compute it by separating it into two contributions. The first comes from node \( i \)'s own activation events, which occur at each timestep \( \Delta t \) with probability \( a_i \Delta t \) (see model definition): after \( T \) timesteps, \( i \) will have activated \( \sim T a_i \) times; for fixed simplex size \( s \), this means it will have made \( T a_i \bar{m} \) interactions (\( \bar{m} = s - 1 \)). The second contribution instead comes from the activations of other nodes: every node \( j \neq i \) will have activated \( T a_j \) times; in each activation of \( j \neq i \), \( i \) was selected with probability \( \bar{m}/(N - 1) \) and, if selected, \( i \) was provided with \( \bar{m} \) interactions. Hence, at time \( T \) node \( i \) will have accumulated \( \kappa_T(i) \) interactions with:

\[
\kappa_T(i) = \bar{m} a_i T + \sum_{j \neq i} \frac{\bar{m} a_j T}{N - 1} \approx \bar{m} T(a_i + \bar{m}(a))
\]  

where the approximation holds for \( N \gg 1 \). For any node
distinct from \( i \), the probability not to have been involved in any of these interactions is \( (1 - 1/(N - 1))^{k_S(i)} \), and hence finally the number of distinct nodes having interacted with \( i \) is

\[
k_{T}^{SAD}(i) = (N - 1) \left[ 1 - \left( 1 - \frac{1}{N - 1} \right)^{k_S(i)} \right] \tag{2}
\]

\[
\approx N \left[ 1 - e^{-\frac{T_m(a_i+<s>)n_i}{N}} \right] \tag{3}
\]

where the approximation holds for large \( N \) and small \( T/N \). The aggregated degree distribution can also be calculated by rewriting the degree distribution as \( P_T(k) = F[a(k)] \frac{da(k)}{dx} \), obtaining:

\[
P_T(k) \sim \frac{1}{\bar{m}T} \frac{1}{1 - \frac{k}{\bar{m}T}} F \left[ \frac{k}{\bar{m}T} - \bar{m}(a) \right] \tag{4}
\]

for small \( k/\bar{m} \) (see Supplemental Material (SM) for the full derivation).

In Figure 2, we compare the predictions for the aggregated degree averaged over all nodes, \( k_{T}^{SAD}(i) \), at fixed \( s \), showing an excellent agreement with numerical simulations. We also compare it with the nAD and eAD models, for which \( k_{T}^{nAD}(i) = N(1 - e^{-T_m(a_i+<s>)n_i}/N) \) \[17\] with \( m = \bar{m} \) for nAD and \( m = \frac{\bar{m}}{T} \) for eAD.

Interestingly, \( k_{T}^{SAD} \) depends on \( \bar{m}^2 \); this implies that, if the simplex size \( s \) is allowed to fluctuate, the size of such fluctuations will in general affect the aggregated degree: in the SM we show this is the case by creating SAD networks where we sample clique sizes from distributions \( p(s) \) with fixed average and variable heterogeneities (as quantified by the ratio between the first two moments of \( p(s) \)). This phenomenology is in contrast with the nAD model, which has no dependence on the second moment of \( s \), while the eAD inherits it from the matching of the number of edges created at each activation (since each activation creates \( m = s(s-1)/2 \) edges, the resulting total number of activations and the integrated degree depend on the fluctuations of \( s \)).

From this aggregated degree point of view, we thus observe a similar behaviour for the SAD and eAD models. Figure 2, however highlights that this is not the whole story, and that the SAD model building mechanism leads to an important structural difference with the eAD model: as each activation creates \( \binom{s}{2} \) interactions that involve only \( s - 1 \) nodes, the size of the largest connected component (GCC) in the SAD integrated until \( T \) grows with \( T \) much more slowly than in the eAD model, for which each activation creates a star reaching \( \binom{s}{2} \) nodes; in fact, it grows in the same way as the nAD model, despite creating many more interactions at each step (the differences appear for \( s > 2 \) as for \( s = 2 \) the three models are the same).

Overall, the structural properties of the SAD model, when seen from an aggregated point of view, present thus both similarities and important differences with activity-driven models with the same numbers of events. To further our analysis, we explore in the following how dynamical processes are impacted by using simplices instead of pairwise interactions as basic building blocks of a temporal network.

**Dynamical processes** We first consider here the paradigmatic susceptible-infected-susceptible (SIS) model for disease spreading \[23\]. In this model, nodes can be either susceptible (S) or infectious (I). Infectious individuals can propagate the disease to susceptible ones at rate \( \beta \) whenever they are interacting, and recover spontaneously at rate \( \mu \), becoming again susceptible.

In a homogeneous population, the epidemic threshold \( \lambda \), separates an epidemic-free state at low values of the parameter \( \lambda = \beta/\mu \) from an endemic state at high values of \( \lambda \).

To obtain the value of the epidemic threshold for the SIS model spreading on top of the SAD model for temporal networks, we resort to a temporal heterogeneous mean-field approach similar to the one used for the AD model \[17\]. In this approach, the nodes are classified according to their activity, and we denote by \( N_a \) the number of nodes with activity \( a \): \( I_a \) and \( S_a \) denote respectively the numbers of infectious and susceptible nodes with activity \( a \) at time \( t \). We thus have \( N_a = S_a + I_a \), \( N = \int daN_a \) is the total population, and \( I = \int daI_a \) the total number of infectious at time \( t \).

Let us consider for simplicity the case of the SAD model with fixed clique size \( s \). The variation during a time step \( \Delta t \) of the number of infectious is given by the following equation, which describes both the evolution of the interactions and of the spreading process:

\[
I_a^{t+\Delta t} - I_a^t = -\mu \Delta t I_a^t + \beta \Delta t S_a a(s-1) \int \frac{da'}{N} I_a^t a'(s-1) \\
+ \beta \Delta t S_a \int \frac{da'}{N} I_a^t a'(s-1) \int \frac{da''}{N} I_a^t a''(s-2). \tag{5}
\]

The first term corresponds to the recovery of nodes with activity \( a \). The second term corresponds to susceptible nodes with activity \( a \) that become active at time \( t \) (with probability \( a\Delta t \)) and create a simplex of size \( s \) (with \( s - 1 \) other nodes) that includes infectious nodes with any activity (hence the integration over \( a' \)). The third term stems from the fact that susceptible nodes with activity \( a \) can be chosen as clique partners by infectious nodes with activity \( a' \) that become active (with probability \( a'\Delta t \)). While these three terms also appear in the case of a spreading process on an AD network, the last term is specific to the SAD model: it describes the cases in which susceptible nodes with activity \( a \) are chosen by a susceptible with activity \( a' \), which becomes active (with probability \( a'\Delta t \)) and creates a simplex that also includes an infectious node with activity \( a'' \).

The dynamics is solved by (i) integrating Eq. (5) over \( a \) and (ii) multiplying Eq. (5) by \( a \) before integrating over \( a \), obtaining coupled evolution equations for \( I' \) and \( \theta' = \int daI_a^t \). Straightforward computations detailed in the SM show that the epidemic threshold is then given
FIG. 3. Epidemic SIS threshold for AD and SAD models. a) Epidemic prevalence as function of $\Lambda = \beta/\mu$: the epidemic transition in the SAD model is delayed as compared with an SIS model on the corresponding eAD model (here $N = 1000$, $s = 4$, $T = 20000$). Vertical lines correspond to the theoretical values of the epidemic thresholds. Node activities were sampled from an activity distribution $F(a) = (a/a_0)^{-\alpha}$, $\alpha = 2.1$ and $a_0 = 5 \cdot 10^{-3}$. We use the same activity distribution in panel b). b) Increasing the average connectivity of the underlying network lowers the epidemic threshold in all models; for the $s$-regular SAD model $\lambda_c$ is always larger than for the corresponding eAD model.

by

$$\frac{\beta}{\mu} > \frac{2}{s(s-1)}(a) + (s-1)\sqrt{s^2(a)^2 + 4(\langle a^2 \rangle - (a)^2)},$$

to compare with the epidemic threshold condition $(6)$

$$\frac{\beta}{\mu} > 1/(m\langle a \rangle + m\sqrt{\langle a^2 \rangle})$$

for an AD model with parameter $m$.

If the sizes of the cliques formed in the SAD model are extracted at random at each activation from a distribution $p(s)$, the r.h.s. of Eq. (5) needs simply to be integrated as $\int ds p(s)$, if the size $s$ is independent of the activity $a$. The epidemic threshold becomes

$$\frac{\beta}{\mu} > \frac{2}{s(s-1)}(a) + \sqrt{\Delta},$$

with

$$\Delta = \langle(s-1)(s-2)\rangle/\langle s-1 \rangle^2 \langle a \rangle + 4\langle s-1 \rangle^2 \langle a^2 \rangle$$

(see details in the SM). Notably, it depends not only on the average clique size but also on the second moment of $p(s)$. This is in contrast with the case of the SIS model on the AD model, in which fluctuations of the numbers of links created at each time step would not change the epidemic threshold ($m$ just being replaced by its average).

Figure 3 shows in panel (a) the result of direct numerical simulations of an SIS model on temporal eAD and SAD networks, showing a good agreement between these simulations and the theoretical values of the epidemic threshold. We moreover compare in panel (b) the epidemic threshold obtained in a SAD model with fixed clique size $s$ with the epidemic threshold obtained in the nAD and eAD models. In the former case, the epidemic threshold is smaller in the SAD model, which can be related to the fact that the SAD model has more interactions than the nAD network. In the latter case –on the contrary– the fact that the $s(s-1)/2$ interactions are created as cliques hampers the spreading process on the SAD with respect to the eAD network, leading hence to a higher epidemic threshold for the SAD case.

We stress here that, even at fixed $s$, if we allow $s$ and $a$ to fluctuate, it is possible to obtain a wide diversity of SAD critical thresholds $\lambda_{SAD}^c$ and also of the ratio $\lambda_{SAD}^c/\lambda_{eAD}^c$. As an example, Fig. 4 shows the ratios obtained when using the activity and clique size distributions measured in each year in the APS dataset: this ratio changes significantly between the data of 1900 and 2015, driven by changes in the activity fluctuations $\beta_a$ until the 1950s and in the co-authorship size fluctuations $\beta_s$ afterwards.

Let us now consider a spreading process of a quite different nature than the SIS model, namely a cascading process. Indeed, while the SIS model is a typical example of "simple contagion" processes, in which contagion can result from a single interaction between a susceptible and an infectious individual, social contagion phenomena are often thought to correspond to more complex processes in which multiple exposures are needed for an idea or an opinion to propagate [24]. While a large number of models describing social contagion have been put forward in the literature and studied on various types of static network structures, few studies have considered social contagion processes on temporal networks. In this context, an interesting case concerns the model of cascades introduced by D. Watts [25] to represent the adoption of a behavior or a product by individuals influenced by their social contacts in a population, generalized to temporal networks in Ref. [26]. In the SM, we compare the evolution of this model on SAD and AD temporal networks. In particular, we show numerically and by analytical arguments that the dynamics can become extremely slow in the SAD with respect to an AD model. We moreover show that the SAD model leads to a very rich phenomenology of the cascading process.

To conclude, we have presented a new model for temporal networks, based on the fact that the fundamental building blocks of many social networks are coherent units of several individuals interacting as a group, rather than dyadic interactions. Our Simplexical Activity Driven model considers indeed agents who, when active, create simplices with other agents, that once aggregated result in a simplicial complex. We have shown how this mechanism leads to fundamental differences with respect to a well-known model in which active agents create sets of dyadic interactions, the activity-driven model. These differences appear not only at the structural level but, most importantly, have strong consequences on how dynamical processes unfold on these networks, as we have
FIG. 4. Ratio $\lambda_{SAD}^{P(t)}/\lambda_{eAD}^{P(t)}$ for co-authorship data. We plot the ratio between the predicted epidemic thresholds for the SAD and corresponding eAD models built using the empirical distributions $F(a)$ and $p(s)$ extracted each year from the APS co-authorship data. The ratio grows between the years 1900 and 2015, driven by $\beta_a = \langle a^2 \rangle / \langle a \rangle$ until the 1950s and by $\beta_s = \langle s^2 \rangle / \langle s \rangle$ afterwards.

Thanks to the simplicity of its definition, our model lends itself to analytical investigations, and can be a starting point for a number of possible refinements, such as adding memory effects, node labels and interacting probabilities depending on these labels, or correlations between activity of an agent and size of the simplicial complex it creates. Moreover, it would be interesting to study further dynamical processes on the SAD model and its variations. Finally, the SAD model constitutes a first null model for the homology of temporal complex systems with high-order interactions. We hope that our work will stimulate research in such directions.

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I. CO-AUTHORSHIP DATA

We consider a dataset containing all publications in American Physical Society (APS) journals from year 1893 to 2016. The dataset is part of the “APS Data Sets for Research” and can be freely obtained from https://journals.aps.org/datasets. The dataset contains 587675 papers by 413513 different authors. Figure S1 illustrates the evolution of the distribution of the number of co-authors as deduced from this dataset: over the course of a few decades, this distribution has evolved from a narrow one in the 1940s to progressively broader distributions in 1960s and up present times, with both average and fluctuations increasing with time. The average number of authors of a paper has steadily increased from 2 to 6 between the 1940s and now. Furthermore, the inset of Fig. S1 shows the temporal evolution of the ratio $\beta_s = \langle s^2 \rangle / \langle s \rangle$ between the first two moments of $p(s)$, highlighting the increase in the heterogeneity of the distribution.

FIG. S1. Distributions of the number $s$ of co-authors of a paper in the APS dataset. The distribution becomes broader in more recent times. Inset: evolution of the ratio $\beta_s = \langle s^2 \rangle / \langle s \rangle$.

II. AGGREGATED DEGREE FULL CALCULATIONS

The number of interactions of $i$ between times 0 and $T$ is given by:

$$k_T(i) = \bar{m}a_i T + \sum_{j \neq i} \bar{m}^2 T a_j \frac{N-1}{N-1} \approx \bar{m}T(a_i + \bar{m}(a))$$

(S1)

where the approximation holds for $N \gg 1$. We can then write $k_T(i)$ as [17]

$$k_T(i) = (N-1) \left[ 1 - (1 - \frac{1}{N-1})^T \bar{m}(a_i + (\bar{m}(a)) \right]$$

(S2)

$$\approx (N-1) \left[ 1 - e^{-\bar{m}(a_i + (\bar{m}(a))} \right]$$

(S3)

Following [17], we write $a_i = \eta x_i$, where $x_i$ is the activity potential and $\eta$ is a free parameter useful when data are available in order to match the average degree in the simulated temporal network to that of the real one. From this we can calculate the activity potential $x_i$ of node $i$ as:

$$x_i = -\frac{N-1}{\bar{m}T \eta} \ln \left( 1 - \frac{k_T(i)}{N-1} \right) - \frac{\bar{m}(a)}{\eta}$$

(S4)

$$\approx \frac{k_T(i)}{\bar{m}T \eta} - \frac{\bar{m}(a)}{\eta}$$

(S5)
FIG. S2. **Structural properties of SAD model.** (a) Average aggregated degree ⟨kT⟩ for the SAD model and corresponding nAD and eAD models for a range of simplex sizes s (for N = 2000, T = 10, activities sampled from P(a) = (a/a0)^α, α = 2.1 and a0 = 5 · 10^-3). ⟨kT⟩ grows quadratically with s in the SAD and in the eAD models and linearly in the nAD model. Inset: ⟨kT⟩ depends on the second moment of s in the SAD model (and in the eAD by inheritance of the fluctuations in s), but not in the nAD model. (b) Temporal growth of the size of the aggregated GCC for the SAD (solid lines), nAD (crosses) and eAD (dashed lines) for various fixed simplex size s. The nAD and SAD models have the same behaviour as expected by design of the node-matched AD model, while the eAD model systematically generates much larger GCC sizes as compared to the corresponding SAD model.

where the approximation again holds for small k/N. Using the expressions above, by simple substitution we obtain the functional form for the integrated degree distribution:

\[
P_T(k) \sim \frac{1}{mT\eta} \frac{1}{1-kN^{-1}} F \left[ -N-1 \frac{1}{mT\eta} \ln \left( 1 - \frac{k}{N-1} \right) - \frac{m(a)}{\eta} \right]
\]  

(S6)

For clarity of illustration, we report here a larger version of Fig. 2 of the main text, adding also the behaviour of kT as a function of the heterogeneity of the distribution of s (see inset Fig. S2h).

**III. SIS MODEL ON THE SAD TEMPORAL NETWORK**

We consider the susceptible-infected-susceptible (SIS) model for disease spreading. In this model, nodes can be either susceptible (S) or infectious (I). Infectious individuals can propagate the disease to susceptible ones at rate β whenever they are interacting, and recover spontaneously at rate μ, becoming again susceptible.

We denote by N_a the number of nodes with activity a. The total number of nodes is N = \( \int daN_a \).

S^t_a and I^t_a denote respectively the number of susceptible and infectious of activity a at time t. We have the simple conservation equation N_a = S^t_a + I^t_a, and the total number of infectious individuals is I^t = \( \int daI^t_a \).
A. Case of a fixed clique size $s$.

As explained in the main text, the time evolution of the number of infectious is given by the following equation:

$$I^{t+\Delta t}_a - I^t_a = -\mu \Delta t I^t_a + \beta \Delta t S_a a(k-1) \int da' \frac{I^t_{a'}}{N}$$

$$+ \beta \Delta t S_a \int da' \frac{I^t_{a'}}{N} (s-1)$$

$$+ \beta \Delta t S_a \int da' \frac{S_{a'}}{N} (s-1) \times \int da'' \frac{I_{a''}}{N} (s-2)$$  \hspace{1cm} (S7)

If we integrate $[5]$ over $a$ we get an equation for $I_t$:

$$I^{t+\Delta t} - I^t = -\mu \Delta t I^t + \beta \Delta t (s-1)\langle a \rangle I^t + \beta \Delta t (s-1)\langle a \rangle \theta^{t} + \beta \Delta t (s-1)\langle s \rangle I^t$$

$$= -\mu \Delta t I^t + \beta \Delta t (s-1)^2\langle a \rangle I^t + \beta \Delta t (s-1)^2\langle a \rangle \theta^{t}$$  \hspace{1cm} (S8)

where $\theta^t = \int da a I^t_a$.

Multiplying $[5]$ by $a$ and integrating we get an equation for $\theta_t$:

$$\theta^{t+\Delta t} - \theta^t = -\mu \Delta t \theta^t + \beta \Delta t (s-1)\langle a \rangle I^t + \beta \Delta t (s-1)\langle a \rangle \theta^{t} + \beta \Delta t (s-1)\langle s \rangle I^t$$

$$= -\mu \Delta t \theta^t + \beta \Delta t (s-1)\langle a \rangle \theta^{t} + \beta \Delta t ((s-1)\langle a \rangle + (s-1)(s-2)\langle a \rangle) I^t$$  \hspace{1cm} (S9)

These equations can be rewritten as

$$\begin{pmatrix}
I^{t+\Delta t} - I^t \\
\theta^{t+\Delta t} - \theta^t
\end{pmatrix} = J
\begin{pmatrix}
I^t \\
\theta^t
\end{pmatrix}$$  \hspace{1cm} (S10)

with

$$J = \begin{pmatrix}
-\mu + \beta(s-1)^2\langle a \rangle & \beta(s-1) \\
\lambda ((s-1)\langle a \rangle + (s-1)(s-2)\langle a \rangle)^2 & -\mu + \beta(s-1)\langle a \rangle
\end{pmatrix}$$  \hspace{1cm} (S11)

The characteristic polynomial of $J$ is

$$(x + \mu)^2 - \lambda k(k-1)\langle a \rangle (x + \mu) + \lambda^2 (k-1)^2 (\langle a \rangle^2 - \langle a^2 \rangle)$$

from which we find its eigenvalues:

$$\Lambda_{\pm} = \left(\lambda k(k-1)\langle a \rangle - 2\mu \pm \lambda k(k-1) \sqrt{k^2\langle a \rangle^2 + 4(\langle a^2 \rangle - \langle a^2 \rangle^2)}\right)/2.$$

The epidemics does not vanish if and only if the largest eigenvalue $\Lambda_+$ is positive. This yields the epidemic threshold condition:

$$\frac{\beta}{\mu} > \frac{2}{k(k-1)\langle a \rangle + (k-1) \sqrt{k^2\langle a \rangle^2 + 4(\langle a^2 \rangle - \langle a^2 \rangle^2)}}$$  \hspace{1cm} (S12)

B. Case of a distribution of clique sizes $P(s)$

Let us assume that the size of the clique created at each activation is taken from a distribution $p(s)$. The size $s$ and the activity of the active node at time $t$ are uncorrelated. With respect to the case of fixed clique size, we then just add an integration on $p(s)$ in the evolution equation of $I^t_a$:

$$I^{t+\Delta t}_a - I^t_a = -\mu \Delta t I^t_a + \beta \Delta t \int ds p(s) S_a a(s-1) \int da' \frac{I^t_{a'}}{N}$$

$$+ \beta \Delta t \int ds p(s) S_a \int da' \frac{I^t_{a'}}{N} (s-1)$$

$$+ \beta \Delta t \int ds p(s) S_a \int da' \frac{S_{a'}}{N} (s-1) \times \int da'' \frac{I_{a''}}{N} (s-2).$$  \hspace{1cm} (S13)
Proceeding as before, we obtain coupled equations for $I^t$ and $\theta^t$, with the matrix $J$ given now by:

$$J = \begin{pmatrix}
-\mu + \beta \langle (s-1)^2 \rangle (a) & \beta \langle s-1 \rangle \\
\beta \langle (s-1) \rangle (a^2) + \langle (s-1)(s-2) \rangle (a^2) & -\mu + \beta \langle s-1 \rangle (a) .
\end{pmatrix}$$

(S14)

The eigenvalues of $J$ are now

$$\lambda_\pm = \left( \lambda \langle s-1 \rangle (a) - 2\mu \pm \lambda \sqrt{\langle (s-1)(s-2) \rangle \langle (s-1) \rangle (a^2) + 4\langle s-1 \rangle (a^2)^2} \right) / 2$$

Imposing that the largest one is positive gives the epidemic threshold condition

$$\frac{\lambda}{\mu} > \frac{2}{\langle s(s-1) \rangle (a) + \sqrt{\langle (s-1)(s-2) \rangle \langle (s-1)(s+2) \rangle (a^2) + 4\langle s-1 \rangle (a^2)^2}}$$

(S15)

C. Ratio between SAD and eAD epidemic thresholds

Using the expressions for $\lambda^{SAD}_c$ and $\lambda^{eAD}_c$, it is possible to write down explicitly the expression for the ratio between the two quantities:

$$\frac{\lambda^{SAD}_c}{\lambda^{eAD}_c} = \frac{\langle a \rangle + \sqrt{\langle a^2 \rangle}}{\langle a \rangle + \sqrt{\langle a^2 \rangle} \sqrt{\Theta}}$$

(S16)

with

$$\Theta = \frac{4\langle s-1 \rangle^2 + \langle (s-1)(s-2) \rangle \langle (s-1)(s+2) \rangle}{\langle s(s-1) \rangle^2} \cdot \frac{\langle a \rangle^2}{\langle a^2 \rangle}$$

(S17)

where for the eAD we used $m = s(s-1)/2$. While the expression above can take values smaller than 1, for meaningful values of the first two orders of $s$ and $a$ ($s \geq 2$, $\langle s^2 \rangle \geq \langle s \rangle^2$) it is always larger or equal to 1, implying that the SAD critical threshold is always higher than that of the corresponding eAD model.

IV. CASCADE MODEL

A. Definition

We consider here the model of cascades introduced by D. Watts [25] and generalized to temporal networks in Ref. [26]. In this model, designed to represent the adoption of a behavior or a product by individuals influenced by their social contacts in a population, agents can be either in state 0 (non-adopters) or 1 (adopters). Agents in state 0 can change state and become adopters if a fraction of their social contacts larger than $\phi \in [0,1]$ (the model parameter) are adopters. In a static network framework, the social contacts are simply the neighbors of an agent. Starting from a certain fraction of adopters, for instance taken at random among all agents, the agents’ states evolve until no non-adopter has a fraction of adopters among his/her neighbors larger than $\phi$: such a configuration is blocked and the cascade stops. The efficiency of the cascade can then be measured by the final fraction of adopters in the population.

Two important modifications need to be considered when the social contacts evolve in time, so that the adoption cascade occurs along a temporal network of interactions:

- First, the model depends on another parameter, namely the length $\theta$ of the time-window during which the social contacts are considered: at time $t$, each non-adopter considers the time-window $(t-\theta,t]$ and becomes adopter if the fraction of his/her number of contacts with adopters among all interactions $s$/he had in this time-window is larger than $\phi$ ($\theta = 1$ corresponds to taking into account only the interactions at time $t$).

- Second, as the contacts are changing over time, a non-adopter could always have the possibility to make enough new contacts with adopters to change state and become adopter: no configuration in which some agents are still non-adopters can be considered as blocked, and the cascade could a priori continue until all agents are adopters.

To measure the efficiency of the dynamics or compare various initial configurations or temporal networks, one should thus for instance fix a maximum time limit for the cascade, or use empirical data of finite length [24].

While a full investigation of the model at varying $\phi$, $\theta$ on the one hand and at different parameters of the SAD temporal network on the other hand (either at fixed $s$ or with a distribution of sizes $p(s)$) is beyond our scope, we simply illustrate here some aspects of the complex phenomenology of the cascading processes. In particular, we show that strong differences appear in the dynamical outcome of the process on SAD and AD temporal networks.
B. Cascades on AD and SAD temporal networks

Figure S3 shows the temporal evolution of the average fraction of adopters in SAD temporal networks with fixed clique size $s$ and in corresponding eAD networks, for $\theta = 1$ and varying $\phi$. In the AD case, the dynamics becomes slower as $\phi$ increases, as can be expected since the fraction of interactions with adopters needed to become an adopter increases, but the slowing down is very limited. In the SAD model in contrast, as $\phi$ increases, the dynamics becomes extremely slow when $\phi$ increases.

The strong difference between the dynamics on AD and SAD networks can be understood as follows. Let us first consider the AD case: at each time step, if an adopter is activated and contacts a non-adopter, then the fraction of adopters that the non-adopter sees is 1, so s/he becomes adopter, whatever the value of $\phi$. If instead the active node is non-adopter, then s/he can become adopter if a fraction larger than $\phi$ of the $\binom{s}{2}$ agents s/he contacts are adopters. While the second mechanism becomes slower if $\phi$ increases, as at early times it is less probable that a non-adopter nodes manages to contact enough adopters to change state, the first mechanism guarantees that the cascade can continue at any value of $\phi$. The situation is quite different for the SAD model. Indeed, at each time step a clique of size $s$ is created. So each agent has the same number of interactions, and a non-adopter can become adopter only if the clique contains more than $(s-1)\phi$ adopters. For instance, if $s = 5$, for $\phi = 0.1$ a non-adopter becomes adopter as soon as one adopter is present in the same clique. For $\phi = 0.3$ however the created clique needs to contain at least two adopters, for $\phi = 0.5$ it needs 3, etc: for $\phi \in \left[\frac{(n-1)}{(s-1)}; \frac{n}{(s-1)}\right]$ a non-adopter needs to be put in interaction with at least $n$ adopters to change state. For any $n$ integer, the dynamics is thus much slower for $\phi \geq n/(s-1)$ than for $\phi < n/(s-1)$. 

FIG. S3. Average fraction of adopters vs. time for cascades unfolding along AD (top plots) and SAD (bottom plots) temporal networks with $N = 500$ nodes, with $s = 5$ (left column) and $s = 8$ (right column). Here $\theta = 1$. 

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C. Effect of the memory span $\theta$

We have checked that this phenomenology is robust for $\theta > 1$. As $\theta$ increases, the process becomes slower on both AD and SAD networks. This is in agreement with the results of [26], who find that the final cascade size on empirical temporal networks of finite duration decreases when $\theta$ increases. This is due to the fact that the number of contacts increases when the time-window to consider $[t-\theta, t]$ becomes larger, but, as the number of nodes in state 1 is small at early times, their fraction during $[t-\theta, t]$ typically decreases and thus it is more difficult for this fraction to be above $\phi$.

D. Effect of the parameter $s$

Figure S4 compares the results of simulations of cascades on AD and SAD temporal networks for various values of $s$ at fixed values of $\phi$, with $\theta = 1$.

For the AD model, as $s$ increases, the cascade dynamics gets faster. This is the result of a competition between 2 phenomena: at fixed $\phi$, if $s$ increases, the probability for an active node in status 0 to have a fraction at least $\phi$ of its neighbors in state 1 is smaller, at least at short times. This would slow down the cascade. However, an active node in status 1 is able to spread its opinion to all the nodes it contacts: in the eAD model, a single active node can change the state of $\binom{s}{2}$ other nodes, a number growing quadratically with $s$, so that this effect dominates and the cascade becomes overall faster.

For the SAD model, the picture is more complicated. If $s$ increases but the value of $\phi$ remains in $[(n-1)/(s-1), n/(s-1)]$ with fixed $n$, then with increased $s$ the spread becomes faster. Equivalently, this corresponds to $s$ increasing within $[1 + (n-1)/\phi, 1 + n/\phi]$ when $s$ crosses the value $1 + n/\phi$ however, the number of nodes in state 1 needed to convince the other nodes in the cliques increases by 1, so the dynamics becomes much slower.

Let us take concrete examples. For $\phi = 0.1$, increasing $s$ leads to faster dynamics until $s = 11$. For $\phi = 0.3$, the intervals $[1 + n/\phi, 1 + (n+1)/\phi]$ to consider are $[1, 4.33], [4.33, 7.67], [7.67, 11]$, etc. So $s = 4$ leads to faster dynamics than $s = 3$ but $s = 5$ becomes slower at short times, $s = 6$ is faster than $s = 5$ and $s = 8$ becomes much slower ($n = 3$ is needed).

For $\phi = 0.5$, the intervals $[1 + (n-1)/\phi, 1 + n/\phi]$ to consider are $[1, 3], [3, 5], [5, 7], [7, 9]$, etc. So $s = 4$ leads to faster dynamics than $s = 3$, $s = 5$ is instead much slower, etc.

Overall, the cascade velocity is non-monotonic with respect to an increase in $s$ for the dynamics on the SAD temporal network.

E. Effect of the variability in clique size

We finally mention the effect of variability in the values of $s$. Let us assume a distribution $p(s)$ centered on a value $s_0$ and with tunable fluctuations around $s_0$, quantified by a coefficient of variation $v$ (ratio between standard deviation and average). On the AD model, an increased variability simply speeds up slightly the cascading process (see Fig. S5). On the other hand, a much more dramatic effect with very strong speed-up of the dynamics can be obtained when the dynamics takes place on the SAD model, as illustrated in Fig. S6. Indeed, let us assume that $\phi \in [(n-1)/(s_0-1); n/(s_0-1)]$ with $n$ not too small, meaning that the dynamics with fixed $s_0$ is slow because a node in state 0 needs to be in contact with at least $n$ nodes in state 1 to change state. Variability in $s$ means that the clique formed can be sometimes smaller than $s_0$, and $\phi$ could sometimes be smaller than $(n-1)/(s-1)$, in which case only $n-1$ nodes in state 1 are required to change state, and the process becomes thus much faster.
FIG. S4. Average fraction of adopters vs. time for cascades unfolding along eAD (left plots) and SAD (right plots) temporal networks with \( N = 500 \) nodes, for various values of \( s \) and \( \phi \). Here \( \theta = 1 \).
FIG. S5. Average fraction of adopters vs. time for cascades unfolding along eAD temporal networks with \( N = 500 \) nodes, for various values of \( s_0 \) (average of \( p(s) \)), \( v \) (coefficient of variation of \( p(s) \)) and \( \phi \). Here \( \theta = 1 \).

FIG. S6. Average fraction of adopters vs. time for cascades unfolding along SAD temporal networks with \( N = 500 \) nodes, for various values of \( s_0 \) (average of \( p(s) \)), \( v \) (coefficient of variation of \( p(s) \)) and \( \phi \). Here \( \theta = 1 \).