## Threshold-based epidemic dynamics in systems with memory

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Abstract – In this article we analyze an epidemic dynamics model (SI) where we assume that there are k susceptible states, that is a node would require multiple (k) contacts before it gets infected. In specific, we provide a theoretical framework for studying diffusion rate in complete graphs and d-regular trees with extensions to dense random graphs. We observe that irrespective of the topology, the diffusion process could be divided into two distinct phases: (i) the *initial* phase, where the diffusion process is slow, followed by (ii) residual phase where the diffusion rate increases manifold. In fact, the initial phase acts as an indicator for the total diffusion time in dense graphs. The most remarkable lesson from this investigation is that such a diffusion process could be controlled and even contained if acted upon within its initial phase.

**Introduction.** – Information diffusion is one of the most common phenomena that occurs on a network and the most elementary model of this process is SI (Susceptible-Infected) [1] and its different variations [2–7]. Epidemic/adoption models have recently found a renewed interest with the formulation of temporal networks and realization that most of the real-world networks are temporal in nature (network structure changes with time). For example, Takaguchi et. al. in [8] presents a model whereby adoption behavior of a node is driven by the number of recent contacts with already adopted individual. Through simulations on real-world temporal networks the authors further show that burstiness [9] affects spreading rate. Similar empirical study was also performed by Karimi et. al. in [10]. A further modification to the model has been proposed by Backlund et. al. in [11] which considers that adoption is driven by the number of contacts with different adopted neighbors within a chosen time instead of a particular neighbor multiple times. Further, information diffusion on temporal networks, more specifically prevalence, have also been studied in [12]. Several other results on the study of epidemics in temporal networks are listed in [13] and on epidemic thresholds in [14–16]. This type of history-dependent thresholding spreading pattern is largely observed in spread of bacterial diseases such as tuberculosis and dysentery [17] and also in peer-to-peer [18] and Bittorrent systems [19]. In [20] the authors show

## that people accept ideas/news after repeated exposure.

The fundamental difference between static and temporal epidemic (SI) models is that in temporal models every agent within a population is not equally susceptible to a disease or equally amenable to a rumor - the one which has been exposed more number of times (in recent past) are more amenable. This difference however is not well formulated and hence not well modeled - the primary contribution of this letter is to succinctly define the problem in terms of a simple model and then theoretically calculate the rate of spread of the epidemics. We consider a spreading model in the lines of [8] where each susceptible node needs to communicate with the infected nodes multiple times to contract infection. More importantly, unlike memoryless systems, we assume that each node comprises a memory which keeps track of the number of contacts it makes with infected ones. Note that in our system memory is a property which allows each node to remember the number of contacts it has already made with the infected ones. While Karimi et. al. studied the effect of burstiness in the diffusion process, we are more interested in estimating both empirically and analytically the diffusion time and rate. Note that this model is completely different from threshold models [21, 22] where a node gets infected when majority of its neighbors are infected or probabilistic SI-models where an infected node, on coming in contact, infects a susceptible node with a probability p, as

those are memoryless systems and the transition depends only on the activity of present time step. Our diffusion model also differs from the Neighborhood Exchange (NE) model proposed in [23]. NE assumes at any given time, an individual will be in contact with an individual-specific number of neighbors with whom disease transmission is possible while our model considers that a node can be in contact with only a single individual. Also NE does not consider the fact that multiple contacts are required to contract infection.

The analytical results are obtained considering simple yet important topologies like the complete graph and the infinite d-regular trees which accounts for two extreme variants of network topology in terms of edge density. An important inference we draw from the theoretical analysis is that irrespective of the topology the diffusion process could be divided into two phases: (i) an initial phase where the diffusion rate is very slow and (ii) a residual phase where the diffusion becomes very fast. This inference is the central contribution of this letter which can help in containing the spread of infectious diseases with minimum overhead if acted upon in the initial phase which we further prove through a detailed empirical study.

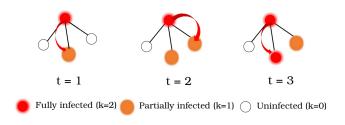


Fig. 1: Proposed diffusion model for k = 2.

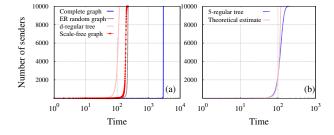


Fig. 2: (a) The number of senders versus time steps for different network topologies (For ER random graph p is 0.005, for d-regular graph d=5 and for BA scale-free graph m i.e., number of edges to attach from a new node to existing nodes is 5) and (b) The theoretical estimate and the simulated result for d-regular trees. The theoretical eastimate is obtained from equation 17.

**Diffusion model.** – Formally, we consider a network topology G = (V, E) where V represents the set of nodes

in the network and E denotes the set of edges between any pair of nodes in V. We initially start with a single infected node in the system. We further assume that for a susceptible node to get infected, k encounters with infected nodes are required. To put it in a simple way we consider that a message M needs to be spread over a network and message M consists of k identical tokens. At each communication instance one token gets transmitted from an infected node to the susceptible node. Therefore, number of tokens (k) in a message corresponds to the number of contacts required for a susceptible node to get infected. Note that for the rest of the paper we will present our diffusion model in terms of messages and tokens.

We assume that at time 0, there is only one sender (infected) node present in the system and it acts as the initiator of the diffusion process. At each discrete time step a sender node randomly selects one of its neighbors and there is a transfer of a token from the sender to the nonsender. A non-sender node becomes a sender only after it receives exactly k tokens (refer to figure 1). The analytical estimation of the diffusion time based on the underlying topology requires a case-by-case examination. We formulate both analytical and empirical results for two extreme variants (in terms of edge density) of networks (a) complete graphs (dense) and (b) infinite regular trees (sparse) while for others we provide empirical results with intuitive justifications.

On Complete graph. — For complete graph we assume the number of nodes in the system to be n. To determine how the number of senders in the system changes with time, we plot the number of senders against time for complete graph in Fig. 2. We observe that the diffusion is initially slow which is then followed by a ramp-up after which the diffusion rate becomes almost exponentially fast. To better analyze the process we divide the process into two phases i) the initial phase and ii) the residual phase.

**Initial phase:** In the spreading process we define the initial phase to be the time between the initiation and the point at which the first sender is created. We define this as  $t_1$ ; we will show later that  $\hat{E}(t_1)$  (expected value of  $t_1$ ) is indeed an indicator for  $\hat{E}(T^*)$  ( $T^*$  - total diffusion time) in case of a complete graph.

Note that analytically deriving  $\hat{E}(t_1)$  assuming a discrete (i.e., for a node the delay between two successive contacts is 1 unit) diffusion model becomes severely complex and hence we adopt a continuous variant of the model. In fact, the calculation of the  $\Pr\{t_1=t\}$  can be treated as an expected time of filling the first urn with k balls in the experiment where we have initially d empty urns (degree of the node, for complete graph  $d \sim n$ ) and at each single time step we add a single ball to one urn chosen randomly. For any k-parts message, by [24] we describe our problem as a unit-time Poisson process. Note that for a poisson process the inter-arrival time follows exponential distribution  $(\lambda)$  and expected number of arrivals in time t is  $\lambda t$ .

Table 1: Summary of the notations used.

Symbol	Definition
$t_1$	Time between initiation and creation of first sender
$T^*$	Total diffusion time
$\hat{E}(t_1)$	Expected $t_1$ (obtained analytically)
$\hat{E}(T^*)$	Expected $T^*$ (obtained analytically)
$Av(t_1)$	Expected $t_1$ (obtained empirically)
$Av(T^*)$	Expected $T^*$ (obtained empirically)
$s_1$	Limiting random variable of $t_1 d^{-\frac{k-1}{k}}$ as $d \to \infty$
$\tau_i$	Scaled time span between creation of $(i-1)^{th}$ sender and $i^{th}$ sender
$s^*$	Limiting random variable for $T^*d^{-\frac{k-1}{k}}$

Let  $X_j(t)$  be a random value describing the number of balls in  $j^{\text{th}}$  urn up to time t. More precisely  $X_j(t) = \sum_{i=1}^N 1$  where N is a random variable with Poisson distribution  $\mathcal{P}(\frac{t}{d})$ . Essentially N represents number of draws of the  $j^{\text{th}}$  urn up to time t if d urns exist in the system. Hence  $\{X_j(t)\}$ s are i.i.ds and  $X_j(t) \sim \mathcal{P}(\frac{t}{d})$ . We formulate the analytical result for complete graph topology through the following theorems 1-5. The various notations used in the paper are further summarized in table 1

**Theorem 1** For a message with k tokens, the expected value of  $t_1$ ,  $\hat{E}(t_1) = \int_0^\infty (1 - P(t_1 \le t)) dt = \int_0^\infty Q(k, \frac{t}{d})^d dt$  where Q(k, u) is a regularized incomplete gamma function and d is the degree.

## Proof.

$$\hat{E}(t_1) = \int_0^\infty (1 - P(t_1 \le t)dt) = \int_0^\infty (1 - (1 - P(t_1 > t))dt) dt$$

$$= \int_0^\infty P(X_j(t) < k)^d dt = \int_0^\infty Q(k, \frac{t}{d})^d dt$$
(1)

where Q(k,u) is a regularized incomplete gamma function i.e.

$$Q(k,u) = \frac{\Gamma(k,u)}{\Gamma(k)} = e^{-u} \sum_{l=0}^{k-1} \frac{u^l}{l!}$$
 (2)

is valid for any natural k and non-negative u.

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Note that  $t_1$  for a Poisson process, is a continuous random variable.

Residual phase: We next proceed to establish the relation between  $\hat{E}(t_1)$  and  $\hat{E}(T^*)$ . Apart from assuming the continuous model, we compute scaled  $t_1$  and  $T^*$  (by  $d^{\frac{k-1}{k}}$ , (this specific scaling function was initially calculated for k=2 and then generalized for higher values)) instead of their explicit values to further aid our analysis. To summarize, we start by computing expected values of scaled  $t_1$  and  $T^*$  considering a continuous (Poisson) model with  $d\to\infty$  and show that the results hold for finite d. Finally we show that the results for the continuous model extend to the discrete model. We begin by showing that  $t_1$  (time to create the first sender apart from the initiator) is an indicator for the diffusion delay  $T^*$  through the following two theorems.

**Theorem 2** For a message with k tokens the random variable  $t_1 d^{-\frac{k-1}{k}}$  converges as  $d \to \infty$  to a limiting random variable  $s_1$  with density  $\frac{x^{k-1}}{(k-1)!}e^{-\frac{x^k}{k!}}$  and expectation  $\hat{E}(s_1) = (k!)^{\frac{1}{k}}\Gamma\left(1+\frac{1}{k}\right)$ .

**Proof.** The proof is based on Poisson clock approximation approach introduced previously. We start from the calculation of the CDF for the random variable  $t_1d^{-\frac{k-1}{k}}$ :

$$F_{t_1d^{-\frac{k-1}{k}}}(x) = P(t_1d^{-\frac{k-1}{k}} \le x)$$

$$= 1 - Q(k, \frac{xd^{\frac{k-1}{k}}}{d})^d$$

$$= 1 - \left(\sum_{i=0}^{k-1} e^{-xd^{-\frac{1}{k}}} (xd^{-\frac{1}{k}})^i / i!\right)^d$$
(3)

where in the last line we used the common simple approximation of Poisson cumulative distribution in long tail.

Since we are interested in limits as  $d\to\infty$ , for  $\exp(-xd^{-\frac{1}{k}})\to 1$  and we can compute limits of  $F_{t_1d^{-\frac{k-1}{k}}}$  as follows:

$$F_{s_1}(x) = \lim_{d \to \infty} F_{t_1 d^{-\frac{k-1}{k}}}(x)$$

$$= \lim_{d \to \infty} 1 - \left(\sum_{i=0}^{k-1} e^{-xd^{-\frac{1}{k}}} (xd^{-\frac{1}{k}})^i / i!\right)^d$$

$$= 1 - e^{(-x^k/k!)}$$
(4)

Now, the density function of  $\tau_1$  can be calculated as -

$$f_{s_1}(x) = \frac{dF_{s_1}}{dx}(x) = \frac{x^{k-1}}{(k-1)!} \exp\left(-\frac{x^k}{k!}\right)$$
 (5)

Further the expectation of  $s_1$  is -

$$\hat{E}(s_1) = \int_0^\infty x f_{s_1}(x) dx = \int_0^\infty (uk!)^{\frac{1}{k}} e^{-u} du 
= (k!)^{\frac{1}{k}} \int_0^\infty u^{\frac{1}{k}} e^{-u} du = (k!)^{\frac{1}{k}} \Gamma\left(1 + \frac{1}{k}\right)$$
(6)

We next compute the expectation of the (scaled) time  $T^*$  till all nodes become senders. We consider  $T_i = t_i d^{-\frac{k-1}{k}}$ . We further assume  $\tau_i$  as the scaled time span between the creation of  $(i-1)^{\text{th}}$  new sender and that of  $i^{\text{th}}$  new sender. Correspondingly  $\tau_i^*$  represents the scaled time for the original process where every node with at least k tokens acts as a sender node. We have  $\hat{E}(\tau_i^*) = \frac{1}{i}\hat{E}(\tau_i) = \frac{1}{i}\left(\hat{E}(T_i) - \hat{E}(T_{i-1})\right)$ . Note that  $\tau_1$  is equal to  $T_1$  as  $T_0$  is 0 and hence  $\hat{E}(s_1)$  equals  $\hat{E}(\tau_1)$ .

**Theorem 3**  $T^*d^{-\frac{k-1}{k}}$  converges to a limiting random variable  $s^*$  with

$$\hat{E}(s^*) = \sum_{i=1}^{\infty} \hat{E}(\tau_i^*) = \hat{E}(\tau_1) \frac{k}{k-1}$$

**Proof.**  $G_i^{(d)}(z) = 1 - F_i^{(d)}(z) = \Pr\left\{t_i > zd^{\frac{k-1}{k}}\right\} = \Pr\left\{T_i > z\right\} \text{(complementary cdf of } T_i\text{). Since } \frac{zd^{\frac{k-1}{k}}}{d} = zd^{-\frac{1}{k}} \text{ we have}$ 

$$G_{i}^{(d)}(z) = \sum_{j=0}^{i-1} {d \choose j} \left( \sum_{l=0}^{k-1} \frac{1}{l!} \left( z d^{-\frac{1}{k}} \right)^{l} e^{-z d^{-\frac{1}{k}}} \right)^{d-j}$$

$$\left( 1 - \sum_{l=0}^{k-1} \frac{1}{l!} \left( z d^{-\frac{1}{k}} \right)^{l} e^{-z d^{-\frac{1}{k}}} \right)^{j}$$

$$= \sum_{j=0}^{i-1} \frac{1}{j!} \left( \frac{z^{k}}{k!} \right)^{j} e^{-\frac{z^{k}}{k!}} (1 + o_{d}(1))$$
(7)

taking limits  $d \to \infty$  and using the abbreviation  $a = \frac{z^k}{k!}$  we obtain for  $G_i(z) = \lim_{d \to \infty} G_i^{(d)}$  and

$$G_i(z) = \sum_{j=0}^{i-1} \frac{a^j}{j!} e^{-a}$$
 (8)

Subsequently, we get for  $\hat{E}(\tau_i)$  $\int (G_i(z) - G_{i-1}(z)) dz$ :

$$\hat{E}(\tau_i) = \int_0^\infty \frac{1}{(i-1)!} \left(\frac{z^k}{k!}\right)^{i-1} e^{-\frac{z^k}{k!}} dz, \qquad (9)$$

$$\hat{E}(\tau_i^*) = \int_0^\infty \frac{1}{i} \frac{1}{(i-1)!} \left(\frac{z^k}{k!}\right)^{i-1} e^{-\frac{z^k}{k!}} dz. \quad (10)$$

For computing  $\sum_{i=1}^{N} \hat{E}\left(\tau_{i}^{*}\right)$  we can exchange integration and summation. Hence we first estimate

$$\lim_{N \to \infty} \sum_{i=1}^{N} \frac{1}{i!} a^{(i-1)} e^{-a} = \frac{1}{a} \left( 1 - e^{-a} \right)$$
 (11)

Transforming variables in the integral as  $y = \frac{z^k}{k!}$  we finally get

$$\hat{E}(s^*) = \frac{(k!)^{\frac{1}{k}}}{k-1} \Gamma\left(\frac{1}{k}\right) = \hat{E}(\tau_1) \frac{k}{k-1}$$
 (12)

We observe from the above result that the expectation of scaled  $T^*$  converges to a value which depends only on k which is constant for a given setting. Hence we conclude that the expectation of  $T^*$  is proportional to  $d^{\frac{k-1}{k}}$  and similarly for  $t_1$ .

The above results are based on the assumption that i is fixed as  $d \to \infty$ . We now proceed to show that the computations hold for finite d for i varying with d. Note that the above formulas hold true for  $i \le f(d)$  as long as  $f(d) = o\left(d^{\frac{1}{k}}\right)$ .

**Theorem 4** Considering that the range of i (number of senders) varies with d (degree) if  $f(d) = d^{\frac{1}{k} - \epsilon}$  for some  $\epsilon > 0$ ,  $\sum_{i>f(d)}^{d} \tau_i^*(d) = o_d\left(\sum_{i\geq 1}^{f(d)} \tau_i^*(d)\right)$ 

**Proof.** We compute first  $\mathbb{E}(\tau_i) = \int_0^\infty \frac{1}{(i-1)!} \left(\frac{z^k}{k!}\right)^{i-1} e^{-\frac{z^k}{k!}} dz$  using again the transformation of variables  $y = \frac{z^k}{k!}$ 

$$\hat{E}(\tau_i) = \frac{1}{(i-1)!} \frac{(k!)^{1/k}}{k} \int_0^\infty y^{i-2+\frac{1}{k}} e^{-y} dy \quad (13)$$

$$= \frac{1}{(i-1)!} \frac{(k!)^{1/k}}{k} \Gamma(i-1+1/k) \quad (14)$$

Using Stirlings approximation we obtain -

$$\frac{\Gamma(i-1+1/k)}{(i-1)!} \simeq e^{-2+\frac{2}{k}} \frac{1}{(i-1)^{1-1/k}}$$
 (15)

The further argumentation is independent of the involved constant coefficients since we only need leading orders.

We have

$$T_{L} = \sum_{1}^{L} \hat{E}(\tau_{i}) = O(1) \cdot \sum_{1}^{L} \frac{\Gamma(i-1+1/k)}{(i-1)!}$$
$$= O(1) \int_{1}^{L} \frac{1}{x^{1-1/k}} dx = O(1) L^{1/k}.$$
 (16)

Note once more that all the computations up to now are in scaled time units. Hence in real time we have  $t_L\sim L^{1/k}d^{1-\frac{1}{k}}.$  Setting  $L\sim d^{\frac{1}{k}-\epsilon}$  we get  $t_L=d^{1-\frac{1}{k}+1/k^2-\frac{\epsilon}{k}}.$  Taking  $t_L$  as unit and taking into account that the total time  $t^*$  (in the scaled process) is according to the results in Erdös and Kaplan [24]  $t^*=(1+o(1))\,d\log d$  we have  $t^*=O\left(1\right)\cdot t_L\cdot d^{\frac{1}{k}-1/k^2+\frac{\epsilon}{k}}\log d.$  But since the acceleration at this point is  $d^{1/k-\epsilon}$  we have for the remaining time (that is the time after the  $d^{\frac{1}{k}-\epsilon}$ -th event) in the accelerated process a contribution of at most  $\tilde{t}_L d^{-1/k^2+\frac{\epsilon}{k}+\epsilon}\cdot \log d=\tilde{t}_L\cdot o_d\left(1\right)$  since  $\epsilon$  can be chosen arbitrary small - here  $\tilde{t}_L$  denotes the time till the  $L^{\rm th}$  event in the accelerated process. This shows that  $\sum_{i>f(d)}^d \tau_i^*\left(d\right) = o_d\left(\sum_{i\geq 1}^{f(d)} \tau_i^*\left(d\right)\right).$ 

The above results show that previous computations (considering  $d \to \infty$ ) give correct limiting values. This indicates that our analysis is able to correctly estimate the diffusion time for a complete graph of finite size.

It now remains to show that the asymptotic estimations for the model with Poisson clock carry over to the discrete time model (which we use for our simulations) defined at the beginning. Note that the discrete time model is actually the Poisson model when looked at in event time steps, where events here are the times when a token is sent

**Theorem 5** If  $t_i$  is the time between the creation of the  $(i-1)^{th}$  and  $i^{th}$  sender and  $\hat{t}_i$  is the corresponding time in the discrete model, then  $\hat{E}(\hat{t}_i) = \hat{E}(t_i)(1 + o_d(1))$ 

**Proof.** Since we have i independent senders all acting with Poisson clocks of intensity 1 we have the time between two tokens sent - denoted in the following by a random variable x - to be an exponential distribution  $Exp\left(i\right)$ .

We index the events by l and observe that  $t_i = \sum_{l=1}^{K_i} x_l$  where  $K_i$  is the random stop time when the  $i^{\text{th}}$  sender is created. In the discrete model i messages are sent simultaneously hence i successive events in the Poisson model correspond to one time step in the discrete model. Hence  $\hat{t}_i = \left\lfloor \frac{1}{i} \cdot K_i \right\rfloor = \frac{1}{i} \cdot K_i \cdot (1 + o_d(1))$ . Since the  $\{x_l\}$ s are i.i.ds we can apply Wald's theorem [25] and get

$$\hat{E}(t_i) = \hat{E}(K_i)\,\hat{E}(x) = \frac{1}{i}\hat{E}(K_i)$$

hence  $\hat{E}(\hat{t}_i) = \hat{E}(t_i) (1 + o_d(1))$  and the analytical results for the poisson model hold for the discrete case as well.

We further simulated our diffusion model on complete graphs to verify our analytical results. For this purpose, we plot in figure 3 the values of average diffusion time  $(Av(T^*))$  and average time to create the first sender  $(Av(t_1))$  respectively as we vary the size of the network. We further report the values of  $Av(T^*)$  and  $Av(t_1)$  for different values of k with network size fixed at 1000. Note that the two quantities  $Av(T^*)$  and  $Av(t_1)$  (the results were averaged over 1000 simulations) exhibit a very similar profile irrespective of the chosen value of k. In the same figure we also plot the function  $d^{\frac{k-1}{k}}$  (represented by  $\hat{E}(t_1)$ ) obtained from theorem 2, suitably scaled by a constant to show how the theoretical results closely follow the numerical simulations.

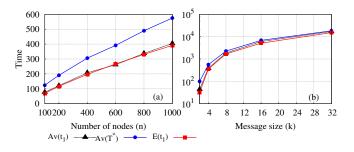


Fig. 3:  $Av(T^*)$  and  $Av(t_1)$  versus (a) the number of nodes with message size k=4 and (b) k for fixed d=1000. For both the plots  $\hat{E}(t_1)=C*d^{\frac{k-1}{k}}$  where  $C=(k!)^{\frac{1}{k}}\Gamma\left(1+\frac{1}{k}\right)$  (refer to theorem 2).

E-R random graph: We further look into **Erdos-Renyi** random graphs [26] and observe that for sufficiently dense graphs (i.e., having high edge probability) the analysis on the complete graph case holds. In this regard we first plot  $Av(T^*)$  (obtained through simulations) and  $\hat{E}(T^*)$  ( $n^{\frac{k-1}{k}}$  scaled by a constant, n is the number of nodes) for different values of k (message size) (refer to figure 4(a)). Clearly the theoretical estimate closely follows the simulated result. As we increase the value of edge probabilities(p) (i.e., make the network more dense) the closer it gets to the theoretical estimate. We further plot  $Av(T^*)$  (scaled by  $\hat{E}(t_1)$ ) for varying p in figure 4(b). The value gets close to 2 with edge probability 1 but remains

close to 2 even for lower values of p. All the results are averaged over 1000 simulations.

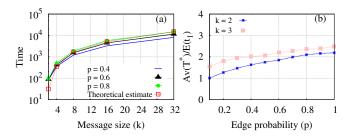


Fig. 4: (a)  $Av(T^*)$  and  $\hat{E}(T^*)$  (suitably scaled) for different values of k (b)  $Av(T^*)$  versus edge probability in Erdos-Renyi random graph for k=2 and k=3. In both cases  $Av(T^*)$  is normalized by  $\hat{E}(t_1)$  which is  $\sqrt{n}$  and  $n^{\frac{2}{3}}$  for k=2 and k=3 respectively.

On d-regular tree:. – We next consider the case of d-regular trees for  $d \geq 3$  (at least 2 children apart from 1 parent) with a distinguished root index 0 which acts as the initial sender. For simplicity we give the root an outdegree of (d-1) by attaching a virtual "mother vertex" to the root which is also a sender but has only one offspring and is not counted in the estimation of sender nodes (this helps us avoid handling the initial steps (i.e., when only the root is having the message) differently from the later steps). Let  $A_{l}(t)$ ,  $0 \le l < k$ , be the number of nodes on the tree which have exactly l packets at time t and have a direct communication link to one of the sender nodes at time t. Note that each of the so defined nodes has exactly one connection to a sender node due to the tree structure and the initial condition of having just one sender at the beginning. We get the following exact linear recursion for the expectation  $a_l(t+1) := \hat{E}(A_l(t+1))$  at time t+1:

$$a_{l}(t+1) = \frac{d-1}{d}a_{l}(t) + \frac{1}{d}a_{l-1}(t), 1 \le l \le k-1$$

$$a_{0}(t+1) = \frac{d-1}{d}a_{k-1}(t) + \frac{d-1}{d}a_{0}(t)$$

Note that for the expected number of sender nodes  $s_t$  at time t, we have

$$s_{t} = \sum_{t' < t} \frac{1}{d} a_{k-1} (t')$$
 (17)

The asymptotic rate of growth of the variables  $\{a_i(t)\}$  as well as  $s_t$  is entirely determined by the value of the largest eigenvalue of the associated transition matrix. The maximal eigenvalue of the associated characteristic polynomial is given by

$$\lambda_{\max} = \frac{d-1}{d} + \left(\frac{d-1}{d} \left(\frac{1}{d}\right)^{k-1}\right)^{\frac{1}{k}} = \frac{d-1 + (d-1)^{1/k}}{d}$$

In figure 2(b) we draw the diffusion dynamics for a 5-regular tree and in the same figure we show that the analytical estimate (obtained from equation 17) of diffusion rate closely resemble the empirical observation.

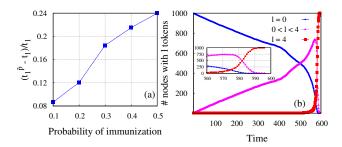


Fig. 5: (a)  $\frac{t_1^{\bar{p}}-t_1}{t_1}$  versus  $\bar{p}$  for a complete graph with 1000 nodes and k=4. (b) Number of nodes at each stage of infection versus time for complete graph of 1000 nodes with k=4. Although the creation of infected nodes is slow initially, the number of partially infected nodes (0 < l < 4) increases rapidly. (inset) Magnified version of the same figure.

**Discussion.** – We have defined the epidemic setting aptly fitting the temporal network system whereby agents remember interactions from the previous time-steps and only adopt an idea after encountering it multiple times. We find that such information diffusion process undergoes two phases with a slow initial phase followed by a very fast residual phase. This behavior is observed irrespective of the underlying topology (refer to figure 2, simulations done for scale-free graphs [27] and ER-random graphs). The reason behind such behavior is that during the process when the first few nodes get fully infected a large fraction of nodes also simulataneously get partly infected as represented in figure 5(b). Once the first set of infected nodes are created these partially infected nodes also get quickly infected and this results in a sudden ramp-up in the diffusion rate.

The above observations indicate that for such systems spreading could be controlled/contained while the system is still in the slow initial phase. Accordingly, we perform an empirical study, whereby, we reduce the level of infection of the agents at one particular time step (say t) and then estimate the time required to obtain the first sender vis-a-vis the time  $t_1$  when such action is not initiated. Reduction of infection means removing one packet from the chosen (say with probability  $\bar{p}$ ) agent, for example, if an agent has acquired j packets at time t, we reduce it to j- 1. In specific we considered a complete graph with 1000 nodes and at  $t = 0.5 * t_1$  we reduce the level of infection in each node with a probability  $(\bar{p})$  and measure the corresponding time (say  $t_1^{\bar{p}}$ ). In figure 5(a) we plot  $\frac{t_1^{\bar{p}}-t_1}{t}$  for different values of  $\bar{p}$ . We observe that creation of the first sender (other than initiator) i.e., beginning of the residual phase (where the rate of spread is increased manifold) could be delayed by almost 20% with a probability of 0.3. We show the experiment by inoculating at one particular time step, however a continuous low-grade innoculation strategy can be initiated and we believe a threshold can be derived whereby the first sender creation can be pushed to infinity. However that can be an interesting research direction to be pursued in the future.

We believe that our findings could open up paths to a number of future studies especially regulating contagion processes in systems with memory. We note that our analysis does not consider the fact that the extent of influence decays with time as observed in several real-world diffusion processes. We believe such investigation calls for additional research efforts.

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## REFERENCES

- [1] Anderson R. M., May R. M. and Anderson B., Infectious diseases of humans: dynamics and control Vol. 28 (Wiley Online Library) 1992.
- [2] WATTS D. J., Proceedings of the National Academy of Sciences, 99 (2002) 5766.
- [3] Dodds P. S. and Watts D. J., Physical review letters, 92 (2004) 218701.
- [4] KRAMER A. D., GUILLORY J. E. and HANCOCK J. T., Proceedings of the National Academy of Sciences, 111 (2014) 8788.
- [5] ARAL S., MUCHNIK L. and SUNDARARAJAN A., Proceedings of the National Academy of Sciences, 106 (2009) 21544.
- [6] TANG M., LIU Z. and LI B., EPL (Europhysics Letters), 87 (2009) 18005.
- [7] Son S.-W., Bizhani G., Christensen C., Grass-Berger P. and Paczuski M., *EPL (Europhysics Letters)*, **97** (2012) 16006.
- [8] TAKAGUCHI T., MASUDA N. and HOLME P., PloS one, 8 (2013) e68629.
- [9] KARSAI M., KIVELÄ M., PAN R. K., KASKI K., KERTÉSZ J., BARABÁSI A.-L. and SARAMÄKI J., Physical Review E, 83 (2011) 025102.
- [10] KARIMI F. and HOLME P., Physica A: Statistical Mechanics and its Applications, **392** (2013) 3476.
- [11] BACKLUND V.-P., SARAMÄKI J. and PAN R. K., Physical Review E, 89 (2014) 062815.
- [12] ROCHA L. E. and BLONDEL V. D., PLoS Comput Biol, 9 (2013) e1002974.
- [13] MASUDA N. and HOLME P., F1000 prime reports, 5 (2013) 6.
- [14] BOGUÑÁ M., CASTELLANO C. and PASTOR-SATORRAS R., Physical review letters, 111 (2013) 068701.
- [15] VAN MIEGHEM P., EPL (Europhysics Letters), 97 (2012) 48004.

- [16] ZHANG Y.-Q. and LI X., EPL (Europhysics Letters), 108 (2014) 28006.
- [17] JOH R. I., WANG H., WEISS H. and WEITZ J. S., Bulletin of mathematical biology, 71 (2009) 845.
- [18] SANGHAVI S., HAJEK B. and MASSOULIÉ L., IEEE Transactions on Information Theory, 53 (2007) 4640.
- [19] QIU D. and SRIKANT R., Modeling and performance analysis of bittorrent-like peer-to-peer networks in proc. of ACM SIGCOMM computer communication review Vol. 34 (ACM) 2004 pp. 367–378.
- [20] ROMERO D. M., MEEDER B. and KLEINBERG J., Differences in the mechanics of information diffusion across topics: idioms, political hashtags, and complex contagion on twitter in proc. of Proceedings of the 20th international conference on World wide web (ACM) 2011 pp. 695–704.
- [21] Granovetter M., American journal of sociology, (1978) 1420.
- [22] MOLLISON D., Epidemic models: their structure and relation to data Vol. 5 (Cambridge University Press) 1995.
- [23] Volz E. and Meyers L. A., Proceedings of the Royal Society of London B: Biological Sciences, 274 (2007) 2925.
- [24] Kaplan N., Journal of Applied Probability, (1977) 212.
- [25] Wald A., The Annals of Mathematical Statistics, 15 (1944) 283.
- [26] Erdős P. and Rényi A., Publicationes Mathematicae (Debrecen), 6 (1959) 290.
- [27] BARABÁSI A.-L. and ALBERT R., science, 286 (1999) 509.