# Diffusion in networks and the virtue of burstiness

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Whether an idea, information, or infection diffuses throughout a society depends not only on the structure of the network of interactions, but also on the timing of those interactions. People are not always available to interact with others, and people differ in the timing of when they are active. Some people are active for long periods and then inactive for long periods, while others switch more frequently from being active to inactive and back. We show that maximizing diffusion in classic contagion processes requires heterogeneous activity patterns across agents. In particular, maximizing diffusion comes from mixing two extreme types of people: those who are stationary for long periods of time, changing from active to inactive or back only infrequently, and others who alternate frequently between being active and inactive.

social networks | diffusion | contagion | bursty | random networks

Networks of interactions are the backbone of a range of diffusion processes from the spread of simple bits of information to the spread of diseases (e.g., refs. 1–10). Whether and how things diffuse depend on the time patterns of when various nodes in a network are active and can interact with each other. Despite the important role of timing in diffusion, little is known about that role. In this paper we provide a theoretical analysis of how the timing of interactions affects a simple diffusion or contagion process.

It is well known that people's activity patterns are far from being independent of time and history. For example, "burstiness"—consecutive periods of activity followed by consecutive periods of silence—has been documented in a multitude of diffusion processes, from email and phone conversations to gene expressions (11–17). It has also been shown that having populations whose activity patterns are time or history dependent can change the outcome of a diffusion process (13, 18–29), for instance with bursty activity patterns dampening diffusion.

However, it is also clear that people differ in the timing of their active periods. For instance, some people check email on a very frequent and intermittent basis, while others have greater time between activities but then spend a longer time active once they are. Thus, to really understand how activity patterns affect diffusion one should at least consider the possibility that different individuals behave differently. As we show, this turns out to make a big difference. We show that heterogeneity in activity patterns across agents is in fact necessary for maximizing the extent of a diffusion and can substantially increase the expected reach of diffusion processes. Heterogeneity is not just a complication in modeling, but actually systematically changes the way that diffusion works. Activity patterns, like bursty ones, that dampen diffusion if everyone adopts them, actually enhance diffusion when matched with opposite behaviors by some other fraction of the population.

The model that we examine is a variation on the widely studied SIR (susceptible, infected, recovered) model (30), which has its roots in the Reed–Frost model (see ref. 31 for background). Some node of a network is the first one infected with a disease or an idea or a meme, etc. The infection then spreads at random through the network. Nodes are either infected or susceptible. They begin as all being susceptible and become infected if they interact with a contagious neighbor. Once infected, agents are contagious for $T$ periods and then cease to be contagious. Thus, diffusion spreads by having an infected and contagious node interacting with any of its neighbors who are susceptible. The process that we analyze is "simple" in that it takes interaction with only one neighbor to become infected (32). The complication is that the spread from one node to another occurs only if they are both active and manage to meet within the time during which the first node is still contagious. This depends on both of their activity patterns.

In our model, the probability that a node is active is not independent of time. On average, nodes are randomly active during any given period with a probability $\lambda > 0$, but the probability is not independent of the history of that node's past behavior. Nodes' active times follow a Markov chain: The probability that a node is active in one period depends on whether it was active last period. In addition, what most distinguishes our analysis is that neighboring nodes can be following completely different Markov processes. For instance, some nodes are more likely to be active if they were active last period, while others are less likely to be active if they were active in the last period.

Three benchmark types of Markov processes figure prominently in our results. We refer to nodes with extreme positive autocorrelation (nodes very likely to remain active for many consecutive periods and then once they switch to remain inactive for many consecutive periods) as "sticky" nodes, nodes with extreme negative autocorrelation (where they change from active to inactive and back very frequently) as "reversing" nodes, and nodes who are just randomly on or off in every period with the same probability independently of history as "Poisson" nodes.

## Significance

The contagion of disease and the diffusion of information depend on personal contact. People are not always available to interact with those around them, and the timing of people's activities determines whether people have opportunities to meet and transmit a germ, idea, etc., and ultimately whether widespread contagion or diffusion occurs. We show that, in a simple model of contagion or diffusion, the greatest levels of spreading occur when there is heterogeneity in activity patterns: Some people are active for long periods of time and then inactive for long periods, changing their availability only infrequently, while other people alternate frequently between being active and inactive. This observation has policy implications for limiting contagious diseases as well as promoting diffusion of information.

To better understand these three types, consider workers in a large firm who spend $\lambda$ of their time near their offices with their doors open for interaction with other coworkers on their team and then $1 - \lambda$ of their time working on projects with closed doors or away from their offices. Let us think of periods as hours. Sticky agents are those who schedule their closed-door time in large clumps, so that they are unavailable for some number of hours in a row and then available with an open door for some number of hours in a row, etc., so that they schedule their sequestered work time in contiguous segments spending days in a row on a project and then days in a row available in the office. If they are available with an open door at some point, they are more likely to be available the next hour, and conversely if they are occupied or away, then that is also likely to persist. Poisson agents are people who just randomly schedule closed-door time with no particular pattern. Reversing agents are people who prefer to alternate, so they work on a project for an hour, then open their door and interact for an hour, and then close their door and work for an hour, etc., alternating project time with interaction time.

We emphasize that we still maintain that the timing of activity is independent across nodes and nodes must all have the same average level of activity—so that every node is active a fraction of $\lambda$ of all periods. In the office example, all workers have the same amount of time with their doors open; it is how they schedule their open-door periods that may differ and matter. The key in our analysis is allowing different nodes to have different time dependencies in their behaviors.

Our main results illustrate that configurations of nodes that maximize the extent of diffusion, as well as the probability of an epidemic, are those that have different Markov chains for different nodes. We show that it is never maximizing to have all nodes follow the same Markov chain: Heterogeneity is necessary to maximize diffusion. We also fully characterize the maximizing structure of heterogeneity for a few simple networks such as chain and star networks, providing the basic intuition as to why it is useful to have heterogeneity and illustrating that it can help. Combining nodes with extreme positive autocorrelation (sticky nodes) with others who have extreme negative autocorrelation (reversing nodes) is optimal in such simple networks. As a by-product, this also shows which structures minimize diffusion (generally homogeneous sticky nodes). Depending on the application, one may wish to maximize or minimize diffusion. Regardless of what one wishes to do, understanding how heterogeneity matters is essential for shaping policy. As the general problem of characterizing the optimal structures for complex networks appears intractable, we analyze some others by simulation.

Fig. 1 illustrates how much of a difference having heterogeneity can make. We examine diffusion on an Erdős–Renyi random network (a network on $n$ nodes where there is an edge between any two nodes with probability $p$, independently across pairs) in which agents are either Poisson (they are active each period with probability $\lambda$) or sticky (they are either active in all periods with probability $\lambda$ or inactive in all periods with probability $1 - \lambda$). Fig. 1 shows how the probability of all nodes becoming infected behaves as we vary the relative fraction of Poisson and sticky nodes (and similar results hold for the expected fraction of infections). Consistent with the previous literature, if all nodes are sticky, diffusion is less likely than when all nodes are Poisson. However, when we allow agents to have heterogeneous behavior, the likelihood of full diffusion is maximized when some agents are sticky and some are Poisson.

To understand why heterogeneity increases diffusion, consider an agent who has recently been infected (and was just active). To maximize the chance of diffusion, it is best if this agent behaves in a positively correlated way—a sticky way—so that she is more
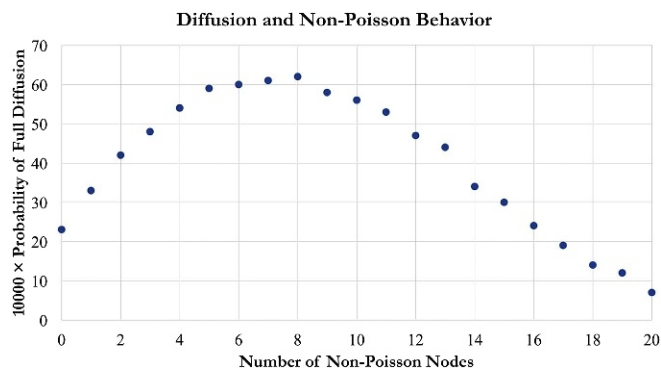


**Fig. 1.** The probability that all nodes get infected as a function of the fraction of non-Poisson (sticky) nodes in an Erdős–Renyi random network, when agents are either Poisson (i.i.d. active in each period) or sticky (they stay in the same state they were in the starting period, with a random starting state). When around 40% of nodes are sticky and the rest are Poisson, the likelihood of a full diffusion is three times more than when all nodes are Poisson. Clearly, mixing bursty behavior with nonbursty behavior substantially helps the diffusion. See *SI Appendix* for details behind the simulation.

likely to remain active during the immediate periods after infection, while she is contagious. On the other hand, when an agent is not yet infected, then being sticky lowers probabilities of transmission, since if a sticky agent happens to be inactive, then there is little chance of becoming active in the near future to become infected. So, sticky agents are poor receivers but good senders. Conversely, reversing agents are good receivers but poor senders. The key is that the gain from matching a good sender and a good receiver outweighs the loss from putting together a poor sender and a poor receiver, since both matchups happen when we pair different types of agents together. There is a positive synergy from a sticky agent's sending ability and a reversing agent's receiving ability: They each maximize each other's chance of a successful transmission when the sticky agent is sending and the reversing agent is receiving. On the other hand, sometimes the roles are switched. The key to the gain from heterogeneity is that there is not much loss from pairing them when the roles are reversed. The sticky agent is a poor receiver no matter who she is paired with and it makes little difference what that choice is. Hence, pairing sticky and reversing agents maximizes the sending advantage of the sticky agents and the receiving advantage of the reversing agents, without much loss from the receiving disadvantage of the sticky agent facing a reversing sender. Optimality requires this sort of pattern.

It is important to emphasize that this is not a small effect: As seen in Fig. 1, changing just under half of the agents in a Poisson population to be sticky increases the chance of full contagion by almost a factor of 3 in a uniformly random network. (We see similar orders of magnitude for getting a fraction of nodes infected, also get even bigger gains from mixtures of reversing and sticky agents, and also explore other metrics for comparison in *SI Appendix*.)

This has implications since diffusion processes are critical in many settings from spreading news about a new program or idea to inhibiting the spread of a disease, false rumor, or computer virus. In some of these settings it may be possible to incentivize agents to even slightly alter activity patterns to enhance diffusion or, in a case in which contagion is detrimental, to dampen the diffusion.

## The Model

There are $n \geq 3$ agents, with labels $i \in N = \{1, \ldots, n\}$ connected in a network represented by a simple graph $G = (N, g)$, where $g \subseteq N^2$ and $ij \in g$ if agent $i$ and agent $j$ are linked.

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Time passes in discrete periods $t \in \{1, 2, \ldots\}$. (We work in discrete time to easily admit negative autocorrelation. For this problem, discrete time seems to simplify rather than complicate the analysis and intuitions.) Agents are either active or inactive in a given period. Activity is independent across agents. An agent is active with a probability $\lambda \in (0, 1)$ in any given period, on average. We assume the long-run average activity levels are the same for all agents. By focusing on agents who are homogeneous in how often they participate, we can isolate the effect of heterogeneities in the timing of participation on diffusion.

In particular, an agent's activity follows a Markov chain. If an agent $i$ is active in period $t$, then she or he is inactive in period $t + 1$ with probability $p_i$ and active with probability $1 - p_i$. Similarly, if an agent $i$ is inactive in period $t$, then she or he is active in period $t + 1$ with probability $q_i$ and inactive with probability $1 - q_i$. This is shown in Fig. 2.

For any fixed $\lambda$, an agent is then completely characterized by $p_i$ or equivalently by $q_i$. In particular, the following equality must hold,

$$\lambda p_i = (1 - \lambda) q_i,$$

which is just the usual balance equation of the Markov chain, given that $\lambda$ is the steady-state probability of activity. Some useful rewritings of this equation are

$$\frac{\lambda}{1 - \lambda} = \frac{q_i}{p_i}, \qquad \lambda = \frac{1}{1 + \frac{p_i}{q_i}}, \qquad q_i = p_i \frac{\lambda}{1 - \lambda}.$$

So, our agents are completely described by $p_i$, given any fixed $\lambda$.

**Three Benchmark Types.** There are three levels of autocorrelation that serve as benchmarks.

A Poisson agent is one who has $p_i = 1 - \lambda = 1 - q_i$. This is an agent who is active at every period with probability $\lambda$; that is, her state is i.i.d. over time.

A sticky agent is one who has $p_i$ and $q_i$ both "near" 0. This is an agent whose state is (almost) perfectly autocorrelated over time. In particular, let sticky agents be those who are either always on (with probability $\lambda$) or always off (with probability $1 - \lambda$). So this is the limit of a Markovian agent as $\min[p_i, p_i \frac{\lambda}{1 - \lambda}] \to 0$, but one that is degenerate.

A reversing agent is one with the maximal possible $p$ and $q$ (maximal negative autocorrelation): So $p = 1$ if $\lambda \leq 1/2$ and $p = (1 - \lambda)/\lambda$ if $\lambda \geq 1/2$. Similarly, $q = 1$ if $\lambda \geq 1/2$ and $q = \lambda/(1 - \lambda)$ if $\lambda \leq 1/2$. Thus, the state of a reversing agent is as negatively serially correlated as possible, switching back and forth between being active and inactive as frequently as possible. In the case in which $\lambda = 1/2$, a reversing agent simply reverses its state every period.

Poisson, sticky, and reversing agents are canonical cases: one with no autocorrelation, one with maximal positive autocorrelation, and the other with maximal negative autocorrelation. Of course, there are other levels of autocorrelation in an agent's state, and we admit arbitrary cases in our general analysis.

**Diffusion.** Some agent is initially infected. All other agents are initially susceptible. Once an agent becomes infected, the agent stays infected forever after. An agent can transmit infection for $T$ periods after being infected. We say that such an agent is "contagious" during those time periods. In each period, an agent who is contagious transmits the infection to a neighbor if and only if both he and his neighbor are active and his neighbor is susceptible.

**Line Networks and Canonical Agents**

We begin our analysis by looking at networks that are "lines"— a tree in which no agent has degree more than two. Fig. 3 is a line network with five nodes. These networks illustrate the main ideas and intuitions and permit a complete characterization of the maximizing configurations when we restrict our attention to the canonical agents.

We begin with an analysis of diffusion with only Poisson and sticky agents—as these are sufficient to provide the basic intuitions about how heterogeneity helps with improving diffusion. After establishing results on optimal configurations with these types, we then add in the reversing agents, showing that optimal configurations mix the extreme agents: sticky and reversing agents. Finally, we turn to an analysis with general agent types.

**Poisson and Sticky Agents.** Let $PS$ denote the probability that a Poisson agent who is infected transmits to a sticky neighbor who is susceptible within $T$ periods; and similarly define $SP$, $PP$, and $SS$. Similarly, let $PPP$ denote the probability that there is full transmission among three Poissons in a line— where transmission must occur within a new $T$ period for each successive interaction. So, once infected, a person can pass the disease or idea along to a neighbor for $T$ periods from the date of the current agent's first infection. Likewise, we define $PSP$, and so forth. We abuse notation and occasionally also use this notation to refer to a particular configuration of agents.

When we consider the transmission in a line, we presume that the initially infected agent is a node at one end and that the agent is then randomly active with probability $\lambda$ in the first period. If we instead assumed that the first agent begins by being active, then the first agent should always be sticky, and that would just push the whole problem back one agent.

In all of the analysis that follows, we presume that agents have the same overall probability of being active, but differ only in timing. Our interest is in seeing how the patterns of timing matter, and holding constant the overall level of activity allows us to isolate how patterns of autocorrelation matter.

**Proposition 1.** *Consider three agents in a line, with all agents being independently active with probability $\lambda \in (0, 1)$ in steady state, and who once infected can transmit for some positive integer number of periods $T$. The configuration of Poisson and sticky agents that maximizes both the expected number of infections and the probability that all agents become infected is uniquely*

- *$PSP$ if $\lambda < \lambda^*$, and*
- *$PPP$ if $\lambda > \lambda^*$,*

*where $\lambda^*$ is the unique solution in $(0, 1)$ to*

$$\lambda = \left[ \frac{1 - (1 - \lambda^2)^T}{1 - (1 - \lambda)^T} \right]^2 \qquad \textbf{[1]}$$
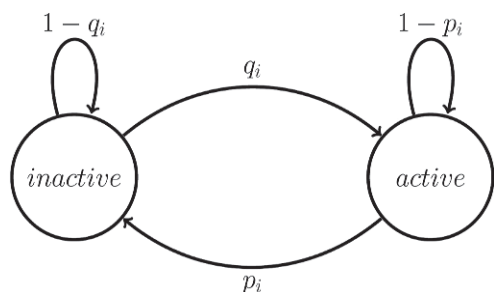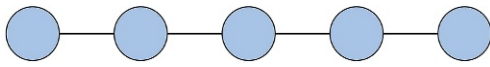
**Fig. 2.** Activity Markov chain of agents.

**Fig. 3.** A line network with five nodes.

*if we are maximizing the probability of total infection and*

$$\lambda = \frac{\left[1 - (1 - \lambda^2)^T\right]\left[2 - (1 - \lambda^2)^T\right]}{\left[1 - (1 - \lambda)^T\right]\left[2 - (1 - \lambda)^T\right]} \qquad [2]$$

*if we are maximizing the expected number of infected nodes.* (*For any $T$, there is a unique fixed solution to* [1] *and* [2] *in* $(0, 1)$, *as we show in the proof.* 1 *and* 0 *are also solutions, but uninteresting ones, as then agents are either always or never active, in which case the time series of their activity is irrelevant.*) *The interior solution of* [2] *is smaller than that of* [1].

The proof of *Proposition 1* and the proofs for all other propositions and theorems are in *SI Appendix*.

To understand the trade-offs that drive heterogeneity note that once an agent is infected, it is best to have that agent be sticky because a recently infected sticky agent remains active while she is contagious. However, when an agent is not yet infected, it is best to alternate states randomly, to enhance coordination probabilities. Sticky agents are poor receivers but good senders, and Poisson agents are good receivers but poor senders. Such dynamics make it optimal to connect a sticky sender and a Poisson receiver. Moreover, the probabilities of transmission have synergies—matching sticky senders with Poisson receivers increases overall probability more than the subsequent loss due to then having to subsequently alternate a Poisson sender with a sticky receiver. Under a wide range of activity levels, the advantages of having heterogeneity outweigh the losses from having the receiver be sticky.

The point of considering the "optimal" configuration is not necessarily to suggest that there is some mechanism designer or planner who can control the system, but to show that heterogeneity enhances diffusion in certain contexts and to understand why this occurs—by showing that it maximizes diffusion, we can see that it definitely enhances diffusion. Our simulations also show that this is not a small effect.

We now show that this intuition extends to longer lines.

**Proposition 2.** *Consider an odd number of agents in a line, with all agents being independently active with probability $\lambda \in (0, 1)$ in steady state, and who once infected can transmit for some positive integer number of periods $T$. Start with one end node being infected and let $\lambda^* \in (0, 1)$ solve* [1] *and $\lambda^{**}$ be the interior solution of $\lambda = \left[1 - (1 - \lambda)^T\right]^2$. Then $0 < \lambda^{**} < \lambda^* < 1$ and the configuration that maximizes the probability of overall infection is*

- *$PSSSS \ldots SP$ if $\lambda < \lambda^{**}$,*
- *$PSPSP \ldots SP$ if $\lambda^{**} < \lambda < \lambda^*$, and*
- *$PPPPP \ldots PP$ if $\lambda > \lambda^*$.*

As we saw in *Proposition 1*, there are similar results for the case of maximizing the expected extent of the diffusion. The cutoff expressions become more complex with longer lines, and so in *Proposition 2* we simply provide the analysis for the probability of overall infection. We can still see the gain from heterogeneity in the following simulation.

To see the extent of the gain from alternation, consider the following results from simulations. We compare the infections in a line of five nodes in which all of the nodes are Poisson to one in which they alternate Poisson and sticky. One of the nodes is picked at random to be infected and we set $T = 2$. We show the comparisons for a full range of $\lambda$. For each of 30 values of $\lambda$ we run 50,000 iterations of drawing a random network and running an infection. The reported values for each $\lambda$ value are the average over the 50,000 iterations.
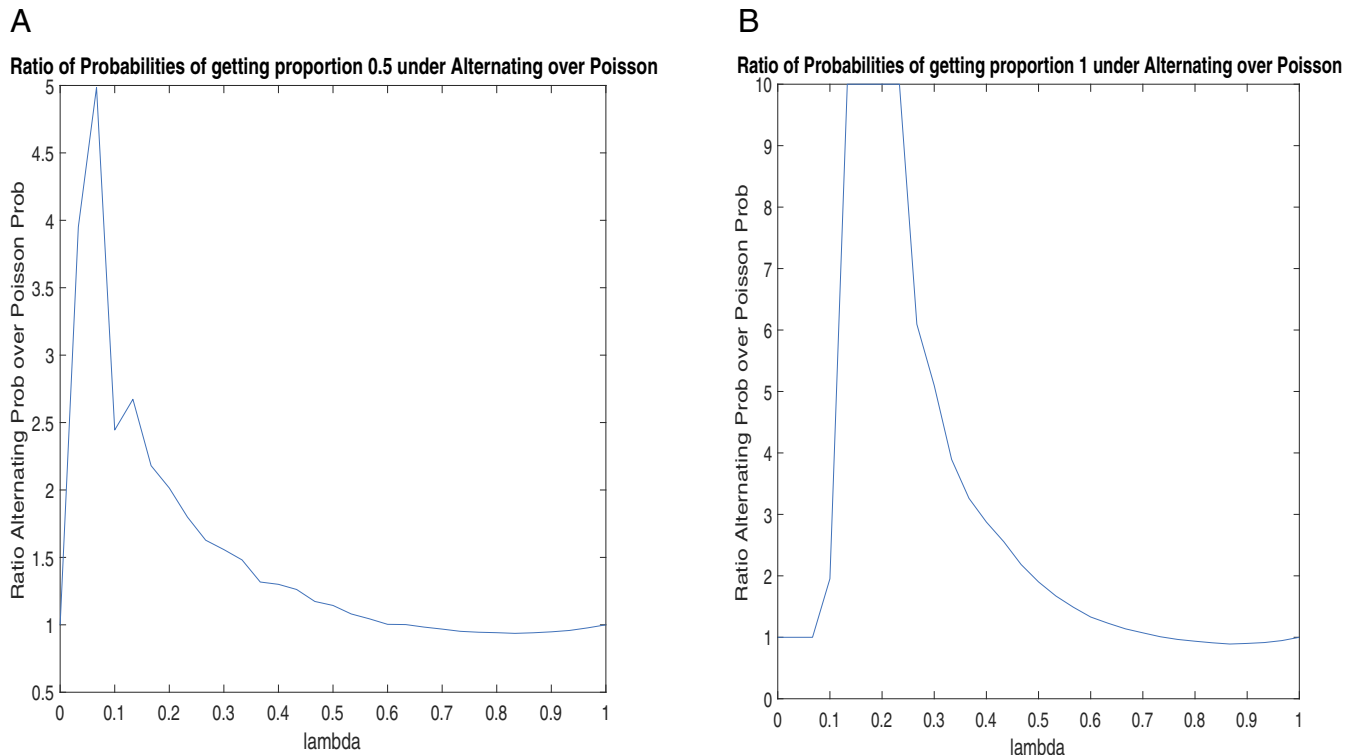
**A**



**B**



**Fig. 4.** The ratio of the infection probabilities under alternating Poisson and sticky nodes over that for all Poisson nodes, on chains of five nodes with one randomly infected. (*A*) Ratio of probability of getting at least half of nodes infected. (*B*) Ratio of probability of getting all nodes infected.

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We see in Fig. 4 that the gains from alternating sticky with Poisson compared with having just Poisson can be very large, more than 400%, while the reverse advantage that appears at high levels of $\lambda$ is relatively negligible.

In the Introduction, we discussed how recent studies showed that bursty (i.e., sticky) behavior slows down the diffusion (19). One may ask whether our results contradict those findings, and the answer is no. In our setting, $SS\cdots S$ is dominated by $PP\cdots P$, which confirms those results. What we prove, however, is that one should not conclude that Poisson behavior is optimal, as those results did not admit heterogeneity. When we consider all possibilities (as in Fig. 1), mixing sticky/bursty behavior with nonbursty behavior substantially improves the diffusion.

We next show that maximizing the probability of a full infection tilts the balance more toward sticky nodes at key junctures or "hubs." To expand on this point, we study the diffusion process on a "star" network. A star network has a central node and $n$ leaves connected to the center. For example, in Fig. 5 we see a star network with four leaves.

**Proposition 3.** *Consider agents in a star network with $n$ leaves, with all agents being independently active with probability $\lambda \in (0,1)$ in steady state, and who once infected can transmit for $T = 2$ periods. Start with some random leaf being infected. Then, for any $\lambda$, there exists some $N$ such that if $n \geq N$, then the configuration that maximizes the probability of a full contagion is to have an $S$ node in the center and $P$ nodes on the leaves.*

To see why *Proposition 3* is true, note the following. First, consider whether the infected leaf infects the center node. This does not depend on how many other leaf nodes there are. Second, consider how the center node interacts with other nodes once infected. Here there is an advantage to having the center be sticky, so that it can transmit in both periods once it is infected. This advantage grows with the number of leaves. Thus, the advantage to having it be sticky as a sender grows with its degree, while the disadvantage of having it be sticky as a receiver does not depend on degree. As degree grows, the advantage can become overwhelming as the expected gain in the diffusion from having the center be sticky compared with Poisson grows in the number of nodes.

*Proposition 2* shows that for $\lambda > \lambda^*$, the configuration that maximizes the expected number of infected nodes does not include $S$ agents. In contrast, *Proposition 3* claims that including sticky behavior is always optimal for agents at sufficiently central junctures. Note that hubs appear in various kinds of networks, from human brain (33, 34) to social networks (35) to computer
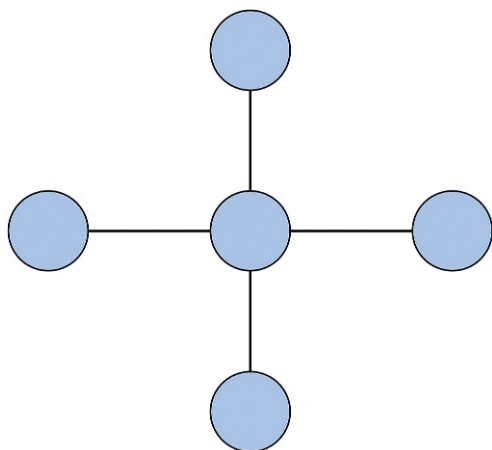
networks (36), and *Proposition 3* suggests that in designing the activity patterns of such nodes with high degrees, autocorrelated behavior can be optimal.

In the next section, we show how "extreme heterogeneity" (i.e., mixing sticky agents with reversing agents, as opposed to mixing sticky and Poisson agents) further improves diffusion.

**Reversing Agents.** Continuing our comparisons, we now consider what happens when we also consider reversing nodes. Reversing nodes work very well when matched with sticky nodes for low values of $\lambda$ and also when mixed with each other if $\lambda$ is very high.

**Proposition 4.** *Consider agents in line and begin with one end node infected, but then randomly active in the first period of its transmission. Suppose that all agents are independently active with probability $\lambda \in (0,1)$ in steady state and once infected can transmit for some positive integer number of periods $T$. If $\lambda < \lambda^*$, then any configuration that maximizes the expected number of infected nodes or the probability of overall infection involves $R$ nodes.*

*Moreover, in the case of $T = 2$, then the optimal configurations involve only $R$ and $S$ nodes (Poisson nodes are not used in the optimal configurations). Those optimal configurations are to have full alternation of the form $RSRSR\ldots SR$ for low values of $\lambda$, all reversing nodes $RRRRR\ldots RR$ for high levels of $\lambda$, and some combinations of a string of $RRRs$ and alternating $SRSR\ldots SR$ for middle values of $\lambda$.*

Having a reversing node following a sticky node maximizes the probability of transmission. For example, if $\lambda > 1/2$, then the probability of transmission from an infected sticky to a reversing node is one. If the reversing node happens to be inactive in one period, then it is active for sure in the next period and then makes contact in at least one of the two periods. Thus, the only loss from having alternating sticky and reversing nodes is from having sticky nodes as receivers, which is biggest for large values of $\lambda$, at which point it is better go entirely to reversing nodes.

Before moving to networks with cycles, we further illustrate our results on a line of five nodes. This shows the differences between various combinations of node types and shows how much improvement comes from including extreme node types and from heterogeneity.

In Fig. 6 we compare the infections in a line of five nodes for various combinations of Poisson, sticky, and reversing nodes. One of the nodes is picked at random to be infected and infected nodes can transmit for $T = 2$. We show the comparisons for a full range of $\lambda$. For each of 30 values of $\lambda$ we run 50,000 iterations of running an infection and average the outcome.

Fig. 6B shows that for the objective of reaching at least half of the nodes, alternating sticky and reversing leads to the best outcomes for $\lambda$ up to about 2/3, and then all reversing is optimal above that. This is as predicted from *Proposition 4*. Thus, sticky— "bursty"—behavior is optimal in combination with reversing for a wide range of values. Fig. 6A shows that for reaching a quarter of the nodes all reversing works for a wider set of values, and reversing and sticky combinations work best only at low values.

## General Networks

We now consider more general networks. Obtaining a full characterization once one introduces cycles into a network appears intractable. The key complication is that a node could be getting contact from several other nodes at once and asynchronously. The full array of possibilities of which nodes become infected when and how that depends on the full network structure and activity patterns of all nodes explodes exponentially. Nonetheless, it is important to make sure that heterogeneity (and bursty)
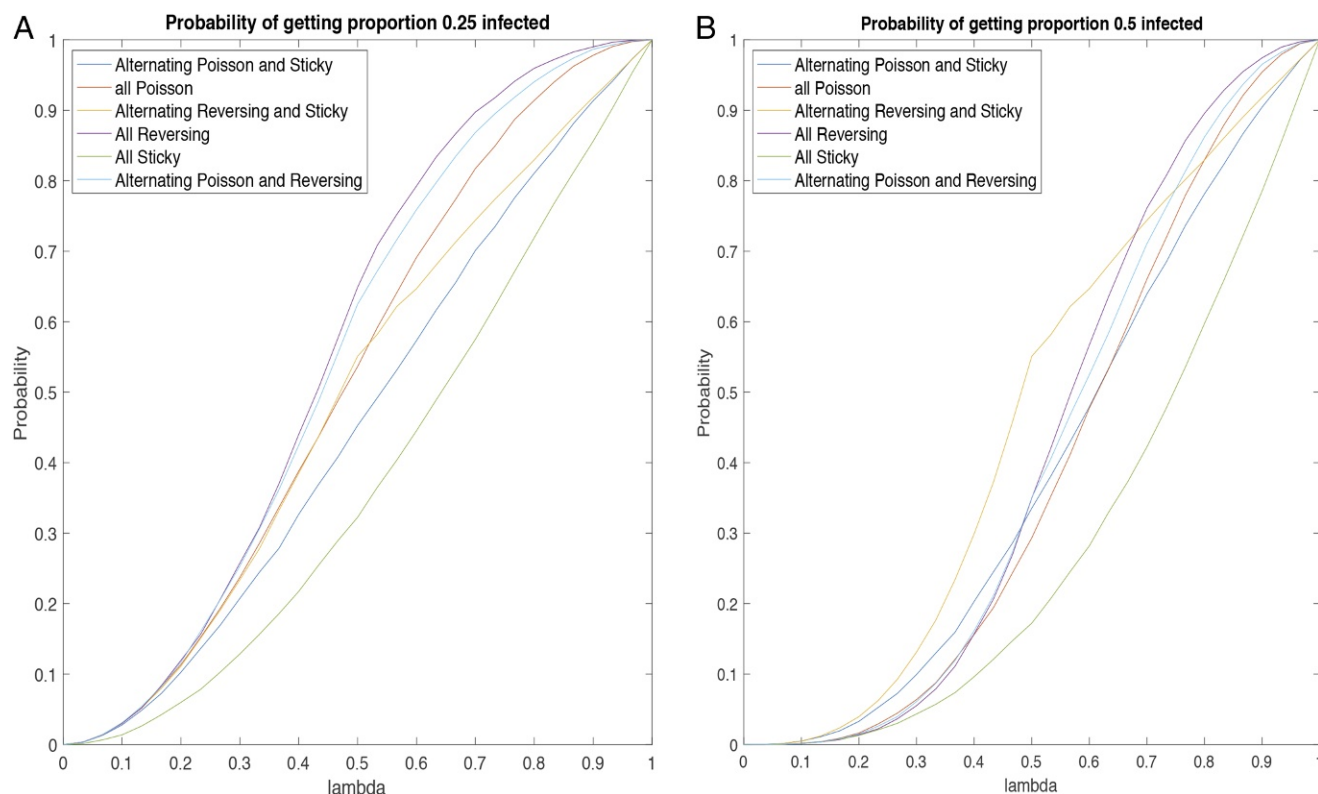


**Fig. 5.** A star network with four leaves.

**Fig. 6.** Comparisons of infection probabilities under various configurations of nodes on lines of five nodes with one randomly infected. (*A*) Probability of getting one-quarter of nodes infected. (*B*) Probability of getting one-half of nodes infected.

behavior can still be diffusion enhancing on such networks. So, our main goal in this section is to show that heterogeneity still plays a substantial role in more complex networks, both through a partial analytic result showing that some heterogeneity enhances diffusion in any network and then via some simulations on some richer networks.

**General Agents and Expected Infection Levels.** We now allow agents to have any $p_i$s, but still all agents have the same long-run probability $\lambda$ of being active to focus on the timing patterns rather than overall levels of activity.

The following result shows that in any network that has some agents who are not in cycles the optimal configuration of agents must involve some sort of alternation/heterogeneity. To make the point that heterogeneity is always optimal, it is sufficient to consider $T = 2$, as the calculations are tractable for that case.

**Proposition 5.** *Consider any path-connected network for which there is at least one node that has degree one (a "leaf") that sits next to a degree two node. Suppose that each agent must be active $\lambda < 1/2$ of the time, independently across agents, and consider $T = 2$. Start with some node other than one of these two nodes being infected. Any configuration of $p_i$s that maximizes either the expected number of infected nodes or the overall probability of full contagion involves $p_i \neq p_j$ for some $i$ and $j$.*

The proof takes advantage of some node that has degree one and its neighbor, which allows us to obtain closed-form expressions for their contagion, fixing the rest of the network. Once nodes enter into cycles, it becomes intractable to calculate the optimal configurations for nodes embedded in cycles.

As *Proposition 5* relies on leaf nodes, it becomes important to also check by simulation that heterogeneity makes a difference in more general networks. To that end we now turn to some simula-

tions to show that the basic intuition that heterogeneity in types can lead to higher rates of contagion extends to some more general networks—and not just occasional leaf nodes—as we verify via some simulations.

**Illustrations on Random and Real-World Networks.** We now examine how diffusion on an Erdős–Renyi network with 40 nodes depends on the mixture of nodes' activity patterns. We consider a network with a probability of 1/4 per link, the expected degree is almost 10, and the network is usually connected and has many cycles. Again, we compare what happens with various configurations of Poisson, sticky, and reversing nodes. Given that the network is random, the various nodes end up randomly located in the network. For each of 30 values of $\lambda$ we run 50,000 iterations and average the outcomes.

Fig. 7*A* shows that a mixture of reversing nodes with sticky nodes does better than any of the other configurations for $\lambda$ up to about 0.3 and after that all reversing nodes take over. This is similar to what we saw for line, and in *Proposition 4*, and here it is more pronounced for getting a fraction of infected nodes of 0.25. Moreover, here the nodes are not explicitly placed in some alternating fashion, but just randomly mixed in the population, and still having sticky mixed with reversing nodes does better for a substantial range of parameters. Interestingly, when we examine the probability of getting half of the nodes infected, then for a middle range of $\lambda$ (near 1/3) it turns out that mixing reversing with Poisson does better than mixing reversing with sticky nodes, as we see in Fig. 7*B*. So, when and what heterogeneity is optimal can depend on the objective and infection rate and network structure.

We also examine how heterogeneity works on a real-world social network. We simulate diffusion with various combinations of Poisson, sticky, and reversing nodes on an Indian village network with 155 nodes, where each node is a household from the
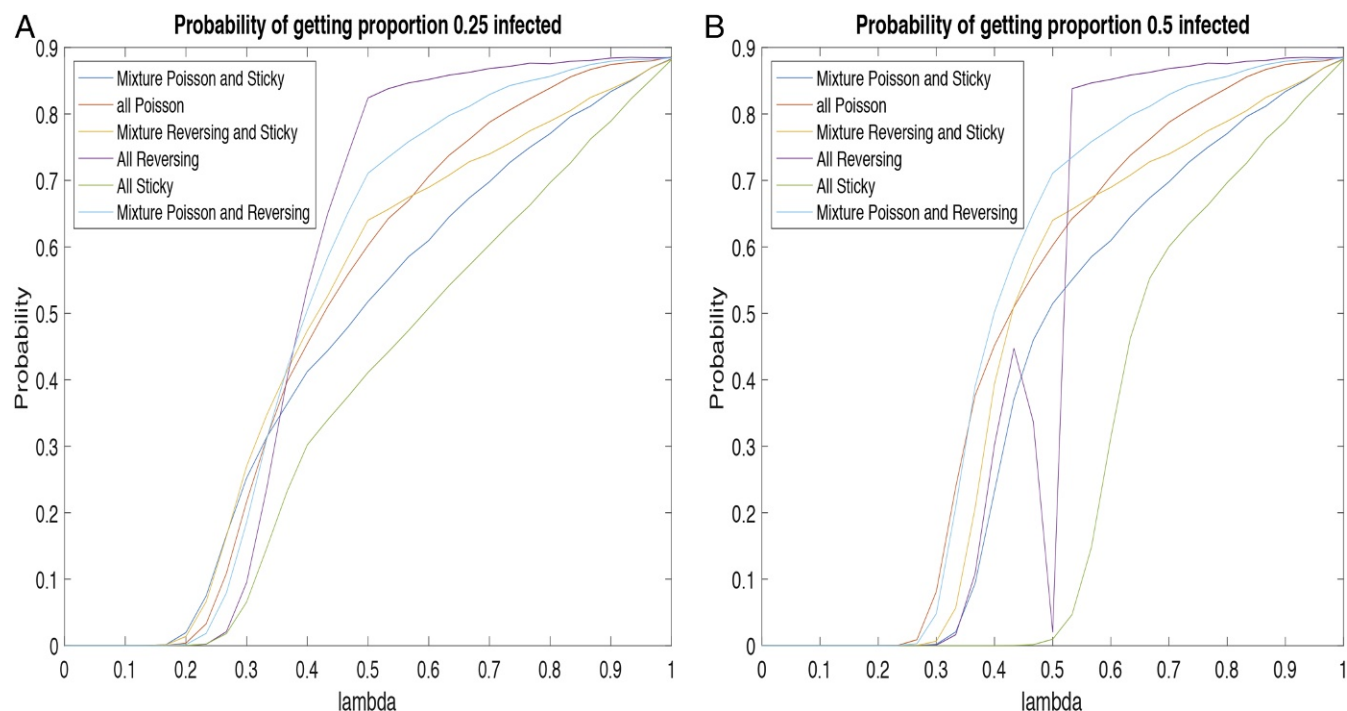
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**Fig. 7.** Comparisons of infection probabilities under various configurations of nodes for Erdős–Renyi random networks on 40 nodes. (*A*) Probability of getting one-quarter of nodes infected. (*B*) Probability of getting one-half of nodes infected.

data of ref. 7 (village no. 77). Again, for each of 30 values of $\lambda$ we run 50,000 iterations of running an infection and average the outcomes. The results are very similar to the Erdős–Renyi random network, as we see by comparing Fig. 8 to Fig. 7.

**Remark.** When calculating the probability of getting proportion 0.5 infected, there is an "anomaly" in both Erdős–Renyi and Indian village network simulations at $\lambda = 0.5$. Figs. 7*B* and 8*B* both admit a sharp decline in the probability of getting at
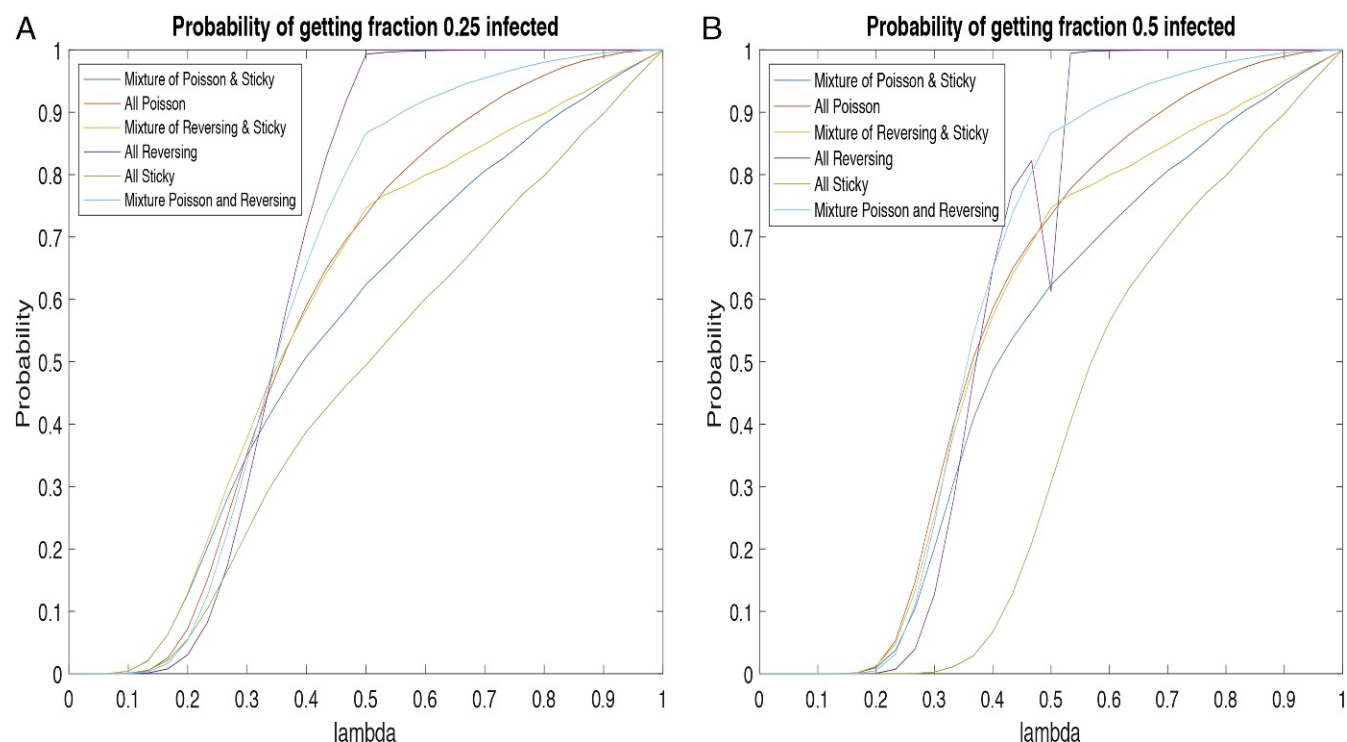


**Fig. 8.** Comparisons of infection probabilities under various configurations of nodes' activity on an Indian village social network. (*A*) Probability of getting one-quarter of nodes infected. (*B*) Probability of getting one-half of nodes infected.

least half of the nodes infected exactly at $\lambda = 0.5$ for the all-reversing case. The reason for this strange behavior is that (only) when $\lambda = 0.5$, reversing nodes no longer have any randomness and then exactly switch from being active to dormant in each period. So when we start with half of the nodes active, they miscoordinate with the other nodes exactly over time and never infect the remaining half. Thus, essentially half the nodes will never be infected, and the only way contagion reaches more than half of the population is if more than half happen to be all path connected to each other and start in the same state, which happens rarely (especially in the Indian village network in which the network fractures into components when one considers only half the nodes).

## A Related Problem

There is a very different problem from diffusion that ends up having a very similar logic applied and helps in further understanding our results, showing their reach. Imagine two people who are lost in a city with no way to communicate. They understand that it would be best for them to find each other by trying to meet at one of the major landmarks. To keep things simple, imagine that the city is New York and they each expect that the logical meeting places are either the Empire State Building or the Statue of Liberty, as in the seminal discussion of focal points in ref. 37. If they both go to each of the landmarks, then there is a chance that they will miscoordinate (38, 39)—going in the opposite order and thus missing each other. If, instead, one of them just goes to one of the landmarks and stays there, while the other alternates and goes to both, then they are sure to meet.

To complete the analogy, and see that heterogeneity does really improve coordination, consider three strategies that a person trying to coordinate with another person might use, which have parallels to our strategies above.

To be concrete, the game is that in each discrete time period each person picks one of $k$ available choices (e.g., which landmark to go to in the above example). The measurement of success is how long it takes until the two people meet.

Consider three strategies that the people can follow, which each have an analogous activity pattern in our analysis above and are so labeled to make the analogy clear.

Poisson (P): In each period an agent makes a choice uniformly at random, hopping from one choice to another with no memory and equal probability on each choice in any given period.

Sticky (S): An agent picks one choice uniformly at random in the first period and sticks with that choice forever.

Reversing (R): At the beginning of time, an agent uniformly and randomly picks an order over the $k$ choices and then chooses them one by one in that order, never visiting a previously visited choice during the first $k$ periods. After $k$ periods, the agent starts over and uniformly at random picks a new ordering over the $k$ choices and visits them in that order for the next $k$ periods and repeats this process each $k$ period indefinitely.

Now let us consider how long it takes two people to first meet when using these strategies. First, it is clear that regardless of which of these strategies they choose, the two agents have the same probability of meeting in the first period—which is simply $1/k$. This then makes it clear that having both agents play S is the worst possible match: If they do not meet in the first period, they will never meet, while under any other combination there is a possibility of meeting in some later period. More generally, it turns out that there is a unique combination that minimizes the expected time to meeting: having one agent play S and the other

play R. This is an analog to our result in *Proposition 4*, which we now state.

Using the labels above, let $SR$ denote the expected time to first meeting when one agent is S and the other is R. With similar notation for other combinations, we have the following:

**Claim 1.** $RS < PS = PP = PR < RR < SS$.

The proof of the claim is not hard.

First, as we already argued $SS$ does strictly worse than any other strategy in terms of expected meeting time.

Second, it is easy to see that any combination with at least one $P$ has a chance of meeting in any given period that is $1/k$ independent of history. Thus, $PP = PS = PR$.

Next comes the key argument that $RS$ does better than $PS$, which then implies that $RS < PS = PP = PR < SS$. $RS$ and $PS$ both have the same probability $1/k$ of meeting in the first period. In period 2, conditional on not having met in the first period, $RS$ has probability $1/(k-1)$ since now R no longer searches a site that S is not choosing, while $PS$ still has probability $1/k$. By period $t < k$, $RS$ has probability $1/(k-t)$ of meeting conditional on not having met yet and probability 1 by period $k$, while $PS$ still has probability $1/k$ in every period. So, $RS$ does strictly better than PS.

The only remaining pairing to consider is $RR$. $RR$ does strictly better than $SS$ as already argued. To see that $RR$ does worse than $PR$ concludes the proof. $RR$ and $PR$ have the same probability of meeting in the first period. In every subsequent period, PR has a probability of $1/k$ of meeting. Instead, $RR$'s probability gets worse in the second period. This is obvious if $k = 2$, since the two people just reverse locations and miss each other if they missed each other in the first period. More generally, the probability that they meet in the second period conditional on not meeting in the first period under $RR$ is $(k-2)/(k-1)^2 < 1/k$. To see this, without loss of generality, let person 1 have made choice 1 and person 2 have made choice 2 in the first period. Now, if the first person chooses choice 2, they will not meet. With the remaining probability of $(k-2)/(k-1)$, under RR the first person goes to a landmark that the second person will possibly visit, and then conditional on that they meet with probability $1/(k-1)$, and so the overall probability is $(k-2)/(k-1)^2$ as claimed. It is easy to extend this argument to see that this probability decays with each period, as the agents do not revisit places to which they have already been, while the other agent is more likely to visit those given that they did not meet before and so the other agent is less likely to have been to those places yet.

## Concluding Remarks

Heterogeneity in activity patterns within a population can substantially enhance diffusion, and matching extreme types of agents next to each other can be optimal. The conclusion that S agents hurt diffusion holds only when one requires all agents to have the same activity patterns, and the relationship between diffusion probability and fraction of agents with S behavior has an inverse-U shape. We show this analytically in simple networks. Given the intractability of finding fully optimal configurations in general networks, we illustrated that the results extend to some more complex networks by simulation.

The point here is not to fully characterize the optimal patterns for diffusion, as it is both intractable and not clear what one would do with such a characterization. The point instead is to show that considering heterogeneity in diffusion processes can have a big impact and to provide intuitions as to why. Natural next steps would be to investigate the further implications of heterogeneity of activity in efficient vaccination policies and enhancement of the diffusion of innovations by picking the best "seeds."

Throughout this paper, we considered a simple model of diffusion—the so-called SIR model. The results may

qualitatively and quantitatively change if one considers different, more complex diffusion models such as the "threshold" diffusion model (40), as some studies have shown that this can result in qualitatively different results in terms of how network structure impacts diffusion (4). How heterogeneous activity patterns impact diffusion in such models seems to be an interesting area for future studies.

Finally, here we examined the optimal structure of agents without constraints on the numbers of different types of agents. In some settings it may be possible to incentivize or choose agents to be S and others to be R. A logical next problem to study is to consider a fixed, relatively small number of S agents (or intermediaries who are "always active") and pick the best "bursts" (or best "intermediaries") for the enhancement of the diffusion. This "optimal bursts" (or "optimal intermediaries") question

also leads to several follow-ups: Is this a computationally hard problem? Would (as in ref. 35 for optimal seeds) a "greedy" algorithm perform well in approximating the optimal placement of S (or intermediary) agents? Similar to the exercise in ref. 7, Which centrality measures perform well in practice for finding the optimal placement of such agents? And, along the lines of ref. 41, How many additional placements would have to be picked at random to prompt a larger diffusion than the optimum?

1. Pastor-Satorras R, Vespignani A (2000) Epidemic spreading in scale-free networks. *Phys Rev Lett* 86:3200–3203.
2. Lopez-Pintado D (2008) Contagion in complex social networks. *Games Econ Behav* 62:573–590.
3. Aral S, Muchnik L, Sundararajan A (2009) Distinguishing influence based contagions from homophily driven diffusion in dynamic networks. *Proc Natl Acad Sci USA* 106:21544–21549.
4. Centola D (2010) The spread of behavior in an online social network experiment. *Science* 329:1194–1197.
5. Jackson MO, Yariv L (2011) Diffusion, strategic interaction, and social structure. *Handbook of Social Economics*, eds Benhabib J, Bisin A, Jackson MO (North Holland, San Diego).
6. Aral S, Walker D (2012) Creating social contagion through viral product design: A randomized trial of peer influence in networks. *Manage Sci* 57:1623–1639.
7. Banerjee A, Chandrasekhar A, Duflo E, Jackson MO (2013) Diffusion of microfinance. *Science* 341:1236498.
8. Akbarpour M, Li S, Gharan SO (2017) Thickness and information in dynamic matching markets, 10.2139/ssrn.2394319.
9. Aral S, Nicolaides C (2017) Exercise contagion in a global social network. *Nat Commun* 8:14753.
10. Gleeson JP, Durrett R (2017) Temporal profiles of avalanches on networks. *Nat Commun* 8:1227.
11. Johansen A (2004) Probing human response times. *Phys A Stat Mech Appl* 338:286–291.
12. Wu Y, Zhou C, Xiao J, Kurths J, Schellnhuber HJ (2010) Evidence for a bimodal distribution in human communication. *Proc Natl Acad Sci USA* 107:18803–18808.
13. Barabasi A-L (2011) *Bursts: The Hidden Patterns Behind Everything We Do, from Your E-mail to Bloody Crusades* (Penguin Group, New York).
14. Holme P, Saramäki J (2012) Temporal networks. *Phys Rep* 519:97–125.
15. Pfitzner R, Scholtes I, Garas A, Tessone CJ, Schweitzer F (2013) Betweenness preference: Quantifying correlations in the topological dynamics of temporal networks. *Phys Rev Lett* 110:198701.
16. Porter MA, Gleeson JP (2016) *Dynamical Systems on Networks* (Springer, Heidelberg).
17. Onaga T, Gleeson JP, Masuda N (2017) Concurrency-induced transitions in epidemic dynamics on temporal networks. arXiv:1702.05054.
18. Belykh IV, Belykh VN, Hasler M (2004) Blinking model and synchronization in small-world networks with a time-varying coupling. *Phys D Nonlinear Phenom* 195:188–206.
19. Vázquez A, et al. (2006) Modeling bursts and heavy tails in human dynamics. *Phys Rev E* 73:036127.
20. Vazquez A, Racz B, Lukacs A, Barabasi A-L (2007) Impact of non-Poissonian activity patterns on spreading processes. *Phys Rev Lett* 98:158702.
21. Iribarren JL, Moro E (2009) Impact of human activity patterns on the dynamics of information diffusion. *Phys Rev Lett* 103:038702.
22. Kumar Pan R, Saramäki J (2011) Path lengths, correlations, and centrality in temporal networks. *Phys Rev E* 84:016105.
23. Karsai M, et al. (2011) Small but slow world: How network topology and burstiness slow down spreading. *Phys Rev E* 83:025102.
24. Hoffmann T, Porter MA, Lambiotte R (2012) Generalized master equations for non-Poisson dynamics on networks. *Phys Rev E* 86:046102.
25. Scholtes I, et al. (2014) Causality-driven slow-down and speed-up of diffusion in non-markovian temporal networks. *Nat Commun* 5:5024.
26. Bick C, Field M (2015) Asynchronous networks and event driven dynamics. arXiv:1509.04045.
27. Li A, Cornelius SP, Liu Y-Y, Wang L, Barabási A-L (2016) The fundamental advantages of temporal networks. arXiv:1607.06168.
28. Holme P, Saramäki J (2012) Temporal networks. *Phys Rep* 519:97–125.
29. Porter MA, Gleeson JP (2014) Dynamical systems on networks: A tutorial. arXiv:1403.7663.
30. Bailey NTJ (1975) *The Mathematical Theory of Infectious Diseases* (Griffin, London).
31. Jackson MO (2008) *Social and Economic Networks* (Princeton Univ Press, Princeton).
32. Centola D, Macy M (2007) Complex contagions and the weakness of long ties. *Am J Sociol* 113:702–734.
33. Bullmore Ed, Sporns O (2009) Complex brain networks: Graph theoretical analysis of structural and functional systems. *Nat Rev Neurosci* 10:186–198.
34. van den Heuvel MP, Sporns O (2013) Network hubs in the human brain. *Trends Cognit Sci* 17:683–696.
35. Kempe D, Kleinberg J, Tardos É (2003) Maximizing the spread of influence through a social network. *Proceedings of the Ninth ACM SIGKDD International Conference on Knowledge Discovery and Data Mining* (ACM, New York), pp 137–146.
36. Cohen R, Havlin S, Ben-Avraham D (2003) Efficient immunization strategies for computer networks and populations. *Phys Rev Lett* 91:247901.
37. Schelling TC (1960) *The Strategy of Conflict* (Harvard Univ Press, Cambridge, MA).
38. Chassang S (2010) Fear of miscoordination and the robustness of cooperation in dynamic global games with exit. *Econometrica* 78:973–1006.
39. Kempe D, Schulman LJ, Tamuz O (2016) Quasi-regular sequences and optimal schedules for security games. arXiv:1611.07169.
40. Granovetter M (1978) Threshold models of collective behavior. *Am J Sociol* 83:1420–1443.
41. Akbarpour M, Malladi S, Saberi A (2017) Just a few seeds more: Value of network information for diffusion, 10.2139/ssrn.3062830.