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DIFFUSION OF NEW PRODUCTS WITH RECOVERING CONSUMERS*

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Abstract. We consider the diffusion of new products in the discrete Bass-SIR model, in which consumers who adopt the product can later "recover" and stop influencing their peers to adopt the product. To gain insight into the effect of the social network structure on the diffusion, we focus on two extreme cases. In the "most-connected" configuration, where all consumers are interconnected (complete network), averaging over all consumers leads to an aggregate model, which combines the Bass model for diffusion of new products with the SIR model for epidemics. In the "least-connected" configuration, where consumers are arranged in a circle and each consumer can be influenced only by the neighbor to the left (one-sided 1D network), averaging over all consumers leads to a different aggregate model which is linear and can be solved explicitly. We conjecture that for any other network, the diffusion is bounded from below and from above by that on a one-sided 1D network and on a complete network, respectively. When consumers are arranged in a circle and each consumer can be influenced by the neighbors to the left and right (two-sided 1D network), the diffusion is strictly faster than on a one-sided 1D network. This is different from the case of nonrecovering adopters, where the diffusion on one-sided and two-sided 1D networks is identical. We also propose a nonlinear model for recoveries and show that consumers' heterogeneity has a negligible effect on the aggregate diffusion.

Key words. marketing, diffusion in social networks, Bass model, SIR model, agent-based models

AMS subject classifications. 91B99, 92D25, 90B60

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1. Introduction.

Diffusion of new products is a fundamental problem in marketing research. The diffusion begins when the product is first introduced into the market and progresses as consumers adopt the product. Here, to *adopt* the product means to buy it (e.g., iPad), download it (e.g., Skype), try it (e.g., Google search), use it (e.g., Facebook), etc.

The first quantitative model of diffusion of new products was proposed in 1969 by Bass [4]. In this model, the diffusion depends on two parameters, p and q, which correspond to the likelihood to adopt the product due to *external influences* of mass media and due to *internal influences* of individuals who have already adopted the product (peer effects/word of mouth), respectively. The Bass model inspired a huge body of theoretical and empirical research [16] (in 2004 the Bass model was named one of the 10 most-cited papers in the 50-year history of *Management Science* [19]). Most of its extensions, however, were aggregate (macroscopic) models. More recently, diffusion of new products has been studied using discrete, agent-based models (ABM) [9, 10, 11, 12, 13, 14]. This kinetic-theory approach has the advantage that it reveals the relation between the (microscopic) behavior of individual consumers and the aggregate market diffusion, and it allows individual-level heterogeneity within both adoption decisions and social networks [17].

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In [7] we introduced the discrete Bass-SIR model for the diffusion of new products. Unlike previous models, it allows for the possibility that adopters will stop influencing their peers after some time. This can occur because they bought the product but stopped using it, because they stopped discussing it with their peers, because their peers became indifferent to their influence, etc. The motivation for this model came from a recent study in which Graziano and Gillingham empirically examined the adoption of solar photovoltaic systems in Connecticut [15]. They observed a strong relationship between adoption and the number of nearby previously installed systems. In particular, they noted that this effect of nearby systems *diminished with time*. This temporal decay of internal influence can be attributed to any of the above reasons. In addition, most firms who install solar panels put up a sign in front of the house announcing the installation, but over time some of these signs disappear. Additional empirical evidence for the temporal decay of internal influence follows from Banerjee et al. [1, 2], who studied a diffusion model in which information is only passed for a finite number of iterations. They found that when using the model, the finite duration of passing information makes a big difference, and that by adding the limits on passing information, the model much more closely matches the data.

The possibility that adopters become noncontagious was previously considered in studies that used SIR-type models. As pointed out in [7], however, *the SIR model is inappropriate for diffusion of new products, and its diffusion dynamics is very different from that of the Bass-SIR model.* In particular, in the SIR model, there is a threshold quantity which determines whether an epidemic occurs or the disease simply dies out. In contrast, in the Bass-SIR model everyone ultimately adopts the product, since even in the absence of internal influences, all consumers eventually adopt due to external effects. This does not mean that the entire population will end up adopting the product, but rather that the Bass-SIR model only takes into account those members of the population who ultimately adopt the product (the "market potential"). Thus, the Bass-SIR model is concerned with the rate at which the aggregate diffusion takes place. For example, a typical application of the Bass-SIR model is to compute the market half-life time $T_{1/2}$ at which the product would be adopted by 50% of its market potential, and to determine how $T_{1/2}$ depends on the network structure and on the recovery rate r.

The focus of this paper is on analyzing the effect of the social network structure on the aggregate diffusion dynamics in the discrete Bass-SIR model, which is a continuous-time Markov chain (CTMC). The paper is organized as follows. In section 2 we review the discrete Bass-SIR model for diffusion of new products with recovering adopters. In section 3 we obtain explicit solutions for the case of purely external adoptions ($q = 0$). Then, for $q > 0$ we show that the effect of recovery on the diffusion depends on the dimensionless variable r/q, where r is the probability of recovery. Thus, when $r \ll q$, recovery only leads to a slightly slower diffusion, whereas when $r \gg q$, diffusion is much slower and is similar to that in the case of purely external adoptions.

In section 4 we consider periodic 1D networks, where consumers are located in a circle. We show that the one-sided 1D model, where each consumer can be influenced only by the neighbor to the left, reduces in the limit of an infinite number of consumers to the *one-sided 1D Bass-SIR model*. This novel reduced model for the aggregate diffusion dynamics consists of four linear ODEs, which can be solved explicitly for the aggregate adoption curve. When each consumer can be influenced by the neighbors to the left and right, taking the limit of an infinite number of consumers leads to a different aggregate model, the *two-sided 1D Bass-SIR model*. We show analytically

and numerically that diffusion in the two-sided case is slightly faster than in the one-sided case. This result is surprising, since in the absence of recovery, the diffusion is identical in both cases.

In the case of a nonspatial (complete) network, where all consumers are interconnected, averaging over all consumers leads to the *nonspatial Bass-SIR model*, which combines the Bass model for diffusion of new products with the SIR model for diffusion of epidemics. Since the one-sided 1D network and the nonspatial network are the least- and most-connected network, respectively, we conjecture that for any other connected network, diffusion is faster than in the one-sided 1D model and slower than in the nonspatial model (section 5). This conjecture is supported numerically for D-dimensional Cartesian networks, scale-free networks, and small-world networks. As the probability for recovery r increases, internal (word-of-mouth) effects become weaker. As a result, the half-life time $T_{1/2}$ increases, and the dependence of the diffusion dynamics on the network structure decreases. Nevertheless, the dependence of $T_{1/2}$ on the network structure *increases* with r for mild values of r.

The assumption that consumers are homogeneous is convenient for the analysis. A more realistic assumption, however, is that each consumer has different parameters p_i, q_i, and r_i. We show that in the case of vertex-transitive networks, the difference between diffusion in the heterogeneous and homogeneous cases is quadratically small in the level of heterogeneity (section 6). Indeed, our simulations reveal that even with 50% heterogeneity, diffusion in the heterogeneous case is only slightly lower than the homogeneous one, both for vertex-transitive networks (periodic Cartesian networks) and for networks which are not vertex-transitive (small-world, scale-free).

In section 7 we relax the assumption that adopters recover independently of other adopters. In analogy with the Bass model, we introduce a nonlinear recovery model, in which consumers can recover both externally (i.e., independently of other consumers) and internally (i.e., because of interactions with recovered/dissatisfied consumers). This situation arises in online social networks, where some people leave because they are unhappy with it, while others leave because their friends are no longer there.

Obviously, social networks are neither complete nor 1D. Nevertheless, the above results should be relevant to diffusion on real social networks. Indeed, in [7] we showed that a small-world structure has a negligible effect on the diffusion, and that diffusion on scale-free networks is equivalent to that on Cartesian grids. In addition, in the case of solar photovoltaic systems, the adoption is predominantly influenced by nearby previously installed systems [15], and so the social network in essentially a 2D Cartesian grid. The 1D Cartesian grid analyzed in this study, therefore, is a reasonable toy model which has the advantage of being amenable to analysis. Finally, our results for the 1D and a complete network are conjectured to provide lower and upper bounds for the diffusion in any social network.

2. Discrete Bass-SIR model. Our starting point is the discrete Bass-SIR model for diffusion of new products with recovering consumers, which was recently introduced in [7]. A new product is introduced to a market with M consumers at time $t = 0$. Initially all consumers are nonadopters. If a consumer adopts the product, he or she becomes a contagious adopter. A contagious adopter can later "recover" and become a noncontagious adopter. The consumers belong to a social network which is described by an undirected or directed graph. Let k_j denote the number of consumers connected to consumer j (the "degree" or "indegree" of node j, respectively), and assume that there are no "isolated" consumers (i.e., $k_j \geq 1$ for all j). If j did not adopt the product by time t, the probability that j will adopt (and thus become

contagious) in $(t, t+\Delta t)$ is

$$\text{Prob}\begin{pmatrix} j \text{ adopts in} \\ (t, t+\Delta t) \end{pmatrix} = \left(p + q\frac{i_j(t)}{k_j} \right) \Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0, \tag{2.1a}$$

where $i_j(t)$ is the number of contagious adopters connected to j at time t. The parameters p and q describe the likelihood that an individual will adopt the product due to *external influences,* such as mass media, and due to *internal influences* by contagious consumers who have already adopted the product (*word of mouth*, *peer effects*), respectively.

The magnitude of internal influences experienced by j increases linearly with the number i_j of contagious adopters connected to j, and is normalized by k_j (see (2.1a)), so that regardless of the network structure, the maximal internal influence that j can experience (when all his or her social connections are contagious adopters) is q. The normalization by k_j allows for a meaningful comparison of different networks. Indeed, in the absence of normalization (i.e., if we set $k_j = 1$ in (2.1a)), it is trivial that adding more connections to a network leads to a faster diffusion. With the normalization by k_j, however, it is not clear, e.g., whether diffusion in the one-sided 1D case is slower than in the two-sided 1D case (see section 4.3).

As in the SIR model, we assume that if j was a contagious adopter at time t, the probability of j recovering and becoming noncontagious in $(t, t+\Delta t)$ is

$$\text{Prob}\begin{pmatrix} j \text{ recovers in} \\ (t, t+\Delta t) \end{pmatrix} = r\Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0. \tag{2.1b}$$

In section 7 we consider a more general model for recovery.

We denote the fraction of nonadopters ("susceptible"), contagious adopters ("infected"), and noncontagious adopters ("recovered") at time t by $S(t)$, $I(t)$, and $R(t)$, respectively. The fraction of adopters (contagious and recovered) is

$$f(t) = I(t) + R(t) = 1 - S(t).$$

Since the product is new, initially all consumers are nonadopters, and so

$$S(0) = 1, \qquad I(0) = R(0) = f(0) = 0. \tag{2.2}$$

3. Preliminary analysis. In general, the effect of internal influences on the adoption curve $f(t)$ depends on the structure of the social network. In this section we derive some results that hold for any network.

3.1. Purely external adoptions. In the absence of internal effects ($q = 0$), relation (2.1a) reduces to

$$\text{Prob}\begin{pmatrix} j \text{ adopts in} \\ (t, t+\Delta t) \end{pmatrix} = p\Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0. \tag{3.1}$$

Therefore, the equations for S, I, and R read

$$S'(t) = -pS, \qquad I'(t) = pS - rI, \qquad R'(t) = rI, \tag{3.2a}$$

where $' = \frac{d}{dt}$, subject to

$$S(0) = 1, \qquad I(0) = 0, \qquad R(0) = 0. \tag{3.2b}$$

The solution of (3.2) is

(3.3a)
$$S = S^{\text{ext}}(t) := e^{-pt}, \quad I = I^{\text{ext}} := p\frac{e^{-rt} - e^{-pt}}{p-r}, \quad R = R^{\text{ext}} := 1 - \frac{pe^{-rt} - re^{-pt}}{p-r}.$$

In particular,

(3.3b)
$$f = f^{\text{ext}}(t;p) := 1 - e^{-pt}.$$

Thus, in the absence of internal effects, recovery does not affect the adoption curve. Recovery does affect, however, the ratio of contagious to recovered adopters. For example,

$$I^{\text{ext}} \sim e^{-rt} - e^{-pt}, \quad R^{\text{ext}} \sim 1 - e^{-rt}, \qquad r \ll p,$$

and

$$I^{\text{ext}} \sim \frac{p}{r}\left(e^{-pt} - e^{-rt}\right), \quad R^{\text{ext}} \sim 1 - e^{-pt}, \qquad r \gg p.$$

Once internal effects are added, recovery affects the adoption curve, since the rate of new internal adoptions depends on I. Indeed, by (2.1a), internal effects accelerate the adoption process, i.e.,

(3.4)
$$f(t;p,q,r) > f^{\text{ext}}(t;p), \qquad t > 0, \quad q > 0.$$

In particular, in the Bass-SIR model (2.1), everyone eventually adopts the product.

3.2. Dimensionless parameter r/q. Since the case of most interest is when the new product spreads predominantly through word of mouth (i.e., $p \ll q$), we rescale time as $t^* := qt$. Hence,

$$f(t;p,q,r) = f(t^*;p^*,r^*), \qquad p^* := \frac{p}{q}, \quad r^* := \frac{r}{q}.$$

This shows that the aggregate effect of recovery depends on the dimensionless parameter $r^* = \frac{r}{q}$. Since $r^* = \frac{Ir\Delta t}{Iq\Delta t}$, this parameter corresponds to the rate of loss (recovery) of contagious adopters over the rate of the creation of new ones (when most consumers are still nonadopters). There are two limiting cases as follows:

- When $r \ll q$, adopters have sufficient time to influence their neighbors before they become noncontagious. Hence, the effect of recovery is small, and diffusion is similar to that in the absence of recovery, i.e., $f(t;p,q,r) \approx f(t;p,q,r=0)$.
- When $r \gg q$, adopters have little time to influence their neighbors before they become noncontagious. Hence, internal effects effectively disappear, and diffusion is driven by purely external adoptions. Therefore, $f(t;p,q,r) \approx f^{\text{ext}}(t;p)$; see (3.3). In particular, diffusion is considerably slower than in the absence of recovery.

Intuitively, as r increases, internal influences last for shorter times, and therefore

1. diffusion becomes slower, i.e.,

   (3.5a) $f(t;p,p,r)$ is monotonically decreasing in r;

2. the dependence of the diffusion on the network structure decreases; i.e., if f_{I} and f_{II} denote the expected fractional adoption in networks I and II, then

   (3.5b) $|f_{\text{I}}(t;p,p,r) - f_{\text{II}}(t;p,p,r)|$ is monotonically decreasing in r.

In particular, as r increases from 0 to ∞, f decreases monotonically from $f(t;p,q,r=0)$ to $f(t;p,q,r=\infty) = f(t;p,q=0,r) = f^{\text{ext}}(t;p)$.

4. 1D networks. We now consider the "least-connected" network, namely, when consumers are located in a circle such that each consumer is only connected to one or two consumers.

4.1. One-sided 1D networks. In the one-sided 1D network, M consumers are located in a circle, and each consumer is only influenced by the neighbor to the left. Since $k_j = 1$, relation (2.1a) reads

$$\text{Prob}\begin{pmatrix} j \text{ adopts in} \\ (t, t+\Delta t) \end{pmatrix} = (p + q\, i_j(t))\, \Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0, \tag{4.1}$$

where $i_j(t) = 1$ if $j-1$ is a contagious adopter at time t, and $i_j(t) = 0$ otherwise.

A priori, finding the aggregate diffusion dynamics requires writing an ODE for the dynamics of each of the 3^M possible configurations.[1] As $M \to \infty$, however, this infinite system can be reduced to a system of four linear ODEs.

LEMMA 4.1. *Consider the discrete Bass-SIR model* (2.1) *on a one-sided* 1*D network. As* $M \to \infty$*, the diffusion dynamics is governed by the* one-sided 1D Bass-SIR model

$$S'(t) = -pS - q\underline{IS}, \qquad \underline{IS}'(t) = pe^{-pt}S + (qe^{-pt} - p - q - r)\underline{IS}, \tag{4.2a}$$

and

$$I'(t) = pS + q\underline{IS} - rI, \qquad R'(t) = rI, \tag{4.2b}$$

subject to

$$S(0) = 1, \qquad \underline{IS}(0) = I(0) = R(0) = 0. \tag{4.2c}$$

Here, $\underline{IS}$ denotes the fraction of pairs where the left consumer is infected and the right consumer is susceptible.[2] Thus, $\underline{IS} \neq I \cdot S$. The dynamics is determined by (4.2a) for S and $\underline{IS}$. Once these two equations are solved, R and I can be recovered from (4.2b).

Proof. We modify the analysis in [9, section 2] as follows. Let (S_k) denote a sequence of k adjacent nonadopters, let (IS_k) denote a sequence of a single contagious adopter and k nonadopters, and let (RS_k) denote a sequence of a single recovered adopter and k nonadopters, i.e.,

$$(S_k) = (\underbrace{S \dots S}_{k \text{ times}}), \qquad (IS_k) = (I\underbrace{S \dots S}_{k \text{ times}}), \qquad (RS_k) = (R\underbrace{S \dots S}_{k \text{ times}}),$$

and let $\underline{S_k}$, $\underline{IS_k}$, and $\underline{RS_k}$ denote the probabilities of these configurations at time t.

A configuration (S_k) cannot be created, as the only possible transformation is $(S) \to (I)$. A configuration (S_k) is destroyed if

1. any of the rightmost $k-1$ S's turns into an I, which happens at a rate of p;
2. a configuration (SS_k) transforms into the configuration (SIS_{k-1}), which happens at a rate of p;
3. a configuration (IS_k) transforms into the configuration (IIS_{k-1}), which happens at a rate of $p+q$; and

[1] Each of the M consumers can be susceptible, infected, or recovered.

[2] Or, equivalently, for any j, the probability that j is infected and $j+1$ is susceptible.

4. a configuration (RS_k) transforms into the configuration (RIS_{k-1}), which happens at a rate of p.

Therefore, the equation for $\underline{S_k}$ is

$$\underline{S_k}'(t) = -(k-1)p\underline{S_k} - p\underline{S_{k+1}} - (p+q)\underline{IS_k} - p\underline{RS_k}, \qquad k = 1, 2, \dots .$$

Since

$$\underline{SS_k} + \underline{IS_k} + \underline{RS_k} = \underline{S_k}, \tag{4.3}$$

the last equation reads

$$\underline{S_k}'(t) = -kp\underline{S_k} - q\underline{IS_k}, \qquad k = 1, 2, \dots . \tag{4.4a}$$

The motivation for (4.4a) is as follows. Any S can change to I at the rate p. Therefore, the overall rate of change due to external effects is $kp\underline{S_k}$. In addition, the leftmost S can change to I due to internal effects if his or her neighbor to the left is an I. Therefore, the overall rate of change due to external effects is $q\underline{IS_k}$.

A configuration (IS_k) is created from (SSS_k) at a rate p, from (ISS_k) at a rate $p+q$, and from (RSS_k) at a rate p. A configuration (IS_k) is destroyed

1. when any of the rightmost $k-1$ S's turns into an I, which happens at a rate of p;
2. when the left S changes to I at a rate of $p+q$; and
3. when the I changes to an R at a rate of r.

Therefore, the equation for IS_k is

$$\underline{IS_k}'(t) = p\underline{S_{k+2}} + (p+q)\underline{IS_{k+1}} + p\underline{RS_{k+1}} - \big((k-1)p + (p+q) + r\big)\underline{IS_k}, \qquad k = 1, 2, \dots .$$

Therefore, by (4.3),

$$\underline{IS_k}'(t) = p\underline{S_{k+1}} + q\underline{IS_{k+1}} - (kp + q + r)\underline{IS_k}, \qquad k = 1, 2, \dots . \tag{4.4b}$$

The motivation for (4.4b) is as follows. (IS_k) are created from (SS_k) at a rate of $p\underline{SS_k}$ due to external effects, and $q\underline{ISS_k}$ are created due to internal effects. Any S can change to I at the rate of p. Therefore, the overall rate of change due to external effects is $kp\underline{IS_k}$. The leftmost S can change to I due to internal effects at the rate of $q\underline{IS_k}$. The I can change to R at the rate of $r\underline{IS_k}$.

Since there are no adopters at $t = 0$, the initial conditions are

$$\underline{S_k}(t=0) = 1, \quad \underline{IS_k}(t=0) = 0, \qquad k = 1, 2, \dots . \tag{4.4c}$$

Therefore, the dynamics is governed by (4.4). This infinite system can be reduced to two coupled ODEs via the substitution[3]

$$\underline{S_k} = e^{-kpt}x(t), \qquad \underline{IS_k} = e^{-kpt}y(t), \qquad k = 1, 2, \dots . \tag{4.5}$$

Indeed, the equations for x and y read

$$x' = -qy, \qquad y' = pe^{-pt}x + (qe^{-pt} - q - r)y, \tag{4.6a}$$

[3]This reduction is not possible for general initial conditions. Fortunately, it is possible for the initial conditions (4.4c), which follow from (2.2).

subject to

$$x(0) = 1, \qquad y(0) = 0. \tag{4.6b}$$

The equation for S' follows from (4.4a) with $k = 1$. By (4.4b) with $k = 1$ and (4.5),

$$\underline{IS}'(t) = p\underline{SS} + q\underline{ISS} - (p+q+r)\underline{IS} = pe^{-pt}S + qe^{-pt}\underline{IS} - (p+q+r)\underline{IS}.$$

The equation for I' is *not* given by (4.4b) with $k = 0$.[4] Rather, a derivation similar to that of (4.4b) shows that $I' = pS + q\underline{IS} - rI$. Finally, since $S + I + R = 1$, then $R' = -S' - I'$. ∎

The one-sided 1D Bass-SIR model (4.2) "identifies" with the nonspatial Bass-SIR model (5.2) if one makes the approximation $\underline{IS} \approx I \cdot S$. This mean-field approximation, however, is very inaccurate, especially when $q \gg p$ [9]. Indeed, the diffusion dynamics in these models can be quite different (see, e.g., Figures 2 and 3B).

Unlike the nonspatial Bass-SIR model (5.2), the one-sided 1D Bass-SIR model (4.2) is *linear*. In fact, we can solve it explicitly as follows.

LEMMA 4.2. *Consider the discrete Bass-SIR model* (2.1) *on a one-sided* 1*D network. Then* $\lim_{M\to\infty} f(t) = f_{1D}^{\text{one-sided}}(t)$*, where*

$$f_{1D}^{\text{one-sided}}(t) = 1 - e^{-(r+q+p)t + \frac{q}{p}(1-e^{-pt})}\left(1 + r\int_0^t e^{(r+q)\tau - \frac{q}{p}(1-e^{-p\tau})}\,d\tau\right) \tag{4.7a}$$

$$= 1 - e^{-pt} + e^{-pt-g(t)}q\int_0^t e^{-r(t-\tau)}e^{g(\tau)}(1-e^{-p\tau})\,d\tau, \tag{4.7b}$$

and $g(t) = qt - \frac{q}{p}(1-e^{-pt})$.

Proof. By (4.6),

$$\ddot{x} = -qy' = -q\left(-(r+q)y + pe^{-pt}x + qe^{-pt}y\right) = -(r+q)x' - qpe^{-pt}x + qe^{-pt}x'$$
$$= -(r+q)x' + q(e^{-pt}x)',$$

and $x'(0) = -qy(0) = 0$. Integrating, one obtains $x' = -(r+q)x + qe^{-pt}x + r$. We can rewrite this as $x' - h(t)x = r$, where $h(t) = -(r+q) + qe^{-pt}$. The solution of this first-order linear ODE is $x(t) = e^{\int_0^t h(s)\,ds}(1 + r\int_0^t e^{-\int_0^\tau h(s)\,ds}\,d\tau)$, where $e^{\int_0^t h(s)\,ds} = e^{\int_0^t [-(r+q)+qe^{-ps}]\,ds} = e^{-(r+q)t + \frac{q}{p}(1-e^{-pt})}$. So by (4.5),

$$f_{1D}^{\text{one-sided}}(t) = 1 - S(t) = 1 - e^{-pt}x(t), \tag{4.8}$$

which proves (4.7a).

We can rewrite $f_{1D}^{\text{one-sided}}(t) = 1 - e^{-rt-pt-g(t)}(1 + r\int_0^t e^{r\tau+g(\tau)}\,d\tau)$. Now,

$$1 + r\int_0^t e^{r\tau+g(\tau)}\,d\tau = 1 + \int_0^t (e^{r\tau})'e^{g(\tau)}\,d\tau$$
$$= 1 + [e^{r\tau}e^{g(\tau)}]_0^t - \int_0^t e^{r\tau}e^{g(\tau)}g'(\tau)\,d\tau = e^{rt}e^{g(t)} - \int_0^t e^{r\tau}e^{g(\tau)}g'(\tau)\,d\tau.$$

[4] This is because when $k = 0$, there is no "left S that changes to I at a rate of $p + q$."

Hence,

$$f_{1D}^{\text{one-sided}}(t) = 1 - e^{-rt-pt-g(t)}\left(e^{rt}e^{g(t)} - \int_0^t e^{r\tau}e^{g(\tau)}g'(\tau)\,d\tau\right)$$
$$= 1 - e^{-pt} + e^{-rt-pt-g(t)}\int_0^t e^{r\tau}e^{g(\tau)}g'(\tau)\,d\tau, \qquad g' = q(1-e^{-pt}),$$

which proves (4.7b). ∎

As expected,

1. when $r = 0$, $f_{1D}^{\text{one-sided}} = 1 - e^{-(q+p)t+\frac{q}{p}(1-e^{-pt})}$, which is the expression derived in [9];
2. when $q = 0$, $f_{1D}^{\text{one-sided}} = f^{\text{ext}}$, in agreement with (3.3b); and
3. $f_{1D}^{\text{one-sided}}(t)$ is monotonically decreasing in r,[5] in agreement with (3.5a).

4.2. Two-sided 1D network. In the two-sided 1D network, M consumers are located in a circle, and each consumer is influenced by the left and right neighbors. Since $k_j = 2$, relation (2.1a) reads

$$\text{Prob}\begin{pmatrix} j \text{ adopts in} \\ (t, t+\Delta t)\end{pmatrix} = \left(p + q\frac{i_j(t)}{2}\right)\Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0, \tag{4.9}$$

where $i_j(t) = 2$ if both $j-1$ and $j+1$ are contagious adopters at time t, $i_j(t) = 1$ if only one of them is contagious at time t, and $i_j(t) = 0$ otherwise. A priori, capturing the diffusion dynamics requires writing an ODE for the dynamics of each of the 3^M possible configurations. As $M \to \infty$, however, this infinite system can be reduced to a system of five linear ODEs as follows.

LEMMA 4.3. *Consider the discrete Bass-SIR model* (2.1) *on a two-sided* 1*D network. As* $M \to \infty$*, the diffusion dynamics is governed by the* two-sided 1D Bass-SIR model

$$\begin{aligned} S'(t) &= -pS - q\underline{IS}, \\ \underline{IS}'(t) &= pe^{-pt}S + \left(\frac{q}{2}e^{-pt} - p - \frac{q}{2} - r\right)\underline{IS} - \frac{q}{2}\underline{ISI}, \\ \underline{ISI}'(t) &= 2pe^{-pt}\underline{IS} + \left(qe^{-pt} - p - q - 2r\right)\underline{ISI}, \end{aligned} \tag{4.10a}$$

and

$$I'(t) = pS + q\underline{IS} - rI, \qquad R'(t) = rI, \tag{4.10b}$$

subject to

$$S(0) = 1, \qquad \underline{IS}(0) = \underline{ISI}(0) = I(0) = R(0) = 0. \tag{4.10c}$$

Here, $\underline{ISI}$ denotes the fraction of triplets where the right and left consumers are infected and the center consumer is susceptible. Thus, $\underline{ISI} \neq I \cdot S \cdot I$. The dynamics is determined by (4.10a) for S, $\underline{IS}$, and $\underline{ISI}$. Once these three equations are solved, R and I can be recovered from (4.10b).

[5] This follows from (4.7b), since $g(t)$ is independent of r.

Proof. The dynamics of $\underline{S_k}$ is governed by

$$\underline{S_k}'(t) = -kp\underline{S_k} - \frac{q}{2}\underline{IS_k} - \frac{q}{2}\underline{S_kI}, \qquad k = 1, 2, \dots . \tag{4.11a}$$

Indeed, any of the S can change to I at the rate of p. Therefore, the overall rate of change due to external effects is $kp\underline{S_k}$. In addition, the leftmost S can change to I due to internal effects at the rate of $\frac{q}{2}\underline{IS_k}$, and the rightmost S can change to I due to internal effects at the rate of $\frac{q}{2}\underline{S_kI}$. Since by symmetry $\underline{IS_k} = \underline{S_kI}$, (4.11a) is equivalent to (4.4a).

The equation for $\underline{IS_k}$ is

$$\underline{IS_k}'(t) = p\underline{S_{k+1}} + \frac{q}{2}\underline{ISS_k} - \left(kp + \frac{q}{2} + r\right)\underline{IS_k} - \frac{q}{2}\underline{IS_kI}, \qquad k = 1, 2, \dots . \tag{4.11b}$$

Indeed, (IS_k) are created from (SS_k) at a rate of $p\underline{SS_k}$ due to external effects, and at a rate of $\frac{q}{2}\underline{ISS_k}$ due to internal effects. Any of the S's can change to I's at the rate of p. Therefore, the overall rate of change due to external effects is $kp\underline{IS_k}$. The leftmost S can also change to I due to internal effects at the rate of $\frac{q}{2}\underline{IS_k}$. The I can change to R at the rate of $r\underline{IS_k}$. Finally, the rightmost S changes to I due to internal effects at the rate of $\frac{q}{2}\underline{IS_kI}$. Similar arguments show that

$$\underline{IS_kI}'(t) = p(\underline{IS_{k+1}}+\underline{S_{k+1}I})+\frac{q}{2}(\underline{IS_{k+1}I}+\underline{IS_{k+1}I})-(kp+q+2r)\,\underline{IS_kI}, \quad k = 1, 2, \dots . \tag{4.11c}$$

Under the substitution

$$\underline{S_k} = e^{-p(k-1)t}S, \quad \underline{IS_k} = e^{-p(k-1)t}\underline{IS}, \quad \underline{IS_kI} = e^{-p(k-1)t}\underline{ISI}, \qquad k = 1, 2, \dots,$$

and using the symmetry $\underline{IS_k} = \underline{S_kI}$, the infinite system (4.11) reduces to the equations for S', $\underline{IS}'$, and $\underline{ISI}'$ in (4.10). Similar arguments show that the equation for I' reads

$$I' = pS + \frac{q}{2}\underline{IS} + \frac{q}{2}\underline{SI} - rI.$$

Since $\underline{IS}= \underline{SI}$, we get the equation for I' in (4.10b). ∎

4.3. $f_{1D}^{\text{one-sided}}(t) < f_{1D}^{\text{two-sided}}(t)$. In [9], Fibich and Gibori showed that when $r = 0$, the diffusion curves in the one-sided and two-sided 1D models are identical, i.e.,

$$f_{1D}^{\text{one-sided}}(t; p, q) \equiv f_{1D}^{\text{two-sided}}(t; p, q).$$

Intuitively, this is because external adoptions are independent of the network structure, and internal adoptions occur through the expansion of 1D clusters (chains) of adopters. Since the internal effect of such a chain in $(t, t+\Delta t)$ is $q\Delta t$ in the one-sided model and $\frac{q}{2}\Delta t + \frac{q}{2}\Delta t$ in the two-sided model, the rates of internal adoptions are identical in both cases. Hence, the diffusion curves are also identical.

The above argument suggests that the diffusion curves in the one-sided and two-sided 1D models should remain identical when adopters are allowed to recover. Surprisingly, however, this is not the case.

LEMMA 4.4. *When $r > 0$, diffusion in the one-sided model is strictly slower than in the two-sided model, i.e.,*

$$f_{1D}^{\text{one-sided}}(t; p, q, r) < f_{1D}^{\text{two-sided}}(t; p, q, r), \qquad t > 0.$$

Proof. Under the substitutions (4.5) and $\underline{IS_kI} = e^{-kpt}z(t)$, the infinite system (4.11) reduces to

(4.12a)
$$x' = -qy, \quad y' = pe^{-pt}x + \left(\frac{q}{2}e^{-pt} - \frac{q}{2} - r\right)y - \frac{q}{2}z, \quad z' = 2pe^{-pt}y + qe^{-pt}z - (q+2r)z,$$

subject to

(4.12b)
$$x(0) = 1, \qquad y(0) = 0, \qquad z(0) = 0.$$

Since $\underline{IS} = \underline{ISS} + \underline{ISI} + \underline{ISR}$ and $\underline{ISR} > 0$ for $t > 0$, then $\underline{ISI} < \underline{IS} - \underline{ISS}$ or, equivalently, $e^{-pt}z < e^{-pt}y - e^{-2pt}y$. Therefore, the solution of (4.12) satisfies

(4.13)
$$x' = -qy, \quad y' > pe^{-pt}x + (qe^{-pt} - q - r)y, \qquad t > 0,$$

subject to (4.6b). We now follow the derivation of (4.8) from (4.6) but replace the equality sign with an inequality wherever needed. Thus, we get that the solution (4.12) satisfies $\ddot{x} = -qy' < -(r+q)x' + q(e^{-pt}x)'$. Integrating, one obtains $x' < -(r+q)x + qe^{-pt}x + r$. We can rewrite this as $x' - h(t)x < r$. Integrating again, one obtains

$$x(t) < e^{\int_0^t h(s)\,ds}\left(1 + r\int_0^t e^{-\int_0^\tau h(s)\,ds}\,d\tau\right).$$

Hence,

$$f_{1D}^{\text{two-sided}}(t) = 1 - S(t) = 1 - e^{-pt}x(t) > 1 - e^{-pt}e^{\int_0^t h(s)\,ds}\left(1 + r\int_0^t e^{-\int_0^\tau h(s)\,ds}\,d\tau\right)$$
$$= f_{1D}^{\text{one-sided}}(t). \qquad \square$$

Intuitively, once recovery occurs, the periodic 1D network is broken into several nonperiodic 1D networks that do not communicate with each other. As we will show elsewhere [6], on 1D networks which are not periodic, diffusion in the two-sided case is strictly faster than in the one-sided case, thus explaining Lemma 4.4. Finally, we note that the difference between the one-sided and two-sided models is quite small (Figures 1A and 3).

4.4. Simulations. Figure 1A confirms the agreement as $M \to \infty$ between the one-sided 1D ABM and the one-sided 1D Bass-SIR model (see Lemma 4.1) and between the two-sided 1D ABM and the two-sided 1D Bass-SIR model (see Lemma 4.3); the agreement is clearest in the inset. The diffusion in the one-sided case is (slightly) slower than in the two-sided case, in agreement with Lemma 4.4. Additional numerical support that $f_{1D}^{\text{one-sided}} < f_{1D}^{\text{two-sided}}$ is given in Figure 3.

Figure 1B shows the dependence of $f_{1D}^{\text{one-sided}}(t)$ on r. As predicted in sections 3.2 and 4.1,

- if $r \ll q$, diffusion is similar to that for $r = 0$;
- $f_{1D}^{\text{one-sided}}(t; r)$ is monotonically decreasing in r; and
- if $r \gg q$, diffusion is similar to that in the absence of internal effects.

5. Lower and upper bounds. In the case of a nonspatial (complete) network where all M consumers are connected to each other, $k_j = M - 1$ and $i_j(t) = M \cdot I(t)$, and so relation (2.1a) reads

(5.1)
$$\text{Prob}\begin{pmatrix} j \text{ adopts in} \\ (t, t+\Delta t) \end{pmatrix} = \left(p + q\frac{M}{M-1}I(t)\right)\Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0.$$

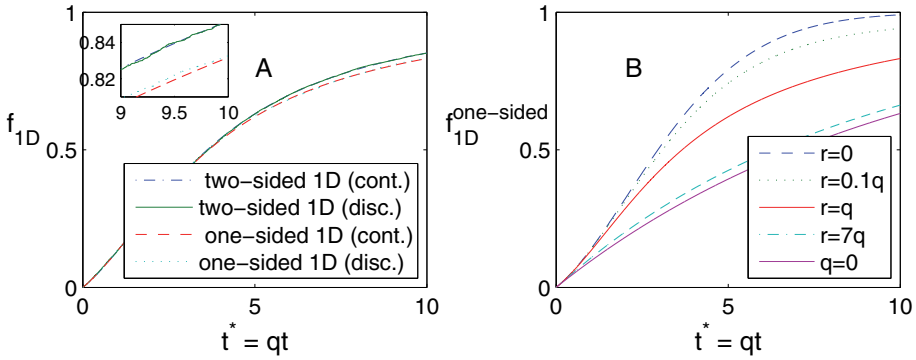

FIG. 1. *Fraction of adopters on* 1*D networks with* $p = 0.01$ *and* $q = 0.1$. A: *Agreement between [a single simulation of] the discrete Bass-SIR model in a one-sided* 1*D network (dots) and the continuous one-sided* 1*D Bass-SIR model* (4.2) *(dashes), and between [a single simulation of] the discrete Bass-SIR model on a two-sided* 1*D network (solid) and the continuous two-sided* 1*D Bass-SIR model* (4.10) *(dashed-dotted). Here* $r = 0.05$ *and* $M = 10{,}000$. B: *The continuous one-sided* 1*D Bass-SIR model* (4.2) *with various values of* r. *Here* $q = 0$ *is* f^{ext}; *see* (3.3b).

As $M \to \infty$, the aggregate diffusion is governed by the *nonspatial Bass-SIR model* [7]

$$S'(t) = -S(p + qI), \qquad I'(t) = S(p + qI) - rI, \qquad R'(t) = rI, \tag{5.2a}$$

$$S(0) = 1, \quad I(0) = 0, \quad R(0) = 0. \tag{5.2b}$$

If $r = 0$, then $R = 0$ and $f = 1 - S$, and so equations (5.2) reduce to the Bass model [4]

$$f'(t) = (1 - f)(p + qf), \qquad f(0) = 0. \tag{5.3}$$

The solution of (5.3) is given by the well-known Bass formula $f_{\text{Bass}}(t) = \frac{1-e^{-(p+q)t}}{1+(q/p)e^{-(p+q)t}}$. There is no explicit solution of (5.2) for $r > 0$.

The 1D and nonspatial cases are the least- and most-connected networks, respectively. Therefore, it was conjectured in [9] that in the absence of recoveries, for "any" network, the fraction of adopters is bounded by $f_{1\text{D}}(t; p, q) < f(t; p, q) < f_{\text{Bass}}(t; p, q)$. Since, however, in the case of recovering consumers $f_{1\text{D}}^{\text{one-sided}}(t) < f_{1\text{D}}^{\text{two-sided}}(t)$ (see Lemma 4.4), we modify this conjecture as follows.

CONJECTURE 5.1. *Consider the discrete Bass-SIR model* (2.1) *on any connected network. As* $M \to \infty$, *the fraction of adopters is bounded by*

$$f_{1\text{D}}^{\text{one-sided}}(t; p, q, r) < f(t; p, q, r) < f_{\text{nonspatial}}(t; p, q, r),$$

where $f_{\text{nonspatial}} = 1 - S$ *and* S *is the solution of* (5.2).

The result of Conjecture 5.1 is not immediate, since, as we add links, the weight of each link decreases (see discussion in section 2). The lower bound was proved in Lemma 4.4 for the case of the two-sided 1D network. In Figure 2 we compute the diffusion numerically for periodic D-dimensional Cartesian networks, where each node is connected to its $2D$ nearest nodes and $\text{Prob}\left(\begin{smallmatrix} j \text{ adopts in} \\ (t,t+\Delta t) \end{smallmatrix}\right) = (p + q\frac{i_j(t)}{2D})\Delta t$; see (2.1a). The diffusion in the 2D and 3D cases is indeed faster than in the one-sided

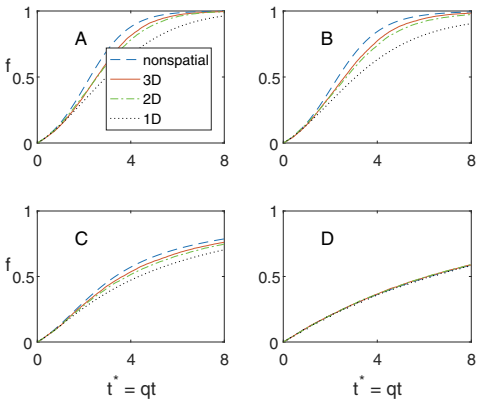

FIG. 2. *Fractional adoption in one-sided* 1*D (dots),* 2*D (dash-dot),* 3*D (solid), and nonspatial (dashes) networks. Here* $p = 0.01$*,* $q = 0.1$*, and* $M = 10{,}000$*.* A: $r = 0$. B: $r = 0.01$. C: $r = 0.1$. D: $r = 0.7$. *Figure reprinted with permission from [G. Fibich, "Bass-SIR model for diffusion of new products in social networks," Physical Review E, volume* 94*,* 032305*,* 2016*]. Copyright* 2016 *by the American Physical Society.*

1D model but slower than in the nonspatial model, in agreement with Conjecture 5.1. The differences among the four networks decrease with r, in agreement with (3.5b). In [7] it was observed numerically that diffusion in scale-free networks in similar, if not identical, to that on Cartesian grids, and that a small-world structure has a negligible effect on the diffusion. This suggests, therefore, that Conjecture 5.1 holds for scale-free and small-world networks.

A useful measure for comparing the diffusion in different networks is the *market half-life time* $T_{1/2} := f^{-1}(1/2)$, i.e., the time for half of the population to adopt. In the absence of internal effects, we have that $f = f^{\text{ext}}$ (see (3.3b)), and so $T_{1/2}^{\text{ext}} = \frac{\ln 2}{p}$. By Conjecture 5.1, for any network with p, q, and r,

$$T_{1/2}^{\text{one-sided 1D}} > T_{1/2} > T_{1/2}^{\text{nonspatial}}. \tag{5.4}$$

Figure 3A shows that (5.4) indeed holds for the two-sided 1D, 2D, and 3D Cartesian networks. In addition, for all networks

1. $T_{1/2}$ is monotonically increasing in r, in agreement with (3.5a); and
2. $T_{1/2} \to T_{1/2}^{\text{ext}}$ as $r/q \to \infty$, since internal effects disappear in the limit (see section 3.2).

In Figure 3B we plot the ratio of the upper and lower bounds in (5.4). Surprisingly, this ratio initially increases with r, and only later decreases monotonically to zero as $r/q \to \infty$. In particular, we have the following observation.

OBSERVATION 5.2. *When* r *is of a comparable magnitude to* q*, recovery increases the dependence of* $T_{1/2}$ *on the network structure.*

This observation also follows from Figure 3C, where we plot the ratio of the half-life times for the one-sided and two-sided 1D models. In that case, however, the maximal difference between the two models is 1.5%.[6]

[6]Observation 5.2 may seem to contradict Figure 2, which shows that the differences among the four models decrease with r. A closer inspection of Figure 2 shows that the vertical distances between the four curves (i.e., the differences in f for a given t) indeed decrease monotonically in r. The horizontal distances between the four curves (i.e., the differences in t for a given f), however, initially increase with r, because the curves become less steep.

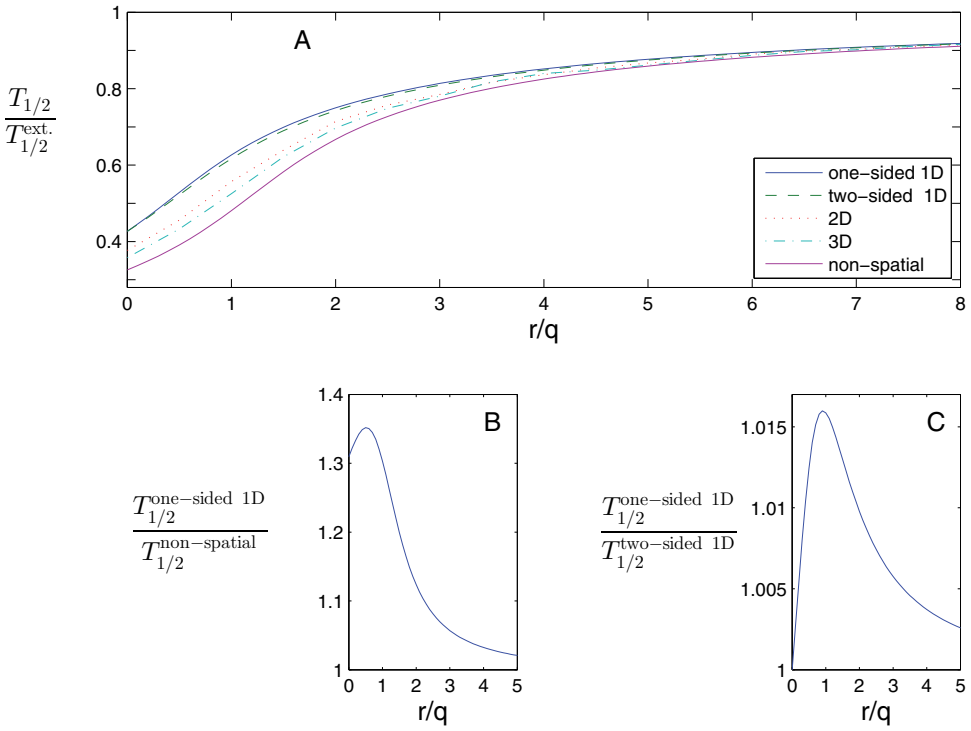

FIG. 3. A: *The market half-time* $T_{1/2}$*, normalized by* $T_{1/2}^{\text{ext}}$*, as a function of* r/q*, in the one-sided* $1D$ *model (solid), two-sided* $1D$ *model (dashes),* $2D$ *model (dots),* $3D$ *model (dashed-dotted), and the nonspatial model (solid). Here* $p = 0.01$ *and* $q = 0.1$. B: *The ratio* $\frac{T_{1/2}^{\text{one-sided 1D}}}{T_{1/2}^{\text{nonspatial}}}$. C: *The ratio* $\frac{T_{1/2}^{\text{one-sided 1D}}}{T_{1/2}^{\text{two-sided 1D}}}$.

6. Heterogeneous consumers. So far, we assumed that consumers are homogeneous; namely, they have the same p, q, and r. While this assumption is convenient for the analysis, a more realistic assumption is that consumer j has its own p_j, q_j, and r_j, i.e.,

$$\text{Prob}\begin{pmatrix} j \text{ adopts in} \\ (t, t+\Delta t) \end{pmatrix} = \left(p_j + q_j \frac{i_j(t)}{k_j}\right)\Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0 \tag{6.1a}$$

and

$$\text{Prob}\{j \text{ recovers in } (t, t+\Delta t)\} = r_j \Delta t + o(\Delta t) \quad \text{as } \Delta t \to 0. \tag{6.1b}$$

Simulations with heterogeneous nonrecovering consumers on nonspatial networks and on periodic 1D and 2D Cartesian networks showed that heterogeneity has a minor effect [13, 9]. By this we mean that the diffusion in the heterogeneous case was close to that in the homogeneous case, with $\bar{p} = \frac{1}{M}\sum_{j=1}^{n} p_j$ and $\bar{q} = \frac{1}{M}\sum_{j=1}^{n} q_j$, even when the level of heterogeneity was significant. This small effect of heterogeneity was explained in [8] as a consequence of the *averaging principle for heterogeneous models*. Exactly the same arguments imply that heterogeneity has a small effect when consumers are allowed to recover.

LEMMA 6.1. *Consider the heterogeneous Bass-SIR model* (6.1) *on a vertex-transitive network.*[7] *Then the adoption curve satisfies*

$$f(t;p_1,\dots,p_M,q_1,\dots,q_M,r_1,\dots,r_M) = f(t;\bar{p},\bar{q},\bar{r})\Big(1+O(\sigma_p^2,\sigma_q^2,\sigma_r^2)\Big),$$

where $\{\bar{p},\bar{q},\bar{r}\}$ *and* $\{\sigma_p,\sigma_q,\sigma_r\}$ *are the mean and standard deviation ("level of heterogeneity") of* $\{p_j\}_{j=1}^M$, $\{q_j\}_{j=1}^M$, *and* $\{r_j\}_{j=1}^M$, *respectively.*

Proof. Following [8], the adoption curve $f(t;p_1,\dots,p_M,q_1,\dots,q_M,r_1,\dots,r_M)$ satisfies the following two conditions:

1. f is twice continuously differentiable in $\{p_i,q_i,r_i\}_{i=1}^M$.
2. f is weakly symmetric in p; i.e., for any $\{p,\tilde{p},q,r\}$ and $i_0 \in \{1,\dots,M\}$, if $p_i = p$ for $i \neq i_0$, $p_{i_0} = \tilde{p}$, $q_i = q$ for all i, and $r_i = r$ for all i, then f is independent of i_0. Similarly, f is weakly symmetric in q and in r.

Indeed, condition 1 can be proved as in [8]. Condition 2 follows from the vertex-transitive property. Hence, the result follows from the averaging principle. ∎

In Figures 4 and 5 we present ABM simulations of the heterogeneous discrete Bass-SIR model on a periodic one-sided 1D network and on a periodic 2D network, respectively, with $p_i = p(1+\eta U(i))$, where U is uniformly distributed in $[-1,1]$, and similarly for q_i and r_i. At the heterogeneity level $\eta = 25\%$, the fractional adoption is nearly identical to the homogeneous one. Even at the heterogeneity level $\eta = 50\%$, the aggregate adoption level is only slightly lower than in the homogeneous case.[8]

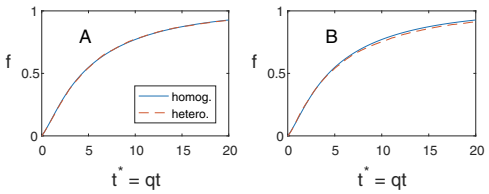

FIG. 4. *Fraction of adopters in [a single simulation of] the heterogeneous discrete Bass-SIR model* (6.1) *on a one-sided* 1*D network (dashes). Dotted line is the homogeneous case. Here* $p = 0.01$, $q = 0.1$, $r = 0.1$, *and* $M = 10{,}000$. *Level of heterogeneity is* $\eta = 25\%$ (A) *and* $\eta = 50\%$ (B).

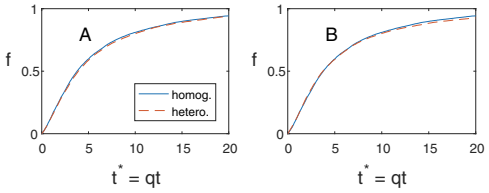

FIG. 5. *Same as Figure* 4 *on a* 2*D network.*

In Figures 6 and 7 we present ABM simulations of the heterogeneous discrete Bass-SIR model on two networks which are not vertex-transitive: a small-world network [18], constructed by adding 5% random long-range connections to a one-sided

[7] A graph G is called *vertex-transitive* if the "view" from any vertex is identical, i.e., if for given any two vertices v_1 and v_2 of G there is some automorphism $f : V(G) \to V(G)$ such that $f(v_1) = v_2$. For example, a nonspatial network and periodic d-dimensional Cartesian networks are vertex-transitive.

[8] The fact that heterogeneity *slows down* the diffusion can be easily proved when $q = 0$. Indeed, by (3.3b), $f^{\text{ext}}(t;p_1,\dots,p_M,r_1,\dots,r_M) = \frac{1}{M}\sum_{j=1}^M (1-e^{-p_j t}) < 1-e^{-\bar{p}t} = f^{\text{ext}}(t;\bar{p},\bar{r})$, where the inequality follows from the fact that for $g(x) = 1-e^{-x}$, $g'' < 0$, and so $\frac{1}{M}\sum_{j=1}^M g(p_j) < g(\sum_{j=1}^M p_j)$.

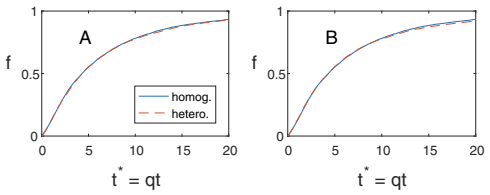

FIG. 6. *Same as Figure* 4 *on a small-world network.*

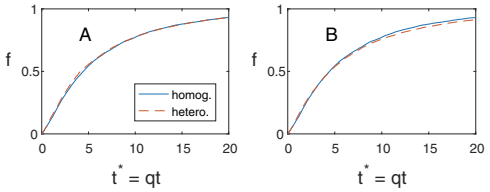

FIG. 7. *Same as Figure* 4 *on a scale-free network.*

1D network, and a scale-free network, constructed using the Barabási–Albert (BA) preferential-attachment algorithm [3] in which each new node makes a single new link, respectively. In both cases, we again observe that heterogeneity has a negligible case on the aggregate diffusion. The result for a small-world network could be expected, since a small-world structure has a negligible effect on the diffusion in the Bass and Bass-SIR models [9, 7]. The result for a scale-free network is less expected and may have to do with the surprising equivalence between diffusion in scale-free and Cartesian networks [7]. Alternatively, it may be an indication that heterogeneity has a negligible effect whenever the number of consumers is sufficiently large.

7. Internal (nonlinear) recoveries. In [5], Cannarella and Spechler analyzed diffusion of online social networks such as Myspace and Facebook. They argued that recoveries (i.e., people leaving the social network) result from interactions between infected (current) members and recovered (past) members. Therefore, they introduced a modified SIR model on a complete network in which the relation $R' = rI$ was replaced with[9]

$$R' = r_{\mathrm{nl}} IR. \tag{7.1}$$

Since $R(0) = 0$, however, under relation (7.1) there will be no recoveries. Hence, they artificially set $R(0) = R_0$, where $0 < R_0 \ll 1$ was a fitted parameter. To avoid this artificial fix and yet allow for nonlinear recoveries, we set

$$R'(t) = (r + r_{\mathrm{nl}} R)I.$$

Thus, in the spirit of the Bass model, adopters can recover independently of others ("external recoveries"), as well as through interactions with recovered people ("internal recoveries"). This leads to the *modified Bass-SIR model*

$$S'(t) = -S(p + qI), \quad I'(t) = S(p + qI) - (r + r_{\mathrm{nl}} R)I, \quad R'(t) = (r + r_{\mathrm{nl}} R)I, \tag{7.2a}$$

$$S(0) = 1, \quad I(0) = 0, \quad R(0) = 0. \tag{7.2b}$$

[9] See [7] for why SIR models are inappropriate for diffusion of new products.

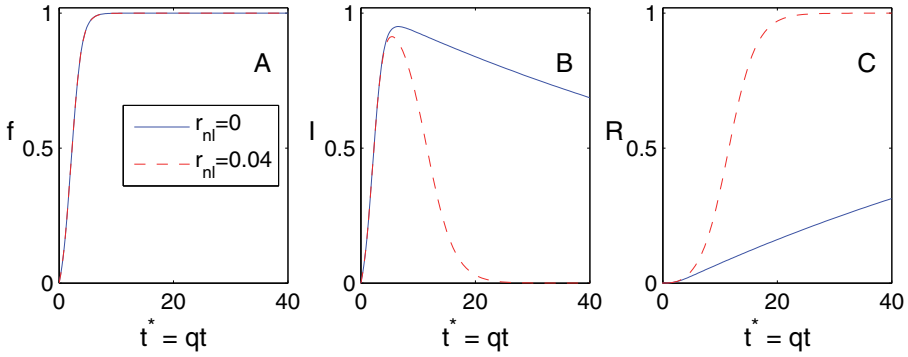

FIG. 8. *The modified Bass-SIR model* (7.2) *with* $p = 0.01$, $q = 0.1$, $r = 0.001$, *and* $r_{\rm nl} = 0$ *(solid), or* $r_{\rm nl} = 0.04$ *(dashes).* A: $f(qt)$. *The two curves are indistinguishable.* B: $I(qt)$. C: $R(qt)$.

Since $R(t) \le 1$, nonlinear internal recoveries can have a dominant effect over linear external ones (i.e., $r_{\rm nl}R \gg r$) only if $r_{\rm nl} \gg r$. To see the dynamics in this case, we set $r = 0.001$, so that external recoveries have a negligible effect, and $r_{\rm nl} = 0.04$, so that $r_{\rm nl} \gg r$. Since $r_{\rm nl} \ll 1$, nonlinear recoveries become important only once most of the population adopts. Hence, the overall adoption $f = I + R$ is unaffected by the nonlinear recoveries; see Figure 8A. Nonlinear recoveries, however, accelerate the transition from infected to recovered, changing it from a linear rate to an exponential one; see Figures 8B and 8C. Therefore, nonlinear recoveries are important if the firm only cares about the number of infected consumers (for example, if being recovered means to stop using the product). If, however, recovered adopters bought the product or still use it, but simply stopped promoting it, the effect of nonlinear recoveries is of much less importance to the firm.

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