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Diffusion and Contagion in Networks with Heterogeneous Agents and Homophily

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Abstract

We study how a behavior (an idea, buying a product, having a disease, adopting a cultural fad or a technology) spreads among agents in an a social network that exhibits segregation or homophily (the tendency of agents to associate with others similar to themselves). Individuals are distinguished by their types (e.g., race, gender, age, wealth, religion, profession, etc.) which, together with biased interaction patterns, induce heterogeneous rates of adoption. We identify the conditions under which a behavior diffuses and becomes persistent in the population. These conditions relate to the level of homophily in a society, the underlying proclivities of various types for adoption or infection, as well as how each type interacts with its own type. In particular, we show that homophily can facilitate diffusion from a small initial seed of adopters.

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JEL Classification Numbers: D85, D83 C70, C73, L15, C45.

1 Introduction

Societies exhibit significant homophily and segregation patterns.[1] How do such biases in interactions affect the adoption of products, contagion of diseases, spread of ideas, and other diffusion processes? For example, how does the diffusion of a new product that is more

[1]For background on homophily and some of its consequences, see McPherson et al. (2001) and Jackson (2008).

attractive to one age group depend on the interaction patterns across age groups? How does the answer depend on the differences in preferences of such groups, their relative sociabilities, and biases in the interactions?

We answer these questions by analyzing a general model of diffusion that incorporates a variety of previous models as special cases, including contagion processes studied in the epidemiology literature such as the so-called SIS model (e.g., Bailey 1975, Pastor-Satorrás and Vespignani, 2001), as well as interactions with strategic complementarities, such as in the game theoretic literature and network games (e.g., Galeotti et al., 2010).[2] Our model incorporates types of individuals who have different preferences or proclivities for adoption, as well as biases in interactions across types.

In particular, we examine whether or not diffusion occurs from a very small introduction of an activity in a heterogeneous and homophilous society. We first concentrate on the focal situation with only two types of agents. Within this case, the most interesting scenario turns out to be one where one type would foster diffusion and the other would not if the types were completely segregated. In that scenario, we show that homophily actually facilitates diffusion, so that having types biased in interactions towards their own types can enhance diffusion to a significant fraction of both types. Having a higher rate of homophily, so that a group is more introspective, allows the diffusion to get started within the group that would foster diffusion on its own. This can then generate the critical mass necessary to diffuse the behavior to the wider society. In contrast, societies exhibiting less homophily can fail to foster diffusion from small initial seeds.

We then move to the general case of many types. Our main characterization theorem generalizes the features from the two-agent case, showing that diffusion relates to a condition on the largest eigenvalue of an interaction matrix which tracks the initial adoption rates of various types of individuals, that is, their adoption rates from small initial seeds. Again, we show that homophily can facilitate diffusion, showing that a sufficient condition is that some type (or group of types) that would adopt on its own is sufficiently homophilous to give the diffusion a toehold. We discuss how this extends the intuitions from the case of two types.

2   An Illustrative Example: The Heterogeneous SIS model with Two Types

To fix ideas and preview some of the insights from the general model, we begin with a case where there are just two types of agents and the contagion follows a simple and well-studied process.

In particular, consider an infectious disease spreading in a population with two groups: the young and the old. Our aim is to analyze whether or not diffusion of the disease occurs.

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[2]For background on diffusion in networks see Newman (2002), Jackson and Yariv (2005, 2007, 2010), López-Pintado (2006, 2008, 2010), Jackson and Rogers (2007) among others.

That is, if we start with a small seed of infected agents, will the infection spread to a significant fraction of both populations and become endemic? In order to answer this question consider the following heterogeneous version of the canonical SIS model.[3]

Agents can be in one of two "states": infected or susceptible. A susceptible agent becomes infected at an independent probability $\nu > 0$ from each interaction with an infected agent. Conversely, with a probability $\delta > 0$ per unit of time an infected individual recovers and becomes susceptible again.[4] The crucial parameter of the model is the relative spreading rate, $\lambda = \frac{\nu}{\delta}$, which measures how infectious the disease is in terms of how easy it is to contract compared to the rate at which one recovers.

An interesting case for our analysis is one where the population is heterogeneous in terms of the proclivities for getting infected. In particular, imagine that the older are more (or less) vulnerable to the disease than the young. More precisely, if λ_1 is the spreading rate of the young and λ_2 of the old, then we allow $\lambda_1 \neq \lambda_2$.

In addition to their age, individuals are also potentially differentiated by the rates at which they interact with other individuals, where "interact" is taken to mean that they have a meeting with an individual which could transmit the infection if one of them is infected and the other is susceptible. In particular, apart from his or her type, each individual is characterized by a degree d; the number of agents the individual meets (and is potentially infected by) every period. Let $P_i(d)$ be the degree distribution of individuals of type i; that is, the fraction of agents of type i that have d meetings per unit of time.

Also, for the purposes of this example, we stick with what is standard in the random network literature, and take the meeting process to be proportionally biased by degree. Thus, conditional on meeting an agent of type i, the probability that he or she is of degree d is proportional to $\frac{P_i(d)d}{\langle d \rangle_i}$, where $\langle d \rangle_i$ is the average degree among type i agents ($\langle d \rangle_i = \sum_d P_i(d)d$).

To capture homophily, let $0 < \pi < 1$ be the probability that a given type i agent (old or young) meets his or her own type, and $1 - \pi$ be the probability of meeting an agent of the other type. For example if the populations are of even size, then having $\pi > 1/2$ means that agents are mixing with their own type disproportionately.

We say that diffusion occurs from a small seed (with a formal definition below) if starting from an arbitrarily small amount of infected individuals (of either type), we end up with a nontrivial steady-state infection rate among the population.

Let $\pi_0 = \frac{1 - \widetilde{d}_1 \lambda_1 \widetilde{d}_2 \lambda_2}{\widetilde{d}_1 \lambda_1 + \widetilde{d}_2 \lambda_2 - 2\widetilde{d}_1 \lambda_1 \widetilde{d}_2 \lambda_2}$, where $\widetilde{d}_i = \frac{\langle d^2 \rangle_i}{\langle d \rangle_i}$.

THEOREM 1 *Diffusion occurs from a small seed in the two type SIS model if and only if one of the following holds:*

1) $\lambda_1 \lambda_2 > \frac{1}{\widetilde{d}_1 \widetilde{d}_2}$ *or*

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[3]The so-called SIS (Susceptible-Infected-Susceptible) model is a basic one used by the epidemiology literature to describe such situations (e.g., Bailey 1975, Pastór-Satorrás and Vespignani, 2000, 2001).

[4]The SIS model allows a recovered person to catch the disease again. An obvious instance is the standard flu.

2) $\lambda_1 \lambda_2 < \frac{1}{\tilde{d}_1 \tilde{d}_2}$ *and* $\pi > \pi_0$.

The proof of the theorem appears in the appendix, and is a special case of our more general results below.

The condition for diffusion in the standard (homogeneous) SIS model is $\lambda > \frac{1}{\tilde{d}}$ (e.g., Pastor-Satorrás and Vespignani, 2001). Thus, we see how this generalizes in the above theorem.

Theorem 1 yields the following straightforward consequences.

COROLLARY **1** *The following statements hold for the two-type SIS model:*

1) If diffusion occurs within each type when isolated (when $\pi = 1$), then it would also occur when there is interaction among the two (when $\pi < 1$).

2) If diffusion does not occur in either of the types when isolated, then it would not occur when there is interaction among the two.

3) If diffusion occurs among one type but not the other when isolated, then it will occur among the whole population if the homophily is high enough.

The most interesting scenario turns out is the last one, such that one of the types would foster diffusion if isolated, whereas the other would not (i.e., $\lambda_1 > \frac{1}{\tilde{d}_1}$ and $\lambda_2 < \frac{1}{\tilde{d}_2}$). In that scenario, homophily either plays no role (that is, when $\lambda_1 \lambda_2 > \frac{1}{\tilde{d}_1 \tilde{d}_2}$) so that any homophily level will allow diffusion, or else it actually facilitates diffusion (that is, when $\lambda_1 \lambda_2 < \frac{1}{\tilde{d}_1 \tilde{d}_2}$ in which case π must exceed π_0).

In the latter case diffusion occurs only if the two types are sufficiently biased in interactions towards their own types (i.e., π is sufficiently large). The intuition for such a result is the following. Having a higher rate of homophily, so that a group is more introspective, allows the diffusion to get started within the group that would foster diffusion on its own. In turn, it can then spread to the wider society.

3 The General Model

With this introduction behind us, we now describe the general model.

3.1 Types and Degrees

Each agent is characterized by his or her degree $d \geq 0$ and type $i \in T = \{1, ..., m\}$.

Since the number of individuals of each type can differ, let $n(i)$ be the fraction of individuals of type i.

An agent's degree d indicates the number of other agents that the agent meets (and is potentially influenced by) before making a decision in a given period. The meeting process is allowed to be directional; i.e., agent h meeting (paying attention to) agent k does not necessarily imply that k pays attention to h. So, although we use the term "meeting," the

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interaction need not be reciprocal. Of course, a special case is one where the interaction is mutual.

Different types may have different distributions in terms of how frequently they meet other agents. In particular, let $P_i(d)$ be the degree distribution of individuals of type i. That is, $P_i(d)$ is the fraction of type i individuals who have d meetings per period. Thus, there can be heterogeneity among agents of a given type, in terms of how social they are.

An agent's type i shapes both the agent's relative interaction rates with other types of agents and the agent's preferences or proclivity for infection. In particular, π_{ij} is the probability that an agent of type i meets an agent of type j in any given meeting. Clearly, $\sum_{j=1}^{m} \pi_{ij} = 1$. The bias in meetings across types is then summarized by the matrix

$$\Pi = \begin{pmatrix} \pi_{11} & \cdots & \pi_{1m} \\ \vdots & \cdots & \vdots \\ \pi_{m1} & \cdots & \pi_{mm} \end{pmatrix}.$$

We assume that Π is a primitive matrix (so that $\Pi^t > 0$ for some t). This ensures that there is at least some possibility for an infection that starts in one group to reach any other, as otherwise there are some groups that are completely insulated from some others.

3.2   The Random Meeting Process

In order to study this system analytically, we examine a continuum of agents, $N = [0, 1]$.

This continuum is partitioned into agents of different types, and then within types, by their degrees.

There are two ways in which the meeting process can be biased: by type and by degree.

In particular, as mentioned above, the relative proportion of a type i agent's meetings with type j is described by the term π_{ij}, which captures relative biases in meetings across types. So, in a given period, an agent of type i with degree d expects to meet $d\pi_{ij}$ agents of type j. Those agents are randomly selected from the agents among type j.

We also allow the meeting process to be biased by degree. The probability that an agent meets an agent of degree d out of those of type j is given by

$$P_j(d)w_j(d),$$

where $w_j(d)$ is a weighting factor. If there is no weighting by degree, then an agent equally samples all agents of type j and $w_j(d) = 1$. This would require a directed meeting process, such that an agent observes members of a given type uniformly at random, independently of their meeting process or sociability. If instead, meetings are proportional to how social the agents of type j are, then $w_j(d) = d/\langle d \rangle_j$, where $\langle d \rangle_j$ is the average degree among type j agents. This latter condition covers cases in which meetings are reciprocal.[5]

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[5]For some details and references for random meeting processes on a continuum, see the appendix of Currarini, Jackson and Pin (2009).

Our formulation also allows for other cases. For simplicity, we assume that $w_j(d) > 0$ for all j and d such that $P_j(d) > 0$.

3.3 The Infection or Adoption Process

In each period an agent is in one of two states $s \in \{0, 1\}$. Either the agent has adopted the behavior and are in state $s = 1$ (active, adopted, infected...), or they have not adopted the behavior and are in state $s = 0$ (passive, non-adopter, susceptible...). The agents' actions are influenced by the actions of others, but in a stochastic manner.

Agents are heterogeneous with respect to their proclivities to adopt the behavior. A passive agent of type i adopts the behavior at a rate described by a function $f_i(d, a)$, where d is the agent's degree (number of meetings per unit of time) and a is the number of agents whom she meets who have adopted the behavior. (Details of the dynamics will be given below.) The reverse process, by which an active agent of type i becomes passive happens at a rate described by a function $g_i(d, a)$. The functions $f_i(d, a)$ and $g_i(d, a)$ are the primitives of the diffusion process and are assumed to satisfy some basic conditions:

A1 $f_i(d, 0) = 0$ for each i and d: a passive agent cannot become active unless she meets at least one active agent.

A2 $f_i(d, a)$ is non-decreasing function in a: the adoption rate is non-decreasing in the number of active agents met.

A3 $f_i(d, 1) > 0$ for each i and some d such that $P_i(d) > 0$. This condition implies that for each type of agent there exists some degree such that the rate of adoption for agents with such a degree is positive when they meet at least one active agent.

A4 $g_i(d, 0) > 0$ for each i and d: it is possible to return from active to passive when all agents met are passive.

A5 $g_i(d, a)$ is non-increasing in a: the transition rate from active to passive is non-increasing in the number of active agents met.

This general model of diffusion admits a number of different models, including models based on best-response dynamics of various games (with trembles) as well as epidemiological models. Here are a few prominent examples of processes that are admitted:

- Susceptible-Infected-Susceptible (**SIS diffusion process**): $f_i(d, a) = \nu_i a$ and $g_i(d, a) = \delta_i$, where $\nu_i \geq 0$ and $\delta_i \geq 0$.

- Myopic-best response dynamics by agents who care about the *relative* play of neighbors (**Relative Threshold diffusion process**): $f_i(d, a) = \nu_i$ if $\frac{a}{d} \geq q$ and $f_i(d, a) = 0$ otherwise. Also $g_i(d, a) = \delta_i$ if $\frac{a}{d} < q$ and $g_i(d, a) = 0$ otherwise, where $\nu_i \geq 0$ and $\delta_i \geq 0$ and $q \in [0, 1]$.

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- Myopic-best response dynamics by agents who care about the *aggregate* play of neighbors (**Aggregate Threshold diffusion process**): $f_i(d,a) = \nu_i$ if $a \geq \min[q,d]$ and $f_i(d,a) = 0$ otherwise. Also, $g_i(d,a) = \delta_i$ if $a < q$ and $g_i(d,a) = 0$ otherwise, where $\nu_i \geq 0$ and $\delta_i \geq 0$ and $q \geq 0$.[6]

- Imitation dynamics when a neighbor is chosen uniformly at random (**Imitation diffusion process**): $f_i(d,a) = \nu_i \frac{a}{d}$ and $g_i(d,a) = \delta_i(1 - \frac{a}{d})$, where $\nu_i \geq 0$ and $\delta_i \geq 0$.

3.4 Steady States and Dynamics

In order to keep track of how diffusion or infection occurs, we analyze a continuous time dynamic, where at any given time $t \geq 0$ the state of the system consists of a partition of the set of agents in "active" and "passive."

As is standard in the literature, we study the continuous system as an analytically tractable alternative to the stochastic discrete system.[7]

Let $\rho_{i,d}(t)$ denote the frequency of active agents at time t among those of type i with degree d. Thus,

$$\rho_i(t) = \sum_d P_i(d)\rho_{i,d}(t)$$

is the frequency of active agents at time t among those of type i, and

$$\rho(t) = \sum_i n(i)\rho_i(t)$$

is the overall fraction of active agents in the population at time t.

The adoption dynamics are described as follows:

$$\frac{d\rho_{i,d}(t)}{dt} = -\rho_{i,d}(t)rate_{i,d}^{1\to0}(t) + (1 - \rho_{i,d}(t))rate_{i,d}^{0\to1}(t), \tag{1}$$

where $rate_{i,d}^{0\to1}(t)$ is the rate at which a passive agent of type i and with degree d becomes active, whereas $rate_{i,d}^{0\to1}(t)$ stands for the reverse transition. In order to compute these transition rates we must calculate first the probability that an agent of type i has of sampling an active agent. Denote this probability by $\widetilde{\rho}_i(t)$. It is straightforward to see that

$$\widetilde{\rho}_i(t) = \sum_j \pi_{ij} \sum_d P_j(d)w_j(d)\rho_{j,d}(t). \tag{2}$$

Given $\widetilde{\rho}_i(t)$ then

$$rate_{i,d}^{0\to1}(t) = \sum_{a=0}^d f_i(d,a)\binom{d}{a}\widetilde{\rho}_i(t)^a(1 - \widetilde{\rho}_i(t))^{(d-a)}$$

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[6]In order to satisfy [A3] in this case, it is necessary to have some probability of degree 1 agents for each type, or else to have $q = 1$.

[7]See Jackson (2008) for discussion of what is known about the approximation.

and

$$rate_{i,d}^{1\to 0}(t) = \sum_{a=0}^{d} g_i(d,a) \binom{d}{a} \widetilde{\rho}_i(t)^a (1 - \widetilde{\rho}_i(t))^{(d-a)}. \tag{3}$$

A *steady-state* is when $\frac{d\rho_{i,d}(t)}{dt} = 0$, which implies that we can write the steady state level $\rho_{i,d}(t)$ as being independent of time. Solving from equation (1) leads to the following necessary condition

$$\rho_{i,d} = \frac{rate_{i,d}^{0\to 1}}{rate_{i,d}^{0\to 1} + rate_{i,d}^{1\to 0}}. \tag{4}$$

If we specify the rates $\widetilde{\rho}_i(t)$ for each type i, then this determines the rates of transition under (3). This in turn, leads to a level of $\rho_{i,d}$ for each i,d under (4) that would have to hold in equilibrium, which in turn determines the rates at which active agents would be met under $\widetilde{\rho}_i(t)$. Thus, replacing equation (4) in equation (2) we find that a steady state equilibrium corresponds to a fixed point calculation as follows:

$$\widetilde{\rho}_i = H_i(\widetilde{\rho}_1 \ldots, \widetilde{\rho}_n), \tag{5}$$

where

$$H_i(\widetilde{\rho}_1, \ldots, \widetilde{\rho}_n) = \sum_j \pi_{ij} \sum_d P_j(d) w_j(d) \frac{rate_{j,d}^{0\to 1}}{rate_{j,d}^{0\to 1} + rate_{j,d}^{1\to 0}}$$

The previous system of equations implicitly characterizes the steady states of the dynamics, since by solving for $\widetilde{\rho}_i$ we can easily find the fraction of adopters of each type ρ_i and ultimately the overall fraction of adopters ρ.

3.5 Diffusion or Contagion from a Small Seed

We now consider the following question which is the central focus of our analysis: If we start with a small fraction of adopters, would the behavior spread to a significant fraction of the population(s)? In other words, we determine the conditions that lead to the diffusion of a new behavior to a significant fraction of the population when there is a small initial perturbation of an initial state in which nobody is infected or has adopted the behavior; so starting from $(\rho_1, \ldots, \rho_n) = (0, \ldots, 0)$.[8]

Thus, in what follows we explore the behavior of the system of (5) near $\widetilde{\boldsymbol{\rho}} = \overrightarrow{0}$; in order to see conditions under which it is a *stable* steady-state.

The system of equations described in (5) can be approximated by a linear system in the neighborhood of $\widetilde{\boldsymbol{\rho}} = \overrightarrow{0}$ as follows:

$$\widetilde{\boldsymbol{\rho}} = A\widetilde{\boldsymbol{\rho}}$$

[8]Notice that the question of moving away from all 1 is completely analogous, simply swapping notation between 0 and 1 throughout the model.

where

$$A = \begin{pmatrix} \frac{\partial H_1}{\partial \widetilde{\rho}_1}\big|_{\widetilde{\rho}=0} & \cdots & \frac{\partial H_1}{\partial \widetilde{\rho}_m}\big|_{\widetilde{\rho}=0} \\ \vdots & \ldots & \vdots \\ \frac{\partial H_m}{\partial \widetilde{\rho}_1}\big|_{\widetilde{\rho}=0} & \cdots & \frac{\partial H_m}{\partial \widetilde{\rho}_m}\big|_{\widetilde{\rho}=0} \end{pmatrix}.$$

As we show in the appendix, filling in for the expressions of $\frac{\partial H_i}{\partial \widetilde{\rho}_j}\big|_{\widetilde{\rho}=0}$, we can rewrite A as

$$A = \begin{pmatrix} \pi_{11}x_1 & \ldots & \pi_{1m}x_m \\ \vdots & \ldots & \vdots \\ \pi_{m1}x_1 & \ldots & \pi_{mm}x_m \end{pmatrix}$$

where

$$x_i = \sum_d P_i(d) w_i(d) d \frac{f_i(d,1)}{g_i(d,0)}.$$

The term x_i is a nicely interpretable factor. It is the relative growth in infection due to type i, but adjusted by the relative rates at which type i's will be met by other agents (so weighted by degrees according to $w_i(d)$).

Note that if when we start with some vector of ρ_j's near 0 (so our approximation is correct), but with positive entries, and then we end up with a new vector that is at least as large as the starting vector, then it must be that 0 is an *unstable* solution.

Definition 1 *There is diffusion from a small seed if and only if for any small $\varepsilon > 0$, there exists some* $\mathbf{v}$ *such that $0 < v_i < \varepsilon$ for all i and $A\mathbf{v} > \mathbf{v}$.*

Thus, diffusion from a small seed requires that beginning any small fraction of initial adopters the "dynamics" lead to a larger fraction of adopters.

We remark that if 0 is unstable relative to some small initial seed $\mathbf{v} > 0$, then it is unstable relative to any small initial seed $\widetilde{\mathbf{v}} > 0$. That is, if $A\mathbf{v} > \mathbf{v}$, then for any $\widetilde{\mathbf{v}} > 0$ there is some t such that $A^t\widetilde{\mathbf{v}} > \widetilde{\mathbf{v}}$. Furthermore, if there is no diffusion with a particular small initial distribution, then there will be no diffusion with any other initial distribution. The next Lemma formalizes such argument.[9]

LEMMA **1** *The condition for the diffusion from a small seed is independent of the distribution across types of the initial seed. That is, if $A\mathbf{v} > \mathbf{v}$ for some $\mathbf{v} > 0$, then for any $\widetilde{\mathbf{v}} > 0$ there is some t such that $A^t\widetilde{\mathbf{v}} > \widetilde{\mathbf{v}}$.*

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[9]This result is partly an artifact of the continuous model approximation. For an analysis of the importance of the specifics of initial adopters, see Banerjee, Chandrasekhar, Duflo and Jackson (2011).

9

4 Analysis

4.1 Two Types

We begin with the analysis of two types, which is a generalization of the results in Section 2.

For now, we stick with a setting where $\pi_{11} = \pi_{22} = \pi$, so that there is a symmetry in how introspective groups are in terms of their meetings.

THEOREM **2** *Let $\pi_0 = \frac{1-x_1 x_2}{x_1 + x_2 - 2x_1 x_2}$. Diffusion occurs if and only if one of the following conditions hold:*

1) $x_1 x_2 > 1$ or
2) $x_1 x_2 \leq 1$ and $\pi > \pi_0$.[10]

The proof of Theorem 2 appears in the Appendix. This result generalizes what was found for the heterogeneous SIS model presented in Section 2. The next corollary presents straightforward consequences of it.

COROLLARY **2** *In the two-type setting*
1) If diffusion occurs within each type when isolated, then it would also occur when there is interaction among the two.
2) If diffusion does not occur among either of the types when isolated, then it would not occur when there is interaction among the two.
3) If diffusion would occur among only one of the types when isolated, then it would occur among the entire population if homophily is high enough.

To see Corollary 2 first note that if there is only one type of agent in the population then the condition for diffusion established by Theorem 2 reduces to the standard condition of $x > 1$. Therefore, diffusion occuring within each type when isolated corresponds to having $x_1 > 1$ and $x_2 > 1$. Those conditions in turn establish part 1) of the corollary as a consequence of part 1) of Theorem 2. If, on the contrary, diffusion does not occur among either of the types when isolated, then $x_1 < 1$ and $x_2 < 1$. Straightforward calculations show that then the condition for diffusion stated in part 2) of Theorem 2 cannot satisfied for any value of $\pi \in (0,1)$. The last part of the corollary follows vacuously if $x_1 x_2 > 1$, and otherwise diffusion occurs if π exceeds π_0, establishing the claim.

4.2 The General Case with Many Types

Consider the following matrix A:

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[10]Note that the second condition implies that either $x_1 > 1$ or $x_2 > 1$.

$$A = \begin{pmatrix} \pi_{11}x_1 & \ldots & \pi_{1m}x_m \\ \vdots & \ldots & \vdots \\ \pi_{m1}x_1 & \ldots & \pi_{mm}x_m \end{pmatrix}.$$

We remark that since $x_i > 0$ for all i (under our assumptions A1-A5), and since Π is primitive and nonnegative, it follows that A is primitive and thus $A^t > 0$ for some t.

We can now state the following result, which generalizes the two-type result to many types.

THEOREM **3** *Diffusion occurs if and only if the largest eigenvalue of A (denoted by μ) is larger than* 1.

The proof of Theorem 3 appears in the appendix.
Corollary 2 generalizes to the m-type case as presented next.

COROLLARY **3** *1) If diffusion from a small seed occurs within each type when isolated, then it would also occur when there is interaction among types.*

2) If diffusion from a small seed does not occur for any of the types when isolated, then it would not occur when there is interaction among them.

3) If there is some type for which $\pi_{ii}x_i > 1$, then there is diffusion from a small seed.

4) If there is a subset of types $S \subset T$ such that $\sum_{j \in S} \pi_{ij}x_j > 1$ for each $i \in S$, then there is diffusion from a small seed.

We first explain why 1) holds, as 2) is a simple variation. If diffusion occurs within each type when isolated then $x_i > 1$ for all i and therefore

$$A > \begin{pmatrix} \pi_{11} & \ldots & \pi_{1m} \\ \vdots & \ldots & \vdots \\ \pi_{m1} & \ldots & \pi_{mm} \end{pmatrix}.$$

It follows that the largest eigenvalue of A is larger than 1 (since the right-hand side matrix is a stochastic matrix and thus has a largest eigenvalue of 1), and the result then follows from Theorem 3.

Next let us explain why 3) and 4) are true, and then discuss the intuition. 3) is clearly a special case of 4), so let us discuss why 4) is true. Given that $\sum_{j \in S} \pi_{ij}x_j > 1$ for each $i \in S$, it follows that for any positive vector u: $[Au]_i$ is greater than $\min_{j \in S} u_j$ for each $i \in S$. Therefore, $\min_{j \in S}[Au]_j > \min_{j \in S} u_j$, and so it must be that if u is the eigenvector corresponding to the maximum eigenvalue,[11] then $Au > u$ and so the eigenvalue is larger than 1.

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[11] Again, recall that A is primitive and thus has a strictly positive eigenvector corresponding to its largest eigenvalue.

1) and 2) of the corollary are fairly intuitive results. Note that in case of just one population, then $x_i > 1$ is the condition that characterizes instability of (diffusion from) no activity. Thus, if all populations are such that they would experience diffusion from a small seed if isolated, then regardless of the interaction pattern there will be diffusion; and similarly if none of them would experience diffusion in isolation, then there cannot be diffusion when they interact.

The less obvious cases are 3) and 4), which show that if some type or group of types has enough interaction with itself to get diffusion going, then diffusion among the entire population will occur. Again, these emphasize the role of homophily in enabling diffusion (infection) from a small seed: if there is some group of types that interacts within itself in a manner sufficient to enable diffusion among that group, then a toehold can be established and diffusion will occur from a small seed.

Another corollary is that if populations are similar so that they have the same infection properties near 0 (i.e., $x_i = x_j = x$ for all i and j), then diffusion properties are determined by whether this growth rate is bigger or smaller than 1.

COROLLARY 4 *If $x_i = x_j = x$ for all i and j, then there is diffusion from a small seed if and only if $x > 1$.*

This corollary then emphasizes that in order for the homophily and particular patterns of interaction to matter, it must be that types are not just heterogeneous in their interaction (the Π matrix), but also in their adoption/infection proclivities. If they all have similar adoption/infection proclivities, then the particular details of who interacts with whom do not affect diffusion from a small seed.

The proof of this corollary is straightforward. Note that

$$A = x\Pi = x \begin{pmatrix} \pi_{11} & \dots & \pi_{1m} \\ \vdots & \dots & \vdots \\ \pi_{m1} & \dots & \pi_{mm} \end{pmatrix}.$$

It follows that the largest eigenvalue of A is larger than 1 if and only if $x > 1$ since Π is a stochastic matrix and has a maximum eigenvalue of 1.

The less obvious cases are thus such that there are some types who would experience diffusion on their own, while others would not. Then the interaction patterns really matter and, as already illustrated for the two-type case, some subtle conditions ensue. A sufficient condition again is that there is sufficient homophily such that infection can take hold within some type, and then it can spread among the population, but more complicated patterns among a number of groups can also possibly lead to diffusion from a small seed.

5   Concluding Remarks

The focus of most of the related literature has been on analyzing the effect that the degree distribution has on diffusion in social networks (see e.g., Jackson and Rogers, 2007, López-

Pintado, 2008, Galeotti and Goyal, 2009, Galeotti et al., 2010.). This paper, however, focuses on the effect of homophily, something which despite its importance has received little attention in the diffusion literature. One of the few exceptions is the paper by Golub and Jackson (2010) which also studies the impact of homophily on some (very different) learning and diffusion processes. There are important differences between our approach and theirs. On the one hand, the diffusion processes analyzed are not the same; we focus on what can be thought of as generalizations of the SIS infection model, whereas Golub and Jackson (2010) analyze models of diffusion based either on shortest paths communication, random walks or linear updating processes. Second, the paper by Golub and Jackson (2010) studies the convergence time to the steady state, whereas we analyze whether there is or not convergence to a state with a positive fraction of adopters.

As a first step to understanding the effect of homophily on diffusion, in this paper we have concentrated on a specific question; namely the spreading of a new behavior when starting with a small initial seed. A central insight here is that homophily can facilitate infection or contagion.

Nevertheless, there are other issues which are left for further work. For example, one could evaluate the size of the adoption endemic state as a function of the homophily level. There homophily might have conflicting effects: although it can facilitate an initial infection, it might be that an increase in homophily can also lead to a decrease in the overall infection rate. Indeed, the eventual fraction of adopters attained in the steady state might depend on the homophily level in complicated ways.

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Appendix

Proof of Theorem 1: The proof of Theorem 1 is a straightforward consequence of the proof of Theorem 2 as seen by substituting the functions $f_i(d, a) = \nu_i a$, $g_i(d, a) = \delta_i$ and $w_i(d) = \frac{d}{\langle d \rangle_i}$ and obtaining the corresponding x_i's. ∎

Proof of Theorem 3: First, note that the system of equations described describing the steady state is

$$\widetilde{\rho}_i = H_i(\widetilde{\rho}_1, \widetilde{\rho}_2, ..., \widetilde{\rho}_m), \tag{6}$$

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where

$$H_i(\widetilde{\rho}_1, \widetilde{\rho}_2, ..., \widetilde{\rho}_m) = \sum_j \pi_{ij} \sum_d P_j(d) w_j(d) \frac{rate_{j,d}^{0 \to 1}}{rate_{j,d}^{0 \to 1} + rate_{j,d}^{1 \to 0}}$$

for $i \in \{1, ..., m\}$.

This is approximated by a linear system in the neighborhood of $(\widetilde{\rho}_1, \ldots, \widetilde{\rho}_n) = (0, \ldots, 0)$ as follows:

$$\widetilde{\boldsymbol{\rho}} = A\widetilde{\boldsymbol{\rho}}$$

where

$$A = \begin{pmatrix} \frac{\partial H_1}{\partial \widetilde{\rho}_1}|_{\widetilde{\rho}=0} & \cdots & \frac{\partial H_1}{\partial \widetilde{\rho}_m}|_{\widetilde{\rho}=0} \\ \vdots & \cdots & \vdots \\ \frac{\partial H_m}{\partial \widetilde{\rho}_1}|_{\widetilde{\rho}=0} & \cdots & \frac{\partial H_m}{\partial \widetilde{\rho}_m}|_{\widetilde{\rho}=0} \end{pmatrix}$$

Note that

$$\frac{\partial rate_{i,d}^{0 \to 1}}{\partial \widetilde{\rho}_i} = \sum_{a=0}^{d} f_i(d,a) \binom{d}{a} \left(a\widetilde{\rho}_i^{\,a-1}(1-\widetilde{\rho}_i)^{(d-a)} + (d-a)\widetilde{\rho}_i^{\,a}(1-\widetilde{\rho}_i)^{(d-a-1)} \right)$$

and therefore

$$\frac{\partial rate_{i,d}^{0 \to 1}}{\partial \widetilde{\rho}_i}|_0 = f_i(d,0) \binom{d}{0} (d-0)(1-0)^{(d-1)} + f_i(d,1) \binom{d}{1} (1-0)^{(d-1)} = d \left[f_i(d,1) + f_i(d,0) \right].$$

Analogously

$$\frac{\partial rate_{i,d}^{1 \to 0}}{\partial \widetilde{\rho}_i}|_0 = d \left[g_i(d,1) + g_i(d,0) \right].$$

Then

$$\frac{\partial H_i}{\partial \widetilde{\rho}_j}|_{\widetilde{\rho}=0} = \pi_{ij} \sum_d P_j(d) w_j(d) \frac{\frac{\partial rate_{j,d}^{0 \to 1}}{\partial \widetilde{\rho}_j}|_0 rate_{j,d}^{1 \to 0}|_0 - rate_{j,d}^{0 \to 1}|_0 \frac{\partial rate_{j,d}^{1 \to 0}}{\partial \widetilde{\rho}_j}|_0}{\left(rate_{j,d}^{0 \to 1} + rate_{j,d}^{1 \to 0} \right)^2 |_0}$$

and thus,

$$A = \begin{pmatrix} \pi_{11} x_1 & \cdots & \pi_{1m} x_m \\ \vdots & \cdots & \vdots \\ \pi_{m1} x_1 & \cdots & \pi_{mm} x_m \end{pmatrix}$$

where

$$x_i = \sum_d P_i(d) w_i(d) d \frac{f_i(d,1) g_i(d,0) - f_i(d,0) g_i(d,1)}{(f_i(d,0) + g_i(d,0))^2}.$$

Given A1, x_i can be rewritten as

$$x_i = \sum_d P_i(d) w_i(d) d \frac{f_i(d,1)}{g_i(d,0)},$$

which is well defined since A4 holds.

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As mentioned in the text, A is primitive since Π is primitive and since A1 and A4 are satisfied implying that $x_i > 0$.[12] Thus, by the Perron–Frobenius Theorem (which applies to primitive matrices) the maximum eigenvalue, denoted μ hereafter, is positive and its corresponding eigenvector, denoted by $\mathbf{u}$ hereafter, is also positive.

We show next that the condition for diffusion from a small seed, or the instability of $\widetilde{\rho} = 0$, corresponds with the condition that the largest eigenvalue of A is larger than 1.

Let us first show that if $\mu > 1$ then $\widetilde{\rho} = 0$ is unstable. Note that if $\mu > 1$ then

$$A\delta\mathbf{u} = \mu\delta\mathbf{u} > \delta\mathbf{u}.$$

Thus, picking small enough δ so that $\delta u_i < \varepsilon$ for each i, satisfies the definition of diffusion from a small seed with $\delta\mathbf{u}$ (or instability of 0).

To see the converse, first consider the case such that $\mu < 1$. Given $\varepsilon > 0$ consider any $\mathbf{v}$ such that $0 < v_i < \varepsilon$ for all i. Suppose that $A\mathbf{v} > \mathbf{v}$. It then follows $A(A\mathbf{v}) > A\mathbf{v} > \mathbf{v}$ as A is nonnegative and has at least one positive entry in each row. Iterating, it follows that that $A^t\mathbf{v} > \mathbf{v}$ for any t. However, choose δ such that $\delta\mathbf{u} > \mathbf{v}$. Given that A is is nonnegative and has at least one positive entry in each row, and both vectors are positive, it follows that $A\delta\mathbf{u} > A\mathbf{v}$, and similarly that

$$A^t\delta\mathbf{u} > A^t\mathbf{v}.$$

Given our previous claim, this then implies that

$$A^t\delta\mathbf{u} > \mathbf{v}$$

for all t. However,

$$A^t\delta\mathbf{u} = \delta\mu^t\mathbf{u} \to 0$$

given that $\mu < 1$, which is a contradiction.

To complete this part of the proof consider the case such that $\mu = 1$. Consider $\varepsilon > 0$. Consider any vector $\mathbf{v}$ such that $\mathbf{v}_i < \varepsilon$. Note that for any small enough $\delta > 0$ the largest eigenvalue of $A - \delta I$ is less than 1. Thus, by the argument above, $(A - \delta I)\mathbf{v}$ is not greater than $\mathbf{v}$. Therefore, $A\mathbf{v}$ is not greater than $\mathbf{v}$. ∎

Proof of Theorem 2: We have already shown that $\widetilde{\rho} = 0$ is unstable if and only if the largest eigenvalue of matrix A is above 1. Let us now complete the proof by examining the eigenvalue in the two-type case. The eigenvalues of a 2×2 matrix are easily computed. Writing

$$A = \begin{pmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{pmatrix},$$

the largest eigenvalue of A is[13]

$$\mu = \frac{(a_{11} + a_{22}) + \sqrt{(a_{11} + a_{22})^2 - 4(a_{11}a_{22} - a_{12}a_{21})}}{2}$$

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[12]In fact, with two types A is a positive matrix since $0 < \pi < 1$.

[13]Note that since A is primitive, its largest eigenvalue is real and positive.

or equivalently

$$\mu = \frac{a_{11} + a_{22} + \sqrt{(2 - a_{11} - a_{22})^2 - 4 + 4a_{11} + 4a_{22} - 4a_{11}a_{22} + 4a_{12}a_{21}}}{2}.$$

Thus, μ is larger than 1 if and only if

$$\frac{a_{11} + a_{22}}{2} > 1 \tag{7}$$

or

$$-1 + a_{11} + a_{22} - a_{11}a_{22} + a_{12}a_{21} > 0. \tag{8}$$

Given that $a_{11} = \pi x_1$, $a_{22} = \pi x_2$, $a_{12} = (1 - \pi)x_2$ and $a_{21} = (1 - \pi)x_1$ then conditions (7) and (8) imply that diffusion (i.e., instability of $\widetilde{\rho} = 0$) occurs if and only if

$$\pi > \frac{2}{x_1 + x_2} \tag{9}$$

or

$$\pi(x_1 + x_2 - 2x_1x_2) + x_1x_2 - 1 > 0. \tag{10}$$

Case 1: $\frac{x_1 + x_2}{2x_1x_2} > 1$. In this case, condition (10) is equivalent to

$$\pi > \frac{1 - x_1x_2}{x_1 + x_2 - 2x_1x_2}$$

and therefore diffusion occurs in this case if and only if

$$\pi > \min\{\frac{1 - x_1x_2}{x_1 + x_2 - 2x_1x_2}, \frac{2}{x_1 + x_2}\}. \tag{11}$$

Case 2: $\frac{x_1 + x_2}{2x_1x_2} < 1$. In this case, condition (10) is equivalent to

$$\pi < \frac{x_1x_2 - 1}{2x_1x_2 - x_1 - x_2}$$

and therefore diffusion occurs in this case if and only if

$$\frac{2}{x_1 + x_2} < \pi \text{ or } \pi < \frac{x_1x_2 - 1}{2x_1x_2 - x_1 - x_2}. \tag{12}$$

Case 3: $\frac{x_1 + x_2}{2x_1x_2} = 1$. In this case, condition (10) simplifies to $x_1x_2 > 1$, and and therefore diffusion occurs in this case if and only if

$$\frac{2}{x_1 + x_2} < \pi \text{ or } x_1x_2 > 1 \tag{13}$$

Let us now show part (1) of Theorem 2.

Suppose that $x_1x_2 > 1$ holds. Then $\frac{x_1 + x_2}{2x_1x_2}$ can fall into any of the cases above. If it were greater than 1, then $\frac{1 - x_1x_2}{x_1 + x_2 - 2x_1x_2} < 0$ which in particular by Case 1 and (11) implies that

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there is diffusion for any $\pi \in (0,1)$. If it were equal to 1, then by Case 3, the result holds. If instead $\frac{x_1+x_2}{2x_1x_2} < 1$ then Case 2 applies. In that case, referring to Figure 1, (x_1, x_2) lies above the upper-most curve,[14] and it is clear that there would exist another profile $(\widehat{x}_1, \widehat{x}_2)$ such that $\widehat{x}_1 \leq x_1$ and $\widehat{x}_2 \leq x_2$ and which lies in the regions considered previously (that is, where $\frac{1-x_1x_2}{x_1+x_2-2x_1x_2} \leq 0$). Therefore diffusion for $(\widehat{x}_1, \widehat{x}_2)$ occurs for all $\pi \in (0,1)$, which in particular implies that for the larger case (x_1, x_2) diffusion would also occur for all $\pi \in (0,1)$ as the largest eigenvalue of a larger matrix is necessarily larger than the largest eigenvalue of a smaller matrix.
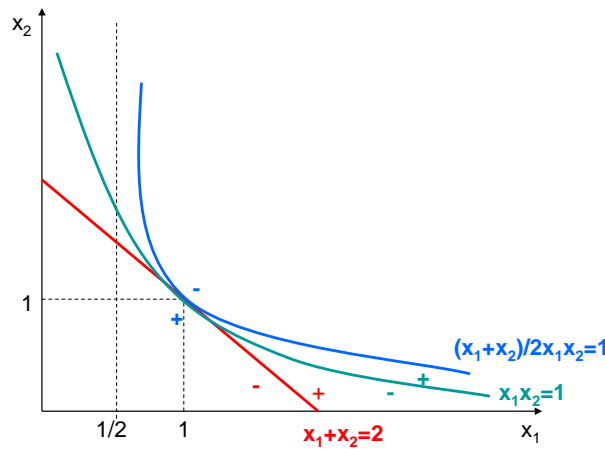
Figure 1: The relationship between the key expressions in the proof of Theorem 2.

Next, we show part (2) of Theorem 2. Suppose that $x_1x_2 \leq 1$. This implies that $\frac{x_1+x_2}{2x_1x_2} > 1$ (see Figure 1) or else that $x_1 = x_2 = 1$ in which Case 3 applies and there cannot be diffusion. Thus, let us analyze the situation where $\frac{x_1+x_2}{2x_1x_2} > 1$ and Case 1 applies. Diffusion occurs if and only if $\pi > \min\{\frac{1-x_1x_2}{x_1+x_2-2x_1x_2}, \frac{2}{x_1+x_2}\}$. Note that if $x_1 + x_2 < 2$ then $\frac{2}{x_1+x_2} > 1$ and therefore diffusion occurs if and only if $\pi > \frac{1-x_1x_2}{x_1+x_2-2x_1x_2}$. If, on the contrary, $x_1 + x_2 \geq 2$ then, it is straightforward to show that $\frac{2}{x_1+x_2} > \frac{1-x_1x_2}{x_1+x_2-2x_1x_2}$ which also implies that diffusion in such a case occurs if and only if $\pi > \frac{1-x_1x_2}{x_1+x_2-2x_1x_2}$. ∎

Proof of Lemma 1: Given the proof of Theorem 3, it follows that if $A\mathbf{v} > \mathbf{v}$ for some $\mathbf{v} > 0$ then $\mu > 1$. Then, choose δ such that $\delta\mathbf{u} < \widehat{\mathbf{v}}$. It follows that $A\delta\mathbf{u} < A\widehat{\mathbf{v}}$ (since A is

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[14]The relative positions of the curves are easily checked, and note the plus and minus signs that indicate whether one is above or below 1 for the corresponding colored expression.

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nonnegative and has at least one positive entry in each row), and similarly that

$$\mu^t \delta\mathbf{u} = A^t \delta\mathbf{u} < A^t \widehat{\mathbf{v}},$$

and the first expression is growing with μ^t. ∎