

Diffusion and protection across a random graph

Andrea Galeotti and Brian W. Rogers*

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Abstract: We study the interplay between the diffusion of a harmful state in a network of contacts and the possibility of individual agents to undertake costly investment to protect themselves against infection. We characterize how equilibrium diffusion outcomes, such as immunization rate and total prevalence, respond to changes in the architecture of the network, and show that these responses depend on the details of the diffusion process.

1 Introduction

We study the diffusion of a harmful state through a population when agents can take preventative action to protect themselves against infection. While the model is stylized, its main ingredients capture important aspects of a number of applications. Nodes in the network could represent firms, which must decide how aggressively to institute firewalls, VPN use, password protocols and the like in order to limit their exposure to viruses and network-based attacks. A second interpretation imagines the population as a network of banks or financial institutions, where links represent the presence of cross-institutional exposure, and the harmful state captures the presence of toxic assets in a bank's portfolio. In this case, the investment consists of committing to sufficient effort to screen assets before engaging in a transaction. Finally, a classical example describes individuals vaccinating in order to prevent human infection of various communicable diseases spreading through social contacts.

Our model has three distinctive features. First, the diffusion process we consider is fairly general and it encompasses, as a special case, the Susceptible-Infected-Susceptible (SIS) framework.¹ Second,

*Galeotti, University of Essex, Department of Economics, Wivenhoe Park, Colchester, Essex CO3SQ, United Kingdom, agaleo@essex.ac.uk; Rogers, Washington University in St. Louis, Department of Economics, brogers@wustl.edu.

¹The SIS model was developed originally to study epidemiological dynamics. More recently it has been adopted by other disciplines to help understand diverse applications such as behaviors, information diffusion, learning dynamics, myopic best response, and imitation dynamics. For classical epidemiological studies of SIS and variants of it see Bailey

we assume that agents are heterogenous in their intensity of interactions, which we call degree. The network of contacts is described by its degree distribution, which we allow to be completely general.

Third, we explicitly model the incentives of agents who are exposed to the diffusion process.² In particular, agents weigh the cost of immunity against the expected cost of exposure to the harmful state during their lifetime, and decide optimally under beliefs that are consistent with the steady-state of the diffusion process, given the investment decisions of the population. We consider both the case in which the investment to protect is taken before agents know their own degree, and the case in which such investment is contingent upon their own degree.³ The former assumption is appropriate when the investment is ex-ante to the diffusion process and difficult to reverse, such as, perhaps, the case of a firm's management deciding on the appropriate investment in firewalls. The latter assumption is more appropriate in the financial example, where large banks understand that they interact with many more banks compared to their smaller counterparts.

These three features, a general diffusion model, general degree distributions and an explicit model of incentives, together allow us to address the following questions: how does a change in the network structure affect immunization patterns, and what is the joint effect on long-run diffusion outcomes such as prevalence? To what extent are such effects independent of the underlying diffusion process, and to what extent do they depend on the specific parameters governing diffusion such as the rate of recovery, the birth/death process, the intensity of infectiousness, and so forth?

Our results characterize how equilibrium diffusion outcomes such as the immunization rate, the infection rate among non-immunized agents, and total prevalence, respond to changes in the architecture of the network. We adopt the notion of first order stochastic dominance in the degree distribution to shed light on the effects of an increase in connectivity in the population; the notion of second order stochastic dominance is used to study the effect of increasing the dispersion of connections. But, in our view, the main contribution of this paper is to point out that, in order to understand how equilibrium diffusion outcomes respond to changes in the social structure, the details of the diffusion process must be taken into consideration. That is, we uncover important interactions between the way in which the diffusion process operates and the way in which network structure affects agents' incentives.

For example, we show that an increase in the connectivity of the network decreases the infection rate among one's neighbors. If, in addition, the degree distribution of one's neighbors increases along

(1975) and Anderson and May (1992). For recent work in economics adopting these frameworks see, for example, Galeotti and Rogers (2013), Jackson and Rogers (2007), López-Pintado (2008) and Jackson and Yariv (2010).

²In the last two decades some progress has been made towards incorporating incentives into epidemiological diffusion models and our paper contributes to this line of research. The literature in economics dates at least to Brito et al. (1991). Important contributions include Kremer (1996), Galeotti and Rogers (2013), Geoffard and Philipson (1996), Goyal and Vigier (2010), Geoffard and Philipson (1997), Bauch and Earn (2004), Boulier et al. (2007), Toxvaerd (2010a) and Toxvaerd (2010b)

³The method of analysis in this setting bears some similarity to the analysis of Galeotti et al. (2010) for their case of strategic substitutes, but the two models are not equivalent and the results are, in fact, different.

with the degree distribution of the population then the equilibrium protection rate increases. These results are robust, in that they hold true for all diffusion processes that we study. However, the effect of connectivity on total prevalence is ambiguous: it depends on the parameters defining the diffusion process. When the diffusion process is characterized by a low recovery rate and low turnover in the population, then we can show that an increase in network connectivity leads to a decrease in total prevalence. For comparison, an increase in network connectivity would unambiguously increase total prevalence in an analogous setting where the level of protection is exogenous (see, e.g., Jackson and Rogers (2007)).

2 Diffusion across a random network

We build on a standard diffusion process involving recovery, death, and impatience. A continuum of agents is distinguished by heterogeneity of their intensity of interactions, which we call degree. More specifically, degree is distributed according to F , so that proportion of agents with degree less than or equal to k is $F(k)$. At any given time, an agent interacts with a randomly chosen set of other agents.

When an agent is born, he makes a decision to protect or not. Formally, the pure action set is $\{P, V\}$, with the interpretation that P represents obtaining protection and V represents the choice to remain vulnerable. Agents with protection remain healthy until they die. Death occurs at rate t (for turnover). Upon death, an agent is replaced with a newborn agent. Vulnerable agents exist in one of two states, S and I , for susceptible and infected, respectively. The transition from I to S happens at rate $r \geq 0$ (for recovery). The transition from S to I depends on the states of an agent's neighbors, the agent's degree, and an infection rate $s \geq 0$.

Let π denote the proportion of agents with protection. Below, we develop a model in which π is dictated by equilibrium choices of entering agents, but at present we treat it as given. Let ρ_k denote the proportion of *vulnerable* degree- k agents who are in state I . Define θ as the probability with which a randomly chosen neighbor of an agent is in state I . Notice that this calculation is different from the overall prevalence, since the degree of a randomly chosen neighbor is distributed according to \tilde{F} , where $P_{\tilde{F}}(k) = kP_F(k)/E_F(k)$ if k is a mass point of F , and where $\tilde{f}(k) = kf(k)/E_F(k)$ otherwise. We therefore have that

$$\theta = (1 - \pi) \int \rho_k d\tilde{F}(k). \tag{1}$$

While the quantities defined above generally vary with time under the dynamics described by the diffusion, we are interested in the steady states of this process. A steady state is described by

time-invariant infection levels at each degree. Formally, we have

$$\frac{d\rho_k}{dt} = (1 - \rho_k)s\theta k - \rho_k(r + t) = 0$$

for every degree k . The first term represents inflow of infection from the mass of susceptible agents, who expect to have θk infected neighbors from whom they become infected at rate s .⁴ The second term represents the outflow of infection due to recovery and death. Solving the above equations produces the steady state condition

$$\rho_k = \frac{s\theta k}{s\theta k + r + t}. \quad (2)$$

We can re-write the steady state condition via substitution of (2) into (1) to obtain

$$1 = (1 - \pi) \int \frac{sk}{s\theta k + r + t} d\tilde{F}(k). \quad (3)$$

We now describe incentives. Agents receive a flow utility of 1 while healthy and $w \in [0, 1]$ while infected. Future payoffs are discounted at rate $d \geq 0$. Protection costs an agent a proportion $(1 - a)$ of his total utility.

At birth, an agent chooses whether or not to protect so as to maximize his expected lifetime payoff. Let us denote the steady state expected continuation values of protection, conditional on having a degree k , by V_k^P , of being susceptible by V_k^S , and of being infected as V_k^I . We have that

$$\begin{aligned} dV_k^P &= a - tV_k^P \\ dV_k^S &= 1 - tV_k^S - s\theta k(V_k^S - V_k^I) \\ dV_k^I &= w - tV_k^I + r(V_k^S - V_k^I). \end{aligned}$$

Solving the above system yields

$$\begin{aligned} V_k^P &= \frac{a}{d + t} \\ V_k^S &= \left(\frac{1}{d + t} \right) \left(\frac{d + t + r + ws\theta k}{d + t + r + s\theta k} \right) \\ V_k^I &= \left(\frac{1}{d + t} \right) \left(\frac{r + w(d + t + s\theta k)}{d + t + r + s\theta k} \right). \end{aligned}$$

Since the continuation value of protection is independent of k , we shall refer to it as V^P . Let us define $G(\theta, k) = \frac{d+t+r+ws\theta k}{d+t+r+s\theta k}$. The equilibrium protection level in the population π must satisfy (i) if $\pi > 0$ then $V_P \geq E_F[V_k^S]$ and (ii) if $\pi < 1$ then $E_F[V_k^S] \geq V_P$. Our goal is to characterize interior

⁴The linear infection rate obtains for low infection rates, s , as is typically relevant, when an agent is infected independently from each of his neighbors.

equilibria, for which, in particular, $E_F[V_k^S] = V_P$, which yields

$$\int G(\theta, k) dF(k) = a. \quad (4)$$

In summary, an interior steady state equilibrium is characterized by a solution (θ, π) to (3) and (4).

We will also be interested in two additional measures of prevalence. First, let ρ denote the average infection rate among all vulnerable agents, so that

$$\rho = \int \rho_k dF(k).$$

Finally we are interested in the total level of prevalence in society, which we denote by

$$\sigma = (1 - \pi)\rho.$$

Following standard arguments, it is easy to show the following.

Observation 1 *For a given level of protection π , distribution of degrees F , and diffusion parameters (d, r, s, t) , it is either the case that (i) there is a unique steady state in which $\theta = 0$, or (ii) there are two steady states, exactly one of which has positive prevalence. In the latter case the unique positive-valued steady state is globally stable.*

Henceforth, when comparing outcomes, we assume that the payoffs, diffusion parameters, and degree distribution are such that the non-trivial steady state exists, and we derive our conclusions based on that steady state.

3 Main results

We maintain in this section the assumption that agents take their protection decision knowing the degree distribution, but before learning their realized degree.

We begin by focusing on the simple example in which all agents have the same degree. This allows us to demonstrate the effects of an increase in connectivity in the simplest possible setting. Further, it serves as a benchmark for comparison to the case in which the protection decision is conditioned on degree, since in a regular graph the two assumptions are equivalent, i.e., the assumption is only relevant to the extent that F has dispersion.

In a regular graph of degree k every agent has the same number of connections and equilibrium can be characterized explicitly. In this case we have $\sigma = \theta = (1 - \pi)\rho_k$.

Solving for an interior steady state equilibrium we obtain

$$\begin{aligned}\theta &= \left(\frac{1-a}{a-w}\right) \left(\frac{d+t+r}{ks}\right) \\ \pi &= 1 - \frac{1}{ks} \left[\left(\frac{1-a}{a-w}\right) d + \left(\frac{1-w}{a-w}\right) (t+r) \right].\end{aligned}$$

To have $\theta > 0$, therefore, requires that $w < a < 1$. To have $\pi > 0$ requires k to be sufficiently large.

Proposition 1 *Fix payoffs and a diffusion process. Consider a regular graph with degree k and a regular graph with degree $k' > k$. Then $\theta' < \theta$, $\pi' > \pi$, $\rho'_k = \rho_k$ and $\sigma' < \sigma$.*

Proof 1 *Immediate from the expressions above.*

In words, we find that an increase in the degree of interaction increases the rate of protection in such a way that the infection rate among vulnerable agents remains constant. As a result, total prevalence, and the infection rate of one's neighbors, both decrease.

We proceed by generalizing this argument. In particular we consider an arbitrary degree distribution F and ask how the equilibrium changes when connectivity is increased in the sense of first order stochastic dominance. A distribution F' first order stochastically dominates a distribution F if $F'(k) \leq F(k)$ for all k , with strict inequality for some k .

Proposition 2 *Fix payoffs and a diffusion process. Let F' FOSD F . Then $\theta' < \theta$ and for every k , $\rho'_k < \rho_k$. If also \tilde{F}' FOSD \tilde{F} then $\pi' > \pi$.*

Proof 2 *Assume for a contradiction that $\theta' \geq \theta$. We have*

$$\begin{aligned}a &= \int_k G(\theta, k) dF(k) \\ &> \int_k G(\theta, k) dF'(k) \\ &\geq \int_k G(\theta', k) dF'(k) \\ &= a\end{aligned}$$

where the first inequality is because G is decreasing in k and the second is because G is decreasing in θ . Once the θ s are ordered the ordering of ρ_k follows immediately by noticing from expression (2) that ρ_k is increasing in θ .

To see the second part of the statement, we examine the steady state condition (3) and observe

that

$$\begin{aligned}
1 &= (1 - \pi) \int \frac{sk}{s\theta k + r + t} d\tilde{F}(k) \\
&< (1 - \pi) \int \frac{sk}{s\theta'k + r + t} d\tilde{F}'(k) \\
&< (1 - \pi) \int \frac{sk}{s\theta'k + r + t} d\tilde{F}'(k).
\end{aligned}$$

where the first inequality follows because the integrand of the steady state condition (3) is increasing in k and \tilde{F}' FOSD \tilde{F} ; the second inequality follows because the integrand of the steady state condition (3) is increasing in θ and $\theta' < \theta$. Hence, $\pi' > \pi$.

The infection rate among one's neighbors decreases when connectivity increases. However, the conclusion that the equilibrium protection rate increases relies on the degree distribution of one's neighbors increasing along with F . In most common families of degree distributions, F and \tilde{F} move together in terms of first order dominance, and in such cases the increase in the protection rate is guaranteed by FOSD of F . But in general the assumption on \tilde{F} is independent of that on F .

In any case, the effect on total prevalence is ambiguous without additional assumptions on F or the diffusion process. This is so because the effect on the average infection rate among vulnerable agents, ρ , is ambiguous: at each degree k the vulnerable agents have a lower infection rate under F' , but higher degree nodes have higher infection rates, and so the shift in F has an opposite upward effect on ρ .

The next result, in tandem with the example that follows, shows that there are interesting interactions between the diffusion process and the social structure. In particular, we demonstrate that when the turnover rate and the recovery rate are both low, a first order shift in the degree distribution allows us to unambiguously order both the protection level and total prevalence.

Proposition 3 *Fix payoffs and a diffusion process. Let F' FOSD F . There exists a positive constant c such that if $t + r \in [0, c]$, then $\pi' > \pi$ and $\sigma' < \sigma$.*

Proof 3 *Assume for a contradiction that $\pi' \leq \pi$. Examining steady state condition (3) we observe that*

$$1 = (1 - \pi) \int \frac{sk}{s\theta k + r + t} d\tilde{F}(k) < (1 - \pi') \int \frac{sk}{s\theta'k + r + t} d\tilde{F}(k).$$

where the inequality follows because, by Proposition 2, $\theta' < \theta$ and, by assumption, $\pi' \leq \pi$. Since

$$\lim_{t+r \rightarrow 0} \int \frac{sk}{s\theta'k + r + t} d\tilde{F}(k) = \lim_{t+r \rightarrow 0} \int \frac{sk}{s\theta'k + r + t} d\tilde{F}'(k) = \frac{1}{\theta'}$$

we have, by continuity, that there exists a positive constant c such that for $t + r \in [0, c]$ it holds that

$$1 < (1 - \pi') \int \frac{sk}{s\theta'k + r + t} d\tilde{F}'(k).$$

and we reach a contradiction with the steady state condition. Finally note that for $t + r$ small then ρ_k is approximately equal to one and therefore $\theta = \sigma = (1 - \pi) > \theta' = \sigma' = (1 - \pi')$, from which the result immediately follows.

That is, if agents live long lives and rarely, if ever, recover from infection, then vulnerable agents spend essentially all of their lives infected. In this case, an increase in connectivity implies an increased incentive to protect, and a consequent decrease in overall prevalence. Mathematically, the fact that ρ_k becomes nearly constant in k shuts down the channel in the model by which differences in degree translate into differences in outcomes. In that sense, we recover exactly the same results as we obtained in the case of regular graphs above.

The following example shows that the particular instances of the diffusion process, where we examine $t + r$ small, is important. It also allows us to elucidate the role of the assumptions in Propositions 2 and 3. Consider the following

Example 1 *We construct an instance for which F' FOSD F , but in which $\pi' < \pi$ and $\sigma' > \sigma$. The diffusion process is given by $(d, r, s, t) = (1, 0, 1, 10)$. Payoffs are given by $(a, w) = (0.9, 0.1)$. The degree distributions are given by the following. F puts equal mass on degrees k_1 and k_3 , while F' puts equal mass on degrees k_2 and k_3 . We set $(k_1, k_2, k_3) = (2, 3, 20)$.*

It is clear that F' FOSD F . Notice, first, that in order for the conclusion of the example to hold, it must be that \tilde{F}' does not FOSD \tilde{F} (by Proposition 2). To see that this is true, one can easily compute that $\tilde{F}'(k_2) = 3/23 > 1/11 = \tilde{F}(k_2)$. That is, the probability that a random neighbor is of the high degree, k_3 , is lower under \tilde{F}' because the low degree nodes have a slightly increased degree.

Second, it must be that $t + r$ is sufficiently large (by Proposition 3). Notice that the combined rate at which agents leave the infected state, through death and recovery, is indeed large (relative to the other rates) in this example. This is important, as otherwise almost all unprotected agents are infected in steady state, regardless of their degree.

Counter to the conventional intuition, an increase in connectivity reduces the equilibrium protection level. This result obtains as a direct consequence of the previous two observations. The fact that $t + r$ is large means that low-degree agents have substantially lower infection rates than higher degree agents. Under \tilde{F}' , the probability that a given neighbor has low degree is higher than under \tilde{F} . While the low degree nodes have slightly higher degree under F' compared to F , and therefore slightly higher infection rates, the dominating effect is that their infection rate is still substantially less than that of the high degree nodes. As a result, the net effect is that the incentive to protect goes down.

Finally, the effect of a denser network combined with a lower rate of protection implies higher total prevalence.

Example 1 highlights one of the contributions of this paper. In particular, if one is interested in understanding how equilibrium diffusion outcomes respond to changes in the social structure, then it is important to recognize that the answer to that question depends on the details of the diffusion process under consideration.

We now turn our attention to a change in the dispersion of connections, while keeping constant the aggregate level of connectivity. For this purpose we consider shifting the degree distribution in the sense of a mean-preserving spread. A distribution F' is a mean preserving spread of F if $E_{F'}[k] = E_F[k]$ and $\int_k F'(k)dk \geq \int_k F(k)dk$ for all k with strict inequality for some k .

Proposition 4 *Fix payoffs and a diffusion process. Let F' MPS F . Then $\theta' > \theta$ and for every k , $\rho'_k > \rho_k$. Furthermore, there exists a positive constant c such that if $t + r \in [0, c]$, then $\pi' < \pi$ and $\sigma' > \sigma$.*

Proof 4 *Assume for a contradiction that $\theta' \leq \theta$. We have*

$$\begin{aligned} a &= \int G(\theta, k) dF(k) \\ &< \int G(\theta, k) dF'(k) \\ &\leq \int G(\theta', k) dF'(k) \\ &= a \end{aligned}$$

where the first inequality follows because $G(\cdot)$ is convex in k and F' MPS F , while the second inequality follows because G is decreasing in θ and, by hypothesis, $\theta' \leq \theta$. Once the θ s are ordered the ordering of ρ_k follows immediately by noticing from expression 2 that ρ_k is increasing in θ . The second part of the statement can be proved using the same argument developed in the proof of Proposition 3 and therefore is omitted.

The intuition behind the results in Proposition 4 echoes the intuitions developed for Proposition 2 and 3. In particular, greater dispersion in the degree distribution implies that the infection rate among one's neighbors is higher. In general, the effects on the immunization rate and total prevalence are ambiguous, but can be ordered if the steady state outcomes of vulnerable agents are not highly sensitive to their degree.

4 Conditioning protection on level of exposure

The analysis above pertains to the case in which the decision to protect is taken before, or at least not conditional upon, the realization of the agent's degree. We now explore the case in which the decision to protect is taken as a function of one's degree.

A strategy profile π now specifies the probability $\pi_k \in [0, 1]$ that an agent with degree k invests in protection. The probability with which a randomly chosen neighbor of an agent is in state I becomes

$$\theta = \int (1 - \pi_k) \rho_k d\tilde{F}(k), \quad (5)$$

and we can write the steady state condition by substituting (2) in (5) as follows

$$1 = \int (1 - \pi_k) \frac{sk}{s\theta k + r + t} d\tilde{F}(k). \quad (6)$$

A strategy profile π is an equilibrium if for all k , (i) $\pi_k = 0$ implies that $V_k^S \geq V^P$, and (ii) $\pi_k = 1$ implies that $V_k^S \leq V^P$.

Proposition 5 *Consider the game in which protection decisions are conditioned on degree. There is an essentially unique equilibrium and the equilibrium is characterized by a threshold \bar{k} such that agents with degree $k > \bar{k}$ protect, agents with degree $k = \bar{k}$ mix, and agents with degree $k < \bar{k}$ remain vulnerable. The threshold \bar{k} solves*

$$G(\theta, \bar{k}) = a \quad (7)$$

Proof 5 *Recall that the expected payoff for a vulnerable agent with degree k is V_k^S , which is decreasing in k , whereas the expected payoff for an agent who decides to protect is V^P , which is independent of k . This implies that an equilibrium is characterized by a threshold as defined above. Therefore, the steady state condition (6) can be written as*

$$1 = \int_{k < \bar{k}} \frac{sk}{s\theta k + r + t} d\tilde{F}(k) + (1 - \pi_{\bar{k}}) \frac{s\bar{k}}{s\theta \bar{k} + r + t} P_{\tilde{F}}(k = \bar{k}). \quad (8)$$

Examination of equation (8) verifies that the RHS is decreasing in θ and increasing in \bar{k} . On the other hand, the equilibrium condition (7) implies that $\theta \bar{k}$ is equal to a given constant determined by parameters. Together, these two observations imply a unique equilibrium. The only exception is when \bar{k} falls in a gap of \tilde{F} . In this case the threshold is not unique, but the behavior of agents of every degree k is nevertheless uniquely determined.

As before, we are interested in how the equilibrium threshold, prevalence, and immunization rate respond to changes in the social structure. Our first observation is that the answers to these questions are, in general, ambiguous. To gain insight into why this is so, consider the examples in

Figure 1. The figure presents three comparisons between neighbor-degree distributions, where in each case \tilde{F}' FOSD \tilde{F} . The equilibrium threshold under F is denoted by \bar{k} , and the one under F' is denoted by \bar{k}' .

The argument works as follows. Assume initially that $\bar{k}' = \bar{k}$. Then, by equation (7), it must be that $\theta' = \theta$, and so, by equation (2), also that $\rho'_k = \rho_k$ for every k . In the first example, \tilde{F}' and \tilde{F} differ only below the threshold degree, where agents choose to remain vulnerable. Since \tilde{F}' shifts mass towards higher such degrees relative to \tilde{F} , and since ρ_k is increasing in k , it must be that, in fact, $\theta' > \theta$ implying that $\bar{k}' < \bar{k}$.

In the second example, \tilde{F}' and \tilde{F} differ only above the threshold \bar{k} , and so the hypothesis that $\bar{k}' = \bar{k}$ and $\theta' = \theta$ are consistent with the change in distribution, as it affects the relative proportions across agents who in any case find it optimal to protect.

In the third example, \tilde{F}' shifts mass away from agents just below the threshold – who have the highest prevalence in society – towards agents just above the threshold, who are protected. Thus under the hypothesis that $\bar{k}' = \bar{k}$, we must have that $\theta' < \theta$, which yields the conclusion that, in fact, $\bar{k}' > \bar{k}$.

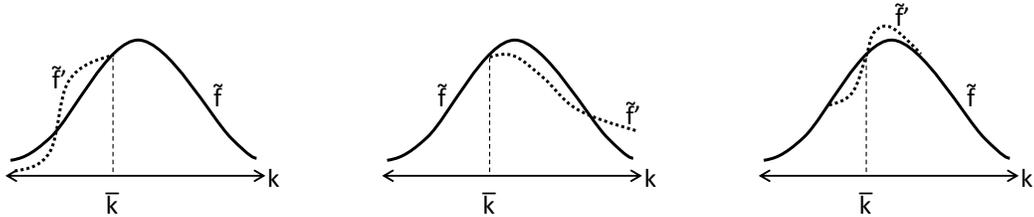


Figure 1: The effects of an FOSD shift are ambiguous. In all three cases \tilde{F}' FOSD \tilde{F} , but in the left panel, $\bar{k}' < \bar{k}$, in the center panel $\bar{k}' = \bar{k}$, and in the right panel $\bar{k}' > \bar{k}$.

We now show that, under appropriate restrictions on the diffusion process, changes in the neighbor-degree distribution do produce clear equilibrium effects. We present the following result. Let $\pi_{\bar{k}}$ and $\pi'_{\bar{k}'}$ be the respective mixing behavior at the threshold degree under F and F' , respectively.

Proposition 6 *Fix payoffs and a diffusion process. Let \tilde{F} and \tilde{F}' be distributions such that $\tilde{F}'(\bar{k}) <$*

$\tilde{F}(\bar{k})$. There exists a positive constant c such that if $t + r \in [0, c]$, then $\bar{k} \leq \bar{k}'$ and $\theta \geq \theta'$.

Proof 6 Suppose that that $\bar{k} > \bar{k}'$. Equilibrium condition (7) then implies that $\theta < \theta'$. Assume now that $t + r = 0$; steady state condition (8) and the assumption that $t = r = 0$ imply that $\theta = \tilde{F}(\bar{k}) - \pi_{\bar{k}} P_{\tilde{F}}(k = \bar{k})$. We then have that

$$\theta = \tilde{F}(\bar{k}) - \pi_{\bar{k}} P_{\tilde{F}}(k = \bar{k}) > \tilde{F}'(\bar{k}) - \pi_{\bar{k}} P_{\tilde{F}'}(k = \bar{k}) > \int_{k < \bar{k}} d\tilde{F}'(k) \geq \tilde{F}'(\bar{k}') - \pi_{\bar{k}'} P_{\tilde{F}'}(k = \bar{k}') = \theta'$$

where the first inequality follows because $\tilde{F}'(\bar{k}) < \tilde{F}(\bar{k})$, the second inequality is trivial, the third inequality follows because $\bar{k}' < \bar{k}$. We thus reach the desired contradiction. The inequalities are strict and do not depend on $t + r$. It is only the first and last equalities, determining the values of θ and θ' , respectively, that use $t + r = 0$. Those equalities come from the steady state condition (8) and so are continuous in $t + r$. Thus the above argument holds when $t + r > 0$ but sufficiently small.

We conclude that $\bar{k} \leq \bar{k}'$ and so, by (7), $\theta \geq \theta'$.

Notice that the hypothesis of Proposition 6 is satisfied when \tilde{F}' FOSD \tilde{F} , and that the result is strong in the sense that it requires the comparison of the degree distributions to hold not everywhere, but instead only at the equilibrium threshold under \tilde{F} . In fact the result also applies to mean preserving spread shifts. To see this, we say that \tilde{F}' is a strong MPS of \tilde{F} if \tilde{F}' MPS \tilde{F} and there exists a T so that $\tilde{F}'(k) > \tilde{F}(k)$ for all $k \leq T$ and $\tilde{F}'(k) \leq \tilde{F}(k)$ for all $k > T$. Now note that if \tilde{F}' is a strong MPS of \tilde{F} and $\bar{k} \leq T$ then $\tilde{F}(\bar{k}) < \tilde{F}'(\bar{k})$ and Proposition 6 implies that $\bar{k} \geq \bar{k}'$ and that $\theta \leq \theta'$.

4.1 The role of knowing one's degree

We are interested in comparing the equilibrium outcomes that obtain when agents make their protection decision before their degree is realized with the equilibrium outcomes that obtain when protection is conditioned on degree. As it turns out, the outcomes cannot generally be ordered. The key intuition is that there exist changes to the neighbor-degree distribution \tilde{F} , that do not affect outcomes in the ex post case (see the middle panel of Figure 1) but that do affect outcomes in the ex ante case.

Example 2 Set $k_1 = 2$ and $\tilde{f}(k_1) = 1/11$, and let there be a high degree k_2 such that $\tilde{f}(k_2) = 10/11$. Set the diffusion parameters to be $(d, r, s, t) = (1, 0, .2, 0)$ and the payoff parameters to be $(a, w) = (.9, 1)$.

Consider first the case in which agents make the protection decision conditional on their degree. In the case that k_2 is sufficiently high to induce protection, the equilibrium threshold is easily solved to be $\bar{k} = 6.875$, producing $\theta = 0.09$. Thus, as long as $k_2 > \bar{k}$ this is the equilibrium outcome.

The proportion of immunized agents is equal to $f(k_2)$. We compare two cases. In the first we have $k_2 = 7$ and in the second we have $k'_2 = 10$. These produce immunization rates of 20/27 and 2/3, respectively.

Consider now the case in which agents must decide to protect before their degree is realized. Setting $k_2 = 7$, one can easily solve for the equilibrium to find $\theta = 0.11$ and $\pi = 0.89$. Thus, when agents make the protection decision *ex ante*, more agents protect, but the chance of contacting an infected agent is higher. This is due to the fact that the set of protected agents is a mixture of high and low degree agents, and therefore the externalities generated by the protection are less effective. When we consider $k'_2 = 10$, we find $\theta = 0.09$ and $\pi = 0.91$. Intuitively, the *ex ante* chance of having a higher degree of 10 induces an even higher protection rate. In this case, the increase in protection lowers the rate of infection among neighbors below the rate that obtains when agents decide after they learn their degree.

The key effect in the example is the following. When agents protect conditional on their degree, there is a certain threshold above which protection is optimal. If an agent has a degree above this threshold, he protects, independently of whether his degree is slightly above or dramatically above the threshold. But in the *ex ante* case, as k_2 increases, the risk of becoming a high degree vulnerable agent becomes very costly and induces a corresponding increase in the protection rate. It is for this reason that the outcomes of the two scenarios cannot generally be ordered.

5 Conclusion

We emphasize that all of our results pertain to an equilibrium setting in which agents make optimal investments in protection, anticipating the consequences of their decisions on the steady state dynamics of the system. Our analysis is intentionally simple, so as to emphasize our finding that the contact pattern of a population interacts in interesting ways with the the nature of diffusion, given a contact pattern. Somewhat surprisingly, this paper presents the only formal study of this question of which we are aware. While the effects of certain changes in contact patterns are robust, others depend on the details of the diffusion process. This suggests that empirical analyses must pay careful attention to both aspects of diffusion dynamics. To increase the force of our results, we have allowed the diffusion process and contact pattern to be fairly general, but there are numerous directions to be pursued by further research. We find particularly interesting possibilities to include endogenizing the network structure, allowing for the possibility of treatment in addition to prevention, and the analysis of a profit seeking provider of protection.

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