Network Science **3** (3): 361–376, 2015. © Cambridge University Press 2015
doi:10.1017/nws.2015.24

Diffusion and protection across a random graph

ANDREA GALEOTTI
Department of Economics, University of Essex, Wivenhoe Park, Colchester, Essex CO3SQ, UK
(e-mail: agaleo@essex.ac.uk*)*

BRIAN W. ROGERS
Department of Economics, Washington University in St. Louis, St. Louis, MO 63130, USA
(e-mail: brogers@wustl.edu.*)*

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 the immunization rate, total prevalence and welfare, 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.[1] 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, on some dimensions, fairly general and it encompasses, as a special case, the linear Susceptible-Infected-Susceptible (SIS) framework.[2] Second, we assume that

[1] Anand et al. (2013) use a related analogy with epidemics in their discussion of market crashes. Naturally, though, viewing financial contagion through the lens of our model will abstract from certain relevant inter-banking effects, including the fact that links are also a source of mutual insurance. There is a very recent literature emerging in economics that studies these issues explicitly. On this, see Cabrales et al. (2014), Acemoglu et al. (2013), and Elliott et al. (2014).

[2] The 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 (1975) and Anderson & May (1992). For recent work in economics adopting these frameworks see, for example, Galeotti & Rogers (2013), Jackson & Rogers (2007), López-Pintado (2008), and Jackson & Yariv (2011).

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

agents are heterogenous in their intensity of interactions, which we call degree. The network of contacts is described by its degree distribution.

Third, we explicitly model the incentives of agents who are exposed to the diffusion process.[3] 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.[4] 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 flexible diffusion model, general degree distribution 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?

We build on a substantial literature that studies diffusion patterns across networks and which has made great progress in moving beyond the classical assumption of uniform mixing in the population. Pastor-Satorras & Vespignani (2001) is a seminal contribution to this field, which studies the case of scale-free networks and so, in particular, sheds light on the effects of heterogeneity of agents' degrees on diffusion outcomes. Pastor-Satorras & Vespignani (2001) also develop the strategy of looking at changes of the degree distribution in terms of stochastic dominance concepts, which has proved very valuable, and which we use, as described below. Subsequent work in this field includes Jackson & Rogers (2007), López-Pintado (2008) and Jackson & Yariv (2006). The model studied here is also related to our earlier work in Galeotti & Rogers (2013), where we first studied the equilibrium effects of protection in a diffusion context. Relative to that analysis, we substantially generalize the diffusion process and, more importantly, introduce the notion of a social network for the purpose of modeling contact patterns.[5] The latter innovation is crucial for all of the results and comparative statics we study here. Finally, the recent work of Goyal & Vigier (2015), while very different, is motivated by similar concerns as our paper. Their contagion model is static, as opposed to the diffusion model we

[3] In 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 & Rogers (2013), Geoffard & Philipson (1996), Geoffard & Philipson (1997), Bauch & Earn (2004), Boulier et al. (2007), Toxvaerd (2010a), and Toxvaerd (2010b).

[4] The method of analysis in this setting bears some similarity to the analysis of Galeotti et al. (2010). See footnote 11 for further details.

[5] Galeotti & Rogers (2013) assume all agents have the same degree, but that agents are separated into two subpopulations, such that within-group and across-group interactions can be varied.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

use, and their focus in on the joint decision between protection and intensity of interaction.

Our results characterize how equilibrium diffusion outcomes such as the immunization rate, the infection rate among non-immunized agents, total prevalence, and welfare 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 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 & 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 $F(k)$.[6] 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. This decision is taken only once and is effective for the duration of the agent's life. 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 z. Upon death, an agent is replaced with a newborn agent with the same degree. 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 $r \geqslant 0$ (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 $s \geqslant 0$.[7]

[6] We are assuming that the underlying network has no-degree correlation and zero clustering. These are strong assumptions that simply the analysis. We discuss them in more details in the concluding remarks.

[7] A natural extension is to assume that the death rate is higher for infected agents than for protected or susceptible agents. We have explored this possibility and found similar qualitative results.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

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

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

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.[8] The second term represents the outflow of infection due to recovery and death. Solving the above equations produces the steady state condition

$$\rho_k = \frac{s\theta k}{s\theta k + r + z}. \tag{2}$$

We can rewrite the steady state condition via substitution of Equation (2) into Equation (1) to obtain

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

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 $g > 0$. At birth, an agent chooses whether or not to protect so as to maximize his expected lifetime payoff.[9] We assume that, by choosing to protect, the agent commits to pay a cost c for every period. In return, protection guarantees not becoming infected. Hence, an agent who chooses to protect gets a flow utility of $a = 1 - c$.[10]

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

[8] The linear infection rate obtains for low infection rates, s, as is typically relevant, when an agent is infected independently from each of his neighbors. We discuss generalizations to this assumption in the Conclusion.

[9] Hence, when agents make the decision to protect or not, they anticipate, given the equilibrium decisions of others, the steady state dynamics, and maximize their utility according to such a belief. This is a strong assumption, but we believe it provides a benchmark that can be used to contrast other behavioral models.

[10] The assumption of commitment is strong but it simplifies greatly the analysis. We remark, however, that in the steady state, the commitment is credible. Hence, what we analyze is still an equilibrium outcome in the case with no commitment.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

being infected as V_k^I. We have that

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

The left-hand sides of the three preceding equations denote the rate at which utility is gained in each of the three states, respectively. The right-hand sides are composed of the flow utility in the state (the first terms) followed by the relevant rates of change multiplied by the corresponding difference in continuation values from the change. For example, the third equation computes the rate of utility to being infected as the flow payoff w, minus the loss of V_k^I from death, which occurs at rate z, plus the gain from recovering of $(V_k^S - V_k^I)$ which occurs at rate r. Solving the above system yields continuation values as a function of the exogenous parameters and the endogenous value of θ, as follows:

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

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

$$\int G(\theta,k)dF(k) = a. \tag{4}$$

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

We will also be interested in two additional measures of prevalence and in measures of welfare, which we now introduce. The average infection rate among all vulnerable agents is

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

and we denote the total level of prevalence in society by

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

Finally, we can think of two natural measures of welfare. One measure is the expected steady-state continuation value of a randomly selected agent at birth. This is equivalent to $E_F[V_k^S]$; since, in equilibrium, $E_F[V_k^S] = V_P = a/(d+t)$, this measure of welfare is independent of the details of the degree distribution. An alternative measure of welfare is the expected steady-state continuation value of a randomly selected agent. Formally

$$W = \pi V^P + (1-\pi)\int \left[\rho_k V_k^I + (1-\rho_k V_k^S\right] dF(k).$$

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

We will provide results on how changes in the degree distribution affect welfare according to expression W.

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 (g, r, s, z), 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

$$\theta = \left(\frac{1-a}{a-w}\right)\left(\frac{g+z+r}{ks}\right)$$
$$\pi = 1 - \frac{1}{ks}\left[\left(\frac{1-a}{a-w}\right)g + \left(\frac{1-w}{a-w}\right)(z+r)\right].$$

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 $\sigma' < \sigma$. Furthermore, the welfare in the regular network with degree k' is higher than in the regular network of degree k.

Proof
The first part of the proposition is immediate from the expressions above. We now show that the welfare increase with the degree of the network. In a regular graph with degree k, we can rewrite the welfare as

$$W_k = \pi V^P + (1-\pi)\left[\rho_k V_k^I + (1-\rho_k)V_k^S\right].$$

The first part of the proposition tells us that $\pi' > \pi$, $\rho_{k'} = \rho_k$. Moreover, by the expression of θ, we know that $k'\theta' = k\theta$, and therefore V_k^I and V_k^S do not change

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

when k moves to k'. Since $V^P > \rho_k V_k^I + (1-\rho_k)V_k^S$ (in fact for equilibrium, $V^P = V_k^S$ and clearly $V_k^S > V_k^I$), it follows that W_k is increasing in π. These observations and the observation that $\pi' > \pi$ implies that $W_{k'} > W_k$. □

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. To understand the result about welfare, note that the continuation utility of a randomly selected non-protected agent is constant in the degree of the network. This follows for two reasons. First, as we have shown, the infection rate among vulnerable agents is constant in the degree. Second, by inspecting the equilibrium expression of θ, it is readily seen that $\theta' k' = \theta k$, which implies that both the continuation payoff of susceptible and of infected agents do not change with the degree of the network. It then follows that to evaluate whether welfare increases with k is sufficient to verify that the protection rate increases with the degree.

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 $F'(k) \leqslant F(k)$ 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
Assume for a contradiction that $\theta' \geqslant \theta$, we have

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

where the first inequality is because G is decreasing in k and the second is because G is decreasing in θ. Once the θs 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

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

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.

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. Similar considerations imply that the effect on welfare of a FOSD shift in the degree distribution is ambiguous.

Notice the role that endogenous protection plays on the prevalence result. In particular, when the network becomes denser (in the sense of FOSD of both F and $\tilde{F}$), if protection was exogenous then prevalence would increase (see, e.g., Pastor-Satorras & Vespignani (2001) and Jackson & Rogers (2007)). When protection is endogenous, higher prevalence causes more agents to protect, which has positive externalities for vulnerable agents, such that their infection rates decrease.

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, total prevalence and welfare.

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 $\pi' > \pi$, $\sigma' < \sigma$ and the welfare associated to F' is higher than the welfare associated to F.

Proof

Assume for a contradiction that $\pi' \leqslant \pi$. Examining steady state condition (3), we observe that

$$1 = (1-\pi)\int \frac{sk}{s\theta k + r + z} d\tilde{F}(k) < (1-\pi')\int \frac{sk}{s\theta' k + r + z} d\tilde{F}(k).$$

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

$$\lim_{z+r\to 0}\int \frac{sk}{s\theta' k + r + z} d\tilde{F}(k) = \lim_{z+r\to 0}\int \frac{sk}{s\theta' k + r + z} d\tilde{F}'(k) = \frac{1}{\theta'}$$

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

$$1 < (1-\pi')\int \frac{sk}{s\theta' k + r + z} d\tilde{F}'(k)$$

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

and we reach a contradiction with the steady state condition. Next, note that for $z + r$ 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.

To conclude the proof, we consider welfare. We denote the welfare under distribution F by W_F. Equilibrium condition implies that $E_F[V_k^S] = V^P$, and therefore

$$\begin{aligned} W_F &= V^P - (1-\pi)E[\rho_k(V_k^S - V_k^I)] \\ &= V^P - (1-w)(1-\pi)E_F\left[\frac{\rho_k}{g+z+r+s\theta k}\right], \end{aligned}$$

where the second equality follows after replacing $V_k^S - V_k^I$. Recall that

$$\rho_k = \frac{s\theta k}{s\theta k + r + z} \text{ if, and only if, } s\theta k = \frac{\rho_k[r+z]}{(1-\rho_k)}$$

and therefore we can rewrite $\frac{\rho_k}{g+z+r+s\theta k} = \frac{\rho_k(1-\rho_k)}{g(1-\rho_k)+z+r}$, and the welfare as

$$W_F = V^P - (1-w)(1-\pi)E_F\left[\frac{\rho_k(1-\rho_k)}{g(1-\rho_k)+z+r}\right].$$

Note that

$$\lim_{r+z\to 0}(1-\pi)E_F\left[\frac{\rho_k(1-\rho_k)}{g(1-\rho_k)+z+r}\right] = \lim_{r+z\to 0}(1-\pi)E_F\left[\frac{\rho_k}{g}\right] = \frac{1}{g}\lim_{r+z\to 0}\theta.$$

Since, by Proposition 2, if F' FOSD F then $\theta' < \theta$, it follows that, at the limit of $z + r \to 0$ the welfare associate to F' is higher than the welfare associated to F. □

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 $z + r$ small, is important. It also allows us to illucidate 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.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

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 $\int_k F'(k)dk \geqslant \int_k F(k)dk$ 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 $z + r \in [0, c]$, then $\pi' < \pi$, $\sigma' > \sigma$, and welfare associated to F' is lower than the welfare associated to F.

Proof

Assume for a contradiction that $\theta' \leqslant \theta$, we have

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

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, $\theta' \leqslant \theta$. Once the θs 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. □

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

The intuition behind the results in Proposition 4 echoes the intuitions developed for Propositions 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. It is again interesting to contrast these findings to models without endogenous protection. For example, Jackson & Rogers (2007) find that the neighbor-infection rate increases following an MPS shift in an SIS model without protection.

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

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

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

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

A strategy profile π is an equilibrium if for all k, (i) $\pi_k = 0$ implies that $V_k^S \geqslant V^P$, and (ii) $\pi_k = 1$ implies that $V_k^S \leqslant V^P$.[11]

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

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

Proof
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

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

[11] The difference between the expected payoff of protecting and the expected payoff of non-protection, $V^P - V_k^S$ is increasing in the degree k, for any given strategy profile. In this sense, the expected payoff of this game exhibits degree complementarities as defined in Galeotti et al. (2010).

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

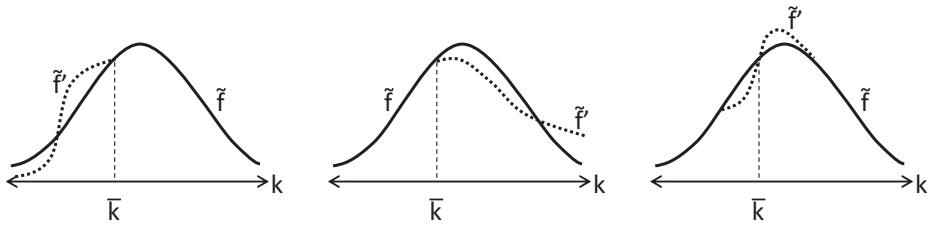


Fig. 1. The effects of an FOSD shift are ambiguous. In all three cases $\tilde{F}'$ FOSD $\tilde{F}$, but in the left panel, $\bar{k}' < \bar{k}$, in the center panel $\bar{k}' = \bar{k}$, and in the right panel $\bar{k}' > \bar{k}$.

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, $\bar{k}' > \bar{k}$.

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 $\bar{k} \leqslant \bar{k}'$ and $\theta \geqslant \theta'$.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

Proof

Suppose 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

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

The first and last equalities follow from the preceding observation about how θ (and θ') are determined when $t + r = 0$. The second equality simply expands $\tilde{F}(\bar{k})$ as the sum of (i) the mass that is less than or equal to $\bar{k}'$, (ii) the mass strictly between $\bar{k}'$ and $\bar{k}$, and (iii) the mass at $\bar{k}$. The strict inequality follows from (i) the assumption that $\tilde{F}'(\bar{k}') < \tilde{F}(\bar{k}')$, (ii) the fact that sum of the remaining terms on the left-hand side are clearly non-negative, and (iii) the fact that the last term on the right-hand side is clearly non-positive.

We thus reach the desired contradiction. The inequality is strict and does 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 $\bar{k} \leqslant \bar{k}'$ and so, by (7), $\theta \geqslant \theta'$.[12] □

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 $k \leqslant T$ and $\tilde{F}'(k) \leqslant \tilde{F}(k)$ for all $k > T$. Now note that if $\tilde{F}'$ is a strong MPS of $\tilde{F}$ and $\bar{k}' \geqslant T$ then $\tilde{F}'(\bar{k}') < \tilde{F}(\bar{k}')$ and Proposition 6 implies that $\bar{k} \leqslant \bar{k}'$ and that $\theta \geqslant \theta'$.

Returning to the question of welfare, notice that because one cannot generally pin down how the threshold degree responds to changes in the degree distribution, one cannot make general conclusions about welfare in this model. In fact, even under the conditions of Proposition 6, which give the problem more structure and, e.g., pin down the direction of change of the threshold degree, the welfare consequences of degree distribution shifts remain generally ambiguous. For example, observe that, following an FOSD shift in $\tilde{F}$, the threshold increases, but the proportion of agents who protect can go either up or down.

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

[12] We thank Nicolas Aguelakakis for pointing out an error in a previous proof.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

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 $(d, r, s, t) = (1, 0, 0.2, 0)$ and the payoff parameters to be $(a, w) = (0.9, 0.1)$.

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 nature of diffusion. 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 to incorporate a generalization of the SIS model and the contact pattern to incorporate general degree distributions. But there are numerous directions to be pursued by further research.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

The diffusion process can be greatly generalized. For example, it would be interesting to explore, instead of the linear adoption rule implicit in the SIS model we study, other possibilities such as concave or convex rules. This could capture, for instance, increasing or decreasing marginal effects of contacting additional infected agents. We refer to López-Pintado (2008) for an analysis of diffusion with concave or convex adoption rules. Although her model is in a non-strategic context, the results nonetheless are suggestive for a strategic model like the one we study here. One could also consider processes in which it is the fraction of infected neighbors that determines the adoption rate, rather than the number of infected neighbors. This is a reasonable assumption in the case of an infection if, for instance, having a higher degree implies spending less time with each contact.

Our analysis assumes that the underlying network has no degree correlation and has zero clustering. These are strong assumptions that allow analytical tractability, but are contrary to empirical facts of most social networks. Degree correlation could be introduced following, for example, Galeotti et al. (2010). Heuristically, we expect that introducing degree correlation would have similar effects to imposing a MPS of the degree distribution: connecting high degree nodes to other high degree nodes would increase their exposure, while connecting low degree nodes to other low degree nodes decrease their exposure. The introduction of clustering would tend to allow pockets of infection to persist more easily in an SIS framework. How clustering affects equilibrium and steady state is unclear and deserves a throughout analysis.

We have also assumed that interactions are undirected. Our results can, however, also be used to understand diffusion and endogenous protection in settings where interactions are directed, e.g., links between web pages and spread of computer viruses. In this case, the relevant distribution is the out-degree distribution of an agent and of a randomly selected agent's neighbor. Under the assumption that in-degrees and out-degrees are uncorrelated, these two distribution are the same. All the results that we have derived can then be applied to this setting under the interpretation that F is the out-degree distribution of a node and the restriction that F and $\tilde{F}$ are the same.

Finally, we find particularly interesting possibilities for future work 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.[13]

References

Acemoglu, D., Ozdaglar, A., & Alireza T.-S. (2013). Systemic risk and stability in financial networks. Technical Report, National Bureau of Economic Research.

Anand, K., Kirman, A., & Matteo M. (2013). Epidemics of rules, rational negligence and market crashes. *The European Journal of Finance*, **19**(5), 438–447.

Anderson, R. M., & May, R. M. (1992). *Infectious diseases of humans: dynamics and control*, Vol. 26, Wiley Online Library.

[13] We refer to Goyal & Vigier (2015) for a reduced form contagion model that allows for both an endogenous level of interaction and an endogenous protection level in the presence of infections, and we refer to Toxvaerd (2010a,b) for analyses that model prevention and treatment more generally.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press

Bailey, N. T. J. (1975). The mathematical theory of infectious diseases and its applications. 2nd edition. *The mathematical theory of infectious diseases and its applications*, (2nd ed.). p. 413. New York: Hanna Press.

Bauch, C. T., & Earn, D. J. D. (September 2004). Vaccination and the theory of games. *Proceedings of the National Academy of Sciences of the United States of America*, **101**(36), 13391–4.

Boulier, B. L., Datta, T. S., & Goldfarb, R. S. (2007). Vaccination externalities. *The BE Journal of Economic Analysis & Policy*, **7**(1), 23.

Brito, D. L., Sheshinski, E., & Intriligator, M. D. (1991). Externalities and compulsary vaccinations. *Journal of Public Economics*, **45**(1), 69–90.

Cabrales, A., Gottardi, P., & Vega-Redondo, F. (2014). Risk-sharing and contagion in networks. CESifo Working Paper Series No. 4715. Available at SSRN: http://ssrn.com/abstract=2425558.

Elliott, M., Golub, B., & Jackson, M. O. (2014). Financial networks and contagion. Available at *SSRN 2175056*.

Galeotti, A., Goyal, S., Jackson, M. O., Vega-Redondo, F., & Yariv, L. (2010). Network games. *The Review of Economic Studies*, **77**(1), 218–244.

Galeotti, A., & Rogers, B. W. (2013). Strategic immunization and group structure. *American Economic Journal: Microeconomics*, **2**(5), 1–32.

Geoffard, P.-Y., & Philipson, T. (1996). Rational epidemics and their public control. *International Economic Review*, **37**(3), 603–624.

Geoffard, P.-Y., & Philipson, T. (1997). Disease eradication: Private versus public vaccination. *The American Economic Review*, **87**(1), 222–230.

Goyal, S., & Vigier, A. (2015). Interaction, protection and epidemics. *Journal of Public Economics*, **125**, 64–69.

Jackson, M. O., & Rogers, B. W. (2007). Relating network structure to diffusion properties through stochastic dominance. *The BE Journal of Theoretical Economics*, **7**(1), 6.

Jackson, M. O., & Yariv, L. (2006). Diffusion on social networks. *Economie Publique*, **16**(1), 69–82.

Jackson, M. O., & Yariv, L. (2011). Diffusion, strategic interaction, & social structure. In *Handbook of Social Economics* (pp. 1–50). Amsterdam, Netherlands: Elsevier.

Kremer, M. (1996). Integrating behavioral choice into epidemiological models of AIDS. *The Quarterly Journal of Economics*, **111**(2), 549.

López-Pintado, D. (March 2008). Diffusion in complex social networks. *Games and Economic Behavior*, **62**(2), 573–590.

Pastor-Satorras, R., & Vespignani, A. (2001). Epidemic spreading in scale-free networks. *Physical Review Letters*, **86**(14), 3200.

Toxvaerd, F. (2010a). Infection, acquired immunity and externalities in treatment. CEPR Discussion Paper No. DP8111. Available at SSRN: http://ssrn.com/abstract=1714883.

Toxvaerd, F. (2010b). Recurrent infection and externalities in prevention. CEPR Discussion Paper No. DP8112. Available at SSRN: http://ssrn.com/abstract=1714884.

https://doi.org/10.1017/nws.2015.24 Published online by Cambridge University Press