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Fabrizio Adriani

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Suzanne Robey
Centre for Decision Research and Experimental Economics
School of Economics
University of Nottingham
University Park
Nottingham
NG7 2RD
Tel: +44 (0)115 95 14763
suzanne.robey@nottingham.ac.uk

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Social distance, speed of containment, and crowding in/out in a network model of contagion

Fabrizio Adriani*

May 19, 2020

Abstract

We study the effects of an intervention aimed at identifying and containing outbreaks in a network model of contagion where social distance is endogenous. The intervention induces a fall in the risk of infection, to which agents optimally respond by reducing social distance. If the intervention relies on infrequent or inaccurate testing, this crowding out effect may fully offset the intervention's direct effect, so that the risk of infection increases. In these circumstances, we show that "slow" interventions – which allow the outbreak to spread to immediate neighbors before being contained – may generate higher ex-ante welfare than "fast" ones and may even "crowd in" social distance. The theory thus identifies a trade off between (i) the swiftness of the intervention and (ii) the scope for crowding out. We show that the nature of this trade off crucially depends on the structure of the underlying social network and prevailing social norms.

JEL Codes: D85, I12, D62.
Keywords: Social Distance, Networks, Containment, Testing, Tracing, Contagion, Offsetting Behavior, Crowding Out.

*ULSB, University of Leicester, fa148@le.ac.uk.

1 Introduction

In the absence of pharmaceutical interventions, containment is the only tool available to policy makers seeking to mitigate the consequences of an epidemic. Within the context of the current COVID-19 pandemic, mass testing and contact tracing have been identified as the two main pillars of any policy seeking to reduce the social and economic impact of indiscriminate lockdowns and quarantines. Against this backdrop, two different sets of problems have been emphasized. First, some behavioral scientists have expressed concerns that mass testing might crowd out private efforts at social distancing.[1] A second concern shared by policy makers is that, given the contagiousness of diseases like COVID-19, interventions need to be fast in order to be effective. For example, experts have argued that traditional contact tracing methods risk falling "one step behind" the outbreak and technological solutions to increase the speed of intervention may be required.[2] In this paper, we argue that, although both concerns are theoretically plausible if taken in isolation, it is unlikely that they both apply at the same time. Indeed, the problem is reminiscent of trade-offs familiar to economists. For instance, the debate on financial crises regularly features the following policy dilemma: on the one hand, slow interventions are ex-post suboptimal since they allow contagion to spread and typically end up being expensive. On the other hand, fast interventions create incentives for excessive risk taking, thus making an intervention ex-ante more likely.

We start off by rehearsing the traditional crowding out argument. In particular, we consider the realistic case where interventions may fail to spot and contain all outbreaks – either because testing is not sufficiently frequent/widespread or because tests produce false negatives with some probability. We show that in this case fast interventions may reduce social distance, increase the expected number of infections and reduce ex-ante welfare. In contrast, under the same circumstances, "slow" interventions – which allow limited contagion before containing the outbreak – have a lower propensity to induce offsetting behavior than fast ones and could even "crowd in" social distancing. Third, and most important, we show that the relative advantage of a fast intervention over a slow one crucially depends on the structure of the underlying social network and on prevailing social norms.

We do this in a model where agents are part of an exogenously given social structure. This may reflect economic, social, or geographical constraints. Within this structure, agents choose which of their links they wish to keep active. Active links provide extra benefits (monetary or non-monetary) but increase the probability of infection. Hence, agents may want

[1] For instance, two leading British health psychologists wrote a letter highlighting similar concerns to England's Chief Medical Officer. *Source*: BBC Science Focus Magazine, April 1st, 2020.

[2] See for instance the white paper by Hart et al. (2020).

to render some of their links inactive to reduce their chances of infection. For instance, while my coauthors are clearly connected to me, during an epidemic some of them may decide to refrain from meeting me in person.

The intuition for the results is provided by the examples in Figures 1 and 2. In Figure 1, nodes A and B, and B and C, have active links. D is in C's social network but their link is currently inactive due to social distancing choices. Suppose that one node among them carries the disease with some probability. If C and D were to make their link active, they would provide a negative externality to A and B, since in the state of nature where D is the initial carrier, the disease could spread to them via C. Crucially, however, when deciding whether to activate his link with D or not, C does not internalize this externality.

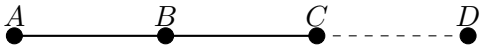


Figure 1: A chain of active links

Consider now an intervention that, with some probability, removes the initial carrier before he can infect others. For instance, individuals may be tested at intervals so that, whenever a positive case is detected, the outbreak is contained before it can spread further. If testing is infrequent or inaccurate, the intervention may reduce risk enough to induce C and D to activate their link but not enough to compensate A and B from the resulting negative externality. Total welfare may thus decrease.

Suppose in contrast that the intervention is too slow to stop the spread immediately and the initial carrier is allowed to infect adjacent nodes before the outbreak is spotted. This affects C's incentive to activate his link with D since, in the state where D is the initial carrier, C would still be infected. Indeed, from C's viewpoint, the incentive to activate a link with D is the same as with no intervention.[3] Yet, this type of intervention is still beneficial since, in the states where A or C is the carrier, one individual is spared infection.

The result, however, crucially depends on the underlying network structure. To see this, contrast the previous example with that in Figure 2. It is still true that, under a slow intervention, C's incentives are the same as with no intervention. However, one can easily verify that, keeping fixed the active links, under a slow intervention everyone is infected in the same states as with no intervention.

These examples show that it is crucial to investigate how the underlying

[3]The same is not true for D, though. Under the intervention, D has a lower probability to be indirectly infected by A and B, so that his incentive to activate the link with C increases, albeit not as much as under a fast intervention. Hence, the extent to which the intervention avoids crowding out depends on whether C or D is more "reluctant" to activate the link. We will come back to this when analyzing the star structure.

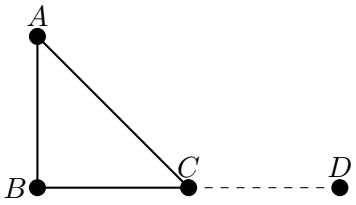


Figure 2: A clique of active links

network structure interacts with the intervention. In this paper, we analyze three very different underlying structures: the star, the cycle graph and the complete graph. In two of these (complete and star) average distances are short, while the other (cycle) has long average distances. One of these (star) is strongly hierarchical, while the others are non-hierarchical.

For relatively sparse networks, like the star and the cycle, we show that there exists a parameter space such that a slow intervention, by avoiding crowding out, increases welfare when a fast one would reduce it. Things are more complex in denser networks. Intuition suggests that a slow intervention is very costly if the network is dense because the outbreak could potentially infect a large number of nodes before being contained. Accordingly, we do find that in some equilibria a slow intervention is dominated by a fast one. However, there are also equilibria where a slow intervention may actually "crowd in" social distancing. To see how crowding in may come about, consider again Figure 2. In the absence of intervention, node A (or B) has a strong incentive to keep his link with C active because he knows that, if he were to cut it, he would still have a high chance of being infected by C via B. A slow intervention, however, reduces the risk of such an "indirect" infection while leaving the risk of a direct one unchanged. The intervention may thus convince A to increase his social distance by cutting the link with C (which in turn increases B's incentive to follow suit). More precisely, when the underlying network is the complete graph, we focus on equilibria featuring collections of isolated cliques. The size of cliques is not fully determined as cliques of different sizes may be stable under the same circumstances. We show that, when agents coordinate on the equilibrium featuring the smallest stable cliques, a fast intervention is always superior. However, if social norms induce agents to coordinate on the equilibrium with the largest stable cliques, a slow intervention that crowds in social distancing may be preferred.

The paper is organized as follows. After a short review of the literature, Section 2 introduces the model. Section 3 considers the benchmark case where agents' behavior is fixed and there is no crowding out. Section 4 provides some general insights, while Section 5 looks at specific network structures. Section 6 concludes. All proofs can be found in the Appendix.

1.1 Related literature

At least since Peltzman (1975), economists have been interested in whether behavioral responses to risk reducing interventions may partially or fully offset the intervention's direct effect.[4] Starting from Kremer (1996), some of the recent works specifically focus on infection prevention and mitigation. These include Geoffard and Philipsons (1996), Gersovits and Hammer (2004), Reluga (2010), Goyal and Vigiers (2015), Greenwood et al. (2019), Toxvaerd (2019), Rowthorn and Toxvaerd (2020). These models however tend to abstract from social structure.[5]

The economic literature on contagion in networks is sizeable and growing. Examples include Cabrales et al. (2012), Goyal and Vigiers (2014), Erol and Vohra (2015), Goyal et al. (2016), Acemoglu et al. (2016a), These works tend to mostly focus on network design and/or resilience and tend to abstract from specific policy interventions. A similar point broadly applies to the literature on financial contagion (see Glasserman and Young (2016) and Acemoglu et al. (2016b) for surveys).

Closer to our setting, Blume et al. (2013) analyze welfare in a static, reduced-form model of contagion in networks (see also Bougheas, 2018). Decentralized equilibria are suboptimal because of the externality outlined in the introduction: when forming new links, agents do not internalize the increase in the probability of infection of their neighbors. Using a variant of Blume et al. (2013), Talamas and Vohra (2020) study the welfare consequences of the introduction of an imperfect vaccine. Although their findings share similarities with our crowding out (but not crowding in) result, our focus is on the trade off between speed of containment and crowding out. This dimension of the problem is clearly absent in the case of a vaccine.

2 Model

The model is a variant of Blume et al. (2013). The main difference is that agents are only allowed to form contacts within an exogenously given underlying network structure. This reflects the different purposes of the two studies: While Blume et al. (2013) look at how contagion shapes network formation, we study how social distancing within per-existing social networks – which may be the result of economic, social, or geographical constraints – responds to containment policies.

There are $N \geq 2$ nodes. Nodes are organized on a graph (the *underlying network*). Nodes can choose to make each of their links active or to keep them inactive. Only links featured in the underlying network can be activated. Links are activated by mutual consent but can be made inactive

[4] See also Hoy and Polborn (2015).

[5] The current pandemic has also prompted a number of works on social distancing and interventions (e.g. Acemoglu et al. 2020; Alvarez et al. 2020; Makris, 2020; Galanis, 2020). These works, however, do not specifically focus on offsetting behavior.

(cut) unilaterally (Jackson and Wolinsky, 1996). An additional active link to a node that already has $n \geq 0$ active links yields a benefit $b\delta^{n+1}$ to the node, with $b > 0$ and $\delta \in (0, 1)$. This implies that there are decreasing returns from active links. These may reflect time constraints or economic considerations.[6] At time 0, there is a probability θN, $\theta \in (0, 1/N]$, that one of the N nodes (the *initial carrier*) may exogenously become infected. If a node becomes infected, the disease may spread through the active links. In particular, we assume that each node is immune with probability $\phi \in (0, 1)$. Immune nodes don't become infected and do not pass the disease to their neighbors. A node is therefore infected if and only if it is either the initial carrier or it is not immune and there is a path of non-immune nodes connecting it to the initial carrier. We assume nodes do not know ex-ante whether they are immune.[7] Finally, becoming infected entails a loss $L > 0$.

Timing is as follows

$t = 0$ Nodes decide which links they want to keep active.

$t = 1$ One node becomes the initial carrier with probability θN.

$t = 2$ All non-immune nodes with at least one active path of non-immune nodes from the initial carrier become infected

$t = 3$ Payoffs are realized

Intervention The above describes the model under no intervention (NI). Under an intervention, at the end of time 1 nodes are tested for infection. If there is an initial carrier, this is identified with probability $\lambda \in (0, 1)$. We refer to λ as the accuracy of the intervention. This reflects how systematically nodes are tested for infection or how accurate the test is. Under a fast intervention (FAST), an initial carrier that is identified is prevented from infecting any other node. Under a slow intervention (SLOW), an identified initial carrier infects his first degree neighbors (provided they are not immune) but the infection does not spread beyond that.

Discussion The model differs from Blume et al. (2013) in a few technical assumptions made for analytical convenience given the fact that we analyze several different network structures. In Blume et al. (2013) all benefits are wiped out if an individual is infected. Similar to Talamás and Vohra (2018), we assume that the benefits are kept but there are decreasing returns

[6] The assumption of an exponential decay for the benefits is stark and unnecessary for the results. That said, in order to have meaningful comparative statics when analyzing the complete graph in Section 5, we do need that beyond a certain number of active links the benefit of an additional link tends to go to zero quite sharply.

[7] This would be the case if immunity were determined by genetic traits that tend to vary in the population. Alternatively, the same would apply if, during previous outbreaks, a large number of infections were asymptomatic and/or tests were initially restricted to clinically severe cases, as in the current epidemic. For instance, using data from China and repatriation flights, Verity et al. (2020) estimate that a large proportion of COVID-19 cases were not identified.

from each additional active link. Although conceptually different, the two assumptions play an analogous role from an ex-ante viewpoint. Also, it turns out that the analysis of cycles and cliques is massively simplified by the assumption that only one individual is exogenously infected. This is similar to the "random adversary attack" in Goyal et al. (2016). Allowing multiple exogenous infections would not qualitatively change the results. Finally, we assume that a fraction of agents is immune. This is again close in spirit to Blume et al. (2013)'s assumption that the infection spreads through active links only with some positive probability, but not exactly identical. On the other hand, the presence of a fraction of immune agents is standard in models of epidemics (see e.g. Toxvaerd, 2020).

3 Preliminaries and benchmark

A node i with n active links becomes infected with probability

$$\pi(i) = \underbrace{\theta}_{\substack{\text{Pr i is the} \\ \text{carrier}}} + \underbrace{(1-\phi)}_{\substack{\text{Pr i not} \\ \text{immune}}} \underbrace{\left[1 - \prod_{j=1}^{n}(1-\pi(j))\right]}_{\substack{\text{at least 1 infected} \\ \text{neighor}}}, \tag{1}$$

where $\pi(j) \in [0,1]$ is neighbor j's probability of being infected, which in turn depends on j's active links. Some examples will be useful in what follows. Conditional on an outbreak hitting a node, a first degree neighbor of the initial carrier has a conditional probability $(1-\phi)$ to become infected. More generally, a node with only one path of active links of distance k from the initial carrier has conditional probability $(1-\phi)^k$ to be infected. A node with k paths from the initial carrier, all of distance 2, is infected if not immune and at least one node out of k is not immune. This occurs with (conditional) probability $(1-\phi)[1-\phi^k]$.

Node i's expected payoff is given by

$$\sum_{j=1}^{n} b\delta^j - \pi(i)L. \tag{2}$$

Under a fast intervention, infection occurs only if the initial carrier is not identified, so that the probability of "endogenous" infection (the second term in 1) is multiplied by $(1-\lambda)$. The same applies under a slow intervention, with the difference that the reduction $(1-\lambda)$ does not apply to the carrier's first degree neighbors.

Consider now ex-ante welfare when active links are given. Total welfare is the sum of expected payoffs, i.e. the total benefits from active links net of the expected loss from infection. Let W_{NI} denote ex-ante welfare in the absence of intervention and let W_I, $I = FAST, SLOW$, denote the same under an intervention. Then, it is clear that

Proposition 1. *In the absence of behavioral responses, if there is at least one active link, then* $W_{FAST} > W_{SLOW} \geq W_{NI}$.

In what follows, we will analyze situations under which the welfare ranking is changed when the active subgraph becomes endogenous. We say that *crowding out* (CO) occurs if nodes add links in response of the intervention (so that the intervention triggers a partially or totally offsetting effect on infection probabilities). We say that crowding out is *full* if the average infection probability increases as a result of the intervention. Clearly enough, an intervention can only reduce welfare if there is full crowding out. Full crowding out, however, is not sufficient for a reduction in welfare since the increase in infection probability might be compensated by the benefits from the additional active links. In general, we have

$$W_I - W_{NI} < 0 \Rightarrow \text{full CO} \Rightarrow \text{CO} \tag{3}$$

Note that, in practice, crowding out is a phenomenon of interest in its own right. For instance, there may be other externalities connected to the total number of infected agents not considered by the model (e.g. strain on the health system). That said, focusing on welfare has the advantage of making explicit the trade off between health and economic activity that is at the centre of the current debate. Finally, we say that the intervention *crowds in* social distance if the number of active links is reduced by the intervention.

4 Generic networks

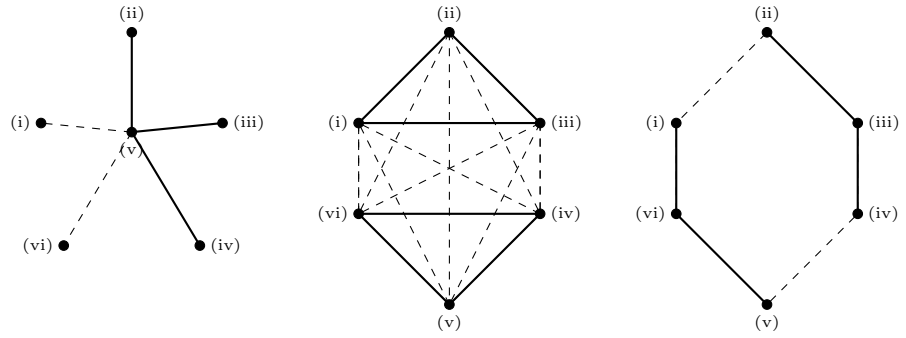
The equilibrium concept we use is pairwise stability (Jackson and Wolinsky, 1996): no node can raise his payoff by cutting one of his links and no two nodes can both benefit (at least one strictly) from activating a link. Before turning to specific network structures, we provide some general insights that help to illustrate the policy dilemma.

Proposition 2. *Fix an equilibrium under NI. Whenever a slow intervention induces two nodes to deviate and activate an additional link, a fast intervention also does. However, the reverse is not true.*

Slow interventions have a lower propensity to trigger crowding out. This is because slow interventions, while reducing the risk of indirect contagion, do not reduce the risk of contagion from a direct link. However, if crowding out does occur and the behavioral response is similar in terms of number of additional active links, then FAST is clearly superior as more nodes are protected. The result is however agnostic about the welfare ranking of the two types of intervention (and how they compare to no intervention). To answer these questions, we need to focus on specific network structures.

5 Specific network structures

In what follows, we will focus on a number of different underlying networks: the star, the cycle graph, and a complete graph as illustrated in Figure 3. The focus of our analysis will be the active graph arising in equilibrium (the solid lines in the Figure).



(a) star (b) complete graph (c) cycle graph

Figure 3: Solid lines represent active links. Dashed lines inactive links.

5.1 Stars

Consider the case where the underlying network is a star as illustrated in Figure 3 (a) and suppose that there are n active links (so that there are $n+1$ active nodes). Note that a peripheral node i is at distance 2 from every other peripheral node and at distance 1 from the hub. Under a fast intervention, his ex-ante probability of infection is

$$\underbrace{\theta}_{\substack{\text{Pr i is}\\ \text{carrier}}} + \underbrace{(1-\lambda)}_{\substack{\text{interv.}\\ \text{fails}}}\underbrace{(1-\phi)}_{\substack{i \text{ not}\\ \text{immune}}}[\underbrace{\theta}_{\substack{\text{hub is}\\ \text{carrier}}} + \underbrace{(1-\phi)}_{\substack{\text{hub not}\\ \text{immune}}}\underbrace{\theta(n-1)}_{\substack{\text{other } n-1\\ \text{carrier}}}] \tag{4}$$

On the other hand, the hub is at distance one from every other node. Hence, he has a higher infection probability,

$$\theta + (1-\lambda)(1-\phi)\theta n. \tag{5}$$

Under NI, the same probabilities are obtained by setting $\lambda = 0$ in (4) and (5). Under SLOW, the probability for a peripheral node is given by (4) plus $\lambda\theta(1-\phi)$, i.e. the probability that the hub is identified as a carrier and the peripheral node is not immune (in which case a peripheral node is infected under SLOW but not under FAST). Since the hub has distance one from all active nodes, his probability of infection under SLOW is the same

as under NI. Interestingly, a slow intervention gives some protection to the peripheral nodes but leaves the hub totally exposed.

Consider now pairwise stability.
(*Note: In the text, we only provide stability conditions for FAST. The conditions for NI are derived by setting* $\lambda = 0$. *Those for SLOW are provided in the Appendix*).

Lemma 1. *Under a fast intervention, a star component with* $n+1$ *active links is pairwise stable iff*

$$\begin{aligned}(1-\phi)\theta(1-\lambda)L\max\left\{\frac{1}{\delta^n},\frac{1+(1-\phi)(n-1)}{\delta}\right\} &\leq \\ b &\leq \\ (1-\phi)\theta(1-\lambda)L\max\left\{\frac{1}{\delta^{n+1}},\frac{1+(1-\phi)n}{\delta}\right\} &\end{aligned} \tag{6}$$

Under the same condition, a star component with $n+1$ *active links is the only pairwise stable component.*

Stability of an $n+1$-member active component requires that the benefits are large enough so that both the hub and the peripheral nodes do not want to deactivate their link – the first inequality in (6) – but not so large that the hub and an isolated node want to activate an additional link – the second inequality.

Lemma 1 points to the existence of two regimes. If δ is sufficiently close to one, the hub – who obtains a benefit $b\delta^{n+1}$ from an additional active link – wants to activate a new link whenever an isolated node wants to but not vice versa. This is because the hub has a higher probability of being infected than an isolated node. Hence, in this case, the size of the active component is determined by the propensity to join of the isolated nodes. We call this the *shy periphery* regime. In contrast, if δ is small or n is large, the returns from an additional links to the hub are small. As a result, the hub is more reluctant to activate links than the isolated nodes. We call this the *shy hub* regime.

How does crowding out occur? Note that in (6), both the upper and lower bounds on b are decreasing in λ and increasing in n. The same applies under a slow intervention. It follows that behavioral responses to any intervention necessarily weakly increase the number of active links. More generally,

Lemma 2. *Consider any intervention (fast or slow). Then, if there are* n *active links when the accuracy is* $\lambda' \geq 0$, *there are* $m \geq n$ *active links under* $\lambda'' > \lambda'$.

A star structure provides the natural setting to illustrate the externality that arises whenever new links become active. Consider what happens when a new link between the core and the periphery becomes active in a star with

n active nodes. For simplicity, let us abstract from intervention. The social benefit from the new link is given by the sum of the hub's benefit and the benefit to the added node, i.e. $b(\delta + \delta^n)$. The social cost is proportional to the increase in the probability of infection for the two nodes that choose to activate their link *plus* the increase for all other $n-1$ existing nodes with active links, i.e.

$$\underbrace{[(1-\phi)\theta + (1-\phi)^2\theta(n-1)]L}_{\Delta \text{ cost for new member}} \quad + \\ \underbrace{(1-\phi)\theta L}_{\Delta \text{ cost for hub}} . + \quad \underbrace{(1-\phi)^2\theta(n-1)L}_{\text{extern. on } n-1 \text{ members}} \tag{7}$$

Pairwise stability requires that $b\delta$ be weakly larger than the first term and that $b\delta^n$ be weakly larger than the second. However, the presence of the externality term implies that the change in social welfare may be negative. This is the mechanism through which an intervention, by increasing the number of active links, may lead to a welfare loss.

5.2 Cycle graphs

Assume that the underlying network is a cycle. Here, we want to derive conditions under which the equilibrium involves a collection of isolated chains, each with n active nodes, as in Figure 3 (c). For simplicity, we abstract from divisibility issues and assume that the total number of nodes N is always divisible as required. Also, in the case of cycles, assuming $\delta < 1$ brings complications without adding insights. Hence, we assume in the text that $\delta = 1$ and relegate the analysis of the $\delta < 1$ case to the Appendix.

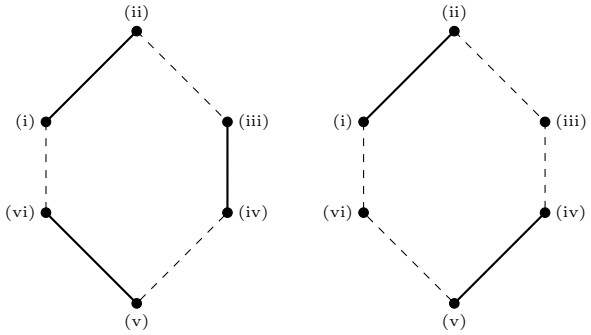
Lemma 3. *Under a fast intervention, an equilibrium with a collection of isolated chains each with n active members exists iff*

$$\theta(1-\lambda)\sum_{i=1}^{n-1}(1-\phi)^i L \leq b \leq \theta(1-\lambda)\sum_{i=1}^{n}(1-\phi)^i L. \tag{8}$$

Under the same condition, any equilibrium is characterized by the presence of n-member chains.

Note that the second statement need not be taken to say that any equilibrium must feature a collection of chains each with n nodes. Whenever n-member chains are stable, an equilibrium may alternate n-member chains and $m \leq n$ chains along the cycle (see e.g. Figure 4). For simplicity, we will focus on the equilibria with the highest and lowest total number of active links (*highest* and *lowest* equilibria for short). The highest equilibrium features a collection of chains each with n nodes. The lowest equilibrium has n-member chains interspersed by isolated agents. In the case depicted in Figure 4, one can verify that both the sequence of chains $\{2,2,2\}$ and

the sequence $\{2, 1, 2, 1\}$ are stable under the same condition (8). From the viewpoint of the isolated nodes in (b), the incentive to activate their link to a pair is the same as for the nodes in (a) to activate their link to another pair. Indeed, this perfect overlap in the conditions under which the two sequences are stable is the main advantage of setting $\delta = 1$. When $\delta < 1$, the isolated nodes have higher incentive to activate a link than other nodes. As discussed in the Appendix, this complicates the analysis of the lowest equilibrium.



(a) pairs (b) pairs/singletons

Figure 4: Possible equilibria with 2-member chains

Similar to the case of stars, note that the bounds on b in (8) are increasing in n and decreasing in λ. As a result, an analogous of Lemma 2 also applies here.

5.3 Complete graphs

Assume that the underlying network is the complete graph. Here, we want to derive conditions under which the equilibrium involves a collection of isolated cliques, each with n active nodes (as in Figure 3). Again, we abstract from divisibility issues.

Lemma 4. *Under a fast intervention, an equilibrium with a collection of isolated cliques with n active members exists iff*

$$\frac{\theta(1-\lambda)(1-\phi)\phi^{n-2}}{\delta^{n-1}}L \leq b \leq \frac{\theta(1-\lambda)(1-\phi)[1+(1-\phi)(n-1)]}{\delta^{n}}L \quad (9)$$

The first inequality in (9) is the condition under which no node wants to cut a link. The second inequality ensures that two nodes belonging to two separate cliques do not want to activate a link. Note that, different from the star structure (and the cycle with $\delta = 1$), the bounds on b do not perfectly partition the parameter space as we increase n, but tend to

overlap (i.e. the lower bound evaluated at $n = k + 1$ is lower than the upper bound evaluated at $n = k$). The result thus points to the existence of multiple equilibria: there exist parameter values where both n-member and m-member, $m > n$, cliques are stable. This indeterminacy is generated by the presence of strategic complementarity. Suppose that i has an active link to j. If j, but not i, were to activate his link with h, i's infection probability would increase but he would not obtain any benefit. Hence, if j activates a link with h, there is a strong incentive for i to follow suit. This complementarity is muted in the cycle and the star since, in both cases, i does not have any link with h to activate.

Note also that, if $\delta \geq \phi$, then the lower bound on b is non-increasing in n (while the upper bound is always increasing). In other words, the incentive to cut links is (weakly) smaller in larger cliques. As a result, the parameter space under which n-member cliques are stable is a subset of the space under which $n + 1$-member cliques are stable, which is in turn a subset of $n + 2$-member cliques and so on. In this case, it is not possible to have meaningful comparative statics on the intervention. In contrast, when $\delta < \phi$, it is worth cutting links if a clique becomes too large. Intuitively, a high rate of immune nodes reduces the chances of a path of non-immunes to the initial carrier, thus providing some protection to nodes not directly linked to the carrier. Since the benefits from active links increase at a rate lower than ϕ, the incentive to cut links increases as the clique becomes larger. Hence, in what follows we will assume

Assumption 1. $\delta < \phi$.

More generally, there may also be equilibria that do not involve collections of isolated cliques. To simplify the analysis, we will thus focus on two equilibria: the one featuring the *smallest* cliques and the equilibrium with the *largest* cliques. These equilibria are particularly representative since, within the class of regular equilibria where all nodes have the same number of active links, the smallest and largest equilibria are the equilibria with the lowest and highest number of active links, respectively.[8] Hence, they capture the maximum and minimum amounts of social distancing that can be achieved in a decentralized equilibrium. In the *smallest* equilibrium, cliques grow in size only when they are destabilized by the fact that two nodes from separate cliques have incentive to activate a new link (the second inequality in 9). In the *largest* equilibrium, n-member cliques are destabilized only when a node wants to cut a link (the first inequality in 9). This suggests that social norms will determine which equilibrium is more likely to emerge. For instance, if cutting links is not considered socially acceptable, we would expect an equilibrium characterized by cliques of larger size. This is because

[8]See Section B in the Appendix. It should be however noted that there may be regular equilibria with the same number of active links as the largest or the smallest that are not collections of cliques.

it is sufficient that a few agents fail to cut a link to a given agent to induce all other agents in the clique to keep their link active.

Different from stars and cycles, in the largest equilibrium the effect of a change in λ on the size of cliques is qualitatively different depending on whether the intervention is fast or slow.

Lemma 5.

1. *Under a fast intervention, both in the largest and the smallest equilibria, if under accuracy λ' each node in a clique has n active links, then under $\lambda'' > \lambda'$ each node in a clique has $m \geq n$ active links.*

2. *Under a slow intervention, if under accuracy λ' each node in a clique has n active links, then under $\lambda'' > \lambda'$ each node in a clique has $m \geq n$ active links in the smallest equilibrium and $m \leq n$ active links in the largest equilibrium.*

While in the smallest equilibrium interventions can only crowd out social distance, in the largest equilibrium social distance is crowded out under a fast intervention but may increase as a result of a slow intervention. The intuition for this "crowding in" result is as follows. In the largest equilibrium, most links are not cut simply because, even if one were to cut a link with a node, he would still have high chances to be infected by the same node via common neighbors. A slow intervention, however, changes this as it reduces the risk of indirect infection but not the direct risk. This makes nodes more willing to cut links, which results in smaller equilibrium cliques.

5.4 Welfare

We now consider whether crowding out under a fast intervention may lead to welfare losses.

Proposition 3. *There always exists λ^* such that a fast intervention increases welfare for all $\lambda > \lambda^*$.*

1. *If the underlying network is a star, then there exists an open set of parameter values such that $W_{NI} > W_{FAST}$.*

2. *If the underlying network is a cycle, then there exists an open set of parameter values such that $W_{NI} > W_{FAST}$ both in the highest and in the lowest equilibrium.*

3. *If the underlying network is complete and the equilibrium involves a collection of isolated cliques, then a fast intervention always increases welfare in the smallest equilibrium, i.e. $W_{FAST} > W_{NI}$ for all parameter values. However, the same does not hold for the largest equilibrium.*

The first statement is obvious: if the intervention is accurate enough, crowding out is never full, so that welfare can only increase. Stars and cycles are similar in the insights they provide. An inaccurate intervention may be enough to induce two nodes to activate an inactive link (with another chain of the cycle or with an isolated node), but not sufficient to compensate the other nodes in the active component of the resulting negative externality. Consider now point 3. In the smallest equilibrium, cliques tend to be too small relative to the social optimum.[9] In this case, even an intervention causing full crowding out always increases welfare. In contrast, in the largest equilibrium there are typically too many active links and the intervention will typically exacerbate a situation of excessive risk taking. Interestingly, this suggests that an intervention is more likely to be beneficial when social norms make agents more at ease with cutting links.[10]

5.5 Tracing fast and slow

We now look at how a slow intervention compares to a fast one depending on the structure of the underlying network.

Proposition 4.

1. *(Complete graphs) Whenever the equilibrium is characterized by isolated cliques, then $W_{FAST} > W_{SLOW}$ for all parameter values in the smallest equilibrium. However, in the largest equilibrium, $W_{SLOW} \geq W_{NI}$ for all parameter values (with strict inequality whenever crowding in occurs), so that $W_{SLOW} > W_{FAST}$ whenever $W_{NI} > W_{FAST}$.*

2. *(Stars) If there are at least 2 active links, in the parameter space such that the "shy hub" regime prevails, there is no crowding out under SLOW and $W_{SLOW} > W_{NI}$ (so that $W_{SLOW} > W_{FAST}$ whenever $W_{NI} > W_{FAST}$).*

3. *(Cycle graphs) If the longest chain has at least 2 active links, there exists an open set of parameters such that full crowding out occurs under FAST but not under SLOW and $W_{SLOW} > W_{NI} > W_{FAST}$.*

When the equilibrium involves a collection of cliques where all nodes are at distance one from each other, a slow intervention cannot improve upon NI in the absence of behavioral responses. In the smallest equilibrium, welfare tends to increase when crowding out occurs. As a result, a slow intervention is always dominated by a fast one. In contrast, in the largest

[9] Optimality requires that n-member cliques should acquire a new member whenever the benefit from an additional active link $b\delta^n$ is larger than $\theta(1-\phi)$. However, for $\lambda = 0$ (i.e. under NI), the second inequality in (9) implies a higher threshold.

[10] A question (not addressed here) is then whether the intervention may also affect social distancing norms. There are plausible reasons to argue both for norms becoming stricter or laxer as a result of the intervention.

equilibrium, cliques tend to be too large relative to the social optimum. A slow intervention that crowds in social distancing is thus always better than NI and may be superior to FAST.

For star structures, a slow intervention is always better than no intervention in the shy hub regime (and thus may improve upon FAST for parameter values). This is because the hub's incentives are unchanged relative to NI – so that there is no issue of crowding out. At the same time, a slow intervention can stop contagion from spreading beyond the hub whenever the outbreak hits a peripheral node. However, it is worth noting that while a slow intervention may be desirable ex-ante, it may become extremely damaging ex-post in the event that the outbreak starts at the core. A better approach would probably combine a slow intervention with high frequency testing of the hub, so that any outbreak starting at (or reaching) the core could be quickly contained. Things are different in the shy periphery regime. In that case, we show in the Appendix that, if δ is sufficiently close to one, a fast intervention dominates a slow one. The reason is that, if δ is close to one, any new active link in the shy periphery regime generates a large surplus for the hub. This effect compensates for the negative externality on existing peripheral members.

Finally, consider cycles. Relative to a fast intervention, a slow one reduces the incentive for the extreme nodes in the chain to destabilize the chain by activating a link with a neighbour who is part of another chain. At the same time, if chains have more than two active nodes, it provides some protection.

6 Concluding remarks

Our results suggest that whether we should be concerned about the speed of an intervention or its possible unintended consequences strongly depends on the underlying network structure and on prevailing social norms. In dense networks, a fast intervention is superior to a slow one if agents coordinate on small groupings. However, when social norms encourage coordination on larger cliques, a slow intervention may result in an ex-ante lower probability of infection as it "crowds in" social distance. In sparser networks, there is less room for crowding in. However, a slow intervention can be superior to a fast one by reducing the scope for crowding out. Finally, in hierarchical networks, a slow intervention fully prevents crowding out when the core is more reluctant to activate links than the periphery. This however comes at a price since, if the core is infected, a slow intervention cannot prevent mass contagion.

An obvious extension of the basic model would consist in introducing other-regarding motives. Note however that, depending on the precise form that social preferences take, these could potentially induce either an increase or a *reduction* in social distancing, as agents partially internalize

their neighbor's benefit from activating mutual links. Another key question is the extent to which interventions may affect social norms and thus "select" for high or low social distancing equilibria.

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A Appendix

A.1 Proof of Proposition 2

Proof. Let $\mathcal{G}$ denote the equilibrium subgraph formed by active links under NI and suppose that nodes i or j or both do not want to activate their link under NI, but both want under the intervention. WLOG, suppose that i does not want to activate the link to j. Let $n \geq 0$ be i's degree under NI. Let $\Delta\pi \in (0,1)$ denote the additional probability of infection for i if he were to activate the link under NI given $\mathcal{G}$. Then, under FAST, the same probability becomes $(1-\lambda)\Delta\pi$. Clearly enough, for i to be willing to activate the link under FAST but not under NI, it must be that

$$(1-\lambda)\Delta\pi < \frac{b\delta^{n+1}}{L} \leq \Delta\pi. \tag{10}$$

Under SLOW, i is infected in the same states as under FAST, except for the state where j is identified as the carrier, in which case if not immune i is infected if under SLOW but not under FAST. This state occurs with probability $\lambda\theta(1-\phi)$. Hence, for i to activate the link under SLOW,

$$(1-\lambda)\Delta\pi + \lambda\theta(1-\phi) < \frac{b\delta^{n+1}}{L} \leq \Delta\pi, \tag{11}$$

Clearly, (10) holds whenever (11) holds but the reverse is not true. □

A.2 Proof of Lemma 1

Proof. Assume that the underlying network is a star and consider an equilibrium characterized by a star subgraph with n active links (i.e. $n+1$ active nodes). For this to be stable, it is necessary and sufficient that

1. No node wants to cut any of their existing active links.
2. Both the hub and isolated nodes do not want to make a new link active.

Each peripheral node has an active link to the hub and is indirectly linked to the other $n-1$ peripheral nodes via the hub. They do not want to cut their active link if

$$\underbrace{-\theta}_{\substack{\text{Pr of inf. if}\\ \text{isolated}}} L \leq b\delta - \underbrace{[\theta + (1-\lambda)(1-\phi)\theta[1+(1-\phi)(n-1)]]}_{\substack{\text{Pr of inf. if}\\ \text{connected}}} L \tag{12}$$

The hub, who has n active links, does not want to cut any of these if

$$b\delta^n \geq (1-\lambda)(1-\phi)\theta L. \tag{13}$$

Rearranging (12) and (13), no node wants to cut an active link if the first inequality in (6) holds. Consider then the incentive to add an active link. A similar argument shows that, for this to be mutually optimal both for the hub and for an isolated node, the second inequality in (6) is necessary and sufficient. Note also that, in expression (6), the upper bound on b for $n=k$ coincides with the lower bound for $n=k+1$. As a result, so long as b is within the bounds, no other star subgraph of size different from n is stable. □

A.3 Proof of Lemma 2

Proof. This follows directly from the fact that both the upper and lower bounds on b in (6) are increasing in n and decreasing in λ. □

A.4 Proof of Lemma 3

Proof. Assume that the underlying network is a cycle and consider an equilibrium characterized by a collection of active chains each with $n-1$ links (i.e. n nodes). Let $k = 1, ...n$ denote a node's position in the chain moving clockwise. For n-member chains to be stable, it is necessary and sufficient that: 1) the two extreme nodes, $k = 1$ and $k = n$, do not want to cut their link (if they do not want to, all non-extreme nodes never want to cut a link for $\delta = 1$); 2) nodes in extreme positions $k = n$ and $k = 1$ in adjacent chains do not want to activate their mutual link. 1) requires that any extreme node i obtains a lower payoff as an isolated node than when having an active link to the chain, i.e.

$$\begin{aligned} -\theta L \leq b - [\underbrace{\theta}_{\substack{\text{Pr i carrier}}} + \underbrace{(1-\phi)\theta(1-\lambda)}_{\substack{\text{Pr i not imm.} \\ \text{i+1 carrier}}} + \underbrace{(1-\phi)^2\theta(1-\lambda)}_{\substack{\text{Pr i \& i+1 not imm.} \\ \text{i+2 carrier}}} + ... \\ ... + (1-\phi)^{n-1}\theta(1-\lambda)]L \quad \Leftrightarrow \\ -\theta L \leq b - \left(\theta + \theta(1-\lambda)\sum_{i=1}^{n-1}(1-\phi)^i\right)L \quad \Leftrightarrow \\ b \geq \theta(1-\lambda)\sum_{i=1}^{n-1}(1-\phi)^i L \end{aligned} \tag{14}$$

As for 2), if node i decides to activate his link with a neighbour who is part of an adjacent chain, his *additional* payoff (on top of what he already obtains from the chain he belongs to) is

$$\begin{aligned} b - [\underbrace{(1-\phi)\theta(1-\lambda)}_{\substack{\text{Pr i not imm.} \\ \text{i-1 carrier}}} + \underbrace{(1-\phi)^2\theta(1-\lambda)}_{\substack{\text{Pr i \& i-1 not imm.} \\ \text{i-2 carrier}}} + ... \\ ... + (1-\phi)^{n}\theta(1-\lambda)]L \quad = \\ b - \theta(1-\lambda)\sum_{i=1}^{n}(1-\phi)^i L \end{aligned} \tag{15}$$

This has to be negative, hence the second inequality in (8). Finally, note that under condition (8) there are no equilibria where the largest chain has $m < n$ members. This is implied by the first inequality in (8); for all $m < n$,

$$\begin{aligned} b - \theta(1-\lambda)\sum_{i=1}^{n-1}(1-\phi)^i L > 0 \quad \Rightarrow \\ b - \theta(1-\lambda)\sum_{i=1}^{m-1}(1-\phi)^i L > 0, \end{aligned} \tag{16}$$

so that any two nodes in extreme positions $k = 1$ and $k = m$ in chains with $m < n$ nodes want to deviate and form a link. Symmetrically, the

second inequality in (8) rules out any equilibrium where the largest chain is of size $m > n$, since an extreme node would want to cut his link. Hence, any equilibrium must have chains of size n. □

A.5 Proof of Lemma 4

Proof. For a collection of totally connected n member cliques to be stable, it must be that: 1) no node has incentive to cut a link and 2) no two agents in two different cliques have incentive to form a link. Consider 1). In a clique of n nodes, each node has $n-1$ active links. Suppose i cuts his $n-1$th link with a node j. i thus forgoes a benefit $b\delta^{n-1}$. His probability of infection is reduced by the fact that he cannot be directly infected by j but only indirectly through their common links. This means that, if j is infected, i is infected iff he is not immune and at least one of their common $n-2$ links are not immune. Hence, 1) requires

$$b\delta^{n-1} \geq (1-\lambda)\theta(1-\phi)L - (1-\lambda)\theta(1-\phi)\underbrace{(1-\phi^{n-2})}_{\substack{\text{Pr at least 1}\\ \text{common link}\\ \text{not immume}}}L, \tag{17}$$

where the first term on the RHS is the probability of being infected by j if the link is not cut and the second is the same if the link is cut. This implies the first inequality in (9). Consider now 2). If node i activates a new link with j, who must belong to a different clique, his probability of infection is affected both by the risk that j is the initial carrier and the probability that j has been infected by a member of his clique. Hence, two nodes i and j in different cliques (who already have $n-1$ links) have no incentive to activate their link if

$$b\delta^{n} \leq \underbrace{(1-\lambda)\theta(1-\phi)}_{\substack{j \text{ is the}\\ \text{carrier}}}L + \underbrace{(1-\lambda)\theta(1-\phi)^2(n-1))}_{\substack{j \text{ infected}\\ \text{by his links}}}L \tag{18}$$

This establishes the second inequality in (9). □

A.6 Stability conditions under SLOW

Consider a **star** subgraph with $n+1$ active nodes. If the hub is the initial carrier and is identified, all nodes that are not immune are infected under SLOW but not under FAST. Hence, the change in infection probability for a peripheral node when activating a new link is the same as under FAST plus the probability of this state, $\lambda\theta(1-\phi)$. The same applies when cutting a link. Since the hub has distance one from all active nodes, his incentives to activate or cut links are the same as under NI. The stability condition

(6) under SLOW thus becomes

$$\begin{aligned}(1-\phi)\theta L \max\left\{\frac{1}{\delta^n}, \frac{1+(1-\lambda)(1-\phi)(n-1)}{\delta}\right\} &\leq \\ b &\leq \\ (1-\phi)\theta L \max\left\{\frac{1}{\delta^{n+1}}.\frac{1+(1-\lambda)(1-\phi)n}{\delta}\right\} &\end{aligned} \tag{19}$$

Consider now **cycles**. For any of the extreme nodes in a chain with n members, both the additional probability of infection when activating a link and the reduction when cutting one are again increased by $\lambda\theta(1-\phi)$ relative to FAST. As already argued in the proof of Lemma 3, when $\delta = 1$, no other node wants to cut a link if the extreme nodes do not want to. As a result, condition (8) becomes

$$\theta\left[(1-\lambda)\sum_{i=1}^{n-1}(1-\phi)^i + \lambda(1-\phi)\right]L \leq b \leq \theta\left[(1-\lambda)\sum_{i=1}^{n}(1-\phi)^i + \lambda(1-\phi)\right]L. \tag{20}$$

Consider now **cliques** with n active nodes when the underlying network is the complete graph. Again, the change in infection probability for node i when activating a link to a node j in a different clique is the same under FAST plus the term $\lambda\theta(1-\phi)$ (reflecting the probability that j is identified as a carrier, in which case i is infected under SLOW but not under FAST). The reduction in the infection probability for i when cutting a link with a node j who is in i's clique is the same as under FAST plus again $\lambda\theta(1-\phi)$. This is because, under SLOW, if i does not cut the link, he is still infected by j when j is identified as carrier. Stability condition (9) accordingly becomes

$$\underbrace{\frac{\theta(1-\phi)[\phi^{n-2}(1-\lambda)+\lambda]}{\delta^{n-1}}L}_{\equiv\underline{b}(n-1;\phi,\lambda)} \leq b \leq \underbrace{\frac{\theta(1-\phi)[1+(1-\lambda)(1-\phi)(n-1)]}{\delta^{n}}L}_{\equiv\overline{b}(n;\phi,\lambda)}. \tag{21}$$

Similar to the case of a fast intervention, the smallest equilibrium features a collection of n-member cliques if

$$\overline{b}(n-1;\phi,\lambda) \leq b \leq \overline{b}(n;\phi,\lambda). \tag{22}$$

The largest equilibrium, features n-member cliques if

$$\underline{b}(n-1;\phi,\lambda) \leq b \leq \underline{b}(n;\phi,\lambda). \tag{23}$$

It is important to note that, different from (22), the upper and lower bounds on b in (23) are increasing in λ, while still increasing in n. This implies that, different from a fast intervention, under the largest equilibrium a slow intervention may actually reduce n relative to NI.

A.7 Proof of Lemma 5

Proof. Point 1. From (9), the stability condition for the smallest equilibrium requires

$$\overline{B}(n-1;\phi,\lambda) \leq b \leq \overline{B}(n;\phi,\lambda), \tag{24}$$

where

$$\overline{B}(n;\phi,\lambda) \equiv \frac{\theta(1-\phi)(1-\lambda)[1+(1-\phi)(n-1)]}{\delta^n} L. \tag{25}$$

The same for the largest equilibrium is

$$\underline{B}(n-1;\phi,\lambda) \leq b \leq \underline{B}(n;\phi,\lambda), \tag{26}$$

where

$$\underline{B}(n;\phi,\lambda) \equiv \frac{\theta(1-\lambda)(1-\phi)\phi^{n-1}}{\delta^n} L. \tag{27}$$

In both cases, both the upper and lower bounds for b are increasing in n and decreasing in λ, which implies that, keeping b fixed, an increase in λ must weakly increase n. Consider now point 2 and compare the above with the same derived for SLOW in (22) and (23). The bounds for b are still increasing in n and, in the smallest equilibrium, decreasing in λ. However, in the largest equilibrium, the bounds are increasing in λ, which implies that in the largest equilibrium the size of the clique must be weakly smaller under λ''. □

A.8 Proof of Proposition 3

Consider the first statement. Note that, since each node's number of active links weakly increases under FAST, the total benefits are always weakly larger under FAST than under NI. Hence, we just need to show that there exists $\lambda^* < 1$ such that the probability of infection is lower under FAST for all $\lambda > \lambda^*$. Suppose that, under NI, node i has n active links. Let $\tilde{\pi}_n \in (0,1)$ denote the probability of node i being infected by neighbors under NI. Clearly enough, if i does not activate any new link, the probability becomes $(1-\lambda)\tilde{\pi}_n < \tilde{\pi}_n$ under FAST, so that welfare must increase for all $\lambda > 0$. Suppose then that i activates $k \geq 1$ new links because of the intervention and let $\tilde{\pi}_{n+k}$ denote the probability of infection that i would face *without* intervention with these additional k links. Finally, let $\tilde{\pi}_M$ the probability of infection under no intervention if i activates all his available links. Then, $\tilde{\pi}_{n+k} \leq \tilde{\pi}_M$. Let $\lambda^* < 1$ be such that $(1-\lambda^*)\tilde{\pi}_M = \tilde{\pi}_n$. Then, for all $\lambda > \lambda^*$, we have $(1-\lambda)\tilde{\pi}_{n+k} < \tilde{\pi}_n$. Since this holds for all nodes i, the result is established.

We now prove points 1-3 starting from stars.

Stars

Proof. Suppose that, under NI, the stable component has n active links (i.e. $n+1$ active nodes). From Lemma 2, the number of active links can only increase under FAST. Hence, suppose that, under FAST, the component has m active links, with $m \geq n$. The total additional benefit generated by the additional links is

$$b \underbrace{\delta(m-n)}_{\substack{\text{benefit} \\ \text{to } n-m \text{ new} \\ \text{members}}} + \underbrace{b \sum_{i=n+1}^{m} \delta^i}_{\substack{\text{benefit} \\ \text{to hub}}}. \tag{28}$$

Compared to $n+1$ nodes under NI, the change in infection probability to the existing n peripheral nodes is given by the probability to be infected by an outbreak started by any of the new $m-n$ nodes net of the probability that *any* outbreak is contained by the intervention, i.e.

$$(1-\phi)\theta[(1-\phi)(m-n) - \lambda(1+(1-\phi)(m-1))] \tag{29}$$

The same for the hub is

$$(1-\phi)\theta[m-n-\lambda m], \tag{30}$$

and the additional probability for the $m-n$ joining nodes is

$$(1-\phi)\theta(1-\lambda)[1+(1-\phi)(m-1)]. \tag{31}$$

Putting all together, the additional social cost is

$$\begin{aligned} &\underbrace{(1-\phi)\theta[(1-\phi)(m-n) - \lambda(1+(1-\phi)(m-1))]nL}_{\text{externality on exist. } n \text{ members}} \quad + \\ &\underbrace{(1-\phi)\theta(1-\lambda)[1+(1-\phi)(m-1)](m-n)L}_{\Delta \text{ cost for new members}} \quad + \\ &\underbrace{(1-\phi)\theta[m-n-\lambda m]L}_{\Delta \text{ cost for hub}} \end{aligned} \tag{32}$$

We now show that there exists an open set of parameter values such that $W_{FAST} < W_{NI}$. For $\epsilon > 0$ suitably small, set δ such that

$$\delta^n = \frac{1}{1+(1-\phi)n}(1-\epsilon). \tag{33}$$

Since $1/\delta^{n+1} > (1+(1-\phi)n)/\delta$, from (6), stability of an active component with $n+1$ nodes requires

$$b \leq (1-\lambda)\frac{(1-\phi)\theta}{\delta^{n+1}}L. \tag{34}$$

Set also

$$b = \frac{(1-\phi)\theta}{\delta^{n+1}}L(1-\epsilon), \tag{35}$$

so that an active component with $n+1$ nodes is (just) stable under NI. Finally, let $\lambda = \ell\epsilon$, with $\ell > 1$, to ensure that

$$\frac{(1-\phi)\theta}{\delta^{n+1}} L(1-\ell\epsilon) < b, \tag{36}$$

so that the $n+1$ active component is unstable under FAST. Hence, for ϵ sufficiently small, the intervention triggers a transition from a component with $n+1$ active nodes to one with $n+2$. Using (33) and (35) and taking the limit for $\epsilon \to 0$, the change in total benefits is

$$\begin{aligned} b(\delta + \delta^{n+1}) = \frac{(1-\phi)\theta}{\delta^n} L + (1-\phi)\theta L \quad = \\ \left[2(1-\phi)\theta + (1-\phi)^2\theta n\right] L. \end{aligned} \tag{37}$$

Using (32) with $m = n+1$, the change in social costs for $\epsilon \to 0$ converges to

$$\begin{aligned} \underbrace{(1-\phi)^2\theta n L}_{\text{extern. on } n \text{ members}} + \underbrace{[(1-\phi)\theta + (1-\phi)^2\theta n] L}_{\Delta \text{ cost for new member}} + \\ \underbrace{(1-\phi)\theta L}_{\Delta \text{ cost for centre}}. \end{aligned} \tag{38}$$

The difference between additional social benefits and additional social costs in the limit is thus equal to the size of the externality,

$$-(1-\phi)^2\theta n L < 0 \tag{39}$$

As a result, by continuity, there exists ϵ^* such that $W_{FAST} < W_{NI}$ for all $\epsilon < \epsilon^*$. □

Cycle graphs

Proof. We now show that there exists an open set of parameter values such that $W_{FAST} < W_{NI}$. Suppose that, in the absence of intervention, b is close to the upper bound of the admissible value for an equilibrium with $n-1$ chains, $n > 2$. Formally, for a suitably small $\epsilon > 0$, let

$$b = \theta \sum_{i=1}^{n-1} (1-\phi)^i L(1-\epsilon) \tag{40}$$

and let $\lambda = \ell\epsilon$, with $\ell > 1$ so that

$$\theta \sum_{i=1}^{n-1} (1-\phi)^i L(1-\ell\epsilon) < \theta \sum_{i=1}^{n-1} (1-\phi)^i L(1-\epsilon) \tag{41}$$

As a result, the intervention destabilizes the sequence of $n-1$-member chains and induces a sequence of n-member chains.

Consider now infection probabilities. For convenience, the argument below will abstract from the nodes' identity. Instead, we will track the change of infection probability for any node in position $k = 1, ..., r$ in r-member chains (where r is the last node of the chain moving clockwise). Let $p(z) := \sum_{i=1}^{z}(1-\phi)^i$ for all $z \geq 1$ and $p(0) = 0$. Under FAST, a node in position $k = 1, .., n$ in a n chain has probability $\theta(1-\lambda)p(k-1)$ to catch an infection spreading clockwise and probability $\theta(1-\lambda)p(n-k)$ to catch one spreading anti-clockwise. His total infection probability is thus

$$\underbrace{\theta}_{\text{exog.}} + \theta(1-\lambda)[\underbrace{p(k-1)}_{\text{clockwise}} + \underbrace{p(n-k)}_{\text{anticlockw.}}] \tag{42}$$

We start with the highest equilibrium. In a sequence of chains of length $n-1$, there are in total $N/(n-1)$ nodes in each of positions $k = 1, ..., n-1$. Similarly, in a sequence of chains of length n, there are N/n nodes in each of positions $k = 1, ..., n$. As a result, the total change in expected infection costs between FAST and NI is

$$\begin{aligned}\Delta C := (1-\lambda)\theta\frac{N}{n}\sum_{k=1}^{n}\left(p(k-1)+p(n-k)\right)L \quad - \\ \theta\frac{N}{n-1}\sum_{k=1}^{n-1}\left(p(k-1)+p(n-1-k)\right)L\end{aligned} \tag{43}$$

The total number of active links in a sequence of n-member chains is

$$\underbrace{\frac{N}{n}}_{\text{number of } n \text{ chains}} \times \underbrace{(n-1)}_{\text{links per chain}} \tag{44}$$

and in a sequence of $n-1$-member chains,

$$\underbrace{\frac{N}{n-1}}_{\text{number of } n-1 \text{ chains}} \times \underbrace{(n-2)}_{\text{links per chain}} \tag{45}$$

As a result, the number of additional links generated by moving from $n-1$ to n sequences is

$$\frac{N}{n(n-1)} \tag{46}$$

Hence $W_{FAST} < W_{NI}$ iff

$$2b\frac{N}{n(n-1)} - \Delta C < 0. \tag{47}$$

Replacing b from (40), ΔC from (43), λ with $\ell\epsilon$, and taking the limit for

$\epsilon \to 0$, we obtain

$$W_{FAST} - W_{NI} = \frac{2N}{n(n-1)}\theta p(n-1)L - \theta\frac{N}{n}\sum_{k=1}^{n}\left(p(k-1)+p(n-k)\right)L \quad + \\ \theta\frac{N}{n-1}\sum_{k=1}^{n-1}\left(p(k-1)+p(n-1-k)\right)L. \quad (48)$$

The above is negative if

$$2p(n-1) - (n-1)\sum_{k=1}^{n}\left(p(k-1)+p(n-k)\right) \quad + \\ n\sum_{k=1}^{n-1}\left(p(k-1)+p(n-1-k)\right) < 0. \quad (49)$$

The LHS reduces to

$$2p(n-1) + \sum_{k=1}^{n}\left(p(k-1)+p(n-k)\right) - 2np(n-1), \quad (50)$$

or, using $\sum_{k=1}^{n}\left(p(k-1)+p(n-k)\right) = 2\sum_{k=1}^{n-1}p(k)$,

$$2\sum_{k=1}^{n-1}p(k) - 2(n-1)p(n-1). \quad (51)$$

Since $p(n-1) \geq p(k)$ for all $k \leq n-1$ (with strict inequality for all $k < n-1$), this must be negative. Hence, by continuity, there exists ϵ^* such that welfare decreases for all $\epsilon < \epsilon^*$.

Consider now the lowest equilibrium. Note that, since in both sequences $\{n, 1, ..., n, 1\}$ and $\{n-1, 1, ..., n-1, 1\}$ there is an isolated node for each chain, the total number of active links in an equilibrium with chains of length $n-1$ is $N(n-2)/n$ and the same for the equilibrium with length n chains is $N(n-1)/(n+1)$. Hence, the change in the number of links is $2N/n(n+1)$. Similarly, the number of nodes in position k is N/n for the sequence $\{n-1, 1, ..., n-1, 1\}$ and $N/(n+1)$ for the sequence $\{n, 1, ..., n, 1\}$. Noting that the infection probability of isolated nodes does not change, (48) becomes

$$\frac{4N}{n(n+1)}\theta p(n-1)L - \theta\frac{N}{n+1}\sum_{k=1}^{n}\left(p(k-1)+p(n-k)\right)L \quad + \\ \theta\frac{N}{n}\sum_{k=1}^{n-1}\left(p(k-1)+p(n-1-k)\right)L. \quad (52)$$

One can then verify that the analogous of (53) for the lowest equilibrium is

$$2\sum_{k=1}^{n-2}p(k) - 2(n-2)p(n-1), \quad (53)$$

which is again negative.

□

Complete graphs

Proof. From Lemma 4, the smallest equilibrium is characterized by n-member cliques under FAST if

$$\overline{B}(n-1;\phi,\lambda) \leq b \leq \overline{B}(n;\phi,\lambda), \tag{54}$$

where

$$\overline{B}(n;\phi,\lambda) \equiv \frac{\theta(1-\phi)(1-\lambda)[1+(1-\phi)(n-1)]}{\delta^n} L. \tag{55}$$

The same for the largest equilibrium is

$$\underline{B}(n-1;\phi,\lambda) \leq b \leq \underline{B}(n;\phi,\lambda), \tag{56}$$

where

$$\underline{B}(n;\phi,\lambda) \equiv \frac{\theta(1-\lambda)(1-\phi)\phi^{n-1}}{\delta^n} L. \tag{57}$$

Assume that, under NI, the equilibrium has n-member cliques. From Lemma 5, a fast intervention will change the size of cliques from n to $m \geq n$. If $m = n$, then it is straightforward to verify that welfare increases. Suppose then that $m > n$. Let us focus first on the smallest equilibrium. When moving from a n-member clique to a m-member one, each nodes adds $m - n$ new links. The change in benefits for any node is thus

$$b \sum_{i=n}^{m-1} \delta^i \geq \frac{\theta(1-\lambda)(1-\phi)}{\delta^{m-1}} L \sum_{i=n}^{m-1} \delta^i \quad \geq$$
$$\theta(1-\lambda)(1-\phi)(m-n)L \tag{58}$$

where the first inequality comes from $b \geq \overline{B}(m-1;\phi,\lambda)$, i.e. stability condition (54) applied to m-member cliques. The second inequality follows from $\delta \leq 1$. Consider now expected infection costs. Compared with an n-member clique under NI, the additional $m - n$ active links increase each node's probability of infection by $(1-\lambda)\theta(1-\phi)(m-n)$. To this, however, we need to subtract the reduction in the probability of being infected by pre-existing links brought about by the intervention. Relative to NI, the additional expected cost of FAST is thus

$$\underbrace{\theta(1-\lambda)(1-\phi)(m-n)L}_{\substack{\Delta \text{ cost of} \\ \text{additional links}}} - \underbrace{\lambda\theta(1-\phi)(n-1)L}_{\substack{\Delta \text{ Pr of inf} \\ \text{from pre-exist. links} \\ \text{due to intervention}}} \tag{59}$$

Since the increase in social benefits is clearly larger than the increase in social costs, $W_{FAST} > W_{NI}$.

We now show that, in the largest equilibrium, there exists an open set of parameter values such that $W_{FAST} < W_{NI}$. For $\epsilon > 0$ but sufficiently small, set

$$b = \frac{\theta(1-\phi)\phi^{n-1}}{\delta^n}(1-\epsilon)L \tag{60}$$

so that a clique with n active members is stable under NI. Let also $\lambda = \ell\epsilon$, with $\ell > 1$, so that

$$\frac{\theta(1-\phi)\phi^{n-1}}{\delta^n}(1-\epsilon)L > \frac{\theta(1-\phi)\phi^{n-1}}{\delta^n}(1-\ell\epsilon)L. \tag{61}$$

This ensures that the intervention makes n-member cliques unstable, so that, for ϵ small, the number of nodes in each clique must increase by one. This implies that, relative to NI, each node will have an additional active link. Using (60), the additional benefit for each node, $b\delta^n$, becomes

$$\theta(1-\ell\epsilon)(1-\phi)\phi^{n-1}(1-\epsilon)L \tag{62}$$

while, setting $m = n+1$ in (59), the extra cost is

$$\theta(1-\ell\epsilon)(1-\phi)L - \ell\epsilon\theta(1-\phi)(n-1)L. \tag{63}$$

Finally, it is straightforward to verify that the difference turns negative for $\epsilon \to 0$. Hence, by continuity, there exists ϵ^* such that $W_{FAST} < W_{NI}$ for all $\epsilon < \epsilon^*$.

□

A.9 Proof of Proposition 4

Proof. **Point 1.** We now show that, in the smallest equilibrium, $W_{FAST} > W_{SLOW}$. If there is no CO, then this is trivially true. Suppose then that, under NI, the smallest equilibrium has stable cliques of n members; under SLOW, the equilibrium has stable cliques of size $f \geq n$, and, under FAST, of size $s \geq n$. Note that, from (9) and (21), the smallest equilibrium is characterized by k-member cliques if

$$\begin{aligned} \overline{B}(k-1;\lambda,\phi) \leq b \leq \overline{B}(k;\lambda,\phi) \quad &\text{(FAST)} \\ \overline{b}(k-1;\lambda,\phi) \leq b \leq \overline{b}(k;\lambda,\phi) \quad &\text{(SLOW)}, \end{aligned} \tag{64}$$

where, under FAST,

$$\overline{B}(k;\lambda,\phi) = \frac{\theta(1-\phi)(1-\lambda)[1+(1-\phi)(k-1)]}{\delta^k}L \tag{65}$$

and, under SLOW,

$$\overline{b}(k;\lambda,\phi) = \frac{\theta(1-\phi)[1+(1-\lambda)(1-\phi)(k-1)]}{\delta^k}L. \tag{66}$$

Note that, for all k, $\overline{b} < \overline{B}$, which implies that cliques must be weakly larger under FAST than under SLOW, i.e. $f \geq s$. Relative to NI, the change in welfare for each node is

$$W_{SLOW} - W_{NI} = \sum_{i=n}^{s-1} \left(b\delta^i - \theta(1-\phi)L \right) \quad = \\ b\sum_{i=n}^{s-1} \delta^i - \theta(1-\phi)(s-n)L, \tag{67}$$

under SLOW and

$$W_{FAST} - W_{NI} = b\sum_{i=n}^{f-1} \delta^i - (1-\lambda)\theta(1-\phi)(f-n)L + \underbrace{\lambda\theta(1-\phi)(n-1)L}_{\substack{\text{Lower Pr of inf} \\ \text{on pre-exist. links}}}, \tag{68}$$

under FAST. Hence,

$$W_{FAST} - W_{SLOW} = b\sum_{i=s}^{f-1} \delta^i - \theta(1-\phi)[(1-\lambda)f - s + \lambda]L. \tag{69}$$

However, from (64), the smallest equilibrium can have stable f-member cliques under FAST only if

$$b\delta^{f-1} \geq (1-\lambda)\theta(1-\phi)[1 + (1-\phi)(f-2)]L. \tag{70}$$

Using this and $\delta \leq 1$, we obtain

$$\begin{aligned} W_{FAST} - W_{SLOW} \geq (1-\lambda)\left[\theta(1-\phi)[1+(1-\phi)(f-2)]\,(f-s)\right. & \quad - \\ \theta(1-\phi)\left[(1-\lambda)f - s + \lambda\right]L & \quad = \\ \theta(1-\phi)[\lambda(s-1) + (1-\lambda)(1-\phi)(f-s)(f-2)]L & \quad > \quad 0. \end{aligned} \tag{71}$$

We now show that in the largest equilibrium $W_{SLOW} \geq W_{NI}$ with strict inequality when crowding in occurs. From Lemma 5, compared to NI, the size of cliques under SLOW is weakly lower. Suppose then that there are n-member cliques under NI and s-member cliques under SLOW, with $n \geq s$. For $n = s$, $W_{SLOW} = W_{NI}$ since every node is at distance one from every other node in the clique. For $n > s$, the change in welfare is

$$W_{SLOW} - W_{NI} = \theta(1-\phi)(n-s)L - \sum_{i=s}^{n-1} b\delta^i. \tag{72}$$

Using (23), the largest equilibrium has stable s-member cliques under SLOW only if

$$b\sum_{i=n}^{s-1} \delta^i \leq \theta(1-\phi)[\phi^{s-1}(1-\lambda) + \lambda]L\frac{\sum_{i=s}^{n-1}\delta^i}{\delta^s} \quad < \\ \theta(1-\phi)[\phi^{s-1}(1-\lambda) + \lambda](n-s)L < \theta(1-\phi)(n-s)L, \tag{73}$$

which implies that $W_{SLOW} > W_{NI}$.

Point 2. This follows from the observation that, under SLOW, the hub is infected in all states under which he is infected under NI. Hence, a slow intervention increases the propensity to join of the periphery but not that of the hub. As already argued in Section A.6, the stability condition (6) for an active component with n members under FAST, under SLOW becomes

$$\begin{aligned}(1-\phi)\theta L \max\left\{\frac{1}{\delta^n}, \frac{1+(1-\lambda)(1-\phi)(n-1)}{\delta}\right\} &\leq \\ b &\leq \\ (1-\phi)\theta L \max\left\{\frac{1}{\delta^{n+1}}, \frac{1+(1-\lambda)(1-\phi)n}{\delta}\right\} &\end{aligned} \tag{74}$$

If $\delta^{-(n-1)} > 1 + (1-\phi)(n-1)$, a slow intervention never leads to CO. However, if $n > 2$, $W_{SLOW} > W_{NI}$ since, in all states where a peripheral node is identified as the carrier, the infection cannot spread beyond the hub and all other peripheral nodes are spared.

Point 3. The argument for point 3 is a minor variation on the one already used for point 2 of Proposition 3. To avoid duplications, we refer the reader to that proof. In particular, instead of (40), now set b such that

$$(1-\ell\epsilon)\theta\sum_{i=1}^{n-1}(1-\phi)^i L < b < \theta\left[(1-\ell\epsilon)\sum_{i=1}^{n-1}(1-\phi)^i + \ell\epsilon(1-\phi)\right]L \tag{75}$$

This ensures that: 1) Under NI and under SLOW the size of stable chains is $n-1$, since $b/L < \theta(1-\lambda)\sum_{i=1}^{n-1}(1-\phi)^i + \lambda\theta(1-\phi) < \theta\sum_{i=1}^{n-1}(1-\phi)^i$; 2) under FAST the size is n since $b/L > (1-\lambda)\theta\sum_{i=1}^{n-1}(1-\phi)^i$. One can then use an argument analogous to the proof of point 2 in Proposition 3 to show that there exsits ϵ^* such that $W_{NI} > W_{FAST}$ for all $\epsilon < \epsilon^*$ both in the highest and the lowest equilibrium. All that remains to show is that, for all ϵ positive but sufficiently small, $W_{SLOW} > W_{NI}$. Note that, since chains have size $n-1$ both under NI and under SLOW, the benefits are identical. Hence, it is enough to compare infection probabilities. Let $\hat{\pi}_k \in (0,1)$ denote the endogenous infection probability under NI for a node in position $k = 1, ..., n-1$ in a chain with $n-1$ nodes. Consider first $k \notin \{1, n-1\}$, (i.e. nodes with two active links). The same probability under SLOW is

$$(1-\lambda)\hat{\pi}_k + 2\lambda\theta(1-\phi) = (1-\ell\epsilon)\hat{\pi}_k + 2\ell\epsilon\theta(1-\phi). \tag{76}$$

Note that, for any node with two active links, $\hat{\pi}_k \geq 2\theta(1-\phi)$ (with strict inequality when there are more than 3 nodes in a chain). In turn, this implies that the chances of infection are weakly lower under SLOW for all $\epsilon > 0$. Consider now $k = 1$ or $k = n-1$. These nodes have only one active link. Their probability of infection under SLOW is

$$(1-\ell\epsilon)\hat{\pi}_k + \ell\epsilon\theta(1-\phi). \tag{77}$$

Since by assumption there are at least two active links (i.e. at least 3 active nodes) in the chain, an extreme node can be infected with positive probability even in states when his neighbor is not the initial carrier. Hence, it must be that, for $k \in \{1, n-1\}$, $\hat{\pi}_k > \theta(1-\phi)$. This is sufficient for $W_{SLOW} > W_{NI}$. As a result, for all $\epsilon > 0$ sufficiently small, both $W_{NI} > W_{FAST}$ and $W_{SLOW} > W_{NI}$, which establishes the result. □

B Further claims

Cycles with $\delta < 1$

There are two main differences with the $\delta = 1$ case. First, it is not anymore true that if an extreme node does not want to cut any link then the non-extreme nodes never want to: they may if δ is sufficiently small. Second, $\delta < 1$ may create a wedge between the conditions under which the sequence of chains $\{n, n, ..., n\}$ and the sequence $\{n, m, n, m, ..., n, m\}$, $m \leq n$ can arise, while, with $\delta = 1$, the stability condition is identical for both sequences.

A sequence of isolated agents is stable iff $b/L \leq (1-\lambda)\theta(1-\phi)/\delta$. Let $p(z) = \sum_{i=1}^{z}(1-\phi)^i$ for all $z \geq 1$ and $p(0) = 0$. Remember that a node in position $k = 1, ...n$ in a n-member chain has probability $\theta(1-\lambda)p(k-1)$ of catching an infection spreading clockwise and probability $\theta(1-\lambda)p(n-k)$ of catching one spreading anticlockwise. Consider any sequence $\{n, m, n, m..., n, m\}$ with $n \geq 2$ and $2 \leq m \leq n$. This is stable iff

$$\theta(1-\lambda)\max\left\{\frac{p(n-1)}{\delta}, \frac{p(n-2)}{\delta^2}\right\} \leq \frac{b}{L} \leq \theta(1-\lambda)\frac{p(n)}{\delta^2} \tag{78}$$

The first inequality says that neither the extreme nodes in the n chain (who have one active link), nor their neighbors in the same chain (who have two) want to cut a link. If these do not want to cut a link, then no other node wants to. The second says that the extreme node in a length m chain does not want to activate a link (if he wants to, his neighbor who belongs to a length n chain always wants to). Note, however, that for $m = 1$ the same condition becomes.

$$\theta(1-\lambda)\max\left\{\frac{p(n-1)}{\delta}, \frac{p(n-2)}{\delta^2}\right\} \leq \frac{b}{L} \leq \theta(1-\lambda)\max\left\{\frac{p(n)}{\delta}, \frac{p(1)}{\delta^2}\right\} \tag{79}$$

This is because, in the second inequality, the new link is with an isolated node, and therefore the benefit for that node is $b\delta$ rather than $b\delta^2$. Note also that

$$\max\left\{\frac{p(n)}{\delta}, \frac{p(n-1)}{\delta^2}\right\} \geq \max\left\{\frac{p(n)}{\delta}, \frac{p(1)}{\delta^2}\right\} \tag{80}$$

with strict inequality if $n > 2$ and δ is sufficiently small. This implies that, for δ sufficiently small, there exist ranges for b where no sequence of the type $\{n, 1, n, 1, ..., n, 1\}$ can emerge. Intuitively, there are parameter values

such that an extreme node in the n chain and the isolated node want to activate a link but, at the same time, in a $n+1$ chain a non-extreme node would want to cut a link, so that neither the sequence $\{n, 1, ..., n, 1\}$ nor the sequence $\{n+1, 1, ..., n+1, 1\}$ are stable. This means that the equilibrium with the lowest number of active links sometimes features the sequence $\{n, 1, ..., n, 1\}$ and sometimes the sequence $\{n, 2, ..., n, 2\}$. To avoid further complications, we only focus on equilibria with equal size chains, i.e. the sequence $\{n, n, ..., n\}$. Note that, even within this class, multiple equilibria arise. This is because, for $\delta < 1$,

$$\max\left\{\frac{p(n)}{\delta}, \frac{p(n-1)}{\delta^2}\right\} < \frac{p(n)}{\delta^2} \tag{81}$$

As a result, in (78), the lower bound for $n = k+1$ is larger than the upper bound for $n = k$. Hence, there exist parameters such that both the sequence $\{n, n, ..., n\}$ and the sequence $\{n+1, n+1, ..., n+1, \}$ are stable. Intuitively, there exist ranges of values for b such that no nodes in n chains want to activate a new link but, at the same time, no nodes in $n+1$ chains want to cut a link. We start from the equilibrium with the highest number of active links. Similar to the case of a complete graph, this is governed by the requirement that no node wants to cut a link. Hence, it implies that the parameter space for the ratio $b/(1-\lambda)\theta L$ is partitioned by

$$\left\{\max\left\{\frac{p(2)}{\delta}, \frac{p(1)}{\delta^2}\right\}, \max\left\{\frac{p(3)}{\delta}, \frac{p(2)}{\delta^2}\right\}, ..., \max\left\{\frac{p(n)}{\delta}, \frac{p(n-1)}{\delta^2}\right\}, ...\right\}, \tag{82}$$

so that, depending on where the ratio falls, we can have sequences of n chains, of $n+1$ chains and so on. In contrast, in the smallest equilibrium, $n+1$ chains only form when two extreme nodes in different n chains want to activate a link. This induces the partition

$$\left\{\frac{p(2)}{\delta^2}, \frac{p(3)}{\delta^2}, ..., \frac{p(n)}{\delta^2}, ...\right\} \tag{83}$$

It is easy to verify that point 2. of Proposition 3 holds also in this case. Indeed, since the statement requires the existence of an open set of parameter values, it is sufficient to repeat the argument in the proof for all δ sufficiently close to one. The same applies to point 3 of Proposition 4.

The largest and smallest equilibria within the class of regular equilibria are characterized by collections of isolated cliques

We first show that, if in any regular equilibrium nodes have $n-1$ active links, then equilibria with isolated cliques where each node has $n-1$ active links must be stable. Consider then an equilibrium where nodes have $n-1$ active links. If all links are active in equilibrium, then the result is proven since there is a clique comprising all nodes. Suppose then that there are two

nodes i and j with an inactive link. The increase in infection probability when the link is activated cannot be larger than in the case where there is no node to which both i and j have an active link (i.e. they have no common neighbor). Then, since both i and j have $n-1$ links, it is necessary (but not suffcient) for stability that

$$b\delta^n \leq (1-\lambda)\theta(1-\phi)[1+(1-\phi)(n-1)]L. \tag{84}$$

Otherwise, i and j necessarily want to activate their link. However, from (9), this implies that in a collection of cliques where all nodes have $n-1$ active links no node wants to activate an additional link. Consider now i's incentive not to cut a link to j. It must be that $b\delta^{n-1} \geq (1-\lambda)\theta(1-\phi)(1-\sigma_{ij})L$ where $\sigma_{ij} \in [0,1]$ denotes the probability that j may infect at least one of i's neighbors with an active link to i. Necessarily, since the component need not be totally connected, it must be that $\sigma_{ij} \leq 1-\phi^{n-2}$, i.e. the probability of being infected by j if all i's neighbors had active links to j. As a result, $b\delta^{n-1} \geq (1-\lambda)\theta(1-\phi)(1-\sigma_{ij})L \geq (1-\lambda)\theta(1-\phi)\phi^{n-2}L$ which, again from (9), implies that in a collection of isolated cliques where every node has $n-1$ active links no node wants to cut a link. As a result, a collection of isolated cliques with n members must be stable. It is immediate to verify that the same argument applies under NI and under SLOW. Endowed with this result, let $\overline{n}$ and $\underline{n}$ be the minimum and maximum number of active links nodes can have in any regular equilibrium, respectively. Then, sticking to our convention that N is always divisible by n, there exists an equilibrium with a collection of isolated cliques where every node has $n \leq \underline{n}$ active links and an equilibrium with a collection of isolated cliques where every node has $n \geq \overline{n}$ active links, which proves the claim.

In star structures, $W_{FAST} > W_{SLOW}$ for δ sufficiently close to one

Consider a star structure and note that the number of active links in the component must be weakly larger under FAST than under SLOW. Let m be the number of active nodes under FAST and $s \leq m$, $s \geq 2$, the number of active nodes under SLOW. Note that, under FAST, each peripheral node is indirectly linked to $m-2$ other peripheral nodes and the hub has $m-1$ active links. Total welfare is the sum of the benefits to the periphery and

to the hub net of the sum of the costs,

$$W_{FAST} = \underbrace{b\sum_{i=1}^{m-1}\delta^i}_{\substack{\text{hub's}\\ \text{benefit}}} + \underbrace{(m-1)b\delta}_{\substack{\text{periphery's}\\ \text{benefit}}} \quad - \\ \underbrace{(1-\phi)\theta(1-\lambda)\left[1+(1-\phi)(m-2)\right](m-1)L}_{\substack{\text{periphery's}\\ \text{cost}}} \quad - \\ \underbrace{(1-\phi)\theta(1-\lambda)(m-1)L}_{\substack{\text{hub's}\\ \text{cost}}} \qquad . \tag{85}$$

The same under SLOW is

$$W_{SLOW} = b\sum_{i=1}^{s-1}\delta^i + (s-1)b\delta \quad - \\ (1-\phi)\theta\left[1+(1-\lambda)(1-\phi)(s-2)\right](s-1)L - (1-\phi)\theta(s-1)L \tag{86}$$

where we note that, as usual, a slow intervention does not protect peripheral nodes when the hub is the initial carrier and never protects the hub. The difference reduces to

$$W_{FAST} - W_{SLOW} = b\sum_{i=s}^{m-1}\delta^i + (m-s)b\delta \quad - \\ (1-\phi)\theta\left(2[(1-\lambda)(m-1)-(s-1)]\right. \quad + \\ \left.(1-\lambda)(1-\phi)[(m-2)(m-2)-(s-2)(s-1)]\right)L. \tag{87}$$

However, from Lemma 1, for a component with m active links to be stable under FAST it must be that

$$b\delta \geq (1-\lambda)\theta(1-\phi)[1+(1-\phi)(m-1)]L \tag{88}$$

Noting that $W_{FAST} - W_{SLOW}$ is increasing in b, replace b in (87) with its lower bound provided by (88) and let $\delta \to 1$ (so that $\sum_{i=s}^{m-1}\delta^i \to m-s$). Rearranging, we obtain

$$W_{FAST} - W_{SLOW} \geq (1-\phi)\theta\left\{\lambda(s-1) \quad + \right. \\ \left.(1-\lambda)\left[2(m-1)(m-s)-(m-2)(m-2)+(s-2)(s-1)\right]\right\}L \tag{89}$$

The term on the first line strictly positive. The term on the second line is increasing in m and is equal to zero for $m = s$. As a result, it must be non-negative for all $m \geq s$. Hence, $W_{FAST} - W_{SLOW} > 0$.