Network Hazard: Moral Hazard in Strategic Network Formation*

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Abstract

While networks offer substantial benefits, they also facilitate the spread of major public threats such as misinformation in social networks, supply chain disruptions, cascading failures of interconnected banks, and epidemics. Efforts by authorities to mitigate contagion can inadvertently diminish agents' incentives to guard against it. This effect is amplified by the network itself, which can counteract the intended benefits of these mitigating measures. Specifically, as more effective tools are deployed to combat contagion, the interplay between mitigation efforts and endogenous network formation create a "network hazard," leading to reduced welfare, increased contagion, and greater volatility.

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Section 5 of this paper subsumes an earlier paper titled Network Hazard.

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1 Introduction

In recent years, networks have become increasingly significant across various markets, giving rise to new and complex challenges. The advent of the internet, for example, has greatly enhanced access to information and facilitated communication. However, it has also exacerbated the spread of misinformation, posing a global challenge. Similarly, globalization has fostered expansive international supply chains and optimized global capital allocation. Yet, it has also made global production systems more vulnerable to local natural disasters and geopolitical risks. In modern banking, financial innovations have improved access to credit and investment opportunities. However, the interbank networks that underpin these functions, such as complex derivative exposures, have introduced systemic risks. Additionally, the growth of a global middle class and increasing incomes, along with advancements in transportation technologies, have boosted mobility and tourism, but have also paved the way for global pandemics. Overall, while a more interconnected world offers numerous benefits, it also introduces significant and proportionate drawbacks. The interdependence of agents within a network creates vulnerabilities that can be transmitted through these connections, raising concerns about widespread contagion.

The specific structure of networked interactions introduces unique externalities that differ from those in standard economic models. A bottleneck in a network or the centrality of a particular agent can disproportionately affect economic outcomes. Therefore, understanding the relationship between contagious threats and the structure of endogenous networks is crucial for accurately assessing risks. For example, endogenous superspreader events posed significant public health threats during the COVID-19 pandemic, while certain public figures have wielded significant influence over polarized societies, particularly through social media. Similarly, highly interconnected banks were at the epicenter of the 2008 financial crisis, which led to unprecedented economic disruption.

Given the complex and widespread effects of contagion, policymakers have employed various instruments—such as content moderation, subsidies, bailouts, and vaccines—to mitigate these impacts. However, this approach raises another critical concern: the role of moral hazard in the endogenous structuring of networks. When agents anticipate the use of such mitigating instruments, they may alter their network connections in ways that heighten the risk of contagion.

This paper argues that these instruments can have unintended consequences, potentially increasing contagion, reducing overall welfare, and heightening volatility due to the endogenous reactions of network structures. Networks tend to amplify externalities; thus, when individual

agents' incentives to protect themselves against contagion are diminished, they are more likely to assume riskier network positions. This collective behavior can amplify contagion externalities, outweighing the positive effects of mitigation efforts.

In the proposed model, each agent decides whether to connect to another designated agent. Exogenous shocks then occur, creating costly externalities that spread through these connections. The total cost for each agent is determined by their Katz-Bonacich centrality. When agents expect that authorities will reduce the rates governing the spread of contagion through external interventions, endogenous centrality can increase to a level that negates the expected welfare benefits of these interventions. The cost of intervention then becomes the dominant effect on expected welfare. Additionally, agents may substitute the direct contagion costs from shocks to their connections for the indirect contagion costs from distant shocks transmitted through their network. This substitution can lead to higher centrality, increased aggregate exposure to central agents, and greater volatility. For instance, more effective or widely available vaccines might encourage larger social gatherings, potentially leading to more significant superspreader events. In the context of bailouts, financial networks may become more interconnected and concentrated around core banks, which could still default with a small probability but have a larger impact on peripheral banks.

Only when mitigating instruments are sufficiently effective—such that the endogenous network becomes highly interconnected and less sensitive to further improvements—do these interventions prove beneficial. This collective form of moral hazard, which reduces welfare or increases volatility through endogenous network reactions, is termed "network hazard." This concept is further explored in stylized models of coordination games, epidemics, and supply chains, confirming its validity and relevance.

Related literature The importance of central agents and network bottlenecks is underscored in the works of Galeotti and Goyal (2010) and Manea (2021). My research contributes to their by highlighting how centrality and network concentration can be self-reinforcing when interventions are introduced. Specifically, central agents tend to become even more central in response to interventions that are initially intended to mitigate the effects of their centrality.

Moral hazard in the context of strategic network formation has also been explored by Erol (2019) in the domain of financial networks. This study examines core-periphery structures, threshold contagion, ex-post bailouts, and a limited scope of second-order counterparty risk. In contrast, this paper investigates more general spillover effects characterized by Katz-Bonacich centrality, considers various types and timings of interventions, and addresses higher-order counterparty risks, both upstream and downstream. The broader implications

are demonstrated through applications in different settings.

Talamàs and Vohra (2020) demonstrate that partially effective vaccines can be detrimental in endogenous networks, as they lead agents to form more connections. My findings diverge by showing that agents tend to form more concentrated networks around central agents rather than increasing the number of links. Additionally, my main analysis extends beyond the scope of independent cascades of infectious diseases.

Empirical validation of the theory presents significant challenges, given that events like pandemics, tsunamis, or financial crises are infrequent, and anticipatory interventions are difficult to observe. However, Celdir and Erol (2023) provide empirical support in the context of pandemics, demonstrating that during the COVID-19 pandemic, higher vaccination rates led to increased foot traffic and, consequently, higher infection rates—resulting in more overall infections and more significant superspreader events as vaccination rates rose. In the context of financial networks, Anderson, Erol and Ordoñez (2022) show that interbank deposit networks became more concentrated around regional Federal Reserve Banks following the establishment of the Federal Reserve System and its discount lending facilities in 1914.

Several related studies examine regulations and interventions in networks without considering strategic network formation. For instance, Jackson and Pernoud (2019) analyze regulatory impacts on investment incentives in financial networks, while Dasaratha, Venkatesh and Vohra (2024), and Bernard, Capponi and Stiglitz (2022) investigate optimal bailout strategies. Galeotti, Golub and Goyal (2020) focus on interventions within fixed networks where coordination games are played.

Structure of the paper Section 2 presents a simplified baseline model and examines its equilibria. Section 3 investigates welfare implications and the concept of network hazard. In Section 4, a more general model is introduced, further illustrating network hazard dynamics. Section 5 provides standalone applications to coordination games, epidemics, and supply chains, which validate the main insights with additional institutional details. Finally, Section 6 offers concluding remarks.

2 Baseline

The economy comprises a mass μ of leaf agents, indexed by $i \in L \subset \mathbb{R}$, and a single root agent, indexed by $i = o$. Each leaf $i \in L$ decides whether to connect to the root, denoted as $a_i = 1$, or not to connect, denoted as $a_i = 0$. Connecting provides a benefit υ to the leaf. The

root cannot refuse any connections. The set $N \subset L$ represents the leaves that are connected to the root, and N is assumed to be measurable.[1]

After connections are formed, each i experiences a shock ω_i, referred to as i's original potential. These shocks are positive, uniformly bounded, independent, and have a mean of ω. Potential spreads throughout the network. Let p_i denote the potential of i. For a leaf $i \in L$, the potential is given by

$$p_i = \omega_i + \delta p_o a_i \tag{1}$$

and for the root o, the potential is

$$p_o = \omega_o + \alpha \int p_j a_j \mathtt{d}j \tag{2}$$

Here, $\delta > 0$ represents the downstream contagion rate from the root to the leaves, and $\alpha > 0$ represents the upstream contagion rate from the leaves to the root. The mean of the shocks ω is called the origination rate. Collectively, these parameters are referred to as contagion rates.[2] The concept of potential is closely related to Katz-Bonacich centrality, a relationship we explore in greater detail in Section 4. The simple network that emerges in the baseline case is illustrated in Figure 1.

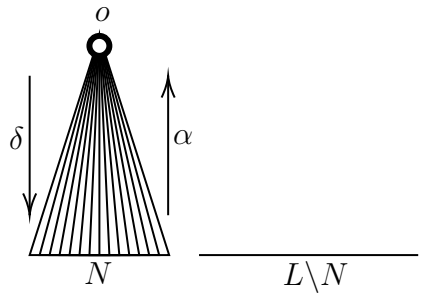


Figure 1: Baseline network

The original potential ω_i represents an intrinsic characteristic of agent i, and i is neutral to it. The excess potential $e_i = p_i - \omega_i$ is a costly externality imposed on i. The payoff for agent i is given by $u_i = \upsilon a_i - e_i$.

An equilibrium is defined as a Nash equilibrium that is stable—meaning that no vanishingly small group of agents can improve their payoffs by deviating jointly. The stability requirement

[1] An arbitrary strategy profile does not necessarily produce a measurable N. A non-measurable N lacks a discrete counterpart and abuses the continuum.

[2] The smallest positive solution to the equations for leaf and root potentials constitutes the network potentials. This solution can be obtained iteratively: each i starts with an original potential ω_i at stage $t = 0$, and at each subsequent stage t, the potentials are updated as $p_{o,t+1} = \omega_o + \alpha \int p_{j,t} a_j \mathtt{d}j$ and $p_{i,t+1} = \omega_i + \delta p_{o,t} a_i$. The limit of this process, $p_i = \lim_{t\to\infty} p_{i,t}$, yields the final potential.

serves to exclude knife-edge Nash equilibria, which are artifacts of the continuum and lack a generic discrete counterpart.[3]

Interpretation The potential represents an individual's evolving state concerning a social or economic issue that poses a costly public threat. This threat propagates through connections. Examples include cascading defaults in financial networks involving derivative or debt contracts, disruptions in supply chains, the spread of infectious diseases, or the dissemination of misinformation in social networks. The potential can be understood as either the magnitude of a cost or the probability of incurring a fixed cost.

When considering the magnitude of a cost, a payment failure within a financial network can trigger a series of further failures across a chain of exposures. The extent of these failures depends on the size of the shortfalls. In supply chains, significant reductions in production capacity or demand can lead to further disruptions, which are proportional to the extent of the supply or demand shortages. Conversely, when considering the probability of a given cost, an agent's likelihood of contracting an infectious disease during an epidemic increases the likelihood that their contacts will also contract the disease. Similarly, the probability of adopting misinformation raises the likelihood that those in contact will also adopt it.

This framework examines the interplay between endogenous centrality and mitigation measures against contagion. In this context, the "root" represents a central entity. For instance, in financial networks, the root could be core banks, while the "leaves" are peripheral banks. In supply chains, the root could be an upstream supplier to downstream producers or consumers. In epidemics, the root might be an individual in a high-traffic location, such as a cashier in a grocery store. In social networks, the root could be a specific platform or a popular public figure on a platform.

Downstream and upstream contagions often exhibit structural differences. To account for this, we consider two separate rates, α and δ. In social networks, interactions between celebrities and fans are typically asymmetric. In supply chains, suppliers face disruptions from demand changes by their buyers, while buyers are vulnerable to production disruptions from their suppliers. In core-periphery financial networks, the nature of exposures between the core and the periphery is often asymmetric.

[3]The vanishingly small group is also assumed to be measurable. Therefore, when N is measurable, payoffs corresponding to the strategy profile and after feasible deviations are well-defined. However, payoffs are not well-defined for arbitrary strategy profiles. Thus, the environment formally constitutes a pseudo-game.

2.1 Baseline equilibrium and welfare

Equilibrium A connected leaf is subject to externalities from the root, while the root is exposed to externalities from all connected leaves. Consequently, each connected leaf is indirectly exposed to every other connected leaf. Specifically, the potential p_o of the root o incorporates its own original potential ω_o as well as the upstream contagion from the leaves, given by $\alpha \int_N p_j \mathtt{d}j$. The aggregate potential of the connected leaves includes their individual shocks, $\int_N \omega_j \mathtt{d}j$, as well as the downstream contagion from the root, $\delta \int_N p_o \mathtt{d}j$. Ultimately, the potential of any agent is a linear combination of shocks. The exposure of an agent to the shocks of agents at distance k is referred to as k-th order contagion. The measure of the connected leaves, $n \equiv |N|$, is called the *connection rate*. For each connected leaf i, we have

$$\begin{aligned} p_i &= \omega_i + \delta p_o = \omega_i + \delta\omega_o + \delta\alpha \int_N p_j \mathtt{d}j = \omega_i + \delta\omega_o + \delta\alpha \int_N \omega_j \mathtt{d}j + \delta^2\alpha \int_N p_o \mathtt{d}j = ... \\ &= \omega_i + \underbrace{\frac{\delta}{1-\alpha\delta n}\omega_o}_{\text{first-order contagion}} + \underbrace{\frac{\delta\alpha}{1-\alpha\delta n}\int_N \omega_j \mathtt{d}j}_{\text{second-order contagion}} \end{aligned} \tag{3}$$

As more leaves connect, the magnitude of the second-order contagion increases. In other words, the net benefit of a connection is influenced by a congestion effect due to second-order contagion. This effect uniquely determines the mass of connected leaves in equilibrium. We denote this mass by

$$\underline{n} \equiv \frac{1}{\alpha\left(\omega + \upsilon\right)}\left(\frac{\upsilon}{\delta} - \omega\right)$$

Proposition 1. *The equilibrium connection rate is* $n^* = \max\left\{0, \min\left\{\mu, \underline{n}\right\}\right\}$.

Increasing the connection rate n raises the expected potential of the root, $\mathbb{E}[p_o]$. Consequently, the net expected benefit of connecting, $\upsilon - \delta\mathbb{E}[p_o]$, decreases until it reaches the *indifference condition*, $\upsilon = \delta\mathbb{E}[p_o]$. By taking the expectation of the externalities described in Equations (1) and (2), we find:

$$\mathbb{E}[p_o] = \omega + \alpha n \mathbb{E}[p_i] = \omega + \alpha n\left(\omega + \delta\mathbb{E}[p_o]\right)$$

Combining this with the indifference condition, we derive $\upsilon = \delta\left(\omega + \alpha n\left(\omega + \upsilon\right)\right)$, which determines the equilibrium connection rate n^*.

It is intuitive that the connection rate increases as the contagion rates are reduced. Notably, the product αn^* is independent of α. This implies that decreasing α does not alter the first-order contagion effect for a connected leaf. However, it does affect the root's first-order

contagion and the leaves' second-order contagion. Therefore, α influences the incentives of the leaves, and thus the connection rate, primarily through its impact on second-order contagion. We further explore contagion effects beyond the second-order in a more detailed framework in Section 4.

Welfare For the remainder of the baseline analysis, assume $\upsilon > \omega\delta$ and $\mu = \infty$ to avoid corner cases of the connection rate, $n^* = 0$ and $n^* = \mu$. The incentives in these corner cases are uninteresting.

Welfare W is defined by the payoffs of the leaves since the root is only a single agent. Thus, welfare has two components: the aggregate connection benefits and the aggregate contagion costs K. The expression for welfare is given by

$$W \equiv \underbrace{\int_L u_i \mathtt{d}i}_{\text{welfare}} = \underbrace{\int_L \upsilon a_i \mathtt{d}i}_{\text{connection benefits}} - \underbrace{\int_L (p_i - \omega_i)\, \mathtt{d}i}_{\text{contagion costs } K} = n\upsilon - n\delta p_o \tag{4}$$

Proposition 2. *Equilibrium welfare is*

$$W^* = \frac{(\upsilon - \delta\omega)}{\alpha\omega\,(1+\delta)}\,(\omega - \omega_o)$$

The variance of the shocks to the leaves averages out in the aggregate. However, a shock to the root has significant aggregate effects on welfare. The deviation of the root's shock from its mean, $|\omega - \omega_o|$, spills over to the leaves.

3 Network hazard

In many cases, a principal—such as a specific government body or a platform's management—has tools to mitigate contagion externalities. Examples of such tools include capital requirements or public liquidity provision in the context of financial contagion, subsidies in supply chains, vaccines or masks in epidemics, and content moderation or search engine algorithms in social networks. Each of these instruments can mitigate contagion within its respective context.

Consider a principal with access to instruments that can reduce the contagion rates α, δ, ω. A mitigation effort that reduces upstream contagion by $\alpha - \hat{\alpha}$ lowers α to $\hat{\alpha}$. Similarly, a mitigation of downstream contagion by $\delta - \hat{\delta}$ reduces δ to $\hat{\delta}$. Mitigation of contagion

origination by $\omega - \hat{\omega}$ scales each ω_i to $\hat{\omega}_i = \omega_i \frac{\hat{\omega}}{\omega}$ ensuring that post-mitigation shocks remain positive. We analyze the mitigation of each contagion rate independently.

For a given contagion rate $\theta \in \{\alpha, \delta, \omega\}$, mitigation of size $\theta - \hat{\theta}$ incurs a cost of $\frac{1}{\eta} c(\hat{\theta}; \theta)$, where $\eta > 0$ is a constant, and c is a decreasing and convex function of $\hat{\theta} \in [\underline{\theta}, \theta]$ with $c(\theta; \theta) = 0$ and $\underline{\theta} > 0$. We refer to η the *efficacy* of the mitigation instrument. Welfare is given by $\tilde{W}(\hat{\theta}; n) = W(\hat{\theta}; n) - \frac{1}{\eta} c(\hat{\theta}; \theta)$ where $W(\hat{\theta}; n)$ is the baseline welfare W (as defined in expression (4)) evaluated under $\hat{\theta}$ instead of θ, keeping all else fixed.

We consider two timing scenarios. The first is the case of commitment, where the mitigation policy is chosen and implemented before any connections are made; this type of mitigation is termed *prevention*. The second scenario involves a lack of commitment, where the mitigation policy is selected and implemented after connections have been established but before the shocks occur; this type of mitigation is referred to as *intervention*. In both cases, the principal aims to maximize the expected welfare $\mathbb{E}[\tilde{W}]$.

3.1 Prevention

Consider a principal who can prevent contagion by reducing the contagion rate θ to $\hat{\theta}$ before any connections are established.

Theorem 1. ***(Prevention and network hazard)*** *The expected welfare decreases, the expected cost of contagion increases, and the variance of welfare increases as the size of the prevention effort increases. Specifically $\mathbb{E}[\tilde{W}^*]$ decreases while $\mathbb{E}[K^*]$ and $\mathbb{V}[\tilde{W}^*]$ increase with $\theta - \hat{\theta}$. Consequently, the optimal policy for the principal is to avoid preventing contagion, implying $\hat{\theta}^* = \theta$.*

Proposition 2 demonstrates that reducing a contagion rate θ increases the connection rate n^* to a degree that offsets the direct welfare gains from reducing θ. Regardless of the extent of prevention, leaves are indifferent between connecting and not connecting. Both connected and unconnected leaves have an expected payoff of 0. Consequently, the expected welfare net of prevention cost is $\mathbb{E}[W] = 0$ in equilibrium. Therefore, expected welfare $\mathbb{E}[\tilde{W}]$ decreases with the size of the prevention effort, indicating that costly prevention is suboptimal.

Volatility is economically significant, and enhancing stability can provide contextual benefits. For example, in the context of epidemics, sharp increases in infection rates can lead to hospitalizations exceeding capacity, which strains the healthcare system. This strain can adversely affect the treatment of other conditions and result in excess deaths. In financial contagion, tail risks can trigger financial crises that may spill over into the real economy. Even

if tail events with low probability do not heavily influence the welfare-maximizing optimal policy, crises of significant magnitude can cause unforeseen disruptions in related areas.

A stronger preventive measure tends to induce *more* volatility without enhancing welfare. This occurs because the first-order contagion cost is more sensitive to changes in contagion rates than the second-order contagion cost, while the incentives of the leaves create a trade-off between these two costs. Reducing contagion rates diminishes the impact of second-order contagion but amplifies the impact of first-order contagion on individual leaves. Additionally, lower contagion rates lead to higher connection rates, meaning that more leaves are affected by the root's shock at a higher rate, thereby increasing aggregate volatility.

To elaborate further, recall expression (4) that breaks down the cost of excess potential into first-order and second-order contagion effects. Let the multiplier of ω_o be $\texttt{FC}$ (First-order Contagion), and the multiplier of the average shock to the leaves be $\texttt{SC}$ (Second-order Contagion).

In equilibrium, leaves connect up to the point where they are indifferent between connecting and not connecting, thereby eroding expected connection benefits to this point of indifference. This condition can be expressed as $\upsilon = (\texttt{FC} + \texttt{SC})\omega$. Due to the recursive nature of contagion and potential, $\texttt{FC} \cdot \omega$ represents the root's (expected) potential transmitted to a leaf at rate δ. Thus, we have $\texttt{FC} = \delta(1 + \texttt{SC})$. Substituting this relationship into the equation gives $\texttt{SC}(1 + \delta)\omega + \delta\omega = \upsilon$. Therefore, $\texttt{SC}$ is weakly increasing, and $\texttt{FC}$ is weakly decreasing with respect to the contagion rates α, δ, and ω.

There is n^* measure of connected leaves in equilibrium. Hence, the aggregate effect of the root's shock on the network is$n^*\texttt{FC}\omega_o$. Since $\texttt{FC}$ is decreasing with respect to the contagion rates, the aggregate effect $n^*\texttt{FC}$ also decreases as the contagion rates decrease.

3.2 Intervention

Consider a principal who can intervene to reduce the contagion rate θ to $\hat{\theta}$ after connections have been made but before shocks have materialized. We first establish a general result regarding the two extremes of the intervention policy, where $\hat{\theta} \in \{\underline{\theta}, \theta\}$.

Proposition 3. *There exists $\overline{\eta} > 0$ and $\overline{n}$ such that for all $\eta \neq \overline{\eta}$, connection rate $n^*_\eta = \overline{n}$ and maximal intervention $\hat{\theta}^*_\eta = \underline{\theta}$ is an equilibrium outcome if and only if $\eta > \overline{\eta}$. For $\eta > \overline{\eta}$, there is no network hazard: $\mathbb{E}[\tilde{W}^*]$ is increasing, whereas $\mathbb{E}[K^*]$ and $\mathbb{V}[\tilde{W}^*]$ are decreasing in η.*

There exists $\underline{\eta} \geq 0$ such that for all $\eta \neq \underline{\eta}$, the baseline connection rate $n^*_\eta = \underline{n}$ and no-intervention $\hat{\theta}^*_\eta = \theta$ is an equilibrium outcome if and only if $\eta < \underline{\eta}$. Also, $\underline{\eta} = 0$ if and only if $c_1(\theta, \theta) = 0$. For $\eta < \underline{\eta}$, efficacy η does not affect welfare.

The contagion rate of interest cannot be reduced below the feasibility threshold $\underline{\theta}$. This constraint causes the mass of connections to be bounded as η varies, with the upper bound being $\overline{n}$. When the efficacy is high enough to achieve maximum connection rate $\overline{n}$ in equilibrium, further increases in efficacy are beneficial. This outcome is intuitive: if the network is inelastic to contagion rates, then reducing these rates decreases the cost of contagion and enhances welfare.

However, when efficacy is below the corresponding cutoff, there is potential for negative consequences due to an elastic connection rate n^*_η. Additionally, there are cases where the cost function does not yield an "interior" equilibrium connection rate. For example, when the cost function is given by $c(\hat{\theta};\theta) = \frac{1}{1-\gamma}(\theta^{1-\gamma} - \hat{\theta}^{1-\gamma})$ with $\gamma \in (0,2)$, all equilibria for any η result in either $n^*_\eta = \underline{n}$ or $n^*_\eta = \overline{n}$. In this example, although there is an interior solution to the leaves' indifference condition between connecting or not, this solution is not stable against deviations by small groups.

We use a commonly applied functional form to explore interior equilibria.

Theorem 2. ***(Intervention and Network Hazard)*** *Let* $c(\hat{\theta};\theta) = \frac{1}{1+\gamma}(\theta - \hat{\theta})^{1+\gamma}$.

Increasing the efficacy of the policy instrument reduces welfare, increases contagion, and increases volatility unless the efficacy is sufficiently high.

There exists a threshold $\tilde{\eta} > 0$ *such that for all* $\eta < \tilde{\eta}$*, there is network hazard in equilibrium:* $\mathbb{E}[\tilde{W}]$ *is decreasing, while and* $\mathbb{V}[\tilde{W}^*]$ *are increasing with respect to* η*.*

Notice that $c_1(\theta,\theta) = 0$ in Theorem 2. Therefore, by Proposition 3, the baseline outcome $(\underline{n},\theta)$ does not emerge as an equilibrium outcome. It is optimal to intervene, meaning $\hat{\theta} < \theta$. A higher η, indicating a more effective intervention tool, reduces $\hat{\theta}^*_\eta$. However, the connection rate n^*_η adjusts and cancels out the expected welfare gains from interventions. The effect of efficacy on welfare and volatility is depicted in Figure ???

Consequently, the cost of intervention becomes the primary factor in expected welfare. The cost of intervention is reduced by higher efficacy η but increased by the larger intervention size $\theta - \hat{\theta}^*_\eta$. The net effect is an increase in the cost of intervention because the marginal aggregate contagion cost is strictly positive. Thus, higher efficacy reduces expected welfare. Additionally, volatility increases as the rising connection rate $n\eta^*$ heightens exposure to the root's shock.

Remark The unintended negative consequences of deploying mitigating measures are referred to as network hazard. This effect, however, has its limits. When $\mu = \infty$, the

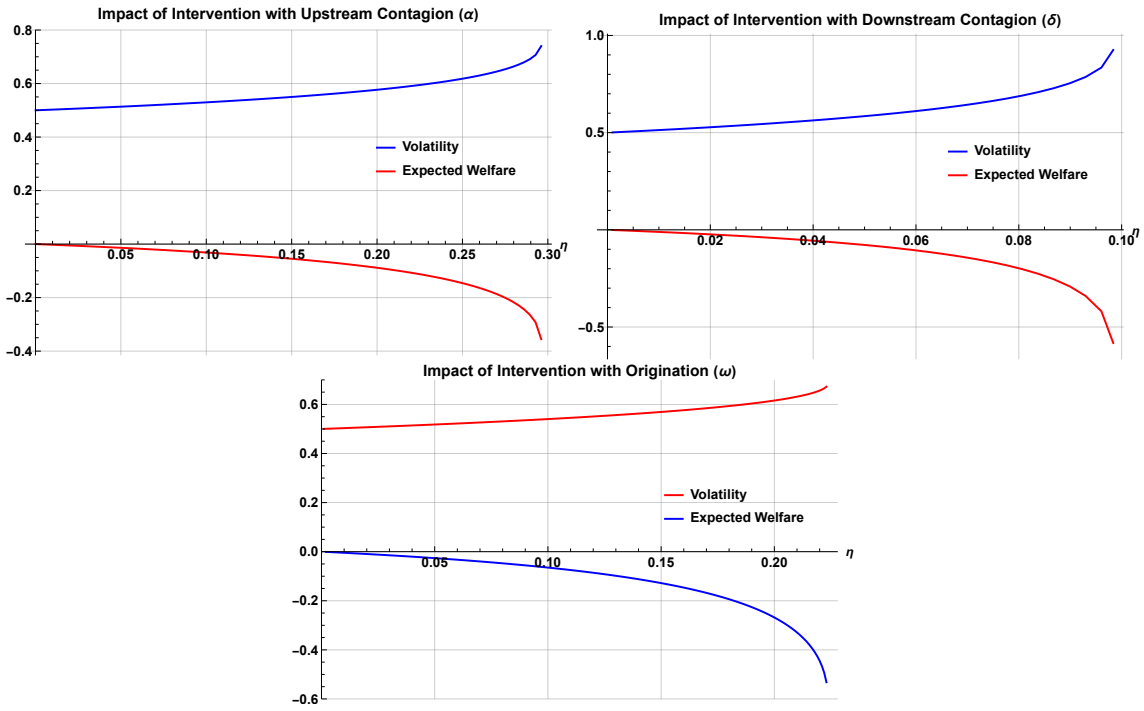


Figure 2: Impact of interventions as efficacy η grows (Values: $\alpha = \delta = \omega = \gamma = 1$, $\upsilon = 2$)

connection rate increases without bound, always counteracting the direct positive impact of the mitigation measures. However, if $\mu < \infty$, and the mitigation tool is sufficiently effective, the connection rate stabilizes at full connectivity, $n^* = \mu$. Beyond this point, further enhancements in the effectiveness of the mitigation tool actually increase welfare and decrease volatility. The key insight is that mitigation tools are only beneficial when their effectiveness is high enough that the network becomes inelastic to their impact. Only then does it make sense to deploy these tools and improve their efficacy further.

3.3 Interference

Interference with contagion rates after shocks have materialized yields no qualitative difference from intervention. However, there is a difference when considering another relevant policy tool. In the context of epidemics, quarantines and stay-at-home orders restrict regular or habitual social events, effectively severing connections. In social media, suspending or censoring a public figure cuts off their existing communication channels with followers. In financial networks, broad-based interventions like the Troubled Asset Relief Program eliminate certain exposures from the financial system.

Now, consider a principal who can mitigate contagion by severing connections. Mitigation by

removing connections is redundant or ineffective if implemented before shocks occur. For a policy f, removing $f(n)$ connections out of n is anticipated by the agents, who would then establish n' connections such that $n = n' - f(n')$. Therefore, we focus on interference with connections after shocks have been realized.

Extend the baseline scenario to allow the principal to cut each connection at a cost κ after the realization of shocks. For simplicity, assume the principal can either cut all n connections or none at all, which aligns with practical applications. The principal's objective is to maximize welfare $\tilde{W}$, defined as the baseline welfare W from expression (4) minus the cost of interference, 0 or κn. The policy is chosen and implemented after shocks have materialized. Shocks are assumed to be i.i.d. and uniformly distributed, with $\omega_i \sim U[0, 2\omega]$. We also require that the equilibrium be robust against a vanishingly small probability of interference failure to rule out some unintuitive cases.[4]

Theorem 3. ***(Interference and network hazard)*** *Welfare increases as the principal's cost of interference rises in a middle range.*

There exist thresholds $\underline{\kappa}$ and $\overline{\kappa}$ such that, in a robust equilibrium, expected welfare increases with κ for $\kappa \in (\underline{\kappa}, \overline{\kappa})$. For $\kappa \geq \overline{\kappa}$, the equilibrium outcome corresponds to the baseline outcome.

Interference is conditional on the realization of the root's shock, ω_o. If ω_o exceeds a certain threshold such that the cost of contagion is greater than the cost of interference, the contagion cost is replaced by the public cost of interference. This imposes an upper bound on the cost of contagion. Leaves connect up to the point of their indifference condition without internalizing the cost of interference.

4 Higher orders of contagion and network hazard

Second-order contagion regulates and determines the connection rate in the baseline scenario. In more complex network structures, shocks can propagate along longer paths, leading to higher orders of contagion. Agents consider these higher orders of contagion when deciding to form connections. Analyzing these incentives becomes more complex in the presence of cycles. With cycles, two agents can have multiple paths of different lengths between them, imposing different orders of contagion on each other. An acyclic network structure avoids this complexity and provides a tidy framework for exploring higher-order contagion. To achieve this, we adopt a recursive and acyclic structure inspired by Elliott, Golub and Leduc (2022).

[4] If $1 - \alpha\delta n$ is close to 0, there is interference irrespective of ω_o. All connections are always severed by the principal and all leaves have 0 payoff. This is an equilibrium, but it does not survive a small probability of interference failing.

Model For a fixed $T \geq 1$, let $L = [0, \mu)^T \subset \mathbb{R}^T$ be the set of leaf agents, and let $B = \cup_{t=0}^{T-1}[0, \mu)^t$ be the set of *bud* agents. We use the convention that $\mathbb{R}^0$ is singleton consisting of the empty vector $o = ()$. The case $T = 1$ represents the baseline scenario.

For each $i \in \mathbb{R}^t$ with $t \geq 1$, denote $i^* \in \mathbb{R}^{t-1}$ the first $t-1$ coordinates of i. This means $i = (i^*, y)$ for some $y \in \mathbb{R}$. Each $i \neq o$ can choose to connect to i^*, denoted by $a_i = 1$, or not to connect, denoted by $a_i = 0$. Bud $i \in B$ is called a *branch* if it chooses $a_i = 1$; otherwise, i is called a *root*.

Let N_i be the set of agents that connect to i, and n_i be the measure of N_i. The potential of i follows a generalization of the process in the baseline case:[5]

$$p_i = \omega_i + \left(\delta p_{i^*} a_i + \alpha \int_{N_i} p_j \mathtt{d}j\right) \tag{5}$$

The shocks and payoffs are identical to their baseline.

The network that emerges from any strategy profile is a forest of rooted directed trees. Agents can choose to become roots if they find the cost of downstream contagion from their potential upstream connection to be too high. The decision of agent i to connect to i^* depends on i^*'s decision to connect to $(i^*)^*$, and so on. Iteratively, up to $2T$'th order contagion is present in the economy.[6]

Discrete analogue In a discrete counterpart of the model, the μ measure of candidate downstream connections for each bud i represents a discrete $m = \lfloor \mu/\epsilon \rfloor$ number of agents, for some small ϵ. In this setup, there are $1 + m + ... + m^{T-1}$ buds and m^T leaves in the economy. The discrete counterpart of the contagion mechanics described in Equation (5) is given by $p_i = \omega_i + \left(\delta p_{i^*} a_i + \alpha\epsilon \sum_{N_i} p_j\right)$. The continuum model can be seen as the limit of the discrete model as $\epsilon \to 0$. This continuum formulation resolves integer problems and provides tractability in the determination of equilibrium potentials and connection rates. After establishing this mapping to the discrete model, we assume $\mu = \infty$ to avoid corner cases of connection rates.

Katz-Bonacich centrality and network games The concept of potential is analogous to Katz-Bonacich centrality. We extend Katz-Bonacich centrality to include a discrete

[5] Similar to the baseline, we pick the smallest positive solution which can be found by the limit of an iterative process.

[6] T'th-order upstream from leaves to o followed by T'th-order downstream from o back to leaves makes up $2T$'th-order contagion.

downstream damping factor δ and a continuous upstream damping factor α. The network induces a linear operator that maps the (random) baseline centrality ω_i to the (random) Katz-Bonacich centrality p_i. Due to linearity and risk-neutrality, the expected baseline centrality ω guides network formation. The continuum population and hierarchical structure allow us to determine the endogenous network and derive the centrality of each agent in a tractable closed form.

It is well known that Katz-Bonacich centrality is related to network games with complementarities. For instance, suppose that after the network is formed, agents play a network game where each agent i chooses an action $q_i \in \mathbb{R}$. The payoff for agent i in the network game is

$$v_i = -\sqrt{(q_i - \omega_i)\left(2(q_i - \omega_i) - \left(\delta q_{i^*} a_i + \alpha \int_{N_i} q_j \mathtt{d}j\right)\right)}$$

This is, i prefers to take an action q_i close to its original shock ω_i, but also prefers to take a higher action q_i if its connections take higher actions q_{i^*} and $(q_j)_{j\in N_i}$. In the nash equilibrium of this game, each i plays $q_i = p_i$, and gets payoff $v_i = -(p_i - \omega_i) = e_i$. Adding υa_i connection benefit, the payoff of i is $\upsilon a_i + v_i = \upsilon a_i - e_i$.

This means that agent i prefers to choose an action q_i close to its original shock ω_i, but also prefers to take a higher action if its connections take higher actions q_{i^*} and $(q_j)_{j\in Ni}$. In the Nash equilibrium of this game, each agent i chooses $q_i = p_i$ and receives a payoff of $v_i = -(p_i - \omega_i) = e_i$. Including the connection benefit υa_i, the total payoff for agent i is $\upsilon a_i + v_i = \upsilon a_i - e_i$.

4.1 Equilibrium network

An instrumental quantity in the describing the equilibrium is the benefit of a connection relative to the downstream transmission rate:

$$\rho \equiv \frac{\upsilon}{\delta}$$

The unique equilibrium network is determined by the following quantities:

$$\begin{aligned} R &= \max(0, \rho - \omega), & B &= \max(0, \rho - \omega - \upsilon) \\ r &= \frac{R}{\alpha(B + \omega + \upsilon)}, & b &= \frac{B}{\alpha(B + \omega + \upsilon)} \\ r' &= \frac{R}{\alpha(\omega + \upsilon)}, & b' &= \frac{B}{\alpha(\omega + \upsilon)} \end{aligned}$$

Theorem 4. *The equilibrium network is unique (up to measure preserving transformations). In this equilibrium, each $i \in B \backslash \mathbb{R}^{T-1}$ has r connections if i is a root and b connections if i is a branch. For $i \in \mathbb{R}^{T-1}$, the values are r' and b'. Specifically:*

- *If $\frac{\upsilon}{\omega} > \frac{\delta}{1-\delta}$, then $r, b, r', b' > 0$. There is a single rooted tree of depth T, starting with $o \in \mathbb{R}^0$. All roots in $\mathbb{R}^t$ are roots of trees of depth $T - t$. Each bud has an expected potential of ρ.*
- *If $\frac{\delta}{1-\delta} \geq \frac{\upsilon}{\omega} > \delta$, then $r, r' > 0 = b = b'$. The network consists of rooted trees of depth 1 (as in the baseline). Roots have an expected potential of ρ, and branches have an expected potential of $\omega + \upsilon$.*
- *If $\delta \geq \frac{\upsilon}{\omega}$, then $r = r' = b = b' = 0$. The network is empty, and each bud has an expected potential of ω.*

The connection rates b, b', r, r' decrease with increasing contagion rates α, δ, and ω.

Whether ρ exceeds the minimum expected potential ω of a connection (i.e., $\frac{\upsilon}{\omega} \geq \delta$) determines whether connecting to an otherwise isolated upstream branch is beneficial. Whether ρ exceeds $\omega + \upsilon$ (i.e., $\frac{\upsilon}{\omega} > \frac{\delta}{1-\delta}$) determines, in equilibrium, whether connecting to a branch with an upstream connection but no downstream connections is beneficial. When ρ is larger than $\omega + \upsilon$, a complex network with long contagion paths emerges. Agents are willing to connect to upstream branches who are themselves connected to their upstream branches and so on, leading to trees of various depths in equilibrium. The unique equilibrium network that emerges in this case is depicted in Figure 3.

In the baseline case, second-order contagion determines the connection rates. In general, connection rates are influenced by all orders of contagion. Regardless of the components contributing to the potential of a bud, downstream agents connect up to their point of indifference. The network configuration adjusts to ensure that potentials are equal to ρ. According to equation (5), connection rates r and b are determined by:

$$\rho = \omega + \alpha\rho r$$
$$\rho = \omega + (\delta\rho + \alpha\rho b)$$

Leaves cannot be connected to, so their potentials do not equilibrate to ρ. Instead, connected leaves have a potential of $\omega + \delta\rho$, as in the baseline case. This relationship determines r' and b'.

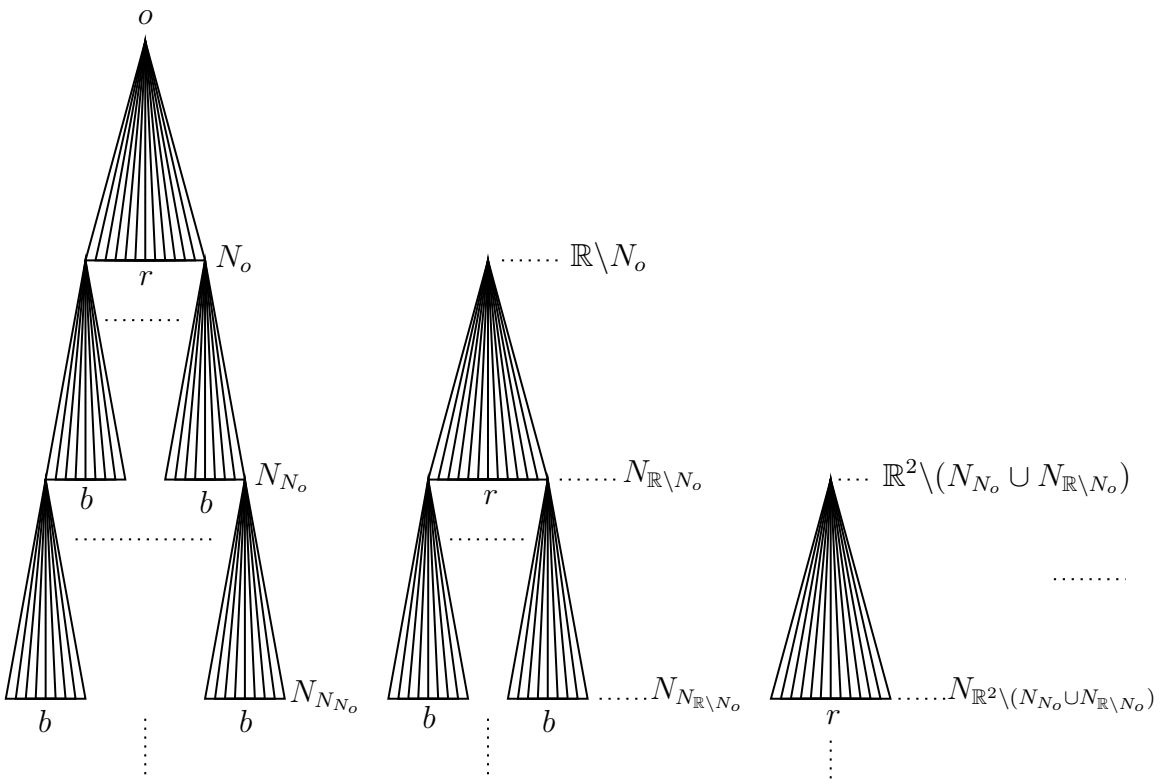


Figure 3: Unique equilibrium network

Theorem 4 shows that the incentives shaping connections have separable effects. First-order downstream contagion reduces connection rates by a *connection multiplier* of $\frac{R}{B}$, and second-order upstream contagion reduces connection rates by a connection multiplier of $\frac{B+\omega+\upsilon}{\omega+\upsilon}$. Roots do not face downstream contagion, so they have $\frac{r}{b} = \frac{r'}{b'} = \frac{R}{B}$ times more downstream connections than non-roots. Final branches (i.e., $\mathbb{R}^{T-1}$) do not experience second-order upstream contagion. Therefore, final branches have $\frac{r'}{r} = \frac{b'}{b} = \frac{B+\omega+\upsilon}{\omega+\upsilon}$ times more downstream connections than non-final branches. These separable effects also allow for a comparison with the baseline case, as roots in $\mathbb{R}^{T-1}$ are similar to the root in the baseline. This similarity is reflected in the fact that $r' = \underline{n}$.

Other network topologies The framework restricts connection decisions to a hierarchical structure, resulting in a unique equilibrium network. It is important to note that even if agents are allowed to connect to any bud, not just their designated predecessor (i.e., i^* for i), the strategy profile described in Theorem 4 is still an equilibrium. This is because all buds and downstream connections of each root are indifferent between connecting and not connecting, making them also indifferent to changing their upstream connection to another bud. In this broader setup, there are other equilibrium networks, including cyclic networks.

4.2 Higher orders of contagion and centrality

For the remainder of this section, assume $\frac{v}{\omega} > \frac{\delta}{1-\delta}$. Under this condition, the equilibrium network described in Theorem 4 consists of deep trees, and higher order of contagion is present. All branches have a potential of ρ, and the connection rate is given by

$$b = \frac{1}{\alpha}\left(1 - \delta\left(1 + \frac{\omega}{v}\right)\right)$$

In an equilibrium tree, each branch has a measure of b connections (except for the root, leaves, and branches that are directly before the leaves). This creates a recursive structure as an equilibrium outcome, allowing us to quantify the effects of shocks based on the distance between the origin of the shock and the affected agent.

Proposition 4. *In equilibrium, for any t, t' such that $t < t' < T$, and for any $i \in \mathbb{R}^t$ and any $j \in \mathbb{R}^{t'}$ with a path to i, the effect of i's original potential ω_i on j's potential p_j is*

$$\frac{\mathtt{d}p_j}{\mathtt{d}\omega_i} = \frac{e^{t'-t}}{1 - \alpha e n_i} + O(e^T)$$

where

$$e \equiv \frac{1 - \sqrt{1 - 4\delta\alpha b}}{2\alpha b}$$

Let ∂_k denote the magnitude of the effect of a shock on leaves at distance k downstream from the origin of the shock. The contagion mechanism in equation (5) gives

$$\partial_k = (\delta\partial_{k-1} + \alpha b\partial_{k+1})$$

This difference equation represents how the endogenous network amplifies shocks. The solution to any such difference equation is exponential in nature. In particular, ∂_k is $O(e^k)$ where e solves the quadratic equation

$$e = \delta + \alpha b e^2$$

The magnitude e represents the effect of shock ω_i on the potential of downstream branches N_i *relative* to the effect of ω_i on i itself. The first component of e is the direct relative downstream effect from i, which is δ. Given that e relatively amplifies ω_i one step downstream, e^2 represents the relative amplification of ω_i two steps downstream. For a given $j \in N_i$, a measure b of agents in N_j, who are subject to the e^2 amplification from i, feedback one step

upstream at rate α on j. This introduces the second component of e, which is the two steps downstream and one step upstream effect, αbe^2.

In the baseline case, there is no αbe^2 term because there are no branches. In the baseline scenario, the relative effect of root o's shock on the leaves is only the direct relative effect δ. This is evident when considering that for $i \in N$, the baseline yields $\frac{\mathtt{d}p_i}{\mathtt{d}\omega_o} = \frac{\delta}{1-\alpha\delta n}$ and $\frac{\mathtt{d}p_o}{\mathtt{d}\omega_o} = \frac{1}{1-\alpha\delta n}$.

4.3 Welfare and network hazard

The set of leaves is infinitely larger than the set of branches, so the payoffs of leaves drive the welfare analysis. The set L is endowed with the Lebesgue measure λ_T in $\mathbb{R}^T$.[7] Welfare is defined as

$$W \equiv \int_L u_i \mathtt{d}\lambda_T(i)$$

The only shock that can potentially create aggregate volatility is ω_o. In equilibrium, shocks to other agents reach at most a uniformly bounded λ_{T-1}-measure of leaves, which is negligible in λ_T. Therefore, welfare volatility is driven by an amplifier of

$$E_T \equiv \frac{\mathtt{d}W}{\mathtt{d}\omega_o}$$

The magnitude of amplification is determined by the network structure and does not depend on the ex-post realization of shocks, as potentials are affine functions of individual shocks.

Proposition 5. *In equilibrium, ex-post welfare is given by*

$$W^* = E_T^*(\omega - \omega_o)$$

where the amplifier E_T^* is determined by

$$\ln E_T^* = T \ln(eb) + O(1)$$

and

$$eb = \frac{1}{2\alpha}\left(1 - \sqrt{(1-2\delta)^2 + (2\delta)^2 \frac{\omega}{\upsilon}}\right)$$

[7] In the discrete counterpart of the model, provided that the 'size' of each agent ϵ is sufficiently small, the welfare of the leaf agents constitutes an arbitrarily large fraction of the total welfare of all agents. Consequently, the optimal policies discussed here are approximately optimal in the discrete model with arbitrarily high precision for sufficiently large populations.

The exact closed-form formula for E_T^* can be found in the proof of the result. Proposition 4 demonstrates that a shock is multiplied by a factor of e when transmitted to a connection one step downstream. Branches on the tree rooted at o have a measure b of downstream connections. Therefore, an *amplifying factor* eb governs the comparative statics of welfare for large T.

Theorem 5. ***(Foundations of network hazard)*** *In equilibrium, the expected welfare is zero. Reducing contagion rates increases welfare volatility, except for the downstream contagion rate when it is already sufficiently low.*

Formally, $\mathbb{E}[W^*] = 0$ *and* $\mathbb{V}\left(W^*\right)^{1/T} = eb - O(1/T)$. *The amplifying factor* eb *is decreasing in* α *and* ω, *and it is decreasing in* δ *if and only if* $2\delta\left(1 + \frac{\omega}{\upsilon}\right) > 1$.

This establishes the foundation for network hazard in the general model. Recall that the earlier results in Section 3 relied on two key forces: (i) agents connect up to the point where expected contagion costs exhaust the net benefits of connections, and (ii) first-order contagion is "steeper" than second-order contagion.

The first principle (i) holds in general, as shown by $\mathbb{E}[W^*] = 0$ in Theorem 5. As a result, both prevention and intervention are ineffective. A larger-scale prevention effort would only increase the cost of mitigation without enhancing expected welfare net of prevention costs. When the principal lacks commitment, an increase in the efficacy of the intervention instrument leads to a larger intervention size, which raises the cost and reduces welfare.

Volatility decreases with contagion rates, similar to the baseline, except for small values of δ. To understand the reason for the non-monotonicity in δ, first recall that in the baseline, the connection rate is given by $n^* = \frac{1}{\alpha(\omega+\upsilon)}\left(\frac{\upsilon}{\delta} - \omega\right)$. Thus $n^*\alpha$ and $n^*\delta$ are weakly decreasing in α and δ. In the extended model, the connection rate is $b = \frac{1}{\alpha}\left(1 - \delta\left(1 + \frac{\omega}{\upsilon}\right)\right)$. The factor $b\alpha$ is weakly decreasing in α but $b\delta$ is non-monotonic. To see why, recall the discussion following Theorem 4, which dissects connection incentives and their consequences on connection rates. The separable effects of upstream and downstream contagion on the incentives are captured by $b = r' \times \frac{B}{R} \times \frac{\omega+\upsilon}{B+\omega+\upsilon}$, and $r' = n^*$. Thus,

$$b = n^* \times \underbrace{\frac{\rho - \omega - \upsilon}{\rho - \omega}}_{\text{1 step downst.}} \times \underbrace{\frac{\omega + \upsilon}{\rho}}_{\text{2 step down and 1 step up}}$$

The third-order (two steps down and one step up) connection multiplier $\frac{\omega+\upsilon}{\rho}$ reflects changes in the mass of connections by accounting for the impact of downstream connections on

downstream connections. This third-order multiplier is increasing in δ, unlike the first-order downstream connection multiplier $\frac{\rho-\omega-\upsilon}{\rho-\omega}$ and the baseline connection rate $r' = n^*$.

This is intuitive: the direct positive effect of reducing the downstream contagion rate δ on first-order contagion is offset by connection incentives. Then, the effect on second-order downstream contagion, which is reflected back via upstream contagion, becomes positive. If δ is already low, n^* is relatively inelastic, so the positive marginal effect of reducing δ through third-order contagion outweighs the negative marginal effect caused by the increased connection rate n^*. Notably, this holds for any arbitrarily large T. The tradeoffs between marginal effects on higher orders parallel those on lower orders due to the recursive structure.

These results confirm the intuition from the baseline model. Unless protective measures are highly effective, they tend to reduce welfare and increase volatility.

5 Network hazard in applications

This section introduces several standalone models from different applications, incorporating additional institutional details to enhance robustness.

5.1 Coordination games and monetary transfers

This section examines monetary incentives in coordination games. For example, advertisers might pay influencers to leverage peer effects, or a manager might design bonuses to enhance group performance.

Consider a setting with two *followers* f_1, f_2, and one *leader* l. In the first stage, links are formed, denoted by $e_{ij} = e_{ji} \in \{0, 1\}$. Forming a link costs $c > 0$ for followers and 0 for the leader, denoted $c_i \in \{0, c\}$ for agent i. Followers are not allowed to link to each other. In the second stage, independent shocks $\theta_i \in \{g, b\}$ are realized. The good shock $g > 0$ occurs with probability $\alpha > 1/2$, while the (sufficiently) large bad shock $b > g$ occurs with probability $1 - \alpha$.

After the shocks are realized, each agent i simultaneously chooses an effort level $a_i \in \{0, 1\}$ with complete information. The shocks represent the cost of effort. Effort a_i applies to all of i's links and costs θ_i per link. If both i and j exert effort and $e_{ij} = 1$, the link generates a benefit yields $\beta > g$ to agent i. Thus, the payoff of agent i in the coordination game (in the second stage, net of cost of links) is $a_i \sum_j (\beta a_j - \theta_i) e_{ij}$.

Agents play the best[8] Nash equilibrium in the second stage. The ex-post payoff of i is

$$u_i = \sum_j \left(a_i \left(\beta a_j - \theta_i\right) - c_i\right) e_{ij}$$

In the first stage, agents form (pairwise) stable[9] networks given the expected continuation payoffs.

Welfare and transfers There is a principal who observes the network and the shocks and then commits to making transfers to each agent based on their effort. Given a transfer scheme $t_i(a_i|\mathbf{e}, \boldsymbol{\theta})$, the payoff of agent i in the coordination game is $u_i + t_i$.[10] The welfare cost of a unit transfer is $1 + z$, where z represents a transaction cost. Welfare is given by

$$w = \sum_i \left(u_i + t_i\right) - (1 + k) \sum_i t_i = \sum_i \left(u_i - z t_i\right)$$

We consider the limit as $z \downarrow 0$. This is equivalent to setting $z = 0$ and choosing the minimal transfer scheme among the optimal transfer schemes under $z = 0$.

Equilibrium If both followers link to the leader, they become indirectly exposed to each other's actions through the leader, making shirking contagious.

Proposition 6. *Assume $4g > 2\beta > 3g$ and $\alpha^2 > \frac{c}{\beta - g} > \alpha^3$. (Other cases are solved in the appendix.)*

In the absence of transfers, the leader has one follower.

In the presence of transfers, the leader has two followers.

Variance of welfare is larger in the presence of transfers than in the absence of transfers.

The stable network is illustrated in Figure 4. When $\beta < 2g$, the benefit from a single link is insufficient to incentivize the leader with two linked followers to exert effort. Therefore, if the leader has two followers and one follower experiences a bad shock, the leader will shirk. This, in turn, causes the other follower to shirk as well. As a result, followers are exposed to second-order contagion. This scenario is sufficiently likely when $\alpha^3 < \frac{c}{\beta - g}$, which prevents the leader from having two linked followers in equilibrium in the absence of transfers.

[8] Top element of the lattice of Nash equilibria.

[9] Pairwise stability requires that no pair of agents has a joint incentive to either cut or add a link, and no single agent has an incentive to cut an existing link.

[10] Given that transfers are conditional individual actions, the best Nash equilibrium is still well-defined.

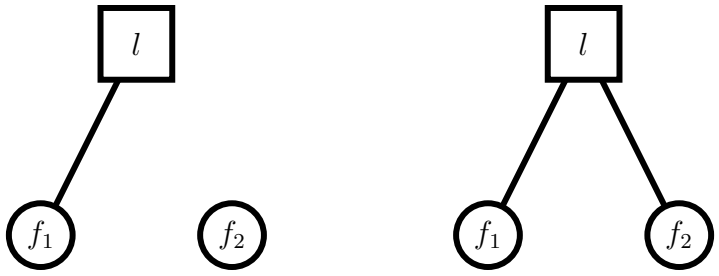


Figure 4: Stable network. Contagion that originates at f_1 can spread to l. This risk prevents f_2 from linking to l. Transfer to l mitigates contagion that originates at f_1. Then f_2 also links with l. But then the effect of contagion that originates at l is exacerbated.

When $2\beta > 3g$ and $\alpha^2 > \frac{c}{\beta - g}$, the precise role of optimal transfers is to mitigate second-order contagion. Provided that the leader has two linked followers, a transfer occurs if and only if one follower, f_i, receives a bad shock while the the other follower, f_j, and the leader have good shocks. The transfer persuades the *leader* to exert effort to prevent contagion, thereby protecting the *other follower* f_j. However, the leader can not be induced to exert effort if it receives a bad shock. As a result, both followers shirk.

Overall, transfers mitigate inefficient contagion ex-post. However, by doing so, transfers reduce market discipline, increase centrality, and expose all followers to the leader's idiosyncratic risk. While mitigating second-order contagion, transfers increase first-order contagion, resulting in greater welfare variance.

5.2 Epidemics and protective measures

This section examines a two-sided matching environment in the presence of an infectious disease. For example, interactions between tutors and students or cashiers and grocery shoppers can lead to the airborne transmission of a disease. Vaccines or masks can mitigate the risk of transmission and alter the matching structure.

There are two types of agents, $t \in \{a, b\}$. There are two type-a agents, $\{a_1, a_2\}$, and two type-b agents, $\{b_1, b_2\}$. Agents of the same type cannot match with each other. Type-a agents do not refuse matches from type-b agents. Each type-b agent, b_i, can match with one type-a agent at a cost 1, or choose not to match at a cost 0. Matching with a_i yields a payoff of v_i to type-b agents, where $v_1 > v_2 > 0$. This means that a_1 is a more preferred match.

Each agent has a probability η of being infected *externally*. Matches can transmit the infections *internally*, with a base transmission probability of τ_0 if one agent in the match is

infected and the other is not. A protective measure reduces the transmission probability by a factor of $m < 1$, lowering it to $\tau = m\tau_0$. The cost of becoming infected is κ.

We use the notion of (strong) stability which is equivalent to strong nash equilibrium in this model.

Equilibrium Since a_1 is preferred, both b-type agents prefer to match with a_1 in the absence of disease. However, when both type-b agents are matched with a_1, one b_i can infect a_1, who can then transmit the infection to the other b_j. This introduces second-order contagion. The efficacy of the protective measure m alters transmission probabilities and influences the network structure as follows.

Proposition 7. *There exist thresholds $m_3 \leq m_2 < m_1$ such that the unique stable network is characterized as follows:*[11]

- *Low efficacy: If $m_1 < m$, there are no matches.*
- *Medium-low efficacy: If $m_2 < m < m_1$, a_1 has one match, and a_2 has no matches.*
- *Medium-high efficacy: If $m_3 < m < m_2$, both a_1 and a_2 have one match each.*
- *High efficacy: If $m < m_3$, a_1 has two matches.*

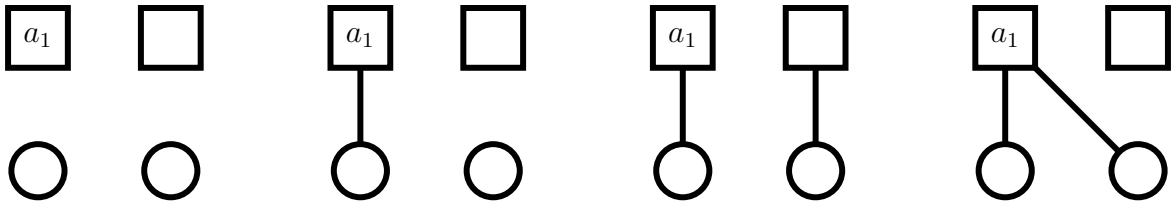
If $\eta < 1/4$, as the protective measure becomes more effective, both the expected number and variance of infections increase at the threshold points $m \in \{m_1, m_2, m_3\}$.

The network is depicted in Figure 5. As m decreases, indicating that the protective measure becomes more effective, the network becomes more interconnected. At the threshold m_1, it becomes individually rational for b_i to match with a_1 provided that b_j does not also match with a_1. At m_2, matching with a_2 similarly becomes rational. However, the expected cost of second-order contagion remains high enough that it is still undesirable for both b_i and b_j to match with the same agent. At m_3, the second-order counterparty risk is sufficiently low, allowing both type-b agents to match with the preferred agent, a_1.

Figures 6 and 7 illustrate the distribution of infections and welfare, respectively.[12] The vertical lines in the figures correspond to m_3. The increments at m_1 and m_2 are related to

[11] $m_1 \equiv m_1^*$, $m_2 \equiv \max\{m_2^*, m^*\} \leq m_3 \equiv \min\{m^{**}, m^*\}$ where $m_i^* \equiv \frac{v_i - 1}{\kappa(1-\eta)\eta\tau_0}$, $m^* \equiv \frac{\sqrt{1+4\frac{v_1-1}{\kappa\eta}}-1}{2(1-\eta)\tau_0}$, $m^{**} \equiv \frac{\sqrt{v_1 - v_2}}{\sqrt{\kappa\eta}(1-\eta)\tau_0}$. Note that we either have $m_2^* < m^* < m^{**}$ or $m_2^* > m^* > m^{**}$ or $m_2^* = m^* = m^{**}$. So $m_3 = m_2 \iff m_2^* \leq m^{**} \iff v_2 - 1 \leq \sqrt{\kappa\eta(v_1 - v_2)}$

[12] $\eta = 0.1, \tau_0 = 0.75, v_1 = 3, v_2 = 2.5, \kappa = 40$



Low efficacy $m > m_1$ | Medium-low efficacy $m_1 > m > m_2$ | Medium-high efficacy $m_2 > m > m_3$ | High efficacy $m_3 > m$

Figure 5: Strongly stable network of matches

Talamàs and Vohra (2020) as the number of matches rise at these thresholds. However, the change at m_3 represents a structural change. One type-b agent switches its match from a_2 to a_1. This occurs precisely because the protective measure becomes effective enough that b_i is less concerned about being infected by contagion originating from b_j and transmitted through a_1.

When both type-b agents match with a_1, the expected number of infections aligns with the increased matching payoffs. More interestingly, both type-b agents are now exposed to the exogenous infection risk of a_1. The variance of infections increases due to the correlation through a_1's infection probability.

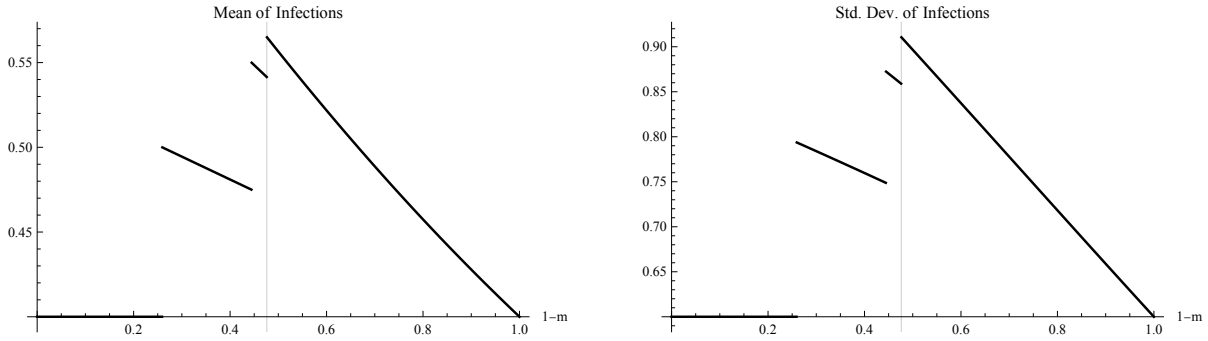


Figure 6: Mean and standard deviation of the number of infections

5.3 Supply chains and subsidies

This section examines inventory risk in supply chains. Downstream buyers face uncertain costs, which leads to upstream suppliers facing uncertain demand and the risk of overproduction. In turn, downstream buyers face the risk of under-provision if the upstream supplier does not risk overproduction. Subsidies to downstream firms' production costs can prevent this chain reaction and alleviate inventory risk.

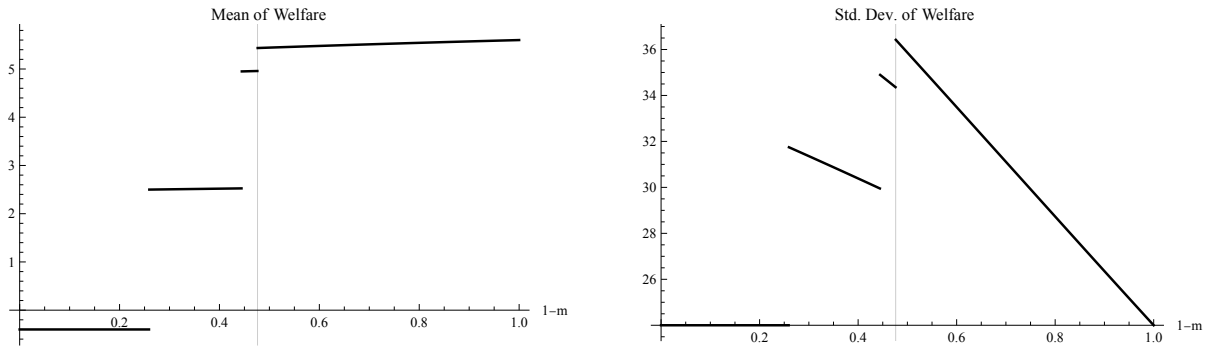


Figure 7: Mean and standard deviation of welfare

Consider two upstream firms, indexed by $u \in U = \{u_1, u_2\}$, which supply substitute but differentiated products to two downstream firms, indexed by $d \in D = \{d_1, d_2\}$. Each downstream firm chooses a specific production technology that is compatible with the input from only one of the upstream firms. Switching costs are sufficiently high such that once a downstream firm chooses an upstream firm's technology, the upstream firm becomes the sole supplier to that downstream firm.

Each upstream firm $u \in U$ requires a distinct *external* input supplied by another market. The price of the external input for firm u is low, $k_u = k$, with probability α_u and high, $k_u = k'$, with probability $1 - \alpha_u$. This price k_u reflects the conditions in the external market. The expected price of the external input of one upstream supplier is slightly lower than for the other, with $\alpha_{u_1} = \alpha_{u_2} + \varepsilon$ for a sufficiently small $\varepsilon > 0$. (The gap can be larger; the small gap is assumed for simplicity and clarity; the general case is discussed in the appendix.) The production function of each $u \in U$ converts q_u units of external input into q_u units of output.

Each downstream form $d \in D$ requires two inputs: an *internal* input supplied by its chosen upstream supplier and an *external* input supplied by another market. These two inputs are perfect complements, meaning that $q_d = \min\{q_d^{in}, q_d^{ex}\}$ units are produced with q_d^{in} units of the internal input and and q_d^{ex} units of external input. The price of the external input is low, $c_d = c$ with probability δ and high, $c_d = c'$, with probability $1 - \delta$.

Each downstream firm $d \in D$ is a monopolistic supplier facing inelastic unit demand for its output. The buyer has a value of p, so d sells at price p and captures all the surplus. Upstream firms sell to downstream firms at price $p' < p$. The fixed price p' reflects the exogenous bargaining power between upstream and downstream firms and it is not subject to renegotiation.

Timing is as follows. First, downstream firms simultaneously choose their technologies (suppliers). Then the prices of the external inputs of upstream firms, k_{u_1}, k_{u_2}, are realized

independently. Then upstream firms build their inventory, q_{u_1}, q_{u_2}. After this, the prices of the external inputs of downstream firms, d_{d_1}, c_{d_2}, are realized independently. Then downstream firms purchase internal and external inputs, and produce q_{d_1}, q_{d_2}. Then consumers buy outputs of the downstream firms.

High prices are assumed to be prohibitive to production: $k', c' > p$. Otherwise, there is no risk. Similarly, low prices are not prohibitive: $k < \delta p'$ and $c < p - p'$. Otherwise, there is no production. Only then firms face *inventory risk*.

If an upstream firm u has two downstream buyers but finds the probability of having 2 units of demand to be low, it will produce only 1 unit to minimize the expected cost of overproduction. But then, if both downstream firms have low costs and each demands 1 unit, the supply is exceeded. In this case, the available unit is supplied on a first-come-first-served basis, with a 1/2 probability for each downstream firm. This risk of under-provision and the competition for the unit supply can discourage downstream firms from choosing the same supplier, even if one supplier is superior to the other.

Denote $D_u \subset D$ denote the downstream buyers of $u \in U$ and $U_d \in U$ denote the upstream supplier of $d \in D$. If u faces a high price $k_u = k'$, it does not produce and has a payoff of $v_u = 0$. Otherwise, u earns

$$v_u = p' \sum_{d \in D_u} q_d - k q_u$$

Downstream d does not produce and has a payoff of $v_d = 0$ if either its upstream supplier U_d faces a high price k' and does not supply, d has high price c' and does not produce, or U_d is the supplier for both downstream firms and supplies only the other downstream firm. Otherwise, d is supplied a unit, produces, and earns

$$v_d = (p - p' - c)\, q_d$$

Welfare and subsidies The economy is not perfectly competitive. So the prices c_d and k_u do not represent the actual cost of producing the corresponding external inputs. Let e_i denote the domestic cost of procuring firm i's external input. The total domestic costs are then $\sum_i e_i q_i$. The total value generated by final consumption is $p \sum_d q_d$, all of which is extracted by firms. So absent subsidies, welfare is

$$\sum_i v_i = p \sum_d q_d - \sum_i e_i q_i$$

Shocks to prices can disrupt efficient production. The government can offer subsidies to firms for their external inputs to restore efficient production. Let s_i denote the subsidy to i per unit of external input purchased. This changes i's payoff to $v_i + s_i q_i$. The total domestic cost of these transfers is $(1+z)\sum_i s_i q_i$, where z represents a transaction cost, such as the cost associated with distortionary taxation needed to fund subsidies. Thus, welfare is given by

$$\begin{aligned} w &= 0 + \sum_i (v_i + s_i q_i) - (1+z)\sum_i s_i q_i \\ &= p\sum_d q_d - \sum_i (e_i + z s_i)\, q_i \end{aligned}$$

We focus on $z \downarrow 0$ to simplify the algebra. Equivalently, $z = 0$ and the government implements the minimal transfer necessary to induce the outcome that would be efficient. Additionally, we assume $e_d = 0$ whereas $e_u = k_u$. This means that the shocks faced by downstream firms stem from financial conditions, while the shocks faced by upstream firms reflect real shocks.[13] This setup highlights the role of subsidies in managing second-order contagion risk faced by downstream firms due to the inventory risk of upstream suppliers.

Equilibrium The chosen supplier of $d \in D$ may face a high price and decide not to produce, creating first-order contagion risk for d. Upstream firms also face first-order contagion if their downstream buyers encounter high prices and do not purchase inputs. Since upstream firms must build inventory in advance, this first-order contagion can lead an upstream firm with two buyers to produce only one unit instead of two, thereby creating second-order contagion for downstream firms by reducing their probability of being supplied.

Proposition 8. *Assume $k > \delta^2 p'$. (Other cases are solved in the appendix.)*

In the absence of subsidies, downstream firms choose different suppliers.

[13] External inputs for downstream firms are produced domestically in imperfectly competitive or monopolistic markets. For example, wages for high-skilled labor, patent rentals, or solar energy are generated using fundamental inputs that have relatively low marginal production costs. Shocks that are financial in nature, such as inflation or financial instability, can increase the prices of these inputs without altering their marginal production costs. A large shock to the price of an external input for a downstream firm reflects financial conditions rather than economic efficiency at the broader economy level. Efficient production can be restored through financial transfers to downstream firms, such as subsidies, to offset the increased prices of their external inputs.

On the other hand, external inputs for upstream firms are produced either internationally or in perfectly competitive domestic markets, where sale prices closely reflect the real costs of production. For instance, if a critical intermediate product produced exclusively abroad becomes more expensive, the domestic economy must absorb this increased cost. Similarly, if a natural disaster disrupts domestic supply chains, the marginal costs of production can rise even if the market for the specific good remains competitive. In this case, a large shock to the price of an external input for an upstream firm represents a shock to the cost of production of the external input, leading to inefficiency in production at the broader economy level.

In the presence of subsidies both downstream firms choose u_1. Each downstream firm d receives a subsidy $s_d = c' - (p - p')$ if u_1 faces a low price of external input.

If $\alpha_{u_1} < 1 - \frac{\delta}{2(1-\delta)}$, the expectation and variance of welfare is higher in the presence of subsidies than in the absence of subsidies.

Upstream firm u_1 is the preferred supplier because it entails lower first-order contagion cost than u_2. However, due to $k > \delta^2 p'$, the expected demand from downstream firms is too low relative to the cost of building enough inventory to supply both downstream firms. Even when both downstream firms choose u_1, u_1 produces only one unit. Thus, each d_i faces the risk of not being supplied. This scenario represents second-order contagion. Since the difference in the exogenous risk α_u between upstream firms does not differ significantly, one downstream firm prefers slightly higher first-order contagion cost over higher second-order contagion cost and chooses u_2.

Because $k' > p$, no upstream firm receives a subsidy when $k_u = k'$. The domestic cost of the external input for upstream firms is too high for efficient production. Downstream firms receive subsidies whenever they face high prices that would otherwise hinder their production, provided their upstream supplier faces low prices. This is because the external inputs of downstream firms are produced at a lower cost, making it efficient to restore production. Since the upstream firms do not face first-order contagion costs due to subsidies, they produce enough to meet the equilibrium demand of their downstream buyers, thereby eliminating second-order contagion. As a result, since the first-order contagion faced by downstream firms due to their supplier's prices remains unchanged, but second-order contagion risk that their supplier transmits is eliminated. Both downstream firms choose the same supplier, u_1.

Expected welfare naturally increases with subsidies since transactions costs are not present. In the absence of subsidies, the presence of second-order contagion prevents downstream firms from choosing the superior supplier. Subsidies remove this inefficiency. However, when both downstream firms choose the same supplier, the idiosyncratic shock to the supplier becomes a source of aggregate volatility. The common supplier is less risky but not entirely risk-free.

6 Conclusion

Networks provide substantial benefits but also introduce significant risks. Their structures in various critical contexts create vulnerabilities that can spread and intensify contagion, such as misinformation, systemic financial crises, global pandemics, and supply chain disruptions. We show that measures intended to mitigate contagion can backfire by promoting riskier network

configurations and increased centrality. A particularly nuanced dynamic is that agents shift their exposure from contagion spreading *through* the network to individual risks *originating* from their direct connections. As mitigation tools become more effective, agents' discipline against contagion weakens, a tendency that is amplified by the network structure, leading to greater contagion, reduced welfare, and increased volatility. This overarching phenomenon, termed "network hazard," represents a collective moral hazard where endogenous network responses undermine the intended effects of interventions.

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A Proofs

A.1 Main model

(Proof of Proposition 1) Straightforward following the paragraphs after the result.

(Proof of Proposition 2) For any given n, welfare is

$$W = \int_L u_i \mathtt{d}i = \int_N u_i \mathtt{d}i = n\upsilon - \int_L (p_i - \omega_i)\, \mathtt{d}i = n\upsilon - n\delta p_o$$

We also have

$$p_o = \omega_o + \alpha \int_N p_i \mathtt{d}i = \omega_o + \alpha n\,(\omega + \delta p_o)$$

so that

$$p_o = \frac{\omega_o + \alpha\omega n}{1 - \alpha\delta n} = \frac{1}{\delta}\left(\frac{\omega_o\delta + \omega}{1 - \alpha\delta n} - \omega\right)$$

In equilibrium, we have $n^* = \frac{1}{\alpha(\omega' + \upsilon)}\left(\frac{\upsilon}{\delta} - \omega\right)$. So welfare is

$$W^* = n^*\,(\upsilon - \delta p_o) = \frac{\upsilon - \delta\omega}{\alpha\,(\omega + \delta\omega)}\,(\omega - \omega_o)$$

(Proof of Theorem 1) By Proposition 2, $\mathbb{E}[W]$ is increasing, $\mathbb{E}[K]$ and $\mathbb{V}[W]$ are decreasing in θ. Then $\mathbb{E}[\tilde{W}]$ is decreasing whereas $\mathbb{E}[K]$ and $\mathbb{V}[\tilde{W}]$ are increasing in $\theta - \hat{\theta}$. So optimal policy is to no prevention.

(Proof of Proposition 3) First note that the principal must pick $\hat{\theta}$ such that the cost of contagion $K(\hat{\theta}, n)$ is not infinite. This corresponds to $\hat{\alpha}\delta n < 1$ or $\alpha\hat{\delta}n < 1$ if $\theta \in \{\alpha, \delta\}$. If $\theta = \omega$ we can assume $\alpha\delta n < 1$. Leaves would not connect up to $\alpha\delta n \geq 1$ because $\underline{\omega} > 0$. So we can focus on finite $K(\hat{\theta}, n)$. We have $K_1'(\hat{\theta}, n) > 0$ and $nK_1'(\hat{\theta}, n)$ is increasing in $\hat{\theta}$ and n.

The problem of the principal is to maximize $-nK(\hat{\theta}, n) - \frac{1}{\eta}c(\hat{\theta}; \theta)$. The derivative is $-nK_1'(\hat{\theta}, n) - \frac{1}{\eta}C'(\hat{\theta}) = \left(\Phi(\hat{\theta}, n) - \eta\right)\frac{nK_1'(\hat{\theta}, n)}{\eta}$ where $\Phi(\hat{\theta}, n) = \frac{-c_1(\hat{\theta}; \theta)}{nK_1'(\hat{\theta}, n)}$. By convexity, $-c_1(\hat{\theta}; \theta) \geq 0$ is decreasing in $\hat{\theta}$. Combining this with $\frac{nK_1'(\hat{\theta}, n)}{\eta^2} > 0$ and the monotonicity of $nK_1'(\hat{\theta}, n)$, we find that Φ is decreasing in $\hat{\theta}$ and n.

Take $\underline{\eta} = \Phi(\theta, n^*) \geq 0$. (Equality holds iff $c_1(\theta;\theta) = 0$.) At $n = n^*$, the solution is $\hat{\theta}_n = \theta$ iff $\eta \leq \Phi(\theta, n^*)$. For $\eta < \Phi(\theta, n^*)$, when an agent or a small group deviates from $n = n^*$, $\hat{\theta} = \theta$ still holds after the deviation. Thus $n = n^*$ is an equilibrium as we established in the baseline. When $\eta = \Phi(\theta, n^*)$, the stability of the solution depends on details of the cost function.

Take $\overline{\eta} = \Phi(\underline{\theta}, n) > 0$. For n, the solution is $\hat{\theta}_n = \underline{\theta}$ iff $\eta \geq \Phi(\underline{\theta}, n)$. Let n^{**} be the solution to the indifferent condition given $\underline{\theta}$. For $\eta > \Phi(\underline{\theta}, \mu)$, when a single leaf or a small group deviates from $n = n^{**}$, $\hat{\theta} = \underline{\theta}$ still holds after the deviation.

(Proof of Theorem 2) Case of $\hat{\alpha}$: Expected welfare is $n\left(\upsilon - \frac{\delta(1+\hat{\alpha}n)\omega}{1-\hat{\alpha}\delta n}\right) - \frac{1}{\eta}\frac{1}{1+\gamma}(\alpha - \hat{\alpha})^{1+\gamma}$. Assume interior solution solution $\hat{\alpha}_n \in (0, \alpha)$. Then the FOC $\frac{\delta(1+\delta)\omega n^2}{(1-\hat{\alpha}_n\delta n)^2} = \frac{1}{\eta}(\alpha - \hat{\alpha}_n)^{\gamma}$ holds. This implies a unique and decreasing $\hat{\alpha}_n$.

The individual cost of contagion is $\frac{\delta(1+\hat{\alpha}_n n)\omega}{1-\hat{\alpha}_n\delta n} \propto \hat{\alpha}_n n \equiv m_n$. Note, the individual cost of contagion must be increasing in n. Otherwise, an arbitrarily small group would deviate together and form connections. The FOC can be restated in terms of m_n and $\hat{\alpha}_n$ as $\frac{\delta(1+\delta)\omega m_n^2}{(1-m_n\delta)^2\hat{\alpha}_n^2} = \frac{1}{\eta}(\alpha - \hat{\alpha}_n)^{\gamma}$. Taking the derivative wrt n, we have $m_n'\left(\frac{1}{m_n} + \frac{\delta}{1-m_n\delta}\right) = \alpha_n'\left(\frac{1}{\hat{\alpha}_n} - \frac{\gamma}{2}\frac{1}{\alpha-\hat{\alpha}_n}\right)$. So n is stable if and only if $\frac{1}{\hat{\alpha}_n} < \frac{\gamma}{2}\frac{1}{\alpha-\hat{\alpha}_n}$.

The stable interior solution is characterized by three conditions: (i) $\frac{1}{\hat{\alpha}_\eta} < \frac{\gamma}{2}\frac{1}{\alpha-\hat{\alpha}_\eta}$, (ii) the FOC $\frac{\delta(1+\delta)\omega n_\eta^2}{(1-\hat{\alpha}_\eta\delta n_\eta)^2} = \frac{1}{\eta}(\alpha - \hat{\alpha}_\eta)^{\gamma}$, and (iii) the indifference condition $\frac{\delta(1+\hat{\alpha}_\eta n_\eta)\omega}{1-\hat{\alpha}_\eta\delta n_\eta} = \upsilon$. By (iii) $m_\eta = \hat{\alpha}_\eta n_\eta < \frac{1}{\delta}$ and m_η is constant in η. Then by (ii) and $n < \frac{1}{\delta\underline{\alpha}}$, we have $\lim_{\eta\to 0}\hat{\alpha}_\eta = \alpha$. Then (i) is automatically satisfied by $\gamma > 0$, for small η. Then the FOC and the indifference condition characterize interior stable equilibria. Note that by $\lim_{\eta\to 0}\hat{\alpha}_\eta = \alpha$ and (iii) imply that $\lim_{\eta\to 0} n_\eta = n^*$ of the baseline,

By the FOC and the indifference condition, the cost of the intervention is

$$\frac{1}{\eta}(\alpha - \hat{\alpha}_\eta)^{1+\gamma} \underset{\text{(FOC)}}{=} \frac{1}{\eta}\left(\eta\frac{\delta(1+\delta)\omega n_\eta^2}{(1-\hat{\alpha}_\eta\delta n_\eta)^2}\right)^{\frac{1+\gamma}{\gamma}} \underset{\text{(indif.)}}{\propto_\eta} \frac{\eta^{\frac{1}{2(\gamma+1)}}}{\hat{\alpha}_\eta}$$

Note that for small η, $\hat{\alpha}_\eta$ is decreasing. This can be observed by taking the derivative of the FOC $\frac{\delta(1+\delta)\omega m^2}{(1-m\delta)^2\hat{\alpha}_\eta^2} = \frac{1}{\eta}(\alpha - \hat{\alpha}_\eta)^{\gamma}$ w.r.t. η, which gives $\frac{1}{\eta} = -\hat{\alpha}_\eta'\left(\frac{\gamma}{\alpha-\hat{\alpha}_\eta} - \frac{2}{\hat{\alpha}_\eta}\right)$. Therefore, the cost of intervention is increasing in η.

The link benefits net of cost of contagion is 0 in expectation. So welfare is decreasing in η. Variance of welfare is $\mathbb{V}\left[n_\eta\left(\upsilon - \frac{\delta(\omega_o+\hat{\alpha}_\eta n_\eta\omega)}{1-\hat{\alpha}_\eta\delta n_\eta}\right)\right] \propto_\eta n_\eta$ which is also increasing in η by $n_\eta' > 0$.

Case of $\hat{\delta}$: Similar arguments work. The FOC is $\frac{n\omega(1+\alpha n)}{\left(1-\alpha\hat{\delta}_n n\right)^2} = \frac{1}{\eta}(\delta - \hat{\delta}_n)^{\gamma}$. By the FOC, for any small η, the individual cost of contagion $\frac{\hat{\delta}_n\omega(1+\alpha n)}{1-\alpha\hat{\delta}_n n}$ is increasing in n at equilibrium $n = n_\eta$, $\hat{\delta}_\eta$

is decreasing and n_η is increasing. Expected welfare is the cost of intervention, given by

$$\frac{1}{\eta}(\delta-\hat{\delta}_\eta)^{1+\gamma} \underset{\text{(FOC)}}{=} \frac{1}{\eta}\left(\eta\frac{n_\eta\omega\left(1+\alpha n_\eta\right)}{\left(1-\alpha\hat{\delta}_\eta n_\eta\right)^2}\right)^{\frac{1+\gamma}{\gamma}} \underset{\text{(indif.)}}{\propto_\eta} \frac{1}{\hat{\delta}_\eta^2\left(\frac{1}{n_\eta}+\alpha\right)}\eta^{\frac{1}{2(\gamma+1)}}$$

which is increasing in η. So welfare is decreasing. Variance of welfare is $\mathbb{V}\left[n_\eta\left(\upsilon-\frac{\delta(\omega_o+\alpha n_\eta\omega)}{1-\alpha\hat{\delta}_\eta n_\eta}\right)\right]\propto_\eta \frac{n_\eta}{1-\alpha\hat{\delta}_\eta n_\eta}\propto_\eta \frac{1}{\hat{\delta}_\eta\left(\frac{1}{n_\eta}+\alpha\right)}$ which is increasing in η.

Case of $\hat{\omega}$: The FOC is $\frac{n(1+\alpha n)}{1-\alpha\delta n}=\frac{1}{\eta}(\omega-\hat{\omega}_n)^\gamma$. For small η, the individual cost of contagion $\frac{1+\alpha n}{1-\alpha\delta n}\hat{\omega}_n$ is increasing in n at equilibrium $n=n_\eta$ for any small η, $\hat{\omega}_n$ is decreasing, and n_η is increasing. The cost of intervention is

$$\frac{1}{\eta}(\omega-\hat{\omega}_\eta)^{1+\gamma} \underset{\text{(FOC)}}{=} \frac{1}{\eta}\left(\eta\frac{n_\eta\left(1+\alpha n_\eta\right)}{1-\alpha\delta n_\eta}\right)^{\frac{1+\gamma}{\gamma}} \propto_\eta \frac{n_\eta\left(1+\alpha n_\eta\right)}{1-\alpha\delta n_\eta}\eta^{\frac{1}{2(\gamma+1)}}$$

which is increasing in η. So welfare is decreasing. The variance of welfare is $\mathbb{V}\left[n_\eta\left(\upsilon-\frac{\delta\left(\frac{\hat{\omega}_\eta}{\omega}\omega_o+\alpha n_\eta\hat{\omega}_\eta\right)}{1-\alpha\delta n_\eta}\right)\right]\propto_\eta \frac{n_\eta\hat{\omega}_\eta}{1-\alpha\delta n_\eta}\underset{\text{(indiff)}}{\propto_\eta}\frac{n_\eta}{1+\alpha n_\eta}$ which is increasing in η.

(Proof of Theorem 3) For a given n, welfare is $n\left(\upsilon-\delta\frac{\omega_o+\alpha n\omega}{1-\alpha\delta n}\right)=n\left(\upsilon+\omega-\frac{\delta\omega_o+\omega}{1-\alpha\delta n}\right)$. So optimal policy is to interfere iff $\upsilon+\omega-\frac{\delta\omega_o+\omega}{1-\alpha\delta n}<-\kappa$. When there is a interference, each agent has 0 payoff. Thus the individual expected payoff is

$$\frac{1-\alpha\delta n}{4\omega\delta}\left(\max\left(-\kappa,\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}\right)^2-\max\left(-\kappa,\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}\right)^2\right)$$

If $-\kappa>\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}$, there is always interference irrespective of ω_o, and each agent has 0 payoff. But such n is not robust to a small probability of interference failing. Each connected leaf would have negative payoff $\upsilon+\omega-\frac{\delta\omega_o+\omega}{1-\alpha\delta n}<-\kappa$ when the interference fails.

If $\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}>-\kappa$, there is never interference. If there is no interference, the baseline's network emerges. But in that network, $\upsilon+\omega=\frac{\omega\delta+\omega}{1-\alpha\delta n}$ so $\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}=(\upsilon+\omega)\left(1-\frac{2\delta+1}{\delta+1}\right)=-(\upsilon+\omega)\frac{\delta}{\delta+1}$. Thus the baseline is an equilibrium when $\kappa>\frac{(\upsilon+\omega)\delta}{\delta+1}\equiv\overline{\kappa}$.

If $\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}<-\kappa<\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}$, expected payoff is

$$\frac{1-\alpha\delta n}{4\omega\delta}\left(\left(\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}\right)^2-\kappa^2\right)$$

This has two roots, $\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}=\pm\kappa$. This is decreasing in n at the solution $\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}=-\kappa$, so the stable solution is $\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}=\kappa$.

Also recall the supposition of the case:

$$-\kappa>\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}=\upsilon+\omega-(2\delta+1)(\upsilon+\omega-\kappa)$$

which is equivalent to $\kappa<\frac{(\upsilon+\omega)\delta}{\delta+1}$.

The solution n_κ is pinned by $\upsilon+\omega-\frac{\omega}{1-\alpha\delta n_\kappa}=\kappa$ and it is decreasing in κ. Denote $x=\frac{\omega}{1-\alpha\delta n_\eta}$ and $y=\upsilon+\omega$. The expected cost of interference is

$$\begin{aligned}&\kappa n_\kappa\mathbb{P}\left[-\kappa>\upsilon+\omega-\frac{\delta\omega_o+\omega}{1-\alpha\delta n_\kappa}\right]=\kappa n_\kappa\frac{1-\alpha\delta n_\kappa}{4\omega\delta}\left(-\kappa-\left(\upsilon+\omega-\frac{2\delta\omega+\omega}{1-\alpha\delta n_\kappa}\right)\right)\\&=\frac{\delta+1}{2\alpha\delta^2}\kappa\left(1-\frac{\omega}{\upsilon+\omega-\kappa}\right)\frac{1}{\upsilon+\omega-\kappa}\left(\frac{\delta}{\delta+1}(\upsilon+\omega)-\kappa\right)\end{aligned}$$

This is decreasing in κ at $\frac{(\upsilon+\omega)\delta}{\delta+1}=\overline{\kappa}$. So there is $\underline{\kappa}$ such that on $\kappa\in(\underline{\kappa},\overline{\kappa})$, welfare is increasing in κ.

(Proof of Theorem 4) Follows by algebra upon the discussion after the result.

(Proof of Proposition 4) Fix t and k. Pick any $i\in\mathbb{R}^t$ and any $j\in\mathbb{R}^{t+k}$ downstream i.

All potentials are linear in all shocks. So denote $\partial_k=\frac{\mathrm{d}p_j}{\mathrm{d}\omega_i}$ the coefficient of ω_i in p_j. Then recursively we have

$$\begin{aligned}\partial_0&=1+\alpha n_i\partial_1\\\partial_1&=\delta\partial_0+\alpha b\partial_2\\&\ldots\\\partial_k&=\delta\partial_{k-1}+\alpha b\partial_{k+1}\\&\ldots\\\partial_{T-t-1}&=\delta\partial_{T-t-2}+\alpha b\partial_{T-t}\\\partial_{T-t}&=\delta\partial_{T-t}\end{aligned}$$

Define a recursive sequence $\alpha bz_k-z_{k-1}+\delta z_{k-2}=0$ whose solution is given by $z_k=A\zeta_1^k+B\zeta_2^k$ where $\zeta_1=\frac{1+\sqrt{1-4\delta\alpha b}}{2\alpha b}>1>\zeta_2=\frac{1-\sqrt{1-4\delta\alpha b}}{2\alpha b}$ and A,B are constants. (Note $\zeta_2=e$.)

Then ∂_k sequence can be found by using a suitable z sequence. Pin down A and B (hence

the z sequence) with

$$z_{T-t}\frac{b'}{b} = \delta z_{T-t-1}$$
$$z_0 = 1 + \alpha n_i z_1$$

Then ∂_k sequence is given by $\partial_k = z_k$ for $k = 0, ..., T-t-1$ and $\partial_{T-t} = z_{T-t}\frac{b'}{b}$.

The conditions that pin A and B are equivalent to

$$\left(A\zeta_1^{T-t} + B\zeta_2^{T-t}\right) b' = b\delta\left(A\zeta_1^{T-t-1} + B\zeta_2^{T-t-1}\right)$$
$$A + B = 1 + \alpha n_i \left(A\zeta_1 + B\zeta_2\right)$$

Then

$$A = \frac{\left(\zeta_2 b' - b\delta\right)}{\left(\zeta_2 b' - b\delta\right)\left(1 - \zeta_1\alpha n_i\right) - \left(\frac{\zeta_1}{\zeta_2}\right)^{T-t-1}\left(\zeta_1 b' - b\delta\right)\left(1 - \zeta_2\alpha n_i\right)}$$
$$B = \frac{\left(\zeta_1 b' - b\delta\right)}{\left(\zeta_1 b' - b\delta\right)\left(1 - \zeta_2\alpha n_i\right) - \left(\frac{\zeta_2}{\zeta_1}\right)^{T-t-1}\left(\zeta_2 b' - b\delta\right)\left(1 - \zeta_1\alpha n_i\right)}$$

Then,

$$\partial_k = z_k = A\zeta_1^k + B\zeta_2^k = \frac{\zeta_2^k}{1 - \zeta_2\alpha n_i}\left(1 + O\left(\left(\zeta_2/\zeta_1\right)^T\right)\right)$$

(Proof of Proposition 5) All potentials are linear in all shocks. So welfare is linear in all shocks. Effects of all shocks but ω_o wash out by law of large numbers. So W is linear in ω_o, with slope E_T by definition. By the indifference conditions $\mathbb{E}[W] = 0$. Combining these we get $W = E_T\left(\omega - \omega_o\right)$.

Regarding $\ln E_T$, continuing with the proof of Theorem 4. We have

$$\partial_T = z_T\frac{b'}{b} = \frac{b'}{b}\left(A\zeta_1^T + B\zeta_2^T\right)$$
$$= \frac{b'}{b}\left(\frac{\left(b\delta\right)\left(\zeta_1 - \zeta_2\right)\zeta_1^{T-1}\zeta_2^{T-1}}{\zeta_1^{T-1}\left(\zeta_1 b' - b\delta\right)\left(1 - \zeta_2\alpha r\right) - \zeta_2^{T-1}\left(\zeta_2 b' - b\delta\right)\left(1 - \zeta_1\alpha r\right)}\right)$$

Note $E_T = rb^{T-2}b'\partial_T$ as there is $rb^{T-2}b'$ λ_T-measure of leaves whose potential's (hence

payoff's) derivative with respect to ω_o is ∂_T. Then using also $\zeta_1\zeta_2 = \frac{\delta}{\alpha b}$ we have

$$E_T = rb^{T-2}b'\partial_T = \frac{\delta r b'^2 (\zeta_1 - \zeta_2)}{b} (\zeta_2 b)^{T-1} \frac{1}{(\zeta_1 b' - b\delta)(1 - \zeta_2 \alpha r) - \left(\frac{\zeta_2}{\zeta_1}\right)^{T-1} (\zeta_2 b' - b\delta)(1 - \zeta_1 \alpha r)}$$

So

$$\ln(E_T) = \ln\left(rb^{T-2}b'\partial_T\right) = T\ln(eb) + o(1)$$

Note

$$\begin{aligned} & e = \delta + \alpha b e^2 \implies \alpha b e = \alpha b \delta + (\alpha b e)^2 \\ & \implies \alpha b e = \delta\left(1 - \delta\left(1 + \frac{\omega}{\upsilon}\right)\right) + (\alpha b e)^2 \implies \\ & be = \frac{1 - \sqrt{1 - 4\delta\left(1 - \delta\left(1 + \frac{\omega}{\upsilon}\right)\right)}}{2\alpha} = \frac{1 - \sqrt{(1-2\delta)^2 + (2\delta)^2 \frac{\omega}{\upsilon}}}{2\alpha} \end{aligned}$$

(Proof of Theorem 5) be is decreasing in α and ω. Regarding δ, be is decreasing in δ if and only if $4\delta\left(1 - \delta\left(1 + \frac{\omega}{\upsilon}\right)\right)$ is decreasing, which is equivalent to $1 < 2\delta\left(1 + \frac{\omega}{\upsilon}\right)$.

A.2 Standalone applications

Notation. Throughout the rest of the appendix we denote by $x = p_1 \circ x_1 \oplus p_2 \circ x_2 \oplus ...$ a random variable that takes value x_i with probability p_i for each i. Also $\mathbb{B}[k, x]$ is the binomial distribution with k tries and x success probability. Welfare in the absence of interventions is denoted w whereas welfare in the presence of interventions is denoted w'.

A.2.1 Coordination games and interventions

(Proof of Proposition 6) Corollary of Propositions 9, 10, 11.

Define parameters more generally. Let α_i, β_i, g_i be heterogenous and depend on the "type" f and l. Assume $\alpha_f \geq \alpha_l$, $\beta_f > g_f$, $\beta_l > g_l$, $2\alpha_f > 1$. Denote $\kappa \equiv \frac{c}{\beta_f - g_f}$, $\omega \equiv \beta_l - 2g_l$, $\omega' = \beta_f - g_f$.

Proposition 9. *The unique stable network is given as follows.*

Under $\omega < 0$, both followers follow if $\alpha_l\alpha_f^2 > \kappa$, only one follow if $\alpha_l\alpha_f > \kappa > \alpha_l\alpha_f^2$, and none follow if $\kappa > \alpha_l\alpha_f$.

Under $\omega > 0$, both followers follow if $\alpha_l\alpha_f > \kappa$, and none follow if $\kappa > \alpha_l\alpha_f$.

Proof. There are three possible networks; no links, one link, two links. As the bad shock b_i is arbitrarily large, any agent with a bad shock chooses $a_i = 0$. Also, if f_i follows and l chooses 0, then f_i chooses 0 because $g_f > 0$.

Under $\beta_l < 2g_l$, if l has two followers and at least one chooses 0, then l chooses 0 by $\beta_l < 2g_l$. This implies that in equilibrium, in a connected component with at least one link, all agents choose 0 if at least one agent has a bad shock, and all agents choose 1 if no agent has a bad shock. Then l's expected payoff as a function of the number of its followers d is $U_{l,d} = \alpha_l \alpha_f^d d\,(\beta_l - g_l)$. By $2\alpha_f > 1$ and $\beta_l > g_l$, $U_{l,d}$ is increasing in $d \leq 2$. So l prefers to have more followers.

The payoff of f_k if both f_1, f_2 are connected to l is $U_{f,2} = \alpha_l \alpha_f^2 (\beta_f - g_f) - c$. If one is connected and the other is not, the one that is connected has $U_{f,1} = \alpha_l \alpha_f (\beta_f - g_f) - c$. Notice $U_{f,1} > U_{f,2}$. Then if $U_{f,2} > 0$ both followers follow l. If $U_{f,1} > 0 > U_{f,2}$, then one follows and the other does not. If $U_{f,1} < 0$ there are no links.

Under $\beta_l > 2g_l$, l chooses 0 if and only if l gets a bad shock or all of its connections get bad shocks. In either case, all agents choose 0. If l gets a good shock, each f chooses with its own shocks: 0 if and only if the shock is bad. Then l's expected payoff as a function of the number of its followers is $U_{l,1} = \alpha_l \alpha_f (\beta_l - g_l)$ or

$$\begin{aligned} U_{l,2} &= \alpha_l \left(\alpha_f^2 2 (\beta_l - g_l) + 2\alpha_S (1 - \alpha_S) (\beta_l - 2g_l) \right) \\ &> \alpha_l \alpha_f^2 2 (\beta_l - g_l) > \alpha_l \alpha_f (\beta_l - g_l) = U_{l,1} \end{aligned}$$

by $2\alpha_f > 1$. So l prefers to have more followers.

When f_i gets a good shock, l chooses 0 only when l gets a bad shock. So the payoff of f_i is $U_{f,1} = \alpha_f \alpha_l (\beta_f - g_f) - c$ if it follows, and 0 otherwise. If $U_{f,1} > 0$, i.e. $\kappa < \alpha_l \alpha_f$ both follow l. Otherwise there are no links.

Proposition 10. *Under $\omega + \omega' < 0$ or $\omega > 0$ there are no transfers. Accordingly, the unique stable network is the same with the one in the absence of interventions.*

Under $\omega' + \omega > 0 > \omega$, $t_l(1|e, \theta) = -\omega$ if l has two followers, for one $i \in \{1, 2\}$, l and f_i have good shocks and f_j has a bad shock. All other transfers are 0 in all other cases of shock realizations and networks. The unique stable network involves two links if $\alpha_l \alpha_f > \kappa$ and no links if $\alpha_l \alpha_f < \kappa$.

Proof. Consider the auxiliary problem of choosing an action profile a to maximize $V = \sum_i \left(a_i \sum_j (\beta_{ij} a_j - \theta_i) e_{ij} \right)$

Given that b_i is large enough, $a_i^* = 0$ if $\theta_i = b_i$. Given this,

$$\begin{aligned} V &= \sum_{i:\theta_i=g_i} a_i \left(\left(\sum_{j\neq i:\theta_j=g_j} \beta_{ij} a_j e_{ij} \right) - d_i g_i \right) \\ &= \sum_{i:\theta_i=g_i} \sum_{j\neq i:\theta_j=g_j} a_i a_j \beta_{ij} e_{ij} - \sum_{i:\theta_i=g_i} a_i d_i g_i \end{aligned}$$

If i has no links, i's action is efficient. So there are no transfers.

If i has links but all of i's links have bad shocks, then $a_i^* = 0$ to save on $\sum_{i:\theta_i=g_i} a_i d_i g_i$ even if i has a good shock.

If i has a good shock and it has a link with a good shock, say j, then there are two cases. If the third agent also has a good shock, there is no need for transfers; all agents choose 1. If the third agent is has a bad shock there are two cases. If the third agents is not connected to i or j, then i and j do not need transfers and they choose 1. So the only case there can possibly be an optimal and positive transfer is when all agents are connected, l and one f_i have good shocks, and other f_j has a bad shock. Due to the complementarities, it is either optimal that l and f_i both choose 0 or they both choose 1. If they both choose 0, $V = 0$. If they both choose 1, $V = V^* \equiv \beta_l + \beta_f - 2g_l - g_f$.

If $V^* < 0$, then $W \leq 0$. Then choosing $t = 0$ implements that optimal action profile. In this case there are never any transfers and the network formed is the same with the absence of interventions.

If $V^* > 0$, optimal action profile is implemented by

$$\begin{aligned} t_l(1) &= (2g_l - \beta_l)^+, \;\; t_l(0) = 0 \\ t_{f_k}(1) &= (g_f - \beta_f)^+ = 0, \;\; t_{f_k}(0) = 0 \\ t_{f_{k'}} &= 0 \end{aligned}$$

If $2g_l < \beta_l$, there is no need for transfers: $t = 0$ and l chooses 1. If $2g_l > \beta_l$, then an f agent has expected payoff $U_{f,1} = \alpha_l \alpha_f (\beta_f - g_f) - c$ regardless of whether the other f agents has a link with l or not. l, conditional on degree d, has expected payoff $U_{l,d} = \alpha_l \alpha_f^d d (\beta_l - g_l)$ which is increasing in d. Thus the unique stable network has two links if $\alpha_l \alpha_f > \kappa$ and 0 links if $\alpha_l \alpha_f < \kappa$.

Proposition 11. *Assume $\omega' > -\omega > 0$ and $\alpha_l \alpha_f > \kappa > \alpha_l \alpha_f^2$. Variance of welfare is larger in the presence of interventions than in the absence of interventions. The change in the expectation of welfare can be positive or negative depending on parameters.*

Proof. Denote $v_f = \beta_f - g_f$, $v_l = \beta_l - g_l$.

$$\begin{aligned} w + c &= (\alpha_l \alpha_f \circ (v_f + v_l) + 0) \\ \mathbb{E}[w] &= \alpha_l \alpha_f (v_f + v_l) - c \\ \text{Val}[w] &= \alpha_l \alpha_f (1 - \alpha_l \alpha_f) (v_f + v_l)^2 \end{aligned}$$

Some algebra yields that in the presence of interventions

$$\begin{aligned} & w' + 2c = \alpha_f^2 \alpha_l \circ (2v_f + 2v_l) \oplus 2(1 - \alpha_f)\alpha_f \alpha_l \circ (v_l - \beta_l + v_f) \\ & \oplus (1 - \alpha_f)^2 \alpha_l \circ 0 \oplus (1 - \alpha_l) \circ 0 \\ & \mathbb{E}[w'] = 2\alpha_f \alpha_l (v_f + v_l - (1 - \alpha_f)\beta_l) - 2c \\ & \text{Val}[w'] = 2\alpha_l \alpha_f (1 - \alpha_f) \left[(v_f + v_l)^2 \right. \\ & \left. + (1 - 2(1 - \alpha_f)\alpha_f) \beta_l + 2 (2\alpha_f - 1) \beta_l (v_l + v_f) \right] \\ & + 4\alpha_l (1 - \alpha_l) \alpha_f^2 (v_f + v_l - (1 - \alpha_f)\beta_l)^2 \end{aligned}$$

Then by rearranging terms we get

$$\begin{aligned} & \text{Val}[w'] - \text{Val}[w] = (1 + 2\alpha_f - 3\alpha_f \alpha_l) (v_f + v_l)^2 + \\ & + 2 (1 - \alpha_f) (1 - 2\alpha_l \alpha_f (1 - \alpha_f)) \beta_l - 4 (1 - \alpha_f) (1 - 2\alpha_l \alpha_f) (v_l + v_f) \beta_l \\ & > 0 \impliedby (1 + 2\alpha_f - 3\alpha_f \alpha_l) (1 - 2\alpha_l \alpha_f (1 - \alpha_f)) > 2 (1 - \alpha_f) (1 - 2\alpha_l \alpha_f)^2 \end{aligned}$$

Denote $x = \alpha_l \alpha_f$ and $y = 1 - \alpha_f$. Then

$$\begin{aligned} & \text{Val}[w'] - \text{Val}[w] > 0 \impliedby \\ & Q[x; y] \equiv -(2y) x^2 + x \left(-3 + 4y^2 + 2y \right) + (3 - 4y) > 0 \end{aligned}$$

Q is a concave quadratic in x. The end points for x are given by $x = \alpha_l \alpha_f \in [0, \alpha_f^2] = [0, (1 - y)^2]$. Given that $y = 1 - \alpha_f < 0.5$, at both end points $x = 0$ and $x = (1 - y)^2$, $Q[0; y]$ and $Q[(1 - y)^2; y]$ are positive. So $Q[x; y]$ is positive.

The difference in mean is

$$\mathbb{E}[w'] - \mathbb{E}[w] = \alpha_f \alpha_l (v_f + v_l - 2(1 - \alpha_f)\beta_l) - c$$

which can be positive or negative. Pick any $\alpha_f > 0.5$, any $\alpha_l < \alpha_f$, any $\beta_f > v_f > \beta_l - 2v_l > 0$, and let $c = \alpha_f \alpha_l (v_f + v_l - 2(1 - \alpha_f)\beta_l) - x$. (Note that $\mathbb{E}[w'] - \mathbb{E}[w] = x$) This clearly implies

all parametric conditions except $\alpha_l\alpha_f > \frac{c}{v_f} > \alpha_l\alpha_f^2$. Note

$$\alpha_l\alpha_f > \frac{\alpha_f\alpha_l\left(v_f + v_l - 2(1-\alpha_f)\beta_l\right) - x}{v_f} > \alpha_l\alpha_f^2$$
$$\iff 0 < 2\beta_l - \frac{v_l}{(1-\alpha_f)} + \frac{x}{(1-\alpha_f)} < v_f$$

Then by assuming

$$0 < 2\beta_l - \frac{v_l}{(1-\alpha_f)} < v_f$$

x can have positive or negative sign. For example, $\alpha_f > 0.5$, $\alpha_l < \alpha_f$, $\beta_f > v_f > \beta_l > 0$ and $v_l < \min\left\{\frac{1}{2}, 2\left(1-\alpha_f\right)\right\}\beta_l$ satisfies all conditions.

A.2.2 Epidemics and protective measures

(Proof of Proposition 7) *Stability part:*

The payoff to a b-type from having no matches is $V_0 = -\kappa\eta$. The payoff to a b-type from being matched to a_j if a_j has no other match is $V_0 + V_j$ where $V_j = v_j - 1 - \kappa(1-\eta)\tau\eta$. The payoff to a b-type from being matched to a_j if a_j has one more match is $V_0 + V_j - \Delta$ where $\Delta = \kappa(1-\eta)\tau(1-\eta)\eta\tau$.

It is easy to see that the stability is characterized as follows. $V_1 < 0$, there are no links. If $V_1 > 0 > \max\{V_2, V_1 - \Delta\}$, then one matched to a_1, the other no matches. If $V_2 = \max\{V_2, V_1 - \Delta\} > 0$, then one to a_1 one to a_2. If $V_1 - \Delta = \max\{V_2, V_1 - \Delta\} > 0$, then both matched to a_1. Regarding m, these bounds correspond to

$$V_i < 0 \iff m_i^* \equiv \frac{v_i - 1}{\kappa(1-\eta)\eta\tau_0} < m$$
$$V_1 - \Delta < 0 \iff m^* \equiv \frac{\sqrt{1 + 4\frac{v_1 - 1}{\kappa\eta}} - 1}{2(1-\eta)\tau_0} < m$$
$$V_1 - \Delta < V_2 \iff m^{**} \equiv \sqrt{\frac{v_1 - v_2}{\kappa\eta(1-\eta)^2\tau_0^2}} < m$$

Then the conditions are: If $m > m_1^*$, there are no links. If $m_1^* > m > \max\{m_2^*, m^*\}$, then one matched to a_1, the other no matches. If $m_2^* > m > m^{**}$, then one to a_1 one to a_2. If $\min\{m^{**}, m^*\} > m$, then both matched to a_1. Note

$$m_2^* > m > m^* \iff V_2 > 0 > V_1 - \Delta \implies V_2 > V_1 - \Delta \iff m > m^{**}$$

meaning $m_2^* > m^* \implies m^* > m^{**}$ by picking $m = m^* + \epsilon$. Also,

$$m_2^* < m < m^* \iff V_2 < 0 < V_1 - \Delta \implies V_2 < V_1 - \Delta \iff m < m^{**}$$

meaning $m_2^* < m^* \implies m^* < m^{**}$by picking $m = m^* - \epsilon$. So we have either $m_2^* > m^* > m^{**}$ or $m_2^* < m^* < m^{**}$.

Consider $m_2^* < m^* < m^{**}$. If $m > m_1^*$, there are no links. If $m_1^* > m > m^*$, then one matched to a_1, the other no matches. If $m^* > m$, then both matched to a_1.

Next consider $m_2^* > m^* > m^{**}$. If $m > m_1^*$, there are no links. If $m_1^* > m > m_2^*$, then one matched to a_1, the other no matches. If $m_2^* > m > m^{**}$, then one to a_1 one to a_2. If $m^{**} > m$, then both matched to a_1.

Thus by defining $m_1 \equiv m_1^*$, $m_2 \equiv \max\{m_2^*, m^*\} \leq m_3 \equiv \min\{m^{**}, m^*\}$, we complete the proof.

Welfare part:

When both b-types are matched to the same a-type, the number of infections X, its mean, and its variance are

$$\begin{aligned}
X = \mathbb{B}[1,\eta] + \Big[& \left(\eta^3 + \eta^2(1-\eta)(4-\tau)\tau + 3(1-\eta)^2\eta\tau^2\right) \circ 3 \\
& \oplus \left(\eta^2(1-\eta)(1-\tau)(3-\tau) + 4\eta(1-\eta)^2\tau(1-\tau)\right) \circ 2 \\
& \oplus \left(\eta(1-\eta)^2(1-\tau)(3-\tau)\right) \circ 1 \oplus (1-\eta)^3 \circ 0 \Big] \\
\mathbb{E}[X] = {} & \eta(4 + \tau(1-\eta)(4 + (2-3\eta)\tau)) \\
\mathrm{Var}[X] = {} & (1-\eta)\eta\Big(12\tau(\tau+1) + 4 - \eta\tau\Big(\tau\big((2-3\eta)^2(1-\eta)\tau^2 \\
& + 8(1-\eta)(2-3\eta)\tau + 45 - 34\eta\big) + 16\Big)\Big)
\end{aligned}$$

When b-type agents are matched to separate a-type agents

$$\begin{aligned}
X = {} & \left[\left(\eta^2 + 2\eta(1-\eta)\tau\right) \circ 2 \oplus (2\eta(1-\eta)(1-\tau)) \circ 1 \oplus (1-\eta)^2 \circ 0\right] \\
& + \left[\left(\eta^2 + 2\eta(1-\eta)\tau\right) \circ 2 \oplus (2\eta(1-\eta)(1-\tau)) \circ 1 \oplus (1-\eta)^2 \circ 0\right] \\
\mathbb{E}[X] = {} & 4\eta\left(1 + (1-\eta)\tau\right) \\
\mathrm{Var}[X] = {} & 4\eta(1-\eta)\left(1 + (3-4\eta)\tau + 2\eta(1-\eta)\tau^2\right)
\end{aligned}$$

When only one b-type agent is matched to an a-type agent, and the others have no matches,

$$
\begin{aligned}
X &= \left[\left(\eta^2 + 2\eta(1-\eta)\tau\right) \circ 2 \oplus \left(2\eta(1-\eta)(1-\tau)\right) \circ 1 \oplus (1-\eta)^2 \circ 0\right] + \mathbb{B}[2,\eta] \\
\mathbb{E}[X] &= 2\eta\left(2 + (1-\eta)\tau\right) \\
\mathrm{Var}[X] &= 2\eta(1-\eta)\left(2 + (3-4\eta)\tau + 2\eta(1-\eta)\tau^2\right)
\end{aligned}
$$

When there are no matches, the X is $\mathbb{B}[4,\eta]$. The expectation is 4η. The variance is $4\eta(1-\eta)$.

Focus on the case of $m_2^* > m^* > m^{**}$. As m goes down, at $m = m_1 = m_1^*$, the network switches from empty to having one link. Expectation and variance clearly increase. At $m = m_2 = m_2^*$, the network switches from one match to two separate matches. Then expectation and variance change by

$$
\begin{aligned}
\Delta\mathbb{E}[X] &= \left[4\left(\eta + (1-\eta)\tau\eta\right)\right] - \left[2\left(2\eta + (1-\eta)\tau\eta\right)\right] = 2(1-\eta)\tau\eta > 0 \\
\Delta\mathrm{Var}[X] &= \left[4(1-\eta)\left(\eta + (3-4\eta)\tau\eta + 2(1-\eta)\tau^2\eta^2\right)\right] \\
&\quad - \left[2(1-\eta)\left(2\eta + (3-4\eta)\tau\eta + 2(1-\eta)\tau^2\eta^2\right)\right] \\
&= 2(1-\eta)\left((3-4\eta)\tau\eta + 2(1-\eta)\tau^2\eta^2\right) > 0
\end{aligned}
$$

At $m = m_3 = m^{**}$ expectation and variance change by

$$
\begin{aligned}
&\Delta\mathbb{E}[X] = \eta(4 + \tau(1-\eta)(4 + (2-3\eta)\tau)) - 4\left(\eta + (1-\eta)\tau^2\eta\right) \\
&= (1-\eta)(2-3\eta)\tau^2\eta > 0 \\
&\Delta\mathrm{Var}[X] = (1-\eta)\eta\Bigg(12\tau(\tau+1) + 3 - \eta\tau\Bigg(\tau\big((2-3\eta)^2(1-\eta)\tau^2 \\
&+ 8(1-\eta)(2-3\eta)\tau + 45 - 34\eta\big) + 16\Bigg)\Bigg) + \eta(1-\eta) \\
&- 4\eta(1-\eta)\big(1 + (3-4\eta)\tau + 2\eta(1-\eta)\tau^2\big) > 0 \iff \\
&- (2-3\eta)^2\tau^4\eta^2 + 8(2-3\eta)\tau^2\eta + \frac{\eta\left(12 - 53\eta + 42\eta^2\right)}{1-\eta} > 0
\end{aligned}
$$

where the last inequality holds by $\eta < 1/4$ and $\tau^2\eta > 0$.

Next consider $m_2^* < m^* < m^{**}$. At $m = m_1^*$, expectation and variance clearly increase. At $m_2 = m_1 = m^*$, network switches from one link to both b-type agents having a match with a_1. Then expectation and variance change by the sum of the two ΔExp and ΔVar terms above, which are both positive. So both changes are positive.

A.2.3 Supply chains and subsidies

(Proof of Proposition 8) Corollary of Propositions 12, 13, 14.

Proposition 12. *If $\frac{\alpha_{u_2}}{\alpha_{u_1}} > 1-\delta+\frac{\delta}{2}$ and $k > \delta^2 p'$, downstream firms choose separate suppliers. Off-the-path, if both downstream firms choose u and u has low cost, it produces 1 and supplies at most one downstream firm.*

If $\frac{\alpha_{u_2}}{\alpha_{u_1}} < 1-\delta+\frac{\delta}{2}$ and $k > \delta^2 p'$, both downstream firms choose u_1. If u_1 has low cost, it produces 1 and supplies at most one downstream firm.

If $k < \delta^2 p'$, both downstream firms choose u_1. If u_1 has low cost, it produces 2 and supplies each downstream firm that has a low cost.

Proof. Take u and consider $D_u = \{d\}$. Conditional on good shocks and being supplied, the downstream firm has ex-post payoff $-c+p-p'$ from production, so it produces if supplied. The supplier u has interim payoff $-k+\delta p' > 0$ from production so it produces and supplies.

Consider $D_u = D$. Conditional on good shocks and being supplied, d has ex-post payoff $-c+p-p'$. The supplier u can produce 1 or 2. If it produces 1, it has interim payoff $-k+2\delta(1-\delta)p'$. If it produces 2, it has interim payoff $-2k+\left(\delta^2+2\delta(1-\delta)\right)p'$. Then it produces 1 if and only if $k > \delta^2 p'$.

Then under $k < \delta^2 p'$, both downstream firms choose u_1 as $\alpha_{u_1} > \alpha_{u_2}$. Under $k > \delta^2 p'$, if both downstream firms choose u_1 they each have ex-ante payoff $\alpha_{u_1}\delta\left(1-\delta+\frac{\delta}{2}\right)(-c+p-p')$. If they choose separate suppliers, the one with smaller payoff has ex-ante payoff $\alpha_{u_2}\delta(-c+p-p')$. Then they choose separate suppliers if and only if $\frac{\alpha_{u_2}}{\alpha_{u_1}} > 1-\delta+\frac{\delta}{2}$.

Proposition 13. *In the presence of subsidies, each downstream firm $d \in D$ receives $s_d = c' - p_D$ if its supplier has low cost. Upstream firms do not receive subsidies. Both downstream firms choose u_1.*

Proof. Given $e_d = 0$ and $e_u = k_u$, welfare is given by $\sum_{d\in D} pq_d - \sum_{u\in U} k_u q_u$. Since $k' > p$, w is maximized by $q_d = 1$ if $k_{u_d} = k$ and $q_d = 0$ otherwise. The minimal subsidies that implement this outcome is $s_d = c' - p_D$ if $c_d = c'$, which induces d to produce, and all other subsidies are 0. Then an upstream firm u with two downstream buyers and a good shock has payoff $q_u(-k+p')$ from producing q_u, so it produces 2. This means both downstream are supplied conditional on their supplier getting a good shock so they both choose u_1 as $\alpha_{u_1} > \alpha_{u_2}$.

Proposition 14. *Suppose that $\alpha_{u_2} \approx \alpha_{u_1} = \alpha < 1 - \frac{\delta}{2(1-\delta)}$ and $k > \delta^2 p'$. Expectation and variance of welfare is larger in the presence of subsidies than in the absence of subsidies.*

Proof. In the absence of interventions,

$$\begin{aligned}
w &= (\alpha_{u_1}\delta \circ (p-k) \oplus \alpha_{u_1}(1-\delta) \circ (-k) \oplus (1-\alpha_{u_1}) \circ 0) \\
&\quad + (\alpha_{u_2}\delta \circ (p-k) \oplus \alpha_{u_2}(1-\delta) \circ (-k) \oplus (1-\alpha_{u_2}) \circ 0) \\
\mathbb{E}[w] &= (\alpha_{u_1} + \alpha_{u_2})(\delta p - k) \\
\mathrm{Var}[w] &= \alpha_{u_1}\delta(1-\alpha_{u_1}\delta)p^2 + \alpha_{u_1}(1-\alpha_{u_1})k^2 - 2\delta\alpha_{u_1}(1-\alpha_{u_1})pk \\
&\quad + \alpha_{u_2}\delta(1-\alpha_{u_2}\delta)p^2 + \alpha_{u_2}(1-\alpha_{u_2})k^2 - 2\delta\alpha_{u_2}(1-\alpha_{u_2})pk
\end{aligned}$$

In the presence of interventions,

$$\begin{aligned}
w' &= \alpha_{u_1} \circ 2(p-k) \oplus (1-\alpha_{u_1}) \circ 0 \\
\mathbb{E}[w'] &= 2\alpha_{u_1}(p-k) \\
\mathrm{Var}[w'] &= \alpha_{u_1}(1-\alpha_{u_1})4(p-k)^2
\end{aligned}$$

Clearly $\mathbb{E}[w'] > \mathbb{E}[w]$. Let $\alpha \approx \alpha_{u_i}$. Then

$$\begin{aligned}
&\mathrm{Var}[w'] > \mathrm{Var}[w] \iff \alpha_{u_1}(1-\alpha_{u_1})4(p-k)^2 > \\
&\alpha_{u_1}\delta(1-\alpha_{u_1}\delta)p^2 + \alpha_{u_1}(1-\alpha_{u_1})k^2 - 2\delta\alpha_{u_1}(1-\alpha_{u_1})pk \\
&+ \alpha_{u_2}\delta(1-\alpha_{u_2}\delta)p^2 + \alpha_{u_2}(1-\alpha_{u_2})k^2 - 2\delta\alpha_{u_2}(1-\alpha_{u_2})pk \\
&\iff \left(2 - \delta\frac{1-\alpha\delta}{1-\alpha}\right) + \left(\frac{k}{p}\right)^2 - (4-2\delta)\left(\frac{k}{p}\right) > 0 \impliedby 1 - \frac{\delta}{2(1-\delta)} > \alpha
\end{aligned}$$