Network Hazard: Resilience and Moral Hazard in Network Formation*

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Abstract

I develop a novel framework for studying network formation with continuum populations. I use the framework to examine contagion and resilience in endogenous networks, with applications to misinformation, supply chains, financial contagion, and epidemics. I analyze the equilibrium effects of policies that mitigate contagion externalities and find that interconnectedness and concentration increase in response to interventions. This general equilibrium response negates the benefits of interventions and creates a "network hazard." Despite interventions that mitigate contagion ex post, contagion and volatility are exacerbated and welfare and resilience are reduced ex ante.

JEL classifications: D85

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

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1 Introduction

The resilience of global economic and political institutions that have shaped the last century faces unprecedented challenges in a changing world. The Future of Growth Report 2024 by The World Economic Forum identifies resilience as one of the four pillars of the future of growth. In the resilience pillar, networks and concentration take center stage, contributing to half of the criteria that make up the resilience scores of countries.[1] The report calls into question national resilience strategies as "...the world grapples with the enormity of global challenges such as climate change, peace and security, financial and economic stability, and the volatility of global health shocks,..."

Networks are central to understanding and addressing these threats to resilience, which itself matters for growth and welfare. Misinformation spreads through social networks, exacerbates polarization, erodes social trust, and threatens democratic institutions (Vosoughi et al. (2018)). The interconnectedness of global supply chains makes them vulnerable to disruptions caused by climate change and geopolitical risks (Katsaliaki et al. (2022)). Complex interdependencies in financial networks create systemic risks, such as those experienced in the 2008 financial crises, as well as those prevented by invoking the systemic risk exception to the deposit insurance limit in the 2023 SVB crises (Acemoglu et al. (2012)). The Covid-19 pandemic demonstrated how global travel patterns and human interaction networks facilitate the rapid transmission of infectious diseases across borders (Kissler et al. (2020)).

The unifying theme of these networked threats is contagion. In assessing policies that mitigate contagion, such as bailouts, systemic risk exceptions, capital regulations, subsidies to critical firms with strategic importance, content moderation and censorship on Internet platforms, mask mandates, and lockdowns, it is essential to understand the endogenous nature of these networks to understand how network topologies react to such policies. It is not possible to accurately predict and assess the consequences of policies without taking into account the endogenous reactions of network structures to mitigation policies.

There is an inherent challenge to evaluating the implications of policies that mitigate network externalities in endogenous networks. Network externalities such as contagion make network formation analysis as intractable as it is important. Each connection in a network creates externalities on incident connections, iteratively reaching a complex and expansive web of

[1] The report is available at https://www3.weforum.org/docs/WEF_Future_of_Growth_Report_2024.pdf Among a total of 25 criteria, the criteria related to networks and contagion are export product concentration, food supply concentration, commodity supply concentration, technology supply concentration, bank concentration, financial system resilience, bank system default risk, cybersecurity index, social polarization, political stability, health workers, and hospital beds.

externalities across all connections. This complexity makes network formation a challenging dimension to examine. However, absent spillovers on incident connections, each connection could be analyzed separately and there would not be any need for a network analysis. So, the very reason to study networks is the reason that makes network formation results, and so relevant policy suggestions, difficult to obtain.

I develop a framework for studying network formation in the presence of a general class of externalities. I employ a continuum population that provides the necessary tractability. The connection of an agent i with an agent j, among the continuum of connections of j, has negligible impact on externalities passed through j. Therefore, i takes the externalities through its potential connections as given and simply contrasts them with the potential connection benefits. The connections that i choose do not have an effect on the aggregate structure of the network. The infinitesimal decisions aggregate up to endogenous aggregate structure and externalities. The agents are *network-takers*. I call this approach *competitive network formation*.

I examine the consequences of interventions in the presence of competitive network formation in a hierarchical economy. Each agent chooses whether to connect to its designated "predecessor" to obtain a given benefit. The resulting network consists of trees of various depths, one of which is shown in Figure 1.

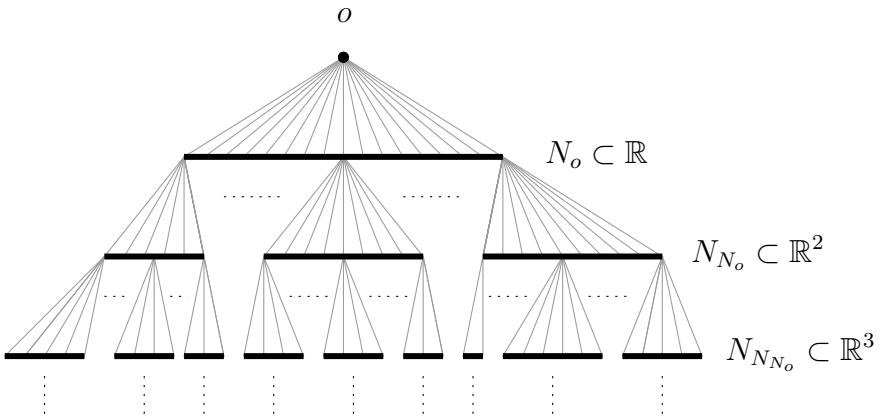

Figure 1: Illustration of a tree in a network which emerges from a strategy profile

After connections are formed, each agent gets an exogenous shock. Each shock starts to spread through connections, decaying at each step at given rates, while propagating negative externalities and harming agents. As a given shock travels across connections, it reaches a higher 'order of contagion' at each step. The shocks resemble the baseline centralities, and the transmission rates resemble the damping factors in the Katz-Bonacich centrality framework.

Before shocks, when more "successors" of an agent choose to connect, the expected centrality of the agent increases at the rate of the expected centrality of connecting successors, which are determined in equilibrium. The net expected benefit of the connection reduces for each successor as more successors connect. This force disciplines connection rates throughout the network. When an agent decides to connect to its predecessor, it understands the expected cost of contagion but does not internalize the exposure it creates between its own existing direct and indirect connections and the direct and indirect connections of its predecessor. This externality binds the connection decisions of all directly or indirectly connected agents to each other.

After connections form, a welfare-maximizing authority can intervene to address contagion and negative externalities. The authority can deploy policy instruments to reduce the transmission rates and the mean of shocks. The authority lacks commitment, and interventions are costly. Moral hazard weakens agents' incentives to protect themselves against contagion in their connection decisions. Interconnectedness and concentration increases, expected welfare decreases, expected cost of contagion increases, and volatility in welfare increases, despite the mitigating effects of interventions. These negative consequences on welfare and resilience constitute what I call "network hazard."

Network hazard emerges not only because the agents form more links, but primarily because they increase the concentration of the network. Absent interventions, the market disciplines connection rates towards agents in key locations in the network, as these key positions are unintentionally conduits of contagion. Mitigating contagion makes agents less disciplined with not only their connections but also with the connections of their connections, etc. Consequently, agents who are less concerned with contagion *through* key agents increase the concentration of connections around key locations. This indirect form of moral hazard is specific to network formation.

Suppressing the potential of key agents to be conduits of contagion increases their potential to originate widespread contagion as an equilibrium response. An endogenous substitution occurs between exposure to high- and low-order contagion. Due to the iterative nature of contagion, the magnitude of high-order exposures relative to the magnitude of low-order exposures decreases when contagion rates are reduced. Therefore, the substitution pushes the high-order exposures down and the low-order exposures up. Consequently, the exposures of 'peripheral' agents to key agents increase, as these exposures are of a lower order than the exposures of peripheral agents to each other *through* the key agents. Higher aggregate exposure to key agents results in higher volatility as the system grows more concentrated around key agents and becomes more vulnerable to the idiosyncratic risks of key agents. Resilience is reduced.

For example, more effective or widely available vaccines can encourage social gatherings, which can potentially lead to superspreader events. A large bank's core position can give it a too-interconnected-to-fail status, which can prompt an additional too-big-to-fail status at the expense of tax payers. Subsidies that protect key firms in production networks can reduce competition and increase industry concentration, increasing the aggregate dependence on a few critical firms.

I also examine alternative timelines of events, as well as an alternative policy tool which severs connections. Similar adverse consequences emerge. Finally, I present alternative scenarios in self-contained models of coordination games, epidemics, and supply chains. These models feature more institutional details and different contagion dynamics, including threshold contagion and independent cascades. The results are robust to these variations.

Related literature Granular networked interactions pose externalities that differ from those in standard economic models. As Galeotti and Goyal (2010) and Manea (2021) highlight, central and bottleneck positions in a network can disproportionately affect aggregate outcomes. My work shows how the influence of key positions in the network can increase in response to future interventions.

There is growing work on interventions in networks. Galeotti, Golub and Goyal (2020) examine optimal interventions subject to a budget within fixed networks in which a coordination game is played.[2] Talamàs and Vohra (2020) studies infectious diseases, independent cascades, pairwise stable networks, and the impact of the density of connections. Erol (2019) studies bailouts, core-periphery financial networks, strongly stable networks, threshold contagion, and second-order externalities. The scope of my work is different. I provide a novel noncooperative framework and explore the consequences of various interventions through centrality and concentration.

Empirical validation of network hazard presents challenges, as events such as pandemics, tsunamis, or financial crises are rare and the anticipation of interventions is generally unobservable. Celdir and Erol (2023) provide empirical support by showing that higher vaccination rates during the Covid-19 pandemic led to higher infection rates, mediated by increased foot traffic at crowded points of interest. In the context of financial networks, Anderson, Erol and Ordoñez (2022) show that interbank deposit networks became more

[2]Several other related studies examine regulations and interventions in fixed financial networks. For example, 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.

concentrated around regional Federal Reserve Banks following the establishment of the Federal Reserve System and its discount lending facilities in 1914.

Graphons are continuum objects that represent the limits of dense graphs. Erol, Parise and Teytelboym (2023) and Parise and Ozdaglar (2023) explore the use of graphons in economic theory as statistical tools to generate random networks.[3] Although sparse graphs are more suitable for most social and economic networks, continuous objects that are economically interpretable and represent sparse graphs are limited. Graphings constitute a candidate, but they represent 'very' sparse graphs with a uniform $O(1)$ bound on degrees (see Lovász (2012)). I introduce a new class of graphs that allow for an arbitrary degree of density or sparsity, have clear economic interpretations, and provide tractability in the analysis of network formation in the presence of a large class of network externalities.

The hierarchical structure I use has similarities to Elliott, Golub and Leduc (2022). In their model, agents face upstream threshold contagion. The top node in the tree represents the aggregate outcome. In my model, agents face linear contagion, both upstream and downstream. The aggregate outcome is determined by the aggregate condition of the bottom nodes. In addition, I study the formation of connections, consider a continuum economy, and incorporate interventions.

Structure of the paper Section 2 presents the hierarchical economy. Section 3 examines the equilibria, interventions, and network hazard. Section 4 explores more detailed intervention tools. Section 5 provides applications in coordination games, epidemics, and supply chains to establish robustness against institutional details. Section 6 offers concluding remarks. Appendix A presents a general framework for network formation in a continuum economy and provides examples and applications. Appendix B contains proofs.

2 Model

The economy is comprised of *bud* and *leaf* agents organized in a hierarchy. A fixed integer $T \geq 1$ is the *depth* of the hierarchy, $L \equiv [0,\mu]^T \subset \mathbb{R}^T$ is the set of *leaf* agents, and $B \equiv \cup_{t=0}^{T-1}[0,\mu]^t$ is the set of *bud* agents. I use the convention that $[0,\mu]^0 \subset \mathbb{R}^0$ is a singleton consisting of the empty vector $o \equiv ()$. For each bud $j \in B$, $S_j = \{j\} \times [0,\mu]$ is the set of *successors* of j. For each $i \in S_j$, j is called the *predecessor* of i and is denoted by $j = i^*$.

[3] See Erol and García-Jimeno (2024) and D'Erasmo, Erol and Ordoñez (2024) for tractable uses of continuum populations in the formation of dense networks.

Each agent $i \neq o$ chooses whether to connect to its predecessor i^*, a decision 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 connected successors of i. The measure of N_i is called the connection rate to i, denoted by n_i.[4] The network emerging from any strategy profile is a forest of rooted directed trees.[5] One example is illustrated in Figure 1.

After connections are formed, each i experiences a shock ω_i, termed the original potential of i. These shocks are positive, uniformly bounded, independent, and have a mean of ω. The potential spreads throughout the network. Let p_i denote the potential of i. For any agent i, the potential is given by[6]

$$p_i = \omega_i + \delta a_i p_{i^*} + \alpha \int a_j p_j \mathtt{d}j. \tag{1}$$

Here, $\delta > 0$ represents the downstream contagion rate and $\alpha > 0$ represents the upstream contagion rate. The mean of the shocks ω is called the origination rate. Together, these parameters are called contagion rates.

The agent $i = (i^*, l_i)$ receives a benefit of $\upsilon(i) = \upsilon - \varepsilon l_i$ from its connection to its predecessor i^*, where $\varepsilon \geq 0$ and $\upsilon > \mu\varepsilon$ are constants. The term ε parameterizes the degree of heterogeneity and υ is the (maximum) benefit of the connection. The connection comes at the cost of exposure to the potential of the predecessor i^*. The payoff of agent i is given by $u_i = a_i \left(\upsilon(i) - \delta p_{i^*}\right)$.

An equilibrium is defined as a Nash equilibrium which is stable, which means that no vanishingly small group of agents can improve their payoffs by deviating jointly. The stability requirement serves to exclude knife-edge Nash equilibria, which are artifacts of the continuum and lack a generic discrete counterpart.[7] I call the model described up to this point the *absence of interventions*.

Welfare and interventions The model with the *presence of interventions* incorporates a specific technology for each contagion rate $\theta \in \{\omega, \alpha, \delta\}$. The corresponding technology can

[4]For simplicity, each N_i is assumed to be measurable. Appendix A provides details on how the payoffs can be defined without imposing measurability.

[5]Rooted directed trees are tree networks wherein all links are oriented in one direction away from a given node. This node is called the root.

[6]The smallest positive solution to the system of equations (1) constitutes the vector of potentials. This solution can be obtained iteratively. Each i starts with its original potential ω_i in stage $t = 0$, and at each subsequent stage t, the potentials are updated as $p_{o,t+1} = \omega_o + \delta p_{i^*,t} a_i + \alpha \int p_{j,t} a_j \mathtt{d}j$. The limit of this process, $p_i = \lim_{t\to\infty} p_{i,t}$, yields the final potential.

[7]The vanishingly small group is also assumed to be measurable, restricted to deviations that result in measurable subsets of connected successors.

reduce the status-quo value of θ to a fraction $\phi_\theta \in (0,1)$.[8] Implementing the technology has a fixed public cost $C_\theta > 0$. The principal decides whether to intervene after the connections are formed before the shocks are realized. For simplicity, I analyze each technology separately.

The set of leaves is infinitely larger than the set of branches, so the payoffs of the leaves drive the welfare analysis. The set L is endowed with the Lebesgue measure λ_T in $\mathbb{R}^T$. Given the reduced or original contagion rates, the aggregate payoff of agents is

$$U = \int_L u_i \mathtt{d}\lambda_T(i).$$

Welfare W is equal to the aggregate payoffs of agents U net of the costs of the intervention, if any.

Interpretations

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

For example, a payment failure within a financial network can trigger a series of additional failures across a chain of exposures. The extent of these failures depends on the size of the shortfalls. So, the potential represents the variable magnitude of a cost. Similarly in supply chains, significant reductions in production capacity or demand can lead to further disruptions, which are proportional to the extent of 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 increases the likelihood that trusting contacts will also adopt it.

My framework examines the interplay between interconnectedness in endogenous networks and mitigation measures against contagion. In this context, buds represent pivotal agents with varying degrees of interconnectedness determined by endogenous connections decisions. For example, in financial networks, buds can be core banks, while leaves are peripheral banks.

[8] In the case of ω, the corresponding technology reduces each shock ω_i to $\phi_\omega \omega_i$. Consequently, the post-intervention shocks are positive.

In supply chains, buds can be upstream suppliers to downstream producers or consumers represented by the leaves. In epidemics, buds might be individuals in high-traffic locations visited by leaves, such as many customers interacting with cashiers in grocery stores, while cashiers also interact with their managers. In social networks, buds can be specific platforms that house many public figures, each of whom has a large number of followers.

Downstream and upstream contagions often exhibit structural differences. So, I consider two separate rates, α and δ for upstream and downstream contagion. In social networks, the interactions between celebrities and fans are asymmetric. In supply chains, suppliers face demand disruptions from their buyers, while buyers are vulnerable to production disruptions from their suppliers. In core-periphery financial networks, exposures between the core and the periphery are asymmetric.

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 the contagion within its respective context.

Connection to network games

It is known that Katz-Bonacich centrality and Eigenvector centrality are connected to network games. Example 1 in Appendix A presents a mapping. In coordination games with quadratic loss utility functions, the best responses are linear. The equilibrium strategy of each agent thus takes the form of a variant of Katz-Bonacich centrality, which is defined by a linear relationship between centralities of connected agents. See Ballester, Calvó-Armengol and Zenou (2006) and Galeotti, Goyal, Jackson, Vega-Redondo and Yariv (2010) for more on network games and centrality measures.

Non-hierarchical economies

A hierarchical network is well suited to study externalities, as it has high a diameter and it is acyclic. Long paths allow for examination of externalities between distant agents. These paths do not intersect, as the network is acyclic. So, externalities are disentangled.

This restriction can be lifted, and agents can be allowed to connect to any other agent, not only their designated predecessor. There is endemic multiplicity in the unrestricted model.

Non-hierarchical equilibrium networks emerge, including cyclic networks. In this sense, the hierarchy restriction can be seen as an equilibrium selection. See Appendix A for the broader setting and an illustration of a cyclic equilibrium network.

3 Network hazard

Buds can choose to become roots if they find that the cost of downstream contagion through their predecessor is too high. Hence, the decision of agent i to connect to i^* depends on i^*'s decision to connect to $(i^*)^*$, and so on. Inductively, agents consider the network externalities that originate from distant agents while making connection decisions. The continuum structure simplifies the analysis of these externalities as no infinitesimal successor can influence the potential of its predecessor.

Theorem 1. ***(Network hazard)*** *Assume $\varepsilon > 0$. There exists a unique equilibrium.*[9]

Assume $\frac{1}{2}\frac{v}{v+\omega} < \delta < \frac{v}{v+\omega}$ and consider an intervention with a contagion rate $\theta \in \{\alpha, \delta, \omega\}$.[10,11]

The expected welfare is lower and the variance of welfare is higher in the presence of interventions than in the absence of interventions, provided that depth T is sufficiently large, intervention cost C_θ is sufficiently small, and heterogeneity ε is sufficiently small.

For any given network, an interim intervention reduces the rate of contagion and creates an interim welfare gain. If the cost of the intervention is not too high, there is an intervention. However, agents anticipate the intervention at the earlier stage when they form their connections. The marginal successors are indifferent under the anticipation of interventions, and so the ex-ante welfare gains from interventions are on the order of ε excluding the cost of interventions. So, expected welfare decreases if heterogeneity ε is low.

Volatility and resilience are economically significant. For example, in the context of epidemics, sharp increases in infection rates can lead to hospitalizations that exceed hospital capacity and strain 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. Disruptions in key firms in global supply

[9] When $\varepsilon = 0$, the equilibrium network *structure* is unique. The equilibrium strategy profile is unique up to measure-preserving permutations of the connection decisions of successors of each predecessor.

[10] When $\delta < \frac{v}{v+\omega}$, the network involves trees of various depths, including one with depth T. When $\delta > \frac{v}{v+\omega}$, the network collapses and all trees have depth 1. Although such a discontinuous change is interesting, high orders of contagion are not present when all trees are of depth 1. So, I ignore this case in the welfare analysis, as my focus is resilience.

[11] When $\delta < \frac{1}{2}\frac{v}{v+\omega}$, reducing δ reduces the welfare variance. The rest of the result holds regardless.

chains can create global cascades that are difficult to predict or measure in advance, as information on the resilience of foreign companies may be limited.

The presence of interventions increases volatility due to a trade-off between various orders of contagion. For example, consider T'th-order downstream contagion from o down to the leaves, as well as $2T$'th-order contagion, starting with T'th-order upstream contagion from the leaves to o followed by T'th-order downstream contagion from o to the leaves. Due to the sequential nature of contagion itself, high orders of contagion are more elastic to contagion rates than low orders because high orders are obtained by amplifying low orders with transmission rates α and δ. Therefore, reducing contagion rates reduces the relative magnitude of high orders of contagion over low orders. But the magnitudes of high- and low-order contagion face trade-offs through the connection incentives. The expected costs of various orders of contagion add up to the net expected connection benefit of a marginal agent. The sensitivity of this benefit is bounded by heterogeneity ε. Therefore, when heterogeneity is low, reducing contagion rates increases the magnitude of low orders of contagion and reduces the magnitude of high orders of contagion. In particular, the downstream contagion from a root to the leaves increases and balances out the reduction in higher-order contagion that starts with upstream contagion to the root, followed by downstream contagion from the root.

This force is more clearly visible in the simple case $T = 1$. In the case of $T = 1$, there is a single root o and a measure μ of leaves. The set of connected leaves exposes o to a first-order upstream contagion of magnitude $\alpha\omega n_o$. This magnitude is amplified by the downstream contagion rate δ, and connected leaves expose each other to second-order contagion of magnitude $\delta\alpha\omega n_o$. In addition, the root exposes the leaves to its shock ω_o, which constitutes a first-order downstream contagion of magnitude $\delta\omega_o$ for each of the n_o-measure of connected leaves. These magnitudes are geometrically amplified by the feedback between potentials by a total factor of $\frac{1}{1-\alpha\delta n_o}$. This amplifying factor is the counterpart of the Leontief inverse matrix.

Second-order contagion, $\texttt{SC} \equiv \frac{\delta\alpha n_o}{1-\alpha\delta n_o}\omega$, and first-order contagion, $\texttt{FC} \equiv \frac{\delta}{1-\alpha\delta n_o}\omega$, constitute the expected downstream contagion $\delta\mathbb{E}[p_o^*]$ from the root to a leaf, which equilibrates the connection benefit of the marginal leaf, $\upsilon - \varepsilon n_o$:

$$\upsilon - \varepsilon n_o = \texttt{FC} + \texttt{SC} = \texttt{FC}\left(1 + \frac{\texttt{SC}}{\texttt{FC}}\right)$$

Since $\texttt{SC}$ is more elastic than FC, $\frac{\texttt{SC}}{\texttt{FC}}$ weakly decreases when the contagion rates are reduced. So $\frac{\texttt{FC}}{\upsilon-\varepsilon n_o}$ increases weakly. Then the magnitude of aggregate first-order downstream contagion, $n_o\texttt{FC}$, increases, provided that ε is small. Since the aggregate exposure to o increases, volatility

increases.

In the remainder of the section, I examine the absence of interventions and build up to the proof of Theorem 1.

3.1 Equilibrium network

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

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

The unique equilibrium network is pinned down by the following quantities:

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

Proposition 1. ***(Equilibrium network)*** *In equilibrium, as $\varepsilon \to 0$, for any T, each $i \in B \backslash \mathbb{R}^{T-1}$ has $r + O(\varepsilon)$ connections if it is a root and $b + O(\varepsilon)$ connections if it is a branch. For $i \in \mathbb{R}^{T-1}$, these values are $r' + O(\varepsilon)$ and $b' + O(\varepsilon)$. Specifically:*

- *If $\rho > \omega + \upsilon + |O(\varepsilon)|$, 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 $\rho - O(\varepsilon)$. Each connected leaf has potential $\omega + \upsilon - O(\varepsilon)$.*
- *If $\omega + \upsilon \geq \rho > \omega + |O(\varepsilon)|$, then $r, r' > 0 = b = b'$. The network consists of rooted trees of depth 1. Each root has an expected potential of $\rho - O(\varepsilon)$. Each non-root agent has an expected potential of $\omega + \upsilon - O(\varepsilon)$.*
- *If $\omega \geq \rho$, then $r = r' = b = b' = 0$. The network is empty and each agent has an expected potential of ω.*
- *Decreasing the contagion rates α, δ and ω increases the connection rates.*

A connected successor of i is exposed to externalities from i, whereas i is exposed to externalities from all of its connected successors. Consequently, each connected successor of i is indirectly exposed to every other connected successor of i. As more of i's successors connect

to i, the expected potential of i increases, and the successors of i pose more externalities on each other through the predecessor i. The marginal successor is indifferent to connecting or not. The indifference condition determines the relationship between the connection rate n_i and the potential of p_i:

$$\upsilon - \varepsilon n_i = \delta \mathbb{E}\left[p_i\right]$$

Whether ρ exceeds the minimum expected potential $\omega + O(\varepsilon)$ determines whether it is beneficial for i to connect to its predecessor i^* provided that i^* has no other connections, neither to its predecessor $(i^*)^*$ nor to any of its successors ($n_{i^*} = 0$).

Whether ρ exceeds $\omega + \upsilon + O(\varepsilon)$ determines whether it is beneficial for i to connect to its predecessor i^* who is connected to its own predecessor $(i^*)^*$ but not to any of its successors ($n_{i^*} = 0$).

When ρ is larger than $\omega + \upsilon + O(\varepsilon)$, a network with long paths emerges, and various orders of contagion are prevalent. Agents are willing to connect to their predecessors, who are themselves connected to their own predecessors, and so on. Trees of various depths emerge in equilibrium. The unique equilibrium network in this case is shown in Figure 2.

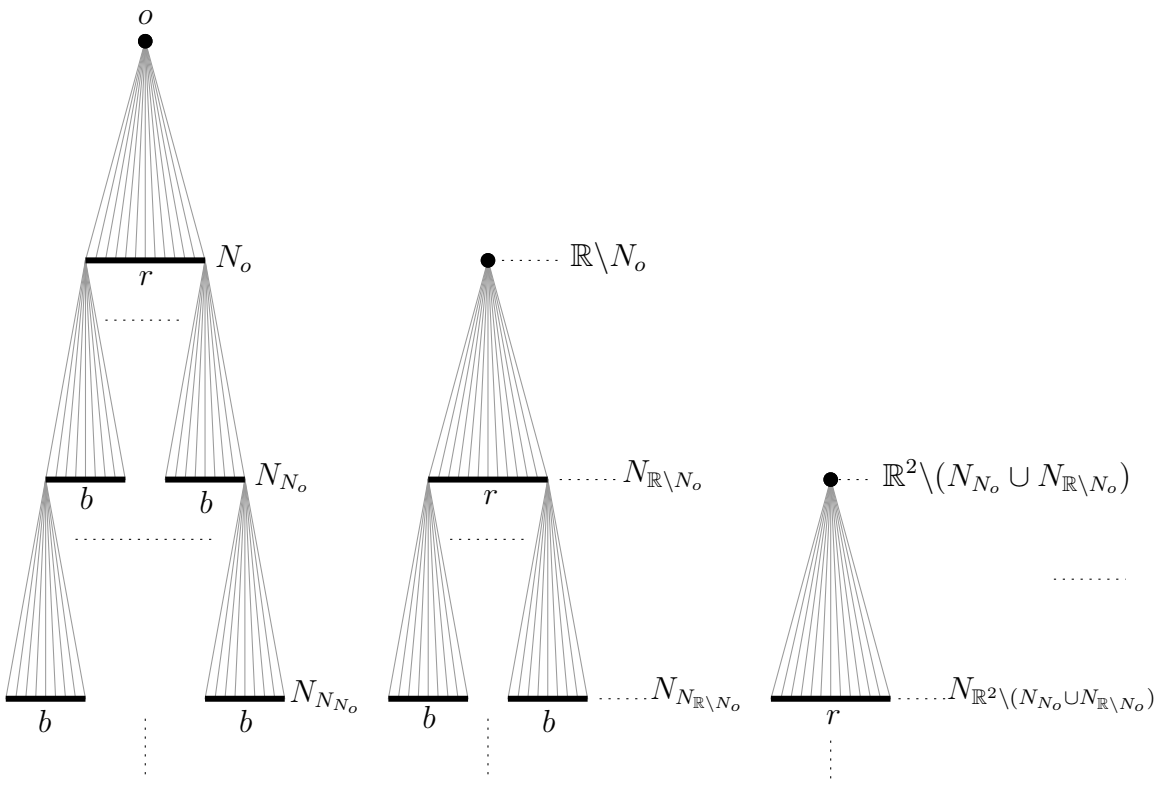

Figure 2: Unique equilibrium network

Each bud has connected successors that bring the expected potential of the bud up to $\rho - O(\varepsilon)$. Then by Equation (1), the connection rate to any branch is

$$\frac{\rho - \omega - \upsilon}{\alpha\rho} + O(\varepsilon) = b + O(\varepsilon)$$

Notably, the product αb is independent of α, up to $O(\varepsilon)$. Decreasing α does not alter the first-order upstream contagion to a branch, as the successors of the branch connect up to the indifference condition of the marginal successor, regardless of the level of α.

Proposition 1 also shows the separable effects of incentives shaping connections. The first-order downstream contagion reduces connection rates by a multiplier of $\frac{R}{B}$, and the second-order upstream contagion reduces connection rates by a 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. The final branches (i.e., $\mathbb{R}^{T-1}$) do not experience second-order upstream contagion. Therefore, the final branches have $\frac{r'}{r} = \frac{b'}{b} = \frac{B+\omega+\upsilon}{\omega+\upsilon}$ times more downstream connections than the non-final branches.

3.2 High orders of contagion

I focus on $\rho > \upsilon + \omega + |O(\varepsilon)|$ for the remainder of the analysis, as this is the case in which there are long paths of exposure. All buds have a potential of approximately ρ, and the approximate connection rate of each branch is

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

The recursive structure allows me to quantify the effects of shocks based on the distance between the origin of the shock and the affected agent downstream the origin.

Proposition 2. ***(Orders of contagion)*** *Fix t and k, and consider $\varepsilon \to 0$ and $T \to \infty$. In equilibrium, for any $i \in \mathbb{R}^t$ and any $j \in \mathbb{R}^{t+k}$ with a path to i, the effect of i's original potential ω_i on j's potential p_j satisfies*

$$\frac{\mathtt{d}p_j}{\mathtt{d}\omega_i} = \frac{e^k}{1 - e\alpha n_i} + O\left(\left(\frac{e}{\delta} - 1\right)^T\right) + O(\varepsilon)$$

where

$$e \equiv 2\delta\left(1 + \sqrt{(2\delta - 1)^2 + (2\delta)^2 \frac{\omega}{\upsilon}}\right)^{-1} < 2\delta$$

When T is large enough, the distance k of the affected agent to the origin of the shock determines the size of the effect. Represent this magnitude with ∂_k. Then Equation (1) 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 is the effect of the shock ω_i on a downstream branch $j \in N_i$, *relative* to the effect of ω_i on i itself. The first component of e is δ , which is the direct relative downstream effect on j. Given that e is the relative amplification of a shock one step downstream, e^2 is the relative amplification of a shock two steps downstream. Then b agents in N_j, who are subject to e^2 amplification from i, feedback one step upstream at rate α onto j. This feedback introduces the second component of e, which is a third-order contagion amplifier, $\alpha b e^2$ – the two steps downstream and one step upstream.

The magnitude of the absolute effect is scaled by the effect of ω_i on i. This is not 1 because of the feedbacks throughout the network. Given that n_i is the measure of connected successors of i, $\frac{1}{1-e\alpha n_i}$ is the multiplier that scales all the relative magnitudes e^k. The term $\frac{1}{1-e\alpha n_i}$ corresponds to the Leontief inverse in the discrete case.

Whether i is connected to its predecessor i^* or not does not affect these magnitudes beyond the equilibrium effect on n_i as i is infinitesimal among the successors of i^*.

Therefore, for large enough T and small enough ε, the effect of ω_i on p_j is $\frac{\mathrm{d}p_j}{\mathrm{d}\omega_i} \approx \frac{e^k}{1-e\alpha n_i}$.

3.3 Welfare and network hazard

Proposition 3. ***(Ex-post welfare)*** *In equilibrium, ex-post welfare satisfies*

$$\lim_{T\to\infty}\lim_{\varepsilon\to 0}\frac{W}{(be)^T} = (\omega - \omega_o)\,O(1).$$

The amplifier

$$be = \frac{1}{2\alpha}\left(1 - \sqrt{(1-2\delta)^2 + (2\delta)^2\,\frac{\omega}{v}}\right)$$

is decreasing in ω, α, and $\delta > \frac{1}{2}\frac{v}{\omega+v}$.

A sharp but long expression of ex-post welfare can be found in the proof of Proposition 3.

Proposition 2 shows that each shock is multiplied by a factor of e when transmitted to a connected successor. The branches of the tree rooted at o have a measure b of downstream connections. Then, the *combined* effect of a shock is amplified by eb when it is transmitted one step downstream, and $(eb)^T$ is the aggregate effect.

Proposition 4. ***(Foundation of network hazard)*** *In equilibrium,*

$$\lim_{\varepsilon \to 0} \mathbb{E}[W] = 0$$

and

$$\lim_{T \to \infty} \lim_{\varepsilon \to 0} \left(\frac{\mathbb{V}[W]}{\mathbb{V}[\omega_o]} \right)^{\frac{1}{2T}} = be$$

This result is the foundation for network hazard. If contagion rates are reduced ex ante, more agents connect to their own successors across the economy, and the average welfare gets re-equilibrated to $O(\varepsilon)$. If agents expect an interim intervention, they form their network accordingly, which justifies the intervention. The combined payoff of agents remains $O(\varepsilon)$, the cost of the intervention is left as the net effect on welfare. Regarding volatility, the aggregate effect of ω_o is mediated by eb, which increases when contagion rates are reduced.

4 Prevention, intervention, and interference

The intervention considered in Section 2 is on the extensive margin. There is either a fixed size intervention or there is no intervention. Now, I examine the size of interventions on the intensive margin. I also explore different timings and different policy tools. For simplicity and precision, I focus on $T = 1$, $\varepsilon = 0$, $\upsilon > \omega\delta$, and large enough μ.

Reducing α to $\hat{\alpha}$ costs $\frac{1}{\eta} c_{\texttt{up}}(\hat{\alpha}; \alpha)$. Reducing δ to $\hat{\delta}$ costs $\frac{1}{\eta} c_{\texttt{dw}}(\hat{\delta}; \delta)$. Scaling shocks $(\omega_i)_{i \in L}$ to $\hat{\omega}_i = \omega_i \frac{\hat{\omega}}{\omega}$ costs $\frac{1}{\eta} c_{\texttt{or}}(\hat{\omega}; \omega)$. I refer to $\eta > 0$ the *efficacy* of the mitigation instrument. For each contagion rate θ among α, δ, ω, the corresponding function c is a decreasing and convex function of $\hat{\theta} \in [\underline{\theta}, \theta]$ with $c(\theta; \theta) = 0$ and $\underline{\theta} > 0$.

The principal can also sever connections to mitigate contagion. The choice is between severing all connections at $\kappa > 0$ cost each or severing no connections. In the context of epidemics, quarantines and stay-at-home orders are non-targeting policies which restrict regular or habitual social events, effectively severing connections. In social networks, suspending or censoring an account removes all connections to followers. In financial networks, broad-based

interventions, such as the Troubled Asset Relief Program, eliminate related exposures of all parties.

Lemma 1. *In the absence of policy tools that mitigate contagion, the equilibrium connection rate is*

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

The equilibrium welfare is

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

The network hazard can be observed in this lemma. The expected welfare is zero. Reducing contagion rates The positive effects of reducing contagion rates are eroded by higher connection rates in response. The variation in the shock of the root spreads to the leaves. In particular,

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

increases when the contagion rates are reduced. So, reducing contagion rates increases contagion costs and increases volatility.

Lemma 1 implies that implementing a costly tool to mitigate contagion before the formation of connections is suboptimal.

Proposition 5. ***(Prevention)*** *Consider a principal who can reduce a contagion rate θ to any $\hat{\theta} \in [\underline{\theta}, \theta]$ before any connections are established.*

As the size of the prevention effort $\theta - \hat{\theta}$ increases, the expected welfare decreases, the expected cost of contagion increases, and the variance of welfare increases.

Consequently, the optimal policy for the principal is to avoid preventing contagion, which implies $\hat{\theta}^* = \theta$.

4.1 Intervention with contagion

Similarly to the original analysis in Section 2, consider a principal who can intervene after the connections are formed, before shocks have materialized. First, I establish a general result regarding the two extremes of the intervention policy, the lowest $\underline{\theta}$ and the highest θ.

Lemma 2. *Consider a principal who can reduce a contagion rate θ to any $\hat{\theta} \in [\underline{\theta}, \theta]$ after all connections are established before any shock has been realized.*

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: the expected welfare is increasing, the expected cost of contagion is decreasing, and the variance of welfare is decreasing in efficacy η.

There exists $\underline{\eta} \geq 0$ such that for all $\eta \neq \underline{\eta}$, the no-intervention 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 $\eta < \underline{\eta}$, the efficacy η does not affect the 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 by $\overline{n}$ as η varies. When the efficacy is high enough to reach the maximum equilibrium connection rate $\overline{n}$, further increases in the efficacy are beneficial. If the network is inelastic to contagion rates, then reducing contagion rates decreases the cost of contagion and enhances welfare.

There are cases in which 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 $n^*_\eta = \overline{n}$.[12]

Proposition 6. ***(Intervention)*** *Let $c(\hat{\theta}; \theta) = \frac{1}{1+\gamma}(\theta - \hat{\theta})^{1+\gamma}$. Consider a principal who can reduce the contagion rate θ to any $\hat{\theta} \in [\underline{\theta}, \theta]$ after all connections are established before any shock has been realized.*

Increasing efficacy η reduces welfare, increases aggregate contagion costs, and increases volatility, unless efficacy is sufficiently high.

The cost function used in Proposition 6 satisfies $c_1(\theta, \theta) = 0$. By Lemma 2, the no-intervention boundary $(\underline{n}, \theta)$ is not an equilibrium outcome. It is optimal to intervene as the marginal cost of an intervention is zero at the no-intervention benchmark. A higher η, indicating a more effective intervention tool, reduces $\hat{\theta}^*_\eta$. The connection rate n^*_η adjusts and the network hazard causes welfare losses and volatility. The welfare consequences are illustrated in Figure 3.

4.2 Interference with connections

Reducing contagion rates after shocks have materialized yields qualitatively similar insights to the case of interventions before shocks. A relevant policy tool is to severe connections,

[12] There is an interior solution to the leaves' indifference condition but this solution is not stable against deviations by small groups.

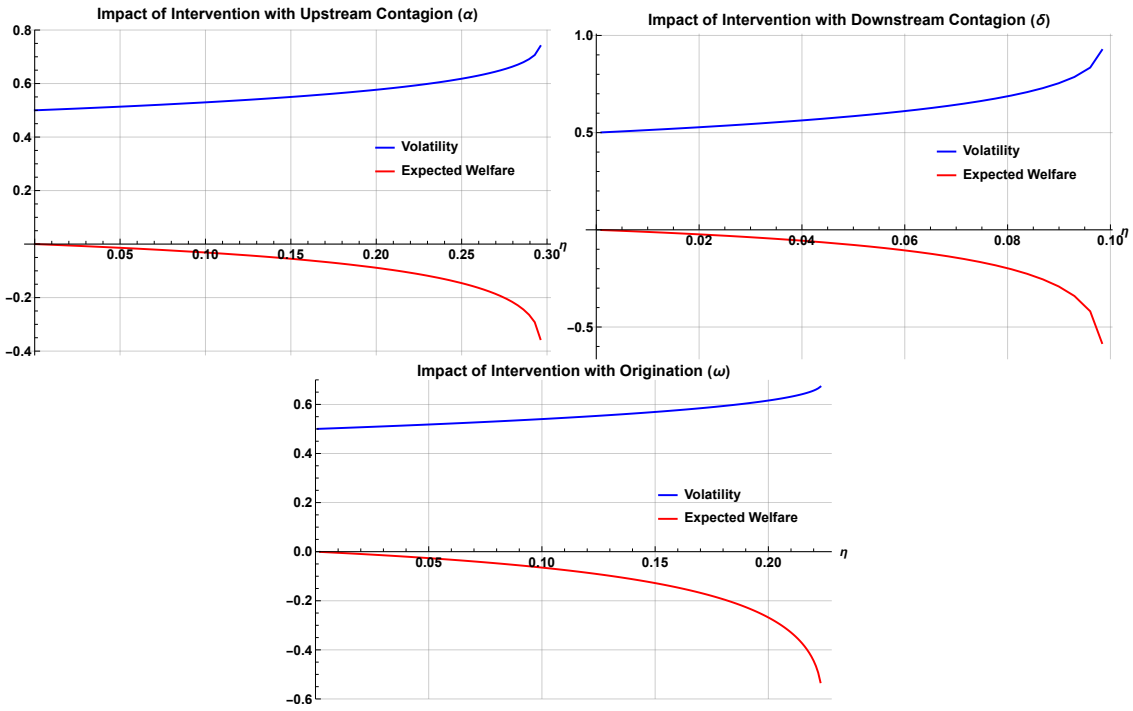

Figure 3: Impact of interventions as efficacy η grows (Values: $\alpha = \delta = \omega = \gamma = 1$, $\upsilon = 2$)

which leads to qualitatively different insights depending on whether it is implemented before or after the shocks. Removing connections is redundant or ineffective if implemented before shocks occur. Let f be the optimal policy. Since agents anticipate that $f(n)$ connections are removed from any n, agents establish n' connections such that $n^* = n' - f(n')$. Removing connections after shocks is useful. Removing connections based on the realization of the shock ω_o removes the downside of contagion in the event of a bad shock ω_o, but does not remove the upside in the event of a good shock ω_o.

Proposition 7. ***(Interference)*** *Assume that the shocks are i.i.d. and uniformly distributed, with* $\omega_i \sim U[0, 2\omega]$.

Consider a principal who can remove all links at cost κn *after the shocks have materialized.*

In a middle range of the unit interference cost κ*, reducing the cost* κ *reduces the expected welfare in robust equilibrium.*[13]

The option of conditional interference limits the public cost of contagion per agent to at most κ. If the contagion cost of an individual exceeds κ, the principal assumes the cost and

[13]The equilibrium must also be robust against a vanishingly small probability that the interference attempt fails. This restriction rules out some unintuitive cases. 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 payoff 0. This outcome constitutes an equilibrium, but it does not survive a small probability of interference failure.

removes the connections. This possibility introduces an incentive to increase connection rates to enjoy the upside without increasing the downside, creating a network hazard.

5 Network hazard in applications

This section introduces several standalone models from different applications, incorporating additional institutional details to enhance the robustness of the broader insight. Each subsequent subsection is self-contained with its own notation.

5.1 Coordination games

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 links of i and costs θ_i per link. If both i and j exert effort and $e_{ij} = 1$, the link generates a benefit that gives $\beta > g$ to agent i. Thus, the payoff of i in the coordination game (in the second stage, net of the cost of links) is $a_i \sum_j (\beta a_j - \theta_i) e_{ij}$.

The agents play the best Nash equilibrium in the second stage.[14] The ex-post payoff of i is

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

In the first stage, agents form (pairwise) stable networks given the expected continuation payoffs.[15]

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

[15] 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.

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 $u_i + t_i$.[16] The welfare cost of a unit transfer is $1 + z$, where z represents the transaction cost. Welfare is given by

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

I consider the limit as $z \downarrow 0$. This specification 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 8. *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.

The variance in 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 of 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 response, 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.

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 other follower, f_j, and the leader have good shocks. The transfer persuades the *leader* to exert effort to prevent contagion, thus protecting the *other follower* f_j. However, the leader cannot be induced to exert effort if it receives a bad shock. As a result, both followers shirk.

In general, transfers mitigate inefficient contagion ex post. However, by doing so, transfers reduce market discipline, increase interconnectedness, 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.

[16] Transfers are conditional individual actions so the best Nash equilibrium is still well-defined.

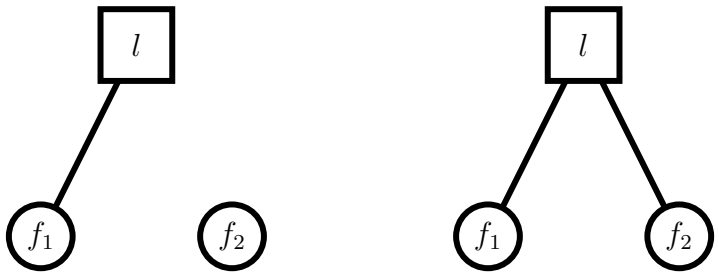

Figure 4: Stable network. Contagion that originates at f_1 can spread to l. This risk prevents f_2 from linking to l. Transfer to l mitigates the contagion that originates in f_1. Then f_2 also links to l. But then the effect of contagion that originates in l is exacerbated.

5.2 Epidemics

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 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 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 $v_1 > v_2 > 0$, making a_1 a more preferred match.

Each agent has a probability η of being infected *externally*. Matches can transmit infections *internally*, with a base transmission probability of τ_0 if one agent in the match is infected and the other not. A protective measure reduces the probability of transmission by a factor of $m < 1$, reducing it to $\tau = m\tau_0$. The cost of becoming infected is κ.

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

Equilibrium Since a_1 is preferred, both agents of type b prefer to match 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 possibility introduces second-order contagion. The efficacy of the protective measure m alters the transmission probabilities and influences the structure of the network as follows.

Proposition 9. *There exist thresholds $m_3 \leq m_2 < m_1$ such that the unique stable network is characterized as follows:*[17]

- *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, the expectation and variance of the number 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 and 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 also does not match 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.

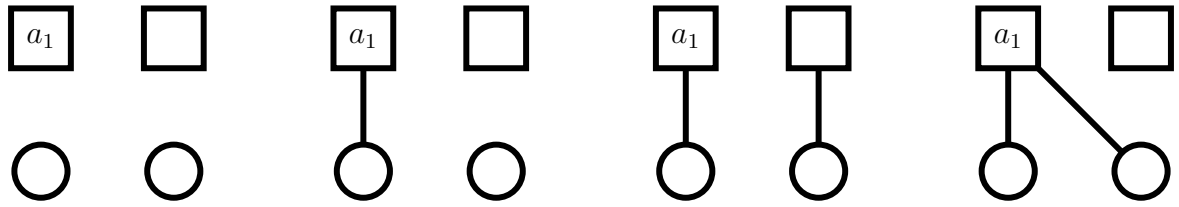

Figure 5: Strongly stable network of matches

Figures 6 and 7 illustrate the distribution of infections and welfare, respectively.[18] The vertical lines in the figures correspond to m_3. The increments at m_1 and m_2 are related to Talamàs and Vohra (2020) as the number of matches increases at these thresholds. However,

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

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

the change at m_3 represents a structural change. One type-b agent switches its match from a_2 to a_1. This switch occurs precisely because the protective measure becomes effective enough so 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 is aligned with the increased matching payoffs. More interestingly, both type-b agents are now exposed to the risk of exogenous infection of a_1. The variance of infections increases due to the correlation through a_1's infection probability.

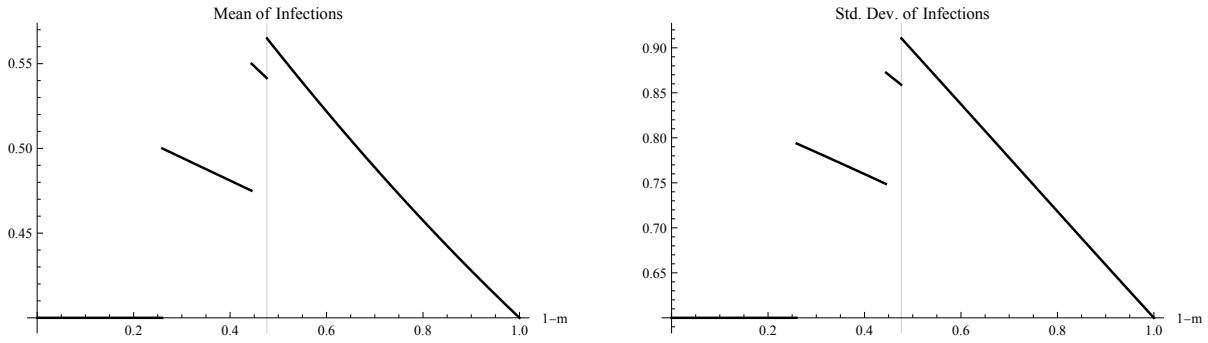

Figure 6: Mean and standard deviation of the number of infections

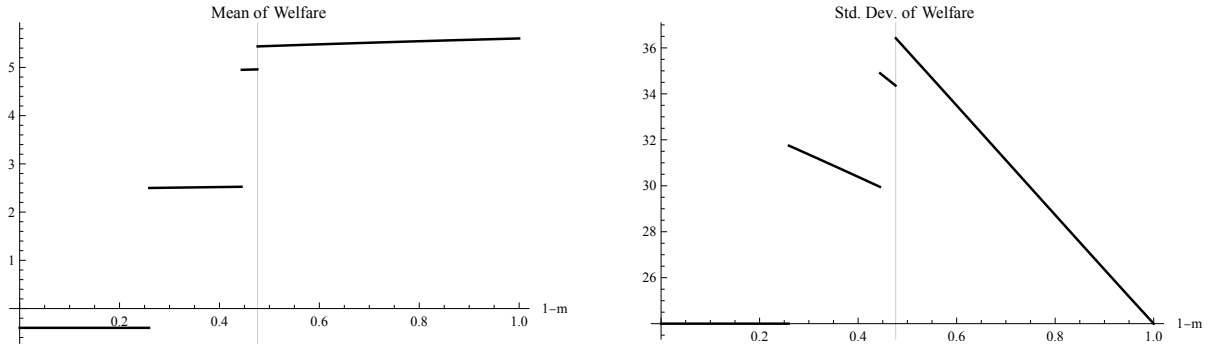

Figure 7: Mean and standard deviation of welfare

5.3 Supply chains

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 underprovision if the upstream supplier does not risk overproduction. Subsidies to downstream firms' production costs can prevent this chain reaction and alleviate inventory risk.

Consider two upstream firms, indexed by $u \in U = \{u_1, u_2\}$, that 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 which is compatible with the input from only one of the upstream firms. Switching costs are sufficiently high, so that once a downstream firm chooses an upstream firm's technology, the upstream firm becomes the sole supplier to the downstream firm.

Each upstream firm $u \in U$ requires a distinct *external* input supplied by another market. The price of the external input for the 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 an 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 and $q_d = \min\{q_d^{in}, q_d^{ex}\}$ units are produced with q_d^{in} units of internal input 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 an inelastic unit demand for its output. The buyer has a value of p, so d sells at a price p and captures all surplus. Upstream firms sell to downstream firms at price $p' < p$. The fixed price p' reflects the exogenous bargaining power between the upstream and downstream firms, and is not subject to renegotiation.

The timing is as follows. First, downstream firms simultaneously choose their technologies (suppliers). Then the prices of the external inputs of the upstream firms, k_{u_1}, k_{u_2}, are realized independently. Then upstream firms build their inventory, q_{u_1}, q_{u_2}. Afterwards, the prices of the external inputs of downstream firms, d_{d_1}, c_{d_2}, are realized independently. Then the downstream firms purchase internal and external inputs, and produce q_{d_1}, q_{d_2}. Then consumers buy the output 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 low, it will produce only 1 units to minimize the expected cost of overproduction.

But then, if both downstream firms have low costs and each demand 1 unit, then the supply is exceeded. In this case, the available unit is supplied on a first-come-first-served basis, with a probability 1/2 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 $D_u \subset D$ the downstream buyers of $u \in U$ and $U_d \in U$ the downstream 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

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

The downstream firm 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 a 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 input. Let e_i denote the domestic cost of the external input of the firm i's. 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 captured by the firms. So, absent subsidies, welfare is

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

The 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 subsidy 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

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

I focus on $z \downarrow 0$ to simplify the algebra. Equivalently, $z = 0$ and the government implements the minimal transfer necessary to induce the efficient outcome. Furthermore, $e_d = 0$, whereas $e_u = k_u$, so the shocks faced by downstream firms stem from financial conditions, while the shocks faced by upstream firms reflect real shocks.[19] This setup highlights the role of subsidies in managing the 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 the 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 cause an upstream firm with two buyers to produce only one unit instead of two, thus creating second-order contagion for downstream firms by reducing their probability of being supplied.

Proposition 10. *Assume $k > \delta^2 p'$. (Other cases are solved in the appendix.)*

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

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 are higher in the presence of subsidies than in the absence of subsidies.

The upstream firm u_1 is the preferred supplier because it has a 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

[19] 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 require relatively low marginal costs of production. Shocks which 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 input.

However, 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 example, 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 increase 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.

not being supplied. This scenario represents second-order contagion. Since the difference in exogenous risk α_u between the upstream firms does not differ significantly, a downstream firm prefers the slightly higher first-order contagion cost over the 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 external input for upstream firms is too high for efficient production. However, the external inputs of downstream firms are produced at a lower cost, making it efficient to restore production. Consequently, downstream firms receive subsidies whenever they face high prices that would otherwise hinder their production, provided their upstream supplier faces low prices. Since 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, the 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 the transaction costs are not present. In the absence of subsidies, the presence of second-order contagion prevents downstream firms from choosing the superior supplier. Subsidies eliminate 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

The interconnected nature of networks poses complex contagious threats despite their benefits. This paper argues that interventions meant to mitigate externalities borne by contagion can create more contagion through moral hazard in network formation. Welfare can decrease and volatility can increase. These adverse consequences of interventions are called network hazard. The network hazard emerges from a substitution between high- and low-order contagion risks through an endogenous reaction of the network structure. This substitution erodes the benefits of interventions and increases the exposure to key agents, resulting in increased volatility.

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A Competitive network formation

Consider an arbitrary set of agents A. Each subset of A^2 is a network and each element of a network is a directed link. A directed link (i, j) is called an out-link of i and an in-link of j. Each network can be represented by a matrix $\mathbf{a}$ where $a_{ij} = 1$ indicates that (i, j) is in the network and $a_{ij} = 0$ indicates otherwise.

For a given network $\mathbf{a}$, the structure of the network shapes externalities between agents. The magnitude of the externality posed by i on its connections is denoted by e_i. These magnitudes are determined by an externality function E. Specifically,

$$e_i = E_i\left((a_{ij}e_j \ , \ a_{ji}e_j \ , \ a_{ij} \ , \ a_{ji})_{j\in A}\right) \tag{2}$$

for each $i \in A$. The externality of an agent depends on the externalities of its connections, which depend on the externalities of their connections, etc. So, E defines the mode of contagion.

Agents form a network given the externalities determined by candidate networks through (2). The payoff for each agent depends on the externalities of the linked agents. This dependence is not necessarily summarized by e_i. Hence, i's payoff is

$$U_i\left((a_{ij}e_j \ , \ a_{ji}e_j \ , \ a_{ij} \ , \ a_{ji})_{j\in A}\right). \tag{3}$$

The externalities can be negative, trading off against direct benefits of links. Motivating examples, such as supply chain disruptions, epidemics, financial contagion, and misinformation, fall into this category. Alternatively, externalities can be positive, trading off against the direct cost of links. For example, connections can be partnerships that are costly to maintain but provide benefits such as information or social capital.

Competitive network formation Let the set of agents be $A \subset \mathbb{R}^D$ for some positive $D \leq \infty$. Two parameters govern the density of the networks of interest. The *in-degree density* is a positive integer $\overline{d} \leq D$. The *out-degree density* is a positive integer $\underline{d} \leq \overline{d}$.

The in-degree density is the dimension of the set of in-links of each agent. Specifically, let $\overline{\mathcal{A}}$ be the algebra generated by the $\overline{d}$ dimensional intervals in A and $\overline{\lambda}$ be the Lebesgue measure of dimension $\overline{d}$. The pre-measure space $\overline{P} = \left(A, \overline{\mathcal{A}}, \overline{\lambda}\right)$ is used for the measurement of the in-links of agents.[20]

Similarly, the *out-degree density* is a positive integer $\underline{d} \leq \overline{d}$. This is the dimension of the set of out-links of each agent. For consistency between in-links and out-links, all out-links are restricted towards a set of *key agents*, $K \subset A$, who live in a $D - (\overline{d} - \underline{d})$ dimensional subspace.[21] Given K, let $\underline{\mathcal{A}}$ be the algebra generated by $\underline{d}$ dimensional intervals in K and let $\underline{\lambda}$ be the $\underline{d}$ dimensional Lebesgue measure. The pre-measure space $\underline{P} = (K, \underline{\mathcal{A}}, \underline{\lambda})$ is used for measuring out-links of agents. Each agent has a finite measure of out-links in $\underline{d}$ dimensions.

Each agent $i \in A$ chooses its out-links unilaterally. The strategies of i are measurable sets in $\underline{P}$. The decision of i to form an out-link to j is denoted by $a_{ij} = 1$. Otherwise, $a_{ij} = 0$. The resulting network is $\mathbf{a} = (a_{ij})_{i,j\in A}$. Given the network $\mathbf{a}$, the resulting in-links of i are denoted $N_i \subset A$.

[20] One can also use sigma-algebras and measure spaces. Some sets in sigma algebras do not have economically meaningful counterparts in finite discrete samples, so I work with pre-measures.

[21] For example, $K = A \cap \left(\mathbb{R}^{D-(\overline{d}-\underline{d})} \times \{v_0\}\right)$ for some $v_0 \in \mathbb{R}^{\overline{d}-\underline{d}}$.

The externality score of i in network $\mathbf{a}$ is

$$e_i \equiv E\left(\overline{\int} a_{ij}e_j \mathtt{d}\underline{\lambda}(j)\ ,\ \overline{\int} a_{ji}e_j \mathtt{d}\overline{\lambda}(j)\ ;\ \theta_i\right) \tag{4}$$

where $\overline{\int}$ denotes the upper Lebesgue integral in the corresponding pre-measure space, and θ_i is the *type* of i. A typical externality function increases with the first and second inputs.

The out-links yield benefits but expose agents to costs proportional to the externalities of out-linked agents. The in-links can also affect this tradeoff. Specifically, the payoff of i given by

$$U\left(\overline{\int} a_{ij}e_j \mathtt{d}\underline{\lambda}(j)\ ,\ \overline{\int} a_{ji}e_j \mathtt{d}\overline{\lambda}(j)\ ,\ \underline{\int} a_{ij}\upsilon\left(i,j\right)\mathtt{d}\underline{\lambda}(j)\ ;\ \theta_i\right) \tag{5}$$

where $\underline{\int}$ is the lower Lebesgue integral in $\underline{P}$, and $\upsilon\left(i,j\right)$ is a measurable function in the product space $\underline{P}\times\overline{P}$ which describes the marginal connection benefit to i from an out-link to j. Note that the externalities and payoffs are well-defined for any strategy profile $\mathbf{a}\in A^2$.

The solution concept is *measurable Nash equilibria* (MNE) – Nash equilibria wherein N_i is measurable in $\overline{P}$ for each $i\in K$.[22,23]

Example 1. (Connection to network games) Consider $E\left(x,y;\theta_i\right)\equiv\left(1-\gamma\right)\theta_i+\gamma\left(x+y\right)$ for some $\gamma\in(0,1)$, and $U\left(x,y,z;\theta_i\right)=\tilde{U}(z)-\gamma\left(1-\gamma\right)\left(x+y-\theta_i\right)^2$.

This specification reflects an environment where agents play a coordination game after forming the network. Firstly, agents form a network and the out-links provide a direct benefit $\tilde{U}\left(\underline{\int} a_{ij}\upsilon\left(i,j\right)\mathtt{d}\underline{\lambda}(j)\right)$ to each agent i. The agents then play a coordination game on the formed network. Each agent i picks $q_i\in\mathbb{R}$ and incurs a cost

$$\left(1-\gamma\right)\left(\theta_i-q_i\right)^2+\gamma\left(\overline{\int} a_{ji}q_j \mathtt{d}\overline{\lambda}(j)+\overline{\int} a_{ij}q_j \mathtt{d}\underline{\lambda}(j)-q_i\right)^2$$

This cost defines a coordination game played on the network. Each agent i prefers to minimize its cost by choosing an action q_i that is close to the actions of connected agents' actions, as well as i's own type θ_i.[24]

[22]Note that any unilateral deviation is allowed – N_is are not restricted to be measurable after deviations. That said, there is no specific incentive to deviate to induce non-measurable sets of in-links because benefits are defined through lower integrals and costs are defined through upper integrals.

[23]Depending on the context, it can be appropriate to impose a stability restriction to rule out profitable group deviations by vanishingly small measures of agent. This can ensure that the equilibrium is not a knife-edge case and has a meaningful generic discrete counterpart.

[24]See Ballester et al. (2006) for more on network games.

The Nash equilibrium of this coordination game is given by $q_i^* = e_i$. After substituting the equilibrium quantities q^* into the payoffs, the resulting equilibrium payoff of i including direct benefits from the network is given by (5).

Example 2. (Linear contagion and regular networks) Take $A = K = [0,1]^D$ and $\underline{d} = \overline{d}$. All agents are ex-ante identical: $\theta_i = \theta$ and $\upsilon(i,j) = \upsilon$ for some constants $\theta, \upsilon > 0$. The externality function is $E(y,z;\theta) = \theta + \delta(y+z)$ where $\delta \in (0,1)$ is a constant. The utility function is $U(x,y,z;\theta) = x - \frac{1}{2}y^2$.

This externality function is the continuous counterpart of the Katz-Bonacich centrality, where θ is the baseline centrality and δ is the damping factor. Katz-Bonacich centrality describes the dynamics of linear contagion.

There is an MNE network where all agents have the same externality score and the same measure of out-links and in-links. This equilibrium can be obtained as follows. First, conjecture the same externality e^* for all agents. When agent i chooses its out-links, it has no effect on any other agent's externality. So i chooses a measure m_i for its out-links, to maximize $m_i\upsilon - \frac{1}{2}(e^* m_i)^2$, taking e^* as given in (5). Then $m_i^* = \frac{\upsilon}{(e^*)^2}$ is the optimal out-degree of i. Then e^* is determined in equilibrium as a fixed point of (4): $e^* = \theta + 2\delta m^* e^*$. Specifically, each agent has an externality of $e^* = \frac{1}{2}\left(\theta + \sqrt{\theta^2 + 8\upsilon\delta}\right)$ and a measure

$$m^* = \frac{1}{16\delta^2\upsilon}\left(\sqrt{\theta^2 + 8\upsilon\delta} - \theta\right)^2$$

of out-links and in-links. The network topology can take various forms. For example, groups of measure $2m^*$ of agents can be organized into bipartite components. Alternatively, groups of measure m^* of agents can be organized into disjoint cliques.

Example 3. (Threshold contagion and cliques) Take $A = K = [0,\mu]^D$ and $\underline{d} = \overline{d}$. All agents are ex-ante identical. Marginal benefits are constant, $\upsilon(i,j) = \upsilon$ for some $\upsilon > 0$. Types are drawn i.i.d. after the formation of the network, $\theta_i \sim U[-rZ, (1-r)Z]$, for some $Z > 0$ and $r \in (0,1)$. The externality function is $E(y,z;\theta_i) = \mathbb{1}[\theta_i < y + z]$. The utility function is $U(x,y,z;\theta) = x \cdot (1 - E(y,z;\theta))$.

The externality function describes the dynamics of the threshold contagion. An externality score $e_i = 1$ means that i is 'infected.' When more connections of i become infected, i is also more likely to become infected.[25] Infection yields 0 payoff. No-infection results in a payoff proportional to the measure of out-links.

[25]There are complementarities between infections. The selection is the smallest solution in the lattice of fixed points in which the smallest set of agents is infected.

There is an MNE network where agents are organized into disjoint cliques of the same size. This equilibrium can be obtained as follows. Conjecture a measure m^* for the measure of agents in each clique. Then there exists some f^* such that in each clique, f^* fraction of agents with the smallest f^*m^* types become infected. Using the externality function and (4), the fraction f^* is determined as $f^* = \mathbb{P}[\theta_i < 2f^*m^*]$. So $f^* = r\left(1 - \frac{2m^*}{Z}\right)^{-1}$. This determines the average expected externality for each agent's connections. If an agent i deviates from its clique and out-links to some m_i measure of agents, given that all other agents are organized into cliques with m^* measure of agents, the expected payoff of i becomes $m_i\mathbb{P}[\theta_i > f^*(m_i + m^*)]$.[26] Then i's optimality condition and (5) yield

$$m^* = \frac{1-r}{2+r}Z.$$

If the risk r increases, the sizes of the cliques shrink.

Example 4. (Hierarchies) Take $A = \mathbb{R}^D$, $K = \mathbb{R}^{D-\Delta} \times \{\mathbf{0} \in \mathbb{R}^\Delta\}$, and $\underline{d} = \overline{d} - \Delta$ for some positive Δ that divides D. Let the externality of each agent be 1 or 2, depending on the externality of its in-links: $E(y, z; \theta_i) = 1 + \mathbb{1}[z > z^*]$ for some constant $z^* > 0$. Let the utility function be $U(x, y, z; \theta) = x - \frac{1}{2}y^2$.

The externality function puts discipline on the out-linking decisions. There is an MNE network where agents are organized into a hierarchy. Let $H_t \equiv \mathbb{R}^{D-t\Delta} \times \{\mathbf{0} \in \mathbb{R}^{t\Delta}\}$ for each $t \geq 0$. Note $H_0 = A$ and $H_1 = K$. For each t, partition $H_t \backslash H_{t+1}$ into sets of measure z^* in $\overline{P}$. For each element h of the partition, pick a measure υ of agents h' in $H_{t-1}\backslash H_t$ in $\underline{P}$, and form an out-link from each agent in h to h'. Finally, from out-links from $\mathbf{0} \in \mathbb{R}^D$ to an arbitrary υ measure of agents in A in $\underline{P}$.[27] In this configuration, each agent has υ measure of out-links in $\underline{P}$ and each key agent has z^* measure of in-links in $\overline{P}$. So, each agent has an externality 1. Given that all other agents have externality 1, choosing $m_i = \upsilon$ measure of out-links in $\underline{P}$ maximizes the payoff i given by $m_i\upsilon - \frac{1}{2}m_i^2$.

Lifting the hierarchy restriction in Section 2 With or without hierarchy restriction, equilibrium networks are defined by the indifference conditions of marginally connected agents. For example, take $\varepsilon = 0$. In any network where each branch agent has potential ρ, no agent has an incentive to deviate. This situation can also occur in a cyclical configuration, as shown in Figure 8.

[26] Recall that strategies are measurable in $\underline{P}$ and the *algebra* is generated by intervals. So, each clique is a finite union of intervals. After a (measurable) deviation to connect to a finite number of intervals, i can be connected to only a finite number of cliques. So, the fraction f^* applies to each clique that i is connected to after the deviation.

[27] Alternatively, take $v(\mathbf{0}, \cdot) < 0$ so $\mathbf{0}$ does not form any out-links.

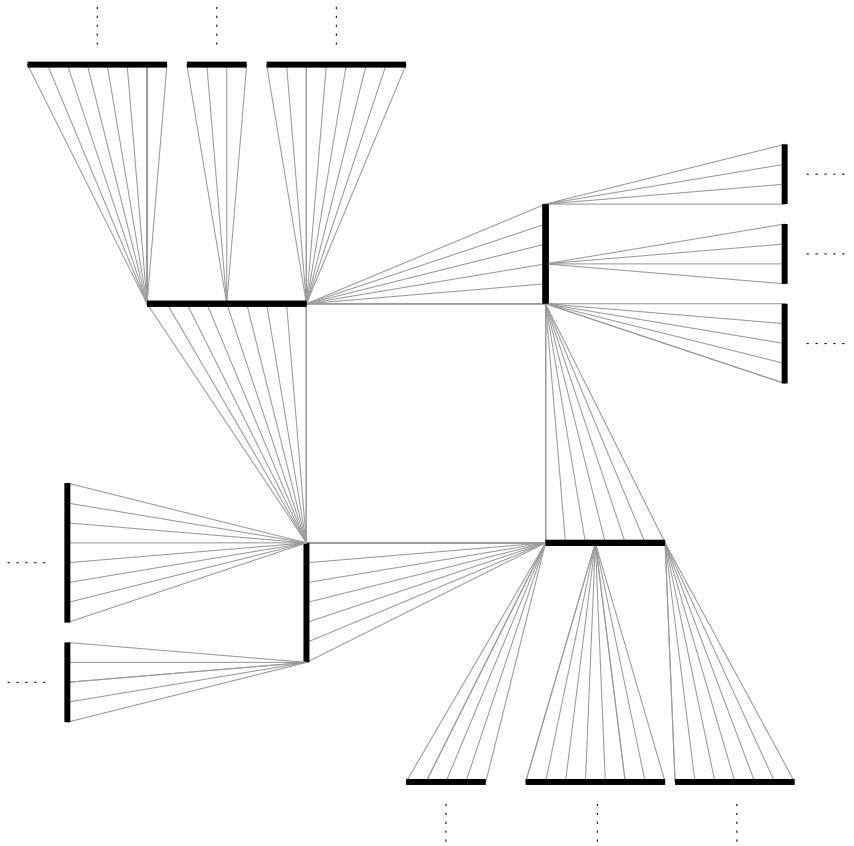

Figure 8: A cyclic network which can emerge as equilibrium when the hierarchy restriction is lifted

B Proofs

B.1 Main model

(Proof of Proposition 1) First consider $\upsilon > \delta(\omega + \upsilon) + |O(\varepsilon)|$.

Take any agent i, and let $j = i^* \in B$. If $n_j = 0$, then $\mathbb{E}[p_j] \leq \omega + \upsilon$ by the rationality of j's connection decision. Then it is rational for i to choose $a_i = 1$ as $\upsilon > \delta(\omega + \upsilon)$. Therefore, regardless of $a_j \in \{0, 1\}$, successors of j connect up to the point that the marginal successor of j is indifferent:

$$\upsilon - \varepsilon n_j = \delta \mathbb{E}[p_j]. \tag{6}$$

Now consider any equilibrium and any rooted tree in the network, say, with depth k. I will show that all agents at any given depth of the tree have the same potential and connection rate. Denote the set of agents at depth k' of the tree by $D_{k'}$.

Start with depth k. Take an agent $i_k \in D_k$ and let $i_{k-1} = i_k^* \in D_{k-1}$. Since $n_i = 0$, we have

$\mathbb{E}[p_{i_k}] = \omega + \delta\mathbb{E}[p_{i_{k-1}}]$. So, all connected successors of i_{k-1} have the same expected potential given by

$$\mathbf{p}(\mathbb{E}[p_{i_{k-1}}]; k) \equiv \omega + \delta\mathbb{E}[p_{i_{k-1}}]$$

Also note that, by (6),

$$n_{i_{k-1}} = \mathbf{n}(\mathbb{E}[p_{i_{k-1}}]) \equiv \varepsilon^{-1}\left(\upsilon - \delta\mathbb{E}[p_{i_{k-1}}]\right).$$

Next, take an agent $i_{k-1} \in D_{k-1}$ and let $i_{k-2} = i^*_{k-1} \in D_{k-2}$. Then by

$$\begin{aligned}\mathbb{E}[p_{i_{k-1}}] &= \omega + \delta\mathbb{E}[p_{i_{k-2}}] + \alpha\mathbf{n}(\mathbb{E}[p_{i_{k-1}}])\mathbf{p}(\mathbb{E}[p_{i_{k-1}}]; k) \\ &= \omega + \delta\mathbb{E}[p_{i_{k-2}}] + \alpha\varepsilon^{-1}\left(\upsilon - \delta\mathbb{E}[p_{i_{k-1}}]\right)\left(\omega + \delta\mathbb{E}[p_{i_{k-1}}]\right) \qquad (7)\end{aligned}$$

Then, all successors of i_{k-2} have the same expected potential $\mathbb{E}[p_{i_{k-1}}]$, denoted by $\mathbf{p}(\mathbb{E}[p_{i_{k-2}}]; k-1)$ as the solution to $\mathbb{E}[p_{i_{k-2}}]$ in (7). Then by (6), all successors of i_{k-2} have the same connection rate, denoted by $\mathbf{n}(\mathbb{E}[p_{i_{k-2}}])$.

Similarly, take an agent $i_t \in D_t$, and let $i_{t-1} = i^*_t \in D_{t-1}$. The same arguments inductively show that all the successors of i_{t-1} have the same expected potential and connection rate.

Regardless of which $i_k \in D_k$ is the starting point, the induction goes up to the root of the tree, i_0. Then, going backward in the induction steps, we find that all agents at depth t have the same potential, denoted by $p(t)$, and the same connection rate denoted by $n(t)$. These satisfy $\delta p(t) = \upsilon - \varepsilon n(t)$ for all $t \leq k-1$, and

$$\begin{aligned}p(0) &= \omega + \alpha n(0)p(1) \\ p(1) &= \omega + \delta p(0) + \alpha n(1)p(2) \\ p(2) &= \omega + \delta p(1) + \alpha n(2)p(3) \\ &\cdots \\ p(k-1) &= \omega + \delta p(k-2) + \alpha n(k-1)p(k) \\ p(k) &= \omega + \delta p(k-1)\end{aligned}$$

Then, $p(k) = \omega + \delta p(k-1)$ and

$$\begin{aligned}
\upsilon &= \delta\omega + \upsilon\alpha n(0) - \varepsilon\left(n(0)n(1)\alpha - n(0)\right) \\
\upsilon &= \delta\left(\omega + \upsilon\right) + n(1)\alpha\upsilon - \varepsilon\left(n(1)n(2)\alpha + n(0)\delta - n(1)\right) \\
\upsilon &= \delta\left(\omega + \upsilon\right) + n(2)\alpha\upsilon - \varepsilon\left(n(2)n(3)\alpha + n(1)\delta - n(2)\right)
\end{aligned}$$

$$\begin{aligned}
\upsilon &= \delta\left(\omega + \upsilon\right) + n(k-2)\alpha\upsilon - \varepsilon\left(n(k-2)n(k-1)\alpha + n(k-3)\delta - n(k-2)\right)... \\
\upsilon &= \delta\left(\omega + \upsilon\right) + n(k-1)\alpha\delta\left(\omega + \upsilon\right) - \varepsilon\left(n(k-1)^2\alpha\delta + n(k-2)\delta - n(k-1)\right)
\end{aligned}$$

Then $p(k) = \omega + \delta p(k-1)$ and

$$\begin{aligned}
n(0) &= \frac{\upsilon - \delta\omega}{\upsilon\alpha} + O(\varepsilon) = r + O(\varepsilon) \\
n(1) &= \frac{\upsilon - \delta\left(\omega + \upsilon\right)}{\upsilon\alpha} + O(\varepsilon) = b + O(\varepsilon) \\
&... \\
n(k-2) &= \frac{\upsilon - \delta\left(\omega + \upsilon\right)}{\upsilon\alpha} + O(\varepsilon) = b + O(\varepsilon) \\
n(k-1) &= \frac{\upsilon - \delta\left(\omega + \upsilon\right)}{\alpha\delta\left(\omega + \upsilon\right)} + O(\varepsilon) = b' + O(\varepsilon)
\end{aligned}$$

(When $k = 1$, $n(0) = \frac{\upsilon - \delta\omega}{\alpha\delta(\omega+\upsilon)} + O(\varepsilon) = r' + O(\varepsilon)$.)

When $\delta\omega + |O(\varepsilon)| < \upsilon < \delta\left(\omega + \upsilon\right)$ it is not individually rational for i to connect to its predecessor i^* if i^* is connected to its predecessor $(i^*)^*$. However, if i^* is not connected to $(i^*)^*$, then it is individually rational for i to connect to i^* provided that i^* has no downstream connections. Therefore, in equilibrium, all root agents have downstream connections up to the point of increasing their potential up to the marginal successor's connection benefit, but there are no branch agents. Each root has a connection rate of $r' + O(\varepsilon)$.

When $\upsilon < \delta\omega$, it is not rational to connect.

(Proof of Proposition 2) Take any $i' \in L$ downstream i. Let the downstream path from i to i' be $i = i_0, i_1, ..., i_{T-t} = i'$. All potentials are affine in shocks. Denote $\partial_t = \frac{\mathrm{d}\mathbb{E}[p_{i_t}]}{\mathrm{d}\omega_i}$ the

coefficient of ω_i in p_{i_t}. Then recursively we have

$$\begin{aligned}
\partial_0 &= 1 + \alpha n_i \partial_1 \\
\partial_1 &= \delta \partial_0 + \alpha \left(b + O(\varepsilon)\right) \partial_2 \\
&... \\
\partial_k &= \delta \partial_{k-1} + \alpha \left(b + O(\varepsilon)\right) \partial_{k+1} \\
&... \\
\partial_{T-t-2} &= \delta \partial_{T-t-3} + \alpha \left(b + O(\varepsilon)\right) \partial_{T-t-1} \\
\partial_{T-t-1} &= \delta \partial_{T-t-2} + \alpha \left(b' + O(\varepsilon)\right) \partial_{T-t} \\
\partial_{T-t} &= \delta \partial_{T-t-1}
\end{aligned}$$

Define the recursive sequence $\{z_t\}_{t=0}^{T-t}$ as $\alpha b z_k - z_{k-1} + \delta z_{k-2} = 0$. The sequence is given by $z_k = Ae_1^k + Be_2^k$ where

$$e_1 = \frac{1 + \sqrt{1 - 4\delta\alpha b}}{2\alpha b} > e_2 = \frac{1 - \sqrt{1 - 4\delta\alpha b}}{2\alpha b}$$

and A, B are constants. Note

$$4\delta \left(1 + \frac{\omega}{\upsilon}\right) \left(1 - \delta \left(1 + \frac{\omega}{\upsilon}\right)\right) < 1 < 1 + \frac{\omega}{\upsilon}$$

which implies $4\delta\alpha b = 4\delta \left(1 - \delta \left(1 + \frac{\omega}{\upsilon}\right)\right) < 1$. So, the roots e_1 and e_2 are real numbers.

Pin down A and B by imposing two conditions on the end points of the sequence $\{z_s\}_{s=0}^{T-t}$:

$$\begin{aligned}
z_0 &= 1 + \alpha n_i z_1 \\
b z_{T-t} &= b' \delta z_{T-t-1}
\end{aligned}$$

Now, the sequence $\{z_s^*\}_{s=0}^{T-t}$ given by $\{z_s^*\}_{s=0}^{T-t-1} = \{z_s\}_{s=0}^{T-t-1}$ and $z_{T-t}^* = z_{T-t} \frac{b}{b'}$ satisfies the recursions of ∂_t modulo the $O(\varepsilon)$ terms. So, the solution to the recursions is given by $\partial_s = z_s^* + O(\varepsilon)$.

The remaining step is to find A, B using the imposed conditions. We have

$$\begin{aligned}
A + B &= z_0 = 1 + \alpha n_i z_1 = 1 + \alpha n_i \left(Ae_1 + Be_2\right) \\
b \left(Ae_1^{T-t} + Be_2^{T-t}\right) &= b z_{T-t} = b' \delta z_{T-t-1} = b' \left(Ae_1^{T-t-1} + Be_2^{T-t-1}\right)
\end{aligned}$$

Then some algebra yields

$$A = \frac{1}{1-\alpha n_i e_2}\left(\frac{1-\alpha n_i e_1}{1-\alpha n_i e_2} - \frac{be_1^{T-t} - b'e_1^{T-t-1}}{be_2^{T-t} - b'e_2^{T-t-1}}\right)^{-1}$$
$$B = \frac{1}{1-\alpha n_i e_1}\left(\frac{1-\alpha n_i e_2}{1-\alpha n_i e_1} - \frac{be_2^{T-t} - b'e_2^{T-t-1}}{be_1^{T-t} - b'e_1^{T-t-1}}\right)^{-1}$$

Then plugging in A and B, we have

$$\begin{aligned}
z_k^* &= Ae_1^k + Be_2^k \\
&= \frac{e_2^k}{1-\alpha n_i e_2}\left(1 - \left(\frac{e_2}{e_1}\right)^{T-1-t-k}\frac{be_2 - b'}{be_1 - b'}\right)\left(1 - \left(\frac{e_2}{e_1}\right)^{T-t-1}\frac{1-\alpha n_i e_1}{1-\alpha n_i e_2}\frac{be_2 - b'}{be_1 - b'e_1}\right)^{-1} \\
&= \frac{e_2^k}{1-\alpha n_i e_2}\left(1 - O\left(\left(\frac{e_2}{e_1}\right)^T\right)\right)
\end{aligned}$$

Therefore

$$\partial_k = \frac{e_2^k}{1-e_2\alpha n_i} + O\left(\left(\frac{e_2}{e_1}\right)^T\right) + O(\varepsilon)$$

Denoting $e \equiv e_2$ completes the proof.

(Proof of Proposition 3 and Proposition 4) To simplify notation, denote $x_\varepsilon = x + O(\varepsilon)$ throughout the proof for any variable x.

Potentials are affine in shocks. The effects of all shocks, except ω_o are negligible. So $W = \mathbb{E}[W] + E \cdot (\omega - \omega_o)$ for some $E > 0$.

The predecessors of the leaves of the trees rooted in $\mathbb{R}^{T-1}$ have r'_ε downstream connections. The marginal connected successor of a root $i \in \mathbb{R}^{T-1}$ is (i, r'_ε). So, the integral of the payoffs of i's downstream connections is $\int_{y=0}^{r'_\varepsilon} \varepsilon y \mathtt{d}y = \varepsilon\frac{1}{2}\left(r'_\varepsilon\right)^2$. For trees rooted at $\mathbb{R}^t$ for $r \leq T-2$, this quantity is $\varepsilon\frac{1}{2}\left(b'_\varepsilon\right)^2$. Denote M_t the λ_T-measure of the leaves of trees that have roots in $\mathbb{R}^t$. Then expected welfare is

$$\varepsilon\frac{1}{2}\left(\left(b'_\varepsilon\right)^2\sum_{t=0}^{T-2} M_t + \left(r'_\varepsilon\right)^2 M_{T-1}\right)$$

Denote f_t the fraction of roots in depth t. Note that for $t \leq T-2$, $M_t = \mu^t f_t r_\varepsilon b_\varepsilon^{T-t-2} b'_\varepsilon$ and for $t = T-1$, $M_{T-1} = \mu^{T-1} f_{T-1} r'_\varepsilon$.

Inductively, we have $1 - f_{t+1} = f_t\frac{r_\varepsilon}{\mu} + (1-f_t)\frac{b_\varepsilon}{\mu}$ for all $t \leq T-2$. So $f_{t+1} = 1 - \frac{b_\varepsilon}{\mu} - f_t\left(\frac{r_\varepsilon - b_\varepsilon}{\mu}\right)$.

So for $t \leq T-1$

$$f_t = \left(1 - \frac{b_\varepsilon}{\mu}\right) \frac{1 - \left(-\frac{r_\varepsilon - b_\varepsilon}{\mu}\right)^t}{1 + \frac{r_\varepsilon - b_\varepsilon}{\mu}}.$$

Then

$$\begin{aligned}\sum_{t=0}^{T-2} M_t &= \sum_{t=0}^{T-2} \mu^t r_\varepsilon b_\varepsilon^{T-t-2} b'_\varepsilon \left(1 - \frac{b_\varepsilon}{\mu}\right) \frac{1 - \left(-\frac{r_\varepsilon - b_\varepsilon}{\mu}\right)^t}{1 + \frac{r_\varepsilon - b_\varepsilon}{\mu}} \\ &= r_\varepsilon b'_\varepsilon \frac{\mu - b_\varepsilon}{\mu + r_\varepsilon - b_\varepsilon} \left(\frac{\mu^{T-1} - b_\varepsilon^{T-1}}{\mu - b_\varepsilon} - \frac{b_\varepsilon^{T-1} - \left(-\left(r_\varepsilon - b_\varepsilon\right)\right)^{T-1}}{r_\varepsilon} \right)\end{aligned}$$

and

$$\begin{aligned}M_{T-1} &= \mu^{T-1} r'_\varepsilon \left(1 - \frac{b_\varepsilon}{\mu}\right) \frac{1 - \left(-\frac{r_\varepsilon - b_\varepsilon}{\mu}\right)^{T-1}}{1 + \frac{r_\varepsilon - b_\varepsilon}{\mu}} \\ &= r'_\varepsilon \frac{\mu - b_\varepsilon}{\mu + r_\varepsilon - b_\varepsilon} \left(\mu^{T-1} - \left(-\left(r_\varepsilon - b_\varepsilon\right)\right)^{T-1}\right)\end{aligned}$$

So

$$\begin{aligned}\frac{\mathbb{E}[W]}{\mu^T} &= \varepsilon \frac{1}{2\mu^T} \left(\left(b'_\varepsilon\right)^2 \sum_{t=0}^{T-2} M_t + \left(r'_\varepsilon\right)^2 M_{T-1} \right) \\ &= \varepsilon \left(\frac{\mu - b}{2\mu\left(\mu + r - b\right)} + O(\varepsilon) \right) \left(\left(r'\right)^3 + \frac{r}{\mu - b} \left(b'\right)^3 + O(\varepsilon) + O\left(\left(\frac{b}{\mu} \right)^T \right) \right) \end{aligned} \tag{8}$$

Next, recall proof of Proposition 2. The leaves whose potentials (hence payoffs) have derivative ∂_T with respect to ω_o has a λ_T-measure of $r_\varepsilon b_\varepsilon^{T-2} b'_\varepsilon$. So $E = r_\varepsilon b_\varepsilon^{T-2} b'_\varepsilon \partial_T$. Recall $\partial_T = z_T^* + O(\varepsilon) = z_T \frac{b}{b'} + O(\varepsilon)$ and

$$\begin{aligned}z_T &= e_1^T \frac{1}{1 - \alpha r e_2} \left(\frac{1 - \alpha r e_1}{1 - \alpha r e_2} - \frac{b e_1^T - b' e_1^{T-1}}{b e_2^T - b' e_2^{T-1}} \right)^{-1} \\ &+ e_2^T \frac{1}{1 - \alpha r e_1} \left(\frac{1 - \alpha r e_2}{1 - \alpha r e_1} - \frac{b e_2^T - b' e_2^{T-1}}{b e_1^T - b' e_1^{T-1}} \right)^{-1} \\ &= \frac{e_2^T}{1 - \alpha r e_2} \left(1 - \frac{b - b' e_2^{-1}}{b - b' e_1^{-1}}\right) \left(1 - \frac{1 - \alpha r e_1}{1 - \alpha r e_2} \frac{b e_2^T - b' e_2^{-1}}{b e_1^T - b' e_1^{-1}}\right)^{-1}\end{aligned}$$

So,

$$E = r_\varepsilon b_\varepsilon^{T-2} b'_\varepsilon \partial_T = r_\varepsilon b_\varepsilon^{T-2} b'_\varepsilon \left(O(\varepsilon) + \frac{b}{b'} \frac{e_2^T}{1-\alpha r e_2} \left(1 - \frac{b - b' e_2^{-1}}{b - b' e_1^{-1}} \right) \left(1 - \frac{1-\alpha r e_1}{1-\alpha r e_2} \frac{b e_2^T - b' e_2^{-1}}{b e_1^T - b' e_1^{-1}} \right)^{-1} \right) \tag{9}$$

Combining (8) and (9) gives ex-post welfare W.

Note that $\delta > \frac{1}{2}\frac{v}{v+\omega}$ implies $e_1, e_2 > 1$, and $\delta < \frac{1}{2}\frac{v}{v+\omega}$ implies $e_1, e_2 < 1$. Then, we have

$$\lim_{\varepsilon \to 0} W = (\omega - \omega_o) (b e_2)^T \frac{r}{b} \frac{1}{1-\alpha r e_2} \left(1 - \frac{b - b' e_2^{-1}}{b - b' e_1^{-1}} \right) \left(1 - \frac{1-\alpha r e_1}{1-\alpha r e_2} \frac{b e_2^T - b' e_2^{-1}}{b e_1^T - b' e_1^{-1}} \right)^{-1}$$

$$\lim_{T \to \infty} \lim_{\varepsilon \to 0} \frac{W}{(b e_2)^T} = (\omega - \omega_o) \frac{r}{b} \frac{1}{1-\alpha r e_2} \left(1 - \frac{b - b' e_2^{-1}}{b - b' e_1^{-1}} \right) \left(1 - \frac{1-\alpha r e_1}{1-\alpha r e_2} \cdot \left\{ \begin{array}{ll} 0 & if\ \delta > \frac{1}{2}\frac{v}{v+\omega} \\ \frac{e_1}{e_2} & if\ \delta < \frac{1}{2}\frac{v}{v+\omega} \end{array} \right\} \right)^{-1}$$

These imply

$$\lim_{\varepsilon \to 0} \mathbb{E}[W] = 0$$

and

$$\lim_{T \to \infty} \lim_{\varepsilon \to 0} \left(\frac{\mathbb{V}[W]}{\mathbb{V}[\omega_o]} \right)^{\frac{1}{2T}} = e_2 b$$

(Proof of Theorem 1) At the point of interventions, the connections rates are fixed. Expected welfare consists of link benefits net of contagion costs. Using the proofs of Proposition 3 and Proposition 4, we find that aggregate connection benefits are $K + H$ where

$$K = \left((v - \varepsilon b'_\varepsilon) \sum_{t=0}^{T-2} M_t + (v - \varepsilon r'_\varepsilon) M_{T-1} \right),$$
$$H = \varepsilon \frac{1}{2} \left((b'_\varepsilon)^2 \sum_{t=0}^{T-2} M_t + (r'_\varepsilon)^2 M_{T-1} \right).$$

The term H is the integral of the connection benefits of the connected leaves over the connection benefit of the marginal connected leaves. The term K is the connection benefit of the marginal connected leaf, integrated over all connected leaves.

In equilibrium, endogenous connection rates increase to the point where the expected contagion cost is exactly K.

At the time of interventions, the connection rates were chosen, but the contagion cost K did not materialize. So, the cost K can be reduced by reducing the contagion rates. K is

bounded away from zero regardless of ε. So, for any given T, there is $C^*_\theta(T)$ independent of ε such that if $C_\theta < C^*_\theta(T)$, then it is optimal to intervene. Given such a T and C_θ, agents foresee the intervention, choose connection rates with respect to the reduced contagion rate $\theta\phi_\theta$. This changes the values to K^* and H^*. K^* is canceled out. Welfare is $H^* - C_\theta$. So, the welfare gain is $O(\varepsilon) - C_\theta$ which is negative for small enough ε.

Furthermore, reducing θ to $\theta\phi_\theta$ increases *be* so that the welfare variance increases by Proposition 4. (The only exception is the case of reducing δ when $\delta < \frac{1}{2}\frac{\upsilon}{\upsilon+\omega}$.)

(Proof of Lemma 1) Straightforward.

(Proof of Proposition 5) Straightforward.

(Proof of Lemma 2) The principal must pick $\hat{\theta}$ such that the cost of contagion denoted by $K(\hat{\theta}, n)$ 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 $\alpha\delta n < 1$. The leaves would not connect up to the point of $\alpha\delta n \geq 1$ 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, $-c_1(\hat{\theta}; \theta) \geq 0$ decreases 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. 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 Proposition 6) Case of $\hat{\alpha}$: The expected welfare is $n\left(\upsilon - \frac{\delta(1+\hat{\alpha}n)\omega}{1-\hat{\alpha}\delta n}\right) - \frac{1}{\eta}\frac{1}{1+\gamma}(\alpha - \hat{\alpha})^{1+\gamma}$. Consider an 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 that the individual cost of contagion must increase 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 $\lim_{\eta\to 0}\hat{\alpha}_\eta = \alpha$ and (iii) imply that $\lim_{\eta\to 0} n_\eta = n^*$ of the case without intervention.

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

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

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 the intervention increases in η.

The connection benefits net of the cost of contagion are 0 in expectation. So, welfare is decreasing in η.

The variance in 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 also increases 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 FOC, for any small η, the individual cost of contagion $\frac{\hat{\delta}_n\omega(1+\alpha n)}{1-\alpha\hat{\delta}_n n}$ increases in n at equilibrium $n = n_\eta$, $\hat{\delta}_\eta$ is decreasing, and n_η is increasing. The expected welfare is the cost of the 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 increases in η. So, welfare is decreasing. The variance in welfare is

$$\mathbb{V}\left[n_\eta\left(\upsilon - \frac{\delta\left(\omega_o+\alpha n_\eta\omega\right)}{1-\alpha\hat{\delta}_\eta n_\eta}\right)\right] \propto_\eta \frac{n_\eta}{1-\alpha\hat{\delta}_\eta n_\eta} \propto_\eta \frac{1}{\hat{\delta}_\eta\left(\frac{1}{n_\eta}+\alpha\right)}$$

that increases 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 $\frac{1+\alpha n}{1-\alpha\delta n}\hat{\omega}_n$ increases 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 Proposition 7) For a given n, the 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, the optimal policy is to interfere iff $\upsilon+\omega-\frac{\delta\omega_o+\omega}{1-\alpha\delta n}<-\kappa$. When there is interference, each agent has 0 payoff. Thus, the expected individual 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 regardless of ω_o, and each agent has payoff 0. However, such n is not robust to a small probability of interference failure. Each connected leaf would have negative payoff $\upsilon+\omega-\frac{\delta\omega_o+\omega}{1-\alpha\delta n}<-\kappa$ when 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 $\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}=\left(\upsilon+\omega\right)\left(1-\frac{2\delta+1}{\delta+1}\right)=-\left(\upsilon+\omega\right)\frac{\delta}{\delta+1}$. Thus the no-intervention outcome is an equilibrium when $\kappa>\frac{(\upsilon+\omega)\delta}{\delta+1}\equiv\overline{\kappa}$.

If $\upsilon+\omega-\frac{2\omega\delta+\omega}{1-\alpha\delta n}<-\kappa<\upsilon+\omega-\frac{\omega}{1-\alpha\delta n}$, the 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 quantity 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:

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

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 decreases in κ. Denote $x=\frac{\omega}{1-\alpha\delta n_\eta}$ and

$y = \upsilon + \omega$. The expected cost of interference is

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

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

B.2 Self-contained applications

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

B.2.1 Coordination games and interventions

(Proof of Proposition 8) Corollary of Propositions 11, 12, 13.

Define the parameters in a more general way. Let α_i, β_i, g_i be heterogeneous 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 11. *The unique stable network is given as follows.*

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

Under $\omega > 0$, both followers follow if $\alpha_l\alpha_f > \kappa$, and none follows 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 $\beta_l > g_l$, $U_{l,d}$ increases 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 receives a bad shock or all 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 12. *Under $\omega + \omega' < 0$ or $\omega > 0$ there are no transfers. Accordingly, the unique stable network is the same as 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 the links of i' 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 has a bad shock, there are two cases. If the third agent is not connected to i or j, then i and j do not need transfers and choose 1. So, the only case where an optimal and positive transfer is possible is when all agents are connected, l and one f_i have good shocks, and the other f_j has a bad shock. Due to complementarities, it is optimal that l and f_i both choose 0 or 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 this optimal action profile. In this case there are never any transfers and the network formed is the same as in 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 an 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 13. *Assume $\omega' > -\omega > 0$ and $\alpha_l \alpha_f > \kappa > \alpha_l \alpha_f^2$. The variance in 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 the parameters.*

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

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

Some algebra yields that in the presence of interventions

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

Then by rearranging terms I get

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

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

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

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

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

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 $c = \alpha_f\alpha_l\left(v_f + v_l - 2(1-\alpha_f)\beta_l\right) - x$. (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

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

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.

B.2.2 Epidemics and protective measures

(Proof of Proposition 9) *Stability part:*

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

The stability is characterized as follows. If $V_1 < 0$, there are no links. If $V_1 > 0 > \max\{V_2, V_1 - \Delta\}$, then one b-type matches a_1, and the other does not match either a_1 or a_2. If $V_2 = \max\{V_2, V_1 - \Delta\} > 0$, then one b-type matches a_1 one mathces a_2. If $V_1 - \Delta = \max\{V_2, V_1 - \Delta\} > 0$, then both b-types match a_1. Regarding m, these bounds correspond to

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

Then the conditions are as follows: If $m > m_1^*$, there are no links. If $m_1^* > m > \max\{m_2^*, m^*\}$, then one matches a_1 and the other does not match. If $m_2^* > m > m^{**}$, then one to a_1 one to a_2. If $\min\{m^{**}, m^*\} > m$, then both match a_1. Note that

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

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

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

means $m_2^* < m^* \implies m^* < m^{**}$ by picking $m = m^* - \epsilon$. So I 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 matches a_1, and the other does not match. If $m^* > m$, then both match a_1.

Next, consider $m_2^* > m^* > m^{**}$. If $m > m_1^*$, there are no links. If $m_1^* > m > m_2^*$, then one matches a_1, and the other does not match. If $m_2^* > m > m^{**}$, then one matches a_1 and the other matches a_2. If $m^{**} > m$, then both match a_1.

Thus, defining $m_1 \equiv m_1^*$, $m_2 \equiv \max\{m_2^*, m^*\} \leq m_3 \equiv \min\{m^{**}, m^*\}$ completes 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

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

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

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

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

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

When there are no matches, 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

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

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

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

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

B.2.3 Supply chains and subsidies

(Proof of Proposition 10) Corollary of Propositions 14, 15, 16.

Proposition 14. *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 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 an interim payoff $-k + 2\delta(1-\delta)p'$. If it produces 2, it has interim payoff $-2k + (\delta^2 + 2\delta(1-\delta))\, p'$. 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 the smaller payoff has an ex-ante payoff of $\alpha_{u_2}\delta\,(-c + p - p')$. Then they choose separate suppliers if and only if $\frac{\alpha_{u_2}}{\alpha_{u_1}} > 1 - \delta + \frac{\delta}{2}$.

Proposition 15. *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 $e_u = k_u$, the welfare is given by $\sum_{d \in D} p q_d - \sum_{u \in U} k_u q_u$. 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 are $s_d = c' - p_D$ 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 that 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 16. *Suppose that $\alpha_{u_2} \approx \alpha_{u_1} = \alpha < 1 - \frac{\delta}{2(1-\delta)}$ and $k > \delta^2 p'$. The expectation and variance of welfare are 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,

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

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

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