Network Hazard and Bailouts*

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Abstract

This paper studies a model of firms with endogenous bilateral exposures and government bailouts. It is shown that the anticipation of bailouts makes firms less concerned with the counterparty choices of their counterparties. This "network hazard" gives rise to large central firms. Bailouts can mitigate contagion but they can not restore output losses. Consequently, idiosyncratic bad shocks to large central firms generate large welfare losses. As such, bailouts create welfare volatility and systemic risk. Surprisingly, moral hazard on risk-return dimension is mitigated by bailouts. Ex-ante regulations can induce discontinuous changes in the network.

JEL classifications: D85, G01, H81.

Keywords: Contagion, Stability, Network Formation, Bailouts, Interventions, Network Reactions, Interconnectedness, Systemic Risk, Volatility, Network Hazard, Moral Hazard

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1 Introduction

The financial crisis of 2008 raised awareness about dangers of systemic risk: the risk that the failure of a few individual firms might, through the interconnectedness of the system, damage the economy as a whole. The structure of the network and shocks are essential for the patterns of contagion (see Acemoglu, Ozdaglar and Tahbaz-Salehi (2015a), Elliott, Golub and Jackson (2014), Anderson, Paddrik and Wang (2018a)). However, most economic networks are formed endogenously by firms who take the possibility of contagion into account while forming interconnections (see Erol and Vohra (2018), Elliott, Hazell and Georg (2018)). Furthermore, the inability of governments to commit to not intervene suggests that an ex-post response to systemic risk, in the form of bailouts, is unavoidable.[1] This paper studies how the anticipation of bailouts affect the network structure and systemic risk in the presence of contagion.

In an interconnected context, bailouts meant to mitigate systemic risk benefit not only the bailout recipients, but also direct or indirect counterparties of bailout recipients. For example, Ford's CEO Alan Mulally pleaded for a bailout of Ford's arch-rival General Motors in his testimony before the Senate Banking Committee in 2008, arguing about the risks of contagion through common suppliers of Ford and General Motors.[2] Similar channels are relevant in the context of financial networks. Until November 2009, Goldman Sachs, UBS, Deutsche Bank, Societe Generale and Merrill Lynch received $62 billion as a result of AIG's bailout.[3,4] Bailouts of failing firms are often criticized because they encourage excessive risk taking by firms that expect to be bailed out. Indeed, much of the literature has focused on moral hazard in individual investment choices. In this paper, we study the network effects of interventions and regulations. In particular, in an interconnected system where firms ben-

[1] See, for example, "The Ripple Effect: Why Failure of the Big Three is not an Option", report by Representative Carolyn B. Maloney, Dec. 2008.

[2] Senate Hearing, Committee on Banking, Housing, and Urban Affairs, Nov. 18, 2008.

[3] "Did Goldman Benefit The Most From AIG Bailout?" by Liz Moyer, Forbes, Jan. 2010.

[4] "In U.S. Bailout of A.I.G., Forgiveness for Big Banks" by Louise Story and Gretchen Morgensen, NY Times, June 2010.

efit from the bailouts of their trading partners, how does the anticipation of bailouts shape the counterparty choices of firms that anticipate such bailouts?

We provide three insights. First, the anticipation of bailouts makes firms less concerned with the counterparty choices of their counterparties. We call this new channel "*network hazard*." Network hazard makes the endogenous network more interconnected and more concentrated. As a result, there is higher systemic risk as well as higher welfare volatility and output volatility in the presence of interventions compared to the absence of interventions. Second, despite network hazard, surprisingly, individual moral hazard on the risk-return dimension is mitigated by bailouts. Finally, tightening ex-ante regulations such as higher capital requirements can trigger abrupt changes in the network structure and discontinuous drops in welfare past a tipping-point.

In the model, firms with existing investments and existing liabilities to external creditors decide to make joint investments. Then firms receive shocks that determine the return on their investments. Firms with bad shocks (bad firms) suffer large losses to their returns, and so they can not repay their external creditors and default. For each firm, joint investments with defaulting firms lead to reduced yields. Accordingly, firms with good shocks (good firms) which have sizable counterparty defaults might become unable to repay external creditors and default. Defaults then cascade. In order to prevent inefficient losses in investments, the government can choose to make targeted capital injections to enable firms to repay their liabilities.

In the absence of interventions, there are two forces at play during network formation. A firm can suffer losses due to its bad counterparties. Call this "first-order counterparty risk." A firm can also suffer losses due to defaults of its good counterparties who are defaulting due to their own bad counterparties. Call this the "second-order counterparty risk." Consider a pair of firms who are counterparties. If each have other counterparties that are not counterparties of the other, each firm in the pair is exposed to second-order counterparty risk through the other. Instead of each firm in the pair having a distinct counterparty, they can have one common counterparty in order

to mitigate second-order counterparty risk through each other. This force pushes counterparties towards having common counterparties, and results in high clustering in the network. The second force is the tradeoff between the first-order counterparty risk and the second-order counterparty risk. Suppose that there is a firm that faces a small exogenous risk of a bad shock and has a large credit line to borrow and make investments. Call this the large firm and the others small firms. All else fixed, a small firm would prefer to become a counterparty with the large firm instead of a small firm in order to reduce its own first-order counterparty risk. Nevertheless, if the large firm has an excessively large number of counterparties, it can transmit contagion and create high second-order counterparty risk for its counterparties. This tradeoff limits small firms' willingness to expose themselves to the large firm in case the large firm is highly exposed to other small firms. As a result, (assuming that the large firm is willing to form a large number of links,) only a limited number of small firms choose to become counterparties with the large firm – the network features only a limited amount of concentration of links around the large firm. Therefore, the network formed in the absence of intervention features high clustering and low concentration.

In the presence of interventions, the sizes of bad shocks determine the structure of optimal interventions. If bad shocks are large (such that the net ex-post returns to investments of bad firms are negative) it is optimal for the government to let bad firms default because their projects are not worth saving. However, good firms have potential high returns to their investments, which justifies their bailout in case they face default. Contagion is stopped by interventions after the initial defaults by bad firms. Since bad firms default and good firms do not as a consequence of optimal interventions, first-order counterparty risk is not altered but second-order counterparty risk is eliminated. On the other hand, if the bad shocks are small, it is optimal to bailout bad firms because their investments are also worth saving. Contagion is stopped immediately by saving bad firms. Then good firms do not need bailouts. In this case, first-order counterparty risk is eliminated by interventions, which also eliminates second-order counterparty risk. In either case of the shocks,

second-order counterparty risk is eliminated. Then, during network formation, both forces outlined earlier become irrelevant. Second-order counterparty risk is eliminated and so there is no need for high clustering. There is no longer a tradeoff between first- and second-order counterparty risks, which makes the large firm obtain a very large number of counterparties. The network features low clustering and high concentration due to the anticipation of bailouts.

This may sound like contagion becomes irrelevant under interventions and that there are no negative effects of the induced network structure. Nevertheless, the large firm, which is now highly interconnected to small firms due to the presence of interventions, can still receive a bad shock. When the large firm gets a bad shock, regardless of whether it receives a bailout or not, the returns to its investments suffer losses. A bailout would reduce further losses from a potential default the large firm, but not eliminate the losses resulting from the bad shock in the first place. Now that the large firm has many investments under interventions, a bad shock to the large firm causes large ex-post welfare losses. Such a bad shock prompts either widespread interventions to the good counterparties of the large firm, or a large and direct intervention to the large firm. The defaults of good firms are prevented with interventions, directly or indirectly, but the welfare and output losses due to the bad shock to the large firm is permanent. Therefore, the performance of a large number of investments and firms become highly correlated through the idiosyncratic risk of the large firm. Welfare volatility, output volatility, and systemic risk are higher in the presence of interventions than in the absence of interventions. We call these adverse effects of bailouts, via the elimination of first- or second-order counterparty risk, "network hazard." Nevertheless, average welfare is higher in the presence of interventions.

We also consider how the anticipation of bailouts interacts with other forms of moral hazard. Suppose that the large firm can choose between two risk-return profiles before network formation: the risky profile or the safer profile. Suppose that this choice can be observed by the firms in the network but not by the external creditors of firms. In this case, the large firm can potentially engage in individual moral hazard by choosing the risky profile and shifting

more risk on to its external creditors. If the large firm chooses the risky profile, it generates high first-order counterparty risk and it will no longer be a preferred counterparty over the small firms. In the light of this, consider the absence of interventions. The large firm can choose the risky profile and end up having a small number of counterparties. Instead, it can choose the safer profile and have a larger but limited number of counterparties. The tradeoff that the large firm faces involves a limited loss in the number of counterparties. The "network stakes" are small. On the other hand, in the presence of interventions, the large firm can choose a risky profile and end up having a small number of counterparties. Instead, it can choose the safer profile and have a very large number of counterparties. The tradeoff involves a large loss in the number of counterparties. The network stakes are large. In this sense, if the large firm finds it optimal to choose the safer profile in the absence of interventions, then it also finds it optimal to choose the safer profile in the presence of interventions. The possibility of interventions allows small firms to use the threat of exclusion and discipline the risk taking by the large firm. In this sense, surprisingly, bailouts can mitigate moral hazard when endogenous counterparty choices and the threat of exclusion are taken into account.

Finally we study ex-ante regulations imposed before the formation of the network from a positive standpoint. For example, the regulator can put an upper bound on total exposures for each firm in the spirit of liquidity or capital requirements, or he can impose taxes. We illustrate that as the regulations get more stringent, past a certain threshold, joint investments fall discontinuously leading to a "phase transition" in the network. This suggests that extreme care is needed when regulating networks.

Literature review. Farhi and Tirole (2012) study systemic bailouts (such as interest rate policy) and collective moral hazard. There, bailouts are not targeted to specific firms, and so firms can become jointly too-big-to-fail by correlating their investment risk. In contrast, here, bailouts are targeted so firms can not trigger bailouts by having correlated investments. Moreover, a firm is not more or less likely to get bailed-out as a function of its counterpar-

ties. The optimal policy either saves good firms or bad firms, where shocks are determined exogenously. In other words, firms can not trigger bailouts by correlating their counterparty choices either. Their counterparty choices become correlated in response to a reduced risk for contagion through network effects. Finally, risk-return type moral hazard is mitigated by the presence of interventions, not exacerbated.

Acemoglu, Ozdaglar and Tahbaz-Salehi (2015a) study how the network structure and the size of shocks affect patterns of contagion. In their model of debt contracts, a creditor A of a defaulting bank B can receive a positive repayment from B, and the amount of repayment depends on the size of the shock to B. Accordingly, the size of the shock to B can matter also for the recovery rates of creditors of A if A also defaults. Size of shocks are essential for patterns of contagion. In our model, links are joint investments and contagion spreads only via the binary default of firms. A bad shock to B makes B default, and the size of the bad shock to B does not affect A's recovery rate. This is the main distinguishing feature of our model compared to the counterparty-risk models of Eisenberg and Noe (2001) and Elliott, Golub and Jackson (2014). This simplicity allows us to characterize stable networks. The role of the size of a bad shock in our model is to determine which firms receive bailouts. Some firms are saved and some not, depending on the size of bad shocks.

Elliott, Hazell and Georg (2018) study the incentives of counterparties to correlate the returns to their investments. They show that the correlation of counterparty risk and the correlation of investment returns tend to be correlated. This is systemic risk-shifting on the external creditors. In our paper, there is no endogenous choice to correlate investment returns. The focus is the effect of interventions on the correlation of counterparty-risk. We also characterize the set of stable networks under limited liability.

Eisert and Eufinger (2018) study how banks who engage in intermediation can benefit from the bailouts of others. In their model, the network topology (extensive margins) is exogenous. Banks are located in different countries and so longer chains of exposures directly lead to a higher probability that contagion

gets stopped along the way. If all banks belonged to one country, there would not be incentives to form long chains. In this paper the network topology is endogenous. Also, firms do not benefit from longer chains of exposures neither in the absence nor in the presence of interventions.

Erol and Ordoñez (2017) study how ex-ante regulations affect the density of the network. Our results on ex-ante regulations extend their analysis to account for various forms of regulations.

This paper contributes to the literature on contagion and systemic risk in economic networks. Acemoglu, Ozdaglar and Tahbaz-Salehi (2015a), Acemoglu, Carvalho, Ozdaglar and Tahbaz-Salehi (2012), Elliott, Golub and Jackson (2014), Glasserman and Young (2015) study contagion in financial and production networks. Acemoglu and Azar (2017), Acemoglu, Ozdaglar and Tahbaz-Salehi (2015b), Erol and Vohra (2018), Elliott, Hazell and Georg (2018) study formation of financial and production networks without interventions. Bernard, Capponi and Stiglitz (2017), Galeotti, Golub and Goyal (2018), and Kanik (2018) study interventions without considering the stability of networks. This paper is the first to investigate network formation in the presence of contagion as well as the anticipation of bailouts. A longer discussion of this literature and the ties to the literature on network games at large can be found in Appendix B.1.1.

On the empirical front, to the best of our knowledge, the effects of interventions on the network structure has not been studied in production networks. As for financial networks, central banks are readily tasked with maintaining financial stability. It is hard to find an example where a government has consistently not intervened in face of systemic risk, so that banks would not anticipate any interventions. Accordingly, one can consider the historical episodes where central banks did not exist to intervene in the first place. Anderson, Erol and Ordoñez (2018b) study bank correspondence networks and the establishment of the Federal Reserve System in 1913. They find that the provision of public liquidity resulted in higher concentration of interbank deposits at regional

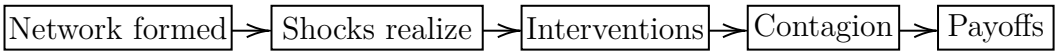
reserve city banks.[5] This is in line with the testable predictions of our model.

Structure of the paper. Section 2 studies firms with bilateral exposures facing contagion and describes the welfare criteria along with optimal interventions. Section 3 introduces the solution concept for network formation and contrasts networks that are formed in the absence and presence of interventions. We call these "network reactions" to the anticipation of interventions. Section 4 studies welfare and systemic risk consequences of the presence of interventions via network reactions, along with the interaction between network hazard and moral hazard. Finally, Section 5 studies ex-ante regulations and resulting phase transitions. Appendix A presents a more general model and corresponding results. Appendix B has extra discussions and extensions. Appendix C includes all proofs.

2 Simplified model

We begin by describing the timing of events in Figure 1. First a network is formed by firms. Then firms receive some shocks that start contagion. Before contagion starts, the government intervenes to mitigate the forthcoming contagion. Finally, given the network, shocks, and interventions, contagion unravels. Proceeding in reverse order, we begin with in Section 2.1 describing and discussing contagion for a fixed network in the absence of interventions. Section 2.2 characterizes optimal interventions by the government for a fixed network and a realization of shocks. Section 2.3 studies the impact of interventions on contagion. We study network formation in Section 3.

Figure 1: Timing of events



[5] They argue that this lead to higher vulnerability in the network. Calomiris et al. (2019) argue that the presence of the Federal Reserve Bank likely increased systemic risk by distorting banks' incentives to manage network liquidity risk. Miron (1988) and Selgin et al. (2012) show that the U.S. economy has seen more volatility since the establishment of the FED.

2.1 Model

Consider firms, financial or not, that borrow from external creditors by issuing debt. This can be via deposit accounts or corporate bonds. Then firms make investments in pairs aside of their private investments. The pairwise investments can be real projects or investments into financial instruments such as swaps that create mutual exposures. After investments, shocks to returns of assets are realized. If firms can repay their liabilities, they stay in business (continuation). Otherwise, they default, which results in the failure of their investments. Failed investments have lowered return for the non-defaulting counterparty. Defaulting firms are protected by limited liability.

Firms and the network: There is a finite set of *firms* $N = \{1, 2, ..., n\}$ with exposures to each other. The *exposure* of i to j is $e_{ji} \in \mathbb{R}_{\geq 0}$. The *network* is $\mathbf{e} = [e_{ij}]_{i,j \leq n}$. (We take $e_{ii} = 0$.) Here e_{ji} is i's contribution into its investment with j, which forms the bases of the exposure of i to j's default. Firm i also has a private investment of fixed size p_i. The total size of investments is $\sum_j e_{ji} + p_i$, which is funded by debt from external creditors, borrowed at a fixed rate r_i.[6] The net liabilities of i above and beyond $(p_i + \sum_j e_{ji})r_i$ is $z_i \geq 0$. Total liabilities are then $(\sum_j e_{ji} + p_i)r_i + z_i$.

Shocks and defaults: The returns to assets of i are stochastic and given by θ_i per unit, net of operational, regulatory, or other costs. The returns are either high or low: $\theta_i \in \Theta_i = \{\theta_{Gi}, \theta_{Bi}\}$ where θ_{Gi} and θ_{Bi} are constants with $\theta_{Bi} < r_i < \theta_{Gi}$.[7] θ_{Gi} is the *good shock* and θ_{Bi} is the *bad shock*. The shocks are drawn jointly from some distribution $\mathcal{D} \in \Delta(\otimes_i \Theta_i)$. We also call firm i *good/bad* when θ_i is good/bad. After shocks are realized and publicly observed, each firm chooses to default or continue.[8] Denote $a_i = 1$ the default choice and $a_i = 0$ the continuation choice.

Payoffs: For firm i, the total size of its investments in which the counterparty

[6] Endogenizing r_i does not change the qualitative results but introduces a lengthy analysis.

[7] We can assume shocks to the liability side as well. For example r_i could be a floating rate. We assume this away for simplicity and focus on shocks to the asset side.

[8] This is without loss of generality. Even if firms are allowed to default selectively on their investments, they either continue with all investments or default on all investments.

has defaulted is $\Sigma_j e_{ji} a_{ji}$. Returns to these assets are reduced to a fraction $1-\eta_i \in (0,1)$ due to the counterparty defaults. Here $1-\eta_i$ is the default recovery rate or η_i represents fire-sale or liquidation costs. The investments of i in which the counterparty has not defaulted sum up to size $\sum_j e_{ji}(1-a_j)$. Then the payoff of i is

$$[-\underbrace{((\Sigma_j e_{ji}+p_i)r_i+z_i)}_{\text{liabilities}}+\underbrace{\theta_i(\Sigma_j e_{ji}(1-\overbrace{a_j}^{\text{counterparty loss}})+\Sigma_j e_{ji}(1-\eta_i)a_j+p_i)}_{\text{assets}}]\underbrace{(1-a_i)}_{\text{default}}$$

Note that only the exposures of i matter for i's payoff, not the exposures to i. Denote $e_i=\Sigma_j e_{ji}$. Also denote $c_i=\Sigma_j e_{ji}a_j$ the size of counterparty defaults on i. Finally denote $\kappa_i=\frac{\theta_{Gi}\eta_i}{\theta_{Gi}-r_i}$ and $\phi_i=\frac{z_i}{\theta_{Gi}-r_i}-p_i$. Then the default payoff is 0 whereas the continuation payoff is

$$\begin{aligned}P_{Ci}(e_i,c_i) &= -\underbrace{(p_i+e_i+z_i)r_i}_{\text{liabilities}}+\underbrace{\theta_{Gi}(p_i+e_i-\eta_i c_i)}_{\text{assets}}\\ &= (\theta_{Gi}-r_i)(e_i-c_i\kappa_i-\phi_i)\\ &\propto e_i-c_i\kappa_i-\phi_i.\end{aligned}$$

Here, c_i is the endogenous outcome of defaults. e_i will be endogenized when we study network formation. The parameters κ_i, ϕ_i, θ_{Gi}, r_i are exogenously given.

Remark: Appendix A presents a general model where the default payoff can be any function $P_{Di}(e_i)$ and the continuation payoff can be any function $P_{Ci}(e_i,c_i)$ that is decreasing in c_i. In our simplified model, $P_{Di}(e_i)\equiv 0$ and $P_{Ci}(e_i,c_i)=(\theta_{Gi}-r_i)(e_i-c_i\kappa_i-\phi_i)$ is an affine function. Appendix B.1.2 describes how the general model can be applied to variations of some established counterparty risk models.

Equilibrium and contagion. Given the realized network and shocks, this game is supermodular. Then, via Topkis' Theorem, the best responses are increasing, where continuation is the higher action and default is the lower action. In return, Tarski's Theorem implies that the set of (Nash) equilibria is a complete lattice. The top element of the lattice, the best equilibrium, corresponds to the outcome of the exogenous contagion dynamics described in our model. The

steps of iterated elimination of strictly dominated strategies are isomorphic to the rounds of contagion.[9]

Contagion unfolds as follows. Since $\theta_{Bi} < r_i$ and liabilities are positive, a bad firm i gets a negative payoff if it continues. Then it chooses to default and gets 0 due to limited liability. In this manner, all bad firms default in the first round of contagion. Denote $\tilde{c}_i = \Sigma_j e_{ji} \mathbb{1}_{\theta_j = \theta_{Bj}}$. Since bad firms default in the first round of contagion, the counterparty losses of a good firm i is $\tilde{c}_i$. In the second round of contagion, a good firm i defaults if its immediate counterparty loss $\tilde{c}_i$ exceeds

$$R_i\left(e_i\right) = \frac{1}{\kappa_i}\left(e_i - \phi_i\right).$$

We call R_i the *resilience function* of i. After some good firms default in the second round of contagion, more good firms can find that their counterparty losses exceed their resilience and choose to default. At each round of contagion, counterparty losses of each firm weakly increases. Contagion stops when no more firms default in a round. Due to the standard Tarski argument, at the end of contagion, each firm has c_i^* total counterparty losses where c_i^* is the counterparty losses of i in the best equilibrium in the lattice. Hence, the outcome of contagion is identical to the best equilibrium in the lattice.

Welfare. Regarding welfare, $(e_i + p_i)r_i + z_i$ are transfers to external creditors who also make up a part of the economy. The repayment does not factor into welfare since it is simply a transfer. Welfare criterion (after shocks, before defaults) is driven by returns from assets. This is

$$W = \sum_i \theta_i(1 - a_i)\Big(p_i + \sum_j e_{ji}(1 - \eta_i a_j)\Big) \tag{1}$$

There are two inefficiencies in the contagion stage. First, if a large number of counterparties of a firm default, liabilities to external creditors force the firm into default, which leads to the failure of efficient investments. Second, when

[9] See Vives (1990) and Milgrom and Roberts (1990) for the lattice Nash equilibria. The Tarski argument is used in many contagion models, including Eisenberg and Noe (2001) and Elliott et al. (2014). Most models of counterparty risk work with the best equilibrium. To the best of our knowledge, the only exception that studies other equilibria in the lattice is Jackson and Pernoud (2019).

a firm defaults, it does not internalize the losses it causes to its counterparties.

2.2 Interventions

Suppose that there is a government who aims to maximize welfare. The government has no budget constraint. After shocks are realized and observed, before contagion, the government can intervene and implement transfers, such as by capital injections or subsidies. Denote $Q_i \geq 0$ the transfer to i. Given transfer rule $Q = (Q_i)_{i \in N}$, the liabilities of i are reduced by Q_i. We call Q optimal if it maximizes W and uses minimal transfers in doing so, given that firms play the best Nash equilibrium of the induced game.[10] Minimal transfer restriction is to ensure robustness against small costs of transfers. Interventions can potentially fix the inefficiency by enabling firms to repay their liabilities and see their efficient investments to maturity. We say a policy Q *saves* i if i receives a positive transfer and continues, whereas it would default under any transfer rule Q' with $Q'_i < Q_i$ and $Q'_{-i} = Q_{-i}$.

Proposition 1. *The unique optimal policy saves all firms with positive shocks who are facing default due to counterparty losses. The transfers make saved firms indifferent between defaulting and continuing. In particular,*

$$Q_i^* = \left[\overbrace{((p_i + e_i)r_i + z_i)}^{liabilities} - \overbrace{\theta_i(p_i + e_i - \eta_i \sum_{j:\theta_j<0} e_{ji})}^{assets}\right]^+ \mathbb{1}_{\theta_i>0}$$

The idea is simple. Firms with negative shocks are unproductive and they default even after receiving a transfer. Then the optimal policy does not entail any transfers to these firms. It is efficient for firms with positive shocks to continue their operations. If such a firm is pushed into default after sizable counterparty losses, this firm can be induced to continuation by appropriate transfers that cover part of its liabilities to external creditors.[11]

We consider two cases in what follow. The first is the case is $\theta_{Bi} < 0$ for all i. We call this the case of *large (bad) shocks*. The second is the case of *small*

[10] Notice that the game is supermodular for any transfer rule.

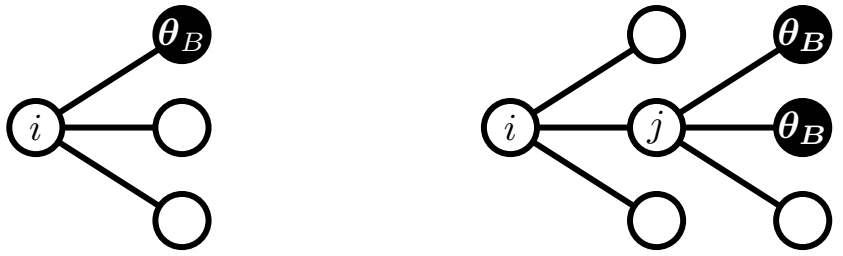
[11] In Appendix B.2.1, we consider an alternative setting in which the government can commit to transfers that are conditional on default choices. Then agents with negative shocks can also be induced into continuation. Results remain similar.

(bad) shocks: $\theta_{Bi} > 0$ for all i. By Proposition 1, under large shocks, the optimal policy is to save all good firms that are facing default due to their bad counterparties. Under small shocks, the optimal policy is to save all bad firms.[12]

2.3 Counterparty risk

In this section we study the impact of interventions on contagion. Two key notions are helpful. *First-order counterparty risk (FCR)* is the risk that losses can be incurred due to defaulting bad counterparties. *Second-order counterparty risk (SCR)* is the risk that losses can be incurred due to defaulting good counterparties who default due to their bad counterparties. These are illustrated in Figure 2. Higher order of counterparty risks can be defined analogously. Clearly, if a firm i faces no FCR, then it faces no SCR either.

Figure 2: First-order and second-order counterparty risks



(a) FCR faced by i (b) SCR faced by i through j

Figure 2-a illustrates a bad shock to one of i's counterparties. In this case, the counterparty defaults, which creates a loss for i. This risk is FCR for i. Figure 2-b illustrates two bad shocks to counterparties of j who is good counterparty of i. These bad shocks, if sufficiently many, can force j into default, which creates a loss for i. This risk is SCR for i.

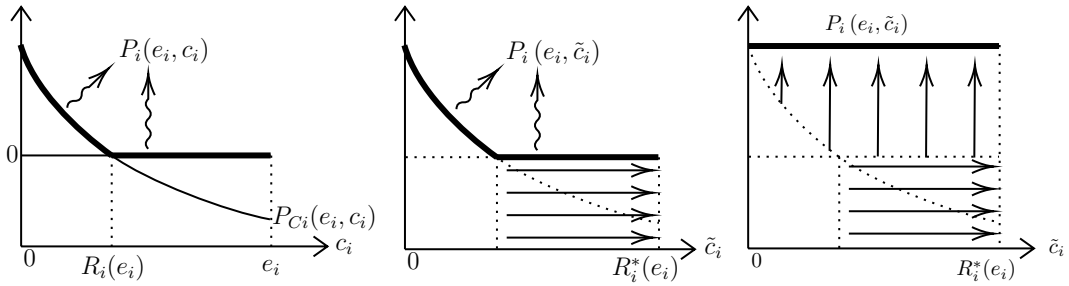
Under large shocks, only good firms in need of interventions are saved. That is, all good firms surely continue at the contagion stage. This eliminates SCR. Nevertheless, bad firms do not receive transfers and they default. Hence FCR is not altered by interventions. Under small shocks though, bad firms are saved and contagion is stopped immediately. Good firms do not need bailouts. Then

[12]Our results easily extend to the cases where for some firms bad shocks are large and for some bad shocks are small. We skip the detailed analysis of the mixed case to save space.

all firms understand that all firms will continue. This eliminates FCR as well as SCR.

In either case, SCR is eliminated. The default threshold (resilience) R is increased artificially by interventions. Firms understand that their counterparties are not going to *transmit* contagion that started elsewhere in the network. Under small shocks, additionally, FCR is eliminated which changes the function P. Firms understand that their counterparties are not going to default, in particular, they are not going to *start* contagion. These are illustrated in Figure 3.

Figure 3: Effects of interventions on counterparty risk



(a) Payoffs and resilience, Absence of interventions

(b) Large shocks, Presence of interventions

(c) Small shocks, Presence of interventions

Figure 3-a shows the payoff and resilience functions in the absence of interventions. If c_i is the extent of counterparty losses, i gets $P_i(e_i, c_i) := \max\{0, P_{Ci}(e_i, c_i)\}$ payoff. Figure 3-b shows the changes in the presence of interventions under large shocks. Only bad firms default and so the payoff function depends on $\tilde{c}_i$ instead of c_i. SCR is eliminated which artificially shifts the resilience function R_i to $R_i^*(e_i) \equiv e_i$. The payoff of i is $P_i(e_i, \tilde{c}_i) := \max\{0, P_{Ci}(e_i, \tilde{c}_i)\}$ because optimal interventions make saved firms indifferent between defaulting and continuing in order to minimize the transfers. Figure 3-c shows the changes in the presence of interventions under small shocks. Now all bad firms are saved so the continuation payoff of a good firm i shifts up to $P_i(e_i, \tilde{c}_i) := P_{Ci}(e_i, 0)$.

3 Network reactions to the anticipation of interventions

Now we move to the analysis of network formation. The notion of stability is given in Section 3.1. Our task is to demonstrate the effects the anticipation of interventions on the network formed. We do this by comparing the

stable networks formed in the absence of interventions (*in AOI*) and in the presence of interventions (*in POI*). To be precise, POI refers to the model we have described thus far whereas AOI refers to the model wherein there is no government to intervene. The first effect of the anticipation of interventions is to make the network more interconnected, studied in Section 3.2. The second effect is to make the network more concentrated, studied in Section 3.3. For each effect, we use the simplest version of the lead application to make the point.

3.1 Stability

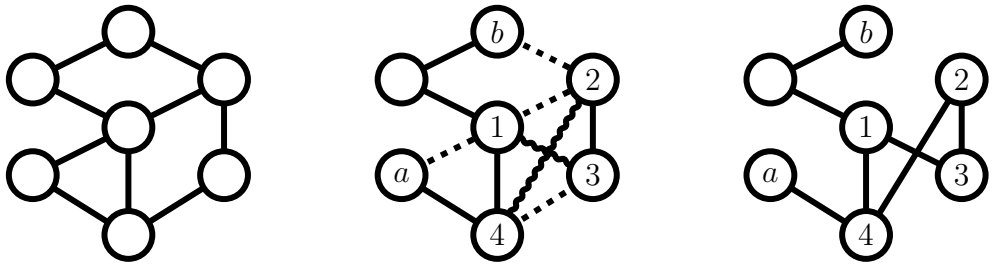
Our focus in this paper is the topology of network rather than the intensity of links. In other words we are interested in endogenous extensive margins rather than endogenous intensive margins. So we take a given non-negative weight matrix $\mathbf{w} = [w]_{i,j \leq n}$. If i and j decide to form a *link*, their exposures to each other are given by $e_{ij} = w_{ij}$ and $e_{ji} = w_{ji}$. If they do not form a link, their exposures are $e_{ij} = e_{ji} = 0$. We denote $\{i, j\} \in E$ whenever i and j form a link. The endogenous part of the network, i.e. the set of extensive margins, is represented by E. If two firms i and j end up forming a link, we call them *counterparties*. We denote N_i the set of counterparties of i.

We assume that firm i can feasibly have exposures at most $\overline{e}_i$, where $\overline{e}_i > 0$ is a constant.[13] We call N_i is feasible for i if $e_i \leq \overline{e}_i$. A network is called *feasible* if each firm has a feasible set of counterparties. Before shocks are realized, firms evaluate a network according to the expectation of their payoffs with respect to shocks, anticipating interventions and contagion that follow.

Consider a feasible network E and a subset N' of firms. A *deviation* by N' from E is one in which N' can simultaneously add any missing links within N', cut any existing links within N', and cut any of the links between N' and N/N'. Figure 4 illustrates a deviation.

[13]The exposure bound can capture the credit line available for the firm to borrow from external creditors. It can also be implied by scarce intangible assets and resources, such as human capital, reputation, relationships, that are needed to form or manage partnerships and investments.

Figure 4: A deviation



(a) Candidate network (b) Deviation (c) After deviation

Figure 4-a shows a candidate network. Figure 4-b shows a deviation by firms 1, 2, 3, and 4. They cut the links $\{1,2\}$, $\{3,4\}$, and add the links $\{1,3\}$, $\{2,4\}$, among themselves. 1 unilaterally cuts a link with non-deviator a and 2 unilaterally cuts a link with non-deviator b. The resulting network after the deviation is shown in Figure 4-c.

A *feasible deviation* by N' is a deviation after which the resulting network is feasible. A *profitable deviation* by N' from E is a feasible deviation in which the resulting network yields strictly higher payoff to all members of N'. A network E is *Strongly Stable* if there are no subsets of N with a profitable deviation from E. A network E is *Pareto Efficient Strongly Stable* if it is Strongly Stable and Pareto efficient.[14] We use *stable* as a shorthand for Pareto Efficient Strongly Stable. We assume that agents form a stable network before shocks.

Going forward we denote $d_i = |N_i|$ the number of counterparties of i (namely the *degree* of i), f_i the number defaulting counterparties of i, and b_i the number of bad counterparties of i.

3.2 Intervention induced interconnectedness

In this section, we show how interventions make the network more interconnected. For this purpose, it suffices to study identical firms.

Assumption 1. *All firms are identical:* $\overline{e}_i = \overline{e}$, $\phi_i = \phi$, $w_{ij} = w > 0$ *for all* i, j. *Firms are not guaranteed to default, but they are subject to contagion risk:* $\overline{e} > \phi > w$ *and* $\kappa \geq 1$. *Shocks are i.i.d., good with probability* $\alpha \in (0,1)$.

Throughout Section 3.2 we maintain Assumption 1. Let $b \sim \mathbb{B}[d, 1-\alpha]$ be a

[14] Versions of strong stability are introduced in Dutta and Mutuswami (1997) and Jackson and Van den Nouweland (2005). See Appendix B.2.2 for a discussion of the solution concept.

Binomial random variable with d independent trials and $1-\alpha$ success probability. Define $V(d) = \mathbb{E}_b[[d - b\kappa - \phi/w]^+]$ and denote $d^* = \arg\max_{dw \leq \overline{e}} V(d)$.[15,16] In order to rule out some typical integer problems we assume that w divides $\overline{e}$ and $2(d^*+1)$ divides n.[17] Fixing these, we study small shocks in Section 3.2.1 and large shocks in Section 3.2.2.

3.2.1 Small shocks and FCR

Consider the case of small shocks. Recall that the optimal policy is to save all bad firms. Thus, FCR is eliminated by interventions.

Proposition 2. *Suppose that Assumption 1 holds and bad shocks are small. In the absence of interventions, a network is stable if and only if it consists of disjoint (d^*+1)-cliques.*[18] *In the presence of interventions, a network is stable if and only if it is $(\overline{e}/w)$-regular.*[19]

The changes in the network structure are illustrated in Figure 5. The network in AOI is given by Figure 5-*a* whereas in POI it is given by Figure 5-*c*. There are two changes to the network in response to the anticipation of interventions. First, the exposures of firms go up from d^*w to $\overline{e}$. Second, cliques dissolve into an interconnected network. Note that $((\overline{e}/w)+1)$-cliques also are $(\overline{e}/w)$-regular, hence stable in POI. So, the change in the structure is driven by a different channel from the one that increases the exposures from d^*w to $\overline{e}$.

The reason the exposures go up is that FCR is eliminated. In choosing their total exposures (proxied by the total number of counterparties), instead of maximizing $\mathbb{E}_b[[d - b\kappa - \phi/w]^+]$ firms now maximize $\mathbb{E}_b[[d - \phi/w]^+]$. The change

[15] x^+ denotes $\max\{x, 0\}$.

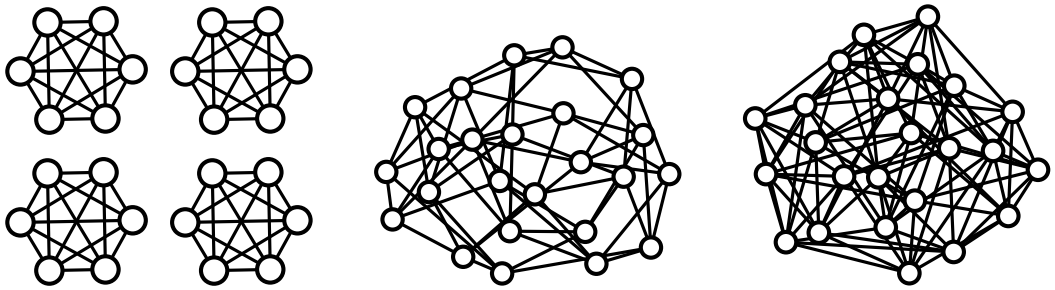
[16] The maximizer d^* is generically unique. We ignore the non-generic cases.

[17] Integer problems are typical in network formation and coalition formation literature. We can still pin down the stable networks without the divisibility assumptions but this effort requires much extra notation and yields no further insight.

[18] A *clique* is subnetwork of firms that are all counterparties of each other. A subnetwork is a subset of firms and all of their links with each other that are present in the original network. *Order* is the number of firms in a subnetwork. A $(d+1)$-*clique* is a clique or order $d+1$. *Disjoint* refers to a subnetwork whose firms are not counterparties with any firm outside the subnetwork.

[19] A *d-regular* network is one in which all firms have d counterparties.

Figure 5: Network reactions to the anticipation of interventions: increased interconnectedness



(a) Absence of interventions

(b) Large shocks, Presence of interventions

(c) Small shocks, Presence of interventions

Figure 5-a illustrates the network formed in AOI. Firms form cliques to increase clustering. Figure 5-b and -c illustrate the networks that are formed in POI under large and small shocks, respectively. When shocks are large and so only good firms are saved, the network becomes interconnected but total exposures of individual firms do not change. When shocks are small and so bad firms are saved, the network becomes interconnected and the exposures per firm go up.

in the network structure is because SCR is eliminated. In AOI, firms desire high clustering to protect themselves against contagion. When they anticipate that, in POI, their good counterparties will continue regardless, firms no longer need to form cliques.

Nonetheless, since SCR implies FCR, it is difficult to disentangle the two effects and formalize this insight. For this reason, next, we study large shocks. Under large shocks, only SCR is eliminated and the effects are better disentangled.

3.2.2 Large shocks and SCR

Consider the case of large shocks. Recall that the optimal policy is to save good firms who are facing default due to their bad counterparties. Thus, SCR is eliminated by interventions.

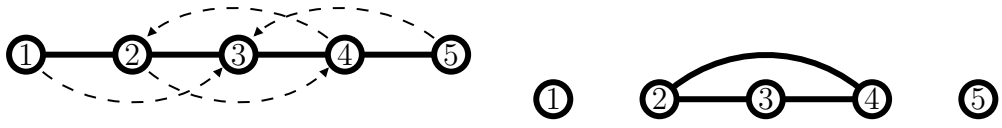
Proposition 3. *Suppose that Assumption 1 holds and bad shocks are large. In the absence of interventions, a network is stable if and only if it consists of disjoint* $(d^* + 1)$*-cliques. In the presence of interventions, a network is stable if and only if it is* d^**-regular.*

The network in AOI and POI are illustrated in Figure 5-*a* and 5-*b*, respectively.

This results makes the contrast between FCR and SCR more clear. When FCR is not altered but SCR is eliminated, firms do not change their total exposures. Nevertheless, the effect on the topology of the network persists. Instead of clusters, firms form any interconnected network.

In fact, the very reason firms form clusters in AOI is the SCR. Consider a candidate network and suppose that two firms i and j have counterparties that are not counterparties of the other, say $i' \in N_i \backslash N_j$ and $j' \in N_j \backslash N_i$. Then a bad shock to i' can make i switch to default on the margin, which creates losses for j. Likewise for j' and i. Then i and j have incentives to connect to only i' or only j'. This needs the consent of i' or j', yet, at a collective level, since all pairs have incentives to have common counterparties, they form dense clusters and maximize the number of their common counterparties. This collective force is pictured in Figure 6 with five firms. When SCR is eliminated by prospective interventions, this channel breaks down. i no longer needs to be concerned with j' because j will be saved even if j' were to marginally push j into default. However, firms are not incentivized (during network formation, by the ex-post interventions after shocks) to form more or less links compared to AOI. Firms with bad shocks are not saved and they default. Since FCR is not altered, exposures of each firm remain d^*w in POI.

Figure 6: Rationale for clustering in the absence of interventions



(a) Chains of exposures lead to redundant SCR

(b) Clustering eliminates redundant SCR with same total exposure

Figure 6-a shows SCR under the lack of clustering. 3 is exposed to 1 via 2 and to 5 via 4. 2 is exposed 4 and 4 is exposed to 2, via 3. In Figure 6-b, firms 2, 3, and 4 eliminate these exposures by clustering together. In the general model in Appendix A, agents form highly clustered networks, but not necessarily disjoint cliques. There it becomes more apparent that cliques in AOI are only a manifestation of a need for clustering.

Now the contrast is more clear. If firms want to have upsides of having counterparties, the downside by direct exposure to counterparties is inevitable. Ac-

cordingly, FCR captures the propensity of firms to form links and determines the density of the network. Eliminating FCR with interventions increases the propensity to form links.

As for SCR, by some collective care in the choice of counterparties, being mindful of the counterparties of their counterparties, firms can eliminate SCR while keeping FCR fixed. Thus SCR influences the topology of the network. Eliminating SCR with interventions reduces market discipline that would lead to the formation of the clusters.

3.3 Intervention induced concentration

Having made the distinction between the effects of FCR and SCR, now we study more elaborate effects of interventions that emerge under heterogeneity. We will focus on large shocks so that only SCR is eliminated. FCR is not altered and the propensity of firms to form links does not change. Moreover, we will make parametric assumptions ensuring that the net present value (NPV) of links is positive for firms, and so firms prefer to form as many links as possible even in AOI. These two will guarantee that firms can not be incentivized by interventions to increase their exposures. Therefore, the sole effect of interventions is to alter which firm want to form links with which firms, without any effect on how many links a firm wants to have. The main channel is the effect of interventions on how firms evaluate the identities and network positions of their counterparties, without any effect on the number of counterparties of firms. In other words, the only channel through which interventions affect the network is the topology or the shape of the network, not the density of the network. This is an intentional modeling choice to illuminate the network effects of interventions.

Assumption 2. *There are two types of firms, large and small. Denote the set of small and large firms S and L, respectively. There are $n-1$ many small firms and one large firm. Potential exposures are given by $w_{ij} = w_S = 1$ if $i, j \in S$ and $w_{ij} = w_L \geq w_S$ otherwise. The exposures of the large firm is not bounded: $\bar{e}_L = \infty$.*[20] *Small firms can afford at least one link with the large*

[20]The large firm can be seen as a firm with access to a high credit limit. This can be a

firm: $\overline{e}_S \geq w_L$. There is a non-trivial probability that the large firm can default due to contagion: $\phi_L \geq w_L$ and $\kappa_L > 1$. (For tractability) Small firms can default with bad shocks but not with counterparty defaults: $\phi_S = 0$ and $\kappa_S = 1$. Denote the size of the good shocks for small firms θ_{GS} and the size of the good shock for the large firm θ_{GL}. Bad shocks are large for all firms.

We maintain Assumption 2 throughout Section 3.3. In order to avoid integer problems, we further assume that $\overline{e}_S$ and $\overline{e}_S - w_L$ are even integers and $n \geq 2(\overline{e}_S + 1)$. Bad shocks are large under Assumption 2 so the optimal policy is to save good firms who face default due to their bad counterparties. Fixing these, we study independent shocks in Section 3.3.1 and correlated shocks in Section 3.3.2.

3.3.1 Emergence of concentration

Assumption 3. *All shocks are i.i.d., good with probability $\alpha \in \left(1 - \frac{1}{\kappa_L}, 1\right)$.*

For the large firm, one link in isolation has net value $w(1 - (1 - \alpha)\kappa_L)$. Since $1 > \alpha > 1 - \frac{1}{\kappa_L}$, links have positive NPV for the large firm. Notice that links have positive NPV also for the small firms because $\kappa_S = 1$.

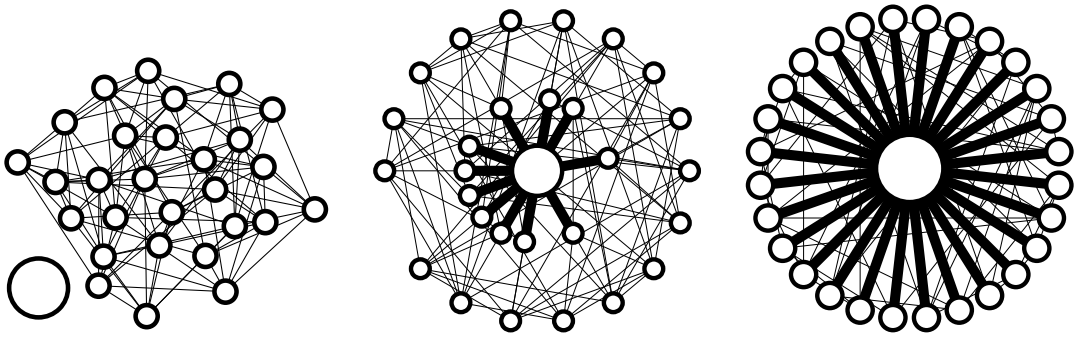
Proposition 4. *Suppose that Assumptions 2 and 3 hold. In the absence of interventions, the following network is stable. The large firm has no counterparties. Each small firms has $\overline{e}_S$ many small counterparties. In any stable network, the large firm has at most $\overline{e}_S$ many small counterparties. All small firms have $\overline{e}_S$ exposure ($\overline{e}_S$ many small counterparties or $\overline{e}_S - \omega_L$ many small and one large counterparty). In the presence of interventions, a network is stable if and only if the large firm is counterparties with all firms and all small firms have $\overline{e}_S - w_L$ many small counterparties.*

Network formed in AOI is illustrated in Figure 7-a and the one formed in POI is shown in Figure 7-c. In AOI, the large firm poses the risk of transmitting contagion, and so small firms do not form any links with the large firm. In

consequence of high collateral or good reputation.

POI, contagion through the large firm is no longer a concern. Pareto efficiency requires that all small firms form links with the large firm. The stable is network "fully concentrated" around the large firm.

Figure 7: Network reactions to the anticipation of interventions: increased concentration



(a) No concentration, Absence of interventions

(b) Bounded concentration, Absence of interventions

(c) Full concentration, Presence of interventions

Figure 7-a illustrates a stable network in AOI under i.i.d. shocks. Small firms either do not connect to the large firm or only a few of them do. In POI under i.i.d. shocks, the network becomes the one shown in Figure 7-c. Small firms are indifferent between forming links with the large firm or other firms and Pareto efficiency ensures that the large firm connects with all small firms.

Figure 7-b illustrates a stable network in AOI under correlated shocks. Small firms solve a tradeoff between low FCR and high SCR by connecting to the large firm. A limited number of small firms connect with the large firm. In POI under correlated shocks, the network becomes the one shown in 7-c. SCR is eliminated and all small firms strictly prefer to connect with the large firm.

At first, one might think this insight depends on α being the same for the large firm and the small firms. Suppose the large firm has α_L probability and small firms have α_S probability of getting good shocks. If α_L is smaller than α_S, small firms would not form links with the large firm neither in AOI nor in POI. The anticipation of interventions does not alter the concentration. If α_L is larger than α_S, then we have two cases. If links of the large firm are positive NPV, i.e. $(1-\alpha_S)\kappa_L < 1$, then all firms form links with the large firm both in AOI and in POI (provided that there are sufficiently many small firms so that LLN can wash away the variance of FCR in AOI). The anticipation of interventions does not alter the concentration. Only when links with the large

firm are negative NPV, i.e. $(1-\alpha_S)\kappa_L > 1$, and $\alpha_L > \alpha_S$, the anticipation of interventions changes the concentration.

So it may appear that this insight is valid only in small networks or when there is symmetry between the large firm and the small firms in terms of their shocks. But these arguments also hinge on the fact that shocks are independent. If shocks are slightly correlated, concentration is significantly increased in response to the anticipation of interventions without any of these restrictions. In this sense, the insight that concentration increases is not an artifact of large and small firms having the same probability of good shocks. This is what we show next.

3.3.2 Increased concentration

Assumption 4. *There is α_L probability that the large firm independently gets a good shock. Shocks of small firms are conditionally independent. There is a high state and a low state. The high state has probability $\sigma \in (0,1)$ and the low state has probability $\sigma' = 1-\sigma$. In the high state, shocks of small firms are i.i.d., good with probability $\alpha_S \in (0,1]$. In the low state, shocks of small firms are i.i.d., good with probability $\alpha'_S \in [0, \alpha_S)$. Also, $\sigma\alpha_S + \sigma'\alpha'_S > 1 - \frac{1}{\kappa_L} > \alpha'_S$ and $\alpha_L > \frac{\sigma\alpha_S^2 + \sigma'\alpha_S'^2}{\sigma\alpha_S + \sigma'\alpha'_S}$.*

Proposition 5. *Suppose that Assumptions 2 and 4 hold. Then there exists $\overline{d}_L > 0$ such that for any n and $\overline{e}_S$, the following hold. Let $\overline{d}_L^* = \min\{\overline{d}_L, n-1\}$. In the absence of interventions, any network in which the large firm has $\overline{d}_L^*$ many small counterparties and small firms have exposure $\overline{e}_S$ ($\overline{e}_S$ many small counterparties or $\overline{e}_S - \omega_L$ many small and one large counterparty) is stable. In any stable network, the large firm has at least $\overline{d}_L^*$ many and at most $\max\{\overline{d}_L^*, \overline{e}_S\}$ many small counterparties whereas small firms have exposure $\overline{e}_S$. In the presence of interventions, a network is stable if and only if the large firm is counterparties with all firms and small firms have exposure $\overline{e}_S$.*

Network formed in AOI is illustrated in Figure 7-b and the one formed in POI is shown in Figure 7-c. In AOI, small firms solve a tradeoff between the reduced FCR from the large firm and the increased SCR through the large firm

in the low state. Small firms then form a "bounded concentration" around the large firm to limit the SCR through the large firm. In POI, SCR is eliminated along with the tradeoff. The network becomes "fully concentrated" around the large firm due to the smaller FCR the large firm poses.

The condition $\sigma\alpha_S + \sigma'\alpha'_S > 1 - \frac{1}{\kappa_L}$ makes the large firm have an unbounded propensity to form links. It is not warranted that small firms want to form links with large firms though. If, hypothetically, there were no SCR through the large firm, by $\alpha_L(\sigma\alpha_S + \sigma'\alpha'_S) > \sigma\alpha_S^2 + \sigma'\alpha_S'^2$, small firms would have preferred to form a link with the large firm over forming w_L links with small firms. However, there is SCR through the large firm in the low state. $\alpha'_S < 1 - \frac{1}{\kappa_L}$ implies, via law of large numbers, that if the large firm has a large number of counterparties, in the low state, it defaults with high probability. In other words, the large firm creates SCR in the low state. Moreover, the SCR through the large firm conditional on the low state increases in the number of the counterparties of the large firm. Here $\overline{d}_L$ is the point at which the FCR benefits and SCR costs of forming a link with the large offset each other.

Note that $\overline{d}_L$ depends only on α_S, α'_S, α_L, and σ (defined in the proof of Proposition 5). If α_L is too large compared to α_S and α'_S (i.e. $\alpha_L > \frac{\sigma\alpha_S^2 + \sigma'\alpha_S'^2}{\sigma\alpha_S}$), the costs of increased SCR through the large firm is always less than the benefit of reduced FCR from the large firm. In this case, $\overline{d}_L = \infty$. All small firms form links with the large firm knowing that the large firm will most likely default in the low state. If, however, α_L is not so large ($\frac{\sigma\alpha_S^2 + \sigma'\alpha_S'^2}{\sigma\alpha_S + \sigma'\alpha'_S} < \alpha_L < \frac{\sigma\alpha_S^2 + \sigma'\alpha_S'^2}{\sigma\alpha_S}$), there is a finite cutoff at which SCR costs exceed FCR benefits. In this case we have $\overline{d}_L < \infty$. Small firms want to form links with the large firm, up to the point that the large firm does not have an exceedingly high probability of defaulting in the low state. Beyond $\overline{d}_L$ (the endogenous finite upper bound on the number of small counterparties that the large firm can have in equilibrium) additional small firms prefer to have links only with small firms rather than the large firm. For a small firm, the value of a link with the large firm is decreasing in the number of counterparties of the large firm, and $\overline{d}_L$ is the point at which the value of a link with the large falls below the value of forming links with w_L many small firms instead. Further discussions of this section can be found

in Appendix B.2.4.

4 Network hazard and moral hazard

In Section 4.1, we discuss welfare and moral hazard consequences of interventions. In Section 4.2, we study systemic risk consequences. In Section 4.3, we extend the model to allow for the large firm to choose its risk-return profile in order to further investigate how interventions interact with moral hazard on risk-return dimension.

4.1 Moral hazard on investment size

We call the adverse consequences of eliminating SCR the *second-order network hazard (SNH).* We call the adverse consequences of eliminating FCR that do *not* emerge when only SCR is eliminated the *first-order network hazard (FNH).*

Welfare. Recall that welfare criterion after shocks before contagion is given by W in Equation (1). Assuming that investments are positive NPV, in all cases we have considered thus far, $\mathbb{E}[W]$ is higher in POI than in AOI. Firstly because interventions fix the inefficient defaults and secondly, the anticipation of interventions (weakly) increase the total size of investments. Ex-ante welfare (after network formation, before shocks) would be $-\sum e_i + \mathbb{E}[W]$ by taking the costs of investments into account. By $r_i > 1$, if investments are positive NPV (for banks), then they are also positive NPV for welfare. Hence, ex-ante welfare is also higher in POI than in AOI.

Moral hazard. By the limited liability, there is already moral hazard in AOI. This can be because outside creditors are protected by deposit insurance which shifts the risk on to the government. It can also be because external creditors can not observe the network structure which means that the risk is shifted on to external creditors. The question is, how does this moral hazard interact with interventions?

First, consider the results in Section 3.2 regarding interconnectedness. Under FNH, investments go up. This means more risk-shifting mainly onto the government and tax-payers who fund the interventions, and potentially onto the

external depositors who fund the investments. Thus FNH exacerbates moral hazard in investment size. SNH, however, does not result in more risk-shifting because firms do not undertake more investments. In this sense, SNH does not directly interact with moral hazard. It is a collective form of lack of market discipline. Then what is the sense in which second-order network hazard is a "hazard?"

For this purpose, consider the results in Section 3.3. The large firm takes on more investments in POI than in AOI. At first, this can be seen similar to FNH since investments go up. Nevertheless the channel is different. We have shown that FNH **increases the propensity** of firms to make investments. This **directly** leads to more risk-shifting. Under SNH, the propensities of firms to make investments do not change. In particular, the large firm always wants to have as many counterparties as possible regardless of AOI or POI. SNH only makes the small firms less concerned with SCR through the large firm, and so more small firms become counterparties with the large firm. This **only enables** the large firm to have more counterparties, without any effect on its propensity to form links. In fact, the propensity of the small firms' do not increase either, and the total size of their investments is not affected. Small firms merely change the identity of their counterparties in response to the anticipation of interventions. Simply because small firms have the same exposure, but now with the large firm, the total size of investments in the economy doubles. SNH in the form of risk shifting in investment size emerges **indirectly** through a pure **network effect, in equilibrium**, not a direct propensity effect. Accordingly, SNH enables more moral hazard whereas FNH incentivizes more moral hazard regarding the size of investments. In this sense, FNH is a typical form of individual moral hazard in a multi-agent environment, but SNH results in a novel form of collective moral hazard specific to networks.

4.2 Volatility and systemic risk

SNH has a second manifestation: volatility and systemic risk. When the large firm is enabled to have more counterparties, the concentration of links around the large firm increases. When concentration increases, a large part of the

economy becomes exposed to the idiosyncratic risk of the large firm. The performance of the whole economy correlates highly with the performance of the large firm. If the large firm gets a good shock, concentration proves beneficial. Many projects yield high return. In the event that the large firm gets a bad shock, the entire set of small firms suffer losses. Inefficient defaults are indeed prevented by interventions, but the large output losses due to bad shock to the large firm is permanent. This part of welfare losses can not be fixed by interventions. Accordingly, welfare volatility and systemic risk emerges in POI. Note that these channels are different from the forms of collective moral hazard that arise under systemic interventions, such as in Farhi and Tirole (2012). Our interventions are targeted, firms do not correlate their risk in order to trigger interventions, and our firms are actually made indifferent between defaulting and continuing. A firm does not even benefit ex-post from its own bailout, but it benefits from the bailouts of its counterparties.

Next we formalize this insight about volatility. Consider one large firm and many small firms with correlated shocks as described in Section 3.3.2. Simulations verify the emergence of volatility: Figure 8 illustrates the probability distribution of welfare. We provide some asymptotic analytical results for the variance of welfare in Proposition 6.

Proposition 6. *Suppose that Assumptions 2 and 4 hold. (For simplicity) Suppose that* $\eta_S = \eta_L =: \eta$, $\theta_{LG} = \theta_{SG} =: \theta_G$.[21],[22] *As* $n \to \infty$, *the limit variance of average welfare is lower in the absence of interventions than in*

[21] To illuminate the effect of the network structure we assume away other heterogeneity pertaining to welfare and take counterparty loss sizes and shock sizes same across large and small agents: $\eta_S = \eta_L =: \eta$, $\theta_{LG} = \theta_{SG} =: \theta_G$.

[22] For analytical results on welfare along the lines of Section 3.3.2 for the networks described in Proposition 3.2, we need to specify one of the networks that can emerge. We consider cliques for the network amongst small firms who are not counterparties with the large firm as a selection amongst all the regular stable networks. Without a deterministic selection, we need to identify a probability distribution over regular networks. Unfortunately, the set of regular networks on a given set of vertices does not have a closed form expression. There are only algorithms that generate this set. Furthermore, even for a given arbitrary regular-network, in order to calculate welfare, one must identify the distribution of the size of the cutset between good and bad firms. This is an open problem in graph theory to the best of our knowledge.

Figure 8: Probability Distribution of Welfare

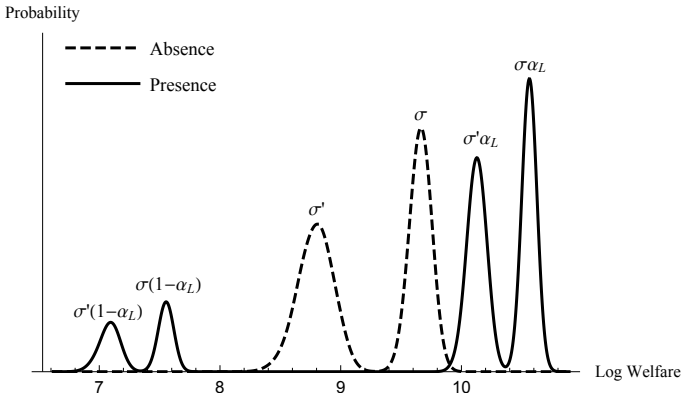


Figure 8 illustrates the probability distribution of welfare in AOI (dashed line) and POI (solid line). In AOI, there is bounded concentration and the large firm's idiosyncratic shock has bounded impact. Welfare is largely determined by the aggregate shock, manifesting the two modes of the distribution. In POI, there is full concentration around the large firm. The welfare, now, is determined largely by both the aggregate shock, but more so by the idiosyncratic shock to the large firm. A bad shock to the large firm results in significantly low welfare. Values used here are as follows: $\sigma = 0.5$, $\alpha_L = 0.8$, $\alpha_S = 0.8$, $\alpha'_S = 0.5$, $n = 200$, $w_L = \phi_L = 4$, $\overline{e}_S = 4$, $\kappa_L = 3$ $\eta_S = \eta_L = 0.9$. In this example, $\overline{d}_L = 1$. If α_L is increased towards 1, $\overline{d}_L$ can go up to 7.

the presence of interventions. The variance in the absence of interventions is solely due to the aggregate shock σ. In the presence of interventions, large firm's idiosyncratic shock contributes to volatility. In particular, the former (absence of interventions) is given by

$$\sigma\sigma'(\alpha_S - \alpha'_S)^2(1 - \eta + \eta(\alpha_S + \alpha'_S))^2(\overline{e}_S\theta_G)^2$$

The latter (presence of interventions) is larger than the former by at least

$$\alpha_L(1 - \alpha_L)\left(1 - \eta + 2\eta(\sigma\alpha_S + \sigma'\alpha'_S)\right)^2(w_L\theta_G)^2.$$

4.3 Moral hazard on the risk-return dimension

Next we study the interaction between interventions and moral hazard in individual risk taking under network effects. In order to study this question, we extend the model to analyze individual risk taking as follows. Suppose that the large firm can choose one of two possible risk-return profiles before network formation. It can either keep (α_L, θ_{GL}) or it can choose a high risk high return profile $(\alpha'_L, \theta'_{GL})$ where $\alpha_L > \alpha'_L$ and $\alpha_L\theta_{GL} < \alpha'_L\theta'_{GL}$. The large

firm has motives to take on more risk since the upside of the high risk profile is higher. This means more risk shifting onto external creditors.[23]

Proposition 7. *Suppose that Assumptions 2 and 4 hold. Further, suppose that* $1 - \frac{1}{\kappa_L} < \alpha'_L < \frac{\sigma\alpha_S^2 + \sigma'\alpha_S'^2}{\sigma\alpha_S + \sigma'\alpha_S}$. *Then moral hazard is mitigated by the presence of interventions: for any primitives under which the large firm chooses the safe profile in the absence of interventions, the large firm chooses the safe profile also in the presence of interventions.*

Since the risk profile is chosen before the network is formed, the large firm can choose the safe profile α_L to induce small firms to form links with itself. The collective behavior of small firms can discipline large firm's risk taking by using the collective threat of not forming links with the large firm. In AOI, stakes of the large firm are low. If it chooses the safe profile, it gets $\overline{d}_L$ links. If it chooses the risky profile, it can not obtain any links but it has high upside, for the relatively small private investment. In POI, stakes are high for the large firm. If it choose the safe profile, it gets $n-1$ links. If it chooses the risky profile, it can not obtain any links but has high upside, for the relatively small private investment. Therefore, if it were optimal to engage in moral hazard in POI, then it would also be optimal in AOI. But the converse is not true. Under second-order network hazard, this compensating force **mitigates** individual moral hazard in risk taking. Interventions, by mitigating SCR, increase the potential stakes of the large firm significantly, which allows small firms to the to discipline the large firm's risk taking. More discussions of this section can be found in Appendix B.2.5.

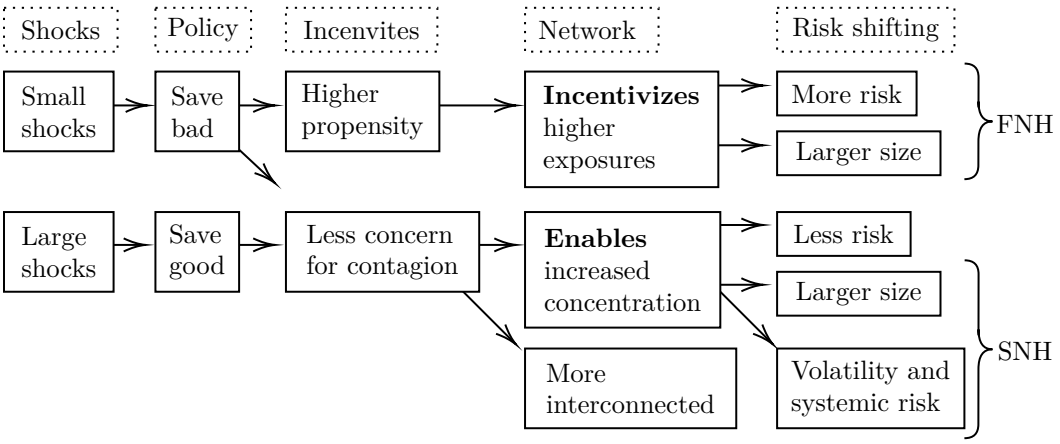
Finally, we summarize Section 4 in Figure 9.

5 Regulation and phase transitions

So far, we have discussed the effects of ex-post interventions. Now we undertake a positive analysis of the effects of ex-ante interventions. Such interventions can take the form of taxes for firms or capital and liquidity requirements

[23] Small firms can also be allowed to choose between two profiles but this choice does not interact with interventions and network hazard. We skip this extension to save space.

Figure 9: Summary of channels and insights



for financial firms. These are interventions that, in spirit, alter the value of exposures and put constraints on total exposures. We illustrate that smooth changes in the intensity of such ex-ante interventions can potentially lead to discontinuous changes in the network structure.

Consider two parameters that govern ex-ante interventions: ψ_E on exposures and ψ'_P on payoffs, described as follows. Firm i has a bound on its exposures $e_i \leq \psi_E \overline{e}$ during network formation. This is in the spirit of capital and liquidity requirements, as studied by Erol and Ordoñez (2017). Firm i's continuation payoff P_{Ci} is altered by ψ'_P as $-(e_i r_i + p_i + z_i) + (1 - \psi'_P)\theta_G(p_i + e_i - \eta_i c_i)$. The parameter ψ'_P can be seen as a tax on the revenue of the firm. Our task here is to understand how the network reacts to changes in ψ_E and ψ'_P.[24]

Assumption 5. *(For simplicity) All firms are identical and $z_i = p_i = 0$ for all firms. Shocks are correlated as follows. There are two states. High state has probability σ, wherein shocks are i.i.d., good with probability α. Low state has probability $\sigma' = 1 - \sigma$, wherein shocks are i.i.d., good with probability $\alpha' < \alpha$.*

We work with Assumption 5 in this section. Since all firms are identical, we can drop the subscripts. Denote $\psi_P = \frac{1}{\eta} - \frac{r}{(1-\psi'_P)\theta_G \eta}$. Then the continuation

[24]Such ex-ante interventions are relevant in other applications as well. For example Erol and García-Jimeno (2019) study how the social structure changes under a coercive government when the government increases the intensity of its surveillance methods.

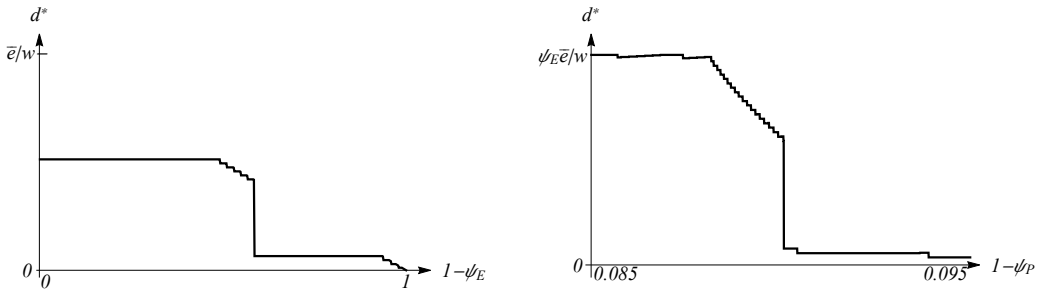
payoff of i is

$$\begin{aligned}
&-(e_i r + z) + (1-\psi')\theta_G(p + e_i - \eta c_i) \\
&\propto \underbrace{\left(\frac{1}{\eta} - \frac{r}{(1-\psi')\theta_G \eta}\right)}_{=:\psi_P} e_i - c_i - \underbrace{\left(\frac{z}{(1-\psi')\theta_G \eta} - \frac{p}{\eta}\right)}_{0} \\
&= \psi_P e_i - c_i
\end{aligned}$$

Notice that if $\psi_P > 1-\alpha'$, links are positive NPV in both states. If $\psi_P \in (1-\alpha, 1-\alpha')$, links are positive NPV in the high state and negative NPV in the low state. If $\psi_P < 1-\alpha$, links are negative NPV in both states.

The unconstraint value function is $V(d;\psi_P) = \mathbb{E}_b[[\psi_P d - b]^+]$ where $b \sim \sigma \circ \mathbb{B}[d, 1-\alpha] \oplus \sigma' \circ \mathbb{B}[d, 1-\alpha']$. The optimal degree of a firm is $d^*(\psi_E, \psi_P) := \arg\max_{dw \leq \psi_E \overline{e}} V(d;\psi_P)$. Our task is study how the exposures of firms, $d^*(\psi_E, \psi_P)w$, change in response to regulation parameters ψ_E and ψ_P? Figure 10 illustrates the discontinuous changes.

Figure 10: Network reactions to regulations: phase transitions



(a) Phase transition in exposure regulation (b) Phase transition in payoff regulation

Figure 10-a shows the discontinuous fall in exposures as the exposure regulation becomes more stringent. As ψ_E falls the exposure bound starts binding and exposures start falling smoothly. Then at a sharp cutoff, exposures fall discontinuously. Figure 10-b shows the discontinuous fall in exposures as the payoff regulation becomes more stringent. Notice that Figure 10-b ranges over a tight interval 0.085 to 0.095. Above 0.095, the exposure bound binds. Around a cutoff, exposures fall smoothly. At the cutoff, the fall is discontinuous. The values used here are $\sigma = 0.3$, $\alpha = 0.9$, $\alpha' = 0.85$, $\overline{e} = 600$, $w = 1$. $\psi_P = 0.09$ is fixed for Figure 10-a and $\psi_E = 5/6$ is fixed for Figure 10-b.

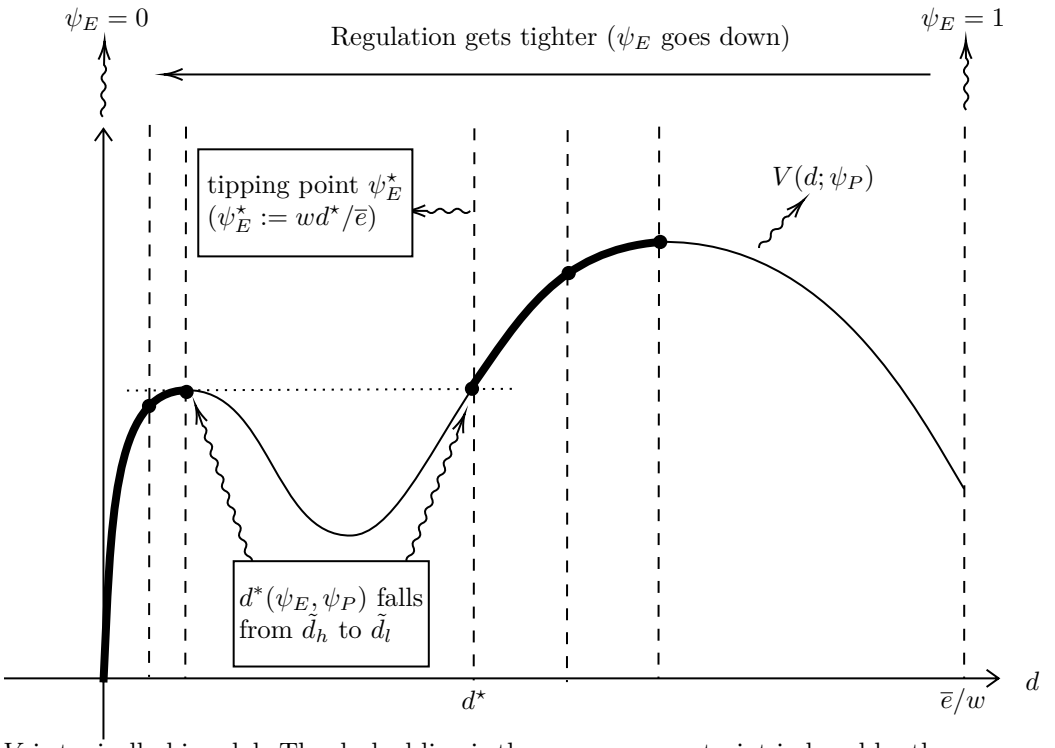
Phase transitions are prevalent in networks. In endogenous economic networks, one might think of such sharp reactions as "bang-bang" results. This is wrong

in our case. In order to understand why, take a closer look at V:

$$V(d;\psi_P) = \sigma\alpha \underbrace{\mathbb{E}_{b_h}[[\psi_P d - b_h]^+]}_{V_h(d;\psi_P)\ \textit{in high state}} + \sigma'\alpha' \underbrace{\mathbb{E}_{b_l}[[\psi_P d - b_l]^+]}_{V_l(d;\psi_D)\ \textit{in low state}}$$

where $b_h \sim \mathbb{B}[d, 1-\alpha]$ and $b_l \sim \mathbb{B}[d, 1-\alpha']$. Call V_h the high state component and V_l the low state component of V. V is typically bimodal with two local maxima. Call the smaller mode $\tilde{d}_l(\psi_E, \psi_P)$ and the higher mode $\tilde{d}_h(\psi_E, \psi_P)$. $\tilde{d}_l$ is close to the maximizer of V_l and $\tilde{d}_h$ is close to the maximizer of V_h. Figures 11 and 12 illustrate the shape of V and the forces behind phase transitions.

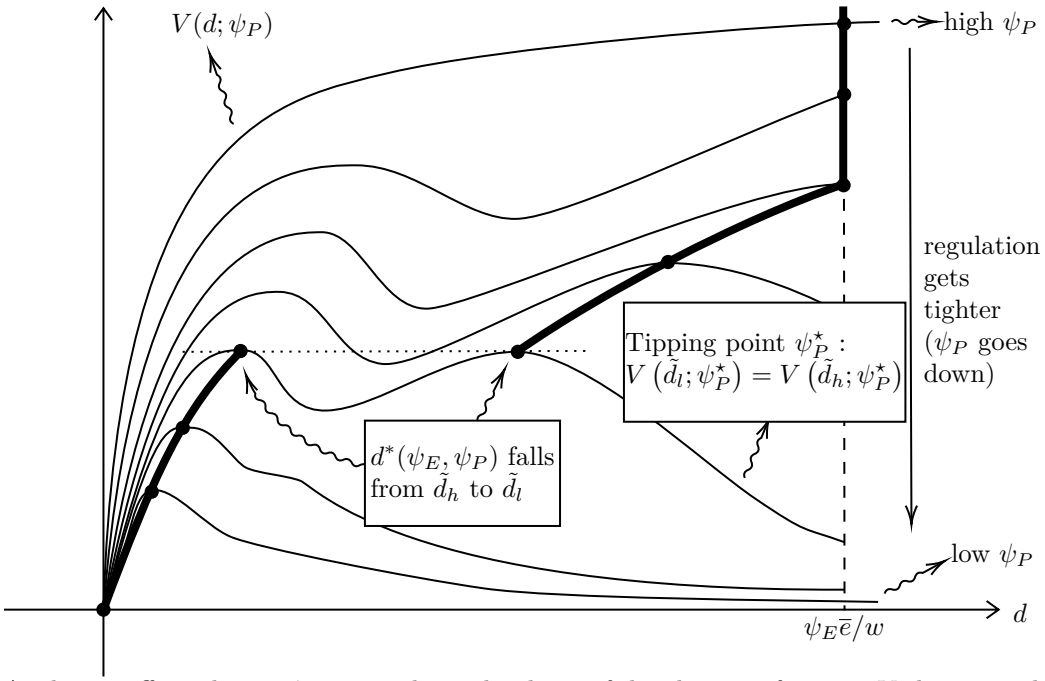
Figure 11: Phase transition in exposure regulation ψ_E



V is typically bimodal. The dashed line is the exposure constraint induced by the exposure regulation. As the regulation gets tighter, the dashed line shifts left. Until the constraint hits $\tilde{d}_h$, the larger local maximizer V, the constrained optimal degree is $\tilde{d}_h$. Then the exposure constraint binds and the exposures start falling smoothly, given by the constraint itself. At a tipping point, the constrained optimal degree falls discontinuously to $\tilde{d}_l$, the smaller local maximizer of V, as the value at the constraint becomes lower than the value at the lower local maximizer.

For the transition in exposure regulation ψ_E, consider the illustration in Figure 11. Fix ψ_P and vary ψ_E. d^* is given by $\min\{\tilde{d}_h, \psi_E\overline{e}/w\} = \psi_E\overline{e}/w$ for large ψ_E.

Figure 12: Phase transition in exposure regulation ψ_P



As the payoff regulation ψ_P gets tighter, the shape of the objective function V changes. The local maximum of V associated with the high state is more sensitive to ψ_P then the local maximum associated with the low state. At a cutoff, the global maximum switches from the right-hand side local maximum to left-hand side local maximum. At this point, optimal exposures fall discontinuously.

As ψ_E falls, d^* falls smoothly. As regulation gets tighter and ψ_E gets smaller, at the tipping point $\psi_E^\star$, d^* falls discontinuously from $\min\{\tilde{d}_h, \psi_E\overline{e}/w\} = \psi_E\overline{e}/w$ to $\min\{\tilde{d}_l, \psi_E\overline{e}/w\} = \tilde{d}_l$ due to the constraint. This is clearly not a bang-bang result. It is a consequence of bimodality, which is a consequence of the aggregate uncertainty.[25]

The transition in payoff regulation ψ_P is more subtle. Consider the illustration in Figure 12. Fix ψ_E and vary ψ_P. The channel is similar to the transition in ψ_E but has an extra element. As regulation gets tighter and ψ_P gets smaller, the local max $V(\tilde{d}_h;\psi_P)$ falls faster than the local max $V(\tilde{d}_l;\psi_P)$. This is because is $\tilde{d}_h > \tilde{d}_l$ and $\alpha > \alpha'$, which makes V_h more sensitive to ψ_P than V_l

[25]Note that Erol and Ordoñez (2017) also study such exposure regulations but their phase transition is not a consequence of aggregate uncertainty.

is. As ψ_P falls, at the tipping point $\psi_P^\star$, $V(\tilde{d}_h;\psi_P)$ falls below $V(\tilde{d}_l;\psi_P)$ and d^* falls from $\tilde{d}_h$ to $\tilde{d}_l$. This also is due to the bimodality, which is a consequence of aggregate uncertainty.

6 Conclusion

We have presented a simple model of firms that expose themselves to the default risk of their counterparties by making joint investments. We have shown that firms protect themselves against contagion by forming network that have high clustering and low concentration. In the presence of interventions, contagion is mitigated by interventions which reduces the concern that firms have for the counterparties of their counterparties. Then the resulting network has low clustering (more interconnected) and high concentration. In turn, idiosyncratic risks of large firms at the center of the network create systemic risk and aggregate volatility. Surprisingly, moral hazard on risk-return dimension is mitigated because peripheral firms can discipline the central firms' risk taking by the threat of exclusion. Ex-ante regulations to curb network hazard might entail discontinuous network reactions. The key channels and insights are applicable to a broader set of applications that feature network games with complementarities.

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A Stability, contagion, and interventions

Section A.1 describes a general framework for contagion and resulting payoff for a given network. Section A.2 establishes a mapping between contagion and network games. Section A.3 discusses applications and interpretations. Section A.4 describes stable networks for various notions of stability. Section A.5 studies interventions and their impact on network formation.

A.1 Model of contagion

Network: There is a finite set of *agents* $N = \{1, 2, ..., n\}$ with exposures to each other. The *exposure* of i to j is $e_{ji} \in \mathbb{R}_{\geq 0}$. The *network* is $\mathbf{e} = [e_{ij}]_{i,j \leq n}$. We take $e_{ii} = 0$. Denote $e_i^{out} = \sum_j e_{ij}$ (the sum of exposures to i), $e_i^{in} = \sum_j e_{ji}$ (the sum of exposures of i), and $e_i = (e_i^{out}, e_i^{in})$.

Shocks: Agents first receive shocks. These shocks can be good or bad. Denote $\theta_i \in \Theta_i = \{\theta_{Gi}, \theta_{Bi}\}$ the *shock to* i. θ_{Gi} is the *good shock* and θ_{Bi} is the *bad shock*. We also call agent i *good/bad* when θ_i is good/bad. There is an aggregate shock $\omega \in \Omega$. Shocks of agents are independent conditional on ω. i gets a good shock with probability $\alpha_i(\omega) \in [0, 1]$. No agent is guaranteed either a good shock or a bad shock: $\mathbb{E}_\omega[\alpha_i(\omega)] \in (0, 1)$ for all i.

Contagion: After shocks, contagion starts and progresses as follows. Agents with bad shocks default. If defaults on a good agent exceeds a threshold, he also defaults. Defaults are irreversible and contagion progresses round by round. Formally, index rounds of contagion with $t \geq 0$. Denote $a_{it} = 1$ if agent i defaults at or before round t and $a_{it} = 0$ otherwise. At round $t = 0$, bad agents default. A good agent i defaults at round $t + 1$ if he has not defaulted before and $c_{it} := \sum_j e_{ji} a_{jt} > R_i(e_i)$. Here R_i is the the *resilience* function of i. When no new agent defaults for the first time, say at $t = t^*$, contagion stops.[26]

Outcome: Denote $a_i = a_{it^*}$. We say i *continues* if $a_i = 0$ and i *defaults* if $a_i = 1$. Denote $c_i = c_{it^*}$, the total size of counterparty defaults on i. All in

[26] This generalizes the standard threshold contagion model to arbitrary weights, arbitrary threshold functions, and arbitrary shock distributions. See Granovetter (1978) and Centola and Macy (2007).

all, at the end of contagion, i continues if and only if i is a good agent and $c_i = \sum_j e_{ji} a_j \leq R_i(e_i)$.

Payoffs: If an agent i defaults, he receives a payoff $P_{Di}(e_i)$ that does not depend on counterparty defaults. If he continues, he receives a payoff $P_{Ci}(e_i, c_i)$ that is weakly decreasing in the size of counterparty defaults c_i. We assume $P_{Di}(e_i) \leq P_{Ci}(e_i, R_i(e_i))$ and $P_{Di}(e_i) < P_{Ci}(e_i, 0)$ so that defaulting is not desirable. In shorthand, i's payoff is

$$P_i(e_i, c_i) = P_{Di}(e_i) + [P_{Ci}(e_i, c_i) - P_{Di}(e_i)]\mathbb{1}_{c_i < R_i(e_i)} \tag{2}$$

Remark: For most of the following results, the payoff and resilience of i can also depend on the aggregate shock ω and the individual shocks to counterparties of i.

A.2 Game theoretical foundations of contagion

Notice that P_i and R_i are two separate primitives of the model. Contagion is governed by R_i, not necessarily the resulting payoffs P_i. In our simplified model, R_i was given by the threshold at which P_{Ci} falls below P_{Di}:

$$R_i(e_i) = \max\{c : P_{Ci}(e_i, c) \geq P_{Di}(e_i)\} \tag{3}$$

When (3) holds, an alternative way to formulate contagion is via a network game of complementarities.[27] Suppose that, in the contagion stage, agents observe all shocks[28] and play a simultaneous-move game and *choose* to continue or default. A bad shock makes it strictly dominant to default. Agents with good shocks best respond to their counterparties. If continuation yields higher payoff, they continue. Then following the Tarski argument as outlined earlier, the best equilibrium of this game corresponds to the outcome of contagion. In this sense, our model encompasses a large class of binary network games.

[27] See Morris (2000), Galeotti et al. (2010).

[28] All results go through as long as agents observe the shocks to themselves and their counterparties. Agent end up forming networks that makes the information about shocks to non-counterparties redundant. See Section B.3.3 for details.

A.3 Interpretations

Threshold contagion models are useful in a wide range of applications.[29] Some examples include the spread of behaviors and trust in communities in sociology and political economy; joint projects and team production in industrial organization; product adoption in marketing; swaps, options, and diversification in finance.[30] Complementarities are the main force underlying these applications. For example, if majority of an agent's friends start using a specific messaging application, past a threshold, the agent can adopt this application as well. Similarly for fashion trends or even political views, peer pressures can lead to adopting a behavior past a certain threshold for adoption among counterparties. Moreover, since our model does not require that Equation (3) holds, it can also be useful to study behavioral forms of contagion.

Our insights are applicable to such scenarios but we pay closer attention to economic networks and financial contagion given the significance of ongoing discussions about systemic risk and bailouts. There are numerous examples of financial contagion models that utilize threshold contagion.[31] The particular manifestation of complementarity in financial networks is that an agent's profitability relies on whether his counterparties uphold their obligations or not. These obligations can take many forms such as simple debt contracts or derivative contracts for insurance purposes. If too many counterparties default on the agent, his yields go down, potentially to a point that can render the agent insolvent, resulting in his inability to uphold his obligations to other agents. Defaults then cascade.

In Section B.1.2, we discuss why our model is appropriate to study various instances of financial contagion. In doing so, we show how variants of three existing models, Eisenberg and Noe (2001), Elliott et al. (2014), and Erol and Ordoñez (2017), can be nested into our model. The main difference of our

[29] See Easley and Kleinberg (2010) and Jackson (2010).

[30] As Centola and Macy (2007) points out, threshold contagion models are in contrast with independent cascades models. The latter is more appropriate for epidemics, rumors, social learning, information transmission etc.

[31] Such as Aymanns et al. (2017), Amini et al. (2016), Cifuentes et al. (2005), Erol and Vohra (2018), and more.

model with existing counterparty-risk models is that contagion is binary. In all existing models, when a counterparty j of an agent i defaults, this hurts i. In our model, the number of counterparties of j that defaulted do not affect i above and beyond making j default. This provides tractability for network formation.

A.4 Stable networks

A.4.1 Stability

Before shocks are realized, agents evaluate a network according to the expectation of their payoffs with respect to shocks, anticipating interventions and contagion that follow.

There is a given non-negative weight matrix $\mathbf{w} = [w]_{i,j \leq n}$. If i and j decide to form a *link*, their exposures to each other are given by $e_{ij} = w_{ij}$ and $e_{ji} = w_{ji}$. If they do not form a link, their exposures are $e_{ij} = e_{ji} = 0$. We denote $\{i, j\} \in E$ whenever i and j form a link. The endogenous part of the network, i.e. binary undirected links, is represented by E. If two firms i and j end up forming a link, we call them *counterparties*. We denote N_i the set of counterparties of i.

We assume that firm i's exposures can feasibly be at most $\overline{e}_i^{in}$ and the exposures to i can feasible be at most $\overline{e}_i^{out}$. We call N_i is feasible for i if $e_i^{out} \leq \overline{e}_i^{out}$ and $e_i^{in} \leq \overline{e}_i^{in}$. A network is called *feasible* if each firm has a feasible set of counterparties. Consider a feasible network E and a subset N' of firms. A *deviation* by N' from E is one in which N' can simultaneously add any missing links within N', cut any existing links within N', and cut any of the links between N' and N/N'. A *feasible deviation* by N' is a deviation after which the resulting network is feasible. Going forward, we will consider various of notions of stability that preclude various feasible deviations. We define in each notion before the corresponding result.

A.4.2 Preliminaries

Assumption 6. *The exposures of agents depend on their own characteristic, not their counterparties: for all i, there exists w_i such that $w_{ji} = w_i$ for all*

j. The payoffs and resiliences of agents do not depend on exposures to them: P_{Di}, P_{Ci}, and R_i are constant in e_i^{out}.

This means that the preferences of i regarding the identities of i's counterparties are driven only by their idiosyncratic risks of bad shocks and their default risks due to their position in the network, not the intrinsic value of their links to i. By abstracting away from differences in values of links, we can better understand the impact shocks and contagion. In Section B.3.1 we relax this and discuss the general case in which i's payoff and resilience depend on the exposures of i and the exposures to i.

Remark: Note that the results in Section 3.3 with the large firm do not satisfy Assumption 6 unless $w_L = w_S$. So the results in Section 3.3 are not corollaries of the general model herein. This model is more in general in the sense that it allows for a large class of payoff and resilience functions than the linear forms in the main body of the paper. Moreover, R and P do not need to be linked to each other as in Equation (3).

We denote $d_i = |N_i|$ the number of counterparties of i (namely the *degree* of i), f_i the number defaulting counterparties of i, and b_i the number of bad counterparties of i. Under Assumption 6, $e_i^{in} = d_i w_i$ and $c_i = f_i w_i$. Moreover, the payoff and resilience does not depend on e_i^{out}. Then, with some abuse of notation, we can use $P_i(d_i, f_i)$, $P_{Ci}(d_i, f_i)$, $P_{Di}(d_i)$, $R_i(d_i)$ instead of $P_i(e_i, c_i)$, $P_{Ci}(e_i, c_i)$, $P_{Di}(e_i)$, $R_i(e_i)$, respectively. The ex-post payoff of i is

$$P_i(d_i, f_i) = P_{Di}(d_i) + [P_{Ci}(d_i, f_i) - P_{Di}(d_i)]\mathbb{1}_{f_i < R_i(d_i)}$$

Let $b_{i\omega} \sim \mathbb{B}\left[d_i, 1 - \alpha_i(\omega)\right]$, where $\mathbb{B}$ is the Binomial CDF. $b_{i\omega}$ represents the number of bad shocks to counterparties of i in state ω. Define the *assortative value function* V_i as

$$V_i(d_i) = \mathbb{E}_\omega\Bigg[\Bigg(\underbrace{(1 - \alpha_i(\omega))}_{\text{bad shock default}} + \underbrace{\alpha_i(\omega)\,\mathbb{P}_{b_{i\omega}}\left[b_{i\omega} > R_i(d_i)\right]}_{\text{contagion default}}\Bigg) P_{Di}(d_i) \qquad (4)$$

$$+ \underbrace{\alpha_i(\omega)\,\mathbb{P}_{b_i}\left[b_{i\omega} \leq R_i(d_i)\right]\mathbb{E}_{b_{i\omega}}\left[P_{Ci}(d_i, b_{i\omega}) \middle| b_i \leq R_i(d_i)\right]}_{\text{continuation with counterparty losses}}\Bigg]$$

Note that $V_i(d_i)$ is not the expected payoff of i. Firstly because network effects and contagion effect i's expected payoff. Secondly, V_i assumes that shocks to

counterparties of i follow the same distribution with the shock to i. In fact, V_i is the payoff of i if it were at the center of a disjoint star subnetwork with agents that have the same shock distribution with i. Define the *assortative degree* of i as $d_i^* := argmax_{d_i w_i \leq \bar{e}_i} V_i(d_i)$.[32]

Define $T_i(d_i) := max\{f_i \leq R_i(d_i) : P_{Ci}(d_i, f_i) > P_{Di}(d_i)\}$. This is the largest counterparty default level that is costly for agent i. Notice that $T_i \equiv \lfloor R_i \rfloor$ is possible but not required. Accordingly, we call T_i the *payoff threshold* for i and R_i the *action threshold*. As the number of counterparty defaults increase, after the payoff threshold T_i, i does not incur any further losses. Distinguishing the payoff threshold and action threshold is going to be helpful in interpreting the effects of interventions described in Section A.5. Interventions will widen the gap between the two thresholds.[33] Denote $\rho_i^* = \lfloor R_i(d_i^*) \rfloor$ and $\tau_i^* = T_i(d_i^*)$.

A.4.3 Globally Stable networks

We first provide some results for the strongest solution concept we consider. A network is *globally stable (GS)* if every agent achieves the highest payoff that he can achieve in any feasible network, i.e. his first-best. Recall that the notion of stability we have used in the simplified model is Pareto Efficient Strong Stability (*PESS*), which is weaker than GS.

Assumption 7. *There exists $\alpha : \Omega \to [0,1]$ such that $\alpha_i(\omega) \equiv \alpha(\omega)$ for all i.*

Naturally, GS networks do not always exist. We relax Assumption (7) after the we establish results for GS networks.

Theorem 1. *Suppose that Assumptions 6 and 7 hold. A network E is GS if and only if*

- *Every agent has its assortative degree: $d_i = d_i^*$ for all i,*
- *There is high clustering: $(|N_i \cap N_j| - \tau_j^*)^+ \geq d_i^* - 1 - \rho_i^*$ for every $\{i,j\} \in E$.*

[32] We assume that V_i admits no indifferences over integers in order to rule out some cumbersome indifferences. Note this is true for typical α, R, and P.

[33] In certain applications, the gap between R_i and T_i can also capture some behavioral inertia before changing the social behavior.

An agent i with degree d_i can not have more than $V_i(d_i)$ expected payoff. Because it is subsumed under V_i that only bad counterparties default. Then the globally maximal payoff of i is $V_i(d_i^*)$ which can not achieved without the assortative degree d_i^*. Conditional on all agents having their assortative degrees, agents must also eliminate SCR. It is not possible to make every agent the center of a star subnetwork. The clustering condition characterizes when when SCR is eliminated for all agents, given that all agents have their assortative degrees. Roughly, the idea is that if $j \in N_i$ has sufficiently many counterparties that are not counterparties of i, then there is an event in which i has less than τ_i bad counterparties but j has more than ρ_j many bad counterparties. At this event, j's action threshold is exceeded and he defaults despite being a good agent. This entails a cost for i since the i's payoff threshold is not yet exceeded. The sufficiency is more elaborate and uses cohesiveness a la Morris (2000).

There are various network structures that satisfy these conditions. Consider identical agents for illustration (and drop the subscripts). Each must have degree d^* and so the network must be d^*-regular. It can not be any d^*-regular network though. Every two agent i and j who are counterparties must have at least $d^* - 1 - (\rho^* - \tau^*)$ many common counterparties. For example, one can construct components with $d^* + 1 + \rho^* - \tau^*$ agents that satisfy the conditions, which are not cliques if $\rho^* - \tau^* > 0$. Also, if $\rho^* - \tau^* \geq d^*/2$, then a $(d^*/2)$-ring network is GS. Note that this is a connected network. Our results differs from the literature that find cliques. The main force here is clustering.

A.4.4 Application to network games

In network games, the best Nash equilibrium requires that the payoff threshold T and the behavior threshold R are identical. Then Theorem 1 has a sharper prediction about the topology of the network. High clustering takes an extreme form of full clustering.

Now $\tau_i^* \equiv \rho_i^*$. Call an agent i *contagious* if $\rho_i^* < d_i^* - 1$ and *non-contagious* if $\rho_i^* \geq d_i^* - 1$. Call two contagious agents i and j *contagion-similar* if $d_i^* = d_j^*$ and $\rho_i^* = \rho_j^*$. Notice that contagion-similarity is an equivalence class.

Theorem 2. *Suppose that Assumptions 6 and 7 hold. For network games ($T \equiv R$), a network is GS if and only if*

- *Every agent has its assortative degree: $d_i = d_i^*$ for all i,*
- *Non-contagious agents are counterparties with only non-contagious agents,*
- *Contagious agents form disjoint cliques each of which consists of contagion-similar agents.*

It is important to highlight the idea that cliques are important only up to providing clustering. In network games wherein the resilience is induced from payoffs, the need for clustering takes the extreme form of inducing disjoint cliques.

A.4.5 Pareto Strongly Stable networks

When the probability of shocks differ across agents, GS networks are not possible to obtain. All agents want to form links with the least risky agents. This creates a tension across agents. In this case we can restore results by resorting to weaker solution concept. A network is *Pareto Strongly Stable (PSS)* if no subset of agents have a feasible deviation that Pareto improves the deviating set. Under PSS, one would expect that least risky agents with form links with each other and so agents will get sorted with respect to how risky they are as counterparties for other agents. In order to establish such a result, we assume that agents can indeed be ranked with respect to their idiosyncratic risk.

Assumption 8. *For every ω, ω', i, j, we have $\alpha_i(\omega) \geq \alpha_j(\omega)$ if and only if $\alpha_i(\omega') \geq \alpha_j(\omega')$.*

For example, there can be some underlying aggregate risk level $\alpha(\omega)$ such that agent i's exposure is altered by its own risk characteristic $s_i \in (0,1)$ as $\alpha_i(\omega) = s_i\alpha(\omega)$. The ranking of s_i's then determine the risk ranking of agents.

Call two agents i and j *risk-similar* if $\alpha_i \equiv \alpha_j$. Call i and j *similar* if they are contagion-similar and and risk-similar. Notice that these are all equivalence relations. Index the equivalence classes induced by similarity with ι. Let n_ι be the number of agents in the equivalence class ι.

Theorem 3. *Suppose that Assumptions 6 and 8 hold.*

A network E is PSS if

- *Every agents has its assortative degree: $d_i = d_i^*$ for all i,*
- *There is high clustering: $|N_i \cap N_j| \geq d_i^* - 1 - \rho_i^* + \tau_j^*$ for every $\{i, j\} \in E$.*
- *Each component*[34] *of the network consists of risk-similar agents.*

In network games ($T \equiv R$), a network is PSS if

- *Every agent has its assortative degree: $d_i = d_i^*$ for all i,*
- *Non-contagious agents are counterparties with only non-contagious and risk-similar agents,*
- *Contagious agents form disjoint cliques with similar agents.*

If $n_\iota \geq d_\iota^* + 1$ for all ι, these are also necessary conditions for PSS.

Agents first get sorted with respect to their risk-similarity. Agents in the risk-similarity class with the least risk form links with only each other and refuse to form links with riskier agents. Given this, agents in the risk-similarity class with the second least risk form links only with each other, refusing the form links with riskier classes. This unravels and agents end up forming links only with their own risk class. In network games, there is a second layer of sorting inside each risk class due to the need for clustering. Agents also get sorted with respect to resilience. Agents with high resilience prefer to match with agents that have high resilience. This also unravels and agents end up forming links only with their own class. Also, as before, cliques emerge for the case of network games. Once again, this stresses the idea that the main force is clustering, not the disconnected nature of the cliques that emerge under network games.

A.4.6 Approximate Strong Stability and general existence

One might worry about the condition $n_\iota \geq d_\iota^* + 1$ in Theorem 3. When $n_\iota \leq d_\iota^*$, agents can not eliminate SCR. Then they need to solve tradeoffs between first-, second-, and even third-order counterparty risks. In this case, even Strongly Stable (SS) networks need not exist, which is shown by an

[34] A component is a disjoint but connected subnetwork.

example in Section B.3.2. Nevertheless, perhaps under some restrictions on payoff and resilience functions, some results could be obtained. But, as long as these assumed functional forms do not take trivial forms that readily eliminate contagion, the tradeoffs between various orders of counterparty risks must be solved. Hence, presumably, there will be SCR in the network formed. Then, in order to solve for the tradeoffs between FCR and SCR, one first needs be able to establish existence of networks that has a specified second-order degree sequence (i.e. the ordered multiset of the numbers of vertices at distance 1 and 2 from each vertex). Note that the second-order degree sequence problem is a simpler problem than finding stable networks, and potentially much simpler since it does not entail resolving the tradeoffs between FCR and SCR. The being said, the second-order degree sequence problem is an open problem in graph theory. Erdős and Miklós (2016) show that it is strongly NP-complete.

Our broader solution to this challenge is to consider an approximate version of strong stability. This way small tradeoffs in high orders of counterparty risks can be ignored. Then, even if no two agents are similar to each other, we can restore existence. In this sense, our insights do not depend on any form of symmetry neither in terms of shocks nor in terms of payoffs.

Given any $\epsilon > 0$, a network is $\epsilon-$*Strongly Stable (ϵ-SS)* if there exists a subset of agents N_ϵ such that $|N_\epsilon| > n(1-\epsilon)$ and no subset of N_ϵ has a feasible deviation in which all members can improve their payoff by more than ϵ. Note that GS $\subset$ PSS $\subset$ PESS $\subset$ SS $\subset$ ϵ-SS.

Assumption 9. *There is a uniform bound $\overline{e} < \infty$ on degrees such that $\overline{e}_i \leq \overline{e}$ for all i. There is some α such that for all i, $\alpha_i(\omega) = s_i \alpha(\omega)$ where $s_i \in (0,1)$ is the risk type of i.*[35] *s_i is independently drawn from a uniform distribution $s_i \sim U[\underline{s}, \overline{s}]$ where $0 < \underline{s} < \overline{s} < 1$. There is some $\tilde{P}$ and $\tilde{R}$ such that for all i, $P_i(\cdot) \equiv \tilde{P}(\cdot; q_i)$ and $R_i(\cdot) \equiv \tilde{R}(\cdot; q_i)$.*[36] *Here q_i is called the contagion-type of i and it is independently drawn from some arbitrary distribution with an*

[35] The essential feature of the shock specification is that Assumption 8 is satisfied: agents can be ranked with respect to their idiosyncratic risks.

[36] For example, one can consider a linear form under network games (along the lines of the lead example): $\tilde{P}_i(d_i, f_i; q_i) = (q_i d_i - f_i)^+$ and $\tilde{R}_i(d_i; q_i) = q_i d_i$.

arbitrary support. For each i, the risk-type s_i and contagion type q_i are drawn prior to network formation and they are publicly observed.

Under Assumption 9, with probability 1, no pairs of agents are risk-similar ($n_\iota = 1$).

Construction: *For all $(d,\rho) \in \mathcal{C} := \left\{(d,\rho) \in \mathbb{Z}_+^2 : \rho \leq d \leq \overline{e}\right\}$, denote $N_{(d,\rho)} = \{i \in N : d_i^* = d, \lfloor R_i(d_i^*) \rfloor = \rho\}$. Call each $N_{(d,\rho)}$ a contagion-class (these are the equivalence classes with respect to contagion-similarity). Note that $\mathcal{C}$ is a finite set since $\overline{e} < \infty$. So there are finitely many contagion-classes. For each contagion-class $N(d,\rho)$, sort all agents in the contagion-class with respect to their risk types. Construct bins of $d+1$ agents in descending order with respect to risk type. Make agents in each bin form a disjoint clique. Each agents in a contagion class $N_{(d,\rho)}$ are sorted into cliques of order $d+1$, there can be at most d agents left as residuals. Residuals can form arbitrary links among themselves.*

This construction results in cliques of various sizes, each of which consists of agents from the same contagion-class and all have close risk type to each other. There is also some residual from each contagion-class. For large enough n, the residual agents make up a vanishing fraction of agents. The construction mimics the earlier sorting and matching arguments.

Theorem 4. *Suppose that Assumptions 6 and 9 hold. For all $\epsilon > 0$, there exists $\underline{n}$ such that for all $n > \underline{n}$, the Construction yields an ϵ-SS network with probability 1.*

We illustrate with an example. Suppose that $\tilde{P}_i(d_i, f_i; q_i) = (q_i d_i - f_i)^+$ and $\tilde{R}_i(d_i; q_i) = q_i d_i$ where q_i is independently drawn from $U[\underline{q}, \overline{q}]$ for some $0 < \underline{q} < \overline{q} < 1$. Under this specification, a potential link $\{i,j\}$ is positive NPV for i if $q_i + \alpha s_j > 1$. Similarly, it is positive NPV for j if $q_j + \alpha s_i > 1$. Yet, there is an upper bound on the degree that each agent can have, and so agents can not typically form links with all potential positive NPV counterparties. Each agent prefers to form links with the least risky agents, i.e. those with the largest s. But then, agents with highest s prefer to match with each other

first, provided that they will not generate SCR for each other. Sorting with respect to risk type unravels and all agents end up being counterparties agents with close-by risk types s. Moreover, in order to eliminate SCR, agents want to form cliques. Yet each agent also wants to have their desired degree. This requires assigning agents with close-by contagion type q to the same cliques so that there is no conflict in terms of their desired clique size. The resulting size of cliques are as follows. For agent i, if $q_i + \alpha s_i > 1$, then V_i is increasing. Then the clique of i has $\overline{e}+1$ agents. If $q_i + \alpha s_i < 1$, V_i has a finite maximizer. Then i's clique has $1 + \min\{\arg\max_{d_i} V_i(d_i), \overline{e}\}$ agents. Note that this can be as small as 2 if q_i and s_i are sufficiently small.

A.5 Interventions and network reactions

Consider a principal who can intervene with contagion. Interventions can take many forms. These can be "soft interventions" that use transfers to induce strategic agents into continuation, such as bailouts and subsidies to firms, compensation to influencers in marketing, etc. Interventions can also be "hard interventions" without strategic components of contagion, such as a an oppressive government arresting opposers, medicine to cure a disease, or quarantining an area to stop the spread. Moreover, in certain applications, transfers that are conditional on continuation actions can be appropriate whereas in some such commitment may be infeasible. For example, in team production in industrial organization, the principal can be a manager trying to incentivize high effort and leverage synergies across workers. But the firms can engage in moral hazard and shirk, despite receiving a transfer. This requires a mechanism design approach and conditional transfers. In marketing and product adoption, the principal can be a brand that is promoting its product against a rival product by purchasing endorsements from social influencers. Then the transfers would be pinned down in a contract and would occur only if endorsement takes place.

Given the variety of scenarios, we take a simple approach. We suppose that the principal can induce good agents to continuation without a cost, which we call "saving." Solely to reduce the length of the analysis, we assume that

bad agents can not be saved.[37] For the objective of the principal, we turn to our simplified model for guidance. Suppose that there is a positive function λ_i such that the objective of the principal, after shocks and before contagion, is given by

$$W = \sum_{i \text{ continues}} (\lambda_i(d_i) - f_i)$$

Denote N_G the set of good agents. For a collection of positive functions $(\zeta_i)_{i \in N}$ and two subsets of good agents $N', N'' \subset N_G$, we say N' *is* $\zeta-$*cohesive relative to* N'' if $|N_i \backslash N''| \leq \zeta_i(d_i)$ for all $i \in N'$. Clearly, the union of two sets that are ζ-cohesive relative to N'' is also $\zeta-$cohesive relative to N''. Denote $\mathcal{N}_{\zeta, N''}$ the largest set N' that is ζ-cohesive relative to N''.

We say N' *is* ζ*-cohesive* if N' is ζ-cohesive relative to itself N'. Notice that union of two ζ-cohesive sets is also ζ-cohesive. Then the union of all ζ-cohesive sets define the maximally ζ-cohesive set, which we denote $\mathcal{N}_\zeta$. For example, in the absence of interventions, the set of agents who continue is characterized by $\mathcal{N}_R$ (a la Morris (2000)).

Denote $R'_i(d_i) = \frac{\lambda_i(d_i)+d_i}{2}$. We will assume that $R'_i(d_i) > \lfloor R_i(d_i) \rfloor$ for all i and d_i, meaning that the principal does not prefer that a continuing agent would default instead.

Theorem 5. *Suppose that Assumption 6 holds and* $R'_i(d_i) > \lfloor R_i(d_i) \rfloor$ *for all* i *and* d_i*. The optimal policy is to save all agents in* $\mathcal{N}_{R'} \backslash \mathcal{N}_{R, \mathcal{N}_{R'}}$*. Consequently,* $\mathcal{N}_{R'}$ *continues and the rest of agents default.*

Inducing a cohesive set to continue is relatively easier than inducing disjoint sets to continue. This is because of the complementarities across agents during contagion and complementarities embedded in the objective of the principal. Saving an agent makes his counterparties more "worthy" of saving. Given the objective function, it is optimal that the set $\mathcal{N}_{R'}$ continues and the rest defaults. In this case, $\mathcal{N}_{R, \mathcal{N}_{R'}}$ does not need saving because their resilience is

[37] Counterparts of results for the case in which bad agents can be saved are available upon request.

not exceeded by the agents outside $\mathcal{N}_{R'}$. The rest of the agents in $\mathcal{N}_{R'}$ need saving.

This intervention policy can be thought of as a dynamic "**counter-contagion**." Suppose that at the first round of contagion, the principal saves all good agents i who have less than $R'_i(d_i)$ but more than $R_i(d_i)$ bad counterparties. Those who have more than $R'_i(d_i)$ many bad counterparties are not saved and they default. Then in the second round of contagion, given all bad agents and the good agents who have not been saved default, agents i who have more than $R_i(d_i)$ but less than $R'_i(d_i)$ many defaulting counterparties are saved, and those who have more than $R'_i(d_i)$ many defaulting counterparties default. The iteration stops at the point where $\mathcal{N}_{R'}$ continues and the rest defaults. This algorithm can tractably pin down the optimal intervention policy in computations.

This argument makes it clear that the role of the interventions is to shift the resilience functions up from R to R' while keeping the payoff functions P and the payoff threshold T the same. The networks that are formed in POI can be found simply as corollaries of Theorems 1, 2, and 3. The only difference is that the clustering condition

$$|N_i \cap N_j| \geq d_i^* - 1 - \rho_i^* + \tau_j^*$$

is replaced with a looser version:

$$|N_i \cap N_j| \geq d_i^* - 1 - \rho_i'^* + \tau_j^*$$

where $\rho_i'^* = \lfloor R'_i(d_i^*) \rfloor$. The effect of interventions is **mitigate** SCR (not eliminate) which loosens the collective discipline and reduces clustering. Once SCR is mitigated by interventions, agents can eliminate the remaining SCR with less clustered networks. In the particular case of network games, one has $T \equiv R$, leading to full clustering. Then interventions bring effective resilience from $T \equiv R$ up to R', but keeps the payoff threshold $T \equiv R$ the same. The cliques dissolve into less clustered network. In the case of our lead example, all good agents were saved because we had $R'(d_i) > d_i$, meaning that $\mathcal{N}_{R'} = N_G$. This lead to an arbitrary regular network. In general, the effect is to reduce clustering.

Similar arguments apply for a counterpart of the Construction and Theorem

4. We skip this to save space. It is also worth noting that if we relax the requirement in Assumption 9 that $\overline{e} < \infty$, one can show that the ϵ-SS networks feature concentration. Anticipation of intervention result in increased the concentration as was the case in our simplified model. This establishes the counterparts of results in Section 3.3. We leave this to future work as it deserves a lengthy analysis.

B Relegated discussions and extensions

Here we provide the discussions that have been relegated. Section B.1 is for the related literature, Section B.2 is for the simplified model, and Section B.3 is for the general model.

B.1 Discussions regarding the literature

B.1.1 Related literature on networks

There is a growing number of recent work regarding contagion and systemic risk in economic networks. Eisenberg and Noe (2001), Acemoglu et al. (2012), Elliott et al. (2014), Acemoglu et al. (2015a), Baqaee (2018), Cabrales et al. (2017) all have different focuses and models to study contagion on fixed networks. Our model does not overlap with any of these but it shares common features with them, most importantly, the complementarities during contagion. Erol and Vohra (2018) and Elliott et al. (2018) are the closest to ours in terms of network formation. They find highly clustered networks but they do not have interventions. We also find high clustering but we also establish concentration results and study how these interact with interventions and regulations. For fixed networks, Leitner (2005), Bernard et al. (2017), and Kanik (2018) study bailouts and bail-ins (banks saving each other). They build on different models and they do not consider network formation. Kanik (2018) also finds that contagion is stopped either at the source (the location of the bad shock) or in the next round of contagion before it spreads.

Relative to network theory at large, this paper contributes to the literature on network games and diffusion. Applications include the cascading defaults of industrial and financial firms; the spread of cultural fads; the adoption of

new products, platforms, political views, etc (see Jackson (2010)). In this literature, Morris (2000) and Galeotti et al. (2006) study network games for fixed networks. Galeotti et al. (2018) study optimal interventions for a given network. Blume et al. (2011) studies endogenous networks under contagion risk. This paper is the first to study network games under interventions for endogenously formed networks. It characterizes strongly stable networks under a general binary network game framework with arbitrary and heterogenous payoff functions under interventions.

Besides these, Papers that study systemic risk given exogenous networks include Allen and Gale (2000), Eisenberg and Noe (2001), Kiyotaki and Moore (1997), Acemoglu et al. (2010), Allen et al. (2012), Blume et al. (2011), Caballero and Simsek (2013), Eboli (2013), Elliott et al. (2014), Freixas et al. (2000), Gai and Kapadia (2010), Gai et al. (2011), Gale and Kariv (2007), Gottardi et al. (2015), Glover and Richards-Shubik (2014), Gofman (2011), Gofman (2017), Kiyotaki and Moore (2002), Vivier-Lirimonty (2006), Acemoglu et al. (2015b), Elliott et al. (2014), and Glasserman and Young (2015). Some that study the efficient ways of stopping contagion for fixed networks are Drakopoulos et al. (2015), Freixas et al. (2000), Minca and Sulem (2014), and Amini et al. (2015). Moreover, Acemoglu et al. (2015b), Goldstein and Pauzner (2004), Moore (2011), Cabrales et al. (2017), Babus (2016), Blume et al. (2013), Kiyotaki and Moore (1997), Lagunoff and Schreft (2001), Zawadowski (2013), Farboodi (2015), Babus and Hu (2017), Chang and Zhang (2016), and Wang (2016)study the formation of networks.

Our theory uses threshold contagion models and strongly stable networks. Strong stability was introduced in Dutta and Mutuswami (1997) and Jackson and Van den Nouweland (2005). As they note, strongly stable networks often do not exist, but when they do, they have nice properties. We characterize variations of strongly stable networks under contagion risk. Most notably we introduce ϵ-Strong stability that yields existence under **arbitrary levels of heterogeneity**. Regarding contagion and network games Morris (2000) considers a simpler version of contagion under homogenous agents and introduces the notion of cohesiveness. He shows that cohesiveness can be used to

pin down the outcome of contagion. We generalize cohesiveness concept to arbitrary levels of heterogeneity, and study network formation as well as interventions. We find that agents form clustered networks that increases the chances of belonging to a cohesive set. We also make use of cohesiveness in identifying optimal interventions in our general theory. The optimal intervention takes the form "dynamic" counter-contagion, which induces an optimally sized cohesive set to resist contagion. Galeotti et al. (2010) consider general network games with arbitrary payoff functions and action spaces. Importantly, they consider agents who are incompletely informed about the network structure. They identify various effects of changing the network. We do not consider incomplete information but we provide stability results. Related to network formation, Goyal and Vega-Redondo (2005) consider a network formation problem with additive payoffs across links with homogenous agents. In case of a unilateral link formation game with no uncertainty in payoffs, they find that an empty network, a complete network, or a network that consists of two cliques is formed, depending on the cost of links. Galeotti et al. (2006) study non-cooperative network formation with heterogenous agents along the lines of Bala and Goyal (2000). They have additive payoffs and their focus is the direct value of links instead of a game played on the network. They find that short average distances and centrality are features of networks formed. Our findings are consistent with this insight. Erol and Vohra (2018) consider additive payoffs with uncertainty in the benefits that are derived from links with homogenous agents, and they show that if the behavior is very contagious, the unique strongly stable network consists of disjoint cliques. Using our framework for binary network games and contagion, we study strongly stable networks (neither unilaterally formed links nor pairwise stable networks), under any payoff function (not necessarily additive), any threshold rule (neither necessarily linear nor necessarily very contagious), uncertainty regarding nodes (not edges), under arbitrary levels of heterogeneity (not homogenous). Regarding interventions, the most related work is by Galeotti et al. (2018) who study interventions with network games with complementarities. They do not consider network formation. Their network game has continuous actions with

a quadratic utility function and the principal has a budget constraint. Our network game is binary and the principal does not face a budget constraint. This simplicity in our paper allows us to characterize strongly stable networks with arbitrary and heterogenous payoff functions.

B.1.2 Threshold contagion in financial networks

As described in the introduction, the key difference of our model with existing counterparty risk models is that in our model contagion spreads only via binary defaults of counterparties, not the particular circumstances under which default occurred. The size of a bad shock does not affect contagion because the losses are incurred by external creditors. This buys tractability which enables us to provide characterization of stable networks. Next we discuss how simplified versions of some existing models of counterparty risk can be nested into our framework.

Eisenberg and Noe (2001) The first example is a variant of Eisenberg and Noe (2001) as follows. Banks have liabilities to external creditors whose claims have seniority over interbank claims. The external cash flow to each bank is either high (good shock) or low (bad shock). Low external cash flows do not suffice to pay the liabilities to external creditors. Then a bad bank can not repay its liabilities and goes bankrupt. Due to the seniority structure, its counterparties recover zero from the defaulting bank. Contagion is triggered by these low external cash flows. If a bank with a high external cash flow suffers a large number of counterparty defaults, its fulfilled interbank claims are reduced to a point that it can not pay its liabilities and it defaults. Assuming a sufficiently small default recovery rate, such as high fire-sale costs, a defaulting bank can only partially repay its external creditors. The counterparties of the defaulting bank recover zero from the defaulting bank. Then defaults propagate through interbank liabilities via contagion dynamics described in our model. The only difference with the standard Eisenberg and Noe (2001) model is the high bankruptcy cost.

Erol and Ordoñez (2017) The second example is Erol and Ordoñez (2017) who study banks that rely on each other to meet their short-term liquid-

ity needs via interbank lending partnerships. Banks have dividend yielding projects that have potential future refinancing needs. A bad shock means large operational costs or large withdrawals of demand deposits that force the bank into liquidating its dividend yielding project. Then a good bank with many defaulting counterparties may find that it costly to raise liquidity on demand in the future. Such expected illiquidity can render an otherwise healthy bank unlikely to meet the refinancing needs of its project, which can incentivize the liquidation of its project to avoid opportunity costs or management costs. These liquidations propagate through interbank lending partners.

Elliott, Golub and Jackson (2014) Our final example is related to Elliott et al. (2014). Suppose that each bank has one pledgable and one non-pledgable asset. Liabilities are larger than the size of the non-pledgable asset and the size of the pledgable asset. Hence, both assets must have high returns to pay the liabilities. This creates incentives for banks to exchange tranches of each others' pledgable assets to diversify against bankruptcy costs (which could potentially be the costly liquidation of a third illiquid asset). A typical balance sheet is pictured in Figure 13.

Banks first get shocks to their non-pledgable assets and then to their pledgable assets. A bank with a low return to its non-pledgable asset (bad shock) faces immediate bankruptcy. This starts contagion. The tranches of the pledgable asset of a defaulting bank generates low yield for the counterparties due to fire-sale costs. These losses reduce the margin of diversification for the good counterparties of bad banks. It becomes less likely that a good bank with many defaulting counterparties have high enough yield from its remaining counterparties that will suffice to pay its liabilities. This triggers defaults and asset devaluations among good banks with sizable counterparty losses. Upon defaults of good banks in the first round of contagion, more good banks suffer counterparty losses and default. Contagion progresses via linkages across good banks in this fashion.

Figure 13: Balance sheet of bank i

Assets	Liabilities
Non-pledgable asset	Liabilities
Pledgable asset	

(a) i has no counterparties

Assets	Liabilities
Non-pledgable asset	Liabilities
Shares left	
Shares from i'	
Shares from i''	
Shares from i'''	

(b) i has three counterparties

Figure 13-a and -b show bank i's balance sheet when it has no counterparties and three counterparties, respectively. If bank i does not have any counterparties, its solvency relies on high returns to both of its assets. If i diversifies its portfolio by exchanging tranches of its pledgable asset with other banks, it needs high return to at least two out of four tranches now owned. This increases the probability of avoiding bankruptcy costs. However, if i's three counterparties get low return to their non-pledgable assets and default before the maturity of their pledgable asset, the tranches owned by i have low return and so i faces bankruptcy regardless of the returns to its own projects.

B.2 Discussions and extensions regarding the simplified model

B.2.1 Interventions with commitment

In an alternative situation where the government has the commitment at the interim stage, injections could be made during defaults conditional on actions taken. Suppose that the government, after shocks are realized and observed, before contagion, can intervene and commit to transfers conditional on the actions taken. Now the transfer to i is $Q_i(a_i)$. Given transfer rule, payoff functions are now $P_{Ci}(e_i, c_i) + Q_i(0)$ if i continues and $P_{Di}(e_i) + Q_i(1)$ if i defaults. This alters the contagion in a different way than unconditional transfers.[38] In principal, firms with negative shocks can also be induced into continuation

[38]Transfer to i depends only on the action of i. This is without loss of generality. For arbitrary transfer rules $(Q_i(a))_{i\in N}$, the induced game $P + Q$ may not be supermodular. Assuming that firms play the best Nash equilibrium whenever it exists and any arbitrary selection among Nash equilibria otherwise, the optimal policy takes the assumed form in which the transfer to i depends only on a_i.

under such conditional transfers. We call Q optimal if it maximizes welfare and uses minimal transfers in doing so.

Suppose that shocks are bounded as follows. For all i, $\theta_{Bi} < \overline{\theta}_B$ and $\theta_{Gi} \in [\underline{\theta}_G, \overline{\theta}_G]$. Here $\underline{\theta}_G > \overline{\theta}_B$, so bad shocks are not desirable. Also $\underline{\theta}_G > 0$ it is efficient for good firms to continue the investments. We have two cases with respect to (with respect to) sizes of bad shocks. As before, the first case is small shocks: $\underline{\theta}_B > 0$. The second case is large shocks: $\overline{\theta}_B < 0$. We also assume that exposures are not too uneven: $\left(\frac{|\overline{\theta}_B|}{\overline{\theta}_G}\frac{1}{\eta_j}\right)e_{ji} \geq e_{ij}$ for all i and j.

Proposition 8. *For small shocks the optimal policy is to save all bad firms. Transfers are*

$$Q_i^*(a_i) = \left[(p_i r_i + e_i r_i + z_i) - \theta_i(p_i + e_i)\right] \mathbb{1}_{\theta_i = \theta_{Bi}, a_i = 0}$$

For large shocks, the optimal policy is to save all good firms who are facing default due to bad counterparties. Transfers are

$$Q_i^*(a_i) = \left[(p_i r_i + e_i r_i + z_i) - \theta_i(p_i + e_i - \eta_i b_i)\right]^+ \mathbb{1}_{\theta_i = \theta_{Gi}, a_i = 0}$$

It is efficient for good firms to continue their operations. If bad shocks are positive, it is also efficient for bad firms to continue operations. In this case, saving bad firms by ensuring that they can meet their liabilities is optimal and they continue. This way, there is no need to save good firms because they do not incur counterparty losses due to bad counterparties. If bad shocks are negative and sufficiently small, then it is efficient for bad firms to default even after considering the fact that saving them would reduce the counterparty losses of others. The welfare loss by inducing bad firms to continue is larger than the welfare gain of their counterparties. Instead, good firms are directly saved via appropriate injections that make sure they can repay their liabilities.

B.2.2 Relegated discussions of Strong Stability

Strong stability is defined differently in Jackson and Van den Nouweland (2005) and Dutta and Mutuswami (1997). In Jackson and Van den Nouweland (2005), deviations that Pareto improve the coalition are precluded, whereas in Dutta and Mutuswami (1997) deviations that strictly improve every member of the coalition are precluded. To minimize confusion we use the name Pareto Strong

Stability for Jackson and Van den Nouweland (2005) and Strong Stability for Dutta and Mutuswami (1997).

The cooperative nature of the solution concept is necessary to capture some notion of mutual consent for link formation. One can, in fact, micro-found strongly stable networks as strong Nash equilibria of a link proposal game as follows. Suppose that agents make proposals to their desired counterparties at cost ε each. Links are formed for mutual proposals. If a link is formed, ε is refunded to its proposers. Then in strong Nash equilibrium of the proposal game, only links that are formed feature proposals. Then, given strong Nash equilibrium network, no agent can form a link without a joint deviation with the potential counterparty. However, every agent can cut an existing link in the strong Nash equilibrium network by withdrawing the proposal. Therefore, the Strong Nash equilibria of the game correspond exactly to SS networks.

This being said, we do not see strong stability as a positive description of how networks are formed. Rather, it should be seen as a selection among other weaker solution concepts, such as pairwise stability.

B.2.3 Relegated discussions of Section 3.2.1

Note that it is possible to have $d^*w = \overline{e}$. If $\overline{e}$ is not small and links are negative NPV, i.e. $(1-\alpha)\kappa > 1$, then $d^*w < \overline{e}$. If, however, links are positive NPV, i.e. $(1-\alpha)\kappa < 1$ or $\overline{e}$ is small, firms want to have as many counterparties as possible. In this case, $d^*w = \overline{e}$. But with a slight additional convexity in the payoffs, this would not be the case anymore. Such convexity can be the consequence of convex management costs, convex link formation costs, or decreasing marginal returns from projects. We work with linear forms for simplicity. The general theory allows for arbitrary functional forms. Under such convexity, even with positive NPV links and large $\overline{e}$, firms form less links in AOI than in POI for small shocks. For large shocks, their degree, again, does not change. The network becomes interconnected for either small or large shocks.

B.2.4 Relegated discussions of Section 3.3.2

Notice that the parametric assumptions governing the shocks must only satisfy $\sigma\alpha_S + \sigma'\alpha'_S > \alpha'_S$ and $\alpha_L > \frac{\sigma\alpha_S^2+\sigma'\alpha_S'^2}{\sigma\alpha_S+\sigma'\alpha'_S}$. The only lower bound imposed on σ is $\sigma > 0$. That is, the correlation can potentially be arbitrarily small. This sheds light on to the emergence of concentration in Section 3.3.1. There, idiosyncratic shocks to the large and small firms had to have the same probability for the anticipation of interventions to increase concentration. In fact, under arbitrarily small correlations, such a sharp restriction is not necessary for increased concentration.

The correlation is helpful in addressing a typical criticism to financial networks. When links are positive NPV, it is argued that dividing institutions into smaller pieces would diversify against the idiosyncratic risk and make the network analysis redundant. This is not the case in our setup. Links are positive NPV unconditionally, but they are negative NPV conditional on the low state. Dividing firms into many smaller pieces only increases the expected losses. This is reflected in the fact at most $\overline{d}_L$ many small firms form links with the large firm regardless of n.

We have taken $\phi_S = 0$ and $\kappa_S = 1$ for tractability. Similar results hold as long as $\phi_S < \phi_L$ ensuring that the network is not fully concentrated around the large firm in AOI. $\kappa_S > 1$ can induce the small firms to reduce their desired exposure reducing the number of their links. Also, having one large firm is not critical here. With multiple large firms that have correlated shocks, similar results go through. If there are multiple large firms that have idiosyncratic shocks, small firms face tradeoffs between first-, second-, and third-order counterparty risk. Weaker solution concepts such as pairwise stability can be used in this case to show that similar networks emerge as stable networks. But this comes at the expense of uniqueness.

B.2.5 Relegated discussions of Section 4.3

First-order network hazard. The picture is different under first-order network hazard. Recall that all bad firms are saved under small shocks (first-order network hazard). Then in POI, in the unique stable network, all small firms

form links with the large firm regardless of the timing of the risk profile choice and the magnitude of α'_L. This is not true in AOI. Therefore, first-order network hazard **exacerbates** individual moral hazard in risk taking.

Alternate timing. Consider $1-\frac{1}{\kappa_L} < \alpha'_L < \frac{\sigma\alpha_S^2+\sigma'\alpha_S'^2}{\sigma\alpha_S+\sigma'\alpha_S} < \alpha_L$ and suppose that the choice of the risk-return profile is made after the network is formed. In this case, small firms understand that whatever network is formed the large firm will choose the risky profile. Then both in AOI and POI, small firms abstain from forming links with the large firm. The network is not altered by the anticipation of interventions. Furthermore, since the large firm does not have any links in either case, it chooses the risky profile regardless. The large firm engages in individual moral hazard. Yet moral hazard does not interact with second-order network hazard.

Alternate parameters. If $\frac{\sigma\alpha_S^2+\sigma'\alpha_S^2}{\sigma\alpha_S+\sigma'\alpha_S} < \alpha'_L < \alpha_L$, the large firm chooses the risky profile in AOI and POI regardless of timing. The risk profile is fixed and so all results in Section 3.3.2 hold. There is indeed more risk shifting on to external creditors due to the choice of the risky profile, hence more moral hazard. But moral hazard does not interact with second-order network hazard and the network structure.

B.3 Discussions and extensions regarding the general model

B.3.1 Heterogenous link benefits

We have worked with Assumption 6 implying that the preferences of i regarding the identities of his counterparties only pertain to the default risk of his counterparties and their network positions. In certain cases, the link to a particular agent j can be more valuable than links to other agents, above and beyond the smaller exogenous default risk of j captured by α_j. Heterogenous values of links is not a force particularly relevant for contagion compared to default risk and network position of agents, but we discuss it nonetheless.

Maintain the first part of Assumption 6 that there exists w_i such that $w_{ji} = w_i$ for all j. Now suppose that the payoff and resilience of i can depend on

exposures to i, $e_i^{out} = \Sigma_{j \in N_i} w_{ij} = \Sigma_{j \in N_i} w_j$. Assume that P_{Ci}, P_{Di}, and R_i are increasing (or decreasing) in e_i^{out}. Now, agents with higher (or lower) w_j are more preferred counterparties for i. Call w_i the exposure type of i.

First consider symmetric with respect to their shocks: there is some α such that $\alpha_i \equiv \alpha$ for all i (Assumption 7). Then, if exposure types are identical for all pairs, GS networks are given by Theorems 1 and 2. If exposure types are different, agents can be ranked with respect to their exposure type and there will be assortative sorting and matching from the best type to the worst. This yields PSS and ϵ-SS networks as in Theorems 3 and 4 (under appropriate counterparts of Assumptions 8 and 9 for exposure types).

If risk types are heterogenous but the risk types and exposure types of agents have the same ranking (in the spirit of Assumptions 8 and 9), version of Theorems 3 and 4 can be restored by the same sorting arguments.

If the risk types α_i and exposure types w_i are all heterogenous and do not admit a common ranking, then there are potential tradeoffs between the FCR from a counterparty and the exposure type of the counterparty. We believe the individual tradeoffs faced by counterparties of i between exogenous risk α_i of i and the exogenous link benefit w_i of i are not particularly relevant for our analysis of contagion and interventions.

B.3.2 Non-existence of SS in small networks

Suppose that there are three agents $1, 2, 3$. Default payoffs are 0. Resilience functions are $R_i(d) \equiv d$ for all agents. The continuation payoffs are

$$P_{1,2}(c,d) = \begin{cases} 2 - \varepsilon c & \text{if } d = 2, \\ -\varepsilon c & \text{if } d = 1, \\ 1 - \varepsilon c & \text{if } d = 0, \end{cases} \qquad P_3(c,d) = \begin{cases} 1 - \varepsilon c & \text{if } d = 2, \\ 2 - \varepsilon c & \text{if } d = 1, \\ -\varepsilon c & \text{if } d = 0. \end{cases}$$

for some small $\varepsilon > 0$. The deviations are described here and shown in Figure 14. 1 and 2 prefer having 2 links to 0 links to 1 link. 3 prefers having 1 link to 2 links to 0 links. In this situation there are no SS networks. The deviations from each candidate network are illustrated in Figure 14. All agents jointly deviate from G_1 to G_2. 3 deviates from G_2 to G_3. 2 deviates from G_3 to G_4.

1 deviates from G_4 to G_1. 1 and 2 jointly deviate from G_5 or G_6 to G_1.

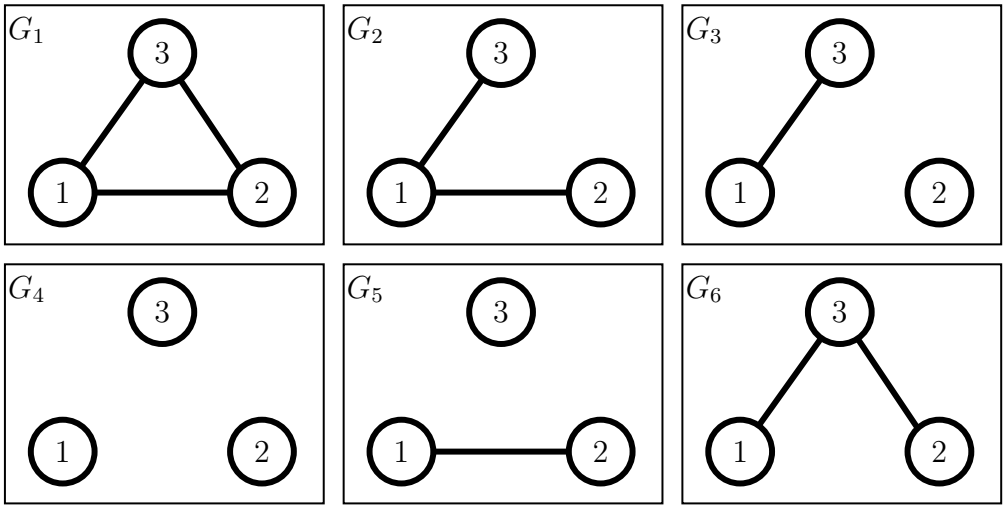

Figure 14: Labels of networks and deviations

B.3.3 Incomplete information on shocks

Vives (1990) also shows that any Bayesian game with supermodular ex-post payoff functions has a maximal pure strategy Bayesian-Nash equilibrium. Now suppose that each agent observes the shocks to itself, its counterparties, and possibly some other agents. Formally, i observes the shocks to a subset $\mathcal{I}_i(E, \boldsymbol{\theta})$ of banks that includes i and all counterparties of i: $i \cup N_i \subset \mathcal{I}_i(E, \boldsymbol{\theta}) \subset N$. Results in our general theory hold identically both for AOI and POI. The key observation is that in AOI, if agents form cliques, the shocks of every agent in the clique are common knowledge across all clique members. Therefore, by forming a clique with its own type, n_i has $V_i(d_i^*)$ expected payoff. Clearly, this payoff cannot be exceeded in any configuration or any information structure because bad counterparties always default. In POI, an agent does not need to know anything more than the shocks of its counterparties. This is so because interventions make sure, under large shocks, all good agents continue and good counterparties do not default due to contagion anyways. Under large shocks, no information is needed by i in POI.

C Proofs

Section C.1 includes the proofs of the general model presented in Appendix A. Section C.2 includes the proofs for the simplified model. Naturally, the simplified model's proofs make use of the results of the general model. Therefore we start with the proofs of the general model to minimize repetition and save space. That being said, some results in the simplified model are not direct corollaries of the results of the general model because specific functional forms allow us to obtain stronger results in the simplified model.

C.1 General model

Proof of Theorem 1

Step 1. We first establish an upper bound on the expected payoff of agents. For any network, i's expected payoff is less than or equal to $V_i(d_i)$. Because the distribution of f_i (weakly) first-order-stochastically dominates the distribution of b_i due to potential spillovers and $P_i(d_i, f_i)$ is (weakly) decreasing in f_i. Also note that this payoff can be achieved by being at the center of a disjoint star network with d_i leaves.

Step 2. Denote $d_{ij} = |N_i \cap N_j|$, $\rho_i = \lfloor R_i(d_i) \rfloor$, and $\tau_i = T_i(d_i)$. We use this step in proofs of other results as well, so we make it a lemma.

Lemma 1. *If i achieves $V_i(d_i)$, then for any $j \in N_i$,*

$$\min\{d_{ij}, \tau_i\} + d_j - 1 - d_{ij} \leq \rho_j. \tag{5}$$

Proof. If i achieves $V_i(d_i)$, then for any $j \in N_i$, in any positive probability event at which $\theta_i = \theta_{Gi}$, $\theta_j = \theta_{Gj}$, and $b_i = \tau_i$, it must be the case that $b_j \leq \rho_j$. Otherwise, in this event, j defaults and so $f_i \geq b_i + 1$. Then i gets less strictly less payoff than $P(d_i, b_i)$ in this event so $V(d_i)$ can not be obtained. Therefore, $b_i = \tau_i$ implies $b_j \leq \rho_j$.

In particular, consider an event such that $\min\{d_{ij}, \tau_i\}$ many agents in $|N_i \cap N_j|$ get bad shocks, $\tau_i - \min\{d_{ij}, \tau_i\}$ many agents in $N_i \backslash (N_j \cup \{j\})$ get bad shocks, all agents in $N_j \backslash (N_i \cup \{i\})$ get bad shocks, and all the remaining agents get

good shocks. Here $b_i = \tau_i$. So we must have $b_j \leq \rho_j$. At this event b_j is equal to $\min\{d_{ij}, \tau_i\} + d_j - 1 - d_{ij}$ so (5) must hold. □

Step 3. A la Morris (2000): a subset N' of agents cohesive if $\theta_{i'} = \theta_{Gi'}$ and $|N_{i'} \cap N'| \geq d_{i'} - \rho_{i'}$ for all $i' \in N'$. Observe that union of cohesive sets are also cohesive. The maximally cohesive set of agents continue and the rest default at the outcome of contagion.

Step 4. If (5) holds for all $j \in N_i$, then i achieves $V_i(d_i)$.

Note that (5) implies $\min\{d_{ij}, b\} + d_j - 1 - d_{ij} \leq \rho_j$ for all $b \leq \tau_i$. Then whenever $b_i \leq \tau_i$, for any $j \in N_i \cap N_G$, the number of good agents in $(N_i \cap N_G) \cup \{i\}$ is at least $d_{ij} + 1 - \min\{d_{ij}, b\}$ which is larger than $d_j - \rho_j$. Therefore, $(N_i \cap N_G) \cup \{i\}$ is a cohesive set. By Step 3, all agents in it continue. In other words, all good counterparties of i continue and all bad counterparties of i default, conditional on $b_i \leq \tau_i$. Then f_i and b_i follow the exact same distribution whenever $b_i \leq \tau_i$. When $b_i > \tau_i$, i's payoff is independent of f_i regardless. Therefore, i achieves $V_i(d_i)$.

Step 5. By Steps 1, 2, and 4, each agent i achieves $V_i(d_i)$ if and only if $(d_{ij} - \tau_j)^+ \geq d_i - 1 - \rho_i$ for every $\{i, j\} \in E$.

Note that $V_i(d_i^*)$ is the globally maximum payoff for agent i by Step 1. To achieve $V_i(d_i^*)$, each i must have $d_i = d_i^*$. Then all agents to achieve their globally maximum payoffs if and only if $d_i = d_i^*$ for all i and $(d_{ij} - \tau_j^*)^+ \geq d_i^* - 1 - \rho_i^*$ for all $\{i, j\} \in E$.

Proof of Theorem 2

(*Sufficiency*) **Step 1.** First we show that the described networks are GS. Take two contagion-similar counterparties i and j. They belong to the same clique so $|N_i \cap N_j| + 1 = d_i^* = d_j^*$. They are contagion-similar so $\rho_i^* = \rho_j^*$. Then $(|N_i \cap N_j| - \rho_j^*)^+ = (d_i^* - 1 - \rho_i^*)^+ \geq d_i^* - 1 - \rho_i^*$. Now take two non-contagious counterparties i and j. They are non-contagious so $\rho_i^* \geq d_i^* - 1$ and $\rho_j^* \geq d_j^* - 1$. Then $(|N_i \cap N_j| - \rho_j^*)^+ = 0 \geq d_i^* - 1 - \rho_i^*$. Therefore by Theorem 1, the network is GS.

(*Necessity*) **Step 2.** We use this step in proofs of other results as well, so we make it a lemma.

Lemma 2. *Consider any network and two counterparties i and j. If i has $V_i(d_i)$ and j has $V_j(d_j)$ payoff, then one of the following must hold:*

- i and j are both non-contagious, or
- i and j are both contagious, they have the identical set of counterparties besides each other ($N_i \backslash \{j\} = N_j \backslash \{i\}$), and they have $\rho_i = \rho_j$.

Proof. Denote $d_{ij} = |N_i \cap N_j|$. By Lemma 1, we have

$$\min\{\rho_i, d_{ij}\} + d_j - d_{ij} - 1 \leq \rho_j,$$

$$\min\{\rho_j, d_{ij}\} + d_i - d_{ij} - 1 \leq \rho_i.$$

If one of i and j, say j, is non-contagious, then $\rho_i \geq d_i - 1 \geq d_{ij}$. Then $\min\{\rho_i, d_{ij}\} = d_{ij}$. Then the latter inequality becomes $d_j - 1 \leq \rho_j$. Thus, j is also non-contagious.

Now consider the case in which both are contagious. $\rho_j < d_j - 1$ so the the former inequality implies $\min\{\rho_i, d_{ij}\} < d_{ij}$. Thus $\min\{\rho_i, d_{ij}\} = \rho_i$. Then the former inequality becomes $\rho_i + d_j - d_{ij} - 1 \leq \rho_j$. Symmetrically, we have $\rho_j + d_i - d_{ij} - 1 \leq \rho_i$. Adding both up to get $d_i + d_j \leq 2(d_{ij} + 1)$. This implies that $d_i = d_j = d_{ij} + 1$. In other words $N_i \backslash \{j\} = N_j \backslash \{i\}$. Putting this back into the inequalities we get $\rho_i \leq \rho_j \leq \rho_i$ meaning $\rho_i = \rho_j$. □

Step 3. Consider any network and suppose that it is GS. All agents must have their assortative degrees: $d_i = d_i^*$. Take two counterparties i and j. Then by Lemma 2, either both are non-contagious or both are from the same contagion-class and have the identical set of counterparties. Therefore, by the connectivity of components, a component can consist of only non-contagious agents or contagious agents. Those that consist of contagious agents must consist of agents from the same contagion-similarity class.

Proof of Theorem 3

This follows the proofs of Theorems 1 and 2. The critical observation is the assortative sorting across agents with respect to risk-type. Agents in the highest ranked risk-class can achieve their first-best payoff only by forming links with each other in the way described by the theorem. If they deviate by forming a link with a lower risk-class their payoff strictly decreases. Deviations among themselves are can not be profitable by Theorems 1 and 2. Given that agents in the highest risk class can not be part of a (Pareto) profitable deviation, the same argument applies to the next highest ranked risk-class for the subnetwork of agents that are not in the highest ranked risk-class. Induction completes the proof for sufficiency. For necessity, $n_\iota \geq d_\iota^* + 1$ implies that there are indeed networks in which agents achieve these payoffs (maximized assortative values), for example by forming cliques with agents from the similarity class. Thus, a PSS network must give all agents at least their maximized assortative values. The same induction argument shows that this is possible only with the networks described.

Proof of Theorem 4

Step 1. For $s \leq s'$ define $V_i'(d_i; s, s')$ as

$$V_i'(d_i, s, s') = \mathbb{E}_\omega \Big[\Big((1 - s'\alpha(\omega)) + s'\alpha(\omega) \mathbb{P}_{b_i} [b_i > R_i(d_i)] \Big) P_{Di}(d_i)$$
$$+ s'\alpha(\omega) \mathbb{P}_{b_i} [b_i \leq R_i(d_i)] \mathbb{E}_{b_i} [P_{Ci}(d_i, b_i) | b_i \leq R_i(d_i)] \Big]$$

where $b_i \sim \mathbb{B}[d_i, 1 - s\alpha(\omega)]$. By definition $V_i'(d_i, s_i, s_i) = V_i(d_i)$. V' is uniformly continuous in s and s'. Also $\mathcal{C}$ is finite. Then there exists ϵ' such that for all i, $|V_i(d_i^*, s, s') - V_i(d_i^*)| < \epsilon$ for any $s, s' \in B_{\epsilon'}(s_i) := (s_i - \epsilon', s_i + \epsilon')$.

Step 2. Take a contagion-class. By SLLN, there exists $\underline{n}'$ such that if $n > \underline{n}'$, for all $s \in [\underline{s}, \overline{s}]$, almost surely, there exists at least $\overline{e}+1$ agents in the contagion-class who have risk type in $B_{\epsilon'/2}(s)$. Since $\mathcal{C}$ is finite, the maximum of all such $\underline{n}'$ over all contagion-classes constitute a uniform $\underline{n}'$ that works for all contagion-classes.

Step 3. Define $B_1 = B_{\epsilon'}(\overline{s} - \epsilon')$, $B_2 = B_{\epsilon'}(\overline{s} - 2\epsilon')$,..., $B_T = B_{\epsilon'}(\overline{s} - T\epsilon')$ where T is such that $\overline{s} - T\epsilon' < \underline{s} \leq \overline{s} - (T-1)\epsilon'$. We complete the proof by

an iteration:

In the first step of the iteration, consider all the bins from all contagion-classes such that the risk-types of agents in these bins are in B_1. By Step 2, there is at least one bin from each contagion-class. By Step 1, they can not improve their payoff by more than ϵ. Then these agents will not be part of any deviation.

In the second step, take out all these agents from the first step. Consider all the remaining bins from all contagion-classes such that the risk-types of agents in these bins are in B_2. Notice that B_1 and B_2 overlap. This is to deal with agents whose bin is partly in B_1 and partly in B_2. Formally, B_t's have radius ϵ' and so, by Step 2, there is at least one bin from each contagion-class. By Step 1 and the fact that the agents in the bins that were taken out in the first iteration do not deviate, agents in the second iteration also do not deviate.

We keep iterating this way. At some iteration (including potentially the first iteration), if a contagion-class runs of bins, meaning that there are only residual agents left in the contagion class, remove this contagion class and continue the iterations. Iteration stops in finite many rounds since n is finite. Denote N_ϵ the set of agents that have been assigned to bins. By the construction of the iteration, all agents in N_ϵ are covered by the iteration, and so agents in N_ϵ do not have deviations among themselves that can improve their by payoff by more than ϵ.

Step 4. Finally, note that the number of residual agents is at most $|\mathcal{C}|\overline{e}$. If n is large enough $|\mathcal{C}|\overline{e}/n < \epsilon$, which completes the proof.

Proof of Theorem 5

We first show that by saving $\mathcal{N}_{R'} \backslash \mathcal{N}_{R,\mathcal{N}_{R'}}$, the set of continuing agents becomes $\mathcal{N}_{R'}$. Conditional on $\mathcal{N}_{R'}$ continuing, an agent i outside $\mathcal{N}_{R'}$ has more than $R_i(d_i)$ counterparties outside $\mathcal{N}_{R'}$. So i defaults. An agent i inside $\mathcal{N}_{R,\mathcal{N}_{R'}}$ has less than $R_i(d_i)$ counterparties outside $\mathcal{N}_{R'}$ so he continues. Agents in $\mathcal{N}_{R,\mathcal{N}_{R'}}$ are saved so they continue.

Step 1. Now we show that this is the optimal policy. Consider the policy of saving agents in X. Denote Y the set of agents that continue under this

policy. Notice that Y is the largest set such that $Y\backslash X$ is R-cohesive relative to Y. Since $Y\backslash X$ is R-cohesive relative to Y, it is also R'-cohesive relative to Y.

Step 2. Suppose that X is not R'-cohesive relative to Y. Then there is some $i \in X$ such that $|N_i\backslash Y| > R'(d_i)$. Then $|N_i\backslash Y| > R(d_i)$. Consider the policy of saving $Y\backslash i$. All of $Y\backslash i$ continue. Agents outside Y still default. On the other hand i defaults because $|N_i\backslash Y| > R(d_i)$. The change in welfare with respect to the policy of saving X is 1) $-(\lambda_i(d_i) - |N_i\backslash Y|)$ from the change through i and 2) $-|N_i \cap Y|$ through the change in i's counterparties in $Y\backslash i$. Note that $|N_i \cap Y| = d_i - |N_i\backslash Y|$. Then total change is $-(\lambda_i(d_i) + d_i - 2|N_i\backslash Y|)$ which is positive because $|N_i\backslash Y| > R'(d_i) \geq \frac{\lambda_i(d_i)+d_i}{2}$. This gives a contradiction. So X must be R'-cohesive relative to Y.

Step 3. Suppose that there is an agent $i \notin Y$ such that $|N_i\backslash Y| \leq R'(d_i)$. Since $i \notin Y$, i defaults, meaning $|N_i\backslash Y| > R(d_i)$. Consider the policy of saving $X \cup i$ instead of X. Then all of $Y \cup i$ continues. Also, $j \in N_i\backslash Y$ now continues if $|N_j\backslash Y| - 1 \leq R_j(d_i) < |N_j\backslash Y|$ (i.e. $\lfloor R_j(d_i) \rfloor = |N_j\backslash Y| - 1$). Then $R'_j(d_j) \geq |N_j\backslash Y|$. The continuation j increases welfare by at least $(\lambda_j(d_j) + d_j - 2|N_j\backslash Y|) \geq 0$. As for i, similarly, since $|N_i\backslash Y| \leq R'(d_i)$, the continuation of i increases welfare by at least $(\lambda_i(d_i) + d_i - 2|N_i\backslash Y|) \geq 0$. A contradiction. So there are no agents $i \notin Y$ such that $|N_i\backslash Y| \leq R'(d_i)$.

Step 4. By Step 1 and 2, both X and $Y\backslash X$ are R'-cohesive relative to Y. Then Y is R'-cohesive relative to Y. That is, Y is R'-cohesive. Then, by Step 3, Y is the maximally R'-cohesive set, $Y = \mathcal{N}_{R'}$. Given that members of $\mathcal{N}_{R'}$ continue, $\mathcal{N}_{R,\mathcal{N}_{R'}}$ do not need saving, so $X = \mathcal{N}_{R'}\backslash\mathcal{N}_{R,\mathcal{N}_{R'}}$.

C.2 Simplified model

Proof of Proposition 1

A firm with a negative shock $\theta_i = \theta_{Bi} < 0$ drops its projects even if it receives transfers. Such firms can not be induced to continuation. Other firms could be defaulting either because the shock is bad ($0 < \theta_i = \theta_{Bi} < r_i$) or they have good shocks ($r_i < \theta_i = \theta_{Gi}$) yet they suffer many counterparty failures. It is

welfare optimal to induce these firms to continuation. Given that firms with negative shocks are defaulting, an firm with a positive shock can not be induced to continuation by a transfer less than Q_i^*. Giving Q_i^* to all firms ensures, in the best Nash equilibrium, that all firms with positive shocks continue.

Proofs of Propositions 2 and 3

First consider Proposition 2.

(*AOI, sufficiency*) Corollary of Theorem 2.

(*AOI, necessity*) Take any network and suppose that it is stable (Pareto Efficient Strongly Stable). Then it is also strongly stable. If there are no firms with payoff $V(d^*)$, d^*+1 many would deviate to forming a disjoint clique and get $V(d^*)$. So there is at least one firm with payoff $V(d^*)$, say i. Suppose that i has at least one counterparty that does not have $V(d^*)$. Let $N' \subset N_i$ have payoff $V(d^*)$ and $N_i \backslash N' \neq \emptyset$ do not. By Lemma 2, all firms in $N' \cup i$ must have the identical set of counterparties. Thus, members of $N' \cup i$ make up a clique (not a disjoint clique) and their only other counterparties are $N_i \backslash N'$. But then, members of $N_i \backslash N'$ form all their missing links among themselves, keep their links with $N' \cup i$, and cut all their links with firms other than $N_i \cup i$. This turns their component into a clique of order d^*+1 and gives them $V(d^*)$, which is a strict improvement for all of $N_i \backslash N'$. Therefore, $N_i \backslash N' = \emptyset$.

So all counterparties of i have payoff $V(d^*)$. By Lemma 2, all counterparties of i have the the identical set of counterparties. This means that the component is a disjoint (d^*+1)-clique.

By iterating this argument for the remaining firms, we see that all components must be disjoint (d^*+1)-cliques.

(*POI, sufficiency*) Corollary of Theorem 2. The only important thing to notice is that since bad firms are being saved, $V(d) = \arg\max_{dw \leq \overline{e}} [d - \phi/w]^+$ which is maximized at $\overline{e}/w$ instead of d^*.

(*POI, necessity*) These are all Pareto efficient networks which, along with sufficiency, completes the for the network part.

For Proposition 3, the proof is similar to nearly identical. The only difference is that since, in POI, good firms continue and bad firms default, firms have degree d^*.

Proof of Proposition 4

Claim 1. In AOI, if a small firm i has $V_i(d_i)$ payoff, it can not be counterparties with the large firm.

Proof. Denote the large firm L. Take a small firm i and suppose that it is counterparties with L and has $V_i(d_i)$ payoff. By Lemma 1, we have

$$\begin{aligned}
\min\{d_i - \phi_S, d_{iL}\} + d_L - d_{iL} - 1 \leq d_L - \phi_L/w_L &\implies \\
\min\{d_i - \phi_S, d_{iL}\} \leq d_{iL} + 1 - \phi_L/w_L < d_{iL} &\implies \\
\min\{d_i - \phi_S, d_{iL}\} = d_i - \phi_S &\implies \\
d_i - \phi_S \leq d_{iL} + 1 - \phi_L/w_L \leq d_i - \phi_L/w_L &\implies \phi_L/w_L \leq \phi_S
\end{aligned}$$

which is a contradiction. □

(AOI, sufficiency) First note that by $\kappa_S = 1$ and $\overline{e}_S > \phi_S$, small firms want to have as many links as possible. By Theorem 2, small firms have no profitable deviations among themselves. By Claim 1, they do not deviate to forming a link with the large firm either. So the network is SS. As for Pareto efficiency, by Claim 1, a small firm has strictly less payoff in any other network in which it has a link with the large firm. Thus the network is Pareto efficient.

(AOI, necessity) By Claim 1, small firms who have links to the large firm do not achieve $V(d_i)$ by the lemma above. If there are more than $\overline{e}_S$ of them, $\overline{e}_S + 1$ of them they can cut their links to all others, and form a clique of their own, and achieve $V(\overline{e}_S)$. So the only candidate for SS networks are those in which the large firm does not have more than $\overline{e}_S$ small counterparties.

Denote S_L the counterparties of L in the candidate network E. Given that the large firm has at most $\overline{e}_S$ counterparties, $\overline{e}_S$ and $\overline{e}_S - w_L$ are even integers, and $n \geq 2(\overline{e}_S + 1)$, Erdős-Gallai Theorem implies that there exists a network in which L is counterparties with S_L, members of S_L have $\overline{e}_S - w_L$ small

counterparties, and all other small firms have $\overline{e}_S$ small counterparties. This means that if E does not give all small firms exposure $\overline{e}_S$, it can me Pareto improved. So the only candidates for PESS networks are the ones described in the result.

(POI, necessity and sufficiency) Since $(1-\alpha)\kappa_L < 1$, the large firm wants to have an unbounded number of small counterparties. In POI, this is the unique Pareto efficient network. By Theorem 1, it is GS, so also SS.

Proof of Proposition 5

Step 1. Since small firms have ex-post payoff function $d - f$, they do not transmit contagion. Also, they want to have as much exposure as possible: $\overline{e}_S$. Since $\sigma\alpha_S + \sigma'\alpha'_S > 1 - \frac{1}{\kappa_L}$, the large firm's expected payoff is strictly increasing in its degree.

Step 2. Take a small firm i. Denote ω_i the event at which the state is high, the large firm has a good shock, and i has a good shock. Denote ω'_i the event at which the state is low, the large firm has a good shock, and i has a good shock. Denote d_L the degree of the large firm. Denote $d_{iL} \in \{0,1\}$ the number of large counterparties of i. Denote $g_{iL} \in \{0,1\}$ the number of continuing large counterparties of i. Then a small firm i's expected payoff is given by

$$\begin{aligned}
&\alpha_L\sigma\alpha_S\left(w_L\mathbb{E}[g_{iL}|\omega_i] + (e_i - d_{iL}w_L)\alpha_S\right)\\
&+ \alpha_L\sigma'\alpha'_S\left(w_L\mathbb{E}[g_{iL}|\omega'_i] + (e_i - d_{iL}w_L)\alpha'_S\right)\\
&+ (1-\alpha_L)\,\sigma\alpha_S^2(e_i - d_{iL}w_L)\\
&+ (1-\alpha_L)\,\sigma'\alpha_S'^2(e_i - d_{iL}w_L).
\end{aligned}$$

L defaults if its threshold is exceeded. Denote

$$\tilde{\mathbb{B}}(d_L) := \mathbb{B}\left[d_L - 1, \frac{d_L - \phi_L/w_L}{\kappa_L}, 1-\alpha_S\right]$$
$$\tilde{\mathbb{B}}'(d_L) := \mathbb{B}\left[d_L - 1, \frac{d_L - \phi_L/w_L}{\kappa_L}, 1-\alpha'_S\right]$$

If $d_{iL} = 1$, $\mathbb{E}[g_{iL}|\omega_i] = \tilde{\mathbb{B}}(d_L)$ and $\mathbb{E}[g_{iL}|\omega'_i] = \tilde{\mathbb{B}}'(d_L)$. If $d_{iL} = 0$, $\mathbb{E}[g_{iL}|\omega_i] = \mathbb{E}[g_{iL}|\omega'_i] = 0$. Then, fixing e_i, the different in i's expected payoff between

being counterparties with the large firm and not is given by

$$w_L \left[\alpha_L \sigma \alpha_S \tilde{\mathbb{B}}(d_L) + \alpha_L \sigma' \alpha'_S \tilde{\mathbb{B}}'(d_L) - (\sigma \alpha_S^2 + \sigma' \alpha_S'^2)\right]$$

Define

$$\overline{d}_L := \sup \left\{ d : \sigma \alpha_S \tilde{\mathbb{B}}(d) + \sigma' \alpha'_S \tilde{\mathbb{B}}'(d) \geq \frac{\sigma \alpha_S^2 + \sigma' \alpha_S'^2}{\alpha_L} \right\}$$

Note that $\tilde{\mathbb{B}}(1) = \tilde{\mathbb{B}}'(1) = 1$. Then by $\alpha_L \geq \frac{\sigma \alpha_S^2 + \sigma' \alpha_S'^2}{\sigma \alpha_S + \sigma' \alpha'_S}$ we have $\overline{d}_L \geq 1$. Recall that we are given $\sigma \alpha_S + \sigma' \alpha'_S > 1 - \frac{1}{\kappa_L} > \alpha'_S$. Then $\alpha_S > 1 - \frac{1}{\kappa_L} > \alpha'_S$. Therefore, $\lim_{d \to \infty} \tilde{\mathbb{B}}(d) = 1$ and $\lim_{d \to \infty} \tilde{\mathbb{B}}'(d) = 0$. Then, if $\sigma \alpha_S \alpha_L > \sigma \alpha_S^2 + \sigma' \alpha_S'^2$, we have $\overline{d}_L = \infty$. Otherwise, $\overline{d}_L$ is bounded.

(Sufficiency)

Denote S the small firms and S_L the counterparties of L.

Step 3. In the candidate network, large firm has $\overline{d}_L^*$ counterparties. Since small firms do not pose SCR, this is the highest payoff for the large firm conditional on having $\overline{d}_L^*$ or less links. It can not be persuaded into any deviation in which it ends up having $\overline{d}_L^*$ or less links.

Step 4. Consider $i \in S_L$. If it cuts the link with the large firm, it can get at most $V_i(\overline{e}_S)$, which is less than what it is getting. So it can not be persuaded into cutting its link with L.

Given that all of S_L will maintain their links with L in any profitable deviation, there is no profitable deviation that reduces i's SCR. Then the only possible profitable deviation is with $S \backslash S_L$. But i already has $\overline{e}_S - w_L$ many small counterparties.

Step 5. Given Steps 3 and 4, the only deviation that are potentially profitable for firms in $S \backslash S_L$, which must include adding a link with L. But since L has $\overline{d}_L^*$ counterparties, small firms get less than $V_i(\overline{e}_S)$ if they form a link with L.

Step 6. Note that this network is also Pareto Efficient by the similar arguments. To maintain L's payoff, it must keep all links. To maintain S_L's payoffs, links with L must stay. If $S \backslash S_L$ form links with L, this reduces their own payoff (and S_L's) payoff.

(Necessity)

Step 7. If L has less than $\overline{d}_L^*$ links, a group of small firms in $S\backslash S_L$ will form links with L to strictly improve their own and L's payoffs. So in any SS network, L can not have less than $\overline{d}_L^*$ counterparties.

Step 8. If L has more than $\max\{\overline{d}_L^*, \overline{e}_S\} \geq \overline{d}_L^*$ counterparties, then members of S_L get less than $V(\overline{e}_S)$. Then since $\max\{\overline{d}_L^*, \overline{e}_S\} \geq \overline{e}_S$, a subset of S_L with $\overline{e}_S + 1$ many firms deviate to form a disjoint clique and improve their payoff. So in any SS network, L can not have more than $\max\{\overline{d}_L^*, \overline{e}_S\}$ counterparties.

Step 9. Given that the large firm has at most $\overline{e}_S$ counterparties, $\overline{e}_S$ and $\overline{e}_S - w_L$ are even integers, and $n \geq 2(\overline{e}_S + 1)$, by Erdős-Gallai Theorem, Pareto efficiency requires that all small firms have $\overline{e}_S$ exposure.

Proof of Proposition 6

By $\alpha_L < \frac{\sigma\alpha_S^2 + \sigma'\alpha_S'^2}{\sigma\alpha_S}$, $\overline{d}_L$ is bounded and the bound is independent of n. Then as n grows, in AOI, average welfare is governed by the small firms who do not have links with the large firm. They are formed into cliques of order $\overline{e}_S + 1$. Then in the limit, by SLLN, average welfare is almost surely given by the random variable

$$\begin{aligned} X_1 &= \sigma \circ \left[\frac{\mathbb{E}_{b_h}\left[(\overline{e}_S + 1 - b_h)(\overline{e}_S - \eta b_h)\right]}{\overline{e}_S + 1}\theta_G\right] \\ &\quad \oplus \sigma' \circ \left[\frac{\mathbb{E}_b\left[(\overline{e}_S + 1 - b_h)(e_S - \eta b_h)\right]}{\overline{e}_S + 1}\theta_G\right] \\ &= \sigma \circ [\underbrace{\alpha_S(1 - \eta(1 - \alpha_S))}_{=:M_1}\overline{e}_S\theta_S] \oplus \sigma' \circ [\underbrace{\alpha_S'(1 - \eta(1 - \alpha_S'))}_{=:M_1'}\overline{e}_S\theta_S] \implies \end{aligned}$$

$$\frac{X_1}{\overline{e}_S\theta_S} = \sigma \circ [M_1] \oplus \sigma' \circ [M_1']$$

Note that $M_1 > M_1'$. In POI, by SLLN, average welfare is almost surely given by the random variable X_2 where

$$\begin{aligned} \frac{X_2}{\overline{e}_S\theta_G} &= (1 - \alpha_L)\sigma \circ \left[\alpha_S\left(1 - \eta\left((1 - \alpha_S)\frac{\overline{e}_S - w_L}{\overline{e}_S} + \frac{w_L}{\overline{e}_S}\right)\right)\right] \\ &\quad \oplus (1 - \alpha_L)\sigma' \circ \left[\alpha_S'\left(1 - \eta\left((1 - \alpha_S')\frac{\overline{e}_S - w_L}{\overline{e}_S} + \frac{w_L}{\overline{e}_S}\right)\right)\right] \\ &\quad \oplus \alpha_L\sigma \circ \left[\alpha_S\left(1 - \eta\left((1 - \alpha_S)\frac{\overline{e}_S - w_L}{\overline{e}_S}\right)\right) + (1 - \eta(1 - \alpha_S))\frac{w_L}{\overline{e}_S}\right] \end{aligned}$$

$$
\begin{aligned}
&\oplus \alpha_L\sigma \circ \left[\alpha'_S\left(1-\eta\left((1-\alpha'_S)\frac{\overline{e}_S-w_L}{\overline{e}_S}\right)\right)+(1-\eta(1-\alpha'_S))\,\frac{w_L}{\overline{e}_S}\right]\\
&=(1-\alpha_L)\sigma\circ\Big[\underbrace{\alpha_S\left(1-\eta\left((1-\alpha_S)\right)\right)}_{M_1}-\underbrace{\eta\alpha_S^2\frac{w_L}{\overline{e}_S}}_{M_2}\Big]\\
&\oplus (1-\alpha_L)\sigma'\circ\Big[\underbrace{\alpha'_S\left(1-\eta\left((1-\alpha'_S)\right)\right)}_{M'_1}-\underbrace{\eta\alpha_S'^2\frac{w_L}{\overline{e}_S}}_{M'_2}\Big]\\
&\oplus \alpha_L\sigma\circ\Big[\underbrace{\alpha_S\left(1-\eta\left((1-\alpha_S)\right)\right)}_{M_1}+\underbrace{\left(1-\eta(1-\alpha_S)^2\right)\frac{w_L}{\overline{e}_S}}_{M_3}\Big]\\
&\oplus \alpha_L\sigma'\circ\Big[\underbrace{\alpha'_S\left(1-\eta\left((1-\alpha'_S)\right)\right)}_{M'_1}+\underbrace{\left(1-\eta(1-\alpha'_S)^2\right)\frac{w_L}{\overline{e}_S}}_{M'_3}\Big].
\end{aligned}
$$

Then

$$
\begin{aligned}
&Var\left(\frac{X_2}{\overline{e}_S\theta_S}\right)-Var\left(\frac{X_1}{\overline{e}_S\theta_S}\right)\\
&=\sigma\alpha_L(1-\alpha_L)(M_2+M_3)^2+\sigma'\alpha_L(1-\alpha_L)(M'_2+M'_3)^2\\
&+\sigma\sigma'\left(M_1-M'_1+(\alpha_S-\alpha'_S)\eta(2\alpha_L-\alpha_S-\alpha'_S)\right)^2-\sigma\sigma'\left(M_1-M'_1\right)^2\\
&>\sigma\alpha_L(1-\alpha_L)(M_2+M_3)^2+\sigma'\alpha_L(1-\alpha_L)(M'_2+M'_3)^2\\
&>\alpha_L(1-\alpha_L)\left(1-\eta+2\eta(\sigma\alpha_S+\sigma'\alpha'_S)\right)^2\left(\frac{w_L}{\overline{e}_S}\right)^2>0
\end{aligned}
$$

Proof of Proposition 7

Q.E.D.

Proof of Proposition 8

Denote C, D, G, B the set of continuing, defaulting, good, bad firms, respectively. For any action profile, represented by C and D, welfare is

$$W=\sum_i\left(e_i-\eta_i c_i\right)\theta_i(1-a_i)=\sum_{i\in C}e_i\theta_i-\sum_{i\in C}\sum_{j\in D}\eta_i e_{ji}\theta_i$$

Denote an efficient action profile C^* and D^*. Denote resulting welfare welfare W^*.

(Large shocks) Consider $\overline{\theta}_B<-\overline{\theta}_G$. Suppose that $C^*\cap B\neq\emptyset$. Denote W' welfare under the action profile in which $C'=C^*\cap G$ continues and $D'=D^*\cup B$

defaults. Then

$$
\begin{aligned}
&W' - W^* \\
&= \sum_{i\in C'}(p_i+e_i)\theta_i - \sum_{i\in C'}\sum_{j\in D'}\eta_i e_{ji}\theta_i - \sum_{i\in C^*}(p_i+e_i)\theta_i + \sum_{i\in C^*}\sum_{j\in D^*}\eta_i e_{ji}\theta_i \\
&= -\sum_{i\in C^*\cap B}(p_i+e_i)\theta_i - \sum_{i\in C^*\cap G}\sum_{j\in C^*\cap B}\eta_i e_{ji}\theta_i + \sum_{i\in C^*\cap B}\sum_{j\in D^*}\eta_i e_{ji}\theta_i \\
&= -\sum_{i\in C^*\cap B}(p_i+e_i)\theta_{Bi} - \sum_{i\in C^*\cap G}\sum_{j\in C^*\cap B}\eta_i e_{ji}\theta_{Gi} + \sum_{i\in C^*\cap B}\sum_{j\in D^*}\eta_i e_{ji}\theta_{Bi} \\
&= -\sum_{i\in C^*\cap B}(p_i+e_i)\theta_{Bi} - \sum_{i\in C^*\cap B}\sum_{j\in C^*\cap G}\eta_j e_{ij}\theta_{Gj} + \sum_{i\in C^*\cap B}\sum_{j\in D^*}\eta_i e_{ji}\theta_{Bi} \\
&= \sum_{i\in C^*\cap B}\theta_{Bi}\Big(-(p_i+e_i) - \sum_{j\in C^*\cap G}\eta_j e_{ij}\frac{\theta_{Gj}}{\theta_{Bi}} + \sum_{j\in D^*}\eta_i e_{ji}\Big) \\
&= \sum_{i\in C^*\cap B}\theta_{Bi}\Big(-p_i - \sum_{j} e_{ji} - \sum_{j\in C^*\cap G}\eta_j e_{ij}\frac{\theta_{Gj}}{\theta_{Bi}} + \sum_{j\in D^*}\eta_i e_{ji}\Big) \\
&= \sum_{i\in C^*\cap B}\theta_{Bi}\Big(-p_i - \sum_{j\in C^*\cap B} e_{ji} - \sum_{j\in C^*\cap G}\Big(e_{ji} + \eta_j e_{ij}\frac{\theta_{Gj}}{\theta_{Bi}}\Big) + \sum_{j\in D^*}(\eta_i - 1)e_{ji}\Big) \\
&= \sum_{i\in C^*\cap B}|\theta_{Bi}|\Big(p_i + \sum_{j\in C^*\cap B} e_{ji} + \sum_{j\in C^*\cap G}\Big(e_{ji} - \eta_j e_{ij}\frac{\theta_{Gj}}{|\theta_{Bi}|}\Big) + \sum_{j\in D^*}(1-\eta_i)e_{ji}\Big) \\
&= \sum_{i\in C^*\cap B}|\theta_{Bi}|\Big(p_i + \sum_{j\in C^*\cap B} e_{ji} + \sum_{j\in C^*\cap G}\Big(e_{ji} - \eta_j e_{ij}\frac{\theta_{Gj}}{|\theta_{Bi}|}\Big) + \sum_{j\in D^*}(1-\eta_i)e_{ji}\Big)
\end{aligned}
$$

which is strictly positive.[39] Contradiction. Thus $C^* \subset G$. Then

$$W^* = \theta_G \sum_{i\in C^*}(e_i - \eta_i c_i)$$

which is strictly increasing in C with respect to set inclusion as long as $C^* \subset G$. Thus $C^* = G$.

This can be implemented by making good firms that are facing default due to bad counterparties in the first round of contagion indifferent between defaulting and continuing. Contagion stops in the first round and there are no further transfers needed. Moreover, any transfer less than this can not make all good firms continue.

[39] This is strict unless there is only one firm in $C^* \cap B$ and it has no counterparties and its private project has size 0. But we can ignore this case because the minimal transfer restriction ensures that a singleton is not saved.

Therefore, bad firms receive no transfer. Good firms who are not pushed to default in the first round also do not receive any transfers. A good firm that would default in the first round then receives $(p_i r_i + e_i r_i + z_i) - \theta_{Gi}(p_i + e_i - \eta_i b_i)$.

(Small shocks) Next consider $\underline{\theta}_B > 0$. Then

$$W^* = \sum_{i \in C^*} (e_i - \eta_i c_i)\, \theta_i$$

is already strictly increasing in $C^* \subset N$ with respect to set inclusion. Then $C^* = N$. The smallest transfer to implement this is to make bad firms indifferent between defaulting or continuing. Since other bad firms will also be saved, there is no need for to compensate a bad firm for the counterparty losses. Then each bad firm i receives $(p_i r_i + e_i r_i + z_i) - \theta_{Bi}(p_i + e_i)$ to induce them to continue.