Contagion of Liquidation on Asset-Trader Network

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Abstract

This paper studies the contagion of bankruptcy through downward price pressure among investors with overlapping portfolios. I calculate the probability of an extensive contagion and the expected bankruptcy rate during such a contagion. System-wide contagion happens only when the diversification of portfolios is in a certain range and. The system exhibits a robust-yet-fragile tendency: when portfolios are relatively well diversified, the probability of a crisis may be small, but the spread of contagion can be extremely extensive, if it happens. This extreme consequence is related to the gradual erosion to investors by multiple rounds of downward price impact, which implies the importance of early government interventions after the initial outbreak of a contagion.

Keywords: Financial stability, contagion, liquidation, network, overlapping portfolios, generating functions

1 Introduction

When distressed financial institutions face bankruptcy, margin calls or regulatory constraints, they may have to sell their assets quickly. Large volume of sales in short period of time may depress the price. This downward price pressure can adversely affect other shareholders, cause more bankruptcy and induce further round of liquidations in other asset markets. In modern financial systems, institutions are interlinked by overlapping portfolios across assets, and the liquidation and rapid price declines may become contagious and spread extensively across markets, as demonstrated by the recent crises. In the US financial crisis 2008, when the housing bubble burst and housing prices declined, home buyers started to default. Various

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mortgage-backed securities held by institutional and private investors also became essentially worthless. These distressed investors, such as Bear Stearns, were forced to liquidate other assets in their portfolios. The Dow Jones Industrial Average declined by 18% as of October 10, 2008. The price effects were felt almost instantly by Europe. In the same week, The FTSE100 declined by 20%. In other financial crises, such as the 1987 stock markets crash, this knock-on price effect also played an important role in transmitting the crisis.

Modern financial systems have become much more complex and interconnected over the past decades. This increasing complexity of interdependence makes the system more susceptible to systemic collapse, while less transparent for investors and policy makers to assess the consequences of contagion. The complexity and fragility of the financial system have fostered a fast growing literature that studies the contagion over financial systems using techniques developed in physics and biology. This literature models the interconnected agents and institutions as networks. Most existing literature on financial networks focuses on the contagion through the channel of direct credit exposures/interbank loans, and very few treat asset prices and the mark-to-market effect as the primary cause of contagion.

In this paper we explore how a shock to an individual investor can give rise to systemic crises through the contagion of liquidation and price effects, and examine how greater complexity affects the probability and extent of contagion and the declines in asset prices. We construct a theoretical model with techniques introduced from the literature on complex networks, and identify a non-monotonic relationship between portfolio diversification and probability of contagion. We show that there exist "tipping points" where a small change in parameters can make significant difference in the extent of contagion. The theoretical results in our model show that the "robust-yet-fragile" phenomenon in Gai and Kapadia (2010) is caused by persistent, multiple rounds of impacts to investors rather than the first wave alone and therefore highlight the importance of early government interventions.

The most well know study of contagion on a simple financial network is Allen and Gale (2000). With four banks with interbank loans, they show that spread of contagion depends crucially on the pattern of interconnectedness between banks - when banks only have exposures to a few others, the counterparty risk is not well diversified and the system is more vulnerable; when every bank has exposures to all other banks, the risk is diversified and contagion is

less likely. Although the insights from simple network structures are seminal, its generality to the real world financial systems is doubtful. As indicated by Cifuentes et al. (2005), in a more complicated network, there is a non-monotonic relationship between connectedness and financial stability: the contagion is small when there are either a few links or when there are many, but is large when the network are moderately connected.

Gai and Kapadia (2010) study more complex networks, and banks linked by interbank loans are modeled as random graph-based networks[1], which can accommodate arbitrary and complex networks. They introduce the techniques of generating function from the literature on complex networks (Strogatz (2001)) and derive a condition for *phase transition*. The phase transition is a threshold, above which a system-wide contagion becomes possible. In addition to the non-monotonicity between network complexity and probability of contagion, they also find a robust-yet-fragile tendency of the financial networks: when portfolios are relatively well diversified, the probability of contagion may be low, but once it happens, it can be extremely widespread. While both Cifuentes et al. (2005) and Gai and Kapadia (2010) incorporate the asset price effects, there is only one generic asset in their models, and contagion still spreads through credit channel per se - without default, there will be no contagion.

In what follows, we investigates the contagion of forced liquidations through the asset prices and mark-to-market effects. In particular, the financial system is modeled as a bipartite random network in which both assets and traders are explicitly represented by different groups of nodes. We call this type of networks the asset-trader network. A simple asset-trader network is shown in Figure 1. This is in contrast to most previous models on financial networks, where the networks only involve agents/banks. This representation allows us to clearly identify the group of shareholders of an asset and the group of assets in an investor's portfolio, respectively. The overlapping of shareholders between two assets, and the overlapping of portfolios of two investors become visual. We can also calculate the price of an asset as a function of the number of shareholders, and evaluate the market-wise price declines in an extensive contagion. To highlight the price effects, I abstract away the direct credit exposures and study a network of pure asset portfolio linkages.

[1]A random network is a graph generated by some random process. In particular, the number of links each node has is determined by a given probability distribution, and who is connected to who is also determined by the random process that implement this distribution.

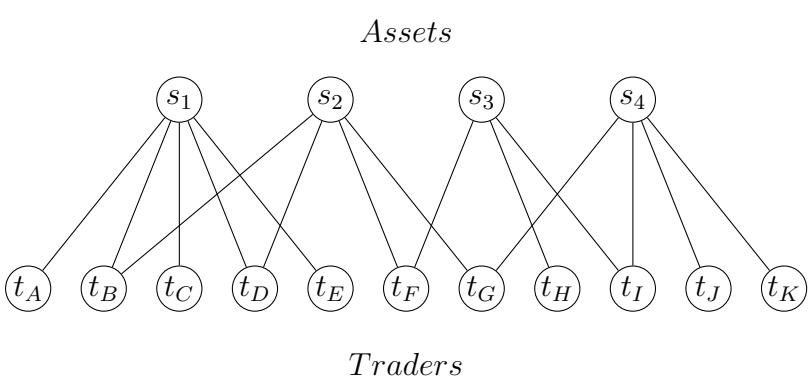


Figure 1: An assets-traders network

Unlike the generic contagion models like Watts (2002), we explicitly define the balance sheets of investors and specify the solvency condition for investors. Following Gai and Kapadia (2010), I use the techniques of generating functions to derive the condition under which the contagion of liquidation will become widespread. While Gai and Kapadia (2010) conjecture a non-monotonic relationship between the extent of contagion and network connectedness from their analytical results, they do not derive an explicit solution for the extent. We use the method introduced in Newman et al. (2001) and explicitly characterize the size of the contagion and show a non-monotonic relationship between this size and the network connectivity. As the average portfolios become more diversified so that the network become better connected, the size of the contagion first increases and then decreases. The results from simulations confirm this non-monotonic relationship and also show a robust-yet-fragile phenomenon similar to that in Gai and Kapadia (2010). With our analytical results we find that the reason underlying this phenomenon is the existence of closed loops in the network and investors subject to multiple rounds of impact of liquidation. From a policy perspective, this implies that early government interventions and bailouts may be crucial in containing the crisis before it cascades system-wide.

I then discuss the effects of front running (predatory trading, see Brunnermeier and Pedersen (2005a) and Carlin et al. (2007a)) on the contagion of liquidation. Based on a simple model, the simulation results suggest that with front running, both the probability of contagion and the extent of the contagion are larger than the benchmark model.

Besides Allen and Gale (2000), Cifuentes et al. (2005) and Gai and Kapadia (2010), our paper is related to a growing literature that studies the contagion of credit risks on banking networks. May and Arinaminpathy (2010) also model the complex banking network with

banks holding several different assets and they use mean-field approximations to provide intuitive explanations. Their assets do not play a crucial role in the contagion and they do not characterize the extent of the contagion analytically. Gleeson et al. (2011) provide a mean-field approximation method by which we can numerically calculate the extent of contagion without Monte Carlo simulations. Their results are fairly accurate compared with simulations and their methods are complementary to ours. Geertsema (2014) constructs a model of fire sale of assets in overlapping portfolios and uses an approximation method to calculate the final equilibrium price after the downward price spirals. Though not explicitly represented as a network, his model is close to ours but he focus on equilibrium price while we focus on the characterization of the number of failed investors.

The linkages and portfolios in our model form randomly and exogenously, and we do not address the issue of endogenous network formation in equilibrium or the design of optimal network topology[2]. Given that there is very limited empirical evidence on the shareholding structures of stock markets and the lack of established theory of optimal portfolio design against contagion of liquidation via price effects, it does not seem necessary to restrict our attention to specific network structures. In addition, our network structures are entirely random and thus accommodate all possible networks. This implies that the results of our model are compatible with all endogenously formed or optimally designed networks.

The rest of the paper are organized as follows: in Section 2.1 we introduce the model and the contagion process. In Section 2.2 we explain how to characterize the contagion process by using the techniques of generating functions. Section 2.4 and 2.3 give the analytical results of the extent of contagion above and below phase transition, respectively, and explain why the extent of contagion is non-monotonic in network connectivity. In Section 2.5 we calculate the probability of contagion. In Section 3 we simulate in the probability and extent of contagion, as well as the asset price declines. We compare the simulated results with analytical results and explain why there is the robust-yet-fragile phenomenon. In Section 4, I discuss the effects of the front running on contagion. A final section concludes.

[2]See Allen and Babus (2008) for a survey on this topic. Leitner (2005) studies a model where agents form a network and linkages can cascade liquidations but also serve as an insurance, because agents may be willing to bail out other agents to prevent the collapse of the whole network. Acemoglu et al. (2014) study a model with exogenous network structures but endogenous investment level on an underlying asset. They characterize the equilibrium and socially optimal investment levels as a function of the structure of the network

2 The Model

2.1 Setting

The complex financial system is represented by a network with S assets and T traders. Each trader is linked to a random number of assets, meaning those assets are in the portfolio of this trader. Each asset is linked to a random number of traders, who are its shareholders. These assets and traders are, in the language of graph theory, *nodes* in the network, and the exposure of a trader to an asset is the *link* in between them. Links are undirected. Since we are primarily interested in financial impact through mark-to-market price effect, we ignore the direct credit exposure between traders and there is no direct linkage between any two traders. We do not need any direct linkage between any two assets. See Figure 1 for a simple example of asset-trader network.

In network literature, a network as described above is called bipartite network. The number of links of a node is called its *degree*. In our asset-trader network, an asset s's degree, denoted d_s, is the number of shareholders of this asset, and a trader t's degree, denoted d_t, is the number of assets in her portfolio. The degrees of assets and traders are random numbers and are governed by their respective degree distributions. The network here is hence a random network and include all possible realizations of networks under the the two degree distributions. The two distributions are arbitrary, though we will assume specific distributions in the numerical simulations.

All assets in the network are illiquid and traders in the network are large shareholders, in the sense that each trader's liquidation can affect the price and bring down the asset price. In a reduced form, the price of an asset s is a strictly increasing function of x_s:

$$\rho(x_s) \tag{1}$$

where x_s is the fraction of shares held by traders in the network. When a trader in the network liquidates, her shares are absorbed by behavioral passive investors outside the network, hence x_s drops. These behavioral investors outside the network are not in our primary interest, and they only passively absorb in-network liquidation[3]. So $\rho(\cdot)$ captures the selling pressure which drives the asset price down. In the rest of this paper, we will only discuss traders in the network.

[3]The behavioral investors outside the network can be interpreted as follows: for the long-term fundamental

To focus on contagion triggered by asset liquidation, traders do not actively or strategically change their asset positions during the process. They change their positions only when asset prices decline and their capital buffer is exhausted, so that they are forced to liquidate. Short sale is not allowed.

Traders are leveraged and when the asset price drops significantly they will become insolvent and be forced to liquidate. A trader t's total asset, A_t, is the market value of her portfolio in the network. Her total liability is her exogenous debt D_t, plus capital K_t. When asset prices change, A_t changes accordingly. The trader remains solvent when $A_t \geq D_t$. When K_t is exhausted by the declines in asset prices, i.e. $A_t < D_t$, the trader becomes insolvent. An insolvent shareholder has to exit the market by liquidating all the assets she holds and is not allowed to re-enter the market.

Let B_s denote the total supply of asset s in the number of shares, and $x_{s,t}$ the fraction of shares held by trader t, with $\sum_t x_{s,t} = x_s$.[4] Hence trader t holds $x_{s,t}B_s$ shares of asset s, which does not change unless she is forced to liquidate. When another shareholder t' liquidates her shares, the in-network fraction of shares drops by $x_{s,t'}$, and the price drops from $\rho(x_s)$ to $\rho(x_s - x_{s,t'})$, and trader t will suffer a mark-to-market loss $L_{s,t,t'} \equiv x_{s,t}B_s\left[\rho(x_s) - \rho(x_s - x_{s,t'})\right]$. Trader t will become insolvent upon the first liquidation of asset s made by trader t' if $L_{s,t,t'} > K_t$.

In what follows, we assume that the number of shares held in the network, x_sB_s, is evenly distributed over each link. So each shareholder of asset s has $\frac{x_sB_s}{d_s}$ shares, where d_s is the initial asset degree and x_s is the initial in-network fraction of shares before anyone liquidates. In addition we assume that the x_s and B_s are identical across assets and capital buffer are identical across traders[5]. Although these assumptions are stylized, they provide a benchmark to demonstrate that contagion can happen even when assets holding is diversified, and we can

investors to be willing to buy the liquidated asset, they must be compensated by lower prices. Or their behavior can be justified by an adverse selection problem. When traders have private information about the asset fundamental value and keep buying the asset, it is natural for the uninformed passive agent to respond by raising the price continuously, as in Kyle (1985). Similar behavioral asset supplies have also been adopted by De Long et al. (1990), where passive investors supply the asset at an increasing price when rational speculators are buying, and by Brunnermeier and Pedersen (2005b) and Carlin et al. (2007b), where long-term traders buy when strategic traders' liquidation pushes down the price and sell when strategic traders' buy-back pushes up the price.

[4]If trader t does not hold asset s, then $x_{s,t} = 0$.

[5]Alternatively, we can assume x_s, B_s and K_t random variables, so that assets and traders becomes heterogenous. But we will lose the tractability of the model and the analytical results will not be available.

analytically characterize the contagion and the relationship between contagion and network structures. In Section 2.6 we will discuss the implications of relaxing these assumptions.

Under the above assumptions, if we drop some of the subscripts, each shareholder of asset s has $\frac{xB}{d_s}$ shares, and a shareholder's mark-to-market loss upon the first liquidation of s (by another shareholder), $L(d_s)$, is

$$L(d_s) \equiv \frac{xB}{d_s}\left[\rho(x) - \rho(x - \frac{x}{d_s})\right] \tag{2}$$

Then the solvency condition is $L(d_s) \geq K$. Since $\rho(\cdot)$ is a strictly increasing function, $L(d_s)$ is strictly decreasing in d_s, and the equation $L(d_s) = K$ has exactly one solution and denote this unique solution d^*. If an asset's degree d_s is greater than or equal to d^*, we have $L(d_s) \leq K$, in which case if one of the shareholder is insolvent and liquidates, the price decline will be small and other shareholders will survive this first round of impact. If an asset's degree is smaller than d^*, we have $L(d_s) > K$. Then, if any of the shareholders liquidates, the mark-to-market loss for the rest of the shareholders will exceed their capital buffer, and all of them will go bankrupt and be forced to liquidate all their portfolios. In this sense, we define the vulnerability of an asset:

Definition 2.1. *An asset s is vulnerable if its degree d_s satisfies*

$$d_s < d^* \tag{3}$$

The links of a vulnerable asset are said to be *infectious* (excluding the incoming link that triggered the liquidations). We assume that $d^* \geq 2$. If we use a simple form of $\rho(\cdot)$: $\rho(x) = \gamma x$, where $\gamma > 0$ is a constant, then the price is linear in x. When a shareholder liquidates, the price drops from γx to $\gamma(x - \frac{x}{d_s})$, and the mark-to-market loss of each remaining shareholder is $\frac{xB}{d_s}\gamma\frac{x}{d_s}$. If this loss is greater than the capital buffer, i.e. $\frac{xB}{d_s}\gamma\frac{x}{d_s} > K$, then this asset is vulnerable. So $d^* = x\sqrt{\gamma BK}$ in this example, and the assumption that $d^* \geq 2$ requires that $x\sqrt{\gamma BK} \geq 2$. In Section 3 we will adopt this simple linear form of $\rho(\cdot)$ in simulations, and let us define $V \equiv x^2B\gamma$, and V is then the initial market value of the shares of an asset held in the network.

Define an indicator function $v(d_s)$,

$$v(d_s) = \begin{cases} 1 & \text{if } 2 \leq d_s < d^*; \\ 0 & \text{if } d_s < 2 \text{ or } d_s \geq d^*. \end{cases}$$

2.2 Contagion and Generating Functions

In this section we use the techniques of generating functions and calculate the size of contagion after a randomly chosen trader fails and liquidates. Let $\{p_i\}$, $i = 0, 1, 2, ...$ be the assets' degree distribution and $\{q_j\}$, $j = 0, 1, 2, ...$ the traders' degree distribution, which are exogenously given. Hence, randomly choose an asset s, the probability that s has i shareholders is p_i; randomly choose a trader t, the probability that t holds j assets is q_j. Let $\mu = \sum_i ip_i$ be the average asset degree, and $\nu = \sum_j jq_j$ be the average asset degree. Since every link connects a trader and an asset, the total number of links in the network calculated from assets and from traders must be the same, i.e. $S\mu = T\nu$.

Time τ is discrete. At $\tau = 0$, a randomly chosen trader is hit by a shock and is forced to liquidate all her portfolio. For the contagion of liquidation spreads to other traders, at least one asset being liquidated must be vulnerable, and when this vulnerable asset is liquidated by the initial chosen trader at $\tau = 0$, all shareholders of this asset will become insolvent and be forced to liquidate all their portfolios at $\tau = 1$.

The probability that a randomly chosen trader has degree j (and hence holds j assets) is q_j, and this probability distribution can be represented by a generating function

$$g_0(x) = \sum_{j=0}^{\infty} q_j x^j \tag{4}$$

The generating functions as such contain all the information of the degree distributions $\{q_j\}$, and are convenient when we want to generate the distribution of the number of assets or traders we can reach at $\tau = 2, 3....$ Appendix A lists some basic properties of the generating functions. Note that

$$g_0(1) = \sum_{j=0}^{\infty} q_j = 1$$

Then, following each link from the initial trader to an asset, we want to know how likely it is vulnerable and how many shareholders it has. We know that, randomly choose an asset, the probability that it has degree i is p_i. However, it is important to note that the degree distribution of an asset reached by following a link is not p_i, because the higher the degree an asset has, the more likely it will be reached by following a randomly chosen link. Therefore

degree distribution of an asset reached as such should be proportional to its degree, i.e. $\propto ip_i$[6]. After normalization, this degree distribution should be

$$P_i \equiv \frac{ip_i}{\sum_{i=0}^{\infty} ip_i} = \frac{ip_i}{\mu}$$

Note that P_0 is always zero because we will never reach a node without any link. Among the i links of a vulnerable asset, one of them is the link that we followed onto that asset, so there are only $i-1$ links that are infectious. Define

$$\begin{aligned} f_{1v}(x) \equiv & \quad \sum_{i=1}^{\infty} P_i v(i) x^{i-1} \\ = & \quad \sum_{2 \leq i < d^*} P_i v(i) x^{i-1} \end{aligned}$$

The second line comes from the fact that $v(i)$ equals 0 for $i = 1$ and $i \geq d^*$, and equals 1 otherwise. Hence, randomly choose a link and follow it to an asset s, the probability that s is vulnerable is $f_{1v}(1)$. But $f_{1v}(x)$ does not capture the the probability that s is safe[7], so we must manually add this probability. The probability that s is safe is $P_1 + \sum_{i \geq d^*} P_i = 1 - f_{1v}(1)$. Therefore the probability distribution of infectious links of an asset reached by following a randomly chosen link is generated by

$$f_1(x) = 1 - f_{1v}(1) + f_{1v}(x) \tag{5}$$

Follow each link of a vulnerable asset, we reach a trader t of degree j with a probability that is proportional to j. So the probability that trader t has $j-1$ outgoing links (excluding the incoming link) is

$$Q_j \equiv \frac{jq_j}{\sum_{j=1}^{\infty} jq_j} = \frac{jq_j}{\nu}$$

and the generating function for this probability is

$$g_1(x) = \sum_{j=1}^{\infty} Q_j x^{j-1} = \frac{g_0'(x)}{\nu} \tag{6}$$

Combine $g_1(\cdot)$ and $f_1(\cdot)$, if this trader t (reached by following a link) liquidates, we can calculate how many t's first-neighbors (traders) will be infected and forced to liquidate via the

[6]See Newman (2003) and Feld (1991) for detailed explanation.

[7]This can be seen from the fact that $f_{1v}(1) < 1$.

vulnerable assets they share with trader t). This probability distribution can be generated by

$$\begin{aligned} G_1(x) =& g_1(f_1(x)) \\ =& Q_1(f_1(x))^0 + Q_2(f_1(x))^1 + Q_3(f_1(x))^2 + ... \end{aligned} \tag{7}$$

The various possibilities contained in $G_1(x)$ is depicted in Figure 2. In Figure 2 it is assumed

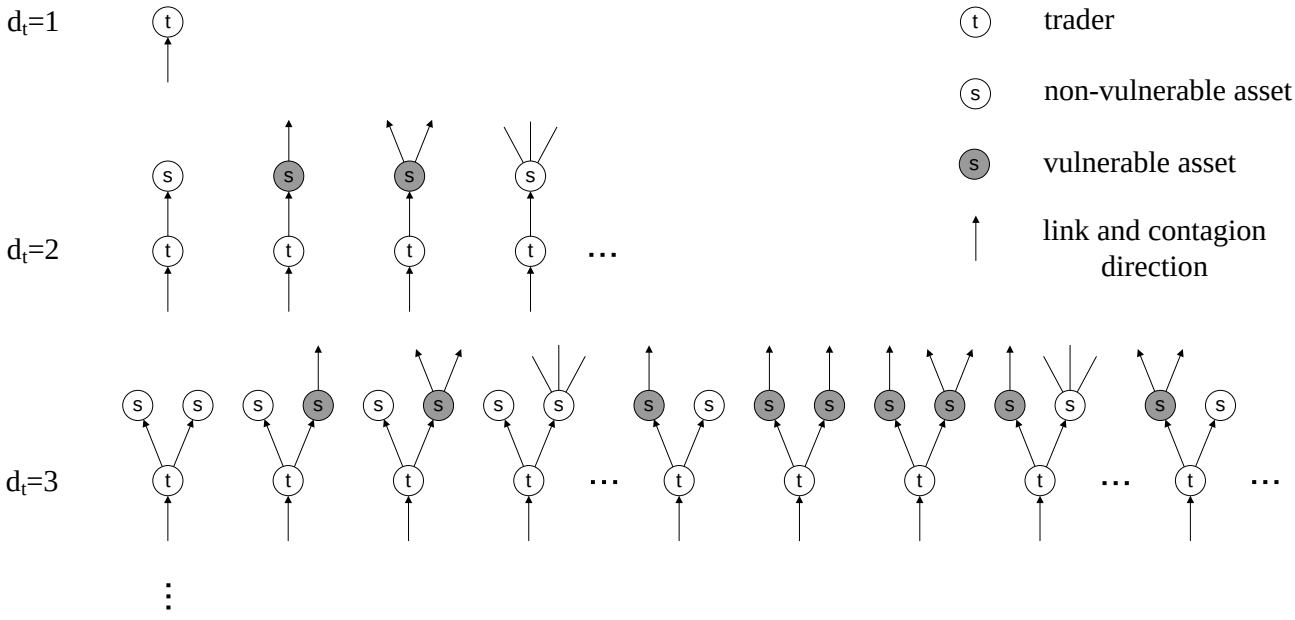


Figure 2: Schematic representation of the $G_1(x)$

that only assets of degree 2 and 3 are vulnerable. In the first row, this trader t (reached by following a link) has degree 1, which is just the link leading to her, and has no link leading out to another asset. In the second row trader t leads to exactly one asset (other than the asset leading onto t), and this asset leads to 0, 1, 2 ... traders (other than t), respectively. In the third row, the trader reached leads to exactly two other assets, the various combinations are depicted. There are of course other possibilities, which are omitted.

To better understand $G_1(x)$, recall that, $G_1(x)$ is a polynomial of x. Like all generating functions, the exponents of x in equation (7) indicates the number of infected neighbors, and the coefficients (after the polynomial expansion) attached to each of the x^i, $i = 0, 1, 2...$, are corresponding probabilities. Also in equation (7) we use the characteristic that if a generating function generates the probability distribution of some property of an object, then the sum of that property over i independent such objects is distributed according to the ith power of the generating function.

Each unit depicted in Figure 2, where trader t reached by following a link leads to a number of assets and then further reaches out a number of links (to other traders), is the smallest but complete episode in the contagion process. It is like a (biological) cell that is the smallest but fully functional unit that can replicate itself. In this sense, we call it the *contagion unit*, and $G_1(\cdot)$ describes the likelihood of different forms of contagion units we can reach by following a link.

At this time we assume that there is no closed loop or cycle in the network, so that each trader will be hit by the spread of liquidation at most once[8]. If the trader survives the first wave of liquidation, she survives till the contagion ends. If loops are allowed, traders will suffer multiple impacts and this will make the contagion more likely and more extensively. This restriction will be relaxed later and we will discuss its implications and compare the simulation results under both of the assumption of single hit (no loop) and multiple hits (loops allowed).

If the liquidation spreads beyond first-neighbors and to the second-neighbors, what happens to the second neighbors is exactly the same as depicted in Figure 2, and we can still use $G_1(\cdot)$ to describe various possibilities that ensue. For the second-neighbors that can be infected by trader t, if we start with a randomly chosen a link (of t) and follow it to t, the probability distribution of this number (plus first-neighbors) is

$$G_2(x) = G_1(xG_1(x))$$

The x between the two layers of G_1 accounts for the first-neighbors. Similarly, the probabilities of contagion to the third, fourth ... neighbors can be described by more layers of nesting of $G_1(\cdot)$.

Now if we follow a link to a trader t, and then to every trader that can be ultimately infected, directly and indirectly, we call this set of traders (connected by vulnerable assets)

[8]The use of probability generating functions requires a tree-like network, i.e. there cannot be any loop in the network. This assumption is not as strong as it looks. The generating function $H_1(x)$ aims to give (the distribution of) the total number traders that can be infected. If there are loops, nodes on the loop may be repeatedly counted. However we only need that there can not be loops between low degree (vulnerable) assets. If high degree assets have loops, nodes on the loop are unlikely to be counted at all, because the high degree nodes (safe assets) obstruct the infection. Notice that the higher the degree, the more likely that the node is in a loop, because it has more links. So most loops have high degree nodes involved, but we do not need to worry about them because they are insulated. The only thing we should worry is the loops consisted of purely low degree assets, though there are not many of them. This whole idea is reflected by the presence of term $v(i)$ in the generating functions.

the *vulnerable cluster*. Let $H_1(x)$ be the generating function of the probability distribution of the size (number of infected traders) of the vulnerable cluster reached by following a link to a trader. Then

$$H_1(x) = xG_1(xG_1(xG_1(\cdots xG_1(x)\cdots)))$$

Traders that share a vulnerable asset must be in the same vulnerable cluster. There can be many vulnerable cluster in the network, and vulnerable clusters are segregate by safe assets (or no link between them at all). The vulnerable cluster plays an important role in our analysis. If any of traders in the vulnerable cluster liquidates, all traders in the cluster will liquidate eventually. Therefore the size of vulnerable cluster essentially determines the contagion at the system level.

The tree-like vulnerable cluster we can reach starting from a link can take many different forms, which are illustrated in Figure 3. Each circle in Figure 3 is a contagion unit and each

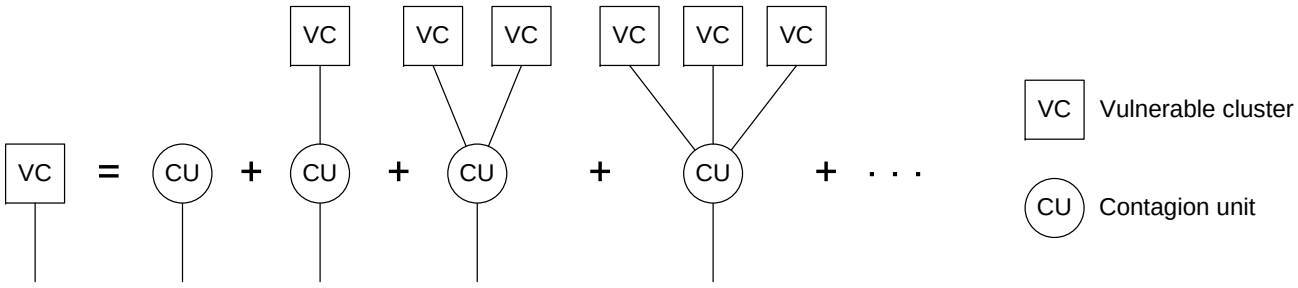


Figure 3: Schematic representation of the vulnerable cluster

square is a vulnerable cluster. On the right hand side, the initial contagion unit reached can emanate 0, 1, 2, 3, ... links, and each link leads to a vulnerable cluster, each of which can also take many different forms, just like the original vulnerable cluster on the left hand side. Each of the vulnerable clusters on the RHS has the same size distribution as the original vulnerable cluster on the LHS. Therefore $H_1(x)$ must satisfy the following self-consistency condition:[9]

$$H_1(x) = xG_1(H_1(x)) \tag{8}$$

Note that in this recursive form of H_1 all the traders infected en route are accounted for by the

[9]That $H_1(x)$ satisfy this recursive form of condition does not necessarily mean that the size of vulnerable cluster goes to infinity. It means that $H_1(x)$ includes all possible size of the cluster, including the infinite large one. See Newman et al. (2001).

x leading the right hand side[10].

The vulnerable cluster described in H_1 starts with a randomly chose link. But we are interested in a contagion that starts with a randomly chosen trader. If a randomly chosen trader is hit by exogenous shock and forced to liquidate all her assets, the distribution of the size of the vulnerable cluster (the total number of traders that can be ultimately infected starting from t, directly or indirectly, through vulnerable assets) is generated by

$$H_0(x) = xg_0(f_1(H_1(x))) \tag{9}$$

The leading x on right hand side accounts for the initial randomly chosen trader.

2.3 The Extent of Contagion Below The Phase Transition

Unfortunately, a closed form solution for $H_0(x)$ does not exist in general. However we can derive the average size of the vulnerable cluster from (8) and (9), which is enough for us to infer the stability of the system. We can calculate the expected extent of contagion $\langle s \rangle$ (in the number of infected traders) by

$$\langle s \rangle = H_0'(1)$$

Since $H_1(x)$ is a standard generating function and it includes all possibilities, all the coefficients in this polynomial sum up to 1, i.e. $H_1(1) = 1$. For the same reason, $f_1(1) = 1$ and $g_0(1) = 1$. From equation (6) we know $g_0'(1) = \nu$. So it follows from equation (9) that

$$\begin{aligned} H_0'(1) =& g_0(f_1(H_1(1))) + g_0'(f_1(H_1(1)))f_1'(H_1(1))H_1'(1) \\ =& 1 + \nu f_1'(1)H_1'(1) \end{aligned} \tag{10}$$

From equation (8) we have

$$H_1'(1) = \frac{1}{1 - G_1'(1)} \tag{11}$$

Substitute equation (11) into (10), we have

$$\langle s \rangle = 1 + \frac{\nu f_1'(1)}{1 - G_1'(1)} \tag{12}$$

[10]They are accounted for by the leading x in G_1 in (7). With this x, we are merely calculating the infected traders "on the farmost border"

From equation (5) we know that $f_1'(1)$ must be finite[11]. Then the expected size of contagion diverges when

$$G_1'(1) = 1 \tag{13}$$

which signifies the phase transition. Recall that $G_1'(1)$ is the average number of links reaching out of a contagion unit (excluding the incoming link). So if this number is smaller than 1, then contagion is expected to die out early. If it is larger than 1, then a giant vulnerable cluster emerges, which occupies a finite fraction of the trader population. In the latter case, if the initial trader hit by the exogenous shock is in the giant vulnerable cluster, then a finite fraction of the network will be infected.

The condition $G_1'(1) = 1$ can be rewritten as $g_1'(1)f_1'(1) = 1$, and then as

$$\sum_i \sum_j ij(i-1)(j-1)p_i q_j v(i) = \nu\mu \tag{14}$$

Suppose the network starts with very low average degrees, e.g. an empty network, and let ν and μ increase[12]. As the average degrees increase, more mass of the degree distributions moves to p_i and q_j with higher i and j. In particular, more mass of p_i moves into the vulnerable range where $v(i) = 1$, so more assets become vulnerable and connect to more traders. This could be a point where $G_1'(1) = 1$ and phase transition could happen. As the average degrees continue to increase, although the network becomes physically better connected, more mass of p_i moves out of the vulnerable range and the diversification comes to dominate. When more vulnerable assets become safe, the giant vulnerable cluster gets disconnected and isolated by these safe assets. This is the second phase transition that could arise, which marks the disappearance of the giant vulnerable cluster and $G_1'(1)$ drops from above to below one. The emergence of the second phase transition implies that the giant vulnerable cluster exists only in between the two transitions, i.e. when average degree is either very low or very high, there is no systemic contagion because there is no giant vulnerable cluster in the network, but when the average degree is in the intermediate range, the contagion will be extensive.

[11]Since there are only finite terms containing x in $f_1(x)$.
[12]Recall that $S\mu = T\nu$.

2.4 The Extent of Contagion Above The Phase Transition

The systemic contagion over the network can be better seen if we can calculate the average size of vulnerable cluster within the contagion window. Since the vulnerable cluster now occupies a finite fraction of traders in the network, its size is in scale with the size of the network. When $T \to \infty$, the size of the vulnerable cluster also diverges. We can calculate the size of vulnerable cluster as a fraction of the population, but the problem is that the probability of looping back to the giant vulnerable cluster can no longer be ignored, so we cannot treat it as a "tree" structure. As the "no-loop" assumption is not applicable, we cannot use the method in Section 2.3 to calculate the size of the giant vulnerable cluster. As Newman et al. (2001) shows, however, this issue can be circumvented by calculating the size distribution of components excluding the giant component, because the non-giant components can still be treated as tree-structures.

Within the contagion window, all traders must belong to either the giant vulnerable cluster or non-giant vulnerable clusters[13]. Randomly choose a trader, the distribution of the size of non-giant vulnerable cluster to which this trader belongs is generated by $H_0(x)$. Then the average fraction of traders that is in non-giant components must be $H_0(1)$, and the average fraction of traders that is in the giant component, $\langle s \rangle$, is

$$\langle s \rangle = 1 - H_0(1)$$

From equation (8) we know $H_0(1) = g_0(f_1(H_1(1)))$. Let $u = H_1(1)$, then from equation (9), $\langle s \rangle$ can be calculated by solving

$$\langle s \rangle = 1 - g_0(f_1(u)) \tag{15}$$

$$u = G_1(u) \tag{16}$$

Now we show that, as the average degree increases, the average size of the giant vulnerable cluster $\langle s \rangle$ first increases and then decreases.

When the network has just entered the contagion window and the average degree starts to increase from the lower bound of the window, it can be shown that $f_1(u)$ is increasing and

[13]Even a trader without any link at all, or a trader whose portfolio consists of safe assets only, can be regarded as a vulnerable cluster of size 1.

$g_0(f_1(u))$ is decreasing. From (16) we know

$$u = g_1(f_1(u)) \tag{17}$$

u is probability that a randomly chosen link leads to a trader that belongs to a non-giant component. $f_1(u)$ is the probability that a randomly chosen link leads to an asset that is not connected to the giant component, i.e. either this asset is safe or it is vulnerable but connected to a non-giant component. Equation (17) says that the probability that a trader (reached by following a randomly chosen link) belongs to a non-giant component, u, must equal the probability that all her assets, if any, are either safe or their contagious links only connect to non-giant components. If any of the contagious links of her assets connects to the giant component, then this case is excluded from (17).

When the average degree increases, more mass in degree distribution P_i moves to higher degree terms (higher i), and more assets become vulnerable. Since $u < 1$ in the contagion window, we know that the function $f_1(\cdot)$ shifts down ($f_1(x)$ becomes smaller for $\forall x, 0 < x < 1$). The intuition is that, since each link of a vulnerable asset has positive probability $(1 - u)$ to connect to (a trader that belongs to) the giant component, then when each vulnerable asset has more links, the chance that its holder is connected to the giant component is higher. Similarly, $g_1(\cdot)$ also shifts down, because each link of this trader has positive probability $(1 - f_1(u))$ to connect to the giant component, then the more assets she has, the higher the chance that she is connected to the giant component. Therefore, if u does not change, the downward shift of $f_1(\cdot)$ and $g_1(\cdot)$ makes a contagion unit less likely to connect to a non-giant component, i.e. $g_1(f_1(u)) < u$ now. Recall that, for non-giant vulnerable cluster, $G_1'(1) = g_1'(1)f_1'(1) < 1$ (even within the contagion window), otherwise this cannot be a non-giant vulnerable cluster. Since $u < 1$, then $G_1'(u) < G_1'(1) < 1$. Then for (17) to hold, u has to decrease. Then $f_1(u)$ must decrease as well when average degree increases.

Now let us check the response of $g_0(f_1(u))$ to changes in average degree. Similar to $g_1(\cdot)$, when average degree increases, more mass in degree distribution q_j moves to higher degree terms (higher j). Since $f_1(u) < 1$, we know that the function $g_0(\cdot)$ shifts down, i.e. it is less likely for a randomly chosen trader not to connect to the giant component. In addition, as shown above, each link of this trader is more likely to connect to the giant component, we

conclude that the fraction of traders that is not in the giant component is declining, i.e. the giant vulnerable cluster is growing disproportionately compared to the network.

As the average degree continues to increase, each asset tends to have more shareholders. More mass in distribution P_i moves out of the vulnerable range, and vulnerable assets turn into safe. $f_1(\cdot)$ shifts up. Eventually u and $f_1(u)$ start to increase. This means that, a randomly chosen link is now more likely to lead to an asset that is not connected to the giant vulnerable cluster. Although $g_0(\cdot)$ is still shifting down, i.e. a random chosen trader tends to have more and more links, but this is dominated by decreasing probability of each of her links to connect to the giant component. Therefore this trader is less likely to connect to the giant component. This shows that the giant vulnerable cluster is shrinking. As the average degree continues to increase and moves out of the contagion window, most of the assets become safe now. The giant component now disappears and there will be no systemic contagion.

The average size of giant vulnerable clusters above phase transition as a function of the average trader degree is shown in Figure 4[14]. These sizes are calculated from equation (15) and (16) and are measured as a fraction of trader population. The average sizes of vulnerable clusters below phase transition are shown in Figure 5. They are calculated from equation (12) and are measured in the number of traders. In Figure 5 the size of vulnerable cluster diverges when the average trader degree is between 0.708 and 8.22, which corresponds to the range in Figure 4 where the giant vulnerable cluster emerges and occupies a finite fraction of trader population. The lower and upper bound correspond to the two phase transitions where $G_1'(1) = 1$. In Section 3 we will examine whether these theoretical results are consistent with those from simulations.

2.5 The Probability of Systemic Contagion

The size of the giant vulnerable cluster closely relates to the probability of a systemic contagion, because, randomly pick trader t, if t is in the giant vulnerable cluster, then the probability of a systemic contagion is one, otherwise it is zero. So theoretically the size of the giant vulnerable cluster as a fraction of the network should coincide the probability of systemic contagion triggered by the liquidation of a random trader.

[14]Assuming Poisson degree distributions, $\rho(x) = x$ and $K/V = 0.01$

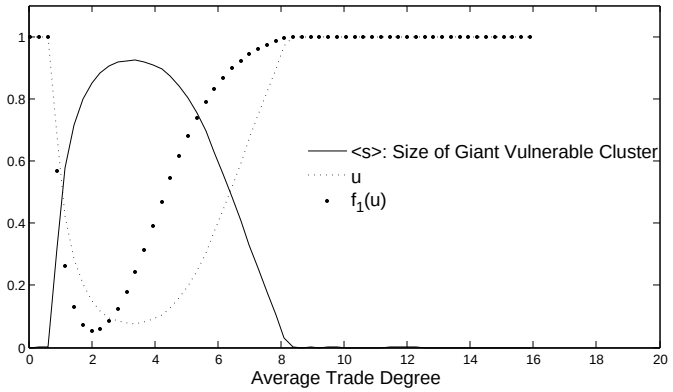


Figure 4: Average size of giant vulnerable cluster above phase transition (as a fraction of trader population)

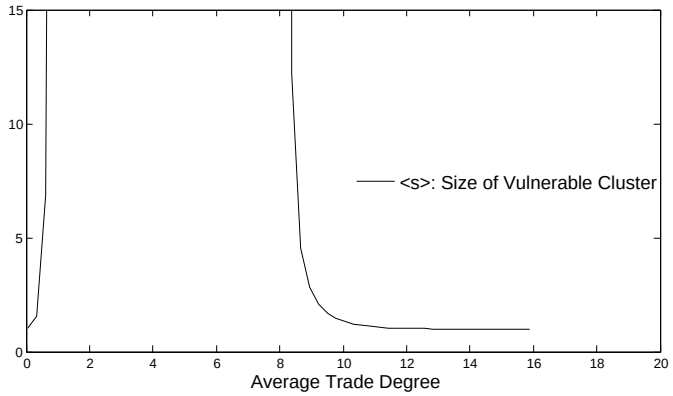


Figure 5: Average size of vulnerable cluster below phase transition (in number of traders)

But the numerical results (Figure 6) show that the size and the probability coincide only when the network connectivity is in the lower range of the contagion window. In the higher range of contagion window, the probability starts to drop whereas the size of the contagion (conditional on contagion breaking out and at least 5% traders infected) continues to increase and almost occupies the entire network. In the range where the extent and probability of contagion diverge, the financial networks exhibit a "robust-yet-fragile" tendency, as Gai and Kapadia (2010) put it, where the the portfolio diversification starts to reduce the probability of an extensive contagion, but given that a contagion already breaks out, there is a substantial portion of the population will be infected. The reason is that the methods of generating functions, including Newman's method, do not take into account of the issue of multiple hits of liquidations. We will discuss this issue in detail in Section 3.2.

In Section 3 we will simulate contagion with traders being immune if they survive their first impact of liquidations and examine whether the extent of contagion is consistent with the probability of contagion. Gleeson and Cahalane (2007), Gleeson (2008), Gleeson et al. (2011) develop a mean-field method to calculate the extent of contagion without simulations. Their methods allow multiple rounds of hits, and the results are quite consistent with those from simulations.

2.6 Implications of Relaxing Some Assumptions

In our model we assumed that the initial market value of each asset held in the network is independent of the degree of asset and the shares are evenly distributed over its shareholders. In reality it might be reasonable that the market value of an asset increases with its number of shareholders. Given a constant capital buffer, this would increase a trader's exposure to risk of liquidation and dilute the benefit from portfolio diversification. On the other hand, we might also expect that a trader's capital buffer to increase with the number of assets in her portfolio. Since we assumed a fixed ratio between total number of assets and total number traders in the network, the ratio between average asset degree, μ, and average trader degree, ν, does not change as μ and ν increase and the network gets better connected. Therefore, as long as the rate at which the initial in-network value of an asset increases with the asset degree is less than the rate at which the capital buffer increases with the trader degree, portfolio diversification

still benefits and our result will still hold qualitatively and equation (13) and (14) continue to have two solutions, though the contagion window might be wider. Conversely, if the former rate exceeds the latter, having more assets will be unambiguously riskier, and the better connected the network the more likely and extensive the contagion.

An uneven distribution of asset shares among shareholders will not change our results qualitatively, though we would expect that the contagion window to be wider because some of the safe assets may become vulnerable to those who hold more shares.

3 Numerical Simulations

3.1 Methodology

To verify our theoretical results, we simulate the contagion process numerically. We assume a random graph in which each possible link between a trader and a asset is present with independent and identical probability p (binomial distribution). This type of random graphs is called Poisson random graph[15]. The networks generated as such allow closed loops and thus accommodate all possible network structures. The Poisson random graph is chosen for simplicity.

We are interested in the expected extent and probability of contagion, and will examine networks with different connectivity. Even when loops are present, in simulations we still examine both the cases where multiple impacts of liquidation are allowed and not allowed. This is to verify that the divergence of extent and probability of contagion is caused by the multiple impacts that turn safe assets into vulnerable. When calculating the extent of contagion, we only count episodes with more than 5% of the traders infected, which is a reasonable signal that the initial exogenous shock has hit within the giant vulnerable cluster. Otherwise, we consider the initial shock missed the giant vulnerable cluster and the small contagion, if any, is irrelevant to the size of the giant vulnerable cluster.

We consider networks with 1000 assets and 2000 traders. The number of assets in a financial system depends on the what can be counted as major (value large enough) and illiquid assets. The number of traders also depends on the definition of major investors. The choice of 1000

[15]The networks with this degree distribution are generated by Configuration Model. See Jackson (2008), Section 4.1.4

assets and 2000 traders seems to be reasonable if we consider large investment banks, hedge funds, insurance companies, etc in the global financial system. Higher numbers of assets and traders in the simulations do not change our results qualitatively. [16]

For simplicity, I assume that the price is proportional to the fraction of the asset held in network, i.e. asset price equals x (we set $\gamma = 1$). Recall that we denoted the initial market value of the portion of an asset held in the network, $x_0^2 B$ (with $\gamma = 1$), as V, where x_0 is the initial fraction of shares held in the network. From equation (2) we know that when asset price is assumed to be proportional to the fraction of the asset held in network, the loss of a trader each time an asset she holds being liquidated by other traders is $B\frac{x_0^2}{d_s^2} = \frac{V}{d_s^2}$, where d_s is the initial degree of that asset. In the case where multiple impacts are allowed, if the accumulated loss of a trader has not exceeded her capital buffer K, then she is still solvent. In the case where multiple impacts are not allowed, the trader becomes immune after the first impact. We also examine the effect of capital buffer (and hence the leverage) on the contagions by varying the ratio K to V.

In the simulations I draw 500 realizations of network for each value of average trader degree ν. In each of these draws, I randomly choose a trader and force her to liquidate all her portfolio. Any neighboring trader whose (accumulated) mark-to-market loss is larger than K is assumed to be bankrupt and must also liquidate all their assets. This process iterates until no new trader fails.

3.2 Simulation Results

The simulation results of the model is shown in Figure 6. By varying the average degree of traders, ν, we have networks with different degree distributions. The average degree of a network is an indicator of how well a network is connected. When multiple liquidation impacts to a trader are allowed, we can see that, if the average degree is either very low or very high, system-wide contagion is not likely to happen. Whereas within a certain window where the average degree is moderate, an extensive contagion is more likely and the extent is non-monotonic in

[16] The use of generating functions requires an infinite network (infinite number of nodes). Watts (2002) shows that the infinite network can be well approximated by a network with 10,000 nodes, and Gai and Kapadia (2010) shows that the results from networks with 1,000 nodes and those from networks with 10,000 nodes agree quite well.

average degree. This confirms the intuition in Section 2.3 and 2.4.

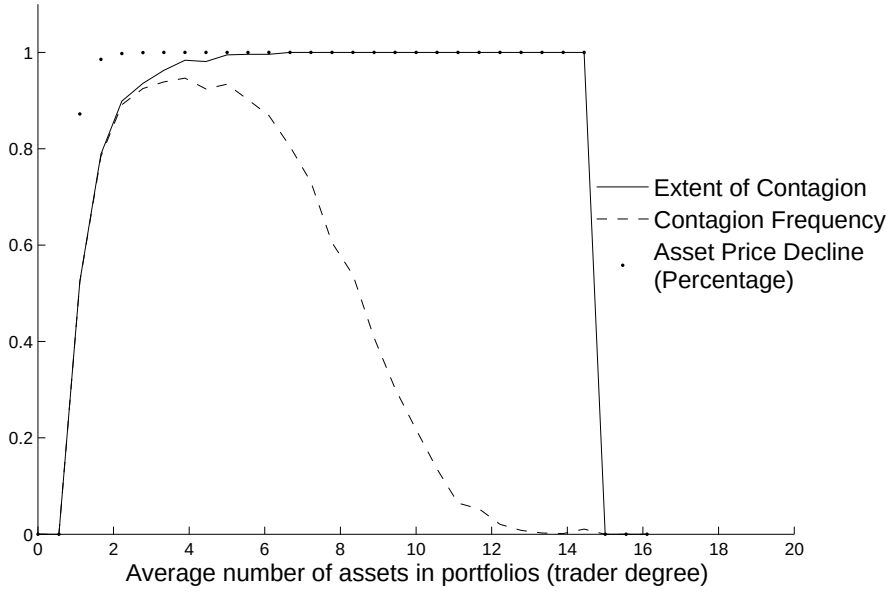


Figure 6: Contagion frequency, conditional extent of contagion and price decline ($K/V = 0.01$, multiple impacts allowed)

Figure 6 also shows the average percentage declines of asset prices conditional on there is a widespread contagion. In calculating the declines all assets are equally weighted. The percentage of price declines are roughly consistent with the extent of contagion, with price declining large than the extent at the lower end of the contagion window. This result can be viewed as what could happen to the stock market indices in a financial crisis, and it suggests that when investors are relatively well diversified, the collapse of asset prices could be significant during a financial crisis, and thus diversification has only limited effects on systemic risks.

In Figure 6 the extent of contagion (conditional on that it has infected more that 5% of the trader population), measured as a fraction of population, is approximately the same as the frequency of contagion at the range of lower average degrees within the window. But at the higher end of the window, though extensive contagion is rare, once it happens almost all traders will fail. Near the upper bond of the contagion window, the extent of the contagion suddenly drops to zero and the contagion disappears. So the financial system exhibits a robust-yet-fragile feature similar to Gai and Kapadia (2010).

If we compare the theoretical size of giant vulnerable cluster in Figure 4 with the simulated extent of contagion in Figure 6, we find that, in the upper range of the contagion window,

the theoretical size falls gradually, compared with the simulated extent of contagion which converges to one (when multiple impacts allowed). This is shown in Figure 7. The main difference between the theoretical model and the model used in previous simulations is whether closed loops are allowed in the network. This implies that the robust-yet-fragile tendency in the financial system is primarily caused by multiple impacts of liquidation to a trader, rather than by the first single impact alone. When the initial contagion starts out, it is very likely that the contagion dies out at early stages because well diversified investors are subject to only minor mark-to-market loss at the beginning, which corresponds to the low frequency. But once the contagion spreads further via vulnerable assets, when loops are present, it starts to backfire over the loops and a trader may be subject to multiple hits of liquidations, including simultaneous and sequential impacts on one or several assets she holds. As the wave of liquidation bounces back and forth on the intertwined financial network, the losses accumulate over time and finally can bring down well diversified investors.

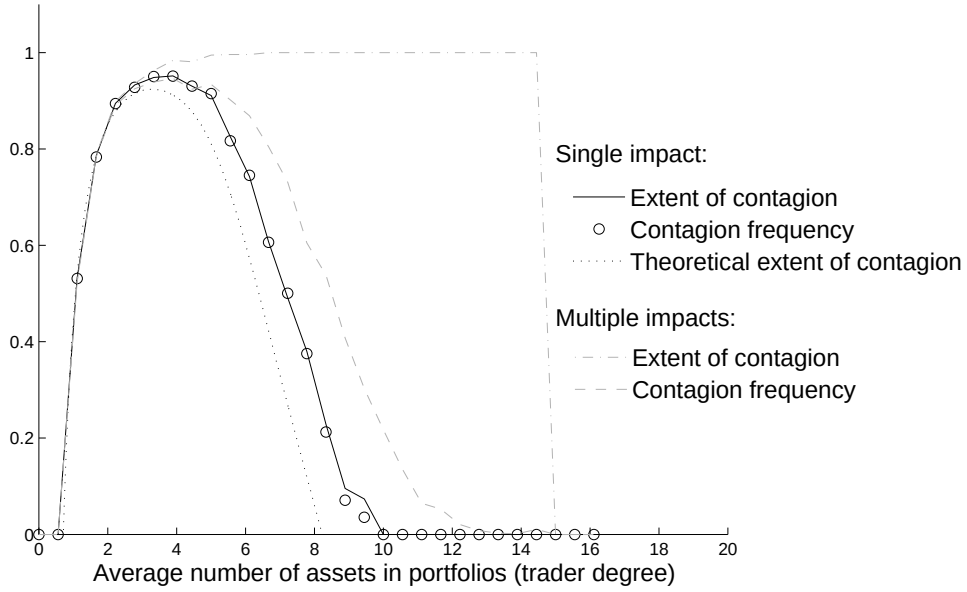


Figure 7: Contagion frequency, conditional extent of contagion and theoretical extent of contagion under single impact ($K/V = 0.01$, compared with multiple impacts)

Figure 7 also shows the simulated extent of contagion under the single impact assumption[17]. The extent of contagion in this case (the size of giant vulnerable cluster) is limited and gradually decreasing when the network gets better connected, which is now consistent with the probability of contagion. The divergence between them under multiple impacts assumption disappears and

[17]In simulations any further mark-to-market loss to a trader after her first impact is ignored.

the two curves are now almost indistinguishable. This confirms our earlier conjecture that multiple impacts are the reason that underlies robust-yet-fragile phenomenon.

The fact that in the higher range of the contagion window (where investors are relatively well diversified) the contagion is limited under the single impact assumption but is extremely extensive once it breaks out when multiple impacts are allowed suggests that most damages are done after the first wave of impacts. This implies that early government intervention can potentially be highly effective in restricting the liquidation in its initial neighborhood. After the initial liquidation breaks out, there could still be time and chances for the government to bail out those who survive but are weakened, before they experience further losses. This could prevent the contagion from spreading to the entire network.

Figure 7 also shows that the theoretical size of giant vulnerable cluster calculated from equation (15) and (16) is largely consistent to the results from simulated extent of contagion under the single impact assumption.

Figure 8 shows how changes in the capital buffer and average degree jointly affect the frequency of extensive contagion. When the capital buffer is high, system risks are low in all average degrees. When the capital buffer declines, probability of contagion reach its peak first within a small window at the low average degree range. As capital buffer drops further, this windows expands to high average degree range. This tells that increasing capital buffer can effectively decrease the probability of widespread contagion; if there is no restriction on the leverage ratios, even very well connected network faces huge system risks.

4 Discussion: Front Running and Contagion

In this section we examine the effect of predation on contagion by allowing some shareholders front running the distressed shareholders.

The front running, or predatory trading, is in line with Brunnermeier and Pedersen (2005a): when some traders are in distress and forced to liquidate illiquid assets, other shareholder who are aware of may take the advantage by selling before the distressed and then later buy back the asset to make a profit. Front running, though generally illegal, has long been suspected on

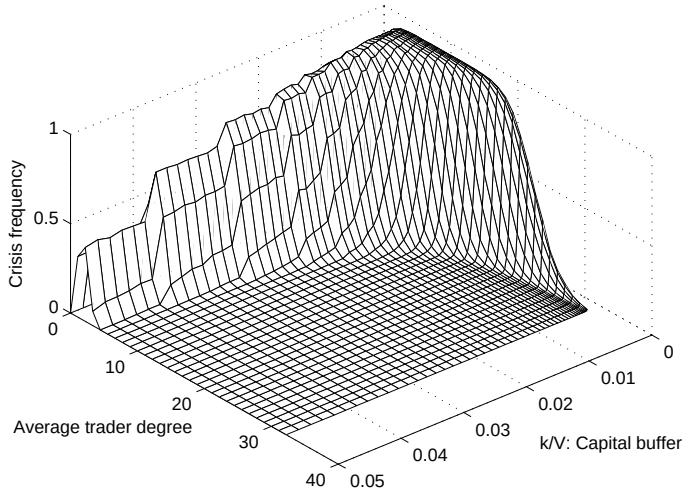


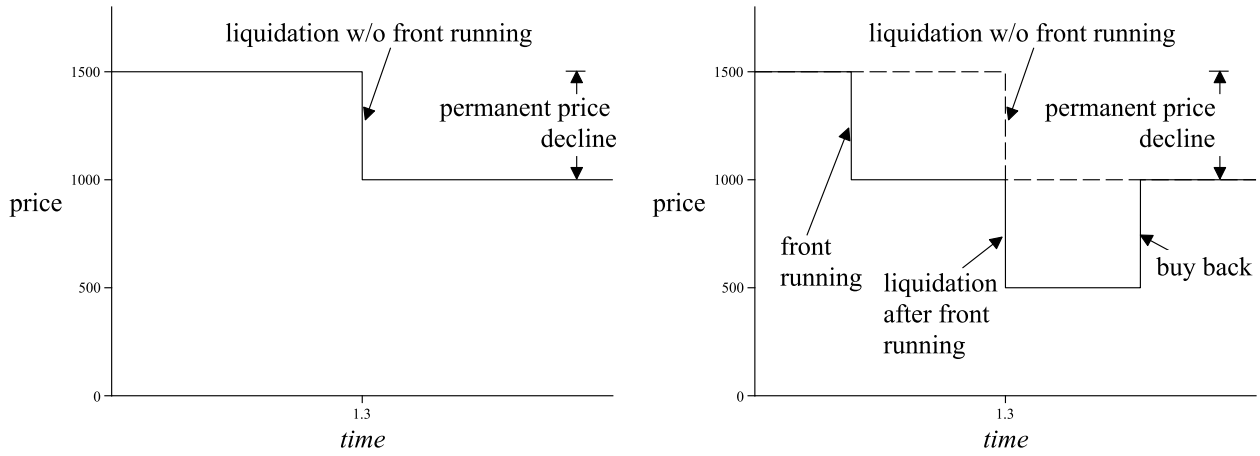
Figure 8: Contagion with various capital buffers

Wall Street. Investigations and convictions appear in newspaper from time to time[18]. There are evidences suggesting that, during the 1998 LTCM collapse, several market participants front run LTCM's liquidation[19].

Figure 9 illustrates a simplified version of the price dynamics during liquidation with front running. In Figure 9a, when some distressed agents are forced to liquidate their positions, without predation the price of an illiquid asset will decline permanently after the forced liquidation. In Figure 9b, if another shareholder (predator) knows about the oncoming liquidation, the predator will try selling before the liquidation, then buy back after the liquidation to her original position. By selling at a higher price at the beginning and buying back after at a lower price, the predator can make a profit. Notice that because of the front running, there is an excessive price decline during the liquidation. At the bottom of this price overshooting where the forced liquidation is about to finish, the price is lower than that without predation. This price overshooting is the reason why we are concerned about predatory behavior in the networks, because it further writes down the asset price during the liquidation and may induce further rounds of distressed liquidation.

The financial network is the same as before and we only consider a very simple model with front running. Whenever there is a distressed trader forced to liquidate, there is exactly

[18]See Khan and Lu (2009)
[19]See Cai (2003)



(a) Without front running (b) With front running

Figure 9: Price dynamics in liquidation

one non-distressed shareholder preys on the distressed. Though stylized, this setting provides a benchmark to show the potential damages that front running could cause. This is also consistent with the fact liquidation is usually highly information sensitive and front running is illegal, so both the distressed shareholder and the predator will want to keep the secret to herself. [20]. The predator sells all her shares of this asset before distressed trader starts to sell, and the predator buys back and restores her initial position after the forced liquidation finishes. The impact of front running and liquidation to shareholders is measured at the bottom of the price overshooting, and all shareholders whose capital buffers are exhausted at this lowest price become insolvent and have to liquidate all their portfolios. The predators are myopic in the sense that they do not forecast whether or not they themselves will be made distressed because of the excessive price declines caused by the predation. When they do find themselves in trouble, they will have to liquidate, just as other shareholders[21].

In the theoretical model where there it is assumed that no closed loop exists in the network, there is at most one distressed shareholder in an asset. Again we assume the simple asset price in the linear form: $\rho(x) = \gamma x$. Since shares are evenly distributed among shareholders, after the predator and distressed have both sold the price decline doubles, and the mark-to-market loss to each remaining shareholder also doubles. Recall that, without predator, this loss is $\frac{V}{d^2}$.

[20]In addition, Brunnermeier and Pedersen (2005a) show that, the more predators, the less profit for each predator can be made out of the predation.

[21]This can be interpreted as predators' incomplete information about the market participants and others' positions, etc.

With one predator, it increases to $\frac{2V}{d^2}$. Consequently, assets with degree $2 \leq d_s < \sqrt{\frac{2V}{K}}$ are vulnerable, i.e. its range expands by $\sqrt{2}$. With more vulnerable assets, it is expected that both the probability and extent of contagion will increase, compared with no front-running cases.

The results from simulations are shown in Figure 10. We see that at the lower end of the

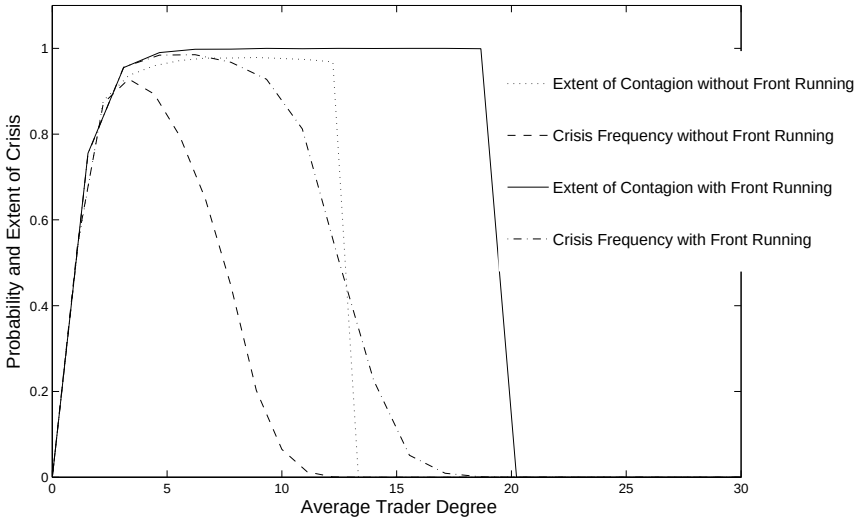


Figure 10: Contagion with front running vs. without front running ($k/V = 0.01$)

average degree, the two models do not differ much. When average degree is beyond 3, the front running makes a difference and outweighs the cases without front running. With front running, the contagion window expands significantly, with both the contagion frequency and the conditional extent of contagion stay near 100% much further than without. This reminds us that, when predatory behavior is common, the asset markets are exposed to much larger risks. The robust-yet-fragile feature is even more evident: crisis may be rare, but if it comes, it will be a real disaster.

5 Conclusion

In this paper I develop a model of contagion of liquidations over complex financial networks. The model applies to any systems of investors connected by their overlapping portfolios and liquidations of one shareholder could adversely affect the market value of portfolios of others, including markets of stock, currencies, securities and commodity. Portfolio diversification may reduce the likelihood of contagion, but it also increases the extent of contagion when liquidation

spread beyond its initial neighborhood. In this case, the failure of the majority of investors is primarily caused by multiple rounds of impacts of liquidations, rather than the first single impact they experience. This model can also incorporate some behavioral aspects such as front running and examine their effects on the contagion.

Our results suggest that when the initial liquidation breaks out, many investors may have felt the impact. But potentially there is still time and opportunities for government to intervene before investors suffer from further rounds of liquidations. If the government can bail out weakened key investors in the network and thus reducing forced liquidations, the damage could be contained in small area rather than spread system wide.

This model provides a preliminary method to evaluate the spread and probabilities of forced liquidation over the asset markets. It would be useful to extend the model by assuming a fixed leverage rather than a fixed value of capital buffer so that an investor's capital is proportional to his portfolio value. It will be interesting to examine the losses and rate of failure for investors with different level of diversification to discuss the optimal level. It would also be highly desirable that one can use actual data on shareholding structures of stock markets to simulate the consequences of an initial shock and the potential contagion that ensues.

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Appendix A: Generating Functions

Let D be a discrete random variable taking values 0, 1, 2, ..., and let $p_i = Prob[D = j]$ for j=0, 1, 2,...

The *probability generating function* of of the distribution, p_i, of the random variable D is

$$f(x) = E(x^D) = \sum_{j=0}^{\infty} P(D = j)x^j = \sum_{j=0}^{\infty} p_i x^j$$

Note that

$$f(1) = \sum_{j=0}^{\infty} p_i = 1$$

The probability distribution p_i can be uniquely determined by the generating function $f(x)$ in the following sense:

$$p_i = \frac{1}{j!}\frac{d^j f(x)}{dx^j}\bigg|_{x=0} = \frac{1}{j!}f^{(j)}(0)$$

Moments. The average over the probability distribution is given by

$$\mu = \langle D \rangle = \sum_{j=0}^{\infty} i p_i = f'(1)$$

and higher moments are given by

$$\langle D^n \rangle = \sum_{j=0}^{\infty} [(x\frac{d}{dx})^n f(x)]|_{x=1}$$

Distribution of sum. If D_1, D_2, ..., D_n are independent discrete random variables with generating functions $f_1(x)$, $f_2(x)$, ..., $f_n(x)$, then the generating function of $D_1 + D_2 + ... + D_n$ is $f_1(x) \cdot f_2(x) \cdot .... \cdot f_n(x)$. For example, if D_1 and D_2 are i.i.d. random variables from distribution p_i, then the distribution of $D_1 + D_2$ is generated by

$$\begin{aligned}
[f(x)]^2 &= [\sum_i p_i x^i]^2 \\
&= \sum_j \sum_k p_i p_k x^{j+k} \\
&= p_0 p_0 x^0 + (p_0 p_1 + p_1 p_0)x^1 + (p_0 p_2 + p_1 p_1 + p_2 p_0)x^2 \\
&\quad + (p_0 p_3 + p_1 p_2 + p_2 p_1 + p_3 p_0)x^3 + ...
\end{aligned}$$