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The Asymmetric Effect of Diffusion Processes: Risk Sharing and Contagion

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Abstract

In this paper we provide a general characterization of diffusion processes, allowing us to analyze both risk-sharing and contagion effects at the same time.

We illustrate the relevance of our theory with reference to the subprime mortgage crisis and more in general to the processes of securitization and interbank linkages. We show that interdependencies in real and financial assets are beneficial from a social point of view when the economic environment is favorable and detrimental when the economic environment deteriorates. In the latter case, private incentives are such that too many linkages are formed, with respect to what is socially desirable. The risk of contagion increases the volatility of the outcome and thus reduces the ability of the financial networks to provide risk-sharing.

Our analysis suggests that a likely major explanation of the subprime mortgage crisis is the process of securitization itself, in addition to the absence of transparency about the characteristics of the underlying assets that the multiple layers of financial intermediation fostered, as commonly claimed.

This may call for a different emphasis on the role of public intervention. While a goal to stabilize the economy in good times should be to disrupt the channels that bring contagion, that is a positive correlation in the returns, in a period of worsening economic conditions our analysis suggests regulatory intervention aimed at disconnecting the economy at crucial nodes. Moreover, we show that policy interventions should be aimed at rescuing institutions, but not their managers. Diminishing the cost of default actually increases the inefficiency due to the divergence between the social and the individual optimum.

KEYWORDS: risk-sharing, contagion, financial crisis, networks

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1 Introduction

Over the last few decades, increased globalization, capital liberalization and financial innovation have prompted a burst of creation of financial linkages between a wide range of different economic actors. These financial linkages are generally put in place intentionally in order to maximize a risk-adjusted expected return, as explained in portfolio theory [Markowitz, 1952]. However, they can also be the result of actions which, although intentional, have different motivations — like obtaining liquidity — or they can be the inevitable byproduct of economic linkages — as in the case of trade relationships.

A crucial question then arises: is an interconnected world a *safer* or a *more dangerous* place to live in? The spread of the 2007 subprime mortgage crisis from the U.S., where it first originated, to other developed economies has highlighted once again the dark side of globalization and financial deregulation.

In the analysis that follows we refer to a *danger* as the risk — both at an individual and at a group level — of being hit by a negative shock that is generated somewhere in the economy. There are two essential ways in which shocks can be diffused through a network. They can be entirely or partially transferred from one node to (one or more of) its neighbors, or they can be transmitted and spread over to other nodes. The first process implies preservation of the original quantity, which is divided among different nodes, while the latter implies its multiplication. We will refer to the first as *risk-sharing*, and to the latter as *contagion*.

Note that starting from a situation where all nodes are homogeneous and a shock hits one node, both processes of risk sharing and contagion imply that the shocked node and its neighbors become more alike as a result of the interaction, that is they imply a positive correlation in outcomes. However, this is obtained in radically different ways: the first process produces reversion to the mean, while the latter produces convergence to the bottom (or to the top). The global inequality decreases monotonically as the shock is diluted, according to the first process, while it first increases and then decreases as the shock is spread over, according to the second.

This definition of contagion implies a causal relationship between shocks, as when an infectious disease is transmitted from one person to the next. This is often modeled as an external effect of the original shock. Our main innovation is to treat it from a statistical point of view, and consider the positive correlation between shocks that it generates. [1]

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[1]Note however that a positive correlation between shocks can also arise as a consequence of a common (external) cause. Our analysis applies also to this case.

To fix the ideas, suppose that there are two risky assets with stochastic returns X_1 and X_2, and two banks B_1 and B_2. Denote with R_1 and R_2 the returns on the investments of these two banks. We speak of a shock as a change in the return on the risky assets. Contagion is modeled as a positive correlation between the shocks, *i.e.* between X_1 and X_2. If the two banks invest in both assets, or if they invest in each other, their returns become more similar (hence R_1 and R_2 are positively correlated), independently from the correlation structure between X_1 and X_2. This is what we term as risk sharing. The possibility of positive correlation (contagion) between X_1 and X_2 is often independent on who owns a claim on what. However, there may be cases in which the ownership structure matters, and there are feedbacks from the upper level R to the lower level X. For instance, the return X (e.g. the interests on a loan, net of the bankruptcy probability) might depend on the decisions of B (e.g. on how much liquidity to provide, and at what interest rate) that in turn depend on R (the financial situation of the bank) that depends on the composition of its portfolio. In this case a positive correlation between X_1 and X_2 can be found as a consequence of direct balance sheet linkages between B_1 and B_2. In our analysis we distinguish between linkages that do not alter the correlation structure in the returns on the underlying assets and those that prompt a positive correlation.

The linkages that may bring contagion, *i.e.* a positive correlation in the returns on different assets, are often deep systemic properties which involve complex feedback between different actors in the economy. As such, they are often neglected by investors and also by the regulation authorities. For instance, although they were already at work during the boom, in the subprime mortgage market they attracted attention only as general economic conditions started to worsen. Higher interest rates implied a decrease in property values, after many years of sustained growth. A high number of borrowers with variable rate mortgages became unable or unwilling to make payments. In order to recover part of their losses, the lenders forced the sale of properties in the housing market. This caused a gigantic increase in the supply of houses, exactly at a time when demand was weak. As a consequence, housing prices suffered even more (first feedback effect). The decline in housing prices implied that other borrowers became unable to refinance their mortgage in more favorable terms, something they were counting on — and that the lenders themselves often encouraged. Hence, many more borrowers made default, putting their properties up for sale. This worsened the financial position of the borrowers, while causing further deterioration in the housing market. Borrowers reacted by restricting the grant of new mortgages and increasing the interest rate on existing variable rate mortgages (second feedback effect), which further

depressed the demand for housing properties and made additional borrowers insolvent. While in previous years the lending behavior of the banks had fueled the increase in housing price, it now contributed toward its plunge.

However, what we aim to show in this paper is that it is the process of securitization, rather than the mechanisms that bring contagion, that played a major role in the subprime mortgage crisis. Securitization allowed banks and other mortgage lenders to pool and repackage their mortgage portfolios and sell them in the financial market as asset-back securities. The increase in the implicit linkages between different economic actors brought by securitization proved an effective means to obtain diversification and risk reduction in good times. However, when the economic conditions started to deteriorate these linkages became detrimental and dangerous, building up a significant risk of systemic failure to the U.S. financial system and imposing a significant a threat to global financial stability.

The rest of the paper is structured as follows: in section 2 we discuss our contribution to the literature; in section 3 we describe our formalization; in section 4 we analyze how risk sharing can be achieved by increasing the connectivity of the system; in section 5 we show how the risk of contagion might modify the risk sharing property of the network; in section 6 we discuss the private incentives to establish connections, while in section 7 we summarize our findings and discuss policy implications.

2 Links with the literature

In the literature, contagion is generally regarded as being synonymous with the propagation of financial distress, due to linkages that facilitate the diffusion of a shock. This reveals that there is some confusion about whether contagion involves the propagation, transmission or transfer of shocks, as these words are often (mis)used interchangeably.

The World Bank provides three definitions of financial contagion: a broad definition ("Contagion is the cross-country transmission of shocks or the general cross-country spillover effects"), a restrictive definition ("Contagion is the transmission of shocks to other countries or the cross-country correlation, beyond any fundamental link among the countries and beyond common shocks") and a very restrictive definition ("Contagion occurs when cross-country correlations increase during crisis times relative to correlations during tranquil times"). [2]

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[2]see the Bank's web page on contagion of financial crisis, at
`http://www1.worldbank.org/economicpolicy/managing%20volatility/contagion/...`

The first definition does not clarify whether the original shock is diluted or not in the diffusion process; hence it could also be applied to what we term risk sharing. The indeterminacy is due to the fact that it does not distinguish between the different levels of the financial chain; that is, it does not consider how shocks in the returns on the underlying assets are related to shocks in the returns to the investors who own them. The second definition also does not distinguish between underlying shocks (to assets) and the implied returns (to investors). Moreover, even if we refer to the underlying assets, the existence of a common cause is explicitly excluded, as is the possibility of a causal relation between the shock due to fundamental links between economic entities, which is often the case. The third definition provided by the World Bank is the closest to the one we use. [Forbes and Rigobon, 2001] and [Claessens et al., 2001] adopt it, when they speak of "a significant increase in cross-market linkages after a shock". The reference to generic cross-market linkages suggests that it does not really distinguish between contagion and diffusion; however, the reference to a dynamic change in those linkages hints at the possibility of positive correlations between the underlying shocks, as we posit: for instance, such an increase may expose other countries to shocks of the same type. The implications they derive are in line with our analysis: "[i]nternational diversification should therefore substantially reduce portfolio risk and increase expected returns. If market correlations increase after a bad shock, however, this would undermine much of the rationale for international diversification" [Forbes and Rigobon, 2001].

Mixing up contagion and diffusion, many authors highlight the existence of a trade-off: they speak of risk sharing when things go right, and of contagion when things get wrong. In this sense, any linkage between economic actors can be a vehicle of contagion. Among these, direct balance sheet linkages between financial institutions are often considered. In a very influential paper, [Allen and Gale, 2000] consider the effects of a liquidity shock in one market when banks in different markets are connected. [3] They show that the cross holdings of deposits is useful to re-allocate liquidity within the banking system, in presence of liquidity preference shocks, *if* there is enough liquidity in the system as a whole. Quite trivially, if liquidity is just badly distributed pooling together all the claims solves the problem. On the other hand, if there is overall excess demand for liquidity interbank linkages let it propagate throughout the system. Instead of having only a few markets facing liquidity shortage, we

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`...definitions.html`. Other definitions of contagion also refer to the diffusion of actions and behavior (e.g. [Morris, 2000])

[3]They consider only four banks/markets, either fully connected or fully disconnected. [Iori et al., 2006] and [Nier et al., 2007] generalize the analysis to more complex network structures.

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have all markets facing liquidity shortage. However, what they do not stress is that a *severe* liquidity shortage in one market becomes less severe, as the shock is diffused (diluted) via interbank linkages. This does not matter only if liquidity shortage (of any size) leads e.g. to a bank run and the eventual collapse of the system, but more generally it is difficult to deny that the size (of a crisis) does matter. [4]

We show that the problem of [Allen and Gale, 2000] is more usefully interpreted as pure risk sharing. In particular, we show that risk sharing is optimal when times are good (when there is no overall shortage of liquidity), but, in their words, it "can turn out to be a disaster" when times are bad. Using the term "contagion" here has only the effect to evocate a negative semantic meaning. A completely different case is when "a shock in one region serves as a signal predicting a shock in another region", which they consider as a different channel for contagion (whose implications they do not examine) but which, in our framework, would be a manifestation of positive correlation between the shocks, and hence a true source of contagion. Similarly, a source of true contagion is identified in the literature on interbank linkages when the negative feedback from the forced sale of assets by distressed institutions on prices and hence on other institutions are explicitly considered (as in [Cifuentes et al., 2005]).

Note that risk sharing may also involve real assets. For instance there are studies of risk sharing behavior in rural environments, with respect to various income and expenditure shocks [Fafchamps and Lund, 2001, Beuchelt et al., 2005, Dercon et al., 2006, De Weerdt and Dercon, 2006], while an application of the central limit theorem to group foraging in animals can be found in [Wenzel and Pickering, 1991].

At a more abstract level the analysis of risk-sharing in networks [Bloch et al., 2006, Bramoullé and Kranton, 2005] generally considers that links are voluntarily formed and focuses on the effect of individual choices on the configuration and stability of the network. In particular, it is generally acknowledged that efficiency requires complete connectivity and full insurance among all members of the network. In contrast, we show that, even if links come at no cost, risk-sharing is beneficial only when the overall economic environment is favorable, while in harsh times it might be better to stay alone.

The role of feedbacks and external effects in determining self-perpetuating dynamics and cascades has commanded even greater attention in the literature,

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[4] A similar trade-off between positive and negative effects of interbank linkages was already investigated in [Bhattacharya and Gale, 1987] and [Rochet and Tirole, 1996], among others.

with applications ranging from innovation to financial markets, cultural fads and social norms (see [Watts, 2004, Jackson, 2006] for a review). The focus is on how the network structure affects individual choices and thus the diffusion process. The key variable is binary at a micro-level (adoption/infection), while continuous at a macro-level (the share of adopters/infected). By contrast, we model contagion as a continuous variable at an individual level (the size of the shock that is transmitted from one individual to another), while its impact at an aggregate level is binary (full or zero adoption/infection), since contagion is assumed to take place instantaneously.

A line of research that is closely related to ours in spirit is the analysis of contagion by means of portfolio theory. In this literature, most contributions rely on some forms of market imperfections to explain why an adverse shock to the returns on one asset can lead to a widespread portfolio rebalance of financial investors with a reduction in other risky asset positions, with the consequence of a price decrease of these assets. For instance, [Calvo and Mendoza, 2000] show that information imperfections leading to market rumors about one asset can lead to sales of other assets, irrespective of their fundamentals. However, portfolio theory can explain global portfolio rebalancing due to local shocks also without recourse to market imperfections [Schinasi and Smith, 2000, Kodres and Pritsker, 2002]. For instance [Goldstein and Pauzner, 2004], similarly to [Kyle and Xiong, 2001], consider the uncertainty about the strategic behavior of other investors and show that a shock in one country might lead to disinvestments in other countries, if investors are characterized by decreasing absolute risk aversion, since the shock reduces investors' wealth and thus make them more risk averse.

The standard definition of contagion according to this approach is comovements in asset prices that are not explained by common fundamentals. Contagion can thus be the result of optimal portfolio allocations made by investors. A strict interpretation of what constitutes an asset would make such a definition coherent with ours (in terms of the example we have discussed above, it would imply a positive correlation between the returns X), except for the fact that we do not distinguish between a fundamental and a speculative value. However, the shares of the banks B in our example also constitute (financial) assets. Comovements in these assets would reflect a positive correlation in R, something that in our approach is not necessarily a result of contagion. Not surprisingly this literature finds that the risk of contagion increases as the level of diversification increases, because the correlation between the returns on investments becomes stronger: contagion is the inevitable by-product of diversification. We reverse this argument, and stress that the possibility of contagion reduces the effectiveness of risk sharing, since diversification works

better the more negatively correlated are the returns on the underlying assets.

A second difference between our approach and the portfolio choice approach is that our model is purely statistical. It does not consider investors' reaction to a change in the value of their portfolios. As such, it can be considered as a model of the "first round" effects of the shocks, where investors that are hit too violently go bankrupt. Portfolios rebalancing, as examined by the literature discussed above, takes place on top of that.

3   A simple diffusion model

There are n financial institutions (hereafter: banks), each with a different investment portfolio subject to a stochastic return X_i. With reference to the U.S. subprime mortgage crisis, think of the portfolio being exclusively composed by mortgages. For simplicity, let's assume that all X_i are drawn from a gaussian vector, with $E(X_i) = \mu, Var(X_i) = \sigma^2$ and $Cov(X_i, X_j) = \rho\sigma^2$, $\rho \geq 0 \ \forall i \neq j$. $\rho = 0$ corresponds to the case when the risk of contagion is null. Note that contagion is neutral on average because positive outcomes can also be propagated (e.g. innovations, self-fulfilling optimistic expectations, *etc*). Alternatively, the random shock might be interpreted as a latent variable connected to the probability of a negative event, a positive realization being equivalent to a reduction in the probability of a negative outcome.

Each institution can either operate in isolation and get a return $R_i = X_i$, or share the risk with other institutions, which can be done either by explicitly buying a stake in the investment portfolio of those institutions or by lending or borrowing money to and from those institutions (in the latter case risk sharing comes from the fact that the loan is not paid back if the borrowing institution goes bankrupt). We will generically refer to these possibly overlapping claims as interbank linkages. We examine as a benchmark the case in which all banks pool their risk together, and face a stochastic return $R_i = R = \bar{X} = \sum_i X_i/n$. This is equivalent to the situation in which they all securitize their mortgages and these asset-backed securities end up (equally mixed) in the portfolios of large investment funds. In this case, $\bar{X}$ is the return on the investment funds.

Hence, risk sharing is modeled as a behavior of the banks, and is summarized by the pooled return R; the possibility of contagion is due to a characteristic of the assets, and is summarized by the correlation coefficient ρ.

If the realized return is less than a threshold θ a financial institution goes bankrupt. [5]   Let $D_i = 1_{[X_i < \theta]}$ be the default indicator for bank i without

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[5]In this model there is no possibility to accumulate reserves (past profits) in order to better face a period of crisis. However, the bankruptcy threshold θ could be negative, to take

interbank linkages, and $D = 1_{[\bar{X} < \theta]}$ be the default indicator with interbank linkages.

The share of expected failures respectively in the absence and presence of interbank linkages are respectively

$$S_{no-link} = E\left[\frac{\sum_i D_i}{n}\right] = \frac{1}{n}\sum_i E[D_i] \tag{1}$$

$$S_{link} = E[D] = P(\bar{X} < \theta) \tag{2}$$

Define the ratio between the two as

$$r = \frac{S_{link}}{S_{no-link}} \tag{3}$$

When we consider the risk of contagion we distinguish two cases. In the first case (*model a*) interbank linkages provide the vehicle for contagion. Think for instance of the case where each bank specializes in providing loans to a specific sector/region. If banks are not linked, a problem in one sector/region does not necessarily affect returns in other sectors/regions. However, if banks are linked a problem in one sector/region may cause a liquidity drain and hence a reduction in the credit supply in other sectors/regions as well, thus contributing to reduce returns everywhere. [6] In this case pooling allows for hedging against the risk of negative outcomes but introduces the possibility of contagion. The structure of the variance-covariance matrix of individual returns $\Sigma(X)$ changes when interbank linkages are considered: $\rho = 0$ when there are no linkages, but $\rho > 0$ when the banks have overlapping claims on each other. [7] In the second case (*model b*) contagion is independent from the existence of interbank linkages: if the housing market tumbles it creates a problem for all mortgage lenders, irrespective (at a first approximation) of the composition of mortgage portfolios. The structure of the variance-covariance matrix of individual returns $\Sigma(X)$ is invariant with respect to the existence

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into account the possibility of a financial institution being able to resist against (limited) losses. This ability could be based on the existence of mandatory or voluntary reserves, or to resort to external debt. Changing the bankruptcy threshold does not affect any of the results of this paper, as long as it remains exogenous.

[6]This might be the result of the vanishing distinction between commercial and investment banking in Anglo-Saxon countries and Japan, in favor of the adoption of the continental European system of universal banking [Rich and Walter, 1993].

[7]More in general, ρ could increases with interbank linkages. This is coherent with the third ("very restrictive") definition of contagion provided by the World Bank, provided that the returns considered are those on the underlying investments, and not those on the total portfolio of the financial institutions who have a claim on them.

of interbank linkages. In particular, this means that $\rho > 0$ even if the banks have no overlapping claims on each other. We show that the only difference between the two cases is that in the latter the variance of outcome without interbank linkages increases. However, the condition under which interbank linkages are beneficial/detrimental remains unaffected.

The following two sections examine the role of risk sharing without and with the risk of contagion.

4 Absence of contagion

In the case of no contagion ($\rho = 0$) there is no difference between *model a* and *model b*. The following proposition holds [8]:

Proposition 1 *If the individual returns are independently distributed and n is sufficiently large, interbank linkages are beneficial (r < 1) if and only if $\mu > \theta$, and are detrimental (r > 1) if and only if $\mu < \theta$.*

Proof   Because all individual X_i are normal, $\bar{X}$ is also normal, irrespective of n, with mean μ and variance $Var(\bar{X}) = \sigma^2/n$. Hence, $S_{link} = P(\bar{X} < \theta) = P(z < -\frac{\mu - \theta}{\sigma/\sqrt{n}})$, where z is a standard normal, and converges either to 0 or to 1 as n grows larger, depending on whether μ is positive or negative. When $\mu = \theta$, S is equal to 0 with probability .5, and is equal to 1 also with probability .5 [9]. On average, half of the population dies.

Because $\rho = 0$, $D_{no-link} = \frac{1}{n}\sum_i P(X_i < 0)$. Since $\sigma^2 > 0$, some $\epsilon > 0$ can always be found such that $\epsilon < P(X_i < 0) < 1 - \epsilon$, and the denominator in eq. 3 remains strictly bound between 0 and n. By the continuity of the limit a size n^* can always be found such that for $n > n^*$ interbank linkages are beneficial if $\mu > \theta$ and detrimental if $\mu < \theta$ [10].   □

Figure 1 exemplifies. Interbank linkages are beneficial if $\mu \gg \theta$ (a), since the probability of getting an insufficient aggregate return is very low. When $\mu \ll \theta$ (c) interbank linkages are likely to produce the collapse of the whole system, while staying alone might lead, although with a low probability, to individual survival. When $\mu = \theta$ (b) the probability of failure is the same, irrespective of whether the banks stay alone or pool their portfolios together.

Note that even without contagion the fate of each bank is not independent from the fate of others, when interbank linkages are present: the returns on

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[8]the proposition can be easily extended to the case of arbitrary distributions of X_i, since by the law of large numbers for a sufficiently big n $\bar{X}$ is well approximated by a normal distribution with mean $\mu = \sum_i \mu_i/n$ and variance $\sigma^2 = \sum_i \sigma_i^2/n^2$

[9]consequently, $Var(S) = .25$ and remains constant irrespective of n

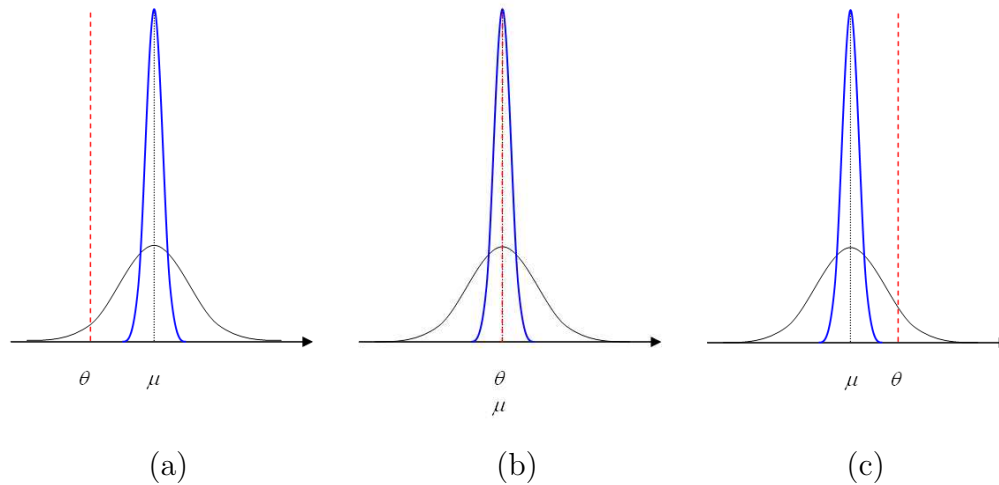[10]this n^* increases as $|\mu - \theta|$ gets smaller, and tends to infinity as $\mu \to \theta$

Figure 1: Distribution of the bank's returns with and without interbank linkages, i.i.d. returns on the underlying assets

the underlying assets are independent, but the returns on the portfolios of the banks are not. This is often called contagion in the literature on interbank linkages, as we pointed out in the introduction, but it is nothing more than pure risk sharing.

The condition $\mu > \theta$ characterizes a well-known trade-off: at each point in time interbank linkages reduce the overall likelihood of failures, but failures imply a system breakdown. This is a manifestation of the "robust yet fragile" nature of many multi-agent systems [Watts, 2002] [11], which may appear stable for long periods of time and easily absorb a number of external shocks, and then suddenly exhibit a large breakdown. Note also that whenever it is not $\mu \gg \theta$, individual failure is just a matter of time anyway. However, it might be the case that (i) the survival threshold θ is only temporarily high, with respect to the average outcome μ, or (ii) the fact that not all of the banks go bankrupt at the same time allows for the replacement of those who exit the market.

5   Presence of contagion

When the possibility of contagion is considered, *model a* and *model b* differ. In the first, $\rho > 0$ irrespective of the existence of interbank linkages; in the second, $\rho > 0$ only if there are interbank linkages.

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[11][Watts, 2002] analyzes this behavior in a model of informational cascades on random networks, where each individual (node) faces a binary decision with externalities

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In any case, with interbank linkages contagion affects the variance of the returns: from the basic theorems about the sum of correlated gaussian variables we have $Var(\bar{X}) = \frac{\sigma^2 + (n-1)\rho\sigma^2}{n}$. [12] The variance of $\bar{X}$ remains bounded: it tends to $\rho\sigma^2$ when $n \to \infty$. Hence, $S_{link} = P(\bar{X} < \theta)$ tends to $P(z < -(\mu - \theta)/\sqrt{\rho}\sigma)$.

5.1 Model a

Under *model a*, the expected number of defaults without interbank linkages is the same as in the no contagion case, since the absence of linkages prevents the diffusion of the shocks. If the individual returns follow identical distributions we can confirm the result of proposition 1: pooling is beneficial if and only if $\mu > \theta$ and detrimental if and only if $\mu < \theta$. The effects of connectivity are simply dampened, with respect to the no contagion case, but never reversed: contagion has the same effect as a reduction in the connectivity of the system. [13]

Proposition 2 *If the existence of interbank linkages allows a positive correlation between the individual returns to manifest, the result of Proposition 1 still holds. However, the effects of pooling are always smaller than in the no contagion case.*

Proof   Since all distributions have the same mean, it is enough to show that when interbank linkages are considered contagion increases the volatility of individual returns, with respect to the case of independent outcomes, but the volatility still remains below the level obtained without interbank linkages [14]: $\sigma^2/n < \frac{\sigma^2 + (n-1)\rho\sigma^2}{n} \leq \sigma^2$. If $\rho = 0$ we get back to the no contagion case of Proposition 1. If $\rho = 1$ the fate of all individuals is identical and pooling together is equivalent to operating in isolation. $\square$

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[12]in order for the variance to be nonnegative it must be $\rho \geq -\frac{1}{n-1}$, which is obviously satisfied when $\rho > 0$.

[13]This proposition is not easily extended to the case of arbitrary distributions of the asset returns. In particular, interbank linkages can be detrimental even if $\mu > \theta$. However, the result that interbank linkages are detrimental if $\mu < \theta$ turns out to be quite general. For instance it can easily be proved when individual outcomes follow a multivariate normal distribution with constant variance-covariance but different means.

[14]a negative value of ρ would produce a reduction in risk. This is the case considered in portfolio theory, but it is clearly not appropriate for the analysis of contagion

5.2 Model b

Model b is the case when there is positive correlation between individual returns ($\rho > 0$) also without interbank linkages. In this case, each D_i follows a dependent Bernoulli distribution, and deriving the expected share of defaults $S_{no-link}$ turns out to be difficult analytically. However, we know how the system behaves in the two extremes $\rho = 0$ and $\rho = 1$. When $\rho = 0$ we are back in the no-contagion case, and proposition 1 holds. When $\rho = 1$, all extractions are identical, and the existence of interbank linkages makes no difference at all. The unconditional expected share of defaults $S_{no-link}$ does not change because the X_i are correlated; only, its variance increases with ρ.

To confirm this intuition, we investigate the effects of an increasing ρ in *model b* by means of a simulation. We consider $n = 100$ assets whose returns X_i follow a multivariate normal distribution with mean μ, variance normalized to 1 and covariance matrix $Cov(X_i, X_j) = \rho, \forall i \neq j$. We also normalize θ to 0. We compare the outcome with and without interbank linkages letting μ and ρ vary. To compute the expected share of defaults we consider, for each combination of μ and ρ, the mean of S_{link} and $S_{no-link}$ over $T = 1,000$ extractions from the multivariate normal distribution.

Figure 2 summarizes the findings: our result that interbank linkages are beneficial (detrimental) only when μ is greater (smaller) than the survival threshold θ still holds. However, as ρ increases, the advantages (disadvantages) of interbank linkages shrink. When ρ approaches unity the outcomes with and without interbank linkages become more similar, as all the extractions are highly correlated with each other. The variance of the share of defaults $Var(S)$ increases. [15]

Figure 3 shows what happens near the threshold θ. When μ is slightly above this threshold ($\mu = .01$), a weak advantage of interbank linkages can still be detected for low levels of ρ (for higher levels, the advantage — which is decreasing in ρ — is shadowed by an increasing variance), while when μ is slightly below the threshold ($\mu = -.01$), the opposite is true. When $\mu = 0$ the expected share of defaults with and without interbank linkages is the same; however, the variance with interbank linkages is higher. [16].

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[15]The level of $S_{no-link}$ under *model a* can be read on the graphs by looking at the red line (absence of interbank linkages) for $\rho = 0$.

[16]In this case the variance of 1 extraction from the multivariate normal distribution of returns, irrespective of the number n of assets and of the level of ρ, is equal to .25. The variance of the average of T extractions, as shown in figure 3, is equal to $.25/T$
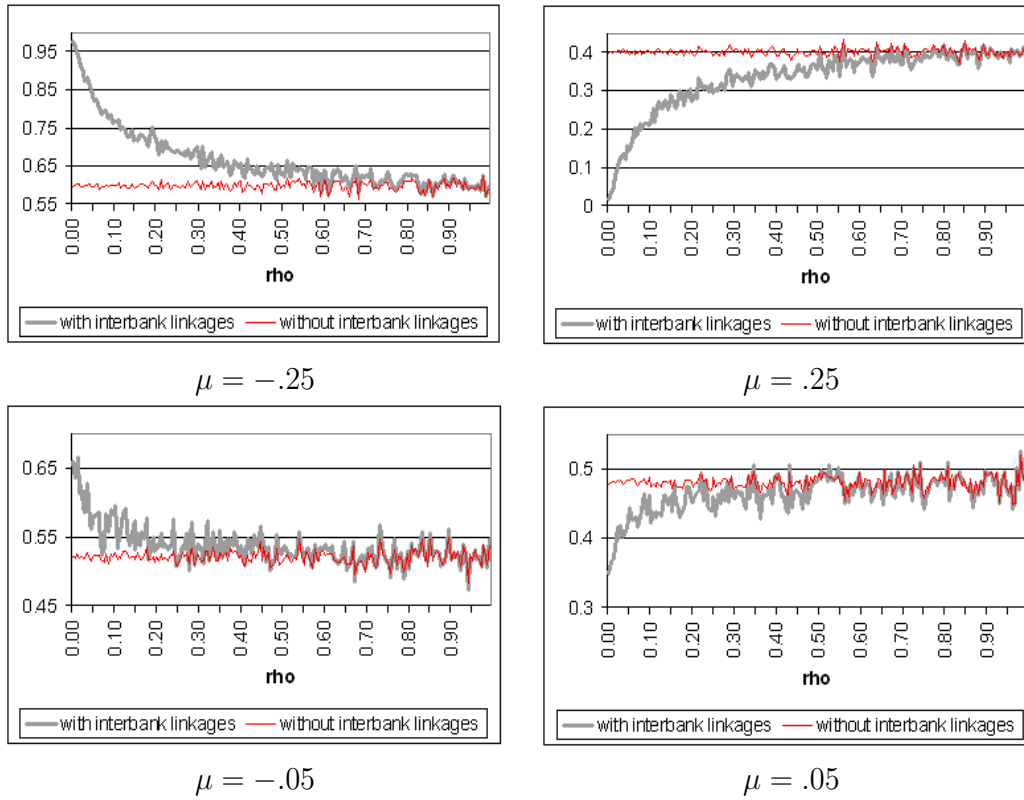
$\mu = -.25$

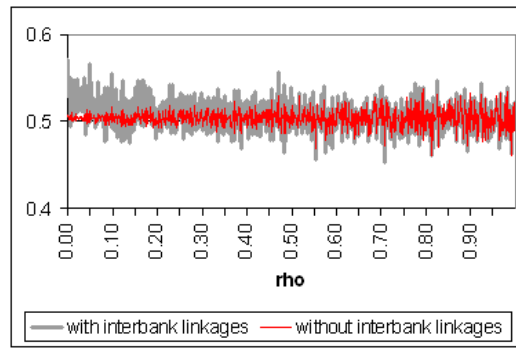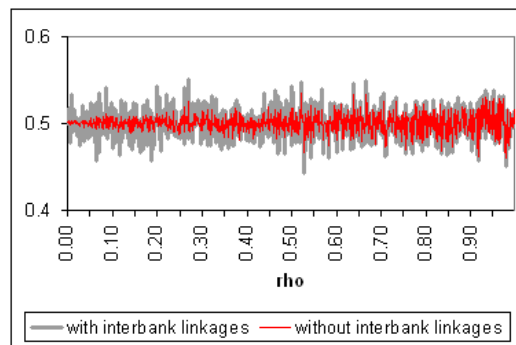$\mu = .25$
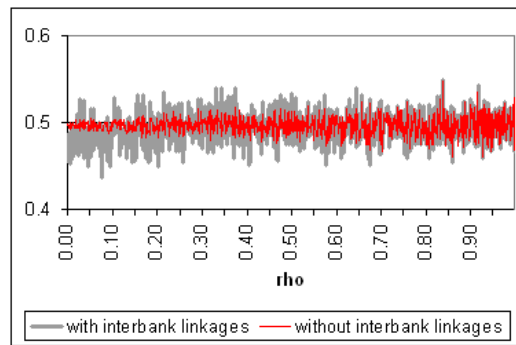
$\mu = -.05$

$\mu = .05$

Figure 2: The effects of contagion, *model b*. Expected share of defaults with and without interbank linkages.

$$\mu = -.01$$

$$\mu = 0$$

$$\mu = +.01$$

Figure 3: The effects of contagion, *model b*. Expected share of defaults with and without interbank linkages.

6   Individual incentives

So far we have provided a normative analysis of whether interbank linkages are favorable, assuming that the goal of society is to minimize bankruptcies. We now briefly discuss the individual incentives to create those linkages. In particular, we argue that it is very likely that too many linkages are formed, since banks are (should be) risk-averse and they also care about their *relative* performance with respect to each other. This is a case of market failure and can provide an argument in favor of public intervention. However, if public intervention goes in the direction of softening the consequences of defaults for bank managers, it could actually make the problem worse.

Of course, a complete analysis would require modeling not only the decision about whether to join the network, but also the dimension of these interbank linkages. This would determine both the size of the network(s) and the level of connectivity between different nodes; in short, it would fully endogenize the network structure, which is something we leave for future research.

In the models described in this paper we have considered that whenever a bank joins the network it becomes indistinguishable from the other banks who have also joined the network; better, it becomes part of a giant super-bank. Coherently with this simplification, we now only consider the private incentives to become part of such a super-bank.

Let

$$U_i = U(R_i) + u(R_i - R) - cD_i \tag{4}$$

be the utility of the bank managers, where R_i is the bank return, $R = \bar{X}$ is the market return, D_i is the default indicator and $c \geq 0$ is the bankruptcy cost. We assume $U', u' > 0, U'', u'' < 0$, and normalize $u(0) = 0$. Hence, the utility of a bank depends positively on its expected returns and on the extent to which its performance beats the market, and negatively on the risk and cost of bankruptcy.

Without interbank linkages, each bank gets an expected utility of

$$E(U)_{no-link} = E(U) + E(u) - cS_{no-link} \tag{5}$$

while if part of the superbank, it gets

$$E(U)_{link} = E(U) - cS_{link} \tag{6}$$

The following proposition then holds:

Proposition 3 *Interbank linkages are formed whenever $\mu \geq m(c)$, with $m(c) \leq \theta$ and $m'(c) \geq 0$. Hence, with respect to the social optimum, too many interbank linkages are formed when the economic conditions are bad ($\mu < \theta$). Moreover, this market failure is bigger the smaller the bankruptcy cost c.*

Proof First, let's note once again that $Var(X_i) \geq Var(\bar{X})$, hence $Var(R_i) \geq Var(R)$. [17] Because of the assumption of risk aversion, this implies that the first term in equation 5, $E(U)$ is always smaller than the corresponding term in equation 6. Moreover, the same assumption of risk aversion implies $E(u) \leq 0$, since $E(R_i) = E(R)$.

If $\mu > \theta$, $S_{no-link} > S_{link}$. All three components of utility are larger with interbank linkages and therefore a giant superbank is formed, in line with the social optimum.

If $\mu < \theta$ however, $S_{no-link} < S_{link}$. In the comparison between $E(U)_{no-link}$ and $E(U)_{link}$, only the third component of utility has reversed its contribution. Therefore, for given values of θ and c interbank linkages are formed whenever $\mu > m$, with $m < \theta$. Since the social optimum would require $m = \theta$, the individual decision whether to join the superbank is inefficient. Because $(S_{link} - S_{no-link})$ is increasing with $(\theta - \mu)$, the threshold m is increasing with c and reaches a maximum at $m = \theta$ when $c \geq c^*$. □

7 Conclusions and policy implications

In this paper we have presented a simple statistical model of diffusion processes, allowing for the analysis of both risk-sharing effects and contagion effects at the same time. Although built on a general framework, our analysis is based on many simplifications: in particular, we assumed that assets and financial actors are homogeneous, and we covered only the extreme cases of fully disconnected and fully connected networks.

From a normative perspective, we have shown that interconnectivity is beneficial, insofar as it reduces the likelihood of individual defaults, but only when the general economic conditions are good. In harsh times, that is when the expected return on investments turn negative, being interlinked becomes socially detrimental: not only is it that the expected number of defaults is higher when the economic agents are connected, but defaults become a systemic failure. However, private incentives are likely to drive individual behavior toward establishing these linkages anyway.

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[17] the only case when equality holds is in *model a* when $\rho = 1$.

The risk of contagion only reduces the effectiveness of interconnectivity to provide risk-sharing: contagion makes risk sharing less beneficial or less detrimental but leaves the conditions in which risk sharing is beneficial or detrimental unaffected. Hence, our analysis suggests that policy makers should focus on disrupting the channels that bring contagion in good times, in order to get the most out of the risk sharing behavior of financial actors, but turn to other goals in period of worsening economic conditions. In particular, regulatory intervention could then be aimed at disconnecting the economy at crucial points by removing some of the linkages between financial actors or, as a last resort, provide targeted intervention to rescue distressed financial institutions. In this case, special attention should be paid not to mitigate the cost of bankruptcy for the managers of these institutions, since this would provide additional incentives to form socially dangerous financial linkages.

We have discussed the model with reference to the U.S. subprime mortagages crisis. Our analysis suggests that a likely major explanation of the crisis is to be found in the process of securitization itself, rather than in the absence of transparency about the characteristics of the underlying assets or in the presence of fraud and misbehavior that the multiple layers of financial intermediation fostered.

Aside from the financial examples discussed above this simple model accounts for a variety of situations, where the interconnectivity between economic agents is important, e.g. research and development or collusive alliances among corporations, international alliances and trading agreements [Jackson, 2004].

To be more concrete, let's consider the case of credit relationships, and think of X as a notional measure of liquidity. Liquidity can be affected by shocks that originated elsewhere in the economy, as (i) a decrease in demand, (ii) an increase in costs, (iii) a technological shock that forces investment, (iv) debtors deferring or delaying payments, (v) creditors urging for repayment or refusing to provide more financing, *etc.* Moreover, a liquidity shock can be passed up to suppliers and creditors and down to clients, by means of variations in prices and quantity and delays in payments and provisions [Stiglitz and Greenwald, 2003]. These client-supplier and debtor-creditor linkages between economic agents ensure that shocks are partially transferred. If one firm runs out of cash, it can delay payments to suppliers, who act as a shock-absorber. This however weakens the position of the suppliers, although they might in turn partially pass this negative shock to other firms. This mechanism can either provide an effective shock absorber, or, if the original shock is big enough, prelude to a widespread default. Contagion — a positive covariance in individual outcomes — can be originated by common demand or supply shocks,

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or by the spread of opinions and expectations in the economy.

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