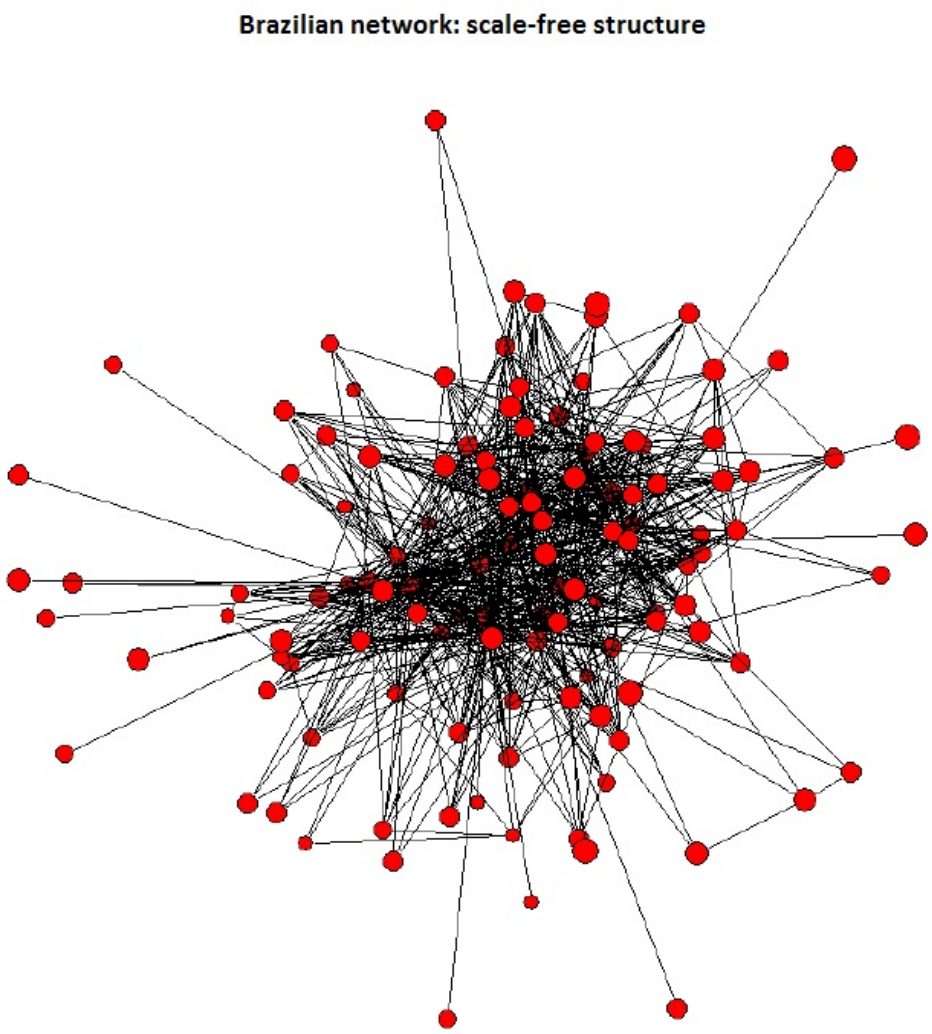
Systemic risk : channels of contagion in financial systems

Rama CONT



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Systemic Risk

- Systemic risk may be defined as the risk that a significant portion of the financial system fails to function properly.
- The monitoring and management of systemic risk has become a major issue for regulators and market participants since the 2008 crisis.
- The financial crisis has simultaneously underlined

· the importance of contagion effects and systemic risk

· the lack of adequate indicators for monitoring systemic risk.

· the lack of adequate data for computing such indicators

Many initiatives under way: new regulations (Basel III), new financial architecture (derivatives clearinghouses), legislation on transparency in OTC markets, creation of Office of Financial Research (US), various Financial Stability Boards

BUT: methodological shortcomings, open questions

Systemic Risk

Various questions:

Mechanisms which lead to systemic risk

Measures / metrics of systemic risk

Monitoring of systemic risk: data type/granularity ?

Management and control of systemic risk by regulators

Need for quantitative approaches and objective criteria to deal with these questions

Channels of contagion: underlying network structure

- Each of these mechanism may be viewed as a contagion process on some underlying "network", but the relevant "network topologies'' and data needed to track them are different in each case:
- 1. Correlation: cross-sectional data on common exposures to risk factors/asset classes for tracking large-scale imbalances
- 2. Balance sheet contagion: network of interbank exposures, cross-holdings and liabilities + capital
- 3. Spirals of illiquidity: network of short-term liabilities (payables) and receivables + 'liquidity reserves'
- 4. Fire sales/ feedback effects: data on *portfolio holdings* of financial institutions across asset classes + capital

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Lecture 1: Contagion and systemic risk in financial networks

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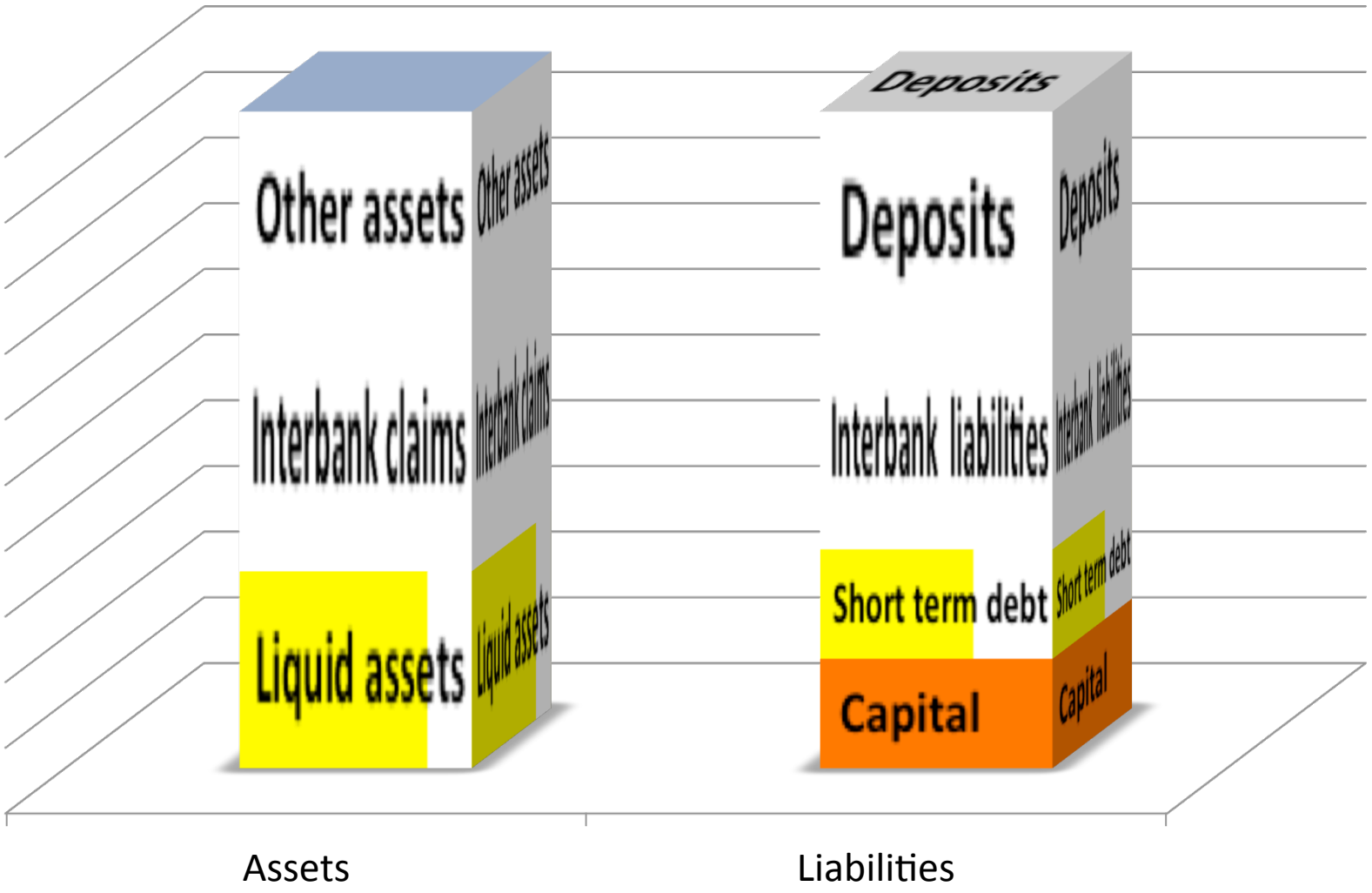
A bank balance sheet



– a *liquidity* buffer l_i

Assets A_i	Liabilities L_i
Interbank assets $\sum_j E_{ij}$ including: Liquid assets l_i^0	Interbank liabilities $\sum_j E_{ji}$ Deposits
Other assets a_i	Capital c_i

Table 1: Stylized balance sheet of a bank.

Insolvency and illiquidity

- Equity + Debt= Total assets.
- Capital= Equity is not to be confused with cash/liquid reserves.
- If Total assets < Debt then firm is insolvent.
- If "liquid assets" < payables, firm is illiquid.
- Liquidity reserves vary rapidly (intraday) while asset and debt values are more "slowly varying".
- Default occurs when firm fails to meet payables (margin call, coupon, interest on debt). This is a liquidity issue.
- A firm can become illiquid even though it is perfectly sovlent. But in principle such a firm will not default: it can borrow against its assets.
- In principle, insolvency does not immediately entail default as long as payments due are met.
- However, when the firm is a bank/ financial institution:

-the regulator monitors capital and may liquidate/restructure bank as insolvency threshold is reached

-most financial institutions hold a very small reserve of liquid assets and cover their short term liquidity needs through short term (overnight) loans. In this situation insolvency (or, indeed, a rumour of insolvency) leads to lenders withdrawing the firms funding and lead to illiquidity

Lender of last resort: if a bank is solvent but temporarily illiquid, the central bank can provide liquidity (lend) to it until liquidity is restored. Bagehot's principle: lend to solvent banks, but at a penalty.

These considerations justify to focus the modeling of bank failure and contagion on insolvency which is the fundamental notion of failure for a bank.

The 'microprudential' approach to financial stability

Traditional approach to risk management and bank regulation: focused on failure/non-failure (solvency, liquidity) of individual banks

Focuses on balance sheet structure of individual banks

Risk of each bank's portfolio is measured using a statistical approach based on historical data: assumes that losses arises due to exogenous random fluctuations in risk factors (stock prices, exchange rates, interest rates, housing prices..)

Main tool for stabilization of system: capital requirements

Based on premise that 'it is enough to supervise the stability of each bank to ensure stability of system'

Ignores links or interactions between market participants which can lead to market instabilities even when banks are 'well capitalized' (Hellwig 1995, 1998; Rochet & Tirole 1996; Freixas, Parigi & Rochet 2000)

Why systemic risk does not reduce to 'marginal risk'

Example 1 (Hellwig): liability chains

Consider a finite set of banks i=1..N, where
i borrows $ 100 from i-1over i years,
i lends $ 100 to i+1 over i+1 years, etc.

Each institution in this "lending chain" is exposed to 'market risk' due to the difference (spread) between the i-year interest rate r(i) and the i+1 year interest rate r(i+1).

Net exposure of i= X(i)=100 (r(i+1)-r(i))

A microprudential approach asks for capital requirements based on a quantile of X(i) or some other tail measure:

Typically interest rate volatility is small (few %) so setting a capital requirement of a few % covers this marginal risk.

However, if bank *i* fails (defaults), then counterparty *i-1* is left completely exposed: exposure in case of default is

E(i)=100 >> X(i)

1998: "Long Term Capital Management"

- Size= 4 billion$, Daily VaR= 400 million $ in Aug 1998.
- Amaranth (2001): size = 9.5 billion USD, no systemic consequence.
- The default of Amaranth hardly made headlines: no systemic impact.
- The default of LTCM threatened the stability of the US banking system $\rightarrow$ Fed intervention
- Reason: LTCM had many counterparties in the world banking system, with large liabilities/exposures.
- **1**: Systemic impact is not about 'net' size but related to exposures/ connections with other institutions.
- **2**: a firm can have a small magnitude of losses/gains AND be a source of large systemic risk

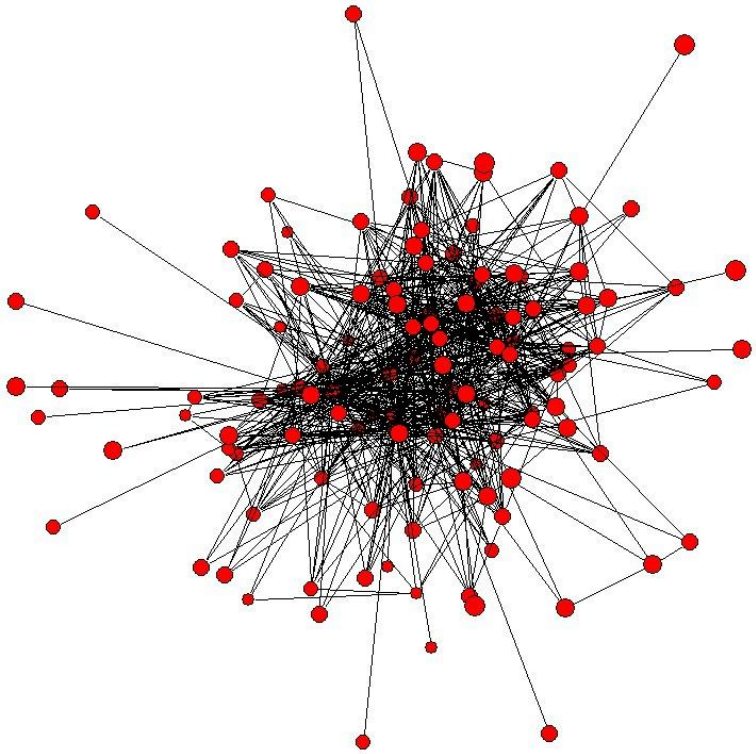
Counterparty networks: interbank exposures

- The relevant setting for studying balance sheet contagion (insolvency cascades) is a network – a weighted, directed graph- whose nodes are financial institutions and whose links represents interbank exposures :

$$E_{ij} = \text{exposure of i to j}$$

= writedown in balance sheet of i when j defaults, taking into account liabilities+ cross-holdings.

- Data on interbank exposures reveal a complex, heterogeneous structure which is poorly represented by simple network models used in the theoretical literature.



Brazilian Interbank network
(Cont, Moussa, Santos 2010)

Measuring systemic risk: why exposures are important inputs

Market-based indicators have been recently proposed for quantifying

- contagion effects: CoVaR (quantile regression of past bank portfolio losses, Adrian & Brunnenmeier 2009)
- the (global) level of systemic risk in the financial system (Lehar 2005, Bodie, Gray, Merton 2008, IMF 2009, Huang, Zhu & Zhou 2010, Acharya et al 2010,..)

Useful for analyzing past/current economic data and should be part of any risk dashboard/ systemic risk tool kit.

Value as forward-looking diagnosis tools? any predictive ability?

Also: market-implied measures capture *market-perceived systemic risk*. Did market prices capture the systemic risk of AIG prior to its collapse?

Network approaches are based on **exposures** which represent potential **future** losses, which can give quite a different picture from past losses.

Even if we believe the Efficient Market hypothesis, market indicators need not reflect exposures, which are not public information.

Regulators, on the other hand, have access to non-public information on exposures and should use such information for stress testing and for computing systemic risk indicators.

Regulatory reforms proposed for mitigating systemic risk include

1. monitoring and control of **nodes** (balance sheets):
   - higher capital ratios: increasing resilience of nodes to potential losses
   - Liquidity Coverage Ratio, Stable Funding Ratio: to prevent default contagion through loss of short term liabilities
   - other: leverage ratio,
2. monitoring and control of **links** : proposals for structural reform
   - Central clearing of OTC derivatives: introduce a new node (Central Counterparty) and replace bilateral OTC exposures by exposures to CCP
   - Ring-fencing (Vickers, Liikanen): separatd the network into a tightly regulated core of retail banks and a less regulated set of other institutions, with strict limits on links between these two subnetworks.

Underlying these proposals is the question of the relation between **network structure** on one hand and the **resilience of the network to shocks** on the other hand.

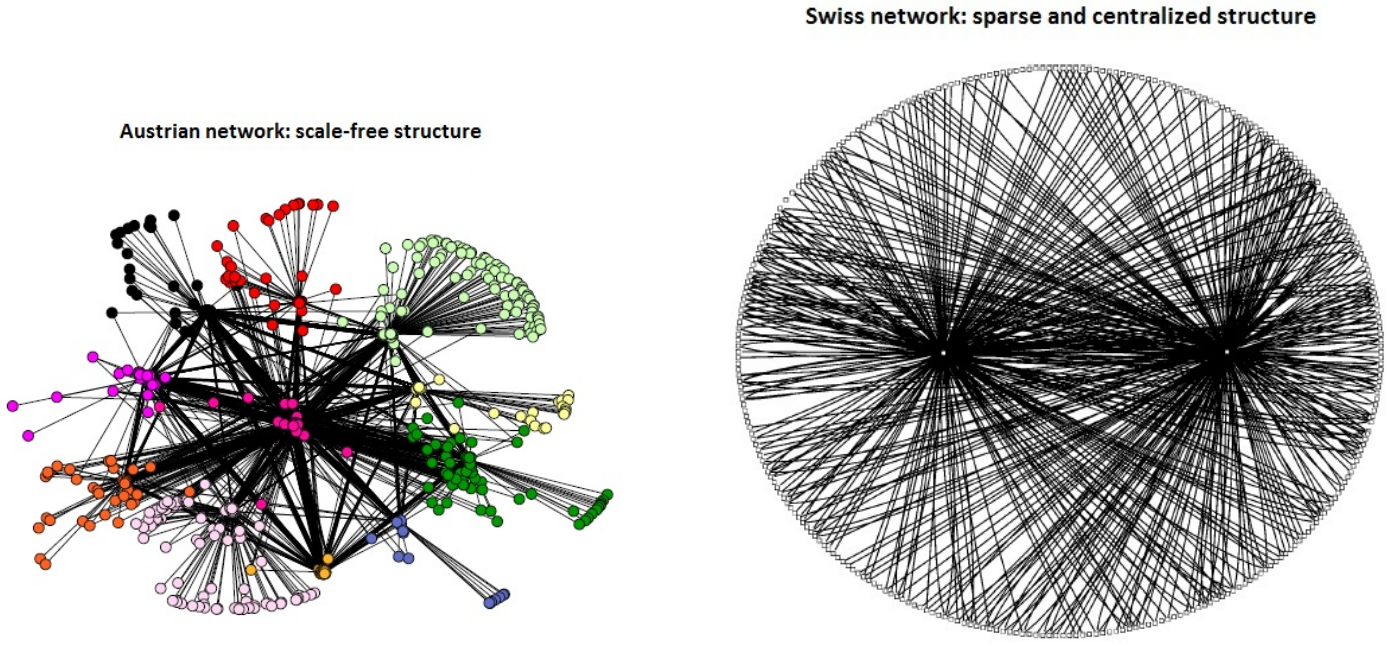


Figure: Network structures of real-world banking systems. Austria: scale-free structure (Boss et al2004), Switzerland: sparse and centralized structure (Müller 2006).

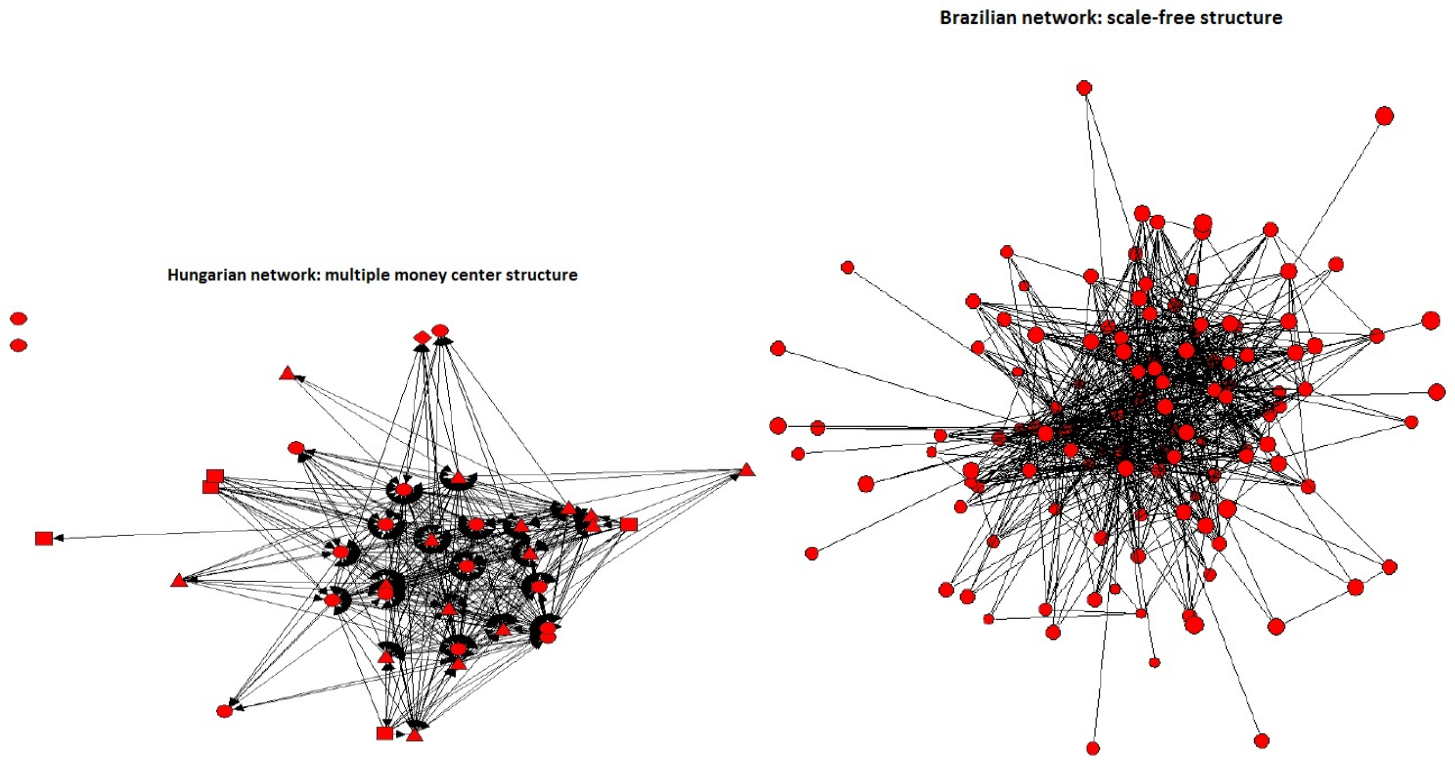


Figure: Network structures of real-world banking systems. Hungary: multiple money center structure (Lubloy et al 2006) Brazil: scale-free structure (Cont, Bastos, Moussa 2010).

The Brazil financial system: a directed scale-free network

- Exposures are reportted daily to Brazilian central bank.
- Data set of all consolidated interbank exposures (incl. swaps)+ Tier I and Tier II capital (2007-08).
- $n \simeq 100$ holdings/conglomerates, $\simeq 1000$ counterparty relations
- Average number of counterparties (degree)= 7
- Heterogeneity of connectivity: in-degree (number of debtors) and out-degree (number of creditors) have heavy tailed distributions

  $$\frac{1}{n}\#\{v, \text{indeg}(v) = k\} \sim \frac{C}{k^{\alpha_{in}}} \qquad \frac{1}{n}\#\{v, \text{outdeg}(v) = k\} \sim \frac{C}{k^{\alpha_{out}}}$$

  with exponents $\alpha_{in}, \alpha_{out}$ between 2 and 3.
- Heterogeneity of exposures: heavy tailed Pareto distribution with exponent between 2 and 3.

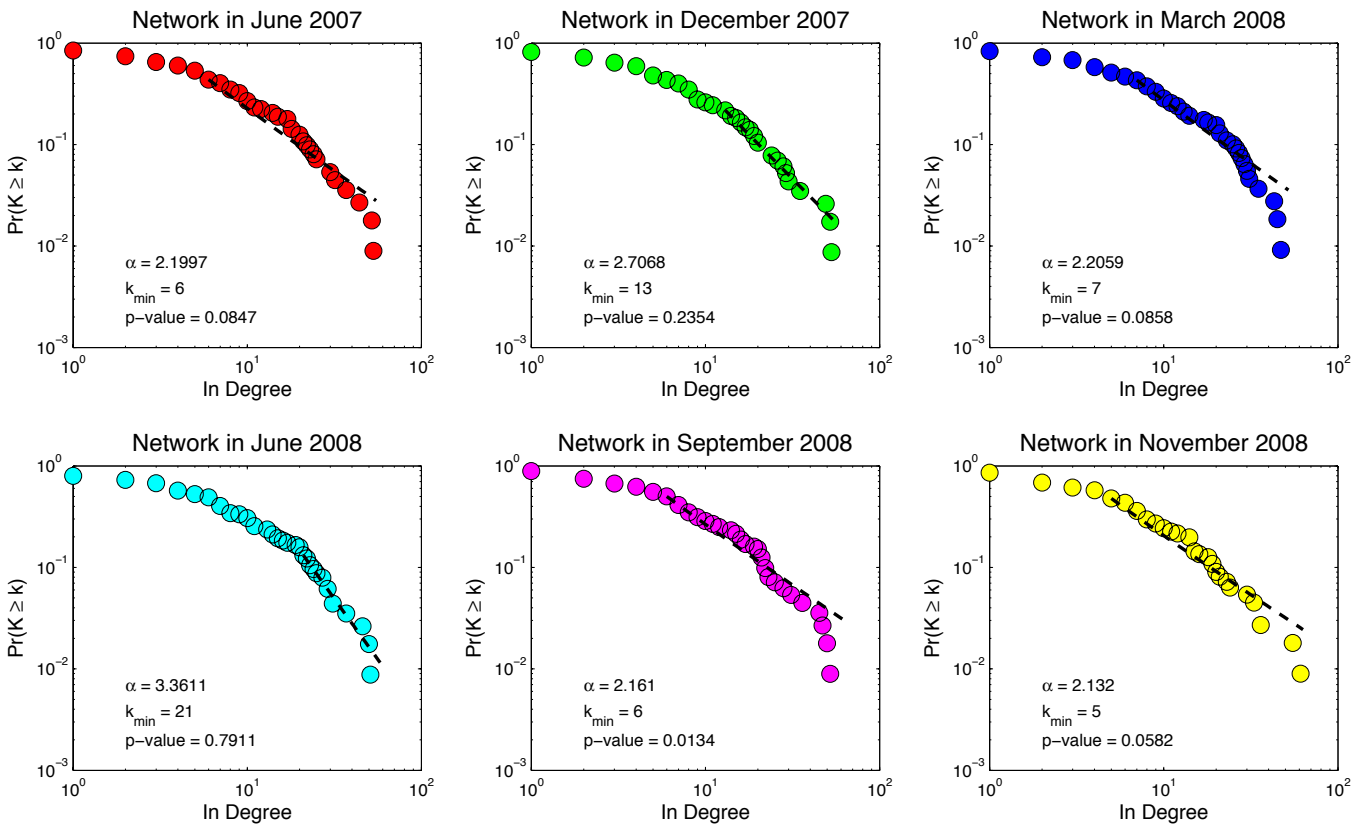


Figure 3: Brazilian financial network: distribution of in-degree.

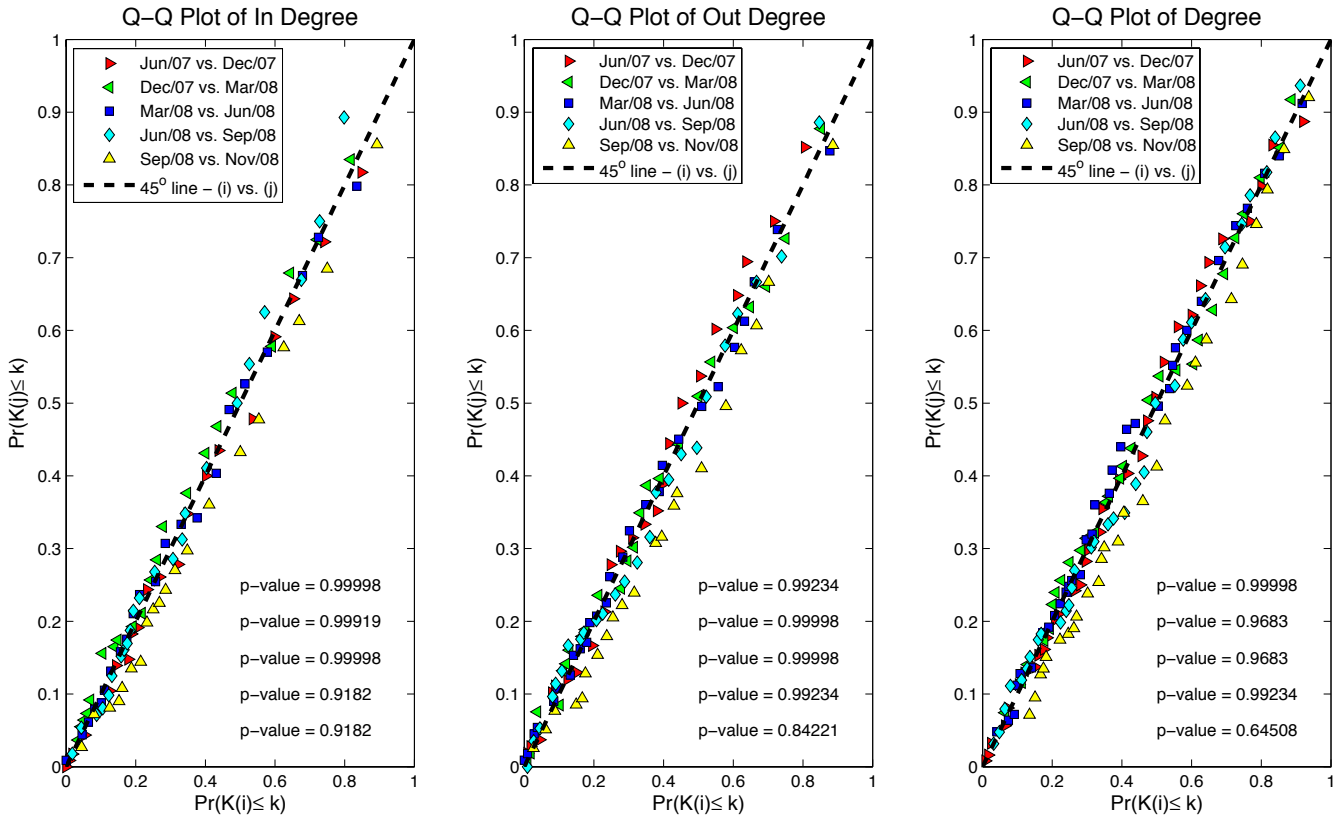


Figure 4: Brazilian financial network: stability of degree distributions across dates.

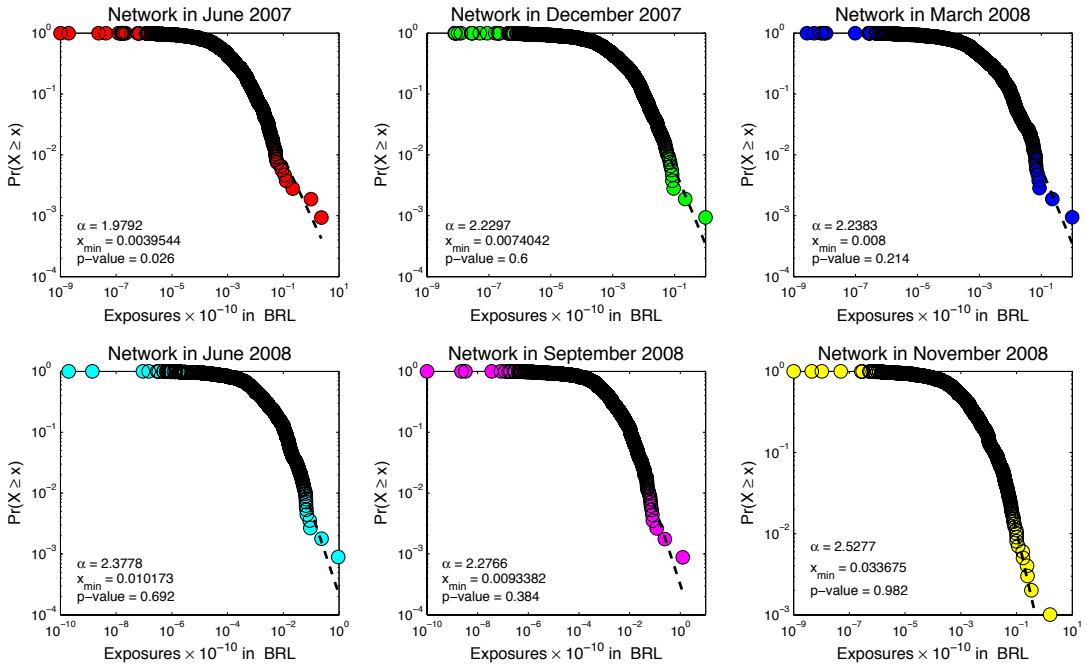


Figure 6: Brazilian network: distribution of exposures in BRL.

Clustering coefficients

Clustering coefficient of a node=

Number of links among neighbors/ Number of *possible* links among neighbors

The ratio is between 0 and 1.

For a complete graph, or for a node immersed in a complete subgraph, the ratio is 1.

For a d-dimensional lattice, clustering $\to 0$ as d increases.

Small world graphs are characterized by small diameter, bounded degree and clustering coefficients bounded away from zero.

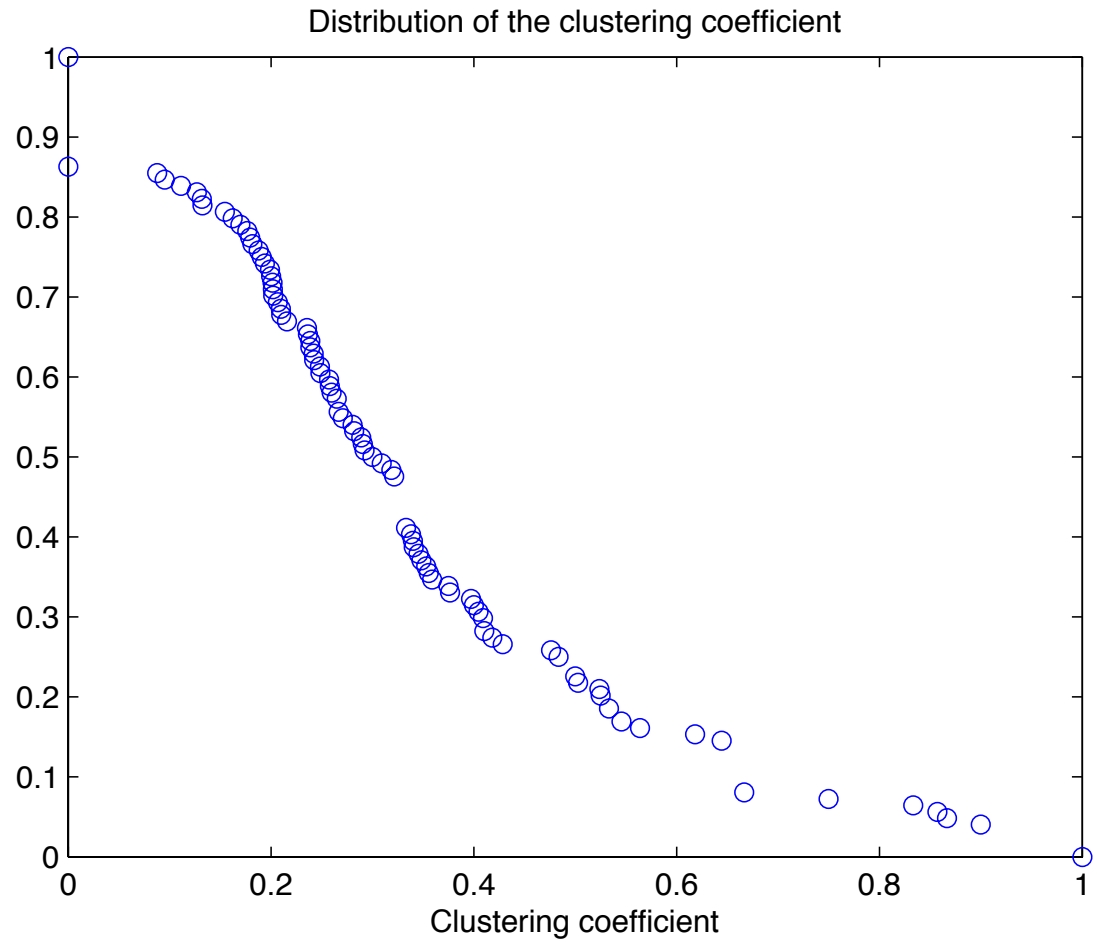


Figure 5: Brazilian network: distribution of clustering coefficient

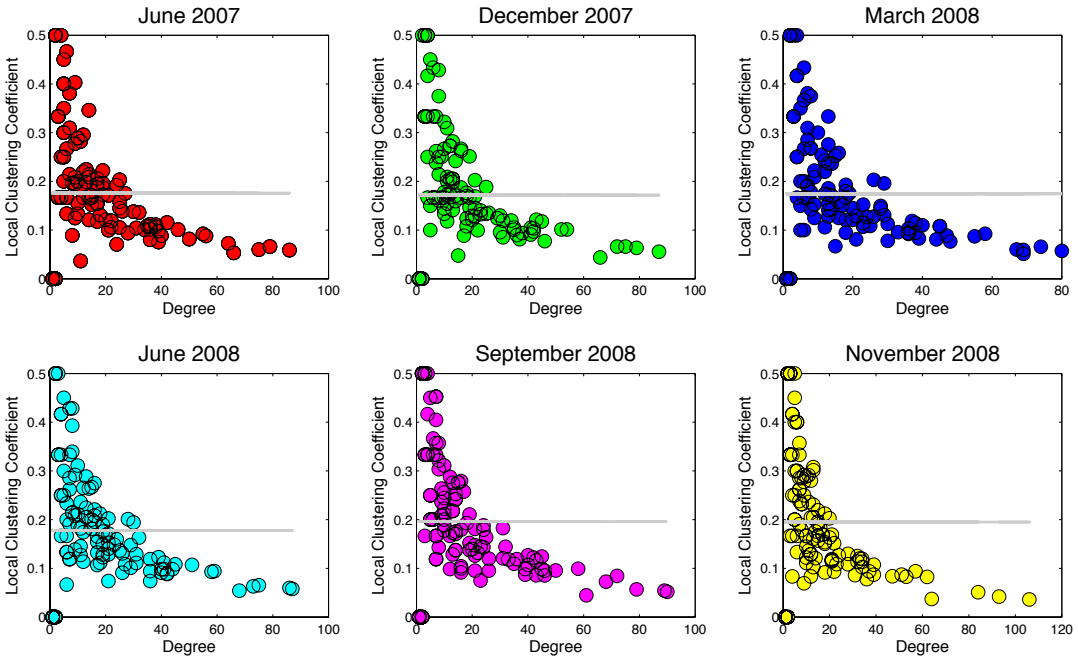


Figure 6: Brazilian financial network: degree vs clustering coefficient. Arbitrarily small clustering coefficients rule out a small world network.

Network models: irrelevant vs irrelevant issues

- Data on interbank exposures reveal a complex, heterogeneous structure which is poorly represented by simple network models used in the theoretical literature.
- Key observation is **HETEROGENEITY** of nodes and exposures, which should warn against the use of simple, homogeneous networks as models for theoretical analysis.
- There has also been a recent outpour of studies on exposure data using the 'network' approach, computing graph-theoretic measures of centrality, PageRank, clustering for interbank networks.
- It is not clear why these measures have any relation with 'systemic risk'
- More precisely, any measure (such as centrality, PageRank,..) which does not use levels of capital as input is, by definition, insensitive to capital requirements and cannot be used in a meaningful analysis of systemic risk

Some Questions

- How does the default of a bank affect its counterparties, counterparties of counterparties,… (domino effect)?
- Which are the banks whose defaults generates the largest systemic loss? : identification of SIFIs
- Can the default of one or few institutions generate a macro-cascade / large-scale instabiity of network?
- How do the answers to the above depend on network structure? Which features of network structure determine its stability/ resilience to contagion?

Previous work: many simulation studies+ analytical results for *average* cascade size on *homogeneous networks (*Watts (2002), Gai & Kapadia (2011),…)

Here: **analytical results on resilience and cascade size (not just average) for general, heterogeneous networks**

Equilibrium analysis: 'clearing vectors'

Consider a situation where all portfolios are simultaneously liquidated and debtholders are paid off completely if possible and if not, proportionally to fraction of debt held. Then, any node unable to pay its liabilities upon liquidation will be declared in default.
(Eisenberg & Noe 2001): Given a matrix of exposures E and capital allocations c, a *clearing vector* is a vector of cash flows p verifying

- limited liability: payments made by a node do not exceed the cash flow available to the node;
- the priority of debt claims: node receives no value until the node is able to completely pay off all of its outstanding liabilities
- proportionality: if default occurs, all liability holders are paid by the defaulting node in proportion to the size of their nominal claim on firm assets.

Recovery rate of each firm at 'equilibrium' is endogenous: it is the ratio of recovered assets to liabilities.
(Eisenberg & Noe 2001): A clearing vector always exists: it may be computed as a fixed point of an iterative "liquidation process".

Equilibrium analysis: 'clearing vectors'

Model motivated by the overnight clearing of interbank payment systems: liabilities are realized as cash flows every day.
Less realistic as a model for bank default or contagion of large bank losses: in reality

- only defaulted firms are liquidated, not all firms.
- in absence of default, there is no liquidation: model cannot deal with large losses, only true defaults.
- liquidation is a lengthy process and, even if eventually a high recovery rate is realized after liquidation ends, defaulted assets are not available/liquid immediately after default.
- many losses leading to insolvency are not realized as cash flows but may be pure 'accounting losses' / writedowns in asset value: immediately after default, these writedowns may be very large, corresponding to very low, or zero (if assets are frozen during liquidation), 'short term' recovery rates.

Ex: Lehman default eventually led to 84% recovery rate but took 6 months, during which dozens of hedge funds exposed to Lehman defaulted!

Cascade models

Another approach is to consider the impact on the network of an exogenous shock: this shock can be

- the default of a node or set of nodes or, more generally
- a loss in asset value across nodes, which may result in a 'downgrade' / loss in creditworthiness of some nodes

- In both situations, counterparties of the node subject to the shock will be affected through writedowns in asset value, so loss is transmitted through the network.
- If the loss transmitted is large relative to the receiving node's capital buffer, new credit events may occur, resulting in a **cascade of losses**.
- Such threshold models of cascade processes were first studied in the context of social networks (Granovetter 1978; Watts 2002) in homogeneous networks with identical node characteristics.

Loss Cascades in a counterparty network

Definition (Loss cascade)

Consider an initial configuration with capital levels $(c(j), j \in V)$. We define the sequence $(c_k(j), j \in V)_{k \geq 0}$ as

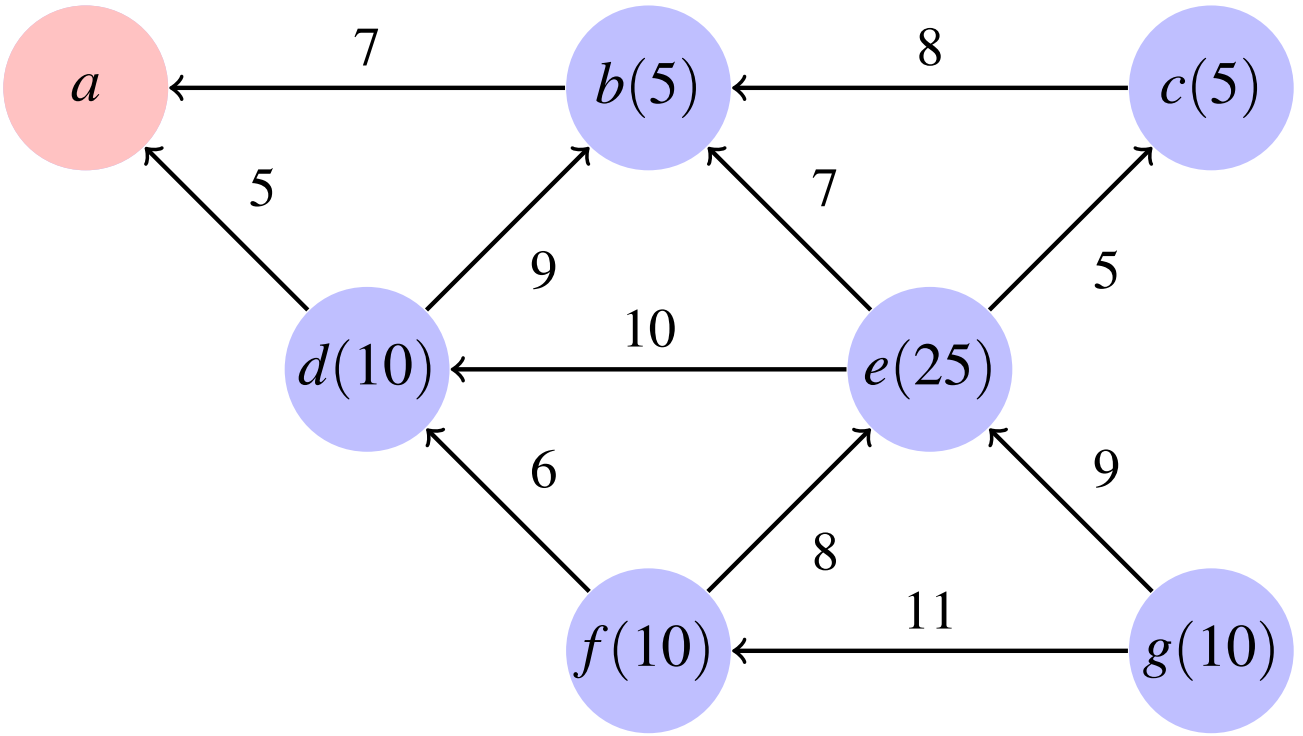
$$c_0(j) = c(j) \qquad \text{and} \quad c_{k+1}(j) = \max(c_0(j) - \sum_{\{i, c_k(i)=0\}} E_{ji}, 0), \tag{1}$$

where R_i is the "recovery rate" for liabilities of institution i after the credit event. $(c_{n-1}(j), j \in V)$, where $n = |V|$ is the number of nodes in the network, then represents the remaining capital once all counterparty losses have been accounted for. The set of insolvent institutions is then given by

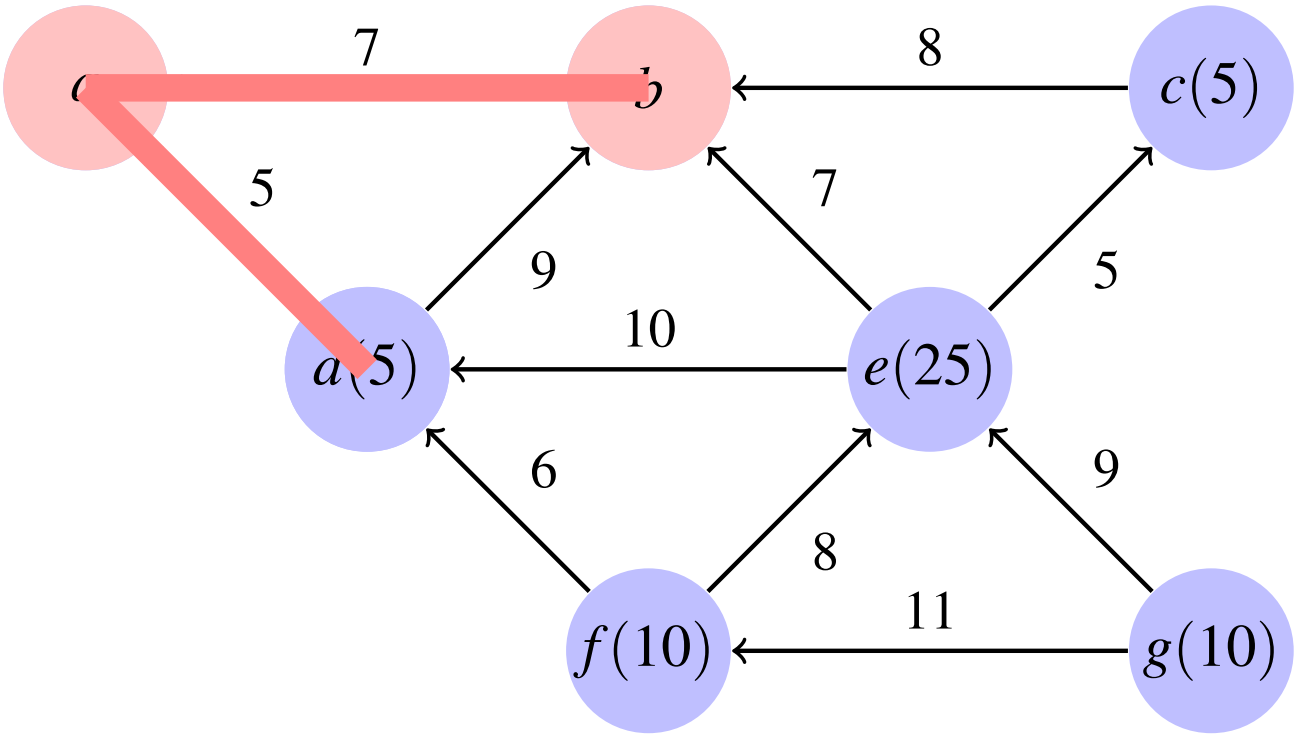
$$\mathbb{D}(c, E) = \{j \in V : c_{n-1}(j) = 0\} \tag{2}$$

With n nodes, the cascade terminates at most after $n - 1$ steps.

Example



Example



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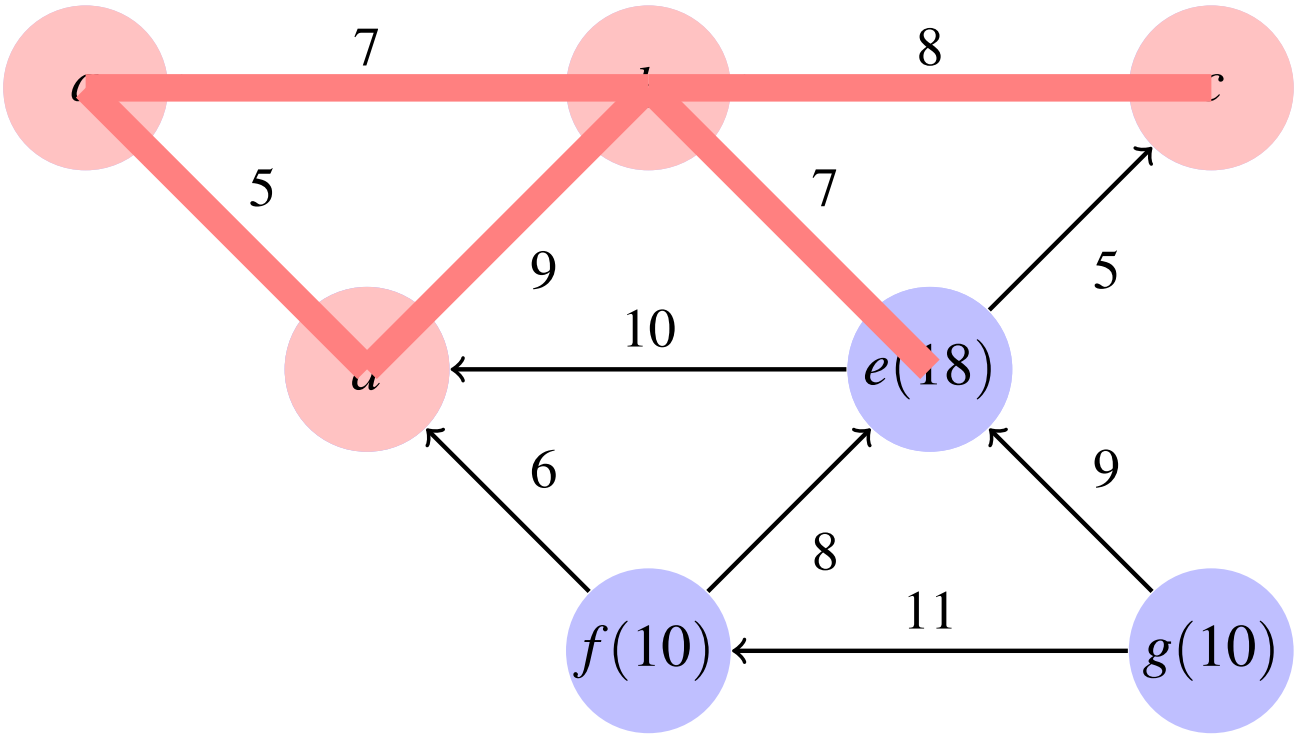
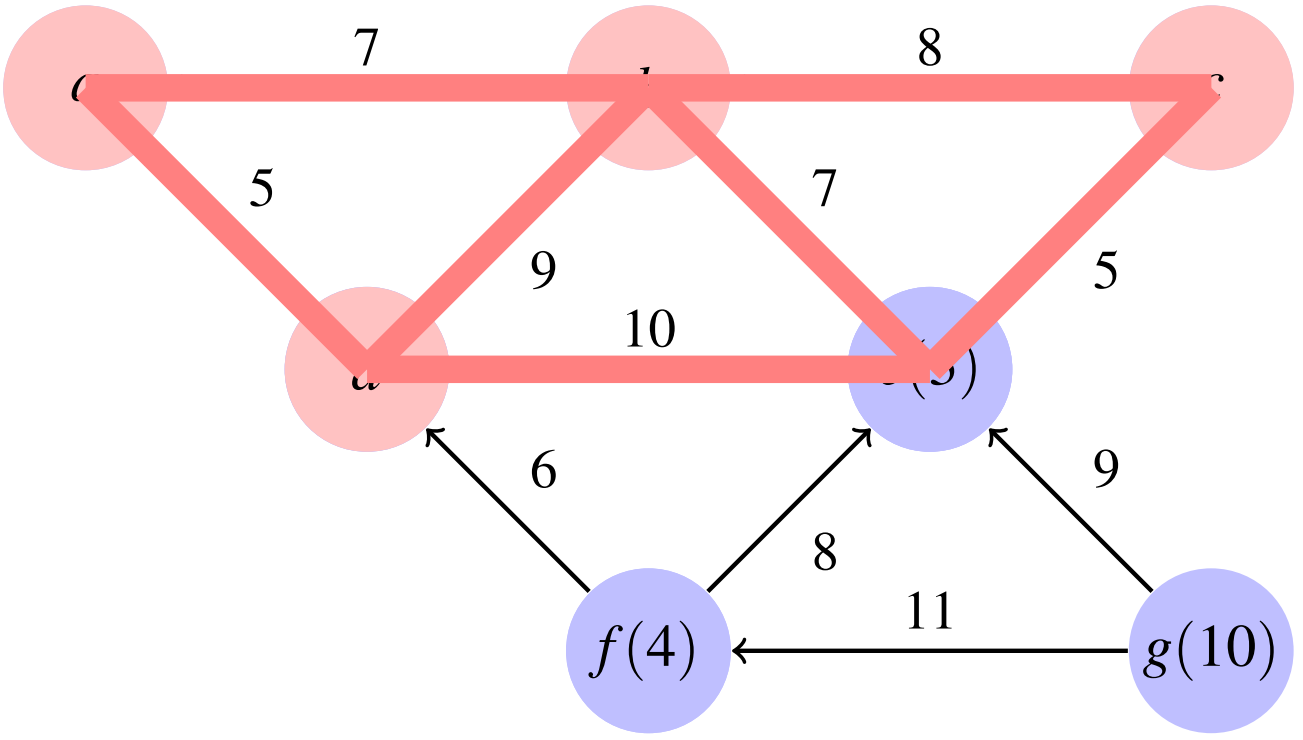
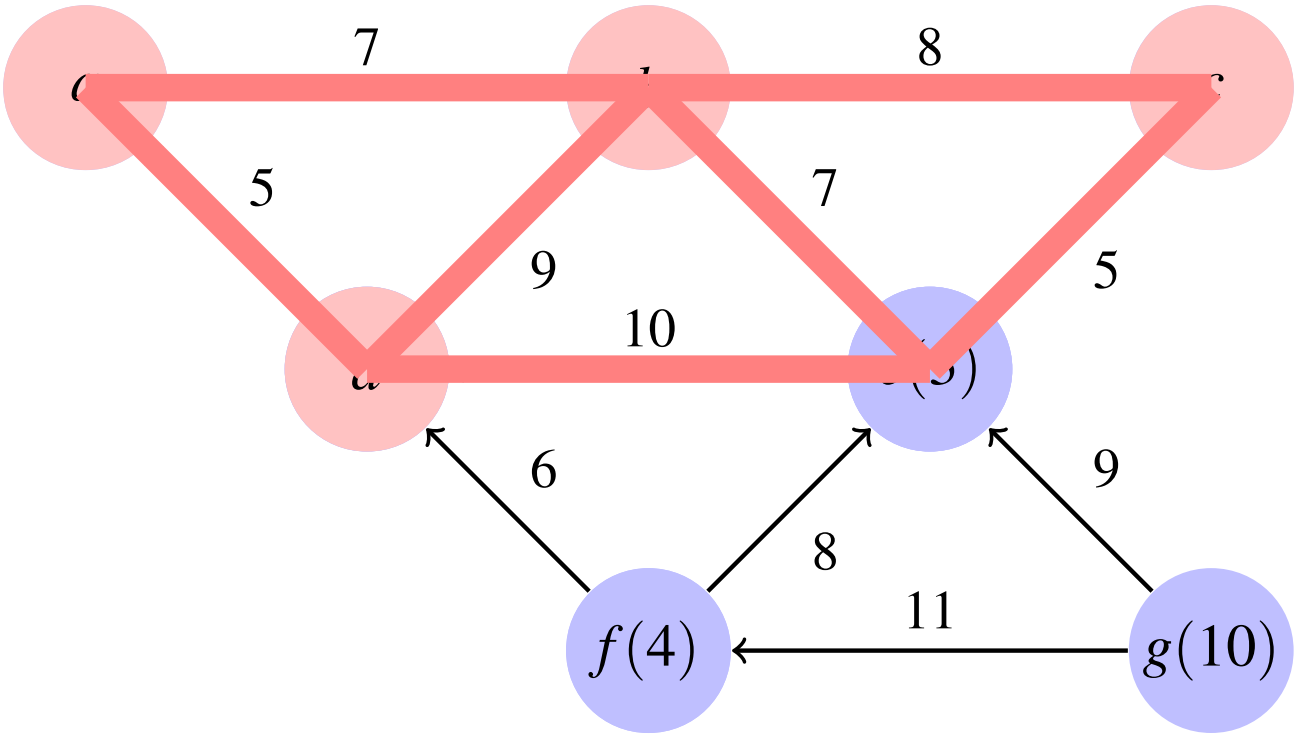


Example



Example

Contagion lasts 3 rounds.
Fundamental defaults: $\{i \mid c^0(i) = 0\} = \{a\}$.
Contagious defaults: $\{i \mid c^0(i) > 0 \;\&\; c^T(i) = 0\} = \{b, c, d\}$.
Total number of defaults: = 4.



Default impact

Definition (Default Impact)

The *Default Impact* $DI(i, c, E)$ of a financial institution $i \in V$ is defined as the total loss in capital in the cascade triggered by the default of i:

$$DI(i, c, E) = \sum_{j \in V} c_0(j) - c_{\text{final}}(j), \tag{3}$$

where $(c_{\text{final}}(j), j \in V)_{k \geq 0}$ is the final level of capital at the end of the cascade with initial condition $c_0(j) = c(j) \quad \text{for} \quad j \neq i \quad \text{and} \quad c_0(i) = 0.$

Default Impact does not include the loss of the institution triggering the cascade, but focuses on the loss this initial default inflicts to the rest of the network: it thus measures the loss due to contagion.

Variants

If one adopts the point of view of deposit insurance, then the relevant measure is the sum of deposits across defaulted institutions:

$$DI(i, c, E) = \sum_{j \in \mathbb{D}(c,E)} Deposits(j).$$

Alternatively one can focus on lending institutions (e.g. commercial banks), whose failure can disrupt the real economy. Defining a set $\mathbb{C}$ of such **core** institutions we can compute

$$DI(i, c, E) = \sum_{j \in \mathbb{C}} c_0(j) - c_{\text{final}}(j)$$

Contagion effects: too rapidly dismissed?

The small magnitude of such "domino" effects has been cited as justification for ignoring contagion (Furfine 2003, Geneva Report 2008).

Such simulations ignore the impact of correlated market shocks on bank balance sheets and, therefore, the compounding effect of market shocks and contagion.

Many studies on domino effects are not based on actual exposures but either look at a subset of exposures (e.g. FedWire) or *estimate/reconstruct* exposures from balance sheet data using maximum entropy methods (Boss et el, Elsinger et al) which result in distributing exposures as uniformly as possible across counterparties. This method can lead to underestimation of contagion effects.

Default impact in a macroeconomic stress scenarios: the Contagion index (Cont, Moussa, Santos 2010)

- Idea: measure the joint effect of economic shocks and contagion by measuring the Default Impact of a node in a macroeconomic stress scenario
- Apply a common shock Z (in % capital loss) to all balance sheets, where Z is a negative random variable
- "Stress scenario" = low values/quantiles of Z
- Compute Default Impact of node *k* in this scenario:

$$DI(\ k,\ c(1+Z)\ ,E)$$

- Average across stress scenarios:

$$CI(k)=E[\ DI(\ k,\ c(1+Z)\ ,E)\ |\ Z < z_q]$$

Forward-looking, based on exposures and stress scenarios

Heterogeneous stress scenarios

Macroeconomic shocks affect bank portfolios in a highly correlated way, due to common exposures of these portfolios.
Moreover, in market stress scenarios fire sales may actually exacerbate such correlations.
In many stress-testing exercises conducted by regulators, the shocks applied to various portfolios are actually scaled version of the same random variable i.e. perfectly correlated across portfolios.
A generalization is to consider co-monotonic shocks generated by a common factor Z:

$$\epsilon(i, Z) = c(i) f_i(Z) \tag{4}$$

f_i are strictly increasing with values in $(-1, 0]$, representing % loss in capital.
A *macroeconomic stress scenario* corresponds to low quantiles α of Z: $\mathbb{P}(Z < \alpha) = q$ where $q = 5\%$ or 1% for example.

The Contagion Index

Definition (Contagion Index)

The Contagion Index $CI(i, c, E)$ (at confidence level q) of institution $i \in V$ is defined as its expected Default Impact in a macroeconomic stress scenario:

$$CI(i, c, E) = \mathbb{E}\left[DI(i, c + \epsilon(Z), E) | Z < \alpha\right] \tag{5}$$

where the vector $\epsilon(Z)$ of capital losses is defined by (**??**) and α is the q-quantile of the systematic risk factor Z: $\mathbb{P}(Z < \alpha) = q$.

Z represents the magnitude of the macroeconomic shock
In the examples given below, we choose for α the 5% quantile of the common factor Z.

Contagion index: simulation-based computation

- Simulate independent values of Z
- Compute Default Impact of node *k* in each scenario as

$$DI(\ k,\ c+\ \varepsilon(Z),E)$$

- Average across stress scenarios given by $Z<\alpha$

$$CI(k)=E[\ DI(\ k,\ c+\varepsilon(Z),E)|\ Z<\alpha]$$

Forward-looking, based on exposures and stress scenarios

Depends on:

- network structure through DI
- Joint distribution F of $\varepsilon(Z)=(\varepsilon_1(Z),\varepsilon_2(Z),\ldots\varepsilon_n(Z))$

Contagion index: empirical results for the Brazilian banking system

In the examples below, we model Z as a negative random variable with a heavy-tailed distribution F and an exponential function for f_i:

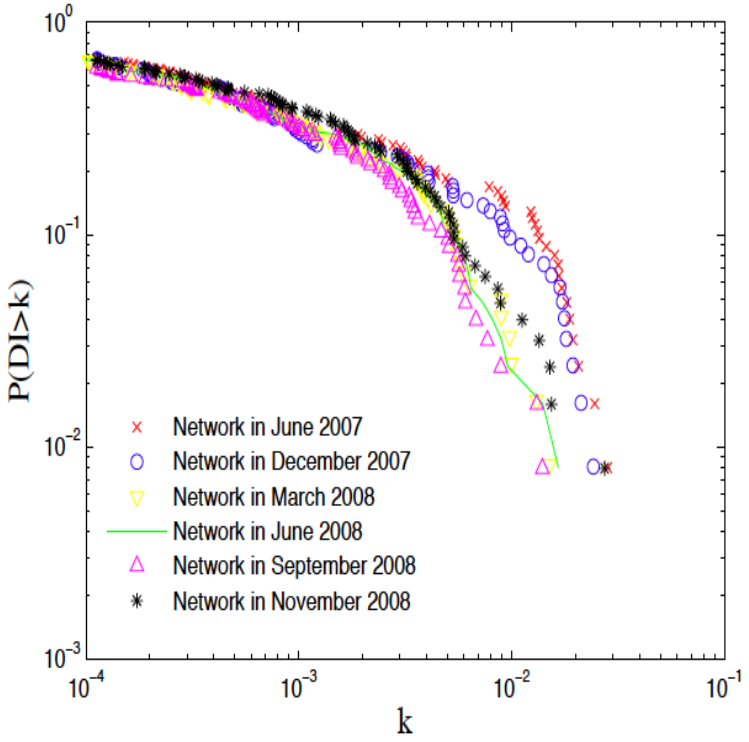
$$\epsilon(i, Z) = c(i)\,(\exp(\sigma_i Z) - 1) \tag{8}$$

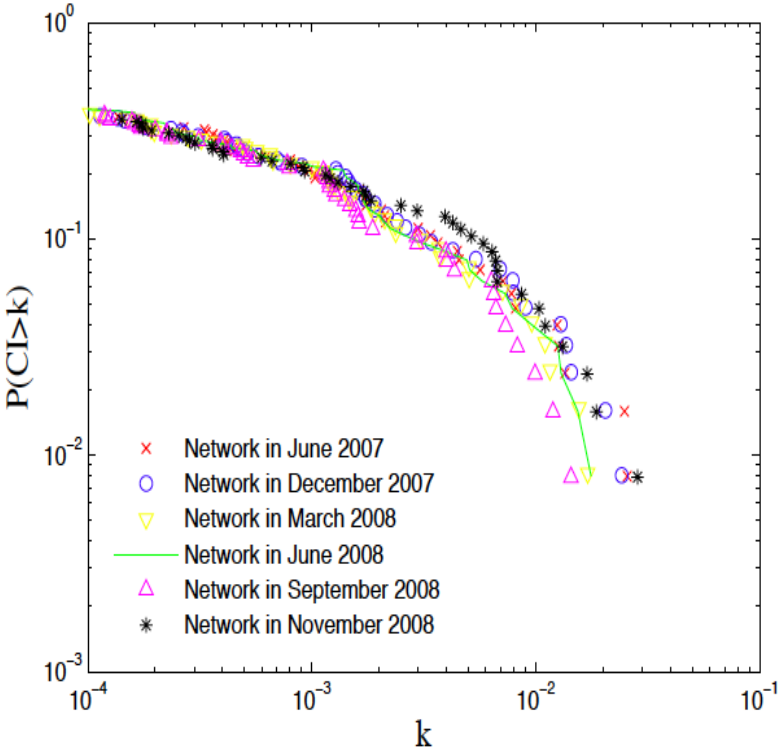
where σ_i is a scale factor which depends on the creditworthiness, or probability of default p_i, of institution i. For example, a possible specification is to choose σ_i such that p_i corresponds to the probability of losing 90% of the Tier 1 capital in a market stress scenario:

$$\sigma_i = -\frac{\log(10)}{F^{-1}(p_i)}. \tag{9}$$

Default probabilities are obtained from historical default rates given by Standard & Poors ratings for the firms at the date corresponding to the simulation.

Contagion index: empirical results for the Brazilian banking system





Empirical results for the Brazilian banking system

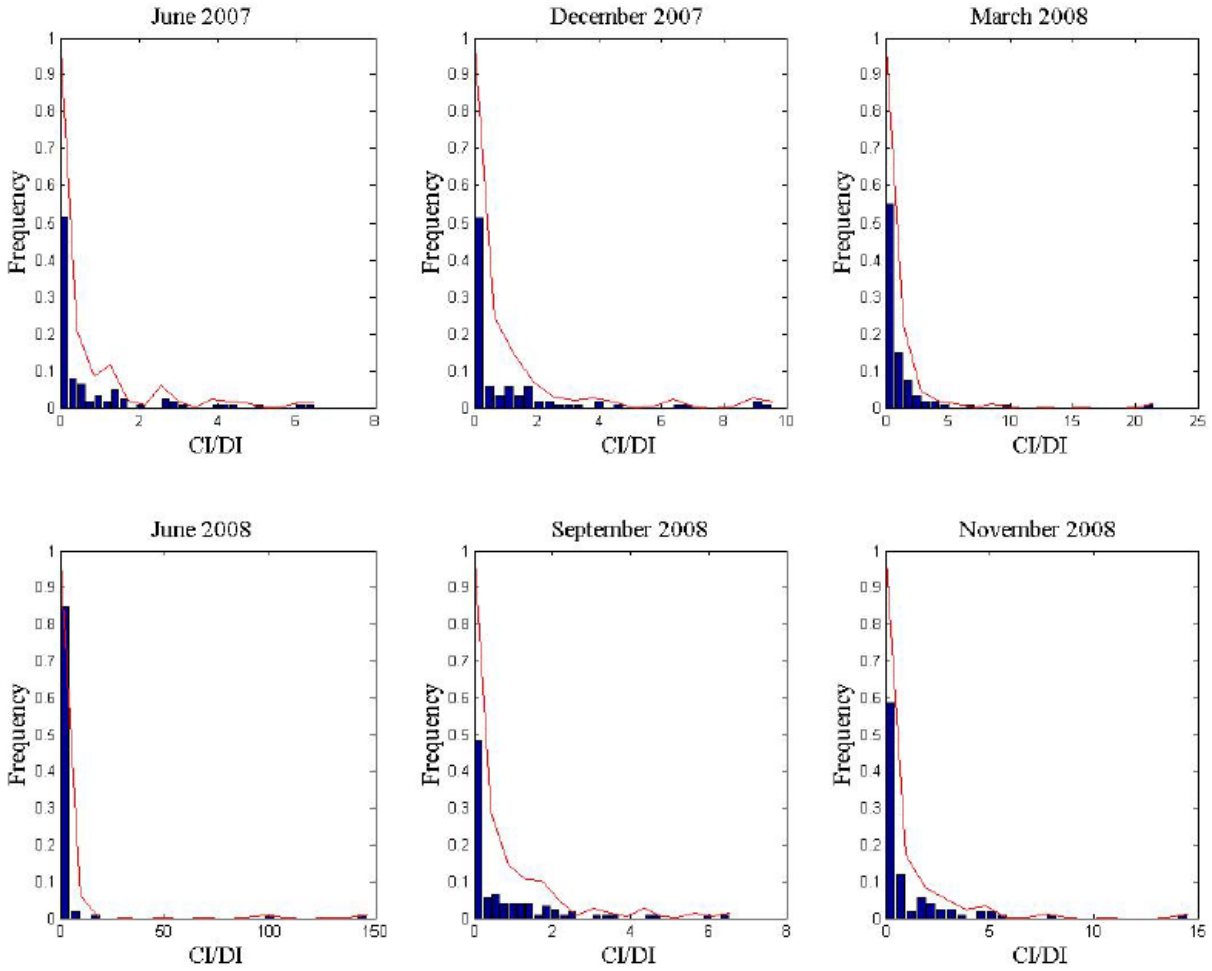


Figure 10: Default impact vs Contagion Index: the Contagion Index can be up to fifteen times larger than the Default Impact for some nodes.

Node	**Contagion index**	**Number of counterparties**	**Total liability**
29	11.31	30	11.84
13	4.45	21	1.12
48	3.58	21	3.32
5	2.67	41	2.13
60	2.38	7	1.45
Network average	0.51	8.98	0.47
Network median	0.08	6.00	0.08

Table 4: Analysis of the five most contagious nodes, when using tier I capital for the time period June 2008. Unit: Billion $.

Role of capital ratios

- Homogeneity: $\forall \lambda > 0, DI(i, \lambda c, \lambda E) = \lambda DI(i, c, E)$.
  Consequence : natural normalization is to express CI, DI as % of total capital
- Monotonicity in capital ratio: Default Impact and Contagion index, as % of initial capital, are (componentwise) increasing functions of ratio of exposures to capital $E(i,j)/c(i)$:

$$\forall i,j \in V, \frac{E(i,j)}{c(i)} > \frac{E'(i,j)}{c'(i)} \Rightarrow \forall k \in V, \frac{DI(k,c,E)}{\sum_i c(i)} \geq \frac{DI(k,c',E')}{\sum_i c'(i)}$$

- BUT: Default Impact and Contagion index are NOT monotone functions of the (usual) capital ratios! One can have

$$\forall i \in V, \frac{\sum_j E(i,j)}{c(i)} > \frac{\sum_j E'(i,j)}{c'(i)} \quad \text{and} \frac{DI(k,c,E)}{\sum_i c(i)} < \frac{DI(k,c',E')}{\sum_i c'(i)}$$

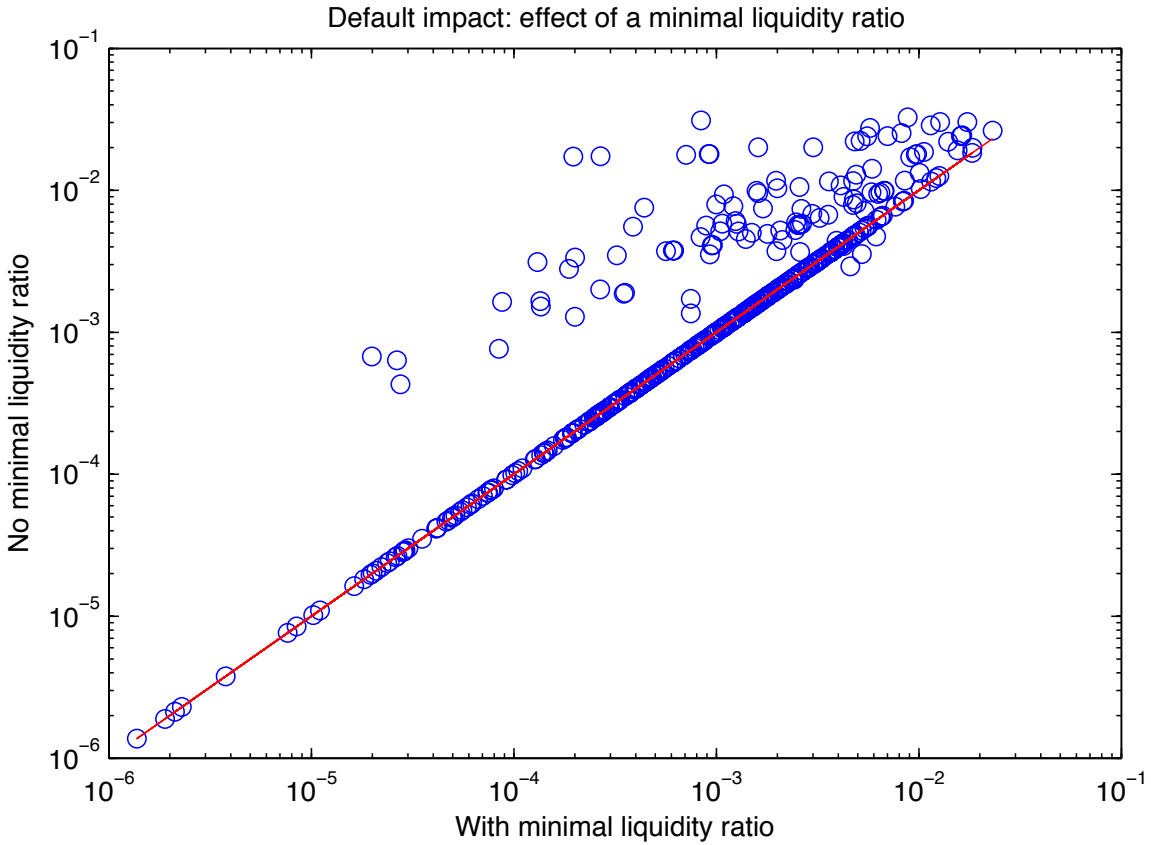


Figure 16: Influence of a minimal capital ratio on default impact: **imposing a minimal capital ratio reduces contagion**.

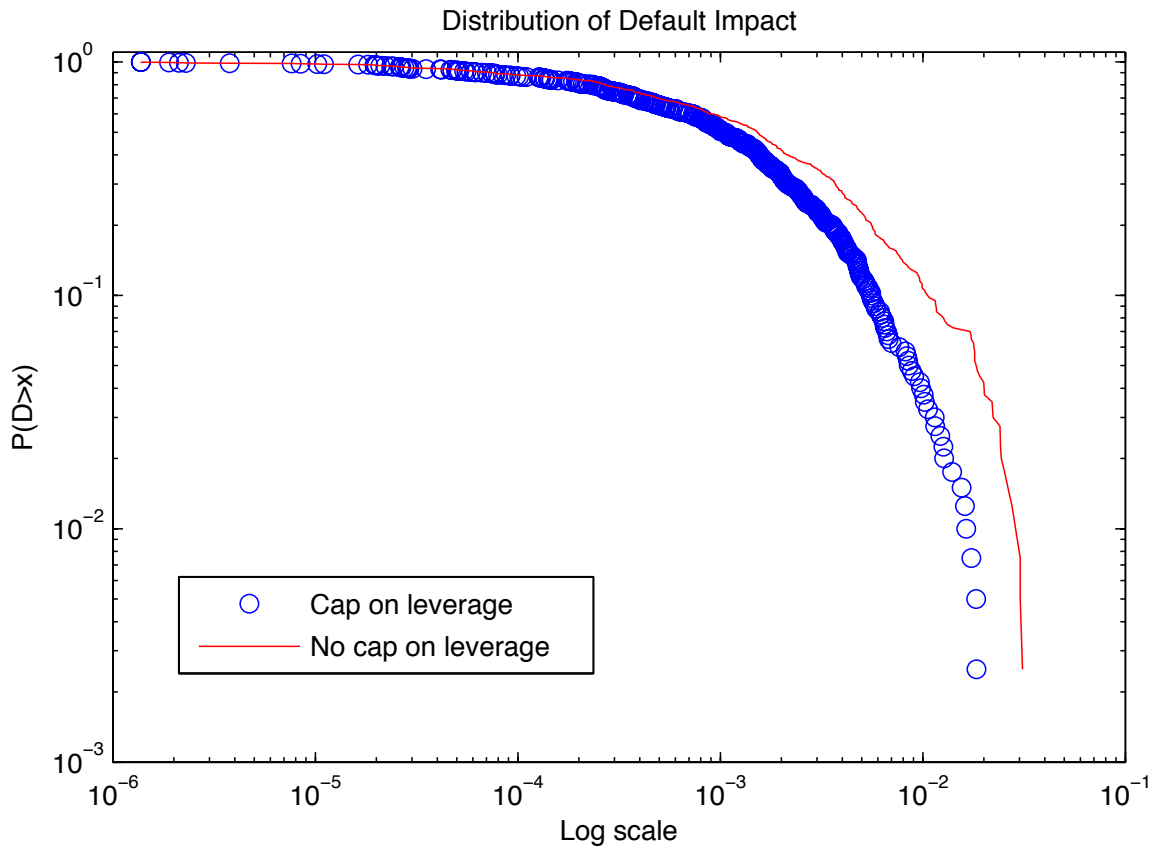


Figure 17: Influence of minimal capital ratio on distribution of default impact: **imposing a minimal capital ratio reduces probability of large systemic losses.**

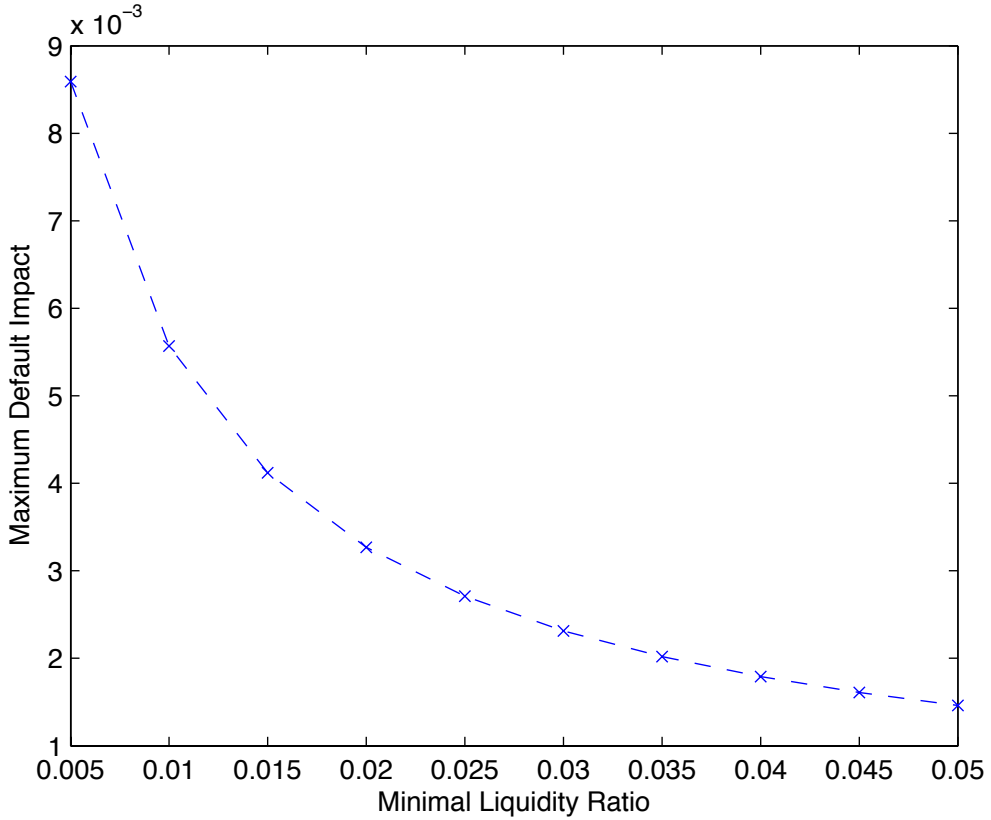


Figure 18: **The worst-case default impact decreases monotonically with the minimal capital ratio**: loss generated by the institution with highest default impact as a function of the minimal ratio of capital to total exposures.

Capital-efficiency of networks

- The lack of monotonicity of the Contagion Index with respect to total capital or capital ratios leads to the question: given a network of exposures and capital allocation, is there a better scheme of capital requirements/allocations which reduces systemic risk (Contagion Indices) without increasing the total level of capital requirements?
- A capital allocation c in the network of exposures E is said to more globally capital-efficient than c' if

$$\sum_i c'(i) > \sum_i c(i) \quad \text{and} \quad \forall k \in V, CI(k, c', E) \leq CI(k, c, E)$$

Such examples exist! But they also arise in empirical data...

Capital requirements and network heterogeneity

- Heterogeneity of network exposures suggests that **homogeneous** capital ratios, as practiced currently, are not necessarily the most efficient.
- Also, the key role played by 'contagious links', defined through high exposure-to-capital ratios $E(i,j)/c(i)$, suggests that the resilience of the network is governed by **concentration of exposures across counterparties**, which the capital ratio

$$\gamma(i) = \frac{c(i)}{\sum_{j \neq i} E(i,j)}$$

  is **not sensitive** to.
- This pleads for capital requirements based not just on $\gamma(i)$ but on the distribution of

$$\left(\frac{c(i)}{E(i,j)}, \quad j \neq i \right)$$

  As an example: capital requirements based on $\max(\frac{c(i)}{E(i,j)}, \quad j \neq i)$

Monitoring nodes or monitoring links?
A new look at capital requirements

Current prudential regulation uses as main tool monitoring and lower bounds for capital ratios defined as c(i)/A(i)

where A(i)= sum of exposures of i+ other assets of i= $\Sigma_j E_{ij}$ +a(i)

Typically a uniform lower bound is imposed on capital ratios for all institutions, *regardless of their size/ systemic risk.*

Capital ratios do not quantify the *concentration* of exposures.

On the other hand:

Simulations show the crucial role of contagious exposures ("weak links") with

$$E_{ij} > c(i) + \varepsilon_i(Z)$$

In other fields (epidemiology, computer network security,..) immunization strategies focus on

- Monitoring or immunizing the most 'systemic' nodes

-strengthening weak links as opposed to uniform or random monitoring.

This pleads for monitoring links representing large relative exposures relative to capital (large value of $E_{ij}/c(i)$)

In a heterogeneous network, this can make a big difference!

Capital requirements targeting 'large exposures'

Our proposal is to target (i.e. impose a lower bound on)

$$\kappa(i) = \max\left(\frac{c(i)}{E(i,j)}, \quad j \neq i\right) \qquad \geq \kappa_{\min}$$

- This has the effect of penalizing the concentration of exposures on a few counterparties.
- Same spirit as regulatory initiatives for monitoring/limiting Large Exposures.

Targeted vs non-targeted capital requirements

We compare, in various empirical and simulated heterogeneous exposure networks, the impact of 4 different capital requirement schemes:

(a) 'Homogeneous' capital ratio:

$$\gamma(i) = \frac{c(i)}{\sum_{j \neq i} E(i,j)} \geq \gamma_{\min}$$

(b) Targeted capital ratios: higher ratios for 5% 'most systemic' institutions (SIFIs):

$$\gamma(i) = \frac{c(i)}{\sum_{j \neq i} E(i,j)} \geq \Gamma_{\min} \qquad \text{if} \quad CI(k) \geq VaR(CI, 95\%)$$

(c) Capital-to-exposure ratio for all institutions:

$$\kappa(i) = \max\left(\frac{c(i)}{E(i,j)}, \quad j \neq i\right) \qquad \geq \kappa_{\min}$$

(d) Capital-to-exposure ratio for SIFIs (5% 'most systemic' institutions):

$$\kappa(i) = \max\left(\frac{c(i)}{E(i,j)}, \quad j \neq i\right) \quad \geq \kappa_{\min} \qquad \text{if} \quad CI(k) \geq VaR(CI, 95\%)$$

Targeted vs non-targeted capital requirements

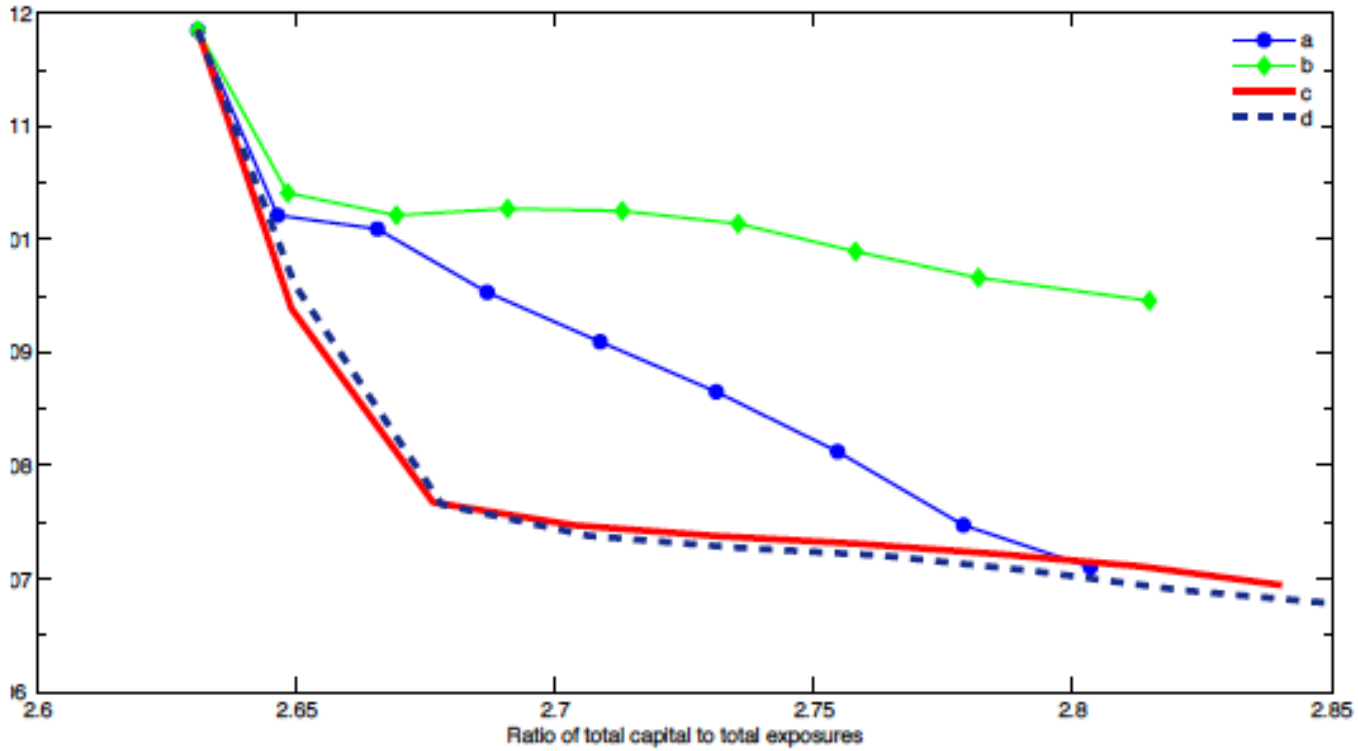
For each scheme, we vary the threshold/limit imposed on the ratios and examine

- The aggregate capital requirement across nodes $\sum_i c(i)$
- The average of Contagion Indices for 5% most systemic nodes $=$ 5% Tail Conditional Expectation $TCE(CI(c,E),5\%)$ of the cross-sectional distribution of the Contagion index.

We will consider a capital requirement $c' = (c'(i), i \in V)$ as more efficient in reducing systemic risk than $c = (c(i), i \in V)$ if it reduces the potential losses from the failure of most systemic institutions without increasing the aggregate level of capital requirements:

$$\sum_{i\in V} c'(i) \leq \sum_{i\in V} c(i) \quad \text{while} \quad TCE(CI(c',E),5\%) < TCE(CI(c,E),5\%)$$

Focusing on weak links: targeted capital requirements



Comparison of various capital requirement policies: (a) minimum capital ratio for all institutions in the network, (b) minimum capital ratio only for the 5% most systemic institutions, (c) uniform capital-to-exposure ratio (d) capital-to-exposure ratio for the 5% most systemic institutions.
(Cont Moussa Santos 2010)

Homogeneous vs inhomogeneous capital requirements

- Targeting SIFIs with higher capital ratios may reduce their probability of failure but, for the aggregate level of capital, does not reduce (in fact, increases) the systemic losses *conditional* on a SIFI failure.
- On the other hand, a lower bound capital-to-exposure ratio has a stronger mitigating effect on systemic losses due to SIFI defaults than a simple capital ratio: it allocates capital in order to strengthen the 'weakest links' in the network.

Summary:

- (i) focus on (weak) links, not nodes.
- (ii) Heterogeneity of network structure makes homogeneous ratios inefficient.

Currently regulators are considering the 'cover one counterparty' rule which amounts to a limit of 100% on capital-to-exposure ratio.
Note that enforcing such a rule does **not** require to observe the entire network.
Implementation involves other issues, in particular identification/tracking of the ultimate beneficiary in financial transactions.

Role of macro-shocks and diversification

Stress scenarios triggered by large values of a risk factor Z:

$$\epsilon(i, Z) = c(i) f_i(Z) \tag{6}$$

$f_i(Z)$ represents the exposure of bank i to this risk factor.

- Monotonicity wrt macro-shocks: greater $|f_i|$ leads to greater values of Contagion Index.
- Contribution of macro shocks to $CI(k, c, E)$ is limited to he set $\{i, f_i(Z).f_k(Z) > 0\}$: tis set is smallest in totally segmented markets, and its size increases with diversification.
- Worst case: in a totally 'globalized'/ diversified market $\{i, f_i(Z).f_k(Z) > 0\} = V$
- Consequence: large-scale diversification increases exposure to systemic risk!
- Diversification reduces the 'volatility'/ marginal risk measure of bank portfolios in non-stress scenarios but.. increases the probability of joint losses in stress scenarios generated by the common risk factor(s) so increases the possibility of contagion.

Conclusion

- Exposures across financial institutions reveal a highly concentrated and heterogeneous network structure with some highly connected large nodes.
- As a consequence of this heterogeneity: contagion can be triggered by a local shock on a node yet result in largesystem-wide losses, concentrated a small sub-graph of **contagious links** between critically important nodes.
- Use metrics based on conditional loss, not expected loss or probability of contagion.
- Homogeneous models may lead to *incorrect insights* on systemic risk.
- **Monitor links, not just nodes** simple indicators based on the **ratio of the largest exposure to capital** can provide a more efficient instrument for monitoring and regulating contagion risk, *without* requiring a detailed observation of network structure.

Contagion in large counterparty networks: analytical results

- Amini, Cont, Minca (2010): mathematical analysis of the onset and magnitude of contagion in a large counterparty network (n->∞)
- Main point: contagion may become large-scale if

$$\sum_{j,k} \frac{\mu(j,k)\, jk}{\lambda} q(j,k) > 1$$

where

μ(j,k)= proportion of nodes with with j debtors, k creditors

λ = average number of counterparties

q(j,k) : fraction of *overexposed* nodes with (j,k) links,

= fraction of nodes with degree (j,k) such that at least ONE exposure exceeds capital

Some questions

Default impact and the amplitude of solvency cascades have been studied by central banks via large scale simulations.

Simulation results indicate a high degree of dependence on the structure of the network but seem difficult to generalize in absence of further insight.

- How does network structure –level of capital requirements, magnitudes and distribution of exposures– influence default contagion in a financial networks?
- Which characteristics of a node/ subnetwork render it dangerous from the point of view of systematic risk?
- How can contagion risk be effectively monitored by the regulator?

Similar issues have been studied in mathematical epidemiology and percolation theory either

-theoretically in a **homogeneous** and/or non-weighted graphs (Bollobas et al, Janson et al, Balogh & Pittel, Wormald,...)

-using simulations or mean-field approximations (Watts & Strogatz, Gai & Kapadia).

Most studies focus on **undirected** graphs.

We tackle these questions analytically, for the case of a general heterogeneous, directed, weighted network, using **asymptotics** and *probabilistic methods*.

The relevance of asymptotics

Most financial networks are characterized by a large number of nodes: FDIC data include several thousands of financial institutions in the US.

To investigate contagion in such large networks, in particular the scaling of contagion effects with size, we can embed a given network in a sequence of networks with increasing size and studying the behavior/scaling of relevant quantities (cascade size, total loss, impact of regulatory policy) when network size increases.

A *probabilistic approach* consists in

-building an ensemble of random networks of which our network can be considered a typical sample

-showing a limit result (convergence in probability or almost sure convergence) of the relevant quantities in the ensemble considered as $n \to \infty$

Analysis of cascades in large networks

We describe the topology of a large network by the joint distribution $\mu_n(j,k)$ of in/out degrees and assume that μ_n has a limit μ when graph size increases in the following sense:

1. $\mu_n(j,k) \to \mu(j,k)$ as $n \to \infty$: the proportion of vertices of in-degree j and out-degree k tends to $\mu(j,k)$).
2. $\sum_{j,k} j\mu(j,k) = \sum_{j,k} k\mu(j,k) =: m \in (0,\infty)$ (finite expectation property);
3. $m(n)/n \to m$ as $n \to \infty$ (averaging property).
4. $\sum_{i=1}^{n} (d_{n,i}^{+})^2 + (d_{n,i}^{-})^2 = O(n)$ (second moment property).

A random network model for asymptotics

To embed out networks in an ensemble of networks with increasing size, we use the *configuration model*

Given a sequence of in/out degrees $d^+_{n,i}$ and out-degrees $d^-_{n,i}$ and exposure matrices (E^n_{ij}), we generate a random ensemble of networks with the same degree sequence by randomly permuting the exposures across links going out of each node

This construction generates random networks with the *same* degree sequences and *same* distribution of exposures, which can be both specified from data.

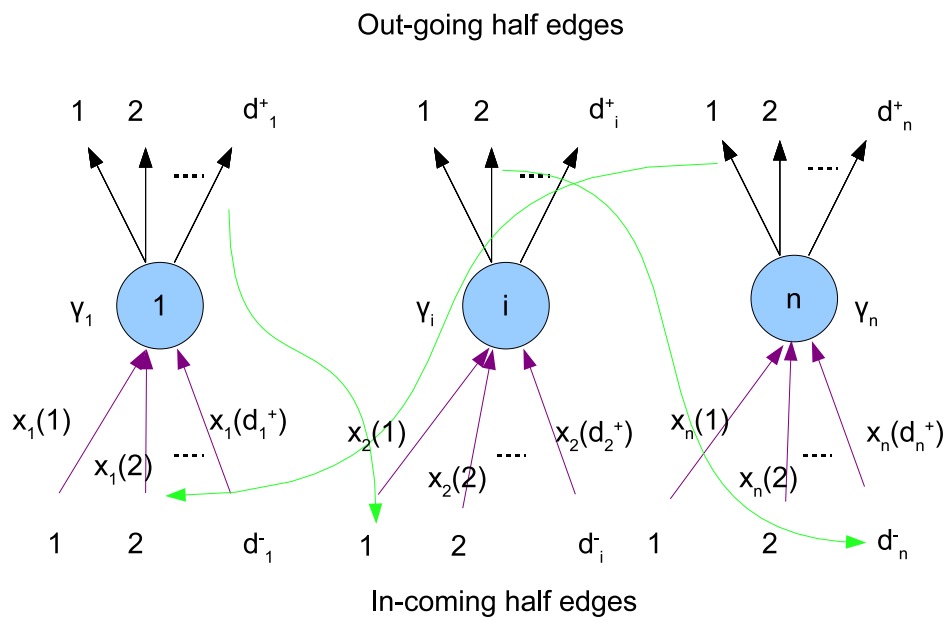


Figure 14: Random configuration model: random matching of incoming half-edges with weighted out-going half-edges.

Contagious links: $i \to j$ is a contagious link if the default of i generates the default of j.

For each node i and permutation $\tau \in \Sigma_{d^+(i)}$, we define

$$\Theta(i,\tau) := \min\{k \geq 0, c_i < \sum_{j=1}^{k} E^n_{i,\tau(j)}\}$$

$\Theta(i,\tau)$ = number of counterparty defaults which will generate the default of i if defaults happen in the order prescribed by τ:

$$p_n(j,k,\theta) := \frac{\#\{(i,\tau) \mid \tau \in \underbrace{\Sigma_j}, \ d_i^{(n)+} = j, \ d_i^{(n)-} = k, \ \Theta(i,\tau) = \theta\}}{n\mu_n(j,k)j!}.$$

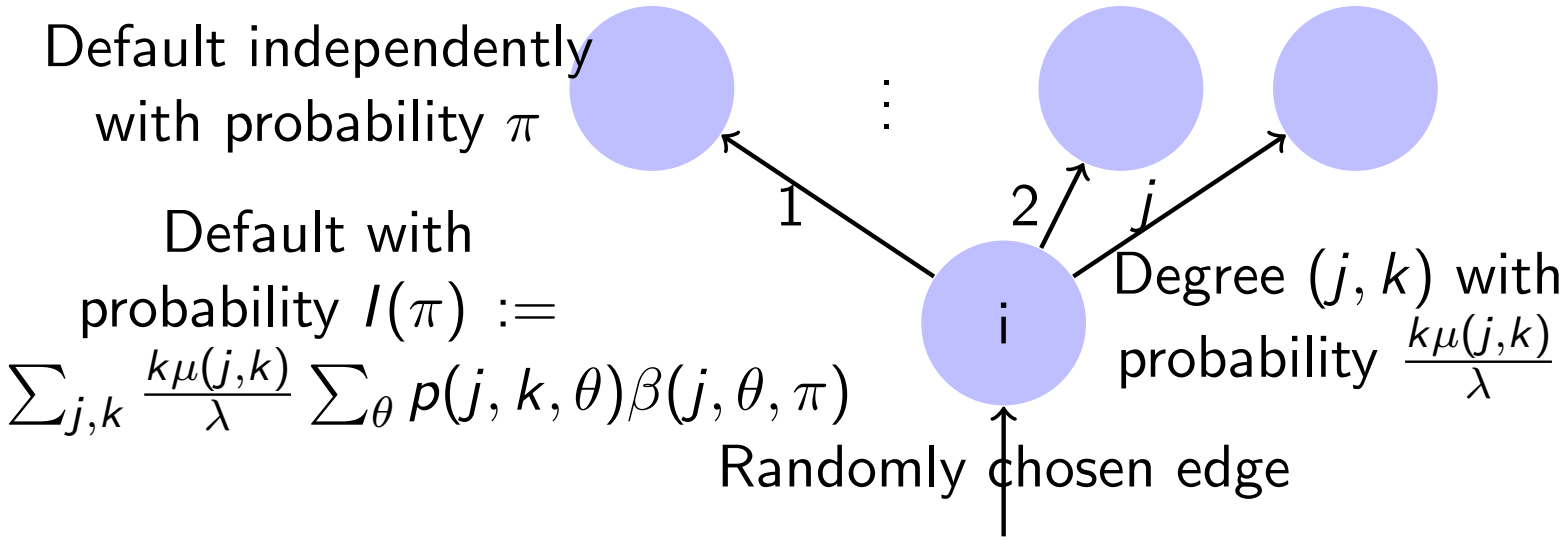
$n\mu_n(j,k)jp_n(j,k,1)$ is the total number of contagious links that enter a node with degree (j,k).

The value $p_n(j,k,1)$ gives the proportion of contagious links ending in nodes with degree (j,k).

The intuition :branching process approximation

Let us define

$$\beta(n,\pi,\theta) := \mathbb{P}(Bin(n,\pi) \geq \theta) = \sum_{j \geq \theta}^{n} \binom{n}{j} \pi^j (1-\pi)^{n-j}.$$



The main idea is that, in a large network, a default cascade is well approximated by an inhomogeneous branching process with rate

$$I(\pi) := \sum_{j,k} \hat{\mu}(j,k) \sum_{\theta=0}^{j} p(j,k,\theta) \overbrace{\beta(j,\pi,\theta)}^{\text{binomial factor}} \quad . \tag{3}$$

which represents the fraction of defaults after one iteration in a cascade where initially a fraction π of nodes default independently.

In particular, the contagion rate in the limit will be a fixed point of I.

$I : [0,1] \mapsto [0,1]$ always has a fixed point. Let π^* be the smallest fixed point of I in $[0,1]$:

$$\pi^* = \min\{\pi \in [0,1] | I(\pi) = \pi\}.$$

Assumptions on the exposure sequence

There exists a function $p : \mathbb{N}_+^3 \to [0,1]$ such that for all $j, k, \theta \in \mathbb{N}$ $(\theta \leq j)$

$$p_n(j,k,\theta) \overset{n\to\infty}{\to} p(j,k,\theta).$$

as $n \to \infty$. Then, $p(j,k,\theta)$ represents the fraction of nodes with degree (j,k) and default threshold θ.
This assumption is fulfilled for example in a model where exposures are exchangeable arrays.

Proposition 1 (Asymptotic fraction of defaults)**.** *Under the above assumptions:*

1. *If $\pi^* = 1$, i.e. if $I(\pi) > \pi$ for all $\pi \in [0, 1)$, then an initial default of a finite subset leads to global cascade where asymptotically all nodes default.*

$$\frac{|D(A, c_n, E_n)|}{n} \xrightarrow{p} 1$$

2. *If $\pi^* < 1$ and furthermore π^* is a stable fixed point of I, then the asymptotic fraction of defaults*

$$\frac{|D(A, c_n, E_n)|}{n} \xrightarrow{p} \sum_{j,k} \mu(j, k) \sum_{\theta=0}^{j} p(j, k, \theta) \beta(j, \pi^*, \theta).$$

Resilience to contagion This leads to a condition on the network which guarantees absence of contagion:

Proposition 2 (Resilience to contagion)**.** *Denote* $p(j,k,1)$ *the proportion of contagious links ending in nodes with degree* (j,k)*. If*

$$\sum_{j,k} k \frac{\mu(j,k)\ j}{\lambda} p(j,k,1) < 1 \tag{11}$$

then with probability $\to 1$ *as* $n \to \infty$*, the default of a finite set of nodes cannot trigger the default of a positive fraction of the financial network.*

The skeleton of contagious links

The converse also holds :

Proposition

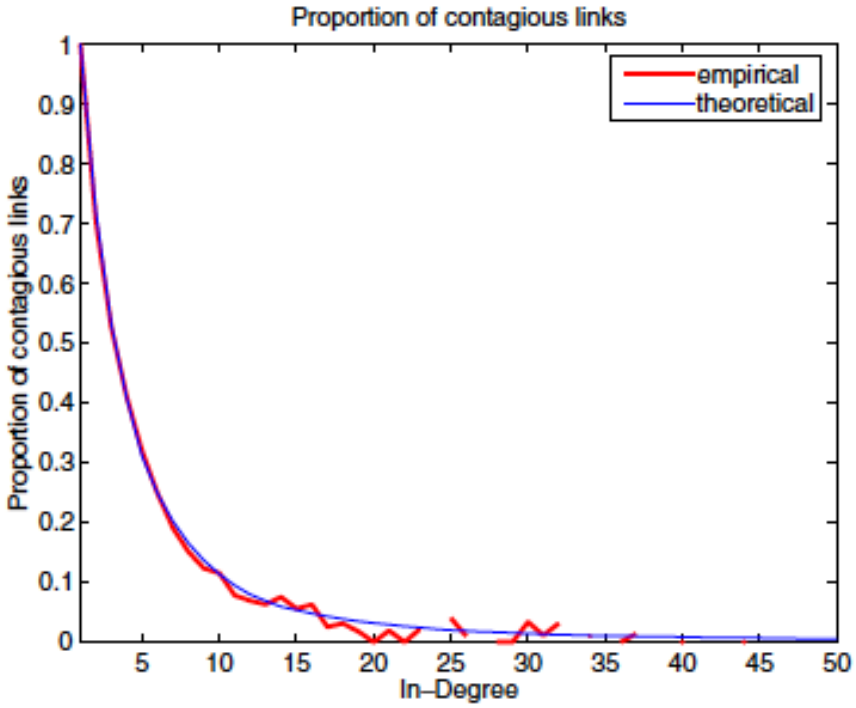
If

$$\sum_{j,k} jk \frac{\mu(j,k)}{\lambda} p(j,k,1) > 1,$$

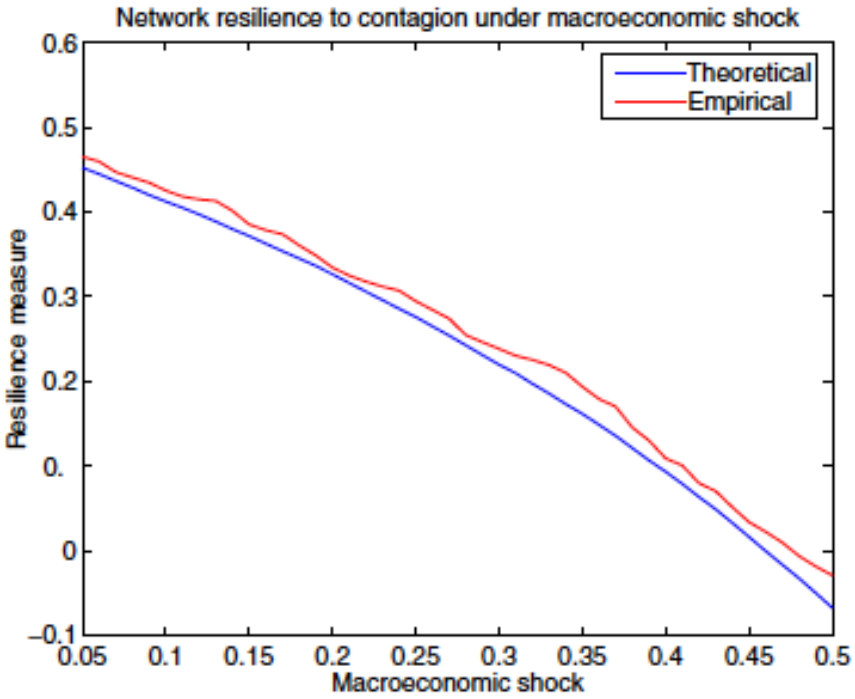
then there exists a connected set χ_n of nodes representing a positive fraction of the financial system, i.e. $|\chi_n|/n \xrightarrow{p} c > 0$ such that, with high probability, any node belonging to this set can trigger the default of all nodes in the set : for any sequence $(c_n)_{n\geq 1}$ such that $\{i, c_n(i) = 0\} \cap \chi_n \neq \emptyset$,

$$\liminf_n \alpha_n(E_n, c_n) \geq c > 0.$$

The relevance of asymptotics



(a)



(b)

Rama CONT: Contagion and
systemic risk in financial networks

Resilience condition:

$$\sum_{j,k} k \frac{\mu(j,k)\ j}{\lambda} p(j,k,1) < 1 \tag{12}$$

This leads to a *decentralized recipe* for monitoring/regulating systemic risk: monitoring the capital adequacy of each institution with regard to its *largest exposures*.

This result also suggests that one need not monitor/know the *entire* network of counterparty exposures but simply the *skeleton/* subgraph of contagious links.

It also suggests that the regulator can efficiently contain contagion by focusing on fragile nodes -especially those with high connectivity- and their counterparties (e.g. by imposing higher capital requirements on them to reduce $p(j,k,1)$).

Contagion process as a Markov chain

Step 1 : description of contagion as a Markov chain

Let $S_n^{j,k,\theta,l}(t)$, $l < \theta \leq j$ be the number of solvent banks with degree (j,k), default threshold θ and l defaulted debtors before iteration t.
Then $(S_n^{j,k,\theta,l}(t), l < \theta \leq j)$ is a Markov chain.
We introduce the additional variables of interest in determining the size and evolution of contagion :

- $D_n^{j,k,\theta}(t)$, the number of defaulted banks at time t with degree (j,k) and default threshold θ,
- $D_n(t)$: the number of defaulted banks at time t,
- $D_n^-(t)$: the number of remaining in-coming edges belonging to defaulted banks.

Transition probabilities

Proposition : $\mathbf{Y}(t) = \left(D^{j,k,\theta}(t), S_l^{j,k,\theta}(t)\right)_{j,k,0\leq l<\theta\leq j}$ is a Markov chain.

Choose an available in-coming half edge belonging to a defaulted node A. Let B be its counterparty.

- B is defaulted, the next state is $\mathbf{Y}(t+1) = \mathbf{Y}(t)$.
- B is solvent of degree (j,k), default threshold θ and this is the $(l+1)$-th deleted out-going edge and $l+1<\theta$. The probability of this event is $\frac{(j-l)S_l^{j,k,\theta}(t)}{m_n - t}$. The changes for next state will be

$$S_l^{j,k,\theta}(t+1) = S_l^{j,k,\theta}(t) - 1,$$
$$S_{l+1}^{j,k,\theta}(t+1) = S_{l+1}^{j,k,\theta}(t) + 1.$$

- B is solvent of degree (j,k), default threshold θ and this is the θ-th deleted out-going edge. Then with probability $\frac{(j-\theta+1)S_{\theta-1}^{j,k,\theta}(t)}{m_n-t}$ we have

$$D^{j,k,\theta}(t+1) = D^{j,k,\theta}(t) + 1,$$
$$S_{\theta-1}^{j,k,\theta}(t+1) = S_{\theta-1}^{j,k,\theta}(t) - 1.$$

Step 2 : Law of Large Numbers : as $n \to \infty$

$$\frac{Y(n\tau)}{n} \xrightarrow{p} y(\tau)$$

where $y(\tau)$ is the solution of a system of ordinary differential equations, which correspond to the non-homogenous spatial branching process previously described.

Step 3 These differential equations can be solved in closed form, yielding the stability/resilience criterion.

A measure for the resilience of a financial network

- Stress scenario: apply a common macro-shock Z, measured in % loss in asset value, to all balance sheets in network
- The fraction q(j,k,Z) of *overexposed* nodes with (j,k) links is then an increasing function of Z
- Network remains resilient as long as $\sum_{j,k} \frac{\mu(j,k)\,jk}{\lambda} q(j,k,Z) < 1$

DEFINITION: Network Resilience = maximal shock Z* network can bear while remaining resilient to contagion

Z* is solution of

$$\sum_{j,k} \frac{\mu(j,k)\,jk}{\lambda} q(j,k,Z) < 1$$

Given network data, Z* computed by solving single equation

Simulation-free stress testing of banking systems

- These analytical results may be used for stress-test the resilience of a banking system, *without* the need for large scale simulation.
- Stress scenario: apply a common macro-shock Z, measured in % loss in asset value, to all balance sheets in network
- Analytical result allow to compute fraction of defaults as function of Z
- Network remains resilient (no macro-cascade) as long as

$$\sum_{j,k} \frac{\mu(j,k)jk}{\lambda} q(j,k,Z) < 1 \Leftrightarrow Z < Z^*$$

An abrupt transition from resilience to non-resilience occurs when shock amplitude reaches Z*: cascade size/ number of defaultsas function of initial shock Z is discontinuous at Z=Z*

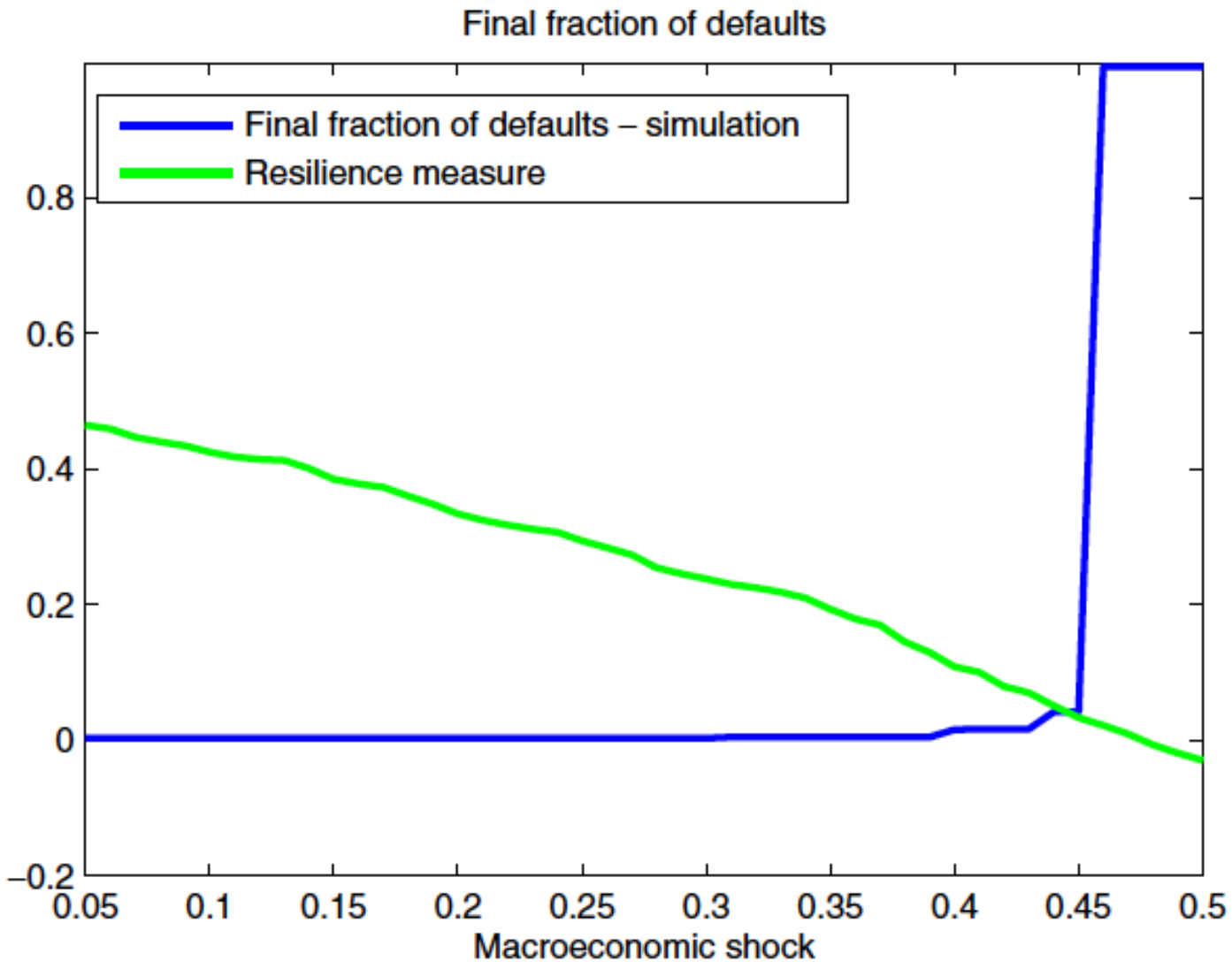
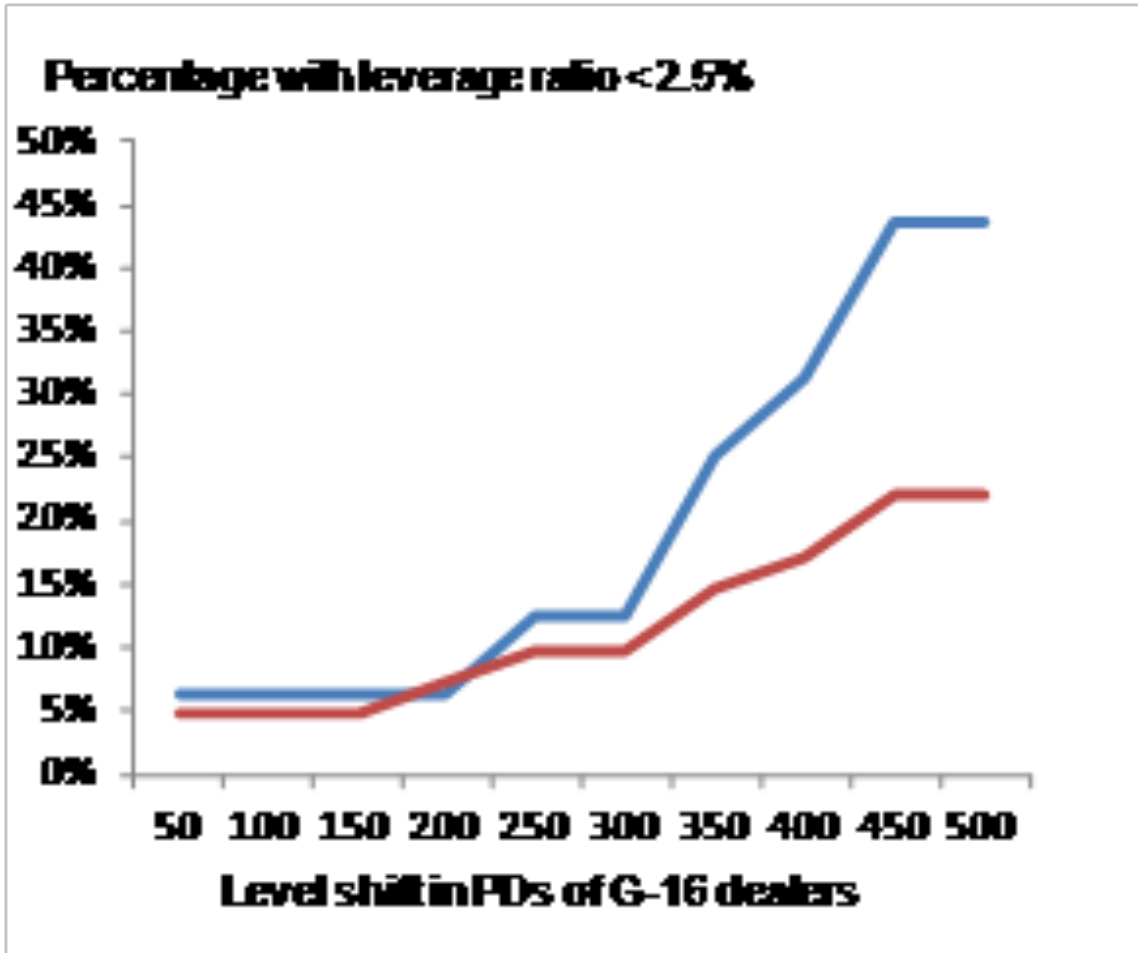


Fig. 7. Final fraction of defaults triggered by an initial fraction of defaults representing 0.1% of the total network.

Stress test of the international OTC dealer network (2013)



Rama CONT: Contagion and systemic risk in financial networks

Interconnectedness and systemic risk: key insights from mathematical modeling

- **Network structure does matter** when studying reisilience and stability of the finacial system: a meaningful model for financial stability should integrate network structure at level of financial institutions exposures.
- Metrics of network resilience/ stability should make use of exposures AND capital levels and should be based on loss contagion, even in absence of default.
- Use **conditional metrics**, which look at system-wide losses conditional on a loss/default/macro-event
- Theory provides links between network structure and resilience and points to key role played by large exposures relative to capital which consitute the weak links along which contagion takes place.
- This pleads to targeted capital requirements based on large exposures, rather than aggregate asset value. Idea: **monitor ratio of large exposures to capital**.
- Network resilience varies ABRUPTLY ('discontinously') with the level of macroeconomic stress/shocks: onset of contagion is 'abrupt'.
- Beware of heterogeneity: Simple, homogeneous models may provide incorrect insights on the link between interconnectedness and systemic risk. In particular increased connectivity may increase or decrease resilience/stability, depending on network details.

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