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Risk Sharing and Contagion**

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Abstract

We provide a general characterization of diffusion processes, allowing to analyze both risk-sharing and contagion at the same time. We show that interdependencies are beneficial when the economic environment is favorable, and detrimental when the economic environment deteriorates. The risk of contagion increases the volatility of outcome and thus reduces the ability of the network to provide risk-sharing.

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1 Introduction

Is an interconnected world a *safer* or a *more dangerous* place to live in? We refer to a *danger* as the risk — both at an individual or at a group level — of being hit by a negative outcome that is generated, either randomly or purposely, somewhere in the economy. There are two essential ways in which outcomes — which can be either material (goods and services, money, *etc.*) or immaterial (information, knowledge, risk or more generally the probability of specific events, *etc.*) flows — 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 spread over. The first process implies *division* of the original quantity, while the latter implies *multiplication*: in the first, total quantity is preserved; in the latter it is increased. We will refer to the first as *risk-sharing*, and to the latter as *contagion*.¹

The two topics have been investigated separately in the literature. The analysis of risk-sharing in networks [Bloch et al., 2006, Bramoullé and Kranton, 2005, e.g.] 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.

Contagion and in particular information cascades have commanded an even greater attention in the literature, 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 the individual level (the amount of the shock that is transferred 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.

¹Note that, starting from a situation where all nodes are homogeneous and a shock hits one node, both mechanisms imply that the shocked node and its neighbors become more alike as a result of the interaction, although in a radically different way: the first 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.

Moreover, in our model individuals have no choice on whether to join the process.

The main contribution of our paper is to provide a general characterization of diffusion processes, allowing to analyze both risk-sharing and contagion at the same time. As a first step, we consider the configuration of the network as exogenous. Moreover, we restrict our attention to the extreme cases of fully connected and fully disconnected graphs. This should provide two benchmarks for the analysis of intermediate cases, which we leave for future research.

2 A prototypical model

There are n croppers, each subject to a stochastic return X_i . They can either live in autarchy, and consume X_i , or pool together their output, and consume $\bar{X} = \sum_i X_i/n$. If they consume less than the starvation threshold θ (normalized to 0) they die. They cannot save for future consumption. In isolation, each X_i follows an independent distribution with $E(X_i) = \mu_i$ and $Var(X_i) = \sigma_i^2 > 0$. When connected, each X_i follows a distribution with $E(X_i) = \mu_i$ and $Cov(X_i, X_j) = \rho_{ij}\sigma_i\sigma_j$, $\rho_{ij} \geq 0 \forall i, j$.

Hence, pooling allows to hedge against the risk of negative outcomes. On the other hand, pooling introduces the possibility of contagion: individual returns can be positively correlated. If a negative outcome occurs somewhere in the economy, its effects are magnified.

In the sharecropping example, contagion might happen because all croppers store their seeds together and they get damaged or lost, or because they all adopt a technology (the seed quality, for instance) that turns out to be inappropriate. With a related metaphor, putting all the eggs in the same basket exposes to the risk of something happening in the basket, or to the basket, which affects all eggs at the same time. The shock to each individual egg is no more independent of the shocks to the other eggs.

The case when $\rho_{ij} = 0, \forall i, j, i \neq j$ corresponds to the case when the risk of contagion is null. Note that even when risk-sharing alone is considered the fate of each individual is not independent of the fate of others, since everybody gets an equal share of the total outcome: *production* is an independent process, while *consumption* is not.

Moreover, note that, on average, contagion is neutral 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.

Define the following average quantities:

$$\mu = \sum_i \mu_i / n \quad (1)$$

$$\sigma = \sum_i \sigma_i^2 / n \quad (2)$$

$$\rho = \frac{2 \sum_i \sum_{j>i} \rho_{ij} \sigma_i \sigma_j}{(n-1) \sum_i \sigma_i^2} \quad (3)$$

In general, we have $E(\bar{X}) = \mu$ and $Var(\bar{X}) = \frac{\sum_i \sigma_i^2 + 2 \sum_i \sum_{j>i} \rho_{ij} \sigma_i \sigma_j}{n^2} = \frac{\sigma^2 + (n-1)\rho\sigma^2}{n}$.

The expected number of failures is $D_{aut} = \sum_i P(X_i < 0)$ in autarchy, while is $D_{pool} = nP(\bar{X} < 0)$ when resources are pooled. Define the ratio between the two as:

$$r = \frac{D_{pool}}{D_{aut}} = \frac{nP(\bar{X} < 0)}{\sum_i P(X_i < 0)} \quad (4)$$

3 Risk-sharing

In the case of no contagion the following proposition holds:

Proposition 1 *If individual returns are independently distributed and n is large, pooling is beneficial ($r < 1$) if and only if $\mu > 0$, and is detrimental ($r > 1$) if and only if $\mu < 0$.*

Proof If the individual returns are independent and n is big enough, \bar{X} is well approximated by a normal distribution with mean μ and variance $Var(\bar{X}) = \sum_i \sigma_i^2 / n^2 = \sigma^2 / n$. Hence, $P(\bar{X} < 0) = P(z < -\frac{\mu}{\sigma/\sqrt{n}})$, where z is a standard normal, which converges either to 0 or to 1 as n grows larger, depending on whether μ is negative or positive. Since $\sigma_i > 0 \forall i$, some $\epsilon > 0$ can always be

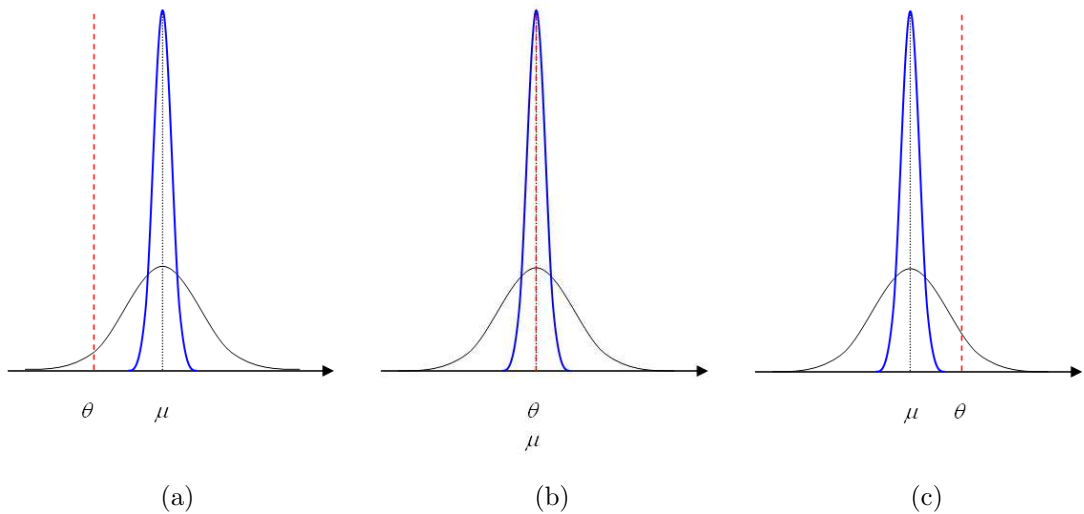


Figure 1: Distribution of autarchic and pooled consumption, i.i.d. returns

found such that $\epsilon < P(X_i < 0) < 1 - \epsilon$, and the denominator in eq. 4 remains strictly bounded between 0 and n . Hence pooling is beneficial if $\mu > 0$ and detrimental if $\mu < 0$. \square

Figure 1 exemplifies, in the case of i.i.d. returns. Pooling is beneficial if $\mu \gg \theta$ (a), since the probability of getting an insufficient aggregate outcome is very low. When $\mu \ll \theta$ (c) pooling is likely to produce the extinction of the whole population, while living isolated might lead, although with a low probability, to survival. When $\mu = \theta$ (b) the probability of failure is the same, irrespective of whether the individual lives in isolation or is integrated in a group.

Note that the condition $\mu > \theta$ characterizes a well-known trade-off: at each point in time pooling reduces 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], 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 the individuals in the population perish at the same time allows for replacement of the unlucky ones.

4 Contagion

If the possibility of contagion is considered ($\rho > 0$) the variance of \bar{X} remains bounded: it tends to $\rho\sigma^2$ when $n \rightarrow \infty$. This variance is always higher than the variance in the no-contagion case, since $\sigma^2 + (n-1)\rho\sigma^2 > \sigma^2$, when $\rho > 0$.² Nothing constrains the numerator in eq. 4 to be bigger or smaller than the denominator. In particular, pooling can be detrimental even if $\mu > 0$.³ Only in special cases, as when the individual returns follow identical distributions, we can confirm the result of proposition 1: pooling is beneficial if and only if $\mu > 0$ and detrimental if and only if $\mu < 0$. Then 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.

Proposition 2 *Suppose the individual returns are identically and normally distributed with mean μ , variance σ^2 and covariance $\rho\sigma^2$, $\rho > 0$. Then, pooling is beneficial (detrimental) if and only if $\mu > 0$ ($\mu < 0$). However, the effects of pooling are always smaller than in the case when $\rho = 0$.*

Proof Since all distributions have the same mean, it is enough to show that contagion increases the volatility of individual returns, with respect to the case of independent outcomes, but the volatility still remains below the autarchy level: $\sigma^2/n < \frac{\sigma^2 + (n-1)\rho\sigma^2}{n} \leq \sigma^2$. If $\rho = 0$ we get back to the no contagion case. If $\rho = 1$ the fate of all individuals is identical and living together is equivalent to living in autarchy. \square

5 Discussion

Aside this rural example⁴ 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].

²A 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.

³The result that pooling is detrimental if $\mu < 0$ is quite general. For instance it can easily be proved when individual outcomes follow a multivariate normal distribution with constant variance-covariance but different means.

⁴risk-sharing networks in rural environments have indeed been analyzed by e.g. [Fafchamps and Lund, 2001, Beuchelt et al., 2005, Dercon et al., 2006, De Weerd and Dercon, 2006], while an application of the CLT to group foraging in animals is [Wenzel and Pickering, 1991]

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 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 transmitted up to suppliers and creditors and down to clients, by means of variations in prices and quantity and delays in payments and provisions [Greenwald and Stiglitz, 2003].

These client-supplier and debtor-creditor linkages between economic agents ensure that shocks are partially transmitted. 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.

Finally, a positive covariance in individual outcomes can be originated by common demand or supply shocks, or by the spread of opinions and expectations in the economy.

6 Extensions

The analysis above covers only the extreme cases of fully disconnected and fully connected networks. From a policy perspective, heterogeneity in the link distribution, with some nodes possibly playing a crucial "hub" function, introduces the possibility of intervening by disconnecting (some parts of) the network as the economic environment deteriorates, that is when interdependencies become more dangerous.

Moreover, while keeping links exogenous, one could investigate how the efficiency of the network (reduction in the number of failures) changes when changing the characteristics (e.g. the mean of individual returns) of the connected individuals.

This is left for future research.

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