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# A biological approach for financial network contagion based on the Susceptible - Infected - Recovered (SIR) model

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*Eloy Fisher\**

## **Abstract**

We will present the purpose, structure and prospective extensions of the Susceptible–Infected–Recovered (SIR) Approach for Financial Network Contagion Model (version 2, FINSIR for short) for NetLogo version 4.1.2. This model seeks to model the behavior and dynamics of Credit Default Swaps (CDS) markets. After framing the FINSIR model, its agents, variables and interactions within a broader set of questions regarding financial markets and the current literature, within this highly restrictive toy computational model, we find that shocks in this financial market exhibit complex evolutionary dynamics that either tend to increasingly fragile states or the elimination of a high number of competitors, in detriment to a more decentralized market order. Given the current incompleteness of the model, we must acknowledge that some of the design assumptions will be approximate and tentative.

**Keywords:** Financial crises, Epidemiological models of financial networks, Agent-based modeling, Credit Default Swaps.

**JEL Codes:** B52, G02.

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## 1. Overview

### *1.1 Preliminaries*

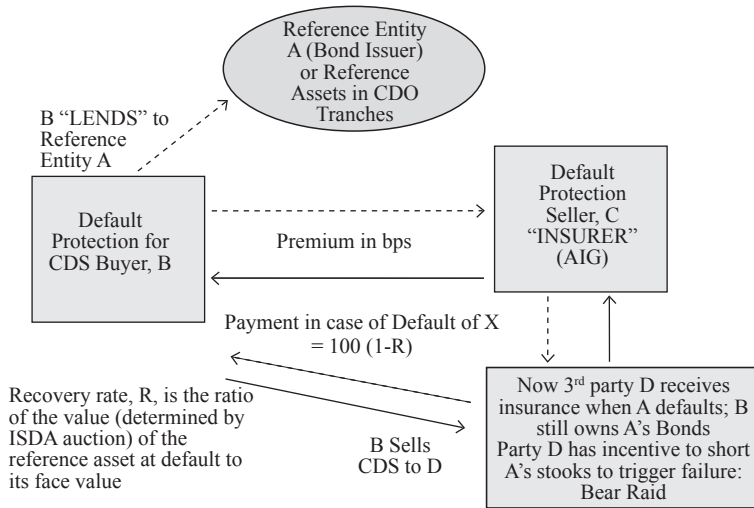
The Great Recession of 2008 demonstrated the vulnerability of financial systems around the world. The trigger for the deepest financial downturn ever since the Great Depression of the 1930's was the meltdown of complex financial derivatives which buttressed balance sheets in firms and households from Wall Street to Main Street. However, even when the intuitive causes for the debacle are well known, standard economic theory was not able to predict how massive, protracted and contagious the crisis would be. Banks across the United States and Europe, heavily indebted in risky assets that lost most of their value over a period of rapid readjustment, were unable to cope with the consequences of their past, risk-seeking behavior.

Although this financial crisis will be studied for years to come, we wish to propose a different approach. Indeed, part of the problem rested in the complexity of these new financial instruments. Through securitization, structured finance was able to substitute largely diversifiable risks for others that were highly systematic. Without a doubt, mindless securitization gave these assets less of a fighting chance to survive a system-wide severe economic downturn relative to other, less sophisticated traditional corporate securities of equal rating (Coval, Jurek and Stafford 2008). When narrow non-systemic considerations are single-handedly used to prompt investing behavior (like during asset bubbles), these assets can provide a nutrient-rich environment for financial contagion.

For this reason, we focus on a specific part of the derivatives market, namely the Credit Default Swap (CDS) market to see how financial contagion could spread. Swap contracts simply call for “An exchange of a series of cash flows” (Bodie, Kane and Marcus 2005: 850) depending to forward contract claims and conditions. On this note, CDS contracts are a specific kind of swap:

...Specified over a period ... with its payoffs linked to a credit event such as a default on debt, restructuring or bankruptcy of the underlying corporate or government entity. The occurrence of such a credit event can trigger the CDS insurance payment by the protection seller who is in receipt of periodic premia from the protection buyer (Markose, Giansante, Gatkowski and Shaghagui 2010: 17)

**Figure 1**  
**How Credit Default Swaps work**



Source: Markose, Giansante, Gatkowski and Shaghagui 2010:17.

We can see in Fig. 1 how CDS work. A CDS buyer (B) lends to a reference entity which issues bonds (or A). However, in order to “insure” against default from A, B buys a CDS from C (or an insurer, exemplified above by the now notoriously famous acronym of AIG). B will pay to C a premium based on the underlying debt exposure to A, and C will pay to B in case A defaults on its loan –this creates a “notional link” between C and B in case of default.

The problem occurs when we introduce another agent, without underlying exposure to A (or D). If B sells its CDS to D without having any financial links or commitment with A, D will receive the “notional link amount” in case A defaults, without holding any bonds or debt with respect to the underlying. This creates a potential for a strategic conflict of interest: given an assessment of A’s exposure, and whether they deem it sustainable or not, companies might be prompted to short-sell A’s stock in order to weaken its balance sheet and drive down its price. If the company, given its existing equity, cannot meet its margin-calls to roll-over loans, this could trigger a default. So, a company which holds A’s stock might trigger this

strategy if it deems that its exposure in equity is less than the potential reward in redeeming the CDS in case of default.

## *1.2 Financial network theory and epidemiological models*

### 1.2.1 Theories of financial networks and contagion

Financial network theory is a new field in economics, one which is gathering increased attention by researchers and policy-makers. Allen and Gale (2002) proved using a small model with four banks that contagion depends most importantly on the interconnection design between banks. Complete networks where all banks diffuse the amount of deposits held by all other banks cause financial shocks to be localized and easily contained. When networks are incomplete, namely when debt is concentrated between a few counter-parties, the system proves to be more fragile.

Haldane (2009) points to new insights on the robustness of networks, borrowing heavily from more in-depth studies which proved that the fragility of what we thought were robust biological ecologies, like rainforests:

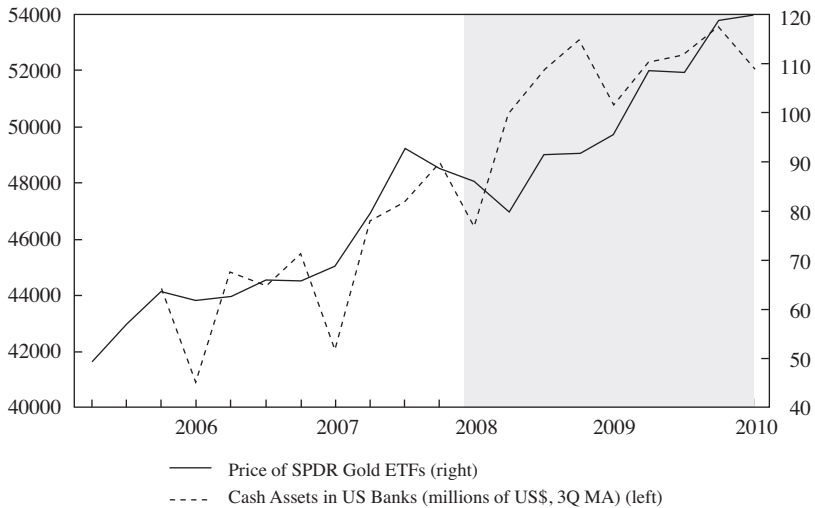
Until recently, mathematical models of finance pointed to the stabilizing effects of financial network completeness. Connectivity meant risk dispersion. Real-world experience appeared to confirm that logic. Between 1997 and 2007, buffeted by oil prices shocks, wars and dotcom mania, the financial system stood tall; it appeared self-regulating and self-repairing. Echoes of 1950s ecology were loud and long... The past 18 months have revealed a system which has shown itself to be neither self-regulating nor self-repairing. Like the rainforests, when faced with a big shock, the financial system has at times risked becoming non-renewable.

In Haldane (2009) four mechanisms seem to be important, and we will try to explain them briefly below to later link them with our computer model. He stresses that connectivity and stability, feedback, uncertainty and innovation are four mechanisms that are at play in financial systems.

With regards to connectivity, financial networks seem to exhibit a tipping point or threshold. Before the critical value, connectivity supports robustness, but beyond it engenders a cascade effect. Feedback, moreover, has a structural and a sociological component – like in epidemiology. Mortality rates in a disease are fixed by the rate of predation of the infecting agent within the host. Nonetheless, transmission varies depending on the channels where contact is allowed, appropriate or demanded by physical context or custom.

Evolutionarily, the transmission responses of “hide or flight” are currently taking two forms in current financial markets: 1) hoarding liquidity (or hide assets) and/or 2) buying “safer” assets, like commodities, gold or silver (flight to assets). The graph below shows how after the initial expectations shock, the appetite for “safer” assets (gold) and liquidity (cash) accelerated during the recession. Although the flight to gold might be a debatable strategy, since it may be fueling another boom-bust scenario, hoarding gold is seen by many investors as a way to hedge against the prospect of devalued currencies, given the massive injections in currencies.

**Figure 2**  
**Cash Assets in US Banks (millions of US\$, 3 month moving average) and price (in US\$) of Gold SPDR Exchange Traded Funds per Quarter 2005 - 2010. NBER recession period shaded gray**



Source: Federal Reserve of the United States, Flow of Funds (L. 109 Q) and Yahoo Finance.

With respect to uncertainty, financial networks generate chains of claims. During times of financial duress and chaos, these chains act as channels for true counter-party exposures. Depending on who or what industry is at the end of the chain, these channels can amplify or restrict uncertainties. With respect to the CDS market specifically, Haldane illustrates:

Consider the case of pricing in the CDS market – an inherently complex, high dimension market. In particular, consider Bank A seeking insurance from Bank B against the failure of Entity C. Bank A faces counterparty risk on Bank B. If that were the end of the story, network uncertainty would not much matter. Bank A could monitor.

Bank B's creditworthiness, if necessary directly, and price the insurance accordingly. But what if Bank B itself has  $n$  counterparties? And what if each of these  $n$  counterparties itself has  $n$  counterparties? Knowing your ultimate counterparty's risk then becomes like solving a high-dimension Sudoku puzzle. Links in the chain, like cells in the puzzle, are unknown – and determining your true risk position is thereby problematic (Haldane 2009: 15).

Finally, diversity in the financial landscape has been overrun by the issuance of debt. Whereas in the past financial institutions would raise money to issuing shares (the equivalent of diversity in the financial eco-system), the reticence to dilute earnings amongst a greater pool of property claimants caused for market capitalization for financial firms to fall almost 90% in the run-up to the crisis (Haldane 2009: 17). Financial innovation was responsible for this phenomena, since the securitization of debt, as substitute for equity, brought great returns at the expense of increasing risk.

All this led to the creation of robust-yet-fragile systems: “while the probability of contagion may be low, the effects can be extremely widespread when problems occur” (Gai and Kapadia 2010: 5). In a highly connected system, losses by a counter-party could be more easily absorbed by other entities. In this case, more connections could lower the probability of contagious default. But these same number of financial linkages can increase the potential for contagion if assets, when toxic, are heavily discounted and this ripples throughout the network. If institutions are lucky enough to survive the first round of defaults, the contagion effects around their network could trigger a second-round of defaults which could prove difficult to avoid.

### 1.2.2 The Susceptible-Infected-Recovered Model as a benchmark for financial contagion

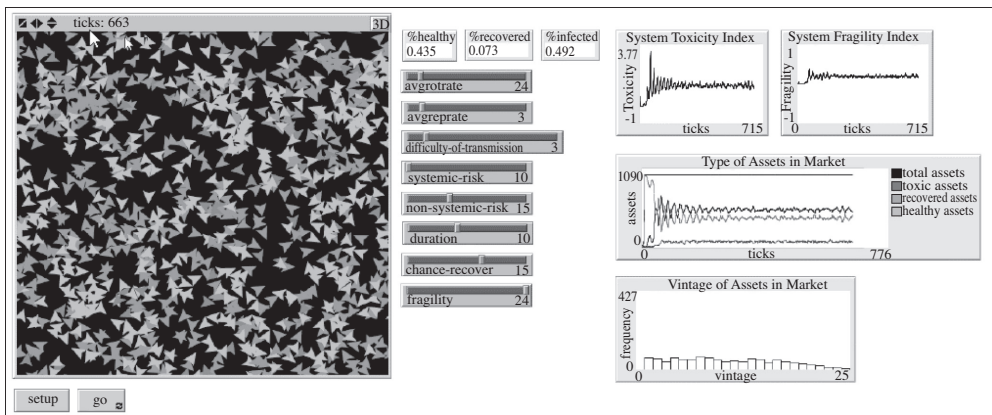
The literature on financial crisis in networks doesn't talk explicitly about the SIR model, yet it hovers in the subtext. Originally devised in 1927, the SIR canonical model seeks to explain the spread of a disease in a population. There are three types of individuals: Susceptible  $S(t)$ , Infected  $I(t)$  and Recovered  $R(t)$ . An infected individual contacts susceptible individuals at a rate  $c$  per susceptible individual, and

(a) is the probability of disease transmission upon contact. Individuals recover at rate  $\rho$  and become susceptible at rate  $\sigma$ . If we include a growth rate of agents within the system ( $\theta$ ) and a death rate in a system  $\delta$ , we can define the model by way of three differential equations as follows:

$$\begin{aligned}
 \left[ \text{Eq. 1} \right] \quad & \frac{dS}{dt} = \theta - dS(t) - acS(t)I(t) + \sigma R(t), \\
 & \frac{dI}{dt} = acS(t)I(t) - \delta I(t) - \rho I(t) \quad , \\
 & \frac{dR}{dt} = \rho I(t) - \sigma R(t) - dR(t) \quad ,
 \end{aligned}$$

For our model, we used a benchmark SIR model as a control experiment and first approximation for financial asset contagion – we will call this model FINSIR v.1. It served only as a preliminary experiment for the development of FINSIR and to assess and devise the current model of financial networks, FINSIR v.1 suffered from many drawbacks, namely the justification of contagion of assets by mere proximity and mingling, with no reference to firm balance sheets. Nonetheless, the model reproduced the cyclical dynamics of the SIR canonical model, which are of interest and are reproduced in an example below.

**Figure 3**  
**Graphical User Interface (GUI) of FINSIR v.1. In NetLogo**



Of interest is the sub-chart above “Type of Assets in Market” which displays some cyclical dampening and evolutionary equilibration, achieved at a given amount of “toxic”, “susceptible” and “recovered” assets. As we will see below, this sort of dynamics were not easily modeled in networks given overarching assumptions about the financial “death” and “recovery” of the firm. Yet, insights of the model above still lie in the subtext of FINSIR v.2.

## 2. Entities and state variables

For FINSIR v.2, instead of using a series of disaggregated homogenous assets floating in some Brownian motion “petri dish” context, agents are financial firms with an underlying debt claim to a single firm. With regards to Figure.1 above, all firms are agent B’s that have a debt with respect to a single agent A (in the model they are labeled as firms-exposure). Also, we include agents like D, which have no underlying exposure with respect to A, and enter the CDS market betting on whether A will default or not on its payments (we call these firms-margin). Both types of firms have assets, which grow at a fixed yield. The price of the CDS, or the spread, is determined by a special auto-regressive process (more on that will be said below). Firms-exposure as stated above, keep an exposure with respect to the underlying firm while firms-margin borrow on the margin. This margin, as commonly defined, is the portion of the purchase price contributed by the firm’s assets, while the remainder is borrowed from the broker. This margin rate sets the amount of debt the firm can handle. Taken as a whole, this asset/debt relationship make up the firm’s quasi-balance sheet. Assets and exposures are randomly assigned.

Furthermore, all firms are enabled to be pessimistic or optimistic based on a cds-discount factor (cdf), state variable that in turn will inform their insertion in the CDS market as sellers or buyers. This cdf will be high for pessimists and low for optimists, and in relative terms, higher for sellers and lower for buyers. This factor will serve to discount or appreciate the spread of the CDS, depending on the bullish or bearishness of the financial firm. For example, if the true price of the spread is a 100 basis points (with respect to the underlying exposure), based on the cdf the price would be  $-cdf \times 100$ . Optimists will have a floating number cdf between 0 and 1, while pessimists will have a floating number cdf between 1 and 2.

Buyers and sellers are determined in the market by a simple algorithm:

$$[\text{Eq. 2}] \quad Price_{CDS} - cdf \cdot Price_{CDS} > Price_{transaction\ cost}$$

This states that if the spread (or price) of the CDS minus its bearish (or bullish) discounted (or appreciated) value is greater than the price of the market transaction costs, the firm will become a buyer of CDS, and if not, the firm will become a seller. The firm will value more the cash flow for granting insurance, since it does not believe the underlying will default. Indeed, this is a simplifying assumption of how the CDS market works, but it captures a lot of the financial literature debate on swap markets.

The financial literature posits that swap market contracts are founded in “comparative advantages” that firms enjoy with respect to financing costs. For example, with respect to interest-rate swaps, lower rated companies may enjoy comparative advantages with respect to variable interest rate markets, while higher rated firms enjoy better fixed interest terms (Hull 2002: 159). These advantages induce firms to transact amongst themselves, if conditions are right. With respect to CDS markets, firms involved do not need to be bearish or bullish only on a matter of pure expectations, but given their ratings, the structure of their assets and liabilities, and how they face day-to-day cash flow operations. Agents create financial links, depending on what side of the transaction they find themselves. These links report the notional value of the CDS exposure (the countervailing cash flow) and allow for cash flow payments based on the aforementioned exposure to be accrued to the counter-party at each time-step. Furthermore, links faced time constraints given their maturity.

Given these industry variables, firms become “toxic” (or infected) and “recovered”, using some insights of the SIR model explained above. However, there are some importance differences: contact in financial firms, as a function of their notional links, is not as clear-cut as in biological models. Biological models treat individuals as entities, infected or not, and abstract from the resource war occurring in their immune systems. In financial firms, the internal processes where their balance sheets are affected are central to the model. For this reason, we must try to keep track of the infection of must cause very distinct consequences for the firms quasi-balance sheets.

When a firm becomes toxic, it heavily discounts CDS spreads and also, the valuation of its assets by way of a toxic discount factor, or *tdf*. This does not mean that firms discount the value of their assets, but that their assets lose value, since these are no longer buttressed against the insurance provided by a CDS asset. Because of this, the equity-to-exposure ratio decreases, but the firm does not become automatically bankrupt (it will do only after it has passed a given threshold, as stated below). This allows for some gray area situations, where firms are allowed

to continue in a zombie-like state, as demonstrated in some model runs – as shown below in Figure 8.

For example, in the model, one of the firms randomly becomes “toxic”. This means that its assets decrease in value by way of what can be a random occurrence like bad management in other branches of its operations, nefarious business practices, etc. Therefore, its payable exposure of CDS to counter-parties decrease in value, as the “toxic” firm cannot meet its obligations. Its CDS no longer buttress the valuation of assets in counter-parties balance sheets. Furthermore, in doing so, it changes its market dynamics and its probabilities of becoming a seller increase as to raise money to meet payables, causing a cascade effect with respect to asset valuation (although this feedback effect is not included in the current model) as more firms press to sell assets.

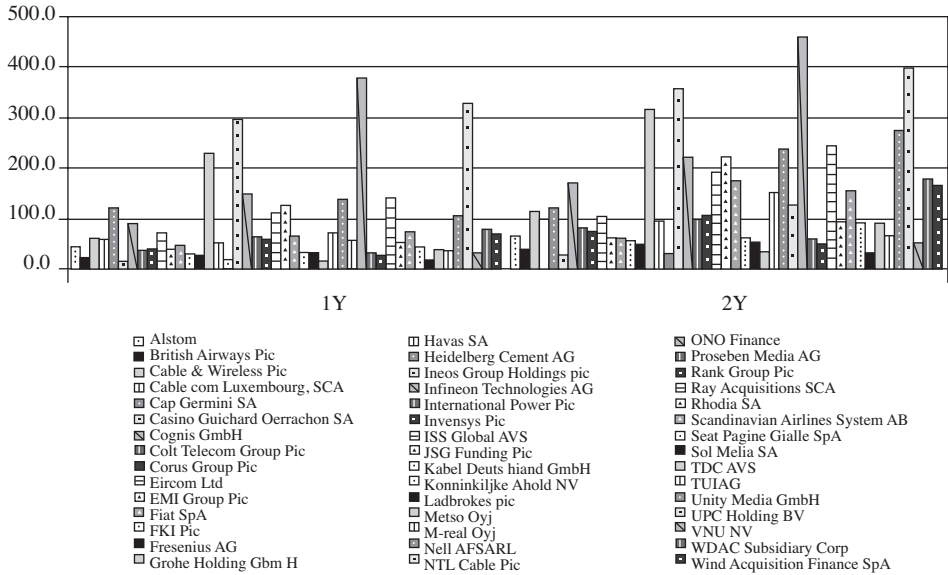
Recovered firms are those who are able to muster a comeback effort, with a probability of recovery  $\gamma$ . However, this recovery is not phenotypical in nature – it affects the discounting of assets within the firm. For example, even in dire systemic circumstances, assets can have a recovery cost. Although counterparties may fail and go bankrupt, asset value can recover when claims become unwound and property rights are clearly delimited between parties to flush out uncertainty – as Haldane noted above. Asset prices can stage a comeback, and in doing so, help firms achieve some breathing room to pay creditors and keep in business. For this reason, recovered firms increase their asset valuation randomly within a range greater than the “toxic” discount. Furthermore, also wired in the model, we have a constant stream of income from bond yields held by firms, which contribute to this re-floating process.

### **3. Process overview and scheduling in FINSIR v.2**

There are two main external processes in FINSIR v.2: The auto-regressive spread process and the market-maker process. Furthermore, agents run other processes internally: a survival process, based on the leverage ratio of the firm, determines whether the agent goes bankrupt and an immune response process which allows for the firm to recover, given a constant stream of income, as all firms are assumed to hold yield-generating bonds.

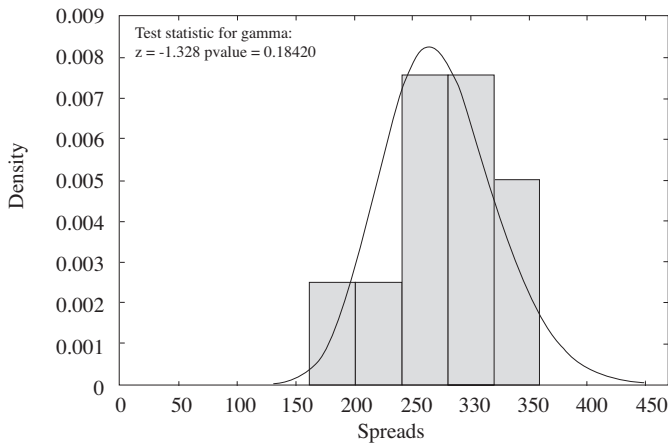
Spreads in FINSIR v.2, or the price of CDS, are determined in the model by a special auto-regressive process. The pricing process for spreads is a complicated affair, since spreads not only reflect the most immediate price, but a forecast of prices given current information. Below we present the distribution of the iTraxx Europe CDS Index, and one and two year spreads of the indexed companies.

**Figure 4**  
**One and Two Year Spreads for companies in the iTraxx Europe CDS Index**



Source:Wilmott Forums

**Figure 5**  
**Distribution of 10 Year Spreads for the iTraxx CDS Index against the gamma distribution**



Source: Wilmott Forums.

**Table 1**  
**Summary Statistics for iTraxx Spreads in Figure 5.**

<i>Mean</i>	<i>Median</i>	<i>Minimum</i>	<i>Maximum</i>
273.166	276.325	181.831	340.000
<i>Std. Dev.</i>	<i>C.V.</i>	<i>Skewness</i>	<i>Ex. Kurtosis</i>
49.0424	0.179533	-0.416366	-0.69

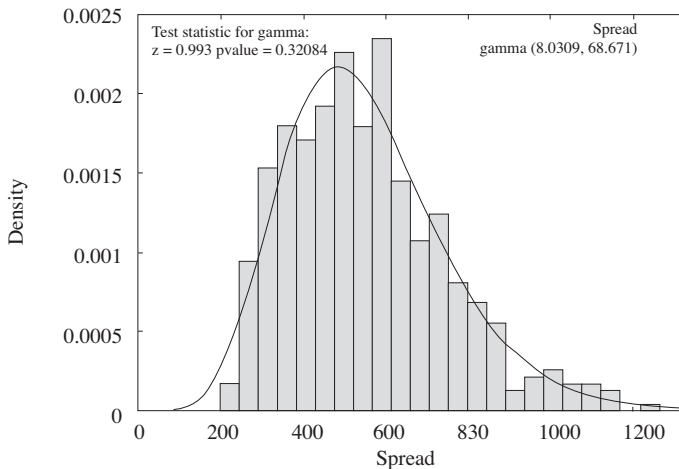
Although the gamma distribution fit above is barely an approximation, it is better than the one offered by a normal distribution. Since real-time CDS data information is hard to come by, we will use the statistics above as a rough approximation to compute our gamma distribution.

For our spreads we will define an AR process with a random gamma distribution where the shape parameter will be, using Table 1 above,  $\theta = mean \times \left(\frac{mean}{variance}\right)$  and the scale parameter  $k = \frac{1}{\left(\frac{variance}{mean}\right)}$  but with an inde-

terminate AR parameter - fluctuating between 0 and 1. For this reason, the process will exhibit an oscillation between a unit root and a stationary process. Below is an example of the spread output for 500 time steps, as computed for FINSIR v.2.

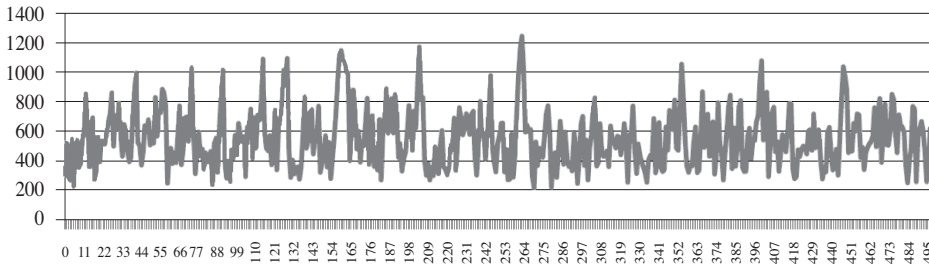
The other important process for the model is the market-maker process.

**Figure 6**  
**Distribution of Spreads for the FINSIR v.2 model**



Source: FINSIR v.2 Model.

**Figure 7**  
**Spreads for the FINSIR v.2 Model**



Source: FINSIR v.2 Model.

This process entails pairing buyers and sellers to create notional financial links between them. The algorithm for this process requires some further explanation. In the first instance, buyers and sellers are divided into two lists. At each point in time, each buyer will look around each seller and see whether a condition for the exchange of a CDS cash flow and the notional hypothetical exposure is met. This condition is:

$$[\text{Eq. 3}] (Exposure_{seller} + Exposure_{buyer})(cdf) < (Assets_{buyer})(tdf)$$

Where the *tdf* is a toxic discount factor which appears when firms become infected. For “toxic” or “infected” firms, the new state condition affects their *cdf* as they grow more pessimistic on CDS market prices, but also affect their own valuation of assets, as they are heavily discounted given that notional amounts payable to the firm quickly disappear, and in order for the firm to meet its notional exposure margins, it is forced to sell its assets. These events help provide a cascading context of financial contagion amongst firms, whether they are or not holders of the underlying exposure.

Moreover, firms without exposure (*firms-margin*) enter into CDS contracts by way of a “sensing” sub-program. In this sense, [Eq. 3] is modified to:

$$[\text{Eq. 4}] (Exp\bar{o}sure_{of\ nearby\ agents} + Exposure_{buyer})(cdf) < (Assets_{buyer})(tdf)$$

Where *Exp̄o*sure is the mean exposure of agents at a distance of 10 patches from the caller. This peculiar introduction of “patches” merits some clarification. In

the model, patches proxy for business “nearness”. Firms do not operate in a vacuum, these respond and act strategically depending on the environment in which they operate. Firms that bet on the exposure of other firms (firms-margin in the model) do so because they have some knowledge about the operations of companies which they deem to consider close to them, whether as competitors or not. The relationship expressed above for firms-margin tries to capture this effect, and the ensuing cascading phenomena of firms which look to their nearest counter-parts for some guidance on their assessments and bets.

Internally, the agent will transact as long as its leverage ratio does not surpass 30%. If the agent has a higher ratio, the firm very likely will be unable to cope with creditor’s claims and disappear. Although this number might be excessive by Basel standards (the standard for banks is 5%), financial firms that went bankrupt during the early days of the crisis had leverage ratios around that number. For example, in the infamous Lehman Brothers 2006 financial report issued by that company before the crisis, the firm nonchalantly recognized that between 2002 and 2006 it had an official leverage position between 23 and 29%. However, it was later acknowledged that its off-books financial position was even more precarious.

The immune process to allow for recovery involved the introduction of a toxic-count variable, very similar to a process rehearsed in *FINSIR v.1*. Here, all infected agents start a toxic-count during their infection which increases by one unit at each time-step. As the toxic-count progresses, the possibility of recovering fades away, but apart from that it does not affect any other variables. This toxic count, as used in other SIR models, only seeks to internally keep a time-clock of the infection and proxy for ensuing difficulties in recovering after a protracted and stubborn infection.

Furthermore, this toxic-count provides a benchmark from which to assess recovery. In order for firms to recover, a random recovery variable (named in the model recovery-possibility) is pitted against the toxic-count. If greater than the toxic-count, the firm recovers, and with it, some of the valuation of its assets, given the justification stated above. However, this valuation after recovery will not be as generous as the one before infection and will oscillate above the toxic discount factor but below the original valuation, given the uncertainty of economic conditions after the infection shock.

#### **4. Initialization**

For the initialization of *FINSIR v.2*, a fixed set of agents is created, localized and a set of state variables are randomized. Furthermore, a number of constants are set

for some of the global variables for convenience. The model as it stands creates 50 financial firms, 25 of those will have an underlying exposure, and the rest will be firms that are betting on the underlying firm's prospects, without having any exposure. Firms are randomly distributed on a given metric space. All firms will have a random number of assets between 0 and 10 000 units. Underlying exposure for those firms with the underlying security will be between 0 and a 1 000 units. As stated above, the cdf will be randomized for all firms between 0 and 2.

Initial parameters set are the initial spread as a random variable between 0 and 0.10. The yield is fixed initially at 0.08, although it can also be randomized. The risk free is set at 0.025, and the transaction cost at 0.01. Finally, toxicity (or the transmission of the infection) via the links is set at 5 – here the controls are inverted, a low “toxicity” allows for higher transmission of infection. The recovery variable is set at 3: a low recovery variable makes recovery more difficult. Some run-time errors occur due to the random structure of buyers and sellers in lists, given their individual cdfs. In some occasions, the market is uneven and buyers and sellers are clustered in one of the two lists, so when the market-maker mechanism starts to review each agent individually, it will run into an empty set. If one of the lists is empty, the program will stop. To solve this problem, another setup initialization is required.

## **5. Design concepts and results**

This short overview on the design concepts will focus on the objectives of firms and the interpretation of their interaction as presented in FINSIR v.2. The objectives of firms are based on rule-of-thumb procedures. In this model, although not strictly profit-optimizers, firms are seeking cash flows without taking into the consideration the notional links being created with their respective counter-parties. In this sense, bounded rationality prompts firms to act, as they are systematically making a crucial incorrect inference (Rubinstein 1998: 41): although the future is uncertain, and the possibilities of a system-wide financial implosion difficult to ascertain, they seek to increase their notional exposure as to increase cash flow receivables.

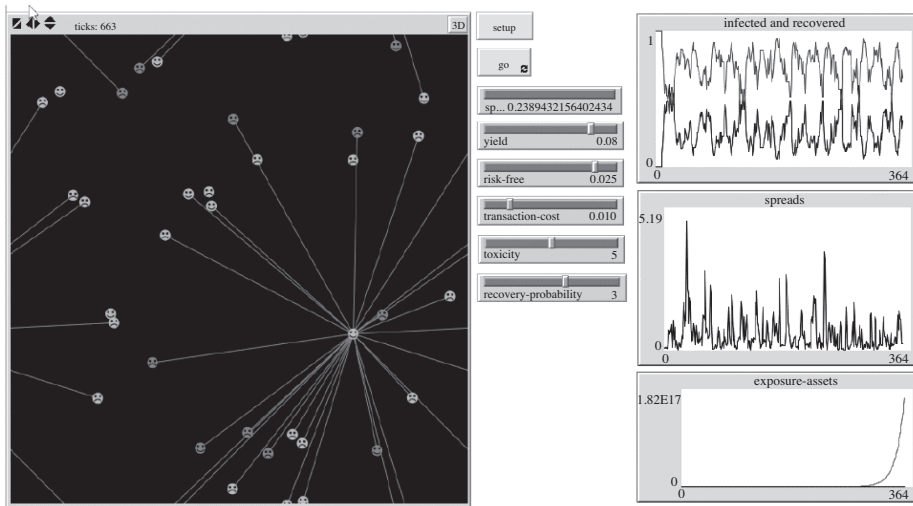
A similar situation proved to be AIG's undoing: deliberately looking to set cash flow premia without due consideration of risk-taking on the notional links created, amidst a crisis which spread rapidly and affected the valuations of assets of the rest of the firms, will cause all involved to fall like one by one, like a house of cards.

This leads, inasmuch it comes to interpreting the interaction mechanisms in the model, to two distinct results, which seem to be in agreement with the robust-

yet-fragile explanations offered by Haldane above. Below an unidentified threshold of interactions, the activation of a toxic shock in the system and the ensuing elimination of weaker firms appears to lock and consolidate firms (as shown below in Figure 8) in a dynamic closely resembling the one in FINSIR v.1, Figure 3. In this case, after determining a set of parameters in the model (namely, the initial spread of the CDS, the bond yields accrued to agents, the transaction costs implicit in the system, the level of toxicity of infection and the possibility of recovery), the model run tends to a sort of dynamic stationary state, very similar to that of some runs in FINSIR v.1. Financial links cluster around a group of very large firms, with enough assets to allow them to maintain their eagerness to buy, willing and able to withstand the system exposure on their balance sheets. Even more so, these companies allow some toxic companies to continue transactions, in a sort of zombie-like state.

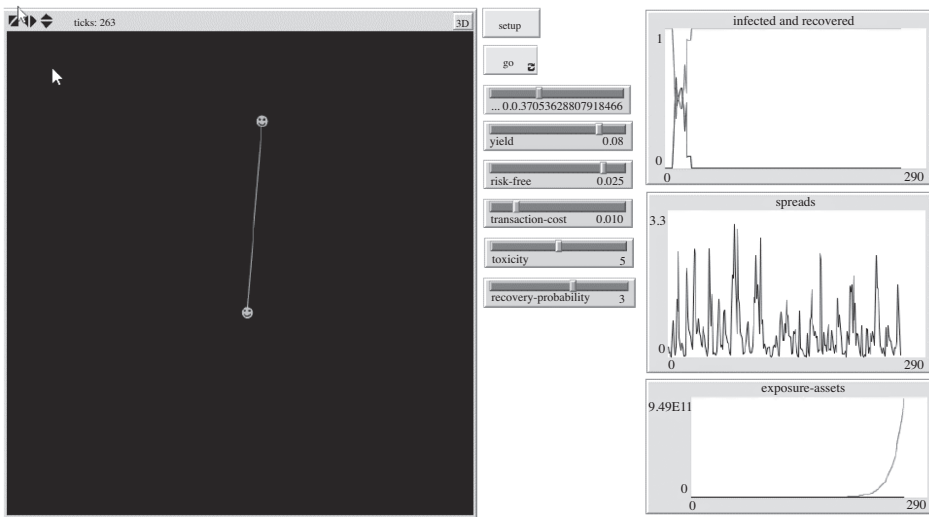
However, it seems that beyond an unidentified threshold (that cascade portal as identified by Haldane and others above), the system assumes an apocalyptic configuration, and only a few firms remain after a violent system adjustment, as seen in Figure 9 below.

**Figure 8**  
**Oscillating dynamics for FINSIR v2 where links cluster around large firms, eager to buy**



Source: FINSIR v.2

**Figure 9**  
**“Apocalyptic” scenario for FINSIR v.2. Most firms disappear**



Source: FINSIR v.2

Even when the causes for these two dynamics are not clear from financial theory, these mirror insights from SIR epidemiological models. We could argue that the determining factor in the first scenario is the incorporation of “toxic” firms within the dynamic equilibrium of the system, one which assumes a clustering pattern around the biggest surviving firms. This scenario would occur during a not-so violent downturn, when infected firms are allowed to continue operations. Without a doubt this will increase the fragility of the system, if another financial crisis ensues. The second, “apocalyptic” scenario occurs when the downturn is violent and adjustment is unforgiving for all firms. Only large firms will survive, and toxic firms will disappear.

## 6. Limitations of FINSIR v.2

There are many problems in trying to apply the SIR Model to financial networks. Financial systems fail to exhibit the same morphology and do not share similar generative processes as biological systems. This limitations were evident during the design of FINSIR v.1 and v.2.

With regards to morphology and structure, contact amongst firms in financial networks is defined with respect to cash flows. These provide the topology from which financial contagion might spread given the intoxication of a firm and its notional asset flows with respect to counter-parties, with little or no regard to symptoms. In the financial world, it is easier to continue rolling over swap positions and posturing a healthy appearance to counter-parties than in biological systems, where contact dynamics, as an increasing function of population, are affected by symptoms (with HIV/AIDS being the notable exception). Financial systems tend to be more subtle.

Furthermore, generative processes involving financial firms exhibit different dynamics than biological systems. For instance, whereas biological entities would be able to reproduce given favorable circumstances of food and climate, financial firms face a different set of constraints, which are not set by their economic capacity but by their relative political bargaining power.

Biology tends not to favor too-big-to-fail entities. In an ecosystem, resource monopolies exert a taxing influence on its surroundings and the potential feedback effects, more often than not, are deemed too costly for the survival of the species. For example, this reality is hard wired into scale laws for thermodynamic allometry and concretely, into Kleiber's law. In financial systems, the political economy of finance allows for loopholes where the size-metabolism constraint is weakened given the relative political bargaining power of financial firms.

For this reason, it is hard to link fitness and reproduction with a successful set of financial networks, given the exogenous nature of the creation of financial firms. Instead of spawning competitors during times of prosperity, financial firms seem to consolidate through mergers and acquisitions (M&A). Even more puzzling, M&A in the financial sector, even during times of prosperity, seems not be driven by increasing profits, performance or stockholder wealth (Piloff & Santomero 1996: 18). Without a doubt, financial firms require a propitious economic context for success; however the sufficient condition for ultimate victory seems to be the relative lobbying might against anti-trust regulations. Division of labor and specialization in financial firms seems to be a more oddity than an incentive for reproduction.

Finally, applying the SIR model to the death of a firm is faced with some problems of its own. Illiquidity in financial firms is a necessary but not sufficient condition for the death of a firm, since the entity might keep illiquid assets and claims that may help it weather bad times, as long as it keeps creditors at bay. And as proven by the recent financial debacle, the determination of insolvency can be a political issue. Brittle accounting rules are no match to organized lobbies, and indeed much of the literature in accounting journals revolves around the discus-

sion of lobbying models for accounting standards (for a short literature review, see Königsgruber 2010).

## **7. Conclusion**

This paper sought to explain the most important aspects of the FINSIR v.2 model and to highlight its operating mechanism as an approximation to model financial contagion. Using the example of the CDS derivative market, the model builds upon a network topology of financial firms involved, whether as holders of exposure of an individual firm or as bettors on the prospective of default of that firm. Later, it seeks to incorporate some insights of the SIR epidemiological model to help visualize the spread of financial crises in that market.

The model provides an interesting first approximation to the dynamics of real derivative markets. Spreads are computed by a process which seeks to mimic, albeit incompletely, their observed progression over medium-term life spans (1 to 2 years). Furthermore, agents react to a combination of expectations, bullish / bearishness and comparative advantages when going into the market for CDS. These rules provide an abridged context for financial link creation. However, whereas other works focus on the creation of these links and their *robust-yet-fragile* topology, this model only seeks to apply a modified version of the SIR model to allow for financial contagion amidst processes of link creation.

In this sense, infected firms pass on their toxicity to other firms via notional cash flows which prove to be fictitious in nature, and in doing so, disrupt firm's expectations and the normal valuation of assets in their quasi-balance sheets. The model suffers from some limitations, especially when trying to model the generative processes, death and morphology of financial firms. Unlike biological systems, financial systems are more subtle and as we have seen in the latest economic downturn, politically driven. For this reason, in order to fully grasp their complexity, another layer of rules and assumptions needs to be wired into the SIR model.

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