INT06: Adaptive Percolation

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Abstract

The spread of infection is considered through percolation methods, including the case of a new adaptive model. We set out the basic formalism for network analysis and contagion. The SIS (Susceptible-Infected-Susceptible) model is analysed as a paradigm example of percolation and more specifically contagion within a network. We present a generalised SIS model that incorporates network adaptation to infection, reliant on the Popularity model of networks. The adaptive SIS model is investigated computationally and the existence of a phase boundary is established. A review of research on financial contagion is given and the lack of an adapted element is noted. Implications and applications of the adaptive SIS model are given for areas of financial contagion and epidemiology amongst others.

Contents

I. Introduction

Network science is a blooming area of research with applications in a hugely broad range of fields leading to an active interdisciplinary movement. The study of nature in network frameworks dates back to the mathematician Euler and his Seven Bridges, thus spawning the mathematical study of networks called Graph Theory[1]. Later Paul Erdős and Alfréd Rényi

[1] I use 'graph' when the discussion is within a more mathematical context but it can be taken as synonymous with 'network'.

published a series of seminal papers on random graphs in which they analytically determined conditions for differing levels of connectivity in their graphs [1–3]. The basic premise of network science can be stated as: many parts of nature can be readily represented by networks, these networks have clear pattens and structures enabling various network classifications, these patterns and structures have consequences for the systems they represent.

Examples of successful and varied applications of network analysis include the friendship networks in schools dependence on ethnicity [4,5], representing interacting proteins in biology [6], statistical mechanics of the network formed by the internet [7], and even a proposed influence on an individual's capability for innovation due to properties of their network of acquaintances [8].

Percolation is a specific subset of network science concerned generally with flow through networks; this incorporates determining when flow is possible, their rates, the ability for a network to sustain a spanning path when nodes or links are removed etc. In physics percolation is used in the study of phase transitions in systems [9–12]. Its use in modelling and analysing the spread of infectious disease in a population is well developed [13–16], as well as more general 'infections' such as the spread of computer viruses, information or opinions through various networks structures like word-of-mouth interactions or email correspondences [17–19].

A limitation of these models is that they lack proper responsive dynamics; the network structure is unaffected by the state of the system. This is reasonable in cases where the infection is not apparent over timescales of transmission such as for asymptomatic diseases, or when the infection simply doesn't cause a change in the behaviour of the host. But many systems of contagion have an inherent adaptation; those with the common cold stay at home so are less likely to meet others and transmit the virus, or the spread of an opinion may be boosted by campaigners proactively persuading others. One area which has had a lot of recent success in applying percolation is modelling financial crashes as the spread of collapse within a network of interdependent banking bodies [20–23]. It will be shown though that the models used thus far do not incorporate an adaptive element to capture the decision making and judgement of the participants involved in forming such economic ties.

In this paper we: 1. show the basic formalism for network analysis and contagion, 2. present a paradigm model of contagion; the SIS model, 3. we define adaptation within the network context, 4. review the most visible papers on financial contagion, 5. introduce a new model of network formation which lends itself to adaptation, 6. present and computationally analyse an adaptive SIS model.

II. Representing Networks

A network consists of a set of *nodes* (vertices, agents) $N = \{1, \ldots, n\}$ and a set of *links* (edges, connections) $G = \{\{i, j\} : i, j \in N\}$. The set of links can also be represent by an $n \times n$ matrix called an *adjacency matrix*;

$$g_{ij} = \begin{cases} 1, & \text{if } \{i, j\} \in G, \\ 0, & \text{otherwise.} \end{cases}$$

We will only consider simple, undirected and unweighted networks meaning no self-links, links are always two-way, and all links are equivalent respectively. This requires $g_{ii} = 0$ for all i, g to be symmetric, and g to contain only 1's or 0's.

The *degree* d_i of a node i is the number of links containing it or equivalently the number of nodes i is linked to. Formally

$$d_i = |\{j | \{i, j\} \in G\}| = \sum_j g_{ij}.$$

The *degree distribution* P of a network is a central object in the characterisation and analysis of networks. It describes the relative frequency of nodes of each degree. Given such a distribution, then $P(d)$ is the fraction of nodes that have degree d. Note that the degree distribution can be a probability distribution from which we can generate a set of degrees to form a network, or it can be a frequency distribution used to describe data from an actual network.

Common degree distributions, which we will be considering, are the *delta* distribution

$P(d) = \delta_{dk}$ which forms a *regular* network in which all nodes have the same degree k. The *Poisson* distribution[2], $P(d) = \frac{\lambda^d}{d!}e^{-\lambda}$ where λ is the mean degree, which form *Erdős-Rényi* networks[3] (ER for short). Lastly the *scale-free* distribution (SF for short), $P(d) = cd^{-\gamma}$ where c is a normalisation factor, which produces *scale-free* networks. SF networks differ from ER networks in that they tend to have many more very high degree nodes and very low degree nodes; the network has a large spread of degrees. Each of these network structures are observed in real world systems [24–26]. See Appendix A for plots of these distributions.

The mean degree and second moment of a network with P are given by $\langle d\rangle = \sum_d P(d)d$ and $\langle d^2\rangle = \sum_d P(d)d^2$ respectively. Diffusion through a network increases with increasing $\langle d\rangle$ [27] since there are more links in the network through which it is possible to flow. So to compare aspects of percolation just between the structures of various networks, we do so while holding their average degrees constant. An important result is that (while holding the mean constant) $\langle d^2\rangle$ increases with increasing spread in the degree distribution $P(d)$ [27], called a mean-preserving spread. The regular degree distribution has no spread, the Poisson has some and the scale-free even more so; this is most visible in Appendix A Figures 12 and 14. This creates an ordering when holding the mean constant; $\langle d^2\rangle_\delta < \langle d^2\rangle_{Po.} < \langle d^2\rangle_{SF}$, the Poisson distribution is a mean-preserving spread of the delta distribution and the scale-free distribution is a mean-preserving spread of both. See Appendix B for further explanation on the ordering of distributions through stochastic dominance.

II.1. The Configuration Model

The configuration model is a procedure by which, given a degree distribution, we can form a random network with corresponding degree frequencies.

i) Take n samples from a given degree distribution to form a degree sequence $\{d_1, d_2, \ldots, d_n\}$. ii) Construct the sequence where node i is listed d_i times; the degree sequence $\{4_1, 1_2, \ldots, 6_n\}$ would result in

$$\{1, 1, 1, 1, 2, \ldots, n, n, n, n, n, n\}.$$

iii) Repeat the following steps until the list is empty: 1. pick two elements at random[4], 2. form the link between the two nodes represented by the pair of elements, 3. delete the elements. This provides a method of forming a random network with a given degree distribution.

Problems inherent in the configuration model are the possibilities of more than one link forming between two nodes, and links connecting a node to itself. The chance of these occurring become increasingly small for large n and a sparse network ($d_{max} \ll n$); we can form an approximate network by removing any multiple or self-links that happen to occur.

III. The SIS Model

III.1. Model Specification

The SIS model is not a fixed network of nodes but an application of network theory to model a collection of agents who have random interactions or meetings with one another overtime, in which an abstract infection can be transmitted.

Consider a collection of agents represented by nodes, all of which can be in one of two states; susceptible or infected. Agents are never removed from the system but recover from infection to return to the susceptible state (hence Susceptible-Infected-Susceptible or SIS). Each agent is described by their degree; the i'th agent has degree d_i. The degree describes the number of interactions with other random agents they will have within a given period, so we call the SIS model a degree-based random meeting model. One should think of the SIS model as a

[2]This distribution is actually an approximation of the distribution formed in the random graphs studied by Erdős and Rényi [1–3]. Take n nodes and form links between them with probability p; this gives a binomial degree distribution $P(d) = \binom{n-1}{d}p^d(1-p)^{n-1-d}$. For large n and small p this approximates to the Poisson distribution.

[3]There are varies elements of randomness in the models considered and to avoid ambiguity we will not call ER networks 'random' networks as is the case in much literature.

[4]It is assumed without real problem for large n that this sequence is even in length.

network defined by a given degree distribution which after every period of time is completely remade randomly. Nodes which are linked in the network for that period denote agents that meet during that period.

We define the infection density $\rho(d)$ to be the fraction of agents with degree d that are infected. Let $P_m(d)$ be the probability of meeting an agent of degree d, and θ be the probability that a given interaction is with an infected node; then $\theta = \sum_d P_m(d)\rho(d)$. For a sparse network of large n formed using the configuration model, the degree of neighbouring nodes are approximately uncorrelated. Then the probability of meeting an agent of each degree $P_m(d)$ is the same as the number of links involving nodes of degree d – $P(d)dn$ – normalised by the total number of links in the system $\sum_d P(d)dn$. Thus $P_m(d) = \frac{P(d)d}{\sum_d P(d)d} = \frac{P(d)d}{\langle d\rangle}$, and so meetings are more likely to be with higher degree agents [15]. This leads to

$$\theta = \sum_d \frac{\rho(d)P(d)d}{\langle d\rangle}. \tag{1}$$

We define the average infection rate[5] ρ as the total fraction of agents in the model that are infected at a given time: $\rho = \sum_d P(d)\rho(d)$. This differs from θ because an individual is more likely to meet another agent if that agent has many meetings.

The final probability of an agent being infected within a given period will be some function of θ, the agent's degree and other parameters describing the specifics of the infection mechanics. For instance the infection could transmit with certainty in meetings between infected and susceptible nodes, or there could be probability of infected per such a meet, or even a threshold on the number of such meetings required for transmission. We choose the simple form of a transmission rate parameter $\nu \in [0, 1]$, which is the probability for transmission of infection in a given meeting between an infected and a susceptible agent. So the final probability for infection in a period is[6]

$$\nu\theta d. \tag{2}$$

We choose recovery to be a simple Markovian property that is the same for all agents: a chance of recovery per period equal to $\delta \in [0, 1]$.

Simulated evolution of this system can be seen in Appendix C.

III.2. Steady-State Infection Rates

Assume a mean-field approximation such that each agent has a fraction of infected neighbouring nodes that matches exactly the density of infected nodes $\rho(d)$. The number that recover is the fraction that are infected multiplied by the recovery rate, and the number that become infected is the number of susceptible agents multiplied by the probability of infection in a given period. So $\frac{\partial\rho(d)}{\partial t} = (1 - \rho(d))\nu\theta d - \rho(d)\delta$. We then solve for steady-states $\frac{\partial\rho(d)}{\partial t} = 0$, so solving for when the number recovering in a period is equal to the number that become infected. This leads to the steady-state equation

$$(1 - \rho(d))\nu\theta d = \rho(d)\delta. \tag{3}$$

Let $\lambda = \frac{\nu}{\delta}$. Solving for infection density gives

$$\rho(d) = \frac{\lambda\theta d}{(\lambda\theta d + 1)}. \tag{4}$$

Substituting this into (1) gives the defining equation for the infection rate θ for the model in a steady-state

$$\theta = \sum_d \frac{P(d)\lambda\theta d^2}{\langle d\rangle(\lambda\theta d + 1)}. \tag{5}$$

Remarks; i) $\theta = 0$ is always a solution corresponding to the steady-state of no infection. ii) $\theta < 1$ for finite λ. iii) The righthand side of (5) is an increasing and convex function of d. The steady-states can be solved exactly for a regular network of degree k; $P(d) = \delta_{dk}$ giving

$$\theta = \sum_d \frac{\delta_{dk}\lambda\theta d^2}{\langle d\rangle(\lambda\theta d + 1)} = \frac{\lambda\theta\langle d\rangle}{\lambda\theta\langle d\rangle + 1} = \rho(k), \tag{6}$$

[5] The notation for infection densities $\rho(d)$ compared with the average infection rate ρ is only distinguished by the presence or lack of the argument (d). This becomes more intuitive once one realises that there are multiple infection densities at any one time, one for each degree, but just one unique average infection at such a time.

[6] Choosing ν such that $max(d)\nu \ll 1$ keeps the probability of infection per period well defined.

where the last equality follows from (4). Again $\theta = 0$ is a solution. Cancelling a factor of θ from (6) then rearranging gives a second solution

$$\theta = 1 - \frac{1}{\lambda\langle d\rangle}, \tag{7}$$

giving a threshold for a non-zero steady infection in a regular network

$$\langle d\rangle > \frac{1}{\lambda}. \tag{8}$$

This result fits with the intuition that higher infection to recovery ratio λ requires a less densely connected network to sustain an infection. The value of the threshold determines the point at which the system undergoes a phase transition, from being capable of sustaining an infection and not.

The analytical formula for the average infection of a regular network is also easily derived:

$$\rho = \sum_d P(d)\rho(d) = \rho(k) = \theta. \tag{9}$$

So it follows that for a non-zero steady state infection in a regular network the average infection is equal to and has the exact form as the infection rate from (7), which is to be expected as the network is uniform; there is an equal probability of meeting any node in a given interaction.

Lopez-Pintado [14] derive a general threshold for non-zero steady-state infections as follows. Define the infection rate evolution function

$$H(\theta) = \sum_d \frac{P(d)\lambda\theta d^2}{\langle d\rangle(\lambda\theta d + 1)}. \tag{10}$$

Remarks; i) $H(\theta) > \theta$ corresponds to increasing infection rate, ii) $H(\theta) < \theta$ to decreasing. iii) $H(\theta) = \theta$ corresponds to the system being in a steady-state. iv) $H(0) = 0$ is always a stead-state as previously noted. v) $H(\theta)$ is an increasing and strictly concave function of θ.

Due to the strictly concave property of the evolution function (and both $H(\theta)$ and θ being bounded by $[0, 1)$), any $H(\theta)$ that begins above the line $H(\theta) = \theta$ must necessarily intersect that very line at a higher value for θ, leading to the existence of a non-zero steady-state. Conversely any $H(\theta)$ that begins below the line $H(\theta) = \theta$ cannot possibly intersect it showing a non-zero steady-state is impossible. These two cases are exactly determined by whether $H'(0) > 1$ or $H'(0) < 1$ respectively. This reasoning is show diagrammatically in Figure 1.

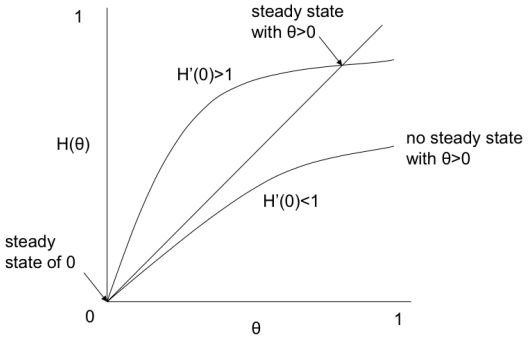

Figure 1: Diagrammatical explication; the gradient of $H(\theta)$ at 0 determines the existence of a non-zero steady-state infection rate. Reproduced from M.O.Jackson [28].

Since

$$H'(\theta) = \sum_d \frac{P(d)d^2}{\langle d\rangle}\frac{1}{(\lambda\theta d + 1)^2}, \tag{11}$$

and

$$H'(0) = \lambda\frac{\langle d^2\rangle}{\langle d\rangle}, \tag{12}$$

thus the general threshold for a non-zero steady-state is

$$\lambda > \frac{\langle d\rangle}{\langle d^2\rangle}. \tag{13}$$

For the regular network $\langle d\rangle^2 = \langle d^2\rangle$, so the result in (8) is recovered. For the Poisson distribution we have $\langle d^2\rangle = \langle d\rangle^2 + \langle d\rangle$ giving a threshold

$$\lambda = \frac{1}{1 + \langle d\rangle}. \tag{14}$$

For a system of infinite size, the scale-free distribution has a divergent $\langle d^2\rangle$ and so will in theory be able to sustain a non-zero infection for any positive λ.

State stability is also readily determined by $H'(0)$. For a small fluctuation in infection rate ε (akin to introducing infection in the system), if $H'(0) > 1$ then $H(\varepsilon) > \varepsilon$ and so the infection rate diverges from zero untill it reaches it's non-zero steady-state which is stable to fluctuations.

If $H'(0) < 1$ then $H(\varepsilon) < \varepsilon$ so fluctuations die and the steady-state with zero infection is stable.

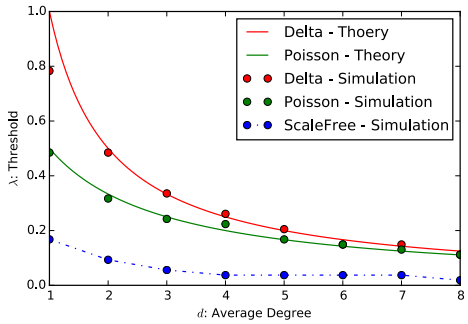

Figure 2: Thresholds for non-zero steady-state infections while varying average degree for various degree distributions, with $n = 1000$.

Figure 2 shows the theoretical predictions of the thresholds for varying average degree for the degree distributions discussed, and the agreeing results from simulation[7]. The thresholds significantly above 0 for the scale-free distribution, which goes against (13), are a result of simulating a finite system.

For a given average degree, the ordering of the thresholds of the distributions corresponds to the opposite of the mean-preserving spread ordering discussed in Section II, because the threshold is inversely proportional to $\langle d^2 \rangle$; $\lambda_{SF} < \lambda_{Po.} < \lambda_{\delta}$. For average degree greater than 2, distinguishing differences in dynamics between the distributions requires a high resolution of λ. Because of this we will now look primarily at networks with $\langle d \rangle = 2$, which exhibit the most variable dynamics over a large range of λ.

Figure 3 plots the theoretical change in steady-state average infection with λ for the regular network from (6), with the simulated steady-state infection rates for all three of the distributions discussed. *Remarks*: i) The threshold values of λ are as predicted by (13); for low λ, $\rho_{\delta} < \rho_{Po.} < \rho_{SF}$, the greater spread in degree leads to greater ability to sustain infection. ii) For high λ this ordering is reversed; $\rho_{SF} < \rho_{Po.} < \rho_{\delta}$, so the less spread distributions sustain a higher average infection.

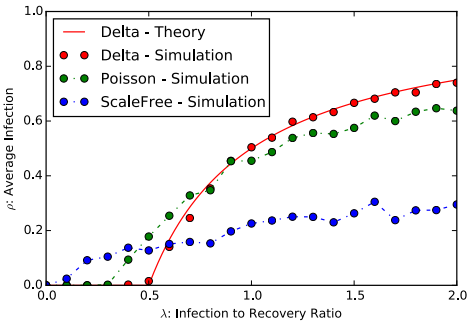

Figure 3: Average infection rate for varying λ, and various degree distributions, with $n = 1000$ and $\langle d \rangle = 2$.

This phenomena is predicted theoretically by Jackson [29], and has an intuitive explanation. In a network with large spread in its degrees there will exist many significantly isolated nodes, as well as a subnetwork of highly connected nodes with a significantly higher average degree than for the network as a whole. As Figure 2 shows that the thresholds decrease rapidly with increasing average degree, so this subnetwork can sustain an infection for low λ and the infection will be mostly contained to the subnetwork. At high λ a densely connected subnetwork is no longer required and infection can propagate through the whole network, yet the very isolated nodes are still highly unlikely to be infected often reducing the steady-state ρ; this is not the case for a regular network which does not contain any significantly isolated nodes and so results in a higher ρ.

III.3. Discussion

Key conclusions: i) There exist precise thresholds for non-zero steady-state infections which are dependent on the degree distribution of a network. ii) Predictions for the ordering of thresholds and steady-state average infection rate is possible in the low and high λ range based on ordering of the degree distributions by spread.

The model and the derived results can be readily used for prediction and guiding policy making in the areas of health care, immunisation, cyber security and opinion spreading, but

[7]The complete code for all simulations used can be found in Appendix G.

they also have further implications than their general predictive ability. For instance, though systems which exhibit scale-free network structure can maintain an infection even with small transmition rates/high recovery rates, they are much more susceptible to targeted vaccination than networks with less degree variance. In the scale-free case it would only be required to immunise the relatively small fraction of highly connected nodes for the infection to die out, compared with a much larger fraction of the equally important nodes in a regular network.

There are a number of limitations of the standard SIS model. Firstly, the networks formed using the configuration model result in neighbours having approximately independent degrees and so not exhibiting any of the detailed network structures seen in real networks, such as nodes clustering into groups and loops of linked nodes. Secondly, the network formation is independent of the state of the infection within the system. This fails to properly capture any effects infection may have on a host's connectivity, as well as any decision making process by agents involved in forming a bond. For instance agents may actively avoid meeting infected agents. This last point in especially relevant to economic interactions in which ties are generally formed with a definite purpose, and is dependent upon the reliability of parties involved.

IV. Adaptation

We aim to tackle the last limitation noted for the SIS model in Section III.3; the SIS model's lack of a reactive element to infection. We chose the SIS model initially because it is well suited to incorporating adaptation due to the repeated network breakdown and formation used to represent the random meetings.

We define adaptation as any change in the network structure in response to the state of part, or the whole of the system. So link removal, creation and rewiring, in direct response to an infection within the network is an example of adaptation. There is large scope for a variety of adaptations[8]. We choose to focus on a single adaptation in which infection is seen as a disadvantage, and thus agents attempt to avoid interactions with other infected agents, or equivalently infected agents have a reduced connectivity. Once an agent has recovered they also recover their initial level of connectivity. We call the generalised form of the SIS model that contains an adaptive element an *adaptive SIS model*.

There is empirical motivation for an adaptation of this type. Illness can naturally limit a host's exposure to others, and in some situations people can autonomously avoid meeting infected others. Anti-virus software can identify sites/emails which are likely to contain malicious software to help users avoid them. Entities forming economic ties make judgements of opposing parties and aim to avoid those that are unstable or failing.

Among the contexts in which adaptation is relevant, one of the most visible and rapidly expanding areas is financial contagion; studying the collapse of financial systems as contagion of failure through a network of connect financial bodies. The effects of financial collapses are detrimental across entire populations of the developed world making its investigation a high priority for policy makers and governments. Financial systems are inherently mathematical and evolve through (generally) logical decision-making, therefore adaption is a vital component of them. Despite this, the most visible papers on the subject do not contain an adaptive element, as shown in the following review.

IV.1. Review of Financial Contagion

Financial crises over the past decades have motivated attempts at understanding the causes and mechanisms responsible by modelling financial collapses as contagion through a network of connected financial bodies ('bank' for short). In this section I aim to introduce the

[8] We can differentiate between two types of adaptation; system-wide and local. A system-wide adaptation would be a change applied uniformly to every node or link in the system. For instance, uniformly reducing the degree of every node in the network once a certain average infection rate is reached would be a system-wide adaptation. A local adaptation is determined and applied at the level of individual nodes or links; once a node becomes infected it reduces it's degree by one, would be an example.

reader to the most visible papers and highlight their most common aspects. See Appendix D for a more in-depth review.

1. *Financial Contagion* - Allen, Gale (2000) [20]. The authors aim to establish whether financial collapse can be explained by contagion across a network of interdependent banks. They apply economic theory to establish a profitably optimal system of interbank loans between four banks. A bank is then shocked by an unexpectedly large set of deposit withdrawals causing the bank to default. The initial bank default deterministically transmits a loss to each of the banks it it indebted to, causing those to default in some cases; this mechanism of contagion is called *counterpart loss*. The authors establish that it is economically understandable that banks form systems in which bank default can be transmitted to others through counter party losses.

2. *Network Models and Financial Stability* - Nier et al. (2007) [21]. The authors aim to investigate how the structure of financial networks affects its susceptibility to systematic breakdown. They model the financial system as a random network representing banks and their interbank loaning. A bank is shocked/made to default at random and this loss transmits through interbank loans (links) via counter party loss. The extent of collapse in the network is analysed for varying network connectivity, size of loan per connection, size of each banks buffer to losses, and system size. The results show non-monotonic changes in the extent of contagion with increasing connectivity, amongst others.

3. *Stability Analysis Of Financial Contagion due to Overlapping Portfolios* - Caccioli et al. (2014) [23]. The authors aim to investigate the stability of a system of banks investing in a set of assets. They model the system as a random bipartite[9] network of banks and assets. Banks default once they incur enough losses on assets. Defaulted banks sell all their assets and the worth of an asset falls as quantities of it are sold, thus transmitting losses through overlapping assets leading to more defaults. Stability is investigated by computing the probability of system collapse for varying parameters. The authors show the existence of various system phase transitions.

The preceding papers all investigate closely related questions, and so contain common elements.

Elements common to all models: i) Static random network. ii) Infection is transmitted deterministically through network links based on the properties of nodes. iii) Nodes do not recover. iv) There is no network reaction to infection. For a fully relevant model of financial contagion which can be used in policy making, some form of an adaptive element is required.

There a number of significant differences inherent in the SIS model compared to financial contagion models: i) Random meeting model, not a static random network. ii) Infection is transmitted randomly, not deterministically based on the properties of nodes. iii) Nodes recover from infection.

We do not propose that an adaptive SIS model is an actual model of financial contagion. Our aim is to make initial ground by presenting a simple but extendable adaptive model of contagion generally, though it could be used as an example for incorporating adaptation in models of financial contagion in the future.

V. The Popularity Model

The configuration model and its use of degree distributions is relatively unsuited for an agent-based adaptation. Firstly altering a node's degree, based on its infected/susceptible state, must be a discrete process. And altering the degree distribution is a system-wide adaptation. We propose the following model, which allows for a continuous change in the connectivity of a single node, as a candidate to incorporate agent-based adaptation.

V.1. Model Specification

This model is a generalisation of the Erdős-Rényi model. In the ER model, links are formed with a probability p. In the popularity model each node is associated with a popularity; link ij is formed with a probability which is a function of the popularities of the nodes i and j.

[9] A bipartite network consists of two sets of nodes, and links are only formed between nodes in differing sets.

Define $P(p)$ to be a probability distribution over $p \in [0,1]$, called a *popularity distribution*; this replaces the degree distribution used in Section II as the defining object in the structure of a network. Random networks with n nodes and $P(p)$ are formed as follows[10]: i) Take n samples from $P(p)$ to form a sequence of *popularities* $\{p_1, p_2, \ldots, p_n\}$. ii) Form links ij with probability $p_i p_j$;

$$\text{Prob}(ij) = p_i p_j. \tag{15}$$

V.2. Degree Distribution Analysis

Even though the degree distribution is not a defining object of networks in the popularity model, it is still central in the analysis of the resultant networks, and enables comparison to networks formed using the configuration model.

Average Degree: Given n nodes and popularity distribution $P(p)$, the average degree of node i is

$$d_i = p_i \sum_{j \neq i} p_j, \tag{16}$$

which for large n is approximately

$$d_i = p_i n \int_0^1 P(p) p \, dp = p_i n \langle p \rangle. \tag{17}$$

Averaging this over all nodes is the average degree for the network, and using the same approximation gives

$$\langle d \rangle = \frac{\sum_i d_i}{n} = \frac{n^2 \langle p \rangle \int_0^1 P(p) p \, dp}{n} = n \langle p \rangle^2. \tag{18}$$

Exact forms of higher order moments require a complete form of the degree distribution.

Degree Distributions: Let $\mathbb{P} = \{p_1, p_2, \ldots, p_n\}$ be the probability sequence for a network. Let $\mathbb{Q} = \{\{p_i, p_j\} | p_i, p_j \in \mathbb{P}\}$ be the set of all subsets of $\mathbb{P}$ with cardinality two (all pairs of popularities). The exact form of the degree distribution is

$$P(d) = \sum_{|S \subseteq \mathbb{Q}| = d} \prod_{\{p_i, p_j\} \in S} p_i p_j \prod_{\{p_k, p_l\} \in \mathbb{Q} \setminus S} (1 - p_k p_l). \tag{19}$$

If $p_i = \tilde{p}$ for all i, the distribution reduces to the binomial distribution with a probability of success per trail $\tilde{p}^2$. Thus the popularity model with constant popularity rightly reduces to the Erdős-Rényi model.

Though the exact form of the degree distribution may not be intractable, initial analysis can be easily gain through computationally creating networks using the popularity model and analysing the resultant degree distributions directly.

Figure 4 shows various Beta(α,β)[11] distributions and the Dirac-Delta distribution δ, from which we will create networks using the popularity model. We can form networks with a given average degree by normalising popularity distributions using the result from (18) and the mean of the popularity distribution[12].

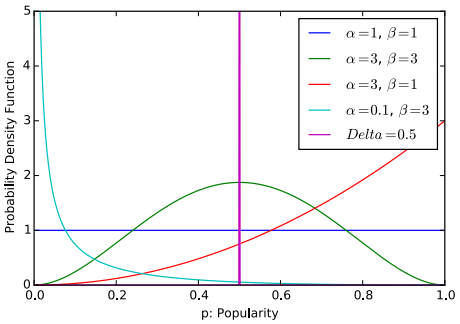

Figure 4: Various popularity distributions using the Beta(α, β) distribution and δ distribution.

Figure 5 shows the resulting degree frequencies for $\langle d \rangle = 20$, while Figure 6 shows these same frequencies in a log-log plot. See Appendix E for further plots of degree frequencies from popularity distributions.

[10] Again I only consider simple, undirected and unweighted networks.

[11] The probability density function for the Beta distribution is given below, where α and β are positive shape parameters.

$$f(x; \alpha, \beta) = \frac{x^{\alpha-1}(1-x)^{\beta-1}}{\int_0^1 u^{\alpha-1}(1-u)^{\beta-1} \, du} = \frac{1}{B(\alpha, \beta)} x^{\alpha-1}(1-x)^{\beta-1}.$$

[12] For the Beta distribution $\langle p \rangle = \frac{1}{1+\frac{\beta}{\alpha}}$.

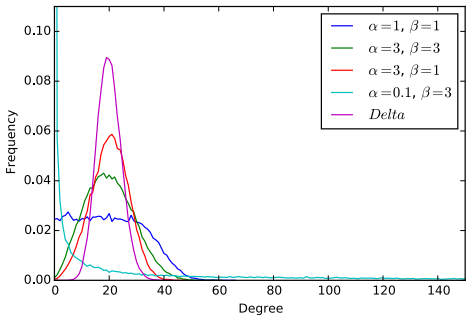

Figure 5: Corresponding resultant degree frequencies for normalised popularity distributions from Figure 4, for $n = 1000$ and $\langle d \rangle = 20$. Averaged over 50 realisations.

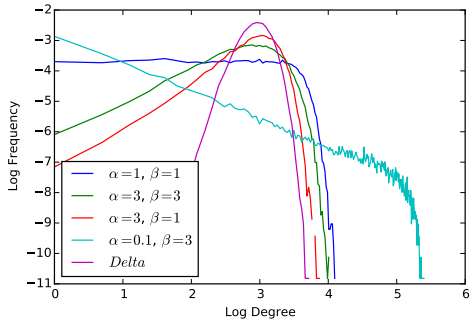

Figure 6: Corresponding resultant log-log degree frequencies for normalised popularity distributions from Figure 4, for $n = 1000$ and $\langle d \rangle = 20$. Averaged over 50 realisations.

Remarks: i) A uniform popularity distribution (Beta(1,1)) results in approximately uniform degree frequencies from zero to $2\langle d \rangle$. ii) A power law popularity distribution (Beta(0.1,3)) creates power law degree frequencies (scale-free networks) as seen by its decreasing linear form up to a limiting degree in the log-log plot. iii) The δ popularity distribution produces a Poisson degree distribution (E-R network). iv) A mean-preserving spread of a popularity distribution generally forms a corresponding mean-preserving spread of degree frequencies. The Beta(3,3) popularity distribution is a mean-preserving spread of the δ popularity distribution and the corresponding resultant ordering in degree frequencies is visible in Figures 5 and 6.

For a significantly fuller investigation of generalised Erdős-Rényi graphs see Södberg [30,31] and Caldarelli et al. [32].

VI. Adaptive SIS Model

VI.1. Model Specification

This model a generalisation of the random-meeting SIS model specified in Section III, in which we replace the defining degree structure with a popularity structure and add an adaptive element.

The system consists of a set of agents which we represent by nodes. Each agent alternates between two states; infected or susceptible. An agent's defining property is its popularity, which defines a popularity sequence for the set of agents. We introduce an adaptation factor $x \in [0, 1]$ which reduces the popularity of infected agents. An agent with popularity p_i has adapted popularity xp_i while infected. The average system infection – the fraction of agents which are infection – is denoted by ρ.

System evolution per period:

i) Agents i, j meet with probability

$$\text{Prob}(ij) = \begin{cases} p_i p_j, & \text{if both } i,j \text{ susceptible,} \\ x p_i p_j, & \text{if one of } i,j \text{ infected,} \\ x^2 p_i p_j, & \text{if both } i,j \text{ infected,} \end{cases}$$

defining a network of meetings generated by the *adapted* popularity sequence.

ii) Infection is transmitted to susceptible agents in meetings with infected agents with probability ν.

iii) Infected agents recover with probability δ.

Note that the outcome for every agent is computed using the *initial* system state for that period. The evolution processes occur simultaneously each period, and so in effect independently.

VI.2. Results

Computation is used to gain results for an initial understanding of the model. We choose to focus on the popularity distributions that differ most in their spread; Beta$(0.1, 3)$, Beta$(1, 1)$ and the δ distribution.

For no adaptation ($x = 1$), results from Section III should be regained for networks with corresponding degree frequencies; the δ popularity distribution results in Appendix F Figure 21 and the Poisson distribution results in Figure 3 show this correspondence. Figure 7 shows the equivalent plot with an adaptation factor $x = 0.7$. *Remarks*: i) Increased thresholds for non-zero infection. ii) Decreased average infection rates. iii) Ordering of thresholds by mean-preserving spreads preserved. iv) Ordering of high λ average infection rates by mean-preserving spreads preserved. v) Systems converging - see Appendix F Figure 22 for further corroboration on their convergence.

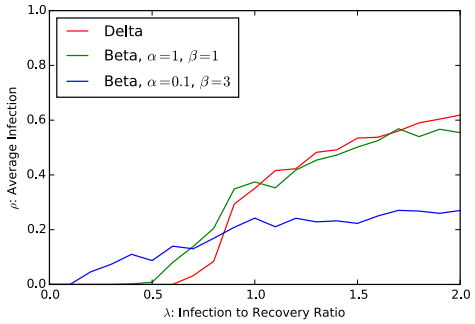

Figure 7: Steady-state average infection for various popularity distributions with varying λ. $n = 1000$, $\langle d \rangle = 2$, $x = 0.7$.

Figures 8, 9 and 10 show heat maps of steady-state average infection ρ in x-λ space for the increasingly spread distributions δ, Beta$(1, 1)$ and Beta$(0.1, 3)$ respectively. All of the plots exhibit a continuous (second-order) phase boundary determining the position of the system's phase transition between sustaining a non-zero infection and not, in x-λ space.

The form of the phase boundaries are dependent upon spread in popularity distribution and the network's resultant average degree[13]. The phase boundaries can generally be ordered by mean-preserving spreads of popularity distributions. Boundaries are more extreme given a greater spread in popularity; compare Figure 10 to 8. We expect that for unbounded λ the phase boundary will tend towards $x = 0$.

Limiting cases: i) For $x = 1$ the system reduces to the non-adaptive model. ii) For $x = 0$, infected agents have a zero probability of meeting others, and no diffusion of infection can occur. iii) For $\lambda = 0$ and a finite recovery rate, infection is transmitted with zero probability and so no diffusion of infection can occur. iv) For zero recovery rate (unbounded λ), infection will eventually spread to the entire system given $x \neq 0$ leading to a stable state with $\rho = 1$. For $x = 0$, the system will be stable with ρ equal to the initial infection fraction of nodes.

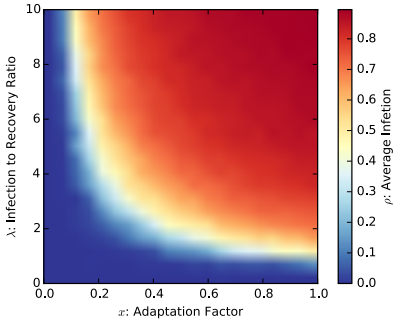

Figure 8: Heat map of the steady-state average infection in a δ popularity distribution system for varying λ and x. $n = 1000$, $\langle d \rangle = 2$.

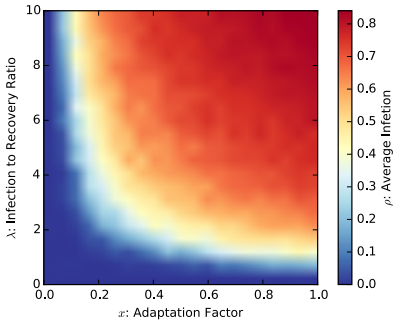

Figure 9: Heat map of the steady-state average infection in a Beta$(1, 1)$ popularity distribution system for varying λ and x. $n = 1000$, $\langle d \rangle = 2$.

[13] Compare Figure 8 and Appendix F Figure 28 to see the effects of increasing a network's average degree.

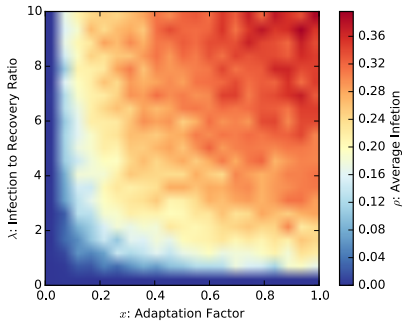

Figure 10: Heat map of the steady-state average infection in a Beta$(0.1, 3)$ *popularity distribution system for varying* λ *and* x*.* $n = 1000$*,* $\langle d \rangle = 2$*.*

VI.3. Discusion

We have been able to study an adaptive model that has wide applications and implications.

At a theoretical level we have produced a guide for theoretical analysis of such complex and adaptive diffusion processes. As well as presenting a readily extendable system that produces interesting and reactive dynamics.

The adaptive SIS model can readily be applied to the epidemiology of symptomatic diseases. For instance if a disease naturally inhibits the host's ability to come into contact with others, or in the policy making regarding the prevention of disease spread; the phase boundaries could guide the extent to which a policy would have to reduce the propensity of infected agents to meet others to ensure the disease dies out. This is especially relevant to bacterial infections and diseases with no known vaccines.

Lastly, the model has particular importance in the area of financial contagion. Though many of the elements from the reviewed research into such models aren't present, our model embodies other pertinent and, as yet, missing dynamics. Firstly, financial bodies form ties bilaterally – both parties must agree – and the decision on whether to form such ties are based on judgements of the opposing parties. The link formation rule in the popularity model (15) captures this bilateral and node-judgmental based decision making. Secondly, we were able to incorporate an adaptive element into this link/decision making process, capturing the propensity for banks to avoid forming ties with unstable/toxic bodies.

VII. Conclusion

The objective of this paper is to present and investigate a reactive diffusion process. We do so by generalising a well established model of a diffusion process to incorporate an adaptive element. We choose the SIS model as it is well suited to incorporating adaptation.

Some of the main contributions of this paper are the introduction of an adaptive model that is relevant and easily extendable. The relationships between the Popularity model and the established configuration model have been investigated. The effects of an adaptive element on thresholds and average rate of infection are shown. The existence of a phase boundary in x-λ space was established.

Suggestions for further possible research: i) Vary x to be greater than 1 to investigate infection spread when those infected increase their connectivity. ii) Investigate other adaptations, for instance adaptations which vary between nodes, or system-wide adaptations. iii) Use a different Popularity model link formation rule than (15), such as a threshold for link formation, or additive popularities, rather than multiplicative, to represent unilateral link formation. iv) Extend the adaptive SIS to better model financial contagion by incorporating a deterministic transmission rule. v) A statistical mechanical investigation into adaptive SIS phase transitions. vi) Compare results from the adaptive SIS model to actual data on the spread of symptomatic diseases. vii) Apply the model to other propagation phenomenon such as the spread of information, opinions and online viral media.

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Appendices

A. Example Degree Distributions

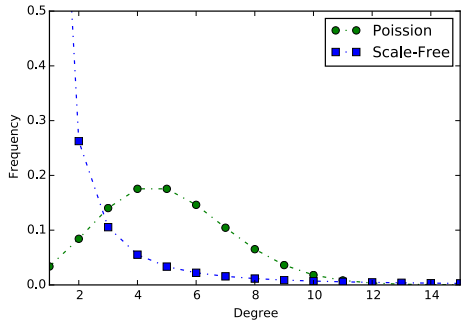

Figure 11: Poisson and scale-free degree distributions for $\langle d \rangle = 5$. The δ distribution would simply be a spike of frequency (height) 1 at $d = 5$.

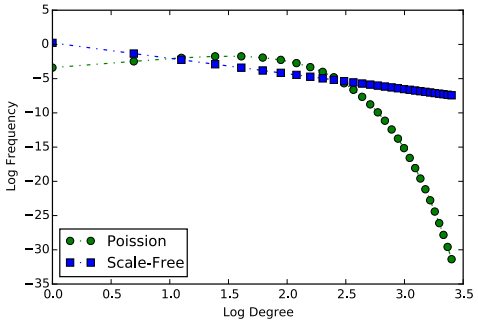

Figure 12: Log-log degree distributions for the Poission and scale-free for $\langle d \rangle = 5$. The δ distribution would simply be a spike of frequency (height) $log(1) = 0$ at $log(d) = 5$. Note the distinctly larger 'tails' of the scale-free distribution showing its greater spread in degree.

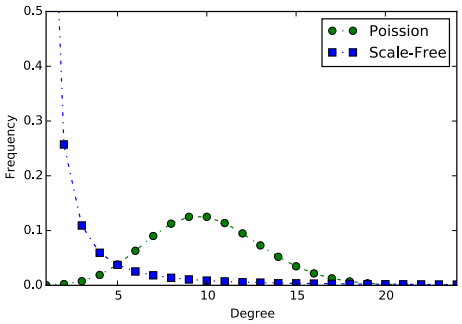

Figure 13: Poisson and scale-free degree distributions for $\langle d \rangle = 10$. The δ distribution would simply be a spike of frequency (height) 1 at $d = 10$.

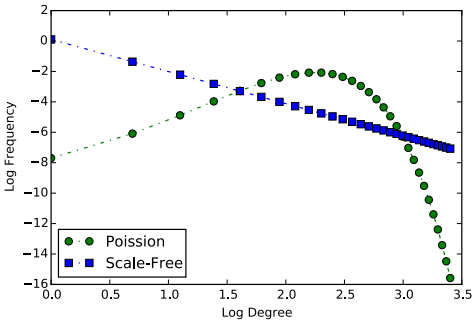

Figure 14: Log-log degree distributions for the Poission and scale-free for $\langle d \rangle = 10$. The δ distribution would simply be a spike of frequency (height) $log(1) = 0$ at $log(d) = 10$. Note the distinctly larger 'tails' of the scale-free distribution showing its greater spread in degree.

B. Stochastic Ordering

Stochastic Ordering is an attempt at ordering probability distributions.[14]

First order stochastic dominance captures that one probability distribution is "bigger" or is "higher" than another; 'rational agents' betting on outcomes betweens distibutions should always choose the dominant distribution. Consider two probability distributions (discrete or continuous) $P(d)$ and $\widetilde{P}(d)$. $P(d)$ first order stochastically dominates $\widetilde{P}(d)$ if

$$\sum f(d)P(d) \geq \sum f(d)\widetilde{P}(d),$$

for all nondecreasing functions[15] f.

$P(d)$ can be formed by shifting probability mass/weight upwards/to-the-right on the $\widetilde{P}(d)$ distribution.

Second order stochastic dominance captures that $P(d)$ has at least a high mean as $\widetilde{P}(d)$ but is more centralised on a single value and so more predictable. $P(d)$ second order stochastically dominates $\widetilde{P}(d)$ if

$$\sum f(d)P(d) \geq \sum f(d)\widetilde{P}(d),$$

for all nondecreasing, concave functions[16] f. First order stochastic dominance implies second order.

A mean-preserving spread is a special case of a distribution $\widetilde{P}(d)$ *being* second order dominated by $P(d)$ in which they have equal means; $\langle d \rangle = \widetilde{\langle d \rangle}$. It removes the first order 'part' (i.e just simply higher gains) and accentuates the predictability due to spread part. It implies

$$\sum f(d)P(d) \geq \sum f(d)\widetilde{P}(d),$$

for all concave functions f, and similarly

$$\sum f(d)P(d) \leq \sum f(d)\widetilde{P}(d),$$

for all convex functions f.

These are stated without proof but with self-persuasion of their validity. The second moment of the degree of the network $\langle d^2 \rangle = \sum_d P(d)d^2$ is the weighted sum over the convex function $f(d) = d^2$ and so $\langle d^2 \rangle$ increases with increasing spread of $P(d)$ while holding its mean constant.

[14] Only gives a partial ordering; for two distributions, neither may dominate the other.

[15] Equivalent conditions:

- $\sum_0^x P(d) \leq \sum_0^x \widetilde{P}(d)$ for all x,
- $\sum_x^\infty P(d) \geq \sum_x^\infty \widetilde{P}(d)$ for all x.

[16] Equivalent conditions:

- $\sum f(d)P(d) \leq \sum f(d)\widetilde{P}(d)$ for all non-increasing, convex functions f,
- $\sum_{z=0}^x \sum_{d=0}^z P(d) \leq \sum_{z=0}^x \sum_{d=0}^z \widetilde{P}(d)$ for all x.

C. Further SIS Results

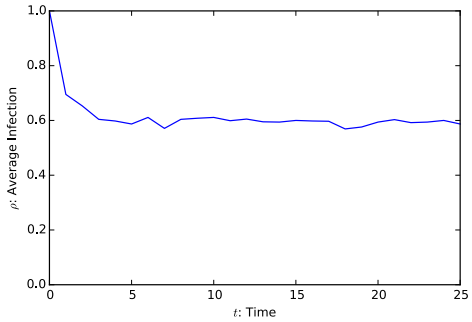

Figure 15: Example of simulated non-zero steady-state evolution of the SIS model; for a δ degree distribution (regular network), $n = 1000$, $\langle d \rangle = 3$, $\lambda = 1$.

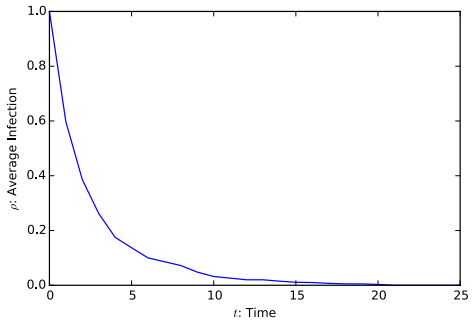

Figure 16: Example of simulated dying out of infection for a sub-threshold SIS model; for a δ degree distribution (regular network), $n = 1000$, $\langle d \rangle = 3$, $\lambda = 0.1$.

D. Extended Financial Contagion Literature Review

1. *Financial Contagion* - Allen, Gale (2000) [20]. i) *Research question*: Can financial crisis be explain by contagion across a network of banks. ii) *General model*: Static directed[17] network of four banks. Links represent bank lending. A pool of consumers that deposit and withdraw funds in banks. System evolves over a one-time series of events at t=0,1,2. Banks aim to maximise more profitable longer term investment (from t=0 to t=2) of deposits, achieved through loaning along the designated links. Loans are later liquidated in order to absorb shorter term consumer withdrawals (at t=1) as required. iii) *Central quantities*: Designated network structure; completely connected, banks paired or a connected loop. Fraction of consumers making short term withdrawals in each bank. iv) *System shock*: Single bank receives an unexpectedly large withdrawal at t=1 for which it currently does not have the funds for, so goes insolvent and defaults on its debts. v) *Contagion mechanism*: Initial bank default transmits losses through the interbank loans (links) causing further possible insolvencies. Call this mechanism *counterparty loss*. vi) *Main results*: It is possible to model financial collapse as contagion of bank default transmitted through interbank loans. iv) *Limitations*: Model is overly specialised.

2. *Network Models and Financial Stability* - Nier et al. (2007) [21]. i) *Research question*: How does the structure of financial networks affect its susceptibility to systematic breakdown. ii) *General model*: Static directional and weighted[18] ER network representing direction and size of interbank loans between banks. Banks hold a finite buffer against loss. iii) *Central quantities*: System size. Total equity within system. Density of interbank loaning (link density). Average size of loans (average link weight). Bank buffer size. iv) *System shock*: Set random bank as insolvent. v) *Contagion mechanism*: Counter party loss. Insolvent banks default on all loans, transmitting losses through interbank loans (links). Banks that receive total losses greater than their buffer become insolvent. vi) *Main results*: Non-linear increase in extent of contagion with decreasing buffer size. Non-monotonic change in extent of contagion with increasing connectivity (link density). Extent of contagion normalised by system size increases with decreasing system size. vii) *Limitations*: Shocks are both idiosyncratic and contained within a single bank, both of which are not the case in practice. Only considered different levels of connectivity within a single network structure type.

3. *Stability Analysis Of Financial Contagion due to Overlapping Portfolios* - Caccioli et al. (2014) [23]. i) *Research question*: Given a network of leveraging[19] banks with overlapping portfolios, how does the system's stability to shocks change with varying system parameters. ii) *General model*: Non-directional weighted bipartite[20] random network. Links represent a bank's investment in an asset. Asset values depreciate exponentially as banks liquidate them. Banks have a finite buffer against losses. A bank is solvent while its liabilities, proportional to its leverage, is less than its total value of assets plus buffer. iii) *Central quantities*: Level of leverage. Diversification; average number of assets each bank invests in (average bank degree). Market crowding; ratio of number of banks to assets. iv) *System shock*: Set random bank as insolvent, or depreciate value of random asset. v) *Contagion mechanism*: Insolvent banks liquidate all their assets, thus driving those asset values down. This leads to further possible insolvency of banks connected through overlapping assets (banks linked through a single asset). vi) *Main results*: It is possible to model financial collapse as contagion through overlapping portfolios. The contagion is self-reinforcing; if initial shock is not absorbed then all banks in the connected network component go insolvent. Non-monotonic change in probability of collapse with increasing diversification; lower and upper threshold on non-zero proba-

[17] Links are one way; it is no longer the case that $g_{ij} = g_{ji}$.

[18] Links are associated with a weight; adjacency matrix g_{ij} now contains a spectrum of values.

[19] Using debt to finance assets. Banks with substantially higher debt than equity are considered to be highly leveraged.

[20] A bipartite network consists of two sets of nodes, and links are only formed between nodes in differing sets.

bility (phase transitions). Decreasing market crowding generally lowers probability of collapse and changes positions of thresholds. Extent of collapse and thresholds independent of shock type. Their exists a threshold on leverage for possibility of collapse. vii) *Limitations*: Passive portfolio management (static system). Doesn't include the counter party loss channel of contagion.

E. Degree Distributions from the Popularity Model

Resultant degree distributions corresponding to popularity distributions.

The remarks noted in Section II hold for these results.

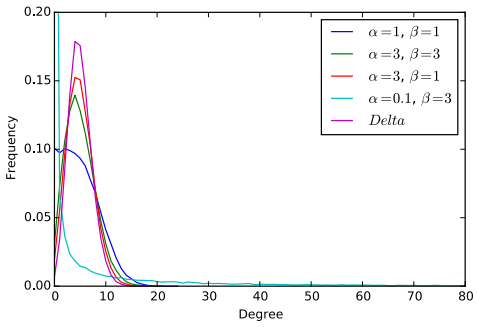

Figure 17: Corresponding resultant degree frequencies for normalised popularity distributions from Figure 4, for $n = 1000$ and $\langle d \rangle = 5$. Averaged over 50 realisations.

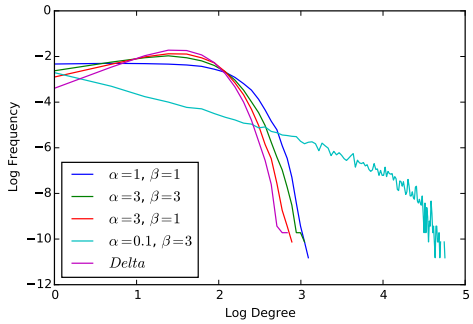

Figure 18: Corresponding resultant log-log degree frequencies for normalised popularity distributions from Figure 4, for $n = 1000$ and $\langle d \rangle = 5$. Averaged over 50 realisations.

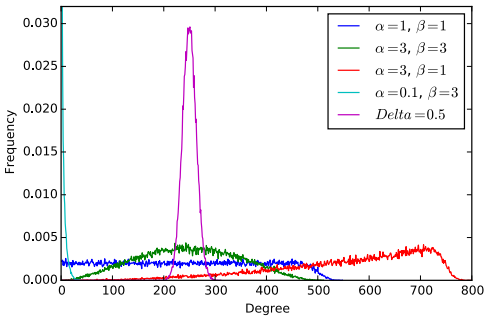

Figure 19: Corresponding resultant degree frequencies for the unnormalised popularity distributions shown in Figure 4, for $n = 1000$. Averaged over 50 realisations.

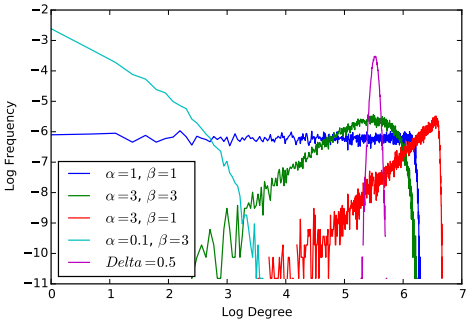

Figure 20: Corresponding resultant log-log degree frequencies for the unnormalised popularity distributions shown in Figure 4, for $n = 1000$. Averaged over 50 realisations.

F. Further Adaptive SIS Results

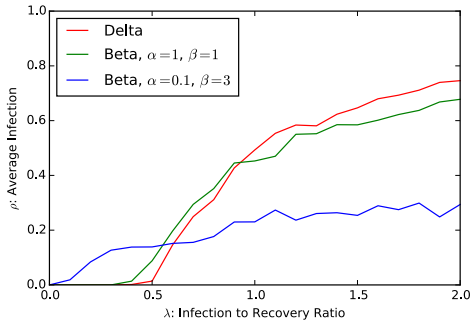

Figure 21: Steady-state average infection for various popularity distributions varying λ. $n = 1000$, $\langle d \rangle = 2$, $x = 1$; no adaptation.

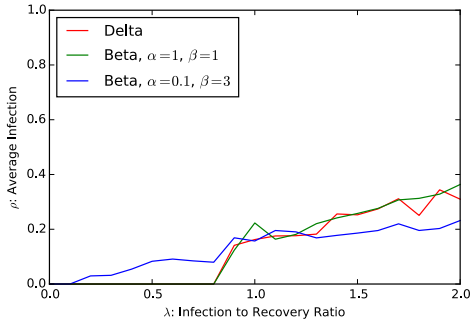

Figure 22: Steady-state average infection for various popularity distributions varying λ. $n = 1000$, $\langle d \rangle = 2$, $x = 0.35$. Note the increased thresholds and decreased average infections, as well as a convergence of the systems.

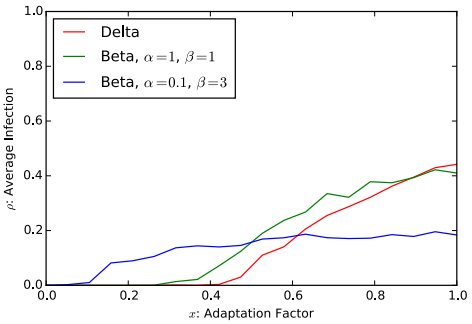

Figure 23: Steady-state average infection for various popularity distributions varying x. $n = 1000$, $\langle d \rangle = 2$, $\lambda = 1$.

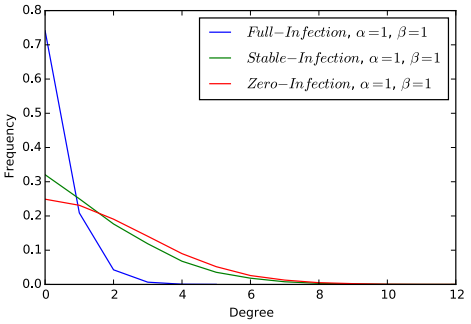

Figure 24: Resultant degree frequencies from adapted popularity sequence for normalised $Beta(1, 1)$ distribution for various average infection . $n = 1000$, $\langle d \rangle = 2$, $x = 0.4$.

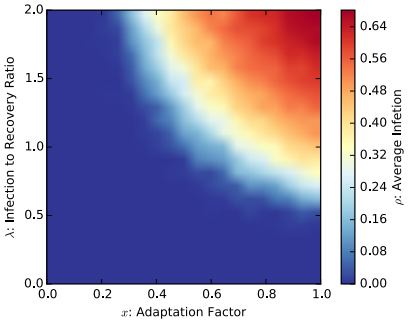

Figure 25: Heat map of steady-state average infection in a δ popularity distribution system for varying λ and x. $n = 1000$, $\langle d \rangle = 2$.

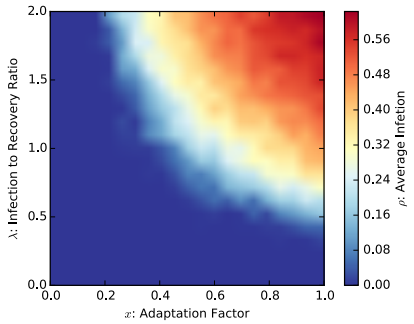

Figure 26: Heat map of steady-state average infection in a Beta$(1, 1)$ popularity distribution system for varying λ and x. $n = 1000$, $\langle d \rangle = 2$.

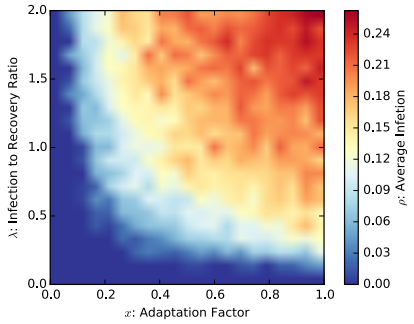

Figure 27: Heat map of steady-state average infection in a Beta$(0.1, 3)$ popularity distribution system for varying λ and x. $n = 1000$, $\langle d \rangle = 2$.

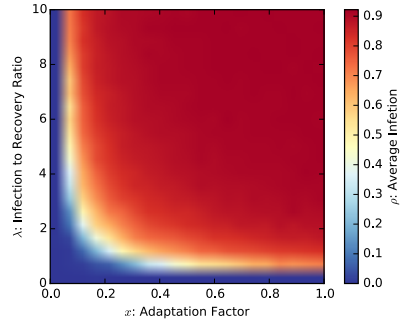

Figure 28: Heat map of steady-state average infection in a δ popularity distribution system for varying λ and x. $n = 1000$, $\langle d \rangle = 8$.

G. Simulations

All simulations and plots were written in Python. Below are links to iPython Notebooks which contain complete code for each simulation, including explanatory comments.

- Degree Distribution Examples: results in Appendix A. `https://gist.github.com/DBCerigo/30dea97eecc461300b08`
- SIS Evolution: results in Appendix C. `https://gist.github.com/DBCerigo/0aeb443640ad86518d39`
- SIS Thresholds: results in Figure 2. `https://gist.github.com/DBCerigo/c79b035be9a5cb428270`
- SIS Steady-State Average Infection: results in Figure 3. `https://gist.github.com/DBCerigo/8af94ff81f0132e03cef`
- Popularity Model resultant Degree Distribtuions: results in Figures 4, 5 and 6, as well as results in Appendix E. `https://gist.github.com/DBCerigo/461242a08f0d6c3bffc0`
- Adaptive SIS Steady-State Average Infections Varying Infection-Recovery Ratio λ: results in Figures 7, 21, 22 and 24. `https://gist.github.com/DBCerigo/ab04a3520960e699cfa4`
- Adaptive SIS Steady-State Average Infections Varying Adaptation Factor x: results in Figure 23. `https://gist.github.com/DBCerigo/8b3a220fa0262ba6ed89`
- Adaptive SIS Heat Maps: results for all heat maps. `https://gist.github.com/DBCerigo/c6ad06a8d000b442af54`