Speed vs Resilience in Contagion

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Abstract

We highlight a trade-off between speed (the rate at which behaviours propagate in the population) and resilience (the measure of initial adopters required for spreading) in models of threshold contagion: contagion is faster in networks where it is harder to initiate contagion. We derive various orderings over networks under which this trade-off is stark. While this trade-off holds between pairs of networks for possibly different contagion thresholds, we also outline conditions under which, for a given contagion threshold, one network is both less resilient and propagates behaviours more quickly; this highlights the role of intermediate links as bulwarks against contagion.

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1 Introduction

Models of threshold contagion, in which a player adopts an action if at least proportion q of her neighbours do the same have played a prominent role in the analysis of how behaviours spread through a networked population.[1] We show that when a population is more resilient (less prone to contagion), contagion is quicker when it does occur. *Resilience* is measured by the minimal measure of agents required to initiate contagion. *Speed* of contagion is measured by the limit rate of contagion in the population if it is initiated. This is because nearby links lower resilience but also lower the speed of contagion.

We prove our results in a continuum population network which allows a clean demonstration of the speed and resilience connection . We develop an ordering over networks on which one can perform comparative statics of contagious dynamics. While this speed-resilience trade-off always holds between any two networks for possibly different contagion thresholds, we also develop conditions under which for a given contagion threshold, one network is both less resilient *and* propagates behaviours more quickly.

Our results complement the work of Morris (2000) who made the observation that with low neighbour growth,[2] the threshold at which contagion obtains tends to be high. In the language of our paper, networks where behaviors spread slowly also tend to be resilient. The influential papers of Centola and Macy (2007) and Centola, Eguíluz, and Macy (2007) highlight the difference between simple and complex contagion and show in simulations that under complex contagion, more uniform rewiring in the ring lattice model of Watts and Strogatz (1998) always speeds up the propagation of contagion, but only up to a point–beyond which contagion might not occur at all. Intuitively, since uniform rewiring replaces

[1] Threshold contagion was analyzed in Morris (2000) (see also Schelling (1973) for an early study of binary choice in a non-networked population, and Blume (1993, 1995); Ellison (1993); Morris (1997) for game-theoretic analyses on graphs). In the language of the influential work of Centola and Macy (2007), threshold contagion is a form of complex contagion where multiple infected neighbours are required for spreading, as opposed to simple contagion where only a single infected neighbour is required.

[2] i.e., when players reached (and hence potentially infected) in t steps grows slower than exponentially.

nearby links with distant links, this has a similar flavor to our results.[3] Our paper provides analytical results which allow us to understand the tradeoff better. In particular, this allows us to analyze richer variations in networks than simply the degree to which links are randomly rewired. For instance, we study the effect of replacing intermediate-distance links with both local and faraway links. It turns out that intermediate links are an important bulwark against contagion, without which the network can simultaneously have low resilience (many local links) while also spreading behaviors quickly (many faraway links).

More recent work such as that of Eckles, Mossel, Rahimian, and Sen (2018) similarly studies contagion on the ring lattice (a discrete analog of our setting). Under uniform rewiring, increasing the rate of rewiring (hence the proportion of long ties) does not speed up threshold contagion as the number of agents grow large.[4] They propose a model of noisy contagion in which with some small probability, an agent might be infected through simple contagion–where only a single infected neighbour is sufficient to be infected–and show that this can speed up contagion substantially. Our paper complements this by proposing an alternate mechanism: instead of uniform rewiring, we study a richer class of networks in which the density of links varies in distance and show that this can matter for limit speeds without noise in infection thresholds.

Our benchmark model involves a continuum population situated on the real line so that there is an infinite measure of agents; working with an infinite measure gives cleaner results. In Appendix A we adapt our results to agents situated on the unit circle, such that there is a bounded measure of agents. With this adaption, our setting is that of a graphon game (Parise and Ozdaglar, 2020); we thus follow the important work of Erol, Parise, and Teytelboym (2020) in studying threshold contagion over a graphon. We show that at each step of the contagion process, the set of infected agents on the graphon–to which our results on the speed resilience tradeoff apply–approximates that on the random graph sampled

[3] In Appendix A we show that the manner in which contagion is sped up under uniform rewiring is particular to small populations: in large populations, the force driving the increase in speed in the simulations of Centola and Macy (2007) and Centola, Eguíluz, and Macy (2007) disappear; rather, the speed-resilience tradeoff operates through a distinct mechanism.

[4] This is because for an uninfected agent situated where the those surrounding her are similarly uninfected, the probability that many of her rewired links happen to be to infected agents vanishes at an exponential rate.

from the graphon.[5] Further, in Appendix B we show that the tradeoffs we identify continue to hold in higher dimensions.

The contagion in our continuum population situated on a line is related to "global game" models of contagion in incomplete information games. The general connection between global games and continuum networks is described in Morris and Shin (2006). "Resilience" corresponds to the existence of high probability approximate common knowledge events and "speed" corresponds to probability of actions getting deleted in each round of iterated deletion.

2 Model

Our base model is of a weighted graph with an infinite measure of agents indexed $i \in \mathbb{R}$. Each agent i allocates weights to his neighbours, where the total weight is normalized to 1. Each agent assigns half his weights to the left and half to right. The weights are translation invariant and symmetric. Thus each agent i assigns weight $G(x)$ to neighbours in the interval $[i, i+x]$; and also $G(x)$ to neighbours in the interval $[i-x, i]$; where G:$[0,+\infty) \to \left[0, \frac{1}{2}\right]$, $G(0) = 0$ and $G(x) \to \frac{1}{2}$ as $x \to +\infty$.

Denote the anti-cumulative distribution as $\overline{G} := \frac{1}{2} - G$., so $\overline{G}(x) = \frac{1}{2} - G(x)$ is the weight agent i assigns to agents in the interval $[x, +\infty)$. .

We will focus on distributions in the following class.

Definition 1. Let $\mathcal{G}$ be the set of cumulative distribution functions G :$[0,+\infty) \to [0, \frac{1}{2}]$ such that for each $G \in \mathcal{G}$,

(i) G admits a bounded density $g : [0,+\infty) \to [0, M]$ for some $M < +\infty$.

(ii) G fulfils strictly decreasing differences i.e., for $x > x' \geq 0$, $g(x) < g(x')$ whenever $g(x') > 0$.

[5] Erol, Parise, and Teytelboym (2020) show that the final set of infected agents in the sampled graph can be approximated arbitrarily well by that on the graphon. We are interested in speed and thus focus on the step-by-step evolution of the contagion process. Erol, Parise, and Teytelboym (2020, Propositions 1 and 2) show that at each time period, if an agent was infected on the graphon, then with high probability they are also infected on the sampled graph; we establish the opposite inclusion.

Part (i) requires that agents do not assign strictly positive weight to any individual neighbour; part (ii) implies some degree of homophily: one might view $\mathbb{R}$ as a one-dimensional space of characteristics — the condition states that each agent has more links to those similar to themselves than to those further away. We note that although we present our results in the main text for a fixed, deterministic graph, our results also hold for random graphs where each agent's links are distributed according to G (see Appendix A). In this context, part (ii) states that each agent has in expectation more nearby than faraway neighbours. [6]Note that for $G \in \mathcal{G}$ since $\lim_{x\to\infty} G(x) = \lim_{x\to\infty} \int_0^x g d\mu = 1/2$ and g is strictly decreasing, this implies $\lim_{x\to\infty} g(x) = 0$.[7]

Time is discrete and infinite, indexed $\mathcal{T} = 0, 1, 2, \ldots$. For time $t \in \mathcal{T}$, we use $I_t \subseteq \mathbb{R}$ to denote the infected players, and use $m_t := \mu(I_t) < \infty$ to denote the measure of infected players. Let $a_t := m_t - m_{t-1}$ denote the additional measure of agents which are infected between periods $t-1$ and t—we refer to this as the speed of contagion at time t. At time $t = 0$, there is a measurable set $I_0 \subset \mathbb{R}$ of measure m_0 which are initially infected. At the end of period t with agents I_t infected, agent $i \in \mathbb{R}$ infected in period $t+1$ if $i \in I_t$ or at least proportion q of her neighbours are infected:

$$\int_{-\infty}^{+\infty} 1[i - x \in I_t] g(x) dx \geq q.$$

We will assume, for simplicity, that I_0 is a closed interval which implies that each I_t is also a closed interval. This follows from strict decreasing differences. (property (ii) of G) and inducting on t. [8]

[6]A similar assumption is used in a recent paper by Frick, Iijima, and Ishii (2022) to study the role of assortativity in causing misperceptions.

[7]While we will focus on the above weighted graph interpretation, there is an alternative unweighted graph interpretation of our model where there is an infinite measure of agents indexed by $(i, k) \in \mathbb{R}\times[0, 1]$. Agent (i, k) is linked to $g(|i - j|)$ of neighbours in the set $\{(j, k) : k \in [0, 1]\}$. We will assume throughout that each agent has a unit measure of neighbours. This is a normalization and does not matter for contagion dynamics because only the proportion of infected neighbours matter.

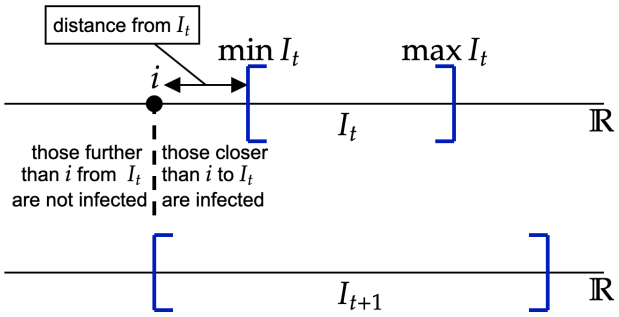
[8]I_o will, in general, not be the smallest set which can precipitate contagion. In Appendix C we derive a lower bound on the minimum size of any measurable set which can precipitate contagion. This lower bound is tight for some graphs, and is directly related to the minimal interval for contagion.

Since I_t is a closed interval for all $\mathcal{T}$, $i \in \mathbb{R}$ is infected at time $t+1$ if either $i \in I_t$ or

$$G\Big(\underbrace{|\min I_t - i| \wedge |i - \max I_t| + \mu(I_t)}_{\text{Distance from interval } I_t + \text{ length of } I_t}\Big) - G\Big(\underbrace{|\min I_t - i| \wedge |i - \max I_t|}_{\text{Distance from interval } I_t}\Big) \geq q$$

where here $|\min I_t - i| \wedge |i - \max I_t|$ is i's minimum distance from the interval I_t. When i is exactly indifferent i.e., the equation above holds with equality, the interval structure of contagion is depicted in Figure 1.

Figure 1: Illustration of intervals $(I_t)_t$



Our initial conditions are thus a tuple (G, m_0, q) comprising the graph weights, the measure of players which are initially infected,[9] and the contagion threshold.

Definition 2. We say that starting from (G, m_0, q), contagion occurs if

$$\lim_{t \to \infty} m_t(G, m_0, q) = +\infty$$

We note that since the measure of initial adopters is finite and the total measure of links each player possesses is finite, contagion never occurs when $q \geq 1/2$. This is the observation made by Morris (2000) though here we work with a continuum of agents. We now introduce our measure of resilience.

[9]Note that exact choice of I_0 does not matter since contagion in our model is invariant to translations.

Definition 3 (Measure of resilience)**.**

$$m_0(G, q) := \inf_{m_0 \in \mathbb{R}_+} m_0 \quad \text{s.t.} \quad \lim_{t \to \infty} m_t(G, m_0, q) = +\infty.$$

Remark 1. Note that we could alternatively fix the initial measure of infected agents and study the maximum threshold under which contagion can occur. [10] More formally, we could instead work with

$$q(G, m_0) := \sup_{q \in (0,1)} q \quad \text{s.t.} \quad \lim_{t \to \infty} m_t(G, m_0, q) = +\infty.$$

Fixing the network G, we see that $q(G, m_0)$ is increasing in m_0 and similarly, $m_0(G, q)$ is increasing in q—these two measures are thus similar. However, we will soon see that speed depends on the contagion threshold q. To compare the speed of spreading among different networks, we will thus fix q and vary the initial measure of infected agents. ■

3 Speed and resilience

We first develop a simple lemma which allows us to obtain tight conditions on whether contagion occurs. The essential idea is that when we restrict our attention to $G \in \mathcal{G}$, if contagion occurs, it does so through the growth of a closed interval of infected agents. In particular, the decreasing differences condition implies that given the set of agents $I_t = [a, b]$ are infected at time t, then for agents i, j such that $b \leq i \leq j$, if j is infected at time $t + 1$, i must be also infected at time $t + 1$. This is because decreasing differences implies that i, being closer to I_t, has a greater proportion of her neighbours in that interval.

Lemma 1. Fix $G \in \mathcal{G}$. Contagion occurs from (G, m_0, q) if and only if

$$G(m_0) > q.$$

Proof. Take $I_0 = [x, x + m_0]$ for some $x \in \mathbb{R}$.

[10] This in the same spirit as Morris (2000) which studied the maximum threshold under which contagion can occur from some finite set of infected agents.

($\Longrightarrow$) Let $a_1/2$ be the additional measure of infected agents at time 1 to the left of the interval I_0. a_1 solves:

$$G(a_1/2 + m_0) - G(a_1/2) = q$$

which is the condition for agent $x - a_1/2$ is exactly indifferent between adopting the action and not. But

$$G(m_0) > q = G(a_1/2 + m_0) - G(a_1/2)$$

which, together with decreasing differences, implies $a_1/2 > 0$.

Now observe that by repeating the argument and the fact that the distribution function G is increasing, $a_{t+1} \geq a_t > 0$ for any $t \in \mathcal{T}$. This is sufficient to conclude that $\lim_{t\to\infty} m_t = \lim_{t\to\infty} \sum_{s=0}^{t} a_s = +\infty$.

($\Longleftarrow$) Consider any player $i < x$ and let $\varepsilon := x - i$. If the condition does not hold,

$$q \geq G(m_0) > G(\varepsilon + m_0) - G(\varepsilon)$$

where the second inequality is from decreasing differences. This implies that any agent $i < x$ is not infected in period 1. By symmetry, a similar argument can be made for agent $i > x + m_0$. Together, this implies that $\lim_{t\to\infty} m_t \leq m_0 < +\infty$. □

Lemma 1 tells us that contagion depends on the behaviour of local links. To see this, rewrite the condition as $m_0 > G^{-1}(pq)$ which also gives an expression for the infimum of the measure of infected agents for contagion to occur:

$$m_0(G, q) = G^{-1}(q) \qquad \textbf{(Min-Seed)}$$

Notice that for small values of q, distributions with a lot of mass concentrated around the middle are more susceptible i.e., requires only a small measure of initial agents to precipitate contagion.

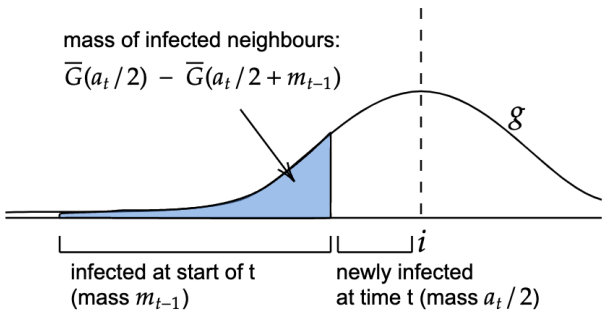
Let us now turn to the question of speed. Recall $a_t = m_t - m_{t-1}$ is the additional measure of infected agents between times t and $t-1$. a_t solves the following

nonlinear difference equation:

$$\overline{G}(a_t/2) - \overline{G}(a_t/2 + m_{t-1}) = q$$

where we recall $\overline{G}(x) = 1/2 - G(x)$ is the anti-CDF of G. This is illustrated in Figure 2 in which the condition for contagion is exactly fulfilled for i, the furthest agent to the right of the infected interval I_{t-1} to be be infected at time t.

Figure 2: Illustrating a_t



If contagion occurs, notice once again that since G is increasing, $a_{t+1} \geq a_t$. However, the additional mass of infected agents per period will be bounded above by what we shall call the limit speed of the process, defined as follows:

$$\begin{aligned} a_\infty &:= \lim_{t\to\infty} a_t \\ &= 2(\overline{G})^{-1}(q) \quad \text{since} \quad \lim_{x\to\infty} \overline{G}(x) = 0. \end{aligned} \tag{Lim-Speed}$$

At this point is is helpful to compare the expressions for resilience and limit speeds. Fixing q, we saw that what matters for resilience is given by the expression in **Min-Seed**, $= m_0(G, q) = G^{-1}(q)$ i.e., the mass of local links; conversely, what matters for the speed is the expression in **Lim-Speed**, $a_\infty(G, q) \propto (\overline{G})^{-1}(q)$ i.e., the mass of faraway links.

Example 1. Consider the case in which each agent's links are distributed according to a normal distribution with variance σ^2 i.e., $G_\sigma(x) = \Phi(x/\sigma) - 1/2$ where we use the subscript σ to track the variance. From **Min-Seed**, and **Lim-Speed**,

we have

$$m_0(G,q) = \sigma \cdot \Phi(q + 1/2) \qquad a_\infty(G,q) = 2\sigma \cdot \Phi(1-q).$$

i.e., the speed resilience tradeoff is (i) linear in σ; and (ii) holds for all contagion thresholds q. Indeed, since the variance of a normal distribution simply shrinks or stretches the distribution while preserving its shape, the contagious dynamics on graphs in this class are simply rescaled versions of each other. ■

The example above parametrized by the normal distribution is quite special. In particular, as we vary σ, each agent's mass of links are shifted toward or away by simply shrinking or stretching the distribution. While this is a sufficient way to obtain the tradeoff, we now provide a general ordering over distributions which guarantee that the tradeoff obtains for every threshold q.

Proposition 1. If $G, G' \in \mathcal{G}$ are such that $G \leq G'$, then for all $q \in (0, 1/2)$,

(i) G is more resilient than G' i.e., $m_0(G,q) \geq m_0(G',q)$; and

(ii) G has a quicker limit speed than G' i.e., $a_\infty(G,q) \geq a_\infty(G',q)$.

Conversely, if $G \not\leq G'$, then (i) and (ii) do not hold. [11]

Proof. Fix any $q \in (0, 1/2)$. $G \leq G'$ implies $G^{-1} \leq G'^{-1}$. Hence from Lemma 1,

$$\begin{aligned} m_0(G,q) &= G^{-1}(q) \\ &\leq G'^{-1}(q) = m_0(G',q). \end{aligned}$$

If contagion does not occur, $a_\infty(G) = a_\infty = 0$; if it does, notice $G \leq G'$ implies $\overline{G} \geq \overline{G}'$ and so $(\overline{G})^{-1} \geq (\overline{G}')^{-1}$. Then from our expression for limit speed,

$$\begin{aligned} a_\infty(G,q) &= 2(\overline{G})^{-1}(q) \\ &\geq 2(\overline{G'})^{-1}(q) = a_\infty(G',q). \end{aligned}$$

[11] The weak inequalities can be replaced with strict inequalities.

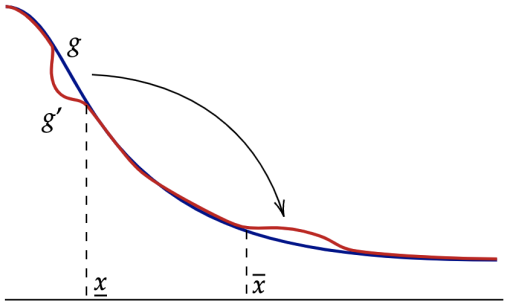
If $G \not\leq G'$, take $x \in [0, \infty)$ such that $G(x) > G'(x)$ and choose $q = G(x)$ so that

$$m_0(G, q) = G^{-1}(G(x)) > G'^{-1}(G(x)) = m_0(G', q)$$

as required. □

The underlying intuition is illustrated in Figure 3: when we move from G to G', this exactly corresponds to shifting mass from nearby links (e.g., those closer than $\underline{x}$) to faraway links (e.g., further away than $\overline{x}$). Proposition 1 states that by shifting mass 'in one direction' in an arbitrary way, this uniformly (i.e., for all contagion thresholds) makes the new distribution G' more resilient but, if contagion occurs, it does so more quickly.

Figure 3: Perturbing G to G'



We have seen that for a particular ordering, the tradeoff is especially stark. We now show that this tradeoff holds in general i.e., for any pair of G, G' though not for every contagion threshold. The insight behind this observation is that starting from a distribution $G \in \mathcal{G}$, any perturbation must alter the network's resilience and speed somewhere i.e., for some contagion threshold. The following proposition formalizes this:

Proposition 2. For $G, G' \in \mathcal{G}$, if $G \neq G'$ then there exists $q, q' \in (0, 1/2)$ such that one is more resilient than the other under q, but has a quicker limit speed than the other under q'.

Proof. Since $G \neq G'$, let us assume, without loss, that $G(x) < G'(x)$ for some $x \in [0, \infty)$. This implies that there exists $y \in (G(x), G'(x))$ such that $G^{-1}(y) >$

$x > G'^{-1}(y)$. Now set $q = y < 1/2$ and by the argument above,

$$\begin{aligned} m_0(G, q) = G^{-1}(q) &= G^{-1}(y) \\ &> G'^{-1}(y) = G'^{-1}(q) = m_0(G', q). \end{aligned}$$

Next, recalling that we defined $\overline{G} = 1/2 - G$ as the anti-cumulative distribution function of G, there exists $z \in (\overline{G}'(x), \overline{G}(x))$ such that $\overline{G'}^{-1}(z) < x < \overline{G}^{-1}(z)$ and setting $q' = z < 1/2$, we have

$$\begin{aligned} a_\infty(G, q') = \overline{G}^{-1}(q') &= \overline{G}^{-1}(z) \\ &> \overline{G'}^{-1}(z) = \overline{G'}^{-1}(q') = a_\infty(G', q') \end{aligned}$$

as required. □

Proposition 2 illustrates a fundamental tension between speed and resilience: we never have a pair of distributions such that, for all contagion thresholds q, one is both more resilient, and has slower limit speeds than the other. The logic underlying this result is specific to threshold models of contagion: since relative rather than absolute measure of infected neighbours is what matters for contagion, less local links (making the distribution more resilient) requires that mass be reallocated toward faraway links (making spread faster).

Proposition 2 developed a particular speed resilience tradeoff for arbitrary networks: when links are shifted in an arbitrary way (thus altering G), the tradeoff obtains for some contagion threshold. Conversely, Proposition 1 developed a general speed resilience tradeoff for a particular ordering over networks: when links are shifted in a 'monotone' way (i.e., toward or away from each agent), we obtain the tradeoff for every contagion threshold. Both of these results were driven by the underlying insight that what matters for resilience are local links while what matters for speed are faraway links. Of course, it is possible to have many local links whilst also having many faraway links. The next proposition shows that by shifting intermediate links both closer and further away, this can make the network less resilient while also facilitating quicker limit speeds.

Proposition 3. For $G, G' \in \mathcal{G}$, suppose that there exists some $\bar{x} \in (0, +\infty)$ such that for all $x \leq \bar{x}$, $G(x) \leq G'(x)$ and for all $x \geq \bar{x}$, $G(xz) \geq G'(x)$. Then

(i) for sufficiently low values of q, G is both more resilient than G' as well as has slower limit speeds; and

(ii) for sufficiently high values of q, G is both less resilient than G' as well as has quicker limit speeds.

Before we prove the claim, let us briefly develop some intuition. The condition in Proposition 3 amounts to a single-crossing condition between G and G': it states that if G crosses G' once from below i.e., if relative to G, G' has: (i) more mass on nearby links; (ii) more mass on faraway links; and (iii) less mass on intermediate links (which follows from (i) and (ii)) then for low contagion thresholds,G' is both more susceptible and allows for quicker limit speeds. This is because, as we previously saw, what matters for resilience are nearby links: as the contagion threshold becomes smaller, the links which matter for resilience are those which are more local. Conversely, what matters for speed are faraway links: as the contagion threshold becomes smaller, the links which matter for speed become further away. For a faraway agent to be infected, that agent must have a substantial measure of links to those which are already infected—but since the agent is faraway, this corresponds exactly to having sufficient mass on the tails of the distribution of her links. Finally, we note that unlike Proposition 1, this 'single crossing' condition on G and G' is not tight—as long as G' is more local links and fatter tails, this is sufficient for G' to be less resilient and have quicker speeds than G for sufficiently small q.

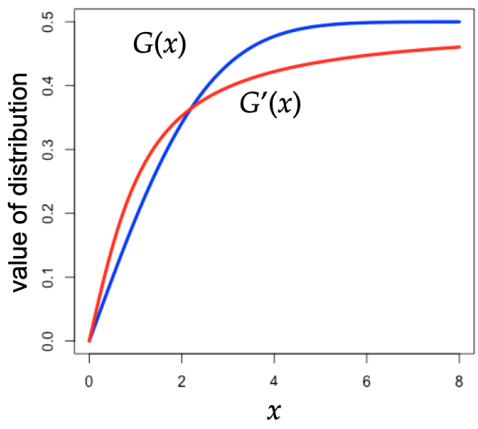
Example 2. Let the distribution of links under G be that of the normal distribution with standard deviation $\sigma = 2$ and G' be that under (standard) Cauchy. [12]

$$G(x) = \Phi(x/2) - 1/2 \qquad G'(x) = (1/\pi)\arctan(x).$$

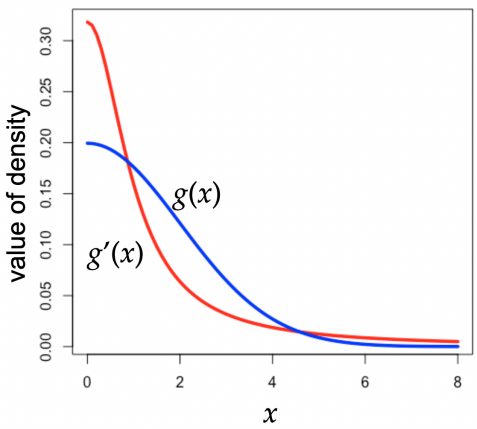
The density of links and distribution functions are illustrated in Figure 4.

[12] i.e., with $\gamma = 1$.

Figure 4: Normal vs Cauchy: distribution and densities



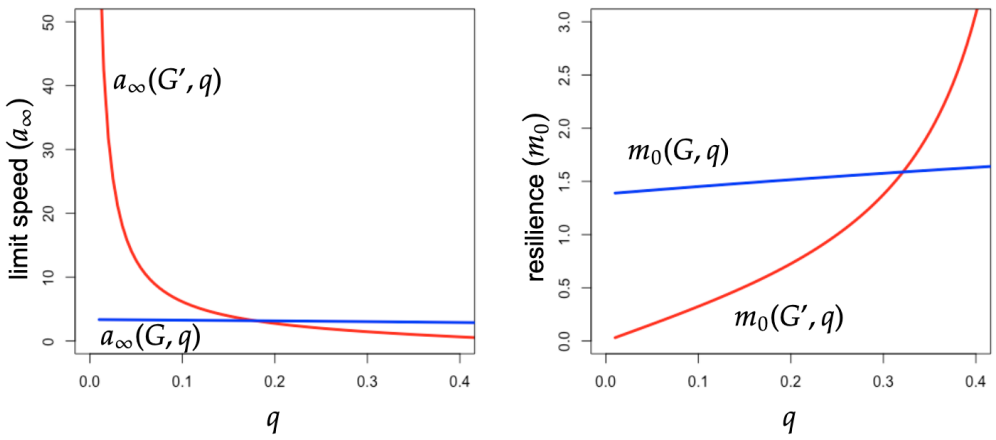
(a) G vs G'



(b) g vs g'

From direct computation, with the expressions **Lim-Speed**, we have that $a_\infty(G', q) = 2 \cdot \tan((1/2 - q) \cdot \pi)$ which implies that $a_\infty(G', q) \simeq 1/q^2$ since the tails of the Cauchy distribution decay polynomially. On the other hand, since the normal distribution has (sub-)Gaussian tails, $a_\infty(G, q) \lesssim (\log(1/q))^{1/2}$. Limit speeds and resilience for a range of contagion thresholds are illustrated in Figure 5.

Figure 5: Normal vs Cauchy: speed and resilience



(a) Comparison of speed

(b) Comparison of resilience

Here we see that for relatively small contagion thresholds ($q \leq 0.15$), G' has quicker limit speeds (panel (a)) but also lower resilience (panel (b)). As the contagion threshold q becomes small, we see that the limit speed under G' quickly outstrips that under G. ■

Proof of Proposition 3. Choose $q' = G(\overline{x})$ and note that by the condition of single crossing at $\overline{x}$ in the proposition, for any $q \leq q'$,

$$\begin{aligned} m_0(G, q) &= G^{-1}(q) \\ &\geq G'^{-1}(q) = m_0(G', q). \end{aligned}$$

with the reverse equality for $q \geq q'$. Similarly, choose $q'' = \overline{G}(\overline{x})$ and for $q \leq q''$ by the condition in the proposition,

$$\begin{aligned} a_\infty(G, q) &= 2\overline{G}^{-1}(q) \\ &\leq 2\overline{G'}^{-1}(q) = a_\infty(G', q) \end{aligned}$$

with the reverse equality for $q \geq q''$. Part (i) follows for thresholds $q \leq q' \wedge q''$;

part (ii) follows for thresholds $q \geq q' \vee q''$. □

4 Concluding remarks

We have highlighted a fundamental tension between the resilience of a networked population to contagion, and the speed at which, conditional on contagion occurring, behaviours propagate. We showed this trade-off in a canonical one-dimensional setting and derived various orders over networks for which the trade-off is particularly stark. Our analysis also highlighted the role intermediate links play as a bulwark against contagion–without them , networks can simultaneously have many local links (reducing resilience) as well as many faraway links (increasing limit speed).

We conclude with two remarks. First, while we studied deterministic contagion dynamics with an infinite measure of agents, our results also apply to settings with a unit measure of agents, or those with discrete random graphs. In Appendix A, we develop this model for the unit circle and show that the essential tradeoffs still obtain. This setting generates a particular class of continuous graphs—graphons—which we show approximates the step-by-step contagion dynamics of random graphs sampled from the graphon (where each agent's links are distributed according to the corresponding distribution G). This problem is studied by an important paper of Erol et al. (2020) who show that the final set of infected agents (i.e., as $t \to \infty$) on the graphon can approximate that of the sampled graph arbitrarily well. Since we are interested in speed, we are interested in the evolution of infected agents over time (i.e., the set of infected agents for each t); to this end we use standard techniques to complete the approximation for each time period. Second, while we worked with networks embedded in a single-dimension, the forces we have highlighted carry over into higher dimensions. In Appendix B, we develop our model for networks embedded in $\mathbb{R}^n$ and show that versions of our results continue to obtain there.

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Appendix to 'Speed vs Resilience in Contagion'

A Unit measure of agents & relation to discrete random graphs

Outline of Appendix A. In Appendix A.1, we outline class of graphs which are compatible with a unit measure of agents arranged on the $[0, 1]$ circle; such graphs can be represented by graphons (Borgs et al., 2008). We then develop a mapping parameterized by a shrinkage factor s which sends graphs we studied in the main text (with potentially infinite support) to graphs which are compatible with the unit circle. In Appendix A.2, we show that this mapping fulfills basic desiderata: in the limit of the map (when $s \downarrow 0$), the values of resilience and limit speeds coincide exactly. In Appendix A.3, we draw on our map and Propositions 1-3 in the main text to show that the tradeoff between resilience and average speed obtains for graphs on the unit circle. [13]In Appendix A.4, we link the contagion dynamics over graphons to that over random graphs sampled from the graphon (Lovász, 2012, Chapter 10). To do so, we extend the results of Erol, Parise, and Teytelboym (2020) to show that contagion on graphons can approximate contagion on the sampled graph at each step of the contagion process arbitrarily well. Collectively, Appendices A.1-A.4 allow us to conclude that the forces we identify in the main text apply more broadly to standard models of random graphs.

A.1 Model

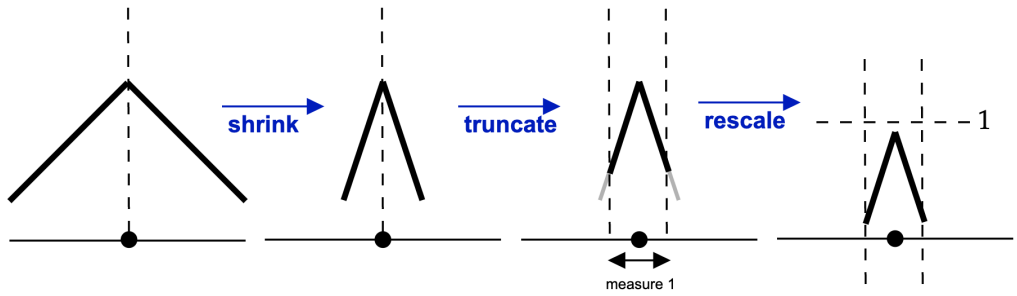
There is now a unit measure of agents indexed $i \in [0, 1]$ arranged on a circle. We will continue to be interested in the class of CDFs $\mathcal{G}$. However, note that since this class allows for distributions which assign positive measure to agents further than distance $1/2$ away, not every distribution in $\mathcal{G}$ will be compatible with the circle model. Instead, the set of CDFs within $\mathcal{G}$ which are compatible

[13]For fixed s, the notion of limit speed is no longer meaningful since conditional on contagion obtaining, the whole population is infected in finite time.

with the circle model is given by those which have zero measure of links to agents beyond distance $1/2$ away which we denote by $\mathcal{G}^T := \{G \in \mathcal{G} : G(1/2) = 1/2\}$. Further note that every distribution $G \in \mathcal{G}^T$ with density $g \leq 1$ corresponds to a graphon (Lovász, 2012); we will make use of this fact at the end of this appendix to show how the results on contagion dynamics developed for a unit measure of agents applies to large but discrete random graphs.

Our goal for this section will be to define a surjective map $G \mapsto G_s^{T,R}$ parametrized by a shrinkage factor $s \in [0, 1]$. The map will proceed in several steps. First, we shrink the graph G; next, we truncate it so that agent i only assigns positive weight to the interval $[i - 1/2, i + 1/2]$; finally, we rescale the density so that i's density over $[i - 1/2, i + 1/2]$ is below 1. The last step ensures that the end product is a graphon. These steps are depicted in Figure 6.

Figure 6: Illustration of $G \mapsto G_s^{T,R}$



For $G \in \mathcal{G}$ and shrinkage factor $s > 0$, define G_s as the rescaled CDF such that

$$G_s(sx) = G(x) \quad \text{for all } x > 0.$$

Further, given $G \in \mathcal{G}$ and $s > 0$, define $G_s^T : [0, 1/2] \to \mathbb{R}_{\geq 0}$ such that for all $x \in [0, 1/2]$,

$$G_s^T(x) = \left(\frac{G_s(x)}{G_s(1/2)} \right) \cdot \frac{1}{2}$$

i.e., we truncate the distribution such that the domain is now $[0, 1/2]$, and rescale masses such that $G_s^T(1/2) = 1/2$, the mass of the original distribution G_σ. Observe that $G \in \mathcal{G}$ implies $G_s, G_s^T \in \mathcal{G}$ for any $s \in (0, 1]$ since rescaling and normalization preserves strict decreasing differences.

Of course, with a unit measure of agents we also need to rescale each agent's measure of links appropriately. To this end, for any $G \in \mathcal{G}$, define

$$G^R(x) = G(x)/R$$

i.e., the graph rescaled by R. We will often apply this rescaling to G_s^T,and denote the result with $G_s^{T,R}$ where the superscript 'T' stands for truncated, and 'R' stands for rescaled. We require that

$$R \geq \frac{dG_s^T}{dx}\Big|_{x=0} = g(0/\sigma)\frac{1/2s}{G(1/2s)} =: R_G(s).$$

This is sufficient to imply

$$g_s^{T,R}(x) \leq g_s^{T,R}(0) \leq \frac{dG_s^T}{dx}\Big|_{x=0} \Big/ R \leq 1$$

for all $x \in [0, 1/2]$, ensuring that the output graph can be represented by a graphon (see Appendix A.4). We will write $G_s^{T,R}$ with the implicit understanding that this is for any value of $R \geq R_G(s)$; the exact value of R is immaterial to contagion dynamics.

A.2 Contagion dynamics on the real line converges to dynamics on the unit circle

We first establish that the contagion dynamics of the graph $G_s^{T,R}$ on the unit circle does, in fact, converge to that of the graph G on the real line.

Proposition 4. Contagion dynamics on $G_s^{T,R}$ over the unit circle converges to the contagion dynamics on G over the real line as $s \downarrow 0$, i.e.,

(i) (Convergence in resilience) $\lim_{s\downarrow 0}\left(\frac{G_s^{T,R}, q)}{s}\right) = m_0(G, q)$; and

(ii) (Convergence in speed) $\lim_{s\downarrow 0} \frac{a_t(G_s^{T,R}, q, sm_0)}{s} = a_t(G, q, m_0)$ for all $t \in \mathcal{T}$.

Proof. For part (i), note that $G_s^{T,R}$ and its inverse is continuous in the supnorm with respect to σ which then implies

$$\begin{aligned}
\lim_{s\downarrow 0} m_0(G_s^{T,R}, q) &= \lim_{s\downarrow 0} (G_s^{T,R})^{-1}(q/R) && \text{(Apply Min-Seed)} \\
&= \lim_{s\downarrow 0} (G_s^{R})^{-1}(q/R) && (\lim_{s\downarrow 0} \|G_s^{T,R} - G_s^R\|_\infty = 0) \\
&= \lim_{s\downarrow 0} (G_s)^{-1}(q) && \text{(Def. of } G^R) \\
&= \lim_{s\downarrow 0} sG^{-1}(q) && \text{(Def. of } G_s) \\
&= \lim_{s\downarrow 0} sm_0(G, q) && \text{(Apply Min-Seed)}
\end{aligned}$$

as required.

For part (ii), we know from part (i) that in the limit, contagion either occurs, or does not occur on both $G_s^{T,R}$ and G. If it does not occur, both sides are zero. If it does, redefine $a_t := a_t(G_s^{T,R}, q, m_0)$ as the speed at time t on the modified graph, and $b_t := a_t(G, q, m_0)$ as the speed at time t on the original graph, and $\tau(G_s^T, q, m_0) := \inf\{t \in \mathcal{T} : m_t(G_s^T, m_0, q) \geq 1\}$ as the first time at which the whole population is infected.

For each $t < \tau(G_s^T, q, m_0)$, a_t is characterized by the non-linear difference equation

$$\overline{G}_s^{T,R}(a_t/2) - \overline{G}_s^{T,R}(a_t/2 + m_t) = q/R.$$

Similarly, b_t is characterized by

$$\overline{G}(b_t/2) - \overline{G}(b_t/2 + m_t) = q.$$

Now combining both equations and taking limits,

$$\begin{aligned}
\overline{G}(b_t/2) - \overline{G}(b_t/2 + m_t) &= \lim_{s\downarrow 0} R \cdot \overline{G}_s^{T,R}(a_t/2) - \lim_{s\downarrow 0} R \cdot \overline{G}_s^{T,R}(a_t/2 + m_t) \\
&= \lim_{s\downarrow 0} \overline{G}_s^{T}(a_t/2) - \lim_{s\downarrow 0} \overline{G}_s^{T}(a_t/2 + m_t) \\
&= \lim_{s\downarrow 0} \overline{G}_s(a_t/2) - \lim_{s\downarrow 0} \overline{G}_s(a_t/2 + m_t) \\
&= \lim_{s\downarrow 0} \overline{G}\left(\frac{a_t/2}{s}\right) - \lim_{s\downarrow 0} \overline{G}\left(\frac{a_t/2 + m_t}{s}\right) \\
&= \overline{G}\left(\lim_{s\downarrow 0} \frac{a_t/2}{s}\right) - \overline{G}\left(\lim_{s\downarrow 0} \frac{a_t/2 + m_t}{s}\right).
\end{aligned}$$

where the second equality is from the definition of G^R,the third equality follows from noting that $\lim_{s\downarrow 0}\|G_s^T - G_s\|_\infty = 0$, and fourth inequality is from the definition of G_s, and the last inequality is from the continuity of G.

We now proceed by induction. For $t = 1$, we can rewrite the above equation to get

$$\overline{G}(b_t/2) - \overline{G}(b_1/2 + m_0) = \overline{G}\left(\lim_{s\downarrow 0} \frac{a_t/2}{s}\right) - \overline{G}\left(\lim_{s\downarrow 0} \frac{a_t/2}{s} + m_0\right)$$

and since $\overline{G}$ is strictly decreasing, we match terms and have $\lim_{s\downarrow 0}(a_t/s) = b_t$. Now for arbitrary $1 < t < \tau(G_s^T, q, m_0)$, supposing that for each $s \leq t-1$, $\lim_{s\downarrow 0}(a_s/s) = b_s$, then notice that $m_{t-1} = m_0 + \sum_{s=1}^{t-1} a_s$, we similarly have

$$\overline{G}(b_t/2) - \overline{G}(b_1/2 + m_{t-1}) = \overline{G}\left(\lim_{s\downarrow 0} \frac{a_t/2}{s}\right) - \overline{G}\left(\lim_{s\downarrow 0} \frac{a_t/2}{s} + m_{t-1}\right)$$

which completes the inductive step. Finally, our argument was for $t < \tau(G_s^T, q, m_0)$ but noting that $\lim_{s\downarrow 0} \tau(G_s^T, q, m_0) = +\infty$, this extends to all $t \in \mathcal{T}$. □

Proposition 4 establishes that the model we worked with in the main text can be viewed as the limit of the circle model with a unit measure of agents as we we rescale each agents' links such that they become increasingly local. Part (i) states that as $s \downarrow 0$, the requisite measure of initial infected agents in the circle model

converges to that of the real line. Part (ii) states that as $s \downarrow 0$, at each time step, the set of infected agents on the circle model under G_s^T converges to that under G. A simple corollary of part (ii) is also that whenever contagion occurs,

$$\lim_{t\to+\infty} \lim_{s\downarrow 0} \frac{a_t(G_s^{T,R}, q, sm_0)}{s} = a_\infty(G, q, sm_0).$$

A.3 Fixed $s > 0$

We now develop our analysis for a fixed shrinkage factor $s > 0$. In such settings there is no meaningful notion of limit speed since, if contagion occurs, there is some finite time after which the whole population is infected. Nonetheless, a version of the speed-resilience tradeoff continues to hold, but with a different measure of speed which we now define.

Define $\tau(G_s^{T,R}, m_0, q) := \inf\{t \in \mathcal{T} : m_t(G_s^T, m_0, q) \geq 1\}$ as the first time at which the whole population is infected and define

$$a_{AVG}(G_s^{T,R}, m_0, q) := \frac{1}{\tau(G_s^{T,R}, m_0, q)} \left(\sum_{t=1}^{\tau(G_s^{T,R}, m_0, q)} a_t(G_s^{T,R}, m_0, q) \right)$$

as the time average of the contagion process. When there is no ambiguity, we drop the arguments from a_{AVG} and τ. It is easy to see that $a_{AVG} \approx \tau^{-1}$ for small s since m_0 and a_t are both $O(s)$.

The following proposition develops approximations for a_{AVG} for small but fixed values of s. The underlying intuition is straightforward: as we take s small, the system is in effect getting larger because at each time step the additional measure of infected agents is order s. This also extends τ, the time taken for the whole population to be infected. But as τ gets large, the network spends a large fraction of the time close to the limit speed, and so the average speed is also well-approximated by the limit speed, and the time taken for the whole population to be infected is similarly well-approximated by the reciprocal of limit speed.

Proposition 5. If contagion occurs from (G, m_0, q) on $\mathbb{R}$, then for any $\varepsilon > 0$ there exists $\tilde{s} > 0$ such that for any $s \leq \tilde{s}$ contagion on the unit circle exhibits the following properties:

(i) $\dfrac{m_0(G_s^{T,R},q)}{s} \in \left[m_0(G,q)-\varepsilon, m_0(G,q)+\varepsilon\right]$; and

(ii) $\dfrac{a_{AVG}}{s} \in \left[a_\infty-\varepsilon, a_\infty+\varepsilon\right]$.

Proof. Part (i) is immediate from Proposition 4 part (i). For part (ii), we know that from part (ii) of Proposition 4, for each $t \in \mathcal{T}$,

$$\lim_{s\downarrow 0} \frac{a_t(G_s^{T,R},q,sm_0)}{s} = a_t(G,q,m_0)$$

for each t. Now for each $t < \tau(G_s^{T,R},q,m_0)$ and any $\eta > 0$, there then exists $\bar{s}_t$ such that for all $s \leq \bar{s}_t$, $|a_t(G_s^{T,R},q,sm_0)/s - a_t(G,q,m_0)| \leq \eta/2$. Take $\bar{s} = \min_{t\leq\tau} \bar{s}_t > 0$. Now consider the contagion dynamics of G on the real line and observe that $\lim_{t\to\infty} a_t(G,q,m_0) = a_\infty$ and that this sequence is increasing. Define $s := \inf\{t \in \mathcal{T} : a_t(G,m_0,q) \geq a_\infty(G,m_0,q) - \eta/2\}$ as the first time the speed of contagion on the real line under G exceeds the limit speed less $\eta/2$, noting that this is independent of s. By the triangle inequality, for $s < \bar{s}$ and $t \geq s$, $|a_t(G_s^{T,R},q,m_0)/s - a_\infty(G,m_0,q)| \leq \eta$.

Now for a given $\eta > 0$, we have

$$\begin{aligned}
\frac{a_{AVG}(G_s^T,q,m_0)}{s} &:= \frac{1}{s\tau(G_s^{T,R},m_0,q)}\left(\sum_{t=1}^{\tau(G_s^{T,R},m_0,q)} a_t(G_s^{T,R},m_0,q)\right) \\
&= \frac{1}{s\tau(G_s^{T,R},m_0,q)}\left(\sum_{t=1}^{s} a_t(G_s^{T},m_0,q) + \sum_{t=s+1}^{\tau(G_s^{T,R},m_0,q)} a_t(G_s^{T,R},m_0,q)\right) \\
&= \frac{m_s(G_s^{T,R},q,m_0) - sm_0}{s\tau(G_s^{T,R},m_0,q)} + \frac{\tau(G_s^{T,R},m_0,q) - s}{\tau(G_s^{T,R},m_0,q)} \cdot a_\infty + O(\eta) \\
&= a_\infty - \frac{s}{\tau(G_s^{T,R},m_0,q)} + \frac{m_s(G_s^{T,R},q,m_0) - sm_0}{s\tau(G_s^{T,R},m_0,q)} + O(\eta) \\
&= a_\infty + O(\tau^{-1}) + O(\eta)
\end{aligned}$$

where the $O(\tau^{-1})$ term in the last inequality is because $m_s(G_s^{T,R},q,m_0) = sm_0 +$

$\sum_{t=1}^{s} a_t(G_s^{T,R}, q, m_0) = O(s)$. Now for any $\varepsilon > 0$, we see that we can choose $\bar{s}$ such that for all $s \leq \tilde{s}$,

$$\left| \frac{a_{AVG}(G_s^{T,R}, q, m_0)}{s} - a_\infty \right| \leq \varepsilon$$

since $\lim_{s \downarrow 0} \tau = +\infty$ and $\eta > 0$ was arbitrary. □

Interpreting the results for fixed $s > 0$ over the unit circle. Let us briefly interpret our results through a simple example.

Example 3. Proposition 5 tells us the orderings developed in the previous section applies to the unit circle for small but fixed values of s. In particular, suppose $G, G' \in \mathcal{G}$ are such that $G(x) < G'(x)$ for all $x > 0$ i.e., G strictly first-order stochastically dominates G'. Proposition 1 in the main text states that G is more resilient ($m_0(G, q) > m_0(G', q)$) but, if contagion occurs, facilitates quicker limit spreading ($a_\infty(G, q) > a_\infty(G', q)$). Proposition 5 tells us that this tradeoff is preserved for $G_s^{T,R}$ and $(G')_s^{T,R}$ on the unit circle for sufficiently small values of s: from Part (i), we have that

$$\begin{aligned} \lim_{s \downarrow 0} m_0(G_s^{T,R}, q)/s = m_0(G, q) &> m_0(G', q) \\ &= \lim_{s \downarrow 0} m_0((G')_s^{T,R}, q)/s \end{aligned}$$

and so for sufficiently small s, $m_0(G_s^{T,R}, q) > m_0((G')_s^{T,R}, q)$. If contagion occurs on both networks, then Part (ii) tells us that for sufficiently small s,

$$a_{AVG}(G_s^{T,R}, m_0, q) > a_{AVG}((G')_s^{T,R}, m_0, q) \quad \text{and} \quad \tau(G_s^{T,R}, m_0, q) < \tau((G')_s^{T,R}, m_0, q)$$

so we continue to obtain a version of Proposition 1, but with average speed instead of limit speed. We can do the same for Propositions 2 and 3. ■

A.4 Approximating discrete random graphs

We now build on the results developed above to approximate the contagion dynamics over large but discrete random graphs.

Definition 4 (Graphon)**.** A graphon is a measurable function $W : [0,1]^2 \to [0,1]$

Notice that for any $G \in \mathcal{G}$ and fixed $s > 0$, $G_s^{T,R}$ corresponds to a (symmetric) graphon in which $W(i, i+x) = W(i, i-x) = g_s^{T,R}(x)$ for $x \in [0,1]$. Let W_G denote the graphon associated with the graph $G \in \mathcal{G}^T$.

Definition 5 (Sampling from graphons)**.** Take a graphon W and an a uniform sample of n nodes from the set $[0,1]$, letting $S = \{i_1, i_2, \dots i_n\}$ be the random collection of sampled nodes. We then connect the nodes i_m and $i_{m'}$ with independent probability $W(i_m, i_{m'})$. We call the result random graph $G(n, W)$ where n is the number of sampled points, and W is the corresponding graphon it is sampled from.

The reader is referred to Lovász (2012, Chapters 8-10) for more detail on the existence, construction, and properties of graphons and sampled random graphs.

Definition 6 (Contagion on sampled graphs)**.** Given the sampled graph $G(n, W)$ and a set of initially infected agents on the sampled graph $I_0^{(n)} \subset S = \{i_1, i_2, \dots i_n\}$, define the sets of infected agents at times $t \in \mathcal{T}$ as $(I_t^{(n)})_{t \in \mathcal{T}}$. Agent x_i is infected at time $t+1$ if either $i \in I_t^{(n)}$ (i.e., i was infected at time t) or $d(i, I_t^{(n)})/d(i) \geq q$ where we use $d(i, I)$ to denote the degree of i among nodes in the set $I \subseteq S$, and $d(i)$:= $d(i, S)$.

We now show that contagious dynamics on the random graph $G(n, W_{G_s^{T,R}})$ is approximated by that on $G_s^{T,R}$ for large n. We start by developing some additional notation to move between the graphon and the sampled graph. Define

$$V^{(n)}(I) = \{i : i \in I \cap S\}$$

as the set of sampled agents who are within the (measurable) set $I \subseteq [0,1]$.

We will initialize $I_0^{(n)} = V^{(n)}(I_0) := \{i : i \in I_0 \cap S\}$ as the set of initially infected agents in the sampled graph. These are agents who (i) are infected in the graphon; and (ii) are drawn in the sample. Since we are interested in speed, our goal will, roughly, be to show that $V^{(n)}(I_t) = I_t^{(n)}$ with high probability i.e., given a set of agents I_t which are infected on the underlying graphon at time t, the set of sampled agents $S \cap I_t$ within this set approximates the set of agents which are infected on the sampled graph $G(n, W)$ at time t.

Proposition 6 (Propositions 1 & 2 of Erol et al. 2020)**.** For any $G \in \mathcal{G}^T$, contagion on the graphon W_G and contagion on the sampled graph $G(n, W_G)$ are such that for all $t \in \mathcal{T}$ and $\epsilon_t, \kappa_t > 0$, there exists N^κ such that for all $n > N^\kappa$, with probability $\geq 1 - \kappa_t$,[14]

$$V^{(n)}(I_t) \subseteq I_t^{(n)}.$$

The Proposition of Erol et al. 2020 gives us one direction of inclusion which is sufficient for them to show that the final set of infected agents on the sampled graph $I_\infty^{(n)}$ is well-approximated by the final set of infected agents on the graphon $V^{(n)}(I_\infty)$.

However, since we are interested in the speed of the process, this states that the speed of contagion under the graphon is weakly lower than that under the sampled graph. However,

We additionally wish to show that for each time period t, the set of infected agents on the graphon approximates that of the sampled graph well i.e., step-by-step approximation. To this end, we will draw on standard probabilistic techniques to show that the opposite inclusion holds with arbitrarily high probability for sufficiently large samples.

Proposition 7. For any $G \in \mathcal{G}^T$, contagion on the graphon W_G and contagion on the sampled graph $G(n, W_G)$ are such that for all $t \in \mathcal{T}$ and $\epsilon_t, \kappa_t > 0$, there exists $N^{\epsilon,\kappa}$ such that for all $n > N^{\epsilon,\kappa}$, with probability $\geq 1 - \kappa_t$

$$I_t^{(n)} \subseteq V^{(n)}(I_t^{\epsilon_t})$$

for some measurable set $I_t^{\epsilon_t} \subseteq I_t$ which ϵ_t-approximates I_t (i.e., $|\mu(I_t^{\epsilon_t}) - \mu(I_t)| < \epsilon_t$).

Proof. We would like to establish that the inclusion holds with probability $\geq 1 - \kappa_t$, and that the set $I_t^{\epsilon_t}$ is ϵ_t measure away from the actual set of infected agents I_t. For the latter goal, although we are only interested in ϵ_t, we will

[14]Note that because we defined threshold contagion as i being infected if greater than or equal to q proportion of her neighbours are infected, we do not need to remove an ϵ-measure set of agents for the approximation; see the discussion following Propositions 1 and 2, and Appendix A.3 of Erol, Parise, and Teytelboym (2020) for details.

construct an accompanying increasing sequence $(\epsilon_s)_{s\leq t}$ where we choose $I_t^{\epsilon_t} = [\min I_t - \frac{\epsilon_t}{2}, \max I_t + \frac{\epsilon_t}{2}]$ for each t i.e., I_t augmented with an additional buffer of length $\epsilon_t/2$ on either side. The idea will be that at each time step t, there might potentially be some agents who are infected on the sampled graph, but not on the graphon; but such agents–if they exist–will accumulate close (within $\epsilon_t/2$ distance) to the infected set on the graphon. By controlling the sequence $(\epsilon_s)_{s\leq t}$ appropriately, we are able to control this accumulation so that they always fall within the approximating set $I_{t+1}^{\epsilon_{t+1}}$.

We proceed by inducting on t for a given sequence $(\epsilon_s)_s$ (which we will choose), and then conclude by showing that we can always choose this sequence to make ϵ_t the desired size. For $t = 0$, there is nothing to prove since by construction $I_0^{(n)} = V^{(n)}(I_0) \subseteq V^{(n)}(I_0^{\epsilon_0})$ for any $\epsilon_0 > 0$. We now show that if the claim is true for time t given ϵ_t, it is also true for time $t+1$ for an ϵ_{t+1}.

For notational simplicity, denote the event $E_t := \left\{ I_t^{(n)} \subseteq V^{(n)}(I_t^{\epsilon_t}) \right\}$ where we suppress dependence on ϵ_t. By the law of total probability,

$$\mathbb{P}(E_{t+1}) = \mathbb{P}(E_{t+1} \mid E_t) \cdot \mathbb{P}(E_t) + \mathbb{P}(E_{t+1} \mid E_t^c) \cdot \mathbb{P}(E_t^c).$$

By induction $\mathbb{P}(E_t) \geq 1 - \kappa_t$ and the second term $\mathbb{P}(E_{t+1} \mid E_t^c) \cdot \mathbb{P}(E_t^c)$ is weakly positive so the main step will be to obtain a lower bound on $\mathbb{P}(E_{t+1} \mid E_t)$. To this end, note

$$\begin{aligned}
\mathbb{P}(E_{t+1}|E_t) =& 1 - \mathbb{P}\left(\bigcup_{\substack{i\in S: \\ i\notin V^{(n)}(I_{t+1}^{\epsilon_{t+1}})}} i \in I_{t+1}^{(n)} \right) \\
\geq& 1 - \sum_{\substack{i\in S: \\ i\notin V^{(n)}(I_{t+1}^{\epsilon_{t+1}})}} \mathbb{P}(i \in I_{t+1}^{(n)}|E_t)
\end{aligned}$$

where the inequality is from the union bound. Now observe that for $i \notin V^{(n)}(I_{t+1}^{\epsilon_{t+1}})$,

i is infected on the sampled graph with conditional probability

$$\begin{aligned}\mathbb{P}(i \in I_{t+1}^{(n)} | E_t) =& \mathbb{P}\left(\sum_{j \in I_t^{(n)}} A_{ij}^{(n)} \geq q \cdot \sum_{j \in S} A_{ij}^{(n)} \mid E_t \right) \\ \leq& \mathbb{P}\left(\sum_{j \in V^{(n)}(I_t^{\epsilon_t})} A_{ij}^{(n)} \geq q \cdot \sum_{j \in S} A_{ij}^{(n)} \right) \\ =& \mathbb{P}\left(\sum_{j \in S} 1(j \in V^{(n)}(I_t^{\epsilon_t})) \cdot A_{ij}^{(n)} \geq q \cdot \sum_{j \in S} A_{ij}^{(n)} \right)\end{aligned}$$

where we define $A_{ij} := 1(\{i \text{ and } j \text{ are connected on the sampled graph}\})$ and the inequality came from the fact that we conditioned on the event $E_t = \left\{ I_t^{(n)} \subseteq V^{(n)}(I_t^{\epsilon_t}) \right\}$ and replaced $I_t^{(n)}$ with $V^{(n)}(I_t^{\epsilon_t})$ in the sum. Now define the random variable

$$X_{ij} := 1\left(j \in V^{(n)}(I_t^{\epsilon_t})\right) \cdot A_{ij}^{(n)} - q A_{ij}^{(n)}$$

noting that X_{ij} and $X_{ij'}$ are independent for $j \neq j'$ (by construction of $G(n, W)$).

Define the operator $B : \mathcal{B}(\mathbb{R}) \to \mathcal{B}(\mathbb{R})$ which maps a set of infected agents at time t to a new set of infected agents at time $t+1$ (e.g., in our notation so far, $B(I_t) = I_{t+1}$). A first observation is that for $i \notin B(I_t^{\epsilon_t})$,

$$\begin{aligned}\mathbb{E}[X_{ij}] =& \int_{j \in I_t^{\epsilon_t}} W(i,j) dj - q \cdot \int_{j \in [0,1]} W(i,j) dj \\ <& 0\end{aligned}$$

where the inequality follows from the fact that $i \notin B(I_t^{\epsilon_t})$ on the graphon. Note that j was uniformly sampled from $[0,1]$, we are implicitly integrating against the uniform density.

But we need to do slightly better than this to obtain a uniform bound on i: what we will do is to choose $I_{t+1}^{\epsilon_{t+1}} \supset B(I_t^{\epsilon_t})$ slightly larger by setting

$$\epsilon_{t+1} = \mu(B(I_t^{\epsilon_t})) - \mu(B(I_t)) + \epsilon_t$$

and since we assumed $i \notin V^{(n)}(I_{t+1}^{\epsilon_{t+1}})$, i is by construction bounded away from

the set $B(I_t^{\epsilon_t})$ by ϵ_t, this tightens our bound further:

$$\begin{aligned}\mathbb{E}[X_{ij}] &= \int_{j\in I_t^{\epsilon_t}} W(i,j)dj - q\cdot\int_{j\in[0,1]} W(i,j)dj \\ &< -f(\epsilon_t) \quad \text{where } f(\epsilon_t) > 0\end{aligned}$$

where here

$$f(\epsilon_t) = q - [G(\mu(B(I_t^{\epsilon_t})) + \epsilon_t/2) - G(\epsilon_t/2)] > 0$$

from strict decreasing differences and the fact that $G(\mu(B(I_t^{\epsilon_t})) = q$ (from the definition of the operator B). Now recall that $|S| = n$ and pick $\delta \in (0, f(\epsilon_t))$ (independent of i). Then note

$$\begin{aligned}\mathbb{P}(i \in I_{t+1}^{(n)}|E_t) \leq& \mathbb{P}\left(\frac{1}{n}\sum_{j\in S} X_{ij} \geq 0\right) \\ \leq& \mathbb{P}\left(\frac{1}{n}\sum_{j\in S} X_{ij} \geq \underbrace{\delta + \mathbb{E}[X_{ij}]}_{<0}\right) \\ \leq& \mathbb{P}\left(\frac{1}{n}\sum_{j\in S} X_{ij} - \mathbb{E}[X_{ij}] \geq \delta\right) \\ \leq& \mathbb{P}\left(\left|\sum_{j\in S} X_{ij} - n\cdot\mathbb{E}[X_{ij}]\right| \geq n\cdot\delta\right) \\ \leq& \exp(-2n\delta^2)\end{aligned}$$

where the last inequality is from McDiarmid/Hoeffding since that differences are bounded by 1. Now note that since our choice of δ did not depend on i, this bound applies to every $i \in S \setminus V^{(n)}(I_{t+1}^{\epsilon_{t+1}})$. Then from before,

$$\begin{aligned}\mathbb{P}(E_{t+1}|E_t) \geq& 1 - \sum_{\substack{i\in S: \\ i\notin V^{(n)}(I_{t+1}^{\epsilon_{t+1}})}} \mathbb{P}(i \in I_{t+1}^{(n)}|E_t) \\ \geq& 1 - n\cdot\exp(-2n\delta^2) =: \eta(n)\end{aligned}$$

which can be made arbitrarily small since the exponential term dominates; the

intuition is that as n grows, there are more nodes which could be infected on the sampled graph although they are not infected on the graphon. However, as n grows, the probability of this event is also decaying exponentially quickly. Now for any $\kappa_{t+1} > 0$,

$$\begin{aligned}\mathbb{P}(E_{t+1}) =& \mathbb{P}(E_{t+1} \mid E_t)\mathbb{P}(E_t) + \mathbb{P}(E_{t+1} \mid E_t^c)\mathbb{P}(E_t^c) \\ \geq& (1 - \eta(n)) \cdot (1 - \kappa_t(n))\end{aligned}$$

and choose n sufficiently large so that $(1 - \delta(n)) \cdot (1 - \kappa_t(n)) \geq 1 - \kappa_{t+1}$.

Finally, our induction step was for a fixed ϵ_t and choosing $\epsilon_{t+1} = \mu(B(I_t^{\epsilon_t})) - \mu(B(I_t)) + \epsilon_t$. Now notice that for any closed interval I $\mu(B(I_t^{\epsilon}))$ is continuous in ϵ for $\epsilon \geq 0.$, and $B(I_t^{\epsilon=0}) = B(I_t)$ so $\lim_{\epsilon \downarrow 0} |\mu(B(I^{\epsilon})) - \mu(B(I))| = 0$. Hence for any $t \in \mathcal{T}$, $\epsilon_t, \kappa_t > 0$, we can construct the sequence $(\epsilon_s)_{s \leq t}$ where we set

$$\epsilon_s = \mu(B(I_{s-1}^{\epsilon_t})) - \mu(B(I_{s-1})) + \epsilon_{s-1}$$

which can be done for sufficiently small ϵ_0 so that our induction argument to obtain the event with probability $\geq 1 - \kappa_t$ does indeed apply. □

Let us take stock. We showed in Appendix A.1-A.4 earlier that Propositions 1,2, and 3 in the main text on the tradeoffs (or lack thereof) between speed and resilience among (infinite) graphs $\mathcal{G}$ is approximated by that among rescaled graphs with a unit measure of agents. Proposition 6 from Erol et al. (2020) and Proposition 7 extends this to random graphs sampled from the corresponding graphon. More explicitly, the contagion dynamics (and hence speed resilience tradeoff) on the graph $G_s^{T,R}$ for $G \in \mathcal{G}$ also applies to the random graph sampled from $W_{G_s^{T,R}}$.

B Higher dimensions

The model in the main text was developed for networks embedded in $\mathbb{R}$. We now show that a version of this tradeoff continues to obtain in higher dimensions.

B.1 Model

Consider a continuum of agents indexed by $\mathbb{R}^n$ for $n \geq 1$. Agent $i \in \mathbb{R}$ has measure of links given by $\mu_i : \mathcal{B}(\mathbb{R}^n) \to [0, +\infty)$ where we use $\mathcal{B}(E)$ to denote the Borel sigma-algebra generated by the set E. We will impose the following regularity assumptions which are analogous to symmetry and translation in variance in the main text.

Assumption 1 (Translation invariance in n dimensions)**.** Take $i, j \in \mathbb{R}^n$ and let $V_{ij} : \mathbb{R}^n \to \mathbb{R}^n$ be the linear operator which sends i to j i.e., $V_{ij}(i) = j$. Then for all $B \in \mathcal{B}(\mathbb{R}^n)$, $\mu_i(B) = \mu_j(V_{ij}(B))$ where we adopt the notation $V_{ij}(B) := \{V_{ij}(k) : k \in B\}$.

Define $\mu := \mu_{\mathbf{0}}$ as the measure centered on the point zero.

Assumption 2 (Rotational symmetry in n dimensions)**.** For any set $E \in \mathcal{B}(\mathbb{R}^n)$, let $R(E) \subseteq \mathbb{R}^n$ be a measure-preserving rotation around the origin. Then $\mu_0(E) = \mu_0(R(E))$.[15]

These assumptions allow us to reduce the network to a single function $G : \mathbb{R}_{\geq 0} \to \mathbb{R}_{\geq 0}$. We work with the Euclidian norm $|| \cdot ||_2$ on $\mathbb{R}^n$. Let $B_\delta(i) := \{k \in \mathbb{R}^n : ||k - i||_2 \leq \delta\}$ denote the $\delta-$ball around the point i. Assumptions 3 and 4 imply that the entire network $\{\mu_i\}_{i \in \mathbb{R}^n}$ is characterised by function $G(\delta) := \mu(B_\delta(0))$ for $\delta \geq 0$. We now define the class of functions G we will focus on.

Definition 7. Define $\mathcal{G}$ as the set of functions $[0, +\infty) \to [0, 1]$ such that for each $G \in \mathcal{G}$,

[15] R is a standard 'rotation matrix'.

(i) G is increasing, $\lim_{x\to\infty} G(x) = 1$, and G is absolutely continuous. This implies there exists a function $g : [0, \infty) \to [0, M]$ for some $M < +\infty$ such that for any $a, b \geq 0$, $a \leq b$,

$$\int_a^b g(x)dx = G(b) - G(a).$$

(ii) $g(x) < g(x')$ whenever (i) $x > x'$; and (ii) $g(x') > 0$.

We will focus on networks in the class $\mathcal{G}$: part (i) provides weak regularity conditions to guarantee that μ is smooth and admits a density g; part (ii) states that each agent has fewer links to neighbours further away. As in the main text, we might think of $\mathbb{R}^n$ as a latent space of traits or opinions—part (ii) then reflects the homophily of the network.

We will consider contagious dynamics of the system given by the tuple (G, I_0, q) in which G characterizes the network in question, I_0 is the initial set of infected agents, and q is the contagion threshold. As in the main text, we assume that I_0 is a compact ball in whatever dimension we work on, and that it is centered around the origin. As such, we write $I_0 = B_{r_0}(0)$ where r_0 is the radius of the initial set of infected agents. We let $m_0 := \mu(I_0)$ be the measure of initially infected agents. Fixing the dimension $n \geq 1 : n \in \mathbb{N}$ we are working on, we can equivalently work with m_0 and r_0. As in the main text, we let $(I_t)_{t\in\mathcal{T}}$ denote the sequence of infected agents, and $m_t := \mu(I_t)$ to denote the measure of infected agents at any time $t \in \mathcal{T}$.

We start with a simple observation that along every time path, the set of infected agents remains a compact Euclidian ball.

Lemma 2. For any $n \geq 1 : n \in \mathbb{N}$ and any $t \in \mathcal{T}$, I_t is a compact ball in $\mathbb{R}^n$.

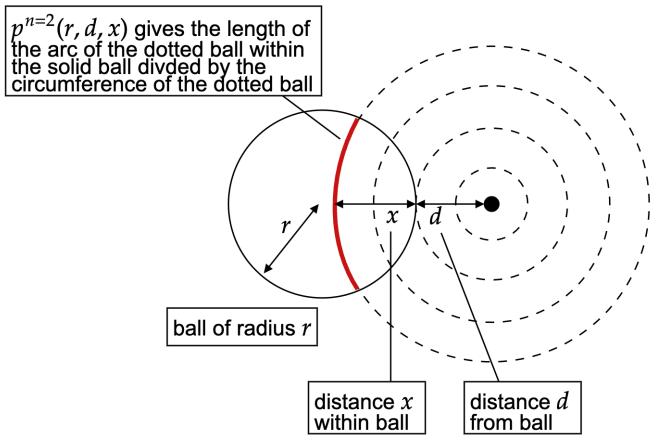
Proof. If $t = 0$ there is nothing to prove since we assumed I_0 was a closed ball. Now suppose that I_t is a closed ball with center normalized at 0. Conclude by observing that for any two agents $i, j \in \mathbb{R}^n$ such that $||i-0||_2 = ||j-0||_2$, at time $t + 1$ either both i and j are infected, or both are not since rotational symmetry guarantees that both have and equal proportion of neighbours infected at time

t. Hence I_{t+1} is a closed ball. Finally, it is not hard to see that the limit speed of this process is finite so for any t, $\mu(I_t)$ is bounded. □

In light of the above lemma, since for each $t \in \mathcal{T}$ the set of infected agents is a closed ball, we can equivalently track the path of contagion through the sequence $(r_t)_{t\in\mathcal{T}}$ where $I_t = B_{r_t}(0)$ i.e., r_t is the radius of the infected set at time t.

We now introduce an additional piece of notation. For dimension $n \geq 1 : n \in \mathbb{N}$, we define $p^n(r, d, x)$ as follows. Suppose there is a closed ball of radius r in $\mathbb{R}^n$ and take any point distance d away from the ball. $p^n(r, d, x)$ gives the proportion of the surface of a hypothetical closed ball of radius $x + d$ centered on that point within the original ball. Figure 7 illustrates this for $n = 2$ in which $p^n(r, d, x)$ gives the ratio of the red arc divided by the circumference of the circle the arc is on.

Figure 7: Illustration of $p^n(r, d, x)$.



For $n = 1$, we have that

$$p^1(r, d, x) = \begin{cases} 1/2 & \text{if } 0 \leq x \leq 2r \\ 0 & \text{otherwise,} \end{cases}$$

which made our analysis in the main text simple. For $n > 1$, there will, in general, be more complicated non-monotonicities captured by p^n. Observe that for each

dimension n,

$$\frac{\partial p^n(r,d,x)}{\partial r} \geq 0, \quad \frac{\partial p^n(r,d,x)}{\partial d} \leq 0,$$

and

$$p^n(r,d,0) = 0, \quad \lim_{x\to+\infty}\left(\lim_{r\to+\infty} p^n(r,d,x)\right) = 1/2.$$

Finally observe that $p^n(r,d,x)$ can be non-monotone in x: for instance, for $n=2$ since the infected agents is a circle, we have that p^n is first increasing in x, and then decreasing thereafter. Finally, observe that p^n homogenous of degree 0:

$$p^n(\lambda r, \lambda d, \lambda x) = p^n(r,d,x)$$

for any $\lambda > 0$.

Now suppose that at time t, there is a ball of radius r_t infected. Under network $G \in \mathcal{G}$ with associated density g, agent distance d away from the infected ball has

$$\int_{x=0}^{x=2r} p^n(r,d,x)g(x+d)dx$$

infected neighbours, noting that this is increasing in r. Analogous to the main text, define $r_0(q,G)$ as the minimum radius of infected agents required to precipitate contagion. We have

$$r_0(G,q) = \inf\left\{r \geq 0 : \int_{x=0}^{x=2r} p^n(r,0,x)g(x)dx \geq q\right\}.$$

Similarly, conditional on contagion occurring, define $a_\infty(q,G)$ as

$$a_\infty(G,q) = \sup\left\{d \geq 0 : \int_{x=0}^{\infty} \lim_{r\to+\infty} p^n(r,d,x)g(x+d)dx \geq q\right\}.$$

Our first result develops an ordering over networks which shows the tradeoff for all contagion thresholds q. This is an analogue of Proposition 1 developed in

the main text, although the conditions are more special than that of first-order stochastic dominance.

Proposition 8. For any dimension n, suppose that G, G' are such that G' is a rescaled version of G i.e.,

$$G(x) = G'(\lambda x) \quad \text{for } \lambda > 1.$$

Then for all $q \in (0, 1/2)$, (i) $\lambda \cdot r_0(G, q) = r_0(G', q)$; and (ii) $\lambda \cdot a_\infty(G, q) = a_\infty(G', q)$.

Before we prove the proposition, it is useful to highlight its connection to Example 1 in the main text: when we 'stretch' G in this way (as when we increase the variance of the normal distribution), we are, in effect, preserving the contagious dynamics, but 'stretching' the metric we are working on. It should therefore be unsurprising that since G and G' are scaled-up/scaled-down versions of each other, that the speed-resilience tradeoff obtains for all contagion thresholds.

Proof. Observe $g(x) = \lambda g'(\lambda x)$ and for any $r_0 > 0$ (radius of the initial ball), we have

$$\begin{aligned}
\int_{x=0}^{x=2r_0} p^n(r, 0, x) g(x) dx &= \int_{x=0}^{x=2r_0} p^n(\lambda r, 0, \lambda x) g(x) dx && (p^n \text{ is HOD0}) \\
&= \int_{x=0}^{x=2r_0} p^n(\lambda r, 0, \lambda x) \lambda g'(\lambda x) dx \\
& && (\text{replacing } g \text{ with } g') \\
&= \int_{y=0}^{y=2\lambda \cdot r_0} p^n(\lambda r, 0, y) g'(y) dy \\
& && (\text{change of var. } y = \lambda \cdot x)
\end{aligned}$$

and part (i) follows from our definition of r_0. Similarly,

$$\begin{aligned}
\int_{x=0}^{\infty} \lim_{r\to+\infty} p^n(r,d,x)g(x+d)dx &= \int_{x=0}^{\infty} \lim_{r\to+\infty} p^n(r,d,x)\lambda\cdot g'(\lambda x+\lambda d)dx \\
&\qquad\qquad \text{(replacing } g \text{ with } g') \\
&= \int_{x=0}^{\infty} \lim_{r\to+\infty} p^n(\lambda r,\lambda d,\lambda x)\lambda\cdot g'(\lambda x+\lambda d)dx \\
&\qquad\qquad (p^n \text{ is HOD0}) \\
&= \int_{y=0}^{\infty} \lim_{r\to+\infty} p^n(\lambda r,\lambda d,y)g'(y+\lambda d)dy \\
&\qquad\qquad \text{(change of var. } y=\lambda\cdot x) \\
&= \int_{y=0}^{\infty} \lim_{r\to+\infty} p^n(r,\lambda d,y)g'(y+\lambda d)dy \\
&\qquad\qquad (\lambda>1)
\end{aligned}$$

and part (ii) follows from our definition of a_∞. □

The ordering in the previous proposition was quite strong, and required that the two graphs, G, G' were rescalings of each other. This was, for instance, fulfilled by Example 1 in the main text since the normal distribution had this property as we control the variance. We now consider the condition of Proposition 1 the main text–that of first-order stochastic dominance–and show that a weaker (i.e., not for all contagion thresholds) version of this tradeoff continues to obtain in n dimensions.

Proposition 9. For any dimension n, suppose that $G \leq G'$. Then there exists $\underline{q}, \overline{q} \in (0, 1/2)$ such that

(i) For $q \leq \underline{q}$, G is more resilient than G'.

(ii) For $q \geq \overline{q}$, G has quicker limit speed than G'.

Proof. The condition implies there is an interval $[0, \underline{x}]$ where $g \leq g'$ pointwise, and an interval $[\overline{x}, +\infty)$ where $g \geq g'$ pointwise. For resilience, choose $\underline{q}$ such

that

$$\int_{x=0}^{x=\underline{x}} p^n(r,d,x)g(x)dx \leq \int_{x=0}^{x=\underline{x}} p^n(r,d,x)g'(x)dx = \underline{q}$$

noting that this can always be done since

$$\int_{x=0}^{x=y} p^n(r,d,x)g'(x)dx$$

is continuous in $\underline{x}$ over the interval $[0,+\infty)$ and starts at zero. Then clearly we have $\underline{x} = r_0(q,G') \leq r_0(q,G)$. This extends to $q \leq \underline{q}$ since $g \leq g'$ over the interval $[0,\underline{x}]$.

For speed, observe that

$$\int_{x=0}^{\infty} \lim_{r\to\infty} p^n(r,d,x)g'(x+d)dx$$

is continuous in d over the interval $[0,+\infty)$, as well as decreasing in d with

$$\begin{aligned}\lim_{d\to+\infty} \int_{x=0}^{\infty} \lim_{r\to\infty} p^n(r,d,x)g'(x+d)dx =& 0 \quad \text{and} \\ \lim_{d\to 0} \int_{x=0}^{\infty} \lim_{r\to\infty} p^n(r,d,x)g'(x+d)dx =& 1/2.\end{aligned}$$

Now pick $d = \overline{x}$, and from the intermediate value theorem we can find $\overline{q}$ such that

$$q' = \int_{x=0}^{\infty} \lim_{r\to\infty} p^n(r,\overline{x},x)g'(x+\overline{x})dx \leq \int_{x=0}^{\infty} \lim_{r\to\infty} p^n(r,\overline{x},x)g(x+\overline{x})dx$$

where the inequality follows from the fact that $g(x) \geq g'(x)$ for all $x \geq \overline{x}$. Then once again noticing that the integral is decreasing in d, we have $\overline{x} = a_\infty(q',G') \leq a_\infty(q',G)$. This extends to $q \geq \overline{q}$ since $g \geq g'$ over the interval $[\bar{x},+\infty)$. □

Note that since p^n is a nonlinear and potentially complicated function, here we are relying on dominance of one density function over the other on some interval to make comparisons. The next result develops an analog of Proposition 3 in the

main text.

Proposition 10. For any dimension n, and $G, G' \in \mathcal{G}$ suppose that there exists some $\bar{x} \in (0, +\infty)$ such that for all $x \leq \bar{x}$, $G(x) \leq G'(x)$ and for all $x' \geq \bar{x}$, $G(x') \geq G'(x')$. Then for sufficiently low values of q, G is both more resilient than G' as well as has slower limit speeds.

Proof. Observe that the condition implies that there exists a pair of thresholds $\underline{z}, \bar{z} \in [0, +\infty)$ such that for $x \leq \underline{z}$, $g(x) \leq g'(x)$ and for $x \geq \bar{z}$, $g(x) \leq g'(x)$. Part (i) proceeds identically to the proof of Proposition 10: there then exists a threshold $q'' \in (0, 1/2)$ such that for all $q \leq q''$, $r_0(G, q) \leq r_0(G', q)$.

For part (ii) on speed, observe that

$$\int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, d, x) g(x + d) dx$$

is continuous in d over the interval $[0, +\infty)$ and decreasing in d with

$$\lim_{d \to +\infty} \int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, d, x) g'(x + d) dx = 0 \quad \lim_{d \to 0} \int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, d, x) g'(x + d) dx = 1/2.$$

Now pick $d = \overline{z}$, and from the intermediate value theorem we can find $q' \in (0, 1/2)$ such that

$$q' = \int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, \overline{z}, x) g(x + \overline{x}) dx \leq \int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, \overline{z}, x) g'(x + \overline{z}) dx$$

where the inequality is because $g(x) \leq g'(x)$ for all $x \geq \bar{z}$. Then since the functional

$$\int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, d, x) g(x + d) dx$$

is decreasing in d, for any $q \leq q'$ we can find $x' \geq \bar{z}$ such that

$$q = \int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, x', x) g(x + x') dx \leq \int_{x=0}^{\infty} \lim_{r \to \infty} p^n(r, x', x) g'(x + x') dx$$

hence $a_\infty(q, G) \leq a_\infty(q, G)$ for all $q \leq q'$. Conclude by picking $q' \wedge q''$ i.e., the minimum of the two thresholds. □

C Minimum seed set

Throughout the main text, we assumed that I_0 is a closed interval. It is not difficult to see that this will not, in general, be the minimum seed required to precipitate contagion: consider, for instance, the case in which starting from $I_0 = [-a, a]$ which precipitates contagion under G, we instead choose $[-a, -\epsilon] \cup [\epsilon, a]$and one can verify that contagion nonetheless obtains for sufficiently small ϵ. This is because g is strictly decreasing on its support, so contagion first occurs over the region $[-\epsilon, \epsilon]$ before spreading to the rest of the real line.

Naturally, one might ask whether we can continue to carve out portions of the original set I_0. Suppose that I_0 is the smallest closed interval on which contagion occurs i.e., $\mu(I_0) = m_0(G, q)$. Define I_0^{min} as the minimum set in $\mathbb{R}$(not necessarily an interval) which can precipitate contagion i.e., I_0^{min} is a solution to the problem

$$\inf_{I_0 \subseteq \mathbb{R}} \mu(I_0) \quad s.t. \quad \lim_{t \to +\infty} \mu(I_t) = +\infty.$$

We now lower-bound the value of the problem:

Proposition 11 (Lower bound on minimum seed set)**.** $\mu(I_0^{min}) \geq 2m_0(G, q/2)$.

Proof. Let I_0 be a minimal interval to precipitate contagion and without loss center $I_0 = [-a, a]$ on the point 0 where $a = \mu(I_0)/2$. Now let I_0^{min} be a measurable set which is sufficient to precipitate contagion. Let $(I_t^{min})_t$ be the sequence of sets of infected agents starting from the initial infected set I_0^{min}. Since contagion occurs, a necessary condition is that there must exist some $i \in \mathbb{R} \setminus I_0^{min}$ for which $i \in I_1^{min}$ i.e., is infected in period 1.

We will show that this necessary condition imposes a lower bound on $\mu(I_0^{min})$ Fix any agent $i \in \mathbb{R} \setminus I_0^{min}$ and observe that the minimum set required for i to be affected cannot be attained; but it can be approached by sets of the form $E_\epsilon := [i - x, i - \epsilon] \cup [i + \epsilon, i + x]$ because of symmetry and strict decreasing differences. The set which attains the infimum is thus of the form $[i - x, i + x]$ where x is chosen so that

$$2G(x) \geq q \implies x = G^{-1}(q/2) = m_0(G, q/2)$$

hence $\mu(I_0^{min}) \geq 2m_0(G, q/2)$ as required. $\square$

Finally note that this bound is tight: take $G(x) = x$ for $x \leq 1/2$ and $G(x) = 1/2$ otherwise.[16] Then observe that

$$2m_0(G, q/2) = G^{-1}(q/2) = 2 \cdot q/2 = q = G^{-1}(q) = m_0(G, q).$$

[16]In the main text we assumed that G has strict decreasing differences for ease of exposition; we could consider $G_\epsilon(x) = x + \epsilon\sqrt{x}$ instead and take ϵ small.