Contagion in Graphons*

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Abstract

The analysis of threshold contagion processes in large networks is challenging. While the lack of accurate network data is often a major obstacle, finding optimal interventions is computationally intractable even in well-measured large networks. To obviate these issues we consider threshold contagion over networks sampled from a graphon—a flexible stochastic network formation model—and show that in this case the contagion outcome can be predicted by only exploiting information about the graphon. To this end, we exploit a second interpretation of graphons as graph limits to formally define a threshold contagion process on a graphon for infinite populations. We then show that contagion in large but finite sampled networks is well approximated by graphon contagion. This convergence result suggests that one can design interventions for large sampled networks by first solving the equivalent problem for an infinite population interacting according to the limiting graphon. We show that, under suitable regularity assumptions, the latter is a tractable problem and we provide analytical characterizations for the extent of contagion and for optimal seeding policies in graphons with both finite and infinite agent types.

Keywords: Networks, Graphons, Contagion, Optimal seeding

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1 Introduction

Many phenomena, such as technology adoption, bank failures, and drug use start with a few initial "seeds" and spread quickly through social and economic networks. Understanding how the extent of the spread depends on the initial seeds and the structure of the network is, therefore, a key policy concern. Some simple contagion processes in which agents require a single exposure to be infected (e.g., the the classic SIR model or the independent cascade model due to Kempe et al. (2003)) do not appear to be particularly sensitive to the location of initial seeds (Akbarpour et al., 2020). On the other hand, complex contagion processes in which agents might require several exposures to get infected, such as the *linear threshold model* due to Granovetter (1978), are very sensitive to initial seeds and can be difficult to analyze, particularly in large networks. In this paper, we propose a novel and tractable way to analyze the linear threshold model over networks sampled from a *graphon*—a general nonparametric network formation model which includes the random graph model due to Erdős and Rényi (1959) and stochastic block models in which agents belong to one of several communities. Our motivation for considering sampled networks is that in many applications detailed data about the network is not available (Breza et al., 2020), but an observer might nevertheless have a theory about the data-generating process (i.e., the stochastic network formation model). We, therefore, analyze whether the contagion outcome in sampled networks can be predicted by exploiting only statistical network information (captured by the graphon) in the large population regime.

Formally, a graphon is a measurable function $W : [0,1]^2 \to [0,1]$. In this paper, we use graphons to represent a stochastic network formation model, however, graphons also have an interpretation as a limit of a sequence of graphs with increasing number of nodes (Borgs et al., 2008; Lovász, 2012). According to this latter interpretation, the $[0,1]$ interval represents a continuum of heterogeneous agents, each associated with a *label* $u \in [0,1]$, so that $W(u,v)$ denotes the level of interaction between labels u and v. Building on this interpretation, our first contribution is to formally define a linear threshold contagion model for a continuum of agents interacting according to a graphon. As in the standard linear threshold model in finite networks, labels in $[0,1]$ can be in one of two states: *infected* and *not infected*. At time $t = 0$ a subset $C_0 \subset [0,1]$ of the labels are exogenously infected as *seeds* (e.g., initial adopters or initial bank failures).[1] In subsequent time steps, labels are infected if they were infected before or if the fraction of their neighbors who are infected exceeds a label-specific threshold

[1] We use $\subset$ to mean weak and $\subsetneq$ to mean strict inclusion.

$\tau(u)$. Formally, a label $u \in [0,1]$ is *exposed* to infection at time $t = 1, 2, 3 \ldots$ if

$$\frac{\int_{C_{t-1}} W(u,v)dv}{\int W(u,v)dv} > \tau(u),$$

where $C_{t-1} \subset [0,1]$ is the set of infected labels at time $t-1$; a label is infected at time t if it was either infected at time $t-1$ or exposed at time t.

As the first main result, we show that the outcome of contagion in large networks sampled from a graphon is well-approximated by the outcome of the graphon contagion process just defined (Theorems 1a and 1b). More precisely, our results show that contagion in a graphon can accurately approximate the entire dynamics (and therefore the speed) of contagion rather than simply the final set of infected agents.

Besides being of independent interest, this convergence result is particularly powerful when we turn to the design of optimal seeding sets. In fact, solving the optimal seeding problem based on exact network data is a computationally hard problem for large populations (Kempe et al., 2003). As a second main result, we propose a novel approach to optimal seeding based on the graphon limiting process. Specifically, we introduce two classes of *optimal seeding problems for graphon contagion processes*: the *max-reach problem* in which the planner wants to select seeds to maximize the spread of contagion given a constraint on the measure of the seed set and the *min-seed problem* in which the central planner wants to select the smallest seed set that achieves a target level of contagion. We derive structural properties of the solution to the graphon seeding problems (Theorem 2) and we show how a planner can exploit such graphon solutions to design seeding policies for sampled networks.

Our approximation and optimal seeding results are particularly useful if one can easily compute the outcome of graphon contagion and the solution to the graphon seeding problems mentioned above. As a third contribution, we show that this is the case if the graphon has a suitable regularity structure. Specifically, we focus on two classes of commonly used graphons. First, we consider stochastic block models in which agents belong to a finite number k of communities ("types"). We show that contagion in a graphon in this class is equivalent to contagion in an auxiliary finite network with $2k$ nodes. As a result, the max-reach problem in a stochastic block model reduces to an optimization problem with only k variables. This reduction offers a clear computational advantage since in general the number of communities k is much smaller than the number of agents. Second, we consider contagion in various structured graphons with an infinite number of agent types. We give clean characterizations of the dynamics of contagion and find analytical solutions to the max-reach and min-seed problems in these structured graphons. Taken as a whole, our illustrations emphasize the tractability offered by the analysis of contagion in graphons.

There is a vast literature on the linear threshold model and on complex contagion more generally. Previous work has analyzed complex contagion in *deterministic networks* (see, e.g., Morris (2000); Kempe et al. (2003); Adam et al. (2012); Lim et al. (2016)), in *partial information settings* (see, e.g., Stein et al. (2017); Wilder et al. (2018); Chin et al. (2022); Eckles et al. (2022)) as well as in *stochastic network models* (see, e.g., Watts (2002); Amini (2010); Lelarge (2012); Moharrami et al. (2016)). Three papers in the latter strand are the closest to our contribution: Jackson and Storms (2019), Rossi et al. (2017), and Sadler (2020). Closest to our work, Jackson and Storms (2019) study equilibria of binary coordination games (that are closely related to threshold contagion) in stochastic block models (a subcase of graphons). They show that such equilibria generate an "atomic" partition of the network that goes beyond standard community structure. Our results are complementary to this paper since (i) the graphon model allows us to explicitly analyze the *dynamics* of the contagion process; (ii) our solution to the max-reach problem is *optimal* in the graphon and (iii) while the exact atomic partition can be challenging to compute in large networks, the *computational tractability* of our graphon-based seeding policy is independent of the sampled network size. The other two papers closest to ours study contagion dynamics in the context of a configuration model, in non-strategic (Rossi et al., 2017) and strategic (Sadler, 2020) settings. Our model is different because instead of the configuration model, we use the graphon model as our stochastic network formation process. This choice has several consequences: (i) while the configuration model generates graphs that are sparse and locally tree-like, graphons encode a large class of (dense) network formation models, such as stochastic block models which can capture the assortativity, community structure, nestedness and clustering of social networks; (ii) we approximate the exact set rather than the total number of infected agents and (iii) our explicit characterization of the limiting process enables a new approach for finding the optimal seed set in large networks.

Finally, our work is related to a growing literature that studies the limiting behavior of network processes by using graphons (see e.g. Vizuete et al. (2020); Caines and Huang (2018); Gao and Caines (2019)).[2] In this literature, Parise and Ozdaglar (2022) is the closest to our paper. Parise and Ozdaglar (2022) adopt a similar approach to ours by first defining a limiting process for an infinite population (in that case a graphon game) and then showing that equilibria of sampled network games (i.e., games played over finite networks sampled from the graphon) can be well approximated by equilibria of the limiting graphon game, for large enough population. We note that the threshold dynamics considered here coincide with the best response dynamics of a coordination

[2]This strand is closely related to models of global games with a continuum of players. See, for example, Morris and Shin (2003, Chapter 5.1) and Morris and Shin (2005).

game (which is an example of a network game). Yet the analysis of Parise and Ozdaglar (2022) cannot be applied in our setting for three main reasons: (i) Parise and Ozdaglar consider smooth and convex network games, instead in coordination games agents have a discrete, $\{0,1\}$ set of actions; (ii) coordination games have multiple equilibria while convergence in Parise and Ozdaglar's model is guaranteed for games with unique equilibria and (iii) we consider the entire dynamics given by the threshold model, instead of only focusing on the equilibrium outcome.

This paper is organized as follows. Section 2 provides an illustrative example of contagion in a graphon corresponding to the Erdős-Rényi model. Section 3 describes our model of contagion in graphons and of corresponding contagion in networks sampled from graphons. Section 4 states a simplified version of our convergence result (for ease of readability). Section 5 introduces optimal seeding problems for contagion in graphons. Section 6 discusses applications of our results to stochastic block models, i.e., the special class of graphons with finite types. Section 7 considers contagion in graphons with infinite number of types. Section 8 concludes the paper. Appendix A states a stronger version of the main convergence result. Appendix B contains technical details for the results in Section 7. Appendix C contains proofs. While in the main text we consider deterministic thresholds, in Appendix D we discuss an example of contagion in graphons with random thresholds.

2 An illustrative example

To develop some intuition, we start our analysis by considering contagion in networks sampled from an Erdős-Rényi random graph model, which can be seen as a special case of a graphon where each edge is independently realized with probability $p \in (0,1]$. We here assume that all agents have the same contagion threshold τ. According to the standard linear threshold model, an agent is exposed at step t if

$$\frac{\text{number of infected neighbours at } t-1}{\text{number of neighbours}} > \tau. \tag{2.1}$$

Intuitively, for a very large number of agents, by applying the law of large numbers, we expect that the number of neighbors for each agent should concentrate around its mean. Recalling that in an Erdős-Rényi model each link is realized with an independent probability p we can then expect (2.1) to be well approximated by

$$\frac{p \times \text{number of infected agents at } t-1}{p \times n} > \tau, \tag{2.2}$$

which is the condition for contagion in a complete network with edge weights p. Hence, in the limit of large populations, the contagion condition simplifies significantly.

To formalize this intuition, we next define a contagion process for infinite populations by assuming that there is a unit mass of agents, which from here on we identify with a *label* in the interval $[0,1]$,[3] and we assume that each label is connected to any other label with weight $p \in (0,1]$.

We then define a threshold contagion process for such an infinite population as follows. Initially, in step $t = 0$, a set $C_0 \subset [0,1]$ of labels is infected as *seeds*. In each subsequent step $t = 1, 2, 3, \ldots$, a label becomes *infected* and is added to set C_t of infected labels at time t when the integral of edge weights with infected neighbors divided by the label's total edge weight exceeds the threshold τ. More formally, a label $u \in [0,1]$ becomes infected at time t if either he was infected at time $t-1$ or if he is *exposed* at time t, that is,

$$\frac{\int_{C_{t-1}} p dv}{\int_0^1 p dv} > \tau. \tag{2.3}$$

In this simple setting, such a model of contagion in a graphon is particularly simple and provides sharp predictions of agents' behavior. Denoting by μ the measure of a set, we obtain:

1. If $\mu(C_0) > \tau$, then (2.3) holds and all labels become infected at the first step;
2. If $\mu(C_0) \leq \tau$, then (2.3) does not hold and no new label becomes infected.

The graphon model predicts a sharp phase transition. If the size of the seed set $\mu(C_0)$ is greater than the threshold τ contagion spreads to the entire $[0,1]$ interval while if $\mu(C_0) < \tau$ no contagion takes place. Figure 1 suggests that this behavior is predictive of the contagion outcome in networks sampled from the Erdős-Rényi model (illustrated for two different choices of p and τ). Specifically, the dashed line in Figure 1 shows the fraction of agents that are infected at the end of the contagion process in the graphon model as a function of the size $\mu(C_0)$ of the initial seed set. The colored lines show the fraction of agents that are infected at the end of the contagion process in networks of different sizes n, sampled according to the Erdős-Rényi model when the initial seed set is composed of $n \times \mu(C_0)$ randomly selected agents. Each line represents the average of infected agents over 50 different network realizations. We can see that for larger n the behavior in the sampled network becomes more and more aligned with the one predicted from the graphon model. In the rest of the paper, we show that the intuition from this simple Erdős-Rényi example can be applied more generally to networks sampled from a graphon.

[3] Henceforth, we use the term "label" to refer to the infinite population setting. The term "agent" is reserved for finite populations.

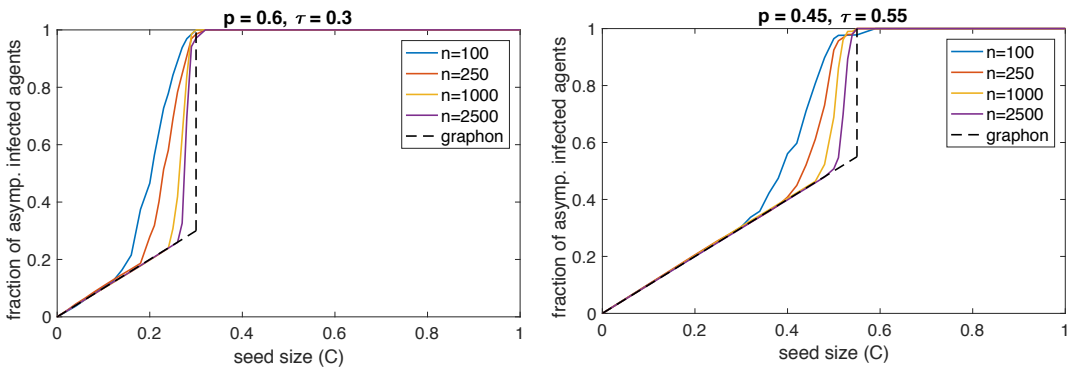


Figure 1: Final fraction of infected agents as a function of the measure $C = \mu(C_0)$ of the initial seed set size C_0 for an Erdős-Rényi graphon and for networks of size n sampled from it (averaged over 50 repetitions) for different values of p and τ.

3 A model of threshold contagion in graphons

In this section, we consider a general class of stochastic network formation processes by using the graphon framework introduced in Lovász (2012, Chapter 10). We first define graphons and sampled networks. We then introduce threshold contagion in a graphon. Finally, we describe threshold contagion in a sampled network.

3.1 Graphons and sampled networks

A *graphon* is a measurable function $W : [0,1]^2 \to [0,1]$ that can be used to describe a probability distribution over the space of networks. The following definition formally connects graphons to stochastic network formation models and illustrates how one can sample from the graphon distribution to construct a sampled network.

Definition 1 (Sampled network)**.** Given any graphon W and an arbitrary number n of nodes, uniformly and independently sample n *labels* $\{u_i^{(n)}\}_{i=1}^n$ from $[0,1]$ and construct a *sampled network* by randomly connecting nodes $i, j \in \{1, \ldots, n\}$ with Bernoulli probability $W(u_i^{(n)}, u_j^{(n)})$. Let $A^{(n)} \in \{0,1\}^{n \times n}$ be the adjacency matrix of such a sampled network.

In Definition 1, the value $W(u, v)$ of the graphon encodes information about the strength of interaction between two arbitrary labels u and v. We say that two labels u and v have the same *type* if $W(u, y) = W(v, y)$ and $W(x, u) = W(x, v)$ for all $x, y \in [0,1]$, that is if u and v interact in the same way with the rest of the labels. The Erdős-Rényi model described in Section 2 can be obtained as a special case of Definition 1 by selecting the constant graphon $W(x, y) = p$. Note that in this case, all labels have the same type. In the following example, we illustrate how graphons can be used to model stochastic block models (SBMs), which have a finite number of different types.

Example 1 (Stochastic block model). Consider networks formed according to a stochastic block model. Agents are divided into k *communities* and denote by π_h the probability that a random agent belongs to the community h, with $\sum_{h=1}^{k} \pi_h = 1$. Agents form links with Bernoulli probability $w_{h_1,h_2} \in [0,1]$ depending on their communities h_1, h_2. In many social networks, for example, agents interact with a higher probability if they are in the same community and a smaller probability if they belong to different communities. Such a community structure can be generated from a piece-wise constant graphon W_{SBM} constructed as follows: Partition $[0,1]$ into k disjoint intervals of labels $\{\mathcal{I}_h\}_{h=1}^{k}$, with $\mu(\mathcal{I}_h) = \pi_h$, and set

$$W_{\text{SBM}}(u,v) = w_{h_1,h_2} \text{ if } u \in \mathcal{I}_{h_1}, v \in \mathcal{I}_{h_2}.$$

By construction, any two labels belonging to the same community have the same type. Hence in stochastic block models, we can use the words "community" and "type" interchangeably. For example, Figure 2 shows three networks obtained by randomly partitioning n agents into two communities (red and blue) with probabilities $\pi_{\text{red}} = 0.40$ and $\pi_{\text{blue}} = 0.60$ and then connecting each pair of agents in the same red or blue community with Bernoulli probability $w_{rr} = w_{bb} = 0.7$, red to blue agents with probability $w_{br} = 0.4$ and blue to red agents with zero probability.

Finally, graphons can be used to encode network formation processes with an infinite number of types, as the next example shows.

Example 2 (Location Model). Suppose that agents (e.g., homeowners or politicians) are located along a $[0,1]$ interval (e.g., a street or a political spectrum) and the interaction between agents i and j is a decreasing function of their distance (e.g., travel time or political differences). If agents interact with a likelihood that depends on relative position then there is a continuum of types, each type corresponding to a specific location. An example of such a graphon is given in Section 7.

3.2 Threshold contagion in graphons

We now define a threshold contagion process in a *graphon* $W : [0,1]^2 \to [0,1]$, with an *initial seed set* $C_0 \subset [0,1]$ and a *threshold function* $\tau : [0,1] \to [0,1]$.[4] Let

$$d(u,X) := \int_0^1 W(u,v)\mathbb{1}_X(v)dv$$

be the mass of neighbours of label u whose labels are in $X \subseteq [0,1]$, namely the degree of u with labels in X. Also denote $d(u) = d(u,[0,1])$ in short. We are primarily interested

[4] In our model we focus on deterministic thresholds. We discuss in Appendix D how results can be generalized to thresholds sampled uniformly at random.

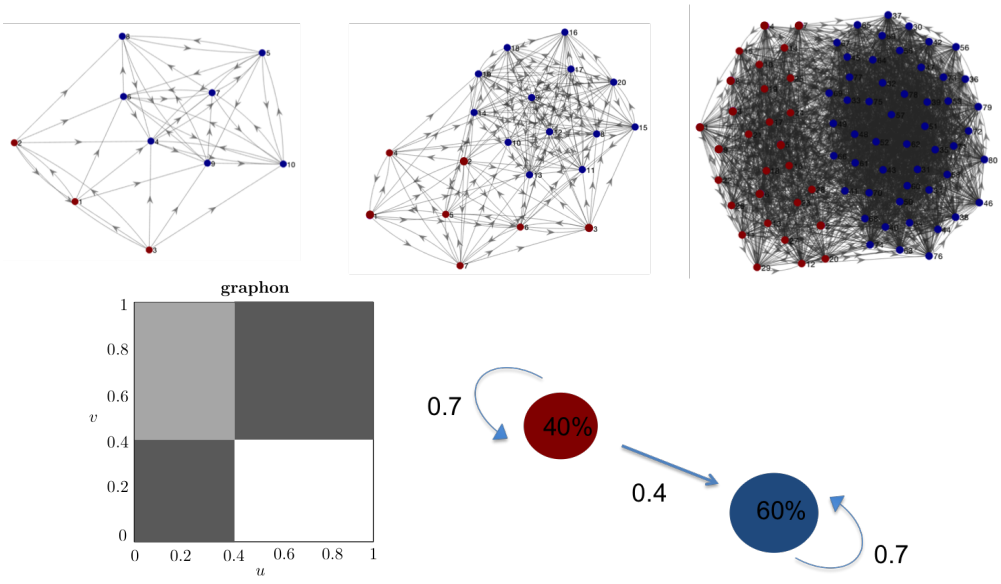


Figure 2: Top: three sampled networks from the stochastic network formation process described in Example 1 for networks of sizes $n = 10, 20, 80$. Bottom left: Illustration of the graphon $W(u, v)$ in Example 1 as a function of both u and v (a linear grayscale colormap is used with white associated to $W = 0$ and black to $W = 1$). Bottom right: a schematic of the interaction among the communities in Example 1; nodes indicate communities, node labels are fractions of agents in the communities, arrows indicate the probability of an agent from a community interacting with agents from another community.

in the infected set of labels at time t denoted by C_t. At every step $t \geq 1$ each label $u \in [0, 1]$ gets infected, and is added to the *infected set* C_t, if either

- the label was infected at the previous stage, that is, $u \in C_{t-1}$; or
- the label belongs to the *exposed set*, denoted by $\hat{C}_t$, which happens when enough of its neighbors are infected, i.e., $d(u, C_{t-1}) > \tau(u)d(u)$.

Hence the set of infected labels at time t is defined as $C_t = C_{t-1} \cup \hat{C}_t$, that is, the union of previously infected and exposed labels. We denote by $C_\infty = \cup_{t=0}^{\infty} C_t$ the final *outcome* of contagion and by $\mu(C_\infty)$ the *reach* or *spread* of contagion (i.e. the measure of C_∞).

3.3 Threshold contagion in sampled networks

The graphon contagion process in Section 3.2 is defined for an infinite population. We next define contagion in sampled networks of finite size n constructed from the graphon according to Definition 1. As in Definition 1, we add superscript $^{(n)}$ in our notation to refer to the sampled network of size n and subscript $_i$ to agent i. Let $[n] = \{1, 2, ..., n\}$ denote the set of agents in a finite population of size n. We define

$$C_0^{(n)} := \{i \in [n] \mid u_i^{(n)} \in C_0\}$$

as the sampled initial seed set, i.e., the set $C_0^{(n)} \subset [n]$ of agents whose label belongs to C_0. Similarly, we can define the threshold of the agent i as the threshold of an agent with label $u_i^{(n)}$, i.e.,

$$\tau_i^{(n)} := \tau(u_i^{(n)}).$$

For $Y \subset [n]$, let $d_i^{(n)}(Y)$ stand for the number of neighbors of i in Y in the sampled network. Denote $d_i^{(n)} = d_i^{(n)}([n])$ the degree of i in short.

Contagion in the sampled network evolves according to the standard linear threshold model of contagion over finite networks. Denoting by $C_t^{(n)}$ the set of infected agents at time t, an agent in the sampled network is infected at $t \geq 1$ if either

- the agent was infected at the previous stage, that is, $i \in C_{t-1}^{(n)}$; or
- the agent belongs to the *exposed set*, denoted by $\hat{C}_t^{(n)}$, which happens when enough of its neighbors are infected, i.e., $d_i^{(n)}(C_{t-1}^{(n)}) > \tau_i^{(n)} d_i^{(n)}$.

As in the graphon case, we let $C_\infty^{(n)} = \cup_{t=0}^{\infty} C_t^{(n)}$ be the set of infected agents at convergence. Note that in finite networks convergence happens in at most n steps.[5]

4 Convergence results

We now show that with high probability one can infer properties of the infected set of agents in the sampled network ($C_\infty^{(n)}$) from the infected set of labels in the graphon process (C_∞). To this end, we make the following regularity assumption on the graphon, the threshold function, and the seed set.

Assumption 1. *The set C_0 is measurable. The threshold function $\tau(u)$ and the graphon $W(u, v)$ are continuous.*

Assumption 1 is somewhat strong as it rules out natural discontinuities (e.g., in stochastic block models), but it allows us to state our main convergence result succinctly. In Appendix A, we relax Assumption 1 to allow for these discontinuities and state a stronger version of the results presented in the main text. For simplicity of exposition, we separate our subsequent analysis in two parts: we first derive a convergence result about nodes that are *infected* in the continuum process (Theorem 1a) and then a result about nodes that are *not infected* in the continuum process (Theorem 1b). Together these two theorems allow us to derive conclusions about the final outcome of contagion in sampled networks based only on the outcome in the continuum process. Specifically, our first result guarantees that when n is large, almost all agents whose labels are in C_∞ get infected with high probability.

[5] Contagion is a monotone process. If at any time step, no new agent is infected, then no agent will get infected at any subsequent time steps. Hence, the contagion process stops after at most n steps.

Theorem 1a. *Suppose that Assumption 1 holds. For all $\varepsilon, \kappa > 0$, there exists $N^{\varepsilon,\kappa}$ and a measurable set C_∞^ε with $C_\infty^\varepsilon \subset C_\infty$, $\mu\left(C_\infty^\varepsilon\right) \geq \mu\left(C_\infty\right) - \varepsilon$ such that for all $n > N^{\varepsilon,\kappa}$, with probability at least $1-\kappa$, all sampled agents whose label is in C_∞^ε are infected in the sampled network.*

Our second main result shows that, conversely, if a label is not infected in the graphon process (i.e. $u \in [0,1] \backslash C_\infty$), then, for n large enough, with high probability the agent with that label is also not infected in the sampled process. To this end, note that the limiting set $D_\infty := [C_\infty]^c$ of labels that are not infected is a *cohesive* set (Morris, 2000), that is, even if all the agents outside D_∞ are infected, the agents in D_∞ are not. Mathematically, for any $u \in D_\infty$

$$d(u, [D_\infty]^c) - \tau(u) d(u) \leq 0. \tag{4.1}$$

To pin down the set of agents whose labels are not infected, we need a condition that is slightly stronger than (4.1). Let us define $\mathcal{D}$ as the set of all measurable subsets of D_∞ for which (4.1) holds strictly. Formally,

$$\mathcal{D} = \{D \subset D_\infty \mid d(u, D^c) - \tau(u) d(u) < 0 \quad \forall u \in D, \} \tag{4.2}$$

and let $\bar{D}_\infty = \cup_{D \in \mathcal{D}} D$. Note that $\bar{D}_\infty$ may not be measurable, but we can define $\mu_\infty = \sup_{D \in \mathcal{D}} \mu(D)$. We can now state the following theorem.

Theorem 1b. *Suppose that Assumption 1 holds. For all $\varepsilon, \kappa > 0$, there exists $N^{\varepsilon,\kappa}$ and a measurable set $\bar{D}_\infty^\varepsilon$ with $\bar{D}_\infty^\varepsilon \subset \bar{D}_\infty$, $\mu\left(\bar{D}_\infty^\varepsilon\right) \geq \mu_\infty - \varepsilon$ such that for all $n > N^{\varepsilon,\kappa}$, with probability at least $1-\kappa$, all sampled agents whose label is in $\bar{D}_\infty^\varepsilon$ are not infected in the sampled network.*

Figure 3 illustrates the statements of Theorems 1a and 1b. Note that the set of agents whose labels are in $C_\infty \setminus C_\infty^\epsilon$ and $\bar{D}_\infty \setminus \bar{D}_\infty^\epsilon$ for which the theorems are silent can be made arbitrarily small in a large enough sampled network. However, there is potentially a "gray area": the behavior of agents whose label falls into $D_\infty \setminus \bar{D}_\infty$ is not pinned down by the graphon model. To see why, note that these labels are not infected because condition (4.1) holds with equality. Therefore, even in large samples, agents whose labels are in $D_\infty \setminus \bar{D}_\infty$, can tip towards being infected or not infected due to any randomness in the realization of links.

It is important however to make two remarks. First, once C_∞ (and thus D_∞) has been calculated, it is straightforward to verify whether $D_\infty = \bar{D}_\infty$, in which case the graphon analysis applies to all labels except for a measure that vanishes with n. Second, it is possible to derive sufficient conditions for $D_\infty = \bar{D}_\infty$ to hold (as discussed in Appendix A). Putting both theorems together, we can conclude that, as

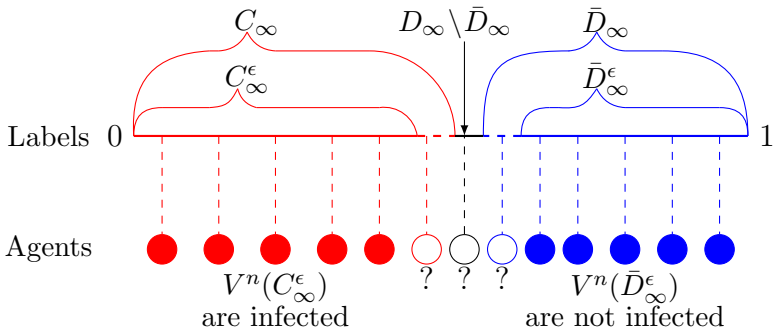


Figure 3: Illustration of Theorems 1a and 1b. The line segment denotes the $[0,1]$ set of labels and the nodes are agents in the sampled networks. Filled nodes are either infected agents (red) or not infected agents (blue). The status of agents with empty nodes (labelled with question marks) is not predicted by Theorems 1a and 1b. $V^n(X)$ is the set of agents in the sampled networks whose label belongs to $X \subseteq [0,1]$.

long as $D_\infty = \bar{D}_\infty$, for any $\varepsilon > 0$ there is N large enough such that approximately $n \times [\mu(C_\infty) \pm \varepsilon]$ agents are infected in any sampled process with $n > N$ agents. In other words, the model of contagion in graphons is a good approximation of network contagion in any large enough sampled network.

The proofs of Theorems 1a and 1b, which use concentration inequalities and the notion of cohesive sets, are given in Appendix C.

5 Optimal seeding problems

Our result in Section 4 shows that the outcome of contagion in a graphon is a good approximation of the outcome of contagion in networks sampled from the graphon. Such convergence result motivates a new approach to intervention design where interventions are designed based on the graphon limit and then applied to the sampled networks (instead of being directly designed for the sampled network, which is a computationally intensive procedure and requires exact network knowledge). This new approach is advantageous if one can easily compute the outcome of graphon contagion and plan optimal interventions for it. We show that this is the case by focusing on two classes of optimal seeding problems for graphons as described next. The tractability of these problems is discussed in Sections 6 and 7 for graphons with finite types and infinite types, respectively.

5.1 Two optimal seeding problems in graphons

To keep the notation simple, from here on, given a graphon $W(u,v)$ and threshold function $\tau(u)$, we denote by $f(C_0)$ the measure of the contagion outcome C_∞ corresponding to the initial seed set $C_0 \subset [0,1]$. We next define two optimal seeding problems in graphons: the max-reach problem and the min-seed problem.

1. **Max-reach:** The first problem we consider is to find the seed set that maximizes final contagion.[6] Suppose that the initial seed can be at most of measure $\rho > 0$. We define the *max-reach contagion problem* as

$$\begin{aligned} f^*_\rho := \sup_{C_0 \subset [0,1]} \quad & f(C_0) \\ \text{s.t.} \quad & \mu(C_0) \leq \rho. \end{aligned} \tag{5.1}$$

   Correspondingly, we say that a set C_0 with $\mu(C_0) \leq \rho$ is an *ε-optimal seed* for the max-reach problem if $f(C_0) \geq f^*_\rho - \varepsilon$. A seed set C_0 is *optimal* for max-reach if it is ε-optimal with $\varepsilon = 0$.

2. **Min-seed:** The second problem we consider is to find the minimum size of the initial seed set that induces a desired level $m \in [0,1]$ of final contagion.[7] We formalize this notion in the *min-seed contagion problem* as follows

$$\begin{aligned} r^*_m := \inf_{C_0 \subset [0,1]} \quad & \mu(C_0) \\ \text{s.t.} \quad & f(C_0) \geq m. \end{aligned} \tag{5.2}$$

   We call a set C_0 *optimal* for min-seed if $f(C_0) \geq m$ and $\mu(C_0) = r^*_m$. We call a set C_0 *limit-optimal* for min-seed if $\mu(C_0) = r^*_m$ and there exists a decreasing nested sequence of sets $(C^k_0)_{k=1}^\infty$ such that $f(C^k_0) \geq m$ for all k and $\cap_k C^k_0 = C_0$. The case $m = 1$ is of special interest as it corresponds to complete contagion. In the following, we call r^*_1 the *resilience* of a contagion process and a corresponding limit-optimal seed a *sensitive infection region*.

While the max-reach and min-seed problems are inspired by corresponding problems studied over finite networks, we note that the graphon model can give rise to new interesting phenomena because contagion in graphons evolves over a continuum of labels.

Example 3. Consider a "hierarchical" graphon in which $W(u,v) = p \times \mathbb{1}_{v<qu}$ for some $q > 0$ (see Figure 4), and $\tau(u) = \tau$ with $\tau q < 1$. In this graphon, lower-indexed labels have a lower degree and higher-indexed labels have a higher degree. Take a seed $C_0 = [0, \varepsilon)$ for some arbitrarily small $\varepsilon > 0$. Note that $d(u) = pqu$ and $d(u, C_0) = p \times \min\{\varepsilon, qu\}$. Hence

$$d(u, C_0) > \tau d(u) \quad \Leftrightarrow \quad \min\{\varepsilon, qu\} > \tau q u \quad \Leftrightarrow \quad u < \frac{\varepsilon}{\tau q}.$$

Consequently, $C_1 = [0, \frac{\varepsilon}{\tau q}) \cap [0,1]$ and iterating contagion we get $C_t = [0, \varepsilon \frac{1}{(\tau q)^t}) \cap [0,1]$

[6] In the context of finite networks, this problem was introduced by Kempe et al. (2003).
[7] In the context of finite networks, this problem was introduced by Long and Wong (2011).

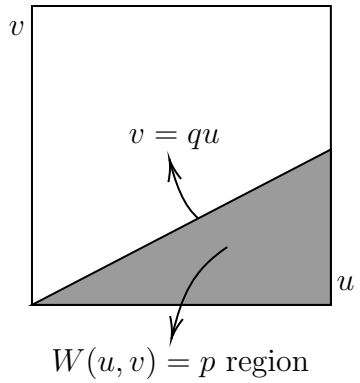


Figure 4: Density plot of the "hierarchical" graphon in Example 3.

leading to $f(C_0) = f([0,\varepsilon)) = [0,1]$. That is, for any $\varepsilon > 0$, the seed $[0,\varepsilon)$ leads to complete contagion. On the other hand, $\{0\} = \cap_\varepsilon [0,\varepsilon)$ does not lead to any contagion because it has zero measure. This example shows that the function f^*_ρ can have a discontinuity at 0: in this case, $f^*_\rho = 1$ for all $\rho > 1$ but $f^*_0 = 0$. Equivalently, this process has zero resilience ($r^*_1 = 0$) and the zero-measure set $\{0\}$ is a sensitive infection region, that is, it is limit-optimal for the min-seed problem with $m = 1$ (this can be verified by setting $C^k_0 = [0, \frac{1}{k})$ for all k.

5.2 Structure of max-reach and min-seed

We conclude this section with a result that describes the general structure of solutions to the max-reach and min-seed problems.

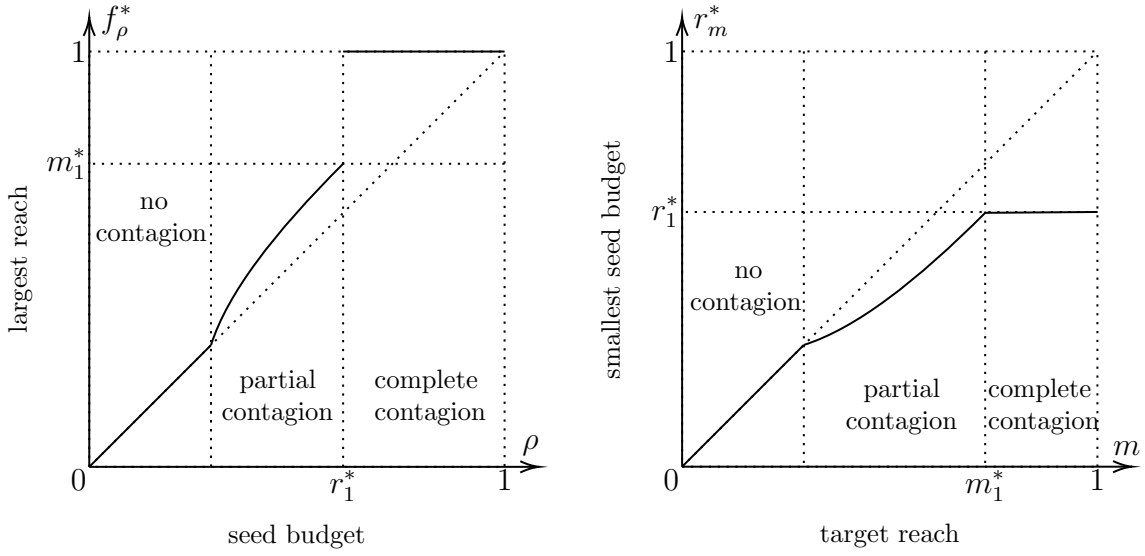
Theorem 2. *The following hold for optimal seeding problems:*

i) $f^*_0 = 0$ *and* $r^*_0 = 0$.

ii) $f^*_\rho - \rho$ *is increasing in* ρ *for* $\rho \in [0, r^*_1)$ *and* $f^*_\rho \equiv 1$ *for* $\rho \in (r^*_1, 1]$.

iii) Let $m^*_1 := \sup_{\rho < r^*_1} f^*_\rho$. *Then* $m - r^*_m$ *is increasing in* m *for* $m \in [0, m^*_1)$ *and* $r^*_m \equiv r^*_1$ *for* $m \in (m^*_1, 1]$.

Theorem 2 makes three intuitive claims. First, when the seed budget is zero, there is no contagion. Second, having a larger budget always increases the possible extent of contagion above and beyond what is added to the budget. Therefore the spread of contagion is supermodular in the initial seed sets when thresholds are deterministic (Jackson and Storms, 2019). Third, complete contagion can be achieved by seeding a set of measures r^*_1. The claims in Theorem 2 are illustrated in Figure 5.



Structure of max-reach Structure of min-seed

Figure 5: Structure of optimal seeding problems (Theorem 2).

6 Contagion in graphons with finite types: Stochastic block models

As we saw in Section 2, in the case of the Erdős-Rényi model, the outcome of contagion in a graphon can easily be computed analytically. In this section, we discuss the computational tractability of graphon contagion for the stochastic block model introduced in Example 1. We show that in stochastic block models one can recover the graphon contagion process exactly from an auxiliary contagion process defined over a finite network with twice as many nodes as the number of communities. Reducing the graphon contagion process to an auxiliary network contagion process is clearly advantageous because the number of communities is typically much smaller than the number of agents in the sampled network. Finally, we show how the reduction to the auxiliary contagion process can help solve the max-reach contagion problem for stochastic block models.

6.1 Contagion dynamics in graphons with finite types

We start our analysis by constructing an auxiliary network. Given a stochastic block model W_{SBM} with k communities $\{\mathcal{I}_h\}_{h=1}^k$ and any seed set C_0, we construct a network with $2k$ nodes. Let i be the index of the nodes in the auxiliary graph, and we distinguish two cases. Each node $i \in \{1, \ldots, k\}$ represents the set of initially uninfected labels in the community $1, \ldots, k$, which has measure $b_i := \mu(\mathcal{I}_i \setminus C_0)$. Each

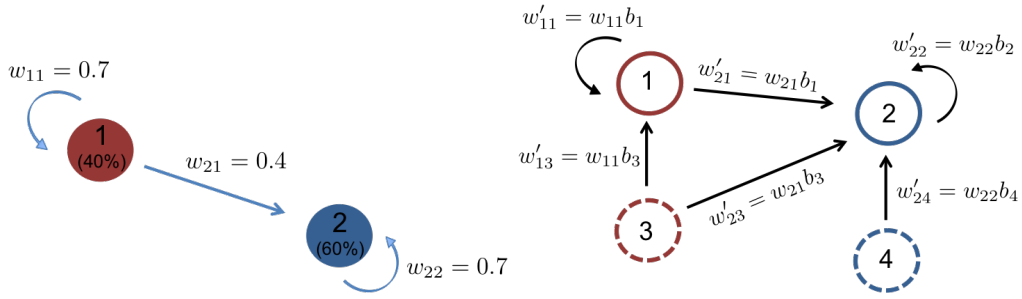


Figure 6: Left: Schematic of the interaction among the two communities in the graphon of Example 1. Nodes indicate communities 1 and 2; node labels show the fraction of agents in each community; labeled arrows represent the probability that an agent from the source community is interacting with an agent from a destination community. Right: Corresponding auxiliary network with seed set $\{3, 4\}$.

node $i \in \{k+1, \ldots, 2k\}$ represents the set of seeds in community $1, \ldots, k$, which has measure $b_i := \mu(\mathcal{I}_{i-k} \cap C_0)$. We then connect nodes in the auxiliary network with weight $w'_{(i,j)} = w_{\{i,j[k]\}}b_j$ (where $j[k] := j$ if $j \leq k$ and $j[k] := j - k$ otherwise). Note that an edge in this graph means that i can be infected by j. Let $A^{\text{aux}} \in \mathbb{R}^{2k \times 2k}$ be the adjacency matrix of this auxiliary network. Figure 6 shows the auxiliary network corresponding to the stochastic block model in Example 1. Armed with this notation, we can state the following result which describes the graphon contagion process for stochastic block models.

Theorem 3. *Consider a graphon process evolving over a stochastic block model W_{SBM} with k communities, seed set C_0 and in which all labels belonging to the same community have the same threshold τ_h for $h \in \{1, \ldots, k\}$. Let C_t^{aux} be the set of infected nodes at time t in the finite contagion process evolving over the auxiliary network $A^{\text{aux}} \in \mathbb{R}^{2k \times 2k}$ corresponding to W_{SBM}, when the initial seed set is $\{k+1, ..., 2k\}$ and the remaining nodes have thresholds $\tau'_i = \tau_i$ for all $i \in \{1, \ldots, k\}$. Then node $i \leq k$ is infected at time t (i.e. $i \in C_t^{\text{aux}}$) in the auxiliary network if and only if the entire block $\mathcal{I}_i$ is infected at time t in the graphon process. Conversely, node $i \leq k$ is not infected on the auxiliary network if and only if $\mathcal{I}_i \setminus C_0$ is not infected.*

Theorem 3 says that in order to know whether a label in a particular community has been infected in a stochastic block model, we only need to know whether the entire community has been infected by looking at the auxiliary network. Note that contagion in the auxiliary network converges in at most k iterations. Hence, an immediate corollary of Theorem 3 is that graphon contagion in the stochastic block model converges in as many iterations as the number of communities. In Figure 7, we present a simulation illustrating the relation between graphon contagion and contagion in sampled networks for a stochastic block model with five communities. As given by Theorems 1a and 1b,

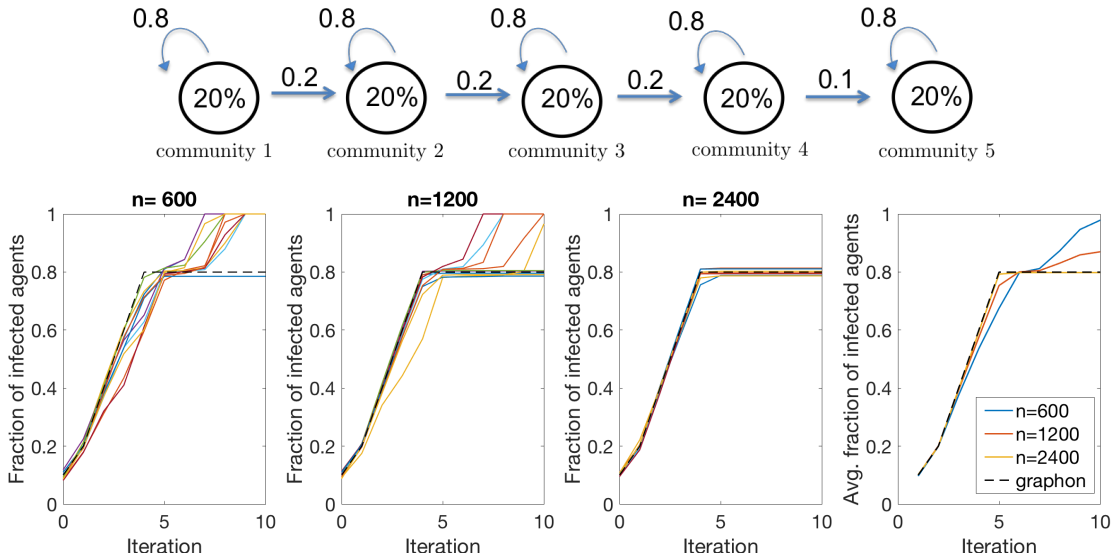


Figure 7: Top: Schematic of the interaction among the five communities of a stochastic block model graphon. Nodes indicate communities; node labels show the fraction of agents in each community; labeled arrows represent the probability an agent from the source community interacting with an agent from a destination community. Bottom: Comparison between contagion in networks with n agents sampled from stochastic block model and contagion in the corresponding graphon for different values of n. Three plots on the bottom left: the evolution of the fraction of infected individuals in 10 repetitions for each value of n. Bottom right plot: the average evolution compared with the graphon. We set $\tau = 0.16$ and initial seed set $C_0 = [0, 0.1]$ corresponding to seeding half of the labels in community 1.

when sampled networks have a large number of agents, contagion in sampled networks closely approximates contagion in a graphon.

6.2 Optimal seeding in stochastic block models

We can now combine insights from Theorems 1a, 1b and 3 in order to analyze the max-reach contagion problem (introduced in Section 5) for stochastic block models. Using Theorem 3, we can reformulate the max-reach contagion problem as an optimization problem in k variables. Specifically, for any initial seed C_0, let $c_i := \mu(C_0 \cap \mathcal{I}_i)$ denote the measure of labels in block i that belong to the initial seed. Recalling that π_i is the fraction of labels that belongs to community i, the vector b (defined in Section 6.1) has components

$$b_i(c) = \begin{cases} \pi_i - c_i & \text{if } i = 1, \dots, k \\ c_i & \text{if } i = k+1, \dots, 2k. \end{cases} \tag{6.1}$$

Let $A^{\text{aux}}(c) \in \mathbb{R}^{2k \times 2k}$ be the adjacency matrix of the auxiliary network obtained for $b(c)$ as in (6.1) and for each label i let $f_i(c)$ be equal to one if label i is infected at the end of contagion over the network with the auxiliary matrix $A^{\text{aux}}(c) \in \mathbb{R}^{2k \times 2k}$ and

initial seed set $\{k+1,\ldots,2k\}$. It then follows from Theorem 3 that

$$f_\rho^* = \max_{c\in[0,1]^k|\sum_i c_i=\rho} \sum_{i=1}^{k}(\pi_i - c_i)f_i(c) + \rho. \tag{6.2}$$

In other words one only needs to decide which portion c_i of the budget to allocate to each community i, which is a k dimensional optimization problem. Let the vector c^* be the maximizer of (6.2). Then any subset C_0^* of $[0,1]$ such that $\mu(C_0^* \cap \mathcal{I}_i) = c_i^*$ is an optimal seed for max-reach problem. Using Theorems 1a and 1b, the solution to the max-reach contagion problem translates into the following graphon-based seeding policy for maximizing contagion in sampled networks: the planner should seed $n \times c_i^*$ randomly selected agents for each community i.

Our seeding policy based on contagion in graphons has two advantages over solving the original seeding problem in the sampled network. First, solving the optimal seeding problem in the sampled network requires knowledge of the realized sampled network, which may be unavailable to the planner. Instead the solution of max-reach problem (6.2) for the stochastic block model is only a function of the relevant parameters of the network formation model (i.e., the connectivity between different communities). Second, solving the optimal seeding problem in the sampled network requires the selection of a set of $n \times \rho$ agents from a possible set of n agents. This is a computationally intractable problem for large n (Kempe et al., 2003). On the other hand, a solution to max-reach problem (6.2) for the stochastic block model requires finding the optimal weight of the edges in the auxiliary network $A^{\text{aux}}(c)$ and is thus a problem in k instead of n variables. Since typically $k \lll n$, even brute force approaches to solving the max-reach problem reformulation (6.2) are computationally tractable.

6.3 Numerical illustration

To illustrate the seeding policy suggested by the graphon model, we solved the max-reach problem (6.2) for the stochastic block model in Figure 7 with a budget $\rho = 0.06$. In this case we obtained $c_1^* = 0.04, c_2^* = c_3^* = 0, c_4^* = 0.02$ as the optimal solution, meaning that if only 6% of the agents can be seeded then for the graphon model it is optimal to seed 4% of the agents in community 1 and 2% of the agents in community 4. Figure 8 (left) shows the performance of this policy when applied to networks sampled from the stochastic block model in Figure 7. In the numerical experiment, for each random network with n nodes, we sampled 300 networks. For each of these sampled networks, we selected as seed $0.04n$ agents at random from community 1 and $0.02n$ agents at random from community 4 and then computed the fraction of infected agents at the end of the contagion process. The solid magenta line

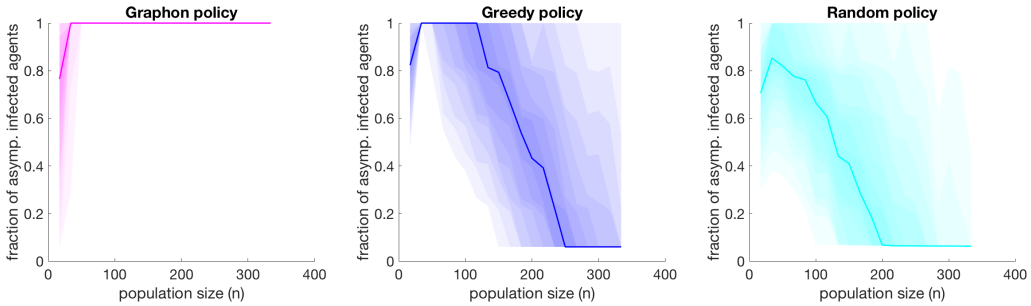


Figure 8: Fraction of asymptotically infected agents as a function of population size when the seed set is designed according to the graphon-based seeding policy (left, magenta), a greedy policy (middle, blue) or random seeding (right, cyan). For each population size n we constructed 300 sampled networks, thus obtaining a distribution of the fraction of asymptotically infected agents. We here report the median of such distribution (solid line) and the quantiles as the shaded region.

shows the median over 300 repetitions. For comparison, the middle and right plots show the median of the same quantity when a greedy and random policy is used. In the greedy policy, suggested by Kempe et al. (2003), the seed set is constructed by iteratively adding one agent at a time (up to $0.06n$ agents) and at each time selecting the agent that has the highest marginal impact on the contagion outcome. In the random policy, $0.06n$ seeds are selected uniformly at random. In all plots, the shaded area shows different quantiles of the corresponding distribution. Note that no shaded magenta area is visible for $n > 100$ because the distribution under the graphon policy is tightly concentrated around one, showing that the seeding policy suggested by the graphon model is optimal for this example. This is clearly not the case for the greedy and random policies, whose performance decreases sharply with n.[8]

7 Contagion in graphons with infinite types

Stochastic block models involve a finite number of types. Consequently, the optimal seeding problem can be reduced to finding the fraction of each type that needs to be seeded, as discussed in Section 6.1. A continuum of types is a more natural assumption in many contexts, such as spatial location, income and wealth, or political views. These continuous characteristics can determine the intensity of interactions in a variety of settings. For the sake of tractability, in the following we focus on a special class of such infinite type models that give rise to interval contagion, as formalized next.

Definition 2. (W, τ) *admits interval contagion* if C_1 is an interval for any interval seed set C_0. (W, τ, C_0) is an *interval contagion process* if (W, τ) admits interval contagion

[8] This is not a contradiction with Kempe et al. (2003) since their results are derived for contagion processes where agents' thresholds are sampled uniformly at random, while here we consider a homogeneous threshold $\tau = 0.16$. See Appendix D for the analysis of graphon contagion with uniform random thresholds.

and C_0 is an interval.

In words, in an interval contagion process, the seed set is an interval of labels and the set of infected labels at any step is also an interval. In light of our interpretation of a continuum of types/labels, seeding an interval corresponds to targeting agents with similar characteristics. This can be seen as a social planner's preference against complexity. The property that any infected interval weakly grows into a new interval (as opposed to generating disconnected infected sets) reflects the presence of homophily: it is easier for labels to infect labels that are closer (i.e, have a similar location, socioeconomic status or ideology). In the remainder of this section, we provide further details on the interval contagion process and its link to homophily, we formalize optimal seeding problems for interval contagion, and we illustrate our results in the context of specific graphons commonly used in the literature. In Appendix B we discuss interval contagion in more detail, characterize general solutions to optimal seeding problems under relatively weak conditions, and revisit the illustrative examples presented in this section.

7.1 Interval contagion in graphons

The following is an immediate necessary and sufficient condition for (W, τ) to admit interval contagion.

Assumption 2. *There exist* cutoff iterators $\alpha : [0,1]^2 \to [0,1]$ *and* $\beta : [0,1]^2 \to [0,1]$ *such that for all* $(a,b) \subset (0,1)$*, for all* $u \in (0,a]$ *and* $v \in [b,1)$*,*

$$d(u,(a,b)) > \tau(u)d(u) \iff u > \alpha(a,b)$$
$$d(v,(a,b)) > \tau(v)d(v) \iff v < \beta(a,b).$$

The idea behind such cutoff iterators is illustrated in Figure 9. Consider an interval (a_0, b_0) of infected labels and a label $u \leq a_0$ that is "to the left" of a_0. The existence of cutoff functions entails that $u \leq a$ becomes infected by (a_0, b_0) if and only if $u > \alpha(a_0, b_0)$. Analogously, label $u \geq b_0$ "to the right" of b_0 gets infected by (a_0, b_0) whenever $u < \beta(a_0, b_0)$.[9] As a result, in the first step of the contagion process a label u is infected if and only if $a_1 < u < b_1$, resulting in C_1 being an interval.

Next, we formalize the link of interval contagion to homophily. Let $d^*(u) \equiv \tau(u)d(u)$. We focus on (W, τ) such that d^* is bounded away from zero.[10]

[9] Lemma 5 in Appendix B shows that $\alpha(a,b)$ is increasing, right-continuous in a and decreasing in b, while $\beta(a,b)$ is increasing, left-continuous in b and decreasing in a.

[10] If $\tau(u) = 0$, u gets infected regardless and so u can be included into the seed. If $d(u) = 0$, the label u is isolated and irrelevant. As a result, it is innocuous to assume that d^* is nonzero.

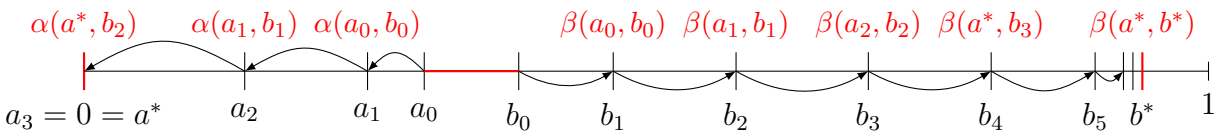


Figure 9: Interval contagion process. $C_0 = (a_0, b_0)$ is the seed set. Cutoff iterators α and β take the contagion process to the final infected set (a^*, b^*) in steps indicated by the arrows, so that $C_1 = (a_1, b_1)$, $C_2 = (a_2, b_2)$, etc.

Definition 3. We say that a graphon W *admits homophily* if for all v, $W(u, v)$ is increasing in $u \in [0, v]$ and decreasing in $u \in [v, 1]$. We say that (W, τ) admits *normalized homophily* if $W^*(u, v) \equiv \frac{W(u,v)}{d^*(u)} \inf(d^*)$ admits homophily.

It is clear that if (W, τ) admits normalized homophily, then Assumption 2 holds. Moreover, the next example shows that when d and τ are constant, homophily suffices for Assumption 2 to hold.

Example 4. (Regular homophilic graphons) Suppose that W admits homophily and $d(u) \equiv d$ and $\tau(u) \equiv \tau$ are constants. Then, by homophily, $d(u, (a, b))$ is increasing on $u \leq a$ and decreasing on $v \geq b$. Therefore cutoff iterators exist, and are given by $d(\alpha(a, b), (a, b)) = \tau d$ (or $\alpha(a, b) = 0$ if $d(0, (a, b)) > \tau d$) and $d(\beta(a, b), (a, b)) = \tau d$ (or $\beta(a, b) = 1$ if $d(1, (a, b)) > \tau d$).

In Appendix B, we provide a condition on the cutoff iterators (called the *single-crossing property*) that guarantees that the interval contagion process on the graphon is particularly tractable. Intuitively, such single-crossing property guarantees that for any fixed right extreme b of the seed set $[a, b]$, the contagion behavior depends in a monotone way on the left extreme a and vice versa for any fixed left extreme a contagion is monotone in b. This is particularly useful because, under this assumption, Proposition 3 in Appendix B proves that the outcome of contagion can only take one of four forms

- No contagion: the final infected set is the same seed set;
- Right-only contagion: the final infected set is $[a_0, 1)$;
- Left-only contagion: the final infected set is $(0, b_0]$;
- Complete contagion: the final infected set is $(0, 1)$ (either starting "to the right" or "to the left").

Under the single-crossing property we are able to analytically tackle max-reach and min-seed problems in a variety of graphons, under the additional constraint that the initial seed is an interval, as formalized next.

Interval max-reach problem For interval contagion, the max-reach problem (5.1) can be reformulated as the *interval max-reach* problem with ρ

$$\tilde{f}^*_\rho := \sup_{a_0 \in [0,1]} \quad f([a_0, a_0 + \rho]), \tag{7.1}$$

where the only optimization variable is the left extreme of the seed set.

Interval min-seed problem For interval contagion, the min-seed problem (5.2) can be reformulated into the *interval min-seed problem* with $m > 0$ in the following way:

$$\begin{aligned} \tilde{r}^*_m := \inf_{a_0, b_0 \in [0,1]^2} \quad & b_0 - a_0 \\ \text{s.t.} \quad & f([a_0, b_0]) \geq m \\ & a_0 \leq b_0, \end{aligned} \tag{7.2}$$

where the only optimization variables are the extremes a_0, b_0 of the seed interval set.

We provide explicit solutions to the interval max-reach and interval min-seed problems for general graphons satisfying the single-crossing property in Appendix B, we next discuss the application of such results to some illustrative examples.

7.2 Analytical examples

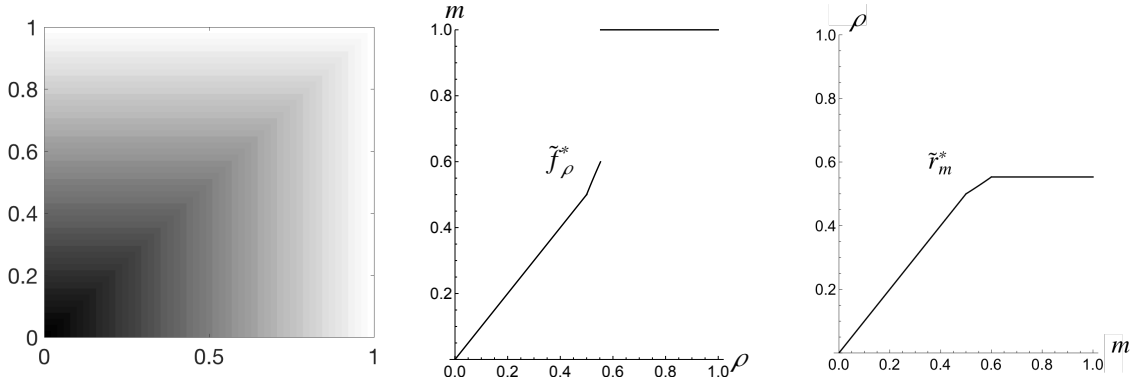
Example 5 (Growing Uniform Attachment (Borgs et al., 2011)). Consider the following network formation process: In each period, a new node arrives, and then every pair of non-adjacent nodes is connected with probability $\frac{1}{n}$. This network can be sampled from the graphon $W_{GUA}(u, v) = 1 - \max\{u, v\}$, illustrated in Figure 10(i).

In this graphon, any label v is most connected to labels less than or equal to v and labels sufficiently close to v are highly connected to v, in line with our homophily interpretation. Lower labels are more connected to all labels, but especially to other lower-label labels, whereas the reverse holds for higher labels. Thus this graphon represents a hierarchical structure. A bit of tedious algebra shows that $d(u) = \frac{1}{2}(1-u^2)$. Pick a constant τ and observe that

$$\frac{W(u, v)}{d^*(u)} = \left(\frac{2}{\tau}\right)\left(\frac{1 - \max\{u, v\}}{1 - u^2}\right)$$

is single-peaked as a function of u, with the peak at v. So (W_{GUA}, τ) admits normalized homophily, and hence interval contagion.

Corollary 1. *Suppose that $W = W_{GUA}$ and $\tau(u) = \tau$. Cutoff iterators exist. The*



(i) Density plot (ii) Max-reach (iii) Min-seed

Figure 10: Growing Uniform Attachment (GUA) graphon (Borgs et al., 2011). Left: density plot for the GUA graphon. Middle: solution to the interval max-reach problem (7.1). Right: solution to the interval min-seed problem (7.2). Thresholds are set at $\tau(u) = \tau = 0.75$.

solution to the interval min-seed problem is:

$$\tilde{r}^*_m = \begin{cases} \frac{\tau}{2-\tau} - \frac{1-\sqrt{\tau^2+(1+\tau)(1-\frac{\tau}{2-\tau})^2}}{1+\tau} & \text{if } m > \frac{\tau}{2-\tau} \\ m - \frac{1-\sqrt{\tau^2+(1+\tau)(1-m)^2}}{1+\tau} & \text{if } \frac{\tau}{2-\tau} > m > 1-\sqrt{1-\tau} \\ m & \text{if } 1-\sqrt{1-\tau} > m \end{cases} .$$

The following set is limit-optimal for the interval min-seed problem:

$$C^*_0 = \begin{cases} \left[\frac{\tau}{2-\tau} - \tilde{r}^*_m, \frac{\tau}{2-\tau}\right] & \text{if } m > \frac{\tau}{2-\tau} \\ [m - \tilde{r}^*_m, m] & \text{if } \frac{\tau}{2-\tau} > m > 1-\sqrt{1-\tau} \\ [0, m] & \text{if } 1-\sqrt{1-\tau} > m \end{cases} .$$

Figure 10(ii) shows the solution to the max-reach problem and Figure 10(iii) shows the solution to the min-seed problem for $\tau = 0.75$. Figure 11 shows a comparison of seeding in sampled networks based on the graphon analysis versus greedy or random. For this illustration we fixed $\tau = 0.75$ and selected a seed budget of $\rho = \tilde{r}^*_1$. The optimal seed set in the graphon is therefore $C^*_0 = \left[\frac{\tau}{2-\tau} - \tilde{r}^*_1, \frac{\tau}{2-\tau}\right]$ and leads to complete contagion. In the graphon policy, to compute the seed set in a sampled network we seeded all agents with $u_i^{(n)} \in C^*_0$. Note that the size of the seed set is random (asymptotically converging to ρn). To have a fair comparison we set the random and greedy seed sets to have exactly the same number of agents as the graphon seed set in each realization. □

We now return to the Location Model described in Example 2.

Example 6 (Location Model (Parise and Ozdaglar, 2022)). The Location Model can be represented by the graphon $W_{LOC}(u, v) = (\min\{u, v\})(1 - \max\{u, v\})$, illustrated

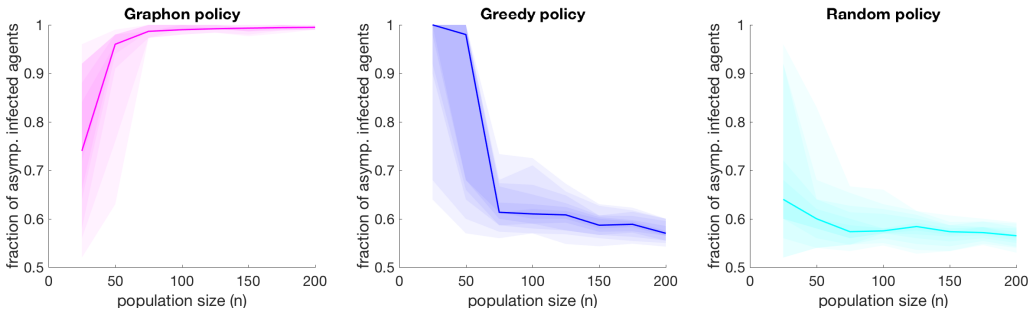


Figure 11: Growing Uniform Attachment (Borgs et al., 2011). Fraction of asymptotically infected agents as a function of population size when the seed set is designed according to the graphon-based seeding policy (left, magenta), a greedy policy (middle, blue) or random seeding (right, cyan). For each population size n we constructed 50 sampled networks, thus obtaining a distribution of fraction of asymptotically infected agents. We here report the median of such distribution (solid line) and the quantiles (from 0.2 to 0.8) as shaded region.

in Figure 12(i).

Some algebra shows $d(u) = \frac{1}{2}u(1-u)$. Pick constant τ and observe that

$$\frac{W(u,v)}{d^*(u)} = \left(\frac{2}{\tau}\right)\left(\frac{\min\{u,v\}(1-\max\{u,v\})}{u(1-u)}\right)$$

is single-peaked in u, with the peak at v. So (W_{LOC}, τ) admits normalized homophily and hence interval contagion.

Corollary 2. *Suppose that $W = W_{LOC}$ and $\tau(u) = \tau$. Cutoff iterators exist. The solution to the interval min-seed problem is:*

$$\tilde{r}^*_m = \begin{cases} \frac{1}{2}\left(\tau + \sqrt{\tau^2 + 4(1-\tau)^2}\right) - (1-\tau) & \text{if } m > \tau \\ \frac{1}{2}\left(\tau + \sqrt{\tau^2 + 4(1-m)^2}\right) - (1-m) & \text{if } \tau > m > 1 - \sqrt{1-\tau} \\ m & \text{if } 1 - \sqrt{1-\tau} > m \end{cases}.$$

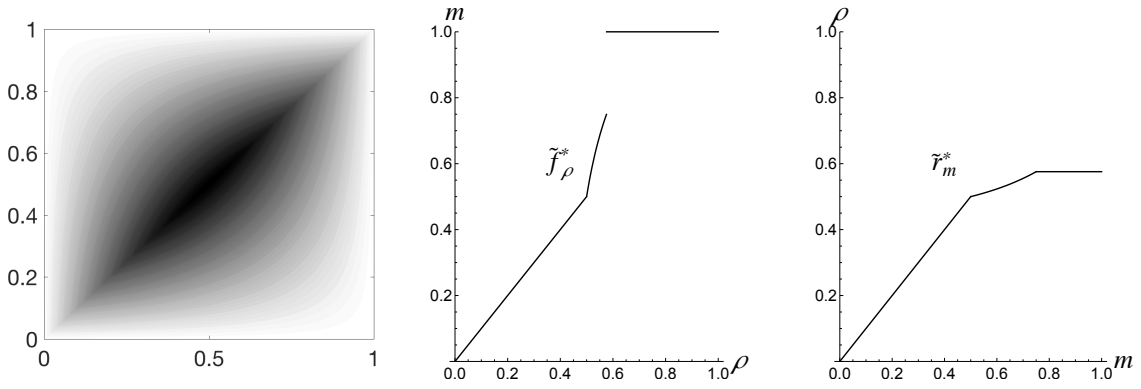
The following is limit-optimal for interval min-seed problem:

$$C^*_0 = \begin{cases} [\tau - \tilde{r}^*_m, \tau] & \text{if } m > \tau \\ [m - \tilde{r}^*_m, m] & \text{if } \frac{\tau}{2-\tau} > m > 1 - \sqrt{1-\tau} \\ [0, m] & \text{if } 1 - \sqrt{1-\tau} > m \end{cases}.$$

or the symmetric intervals around 0.5.

Figure 12(ii) shows the solution to the max-reach problem and Figure 12(iii) shows the solution to the min-seed problem for $\tau = 0.75$. Figure 13 shows a comparison of seeding in sampled networks based on the graphon analysis versus greedy or random.

□



(i) Density plot (ii) Max-reach (iii) Min-seed

Figure 12: Location Model (Parise and Ozdaglar, 2022). Left: density plot for the GUA graphon. Middle: solution to the interval max-reach problem (7.1). Right: solution to the interval min-seed problem (7.2). Thresholds are set at $\tau(u) = \tau = 0.75$.

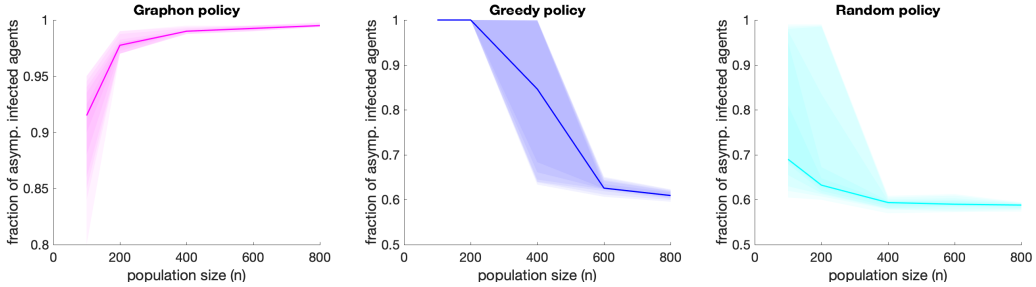


Figure 13: Location Model (Parise and Ozdaglar, 2022). Fraction of asymptotically infected agents as a function of population size when the seed set is designed according to the graphon-based seeding policy (left, magenta), a greedy policy (middle, blue) or random seeding (right, cyan) for the budget $\frac{1}{2}\left(\tau + \sqrt{\tau^2 + 4(1-\tau)^2}\right) - (1-\tau)$. For each population size n we constructed 20 sampled networks, thus obtaining a distribution of the fraction of asymptotically infected agents. We here report the median of such distribution (solid line) and the quantiles (from 0.2 to 0.8) as a shaded region.

8 Conclusion

In this paper, we introduced a model of threshold contagion in graphons. Our key result showed that contagion in a graphon closely approximates contagion in sampled graphs. This suggests a new procedure for intervention design where the seed set is evaluated for the graphon process and then applied to sampled networks. We showed that calculating the optimal seed set for the graphon model is often analytically tractable, overcoming the computational intractability of intervention design for large sampled networks.

We can see several directions for further work. First, one could analyze other contagion processes in graphons, such as the independent cascade model due to Kempe et al. (2003) or the classic SIR model used extensively for disease spread (e.g., Acemoglu et al. (2021)). Second, it is worth better understanding under what conditions

arbitrarily small seed sets—sensitive infection points—can result in a positive fraction of the labels being infected. Third, since our results imply that contagion in graphons well approximates every step of the contagion process, the graphon model could be used to study the speed of contagion (e.g., Koh and Morris (2022)). Finally, we hope that our model could serve as a useful starting point for richer economic models, such as network formation in the presence of shocks (e.g., Erol (2019)).

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APPENDIX

A Convergence results

In this section we consider a generalization of the convergence results presented in Theorems 1a and 1b that hold under the following less restrictive assumption.

Assumption 3. *We assume the following:*

- *The set C_0 is measurable.*
- *The threshold function $\tau(u)$ is piece-wise continuous, $u \mapsto W(u,v)$ is piece-wise continuous for almost all v (i.e., there is a set D_v with $\mu(D_v) = 0$ such that $u \mapsto W(u,v)$ is piece-wise continuous in u for any $v \in [0,1] \backslash D_v$) and the set of discontinuities, $D_u := \{u \mid \exists v \in [0,1]\backslash D_v \text{ s.t. } W(u,v) \text{ or } \tau(u) \text{ are not continuous in } u\}$, has measure zero.*
- *The set $[0,1]\backslash D_u$ is open.*

Clearly, Assumption 3 implies Assumption 1. To compactly state our following results for any set of labels $X \subset [0,1]$ we define $V^{(n)}(X) := \{i \in [n] \mid u_i^{(n)} \in X\}$ to be the set of (sampled) agents whose label is in the set $X \subset [0,1]$.

A.1 Infected labels and infected agents

Consider the set C_∞ of labels that are infected in graphon contagion. Our first result is that "almost all" sampled agents whose labels are in C_∞ will be infected in any sampled network with large enough population.

Theorem 1a′. *Suppose that Assumption 3 holds. For all $\varepsilon, \kappa > 0$, there exists $N^{\varepsilon,\kappa}$ and a measurable set C_∞^ε with $C_\infty^\varepsilon \subset C_\infty$, $\mu(C_\infty^\varepsilon) \geq \mu(C_\infty) - \varepsilon$ such that for all $n > N^{\varepsilon,\kappa}$, with probability at least $1-\kappa$*

$$V^{(n)}(C_\infty^\varepsilon) \subset C_\infty^{(n)},$$

that is, agents whose labels are in C_∞^ε will be infected in the sampled network.

To prove Theorem 1a′, we show that the graphon model is a good approximation of both the infected set and the exposed set of agents *at every step* t of the contagion process. Theorem 1a′ follows immediately as a consequence of the following two propositions.

Proposition 1. *(Infected set at time t) Suppose that Assumption 3 holds. For all $t \geq 0$ and $\varepsilon_t, \kappa_t > 0$, there exists $N_t^{\varepsilon_t,\kappa_t}$ and a measurable set $C_t^{\varepsilon_t}$, with $C_t^{\varepsilon_t} \subset C_t$, $\mu(C_t^{\varepsilon_t}) \geq \mu(C_t) - \varepsilon_t$ such that for all $n > N_t^{\varepsilon_t,\kappa_t}$, with probability at least $1-\kappa_t$*

$$V^{(n)}(C_t^{\varepsilon_t}) \subset C_t^{(n)}.$$

Proposition 2. *(Exposed set at time t) Suppose that Assumption 3 holds. For all $t \geq 1$ and $\varepsilon_t, \kappa_t > 0$, there exists $\hat{N}_t^{\varepsilon_t,\kappa_t}$ and a measurable set $\hat{C}_t^{\varepsilon_t}$, with $\hat{C}_t^{\varepsilon_t} \subset \hat{C}_t$, $\mu\left(\hat{C}_t^{\varepsilon_t}\right) \geq \mu\left(\hat{C}_t\right) - \varepsilon_t$ such that for all $n > \hat{N}_t^{\varepsilon_t,\kappa_t}$, with probability at least $1-\kappa_t$*

$$V^{(n)}\left(\hat{C}_t^{\varepsilon_t}\right) \subset \hat{C}_t^{(n)}.$$

In words, Propositions 1 and 2 say that if a label is infected (resp. exposed) at time t in the graphon process then, for large n, at time t the agent with that label is infected (resp. exposed) with high probability in the sampled process. We prove Propositions 1 and 2 jointly by induction in Appendix C). To derive some intuition, note that Proposition 1 holds at time $t = 0$ by construction. The main step in our proof is to show that if Proposition 1 holds for time $t-1$ then Proposition 2 holds for time t. Note that the statements in Propositions 1 and 2 hold for almost all labels except a set of measure ε, which can be made as small as necessary by increasing n. To understand why we need to exclude a set of measure ε consider Proposition 2. In order to determine whether an agent with label $u_i^{(n)}$ who is exposed at time t in the graphon process, i.e., $u_i^{(n)} \in \hat{C}_t$, is also exposed in the sampled process we need to determine whether

$$d_i^{(n)}(C_{t-1}^{(n)}) - \tau_i^{(n)} d_i^{(n)} > 0.$$

For a given label $u_i^{(n)}$, we know that

$$d_i^{(n)}(C_{t-1}^{(n)}) = \sum_{j \in C_{t-1}^{(n)}} A_{ij}^{(n)} \quad \text{and} \quad d_i^{(n)} := \sum_j A_{ij}^{(n)}$$

are the sum of random variables $A_{ij}^{(n)} = \text{Ber}(W(u_i^{(n)}, u_j^{(n)}))$ induced by $u_j^{(n)}$. Our main argument is to use concentration inequalities to show that, for large n, the two sums accumulate so that

$$\frac{d_i^{(n)}(C_{t-1}^{(n)})}{n} \approx d(u_i^{(n)}, C_{t-1}) \quad \text{and} \quad \frac{d_i^{(n)}}{n} \approx d(u_i^{(n)}),$$

where the approximation improves for a large n. Hence, we have that

$$T^{(n)} := d_i^{(n)}(C_{t-1}^{(n)}) - \tau_i^{(n)} d_i^{(n)} \approx n(d(u_i^{(n)}, C_{t-1}) - \tau(u_i^{(n)}) d(u_i^{(n)})) =: nT.$$

Since $u_i^{(n)} \in \hat{C}_t$, T is strictly positive, but could be arbitrarily close to zero. By removing a set of measure ε from $\hat{C}_t$ we make sure that T is bounded away from zero by a positive quantity so that the perturbed term $T^{(n)}$—which accumulates around T—is positive for n large enough.

A.2 Labels and agents who are not infected

Next, we consider labels that belong to the set $\bar{D}_\infty$. Under the more general assumption 3, Theorem 1b can be reformulated as follows.

Theorem 1b′. *Suppose that Assumption 3 holds. For all $\varepsilon, \kappa > 0$, there exists $N^{\varepsilon,\kappa}$ and a measurable set D_∞^ε with $D_\infty^\varepsilon \subset \bar{D}_\infty$, $\mu(D_\infty^\varepsilon) \geq \mu(\bar{D}_\infty) - \varepsilon$ such that for all $n > N^{\varepsilon,\kappa}$, with probability at least $1 - \kappa$*

$$V^{(n)}(D_\infty^\varepsilon) \subset D_\infty^{(n)},$$

that is, agents whose labels are in D_∞^ε will not be infected in the sampled network.

We conclude this section by noting that if $D_\infty \setminus \bar{D}_\infty$ does not have zero measure, then we need to be careful about indices that fall in $D_\infty \setminus \bar{D}_\infty$. Indeed, on the graphon,

C_∞ gets infected and D_∞ does not. When one samples from the graphon, the points on $D_\infty \setminus \bar{D}_\infty$ can behave erratically. We next derive a sufficient condition for $D_\infty \setminus \bar{D}_\infty$ to have zero measure.

Remark 1. Suppose that there exists $\ell > 0$ such that for any $u \in D_\infty$, we have that

$$d(u, [D_\infty]^c) - \tau(u)d(u) < -\ell, \tag{A.1}$$

then $D_\infty = \bar{D}_\infty$.

We also note that once C_∞ has been calculated, it is straightforward to verify whether $\mu(D_\infty \setminus \bar{D}_\infty) = 0$ or the stronger condition (A.1) in Remark 1 holds, as the following example shows.

Example 7. Let us consider again the Erdős-Rényi model with connection probability p and constant threshold function $\tau(u) = \tau$ discussed in Section 2. Fix any seed set C_0 of measure ρ. We next show that the assumption $\mu(D_\infty \setminus \bar{D}_\infty) = 0$ is generic in the sense that it holds for almost all thresholds τ. Recall that we have two possible contagion outcomes:

1. if $\rho - \tau > 0$, then $C_\infty = [0, 1]$ (all labels are infected);
2. if $\rho - \tau \leq 0$, then $C_\infty = C_0$ (no new label is infected).

Condition $\mu(D_\infty \setminus \bar{D}_\infty) = 0$ trivially holds in the first case since $D_\infty = \bar{D}_\infty = \emptyset$. We focus on the second case and show that $\mu(D_\infty \setminus \bar{D}_\infty) = 0$ holds whenever $\rho - \tau < 0$. In the second case, we have that $[D_\infty]^c = C_\infty = C_0$, hence

$$d(u, [D_\infty]^c) - \tau d(u) = p\rho - \tau p = p(\rho - \tau) < 0$$

for all $u \in D_\infty$ and condition (4.2) is met with $\bar{D}_\infty = D_\infty$ generically for all $\tau \neq \rho$.

Therefore, we cannot apply Theorems 1a′ and 1b′ for this example only in the non-generic case when $\tau = \rho$. In that case, $\mu(D_\infty \setminus \bar{D}_\infty) = \mu(D_\infty)$. To see this, note that for $\tau = \rho$ and for any $\bar{D}_\infty \subset D_\infty$, it must be $\mu([\bar{D}_\infty]^c) \geq \mu([D_\infty]^c) = \mu(C_\infty) = \rho$. Hence the left hand side of condition (4.2) is greater or equal to $p(\rho - \tau) = 0$ and condition (4.2) cannot hold. Hence, when $\tau = \rho$, we have that $\bar{D}_\infty = \emptyset$.

B Contagion in graphons with infinite types

B.1 Interval contagion

Under Assumption 2, if at time step t the infected set is the interval (a_t, b_t), then at $t + 1$ the infected set becomes the interval (a_{t+1}, b_{t+1}) where

$$a_{t+1} = \min\{a_t, \alpha(a_t, b_t)\} \leq a_t, \tag{B.1}$$

$$b_{t+1} = \max\{b_t, \beta(a_t, b_t)\} \geq b_t. \tag{B.2}$$

The existence of cutoff functions thus guarantees that contagion can be succinctly summarized (using (B.1) and (B.2)) by a growing interval described by the two *contagion sequences* $(a_t)_{t\geq 0}$ and $(b_t)_{t\geq 0}$ that start from a_0 and b_0 and that correspond to the left and right endpoint of the infected interval at time t. Note that by construction, $(a_t)_{t\geq 0}$ is a (weakly) decreasing and bounded sequence and $(b_t)_{t\geq 0}$ is a (weakly) increasing and

bounded sequence.[11] Let the limit of these sequences be a^* and b^*. The outcome of contagion is then given by (a^*, b^*). In Figure 9, after interval (a_1, b_1) is infected the values of the cutoff iterators change to $\alpha(a_1, b_1)$ and $\beta(a_1, b_1)$ and in the next step all labels between $a_2 = \alpha(a_1, b_1)$ and $b_2 = \beta(a_1, b_1)$ are infected. The limit of the contagion sequence in the example is $(a^*, b^*) = [0, b^*)$.

Our goal is to characterize the interval of infected labels (a^*, b^*) in terms of the interval of initial seeds (a_0, b_0). To this end, we focus on a particularly tractable set of cutoff iterators that satisfy the following assumption.

Assumption 4. *(Single-crossing property) The cutoff iterators satisfy the single-crossing property if:*

- *for all $b \in [0,1]$, there exists a crossing function $\alpha^\dagger(b) \in [0,1]$ such that for all $a \in (0, b)$ [if $a < \alpha^\dagger(b)$: $\alpha(a,b) < a$] and [if $a > \alpha^\dagger(b)$: $\alpha(a,b) > a$],*
- *for all $a \in [0,1]$, there exists a crossing function $\beta^\dagger(a) \in [0,1]$ such that for all $b \in (a, 1)$ [if $b < \beta^\dagger(a)$: $\beta(a,b) < b$] and [if $b > \beta^\dagger(a)$: $\beta(a,b) > b$].*

Assumption 4 is also quite natural and simply says that for any fixed right extreme b of the seed set, the contagion behavior depends in a monotone way on the left extreme a. Specifically, there is a threshold $\alpha^\dagger(b)$ such that if the left extreme is smaller than $\alpha^\dagger(b)$ the infection propagates ($\alpha(a,b) < a$), while otherwise it stops ($\alpha(a,b) > a$). Similar arguments hold in terms of b for fixed a. We show in Lemma 8 in Appendix B that $\alpha^\dagger$ and $\beta^\dagger$ are both increasing. Therein, we also prove that both the examples of graphons discussed in Section 7.2 satisfy Assumptions 2 and 4.

Figure 14 illustrates cutoff iterators that satisfy the single-crossing property and shows that, under Assumptions 2 and 4, when one extreme is fixed contagion evolves in a simple way. Focusing on the case when b is fixed, it is possible to show that either contagion stops immediately or it spreads until $a^* = 0$. Similarly, when a is fixed contagion either stops immediately or it spreads until $b^* = 1$. During the actual contagion process both ends of the interval change simultaneously, still it is possible to show that only four outcomes are possible, as proven next.

Proposition 3. *Suppose that given a graphon $W(u,v)$ and threshold function $\tau(u)$, the cutoff iterators α and β exist and satisfy the single-crossing property. Then, given an initial seed (a_0, b_0), the outcome of contagion (a^*, b^*) is given by one of the following modes:*

1. *No contagion: If $a_0 \geq \alpha^\dagger(b_0)$ and $b_0 \leq \beta^\dagger(a_0)$, then $a^* = a_0$ and $b^* = b_0$.*
2. *Right-only contagion: If $a_0 \geq \alpha^\dagger(1)$ and $b_0 > \beta^\dagger(a_0)$, then $a^* = a_0$ and $b^* = 1$.*
3. *Left-only contagion: If $a_0 < \alpha^\dagger(b_0)$ and $b_0 \leq \beta^\dagger(0)$, then $a^* = 0$ and $b^* = b_0$.*
4. *Complete contagion: If (i) [right-first: $a_0 < \alpha^\dagger(1)$ and $b_0 > \beta^\dagger(a_0)$] or (ii) [left-first: $a_0 < \alpha^\dagger(b_0)$ and $b_0 > \beta^\dagger(0)$], then $a^* = 0$ and $b^* = 1$.*

To develop some intuition for Proposition 3 let us consider Case 1. Since $a_0 \geq \alpha^\dagger(b_0)$ no label smaller than a_0 is exposed at the first iteration (cf. Figure 14(i)) and since $b_0 \leq \beta^\dagger(a_0)$ no label greater than b_0 is exposed at the first iteration. Therefore, no new label is exposed and contagion stops immediately.

[11] From here on we omit the word weakly from (weakly) increasing/decreasing and we instead specify strictly increasing/decreasing if we want to exclude the equality sign.

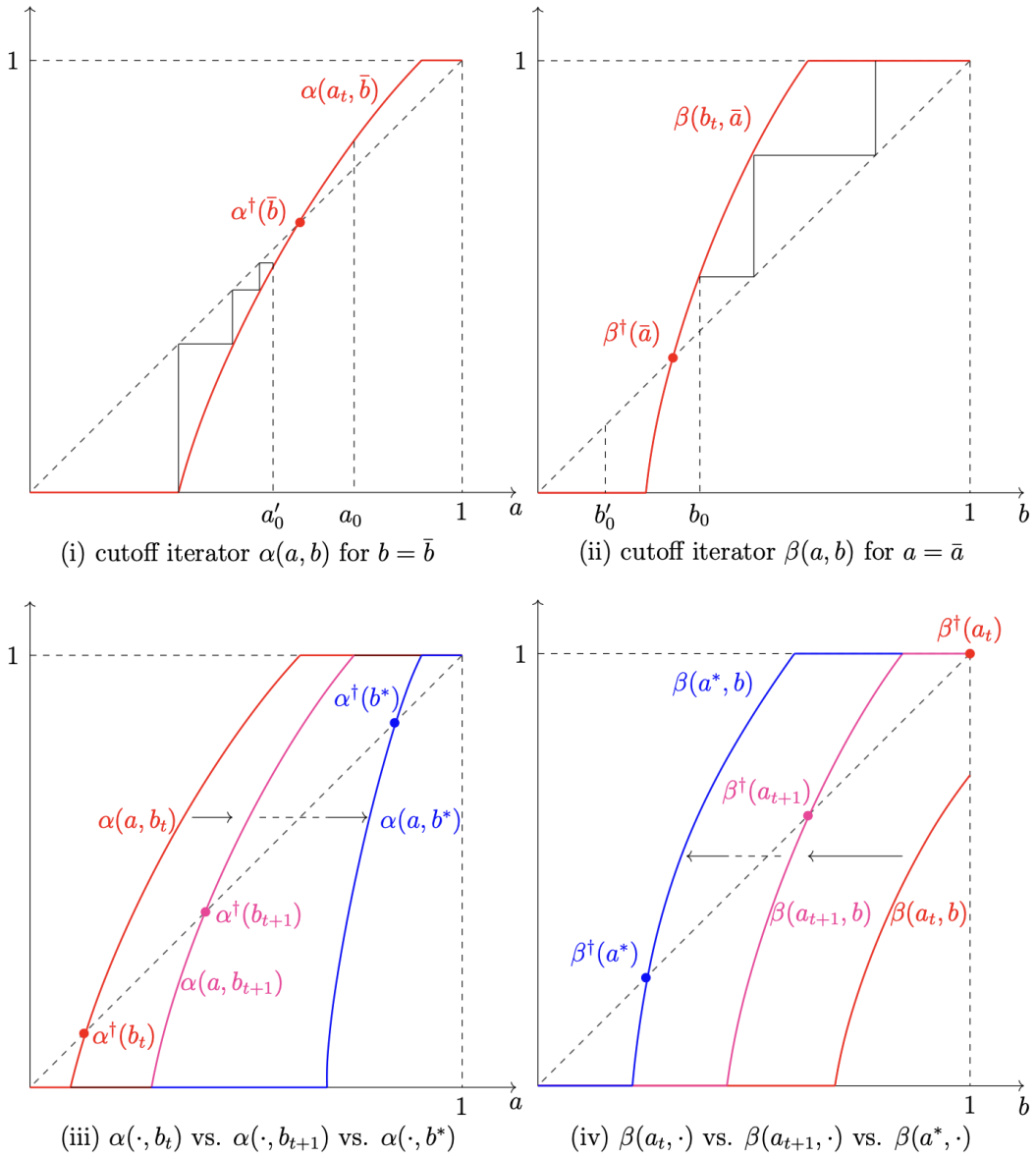

Figure 14: Phase diagrams of single-crossing cutoff iterators.

Consider first the case when *one extreme of the interval is fixed*. Figure 14(i) illustrates $\alpha(\cdot, \bar{b})$, for some fixed right end $\bar{b}$. Starting from a_0 one can define the auxiliary sequence $\{a_t^{\bar{b}}\}_{t=0}^{\infty}$ where $a_0^{\bar{b}} = a_0$ and $a_{t+1}^{\bar{b}} = \min\{a_t^{\bar{b}}, \alpha(a_t^{\bar{b}}, \bar{b})\}$. Note that if we start from $a_0 > \alpha^\dagger(\bar{b})$ the single-crossing property requires that $\alpha(a_0, \bar{b}) > a_0$. Therefore, the contagion process stops, and $a^* = a_0$. In other words, a_0 is a fixed point of the auxiliary sequence. On the other hand, suppose that we start from $a_0' < \alpha^\dagger(\bar{b})$. Using the single-crossing property that $\alpha(a, \bar{b}) < a$ for all a and that $\alpha(a, \bar{b})$ is right-continuous, the auxiliary sequence is strictly decreasing and converges to $a^* = 0$. Hence all labels in the interval $(0, \alpha^\dagger(\bar{b}))$ are infected. Figure 14(ii) illustrates the same process when the left end of the interval is fixed at $\bar{a}$ and $\beta(\bar{a}, \cdot)$ iterates.

During the actual contagion process, both ends of the contagion interval are *changing simultaneously*. Figure 14(iii) illustrates what happens to $\alpha(\cdot, b_t)$ when b_t increases to b_{t+1}. Using a similar argument to the one above, if one were to fix b_t then the interval $(0, \alpha^\dagger(b_t))$ would be infected. When b_t increases to b_{t+1} however $\alpha^\dagger(a, b_t)$ falls for all a, so the cutoff $\alpha^\dagger(b_{t+1})$ is higher than $\alpha^\dagger(b_t)$. Therefore, any label between $\alpha^\dagger(b_t)$ and $\alpha^\dagger(b_{t+1})$ will end up being infected. The process iterates until all labels in $(0, \alpha^\dagger(b^*))$ are infected.

Figure 14(iv) illustrates how $\beta^\dagger(a)$ changes as a decreases from a_t to a^*.

Suppose instead that $a_0 < \alpha^\dagger(b_0)$, in this case all the labels in $(\alpha(a_0, b_0), a_0)$ are exposed. Starting from a_0, define the auxiliary sequence $\{a_t^{b_0}\}_{t=0}^\infty$ where $a_0^{b_0} = a_0$ and $a_{t+1}^{b_0} = \min\{a_t^{b_0}, \alpha(a_t^{b_0}, b_0)\}$. In this auxiliary sequence, we are allowing only labels that are at the left of a_0 to get infected. Clearly, if a label is infected in the auxiliary sequence, it is infected also in the original contagion process. Since the single-crossing property gives us that $\alpha(a, b_0) < a$ for all $a < \alpha^\dagger(b_0)$ and we have that $\alpha(a, b_0)$ is right-continuous, the auxiliary sequence is strictly decreasing and converges to 0. This implies that $a^* = 0$. We now need to distinguish two cases. If $b_0 \leq \beta^\dagger(0)$ at no point in the process labels greater than b_0 are exposed and the final contagion outcome is $(0, b_0)$; this corresponds to Case 3 in Proposition 3. If instead $b_0 > \beta^\dagger(0)$, then by the single-crossing property and left continuity of $\beta(0, b)$, the auxiliary sequence $\{b_t^0\}_{t=0}^\infty$ where $b_0^0 = b_0$ and $b_{t+1}^0 = \max\{b_t^0, \beta(0, b_t^0)\}$ converges to 1 (cf. Figure 14(iii)). Intuitively, the fact that the process where we first allow contagion on the left and then on the right leads to complete contagion implies that complete contagion occurs also in the original process when both extremes change simultaneously (because the order of contagion does not matter).[12] Hence, we must have that $b^* = 1$ and the entire interval $[0, 1]$ is infected at the end of the contagion process; this corresponds to Case 4(ii) in Proposition 3. Cases 2 and 4(i) work analogously by first applying the cutoff operator to labels on the right and then on the left of (a_0, b_0).

In summary, for cutoff iterators that satisfy the single-crossing property, if contagion starts either to the left or to the right of initial seed set, it will continue until it infects all the labels on that side. To check for complete contagion, we can simply check whether contagion starts on one side and whether, after that, it starts on the other side.

B.2 Optimal seeding

In this section we show how Proposition 3 enables the solution of the interval max-reach and min-seed problems when cutoff iterators exist and satisfy the single-crossing property.

Max-reach

Denote by A_ρ the set of values of a_0 that lead to complete contagion, corresponding to Case 4 in Proposition 3. Then

$$A_\rho := \{a_0 \mid a_0 < \alpha^\dagger(1), a_0 + \rho > \beta^\dagger(a_0)\} \cup \{a_0 \mid a_0 < \alpha^\dagger(a_0 + \rho), a_0 + \rho > \beta^\dagger(0)\}.$$

If complete contagion is possible with a seed budget of ρ, then A_ρ is non-empty and any seed that corresponds to any element of A_ρ is optimal. Otherwise, we must consider whether partial contagion is possible. Proposition 3 tells us that partial contagion can be either right-only or left-only. With right-only contagion (Case 2), the outcome of contagion is $(a_0, 1]$. So the problem boils down to finding the smallest a_0 such that b_0 can be taken to be larger than $\beta^\dagger(0)$ with a budget of ρ. This way, contagion iterates until b_t converges 1. The case of left-only contagion (Case 3) works analogously. This discussion proves the next result.

[12] This argument is not entirely rigorous because the first sequence $\{a_t^{b_0}\}_{t=0}^\infty$ may take infinite time to converge, we provide a rigorous proof of these statements in Appendix C.

Proposition 4. *Under Assumptions 2 and 4, if $A_\rho \neq \emptyset$ then $\tilde{f}^*_\rho = 1$ and any set $[a_0, a_0 + \rho]$ with $a_0 \in A_\rho$ is an optimal seed set. If instead $A_\rho = \emptyset$ and we define*[13]

$$\tilde{f}^{[2]}_\rho := \sup_{a_0 \in [0,1]} \; 1 - a_0 \quad \text{s.t.} \quad a_0 \geq \alpha^\dagger(1), \; \rho + a_0 > \beta^\dagger(a_0)$$

$$\tilde{f}^{[3]}_\rho := \sup_{a_0 \in [0,1]} \; a_0 + \rho \quad \text{s.t.} \quad a_0 < \alpha^\dagger(a_0 + \rho), \; \rho + a_0 \leq \beta^\dagger(0)$$

then $\tilde{f}^*_\rho = \max\{\rho, \tilde{f}^{[2]}_\rho, \tilde{f}^{[3]}_\rho\}$.

Min-seed

Theorem 4. *Under Assumptions 2 and 4,*

$$\tilde{r}^*_m = \min\left\{ \inf_{0 \leq x < \max\{1-m, \alpha^\dagger(1)\}} \left(\beta^\dagger(x) - x\right)^+, \inf_{1 \geq x > \min\{m, \beta^\dagger(0)\}} \left(x - \alpha^\dagger(x)\right)^+ \right\}. \tag{B.3}$$

Consider any $\tilde{a}^*_0$ and $\tilde{b}^*_0$ respectively from the arguments of the first and second infimum terms in Eq. (B.3).[14] *If $\tilde{r}^*_m$ is equal to the first* inf*, then $[\tilde{a}^*_0, \tilde{a}^*_0 + \tilde{r}^*_m]$ is limit-optimal. If $\tilde{r}^*_m$ is equal to the second* inf *then $[\tilde{b}^*_0 - \tilde{r}^*_m, \tilde{b}^*_0]$ is limit-optimal.*

Recall from Proposition 3 that right-only or right-first complete contagion happens if and only if $b_0 > \beta(a_0)$. Then the smallest seed size that achieves right-only or right-first complete contagion requires taking $b_0 = \beta(a_0) + \epsilon$ (or $a_0 + \epsilon$ if $\beta(a_0) < a_0$). Then for a given a_0, the smallest seed size to achieve right-only or right-first complete contagion is $\left(\beta^\dagger(a_0) - a_0\right)^+ + \epsilon$. Given that right-only or right-first complete contagion happens with such a seed, the outcome is $(0, 1)$ if $a_0 < \alpha^\dagger(1)$ and it is $(a_0, 1)$ otherwise. Then the solution to the interval min-seed problem with the constraint that right-only or right-first complete contagion happens is given by the first inf term in the expression for $\tilde{r}^*_m$. The same argument applies for the second inf term for the case in which left-only or left-first contagion happens. Combining the two terms gives us the complete solution to the min-seed problem. Using this solution, we can also find the limit-optimal seed set for the interval min-seed problem.

[13] We define the sup of an empty-set to be 0. Note that the optimization problems in Proposition 4 are defined as supremum and not maximum hence an optimal seed set might not exists. However, by definition of supremum for any $\varepsilon > 0$ there must exists a feasible $a^{[2]}_{0,\varepsilon}$ such that $1 - a^{[2]}_{0,\varepsilon} > \tilde{f}^{[2]}_\rho - \varepsilon$ and a feasible $a^{[3]}_{0,\varepsilon}$ such that $a^{[3]}_{0,\varepsilon} + \rho > \tilde{f}^{[3]}_\rho - \varepsilon$. If $\tilde{f}^*_\rho = \tilde{f}^{[2]}_\rho$ one can then define an ε-optimal seed set as $[a^{[2]}_{0,\varepsilon}, a^{[2]}_{0,\varepsilon} + \rho]$. Instead, if $\tilde{f}^*_\rho = \tilde{f}^{[3]}_\rho$ an ε-optimal seed set is $[a^{[3]}_{0,\varepsilon}, a^{[3]}_{0,\varepsilon} + \rho]$.

[14] For $A \subset [0,1]$, $x^* \in \arg\inf_{x \in A} g(x)$ if there exists a sequence $(x_t)_t$ in A such that $\lim_t x_t = x^*$ and $\lim_t g(x_t) = \inf_{x \in A} g(x)$. Note that we are not requiring x^* to belong to A hence arg inf is non-empty.

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C Proofs

C.1 Algorithmic definitions of contagion processes

Denote

$$r(u, C_{t-1}) := \frac{d(u, C_{t-1})}{d(u)}$$

be the fraction of neighbors of label u that are infected in step $t-1$.

We can write the fraction of infected neighbors in the sampled network at time t as

$$r^{(n)}(i, C_{t-1}^{(n)}) := \frac{d^{(n)}\left(i, C_{t-1}^{(n)}\right)}{d^{(n)}(i)} := \frac{\sum_{j \neq i} A_{ij}^{(n)} \mathbb{1}_{C_{t-1}^{(n)}}(j)}{\sum_{j=1}^{n} A_{ij}^{(n)}}.$$

Algorithm 1: Contagion in a graphon

```
initialize C_0 ⊂ [0,1]
for t = 1, ..., ∞ do
    for u ∈ [0,1] do
        if u ∈ C_{t-1} or r(u, C_{t-1}) > τ(u) then
            u ∈ C_t
        end
    end
end
```

Algorithm 2: Contagion in a sampled network

```
initialize C_0^(n) ⊂ [n]
for t = 1, ..., n do
    for i = 1, ..., n do
        if i ∈ C_{t-1}^(n) or r^(n)(i, C_{t-1}^(n)) > τ_i^(n) then
            i ∈ C_t^(n)
        end
    end
end
```

C.2 Auxiliary lemmas for Proposition 1 and Proposition 2

Lemma 1. *Let $\phi : [0,1] \to [-1,1]$ be a measurable function. For any $\ell \in [-1,1]$, define the strict upper contour set of ϕ at ℓ as $\phi^{-1}((\ell,1]) = \{u \in [0,1] \mid \phi(u) > \ell\}$. Then $\mu\left(\phi^{-1}((\ell,1])\right)$ is right-continuous in ℓ.*

Lemma 1 can be proven following the same arguments as for the right-continuity of CDFs, hence its proof is omitted.

Lemma 2. *Consider any measurable set C. If Assumption 3 holds, then $\phi(u) := \int_C W(u,v)dv - \tau(u)\int_0^1 W(u,v)dv$ is piece-wise continuous (i.e. can be discontinuous only in D_u).*

Proof. We show that $\int_C W(u,v)dv$ is piece-wise continuous in u. Specifically, we prove that it is continuous for any $u \in [0,1]\backslash D_u$. The rest follows similarly. We need to prove that for any $u \in [0,1]\backslash D_u$, $u_n \to u$ implies $\int_C W(u_n,v)dv \to \int_C W(u,v)dv$. For all $v \in [0,1]\backslash D_v$ and $u \in [0,1]\backslash D_u$, $W(u,v)$ is continuous in u, hence $f_n(v) := W(u_n,v)\mathbb{1}_C(v) \to W(u,v)\mathbb{1}_C(v) =: f(v)$. Thus $f_n \to f$ pointwise (except for a set D_v of zero measure). Also note that $|f_n(v)| \leq 1$ for all $v \in [0,1]$, for all n. Then by Lebesgue's Dominated Convergence Theorem

$$\lim_{n\to\infty} \int_C W(u_n,v)dv = \lim_{n\to\infty} \int_0^1 f_n(v)dv = \int_0^1 f(v)dv = \int_C W(u,v)dv$$

for all $u \in [0,1]\backslash D_u$. □

Lemma 3. *Suppose that $\phi : [0,1] \to [-1,1]$ is piece-wise continuous (i.e. it is discontinuous only in a set D_u of zero measure). Then $\phi^{-1}((\ell,1])$ is measurable for any $\ell \geq 0$.*

Proof. Since ϕ is piece-wise continuous it is measurable and thus $\phi^{-1}((\ell,1])$ (i.e. the pre-image of the measurable set $(\ell,1]$ is measurable). □

Lemma 4. *If Assumption 3 holds, then C_t is measurable for all $t \geq 0$.*

Proof. We proceed by induction, first note that C_0 is measurable by Assumption 3. We then show that if C_{t-1} is measurable then C_t is measurable. To this end, define $\phi_{t-1}(u) := \int_{C_{t-1}} W(u,v)dv - \tau(u)\int_0^1 W(u,v)dv$ and note that, since C_{t-1} is measurable, $\phi_{t-1}(u)$ is piece-wise continuous by Lemma 2. As a consequence $\hat{C}_t = \phi_{t-1}^{-1}((0,1])$ is measurable by Lemma 3 . Finally $C_t = C_{t-1} \cup \hat{C}_t$ is the union of measurable sets and is thus measurable. □

C.3 Proofs of Proposition 1 and Proposition 2

We prove Proposition 1 and Proposition 2 jointly by induction.

- **Step 1:** Proposition 1 holds for $t = 0$.

  To prove this statement for any ε_0, set $N_0^{\varepsilon_0} = 0$, $C_0^{\varepsilon_0} = C_0$. By definition, $V^{(n)}(C_0^{\varepsilon_0}) = V^{(n)}(C_0) = C_0^{(n)}$. Hence Proposition 1 holds for $t = 0$ and any $\kappa_0 \geq 0$.

- **Step 2:** We next prove that if Proposition 1 holds for $t' = t-1$, then Proposition 2 holds for time t.

  - **Step 2.1**: We start by showing that for any ε_t it is possible to construct a measurable set $\hat{C}_t^{\varepsilon_t}$ such that:

    P1) $\hat{C}_t^{\varepsilon_t}$ is contained in $\hat{C}_t$ and differs by it at most of measure ε_t;

    P2) any $u \in \hat{C}_t^{\varepsilon_t}$ has susceptibility bounded away from zero, meaning that there exists $\ell_t > 0$ such that $d(u, C_{t-1}) - \tau(u)\, d(u) > \ell_t$.

    To this end,

* Define $\phi(u) := d(u, C_{t-1}) - \tau(u) d(u)$ and $M(\ell) := \phi^{-1}((\ell, 1])$. By Lemmas 2 and 4 ϕ is piece-wise continuous and thus measurable, hence $\mu(M(\ell))$ is right-continuous by Lemma 1 and $lim_{\ell \downarrow 0} \mu(M(\ell)) = \mu(M(0))$. Note that $M(0) = \hat{C}_t$ by definition. By right-continuity, given ε_t, there exists $\ell_t > 0$ such that $0 \leq \mu\left(\hat{C}_t\right) - \mu(M(\ell_t)) \leq \varepsilon_t$. Set $\hat{C}_t^{\varepsilon_t} = M(\ell_t)$ which is measurable by Lemma 3.
* Then
$$\hat{C}_t^{\varepsilon_t} = M(\ell_t) \subset M(0) = \hat{C}_t,$$
$$\mu\left(\hat{C}_t^{\varepsilon_t}\right) = \mu(M(\ell_t)) \geq \mu\left(\hat{C}_t\right) - \varepsilon_t,$$
proving P1).
* Finally, take any $u \in \hat{C}_t^{\varepsilon_t}$. Since $\hat{C}_t^{\varepsilon_t} = M(\ell_t)$, by construction, $d(u, C_{t-1}) - \tau(u) d(u) > \ell_t$, thus proving P2).

– **Step 2.2:** We next show that for n large enough with high probability all agents in the newly constructed set $\hat{C}_t^{\varepsilon_t}$ are exposed in the sample. More precisely, we aim at showing that with probability at least $1 - \kappa_t$
$$u_i^{(n)} \in \hat{C}_t^{\varepsilon_t} \quad \Rightarrow \quad i \in \hat{C}_t^{(n)}.$$
We do this in several steps.
* By definition $i \in \hat{C}_t^{(n)}$ if and only if
$$T := \sum_{j \in C_{t-1}^{(n)}} A_{ij}^{(n)} - \tau_i^{(n)} \sum_{j=1}^{n} A_{ij}^{(n)} > 0.$$
Fix $\kappa_{t-1} = \frac{\kappa_t}{2}$ and $\varepsilon_{t-1} = \frac{\ell_t}{2}$ (where ℓ_t is as defined in the construction of the set $\hat{C}_t^{\varepsilon_t}$ in step 2.1). By induction, for any $n > N_{t-1}^{\varepsilon_{t-1}, \kappa_{t-1}}$ with probability at least $1 - \kappa_{t-1}$
$$T = \textstyle\sum_{j \in C_{t-1}^{(n)}} A_{ij}^{(n)} - \tau_i^{(n)} \sum_j A_{ij}^{(n)} \overset{\text{induction step}}{\geq} \underbrace{\textstyle\sum_{j \in V^{(n)}(C_{t-1}^{\varepsilon_{t-1}})} A_{ij}^{(n)}}_{T_1(u_i^{(n)})} - \tau_i^{(n)} \underbrace{\textstyle\sum_j A_{ij}^{(n)}}_{T_2(u_i^{(n)})}, \tag{C.1}$$
where we used that $V^{(n)}(C_{t-1}^{\varepsilon_{t-1}}) \subset C_{t-1}^{(n)}$. We aim at showing that for n large $\frac{T_1(u_i^{(n)})}{n} \approx d\left(u_i^{(n)}, C_{t-1}^{\varepsilon_{t-1}}\right)$ and $\frac{T_2(u_i^{(n)})}{n} \approx d\left(u_i^{(n)}\right)$. To this end, let us define the functions
$$E_1(u) := \sum_j \mathbb{1}(u_j^{(n)} \in C_{t-1}^{\varepsilon_{t-1}}) W(u, u_j^{(n)}) \quad \text{and} \quad E_2(u) := \sum_j W(u, u_j^{(n)}),$$
so that for example $E_2(u_i^{(n)}) := \sum_j W(u_i^{(n)}, u_j^{(n)}) = \sum_j \mathbb{E}[A_{ij}^{(n)}]$ where the expectation is over the Bernoulli realization of the links. We aim at showing that:
i) $\frac{T_2(u_i^{(n)})}{n}$ accumulates around its mean $\frac{E_2(u_i^{(n)})}{n}$

ii) $\frac{E_2(u_i^{(n)})}{n}$ converges to $d\left(u_i^{(n)}\right)$,
(and similarly for T_1).

* Proof of i): Consider any $\kappa, \epsilon > 0$. We next aim at proving that there exists $\tilde{N}_1$ large enough such that for all $n > \tilde{N}_1$ and for any $\{u_j^{(n)}\}_{j=1}^n$, with probability at least $1 - \kappa/2$

$$\frac{1}{n}|T_1(u_i^{(n)}) - E_1(u_i^{(n)})| = |\frac{1}{n}\sum_{j:u_j^{(n)}\in C} A_{ij}^{(n)} - \frac{1}{n}\sum_{j:u_j^{(n)}\in C}\mathbb{E}[A_{ij}^{(n)}]| \leq \epsilon/2, \quad \text{for all } i, \tag{C.2}$$

where the expectation is over the Bernoulli realization of the links and $C = C_{t-1}^{\varepsilon_{t-1}}$ (for the T_2 term set $C = [0,1]$ and repeat the same argument). To this end let us define the new random variables

$$\tilde{A}_{ij}^{(n)} = \begin{cases} A_{ij}^{(n)} & \text{if } u_j^{(n)} \in C \\ 0 & \text{otherwise} \end{cases}$$

and note that

$$|\frac{1}{n}\sum_{j:u_j^{(n)}\in C} A_{ij}^{(n)} - \frac{1}{n}\sum_{j:u_j^{(n)}\in C}\mathbb{E}[A_{ij}^{(n)}]| = |\frac{1}{n}\sum_{j=1}^n \tilde{A}_{ij}^{(n)} - \frac{1}{n}\sum_{j=1}^n \mathbb{E}[\tilde{A}_{ij}^{(n)}]|.$$

Moreover note that for each fixed i the variables $\{\tilde{A}_{ij}^{(n)}\}_{j=1}^n$ are independent and bounded between 0 and 1. It follows by Hoeffding's inequality that

$$\mathbb{P}[|\frac{1}{n}\sum_{j=1}^n \tilde{A}_{ij}^{(n)} - \frac{1}{n}\sum_{j=1}^n \mathbb{E}[\tilde{A}_{ij}^{(n)}]| > \epsilon/2] < 2\exp(-2(\epsilon/2)^2 n).$$

By the union bound,

$$\mathbb{P}[|\frac{1}{n}\sum_{j=1}^n \tilde{A}_{ij}^{(n)} - \frac{1}{n}\sum_{j=1}^n \mathbb{E}[\tilde{A}_{ij}^{(n)}]| \leq \epsilon/2, \forall i] > 1 - 2n\exp(-2(\epsilon/2)^2 n).$$

The conclusion follows by setting $\tilde{N}_1$ such that $2\tilde{N}_1\exp(-2(\epsilon/2)^2\tilde{N}_1) < \kappa/2$.

* Proof of ii): The functions

$$E_1(u) := \sum_j \mathbb{1}(u_j^{(n)} \in C_{t-1}^{\varepsilon_{t-1}}) W(u, u_j^{(n)}) \quad \text{and} \quad E_2(u) := \sum_j W(u, u_j^{(n)})$$

are sum of iid random variables since the $u_j^{(n)}$ are iid random variables

uniformly distributed in $[0,1]$. Note that

$$\mathbb{E}\left[W(u,u_j^{(n)})\right] = \int_0^1 W(u,w)dw = d(u),$$
$$\mathbb{E}\left[\mathbb{1}(u_j^{(n)} \in C_{t-1}^{\varepsilon_{t-1}})W(u,u_j^{(n)})\right] = \int_0^1 \mathbb{1}(w \in C_{t-1}^{\varepsilon_{t-1}})W(u,w)dw = d(u, C_{t-1}^{\varepsilon_{t-1}}).$$

By the uniform law of large number[15] (uniform in u) for any $\epsilon > 0$ and for any $\kappa > 0$ there exists $\tilde{N}_2$ such that for any $n \geq \tilde{N}_2$ with probability $1 - \kappa/2$

$$sup_{u\in[0,1]} \left|\frac{1}{n}E_2(u) - d(u)\right| < \epsilon/2 \tag{C.3a}$$

and similarly

$$sup_{u\in[0,1]} \left|\frac{1}{n}E_1(u) - d(u, C_{t-1}^{\varepsilon_{t-1}})\right| < \epsilon/2. \tag{C.3b}$$

Hence in particular

$$\begin{aligned} |\frac{1}{n}E_1(u_i^{(n)}) - d(u_i^{(n)}, C_{t-1}^{\varepsilon_{t-1}})| \leq \epsilon/2, \quad \text{for all } i, \\ |\frac{1}{n}E_2(u_i^{(n)}) - d(u_i^{(n)})| \leq \epsilon/2, \quad \text{for all } i. \end{aligned} \tag{C.4}$$

* Combining (C.2) and (C.4), we obtain that for all $n \geq \tilde{N} := \max\{\tilde{N}_1, \tilde{N}_2\}$ with probability $1 - \kappa$

$$\begin{aligned} T_1(u_i^{(n)}) &> nd\left(u_i^{(n)}, C_{t-1}^{\varepsilon_{t-1}}\right) - n\epsilon \\ T_2(u_i^{(n)}) &< nd\left(u_i^{(n)}\right) + n\epsilon \end{aligned} \tag{C.5}$$

for all i.

* Set $\kappa = \frac{\kappa_t}{2}$ and $\epsilon = \frac{\ell_t}{4}$. Plugging-in the bounds derived in (C.5) in (C.1), using the fact that $d\left(u, C_{t-1}^{\varepsilon_{t-1}}\right) \geq d\left(u, C_{t-1}\right) - \varepsilon_{t-1}$ and the union bound, we get that for all $n > \hat{N}_t^{\varepsilon_t,\kappa_t} = \max\left\{N_{t-1}^{\varepsilon_{t-1},\kappa_{t-1}}, \tilde{N}\right\}$ with probability at least $1 - \kappa_{t-1} - \kappa = 1 - \frac{\kappa_t}{2} - \frac{\kappa_t}{2} = 1 - \kappa_t$

$$\begin{aligned} T &\geq n\left[d\left(u_i^{(n)}, C_{t-1}^{\varepsilon_{t-1}}\right) - \tau_i^{(n)} d\left(u_i^{(n)}\right) - \epsilon - \tau_i^{(n)}\epsilon\right] \\ &= n\left[d\left(u_i^{(n)}, C_{t-1}\right) - \varepsilon_{t-1} - \tau\left(u_i^{(n)}\right) d\left(u_i^{(n)}\right) - 2\epsilon\right] \\ &> n\left[\ell_t - \varepsilon_{t-1} - 2\epsilon\right] = n\ell_t\left[1 - \frac{1}{2} - 2\frac{1}{4}\right] = 0 \end{aligned}$$

as desired.

[15] Define $F\left(x|\theta\right) = W\left(\theta, x\right)$ and $F\left(x|\theta\right) = \mathbb{1}(x \in C_{t-1}^{\varepsilon_{t-1}})W\left(\theta, x\right)$ for E_2 and E_1, respectively. Then $F(x \mid \theta)$ is piecewise continuous in θ for almost all x and measurable in x for each θ, hence uniform law of large numbers applies. Additionally, we use the union bound to bound the two quantities in (C.3) simultaneously.

* Overall we have shown that for all $n > \hat{N}_t^{\varepsilon_t,\kappa_t}$ with probability at least $1-\kappa_t$

$$u_i^{(n)} \in \hat{C}_t^{\varepsilon_t} \quad \Rightarrow \quad i \in \hat{C}_t^{(n)}.$$

- **Step 3.**

  Finally, we prove that if Proposition 1 holds for $t' = t-1$ and Proposition 2 holds for $t' = t$, then Proposition 1 holds for $t' = t$.

  - Recall that $C_t^{(n)} = C_{t-1}^{(n)} \cup \hat{C}_t^{(n)}$ and $C_t = C_{t-1} \cup \hat{C}_t$ (the infected at time t are the union of the infected at time $t-1$ and the exposed at time t) .
  - For any ε_t, κ_t by Proposition 1 applied to $t' = t-1$, there exists $N_{t-1}^{\varepsilon_t/2,\kappa_t/2}$ and $C_{t-1}^{\varepsilon_t/2}$ such that $\mu(C_{t-1}^{\varepsilon_t/2}) \geq \mu(C_{t-1}) - \varepsilon_t/2$ and for all $n > N_{t-1}^{\varepsilon_t/2,\kappa_t/2}$ with probability $1-\kappa_t/2$

$$V^{(n)}(C_{t-1}^{\varepsilon_t/2}) \subset C_{t-1}^{(n)};$$

  - Similarly, for any ε_t, κ_t by Proposition 2 applied to $t' = t$, there exists $\hat{N}_t^{\varepsilon_t/2,\kappa_t/2}$ and $\hat{C}_t^{\varepsilon_t/2}$ such that $\mu(\hat{C}_t^{\varepsilon_t/2}) \geq \mu(\hat{C}_t) - \varepsilon_t/2$ and for all $n > \hat{N}_t^{\varepsilon_t/2,\kappa_t/2}$ with probability $1-\kappa_t/2$

$$V^{(n)}(\hat{C}_t^{\varepsilon_t/2}) \subset \hat{C}_t^{(n)};$$

  - Let us define $N_t^{\varepsilon_t,\kappa_t} := \max\{N_{t-1}^{\varepsilon_t/2,\kappa_t/2}, \hat{N}_t^{\varepsilon_t/2,\kappa_t/2}\}$ and $C_t^{\varepsilon_t} = C_{t-1}^{\varepsilon_t/2} \cup \hat{C}_t^{\varepsilon_t/2}$. Note that since both $C_{t-1}^{\varepsilon_t/2}$ and $\hat{C}_t^{\varepsilon_t/2}$ are measurable so is their union.
  - Moreover, for any $n > N_t^{\varepsilon_t,\kappa_t}$, $\mu(C_t^{\varepsilon_t}) \geq \mu(C_t) - \varepsilon_t$ and with probability $1-\kappa_t$

$$V^{(n)}(C_t^{\varepsilon_t}) = V^{(n)}(C_{t-1}^{\varepsilon_t/2} \cup \hat{C}_t^{\varepsilon_t/2}) = [V^{(n)}(C_{t-1}^{\varepsilon_t/2}) \cup V^{(n)}(\hat{C}_t^{\varepsilon_t/2})] \subset [C_{t-1}^{(n)} \cup \hat{C}_t^{(n)}] = C_t^{(n)}.$$

  This concludes the induction and proves Propositions 1 and 2.

C.4 Proof of Theorem 1a′

- Consider the contagion process in the graphon. The sequence $\mu(C_t)$ is increasing and is upper bounded by 1. Hence it is convergent, let μ^* be its limit. Clearly, it must be $\mu^* = \mu(C_\infty)$. By definition of limit, for any $\varepsilon > 0$, there exists a time $T > 0$ such that $\mu(C_T) > \mu(C_\infty) - \varepsilon/2$.
- By Proposition 1, there exists $N_T^{\varepsilon/2,\kappa}$ and $C_T^{\varepsilon/2}$ such that $\mu(C_T^{\varepsilon/2}) \geq \mu(C_T) - \varepsilon/2$ and for all $n > N_T^{\varepsilon/2,\kappa}$ with probability at least $1-\kappa$,

$$V^{(n)}(C_T^{\varepsilon/2}) \subset C_T^{(n)}.$$

- Let $N^\varepsilon := N_T^{\varepsilon/2,\kappa}$ and $C_\infty^\varepsilon := C_T^{\varepsilon/2}$. Then $C_\infty^\varepsilon = C_T^{\varepsilon/2} \subset C_T \subset C_\infty$ and $\mu(C_\infty^\varepsilon) = \mu(C_T^{\varepsilon/2}) \geq \mu(C_T) - \varepsilon/2 \geq \mu(C_\infty) - \varepsilon$. Moreover

$$V^{(n)}(C_\infty^\varepsilon) = V^{(n)}(C_T^{\varepsilon/2}) \subset C_T^{(n)} \subset C_*^{(n)}.$$

C.5 Proof of Theorem 1b′

- First of all note that since $\mu_\infty = \sup_{D\in\mathcal{D}} \mu(D)$ for any $\epsilon > 0$ there exists a set $\bar{\bar{D}}_\infty \in \mathcal{D}$ such that $\mu(\bar{\bar{D}}_\infty) \geq \mu_\infty - \epsilon/2$.
- Next note that since $\bar{\bar{D}}_\infty \in \mathcal{D}$, $\phi(u) := d(u, \bar{\bar{D}}_\infty^c) - \tau(u)d(u) < 0$ for all $u \in \bar{\bar{D}}_\infty$. Let $\tilde{\phi}(u) := -\phi(u)$. Then $\bar{\bar{D}}_\infty := \tilde{\phi}^{-1}((0,1])$. Similar arguments as in the proof of Theorem 1 show that for any $\epsilon > 0$ it is possible to construct a measurable set $\bar{D}_\infty^\epsilon \subset \bar{\bar{D}}_\infty$ such that $\mu(\bar{D}_\infty^\epsilon) \geq \mu(\bar{\bar{D}}_\infty) - \epsilon/2 \geq \mu_\infty - \epsilon$ and there exists $\ell > 0$ such that $d(u, \bar{\bar{D}}_\infty^c) - \tau(u)d(u) < -\ell$ for all $u \in \bar{D}_\infty^\epsilon$.
- We next show that with probability $1-\kappa$ for n large enough, $V^{(n)}\left(\bar{D}_\infty^\varepsilon\right)$ is a cohesive set. To this end, we need to prove that if $u_i^{(n)} \in \bar{D}_\infty^\varepsilon$ then

$$T_i := \underbrace{\sum_{j|u_j^{(n)}\in[\bar{D}_\infty^\varepsilon]^c} A_{ij}^{(n)}}_{T_3(u_i^{(n)})} - \tau_i^{(n)} \underbrace{\sum_j A_{ij}^{(n)}}_{T_2(u_i^{(n)})} \leq 0.$$

  Note that $T_2(u_i^{(n)})$ is as defined in (C.1) and $T_3(u_i^{(n)})$ has the same structure as $T_1(u_i^{(n)})$. In a similar way we can conclude that for any $\kappa > 0$ and $\delta > 0$ there exists $N_{\delta,\kappa}$ such that if $n > N_{\delta,\kappa}$ with probability at least $1-\kappa$ for all i

$$\begin{aligned} T_3(u_i^{(n)}) &< nd\left(u_i^{(n)}, [\bar{D}_\infty^\varepsilon]^c\right) + n\delta \\ T_2(u_i^{(n)}) &> nd\left(u_i^{(n)}\right) - n\delta. \end{aligned} \tag{C.6}$$

  Hence

$$\begin{aligned} T_i &= n\left[\frac{1}{n}T_3(u_i^{(n)}) - \tau_i^{(n)}\frac{1}{n}T_2(u_i^{(n)})\right] \\ &< n\left[d(u_i^{(n)}, [\bar{D}_\infty^\varepsilon]^c) + \delta - \tau_i^{(n)}(d(u_i^{(n)}) - \delta)\right] \\ &\leq n\left[-\ell + 2\delta\right] \end{aligned}$$

  where we used $d(u_i^{(n)}, [\bar{D}_\infty^\varepsilon]^c)) - \tau_i^{(n)} d(u_i^{(n)}) < -\ell$ since $u_i^{(n)} \in \bar{D}_\infty^\varepsilon$. By setting, $\delta < \frac{\ell}{2}$ and $n > N^{\epsilon,\kappa} = N_{\delta,\kappa}$ we get $T_i < 0$ as desired.
- Since $V^{(n)}\left(\bar{D}_\infty^\varepsilon\right)$ is a cohesive set and none of the agents in $V^{(n)}\left(\bar{D}_\infty^\varepsilon\right)$ is initially infected, these agents cannot be infected during the process, thus terminating the proof.

C.6 Proof of Theorem 2

To keep the notation simple, from here on, given a graphon $W(u,v)$ and threshold function $\tau(u)$, we denote by $\chi(C_0)$ the graphon contagion outcome C_∞ corresponding to the initial seed set $C_0 \subset [0,1]$ and we recall that $f(C_0) = \mu(\chi(C_0))$.

i) It is clear that $f_0^* = 0$ and $r_0^* = 0$ since starting from a zero measure set contagion does not iterate.

ii) Take any ρ such that $\rho < r_1^*$. In this case, it is not possible to achieve complete

contagion, and thus $f^*_\rho < 1$. Take any $\epsilon < 1 - f^*_\rho$. Take a sequence $(C^t_0)_t$ such that $\mu(C^t_0) \leq \rho$, $f(C^t_0) \uparrow f^*_\rho$. For each t, $f(C^t_0) \leq f^*_\rho < 1 - \epsilon$. Then there exists a set Z_t with measure ϵ in the complement of $\chi(C^t_0)$. Denote $Z'_t = C^t_0 \cup Z_t$. Z'_t has measure at most $\rho + \epsilon$. Then $f(Z'_t) \leq f^*_{\rho+\epsilon}$. Also $\chi(Z'_t) \supset \chi(C^t_0) \cup Z_t$ and so $f(Z'_t) \geq f(C^t_0) + \epsilon$. Therefore, $f^*_{\rho+\epsilon} \geq f(C^t_0) + \epsilon$. Taking the limit, we find $f^*_{\rho+\epsilon} \geq f^*_\rho + \epsilon$. This proves that $f^*_\rho - \rho$ is increasing for $\rho \in [0, r^*_1)$. It is clear that for $\rho > r^*_1$, $f^*_\rho \equiv 1$.

iii) Regarding $m - r^*_m$ we can use the same logic. First, take $m > m^*_1$. By definition of m^*_1 it is not possible to reach a set of measure m with an initial seed of measure less than r^*_1, on the other hand for any $\rho > r^*_1$ it is possible to find an initial set of measure ρ that induces complete contagion. Hence it must be $r^*_m = r^*_1$.

Take $m < m^*_1$, by definition of m^*_1, it must be $r^*_m < r^*_1$ (there exists $\bar{\rho} < r^*_1$ s.t. $f^*_{\bar{\rho}} > m$ hence $r^*_m \leq \bar{\rho} < r^*_1$) which implies $f^*_{r^*_m} < 1$. Let $\epsilon < 1 - f^*_{r^*_m}$. Take a sequence C^t_0 such that $f(C^t_0) \geq m - \epsilon$ and $\mu(C^t_0) \downarrow r^*_{m-\epsilon}$. Take the union of C^t_0 with an ϵ measure set Z_t inside the complement of $\chi(C^t_0)$ (which exists since $f^*_{r^*_{m-\epsilon}} \leq f^*_{r^*_m} < 1 - \epsilon$). Call this union Z'_t. We have $f(Z'_t) \geq f(C^t_0) + \epsilon \geq m$. Then $\mu(Z'_t) \geq r^*_m$. Also, $\mu(Z'_t) = \mu(C^t_0) + \epsilon \downarrow r^*_{m-\epsilon} + \epsilon$. Hence, $r^*_{m-\epsilon} + \epsilon \geq r^*_m$ which completes the proof.

C.7 Proof of Theorem 3

As first step we partition the labels into $2k$ classes $\{\mathcal{U}_j\}_{j=1}^{2k}$ where $\mathcal{U}_j = \mathcal{I}_j \setminus C_0$ for $j = 1, \ldots, k$ is the set of labels in community j that do not belong to the seed set and $\mathcal{U}_j = \mathcal{I}_{j-k} \cap C_0$ for $j = k+1, \ldots, 2k$ is the set of labels in community j that belong to the seed set. It is immediate to see that labels within the same class have always the same state (i.e. either the entire class is infected or nobody in the class is infected). Take then a label u in class $i \leq k$, such label (and therefore its entire class) is infected at time t if either it was infected at time $t-1$ or if it is exposed, meaning

$$\begin{aligned} & d(u, C_{t-1}) - \tau_i d(u) > 0 \\ \Leftrightarrow & \int_0^1 W_{\text{SBM}}(u,v) \mathbb{1}_{C_{t-1}}(v) dv - \tau_i \int_0^1 W_{\text{SBM}}(u,v) dv > 0 \\ \Leftrightarrow & \sum_{j=1}^{2k} \int_{\mathcal{U}_j} w_{ij[k]} \mathbb{1}_{C_{t-1}}(v) dv - \tau_i \sum_{j=1}^{2k} \int_{\mathcal{U}_j} w_{ij[k]} dv > 0 \\ \Leftrightarrow & \sum_{j=1}^{2k} w_{ij[k]} b_{j[k]} \mathbb{1}_{C^{aux}_{t-1}}(j) - \tau_i \sum_{j=1}^{2k} w_{ij[k]} b_{j[k]} > 0 \end{aligned} \tag{C.7}$$

where $\mathbb{1}_{C^{aux}_{t-1}}(j) = 1$ if class j was infected at time $t-1$. The last equation in (C.7) is exactly the contagion process in the auxiliary network.

C.8 Proof of Proposition 3

We start with some lemmas. Denote $\Delta_\alpha = \{(a,b) \in [0,1]^2 : \alpha(a,b) < a\}$ and $\Delta_\beta = \{(a,b) \in [0,1]^2 : \beta(a,b) > b\}$.

Lemma 5. *Monotonicity of iterators: $\alpha(a,b)$ is increasing in a and decreasing in b on Δ_α. $\beta(a,b)$ is decreasing in a and increasing in b on Δ_β.*

Continuity of iterators: $\alpha(a,b)$ is right-continuous in a and left-continuous in b on Δ_α. $\beta(a,b)$ is right-continuous in a and left-continuous in b on Δ_β.

Proof. Note that $d(u,(a,b))$ is an integral of a non-negative function from a to b, hence it is continuous in (a,b), decreasing in a, and increasing in b.

Monotonicity: Take $(a,b),(a',b) \in \Delta_\alpha$ such that $a < a'$. Then for any u, we have $d(u,(a,b)) \geq d(u,(a',b))$ and for any $u \in (0,a]$, $\mathbf{1}_{u>\alpha(a,b)} \geq \mathbf{1}_{u>\alpha(a',b)}$. This implies that $\alpha(a,b) \leq \alpha(a',b)$. Similar arguments follow for the remaining monotonicity properties.

Continuity: Note that $d(u,(a,b))$ is continuous in a and b as it is an integral from a to b. Then for all u, there exists $m(u,b) \geq 0$ such that $d(u,(a,b)) > \tau(u)d(u)$ iff $a < m(u,b)$. Therefore, for all $u \in (0,a]$, $a < m(u,b)$ iff $u > \alpha(a,b)$. Now, for a fixed b, take a discontinuity point a_0 of $\alpha(\cdot,b)$ such that $(a_0,b) \in \Delta_\alpha$, if any exists. As α is increasing in a on Δ_α, left and right limits of $\alpha(a,b)$ at $a = a_0$ exist. Denote them $\alpha(a_0^-,b)$ and $\alpha(a_0^+,b)$. By the property $[a < m(u,b)$ iff $u > \alpha(a,b)]$, $m(u,b) = a_0$ for all $u \in [\alpha(a_0^-,b), \alpha(a_0^+,b)]$. Then $a_0 \not< m(u,b)$ for $u \in u \in [\alpha(a_0^-,b), \alpha(a_0^+,b)]$, implying that $u \not> \alpha(a_0,b)$ for $u \in [\alpha(a_0^-,b), \alpha(a_0^+,b)]$. Therefore, $\alpha(a_0,b) \geq \alpha(a_0^+,b)$. Thus, $\alpha(a_0,b) = \alpha(a_0^+,b)$. This shows that α is right-continuous in a on Δ_α. Similar arguments can be used to show the remaining continuity properties. □

Denote $\bar{\alpha}(a,b) := \min\{a, \alpha(a,b)\}$ and $\bar{\beta}(a,b) := \max\{b, \beta(a,b)\}$. Further note that $\alpha \equiv \bar{\alpha}$ on Δ_α and $\beta \equiv \bar{\beta}$ on Δ_β. Recall how we have defined contagion sequences: we start with the seed $(a_0,b_0) \subset [0,1]$, and iterate a_t with $\bar{\alpha}$ and b_t with $\bar{\beta}$. Since these are (weakly) monotone and bounded sequences, the limits exist and we denote them by a^* and b^*. The fact that α and β are right-continuous in a and left-continuous in b (see Lemma 5) along with the fact that $(a_t)_{t\geq 0}$ is decreasing and $(b_t)_{t\geq 0}$ is increasing, guarantees that $a^* = \bar{\alpha}(a^*,b^*)$ and $b^* = \bar{\beta}(a^*,b^*)$.

Note that in the contagion sequence both arguments of $\bar{\alpha}$ and $\bar{\beta}$ change in time. It is useful to define two *auxiliary sequences* where only one argument changes in time. Specifically, construct the *auxiliary sequence* $(\tilde{a}_t)_{t\geq 0}$ given by $\tilde{a}_0 = a_0$, $\tilde{a}_t = \bar{\alpha}(\tilde{a}_{t-1}, b^*)$. Similarly, construct $(\tilde{b}_t)_{t\geq 0}$ given by $\tilde{b}_0 = b_0$, $\tilde{b}_t = \bar{\beta}(\tilde{b}_{t-1}, a^*)$.

Lemma 6. *$\lim_t \tilde{a}_t = a^*$ and $\lim_t \tilde{b}_t = b^*$.*

Proof. By the monotonicity properties of α and β in Lemma 5, we can prove $[\tilde{a}_t \leq a_t$ for all $t]$. In fact, by induction: $\tilde{a}_{t-1} \leq a_{t-1}$ and $b_{t-1} \leq b^* \implies \tilde{a}_t = \bar{\alpha}(\tilde{a}_{t-1}, b^*) \leq \bar{\alpha}(a_{t-1}, b_{t-1}) = a_t$. Moreover, $[\tilde{a}_t \geq a^*$ for all $t]$ since: $\tilde{a}_t = \bar{\alpha}(\tilde{a}_{t-1}, b^*) \geq \bar{\alpha}(a^*, b^*) = a^*$. Thus $a^* \leq \tilde{a}_t \leq a_t$ for all t, which implies that $\lim_t \tilde{a}_t = a^*$. Similarly, the limit of $(\tilde{b}_t)_{t\geq 0}$ is b^*. □

Now we can work with the auxiliary sequences in order to identify necessary conditions for the limits a^* and b^*.

Lemma 7. *Limit properties: If $a_0 \geq \alpha^\dagger(b^*)$, then $a^* = a_0$. If $a_0 < \alpha^\dagger(b^*)$, then $a^* = 0$. If $\beta^\dagger(a^*) \geq b_0$, then $b^* = b_0$. If $\beta^\dagger(a^*) < b_0$, then $b^* = 1$.*

Proof. If $a_0 \geq \alpha^\dagger(b^*)$, by Assumption 4 and right continuity of $\alpha(\cdot, b^*)$, $\alpha(a_0, b^*) \geq a_0$. Then $\tilde{a}_1 = \overline{\alpha}(a_0, b^*) = a_0$. So, $a_0 = \tilde{a}_1 = ... = a^*$. So the limit is $a^* = a_0$.

If $a_0 < \alpha^\dagger(b^*)$, by Assumption 4, $\alpha(a_0, b^*) < a_0$. Then $\tilde{a}_1 = \overline{\alpha}(a_0, b^*) < a_0 < \alpha^\dagger(b^*)$ unless $a_0 = 0$. Similarly, $\tilde{a}_2 = \tilde{\alpha}(\tilde{a}_1, b^*) < \tilde{a}_1$, unless $\tilde{a}_1 = 0$. So $(\tilde{a}_t)_t$ strictly decreases

unless it hits 0. Recall that the limit of $\{\tilde{a}_t\}$ is a^*. Suppose that $a^* > 0$. Recall that α is right-continuous and increasing in a. Thus $\bar{\alpha}$ is right-continuous and increasing in a. Then $a^* = \lim_{t\to\infty} \tilde{a}_{t+1} = \lim_{t\to\infty} \bar{\alpha}(\tilde{a}_t, b^*) = \bar{\alpha}(a^*, b^*)$ and thus $a^* = \alpha(a^*, b^*)$. This is a contradiction since by $0 < a^* \leq a_0 < \alpha^*(b^*)$ and Assumption 4, $a^* > \alpha(a^*, b^*)$. Hence it must be $a^* = 0$.

Similarly, if $\beta(a^*, b_0) \leq b_0$, then $b^* = b_0$. If $\beta(a^*, b_0) > b_0$, then $b^* = 1$. □

Lemma 8. *Monotonicity of crossing functions: $\alpha^\dagger(b)$ and $\beta^\dagger(a)$ are increasing.*

Proof. If $b \geq b'$, then $\alpha(a, b) \leq \alpha(a, b')$. Under Assumption 4, this implies that $\alpha^\dagger(b) \geq \alpha^\dagger(b')$. This is, $\alpha^\dagger$ is increasing under Assumption 4. Similar arguments apply to $\beta^\dagger$. See Figure 14 for a visualization. □

Now we are ready to state the proof of Proposition 3 using Lemma 7 and 8:
Case 1
This can be proven as detailed after the statement of Proposition 3 in the main text.
Case 2 and Case 4a
Recall that $\beta^\dagger$ is an increasing function (Lemma 8) and $a^* \leq a_0$. Hence $\beta^\dagger(a^*) \leq \beta^\dagger(a_0)$. The condition $b_0 > \beta^\dagger(a_0)$ thus implies $b_0 > \beta^\dagger(a^*)$ which by Lemma 7 leads to $b^* = 1$. We now distinguish two cases.
Case 2: $a_0 \geq a^\dagger(1)$ implies $a_0 \geq a^\dagger(b^*)$ and by Lemma 7 $a^* = a_0$.
Case 4a: $a_0 < a^\dagger(1)$ implies $a_0 < a^\dagger(b^*)$ and by Lemma 7 $a^* = 0$.
Case 3 and Case 4b
Recall that $\alpha^\dagger$ is an increasing function and $b_0 \leq b^*$; hence $\alpha^\dagger(b_0) \leq \alpha^\dagger(b^*)$. The condition $a_0 < \alpha^\dagger(b_0)$ thus implies $a_0 < \alpha^\dagger(b^*)$ and by Lemma 7 we obtain $a^* = 0$. We now distinguish two cases.
Case 3: $b_0 \leq \beta^\dagger(0)$ implies $b_0 \leq \beta^\dagger(a^*)$ and by Lemma 7 we obtain $b^* = b_0$.
Case 4b: $b_0 > \beta^\dagger(0)$ implies $b_0 > \beta^\dagger(a^*)$ and by Lemma 7 we obtain $b^* = 1$.

C.9 Proof of Theorem 4

We distinguish two cases:

- Complete contagion ($m = 1$)
  Proposition 3 implies that complete contagion can happen in four cases.
  1. $a_0, b_0 \in [0, 1]$ and conditions of case 4a hold. In this case we define

$$\tilde{r}_1^{[4a]} := \begin{bmatrix} \inf\limits_{a_0,b_0\in[0,1]^2} & b_0 - a_0 \\ \text{s.t.} & 0 \leq a_0 < \alpha^\dagger(1) \\ & 1 \geq b_0 > \beta^\dagger(a_0) \\ & a_0 \leq b_0 \end{bmatrix} = \inf_{0\leq a_0<\alpha^\dagger(1)} \left(\inf_{1\geq b_0>\beta^\dagger(a_0), b_0\geq a_0} b_0 - a_0 \right) \tag{C.8}$$

$$= \inf_{0\leq a_0<\alpha^\dagger(1)} \left(\beta^\dagger(a_0) - a_0\right)^+ = \inf_{0\leq x<\alpha^\dagger(1)} \left(\beta^\dagger(x) - x\right)^+. \tag{C.9}$$

  2. $a_0, b_0 \in [0, 1]$ and conditions of case 4b hold

In this case we define

$$\tilde{r}_1^{[4b]} := \begin{bmatrix} \inf\limits_{a_0,b_0\in[0,1]^2} & b_0 - a_0 \\ \text{s.t.} & 0 \le a_0 < \alpha^\dagger(b_0) \\ & 1 \ge b_0 > \beta^\dagger(0) \\ & a_0 \le b_0 \end{bmatrix} = \inf_{1\ge b_0>\beta^\dagger(0)} \left(\inf_{0\le a_0<\alpha^\dagger(b_0), a_0\le b_0} b_0 - a_0 \right) \tag{C.10}$$

$$= \inf_{1\ge b_0>\beta^\dagger(0)} \Big(b_0 - \alpha^\dagger(b_0)\Big)^+ = \inf_{1\ge y>\beta^\dagger(0)} \Big(y - \alpha^\dagger(y)\Big)^+. \tag{C.11}$$

3. $a_0 = 0$ and conditions of case 2 hold
   This can only happen if $\alpha^\dagger(1) = 0$. In this case we define

$$\tilde{r}_1^{[2]} := \inf_{b_0>\beta^\dagger(0)} b_0 = \beta^\dagger(0) \ge \tilde{r}_1^{[4b]}. \tag{C.12}$$

4. $b_0 = 1$ and conditions of case 3 hold
   This can only happen if $\beta^\dagger(0) = 1$. In this case we define

$$\tilde{r}_1^{[3]} := \inf_{a_0<\alpha^\dagger(1)} 1 - a_0 = 1 - \alpha^\dagger(1) \ge \tilde{r}_1^{[4a]}. \tag{C.13}$$

Hence $\tilde{r}_1^* = \min\{\tilde{r}_1^{[2]}, \tilde{r}_1^{[3]}, \tilde{r}_1^{[4a]}, \tilde{r}_1^{[4b]}\} = \min\{\tilde{r}_1^{[4a]}, \tilde{r}_1^{[4b]}\}$.

- Partial contagion ($m < 1$)
  Proposition 3 implies that partial contagion can happen in three cases.

  1. *The conditions of case 2 hold:*
     In this case $a^* = a_0$ and $b^* = 1$. Suppose first that we want to have final contagion of exactly m' (instead of $\ge m$). We can then define

$$\tilde{r}_{m'}^{[2]} := \begin{bmatrix} \inf\limits_{a_0,b_0\in[0,1]^2} & b_0 - a_0 \\ \text{s.t.} & 1 - a_0 = m' \\ & a_0 \ge \alpha^\dagger(1) \\ & b_0 > \max\{a_0, \beta^\dagger(a_0)\} \end{bmatrix}. \tag{C.14}$$

     Note that this problem is well-defined only if $1 - m' \ge \alpha^\dagger(1)$. In this case

$$\tilde{r}_{m'}^{[2]} = \begin{bmatrix} \inf\limits_{b_0\in[0,1]} & b_0 - (1 - m') \\ \text{s.t.} & b_0 > \max\{1 - m', \beta^\dagger(1 - m')\} \end{bmatrix} = \max\{0, \beta^\dagger(1-m') - (1-m')\}. \tag{C.15}$$

Then

$$\tilde{r}_m^{[2]} := \begin{bmatrix} \inf\limits_{a_0,b_0\in[0,1]^2} & b_0 - a_0 \\ \text{s.t.} & 1 - a_0 \geq m \\ & a_0 \geq \alpha^\dagger(1) \\ & b_0 > \max\{a_0, \beta^\dagger(a_0)\} \end{bmatrix} = \inf_{1-\alpha^\dagger(1)\geq m'\geq m} \tilde{r}_{m'}^{[2]}$$
$$= \inf_{1-\alpha^\dagger(1)\geq m'\geq m} \max\{0, \beta^\dagger(1-m') - (1-m')\}$$
$$= \inf_{\alpha^\dagger(1)\leq x\leq 1-m} \max\{0, \beta^\dagger(x) - x\}.$$

2. *The conditions of case 3 hold:*
   With the same arguments as above it can be shown

$$\tilde{r}_m^{[3]} := \begin{bmatrix} \inf\limits_{a_0,b_0\in[0,1]^2} & b_0 - a_0 \\ \text{s.t.} & b_0 \geq m \\ & a_0 < \min\{b_0, \alpha^\dagger(b_0)\} \\ & b_0 \leq \beta^\dagger(0) \end{bmatrix}$$
$$= \inf_{m\leq y\leq \beta^\dagger(0)} \Big(y - \alpha^\dagger(y)\Big)^+.$$

3. *The conditions of case 1 hold:*
   In this case, the initial seed set has measure m

Overall $\tilde{r}_m^* = \min\{m, \tilde{r}_m^{[2]}, \tilde{r}_m^{[3]}, \tilde{r}_1^{[4a]}, \tilde{r}_1^{[4b]}\} = \min\{\tilde{r}_m^{[2]}, \tilde{r}_m^{[3]}, \tilde{r}_1^{[4a]}, \tilde{r}_1^{[4b]}\}$, which leads to (B.3).

We next compute the limit-optimal set. First, for some $A \subset [0,1]$ and a function $g : [0,1] \to \mathbb{R}$, define arg inf operator as follows. $x^* \in \arg\inf_{x\in A} g(x)$ if there exists a sequence $(x_t)_t$ in A such that $\lim_t x_t = x^*$ and $\lim_t g(x_t) = \inf_{x\in A} g(x)$. Note that such x^* belongs to $[0,1]$ but does not necessarily belongs to A. Moreover, $\arg\inf_{x\in A} g(x)$ is non-empty. This can be seen as follows. There exists a sequence $(\tilde{x}_t)_t$ in A such that $\lim_t g(\tilde{x}_t) = \inf_{x\in A} g(x)$. The interval $[0,1]$ is compact, so $(\tilde{x}_t)$ has a convergent subsequence, say $(x_t)_t$. Then $\lim_t x_t \in \arg\inf_{x\in A} g(x)$. Notice that the sequence (x_t) in the definition of arg inf can be taken to be monotonic without loss of generality. arg sup can be defined analogously.

First consider the case of $\tilde{r}_m^* = \tilde{r}_1^{[4a]}$. Take any $a_0^* \in \arg\inf_{0\leq x<\alpha^\dagger(1)} \big(\beta^\dagger(x) - x\big)^+$. Take a monotonic sequence $(\tilde{a}_0^t)_t$ in $[0, \alpha^\dagger(1))$ with $(\tilde{a}_0^t)_t \to a_0^*$ and $\big(\beta^\dagger(\tilde{a}_0^t) - \tilde{a}_0^t\big)^+ \to \tilde{r}_1^{[4a]}$. Set $b_0^t = a_0^* + \tilde{r}_1^{[4a]} + \epsilon_t$ for some $\epsilon_t \downarrow 0$. Then

$$b_0^t > a_0^* + \tilde{r}_1^{[4a]} = \lim_{t'} \left(\tilde{a}_{t'} + \Big(\beta^\dagger(\tilde{a}_0^{t'}) - \tilde{a}_{t'}\Big)^+\right) \geq \lim_{t'} \beta^\dagger(\tilde{a}_0^{t'}).$$

If $(\tilde{a}_0^t)_t \uparrow a_0^*$, then set $a_0^t = \tilde{a}_0^t$. Then we have $a_0^t = \tilde{a}_0^t < \alpha^\dagger(1)$ and $b_0^t > \lim_{t'} \beta^\dagger(\tilde{a}_0^{t'}) \geq \beta^\dagger(\tilde{a}_0^t)$ because $\beta^\dagger$ is an increasing function (Lemma 8) and $(\tilde{a}_{t'})_{t'}$ is an increasing sequence. If $\tilde{a}_t \downarrow a_0^*$, then set $a_0^t = a_0^*$. Then we have $a_0^t = a_0^* \leq \tilde{a}_0^1 < \alpha^\dagger(1)$ and $b_0^t > \lim_{t'} \beta^\dagger(\tilde{a}_0^{t'}) \geq \beta^\dagger(a_0^*) = \beta^\dagger(a_0^t)$ because $\beta^\dagger$ is an increasing function and $(\tilde{a}_0^{t'})_{t'}$

is a decreasing sequence with limit $a_0^* = a_0^t$. In both cases, we have $a_0^t < \alpha^\dagger(1)$ and $b_0^t > \beta^\dagger(a_0^t)$. Thus, $f(([a_0^t, b_0^t])_t) = 1$ (by Proposition 3). Also, in both cases $([a_0^t, b_0^t])_t$ is a decreasing nested sequence with intersection $[a_0^*, a_0^* + \tilde{r}_1^{[4a]}]$, which has measure $\tilde{r}_1^{[4a]}$. Then by definition, $[a_0^*, a_0^* + \tilde{r}_1^{[4a]}]$ is limit-optimal for min-seed.

Next consider the case of $\tilde{r}_m^* = \tilde{r}_1^{[4b]}$.Take $b_0^* \in \arg\inf_{1 \geq y > \beta^\dagger(0)} \left(y - \alpha^\dagger(y)\right)^+$. Take a monotonic sequence $(\tilde{b}_0^t)_t$ in $(\beta^\dagger(0), 1]$ with $\tilde{b}_0^t \to b_0^*$ and $\left(\tilde{b}_0^t - \alpha^\dagger(\tilde{b}_0^t)\right)^+ \to \tilde{r}_1^{[4b]}$. Set $a_0^t = b_0^* - \tilde{r_1}^{[4b]} - \epsilon_t$ for some $\epsilon_t \downarrow 0$. Then we have

$$a_0^t < b_0^* - \tilde{r}_1^{[4b]} = \lim_{t'} \left(\tilde{b}_0^{t'} - \left(\tilde{b}_0^{t'} - \alpha^\dagger(\tilde{b}_0^{t'}) \right)^+ \right) \leq \lim_{t'} \alpha^\dagger(\tilde{b}_0^{t'}).$$

If $\tilde{b}_0^t \downarrow b_0^*$, set $b_0^t = \tilde{b}_0^t$. Then we have $b_0^t = \tilde{b}_0^t > \beta^\dagger(0)$ and $a_0^t < \lim_{t'} \alpha^\dagger(\tilde{b}_0^{t'}) \leq \alpha^\dagger(b_0^t)$ because $\alpha^\dagger$ is an increasing function (Lemma 8) and $(\tilde{b}_0^{t'})_{t'}$ is a decreasing sequence. If $\tilde{b}_0^t \uparrow b_0^*$, set $b_0^t = b_0^*$. Then we have $b_0^t = b_0^* \geq \tilde{b}_0^1 > \beta^\dagger(0)$ and $a_0^t < \lim_{t'} \alpha^\dagger(\tilde{b}_0^{t'}) \leq \alpha^\dagger(b_0^*) = \alpha^\dagger(b_0^t)$ because $\alpha^\dagger$ is an increasing function and $(\tilde{b}_0^{t'})_{t'}$ is an increasing sequence with limit $b_0^* = b_0^t$. In both cases we have $b_0^t > \beta^\dagger(0)$ and $a_0^t < \alpha^\dagger(b_0^t)$. Thus, $f(([a_0^t, b_0^t])_t) = 1$ (by Proposition 3). Also, in both cases $([a_0^t, b_0^t])_t$ is a decreasing nested sequence with intersection $[b_0^* - \tilde{r}_1^{[4a]}, b_0^*]$, which has measure $\tilde{r}_1^{[4b]}$. Then by definition, $[b_0^* - \tilde{r}_1^{[4a]}, b_0^*]$ is limit-optimal for min-seed.

Consider the case of $\tilde{r}_m^* = \tilde{r}_m^{[2]}$. Take any $a_0^{**} \in \arg\inf_{\alpha^\dagger(1) \leq x \leq 1-m} \left(\beta^\dagger(x) - x\right)^+$. Take a monotonic sequence $(\tilde{a}_0^t)_t$ in $[\alpha^\dagger(1), 1-m]$ with $(\tilde{a}_0^t)_t \to a_0^{**}$ and $\left(\beta^\dagger(\tilde{a}_0^t) - \tilde{a}_0^t\right)^+ \to \tilde{r}_m^{[2]}$. Note that $a_0^{**} \in [\alpha^\dagger(1), 1-m]$. Set $b_0^t = a_0^{**} + \tilde{r}_m^{[2]} + \epsilon_t$ for some $\epsilon_t \downarrow 0$. Then

$$b_0^t > a_0^{**} + \tilde{r}_m^{[2]} = \lim_{t'} \left(\tilde{a}_{t'} + \left(\beta^\dagger(\tilde{a}_0^{t'}) - \tilde{a}_{t'} \right)^+ \right) \geq \lim_{t'} \beta^\dagger(\tilde{a}_0^{t'}.$$

If $(\tilde{a}_0^t)_t \uparrow a_0^{**}$, then set $a_0^t = \tilde{a}_0^t$. Then $b_0^t > \lim_{t'} \beta^\dagger(\tilde{a}_0^{t'}) \geq \beta^\dagger(\tilde{a}_0^t)$ because $\beta^\dagger$ is an increasing function and $(\tilde{a}_{t'})_{t'}$ is an increasing sequence. If $\tilde{a}_t \downarrow a_0^{**}$, then set $a_0^t = a_0^{**}$. Then we have $b_0^t > \lim_{t'} \beta^\dagger(\tilde{a}_0^{t'}) \geq \beta^\dagger(a_0^{**}) = \beta^\dagger(a_0^t)$ because $\beta^\dagger$ is an increasing function and $(\tilde{a}_0^{t'})_{t'}$ is a decreasing sequence with limit $a_0^{**} = a_0^t$). In both cases, we have $a_0^t \in [\alpha^\dagger(1), 1-m]$ and $b_0^t > \beta^\dagger(a_0^t)$. Thus, $f(([a_0^t, b_0^t])_t) = 1 - a_0^t$ (by Proposition 3). In both cases, $1 - a_0^t \geq 1 - a_0^{**} \geq m$, and so $f(([a_0^t, b_0^t])_t) \geq m$. Also, in both cases $([a_0^t, b_0^t])_t$ is a decreasing nested sequence with intersection $[a_0^{**}, a_0^{**} + \tilde{r}_m^{[2]}]$, which has measure $\tilde{r}_m^{[2]}$. Then by definition, $[a_0^{**}, a_0^{**} + \tilde{r}_m^{[2]}]$ is limit-optimal for min-seed

Finally consider the case of $\tilde{r}_m^* = \tilde{r}_m^{[3]}$. Take $b_0^{**} \in \arg\inf_{m \leq y \leq \beta^\dagger(0)} \left(y - \alpha^\dagger(y)\right)^+$. Take a monotonic sequence $(\tilde{b}_0^t)_t$ in $[m, \beta^\dagger(0)]$ with $\tilde{b}_0^t \to b_0^{**}$ and $\left(\tilde{b}_0^t - \alpha^\dagger(\tilde{b}_0^t)\right)^+ \to \tilde{r}_m^{[3]}$. Note that $b_0^{**} \in [m, \beta^\dagger(0)]$ .Set $a_0^t = b_0^{**} - \tilde{r}_m^{[3]} - \epsilon_t$ for some $\epsilon_t \downarrow 0$. Then we have

$$a_0^t < b_0^{**} - \tilde{r}_m^{[3]} = \lim_{t'} \left(\tilde{b}_0^{t'} - \left(\tilde{b}_0^{t'} - \alpha^\dagger(\tilde{b}_0^{t'}) \right)^+ \right) \leq \lim_{t'} \alpha^\dagger(\tilde{b}_0^{t'}).$$

If $\tilde{b}_0^t \downarrow b_0^{**}$, set $b_0^t = \tilde{b}_0^t$. Then we have $a_0^t < \lim_{t'} \alpha^\dagger(\tilde{b}_0^{t'}) \leq \alpha^\dagger(b_0^t)$ because $\alpha^\dagger$ is an increasing function and $(\tilde{b}_0^{t'})_{t'}$ is a decreasing sequence. If $\tilde{b}_0^t \uparrow b_0^{**}$, set $b_0^t = b_0^{**}$. Then we have $a_0^t < \lim_{t'} \alpha^\dagger(\tilde{b}_0^{t'}) \leq \alpha^\dagger(b_0^{**}) = \alpha^\dagger(b_0^t)$ because $\alpha^\dagger$ is an increasing function and $(\tilde{b}_0^{t'})_{t'}$ is an increasing sequence with limit $b_0^{**} = b_0^t$. In both cases we have $b_0^t \in$

$[m, \beta^\dagger(0)]$ and $a_0^t < \alpha^\dagger(b_0^t)$. Thus, $f(([a_0^t, b_0^t])_t) = b_0^t$ (by Proposition 3). In both cases, $b_0^t \geq b_0^{**} \geq m$, and so $f(([a_0^t, b_0^t])_t) \geq m$. Also, in both cases $([a_0^t, b_0^t])_t$ is a decreasing nested sequence with intersection $[b_0^{**} - \tilde{r}_m^{[3]}, b_0^{**}]$, which has measure $\tilde{r}_m^{[3]}$. Then by definition, $[b_0^{**} - \tilde{r}_m^{[3]}, b_0^{**}]$ is limit-optimal for min-seed.

C.10 Proof of Corollary 1

Degrees and cutoff iterators

Consider the GUA graphon $W_{GUA}(u, v) = 1 - \max\{u, v\}$.

$$\begin{aligned} d(u) &= \int_0^1 W_{GUA}(u, v)dv = \int_0^1 (1 - \max\{u, v\})\, dv \\ &= \int_0^u (1 - u)\, dv + \int_u^1 (1 - v)\, dv \\ &= u(1 - u) + \frac{1}{2}(1 - u)^2 = \frac{1}{2}(1 - u^2). \end{aligned}$$

For $u \leq a$,

$$d(u, (a, b)) = \int_a^b (1 - \max\{u, v\})\, dv = \int_a^b (1 - v)\, dv = \frac{1}{2}((1 - a)^2 - (1 - b)^2)$$

$$\begin{aligned} d(u, (a, b)) > \tau d(u)((1 - a)^2 - (1 - b)^2) &> \tau(1 - u^2) \\ \iff u^2 &> 1 - \tau^{-1}((1 - a)^2 - (1 - b)^2) \\ \iff u &> \sqrt{\max\{0, 1 - \tau^{-1}((1 - a)^2 - (1 - b)^2)\}} = \alpha(a, b) \end{aligned}$$

For $u \geq b$,

$$d(u, (a, b)) = \int_a^b (1 - \max\{u, v\})\, dv = \int_a^b (1 - u)\, dv = (1 - u)(b - a)$$

$$\begin{aligned} d(u, (a, b)) > \tau d(u)(1 - u)(b - a) &> \frac{\tau}{2}(1 - u^2) \\ \iff u &< 2(b - a)\tau^{-1} - 1 \\ \iff u &< \min\left\{1, \max\left\{0, 2(b - a)\tau^{-1} - 1\right\}\right\} = \beta(a, b) \end{aligned}$$

Crossing functions

For any $a, b \in [0, 1]$

$$\begin{aligned} \alpha(a, b) < a &\iff 1 - \tau^{-1}((1 - a)^2 - (1 - b)^2) < a^2 \\ &\iff \tau(1 - a^2) < (1 - a)^2 - (1 - b)^2 \\ &\iff 0 < (1 + \tau)a^2 - 2a + \left(1 - \tau - (1 - b)^2\right) \\ &\iff \left[a < \frac{1 - \sqrt{\tau^2 + (1 + \tau)(1 - b)^2}}{1 + \tau}\right] \vee \left[a > \frac{1 + \sqrt{\tau^2 + (1 + \tau)(1 - b)^2}}{1 + \tau}\right] \end{aligned}$$

Hence

$$\alpha^{\dagger}(b) = \max\left\{0, \frac{1-\sqrt{\tau^2+(1+\tau)(1-b)^2}}{1+\tau}\right\}$$

Moreover

$$\begin{aligned}\beta(a,b) < b &\iff 2(b-a)\tau^{-1} - 1 < b \\ &\iff \left(2\tau^{-1}-1\right)b < 1 + 2\tau^{-1}a \\ &\iff b < \frac{1+2\tau^{-1}a}{2\tau^{-1}-1}\end{aligned}$$

Hence

$$\beta^{\dagger}(a) = \min\left\{1, \frac{2a+\tau}{2-\tau}\right\}$$

Therefore, α and β both satisfy single-crossing.

The solution to interval min-seed

$$\begin{aligned}\tilde{r}^*_m &= \min\left\{\inf_{0\le x<\max\left\{1-m,\alpha^{\dagger}(1)\right\}}\left(\beta^{\dagger}(x)-x\right)^+, \inf_{1\ge x>\min\left\{m,\beta^{\dagger}(0)\right\}}\left(x-\alpha^{\dagger}(x)\right)^+\right\} \\ &= \min\left\{\inf_{1\ge x>\min\left\{m,1-\alpha^{\dagger}(1)\right\}}\left(\beta^{\dagger}(1-x)-(1-x)\right)^+, \inf_{1\ge x>\min\left\{m,\beta^{\dagger}(0)\right\}}\left(x-\alpha^{\dagger}(x)\right)^+\right\} \\ &= \min\left\{\inf_{1\ge x>\min\left\{m,\frac{2\tau}{1+\tau}\right\}}\min\left\{x, \frac{\tau}{2-\tau}(2-x)\right\}, \inf_{1\ge x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\left(x-\alpha^{\dagger}(x)\right)^+\right\} \\ &= \min\left\{\min\left\{\min\left\{m,\frac{2\tau}{1+\tau}\right\},\frac{\tau}{2-\tau}\right\}, \inf_{1\ge x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\left(x-\alpha^{\dagger}(x)\right)^+\right\} \\ &= \min\left\{m, \frac{\tau}{2-\tau}, \inf_{1\ge x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\left(x-\alpha^{\dagger}(x)\right)^+\right\} \\ &= \inf_{x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\left(x-\alpha^{\dagger}(x)\right)^+.\end{aligned}$$

This implies that the optimal seed always involves left contagion or no contagion.

$$\begin{aligned}\tilde{r}^*_m &= \inf_{x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\left(x-\alpha^{\dagger}(x)\right)^+ \\ &= \inf_{x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\left(x-\max\left\{0,\frac{1-\sqrt{\tau^2+(1+\tau)(1-x)^2}}{1+\tau}\right\}\right)^+ \\ &= \inf_{x>\min\left\{m,\frac{\tau}{2-\tau}\right\}}\begin{cases}x-\frac{1-\sqrt{\tau^2+(1+\tau)(1-x)^2}}{1+\tau} & x\ge 1-\sqrt{1-\tau} \\ x & x<1-\sqrt{1-\tau}\end{cases}.\end{aligned}$$

Note that x and $x-\frac{1-\sqrt{\tau^2+(1+\tau)(1-x)^2}}{1+\tau}$ are both increasing functions. Thus

$$\tilde{r}_m^* = \begin{cases} \frac{\tau}{2-\tau} - \frac{1-\sqrt{\tau^2+(1+\tau)(1-\frac{\tau}{2-\tau})^2}}{1+\tau} & m \geq \frac{\tau}{2-\tau} \\ m - \frac{1-\sqrt{\tau^2+(1+\tau)(1-m)^2}}{1+\tau} & \frac{\tau}{2-\tau} > m \geq 1-\sqrt{1-\tau} \\ m & 1-\sqrt{1-\tau} > m \end{cases} \tag{C.16}$$

Resilience and complete contagion

We can see that the resilience is $\tilde{r}_1^* = \frac{\tau}{2-\tau} - \frac{1-\sqrt{\tau^2+(1+\tau)(1-\frac{\tau}{2-\tau})^2}}{1+\tau}$. The corresponding limit-optimal seed is

$$\left(\frac{1-\sqrt{\tau^2+(1+\tau)\left(1-\frac{\tau}{2-\tau}\right)^2}}{1+\tau}, \frac{\tau}{2-\tau} \right).$$

Partial contagion

It is not possible to have contagion of exact size $m' \in (\frac{\tau}{2-\tau}, 1)$. Hence $\frac{\tau}{2-\tau}$ is a discontinuity point in terms of the size of contagion.

Contagion of exact size $m' \in \left(1-\sqrt{1-\tau}, \tau\right)$ is achieved with the limit-optimal seed set

$$\left(\frac{1-\sqrt{\tau^2+(1+\tau)\left(1-m'\right)^2}}{1+\tau}, m' \right).$$

Contagion of exact size $m' < 1-\sqrt{1-\tau}$ is possible only when there is no contagion. This can be achieved with any seed of size m'.

Interval max-reach

- If $\rho > \frac{\tau}{2-\tau} - \frac{1-\sqrt{\tau^2+(1+\tau)(1-\frac{\tau}{2-\tau})^2}}{1+\tau}$, by equation (C.16), $\tilde{f}_\rho^* = 1$.
- If $\frac{\tau}{2-\tau} - \frac{1-\sqrt{\tau^2+(1+\tau)(1-\frac{\tau}{2-\tau})^2}}{1+\tau} > \rho > 1-\sqrt{1-\tau}$, then $\tilde{f}_\rho^*$ is given by $\rho = \tilde{f}_\rho^* - \frac{1-\sqrt{\tau^2+(1+\tau)(1-\tilde{f}_\rho^*)^2}}{1+\tau}$, which is equivalent to

$$\tilde{f}_\rho^* = \frac{\rho(1+\tau) - \sqrt{(\rho-\tau)^2+\rho^2\tau}}{\tau}$$

- If $\rho < 1-\sqrt{1-\tau}$, then $\tilde{f}_\rho^* = \rho$ for any seed.

C.11 Proof of Corollary 2

Degrees and cutoff iterators

$W_{MM}(u,v) = \min\{u,v\}(1-\max\{u,v\})$.

$$\begin{aligned} d(u) &= \int_0^1 W_{MM}(u,v)dv = \int_0^1 \min\{u,v\}(1-\max\{u,v\})\,dv \\ &= \int_0^u \min\{u,v\}(1-\max\{u,v\})\,dv + \int_u^1 \min\{u,v\}(1-\max\{u,v\})\,dv \\ &= \int_0^u v(1-u)\,dv + \int_u^1 u(1-v)\,dv = \frac{u^2}{2}(1-u) + u\frac{(1-u)^2}{2} = \frac{u(1-u)}{2}. \end{aligned}$$

For $u \le a$,

$$d(u,(a,b)) = \int_a^b \min\{u,v\}\,(1-\max\{u,v\})\,dv = \int_a^b u\,(1-v)\,dv = u\frac{(1-a)^2-(1-b)^2}{2}$$

$$d(u,(a,b)) < \tau d(u) \iff u < 1-\tau^{-1}((1-a)^2-(1-b)^2)$$

Hence

$$\alpha(a,b) = \max\left\{0, 1-\tau^{-1}((1-a)^2-(1-b)^2)\right\}.$$

For $u \ge b$,

$$d(u,(a,b)) = \int_a^b \min\{u,v\}\,(1-\max\{u,v\})\,dv = \int_a^b v\,(1-u)\,dv = (1-u)\,\frac{b^2-a^2}{2}$$

$$d(u,(a,b)) > \tau d(u) \iff u < \tau^{-1}(b^2-a^2)$$

Hence

$$\beta(a,b) = \min\left\{1, \tau^{-1}(b^2-a^2)\right\}.$$

Crossing functions

For all $a, b \in [0,1]$,

$$\begin{aligned}
\alpha(a,b) < a &\iff 1-\tau^{-1}((1-a)^2-(1-b)^2) < a \\
&\iff 0 < (1-a)^2 - \tau(1-a) - (1-b)^2 \\
&\iff \left[1-a < \frac{1}{2}\left(\tau - \sqrt{\tau^2+4(1-b)^2}\right)\right] \vee \left[1-a > \frac{1}{2}\left(\tau + \sqrt{\tau^2+4(1-b)^2}\right]\right) \\
&\iff 1-a > \frac{1}{2}\left(\tau + \sqrt{\tau^2+4(1-b)^2}\right) \\
&\iff a < 1-\frac{1}{2}\left(\tau + \sqrt{\tau^2+4(1-b)^2}\right)
\end{aligned}$$

hence

$$\alpha^\dagger(b) = \max\left\{0, 1-\frac{1}{2}\left(\tau + \sqrt{\tau^2+4(1-b)^2}\right)\right\}$$

and

$$\begin{aligned}
\beta(a,b) < b &\iff \tau^{-1}(b^2-a^2) < b \\
&\iff b^2 - \tau b - a^2 < 0 \\
&\iff \left[b > \frac{1}{2}\left(\tau - \sqrt{\tau^2+4a^2}\right)\right] \wedge \left[b < \frac{1}{2}\left(\tau + \sqrt{\tau^2+4a^2}\right)\right] \\
&\iff b < \frac{1}{2}\left(\tau + \sqrt{\tau^2+4a^2}\right) = \beta^\dagger(a)
\end{aligned}$$

hence

$$\beta^\dagger(a) = \min\left\{1, \frac{1}{2}\left(\tau + \sqrt{\tau^2 + 4a^2}\right)\right\}.$$

Therefore, α and β both satisfy single-crossing.

The solution to the interval min-seed

In general, we have

$$\tilde{r}^*_m = \min\left\{\inf_{1\geq x>\min\left\{m,1-\alpha^\dagger(1)\right\}}\left(\beta^\dagger(1-x)-(1-x)\right)^+, \inf_{1\geq x>\min\left\{m,\beta^\dagger(0)\right\}}\left(x-\alpha^\dagger(x)\right)^+\right\}$$

Note that $\inf_{x>\min\left\{m,1-\alpha^\dagger(1)\right\}}\left(\beta^\dagger(1-x)-(1-x)\right)^+$ and $\inf_{x>\min\left\{m,\beta^\dagger(0)\right\}}\left(x-\alpha^\dagger(x)\right)^+$ are identical problems (as the MM graphon is symmetric). So

$$\begin{aligned}
\tilde{r}^*_m &= \inf_{1\geq x>\min\left\{m,\beta^\dagger(0)\right\}}\left(x-\alpha^\dagger(x)\right)^+ \\
&= \inf_{1\geq x>\min\{m,\tau\}}\left(x-\max\left\{0,1-\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-x)^2}\right)\right\}\right)^+ \\
&= \inf_{1\geq x>\min\{m,\tau\}}\left(\begin{cases}\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-x)^2}\right)-(1-x) & \text{if } x>1-\sqrt{1-\tau}\\ x & \text{if } x<1-\sqrt{1-\tau}\end{cases}\right)
\end{aligned}$$

Both x and $\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-x)^2}\right)-(1-x)$ are increasing in x. So

$$\tilde{r}^*_m = \begin{cases}\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tau)^2}\right)-(1-\tau) & \text{if } m>\tau\\ \frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-m)^2}\right)-(1-m) & \text{if } \tau>m>1-\sqrt{1-\tau}\\ m & \text{if } 1-\sqrt{1-\tau}>m\end{cases} \tag{C.17}$$

Resilience and complete contagion

We can see that the resilience is $\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tau)^2}\right)-(1-\tau)$. The two following sets are both limit-optimal seeds

$$\left(1-\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tau)^2}\right),\tau\right)$$

$$\left(1-\tau,\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tau)^2}\right)\right).$$

Partial contagion

It is not possible to have contagion of exact size $m'\in(\tau,1)$. Hence τ is a discontinuity point in terms of the size of contagion.

Contagion of exact size $m'\in\left(1-\sqrt{1-\tau},\tau\right)$ is achieved optimally with either one of the two limit-optimal seeds

$$\left(1-\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-m')^2}\right),m'\right)$$

$$\left(1-m', \frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-m')^2}\right)\right)$$

Contagion of exact size $m' < 1-\sqrt{1-\tau}$ is possible only when there is no contagion. This can be achieved with any seed of size m'.

Interval max-reach

- If $\rho > \frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tau)^2}\right)-(1-\tau)$, by equation (C.17), $\tilde{f}^*_\rho = 1$.
- If $\frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tau)^2}\right)-(1-\tau) > \rho > 1-\sqrt{1-\tau}$, then $\tilde{f}^*_\rho$ is given by $\rho = \frac{1}{2}\left(\tau+\sqrt{\tau^2+4(1-\tilde{f}^*_\rho)^2}\right)-(1-\tilde{f}^*_\rho)$, which is equivalent to $\tilde{f}^*_\rho = 1-\frac{\rho(\tau-\rho)}{2\rho-\tau}$.
- If $\rho < 1-\sqrt{1-\tau}$, then $\tilde{f}^*_\rho = \rho$ for any seed.

D Contagion in graphons with heterogeneous thresholds

Here, we consider contagion in an Erdős-Rényi graphon in which the threshold function is $\tau(u) = u$. In this case, the threshold of every label is heterogeneous. Moreover, in a sampled network, this corresponds to the case in which agents' thresholds are drawn uniformly at random as in Kempe et al. (2003) and Lim et al. (2016). We start by calculating the fraction of labels that will be infected in step t.

Initial seed: Let $C := \mu(C_0)$ denote the measure of the set C_0. Then a fraction C of labels is infected initially, and a fraction $1-C$ of labels remains uninfected. Let us focus on x_t, the fraction of the initially unseeded labels that become infected. At the end of the initial step, we have that $x_0 = 0$.

Step 1: Since $\int_{C_0} pdv / \int_0^1 pdv = \mu(C_0)$, all unseeded labels with a threshold less than $\mu(C_0) = C$ are infected in the first step. Recall that thresholds are uniformly distributed in $[0,1]$, hence the probability that a label has a threshold less than C is simply C. Since there is a fraction of $1-C$ labels that are not in the seed set, we can expect a fraction of $x_1 = C(1-C)$ labels to be newly infected in Step 1. Therefore, there is a fraction of $C+x_1$ infected labels in total at the end of Step 1.

Step 2: All unseeded labels with a threshold less than $\mu(C_1) = C+x_1$ get infected. Again the probability of having such a threshold is $C+x_1$ and there are $1-C$ candidates (note that here we do not distinguish whether a label was already infected in Step 1). Hence, there is a fraction of $x_2 = (C+x_1)(1-C)$ labels that were not in the seed set and are infected in Step 2. Therefore, there is a fraction of $C+x_2$ infected labels in total at the end of Step 2.

$\vdots$

Step t: All remaining labels with a threshold less than $\mu(C_{t-1}) = C+x_{t-1}$ get infected. Hence, there is a fraction of $x_t = (C+x_{t-1})(1-C)$ labels that were not in the seed set and are infected in Step t. Therefore, there is a fraction of $C+x_t$ infected labels in total at the end of Step t. Figure 15 illustrates the first three steps of the process when $C = \frac{1}{2}$. Note that the contagion process in the graphon does not depend on p in this example (consistent with the fact that p simplifies in Eq. (2.3)).

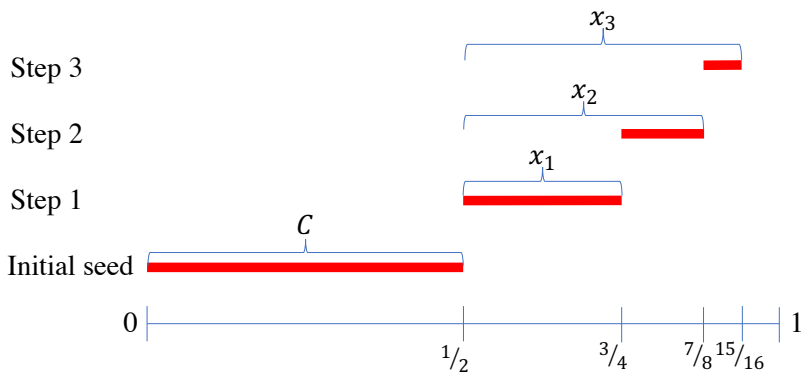

Figure 15: First three steps of contagion in the graphon with $C = \frac{1}{2}$.

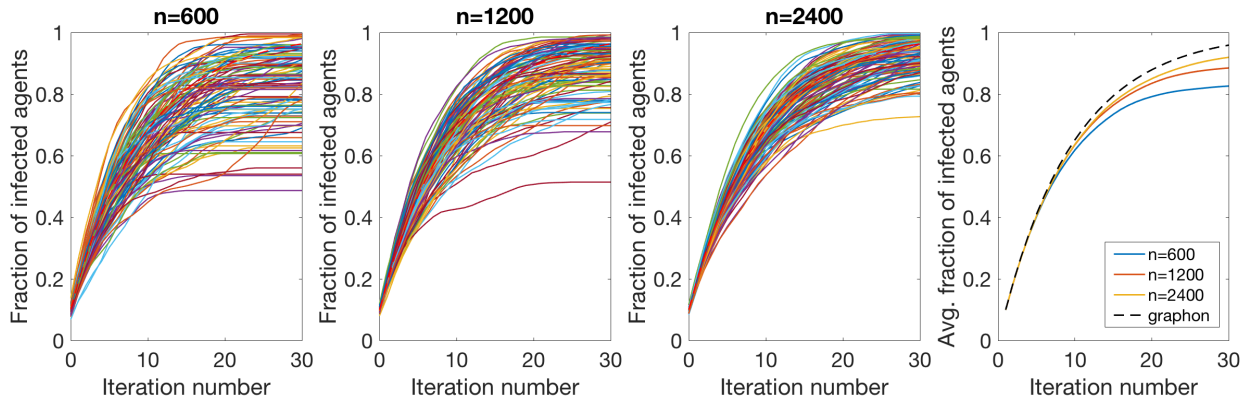

Figure 16: Comparison between contagion in networks with n agents sampled from a Erdős-Rényi model (with $p = 0.1$ and $C = 0.1$) for different values of n and contagion computed according to formula (D.1) . For each value of n we show the evolution of the fraction of infected individuals in each of the 100 repetitions (three plots on the left) and the average evolution compared with formula (D.1) (right).

More generally, we can write

$$x_t = (1 - C)(x_{t-1} + C),$$

with $x_0 = 0$. We can solve this recursive equation to obtain that

$$x_t = (1 - C)(1 - (1 - C)^t)$$

of the initially unseeded labels are infected at time t. Therefore, the total fraction of labels that are infected at time t in the infinite population limit is

$$C + x_t = 1 - (1 - C)^{t+1}. \tag{D.1}$$

In our approximation, as $t \to \infty$, the fraction of infected labels goes to 1 for any positive fraction of initial seeds and for any $p \in (0, 1]$.

Figure 16 (right) compares the contagion process averaged across 100 sampled networks to our analytical approximation for a continuum of labels. We can see that the approximation works well for reasonable values of p and number n of agents and accurately captures most of the dynamics of contagion.