Bootstrap Percolation on Geometric Inhomogeneous Random Graphs*

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Abstract

Geometric inhomogeneous random graphs (GIRGs) are a model for scale-free networks with underlying geometry. We study bootstrap percolation on these graphs, which is a process modelling the spread of an infection of vertices starting within a (small) local region. We show that the process exhibits a phase transition in terms of the initial infection rate in this region. We determine the speed of the process in the supercritical case, up to lower order terms, and show that its evolution is fundamentally influenced by the underlying geometry. For vertices with given position and expected degree, we determine the infection time up to lower order terms. Finally, we show how this knowledge can be used to contain the infection locally by removing relatively few edges from the graph. This is the first time that the role of geometry on bootstrap percolation is analysed mathematically for geometric scale-free networks.

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1 Introduction

One of the most challenging and intriguing questions about large real-world networks is how activity spreads through the network. "Activity" in this context can mean many things, including infections in a population network, opinions and rumours in social networks, viruses in computer networks, action potentials in neural networks, and many more. While all these networks seem very different, in the last two decades there was growing evidence that most of them share fundamental properties [4, 24]. The most famous property is that the networks are *scale-free*, i.e. the degrees follow a power-law distribution $\Pr[\deg(v) \geq d] \approx d^{1-\beta}$, typically for some $2 < \beta < 3$. Other properties include a large connected component which is a small world (poly-logarithmic diameter) and an ultra-small world (constant or poly-loglog average distance), that the networks have small separators and a large clustering coefficient. We refer the reader to [15] for more detailed discussions.

Classical models for random graphs fail to have these common properties. For example, Erdős-Rényi graphs or Watts-Strogatz graphs do not have power-law degrees, while Chung-Lu

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graphs and preferential attachment (PA) graphs fail to have large clustering coefficients or small separators. The latter properties typically arise in real-world networks from an underlying geometry, either spatial or more abstract, e.g., two nodes in a social networks might be considered "close" if they share similar professions or hobbies. It is well-known that in real-world networks the spread of activity (of the flu, of viral marketing, ...) is crucially governed by the underlying spatial or abstract geometry [41]. For this reason, the explanatory power of classical models is limited in this context.

In recent years models have been developed which overcome the previously mentioned limitations, most notably *hyperbolic random graphs* (HypRGs) [13, 12, 10, 44] and their generalisation[1] *geometric inhomogeneous random graphs* (GIRGs) [15][2], and *spatial preferential attachment* (SPA) models [2, 22, 35]. Apart from the power-law exponent β, these models come with a second parameter $\alpha > 1$, which models how strongly the edges are predicted by their distance. Due to their novelty, there are only very few theoretical results on how the geometry impacts the spreading of activity through these networks.

In this paper we make a first step by analysing a specific process, *bootstrap percolation* [20], on the recent and very general GIRG model. In this process, an initial set of *infected* (or *active*) vertices iteratively infects all vertices which have at least k infected neighbours, where $k \geq 2$ is a parameter. It was originally developed to model various physical phenomena (see [1] for a short review), but has by now also become an established model for the spreading of activity in networks, for example for the spreading of beliefs [32, 25, 48, 45], behaviour [30, 31], or viral marketing [38] in social networks (see also [19]), of contagion in economic networks [6], of failures in physical networks of infrastructure [52] or compute architecture [39, 28], of action potentials in neuronal networks (e.g, [47, 49, 5, 21, 50, 43, 26, 27], see also [40] for a review), and of infections in life networks [25].

1.1 Our contribution

We investigate bootstrap percolation on GIRGs with an expected number of n vertices. We fix a ball B in the underlying geometric space, and we initially infect each vertex in B independently with probability ρ. In this way, we model that an infection (a rumour, an opinion, ...) often starts in some local region, and from there spreads to larger parts of the network. In Theorem 1 we determine a threshold ρ_c such that in the *supercritical case* $\rho \gg \rho_c$ whp[3] a linear fraction of the graph is infected eventually, and in the *subcritical case* $\rho \ll \rho_c$ infection ceases immediately. In the *critical case* $\rho = \Theta(\rho_c)$ both options occur with non-vanishing probability. If there are enough (at least k) "local hubs" in the starting region, i.e. vertices of relatively large expected degree, then they become infected and facilitate the process. On the other hand, without such local hubs the initial infection is not dense enough, and comes to a halt.

For the supercritical case, we show that it only takes $O(\log\log n)$ rounds until a constant fraction of all vertices is infected, and we determine the number of rounds until this happens up to a factor $1 \pm o(1)$ in Theorem 2. For the matching lower bound in this result, we need the technical condition $\alpha > \beta - 1$, i.e. edge-formation may not depend too weakly on the

[1] It is non-obvious that GIRGs are a generalization of HypRGs, see [15, Theorem 6.3].

[2] Other than in [15] we do not condition on the number of vertices to be exactly n, which leads to slightly less technical proofs.

[3] with high probability, i.e. with probability tending to 1 as $n \to \infty$. All unspecified limits and asymptotics will be with respect to $n \to \infty$. For example, for a function $f = f(n)$ the notation $f = O(1)$ means that there is $n_0 > 0$ and an absolute constant $C > 0$ that depends only the constant parameters $\alpha, \beta, d, w_{\min}, k$ of the model, such that $f(n) \leq C$ for all $n \geq n_0$. Similarly, $f = \omega(1)$ means $\lim_{n\to\infty} f(n) = \infty$ etc.

geometry. Notably, if the starting region B is sufficiently small then the number of rounds agrees (up to minor terms) with the average distance in the network. In particular, it does not depend on the infection rate ρ, as long as ρ is supercritical.

Finally we demonstrate that the way the infection spreads is strongly governed by the geometry of the process, again under the assumption $\alpha > \beta - 1$. Starting from B, the infection is carried most quickly by local hubs. Once the local hubs in a region are infected, they pass on their infection **a)** to other hubs that are even further away, and **b)** locally to nodes of increasingly lower degree, until a constant fraction of all vertices the region is infected. Indeed, given a vertex v (i.e. given its expected degree and its distance from B), and assuming that v is not too close to B, we can predict whp (Theorem 4) in which round it will become infected, again up to a factor $1 \pm o(1)$. In real applications such knowledge is invaluable: for example, assume that a policy-maker only knows initial time and place of the infection, i.e. she knows the region B and the current round i. In particular, she does not know ρ, she does not know the graph, and she has no detailed knowledge about who is infected. Then we show that she is able to identify a region B' in which the infection can be quarantined. In other words, by removing (from round i onwards) all edges crossing the boundary of B' whp the infection remains contained in B'. The number of edges to be deleted is relatively small: it can be much smaller than n (in fact, any function $f = f(n)$ satisfying $f = \omega(1)$ can be an upper bound, if i and $\mathrm{Vol}(B)$ are sufficiently small), and it is even much smaller than the number of edges *inside* of B', as was already noted in [15].

1.2 Related work

The GIRG model was introduced in [15], and we rely on many results from this paper. The average distance of a GIRG (which, as we show, agrees with the time until the bootstrap percolation process has infected a constant portion of all vertices) was determined in [16] in a much more general setup.

Bootstrap percolation has been intensively studied theoretically and experimentally on a multitude of networks, including trees [20, 9], lattices [3, 8], Erdős-Rényi graphs [36], various geometric graphs [49, 42, 14, 29], and scale-free networks [23, 11, 7, 38]. On geometric scale-free networks there are some experimental results [18], but little is known theoretically. Recently, Candellero and Fountoulakis [17] determined the threshold for bootstrap percolation on HypRGs (in the threshold case $\alpha = \infty$, cf. below), but they assumed that the initial infection takes place *globally*, i.e. whether any vertex is infected initially is independent of its position, and not *locally* as in our paper, where no vertex outside of a certain geometric region is infected initially. This has two major consequences. Firstly, in the global setting, the (expected) number of initially infected vertices needs to be polynomial in n in order for the infection to start spreading significantly; while in our setting every ball containing an expected number of $\omega(1)$ vertices can initiate a large infection whp. Secondly, using our knowledge about how the process evolves in time with respect to the geometry, we show that the infection time of any vertex is mainly governed by its geometric position and its weight. On the other hand, with a global initial infection the infection times only depend on the expected degrees. Note that we do not encode these expected degrees as geometric information (in contrast to [17]), but rather in the weights. Similarly, the questions studied in this paper do not apply for non-geometric random graph models.

While there is plenty of experimental literature and also some mean-field heuristics on other activity spreading processes on geometric scale-free networks (e.g., [51, 53, 34, 54, 33, 46]), rigorous mathematical treatments are non-existent with the notable exception of [37], where rumour spreading is analysed in an SPA model with a push and a push&pull protocol.

2 Model and notation

Graph model

A GIRG is a graph $G = (V, E)$ where both the vertex set V and the edge set E are random. Each vertex v is represented by a pair (x_v, w_v) consisting of a *position* x_v (in some *ground space*) and a *weight* $w_v \in \mathbb{R}_{>0}$.

Ground space and positions. We fix a (constant) dimension $d \geq 1$ and consider the d-dimensional torus $\mathbb{T}^d = \mathbb{R}^d/\mathbb{Z}^d$ as the ground space. We usually think of it as the d-dimensional cube $[0,1]^d$ where opposite boundaries are identified and measure distances by the ∞-norm on $\mathbb{T}^d$, i.e. for $x, y \in [0,1]^d$ set $\|x - y\| := \max_{1 \leq i \leq d} \min\{|x_i - y_i|, 1 - |x_i - y_i|\}$.

The set of vertices and their positions are given by a homogeneous Poisson point process on $\mathbb{T}^d$ with intensity $n \in \mathbb{N}$. More formally, for any (Lebesgue-)measurable set $B \subseteq \mathbb{T}^d$, let $V \cap B$ denote (with slight abuse of notation) the set of vertices with positions in B. Then $|V \cap B|$ is Poisson distributed with mean $n\mathrm{Vol}(B)$, i.e. for any integer $m \geq 0$ we have

$$\Pr\left[|V \cap B| = m\right] = \Pr[\mathrm{Po}\left(n\mathrm{Vol}(B)\right) = m] = \frac{(n\mathrm{Vol}(B))^m \exp(-n\mathrm{Vol}(B))}{m!},$$

and if B and B' are disjoint measurable subsets of $\mathbb{T}^d$ then $|V \cap B|$ and $|V \cap B'|$ are independent. Note in particular that the total number of vertices $|V|$ is Poisson distributed with mean n, i.e. it is also random. An important property of this process is the following: Given a random vertex[4] $v = (x_v, w_v)$, if we condition on $x_v \in B$, where B is some measurable subset of $[0,1]^d$, then the position x_v is uniformly distributed in B.

Weights. For each vertex, we draw independently a weight from some distribution $\mathcal{D}$ on $\mathbb{R}_{>0}$. We say that the weights follow a *weak power-law* for some exponent $\beta \in (2,3)$ if a $\mathcal{D}$-distributed random variable D satisfies the following two conditions: There is a constant $w_{\min} \in \mathbb{R}_{>0}$ such that $\Pr\left[D \geq w_{\min}\right] = 1$, and for every constant $\gamma > 0$ there are constants $0 < c_1 \leq c_2$ such that

$$c_1 w^{1-\beta-\gamma} \leq \Pr\left[D \geq w\right] \leq c_2 w^{1-\beta+\gamma} \tag{1}$$

for all $w \geq w_{\min}$. If this condition is also satisfied for $\gamma = 0$, then we say that the weights follow a *strong power-law*.

Edges. Next we fix a constant $\alpha \in \mathbb{R}_{>1} \cup \{\infty\}$. Then (conditional on the Poisson point process) two distinct vertices $u = (x_u, w_u)$ and $v = (x_v, w_v)$ form an edge independently of all other pairs with probability $p(x_u, x_v, w_u, w_v)$, where the function p satisfies

$$p(x_u, x_v, w_u, w_v) = \Theta(1) \min\left\{\left(\frac{w_u w_v}{\|x_u - x_v\|^d n}\right)^\alpha, 1\right\},$$

if $\alpha < \infty$. In the *threshold model* $\alpha = \infty$ we instead require that p satisfies

$$p(x_u, x_v, w_u, w_v) = \begin{cases} \Omega(1) & \text{if } \|x_u - x_v\| \leq C_1 \left(\frac{w_u w_v}{n}\right)^{1/d} \\ 0 & \text{if } \|x_u - x_v\| > C_2 \left(\frac{w_u w_v}{n}\right)^{1/d} \end{cases}$$

for some constants $0 < C_1 \leq C_2$. Note that for $C_1 \neq C_2$ the edge probability may be arbitrary in the interval $\left(C_1 \left(\frac{w_u w_v}{n}\right)^{1/d}, C_2 \left(\frac{w_u w_v}{n}\right)^{1/d}\right)$.

[4] By abuse of notation, x_v and w_v may either denote random variables or values.

Bootstrap percolation

Let $k \geq 2$ be a constant, let $B_0 \subseteq \mathbb{T}^d$ be measurable, and let $0 \leq \rho \leq 1$. Then the bootstrap percolation process with *threshold* k, *starting region* B_0, and *initial infection rate* ρ is the following process. For every integer $i \geq 0$ there is a set $V^i \subseteq V$ of vertices which are *infected* (or *active*) at time i. The process starts with a random set $V^0 \subseteq V$ which contains each vertex in $V \cap B_0$ independently with probability ρ, and which contains no other vertices. For all integers $i \geq 0$ we then define the set V^{i+1} iteratively by

$$V^{i+1} := V^i \cup \left\{ v \in V \;\middle|\; v \text{ has at least } k \text{ neighbours in } V^i \right\}.$$

Moreover, we set $V^\infty := \bigcup_{i \in \mathbb{N}} V^i$, and for convenience of notation we extend this definition to real parameters $i \in \mathbb{R}_{>0}$ by setting $V^i := V^{\lceil i \rceil}$. For a vertex $v \in V$, we define its *infection time* as $L_v := \inf \left\{ i \geq 0 \;\middle|\; v \in V^i \right\}$ and $L_v := \infty$ if the infimum does not exist.

We denote by $\nu = \nu(n) := n\mathrm{Vol}(B_0)$ the expected number of vertices in B_0. Throughout the paper we will assume that B_0 is a ball, which is – without loss of generality due to symmetry of $\mathbb{T}^d$ – centred at 0. Moreover, we will assume that $\nu = \omega(1)$.

Further notation

We denote the neighbourhood of a vertex $v \in V$ by $N(v) := \{u \in V \mid \{u, v\} \in E\}$. Furthermore, for any two sets of vertices U_1 and U_2, we denote the set of edges between them by $E(U_1, U_2) := \{e = \{u_1, u_2\} \mid u_1 \in U_1, u_2 \in U_2\}$. For any $\lambda \geq 0$ and any closed ball $B \subseteq \mathbb{T}^d$ of radius $r \geq 0$ centred at 0 we denote by λB the closed ball of radius λr around 0. By abuse of notation, if $S \subset V$ and $B \subseteq \mathbb{T}^d$ then $S \cap B := \{v \in S \mid x_v \in B\}$.

3 Main results

First of all we show that bootstrap percolation on a *GIRG* has a threshold with respect to the initial infection rate ρ. Since HypRGs are a special instance of GIRGs, this contains in particular the result of [17] on (threshold) HypRGs, where the case $\nu = n$ was studied.

▶ **Theorem 1.** *Consider a bootstrap percolation process on a GIRG $G = (V, E)$ with constant parameters $\alpha, \beta, d, w_{min}, k$, initial infection rate $\rho = \rho(n) \in [0, 1]$, and initial infection region B_0 with volume ν/n, where $\nu = \nu(n) = \omega(1)$. We set*

$$\rho_c = \rho_c(n) := \nu^{-\frac{1}{\beta - 1}}.$$

If the weights follow a strong power-law, then as $n \to \infty$ we have:

- **(i)** *If $\rho = \omega(\rho_c)$, then $|V^\infty| = \Theta(n)$ whp.*
- **(ii)** *If $\rho = \Theta(\rho_c)$, then $|V^\infty| = \Theta(n)$ with probability $\Omega(1)$, but also $V^\infty = V^0$ with probability $\Omega(1)$.*
- **(iii)** *If $\rho = o(\rho_c)$, then $V^\infty = V^0$ whp.*

If the weights follow a weak power-law, then as $n \to \infty$ we have:

- **(iv)** *If there is a constant $\delta > 0$ such that $\rho \geq \rho_c^{1+\delta}$, then $|V^\infty| = \Theta(n)$ whp.*
- **(v)** *If there is a constant $\delta > 0$ such that $\rho \leq \rho_c^{1-\delta}$, then $V^\infty = V^0$ whp.*

Whenever we refer to the *supercritical* regime we mean case (i) and (iv). Similarly, (iii) and (v) form the *subcritical* regime and (ii) is the *critical* regime. Note in particular that there is a supercritical regime regardless of how small the expected number ν of vertices in the starting region is, provided that $\nu = \omega(1)$. This is in sharp contrast to non-geometric

graphs like Chung-Lu graphs, where the expected number of initially infected vertices must be polynomial in n (if the set of initially infected vertices is chosen at random).

Indeed the proof of Theorem 1 will grant a deeper insight into the evolution of the process. Since the process whp stops immediately in the subcritical regime, we may restrict ourselves to the other cases. We show a doubly logarithmic upper bound on the number of rounds until a constant fraction of all vertices are infected. Furthermore, we prove that this bound is tight up to minor order terms if the influence of the underlying geometry on the random graphs is sufficiently strong ($\alpha > \beta - 1$). Remarkably, the bounds do not depend on the initial infection rate ρ, as long as ρ is supercritical. Moreover, if the expected number ν of vertices in the starting region is sufficiently small (if $\log\log\nu = o(\log\log n)$), then the bound coincides with the average distance in the graph, again up to minor order terms.

▶ **Theorem 2.** *In the situation of Theorem 1, let $\varepsilon > 0$ be constant and set*

$$i_\infty := \frac{\log\log_\nu n + \log\log n}{|\log(\beta-2)|}.$$

Then in the supercritical regime whp, and in the critical regime with probability $\Omega(1)$ we have $|V^{(1+\varepsilon)i_\infty}| = \Theta(n)$, as $n \to \infty$.
If furthermore $\alpha > \beta - 1$ and $\nu = n^{o(1)}$ then in all regimes we have whp $|V^{(1-\varepsilon)i_\infty}| = o(n)$, as $n \to \infty$.

In fact, we can still refine the statement of Theorem 2 tremendously, at least in the case $\alpha > \beta - 1$. In the following, we determine for every fixed vertex v its infection time L_v, up to minor order terms (with the restriction that v may not be too close to the starting region). We will show that it is given by the following expression (see also Remark 6 below).

▶ **Definition 3.** For any $x \in \mathbb{T}^d \setminus B_0$ and $w \in \mathbb{R}_{>0}$ we define

$$\Lambda(x,w) := \begin{cases} \max\left\{0,\ \frac{\log\log_\nu\left(\|x\|^d n/w\right)}{|\log(\beta-2)|}\right\}, & \text{if } w > (\|x\|^d n)^{1/(\beta-1)}, \\ \frac{2\log\log_\nu(\|x\|^d n) - \log\log_\nu w}{|\log(\beta-2)|}, & \text{if } w \le (\|x\|^d n)^{1/(\beta-1)}. \end{cases} \tag{2}$$

In the first case we use the convention that the second term is $-\infty$ if $\|x\|^d n/w < 1$, and thus does not contribute to the maximum.

Note that in the second case, the sign of $\log\log_\nu w$ may be either positive or negative. However, then we have the lower bound $\Lambda(x,w) \ge \log\log_\nu(\|x\|^d n)/|\log(\beta-2)| + O(1)$ due to the upper bound of w and thus, in particular $\Lambda(x,w) \ge 0$, since $x \in \mathbb{T}^d \setminus B_0$.

▶ **Theorem 4.** *Assume we are in the situation of Theorem 1 in the supercritical regime. Let $v = (x_v, w_v)$ be any fixed vertex such that $x_v \in \mathbb{T}^d \setminus B_0$, $w_v = \omega(1)$ and $\Lambda(x_v, w_v) \le \log_2(\|x_v\|^d n/\nu^{2/(\beta-2)})$. Then, as $n \to \infty$, the infection time L_v satisfies whp*

$$L_v \le (1 + o(1))\Lambda(x_v, w_v) + O(1).$$

If additionally $\alpha > \beta - 1$ then, as $n \to \infty$ we also have whp

$$L_v \ge (1 - o(1))\Lambda(x_v, w_v) - O(1).$$

As in Theorem 2, the bounds do not depend on the initial infection rate ρ, as long as it is supercritical.

▶ Remark 5. The technical restrictions in Theorem 4 are necessary: if a vertex v has weight $w_v = O(1)$ then the number of neighbours is Poisson distributed with mean $\Theta(w_v)$ (see Lemma 8), so v is even isolated with probability $\Omega(1)$. In particular, we cannot expected that whp v is ever infected.

The restriction $\Lambda(x_v, w_v) \leq \log_2(\|x_v\|^d n/\nu^{2/(\beta-2)})$ ensures that v is not too close to the starting region. If v is too close, then it may have neighbours inside of B_0, and in this case it does depend on ρ when they are infected. (And of course, this process iterates.) The term $\log_2(\|x_v\|^d n/\nu^{2/(\beta-2)})$ is not tight and could be improved at the cost of more technical proofs. However, there are already rather few vertices that violate the condition $\Lambda(x_v, w_v) \leq \log_2(\|x_v\|^d n/\nu^{2/(\beta-2)})$. For example, recall that it only takes $O(\log\log n)$ steps until a constant fraction of all vertices are infected. At this time, we only exclude vertices which satisfy $\|x_v\|^d n \leq \nu^{2/(\beta-2)} \cdot (\log n)^{O(1)}$, so the expected number of affected vertices is also at most $\nu^{2/(\beta-2)} \cdot (\log n)^{O(1)}$. Even this is a gross overestimate, since the vertices close to the origin have much smaller infection times L_v, and thus only very few of them are affected by the condition.

▶ Remark 6. The first case in Definition 3 is not needed if we restrict ourselves to vertices as they typically appear in GIRGs. More precisely, as we will see in Lemma 10, whp all vertices $v = (x_v, w_v) \in V \cap (\mathbb{T}^d \setminus B_0)$ satisfy $w_v \leq (\|x_v\|^d n)^{1/(\beta-1-\eta)}$ where $\eta > 0$ is an arbitrary constant. In the border case $(\|x_v\|^d n)^{1/(\beta-1)} \leq w_v \leq (\|x_v\|^d n)^{1/(\beta-1-\eta)}$ both expressions in (2) agree up to additive constants, i.e.

$$\Lambda(x_v, w_v) = \frac{2\log\log_\nu(\|x_v\|^d n) - \log\log_\nu w_v}{|\log(\beta-2)|} \pm O(1). \tag{3}$$

Therefore, we could also use (3) as definition for Λ if we would exclude vertices which are unlikely to exist in Theorem 4 .

Finally, we give a strategy how to contain the infection within a certain region when only the starting set and the current round are known, but not the set of infected vertices. Note that the number of edges that need to be removed is substantially smaller than the expected number $\widetilde{\nu}_i$ of vertices in a containment area $\widetilde{B}_i$, see Definition 11.

▶ **Theorem 7.** *Assume that we are in the situation of Theorem 1, and that $\alpha > \beta - 1$. If the starting region B_0 is known, then by removing all edges crossing the boundary of $\widetilde{B}_i$ before round $i+1$, whp (as $n \to \infty$) the infection is contained in $\widetilde{B}_i$. The expected number of edges crossing the boundary of $\widetilde{B}_i$ is $\widetilde{\nu}_i^{\max\{3-\beta, 1-1/d\}+o(1)}$.*

4 Basic properties of GIRGs

In this section we list briefly some basic properties of GIRGs (without proofs). The first lemma, based on [16, Lemma 4.4 and Theorem 7.3], tells us that the expected degree of a vertex equals its weight, up to constant factors. Moreover, it gives the marginal probability that two vertices u, v of fixed weights but random positions in $\mathbb{T}^d$ are adjacent. This probability remains the same if the position of one (but not both) of the vertices is fixed.

▶ **Lemma 8.** *Let $v = (x_v, w_v)$ be a vertex with fixed weight and position. Then $\deg(v)$ is Poisson distributed with mean $\Theta(w_v)$. Moreover, if $u = (x_u, w_u)$ is a vertex with fixed weight, but with random position $x_u \in \mathbb{T}^d$, then*

$$\Pr[\{u,v\} \in E \mid w_u, w_v, x_v] = \Theta\left(\min\left\{\frac{w_u w_v}{n}, 1\right\}\right). \tag{4}$$

Note in particular that the right hand side of (4) *is independent of* x_v*, so the same formula still applies if also the position* x_v *of* v *is randomised.*

We often need to bound the expected number of neighbours of a given vertex in some geometric region, which we may do by the following lemma.

▶ **Lemma 9.** *Let* $\eta > 0$ *and* $C > 1$ *be constants, define* $m := \min\{\alpha, \beta - 1 - \eta\}$ *and consider a closed ball* $B \subseteq \mathbb{T}^d$ *of radius* $r > 0$ *centred at* 0*. Let* $v = (x_v, w_v)$ *be a vertex with fixed weight and position. Then*

$$\mathbb{E}\left[|N(v) \cap B|\right] = O(n\mathrm{Vol}(B)) \cdot \begin{cases} \min\left\{\frac{w_v}{n\mathrm{Vol}(B)}, 1\right\}, & \text{if } \|x_v\| \le Cr, \\ \min\left\{\left(\frac{w_v}{\|x_v\|^d n}\right)^m, 1\right\} & \text{if } \|x_v\| \ge Cr. \end{cases}$$

The last lemma states that whp there are no vertices whose weight is much larger than their distance from the origin.

▶ **Lemma 10.** *Let* $\eta > 0$ *be a constant and consider a closed ball* $B \subseteq \mathbb{T}^d$ *of radius* $r > 0$ *centred at* 0*, satisfying* $n\mathrm{Vol}(B) = \Theta(r^d n) = \omega(1)$*. Then with probability* $1 - (r^d n)^{-\Omega(\eta)}$ *there is no vertex* $v = (x_v, w_v)$ *with* $x_v \in \mathbb{T}^d \setminus B$ *and* $w_v \ge (\|x_v\|^d n)^{1/(\beta-1-\eta)}$*.*

5 Proof outline

5.1 Intuition

Due to space limitations we can only give a very rough sketch of the main ideas. We warn the reader that the statements as they are formulated in this section are not literally true, but they are only true if appropriate error margins (slightly smaller/larger weights or regions) are taken into account. The same holds for definitions within this section. The rigorous definitions and statements with full technical details can be found in Section 5.2 and 5.3.

For the subcritical regime, we distinguish between high-weight vertices ($w_v = \omega(w_0)$, where $w_0 := \nu^{1/(\beta-1)}$) and low-weight vertices ($w_v = O(w_0)$). By an easy computation, the expected number of low-weight vertices in B_0 that are infected in round 1 is $o(1)$, so by Markov's inequality no low-weight vertex becomes infected whp. On the other hand, whp no high-weight vertex exists in B_0, and the expected number of infected vertices outside of B_0 is also $o(1)$ because they are too far away from infected vertices. In order words, whp no vertex is infected in round 1.

In the critical regime, the calculation is similar, but if there exist vertices of weight $\Theta(w_0)$ then these vertices are infected with probability $\Omega(1)$. The number of vertices of weight $\Theta(w_0)$ is Poisson distributed with mean $\Theta(1)$, so it may happen (both with probability $\Omega(1)$) that either no such vertex exists (so percolation stops) or that there are at least k such vertices, and all of them are infected. In the supercritical regime, whp k vertices of weight (slightly less than) w_0 are infected. Whp, these k vertices infect all other vertices of similar weight in two more rounds. This is sufficient to start an avalanche of infection, and for the rest of this section we will restrict ourselves to this case.

If the infection gets started, then it evolves as follows. Let $\zeta := 1/(\beta - 2) > 1$, and consider the sequence B_i of nested balls of volume ν_i/n centred at 0, where $\nu_i := \nu^{\zeta^i}$. Then in the i-th round, all vertices of weight roughly $w_i := \nu_i^{1/(\beta-1)}$ in B_i are infected. In the next round, whp the vertices of weight w_i in B_i infect all vertices of weight w_{i+1} in B_{i+1}, thus spreading the infection to new regions. Note that this statement is easy to prove inductively since we assumed that *all* vertices of weight w_i in B_i are infected, so for the vertices in B_{i+1}

it suffices to count the number of neighbours of a certain weight in B_i, which is a Poisson distributed random variable. This gives a lower bound on how fast the infection spreads geometrically. It can not spread faster since whp there are no edges from B_i to $\mathbb{T}^d \setminus B_{i+1}$. This latter fact already allows us to execute a containment strategy.

On the other hand, if in round j every vertex of weight w in some region has a large probability to be infected, then in round $j+1$ every vertex of weight at least $w^{1/\zeta}$ in this region has a large (though slightly smaller) probability to be infected. To prove this formally, we consider a vertex of weight $w^{1/\zeta}$. Such a vertex (but not vertices of smaller weight) has at least w^δ neighbours of weight w, with probability at least $1 - \exp[-w^\delta]$. So we pick k such neighbours, and bound the probability that at least one of them is *not* infected by a union bound. In this way, we lose a factor of k in each round, but by going through the proof details it turns out that this factor is still negligible compared to the error term $\exp[-w^{\delta/\zeta}]$.

Complementing this infection pathway by a matching upper bound is the most challenging and technical part of the proof. In round $i-1$ there is no infected vertex in B_i, so it is not hard to argue that in round i only vertices of large weight in $\mathbb{T}^d \setminus B_{i-1}$ are infected. However, in subsequent rounds it does happen that vertices of very small weight in $\mathbb{T}^d \setminus B_{i-1}$ become infected. Fortunately, this only happens with rather small probability, which we can explicitly bound (Theorem 13 (f)) as a function of the weight. Once we have such a bound in some round, we use that whp no vertex in $\mathbb{T}^d \setminus B_{i-1}$ (not too close to the boundary) has strictly more than one neighbour in B_{i-1}. Therefore, in order to be infected, at least one of its neighbours in $\mathbb{T}^d \setminus B_{i-1}$ must have been infected in the previous round, and we can bound the probability of this event by the expected number of previously infected neighbours in $\mathbb{T}^d \setminus B_{i-1}$. It turns out that this simple bound is sufficient to provide the desired matching upper bound, safe quite some technical details which we omit.

We remark that it is in this last step where we use the assumption $\alpha > \beta - 1$ since otherwise there do exist vertices in $\mathbb{T}^d \setminus B_{i-1}$ that have several neighbours in B_{i-1}, and these vertices exist in a substantial part of B_i. Even worse, in some (large) subregion of B_i, the number of infections in round $i+1$ that come from neighbours in B_{i-1} dominates the number of infections that come from neighbours in B_i. For investigating the case $\alpha \le \beta - 1$ (which we don't in this paper), it will no longer be possible to use a bound on the infection probability that is uniform within $\mathbb{T}^d \setminus B_{i-1}$, or within $B_i \setminus B_{i-1}$.

Once the claims outlined above are proven (Theorem 13 and 14) we have almost complete control over the process. In particular, for a each vertex v with fixed weight and position (outside of the starting region B_0), and for each round j we have lower and upper bounds for the probability that v has already become infected by round j. We can thus compute rounds j_1, j_2 for which the probability is at most $o(1)$ and at least $1 - o(1)$, respectively, and we find that these rounds coincide up to lower order terms. It is still rather complicated to actually perform the calculations of j_1 and j_2 due to the many technical details which we omitted in this outline, but no further knowledge about the infection process is required.

5.2 Formal statements and sketch of proofs

In this section we will give two theorems which describe the geometrical evolution of the process in detail, and which make the intuitions from Section 5.1 precise. Theorem 13 states that **a)** certain regions cannot be reached too early by the infection, and **b)** within an infected region, vertices of too low weight have a small probability to be infected early. Hence, the theorem gives an *upper bound on the speed* of the infection process. Note that this already gives the quarantine statement (Theorem 7), see Section 5.3 for details. Afterwards, Theorem 14 gives lower bounds on the probability that a vertex in a given region is infected

at a given time, in the supercritical case. In particular, a vertex of weight $w_v = \omega(1)$ will eventually be infected whp. Thus the theorem provides a *lower bound on the speed* of the process. This lower bound also applies in the critical regime if in the first step sufficiently many heavy vertices became infected, an event which holds with at least constant probability.

In Section 5.1 we introduced balls B_i which essentially correspond to the region of infected vertices in round i. For the formal statements we need slightly smaller and larger balls, which we now define formally. In general, $\tilde{\nu}_i, \tilde{B}_i$ etc. will denote the upper bound variants.

▶ **Definition 11.** For all $0 < \varepsilon < \zeta = 1/(\beta-2)$ and all $i \geq 0$, we set

$$\nu_0 := \nu \qquad \text{and} \qquad \nu_i = \nu_i(\varepsilon) := \nu_0^{(\zeta-\varepsilon)^i},$$
$$\widetilde{\nu}_0 = \widetilde{\nu}_0(\varepsilon) := \nu^{(\beta-1)/(\beta-2)+\varepsilon} \qquad \text{and} \qquad \widetilde{\nu}_i = \widetilde{\nu}_i(\varepsilon) := \widetilde{\nu}_0^{(\zeta+\varepsilon)^i}$$

We define $B_i := B_i(\varepsilon)$ and $\widetilde{B}_i := \widetilde{B}_i(\varepsilon)$ to be the closed ball centred around 0 of volume $\min\{\nu_i(\varepsilon)/n, 1\}$ and $\min\{\widetilde{\nu}_i(\varepsilon)/n, 1\}$, respectively. Note that $B_i(\varepsilon) \subseteq \widetilde{B}_i(\varepsilon')$ for all $i \geq 0$ and all $0 < \varepsilon, \varepsilon' < \zeta$.

First we give an upper bound on the speed of the process. For a formal statement, we define the following families of "good" events.

▶ **Definition 12.** Let $\varepsilon > 0$ be a constant and let $\eta = \eta(\varepsilon) > 0$ be a constant which is sufficiently small compared to ε. Moreover, let $h = h(n)$ be a function satisfying $h(n) = \omega(1)$, $h(n) = o(\log n)$, and $h(n) = \nu^{o(1)}$. Then for all $i, \ell, j \geq 0$ we define the following families of events:

- $\mathcal{E}(i) := \{V^i \cap (\mathbb{T}^d \setminus \widetilde{B}_i) = \emptyset\}$;
- For all $w \geq w_{\min}$ let $S(w,\ell) := \{v \in V^{\leq \ell} \mid w_v \geq w\}$. We set

$$\mathcal{F}(\ell, w) = \mathcal{F}_{\varepsilon,\eta,h}(\ell, w) := \left\{ |S(w,\ell)| \leq h^\ell w^{2-\beta+\eta} \widetilde{\nu}_0^{1-(\zeta+\varepsilon)^{-\ell}(\beta-1)^{-1}} \right\},$$

  and $\mathcal{F}(\ell) = \mathcal{F}_{\varepsilon,\eta,h}(\ell) := \bigcap_{w' \geq w_{\min}} \mathcal{F}(\ell, w')$;
- $\mathcal{G}(j) = \mathcal{G}_{\varepsilon,\eta,h}(j) := \bigcap_{j'=0}^{j} (\mathcal{E}(j') \cap \mathcal{F}(j'))$.

In other words, $\mathcal{E}(i)$ means that no vertex outside of $\widetilde{B}_i$ is infected at time i, and $\mathcal{F}(\ell)$ is the event that there are not "too many" vertices which have small weight, are close to the starting region, and are infected at time ℓ. Finally, $\mathcal{G}(j)$ is the event that all "good" events hold up to time j.

▶ **Theorem 13.** *Let ε, η, h be given as in Definition 12 and assume $\alpha > \beta - 1$. Then, for sufficiently large n,*

(a) *$\mathcal{E}(0)$ is always satisfied;*
(b) *$\Pr[\mathcal{F}(0)] \geq 1 - O(h^{-1})$;*
(c) *For all $i \geq 1$ we have $\Pr[\mathcal{E}(i) \mid \mathcal{G}(i-1)] \geq 1 - h^{-\Omega(i)}$;*
(d) *For all $\ell \geq 1$ we have $\Pr[\mathcal{F}(\ell) \mid \mathcal{G}(\ell-1)] \geq 1 - h^{-\Omega(\ell)}$;*
(e) *Whp, the events $\mathcal{G}(j)$ hold for all $j \geq 0$;*
(f) *For all $i \geq 1$ and $\ell \geq 0$, and for every fixed vertex $v = (x_v, w_v)$ such that $x_v \in \mathbb{T}^d \setminus 2^{\ell+1}\widetilde{B}_{i-1}$ and $w_v \geq w_{min}$ we have*

$$\Pr\left[v \in V^{i+\ell} \,\middle|\, \mathcal{G}(i+\ell-1)\right] \leq w_v 2^{\ell d} \widetilde{\nu}_i^{-(\zeta+\varepsilon)^{-\ell-2}/(\beta-1)}.$$

The theorem can be proven by induction on $i + \ell$, with the strategies from Section 5.1, and using the lemmas from Section 4. We next state the complementary lower bound.

For all $i, \ell \geq 0$, let $w_{i,\ell} = w_{i,\ell}(\varepsilon) := \nu^{(\zeta-\varepsilon)^{i-\ell}/(\beta-2)}$, and let U_i be the set of vertices in B_i of weight at least $w_{i,0}$. Furthermore, we denote by $\mathcal{H}(i)$ the event that in round $i+3$ all vertices in U_i are infected.

▶ **Theorem 14.** *Let $0 < \varepsilon < \zeta$ and $\eta = \eta(\varepsilon) > 0$ be sufficiently small. Assume that we are in the supercritical case, or instead that $|U_i \cap V^1| \geq k$. Then the following is true:*

(a) *Whp $|U_i| = \nu_i^{\Omega(\eta)}$ and $|U_i| = O(\nu_i)$ uniformly for all $i \geq 0$.*

(b) *Whp all the events $\mathcal{H}(i)$ occur.*

(c) *There exist constants $C_0, C_1, C_2 > 0$ such that the following holds: Let $v = (x_v, w_v)$ be any vertex with fixed position and weight and let $i, \ell \geq 0$ be such that $x_v \in B_i$ and $w_v \geq \max\{w_{i,\ell}, C_0\}$. Then for sufficiently large $n \in \mathbb{N}$,*

$$\Pr[v \in V^{i+3+\ell} \mid \mathcal{H}(0), \dots, \mathcal{H}(i)] \geq 1 - \exp\left[-C_1 \nu_i^{C_2(\zeta-\varepsilon)^{-\ell}}\right].$$

Again, the theorem can be proven inductively, with the strategies from Section 5.1.

5.3 Proof sketches for main results

In this section we highlight the main steps used to deduce the results from Section 3 from Theorem 13 and Theorem 14.

Threshold and speed of the process: Theorem 1 and Theorem 2

We split the (combined) proof into six claims:

We first show the second statement of Theorem 2, so let $0 < \varepsilon < \zeta$ be a constant.

▶ **Claim 15.** *Assume that $\alpha > \beta - 1$ and $\nu = n^{o(1)}$, then $|V^{(1-\varepsilon)i_\infty}| = o(n)$ whp.*

We define integers $i \geq 0$ and $\ell \geq 0$ such that $i + \ell \geq (1-\varepsilon)i_\infty$ and $\widetilde{\nu}_i = n^{1-o(1)}$ but $2^i \widetilde{\nu}_i = o(n)$. Then whp there are only $o(n)$ vertices inside of $2^\ell \widetilde{B}_i$, by Markov's inequality, and for vertices outside we obtain the corresponding bound from Theorem 14 (f).

In the subcritical regime, (iii) or (v), we will indeed show that whp the process does not infect any vertices in the first step and therefore terminates immediately.

▶ **Claim 16.** *$V^1 = V^0$ whp.*

Since the initial infection occurs only within B_0 and all vertices have the same probability of being infected, the number of neighbours of a given vertex is Poisson distributed, and we can bound the mean by Lemma 9. Thus we can compute the expected number of vertices in $V^{=1}$, which is $o(1)$, and this proves the claim by Markov's inequality.

Next we show that in the critical regime, (ii), with constant probability no further vertices ever become infected.

▶ **Claim 17.** *$V^1 = V^0$ with probability $\Omega(1)$.*

The number of "heavy" vertices of weight $\Theta(w_0)$ is Poisson distributed with expectation $\Theta(1)$. Thus, with probability $\Omega(1)$ there are no heavy vertices. Conditioned on this event, the calculations of the subcritical regime carry over.

On the other hand, also with probability $\Omega(1)$, at least k heavy vertices exist. Each such vertex is infected in the first round with probability $\Omega(1)$ by vertices very close to it, and all these events are positively associated. This proves the following claim.

▶ **Claim 18.** *$V^1 \cap B_0$ contains at least k heavy vertices with probability $\Omega(1)$.*

Next we assume that we are in the supercritical regime (i) or (iv), or in the critical regime (ii) where we also assume that at least k heavy vertices are infected in the first round. Then we need to show the following claim.

▶ **Claim 19.** $|V^{(1+\varepsilon)i_\infty}| = \Omega(n)$ *in expectation and whp.*

We (carefully) choose $i \geq 0$ and $\ell \geq 0$ such that $i+\ell \leq (1+\varepsilon)\, B_i = \mathbb{T}^d$ and $w_{i,l} = O(1)$. Then Theorem 14 (c) tells us immediately that every vertex of weight at least C has probability $\Omega(1)$ to be infected by time $i + \ell + 3$, and since the expected number of such vertices is $\Omega(n)$, thus proving the claim for the expectation. The whp statement follows from a small technical alteration of the previous argument, which we omit for space limitations.

Now Theorem 1 follows from Claims 16, 17, 18 and 19, while Theorem 2 is proven by Claims 15 and 19.

Infection times: Theorem 4

With our previous results, the idea is very simple: given a v vertex which satisfies the assumptions of Theorem 4 we show an lower bound on its infection time L_v by Theorem 14 and an upper bound by Theorem 13, respectively. The details become quite long and technical, and are therefore omitted.

Quarantine strategies: Theorem 7

By Theorem 13, whp there is no vertex outside of $\widetilde{B}_i$ which is infected in round i. Therefore, it suffices to (permanently) remove by the end of round i all edges that cross the boundary of $\widetilde{B}_i$, i.e. all edges in $E(\widetilde{B}_i, \mathbb{T}^d \setminus \widetilde{B}_i)$. Using an argument very similar to the one used in [15, Lemma 7.1 and Theorem 7.2], where the number of edges cutting a grid is considered, we can bound $|E(\widetilde{B}_i, 2\widetilde{B}_i)|$ the expected number of close-range edges by $\widetilde{\nu}_i^{\max\{3-\beta,1-1/d\}+o(1)}$. On the other hand using Lemma 9, we can estimate the expected number $|E(\widetilde{B}_i, \mathbb{T}^d \setminus 2\widetilde{B}_i)|$ of long-range edges by $\widetilde{\nu}_i^{3-\beta}$ and the result follows. We omit the details.

6 Concluding remarks

We have shown that in the GIRG model for scale-free networks with underlying geometry, even a small region can cause an infection that spreads through a linear part of the population. We have analysed the process in great detail, and we have determined its metastability threshold, its speed, and the time at which individual vertices becomes infected. Moreover, we have shown how a policy-maker can utilise this knowledge to enforce a successful quarantine strategy. We want to emphasise that the latter result is only a proof of concept, intended to illustrate the possibilities that come from a thorough understanding of the role of the underlying geometry in infection processes. In particular, we want to remind the reader that bootstrap percolation is not a perfect model for viral infections (though it has been used to this end), but is more adequate for processes in which the probability of transmission grows more than proportional if more than one neighbours is active, like believes spreading through a social network ("What I tell you three times is true."), or action potential spreading through a neuronal network.

Therefore, this paper is only a first step. There are many other models for the spread of an infection, most notably SIR and SIRS models for epidemiological applications, and we have much yet to learn from analysing these models in geometric power-law networks like GIRGs. From a technical point of view, it is unsatisfactory that our analysis does not include the case $\alpha \leq \beta - 1$. We believe that also in this case, the bootstrap percolation process is essentially governed by the geometry of the underlying space, only in a more complex way. Understanding this case would probably also add to our toolbox for analysing less "clear-cut" processes.

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