

## Clusters in an Epidemic Model with Long-Range Dispersal

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In the presence of long-range dispersal, epidemics spread in spatially disconnected regions known as clusters. Here, we characterize exactly their statistical properties in a solvable model, in both the supercritical (outbreak) and critical regimes. We identify two diverging length scales, corresponding to the bulk and the outskirts of the epidemic. We reveal a nontrivial critical exponent that governs the cluster number and the distribution of their sizes and of the distances between them. We also discuss applications to depinning avalanches with long-range elasticity.

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Catastrophic events such as avalanches, material failure, and initial-stage epidemic outbreaks often occur as a chain reaction. Their simplest model was that of Bienaymé and Galton-Watson (BGW) [1,2], originally conceived for genealogy. In a continuous time version one starts with a single infected individual. During a short time lapse  $dt$  each infected individual recovers with probability  $\gamma dt$ , and causes a new infection with probability  $\beta dt$ . On average, each infection generates  $R_0 = \beta/\gamma$  new ones:  $R_0$  determines the fate of the epidemic. When  $R_0 < 1$ , it goes to extinction rapidly. When  $R_0 > 1$ , the size of the population that has been infected up to time  $t$  grows exponentially,  $S \sim e^{(\beta-\gamma)t}$ , as in the initial outbreak stage of an epidemic. At the critical point,  $R_0 = 1$ , the probability that the epidemic has survived up to time  $t$  decreases as  $\sim 1/t$ , and in that case it will have infected  $\sim t^2$  individuals. As a result,  $S$  has strong fluctuations and has a power law distribution  $P(S) \sim S^{-3/2}$  with a cutoff at  $S_{\max} \sim t^2$ . The critical case mimics the scale free behavior displayed by avalanches in disordered materials, i.e., the propagation of an instability which triggers further instabilities via elastic interaction [3].

The BGW model ignores the spatial spreading of the epidemic. Branching diffusion models consider that infected individuals also perform some random walk in a  $d$  dimensional space, independently of recovery and infection. Often, one specifies the random walk to be a short-range Brownian motion. Then the region affected by the epidemic is a connected set, whose geometric properties have been characterized [4–10]. For instance, at criticality, the radius  $\xi$  of this set grows as  $\xi \sim S^{1/4}$ .

However, Brownian diffusion models cannot capture the *long-range* dispersal that ubiquitously occurs in nature, due to, e.g., wind, ocean currents, and air traffic [11–17], spreading an epidemic far from its origin. A similar situation is observed in disordered materials where

long-range interactions can trigger disconnected avalanches, e.g., in the propagation of crack fronts [18–21], wetting lines [22–24], or plasticity [25,26]. In this Letter, we model the long-range dispersal of the infected individuals as follows: during  $dt$ , an individual jumps from  $x$  to  $x'$  with probability  $p_\alpha(x-x')d^d x' dt$ , where  $p_\alpha(x)$  decays as a power law at large distances:

$$p_\alpha(x) = \frac{\theta(|x| - \epsilon)}{|x|^{\alpha+d}}, \quad \alpha > 0. \quad (1)$$

Here  $|x|$  is the Euclidean norm,  $\theta$  is the Heaviside step function, and  $\epsilon \ll 1$  is a short-distance cutoff. Similar long-range models have been studied on a lattice, where the outbreak always displays a subexponential growth [27–33]. Here, we assume an infinite pool of susceptible individuals everywhere, which ensures an exponential outbreak when  $R_0 > 1$ .

A typical epidemic obtained from a numerical simulation of our model is shown in Fig. 1. One may distinguish two regions characterized by distinct length scales. The *bulk*, of radius  $\xi$ , contains most of the infections. Farther away, a sparser *outskirt* of radius  $D$  contains all the remaining infections. The existence of the outskirts is a consequence of the long-range jumps. One aim of this Letter is to obtain how  $\xi$  and  $D$  scale with the infected population  $S$ . Another fundamental consequence of long-range dispersal is the presence of *clusters*, i.e., spatially disconnected regions affected by the epidemic. As is apparent from Fig. 1, the clusters vary in sizes, and their spatial distribution is not uniform. The second goal of this Letter is to introduce a method to properly define the clusters. We then characterize their random geometry: how the number of clusters grows with  $S$ , how their sizes are distributed, what distances are separating them, etc. Our exact results are obtained by the analysis of a nonlinear “instanton”

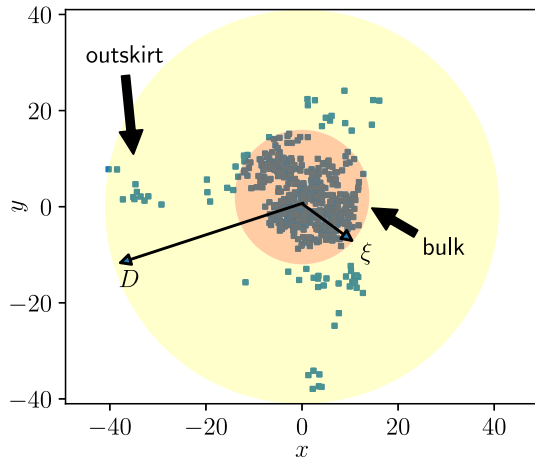


FIG. 1. Spatial distribution of a critical epidemic started at the origin, totaling 1000 infections. Because of the long-range dispersal [1],  $\alpha = 1.5$  of infected individuals, the points visited form disconnected clusters. The bulk of radius  $\xi$ , concentrating a majority of infections, is surrounded by a sparse outskirts, containing *all* infections.

equation. We stress that our methods are applicable to real-world data. As a proof of principle, we tested our theory against the Covid-19 outbreak data in the U.S. Remarkably, a prediction of our model, Eq. (12) below, describes well the spatial distribution of the clusters during the first week of March 2020 (see the Supplemental Material [34]).

The epidemic model introduced above provides a discrete realization, equivalent, near criticality [40], to the mean-field theory [41,42] describing the spatial structure of the avalanches of slowly driven elastic interfaces in a disordered medium. In crack experiments, clusters have been directly observed [43], and their number and size distribution have been characterized [44,45]. These works proposed that these properties are fully encoded in the global properties of the crack front, e.g., in its roughness exponent [46–48]. Here, we make a first step at examining this issue analytically; our results indicate that the cluster statistics probably involve a new independent exponent. In what follows, we report our main results, and sketch the main points of their derivation; see the Supplemental Material [34] for details.

*Bulk and outskirts.*—We first determine the length scales of the bulk  $\xi$  and outskirts  $D$ , by simple arguments. We consider our model with a single infected individual at the origin initially ( $t = 0$ ). At criticality ( $R_0 = 1$ ), the bulk length  $\xi$  can be estimated as the *typical* displacement of a random walk with jump distribution [Eq. (1)]. When  $\alpha < 2$ , we have a Lévy flight, and thus

$$\xi \sim t^{\frac{1}{\alpha}} \sim S^{\frac{1}{2\alpha}}, \quad \alpha < 2, \quad (2)$$

where the last estimate comes from the scaling  $S \sim t^2$ . When  $\alpha > 2$ , we recover the short-range behavior

$\xi \sim \sqrt{t} \sim S^{\frac{1}{4}}$  [42,49]. On the other hand, the outskirts' diameter  $D$  is estimated as the *farthest* jump among  $\sim S$  independent attempts:

$$D \sim S^{\frac{1}{2}}. \quad (3)$$

Hence, the outskirts is much larger than the bulk if  $\alpha < 4$ : only for  $\alpha > 4$  do we completely recover a short-range behavior, with  $D \sim \xi \sim S^{1/4}$ . This is already a surprise, as naively one would expect a short-range takeover at  $\alpha = 2$ .

In the supercritical regime, the argument for the outskirts diameter  $D$  and the result [Eq. (3)] still hold. The scaling of the bulk size  $\xi$  is different. Indeed, the infected population grows exponentially,  $S \sim e^{(\beta-\gamma)t}$ . As a consequence, the density of infected individuals is exponentially large at the epicenter,  $x = 0$ , and decays as  $\sim S|x|^{-\alpha-d}$  [Eq. (1)]. The bulk extent is then determined by the distance,  $|x| = \xi$ , at which the density reaches unity:

$$\xi \sim S^{\frac{1}{\alpha+d}} \sim e^{\frac{\beta-\gamma}{\alpha+d}t} \quad (4)$$

Note that, when  $R_0 > 1$ , the separation of scales  $D \gg \xi$  remains for any  $\alpha$ , and the short-range behavior with a linear growth  $\xi \propto t$  is never recovered. This is in contrast with lattice models [27,28], where a reduction to short range does happen at  $\alpha = d + 1$ .

*Defining clusters.*—In our model, the ensemble of positions ever occupied by an infected individual up to time  $t$  is a finite set, as only a finite number of jumps have occurred. How do we define its clusters? For simplicity we focus on one and two dimensions. We introduce a coarse-graining scale  $b \gg \epsilon$ , and thicken each point by a patch of size  $b$ —an interval of length  $b$  in 1D, and a square of size  $b$  in 2D—centered at that point; see Fig. 2. The patches attached to different points can then overlap and form clusters. To characterize their spatial distribution, we introduce the following observables: (i) the number of clusters  $N_c$ ; (ii) the length and area of individual clusters,  $\ell_c$  in 1D and  $\mathcal{A}_c$  in 2D, whereby the sum of all  $\ell_c$  ( $\mathcal{A}_c$ ) is the epidemic's *extension*  $\ell$  (area,  $\mathcal{A}$ , respectively); and (iii) the distances between clusters. In 1D, a natural choice is the distribution of gaps (Fig. 2). It is not hard to see that the number of gaps larger than  $g$  is related to the cluster number with  $b = g$ :

$$N_c(b = g) = (\text{number of gaps} > g) + 1. \quad (5)$$

In 2D, the notion of gaps is not obvious, and we take  $N_c(b = g)$  as a probe of the distances between clusters. We obtained the  $b$  dependence of all the quantities; for conciseness, we report results with  $b = 1$  unless otherwise stated.

*Clusters at criticality.*—When  $R_0 = 1$ , statistical fluctuations are strong, and there are various ways of averaging. Here, we focus on averages conditioned on a large infected

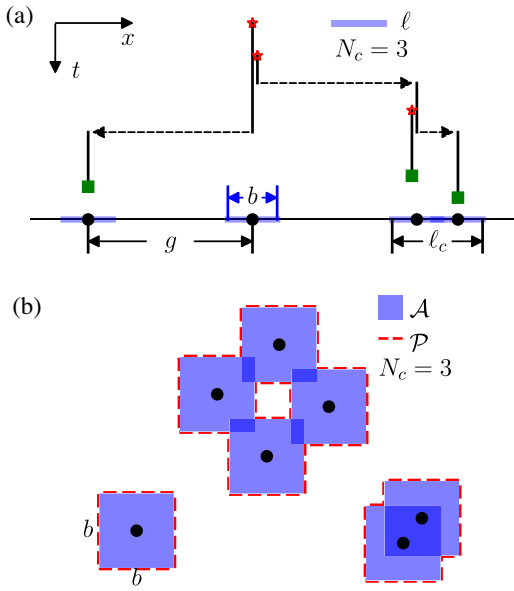


FIG. 2. (a) Illustration of an epidemic in 1D. An infection (recovery, jump) is indicated by a red star (green square, dashed line, respectively). The points visited are coarse grained by an interval of length  $b$ . They form  $N_c = 3$  clusters, with total extension  $\ell = \sum \ell_c$ . The gaps  $g$  are defined independently of  $b$ . (b) In 2D, a point is coarse grained by a square of side  $b$ , to define the cluster number  $N_c (= 3)$ , the area  $\mathcal{A}$ , and the perimeter  $\mathcal{P}$ . Note that in 2D, a cluster can be nonconvex and have holes.

population  $S$  (assuming nonextinction), denoted as  $\langle \mathcal{O} \rangle_S$  for an observable  $\mathcal{O}$ . From the  $S$ -conditioned averages, we can obtain the asymptotics of the average over all realizations up to time  $t$ , using

$$\langle \mathcal{O}(t) \rangle \sim \int^{S_{\max}} P(S) \langle \mathcal{O} \rangle_S dS, \quad S_{\max} \sim t^2. \quad (6)$$

Thus, if  $\langle \mathcal{O} \rangle_S \sim S^a$ ,  $\langle \mathcal{O}(t) \rangle \sim t^{\max(2a-1, 0)}$  (See Table I of the Supplemental Material [34] for results).

We have seen that when  $\alpha < 4$ , the outskirts is much larger than the bulk, and we expect many clusters. Interestingly, the interval  $\alpha \in (0, 4)$  is divided into several regimes, with qualitatively different behaviors of  $\langle \ell \rangle_S$ ,  $\langle \mathcal{A} \rangle_S$ , and  $\langle N_c \rangle_S$ . Let us start with the most nontrivial one,  $\alpha \in (d/2, d)$ . There, we find that the average extension and area are related to the bulk extent in a rather expected way:

$$\langle \ell \rangle_S \sim \xi, \quad \langle \mathcal{A} \rangle_S \sim \xi^2. \quad (7)$$

It is worth noting that the above quantities are independent of  $b$  for a large range of  $b$ ; see Eq. (15) below. Now, the average number of clusters scales with  $\xi$  via a new and nontrivial exponent

$$\langle N_c \rangle_S \sim \xi^\chi, \quad \alpha < \chi < d. \quad (8)$$

The exponent  $\chi$  is a function of  $\alpha$  and  $d$ , and determined by a transcendental equation given in the Supplemental

Material [34] together with a plot. It satisfies  $\alpha < \chi < d$ , which means that the number of clusters grows with  $S$  but remains much lower than the area or extension. Thus, the cluster areas  $\mathcal{A}_c$  and extensions  $\ell_c$  must have broad distributions (with divergent mean as  $S \rightarrow \infty$ ). Computing them is beyond the reach of the present techniques. However, assuming that they follow a single power law in the interval  $[1, \xi^d]$ , we can surmise their exponent [45]:

$$P(\ell_c) \sim \ell_c^{-\chi-1}, \quad P(\mathcal{A}_c) \sim \mathcal{A}_c^{-\chi/2-1}. \quad (9)$$

Concerning the gaps between clusters, we found that  $\langle N_c(b=g) \rangle_S$  has two regimes with distinct power laws:

$$\frac{\langle N_c(b=g) \rangle_S}{\sqrt{S}} \sim \begin{cases} (g/g_c)^{\frac{d(\chi-\alpha)}{d-\alpha}} & 1 \ll g \ll g_c \\ (g/g_c)^{\frac{ad}{d+\alpha}} & g_c \ll g \ll D \end{cases}, \quad (10)$$

where  $g_c = \xi^{1-\alpha/d}$  is the crossover gap length. The two gap regimes  $g \ll g_c$  and  $g \gg g_c$  correspond to gaps in the bulk and in the outskirts, respectively. To better understand this result, let us consider one dimension [50]. Observe that the total length of the gaps no greater than  $g_c$  is exactly the bulk size:

$$\sum (\text{gaps} \leq g_c) \sim g_c \langle N_c(b=g_c) \rangle_S \sim \xi. \quad (11)$$

Now, if we consider all the bulk gaps up to a size  $g \ll g_c$ , their number is almost  $N_c(b=1)$ , but their total size is a negligible fraction of the bulk. On the other hand, the outskirts gaps are a minority in number, but their total size is much greater than the bulk size. Of course, there is no sharp transition between bulk and outskirts, but rather a smooth crossover. Indeed, in Fig. 3, we show that to demarcate the two power laws requires several orders of  $g/g_c$ . Otherwise, one may observe a ‘‘compromise’’ of the theoretical predictions.

So far we focused on the regime  $\alpha \in (d/2, d)$ . The other ones are simpler. In a nutshell, for strong long-range dispersal ( $\alpha < d/2$ ), the clusters become atomic and have a finite size on average. Therefore we have  $\langle N_c \rangle_S \sim S$ , and  $\langle \ell \rangle_S, \langle \mathcal{A} \rangle_S \sim S$  as well. For weak long-range dispersal ( $\alpha > d$ ), the bulk becomes more compact, and gaps of size  $\gtrsim 1$  exist only in the outskirts. See the Supplemental Material [34] for a detailed discussion.

*Clusters of an outbreak.*—In the supercritical ( $R_0 > 1$ ) regime, the statistical fluctuations are weak. We can thus consider the averages up to  $t$ , which are dominated by realizations with an infected population  $S \sim e^{(\beta-\gamma)t}$ . Recall that the bulk and outskirts diameter grow exponentially as Eqs. (3) and (4), for any  $\alpha > 0$ . Now, the cluster structure of an outbreak is also simpler, and we found the same qualitative picture for any  $\alpha$ . The bulk is compact and has no large gaps. Its extension is  $\langle \ell \rangle \sim \xi$ ,  $\langle \mathcal{A} \rangle \sim \xi^2$ , where  $\xi \sim S^{1/(d+\alpha)} \sim e^{(\beta-\gamma)t/(d+\alpha)}$  [Eq. (4)]. The outskirts is sparse, and has an exponential number of clusters,  $\langle N_c \rangle \sim \xi^d$ .

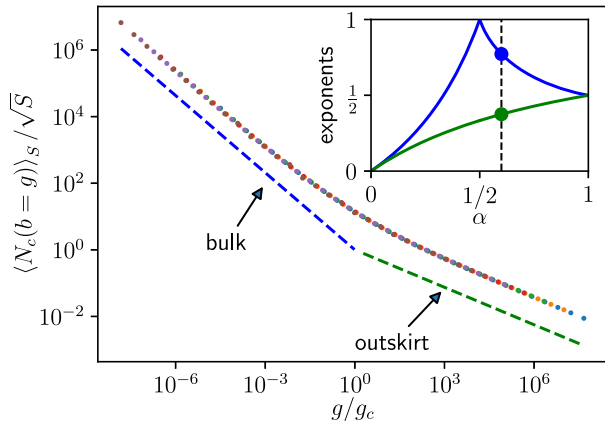


FIG. 3. Gap distribution in 1D with  $\alpha = 0.6$ , obtained by numerical solution of Eq. (13) [34]. Data points with various sizes  $S = 10^{20}, \dots, 10^{28}$  are collapsed using Eq. (10). The dashed lines indicate the predicted exponents in two regimes. Inset: dependence of the two exponents on  $\alpha$ .

Notably, their spatial structure is time independent: the gap distribution is stationary up to a normalization and a cutoff,

$$\langle N_c(b=g) \rangle \sim \xi^d g^{-\frac{d\alpha}{\alpha+d}}, \quad g \ll D. \quad (12)$$

In the Supplemental Material [34], we tested this prediction against Covid-19 data, finding an encouraging agreement.

*Method.*—We highlight some key points of our analytical approach. The main object is a function  $F(x, t|b)$ , which is the probability that  $x$  belongs to the patch of some point visited before  $t$ . A standard backward recursion argument shows that  $F$  satisfies a semilinear “instanton” equation [42,51–54]:

$$\partial_t F = \mathcal{D}^\alpha F + (\beta - \gamma)F - \beta F^2, \quad F|_{t=0} = 0, \quad (13)$$

for any  $x$  outside the patch of the origin; inside that,  $F = 1$ . Here  $(\mathcal{D}^\alpha f)(x) := \int p_\alpha(x-y)[f(y) - f(x)]d^d y$  is the “fractional diffusion” term. From the solution  $F$ , we can obtain the area (extension) by integrating it over the plane (line). The cluster number is obtained by differentiating with respect to  $b$ . In 1D, we have

$$N_c(b) = \partial_b \ell(b). \quad (14)$$

A similar trick exists in 2D [34].

Therefore, the problem boils down to the asymptotic analysis of Eq. (13). In the supercritical regime, the exponential spreading of its traveling wave solution follows from existing rigorous results [55]; for a self-contained derivation and our results on clusters, see the Supplemental Material [34]. In Fig. 4, we plot the front profile. Note that it decays as a power law, and does not have a characteristic width. In contrast, in traveling wave equations with short-range diffusion, the wavefront position has linear growth in time, and its width is of order unity.

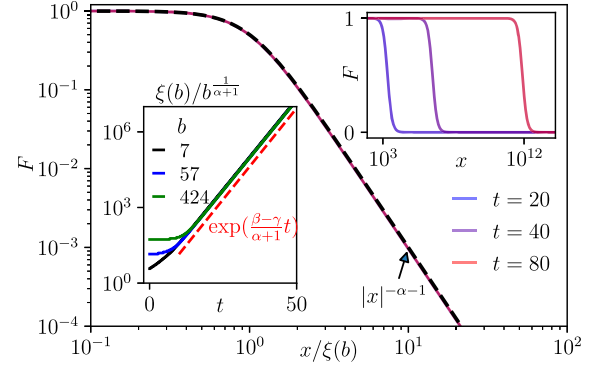


FIG. 4. Traveling wave solution to Eq. (13) in the supercritical regime ( $\alpha = 2, \beta = 1, \gamma = 0, b = 7$ ). The solution at  $t = 20, 40, 80$  (upper inset) collapsed onto the front profile  $F(x, t) = f[x/\xi(b)]$ ,  $f(y) = 1/(1+y^{1+\alpha})$  (black dashed curve). The front position  $\xi(b)$  is defined by  $F[\xi(b)] = 1/2$ . Its time dependence is plotted in the lower inset for three values of  $b$ . The collapse confirms the  $b$  dependence of the front position [34].

The results at criticality follow from the stationary solution of Eq. (13). The solution in the regime  $\alpha \in (d/2, d)$  involves a noteworthy feature. To discuss that without going into technical details, consider the following puzzle, say in 1D. Recall that the cluster number and the extension are related by a  $b$  derivative Eq. (14). Then, how can they scale differently:  $\ell \sim \xi$ ,  $N_c \sim \xi^\ell \ll \xi$ ? The crux is that the leading asymptotics of  $\ell$  is  $b$  independent, while  $N_c$  derives from a subleading term:

$$\langle \ell(b) \rangle_S = c_0 \xi + c_1 (b) \xi^\ell, \quad (b \ll g_c) \quad (15)$$

where  $c_0$  is  $b$  independent. To extract the cluster statistics from the solution of Eq. (13), it is necessary to identify its *subleading* asymptotics, in addition to the previously known leading one [56]. This mathematical detail has a physical interpretation: cluster statistics are associated with irrelevant perturbations in the sense of the renormalization group. During the coarse-graining process, the clusters merge, and information about them is gradually erased.

*Conclusion.*—We have characterized the clusters of an epidemic model with long-range dispersal, which is equivalent near criticality to the mean-field theory of depinning avalanches with long-range elasticity. We found that two diverging length scales—the bulk and the outskirts—emerge in both supercritical and critical regimes. In the latter, the bulk can have a rich structure with broadly distributed cluster sizes as well as gap sizes. Our analytical approach based on the instanton equation can be extended to study the effect of inhomogeneous networks [14], realistic mixing patterns [14,57], superspreading events [58], or the regions where the epidemic is still active at time  $t$  [59,60]. It will be also interesting to see how much the qualitative features revealed here appear in other epidemic models, e.g., contact point processes [61,62]. Finally, concerning depinning



avalanches, our model provides a mean-field description which should be quantitatively correct for realistic long-range systems when  $d \geq 2\alpha$ . To describe these systems for  $d < 2\alpha$ , loop corrections to mean-field theory should be taken into account. In particular, our results imply a cluster number distribution  $P(N_c) \sim N_c^{-\mu}$  where  $\mu = \alpha/\chi + 1$  for  $\alpha \in (d/2, d)$ . At the critical dimension  $d = 2\alpha$ , we recover the BGW value  $\mu = 3/2$ , but in our model  $\mu > 3/2$  is a new exponent when  $d < 2\alpha$ . Meanwhile, numerical studies [45] of realistic models suggest that  $\mu \approx 3/2$  for all  $d < 2\alpha$ . It will be interesting to see how to retain the “dangerously irrelevant” cluster statistics in the field theory and whether the loop corrections can account for this numerical observation.

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