

# Contact process with a defect: universal oasis, nonuniversal scaling

Zvi Miller<sup>1</sup> and Nadav M. Shnerb<sup>1</sup>

<sup>1</sup>*Department of Physics, Bar-Ilan University, Ramat-Gan 52900, Israel*

## Abstract

The extinction transition in the presence of a localized quenched defect is studied numerically. When the bulk is at criticality, the correlation length diverges and even an infinite system cannot "decouple" from the defect. The results presented here suggest that, in 1+1 dimensions, the critical exponent  $\delta$  that controls the asymptotic power-law decay depends on the strength of the local perturbation. On the other hand, the exponent was found to be independent of the local arrangement of the defect. In higher dimensions the defect seems to induce a transient behavior that decays algebraically in time.

## I. INTRODUCTION

In a recent article, the results of a spatial-genetic study of honeybees in the Sahara desert [1] have been published. The authors concluded that these bees (*Apis mellifera*) first arrived to the desert around 10,000 years ago, when the Sahara was a green savannah. Although the contemporary Sahara is inhospitable to these bees, they do survive in many **oases** that litter the desert. The same scenario holds for many existing communities of animals: their habitat is made of relatively small patches, and the sustainability of the whole system is determined by the rates of local extinction and recolonization. When migration among "oases" is relatively weak, such a system is known as a *metapopulation* [2].

In the study of metapopulation persistence, many researchers are using the stochastic patch occupancy models (SPOM) [3–5] as a paradigmatic framework. These models assume that each habitat patch is either occupied or empty, i.e., that the timescale in which the population reaches its local carrying capacity equilibrium is relatively small. Neglecting the details of the in-site dynamics the theory is then focused on extinction and recolonization events. In the language of stochastic dynamics, we are dealing with a contact process [6, 7] taking place in heterogenous environment. While the properties of the contact process and its generalizations are quite well understood when it takes place on a uniform lattice, much less is known in the presence of a quenched disorder. In this paper we are dealing with a very simple case: a (generalized) contact process perturbed by a local heterogeneity. Interestingly it turns out that even a local defect may alter the behavior of the whole system at the most important parameter regime, close to the extinction transition.

On a homogenous lattice the contact process is known to admit an extinction transition when the birth rate is below some (nonuniversal) threshold. For any extinction transition on a homogeneous substrate and a single absorbing state, Grassberger [8] and Janssen [9] conjectured that the microscopic details of the stochastic process are irrelevant close to the extinction point and the transition belongs to the directed percolation (DP) universality class. The basic rationale behind this conjecture is that a spatially extended system decomposes, close to the transition, into active and inactive zones, where after each typical period of time there is certain probability for an active state to die, to survive, or to infect its inactive neighbors. If these regions are considered as lattice points on a  $d$  dimensional array, the chance of an active site to survive or to infect its neighbors within a unit time is equivalent

to the chance that a bond exists between a lattice point at time  $t$  and its neighbors in a subsequent replica of the system at  $t + 1$ . Accordingly, the extinction transition happens when the bond density is exactly at threshold for an infinite cluster in a  $d + 1$  dimensional system, and the transition belongs to the directed percolation universality class in  $d + 1$  dimensions.

The Grassberger-Janssen conjecture has proven to be extremely robust, and a large number of stochastic models that admit an extinction transition were shown to belong to the DP equivalence class if the substrate is homogenous [10, 11]. It was further shown that spatio-temporal substrate noise (i.e., birth-death rates that fluctuate in space and time with only short range correlations) is an irrelevant perturbation close to the transition, so small noise is averaged out and leaves the DP transition unaffected [10], although it can change the location of the transition point [13]. The case of diffusively correlated disorder [16, 17] was considered recently by Dickman [18] and by Evron et. al. [19] with somewhat different results: while [18] reported that the critical exponents differ from those of DP transition, in [19] it was suggested that only the *off-transition* behavior of the system is governed by local adaptation to favored regions, since the scaling function describing the behavior away from the transition shows significant deviations from the known DP behavior.

In practice, however, it is hard to avoid quenched heterogeneities from the substrate on which the process takes place, and this is true in particular for metapopulations [2] or the spread of an epidemic [20, 21]. It turns out that quenched (time independent) disorder is a relevant perturbation [12] and seems to change the nature of the transition.

When the system is subject to a global disorder (the spatial range in which the heterogeneity occurs is not compact) a Griffiths phase exists between the active and the inactive regions [14]. In the parameter region that corresponds to the Griffiths phase the survival of an occupied patch depends on the local properties of the substrate, *not* on activation by neighboring regions. In particular, for each time scale the process stays alive due to the spatial domains that admit high carrying capacity [14, 15]. Although stochastic fluctuations guarantee extinction for any localized active patch in the absence of migration [22–24], the time to extinction grows exponentially with the carrying capacity of the spatial domain. This implies that exponentially rare spatial patches, with high birth rate, support the population for exponentially long times. An optimization argument [14] shows that in such a case the survival until  $t$  is dominated by rare spatial fluctuations of linear size  $L \sim \log(t)$ ;

accordingly, the density falls algebraically with time. The Griffiths phase is located between an extinction region, where essentially no oases exist, and the active phase, where good oases infect each other to yield a never dying process.

In this paper we intend to focus on a much simpler problem: the contact process in the presence of a localized (compact) spatial heterogeneity, like an oasis in the desert or an ecological trap (sink habitat). When the bulk system is off criticality it is segregated into effective patches of size  $\xi$ , and the effect of a compact inhomogeneity extends only to this distance, so it cannot change the long-time properties of an infinite sample. The situation changes when the bulk system is at criticality: in that case the correlation length decay obeys a power-law, and in principle a single oasis (or sink) may affect the properties of the bulk. Here we present numerical evidences suggesting that this is indeed the case, at least in one dimension. A similar phenomenon characterizes the physics of the zero temperature Kondo effect, when a single magnetic impurity changes the behavior of an infinite system as it dictated the density of states close to the Fermi level where correlation length diverges.

Before proceeding to the results, let us describe the numerical procedure. We have simulated a spatial version of the SIS model for epidemics, which is a simple generalization of the contact process, utilizing the simulation technique presented in [21, 28]. On each site there are  $N$  agents, from which  $N - I$  are susceptible and  $I$  are infected. If the infection rate is  $\alpha$  and the recovery rate is  $\beta$ , the chance of an infected agent to recover within  $\Delta t$  is  $\beta\Delta t$ . On a one dimensional lattice a susceptible agent on the  $n$ -th site has  $I_n$  infected neighbors in its own site and  $I_{n\pm 1}$  in the neighboring sites. If the chance to infect a local susceptible is  $\alpha(1 - \chi)/N$  and the chance to infect a neighbor susceptible is  $\alpha\chi/(2N)$ , the average number of infections on the  $n$ -th site within  $\Delta t$  is given by  $q_1(N - I_n)$  where  $q_1 = \Delta t\alpha [(1 - \chi)I_n + \chi I_{n+1}/2 + \chi I_{n-1}/2]$  and the average number of recoveries is  $q_2 I_n$  where  $q_2 = \Delta t\beta I_n$ . Once these numbers were calculated for all the chain, the system is updated by drawing, for any site, two numbers from a binomial distribution, one with an average  $q_1(N - I - n)$  and the other with an average  $q_2 I_n$ .  $I_n(t)$  is then updated to  $I_n(t + \Delta t) = I_n(t) + B(q_1, N - I_n) + B(q_2, I_n)$ . This procedure may be considered as a stochastic Euler integration of the system dynamics, and it converges to the pure contact process in the limit  $N = 1$ ,  $\chi = 1/2$ . Without loss of generality we have taken  $\beta = 1$  in all cases considered below, and the generalization to higher dimensions is trivial.

## II. DEFECT DEPENDENT DECAY EXPONENT IN ONE DIMENSION

Let us start presenting our results for the contact process,  $N = 1$ . The critical birth (infection) rate in that case is known to be  $\alpha_c = 3.2978$ . For a homogenous system at criticality the survival probability, i.e., the chance of a cluster, grown from a single seed, to be active after  $t$  steps, is given by  $P(t) = t^{-\delta}$ , where the 1d value is  $\delta_0 \approx 0.159$ .

Figure 1 shows what happens when a single site for which  $\alpha = \alpha_c + \delta\alpha$  has been introduced into a system which is otherwise at criticality (see schematic sketch in the upper-right part of the figure). When the process is ignited with a single particle at the heterogeneity (arrow),  $P(t)$  is still a straight line on a double logarithmic plot, but the slope depends on  $\Delta\alpha$ : for source (oasis) the slope is smaller than  $\delta_0$  and for sink the decay is faster. This indicates the failure of the critical system to decouple from the defect when the correlation length diverges. Figure 2 shows  $\delta(\Delta\alpha)/\delta_0$  between a perfect sink and a very strong source.

What happens when the process is ignited away from the heterogeneity? If the oasis/sink is located at the origin and the process starts when a seed (a single infected person) is positioned at  $x$ ,  $P(t)$  must decay with the exponent  $\delta_0$  until the system starts to "feel" the oasis, then cross over to the appropriate value of  $\delta$ . When the system is at criticality the spread of a perturbation is controlled by the ratio between the spatial and the temporal correlation length, i.e., a perturbation is traveling a distance  $x$  within a time  $t \sim x^z$ , where for a 1d DP transition  $z = 1.58$ . This implies that after a time  $t$  the source/sink affects a distance  $x \sim t^{0.63}$ .

Figure 3 illustrates the effect of the distance from the transition point. Here the seed was deposited at  $\Delta x = 15$  where the origin is an oasis, a sink or a regular site for which  $\alpha = \alpha_c$ .  $P(t)$  for all cases is identical until the system starts to get some information about the structure at the origin, then it splits into three different slopes (left panel). In the right panel there is a sink at the origin and  $P(t)$  is shown for different distances of the seed from the heterogeneity: one sees that the short time decay has the exponent of a homogenous system, the long time decay also have the same slope, but the crossover time depends on the distance.

The same behavior is depicted in Fig. 4. Here the local particle density  $\rho(x, t)$ , starting from a fully occupied lattice, is shown: a snapshot at  $t = 1000$  for different types of special site (inset) and consecutive time frames for a system with a sink. Since at the close region

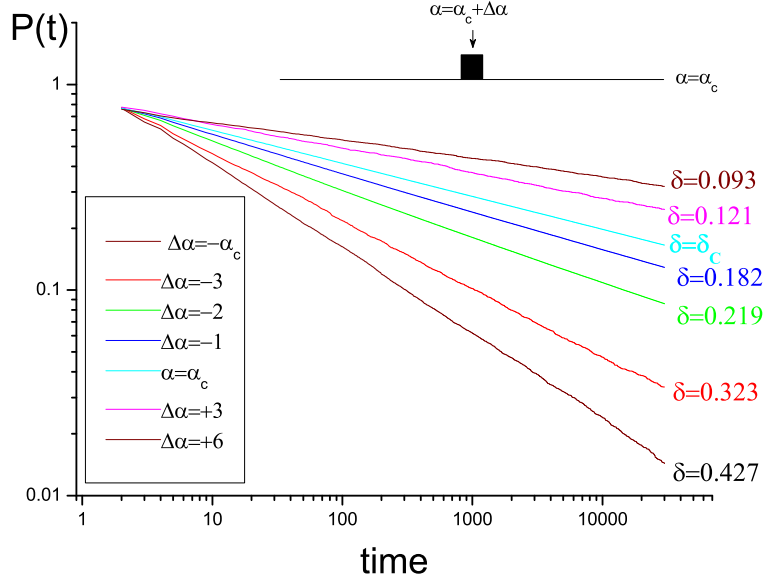


FIG. 1: The survival probability  $P(t)$  vs.  $t$  on a double logarithmic plot for a system of  $L=5000$  sites with periodic boundary conditions. Only a single particle is allowed on each site, so the process is equivalent to the contact process. At the bulk the infection rate is  $\alpha_c = 3.2978$ , and only on the "exceptional" site at the origin  $\alpha = \alpha_c + \Delta\alpha$ . When the system is homogenous the slope is  $\delta_0 \sim 0.16$ , as expected from the theory. If on the central site  $\alpha < \alpha_c$  (sink) the slope is larger, and for an oasis ( $\alpha > \alpha_c$ ) it is smaller. The results here reflect an average over  $10^6$  processes, all ignited with a single agent on the source/sink site.

$\rho(t)$  decays like  $t^{-\delta(\Delta\alpha)}$  and in the far region  $\rho(t) \sim t^{-\delta_0}$ , on a double logarithmic scale a straight line appears for  $\rho(x)$  in the intermediate region, with a slope

$$s = \frac{\log(\rho_0 t^{-\delta(\Delta\alpha)}) - \log(\rho_0 t^{-\delta_0})}{\log(t^{z/2})} \sim 2 \frac{\delta(\Delta\alpha) - \delta_0}{z}. \quad (1)$$

This slope agrees with the measurements from our numerical experiments.

### III. UNIVERSAL RESPONSE TO LOCAL HETEROGENEITY

Surprisingly, our numerics suggest that the effect of a localized heterogeneity is also *universal*, i.e., it is independent of the spatial structure of the source/sink and of  $N$ , the carrying capacity of a site. This feature is demonstrated in Figure 5: here the heterogeneity

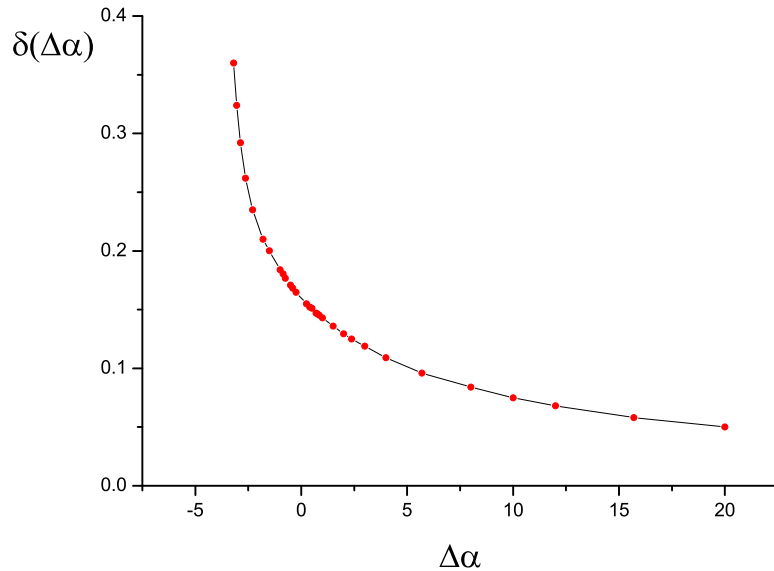


FIG. 2: The decay exponent  $\delta(\Delta\alpha)$  vs. the strength of the heterogeneity  $\Delta\alpha$ , from a sink where the birth rate is zero ( $\Delta\alpha = -\alpha_c$ , i.e.,  $\alpha = 0$ ) to a very strong oasis.

is made of two exceptional sites, and the distance between the two,  $2d$ , is a free parameter. When the process starts with a seed between the two sites, the short time decay of  $P(t)$  is, of course, nonuniversal. However, after the process "probes" the two sites the long time behavior is universal and independent of the distance between the sinks. We have carried out different numerical experiments, with two or three, equal or unequal, exceptional sites (and for two sources, source and sink, two sinks with different  $\alpha$ s etc.). In all of them the tail exponent is independent of the spatial structure of the compact heterogeneity.

The same observation holds if one allows more than one agent at any site. In Fig. 6  $P(t)$  is plotted for  $N = 50$ . There is a transient region in which the system followed the deterministic decay rate  $P \sim 1/t$  (as suggested by the equation  $\dot{I} = \alpha\chi\nabla^2 I - \alpha I^2$ , which is the deterministic limit of the stochastic process defined above [28]). However, beyond this transient region the stochastic exponent controls the decay, with different exponents for the homogenous and the inhomogeneous case. The values of the asymptotic decay  $\delta$  seems to be very close for  $N = 1$ ,  $N = 10$  (not shown) and  $N = 50$  (differences smaller than 0.02, which is more or less the numerical error in the measurement of the slope), so it seems that the exponent is independent of  $N$  either.

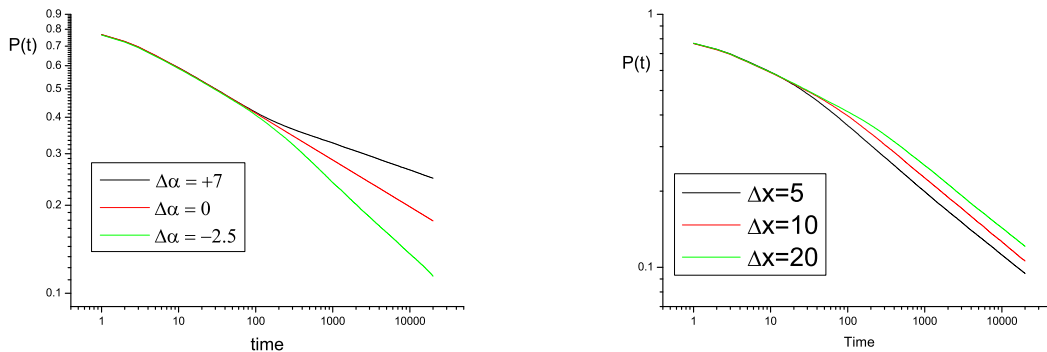


FIG. 3: Left panel:  $P(t)$  vs.  $t$  (loglog scale) for a contact process that starts with one agent at a distance  $\Delta x = 15$  from the heterogenous site. Three cases are compared: an oasis ( $\Delta\alpha = +7$ ), a sink ( $\Delta\alpha = -2.5$ ) and a homogenous system. Right panel: same, with a sink ( $\Delta\alpha = -2.5$ ) at the origin, where the three lines correspond to different  $\Delta x$ .

#### IV. TWO AND THREE SPATIAL DIMENSIONS: CONVERGENCE TO THE HOMOGENEOUS CRITICAL BEHAVIOR

All the results presented so far are for a 1d system. In higher dimensions the situation is different: here the effect of a compact heterogenous source/sink manifests itself only as a transient, but the asymptotic behavior of  $P(t)$  follows the exponent  $\delta_0$  that characterizes the homogenous system, as shown in Figures 7. Careful examination of the results seems to indicate that the long time decay is a combination of two exponent of the form:

$$P(t) \sim t^{-\delta_0} (1 + At^{-\delta_1}) \quad (2)$$

for a sink and

$$P(t) \sim \frac{t^{-\delta_0}}{(1 + At^{-\delta_2})} \quad (3)$$

for a source, i.e., that the transient behavior itself decays algebraically in time, with an exponent larger than  $\delta_0$ .

The qualitative difference between the behavior in 1d and in higher dimension suggest that the effect of a single defect has to do with the chance of a random walker to return to the origin. At criticality the average spatial size of the contact process is not shrinking or growing, and since there is no preferred direction the process visits spatial points at random (this feature has been already considered in the theory of persistence time [25]).



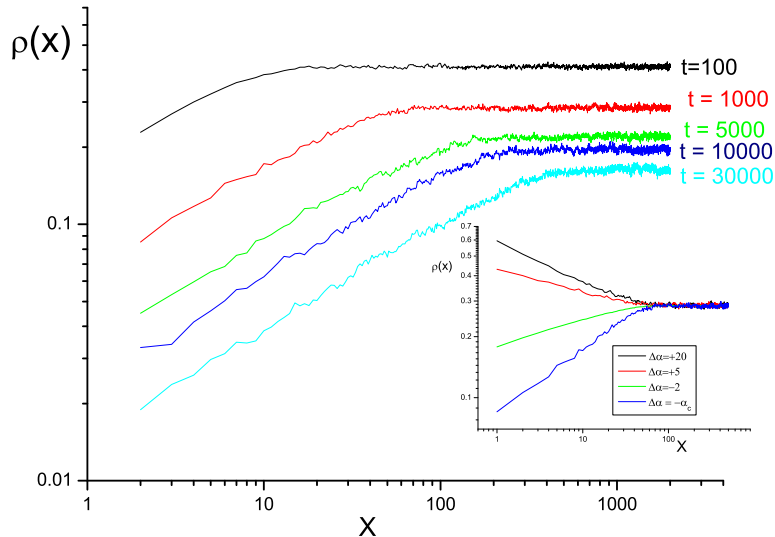


FIG. 4: The main panel shows a few "snapshots" of the spatial density profile,  $\rho(x)$ , for a contact process that starts from a fully occupied lattice with a sink ( $\Delta\alpha = -\alpha_c$ ) at the origin. Each line reflects the average results of 1000 histories. While in the far region the density decays algebraically with  $\delta_0$ , in the close region the decay is faster. The region affected by the sink is also growing. The inset shows the density profile at fixed time,  $t = 1000$ , for different types of heterogeneity at the origin. Clearly, in the far region there is no difference, and the distance in which the sink/source affects the system is independent of its strength and character.

Since in above 2d there is a good chance that the process never returns to the oasis, and 2d is marginal, only in 1d the oasis changes the critical exponent.

## V. DISCUSSION

Along this paper we have dealt with a single oasis/sink coupled to a critical bulk. This problem is quite similar to the one recently considered by Barato and Hinrichsen [26], in which an inert spatial system (at the bulk there are no birth-death processes, only diffusion) is coupled to a single active site on the boundary. In this setup the active site admits only one agent ( $N = 1$ ) with and is "critical" in the sense that the birth rate on this site is equal to the death rate. These authors reported a decay exponent  $1/6$  for that system. Later,

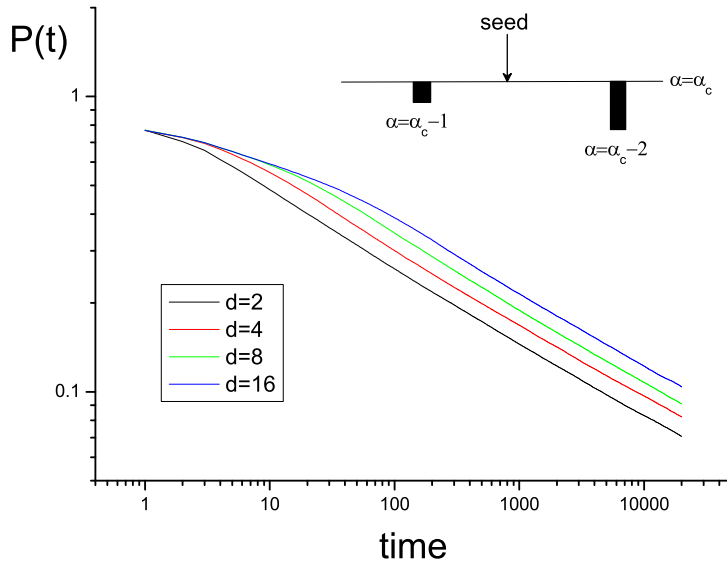


FIG. 5: The survival probability  $P(t)$  vs.  $t$  for a contact process. Here the bulk is at criticality with two, unequal, sink sites, one with  $\alpha = \alpha_c - 1$  and for the other  $\alpha = \alpha_c - 2$ . The process starts when a single seed is introduced at the origin, which is the midpoint between the two sinks, with a distance  $d$  from the seed to any of the heterogeneities. As  $d$  increases, the time it takes to the process to interact with the sinks is growing, still the asymptotic behavior is the same (the slope of all these lines is the same within the measurement error).

Burov and Kessler [27] have solved analytically the problem for  $N = \infty$  and found  $\delta = 1/4$ ; For any finite  $N$  their numerics indicated algebraic decay with different exponent.

In some sense, the model considered here and the model of [26, 27] are similar, as they both deal with an exceptional site coupled to a critical bulk. The main difference is that in our model the bulk is active and the criticality is "quantum", while the model considered before has an inert bulk. Still it will be interesting to check the response of the inert bulk model to a spatially structured defect.

An interesting question that may be asked in general with regard to these systems is about their deterministic limit, i.e., the behavior when  $N \rightarrow \infty$ . For the deterministic system the critical point is at  $\alpha = 1$  and a single defect coupled to a critical medium satisfies an equation

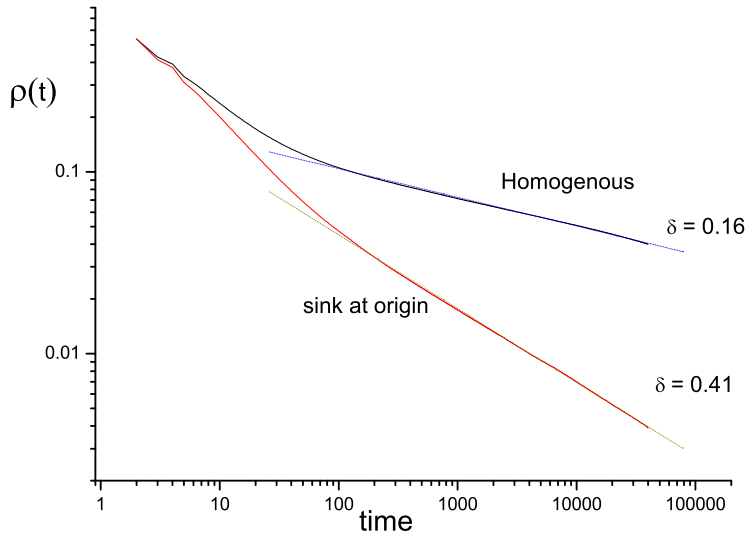


FIG. 6: The density  $\rho(t)$  vs. time for a system with carrying capacity of 50 agents per site. With  $\chi = 0.2$ , the transition for the homogenous system has been identified at  $\alpha_c = 1.1875$ . At criticality (black line) the decay starts with exponent  $\delta = 1$ , as expected from the mean-field equations, then when the stochasticity becomes important it crosses over to  $\delta_0$  of the DP transition (the dashed line is the linear fit for the tail). On the other hand, if a sink (site with  $\alpha = 0$ ) is introduced at the origin the long-time exponent is larger (red line) and the best fit to the tail (dashed) yields  $\delta = 0.41$ . The results reflect an average over  $10^6$  histories.

like

$$\dot{I} = \nabla^2 I + \alpha(x)I - I^2,$$

where  $\alpha(x)$  takes nonzero values at the spatial region associated with the defect. Since any potential well in 1d supports a localized state, so does the linearized evolution operator  $\mathcal{L} = \nabla^2 + \alpha(x)$ . Thus for any oasis, with any spatial structure, the system must admit a localized colony (its amplitude is dictated by the nonlinear term) that lives forever [29].  $P(t, N)$  is thus singular at the deterministic limit. In higher dimensions, on the other hand,

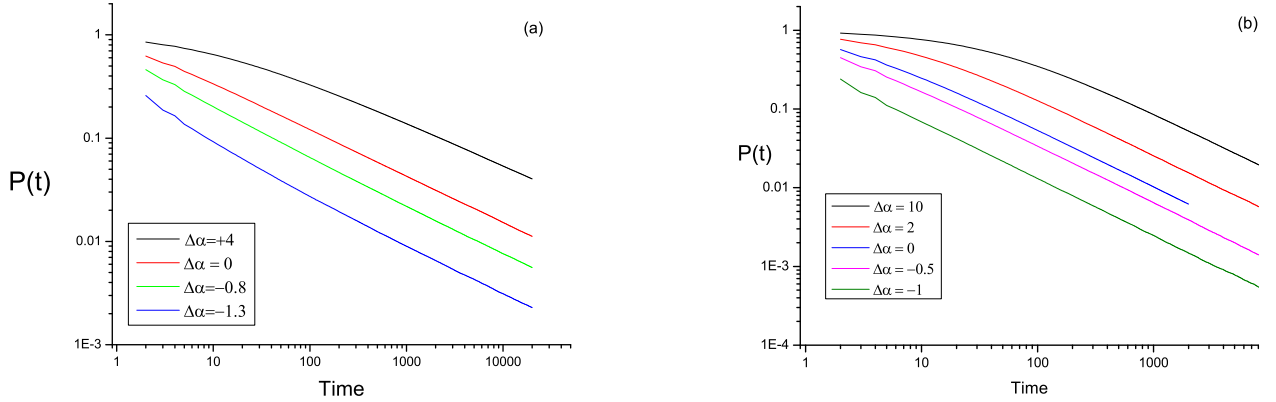


FIG. 7: The survival probability for a process initiated with a single seed at the origin for 2d (a) and 3d (b). The site at the origin has different reproduction rate. Either for source or for sink, the long-time decay seems identical and converge to the known values for the DP exponents in these dimensions. The bulk values of  $\alpha_c$  are 1.6488 for 2d and 1.3168 for 3d.

not any oasis supports a localized state, so the deterministic limit may be non-singular.

- 
- [1] T. Shaibi and R. Moritz, *Conservation Genetics* **11**, 2085-2089 (2010).
  - [2] Hanski I *Metapopulation Ecology*. Oxford University Press, Oxford, New York (1999).
  - [3] R. Etienne, C.J.F. ter Braak, C.C. Vos *Application of stochastic patch occupancy models to real metapopulations*, Elsevier, pp 105–132 (2004).
  - [4] A. Moilanen, SPOMSIM: software for stochastic patch occupancy models of metapopulation dynamics. *Ecological modeling* **179**,533 (2004)
  - [5] A. Moilanen and I. Hanski *Ecology* **79** 2503 (1998).
  - [6] T. E. Harris, Contact interactions on a lattice, *Ann. Prob.* **2**, 969 (1974).
  - [7] J. Marro and R. Dickman, *Nonequilibrium phase transitions in lattice models*, Cambridge University Press, Cambridge, 1999.
  - [8] P. Grassberger, *Z. Phys.* **B 47**, 365 (1982).
  - [9] H. K. Janssen, *Z. Phys.* **B 42**, 151 (1981).

- [10] H. Hinrichsen, *Adv. Phys.* **49**, 815 (2000).
- [11] L. Lübeck, *Int. J. Mod. Phys. B* **18**, 3977 (2004).
- [12] H. K. Janssen, *Phys. Rev. E* **55**, 6253 (1997).
- [13] J. E. Keymer et. al., *The American Naturalist* **156**, 478 (2000).
- [14] A. G. Moreira and R. Dickman, *Phys. Rev. E* **54**, R3090 (1996).
- [15] R. Cafiero, A. Gabrielli, and M. A. Muñoz *Phys. Rev. E* **57**, 5060 (1998); J. Hooyberghs, F. Iglói, and C. Vanderzande, *Phys. Rev. Lett.* **90**, 100601 (2003); T. Vojta and M. Dickison *Phys. Rev. E* **72**, 036126 (2005).
- [16] N.M. Shnerb, Y. Louzoun, E. Bettelheim and S. Solomon, The Importance of Being Discrete - Life Always Wins on the Surface, *Proc. Natl. Acad. Sci.*, **97** 10322 (2000); N.M. Shnerb, E. Bettelheim, O. Agam, S. Solomon and Y. Louzoun, Adaptation of Autocatalytic Fluctuations to Diffusive Noise, *Phys. Rev. E* **63**, 21103 (2001); S. Moalem and N. M. Shnerb, Unbounded autocatalytic growth on diffusive substrate: The extinction transition, *Phys. Lett. A* **366** 339 (2007); H. Kesten, V. Sidoravicius, Branching random walk with catalysts, *Electr. J. Prob.* **8**, 1 (2003).
- [17] R. Kree, B. Schaub, and B. Schmittmann, Effects of pollution on critical population dynamics, *Phys. Rev. A* **39**, 2214(1989); N. V. da Costa, U. L. Fulco, M. L. Lyra, and I. M. Gléria, Criticality of a contact process with coupled diffusive and nondiffusive fields, *Phys. Rev. E* **75**, 031112 (2007); J. E. de Freitas, L. S. Lucena, L. R. da Silva, and H. J. Hilhorst, Critical behavior of a two-species reaction-diffusion problem, *Phys. Rev. E* **61**, 6330 (2000); I. Dornic, H. Chaté, and M. A. Muñoz, Integration of Langevin Equations with Multiplicative Noise and the Viability of Field Theories for Absorbing Phase Transitions, *Phys. Rev. Lett.* **94**, 100601 (2005).
- [18] Ronald Dickman, A contact process with mobile disorder, *J. Stat. Mech.* **8**, P08016 (2009).
- [19] Directed percolation and the Extinction transition on diffusive substrate Galit Evron, David A. Kessler and Nadav M. Shnerb, *Physica A* **389** 428 (2010).
- [20] V. Colizza, R. Pastor-Satorras, A. Vespignani, *Nature Physics* **3**, 276-282 (2007); V. Colizza, A. Vespignani, *Phys. Rev. Lett.* **99**, 148701 (2007); V. Colizza, A. Barrat, M. Barthelemy, A.-J. Valleron and A. Vespignani, *PLoS Medicine* **4**(1): e13 (2007).
- [21] Yossi Ben-zion, Yahel Cohen and Nadav M. Shnerb, *Journal of theoretical biology* **264**, 197-204 (2010).

- [22] D.A. Kessler and N.M. Shnerb, *Journal of Statistical Physics* **127** 861 (2007).
- [23] A. Kamenev and B. Meerson *Phys Rev E* **77** 061107 (2008).
- [24] O. Ovaskainen and B. Meerson, *Trends in Ecology and Evolution* **25**, 643 (2010).
- [25] H. Hinrichsen and H.M. Koduvely, *The European Physical Journal B - Condensed Matter and Complex Systems*, **5**, 257-264 (1998).
- [26] A. C. Barato and H. Hinrichsen, *Boundary-Induced Nonequilibrium Phase Transition into an Absorbing State*, *Phys. Rev. Lett.* 100, 165701 (2008).
- [27] S. Burov and D.A. Kessler, *Non-Universal Extinction Transition for Boundary Active Site*, *cond-mat* 1001.1094 (2010).
- [28] D.A. Kessler and N.M. Shnerb, *Novel Exponents Control the quasi-deterministic limit of the extinction transition*, *J. Phys. A: Math Theor.* **41**, 292003(FTC) (2008).
- [29] D. R. Nelson and N. M. Shnerb, *Non-Hermitian localization and population biology*, *Phys. Rev. E* **58**, 1383 (1998).