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Microscopic noise, adaptation and survival in hostile environments

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Abstract. The survival of autocatalytic agents in hostile environments depends on their ability to adapt their spatial configuration to local fluctuations. A model of diffusive reactants that extract the advantage of spatio-temporal fluctuations associated with the stochastic wandering of diffusive catalysts is discussed. Two arguments are presented for the basic processes behind this extraordinary behavior. In the first, the local colonies that evolve around any spatially advantageous region overlap in space-time and an infinite directed percolation cluster emerges. The second argument is based on the return probability of a diffusive agent that is shown to yield finite density of active "oases" with an exponentially large contribution to the reactant population. The different range of applicability of these survival lower bounds to small systems is discussed.

PACS. 05.40.-a Fluctuation phenomena, random processes, noise, and Brownian motion – 64.60.Ak Renormalization-group, fractal, and percolation studies of phase transitions – 64.60.Ht Dynamic critical phenomena

1 Introduction

The stochastic effects associated with microscopic noise in spatially extended reactive systems have recently attracted a lot of interest [1–5]. These effects have been considered in a variety of contexts, including biochemical networks dynamics, ecology, population dynamics, disease control, and chemical reaction kinetics. The common analysis of these systems is based on rate equations, i.e., deterministic partial differential equations that assume large concentrations of individual reactants that are believed to allow for the use of continuous variables. Crossing to an agent based, spatially extended, stochastic picture, one finds a variety of new effects, unseen by the "mean field" deterministic formulation. In particular, the survival (or extinction) conditions for reactants may be shifted, leading to a new type of extinction transition. It was hypothesized that, generically, these transitions fall into the equivalence class of the directed percolation transition [6].

In previous publications [5,7], the quite unexpected resilience of a system that contains autocatalytic agents and immortal catalysts has been considered, and adaptation of the autocatalytic fluctuations to diffusive noise has been shown to lead to results that differ strongly from the PDE predictions. The system contains two species: catalyst agents (A) wandering around, and diffusive reactants (B) admitting an internal decay mechanism and yielding an offspring only in the presence of the catalyst. It turns out that the reactants may survive even below the mean field limit due to the Poissonian fluctuations associated with the discrete nature of the catalysts. These fluctuations allow (in a large enough system) regions to have a positive growth rate even if the decay process is very fast. As this exponential growth is spatially correlated to randomly occurring catalyst hotspots ("oasis" regions), the reactants tend to concentrate around these favored regions and multiply even further. These results turn out to be of importance for a wide range of applications, and a few generalizations of this "AB model" have been discussed recently [8].

This surprising result and its importance for understanding the resilience of various systems raises an essential question: what are the crucial assumptions underlying this result? In particular, the requirement for an arbitrarily large area of the surface is of relevance to real systems. In statistical mechanics terms, this evokes the issue or the commutativity between infinite volume and infinite time limits. It was shown [9] that, quite generally, there is a difference between the average and the typical local concentrations of the reactants in this system, and the average

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may reflect rare events of zero measure, such that it remains finite, or even diverges, while the probability for survival at a point may approach zero. In light of these results, the need for a simple theory that connects the rare events into the generic scenario is obvious. This paper is devoted to the presentation of two theoretical arguments that ensure the proliferation of the reactants below the rate equation threshold and are applicable, at least at some parameter range, to small systems.

The first argument presented here is based on the plausibility assumption used to translate the AB model to a model of directed percolation. The second argument is even more radical, as it depends on the growth at a fixed location, taking into account the diffusive correlations of the catalysts.

2 The AB model

In the AB model [5,7], the disagreement between the realistic stochastic process and the deterministic PDEs that pretend to describe it is emphasized for a very simple and generic system. The system includes two species: an immortal catalyst A only randomly diffusing in space, and a reactant agent B decaying with rate δ_B and proliferating in the presence of A-s at rate $\beta_B N_A$, where N_A is the number of A agents at the reactant spatial location (local density of A). Both A and B undergo diffusion with rates D_A and D_B respectively. Schematically, the local reactions considered are:

$$\begin{aligned} B &\longrightarrow \oslash \\ B + A &\longrightarrow B + B + A. \end{aligned} \tag{1}$$

The continuum approximation of this process involves the mean-field rate equations for the densities of A and B, $a(x,t)$ and $b(x,t)$ respectively. The rate equations are,

$$\begin{aligned} \frac{\partial a(x,t)}{\partial t} &= D_A \nabla^2 a(x,t) \\ \frac{\partial b(x,t)}{\partial t} &= D_B \nabla^2 b(x,t) - (\delta_B - \beta_B a(x,t)) b(x,t), \end{aligned} \tag{2}$$

admitting a very simple solution. Since only A diffuses, the catalyst density becomes spatially uniform μ_A (where μ_A is the average A density) after some time, and the dynamic of the reactants B is given by the linear equation,

$$b(x,t) = D_B \nabla^2 b(x,t) - M b(x,t), \tag{3}$$

where $M \equiv \delta_B - \beta_B \mu_A$ is the decay/growth rate of the system, depending on its sign. Thus, the mean field theory predicts a phase transition at $M = 0$. For positive M, the reactant concentration decays exponentially, while negative M yields exponential growth (proliferation). Of course, in realistic systems, one expects some saturation mechanism that prevents explosion, perhaps in the form of B agents competition for resources. However, for simplicity's sake, in the present work we neglect such a term, so the system admits two attractors, $b = 0$ and $b = \infty$.

The unexpected resilience of the B population in the agent based version of the AB model can be traced to the following mechanism: any arbitrary B death rate (leading to arbitrary local exponential decay) can be balanced by the growth induced by a large enough A aggregate at the same location. This effect could, in principle, be washed away by the fact that, due to A diffusion, the large A aggregates are short lived (the probability for an A to remain a time t at a give location decays with t). However, there are 3 main effects that support the survival of the B population:

1. even if an A aggregate decays, there is a certain probability that, by the time of its decay, another A aggregate will arise in its neighborhood. This will ensure the descendance of the B's generated by the first aggregate;
2. the exponential growth of the B population around large enough A aggregates compensates in the growth expectation the exponential decay of the aggregate survival probability;
3. in one or two dimensions, the probability for an A that left the aggregate to eventually return is 1.

Each of these processes is affected differently by the finite size. In the following sections, we will study each of them separately. We will first address and quantify the first effect, and then address the last two.

We briefly review here the main features of the AB model for discrete agents [5,7]. On each site x there exists at any time t an arbitrary number $N_A(x,t)$ of particles of type A and an arbitrary number $N_B(x,t)$ of particles of type B. Given an initial configuration $\{N_A(x,0), N_B(x,0)\}$, one iteratively generates the subsequent configurations $\{N_A(x,t), N_B(x,t)\}$ according to the following rules:

1. the particles A never "die" or "get born". They can jump on any of the d-dimensional sites neighboring their current location with a hopping probability of $D_A/2d$ for each neighbor;
2. the particles B can jump to any of the neighbors, with a hopping probability of $D_B/2d$;
3. the B's "die" with probability per unit time δ_B;
4. any pair of A and B located on the same lattice site can generate a new B with probability per unit time β_B.

In the following sections, some of the argumentation deals also with the generalized AB model, where A creation-annihilation processes are also allowed, keeping μ_A constant. The particular form of these reactions is $A \to \emptyset$ at rate δ_A and $\emptyset \to A$ at rate β_A, such that $\mu_a = \beta_A/\delta_A$.

3 Proliferation based on static catalysts

As noted in [7] and [9], in a case of static catalysts ($D_A = 0$) proliferation always takes place for an infinite size system. Random distribution of the catalysts A implies spatial Poissonian fluctuations of the local growth

rate, and this, in turn, implies that for large enough samples there will be finite density of "oases" (spatial regions where the overall growth rate is positive) no matter how large δ_B. If there is no dynamic for the catalysts, at least a finite fraction of these sites becomes "active" (i.e., yield a flourishing colony of reactants), resulting in the proliferation phase.

3.1 Single oasis and colony dynamics

The case of local growth of diffusive reactants on a spatial domain with a single active site may be solved and yields a simple and intuitive framework. For the sake of simplicity, we present an off lattice evaluation with some assumptions about the shape and size of the active site, but a possible generalization for the other cases is straightforward and the basic intuition is the same. At the end of this subsection, the translation of the results to a discrete lattice is presented. Clearly, even if the A agents are standing still, the treatment presented here is approximate, as it neglects the discrete nature of the B agents. However, as long as the proliferation leads to a large number of reactants at the oasis — and this must be the case for an unbounded model — this inattention is not consequential.

The growth around an active site is described by the equation,

$$\frac{\partial b(\mathbf{x},t)}{\partial t} = \widetilde{D}_B \nabla^2 b(\mathbf{x},t) + g(\mathbf{x}) b, \tag{4}$$

where $g(x)$ is a parameter that incorporates all the parameters that effects the growth, $g(x) = \beta_B N_A(x) - \delta_B$. A single active site of radius R corresponds to:

$$g(r) = \begin{cases} g_0 & r < R \\ -g_1 & r > R \end{cases} \tag{5}$$

where g_0 and g_1 are positive. This linear problem may be solved (see [8], Appendix A) using spherically symmetric functions in the physical dimensions.

Equation (4) is linear and its solution involves the presentation of a complete set of eigenfunctions $\phi_n(r)$ with the corresponding eigenvalues Γ_n. Accordingly, any initial state $b(x,0)$ may by written as a superposition of the eigenfunction $b(x,0) = \sum_n \alpha_n \phi_n$ and its time evolution is given by,

$$b(x,t) = \sum_n \alpha_n \phi_n e^{\Gamma_n t}. \tag{6}$$

Solving (4), (which is equivalent to the Schrödinger equation for a single quantum particle in a potential well) one finds that there are two types of eigenfunctions. Localized eigenfunctions decay exponentially out of the "oasis", $\phi_n(r) \sim \exp(-\kappa_n r)$, and admit positive eigenvalues, while extended eigenfunctions have zero support on the oasis and allow only negative values of Γ. The profile of a positive eigenfunction is thus,

$$\phi_n(r,t) \sim e^{-\kappa r + \Gamma_n t}, \tag{7}$$

so a level point (a point of constant height $\phi(r,t) = \text{const.}$ on the profile) travels away from the oasis with velocity $v = \Gamma/\kappa$. If the B agents dynamic is approximated as a continuum dynamic [like in (4)] with a finite *threshold*, this threshold dictates the level point discussed above. However, as long as the concentration of the reactants on the oasis is large (compared to unity), the velocity is threshold independent. Clearly, our interest lies not in all the spectrum of the linearized evolution operator; but rather, only in its fastest growing (maximal Γ) state.

Let us consider now the details of the fastest growing state in the physical dimensions. First we note that in $1d$ there is *always* a localized solution with positive eigenvalue, independent of the reactants diffusion constant, while in three dimensions this is not the case, and for any oasis there is no bound state if the reactant diffusion is large. In $2d$ the situation is marginal, since this is the critical dimension for the return of a random walker: there is a positive eigenvalue for each oasis, independent of the reactant diffusion rate, but for very weak island ($g_0 R^2/\widetilde{D}_B \ll 1$) the growth rate $\Gamma = \widetilde{D}_B \exp(-4\widetilde{D}_B/g_0 R^2)/R^2$ approaches zero exponentially as diffusion grows. Another peculiarity of the $d = 2$ case is logarithmic corrections to the exponential decay of the spatial profile far away from the oasis and the corresponding correction to the velocity of the colony.

In the limit of a "strong" oasis ($g_0 R^2/\widetilde{D}_B \gg 1$) it is possible to write down the asymptotic velocity of the colony's front as,

$$v = \sqrt{\widetilde{D}_B (g_0 - \theta^2 \widetilde{D}_B / R^2)}, \tag{8}$$

where $\theta = \pi/2, [z_0^{(1)}]$ and π in one, two and three dimensions correspondingly ($[z_0^{(1)}] = 2.402...$ is the first zero of the zero order Bessel function).

The translation of the above results to the discrete lattice dynamics is trivial, as the basic length scale of is now the lattice constant l_0. The diffusion coefficient of the lattice is just a hopping rate and is related to the continuum diffusion by $D_B \equiv \widetilde{D}_B / l_0^2$. The velocity, again, is given by the continuum velocity multiplied by l_0, where D_B should be plugged instead of $\widetilde{D}_B$. The radius of the oasis is of order l_0, but a better approximation for the coefficient is $\theta^2 \sim 2d$.

3.2 Finite size effects for autocatalytic growth on heterogeneous substrates

Following the analysis of the single oasis problem, let us discuss, still for immobile catalysts, the effects of finite sample size and sample to sample fluctuations, where in this subsection a lattice of N spatial sites is considered. As shown in the last section, if the number (m) of catalysts at a point is larger than some critical number m_c (that depends on the dimension of the system and the reactants' death rate) an oasis occurs where a B colony grows. The presence of subthreshold catalyst density around the oasis

has a minor effect on θ^2 and is ignored. As the A-s are randomly distributed, the probability to find m catalysts at a lattice point obeys the Poisson distribution:

$$P(N_A(x,t)=m)=\frac{e^{-\mu_A}{\mu_A}^m}{m!}. \quad (9)$$

Thus, the probability for an active oasis is the sum over (9) from m_c to infinity:

$$P(m>m_c)=1-\frac{\Gamma(m_c,\mu_A)}{\Gamma(m_c)}. \quad (10)$$

If the number of lattice sites is much larger than $1/P(m > m_c)$, one expects a finite density of active sites in each random sample. However, if the number of lattice sites is much smaller than $1/P(m > m_c)$, only (exponentially) rare samples will be "active" (i.e., contain at least one oasis), while all the others are inactive, and the typical case (determined by the inactive samples) differs from the average (determined by exponentially rare fluctuations). In terms of the lattice constant l_0, the typical distance between oases is $R \sim l_0 \left(P(m > m_c)\right)^{-d}$, and if the probability to find an active site is small, $P(m > m_c)$ may be approximated by P_{m_c}.

4 Directed percolation and the proliferation phase

As already discussed in [7,9], a large enough sample with frozen catalysts always supports the proliferation of the reactants, as there is no bound (in this model) for the growth of a colony based on a single oasis. The case of diffusive catalysts is different: here the oasis is unstable, since any individual local fluctuation of the A concentration should decay in time. The main question here concerns the lifetime of a fluctuation. Given μ_A, the average number of catalysts per site, and a fluctuation size $m > m_c$, what is the typical time until m decays to m_c? In a case of *continuous* field diffusion with random initial conditions, this problem is known as the problem of persistence diffusion. The probability of a fluctuation to maintain its sign (i.e., that it never crosses the average) until t has been shown to obey a power law distribution, $P(t) \sim t^{-\theta}$, where θ depends upon the dimension of the system [10,11]. This power law behavior has also been demonstrated for the probability of not crossing arbitrary values that differ from the average, with different θ [12]. However, the problem considered here differs from these persistent diffusion cases as it involves the stochastic wandering of *discrete* agents (the A-s). At equilibrium, the system is invariant under time translation, correlation functions depend only on time differences, and the stochastic noise leads to a crossover from a power law (far from equilibrium) to an *exponential* decay at equilibrium. This crossover will be discussed elsewhere, but for the sake of this work, let us try to find a lower bound to the persistence of the oasis. We look only at oases just above the critical number of catalysts m_c, such that, as soon as a single catalyst leaves, the B-s growth rate at this spatial location becomes non-positive. Thus, the lower bound for the lifetime is simply the inverse hopping rate at the critical density $\sim 1/m_c D_A$. This is so because, in the worst case, any catalyst that leaves the oasis never returns, and the diffusion plays the same role as death rate for the A-s in the active site. This idea may be extended to include other processes in the catalysts' dynamic; for example, if the A-s decay with probability δ_A the lower bound is $1/[m_c(D_A + \delta_A)]$ and so on.

4.1 From single oasis to directed percolation

At this point, we gather all the information from the above sections in order to build a directed percolation picture for the AB extinction transition. In the limit of an infinite sample we already have a finite density, $1/P(m > m_c)$, of active sites, and the lifetime of such an active site is approximated by a constant $1/m_c D_A$. In a space time diagram the active sites may thus be approximated as "rods" of constant length. Around each of these rods a B-colony is developed, with a size that grows linearly in time, so these colonies (in 1+1 dimensions, for example) look like triangles centered around each of these rods.

Within this picture, the problem of extinction-proliferation transition is translated into the problem of directed percolation of the space time triangles (in 1+1, or cones in 2+1 etc.). The density of the triangles is determined by the A agents' statistics, their length (lifespan) by the catalysts dynamics, and their width by the reactants growth and diffusion rate.

As described above, given the parameters δ_B, μ_A, β_B and the hopping rate for each of the species, a site is active if $N_A(x) \gtrsim \delta_B/\beta_B$ (as explained above, in $2d$, this condition is sufficient for the existence of an oasis, where a colony may grow). An estimate for the lifetime of such an island is $\beta_B/[\delta_B(\delta_A + D_A)]$. We now take the limit of "strong" oasis, (8), in order to get an order of magnitude estimation. Given that the number of catalysts needed at a single spatial point is not large, fluctuations of $O(1)$ correspond to the strong oasis limit, assuming that $g_0 - 2dD_B$ is, again, of order one. If this is the case, the velocity of the colony front is $\sqrt{D_B \beta_B l_0^2}$, and thus the radius of the colony when the oasis disintegrated is $R_0 = \beta_B \sqrt{D_B \beta_B l_0^2}/[\delta_B(\delta_A + D_A)]$. On the other hand, the typical distance between neighboring oases scales like the inverse probability, given by equation (9), and in $2d$ for rare oases this will be given roughly by $R_1 = \exp[\delta_B/(2\beta_B)]$. The filling fraction of the sample is given by the ratio $p = (R_0/R_1)^d$. Thus, the fate of the system is determined by the relation between p and p_c^d, the critical filling fraction for directed percolation at certain dimensionality. Clearly, p grows with β_B and D_B, as the size of the colony around the oasis is larger. p decays with δ_B, δ_A and D_A, the limiting factors for the size and the lifetime of the colony.

As we have already mentioned, this argument only sketched approximate "lower bound" conditions for the survival of the reactants below the mean field threshold.

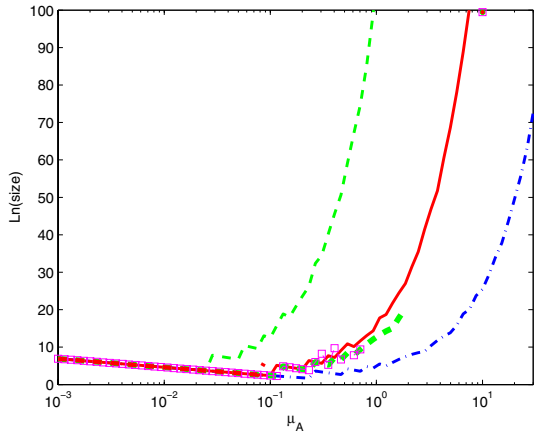

Fig. 1. Effect of scale. The mean field description of the AB model (either with or without catalyst death) is only a function of $M = \delta_B - \mu_A\beta_B$. These systems perform very differently in the strong (low values of μ_A and high values of β_B) and weak (high values of μ_A and low values of β_B) coupling limits. A clear difference is the effect of the system size. Each curve represents the system size required for survival for a given value of M ($\delta_B = 1.0$ and $\beta_B\mu_A$ are 0.05, 0.2 and 0.5 in the long dashed, full and dashed-doted line respectively). At high values of μ_A, a large system is required in order to find a fluctuation large enough to allow survival. At low values of μ_A, only single A agent is required to allow survival.

The effects of rare events may lead to the appearance of long living, localized colonies as discussed in the next section. Our discussion also neglects the space-time correlation among islands: as the diffusion of an A catalyst around the location of large concentration is correlated, the lifetime of a colony may thus be larger then expected as the reactant concentration follows the catalysts.

An important example may be found in the case of single A based proliferation, i.e., where a site is active (admits local growth) if it contains a single catalyst. In this case, the considerations presented above regarding the lifetime of an oasis are irrelevant, since there are strong correlations between the spatial location of different oases (in fact, the next active point appears in an adjusting site due to the diffusive wandering of the catalyst). This phenomenon manifests itself in Figure 1: at the single A regime, the extinction transition is almost independent of the system size (the critical size grows linearly with the inverse density of the catalysts). In the opposite regime, i.e., where an active site requires a large number of catalysts, the corresponding fluctuations are exponentially rare, implying strong dependence on the system size. In the next section, this single A proliferation based limit will be considered in detail.

5 Local criteria for population growth

In the previous section, we have described a B-population survival mechanism based on a directed percolation analysis. That analysis neglected the spatio-temporal correlations in the number $N_A(x,t)$ of catalysts A at various sites x and times t. The effect of an A diffusing away from a site x was treated as the irreversible death of that A for what concerned the estimation of the number of B's on that site (denoted by $N_B(x,t)$).

In this section we estimate the expected value of $N_B(x,t)$ ($E[N_B(x,t)]$), while taking into account the probability, $Q_d(t)$, that an A originating at time $t=0$ on site x will return to x at least once in the time interval $[0,t]$. The main result of the present section is a sufficient condition,

$$\beta_B > \beta_{min} = D_A(1 - Q_d(\infty)), \tag{11}$$

for the (at least) exponential growth (rather then decay) of the average number of B's in the system. As $Q_1(\infty) = Q_2(\infty) = 1$ (see Fig. 2), in 1 and 2 dimensions $\beta_{min} = 0$, thus the total B population always growth.

To show that equation (11) indeed constitutes a sufficient condition for an increase of the average B population, the following lower bound arguments are invoked. First, we neglect the contribution to $N_B(x,t)$ of B's born on a different site $y \neq x$ or B-s returning to x after having left it. Thus, for the purpose of the present section, we take the "lower bound" assumption that an agent B disappears once it leaves its site of origin x. Therefore, the effect over a time interval $[0,\, t]$ of the B's diffusion is simply $\exp\{-tD_B\}$, similar to the effects of B's death $\exp\{-t\delta_B)$. In the absence of catalysts, the lower bound for the expected B population $N_B(x,t)$ would be:

$$E[N_B(x,t)] \geq N_B(x,0)exp\{-t(\delta_B + D_B)\}. \tag{12}$$

In the presence of catalysts, we also limit ourselves to the contribution of the A-s that, at $t=0$ were located at x and neglect all other catalysts. Thus, from now on, $N_A(x,t)$ is the number A's that reside on x at time t but also resided on x at $t=0$. Since those (and previous) assumptions work against the B population survival, they are legitimate in the computation of a sufficient condition for the B growth/survival.

The jumps, deaths, and births of different B-particles are independent [9]. Therefore their effect on $E[N_B(x,t)]$ factorizes, and we can evaluate their contributions separately and only in the end multiply them to obtain the necessary condition of growth for $E[N_B(x,t)]$, using the inequality,

$$E[N_B(x,t)] \geq N_B(x,0)\exp\{-(\delta_B + D_B)t\} \times E[\exp(\beta_B \int^t N_A(x,\tau)d\tau)]. \tag{13}$$

Furthermore, the A-particles perform simple random walks independent of one another. Their contributions in the product (13) factorize into the product of $N_A(x,0)$ single A contributions,

$$E[N_B(x,t)] \geq N_B(x,0)\exp\{-(\delta_B + D_B)t\} \times \prod_{k=1}^{N_A(x,0)} \left(E[\exp(\beta_B \int^t n_k(x,\tau)d\tau)]\right), \tag{14}$$

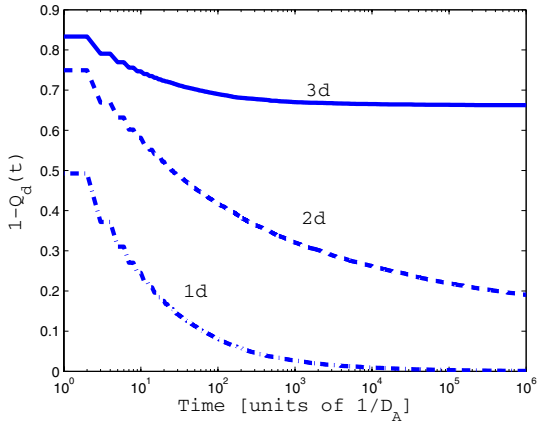

Fig. 2. A dynamics in 1, 2 and 3 dimensions. The drawn curves represents one minus the probability that A agents diffusing from a given point would return to this point at least once within a period of t. The probability of not returning converges rapidly to zero in one dimension, more slowly in two dimensions, and converges to a finite values in three and more dimensions.

where $n_k(x,\tau)$ equals one when the k'th A agent is present at x and zero otherwise.

Let us concentrate on a single agent (say, the agent with $k=1$). Defining,

$$Z(t) \equiv E[\exp(\beta_B \int^t n_1(x,\tau)d\tau)],$$

equation (14) may be written as:

$$E[N_B(x,t)] \geq N_B(x,0)\exp\{-(\delta_B+D_B)t\}Z(t)^{N_A(x,0)}. \tag{15}$$

So the sufficient condition for average growth is:

$$(\delta_B+D_B)t < N_A(x,0)\ \ln Z(t). \tag{16}$$

Clearly,

$$E[\exp(\beta_B \int^t n_1(x,\tau)d\tau)] \geq E[\exp(\beta_B\tau_1)], \tag{17}$$

where τ_1 is the time between $t=0$ and the first jump of the A agent out of $x=0$. The probability distribution function for τ_1 is exponential, $P(\tau_1) = D_A\exp(-D_A\tau_1)$, and hence, by integrating over τ_1,

$$Z(t) \equiv E[\exp(\beta_B \int^t n_1(x,\tau)d\tau)] \geq \frac{D_A[e^{(\beta_B-D_A)t}-1]}{\beta_B-D_A}. \tag{18}$$

Now let us make a distinction between two situations. If $\beta_B > D_A$ we can get the lower bound directly. For a large enough t, $\ln Z(t) \sim (\beta_B - D_A)t$ up to a additive, time independent term. Thus, a sufficient condition for an average growth will be

$$N_A(x,0)(\beta_B - D_A) > (\delta_B + D_B) \tag{19}$$

or

$$N_A(x,0) > N_0 = (\delta_B+D_B)/(\beta_B-D_A). \tag{20}$$

Conversely, if $D_A > \beta_B$, one has to consider the effect of multiple returns of the same A-agent to x. We now estimate the contribution to $Z(t)$ of the return of a single A to its initial location. If the condition stated above (11) holds,

$$Q_d(\infty)\frac{1}{1-\beta_B/D_A} > 1, \tag{21}$$

one can define a large enough return time τ_2, followed by sojourn time τ_1, such that the contribution of such single-return events to Z exceeds some predetermined value $\exp(\eta)$ (with $\eta > 0$):

$$\exp(\eta) = Q_d(\tau_2)\frac{[1-e^{(\beta_B-D_A)\tau_1}]}{1-\beta_B/D_A} > 1.$$

The contribution of a succession of N such events to Z is $\exp(\eta N)$. Since this contribution is positive for each $\tau_2+\tau_1$ step, and since all steps are independent, the fact that the expectancy of $Z(t)$ grows in each such step ensures that $E(Z(t))$ will grow indefinitely. As $N = t/(\tau_2+\tau_1)$, the contribution to Z is now growing in time,

$$Z \sim \exp\left(\frac{\eta}{\tau_1+\tau_2}t\right) \tag{22}$$

where η is some numerical factor. This yields the average local proliferation condition,

$$N_A(0)\ \eta/(\tau_1+\tau_2) > \delta_B + D_B. \tag{23}$$

Since the A-s are initially Poissonianly distributed, there will always be a finite density of points for which $N_A(x,0)$ satisfy the condition (23).

6 Conclusions

The discrete proliferating model studied in the present paper reveals the importance of spatial effects and sample size on the survival of an autocatalytic system in stochastic environments. Beyond the conceptual issues, there are many concrete applications of the present work to biology, economics, and social sciences. For instance, the present results shed new light on the dangers posed by the shrinking and fractionalizing of natural habitats upon the survival of dependent species. The same mathematical formalism represents the problems related to the sustainability of the present world economic system [16], where the effects of the market size and globalization are presently under close scrutiny and heated debate. In the current work, we have proposed a mathematical treatment of the habitat/economy size effects on systems' resilience and sustainability. As opposed to many cases where the mean field arguments (using average agents density or a continuous distributions, with rate equations as a mathematical tool) are applicable, the dynamics of the present systems may be dominated by rare spatio-temporal fluctuations. This means that the survival of species or economies under hostile conditions is still possible through the rare occurrence of lucky events. Two survival mechanisms have

been pointed out: the first, and the simplest, relies on the presence of a static, random distribution of catalysts, where the relevant spatial scale is inversely proportional to the chance of finding a single oasis. If the environmental conditions are not static, any oasis is bound to decay, and the survival is based on percolation of the reactants among the short-lived favored locations. In that case, the relevant spatial scale needed for survival depends on the space-time inhomogeneity; to allow for the directed percolation survival, the system should be much larger than the radius of a single colony R_0. Note that, in this regime, even at infinite system size the B population may become extinct if the average catalyst density falls below the percolation threshold. The second survival mechanism, considered in the previous section, is based on rare events where a dynamic environment remains, by chance, static for a relatively long time. While such events are exponentially rare, their contribution to the survival of the reactants is exponentially large. The present work suggests the performance of a meta analysis that would provide the probabilities of survival in specific systems in the presence of various constraint. For example, one may compare living habitats consisting of disconnected patches with systems of the same total size, but made of a single connected component, as has been recently done in various experiments [14]. We present, in other publications, some studies that confront the present theoretical results with empirical data from markets [15], economics [16], population dynamics and other branches of science.

Appendix A: Difference between A diffusion and A death

6.1 Dynamics of non-static catalyst

Let us look without loss of generality at the population $N_A(t) = N_A(x = 0, t)$, and assume an initial value of $N_A(0)$. In contrast with the dying A agents, the diffusing A catalysts can come back at time t with a probability $P_R(D_A t)$. The cumulative probability asymptotically reaches 1 in one and two dimensions and saturates at finite values for higher dimensions (Fig. A.1). The only difference between diffusion and A annihilation is that diffusing A can return, while dying A never do. The A dynamics can be approximated by:

$$\dot{a(t)} = -(\delta_A + D_A)a(t) + \beta_A + D_A\mu_A + \int_{\tau=0}^{\tau=t} a(t-\tau)P_R(D_A\tau)e^{-\delta_A\tau}d\tau. \quad (24)$$

Let us assume a large A fluctuation $a(0) \gg mu_A$. We can ignore the contribution of the mean and obtain the following function for the A population,

$$a(t-\tau) = a(t)e^{(D_A+\delta_A-D_AC(D_A\tau))\tau}, \quad (25)$$

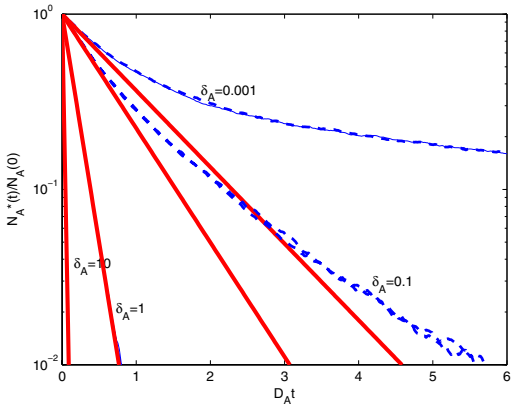

Fig. A.1. 1D combination of A agent death and diffusion. One can see that the precise solution (dashed lines) and the solution ignoring A returns (full lines)are similar until a tenfold decrease in A and then the full solution converges to a line with a slope similar to the one of the destruction rate only.

where $C(D_A\tau)$ represents the fact that diffusing A agents can return. 24 then becomes:

$$\dot{a(t)} = -(\delta_A+D_A)a(t)+\beta_A+D_A\mu_A + a(t)\int_{\tau=0}^{\tau=t} e^{(\delta_A+D_A-D_AC(D_A\tau))\tau}P_R(D_A\tau)e^{-\delta_A\tau}d\tau. \quad (26)$$

If we define $C(D_At)$ to be the function solving,

$$C(D_At) = \frac{1}{D_A}\int_{\tau=0}^{\tau=t} e^{(D_A(1-C(D_A\tau)))\tau}P_R(D_A\tau)d\tau, \quad (27)$$

and assume that $C(t)$ is changing slowly, we obtain from 25 and 27 an approximate solution for 24:

$$\dot{a(t)} \sim -(\delta_A + D_A(1 - C(D_At)))a(t) + \beta_A + D_A\mu_A \quad (28)$$

$$e^{(C(t)-1)t}\frac{dC(t)}{dt} = P_R(1,t). \quad (29)$$

This produces a step like function for $C(t)$ and an approximate solution of:

$$a(t) = (a(0) - \frac{\beta_A}{\delta_A}) * e^{-(\delta_A+D_A(1-C(D_At)))t} + \frac{\beta_A}{\delta_A}. \quad (30)$$

One can clearly see in Figure A.1 that this estimate produces a very close approximation of the precise process. Thus, the only difference between diffusion and A annihilation is a correction to the diffusion term, which lessens its destructive effect. Interestingly, $C(t)$ is small for low values of t and rises sharply to unity for high values, such that, for short times, the effect of diffusion is similar to death, while for long times it is completely negligible. In order to reach the limit where diffusion can be canceled, one has to wait for a time long enough that the aggregate size would decrease more than tenfold. In a very large system, this situation is realistic, as shall be further discussed, but in typical systems, the probability of finding such aggregates is very small. If we only treat the short time limit, we can assume that $C(t) = 0$ and treat diffusion and death equally.

Appendix B: Directed percolation: full treatment

Here we reproduce and enlarge a part of the results mentioned in [8]. The directed percolation argument raised in [8] is that the B population in the system will survive if and only if the d dimensional volume containing a B population surrounding an A aggregate will contain a new A aggregate when the original A aggregate disappears. More precisely, the argument is that the B density distribution around an A aggregate is approximately:

$$b(r,t) \sim b(0,0)e^{(\beta_B a(t)-\delta_B-D_B)t-\sqrt{\frac{(\beta_B a(t)-D_B)}{D_B}}r}. \quad (31)$$

The B isodensity line advances with a velocity of $\frac{(\beta_B a(t)-\delta_B-D_B)}{\sqrt{\frac{(\beta_B a(t)-D_B)}{D_B}}}$. The condition for growth at $x=0$ (the A aggregate position) is $a(t) > \frac{\delta_B+D_B}{\beta_B}$. Thus, given an initial A density, we can approximate that the B population will grow at $x=0$ until

$$t_{end} = \frac{\ln\left(\frac{a(0)-\frac{\beta_A}{\delta_A}}{\frac{\delta_B+D_B}{\beta_B}-\frac{\beta_A}{\delta_A}}\right)}{(\delta_A+D_A)}$$

(as we approach the mean field survival threshold, t_{end} approaches infinity, reproducing the mean field survival). The maximal island radius can be approximated by:

$$r(t) = \int_{(\beta_B a(t)-\delta_B-D_B)>0} \frac{(\beta_B a(t)-\delta_B-D_B)}{\sqrt{\frac{(\beta_B a(t)-D_B)}{D_B}}}dt. \quad (32)$$

If we set $a(0)=m$, we can substitute the value of $a(t)$ to obtain:

$$r(t) = \int_0^{t_{end}} \frac{(\beta_B(m-\mu_A)e^{-d_A t}+\beta_B\mu_A-\delta_B-D_B)}{\sqrt{\frac{(\beta_B m-\mu_A)e^{-d_A t}+\beta_B\mu_A-D_B)}{D_B}}}dt, \quad (33)$$

where we have defined: $\mu_A = \beta_A/\delta_A$ and $d_a = \delta_A + D_A$. In a space time diagram the active sites may be approximated as "rods" of constant length. Around each of these rods a B-colony is developed. The extinction-proliferation transition is translated into a directed percolation question. Taking into account only A aggregates with at least m A-agents, the average radius between such aggregates in d dimensions can be approximated by:

$$R_m^d = e^{\frac{\beta_A}{\delta_A}}m!\frac{\delta_A}{\beta_A}^m. \quad (34)$$

The survival condition thus becomes [1] :

$$r(t_{end}(m))^d > P_c e^{\frac{\beta_A}{\delta_A}}m!\frac{\delta_A}{\beta_A}^m. \quad (35)$$

[1] This equation has an analytical solution, defining $x = \frac{\delta_B}{|\beta_B\mu_A-D_B|}$. and $w = \frac{\beta_B m-D_B)}{|\beta_B\mu_A-D_B|}$ to obtain $f(x,w) = 2(\sqrt{w}-\sqrt{x}) + (1-x)(\ln(\frac{\sqrt{w}-1}{\sqrt{w}+1}) - \ln(\frac{\sqrt{x}-1}{\sqrt{x}+1})$ or $f(x,w) = 2(\sqrt{w}-\sqrt{x}) + \frac{m_0 x}{\delta_B}(\text{arctag}\sqrt{w} - \text{arctag}\sqrt{x})$ depending on the sign of $\beta_B\mu_A - D_B$):$f(x,w) > \frac{D_A+\delta_A}{\sqrt{|D_B(\beta_B\mu_A-D_B)|}}0.7\sqrt{e^{\mu_A}m!\frac{1}{\mu_A}^m}$

In order for the system to survive, there should be at least one such m. For a given set of parameters $(\delta_B, \beta_B, \mu_A, D_B)$ there is a cutoff value of $\delta_A + D_B$, under which the infinite system survives, given by

$$\max_{m<m_V} r(t_{end}(m))^d - P_c e^{\frac{\beta_A}{\delta_A}}m!\frac{\delta_A}{\beta_A}^m > 0, \quad (36)$$

where m_V is the highest value of m fulfilling $VP(a(0,t) = m) \gg 1$. A close inspection of the solution of (36) shows that for high β_B and low μ_A values, the system is dominated by low values of m and the effect of increasing the system size rapidly saturates. For low β_B values and high μ_A values, the system is dominated by high m values. The presence of such high m values is a function of the system size. In such systems, the B survival probability will keep increasing with system size.

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References

1. J.S. van Zon, P.R. ten Wolde, Phys. Rev. Lett. **94**, 128103 (2005)
2. Y. Togashi, K. Kaneko, Phys. Rev. E **70**, 020901(R) (2004)
3. E. Brunet, B. Derrida, J. Stat. Phys. **103**, 269 (2001)
4. J.L. Cardy, U.C. Tauber, Phys. Rev. Lett. **77**, 4780 (1996); J.L. Cardy, U.C. Tauber, Jour. Stat. Phys. **90**, 1 (1998)
5. N.M. Shnerb, Y. Louzoun, E. Bettelheim, S. Solomon, Proc. Natl. Acad. Sci. **97**, 10322 (2000)
6. P. Grassberger, Z. Phys. B: Condens. Matter **47**, 465 (1982); H.K. Janssen, Z. Physik. **42**, 141 (1981)
7. N.M. Shnerb, E. Bettelheim, Y. Louzoun, O. Agam, S. Solomon, Phys. Rev. E **63**, 021103 (2001)
8. A. Agranovich, Y. Louzoun, N. Shnerb, A. Moalem, J. Theoretical Biolgy **241**, 309 (2006)
9. H. Kesten, V. Sidoravicius, Electr. J. Prob. **8**, 1 (2003)
10. S.N. Majumdar, C. Sire, A.J. Bray, S.J, Cornell, Phys. Rev. Lett. **77**, 2867 (1996)
11. B. Derrida, V. Hakim, R. Zeitak, Phys. Rev. Lett. **77**, 2871 (1996)
12. I. Dornic, C. Godrche, J. Phys. A **31**, 5413 (1998)
13. F. Dyson, Rev. Mod. Phys. **51**, 447 (1979)
14. M. Holyoak, S.P. Lawler, Ecology **77**, 1867 (1996); S.P. Ellner et al., Nature **412**, 538 (2001)
15. J. Goldenberg, B. Libai, Y. Louzon, D. Mazursky, S. Solomon, *Technological Forecasting and Social Change*, 71/9 Nov. (2004)
16. Y. Louzoun, S. Solomon, J. Goldenberg, D. Mazursky, Artificial Life **9**, 357 (2003)