Power-law relaxation of spatially disordered stochastic cellular automata and directed percolation

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(Received 24 December 1987)

This paper concerns a large class of two-state stochastic cellular automata (SCA) with inhomogeneous transition probabilities, related to generalized directed percolation (DP) on lattices with random linear defects along the preferred direction. These models were previously shown to have universal critical behavior distinct from that of standard DP. Here, I analyze the relaxation to the "vacuum," the only absorbing state of such SCA. Asymptotic power-law decay with a variable exponent is derived for a substantial region in the parameter space of simple D = 1 models. Disordered SCA in D = 2 and 3 are more complicated, but similar power-law decay is still established by means of lower and upper bounds. The long-tailed relaxation is due to rare, very slowly decaying clusters. This is similar to the mechanism that causes the "Griffiths' phase" in disordered spin models.

I. INTRODUCTION

Stochastic cellular automata (SCA) can be useful models for collective behavior and phase transitions in nonequilibrium systems, capturing just the barest essentials of large interacting systems. However, our present understanding of the statistical physics of SCA, lacking Hamiltonians and detailed balance, is limited compared to that of equilibrium spin models. This paper concerns a broad family of SCA that model, e.g., autocatalytic propagation phenomena in physics, astrophysics, chemistry, and biology. Several types of SCA from this family have been studied in recent years.¹⁻⁵ All such SCA consist of cells with two states (0,1) and have parallel dynamics defined by transition probabilities depending on the states of cells within a finite range. An essential characteristic is the existence of exactly one global absorbing state (the "vacuum", all 0's). An additional "active" phase can exist when the interactions between cells are large enough for a finite density of 1's to propagate for unlimited lengths of time.

If such SCA are restricted to have the translation invariance of a lattice in *D*-dimensional space, then their critical behavior is in the universality class of directed percolation (DP) on the (D + 1)-dimensional lattice forming the discrete space-time of the SCA.^{1,2} The most general family of SCA models from which I start here can be defined on networks with almost any fixed spatial structure. These general models are referred to as "DP-SCA" since their histories (configurations in space-time) are identical to those of generalized DP models in which the site or bond densities are still homogeneous along the preferred direction (time), but not along the (spatial) directions perpendicular to it.

There are many possible choices of specific inhomogeneous spatial structures for DP-SCA. Here, I concentrate on random structures. It has been argued² and shown numerically in a previous paper¹ that fixed ("quenched") spatial randomness in DP-SCA (random bond or cell dilution, or differences in local transition probabilities) leads to a new universality class with critical exponents clearly distinct from those of DP. This is probably important for applications, since most practical realizations occur in media with some form of structural inhomogeneity.

Here, I derive results for the relaxation of spatially disordered DP-SCA to the vacuum. The asymptotic decay is shown to be a power law in a large part of the phase diagram. The value of the exponent depends on the strength and type of the quenched disorder incorporated into the local transition rules. This is markedly different from the relaxation of homogeneous DP-SCA, which is exponential throughout the vacuum phase.²⁻⁵ The nonexponential relaxation of the disordered models has a simple physical basis: it is due to the long-lived contributions from rare regions in which the local connectivity or transition rules are accidentally above the critical point for existence of an active phase in infinite systems. A similar mechanism causes "Griffiths' phases"^{6,7} in randomly disordered spin models, but the effects on DP-SCA are quantitatively different because the latter are nonequilibrium models.

The plan of the paper is as follows. In Sec. II, spatially disordered DP-SCA are defined and their basic properties summarized, setting the stage for the main problems and results of the paper. In Sec. III, the relaxation of onedimensional disordered DP-SCA is analyzed, yielding exact asymptotic forms for cell diluted models and useful lower and upper bounds for more general types of disorder. In Sec. IV, the analysis is extended to disordered DP-SCA in higher dimensions, which leads to slightly more complicated but essentially similar results. The conclusions are discussed in Sec. V.

II. DP-SCA MODELS, DEFINITION AND BASIC PROPERTIES

The general family of fixed-structure DP-SCA can be described as follows. First consider a *D*-dimensional lattice with links between pairs of vertices lying within a fixed finite distance. An SCA "cell" is identified with each vertex, and two "couplings" (one per direction) with each link. Each cell *i* carries a two-state variable $s_i \in \{0,1\}$, and *z* finite-range couplings $c_{ij} \ge 0$. The dynamics is defined by time-invariant transition probabilities ("rules")

$$P(s_i(t+1)=1)=F_i\left(\sum_j c_{ij}s_j(t)\right),$$

with $F_i(x=0)=0$ and $0 < F_i(x>0) < 1$. The restrictions on the $F_i(x)$ guarantee strictly stochastic rules, except for the single deterministic rule F(0)=0, giving a unique global absorbing state: the vacuum, $s_i = 0 \quad \forall i$. Fixed (quenched) disorder is represented by letting a set of random parameters control the local rules via the F_i or c_{ii} . For instance, random-coupling dilution can be introduced by taking $c_{ij} = cr_{ij}$, with fixed random parameters $r_{ii} \in \{0,1\}$. Independently of the couplings, the total input x of a cell can assume at most $Z = 2^{z} - 1$ distinct positive values, so the specification of a generic DP-SCA model requires Z values of $F_i(x > 0)$ per cell. Formally, one could do without the c_{ii} altogether, specifying the rules directly in terms of the Z nontrivial configurations that can occur in each set of z neighbors. However, the present notation is more perspicuous when additional structure in the couplings reduces the number of independent stochastic rules per cell. For example, strictly totalistic models $(c_{ii} = c_i)$ have z such rules per cell, and DP-SCA with spatial translation invariance $[c_{ii}=c]$ and $F_i(x) = F(x)$ have only z such rules for the whole model.

Below, the extensive number of parameters required in defining a disordered model will be reduced to only a few by specifying a distribution from which the local rule parameters are drawn at random, and interest will focus on behavior averaged over the corresponding realizations of the disorder.

If all c_{ij} and $F_i(x > 0)$ are close to 0, the vacuum is eventually reached from all initial conditions. Clearly, the rate of reaching the vacuum is a nonincreasing function of each parameter. In fact, the model can remain in an active phase when its parameters are sufficiently close to 1. This phase corresponds to an invariant probability measure (additional to the vacuum) on states with a finite density of 1's; this measure can be reached with a positive probability from all initial conditions except the vacuum. The part of the parameter hypercube in which an active phase exists is separated by a critical hypersurface from that in which only the (Dirac) measure on the vacuum state exists. There could be more phases⁸ at higher parameter values, but this paper deals with phenomena at or below the first transition. Presently, rigorous results about DP-SCA exist only for regular (1 + 1)-DP (Ref. 4) and the related "contact" model,⁵ but a considerable body of results from numerical transfer matrix, Monte Carlo (MC), and renormalization-group techniques, as well as field theory, has accumulated over recent years.^{2,3} Naturally, the issue of characterizing universal features of the phase transition in the various DP-SCA models arises. The relevant results are summarized here to provide the background for the problem of nonexponential decay to the vacuum in DP-SCA with quenched disorder.

For D < 4, there are now two known universality classes^{1,2} within the general family of DP-SCA as defined above. Membership in one or the other depends on the presence or absence of spatial randomness. The more familiar class (which is identical to that of DP in spacetime) contains the models with spatial translation invariance. This implies *i* independence and *i*-*j* symmetry of all couplings c_{ij} and rules F_i , as well as the topology of a periodic lattice. For example, choosing all $F_i(x) =$ $1 - \exp(-x)$ and $c_{ii} = c$ for nearest neighbors on a regular (D) lattice yields bond-DP on a (D+1) lattice with bond density $1 - \exp(-c)$, whereas one obtains site-DP with site density p when taking, e.g., $F_i(x > 0) = p$ and $c_{ii} = 1$. More generally, DP universality applies to the critical hypersurface lying strictly in the interior of the z-parameter hypercube spanned by the z transition probabilities that fully specify a DP-SCA model with translation invariance.

By contrast, generic DP-SCA having uncorrelated randomness in their local couplings or transition rules cannot be in the DP universality class for D < 4, as has been shown^{1,2} by a Harris-type argument,⁹ adapted to fit SCA models. Roughly speaking, one asks whether the spatial correlation length of the dynamical patterns of 1's near criticality diverges fast enough for the local differences in the critical point due to the fixed disorder to become averaged out. The answer is no (in D < 4) if one assumes that the standard DP exponents apply also to DP-SCA models with a finite amount of disorder. Thus the assumption must be inconsistent, raising the question of whether the phase transition becomes smeared out, or survives with other critical exponents, possibly forming a new universality class of disordered DP-SCA models.

In a previous paper,¹ this question was studied for D=1 and 2 models through extensive MC calculations. The second-order transition was shown to persist and new critical exponents were found. Universality was confirmed within numerical accuracy for models with various types and strengths of structural disorder. Note that the active phase ceases to exist at all if the disorder cuts the network of SCA cells into finite clusters, since each of these has a finite probability per time step of reaching the vacuum state.

III. RELAXATION OF D=1 MODELS

The structure of one-dimensional DP-SCA is simple enough to allow exact results to be obtained for the asymptotic relaxation behavior of models with random cell dilution. More general types of disorder lead to similar behavior, but the analysis usually yields only lower bounds to the decay function, supplemented by upper bounds in favorable cases.

A. Cell dilution: Exact asymptotic forms

The simplest disordered D=1 DP-SCA are exemplified by models with nearest-neighbor couplings $c_{ij}=1$ for |i-j|=1, and $F_i(x>0)=cr_i$, where the $r_i \in \{0,1\}$ are fixed random variables chosen from the distribution $f(r)=p\delta(r-1)+(1-p)\delta(r)$. This represents random cell dilution, splitting the model into random-length strings of nearest-neighbor connected SCA cells for any p < 1. The probability of occurrence of *n*-cell strings is $P_n = p^n (1-p)^2$. One recovers regular D=1 DP-SCA on the edge p=1 of the p-c phase diagram, with a (DP-class) phase transition occurring at $c = c^*$. The general-p models relax to the vacuum not only for $(p=1, c < c^*)$, but for all (p < 1, c < 1) as well, because a finite-length DP-SCA string has a finite chance per time step of reaching the vacuum. In fact, there are two time scales 5(a) when $1 > c > c^*$. In a few time steps, a string of length n >> 1settles into a metastable form of the active phase (carrying a finite density of 1's), while-on a macroscopic time scale-it decays to the vacuum through rare, exceptionally large fluctuations. The probability that an n string has not yet reached the vacuum at time t is^{5(a)} $M_n(t) \sim \exp(-t/T_n)$, with $T_n \sim \exp(an) = A^n$, where $a = \ln(A) \ge 0$ depends on c. Although a(c) is not generally available in closed form, one can verify that it is increasing on the interval $c^* < c < 1$, diverges as $a(c) \sim -\ln(1-c)$ for $c \rightarrow 1$, and vanishes as $a(c) \sim \xi_s^{-D} \sim (c-c^*)^{Dv_s}$ for $c \rightarrow c^*$ (the latter is from finite-size scaling). Note that T_n only depends exponentially on *n* for $c > c^*$, where an infinitely long string could support an active phase. For $c < c^*$, T_n tends to a finite value $\sim \xi_t$ for $n \gg \xi_s$, whereas $T_n \sim n^{v_t/v_s}$ for $c = c^*$. I will now show that the exponential growth of T_n with *n* for $c > c^*$ gives rise to the slow phase, a region in parameter space where the model relaxes via a power law.

The quantities of interest are the decay function $M(t) = \sum_n nP_nM_n(t)$, defined as the fraction of cells with $s_i(t) = 1$ when started from $s_i(0) = 1 \forall i$, and the effective decay time $T = \sum_t M(t)$. Thus one has

$$T = \sum_{t} \sum_{n} n P_n M_n(t) = \sum_{n} \left[n P_n \sum_{t} M_n(t) \right] \sim \sum_{n} n P_n T_n ,$$
(1)

where the *n* sums exclude the infinite connected network that may be present in some of the diluted D > 1 models to be treated later.

For the cell diluted D = 1 model, the sums can be done easily,

$$T \sim (1-p)^2 \sum_{n} n (Ap)^n = Ap (1-p)^2 \frac{d}{d (Ap)} \left[\sum_{n} (Ap)^n \right],$$
(2)

$$T \sim Ap(1-p)^2(1-Ap)^{-2}, p < 1/A$$
.

One can view the divergence of T for $p \rightarrow 1/A$ as a genuine dynamical phase transition,¹⁰ with critical exponent $\theta = -2$, and occurring on a critical line p = 1/A, which connects the points (p=0,c=1) and $(p=1,c=c^*)$ in the *p*-*c* phase diagram and goes as $p \sim (1-c)$ for $p \rightarrow 0$ and as $1-p \sim (c-c^*)^{Dv_s}$ for $p \rightarrow 1$.

The asymptotic behavior of M(t) can be obtained as

$$M(t) = \sum_{n} n P_n M_n(t) \sim \sum_{n} n p^n \exp[-t \exp(-an)] .$$
 (3)

Tentatively replacing the sum by the integral

$$M(t) \sim \int_0^\infty dn \ n \exp[-bn - t \exp(-an)]$$

where $b = -\ln(p)$, (4)

enables the large-t behavior to be found via Laplace's method. The square bracket in Eq. (4) has its quadratic maximum at $n = (1/a)\ln(at/b)$, with second derivative -ab. Thus, to justify the transition from (3) to (4), one needs to have $t \gg (Ab)/a$ and $ab \ll 1$. Only the latter condition can be restrictive. Supposing for the moment that $ab \ll 1$, one obtains

$$M(t) \sim (at/b)^{-b/a} \ln(at/b), \quad t \to \infty \quad . \tag{5}$$

If ab > 1, then the discreteness of n in Eq. (3) becomes noticeable, resulting for $ab \to \infty$ in a log-periodic stepwise decay, but with envelope as in Eq. (5). In any case, M(t)can be said to show power-law decay with exponent -b/a. The exponent is finite and negative inside the parameter region (0 , which proves theexistence of the slow phase for this simplest model.Moreover, <math>b/a vanishes continuously if $c \to 1$ or $p \to 1$, and diverges if $c \to c^*$ or $p \to 0$, controlled by the respective behaviors of a(c) and b(p). The logarithmic nextto-leading factor in M(t) may appear hard to verify in an experiment or numerically, but it is directly related to the exponent θ describing the divergence of T for $a \to b$. This can be shown by integrating functions similar to the tail (5) of M(t),

$$\int_{1}^{\infty} dt \ t^{-b/a} (\ln t)^{q} = \int_{0}^{\infty} dx \ x^{q} \exp\{[1 - (b/a)]x\}$$
$$= \Gamma(q+1)[(b/a) - 1]^{-1-q}, \qquad (6)$$

using the substitution $x=\ln t$. Clearly, the logarithmic factor must occur with q=1 to recover the exact exponent $\theta = -2$ of the decay-time divergence.

Different asymptotics for M(t) and T apply to the edges of the *p*-*c* diagram, but the results are trivial or well known, except on the line $(c = c^*, 0 . Then <math>T_n \sim n^{v_t/v_s}$ for large *n*, leading to stretched exponential decay of $M(t) \sim \int_0^\infty dn \ n \exp[-bn - t/T_n]$. One obtains

$$\ln[M(t)] \sim -t^{v_s/(v_t+v_s)}, \qquad (7)$$

the asymptotic behavior of the integral being determined (Laplace) by the maximum of the square bracket occurring at

$$n = [(v,t)/(v,b)]^{v_s/(v_t+v_s)}$$

Algebraic next-to-leading factors in M(t) have been dropped from Eq. (7).

B. General disorder: Power-law bounds

Less complete but useful results are obtained for models with more general disorder. Asymptotic power-law lower bounds for M(t) can be found in all cases, but these bounds are not usually tight throughout the phase diagram. In some cases, power-law upper bounds can also be found. For example, take random dilution of couplings: $F_i(x > 0) = c \ \forall i, c_{ij} = r_{ij}$ only if |i-j| = 1, with the r_{ij} again drawn randomly from the distribution $f(r)=p\delta(r-1)+(1-p)\delta(r)$. To get a lower bound M'(t), one counts only the contributions to M(t) coming from strings in which each cell carries both couplings. The decay of these internally homogeneous strings has already been analyzed in Sec. III A. The only difference is that *n*-cell strings now occur with a probability $P_n \sim p^{2n}$, because two r_{ij} per cell equal 1. Thus M'(t) also decays as in Eq. (5), but with exponent -b'/a, where $b'=-2\ln(p)$ and *a* is as before. An upper bound M''(t) is found by considering the strings ending in cells without any couplings, and overestimating their decay time by taking it equal to that of a homogeneous string of the same length. Thus M''(t) again decays algebraically, with exponent -b''/a, where now

$$b'' = -\ln[1-(1-p)^2]$$

and a is as before. Note that $0 < b'' \le b'$. The true asymptotic decay M(t) (modulo any staircase ripple) then must be a power law with exponent -b/a, where $b'' \le b \le b'$.

For most models, useful upper bounds are very hard to obtain analytically, contrary to lower bounds. This can be illustrated already in a simple model having weak and strong cells instead of dilution. Take $c_{ij} = 1$ for |i-j| = 1 and $F_i(x > 0) = cr_i$, where the r_i have distribution $f(r) = p\delta(r-1) + (1-p)\delta(r-k)$ with 0 < k < 1. Observe that the decays $M^{(k)}(t)$ of any pair of such models which are identical except for their k values are ordered in the sense that k < K implies $M^{(k)}(t) < M^{(K)}(t)$, as long as $M^{(K)}(t) > 0$. This follows from the fact that the space-time sites with $s_i(t) = 1$ of the k model are a proper subset of those of the K model. Similarly, a p ordering exists for models with the same k, but different p. These orderings are helpful in the construction of bounds and in restricting the parameter space where long-tailed relaxation can occur. Specifically, no such slow phase can exist for $c < c^*$ in any k < 1 model, since the decay of the k=1model provides an exponentially decaying upper bound. On the other hand, for $c > c^*$, the decay of the k = 0(cell-dilution) model gives a power-law lower bound [Eq. (5)] for the M(t) of all k > 0 models. This suffices to prove the existence of a slow phase for $c > c^*$ in the k > 0models, provided that they relax to the vacuum at all. However, this condition is false for some of the models, e.g., the ones with $c > c^*/k$ at any p. These support an active phase, since their decay is bounded below by that of the same model with p = 0, which is identical to a p = 1(standard DP) model with $c > c^*$. This narrows down the parameter region for the slow phase to $c^* < c < c^*/k$ with 0 . Are there any models in this region thatdecay to the vacuum? Heuristic arguments based on the k and p ordering of $M(\infty)$ in the active phase suggest that the active phase disappears for c below a critical line C(p) in the *c*-*p* diagram, where C(p) is smooth and strictly decreasing between the exactly known end points $C(0) = c^*/k$ and $C(1) = c^*$, but no explicit calculation of C(p) is available. Attempts at constructing a lower bound for C(p) have worked only for $k, p \ll 1$ so far, and are hardly worth pursuing here. However, numerical (MC) evidence¹ strongly indicates the existence of a region within the rectangle (0 where the models decay to the vacuum. This region then is the slow phase.

DP-SCA with coupling or cell parameters drawn from a continuous random distribution can also be shown to have decay bounded below by a power law. For example, take $F_i(x) = 1 - \exp(-x) \forall i$ and $c_{ij} = cr_{ij}$ for |i-j| = 1, with the r_{ij} chosen from a smooth distribution f(r), with f(r) > 0 for $0 < r_{\min} < r < r_{\max}$, scaled so that $\langle r \rangle = 1$, and having standard deviation d. Standard bond-DP is recovered in the special case d = 0. For d > 0 one still expects to find a transition between an active and a vacuum phase, occurring at a value of c depending smoothly on d. MC results reported previously¹ support this, and yield new critical exponents characteristic of D=1 disordered DP-SCA. Lower bounds M'(t) for these models are obtained by considering the decay of supercritical strings of cells, i.e., strings in which all couplings have $r_{ij} > R$, for an $R > c^*/c$. The set of such "S strings" is nonempty if $c > c^* / r_{max}$. The probability of occurrence of S strings of length *n* is $P_n \sim \exp(-bn)$, where now $b = -\ln \int_{R}^{\infty} dr f(r)$. The decay $M_n(t)$ of an *n*-cell S string is bounded below by the decay $M'_n(t)$ of a homogeneous *n*-cell string in which each $r_{ij} = R$. The latter strings have $M'_n(t) \sim \exp(-t/T_n)$, where $T_n \sim \exp(an)$, with a > 0. As long as the set of S strings is nonempty, b is finite and a power-law lower bound M'(t), of the form of Eq. (5), is derived for the decay of the S strings, which in turn is a lower bound for M(t) of the whole model. If an explicit form of f(r) and a(R) is known, then R can be chosen variationally, optimizing the lower bound by minimizing the value of b/a. In any case, b and a are increasing functions of R, with b=0 at $R=r_{\min}$ and $b=\infty$ at $R = r_{\text{max}}$, while a = 0 at $R = c^*/c$ and $a \to \infty$ as $R \to \infty$. In general, the bound is not expected to be tight and it will become irrelevant if an active phase occurs for cabove a critical line C(d) in a region of the *c*-*d* diagram where S strings exist. On the other hand, the bound will become tight near the lower edge of the slow phase at $c \rightarrow c^* / r_{\text{max}}$. The slow phase occupies the whole phase diagram if f(r) has an unbounded upper tail. It may, however, be difficult to see this in practice since the power-law tail of M(t) has a large exponent for $c \ll c^*$.

IV. RELAXATION OF D > 1 MODELS

Exact analysis of disordered DP-SCA in D > 1 is prohibitively difficult. For D > 4, one expects only trivial effects of quenched disorder on the critical exponents since the model is above its upper critical dimension. D=4 is an undecided marginal case. However, nonexponential relaxation effects are expected in any D, and appropriate bounds will be derived despite the problems preventing a full analysis. As in Sec. III, models with dilution-type spatial disorder are analyzed in some detail first. Lower and upper bounds with asymptotic powerlaw decay are found. Finally, the occurrence of similar effects in DP-SCA models with more general types of disorder is indicated.

Cell diluted DP-SCA models in D > 1 can be defined similarly to those in D=1, e.g., by setting $F_i(x)=0$ with probability 1-p, leaving a fraction p of cells with $F_i(x > 0) = c$, and taking $c_{ii} = 1$ for i - j within a finite range. In terms of a DP problem on a (D+1) space-time lattice, one removes timelike lines of sites. An important feature in D > 1 is the existence of a critical $p = p^*$ below which the fixed random network of remaining SCA cells no longer forms a statically percolating structure.¹¹ For $p > p^*$, the structure contains at least one path to infinity, guaranteeing that the model has an active phase at least for $c > c_1^*$, the critical point of the p=1 model in D=1. One expects the active phase to disappear for c below a critical line $C_D(p)$ that decreases smoothly to the DPclass critical point c_D^* at p=1. This has been confirmed by MC results,¹ and new critical exponents were found for the transition along the line for $p^* . With$ respect to the relaxation, one expects a slow phase only for $c > c_p^*$. For $p > p^*$ its upper boundary must be the critical line $C_D(p)$, whereas it would exist up to c=1 for $p < p^*$. It is easier to analyze the slow relaxation behavior for $p < p^*$ where no active phase can occur, but the lower bounds that will be derived next are also applicable to $p > p^*$.

To find nonexponential asymptotic lower bounds for M(t), consider the relaxation of exceptionally dense, roughly spherical *n*-cell clusters on which the DP-SCA model decays to the vacuum on a time scale growing as $T_n \sim \exp(an)$ for $c > c_D^*$. Such exponential growth occurs because a (locally) supercritical cluster decays only through coherent fluctuations involving all its cells, instead of via a diffusing domain wall as in an Ising model (cf. Sec. V). Such compact clusters occur with probability $\ln(P_n) = -bn - b'm + O[\ln(n)]$, where $b \sim -\ln p$, $b' \sim -\ln(1-p)$, and $m = n^{(D-1)/D}$. The three factors in P_n stem from contributions of the (dense) bulk, the (empty) outer surface and the multiplicity of cluster shapes with the same T_n . The long-tailed relaxation of these dense clusters gives a lower bound M'(t) for M(t),

$$M'(t) = \sum_{n} nP_{n} \exp(-t/T_{n})$$

$$\sim \int_{0}^{\infty} dn \exp[-bn - t \exp(-an) - b'n^{(D-1)/D}],$$
(8)

where algebraic factors in the integrand can now be neglected. Again using Laplace's method, one finds the asymptotic forms determined by the maximum of the square bracket, occurring at $n = (1/a)\ln(at/b) + O[(\ln t)^{-1/D}]$. This yields

$$\ln[M(t)] > \ln[M'(t)] \sim -(b/a)\ln(at/b) + [(1/a)\ln(at/b)]^{(D-1)/D}.$$
(9)

The dominant contribution to M'(t) is the same power law as in D=1, but the next important factor now is not logarithmic, but an intermediate form that may perhaps be called "stretched power law."

Related to this is the equally unusual behavior of the corresponding lower bound T' of the decay-time T

$$T > T' \sim \sum_{n} n P_{n} T_{n} \sim \int_{0}^{\infty} dn \exp\{(a-b)n - b' n^{(D-1)/D} + O[\ln(n)]\}, \quad (10)$$

from which one sees that T' now goes to a finite value (depending on b') as $a \rightarrow b$ from below, and then jumps discontinuously to ∞ for a > b. Note the possibility that T already diverges for smaller values of p and c than those giving a = b, due to contributions from other less dense clusters than have been taken into account here. One could even wonder whether the leading factor of M(t) might be slower than a power law. To disprove this, one can construct a power-law upper bound M''(t)with exponent -b''/a where $0 < b'' \le b$ for all $c > c^*$ and $p < p^*$. The idea is to count all the clusters, instead of just the compact ones, and to use the decay time of the slowest (compact) n cluster as upper bound for the decay time of the unrestricted n clusters. For any $p < p^*$, the number density of (unrestricted) n-cell clusters decreases exponentially for large n^{11} . Thus $\ln(P_n) \sim -b''n$ with 0 < b'' < b. The decay time of any *n*-cell cluster is bounded above by the usual $T_n \sim \exp(an)$ of a compact cluster of the same mass. Hence M''(t) has the same form as Eq. (4), leading to the power-law upper bound announced above. The asymptotic decay of M(t) (modulo any staircase terms) then also is a power law, but a finite range of exponents is left open. Note that the M''(t) upper bound does not exist for $p > p^*$, where it is swamped by contributions from the infinite cluster. The lower bound M'(t)becomes irrelevant for $c > C_D(p)$.

Models with coupling dilution can be attacked by a combination of the methods just used for cell dilution with those for coupling diluted D=1 models. It may therefore suffice to explain the strategy without repeating the details of the calculations.

A power-law lower bound can be derived by considering compact clusters in which each cell carries all its zcouplings. A similar upper bound is found by counting all clusters in which a cell is considered connected to another if their common link carries at least one of its (directional) couplings. The decay time for a cluster is again overestimated by taking it equal to that of the cluster having the same cells, but each carrying all z couplings. Note that this upper bound exists only for pbelow the critical point of static diode percolation.¹²

The D > 1 models with disorder variables drawn from continuous distributions can be treated by analogy with those in D=1. Again, one obtains weaker results than with dilution, but still finds power-law lower bounds for $c > c^*/r_{max}$.

V. DISCUSSION

It has been shown that power-law relaxation is a general feature of the decay to the vacuum state in DP-SCA models with quenched disorder. The basic physical mechanism underlying this power-law decay is simply the existence of large clusters of cells in which the interactions are above the critical point at which an infinite system can support an active phase in addition to the vacuum. The number of such clusters falls off exponentially with their size, but their characteristic decay time grows exponentially.

This mechanism is similar to the one responsible for the Griffiths' phase in disordered spin models. However, the analogy in the phenomena caused by it is not very strict, due to the lack of symmetry between the vacuum and the active phase in SCA. There is no clean distinction between the coupling of disorder variables to the local values of the transition point or the order parameter (the density of 1's). Therefore in DP-SCA one cannot clearly distinguish the equivalents of either random-field or random-coupling models as one can in Ising systems.

The actual shape of the relaxation tails is also different: disordered Ising models such as diluted ferromagnets or spin glasses in their Griffith's phase relax as $\ln[M(t)] > (\ln t)^{D/(D-1)}$, faster than a power law, but slower than a stretched exponential.^{6,7} A recent derivation for *m*-vector spins⁶ in the $m \to \infty$ limit yields Kohlrausch-type stretched exponential decay. Comparing SCA to Ising systems, the difference can be traced to the fact that a "droplet" in a spin system overturns by the movement of a domain wall across it. The ratelimiting step is the creation of the wall, requiring a free energy that scales as the (D-1)th power of the droplet diameter. In SCA, one needs a coordinated fluctuation involving essentially all the cells in the locally supercritical cluster to bring it to the vacuum state. Even moving a wall across it has to go against the positive average growth speed of the local active phase, which requires a coordinated sequence of local fluctuations. One can also look at the configurations in D+1 space-time (the histories of the model). Any SCA defines a Hamiltonian on its set of histories,¹³ which can thus be viewed as equilibrium configurations of a special Ising (or Potts) model, usually with multispin interactions, on a D + 1 lattice. In our case, a cluster of DP-SCA cells becomes a corresponding bar-shaped (D+1) volume, and decay to the vacuum is equivalent to creation of a D-dimensional domain wall across the bar. Its free-energy cost, which translates back for SCA as the logarithm of the decay rate to the vacuum, scales with the number of sites in the wall. This makes the decay-time scale grow exponentially with the D volume of the SCA cluster. In the vicinity of the critical point, the D volume of the cluster gets rescaled by the spatial correlation volume ξ_s^D .

Experimental observation of slow relaxation of spin models in the Griffiths' phase is notoriously difficult,⁷ but this may be much easier for some of the DP-SCA studied here. Numerical work on strongly diluted DP-SCA in D=2 showed very strong tails,¹ with $T > 10^5$, exceeding the limitations of available computer time. Also, in view of the possible application of DP-SCA as models of replicating organisms or autocatalytic chemical reactions, one may hope that the phenomena analyzed here could be of relevance to realistic systems in which such entities survive for long periods in inhomogeneous media.

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