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New Universality for Spatially Disordered Cellular Automata and Directed Percolation

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Stochastic cellular automata (SCA) with fixed, but randomly chosen, probabilities (spatial disorder) are studied in D dimensions. For zero disorder, the present SCA reduce to directed percolation in $(D+1)$ space-time, but finite spatial disorder is incompatible with the critical exponents of directed percolation. Monte Carlo calculations of the SCA on several disordered structures in $D=1$ and $D=2$ yield new universal exponents. I also discuss the phase diagram of "diluted" SCA, which contains a multicritical point and the SCA analog of a "Griffiths phase" with nonexponential relaxation.

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Directed percolation (DP) has been studied widely over the last few years, both because of its intrinsic interest as a purely geometrical statistical physics model with its own universal critical behavior, and because of its range of possible applications in physics, chemistry, and biology.[1-4] It was realized from the beginning that DP clusters on $(D+1)$-dimensional lattices are equivalent to "histories" (patterns in space-time) of interacting Markov processes on lattices in a D-space. Very recently, the implications of this equivalence for the behavior of stochastic cellular automata (SCA) with totalistic[5] rules (i.e., depending only on the sum of inputs) and one absorbing state ("vacuum") were studied.[6] In summary, all such SCA (homogeneous in space and time) turn out to be in the universality class of DP. In addition, the histories of any SCA can be mapped onto the equilibrium configurations of a generalized Ising model with intriguing special properties of its partition function.[7] This may bring techniques and results of equilibrium statistical physics to bear on nonequilibrium problems[8] which are in general much less understood, but which have many important applications. In turn, some applications of SCA can lead one to study interesting extensions of existing models.

In this Letter I study the effect of fixed spatial inhomogeneity on SCA having rules that are locally equivalent to DP in space-time[3,6] (DP-SCA). Such spatial disorder occurs in the reality of many SCA applications. This simple extension leads to new universality classes in $D<4$ and to a rich set of new SCA physics including multicriticality and a "Griffiths phase."

To see this, consider SCA models inbedded in D-dimensional space. Each of the SCA cells i has two local states $s_i \in \{0,1\}$, and z finite-range couplings $c_{ij} > 0$. Going beyond strictly totalistic rules, a DP-SCA model is specified by its time-invariant, local, state transition probabilities

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

with $F_i(x \leq 0)=0$ and $0 < F_i(x>0) < 1$. Note the restrictions on $F(x)$ which represent the single deterministic rule causing the "vacuum" ($s_i=0\ \forall\ i$) to be the unique absorbing state of the process. In general, there are 2^z-1 positive probabilities per cell, specified by the z couplings c_{ij} and the functions $F_i(x)$, which is smooth on $x>0$ in most applications, although this is not required. One can recover models in the DP universality class[1] by imposing the additional restrictions of i independence and i-j symmetry. For example, setting $F_i(x)=1-\exp(-x)$ and $c_{ij}=c$ for nearest neighbors on a regular lattice yields bond DP on a $(D+1)$ lattice with bond density $1-\exp(-c)$. Likewise, with $F_i(x>0)=p$ and $c_{ij}=1$ one gets site DP with site-density p. General i,j-independent (zero disorder) DP-SCA models are defined by z positive probabilities and DP universality is found to apply in the vicinity of all parts of the critical hypersurface that lie strictly in the interior of the z-parameter hypercube.[6]

Spatial disorder is introduced by letting the local DP-SCA probabilities depend randomly on i and j. This breaks the translation invariance and the local i-j symmetry in the D space without introducing global spatial anisotropy or affecting the time invariance of the rules or the special role of the vacuum. On the $(D+1)$-space-time lattice, a generalized DP problem emerges with randomness which is fully correlated along the preferred axis (time), but uncorrelated along the other D dimensions.

The first question that suggests itself is whether even small spatial disorder is compatible with the universality class of standard DP. An argument in the style of Harris[9] shows that this is not the case: Assume that there were a transition with the normal exponents and let the disorder, parametrized by r, couple smoothly to the critical value c^* of some global SCA rule parameter c. Because of the time-invariant rules, the fluctuations $\sigma(r) \sim \sigma(c-c^*)$ that affect the large space-time clusters depend only on their spatial corre-

lation length $\xi_s \sim (c-c^*)^{-\nu_s}$. Thus,

$$\sigma(c-c^*) \sim \sigma(r) \sim \xi_s^{-D/2} \sim (c-c^*)^{D\nu_s/2}.$$

Self-consistency requires that the fluctuations go to zero faster than $c-c^*$ near criticality, and so one should have $D\nu_s > 2$. However, ν_s is approximately 1.1, 0.8, 0.6, and 0.5 for $D=1$, $=2$, $=3$, and ≥ 4, respectively, and so one finds a contradiction ($D \geq 4$ is marginal). The inconsistency with the zero-disorder DP exponents raises the question of whether new universality classes exist; there could also be just a smeared-out transition. I have studied this question for $D=1$ and $D=2$ using Monte Carlo (MC) calculations.

In $D=1$, cyclic arrays of up to $N=2048$ cells i were updated according to the above SCA rules with $c_{ij} > 0$ for $j \in \{i-1, i+1\}$. In the model, where $c_{ij} = c \; \forall \; i$ defines the global parameter c representing the average SCA coupling strength, the (cell-based) disorder was introduced by letting $F_i(x) = 1 - \exp(-xr_i)$. The r_i, with $0 < r_{\min} < r_i < 1$, are N independent random variables assigned to the N cells i. In $D=1$, the r_i must be positive to prevent the space-time lattice from being cut into strips of finite width on which no transition is possible. I have used binary as well as uniform r distributions with $r_{\min} = \frac{3}{4}$, $\frac{1}{2}$, and $\frac{1}{4}$. In addition, calculations were done on the similar case with $c_{ij} > 0$ also if $i=j$, on the model with $F_i(x>0) = cr_i$ and $c_{ij} = 1$, as well as on models with coupling-disorder specified by zN random values $c_{ij} = cr_{ij}$ and $F_i(x) = 1 - \exp(-x)$. All these disordered models are found to have the same critical behavior, ruled by exponents that are mostly very different from the ones found for zero disorder. The exponents computed directly from the MC data are as follows.

(a) The density exponent β, found from the relation $\langle n(t) \rangle_r \sim (c-c^*)^{\beta}$ for $c \geq c^*$ and $t \gg \xi_t$, where $n(t)$ is the relative number of cells with $s_i(t) = 1$ after starting from $s_i(0) = 1 \; \forall \; i$. The $\langle \cdots \rangle_r$ average is over 512 independent realizations of the disorder. Near criticality, data were taken after up to 10^5 timesteps to avoid transient artifacts.

(b) The spreading dimension[10] $\hat{d}$, from the relation $\langle \int_0^T m(t)\,dt \rangle_r \sim T^{\hat{d}}$ for $T \gg 1$, at $c=c^*$, and with only $s_0(0) = 1$. The quantity $m(t)$ counts the cells with $s_i(t) = 1$, averaged over only the "large" space-time clusters, e.g., those that did not yet relax to the vacuum at $t=T$. The spreading dimension is connected to other exponents via (hyper)scaling relations

$$\hat{d} = 1 + (D\nu_s - \beta)/\nu_t = (\beta + \gamma)/\nu_t.$$

(c) The (inverse) dynamic exponent ν_s/ν_t, from $\langle m(t) \rangle_r / \langle n(t) \rangle_r \sim t^{D\nu_s/\nu_t}$ for $t \gg 1$ at $c=c^*$. This relation follows from the behavior of $\langle n(t) \rangle_r \sim t^{-\beta/\nu_t}$ at $c=c^*$. There are smaller corrections to scaling on $m(t)/n(t)$ than on $m(t)$ or $n(t)$ separately.

TABLE I. Critical exponents of DP/SCA in $D=1$.

	Disorder (this work)	No disorder (this work)	(1+1) DP (Refs. 1 and 10)
$\hat{d}$	1.28 ± 0.03	1.46 ± 0.02	1.46 ± 0.03
β	1.75 ± 0.1	0.28 ± 0.015	0.273 ± 0.003
ν_s/ν_t	0.72 ± 0.03	0.63 ± 0.02	0.632 ± 0.001
ν_s	2.85 ± 0.25	1.05 ± 0.1	1.100 ± 0.005
ν_t	4.0 ± 0.5	1.65 ± 0.2	1.734 ± 0.002

The values of these exponents and of a few others computed via the scaling relations[1] appear in Table I. The exponents for zero disorder, extracted from MC data in exactly the same way, are also given so as to allow the errors in the method to be gauged. The estimated error ranges given are about equally due to the finite statistics and to systematic effects of corrections to scaling and the inaccurately known values of the critical point. Selected values from the literature on $(D+1)$ DP are also listed. Note that with the much larger value of ν_s, the disordered model no longer violates Harris's criterion. The dynamic exponent ν_s/ν_t changes much less, but the difference still seems significant.

In $D > 1$, spatial disorder can be introduced in the same way as was done in $D=1$, but here it is possible to let $r_{\min} = 0$. For instance, let r be a binary random variable with distribution

$$f(r) = p\delta(r-1) + (1-p)\delta(r-0).$$

Again one can let each value of r control the local SCA rules through each c_{ij}, or through each $F_i(x)$. Thus, one defines models of "diluted" SCA in which the density of the fixed set of remaining cells or couplings is p. In terms of a DP problem in $(D+1)$ space-time, one eliminates whole timelike rows of sites and/or bonds. There is a critical $p=p^*$ below which the network of SCA cells and bonds does not form a (static) percolating structure. No SCA phase transition is possible on such a fragmented network. For $p \geq p^*$, though, one expects to get a DP-SCA phase transition since the spatial structure contains at least one path to infinity and we have just seen that there is a DP-SCA phase transition even on disordered $D=1$ structures. In fact, the critical coupling $c_D^*(p)$ of DP-SCA on a diluted, but spatially percolating, structure in $D > 1$ should lie between $c_D^*(1)$ and $c_1^*(1)$, i.e., the values for the corresponding undiluted systems in D and 1 dimensions. One expects a phase diagram as in Fig. 1.

Reduction to well-known models occurs along two edges of the figure. For $p=1$ (no dilution) one has $(D+1)$ DP, and for $c=1$ (strong-coupling limit) one gets deterministic propagation on random structures in D-space.[11] There are several regions with interesting

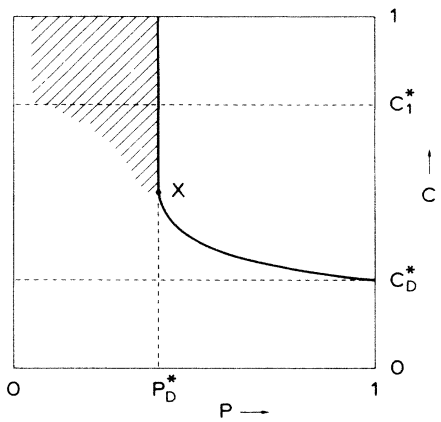

FIG. 1. Phase diagram of DP-SCA on diluted structures in $D > 1$, having cell density p and coupling strength c ($c = 1$ is the strong-coupling limit). The vacuum phase is below and to the left of the critical line (solid line). The multicritical point is at X. In the striped region (contained in the Griffiths phase), the relaxation has particularly strong long-time tails.

new behavior in the rest of the diagram. Starting from $p = 1$, one expects to see a narrow region where the critical phenomena show crossover from DP universality to the new disordered DP-SCA class, which is then expected to govern the behavior near the critical line for $p > p^*$, ending in the multicritical point X.

I have studied this for $D = 2$ by MC, computing the critical exponents similarly to the $D = 1$ case. For $\hat{d}$ and ν_s/ν_t, averaging was done over 1024 randomly diluted structures imbedded in either square or triangular lattices of up to 128^2 cells, using toroidal boundaries. The propagation of the $s_i = 1$ states was followed for up to 4096 timesteps. To compute β, 128 structures and decays of up to 3×10^4 timesteps were used. The random structures that were used had cell densities $p = 1.0$, 0.95, 0.9, 0.8, and 0.7, well above the static percolation threshold p^*.

Well-defined phase transitions were found in all cases, and the power-law behavior of the singular variables as functions of c or t was analyzed as in the $D = 1$ case. Again, the critical exponents of the disordered models were found to be quite different from the ones without disorder. Crossover phenomena were seen in the model with $p = 0.95$. In addition, some models with $r_{\min} = \frac{1}{2}$ ("weak" instead of absent cells) were studied. These cases again showed the same exponents, confirming the existence of the universality class characterized by the exponents in the left-hand column of Table II.

Following the critical line for $p \to p^*$, one reaches a multicritical point X. From there on, the line coincides with $p = p^*$ and the critical behavior is controlled by that of static percolation, but several nontrivial effects can occur. For $c > c_1^*$ the situation is clear. The SCA process propagates with positive velocity to any cell in the spatially infinite cluster and eventually dies out on any finite cluster (but see below). However, one expects the multicritical point to have strictly $c(X) < c_1^*$. This is in contrast to diluted-spin models where $T_c \to 0$ (its $D = 1$ value) as $p \to p^*$. The critical coupling of DP-SCA will not approach its $D = 1$ value as $p \to p^*$, despite the finite ramification of the backbone and the diverging total length $L \sim (p - p^*)^{-1}$ of its "red"[12] (cut-set) links. The explanation lies in the "random necklace"[13] structure of the incipient infinite cluster in which "red" links and self-similarly structured blobs of larger sizes occur in fully random sequence. This lowers the value of $c(X)$, even if one takes into account only the bypasses formed by three-cell blobs.

TABLE II. Critical exponents of DP-SCA in $D = 2$.

	$p^* < p < 1$ (this work)	$p = 1$ (this work)	(2+1) DP (Refs. 1 and 4)
$\hat{d}$	1.61 ±0.05	1.81 ±0.04	· · ·
β	1.10 ±0.05	0.63 ±0.03	0.57; 0.62 ±0.06
ν_s/ν_t	0.58 ±0.03	0.64 ±0.03	· · ·
ν_s	1.17 ±0.1	0.84 ±0.05	0.75; 0.85
ν_t	2.0 ±0.15	1.3 ±0.1	1.27; 1.13 ±0.12

Crossing the critical line for $c_1^* > c > c(X)$ may be controlled by backbone exponents if the SCA process cannot penetrate into the dangling ends anymore, but this depends on unknown details of the structure of the dangling ends. Numerical results of DP-SCA on large percolation clusters indeed show that $c(X)$ is much below c_1^*, by as much as a factor of 2, although the accuracy is so far too low to determine the more subtle multicritical properties such as the crossover scaling or the shape of the phase boundary for small positive $p - p^*$. It is difficult to study these numerically because of slow relaxation and finite-size problems on the backbone, which is a sparse structure ($D \cong 1.6$). Further work on this problem is clearly needed.

Finally, it remains to discuss briefly the decay to the vacuum state of subcritical disordered DP-SCA. For $c < c_D^*$, exponential decay occurs on a time scale depending on c and p, but the situation is more complex for larger c. Take the region $p < p^*$ for ease of discussion. For $c > c_D^*$, the average DP-SCA lifetime on a compact (dense) cluster of N cells (N large) grows as $\sim \exp(aN)$, with a depending on c. On the other hand, such clusters occur with probability $\sim \exp(-bN)$, with b depending on p. This causes the relaxation at long times to be determined by the very rare but very slow compact clusters. The number of cells with $s_i(t) = 1$ [starting from $s_i(0) = 1 \; \forall \; i$] is

then bounded below (modulo logarithmic factors) by[14]

$$M(t) \sim \int_0^\infty dN\, N \exp(-bN) \times \exp[-t\exp(-aN)] \sim t^{-b/a}$$

for $t \to \infty$. The amplitude of the long tail is generally quite small for $c < c(X)$. However, as c gets larger, less compact clusters also get exponentially N-dependent lifetimes. For $c > c_1^*$, even the large, lattice-animal-type clusters at $p \ll p^*$ contribute to the long-time tail. The occurrence of such effects can be considered as the SCA analog of the "Griffiths phase"[15] in diluted-spin systems. In spin systems, the experimental signatures of such a phase can be hard to find, but it should be easier to see them in DP-SCA with $c > c_1^*$. There is preliminary support for this picture in MC results, even with p down to 0.02. Decays with strong long tails ($t > 10^5$) are seen in the parameter region that is striped in Fig. 1. The lower edge of this region occurs around values of $c(p)$ that increase smoothly from $c(X)$ to c_1^* for $p < p^*$, instead of showing a jump from $c(X)$ to 1. The amplitude of the long tail decreases quickly for c below the striped region and for $p < 0.01$.

In conclusion, the numerical results reported here characterize the new universality for spatially disordered DP-SCA and show that many of the critical exponents in $D = 1$ and 2 differ strongly from the corresponding zero-disorder ones. It would be interesting to look for this disorder effect in field theories of the problem, e.g., via expansions in $\epsilon = 4 - D$. For the diluted model in $D > 1$, a new multicritical point and Griffiths phase have been identified, both of which invite further study. Also, one would like to have the exponents for $D = 3$. As regards applications, the present model and its extensions should describe, e.g., the spread of autocatalytic chemical reactions,[16] replicating organisms, or infections without permanent immunity in random media, as well as the development of cooperative firing in young, mostly excitatory, neural networks.[3] Finally, the model allows interesting generalizations. Even with the present local DP-SCA rules, temporal instead of spatial disorder can be introduced; new exponents are again expected.[6] Furthermore, one can let some c_{ij} go negative and study frustrated SCA. The latter should be good models of disordered networks of excitatory and inhibitory neurons in the brain. For small densities of the inhibitory cells, one expects to be still in the disordered DP-SCA universality class described here,[17] but new, more complex behavior can occur with larger inhibitory densities. Numerical studies of such models are underway.

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