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Violation of scaling in the contact process with quenched disorder

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We study the two-dimensional contact process (CP) with quenched disorder (DCP), and determine the static critical exponents β and ν_{\perp} . The dynamic behavior is incompatible with scaling, as applied to models (such as the pure CP) that have a continuous phase transition to an absorbing state. We find that the survival probability (starting with all sites occupied), for a finite-size system at the critical point, decays according to a power law, as does the *off-critical* density autocorrelation function. Thus the critical exponent ν_{\parallel} , which governs the relaxation time, is undefined, since the characteristic relaxation time is itself undefined. The logarithmic time dependence found in recent simulations of the critical DCP [A. G. Moreira and R. Dickman, Phys. Rev. E **54**, R3090 (1996)] is further evidence of violation of scaling. A simple argument based on percolation cluster statistics yields a similar logarithmic evolution. [S1063-651X(98)02702-0]

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I. INTRODUCTION

Phase transitions between an absorbing state (one admitting no further evolution), and an active regime occur in models of autocatalytic chemical reactions, epidemics, and transport in disordered media [1]. Paradigms of this sort of transition are the contact process (CP) [2] and its simultaneous-update counterpart, directed percolation (DP) [3]. Since many-particle systems often incorporate frozen-in randomness, it is natural to investigate the effect of quenched disorder on an absorbing-state transition. Thus, some years ago, Noest observed that the critical behavior of disordered directed percolation is quite different from that of pure DP [4]. We recently studied the CP with quenched disorder in the form of random site dilution, and found logarithmic time dependence at the critical point [5]. For example, P(t), the probability of survival, starting from a single active site, follows $P \sim 1/(\ln t)^a$ for large t. Such a form is incompatible with the scaling hypothesis that applies quite generally to absorbing-state transitions [6]. This violation of scaling is consistent with Janssen's recent field-theoretic analysis, which shows that the resulting renormalization group equations have only runaway solutions [7]. Here we present further results bearing on the violation of scaling. In particular, we find that the exponent $\nu_{||}$ does not exist for the diluted contact process (DCP). We also propose an explanation for logarithmic behavior at the critical point.

In the CP on the square lattice, each site is either vacant or occupied by a particle. Particles are created at vacant sites at rate $\lambda n/4$, where *n* is the number of occupied nearest neighbors, and are annihilated at unit rate, independent of the surrounding configuration. The order parameter is the particle density ρ ; it vanishes in the vacuum state, which is absorbing. As λ is increased beyond $\lambda_c = 1.6488(1)$, there is a continuous phase transition from the vacuum to an active steady state; for $\Delta \equiv \lambda - \lambda_c > 0$, the stationary density $\overline{\rho} \sim \Delta^{\beta}$. In the vicinity of the critical point the characteristic relaxation time $\tau \sim |\Delta|^{-\nu_{\parallel}}$, and the correlation length diverges as $\xi \sim |\Delta|^{-\nu_{\perp}}$. In the DCP, a fraction *x* of the sites are diluted at random, and the birth-and-death process defining the CP is restricted to nondiluted sites. (Further details on the DCP may be found in Ref. [5].)

To provide the necessary background we summarize the scaling behavior of the CP and allied models [6]. Consider first the evolution from an initial configuration with just a single particle at the origin. The conditional probability of finding a particle at r, given that at time zero there was a particle at the origin, and that all other sites were vacant, obeys

$$\rho(r,t;0,0) \simeq t^{\eta - dz/2} F(r^2/t^z, \Delta t^{1/\nu_{||}}).$$
(1)

Similarly the survival probability is expected to follow

$$P(t) \simeq t^{-\delta} \Phi(\Delta t^{1/\nu \parallel}).$$
⁽²⁾

(*F* and Φ are scaling functions.) At the critical point $(\Delta = 0)$, Eq. (2) implies $P(t) \sim t^{-\delta}$, while Eq. (1), when integrated over space, yields a mean population $n(t) \sim t^{\eta}$. If we take the second moment (in space) of $\rho(r,t;0,0)$ with $\Delta = 0$, we obtain

$$R^{2}(t) \sim \frac{t^{\eta - dz/2}}{n(t)} \int x^{2} f(x^{2}/t^{z}) d^{d}x \propto t^{z}.$$
 (3)

For $\Delta > 0$, the survival probability attains a finite asymptotic value: $\lim_{t\to\infty} P(t) \equiv P_{\infty} \sim \Delta^{\beta'}$ [8,9]. Several scaling relations can be derived, in particular,

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(4)

$$z=2\nu_{\perp}/\nu_{\parallel}$$

and

$$\delta = \beta / \nu_{||}, \qquad (5)$$

as well as the hyperscaling relation

$$4\,\delta + 2\,\eta = dz.\tag{6}$$

Equations (1)–(3) describe spreading from a single seed. Consider, on the other hand, a system at the critical point, with all L^d sites initially occupied, and let $P_m(t)$ be the survival probability starting from this maximally occupied state. At short times $P_m = 1$ and the density is governed by the power law $\rho(t) \sim t^{-\delta}$. Following this initial phase, P_m decays exponentially, with a characteristic time $\tau \sim L^{\nu \parallel / \nu_\perp}$, and the density in the surviving sample attains a quasistationary value $\rho_s \sim L^{-\beta/\nu_\perp}$. The scaling results summarized above have been amply confirmed for the CP and other models with a unique absorbing configuration, and have been extended to models possessing multiple absorbing configurations [9,10].

In our recent study of the DCP, we found that P(t), n(t), and $R^{2}(t)$ display logarithmic time dependence at the critical point, which is incompatible with the scaling forms, Eqs. (1) and (2), describing the pure model. In this work we report further results on static and dynamic critical properties, in an effort to determine the extent of the scaling violation, and to understand its origin. In Sec. II we analyze the stationary density (in the supercritical regime), and the quasistationary density (at the critical point), to obtain estimates of β and ν_{\perp} , and also examine the stationary density-density correlation function, which decays algebraically. Section III concerns the survival probability P_m starting from a maximally occupied state; it also decays algebraically, roughly as predicted by a simple probabilistic picture. Another simple argument is presented in Sec. IV, for the logarithmic decay of the survival probability. We conclude, in Sec. V, with a discussion of our main results, and of the reason for violation of dynamic scaling in the DCP.

II. STATIC BEHAVIOR

A. Stationary density

We determined the stationary density $\overline{\rho}$ for dilutions x = 0.05, 0.1, 0.2, 0.3, and 0.35, by the following simulation procedure. After generating a disorder configuration on a lattice of $L \times L$ sites, and initially occupying all nondiluted sites, we permit the system to relax for a time t_R , and then accumulate data on the density for a period of duration t_s . This process is repeated N_T times (with a new disorder configuration for each trial), and the density $\rho(t)$, computed over the N_S trials that survive up to time $t_R + t_S$, is examined to verify that sufficient time has been allowed for relaxation. (If not, t_R is increased accordingly.) We then take the mean density for each trial during the observation time $t_R \leq t \leq t_S$, and compute the mean and standard deviation over the sample of N_S independent trials. We used lattice sizes L=100, 200, 400, and 800, increasing L as we approachedthe critical point, and checking (for $L \leq 400$) that estimates



FIG. 1. Stationary density ρ vs $\Delta \equiv \lambda - \lambda_c$ in the diluted contact process. \times : x = 0.05; \blacksquare : x = 0.10; \bigcirc : x = 0.20; \Box : x = 0.30; \bullet : x = 0.35. Figures denote the slopes of the various straight lines.

for the stationary density agreed for two lattice sizes. The sample size N_S ranged from 50 to 100. We employed $t_R = t_S = 5 \times 10^4 - 2.5 \times 10^5$, the larger values reflecting slower relaxation near $\lambda_c(x)$. [We use the critical point estimates $\lambda_c(x)$ obtained via time-dependent simulations in Ref. [5].]

Our results for the stationary density, shown in Fig. 1, reveal a crossover between the DP value of $\beta \approx 0.58$ at small dilutions (x = 0.05 and 0.1), and a larger exponent as one approaches λ_c . The data for $x \ge 0.1$ yield exponents in the range 0.89–0.98, suggesting that $\beta = 0.93(5)$. (Figures in parentheses denote statistical uncertainties — one standard deviation.) We also find that $\overline{\rho^2} \sim \Delta^{2\beta}$ with $2\beta = 1.84(4)$. Thus the order parameter exponent agrees, to within uncertainty, with our earlier result $\beta' = 0.99(3)$ for the exponent governing the ultimate survival probability [5]. (For the pure CP, $\beta' = \beta \approx 0.58$.) While our estimate is not far from Noest's result, $\beta = 1.10(5)$, we regard it as excluding that value; we do not feel, however, that our data rule out $\beta = 1$.

B. Quasistationary density

Rather than studying spatial correlations directly, we determine ν_{\perp} by analyzing the quasistationary density ρ_s at the critical point as a function of the system size; this yields β/ν_{\perp} , as noted in Sec. I. We studied lattice sizes varying from L=8 to L=128, averaging over 2×10^3 to 10^5 independent runs, of duration $t_s = 10^3$ to $t_s = 10^5$. (The larger sample sizes and longer run times apply to the larger L values.) We show in Fig. 2 a log-log plot of $\rho_s(\lambda_c, L)$ versus L for dilutions ranging from 0.02 to 0.35, along with the slopes of linear least-squares fits to the data for $L \ge 16$. (The uncertainty in the slope ranges from 1 to 3%.) For x < 0.1, the scaling of ρ_s is similar to that found in the pure CP, for which $\beta/\nu_{\perp} = 0.80(3)$. But for larger dilutions we see a steady increase in the slope; we estimate $\beta/\nu_{\perp} = 0.93(3)$ for the DCP. Combining this with our estimate $\beta = 0.93(5)$, we obtain $\nu_{\perp} = 1.00(9)$ for the DCP. This is lower than, but still consistent with, Noest's earlier estimate of 1.17(10) [4]. On the other hand, a theorem of Chayes *et al.* requires $d\nu_{\perp} \ge 2$ for models with quenched disorder, or $\nu_{\perp} \ge 1$ here [11]; the



FIG. 2. Quasistationary density $\rho_s(\lambda_c, L)$ vs *L*, for dilutions x=0.02 (\blacksquare); 0.05 (\triangle); 0.10 (\times); 0.20 (\bigcirc); 0.30 (\diamond), and 0.35 (\Box). The lines are linear fits to the last four data points in each set; figures indicate the slope.

upper range of our estimate is consistent with this result. [Note that if we use our earlier estimate, $\beta' = 0.99(3)$ in place of our β value, we obtain $\nu_{\perp} = 1.06(7)$.]

C. Stationary density-density correlation function

The stationary density-density correlation function is defined by

$$C(t) = \langle \sigma_i(t_0 + t) \sigma_i(t_0) \rangle, \tag{7}$$

where $\sigma_i(t) = 1$ (0) if site *i* is occupied (vacant) at time *t*, and the average is over realizations of the process *and* over disorder. It is understood that t_0 is sufficiently large that the right-hand side (rhs) is independent of t_0 . For the undiluted CP, the rhs is then independent of *i* as well, and $\Delta C \equiv C(t) - \overline{\rho^2} \sim \exp(-t/\tau)$ for large *t*. τ is a characteristic relaxation time diverging as $\tau \sim \Delta^{-\nu||}$ near the critical point.

We studied the density-density correlation function in the DCP at x=0.1 and 0.3, evaluating the rhs of Eq. (7) for a *single* site *i* (the first nondiluted site to be generated), on $L \times L$ lattices with periodic boundaries, using 500–2000 independent realizations of the disorder. Figure 3 (inset) shows a typical evolution, with C(t) slowly approaching an asymptote, C_{∞} . The main graph shows that the excess, $\Delta C \equiv C(t) - C_{\infty} \sim t^{-b}$, so it cannot be characterized by a relaxation time. The exponent *b* varies from about 0.7 to 0.8 well above λ_c , to about 0.5 near λ_c . Note that for $b \leq 1$, even the alternative expression

$$\tau \equiv \int_0^\infty dt \Delta C(t), \tag{8}$$

similar to that employed by Noest, is undefined. We also observe a power-law approach (with an exponent of about



FIG. 3. Main graph: excess density-density correlation function vs time in the DCP for x=0.3 and $\lambda=2.60$ (top), 2.70 (middle), and 2.47 (bottom). The inset shows *C* versus *t* for x=0.3 and $\lambda=2.70$.

1/2) of the critical quasistationary density ρ_s , discussed in the preceding subsection, to its asymptotic value.

III. DYNAMIC BEHAVIOR

Consider the pure CP on a lattice of L^d sites, starting with all sites occupied. Since we are dealing with a finite system, there is a well-defined lifetime $\tau(\Delta, L)$ and the survival probability $P_m(t) \propto \exp[-t/\tau(\Delta, L)]$ for large *t*. Just at the critical point the lifetime has a power-law dependence on *L*: $\tau(0,L) \sim L^{\nu_{\parallel}/\nu_{\perp}}$. For $\Delta > 0$, vacating $(L/\xi)^d$ independent regions simultaneously is an exponentially rare event, and we expect the scaling form

$$\tau(\Delta, L) \sim L^{\nu_{\parallel}/\nu_{\perp}} \exp[c(L\Delta^{\nu_{\perp}})^d]$$
(9)

(*c* is a constant), as is confirmed by the data shown in Fig. 4. (The data also appear to scale for $\Delta < 0$, but with a different scaling function.)



FIG. 4. Semilogarithmic scaling plot of the lifetime in the *pure* CP. Squares: $|\Delta| = 0.01$; diamonds: $|\Delta| = 0.02$; \bullet : $|\Delta| = 0.05$.



FIG. 5. Survival probability P_m versus time in the critical DCP starting from a maximally occupied state, for dilution x=0.1 and $\lambda = \lambda_c = 1.8464$. +: L=32; $\bigcirc: L=64$; $\diamond: L=128$.

We studied the relaxation from a maximally occupied state (all nondiluted sites occupied) in the critical DCP. Figures 5 and 6 show $P_m(t)$ for x = 0.1 and 0.3, respectively, for L = 32, 64, and 128. (We studied samples of 5000, 2000, and 1000 trials for L=32, 64, and 128, respectively) From the figures it appears that following the initial stage, the survival probability crosses over to a nonuniversal power law, with an exponent that decreases with x and with L. (For x=0.1we find $P_m \sim t^{-a}$ with a = 1.8, 1.1, and 0.86 for L = 32, 64, and 128, respectively; for x = 0.3 the corresponding powers are 0.71, 0.55, and 0.37.) Since the asymptotic decay of P_m is nonexponential, there is no characteristic lifetime for the process. The initial stage, during which $P_m = 1$, is characterized by a correlation length $\xi(t) \le L$. During this phase we find $n_m \sim t^{-\delta}$ with $\delta \simeq 0.47$, as for the pure model. Once P_m starts to decay, n_m crosses over to a different, nonuniversal power law, as seen in Fig. 7.

While the P_m data are certainly inconsistent with exponential decay, a slight downward curvature suggests a faster than power-law decay. In fact, somewhat more linear plots are obtained using the form $\ln P_m \propto -(\ln t)^a$, with *a* in the range 1.5–3 depending on the data set. The data are too noisy to permit a definite conclusion regarding the form of



FIG. 6. Same as Fig. 5, but for x = 0.3, $\lambda = \lambda_c = 2.47$.



FIG. 7. Mean number of particles n_m in the critical DCP with x=0.3. Symbols as in Fig. 5.

 P_m , but it is interesting that a simple argument yields the modified power law. Because of fluctuations, the actual fraction of diluted sites in a given sample differs from x by dx, which is a Gaussian random variable with mean zero and variance $x(1-x)/L^d$, by the central limit theorem. If we now ignore the spatial inhomogeneity of the disorder, and ascribe the effective distance from criticality, Δ , of a sample to the fluctuation dx, we find that Δ is likewise Gaussian:

$$P(\Delta) \sim \exp(-bL^d \Delta^2), \tag{10}$$

with

$$b^{-1} = 2x(1-x) \left(\frac{d\lambda_c}{dx}\right)^2.$$
 (11)

If we assume that the lifetime $\tau(\Delta, L)$ scales as in the pure model, Eq. (9), the survival probability is given by

$$P_m(t) \sim \int d\Delta \exp\left(-bL^d\Delta^2 - \frac{t}{\tau(\Delta,L)}\right).$$
 (12)

Maximizing the argument of the exponential to extract the leading behavior at large t, we obtain

$$\ln P_m \sim -bL^{d-2/\nu_{\perp}} (\ln \tilde{t})^{2/d\nu_{\perp}}, \qquad (13)$$

where $\tilde{t} \equiv t/(bL^{\nu_{\parallel}/\nu_{\perp}})$. [The exponent $2/d\nu_{\perp} \approx 1.37$ for d=2. To evaluate the rhs of Eq. (13) we require $d\lambda_c/dx$; from the data reported in Ref. [5], we obtain values of 2.256 and 4.393 for x=0.1 and x=0.3, respectively.] In Fig. 8 we test the scaling prediction by plotting $(L^{2/\nu_{\perp}-2}/b) \ln P_m$ versus $(\ln \tilde{t})^{1/\nu_{\perp}}$. For x=0.3 the data collapse for different L values is quite good. For x=0.1 the data do not collapse, but Eq. (13) nevertheless appears to account for much of the L dependence. The x dependence of P_m , by contrast, is not predicted correctly by the scaling argument. [The reason for this is unclear, though one may speculate that the factor c in Eq. (9) depends upon x in some unknown manner.]

Our estimate for $P_m(t)$, which treats fluctuations in the disorder as if they were spatially homogeneous, and uses pure-model scaling, is clearly inadequate to deal with the true subtlety of the DCP. That it may yet contain some germ



FIG. 8. (a) Scaling plot of the data of Fig. 5 (x=0.1); (b) Scaling plot of the data of Fig. 6 (x=0.3). $r=1/\nu_{\perp}=1.37$; $s=2-2/\nu_{\perp}=0.74$; \tilde{t} is defined in the text. Symbols as in Fig. 5.

of truth is suggested by our finding that when we do not average over disorder, the decay of P_m is *exponential*, as in the pure CP, but with a lifetime particular to the disorder set generated. (One is naturally interested in knowing the distribution of the relaxation time. This poses a formidable numerical task that we hope to address in future work.) In summary, the relaxation of the DCP from a maximally occupied state is similar to that of the pure model during the initial stage, in which correlations have yet to grow to the size of the system. But afterward the evolution follows nonuniversal power laws (or modified power laws) and we cannot define a characteristic lifetime.

IV. CRITICAL DYNAMICS

In this section we propose a simple explanation for how logarithmic time dependence arises in the critical DCP. Consider the survival probability P(t) starting from a single occupied site or seed located at the origin, \mathcal{O} . Clearly, the trials that contribute to P(t) at large t are those in which the seed happens to fall in a large, favorable region. To make the notion of a "favorable region" somewhat more precise, imagine taking the disorder configuration on a cube of L^d sites, filling space with periodic copies, and running the contact process with $\lambda = \lambda_c(x)$ on this lattice. For some disorder configurations — the favorable ones — the process will in fact be *supercritical*, because the fraction of diluted sites is < x, or because of a particularly advantageous arrangement of the diluted sites. Such regions are characterized by an "effective distance from criticality" $\Delta_{eff} > 0$. Any disorder configuration contains both favorable and unfavorable regions. If the seed lies in a favorable region, we can define its "domain" as the maximal connected favorable region containing \mathcal{O} . (The domain is surrounded by unfavorable regions, which impede the spread of the process.) For simplicity, we suppose that on a domain of V sites, having some $\Delta_{\text{eff}} > 0$, the DCP has a lifetime $\tau \sim \exp[cV\Delta_{\text{eff}}^{d\nu_{\perp}}]$, as it would on a compact region, in the pure model. From the central limit theorem, the typical value of Δ_{eff} on a domain of V sites $\sim V^{-1/2}$, yielding a lifetime $\tau \sim \exp[cV^{1-d\nu_{\perp}/2}]$. At time t, only trials whose seeds happen to fall in a domain with $\tau \ge t$, or $V \ge c'(\ln t)^{2/(2-d\nu_{\perp})}$ survive.

It remains to estimate the probability p(V) that O belongs to a domain of V sites. To do this, note that a domain is a kind of percolation cluster. The precise definition of the sites in this percolation problem is unclear (we might imagine averaging over small blocks of sites in the original lattice), as is the connectivity rule (next-nearest-neighbor blocks, for example, might effectively be connected). But we should expect the associated percolation model to be isotropic and of finite range. Moreover, domain percolation must be *critical* at $\lambda_c(x)$. If it were supercritical, the contact process would be able to spread into an unbounded domain, and so would itself be supercritical. Similarly, if domains were subcritical, their size distribution would decay exponentially, and the CP would be subcritical. At the critical point, the domain size is power-law distributed: $p(V) \sim V^{-(\tau_p^{-1})}$ for large V, with τ_p the usual percolation cluster-size exponent ($\tau_n = 187/91$ in two dimensions). Combining this result with the lifetime estimate, we have

$$P(t) \sim \int_{c'(\ln t)^{2/(2-d\nu_{\perp})}} \frac{dV}{V^{\tau_p - 1}} \sim (\ln t)^{-2(\tau_p - 2)/(2-d\nu_{\perp})}.$$
(14)

As in the argument (Sec. III) for the survival probability starting from a maximally occupied state, the effect of inhomogeneity in the disorder is greatly oversimplified. Inserting the known values of ν_{\perp} and of τ_p , we obtain $P(t) \sim (\ln t)^{-0.2}$, whereas the exponent we observed in simulations [5] is much larger, and *nonuniversal*, ranging from about 2.7 at x=0.35 to 4.6 at x=0.1. Thus we offer the above argument without any claim of quantitative validity, but rather to show how a simple treatment of disorder leads naturally to logarithmic time dependence, and in the hope that it may form the basis for a more convincing approach.

The probabilistic arguments suggest that it may be possible to understand how anomalous dynamics arises from an average over disorder. Here it is important to recall Noest's analysis of contributions from exponentially rare, favorable disorder configurations to the survival probability P(t). By deriving upper and lower bounds on the survival probability, he was able to prove power-law decay of P(t) in a Griffiths phase for $\lambda_c(x) > \lambda > \lambda_c(0)$ [12]. Arguments of a somewhat similar nature were advanced by Bray in his discussion of the relaxation of diluted spin models [13].

V. DISCUSSION

We have found that some aspects of the diluted contact process exhibit the same sort of critical behavior — albeit with different exponents — as seen in the pure model. Other features — spreading from a seed, the density-density autocorrelation function C(t) and the survival probability $P_m(t)$, starting from a maximally occupied state—do not follow the usual scaling, and are nonuniversal. The anomalous properties are all connected with dynamics: normally, C(t) and $P_m(t)$ decay exponentially, with the diverging lifetime serving to define the exponent $\nu_{||}$ through $\tau \sim \Delta^{-\nu_{||}}$. Here no such definition is possible. Consistent with this, the spreading exponents δ , z, and η , which are formally zero, are connected to $\nu_{||}$ via the scaling relations Eqs. (4)–(6). Since β and ν_{\perp} are in fact finite, Eqs. (4) and (5) suggest that $\nu_{||}$ is infinite. Given Janssen's recent results [7], it is of interest to know whether our finding of power-law static behavior, but anomalous time dependence, is compatible with a fieldtheoretical analysis.

Some insight into the violation of dynamic scaling may be gained by returning to Eq. (9): the exponential dependence of the lifetime (in the pure CP) upon L and Δ suggests an extreme sensitivity of dynamic behavior to disorder. We might expect dynamics to be dominated by the extremes of local fluctuations in the disorder. The result is that a property such as the relaxation time τ for the density-density autocorrelation function is *non-self-averaging*, i.e., it does not converge to a limiting value even as $L \rightarrow \infty$ [14,15]. Another manifestation of non-self-averaging is the dependence of P(t) and n(t), even at long times, on the location of the seed at time zero. These features are dominated by local fluctuations, rather than by the properties of a "typical" disorder configuration. This in turn suggests that further insight may be gained by studying critical behavior for *fixed* disorder configurations, in order to determine the statistical distributions of various system properties, and of the domains defined in Sec. IV. In this way the primitive probabilistic arguments for P_m and P(t) could be honed into a quantitative description of how anomalous behavior emerges in the average over disorder.

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