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Random Fields at a Nonequilibrium Phase Transition

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We study nonequilibrium phase transitions in the presence of disorder that locally breaks the symmetry between two equivalent macroscopic states. In low-dimensional equilibrium systems, such *random-field* disorder is known to have dramatic effects: it prevents spontaneous symmetry breaking and completely destroys the phase transition. In contrast, we show that the phase transition of the one-dimensional generalized contact process persists in the presence of random-field disorder. The ultraslow dynamics in the symmetry-broken phase is described by a Sinai walk of the domain walls between two different absorbing states. We discuss the generality and limitations of our theory, and we illustrate our results by large-scale Monte Carlo simulations.

Impurities, defects, and other types of quenched disorder can have drastic effects on the long-time and large-distance behavior of many-particle systems. For example, disorder can modify the universality class of a critical point [1,2], change a phase transition from first order to continuous [3–5], or smear a sharp transition over an interval of the tuning parameter [6]. Particularly strong effects arise from disorder that locally breaks the symmetry between two equivalent macroscopic states while preserving the symmetry globally (in the statistical sense). As this type of disorder corresponds to a random external field in a magnetic system, it is usually called random-field disorder. Recently, a beautiful example of a random-field magnet was discovered in $LiHo_xY_{1-x}F_4$ [7–9]. Random-field disorder naturally occurs when the order parameter breaks a real-space symmetry such as in nematic liquid crystals in porous media [10] and stripe states in high-temperature superconductors [11].

Imry and Ma [12] discussed random-field effects at equilibrium phase transitions based on an appealing heuristic argument. Consider a uniform domain of linear size L in d space dimensions. The free energy gain due to aligning this domain with the (average) local random field behaves as $L^{d/2}$ while the domain wall energy is of the order of L^{d-1} [13]. For $d < 2$, the system thus gains free energy by forming finite-size domains that align with the random field. In contrast, for $d > 2$, the uniform state is preferred. Building on this work, Aizenman and Wehr [5] proved rigorously that random-field disorder prevents spontaneous symmetry breaking in all dimensions $d \leq 2$ for Ising symmetry and $d \leq 4$ for continuous symmetry. Thus, random fields destroy an equilibrium phase transition in sufficiently low dimensions.

In nature, thermal equilibrium is rather the exception than the rule. Although equilibrium is an excellent approximation for some systems, many others are far from equilibrium and show qualitatively different behaviors. In recent years, phase transitions between different nonequilibrium states have attracted considerable attention. Examples can be found in population dynamics, chemical reactions, growing surfaces, granular flow, as well as traffic jams [14–18]. It is therefore important to study random-field effects at such nonequilibrium phase transitions. Are these transitions destroyed by random fields just like equilibrium transitions?

In this Letter, we address this question for a prominent class of nonequilibrium phase transitions, viz., absorbing state transitions separating active, fluctuating states from inactive, absorbing states where fluctuations cease entirely. We develop a heuristic argument showing that random-field disorder which locally favors one of two equivalent absorbing states over the other does not prevent global spontaneous symmetry breaking in any dimension. The random fields thus do not destroy the nonequilibrium transition. In the symmetry-broken phase, the relevant degrees of freedom are domain walls between different absorbing states. Their long-time dynamics is given by a Sinai walk [19] leading to an ultraslow approach to the absorbing state during which the density of domain walls decays as $\ln^{-2}(t)$ with time t (see Fig. 1). We also study the
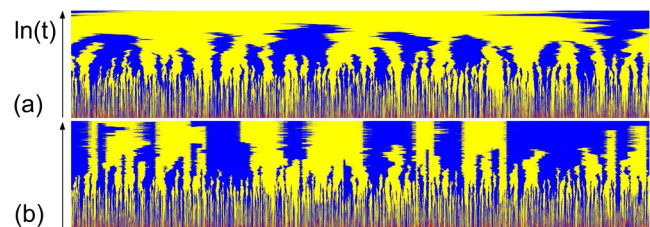

FIG. 1 (color online). Time evolution of the generalized contact process in the inactive phase (a) without ($\mu = 5/6$) and (b) with random-field disorder ($\mu_h = 1$, $\mu_l = 2/3$). I_1 and I_2 are shown in yellow and blue (light and dark grey). Active sites between the domains are marked in red (middle grey). The difference between the diffusive domain wall motion (a) and the much slower Sinai walk (b) is clearly visible (part of a system of 10^5 sites for times up to 10^8).

behavior right at the critical point where we find even slower dynamics.

In the remainder of the Letter, we sketch the derivation of the results, and we support them by Monte Carlo simulations. For definiteness, we first consider the generalized contact process with two absorbing states [20] in one dimension. We later argue that our heuristic argument applies to an entire class of absorbing state transitions.

The (simple) contact process [21] is a prototypical model featuring an absorbing state transition. Each site of a d-dimensional hypercubic lattice is either in the active (infected) state A or in the inactive (healthy) state I. The time evolution is a continuous-time Markov process with infected sites healing at a rate μ while healthy sites become infected at a rate $\lambda m/(2d)$ where m is the number of infected nearest neighbors. The long-time behavior is governed by the ratio of λ and μ. If $\mu \gg \lambda$, healing dominates over infection, and all sites will eventually be healthy. The absorbing state without any infected sites is thus the only steady state. For $\lambda \gg \mu$, the infection never dies out, leading to an active steady state with a nonzero density of infected sites. The absorbing and active steady states are separated by a nonequilibrium transition in the directed percolation (DP) [22] universality class.

Following Hinrichsen [20], we generalize the contact process by allowing each site to be in one of $n + 1$ states, the active state A or one of n inactive states I_k ($k = 1, \ldots, n$). The time evolution of the generalized contact process (GCP) is conveniently defined [20] via the transition rates for pairs of nearest-neighbor sites,

$$w(AA \rightarrow AI_k) = w(AA \rightarrow I_kA) = \bar{\mu}_k/n, \qquad (1)$$

$$w(AI_k \rightarrow I_kI_k) = w(I_kA \rightarrow I_kI_k) = \mu_k, \qquad (2)$$

$$w(AI_k \rightarrow AA) = w(I_kA \rightarrow AA) = \lambda, \qquad (3)$$

$$w(I_kI_l \rightarrow I_kA) = w(I_kI_l \rightarrow AI_l) = \sigma, \qquad (4)$$

with $k, l = 1, \ldots, n$ and $k \neq l$. All other rates vanish. The GCP defined by Eqs. (1)–(4) reduces to the simple contact process if we set $n = 1$ and $\bar{\mu}_k = \mu_k = \mu$ (up to rescaling all rates by the same constant factor [23]). The transition of Eq. (4) permits competition between different inactive states as it prevents different domains from sticking together. Instead, they can separate, and the domain walls can move. We now set $\bar{\mu}_k = \mu_k$ and $\lambda = \sigma = 1$ to keep the parameter space manageable [24]. This also fixes the time unit. Moreover, we focus on $d = 1$ and $n = 2$.

The long-time behavior again follows from comparing the infection rate λ with the healing rates μ_1 and μ_2. Consider two equivalent inactive states, $\mu_1 = \mu_2 = \mu$. For small μ, the system is in the active phase with a nonzero density of infected sites. In this fluctuating phase, the symmetry between the two inactive states I_1 and I_2 is not broken; i.e., their occupancies are identical. If μ is

increased beyond $\mu_c^0 \approx 0.628$ [20,25], the system undergoes a nonequilibrium phase transition to one of the two absorbing steady states (either all sites in state I_1 or all in state I_2). At this transition, the symmetry between I_1 and I_2 is spontaneously broken. Its critical behavior is therefore not in the DP universality class but in the so-called DP2 class which, in $d = 1$, coincides with the parity conserving class [26]. If $\mu_1 \neq \mu_2$, one of the two inactive states dominates for long times, and the critical behavior reverts back to DP.

We introduce quenched (time-independent) disorder by making the healing rates $\mu_k(\mathbf{r})$ at site $\mathbf{r}$ independent random variables governed by a probability distribution $W(\mu_1, \mu_2)$. As we are interested in random-field disorder which locally breaks the symmetry between I_1 and I_2, we choose $\mu_1(\mathbf{r}) \neq \mu_2(\mathbf{r})$. Globally, the symmetry is preserved in the statistical sense implying $W(\mu_1, \mu_2) = W(\mu_2, \mu_1)$. An example is the correlated binary distribution

$$W(\mu_1, \mu_2) = \frac{1}{2}\delta(\mu_1 - \mu_h)\delta(\mu_2 - \mu_l)$$
$$+ \frac{1}{2}\delta(\mu_1 - \mu_l)\delta(\mu_2 - \mu_h) \qquad (5)$$

with possible local healing rate values μ_h or μ_l [27].

To address our main question, namely whether the random-field disorder prevents the spontaneous breaking of the global symmetry between the two inactive states and thus destroys the nonequilibrium transition, we analyze the large-μ regime where all healing rates are larger than the clean critical value μ_c^0. In this regime, almost all sites quickly decay into one of the two inactive states I_1 or I_2. The relevant long-time degrees of freedom are domain walls between I_1 and I_2 domains. They move via a combination of the process of Eq. (4) which creates an active site at the domain wall and the process of Eq. (2) which allows this active site to decay into either I_1 or I_2. Because of the disorder, the resulting domain wall hopping rates depend on the site $\mathbf{r}$. Importantly, the rates for hopping right and left are different because the underlying healing rates $\mu_1(\mathbf{r})$ and $\mu_2(\mathbf{r})$ are not identical.

The long-time dynamics in the large-μ regime is thus governed by a random walk of the domain walls. Due to the local left-right asymmetry, this random walk is not a conventional (diffusive) walk but a Sinai walk [29]. The typical displacement of a Sinai walker grows as $\ln^2(t/t_0)$ with time t [19] (t_0 is a microscopic time scale), more slowly than the well-known $t^{1/2}$ law for a conventional walk (see Fig. 1). When two neighboring domain walls meet, they annihilate, replacing three domains by a single one. Domain walls surviving at time t thus have a typical distance proportional to $\ln^2(t/t_0)$. The domains grow without limit, and their density decays as $\ln^{-2}(t/t_0)$. In the long-time limit, the system reaches a single-domain state; i.e., either all sites are in state I_1 or all in I_2. This implies that the symmetry between I_1 and I_2 is spontaneously

broken (which of the two absorbing states the system ends up in depends on details of the initial conditions and of the stochastic time evolution). The nonequilibrium transition consequently persists in the presence of random-field disorder.

It is important to contrast the domain wall dynamics in our system with that of a corresponding equilibrium problem such as the random-field Ising chain (whose low-temperature state consists of domains of up and down spins [30]. The crucial difference is that the inactive states I_1 and I_2 in our system are absorbing: active sites and new domain walls never arise in the interior of a domain. In contrast, inside a uniform domain of the random-field Ising chain, a spin flip (which creates two new domain walls) can occur anywhere due to a thermal fluctuation. This mechanism limits the growth of the typical domain size to its equilibrium value dictated by the Imry-Ma argument [12], and thus prevents spontaneous symmetry breaking.

To verify these heuristic arguments and to illustrate the results, we perform Monte Carlo simulations [25] of the one-dimensional GCP with random-field disorder. We use system sizes up to $L = 10^5$ and times up to $t = 2 \times 10^8$. The random-field disorder is implemented via the distribution of Eq. (5) with $1.5\mu_l = \mu_h \equiv \mu$. Our simulations start from a fully active lattice (all sites in state A), and we monitor the density ρ of active sites as well as the densities ρ_1 and ρ_2 of sites in the inactive states I_1 and I_2, respectively. Figure 2 presents an overview of the time evolution of the density ρ.

We now focus on the curves with healing rates $\mu \gtrsim 1.0$ for which both $\mu_h = \mu$ and $\mu_l = 2\mu/3$ are larger than the clean critical value μ_c^0. The inset of Fig. 2 shows that the density continues to decay to the longest times studied for all these curves. However, the decay is clearly slower than a power law. To compare with our theoretical arguments, we note that active sites only exist near domain walls in the large-μ regime. We thus expect the density of active sites
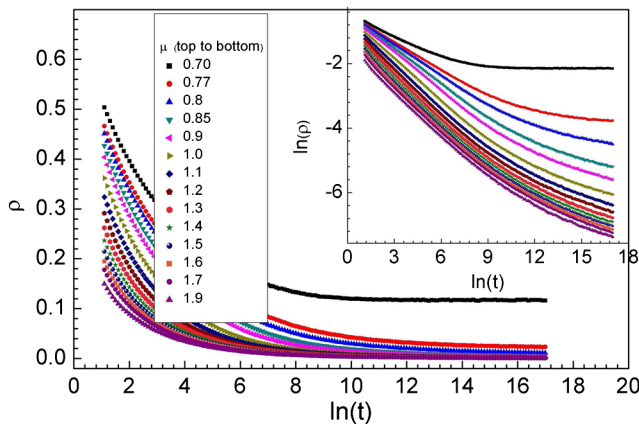
to be proportional to the domain wall density, yielding $\rho \sim \ln^{-2}(t/t_0)$. To test this prediction we plot $\rho^{-1/2}$ vs $\ln(t)$ in Fig. 3; in such a graph the expected behavior corresponds to a straight line. The figure shows that all curves with $\mu > 1$ indeed follow the prediction over several orders of magnitude in time.

In addition to the inactive phase, we also study the critical point. To identify the critical healing rate μ_c, we extrapolate to zero both the stationary density $\rho_{\mathrm{st}} = \lim_{t\to\infty} \rho(t)$ in the active phase and the inverse prefactor of the $\ln^{-2}(t/t_0)$ decay in the inactive phase. This yields $\mu_c \approx 0.80$ (see inset of Fig. 4). At this healing rate, the density decay is clearly slower than the $\ln^{-2}(t/t_0)$ law governing the inactive phase. This extremely slow decay and the uncertainty in μ_c prevent us from determining the functional form of the critical $\rho(t)$ curve unambiguously. If we assume a time dependence of the type $\rho(t) \sim \ln^{-x}(t/t_0)$ we find a value of $x \approx 0.5$. Moreover, from the dependence of the stationary density on the healing rate, $\rho_{\mathrm{st}} \sim (\mu_c - \mu)^\beta$, we obtain $\beta \approx 1.5$. The values of x and β should be considered rough estimates. An accurate determination of the critical behavior of the GCP with random-field disorder requires a significantly larger numerical effort and remains a task for the future.

In summary, we have shown that the nonequilibrium phase transition of the one-dimensional GCP survives in the presence of random-field disorder, in contrast to one-dimensional equilibrium transitions that are destroyed by random fields. In the concluding paragraphs, we discuss the generality and limitations of our results.

The crucial difference between random-field effects in equilibrium systems such as the random-field Ising chain and in the GCP is the absorbing character of the inactive states I_1 and I_2 in the latter. The interior of an I_1 or I_2 domain is *dead* as no active sites and no new domain walls can ever arise there. In contrast, in an equilibrium system, pairs of new domain walls can appear in the interior of a
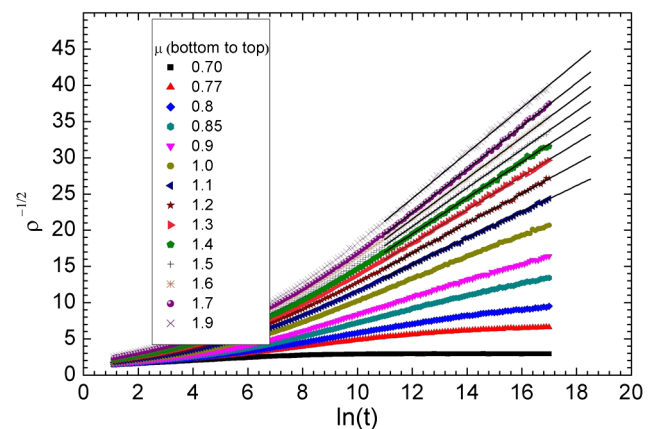

FIG. 2 (color online).   Density ρ vs time t for several values of the healing rate μ. The data are averages over 60 to 200 disorder configurations. Inset: The log-log plot shows that the density decay is slower than a power law for all μ.

FIG. 3 (color online). $\rho^{-1/2}$ vs $\ln(t)$ for several values of the healing rate μ. The solid straight lines are fits to the predicted behavior $\rho \sim \ln^{-2}(t/t_0)$.
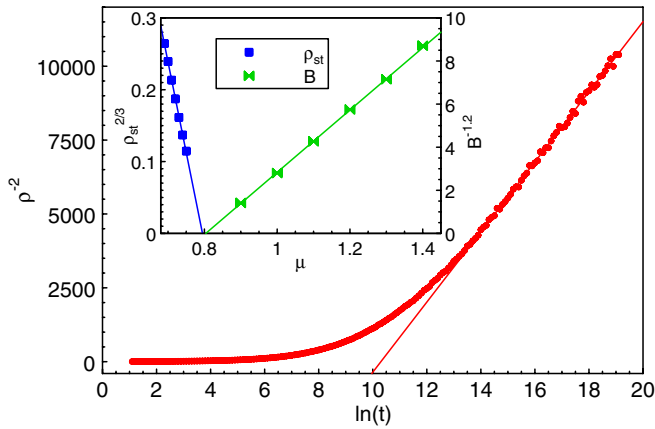
FIG. 4 (color online).   Density vs time at the critical healing rate $\mu_c = 0.8$, plotted as $\rho^{-1/x}$ vs $\ln(t)$ with $x = 0.5$. The solid line is a fit to $\rho(t) \sim \ln^{-x}(t/t_0)$. Inset: Identifying μ_c from the stationary density ρ_{st} in the active phase and the prefactor of the $\rho = B\ln^{-2}(t/t_0)$ decay in the inactive phase.

uniform domain via a thermal fluctuation. This limits the growth of the typical domain size to the Imry-Ma equilibrium size and thus destroys the equilibrium transition (in sufficiently low dimensions). We expect our results to hold for all nonequilibrium phase transitions at which the random-field disorder locally breaks the symmetry between two absorbing states. Other nonequilibrium transitions may behave differently. For example, our theory does not apply if the random fields break the symmetry between two active states.

In the symmetry-broken inactive phase, the dynamics of the GCP with random-field disorder is ultraslow. It is governed by the Sinai random walk of domain walls between the two inactive states. This leads to a logarithmic time decay of the densities of both domain walls and active sites. Note that the Sinai coarsening dynamics has been studied in detail in the equilibrium random-field Ising chain [31] where it applies to a transient time regime before the domains reach the Imry-Ma equilibrium size.

Although our explicit results are for one dimension, we expect our main conclusion to hold in higher dimensions, too. In the interior of a uniform domain of an absorbing state, new active sites (and new domain walls) cannot arise in any dimension. Moreover, the Imry-Ma mechanism by which the random fields destroy an equilibrium transition becomes less effective in higher dimensions. Indeed, Pigolotti and Cencini [32] report spontaneous symmetry breaking in a model of two species competing in a two-dimensional landscape with local habitat preferences. To further study this question, we plan to introduce random fields into our simulations of the two-dimensional GCP [33].

Finally, we turn to experiments. Although clear-cut realizations of absorbing state transitions were lacking for a long time [34], beautiful examples were recently found in turbulent liquid crystals [35], driven suspensions [36,37],

and superconducting vortices [38]. As they are far from equilibrium, biological systems are promising candidates for observing nonequilibrium transitions. A transition in the DP2 universality class (as studied here) occurs in a model of competing bacteria strains [39] which accurately describes experiments in colony biofilms [40]. Random-field disorder could be realized in such experiments by environments that locally favor one strain over the other.

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