Effects of excluded volume interaction on diffusion-reaction processes in crowded environments

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In nonequilibrium phase transitions of reaction-diffusion processes, the irrelevance of excluded volume interaction for the critical properties generally has been accepted due to the rare probability of multiple occupancy at criticality. Moreover, this belief is common sense in scale-free (SF) networks, which correspond to infinite dimensional irregular structures. However, the conventional belief is not satisfied in crowded environments in which the total number of particles is preserved in time. In this paper, we show, by investigating a typical process for epidemic spreading in crowded environments, that excluded volume interaction indeed changes critical behaviors in one dimension and surprisingly even mean-field behaviors in SF networks.

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I. INTRODUCTION

Crowded environments, including a large number of particles, are ubiquitous in nature and are of practical importance in a wide range of phenomena in physics, chemistry, and biology [1–5]. Examples for such environments range from cells and narrow channels, such as zeolites [2] and nanosize pores [3] to cities with large populations. In such environments, the high density of reactants makes excluded volume or hard-core (HC) interaction important as in biochemical reactions in cells [4] and driven diffusive systems in one dimension [5].

There is much evidence for the important effects of the HC interaction in crowded environments. Nevertheless, the HC interaction generally has been believed to be irrelevant in reaction-diffusion systems undergoing continuous transitions. The reason is that the density of particles is so low near criticality that multiple occupancy on a site is unlikely. This conventional belief has been confirmed by the recent success of bosonic field theory on reaction-diffusion systems [6]. Only few exceptions have been reported so far [7]. However, when the number of particles is conserved, crowded environments do not satisfy the conventional belief, and thus, there are no physical reasons that bosonic and HC particle systems share the same critical behaviors in crowded environments. In this context, it is particularly important to explore the effect of the HC interaction on the universality of nonequilibrium phase transitions in crowded environments.

In this paper, we explore the effect of the HC interaction on the critical behaviors of nonequilibrium absorbing phase transitions (APTs) under particle conservation. APTs are a prototype of nonequilibrium phase transitions widespread in physics, chemistry, biology, and sociology [8]. The universality of APTs is classified according to various physical properties, such as conservation laws and symmetries [8]. The directed percolation (DP) class is the most well-established and most of studied models belong to the DP class [8].

When activity spreads upon contact with nonorder fields via diffusion, the conservation of the total number of particles plays an important role and leads to various non-DP critical behaviors depending on the diffusion rate of the nonorder fields [9–14]. Various reaction-diffusion processes coupled to nonorder fields have been studied extensively by means of bosonic field theory and numerical simulations in connection with self-organized criticality, autocatalytic chemical reactions, epidemic spreading in lattices [9–13], and even in scale-free (SF) networks [14]. In such processes, reactions take place in crowded environments even at criticality due to the conservation, which does not satisfy the conventional belief for systems without the conservation. This kind of process can be encapsulated in the so-called diffusive epidemic process (DEP) of two species defined by two reactions; spontaneous recovery $A \to B$ and infection upon contact $A + B \to A + A$ [12]. Hence, the DEP is a good test bench for the effect of the HC interaction on APTs in the crowded environment.

We study the driven DEP of HC particles in SF networks with degree distribution $P(k) \sim k^{-\gamma}$ and one dimension ($d = 1$) because the bias of motion is a useful and important probe for testing the robustness of theoretical results for bosonic systems. In SF networks, topological bias to hub nodes are naturally induced by heterogeneous degree distributions [15]. The topological bias drives particles to hub nodes, and then, compact clusters are formed around the hub nodes due to the HC interaction. Since motion is impossible in such clusters, DEP dynamics is reduced to the contact process (CP), which is a typical model of the DP class. In SF networks, the CP undergoes APTs at a finite infection rate for $\gamma > 2$. On the other hand, in the bosonic counterpart, particles freely diffuse and pile up on hub nodes. This free diffusion in the bosonic counterpart always makes the system, in endemic states, $2 < \gamma \leqslant 3$ [14]. As a result, the interplay of topological bias and the HC interaction completely changes the critical behaviors of the DEP in SF networks. We analytically show the formation of a compact cluster for nodes with a degree larger than a certain crossover value, and resultant critical behavior is just that of the CP. We also numerically confirm the analytical predictions.

The results in SF networks suggest that the interplay of bias and the HC interaction is relevant to critical behavior in crowded environments. To examine the effect of the interplay in regular lattices, we investigate the driven DEP in $d = 1$. We apply an external field driving all particles in the same direction. As in SF networks, we find that the interplay indeed

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affects the critical behavior, and the resultant critical behavior belongs to the DP class, which is different from the non-DP behavior of the bosonic counterpart. Unlike SF networks, big compact clusters around special sites cannot form in $d = 1$. Instead, the interplay causes nontrivial blocking effects and causes a direct infection between the nearest neighbors, which are dominant over the spreading by motion.

Our results suggest that the conventional belief for the HC interaction no longer holds in crowded environments and the interplay of bias and the HC interaction can change the universality of APTs. In particular, in SF networks, it is naturally believed that the HC interaction is irrelevant to critical phenomena because networks correspond to infinite dimensional irregular structures. It was shown that reaction-diffusion processes of single species were well described by bosonic mean-field (MF) theory in SF networks [16] and regular lattices [17]. However, our results in networks suggest that the HC interaction plays an important role in multispecies systems and provide physical phenomena that cannot be described by a bosonic MF theory.

This paper is organized as follows. We introduce the DEP in Sec. II and present a MF analysis and simulation results in SF networks in Sec. III. We present simulation results in one dimension in Sec. IV. We finally summarize our findings with some discussions in Sec. V.

II. DEP OF HC PARTICLES

DEP of HC particles (HC DEP) consists of two species corresponding to infected (A) and healthy (B) individuals. Both species diffuse with rates D_A and D_B. Hoppings to occupied sites are forbidden by the HC interaction. An A particle spontaneously recovers with rate λ; $A \to B$. With the unit rate, an A particle selects one of the nearest neighbors. If the selected site is occupied by B, then B is infected; $A + B \to A + A$. The reactions conserve the total density of particles $\rho = a + b$, where $a(b)$ is the density of $A(B)$ particles. The HC DEP of $\rho = 1$ is a CP, the prototype for the DP class [8]. For a fixed λ, the DEP undergoes APTs at a critical density ρ_c from active states ($a > 0$) into absorbing ones ($a = 0$). In APTs, the order parameter a, spatial correlation length ξ, and relaxation time τ scale with the distance from criticality Δ as $a \sim \Delta^{\beta}$, $\xi \sim |\Delta|^{-\nu_\perp}$ and $\tau = |\Delta|^{-\nu_\parallel}$ for $\Delta \to 0$ [8].

For the normal diffusion, universality of the DEP is known to depend on the value of $\mu = (D_A - D_B)/D_A$ in regular lattices [11,12]. For $d < d_c(= 4)$, the DEP belongs to the conserved DP (C-DP) class for $\mu = 1$ in $d = 2$ [11]. For $\mu < 1$, bosonic field theory predicted two distinct critical behaviors, which were different from both DP and C-DP classes for $0 \leqslant \mu < 1$ and discontinuous transition for $\mu < 0$ [12]. However, numerical studies yielded conflicting estimates of scaling exponents with the theoretical predictions except for z, so the universality is not firmly established yet [13]. For $d > d_c$, the exponents take standard MF values; $\beta = \nu_\parallel = 1$ and $\nu_\perp = 1/2$ [12]. In SF networks with $P(k) \sim k^{-\gamma}$, the bosonic DEP exhibits the standard MF behaviors for $\gamma > 3$, but no transitions, i.e., $\rho_c = 0$, for $2 < \gamma \leqslant 3$ due to the strong heterogeneity of degree distributions, i.e., hub structures [14].

III. DEP OF HC PARTICLES IN SF NETWORKS

First, we report the results for $D_A = D_B$ in SF networks. The generalization for the $D_A \neq D_B$ case is straightforward and gives the same results. By developing a MF theory for HC DEP in SF networks, we will show that the HC DEP undergoes APTs for $\gamma > 2$ unlike the bosonic counterpart and shares the same MF critical behavior with the CP in the networks [18,19]. Networks consist of nodes connected by links. The degree k of a node is defined as the number of links connected to other nodes. As usual, we employ the noiseless degree-dependent MF theory in which one neglects the fluctuations in densities for nodes with the same k [20]. In the MF theory, one considers the average density of A particles a_k and b_k of B's on a node with degree k. Total density ρ_k on a node with k is given as $\rho_k = a_k + b_k$. Then, the density of A particles is obtained as $a(t) = \sum_k a_k(t)P(k)$ and is obtained similarly for $b(t)$. We obtain the rate equation of a_k as

$$\partial_t a_k = D\left[\nabla_A^2\right] - \lambda a_k + b_k k \sum_{k'} T_{kk'} a_{k'} P(k'|k), \tag{1}$$

and we obtain the rate equation similarly for $b_k(t)$. We define $[\nabla_A^2] \equiv k(1-\rho_k)\sum_{k'} T_{kk'} a_{k'} P(k'|k) - a_k k \sum_{k'} T_{k'k}(1-\rho_{k'})P(k'|k)$ as the diffusion terms of A's under the HC constraint. $T_{kk'}$ is the hopping probability from a node with k' to one with k, and $P(k'|k)$ is the conditional probability of a node with k being connected to one with k'. With $T_{kk'} = 1/k'$ and $P(k'|k) = k'P(k')/\langle k\rangle$ for uncorrelated networks, one obtains

$$\partial_t a_k = -\lambda a_k - \widetilde{D} a_k + [b_k + D(1-\rho_k)]\widetilde{k} a, \tag{2}$$

$$\partial_t b_k = \lambda a_k - \widetilde{D} b_k - b_k \widetilde{k} a + D(1-\rho_k)\widetilde{k} b, \tag{3}$$

where $\widetilde{k} = k/\langle k\rangle$ and $\widetilde{D} = D(1 - \langle k\rho_k\rangle/\langle k\rangle)$. In the steady states, ρ_k provides information for the distribution of particles. Summing up Eqs. (2) and (3), we obtain ρ_k in the steady states as

$$\rho_k = \frac{k/k_c}{1 - \langle k\rho_k\rangle/\langle k\rangle + k/k_c}. \tag{4}$$

$k_c = \langle k\rangle/\rho$ is the crossover k above which $\rho_k \approx 1$. As expected, the HC constraint bounds $\rho_k \leqslant 1$, and $\langle k\rho_k\rangle \leqslant \langle k\rangle$. It results in the reduced diffusion rate $\widetilde{D} < D$. More importantly, compact clusters of $\rho_k = 1$ are formed around nodes with $k \gg k_c$. In such compact clusters, diffusion is impossible so that only the reactions determine the critical behavior of the clusters. For $k \ll k_c$, however, particles mainly diffuse due to $\rho \ll 1$, so the contributions of the nodes with $k \ll k_c$ to the critical behavior of the whole system should be negligible. As a result, HC DEP should exhibit the same critical behavior as the CP in SF networks [18,19]. It physically means that the topologically induced natural bias drives particles to hubs, and then, compact clusters are formed around the hubs by the HC interaction. These naturally made compact clusters indeed change the critical behavior of the DEP in SF networks.

In order to determine the critical behavior, we obtain the self-consistent equation for the density a in the steady state. By setting $\partial_t a_k = 0$ in Eq. (2) and defining $\widetilde{\lambda} = 1/(\lambda + \widetilde{D})$, one obtains, from $a = \sum_k a_k P(k)$,

$$a = \frac{\widetilde{\lambda} a}{\langle k\rangle} \int_1^\infty \frac{[(1-D)\rho_k + D]k P(k) dk}{1 + \widetilde{\lambda} a k/\langle k\rangle}. \tag{5}$$

For $\rho_k = 1$, Eq. (5) is the density of the CP [18]. With a fixed ρ, we calculate λ_c and β. The integral of the first term I_1 in Eq. (5) is $I_1 \simeq (1-D)\langle k\rho_k\rangle$. Integrating the second term, one obtains $\frac{a\kappa_c - I_1}{(\gamma-1)D} = \frac{1}{\gamma-2} - \frac{1}{\kappa_c(\gamma-3)} + \frac{(\gamma-4)\kappa_c^{2-\gamma}}{(\gamma-2)(\gamma-3)}$ with $\kappa_c = \langle k\rangle/\widetilde{\lambda}a$. Comparing the second term with the third, one obtains $\lambda_c = \frac{\langle k\rho_k\rangle}{\langle k\rangle}$, and $a \sim (\lambda_c - \lambda)^\beta$ with $\beta = 1$ for $\gamma > 3$ and $1/(\gamma-2)$ for $2 < \gamma \leqslant 3$. We note $0 < \lambda_c \leqslant 1$ even for $\gamma \leqslant 3$ because of $\rho_k \leqslant 1$ for all k unlike the bosonic DEP with $\lambda_c = \infty$ for $\gamma \leqslant 3$.

At λ_c, $a(t)$ decays as $t^{-\beta/\nu_\parallel}$. $\nu_\parallel$ can be obtained from the rate equation of $a(t)$. From Eq. (2), we get $da/dt = (\lambda_c - \lambda)a(t) - a(t)\langle ka_k(t)\rangle/\langle k\rangle$. From the quasistatic approximation [21] or setting $\partial_t a_k = 0$ in Eq. (2), one obtains the integral form of $\langle ka_k(t)\rangle$. Finally, with the same manner employed to evaluate the integral in Eq. (5), we obtain

$$da(t)/dt = (\lambda_c - \lambda)a - ca^2 - ga^{\gamma-1}, \tag{6}$$

where c and g are nondiverging coefficients. Equation (6) is just the MF equation for the CP proposed in Ref. [19]. At λ_c, one readily finds $\beta/\nu_\parallel = 1$ for $\gamma > 3$ and $1/(\gamma-2)$ for $2 < \gamma \leqslant 3$, so $\nu_\parallel = 1$ for $\gamma > 2$. In the steady state, Eq. (6) also gave the same β we already found. For $D_A \neq D_B$, one easily obtains da/dt by replacing D with D_A in Eq. (2), which gives Eq. (6) with different coefficients. ρ_k only changes the dependence of λ_c on diffusion rates and ρ, for instance, $\lambda_c = \rho$ for $D_B = 0$. Hence, the MF critical behaviors for $D_A \neq D_B$ are identical to those for the $D_A = D_B$ case. In conclusion, the HC DEP undergoes APTs for $\gamma > 2$ unlike the bosonic counterpart and shares the same MF critical behavior with the CP in SF networks.

To confirm the analytic results, we perform Monte Carlo (MC) simulations in deterministic annealed SF networks [22]. We used networks with $\gamma = 2.5$, $k_{\min} = 3$, $\langle k\rangle = 7.3$, and size N up to 10^6. The density ρ is set as $\rho = 0.8$. For $D_A = D_B = 1$, we first measure ρ_k and $\langle k\rho_k\rangle$ in the steady states starting from random distributions and obtain $\langle k\rho_k\rangle = 6.4$. We can see the simulation result coincides with ρ_k from Eq. (4) as shown in Fig. 1(a). For homogeneous initial conditions of $a(0) = \rho$, we measure $a(t)$ up to $t = 200$ time steps by averaging over 100 independent runs. We first decide the critical value p_c of healing probability $p = \lambda/(1+\lambda)$ for a fixed ρ. We examine the effective exponent defined as

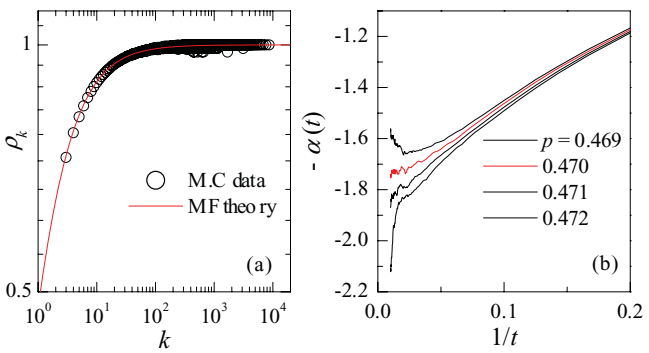

FIG. 1. (Color online) The plot of (a) ρ_k and (b) $\alpha(t)$ for $\gamma = 2.5$, $\rho = 0.8$, and $N = 10^6$. (a) Symbols and a solid line correspond to simulation data and ρ_k of Eq. (4). (b) Each line from top to bottom corresponds to the line of p indicated in the legend.

$\alpha(t) = -\ln[\rho(mt)/\rho(t)]/\ln m$, which exhibits upward (downward) curvature in the active (absorbing) phase. At p_c, $a(t)$ decays as $t^{-\beta/\nu_\parallel}$ in the limit $N \to \infty$, so $\alpha(t)$ approaches $\beta/\nu_\parallel$. In Fig. 1(b), we plot $\alpha(t)$ using $m = 2$. We estimate $p_c = 0.470(1)$ and $\beta/\nu_\parallel = 1.74(1)$. Our estimate is still smaller than the MF value $\beta/\nu_\parallel = 1/(\gamma-2) = 2$ for $\gamma = 2.5$. However, we confirm α increases with N, $\alpha = 1.4(1)$ and $1.6(1)$ for $N = 10^4$ and 10^5, which reflects the strong finite-size effects of the networks. Hence, we expect $\beta/\nu_\parallel = 2$ in the limit $N \to \infty$. We also confirm similar behavior for $D_A \neq D_B$ and, thus, confirm the MF predictions.

IV. DRIVEN DEP IN ONE DIMENSION

Next, we report simulation results in $d = 1$ in the presence of external driving fields to show the effects of the HC interaction in crowded environments. All particles are driven in the same direction on a ring of size L. Here, $D_A(D_B)$ is defined as the hopping rate of $A(B)$ particles. We perform simulations with two types of initial conditions, a homogeneous condition $a(0) = \rho$ and a localized one $a(0) = 1/L$.

For the homogeneous condition, $a(t,L)$ satisfies $a(t,L) = t^{-\alpha}F(t/L^z)$ with $\alpha\nu_\parallel = \beta$ at criticality, which allows the finite-size scaling (FSS) analysis [8]. We set $\rho = 0.2$ and obtain simulation results for $\mu = 1, 1/2, 0, -1$. From FSS analysis using L up to 8×10^3, we find $z = 1$ and $\alpha = \beta/\nu_\perp \approx 1/2$ for $\mu \neq 0$. Since the finite-size effects start to come into play at the relaxation time $\tau \sim L$ for $z = 1$, FSS analysis for systems of $z > 1$ is impossible due to strong finite-size effects. However, for $\mu = 0$, we observe that the finite-size effects are not strong enough to hinder FSS analysis. We measure $p_c = 0.034\,65(7)$ and estimate $\alpha = 0.173(5)$, $\beta/\nu_\perp = 0.25(1)$, and $z = 1.60(5)$ (see Fig. 2). These estimates are comparable to DP values; $\beta/\nu_\parallel = 0.16$, $\beta/\nu_\perp = 0.252$, and $z = 1.58$ [8].

For the localized condition, one obtains the dynamical scaling behavior of activity spread, starting from a single A particle in the sea of randomly distributed B's. We measure the number of A particles $N_A(t)$ averaged over all samples,

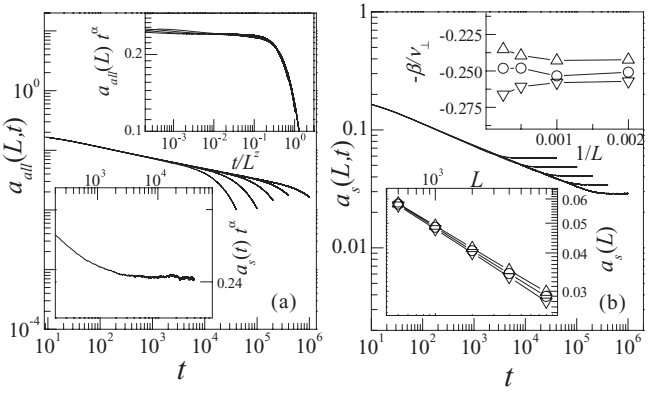

FIG. 2. (a) $a_{\rm all}$ is a averaged over all runs. The upper inset shows the data collapsing of $a_{\rm all}$ with $\alpha = 0.172$, $z = 1.6$. The lower inset shows the scaling plot of $L = 8000$ data with $\alpha = 0.172$. (b) a_s is a averaged over surviving runs. The upper inset shows the successive slope of the steady state $a_s(L)$ in the lower inset. In the insets, line symbols from top to bottom correspond to $p = 0.034\,57, 0.034\,65(p_c), 0.034\,73$.

TABLE I. Critical exponents obtained from localized initial conditions. DP, C-DP, and anisotropic C-DP (A-C-DP) values are taken from Refs. [8] and [10]. Figures in parentheses denote statistical uncertainties.

D_A	D_B	μ	p_c	η	δ	α	$2/z$
1.0	0.0	1	0.094 55(2)	0.14(1)	0.34(1)	0.17(1)	1.30(2)
1.0	0.5	1/2	0.067 08(8)	0.09(3)	0.39(2)	0.17(2)	1.29(2)
1.0	1.0	0	0.034 50(3)	0.36(2)	0.15(2)	0.16(1)	1.32(3)
0.5	1.0	−1	0.1006(1)	0.05(5)	0.45(5)	0.08(3)	1.1(1)
DP				0.3137	0.159	0.159	1.265
C-DP				0.35	0.170	0.141	1.436
A-C-DP				0	1/2	1/2	2

the survival probability $P_s(t)$, and the mean squared spreading distance $R^2(t)$ averaged over surviving runs, which scale as $N_A \sim t^{\eta}$, $P_s \sim t^{-\delta}$, $R^2 \sim t^{2/z}$ at criticality [8]. In addition, we measure $a(t)$ inside the spreading region of size $R(t)$ defined as $a(t) = \langle N_A(t)/R(t)\rangle$, which should scale as $t^{-\alpha}$ at p_c. We measure observables up to time $t_{\max}(\ll L)$. Since A particles cannot propagate ballistically from the starting point, we, in principle, remove the finite-size effects [8]. We set $(t_{\max}, L) = (10^5, 10^6)$ for $\mu < 1$ and $(10^6, 10^7)$ for $\mu = 1$. We summarize our results in Table I.

For $\mu \geqslant 0$, η and δ are not universal, but the sum $\eta + \delta$ agrees with the DP value. This dynamical property has been found in DP models with infinitely many absorbing states, such as pair contact process [8]. In addition, α and $2/z$ are universal and agree with the values of the DP class. For $0 \leqslant \mu < 1$, bosonic field theory predicts $z = 2$, $\nu_\perp = 2/d$ [12]. For $\mu = 1$, our results are different from those of the C-DP and A-C-DP classes [10]. As a result, we are convinced that driven HC DEP in $d = 1$ belongs to the DP class for $\mu \geqslant 0$. For $\mu = -1$, we expect $2/z = 1$ for $t \to \infty$. Our estimate of α for $\mu = -1$ also agrees with $\beta/\nu_\parallel = 0.07(2)$ for the isotropic DEP with $\mu > 0$ [13]. Hence, the transition for $\mu < 0$ is continuous and belongs to the class of $0 < \mu < 1$. As a result, the interplay of the HC interaction and bias has no effect on the critical behavior of the DEP for $\mu < 0$. It can be understood from the fact that B particles move faster than A's, so the relative motion of A's plays a crucial role in infection as in isotropic cases.

V. SUMMARY

To summarize, we showed that the interplay of the HC interaction and bias caused nontrivial blocking effects and changed the universality of APTs in crowded environments. The blocking effects appear as compact clusters in SF networks and fragmented small clusters in $d = 1$. In such environments, the direct infection between the nearest neighbors dominates the diffusion-mediated spreading of activity, and thus, the critical behavior is determined by the reactions themselves. The DEP is generalized by the reaction scheme $mA + kB \to (m + k)A$, $nA \to (n - \ell)A + \ell B$ [23]. Our results suggest that the generalized DEP of the HC particles with the driving field should exhibit the corresponding critical behavior characterized by reactions in compact clusters.

In addition, our results are of practical importance for disease spreading in the systems with SF network structures. Some diseases, such as cholera, spread by contagion between individuals inside local regions, which would naturally be described by mobile HC particles. In contrast, others, such as severe acute respiratory syndrome, which spread between nations or cities are intrinsically bosonic [16]. Since populations in such networks are fixed, at least on average, our results suggest that the spreading phenomena in crowded environments could completely be different depending on the scale of networks in which diseases spread.

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