Disorder and nonconservation in a driven diffusive system

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We consider a disordered asymmetric exclusion process in which randomly chosen sites do not conserve particle number. The model is motivated by features of many interacting molecular motors such as RNA polymerases. We solve the steady state exactly in the two limits of infinite and vanishing nonconserving rates. The first limit is used as an approximation to large but finite rates and allows the study of Griffiths singularities in a nonequilibrium steady state despite the absence of any transition in the pure model. The disorder is also shown to induce a stretched exponential decay of system density with stretching exponent $\phi=2/5$.

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I. INTRODUCTION

Driven diffusive systems serve as simple models for collective phenomena ranging from traffic flow to molecular motors. Moreover, they provide tractable examples of systems far from thermal equilibrium. Studies of one-dimensional driven diffusive systems have shown that many interesting phenomena, which are typically not observed in one-dimensional systems in thermal equilibrium, exist. Prominent examples are boundary induced phase transitions and spontaneous symmetry breaking; for reviews, see, e.g., [1–3].

Most studies have considered systems in which the dynamics are the same everywhere in the system or systems where the dynamics are modified only at the boundaries. However, when trying to relate these systems to many interacting molecular motors, the effects of nonconservation and disorder (i.e., spatial heterogeneity in the dynamics) cannot be ignored in many cases.

Indeed there have been some studies on the effects of disorder on driven diffusive systems. For example, the effect of assigning a disordered quenched rate to each particle has been studied in [4–10] on a ring geometry. Exact solutions show that at high enough densities a macroscopic number of particles jam behind the slowest particle in the system. The phase transition between the jammed and nonjammed phase is similar to a Bose-Einstein condensation. Work has also been done on an asymmetric exclusion process on a ring where the quenched hopping rates between neighboring sites are drawn at random [8,9,11]. For molecular motors moving along a disordered substrate this seems to be the relevant scenario [12,13]. It was argued, based on numerics and mean-field solutions, that at high densities the system phase separates into a region of high density coexisting with a low density region. Finally, the combined effect of random hopping rates and open boundary conditions was considered in [14,15]. In [14] it was argued using numerics that the location of phase transition lines may be sample dependent. In [15] mean-field arguments and numerics indicate the existence of shifts in phase boundaries which, by analogy with equilibrium systems, are expected to be accompanied by emergent Griffiths regions. A review of the effects of disorder in exclusion models has been given in [16].

In this paper we consider another type of disorder. We study an asymmetric exclusion process (ASEP) where *nonconserving* sites are chosen at random along the lattice. At these sites particles may attach and detach with specified rates which may also be drawn at random. Thus there are two components to the disorder. A feature of this disorder is that it allows a detailed account of the way in which Griffiths singularities can arise in nonequilibrium steady states. In equilibrium the mechanism leading to Griffiths singularities is well understood: the disorder, e.g., dilution, breaks the system into pure regions and large pure regions may give rise to the exponentially suppressed Griffiths singularities. In the present case, in the limit of high attachment and detachment rates, the nonconserving sites break the system into driven conserving domains.

Nonconservation of particles in driven systems without disorder has previously been considered in the context of molecular motors. The idea is that molecular motors move in a preferred direction along a filament and are able to attach to and detach from the filament. In the work so far all sites are nonconserving [17–20]. The motivation for the model we study here comes from the fact that some molecular motors attach and detach only at certain sites.

More specifically, we give a very simplified description of many interacting RNA polymerase (RNAp) motors acting on a prokaryotic DNA *in vitro*. Prokaryotic RNA polymerase can initiate without regulatory proteins; namely, RNAp left in a solution with DNA can produce RNA even if the specific proteins which regulate its action (for example, by enhancing the initiation rates) are not present. RNAp motors can enter and leave the DNA in order to transcribe RNA molecules at specific sites, referred to as promoter and termination sites, respectively.

We consider a lattice model in which periodic (open) boundary conditions correspond to closed (open) prokaryotic DNA. In the lattice model binding of an RNAp motor to a promoter corresponds to a particle entering the system. The unbinding at the termination site corresponds to a particle leaving the system. In the absence of regulatory proteins the rate of entering the DNA depends on the details of the promoter sites. In such systems the RNAp motors do not usually move from one gene to another. In the lattice model this would correspond to particles not moving from one stretch of

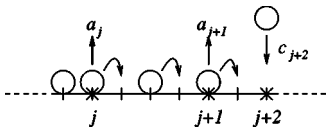

FIG. 1. Allowed dynamics of the disordered model. The disorder sites are marked with an asterisk. See text for details.

conserving sites to a neighboring one, i.e., the limit in which the detachment rates are large at the nonconserving sites. We comment that in principle RNAp motors may move in different directions along the DNA when transcribing different messenger RNAs, corresponding to particles moving in different directions along different conserving stretches. However, in the limit when the detachment rate is large this will not influence most of the results described in the paper. Of course, our assumption of randomly distributed lengths of genes (or conserving segments) is not expected to hold. Moreover, the motion of RNAp might depend on the sequence on which it is moving and we have neglected the binding mechanism to the promoter site. The latter is a subject of much debate [21]. However, the model provides a starting point for analyzing more realistic situations similar in spirit to Ref. [22] which has motivated the introduction of the asymmetric exclusion model (see also [23]).

The paper is organized as follows. In Sec. II, we define the model and discuss two limits that are exactly soluble. In Sec. III we show that the disorder induces Griffiths-like singularities as the rates for entering and leaving the lattice are changed. More significantly, it is shown in Sec. IV that the presence of the nonconserving sites leads to anomalous relaxation of the system toward the steady state. Specifically, we argue that decay of measurable quantities decay as a function of time t as a stretched exponential $\exp(-ct^{\phi})$, where c is a nonuniversal constant and $\phi=2/5$. The results are verified numerically. We conclude in Sec. V.

II. MODEL

The model we consider is a disordered generalization of the ASEP. The pure ASEP is defined on a one-dimensional lattice containing L sites and with periodic boundaries. The lattice is occupied by particles subject to an exclusion interaction, which prohibits multiple occupancy of any site. These particles hop with rate 1 to the nearest neighbor site to the right, provided it is empty, and so the total particle number N is conserved. We introduce nonconservation into this model by allowing, at certain sites (which we will call "disorder" sites), processes that do not conserve the total particle number N. Hence each site l ($l=1,\ldots,L$) in the pure model remains a pure site with probability p, or becomes a disorder site with probability $(1-p)$. Now, at the disorder sites, labeled by $j=1,\ldots,P$, particles attach with rate c_j or detach with rate a_j. In general, we wish to consider heterogeneous rates for the nonconserving processes. The dynamics are illustrated in Fig. 1.

To study the model we first consider limits that can be solved exactly. Later, using numerics, we argue that the results are generic. Exact solubility arises when the steady state densities at the disorder sites are determined solely by the attachment and detachment processes. In these cases, since the system is composed of conserving domains of the chain in contact with disorder sites at the boundaries of each domain, the steady state can be written as a product of boundary driven ASEPs in which the densities of the boundary reservoirs are given by the disorder site densities ρ_j.

Before turning to the disordered ASEP under consideration we recap some facts, which will be useful later, about the boundary driven ASEP. For the boundary driven ASEP, in which particles are injected at the left-hand boundary site with rate α (provided it is vacant) and removed from the right-hand boundary site with rate β, exact steady state weights for particle configurations can be obtained using a matrix product ansatz [24]. In this ansatz, particle configurations are represented as a product of matrices $X_1\cdots X_L$ where $X_l=D$ (E) if site l is occupied (vacant). The steady state weight of a configuration is given by $\langle\alpha|X_1\cdots X_L|\beta\rangle$ provided the matrices D and E and the vectors $\langle\alpha|$ and $|\beta\rangle$ satisfy the relations

$$DE = D + E \equiv C, \quad \alpha\langle\alpha|E = \langle\alpha|, \text{ and } \beta D|\beta\rangle = |\beta\rangle. \quad (1)$$

From these relations exact expressions for the normalization $\langle\alpha|C^L|\beta\rangle$ can be derived which show that the model undergoes a second order phase transition: when both α and $\beta \geqslant 1/2$ the system is in a maximum current phase, otherwise it is in one of two low current phases. The phase transition between the low current phases is first order. The rates α and $1-\beta$ represent the densities of particles in reservoirs connected to the boundary sites.

Next, we use the known results for the boundary driven ASEP to study the disordered case. As stated above, there are limits where the steady state weight of the disordered model factorizes into a product over boundary driven ASEP weights. The two exactly soluble limits are as follows.

$c_j, a_j \to \infty$, with c_j/a_j fixed. Specifically we let $c_j, a_j \to \infty$ in a system of finite size then calculate the steady state $(t\to\infty)$. In this limit, each disorder site j acquires a density ρ_j determined solely by c_j and a_j which obeys the equation of motion,

$$\frac{\partial \rho_j}{\partial t} = (1-\rho_j) + a_j\rho_j. \quad (2)$$

Therefore in the steady state

$$\rho_j = \frac{c_j}{c_j + a_j}. \quad (3)$$

If we define n_j to be the number of sites between disorder sites j and $j+1$ (i.e., the length of the jth conserving domain), then the normalization $Z_L(\{n_j\})$ (which is the sum over the steady state weights of all particle configurations on sites excluding the disorder sites), for a given configuration of the disorder sites $\{n_j\}=n_1,\ldots,n_P$, factorizes into a product over normalizations for the boundary driven ASEP:

$$Z_L(\{n_j\}) = \prod_{j=1}^{P} \langle \rho_j | C^{n_j} | 1-\rho_{j+1} \rangle \tag{4}$$

where $\rho_{P+1}=\rho_1$.

$c_j, a_j \to 0$, *with* c_j/a_j *fixed*. Specifically we let $c_j, a_j \to 0$ in a system of finite size and then calculate the steady state. In this limit the time between each attachment/detachment event tends to infinity. Therefore, after each event the system reaches a homogeneous steady state of the pure ASEP with periodic boundaries. Thus the system density $\rho = N/L$ satisfies the equation of motion

$$\frac{\partial \rho}{\partial t} = \left[\sum_{j=1}^{P} c_j \right] (1-\rho) + \left[\sum_{j=1}^{P} a_j \right] \rho. \tag{5}$$

Therefore in the steady state the system density is given by

$$\rho = \frac{\sum_{j=1}^{P} c_j}{\sum_{j=1}^{P} (c_j + a_j)}. \tag{6}$$

Because the steady state is homogeneous, all sites, including disorder sites, have the same steady state density ρ. Moreover, the steady state factorizes and there are no correlations between sites.

One can also obtain this result by considering the attachment and detachment as perturbations that connect different steady states (labeled by particle number) of the pure system. The weights of these steady states satisfy a balance condition which yields Eq. (6) and the factorization property [25].

Therefore, one can write the normalization in a form similar to Eq. (4):

$$Z_L(\{n_j\}) = \prod_{j=1}^{P} \langle \rho | C^{n_j} | 1-\rho \rangle. \tag{7}$$

This is because in this case D and E are given by the scalars $1/(1-\rho)$ and $1/\rho$.

Thus we see that in the two limits considered the nonconservation factorizes the steady state into a product of conserving domains. In the following we will use the factorized form (4) with ρ_j given by Eq. (3) as an approximation for the case where c_j, a_j are large but finite, which is relevant for the model of molecular motors. In this case, as one expects for RNAp, particles enter the lattice at specific sites and unbind at the next disordered site. This approximation has a mean-field character, in the sense that correlations are factorized about the disorder sites; however, all correlations within conserving domains are retained.

III. GRIFFITHS SINGULARITIES

We can exploit known properties of the normalization of the boundary driven ASEP to demonstrate the existence of Griffiths-type singularities in the disordered ASEP [26,27]. As an illustrative example, we consider binary disorder at disorder sites, such that

$$\rho_j = \frac{c_j}{a_j + c_j} = \begin{cases} u \text{ with probability } q, \\ v \text{ with probability } 1-q. \end{cases}$$

This is the simplest choice of disorder for which Griffiths singularities occur. Generalizations to more complicated situations are straightforward.

Using Eq. (4) the steady state normalization satisfies

$$\ln Z_L = \sum_{j=1}^{P} W_{n_j}(\rho_j, 1-\rho_{j+1}), \tag{8}$$

where $W_n(\rho_j, 1-\rho_{j+1}) = \ln \langle \rho_j | C^{n_j} | 1-\rho_{j+1} \rangle$. In order to perform the disorder average, we write Eq. (8) as a sum over domain sizes; hence

$$\ln Z_L = \sum_{n=0}^{\infty} [\nu_{u,1-v}(n) W_n(u,1-v) + \nu_{u,1-u}(n) W_n(u,1-u) + \nu_{v,1-v}(n) W_n(v,1-v) + \nu_{v,1-u}(n) W_n(v,1-u)] \tag{9}$$

where $\nu_{\alpha,\beta}(n)$ is the number of conserving domains of size n bounded by disorder sites at densities α and $1-\beta$.

We can average over the configurations of the $\nu_{\alpha,\beta}(n)$ by calculating the expectation values $\langle \nu_{\alpha,\beta}(n) \rangle$ in the thermodynamic limit (the angular brackets denote a disorder average). This is achieved by observing that $\lim_{L\to\infty} L^{-1} \langle \nu_{\alpha,\beta}(n) \rangle$ is just the probability that a site is part of an n-site conserving domain bounded by disorder sites with densities α and $1-\beta$; hence

$$\lim_{L\to\infty} L^{-1} \langle \ln Z_L \rangle = (1-p)^2 \sum_{n=0}^{\infty} p^n \{ q^2 W_n(u,1-u) + q(1-q) \times [W_n(u,1-v) + W_n(v,1-u)] + (1-q)^2 W_n(v,1-v) \}. \tag{10}$$

The form of Eq. (10) is typical of systems that exhibit Griffiths singularities. In equilibrium, these singularities are usually inferred from the properties of the Yang-Lee zeros of the partition function—we can use the known properties of the Yang-Lee zeros of the analogous quantity, the normalization [28], to show how Griffiths singularities arise in the disordered nonequilibrium model: For fixed $u \geqslant 1/2$ say, in the complex v plane and for n arbitrarily large, the zeros of $\langle u | C^n | 1-v \rangle$ accumulate arbitrarily close to the point $v=1/2$ on the real axis. Therefore there exists a singularity in $W_n(u,1-v)$ arbitrarily close to the point $v=1/2$ which is exponentially suppressed (by a factor p^n). Thus, such a Griffiths-type singularity follows whenever u and v are such that at least one of the $W_n(\alpha,\beta)$ in Eq. (10) lies on the phase boundary of the ASEP, i.e., whenever u and/or $v=1/2$.

One can go further and consider disorder in the c_j's and a_j's explicitly. For instance, if both c_j and a_j are drawn from binary distributions, then the densities at the disorder sites can assume one of four possible values, each with a different probability in general. However, Griffiths singularities still arise whenever any one of these values for the density is $1/2$, as before. It is also straightforward to use standard arguments

from the study of dilute systems to show that the correlation length remains finite at the Griffiths singularity, as is the case in equilibrium systems.

Note that the pure limit, $p=1$, of our system, corresponds to the ASEP on a ring. This is a system that does not exhibit any phase transition. Thus Griffiths singularities emerge in the disordered system despite the absence of a transition in the pure model. This is different from the usual behavior in equilibrium. Although in this case particles are conserved in the pure system and the introduction of disorder breaks this conservation law, one could also consider an open boundary driven ASEP as the pure model [29].

To summarize, we have seen that the nonconservation of particles at certain sites leads to a factorization of the steady state. It is this factorization property that leads to the possibility of finding Griffiths singularities. The nonconserving sites act as particle reservoirs at the boundaries of conserving domains (domains in which the particle dynamics are those of the pure ASEP); the densities of these reservoirs are determined by the attachment and detachment rates. The existence of Griffiths singularities then, given the factorized form of the steady state, depends on the properties of the Yang-Lee zeros of the normalization of the boundary driven ASEP. Such zeros, and therefore also Griffiths singularities, are found only when the densities at the disorder sites carry some spatial dependence. For homogeneous disorder site densities, the normalization of the boundary driven ASEP is given by a product measure (i.e., the matrices D and E are given by scalars) and there are no Yang-Lee zeros. This spatial dependence need not be disorder in the attachment and detachment rates, as considered above—one can choose rates with a periodic modulation for example, and still generate Griffiths singularities.

IV. DYNAMICS: STRETCHED EXPONENTIAL DECAY OF THE DENSITY TO ITS STEADY STATE VALUE

In the pure boundary driven ASEP, whenever the system is in or at the boundary of the maximum current phase, the system density decays with time to its stationary value as an exponential with a decay constant that depends on system size L as L^z, where $z=3/2$ is the dynamic exponent [30,31]. In the low current phase when the boundary injection and ejection rates are equal, a shock exists in the steady state, and the dynamic exponent $z=2$. Otherwise, in the low current phases the relaxation time is finite [30] and does not depend on L. Hence, in the disordered model, whenever contributions to the normalization (10) are in the maximum current phase, the decay of the system density will be determined by the relaxation of these conserving domains.

For the following analysis, it is sufficient to consider disorder only in the location of the disorder sites: we consider homogeneous attachment and detachment rates, i.e., $c_j=c$ and $a_j=a$. In the case where $c=a$, Eq. (3) gives $\rho=1/2$ at which point conserving domains are on the boundary of the maximum current phase; otherwise the conserving domains are in the low current phases. Thus only when $c=a$ do we expect the decay constant associated with a conserving domain to depend on its size [31].

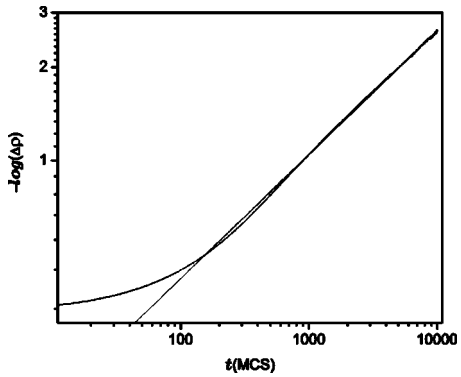

FIG. 2. Log-log plot of the decay of the density with time for $a_j=c_j=10$; the initial condition was an empty lattice. The base of log is ln.

Therefore for $c=a$, in a conserving domain of length n, we assume that the particle density $\rho_n(t)$ decays to its steady state value as

$$\delta\rho_n(t) \equiv \rho_n(t) - 1/2 \sim e^{-\Delta_n t}, \tag{11}$$

where $\Delta_n=\Delta_0 n^{-3/2}$. In the disordered case, we need to sum over configurations of the disorder sites. This is achieved in the same way as in the previous section so, in the thermodynamic limit, the decay of the system density $\rho(t)$ becomes

$$\delta\rho(t) \sim \sum_{n=0}^{\infty} p^n e^{-\Delta_n t}, \tag{12}$$

where we retain only the n dependence, as is sufficient to determine the dominant contribution to the form of the relaxation. If we convert the sum into an integral and consider late times so that the integral can be evaluated at the saddle point, we obtain

$$\delta\rho(t) \sim \exp(-ct^{\phi}), \tag{13}$$

where c is a constant and $\phi=(1+z)^{-1}=2/5$.

Equation (13) predicts the decay of the density up to some prefactor, a power law in t, with an exponent peculiar to the decay of the density. The stretching exponent ϕ should be universal, however, in the sense that other correlation functions, e.g., the current, should reach their stationary values with the same stretched exponential decay. This result should be valid for more general types of disorder whenever one has conserving domains in the maximal current phase.

In Figs. 2 and 3 we show the results of simulations. The simulations were run on systems of 10 000 sites with periodic boundary conditions and averaged over 1000 histories of the dynamics, starting from an empty lattice and with the same realization of disorder. The probability of a site to be pure was choosen to be $p=0.95$. The decay of the averaged system density $\delta\rho(t)$ is shown in Fig. 2 for $a_j=c_j=10$, and in Fig. 3 for $a_j=c_j=1$. The noise at long times in Fig. 3 is due to the small densities and their enhancement by the logarithmic scale. In both cases the straight line $t^{2/5}$ is given for reference. Figure 2 shows very good agreement with the predicted stretched exponential decay, and even in Fig. 3, where c_j and a_j are not large, the agreement is still quite good. Thus

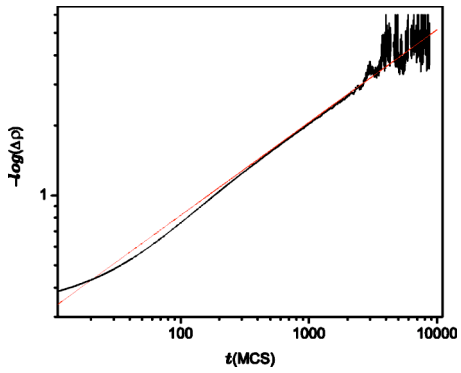

FIG. 3. Log-log plot of the decay of the density with time for $a_j=c_j=1$; the initial condition was an empty lattice. The base of log is ln.

it appears that our result for the stretching exponent holds for finite rates, although its derivation is only exact in the limit of infinite attachment and detachment rates.

V. CONCLUSION

In this work we have studied an ASEP with disorder sites where particles are not conserved. This may provide a basis for a more realistic model for interacting molecular motors such as RNAp. We have used an approximation, exact in the limit of infinite nonconserving rates, the underlying assumption behind which is that the system factorizes into conserving domains. According to the ratios of the attachment and detachment rates these domains assume different phases of the ASEP with open boundaries.

Within this approximation we can explicitly identify Griffiths singularities. These arise when there are large conserving domains, on the boundary of the maximal current phase. An interesting feature is the prediction of a Griffiths singularity despite the absence of a transition in the pure system.

More generally one might ask under what conditions Griffiths singularities arise in nonequilibrium steady states. In equilibrium systems Griffiths singularities are understood in terms of Yang-Lee zeros of the partition function. In nonequilibrium systems one does not have an energy function; nevertheless, one can often identify a quantity that plays the role of a partition function, for example, the normalization (4), and recently there has been progress in understanding the zeros of such quantities [28].

When there is a spectrum of maximal-current conserving-domain sizes, we have demonstrated that correlation functions undergo a stretched exponential decay with a stretching exponent predicted to be $\phi=2/5$. Moreover, simulations suggest this result holds for a wide range of attachment and detachment rates. A related stretched exponential decay has already been observed of autocorrelations in a bond diluted symmetric exclusion process on a ring [32] (in this case $z=2$ so $\phi=1/3$).

In respect of the biological motivation of the model, as is the case in equilibrium, one would not expect to observe any Griffiths singularity directly. However, the factorization property of the steady state also affects the dynamics in a way one might hope to observe in real in vitro experiments. Of course, in experiments, several factors might have to be incorporated into the model to make it more realistic. Examples are the distribution of pure domains, the mechanism of binding to the promoter sites, and disorder in the hopping rates. However, we expect our results to be robust as long as a maximal current phase in the pure domains can be attained and the distribution of domains is Poissonian over a wide range. In particular, given a specific domain size distribution it would be easy to obtain, using the methods described in the text, the density decay as a function of time.

It would be instructive to develop further the approximation that the steady state factorizes about the disorder sites. As we saw in Sec. II this approximation is exact in two limits, and we gave expressions for the densities at the disorder sites. It would be interesting to develop a scheme that interpolates between these two limits. Also of interest would be a better understanding of the correlations between the conserving domains which may exist away from the two exact limits and their effect on Griffiths singularities.

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