The Totally Asymmetric Simple Exclusion Process: Applications to Molecular Motor Modeling

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We explore the totally asymmetric simple exclusion process (TASEP) as a possible generic model of molecular motor movement along a filament. In addition to the usual asymmetric particle hopping dynamics of the TASEP, we propose to allow the motors to switch between two parallel filaments and to randomly attach and detach from the lattice (Langmuir kinetics). We allow for variability in the rates at which the molecular motors move along the filament and analyze the response of our model to single-site defects. Both Monte Carlo simulations and mean field solutions to the model are computed. We find that each of the additional processes qualitatively impacts the distribution of molecular motors along the filament, resulting in new phases and effects not present in the original TASEP model. The addition of switching between the two filaments minimizes the effect of defects. Asymmetric switching and moving rates add greater variability to the possible motor density distributions and responses to a defect. We relate our results to biological systems and propose various additional experimental tests of our model.

PACS numbers:

I. INTRODUCTION

The TASEP was first introduced as a lattice model for the motion of a ribosome along a piece of mRNA during translation [1]. In its most basic form, the model consists of N lattice sites in one dimension. Particles are added at the left boundary of the lattice at some rate α and removed from the right boundary with rate β. In between, a particle at a site i will have a probability p of hopping to the right (hence, the model is asymmetric), i.e. to the $i+1$-th site, *provided that* the site is not occupied (i.e. the particles *exclude* each other from occupying the same site). Typically, we set $p = 1$. This kind of toy model serves as a description of a ribosome moving from codon to codon on an mRNA strand. Namely, we can think about each particle on the lattice as a ribosome. Just as the particle, the ribosome will attach to the mRNA at the start codon (i.e. the left boundary of the lattice). Then, the ribosome will move along the mRNA strand in the 5' to 3' direction, translating one codon in the mRNA at a time. This naturally corresponds to the asymmetric nature of the TASEP dynamics. Finally, the TASEP captures the most basic ribosome-ribosome interaction by not allowing two ribosomes to occupy the same codon (lattice site). The simplicity of this model allows it to be expanded far beyond its first indended use. Since its first incarnation, extensions of the TASEP have been used to model a variety of biological systems, such as molecular motor movement along a microtubule, ant-trail dynamics, and even the process of sequence matching [2, 3].

Since its first appearance in the literature, the original TASEP model has been augmented with various processes to make it a more realistic model. An important modification that is relevant to the study of molecular motors is the addition of *Langmuir kinetics*. These kinetics allow for the addition and removal of particles at each lattice site with some probability rates b and a. This is particularly relevant for molecular motors since they are able to attach and detach from their associated filaments, or "tracks". In fact, there is a high variability in the rate at which this association/dissociation occurs. For example, kinesin-1 movement along a microtubule is highly processive and the motor rarely detaches from its track. Conversely, myosin II does not stay attached to an actin filament for too long in a muscle cell in order to prevent different myosin motors from interfering with each other [4]. Thus, variability in attachment and detachment rates is connected to the biological function of the motor. Moreover, the cell sometimes actively participates in the regulation of these rates. For example, studies of kinesin and dynein motors have shown that the cell can regulate the attachment and detachment rates a and b directly via a microtubule binding protein called tau [5]. Another effect that can be added to the TASEP model is the use of multiple, coupled one dimensional lattices. In particular, we can couple two TASEP "lanes" together by allowing particles to hop back and forth between the lanes with some characteristic rates s_1 and s_2. Such a process is biologically relevant because molecular motors, such as kinesin, move along a set of parallel tracks. In particular, it is known that microtubulin consists of about 13 protofilaments which twist together in a helical structure. Kinesin and dynein motors run along these individual protofilaments and are able to switch to adjacent filaments to avoid colliding with each other. In particular, studies suggest that kinesin motors, being more tightly bound to a protofilament, are able to "push" a dynein motor out of the way to an adjacent protofilament [6]. Lane switching dynamics are also particularly relevant in the description of the microtubule and

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actin cytoskeleton network present in neuronal cells. In these cells, single molecular motors are able to move between different tracks consisting of microtubulin or actin. Goode et al. suggest that it is possible that motors of various types form "heteromotor" complexes that allow the transport of single objects along two different cytoskeletal tracks [7].

Even the simplest TASEP dynamics are complicated by the fact that they already move beyond the realm of equilibrium statistical mechanics. This is because the TASEP violates detailed balance. Namely, consider any configuration $\mathcal{C} = \{ n_i \mid i = 1, 2, \dots N \}$ of the lattice, where $n_i = 1$ if the i-th site is occupied and 0 otherwise. Then, let $P_t(\mathcal{C})$ be the probability that at time t we have a configuration $\mathcal{C}$. This probability will satisfy a *master equation* which describes its time evolution. The equation is given by

$$\frac{\partial}{\partial t} P_t(\mathcal{C}) = \sum_{\mathcal{C}'} \left[\mathcal{W}_{\mathcal{C}' \to \mathcal{C}} P_t(\mathcal{C}') - \mathcal{W}_{\mathcal{C} \to \mathcal{C}'} P_t(\mathcal{C}) \right], \qquad (1)$$

where $W_{\mathcal{C} \to \mathcal{C}'}$ is a probability *rate* of transitioning from configuration $\mathcal{C}$ to configuration $\mathcal{C}'$. For an equilibrium system, we know that for large times t, we approach a *steady-state Boltzmann distribution*, which necessarily satisfies the detailed balance condition

$$\mathcal{W}_{\mathcal{C}' \to \mathcal{C}} P_t(\mathcal{C}') = \mathcal{W}_{\mathcal{C} \to \mathcal{C}'} P_t(\mathcal{C}). \qquad (2)$$

This condition is guaranteed by the usual Boltzmann relation from statistical mechanics

$$\frac{P_t(\mathcal{C})}{P_t(\mathcal{C}')} = e^{\beta[\mathcal{H}(\mathcal{C}') - \mathcal{H}(\mathcal{C})]}, \qquad (3)$$

where $\mathcal{H}(\mathcal{C})$ is the energy of configuration $\mathcal{C}$ [9]. When a system satisfies detailed balance, we can use powerful tools from equilibrium statistical mechanics, such as various thermodynamic relations, fluctuation-dissipations theorems, and so on. We can write down a partition and entropy function, and have a complete statistical description of the system. However, biological processes are often very far from equilibrium. Indeed, these systems are *open* and are constantly driven by various energy sources (e.g. ATP to ADP transitions). Thus, we need to go back to a basic microscopic description of these systems in order to derive various statistical behaviors. To this day, a systematic formulation of non-equilibrium processes remains an open problem. TASEP models and their relatives, like the Ising model in equilibrium statistical mechanics, attempt to highlight the generic statistical rules which govern these open systems. By analyzing a variant of the TASEP model, we hope to say something about molecular motor movement, which, being a type of *active* transport, is a non-equilibrium process.

To highlight the difference between TASEP and other statistical models, it is useful to consider the hopping model for molecular motor movement proposed by Fisher and Kolomeisky [8]. In that model, a molecular motor moves along a one-dimensional lattice by transitioning through N internal states at each lattice point. Namely, after the motor transitions through N internal states, it moves on to the adjacent lattice site. These transitions are characterized by asymmetric forward and backward rates which push the motor preferentially along one direction on the lattice. Although this particular model does not satisfy detailed balance explicitly, it is sufficiently close that the authors are able to use various relations, such as the Einstein relation $\mu = k_B T / D$. This equation relates the diffusion constant D to the mobility μ and temperature T, which allows one to calculate a friction force $F = \mu v$ (for an average motor velocity v) that is interpreted as the driving force of the molecular motor. This relation, however, is only valid close to equilibrium and the authors of the model acknowledge that it may not always be applicable [8]. Conversely, in the analysis of a TASEP model, we are not able to make use of these powerful relations and are mostly constrained to looking at Monte Carlo simulations of the microscopic dynamics or solving mean field equations derived from the Master Equation (Eq. 1). Indeed, even the concept of a single temperature is not useful in these far-from-equilibrium models because often the steady state solution of the model (the $\partial_t P_t(\mathcal{C}) = 0$ solution in Eq. 1) includes a macroscopic flux of particles mediated by a temperature *gradient* [9]. The advantage of a TASEP model is that it includes additional processes, such as motor-motor interactions, which are not present in the hopping model. Also, being an explicitly non-equilibrium model, we expect that the TASEP provides a better generic and conceptual description of molecular motor movement since it does not rely on proximity to equilibrium to get relevant results.

II. THE MODEL

In this study, we modify the model introduced by Parmeggiani et al. by introducing an additional lane and defects. The introduction of a second lane was done recently by Wang et al. [14]. We propose combining both Langmuir kinetics, a two-lane TASEP, and defects. As discussed in the previous section, these additional processes have important biological consequences. So, we include both the different rates p_1 and p_2 with which the particles move along the two different lanes in the model and switching rates s_1 and s_2 between the two lanes. We were not able to find a model which included all of these effects in the literature. The diagrammatic description of the proposed model is given in Fig. 1. The model is implemented using a Monte Carlo simulation and random-sequential updates. The latter refers to picking a random site on the lattice (both lanes), and applying an "update rule." In our model, if the site that is chosen during the random update is not a boundary site and is empty, then we add a particle to the site with probability a. If the site is filled, we remove a particle with probability b. If

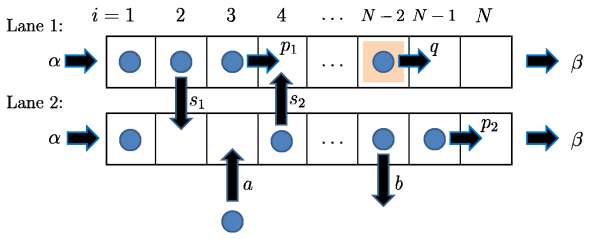

FIG. 1: The proposed molecular motor model has motors attaching to one end of a filament at rate α and leaving from the other end with rate β. The motors detach and attach to the filament with rates b and a, respectively. The motors move along the filament with rate $p_{1,2}$ and with rate q in the defects. The motors switch lanes with rates s_1 and s_2, depending on which lane the motor is in.

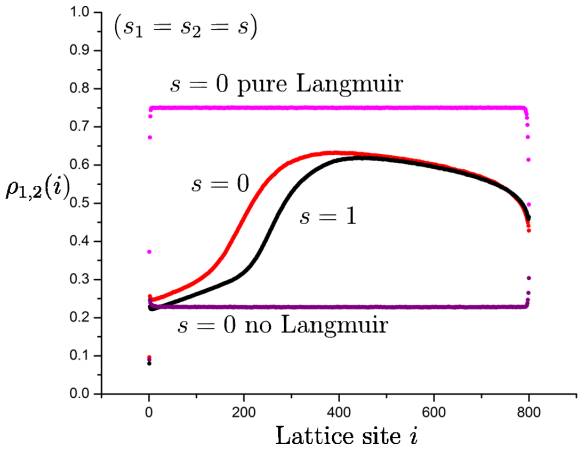

FIG. 2: The average particle density at each lattice site i for $N = 800$ sites. The parameter values used for regime with two coexisting phases were $p_1 = p_2 = 1$, $\alpha = 0.2$, $\beta = 0.6$, $a = 0.3/(N-2)$, and $b = 0.1/(N-2)$. The "pure Langmuir" line both a and b were increased $N-2$-fold. For the "no Langmuir" regime, we had $a = b = 0$. No defects were introduced.

neither of these things occur, then, if the site is filled, we move the particle to the right if the adjacent site is empty (with probability p_1 or p_2 depending on the lane). Otherwise, we move the particle to the other lane (if the corresponding site is open) with probability s. If we hit a boundary site during our random sequential updates, for the left (right) boundary, we add (remove) a particle with probability α (β) if the site is empty (full). Also, in the left boundary sites, if the site is filled and the adjacent site is empty, we move the particle to the right with probability p_1 or p_2. If none of these things can happen, i.e. if there is a "traffic block" in both lanes, then we simply move on to the next random lattice site. An important aspect of Monte Carlo simulations using random-sequential updating is the fact that the rates a and b must be rescaled relative to α and β. We recognize that a and b must be both divided by $N-2$ because we apply the update rule with a and b to $N-2$ sites while the α and β rates are only applied at the left and right boundary. Also, since transient solutions are typically difficult to characterize, we only analyze the *steady state* behavior of the model. This is done by allowing the Monte Carlo simulation to run through many time steps (about $10^5 N$) and achieve a single steady state before we extract any data from the simulation.

To test the model, we first try to replicate the results of Parmeggiani et al. In that study, a phase was discovered in which the competing Langmuir (a and b rates) and boundary dynamics (α and β rates) induce a coexisting high and low density phase with a sharp boundary [10]. We were able to find this phase using the suggested values $\alpha = 0.2$, $\beta = 0.6$, $a = 0.3/(N-2)$, and $b = 0.1/(N-2)$ from that study, where $N = 800$ is the number of lattice sites used in the simulation. In Fig. 2, we see that we indeed get this interesting phase. The two coexisting phases in Fig. 2 can be understood by appealing to previous analytic results for the particular values of the parameters we used. Namely, from the complete analytic solution to the TASEP master equation, we know that the density of particles in the basic TASEP, away from the boundary, is given by $\rho(i) = \alpha = 0.2$ in the so-called "low density phase," for which $\alpha \leq \min\{\beta, 1/2\}$ [11]. Thus, we see that the left side of the lattice is dominated by the boundary TASEP dynamics due to α. This is confirmed by the "no Langmuir"-labeled line in Fig. 2. Conversely, on the right side, we see the effects of the Langmuir dynamics. Namely, we know that for pure Langmuir dynamics, the density of the particles on the lattice is given by the expression $\rho(i) = K/(1+K)$, where $K \equiv a/b$ [10]. In our case, we have that $K = 3$. Thus, the density we expect to get from the Langmuir dynamics is $\rho(i) = 3/4$. This is confirmed by the "pure Langmuir"-labeled line in Fig. 2.

These results have a biological interpretation. Namely, our model suggests that the distribution of molecular motors along a filament can be regulated by varying the rates associated with the dynamics of the motors. Since these rates can be regulated by the cell through various microtubule-associated proteins (MAPs) such as tau, the various phases exhibited by our model may have important analogues in biological systems [4]. For example, several studies have shown that kinesin motors tend to clump near the end of a microtubule toward which the motors move. Moreover, a recent experiment by Nishinari et al. suggests that domain walls, or "shocks", between high and low concentrations of molecular motors form along a microtubule [12, 13]. This kind of asymmetric distribution of motors is qualitatively confirmed by our model.

Although it is difficult to find an analytically tractable solution to the master equation of our particular model, it is possible to perform a mean field analysis. Namely, we can consider the particle occupation number $n_{j,i}$ in the j-th lane and i-th site on our $N \times 2$ lattice. Then, if we include all of the relevant processes of particle hopping and association/disassociation, we find from the master

equation that the average density satisfies

$$\begin{aligned}\frac{d\langle n_{1,i}\rangle}{dt} &= p_1\langle n_{1,i-1}(1-n_{1,i})\rangle - p_1\langle n_{1,i}(1-n_{1,i-1})\rangle \\ &+s_2\langle n_{2,i}n_{2,i+1}(1-n_{1,i})\rangle + a\langle 1-n_{1,i}\rangle \\ &-d\langle n_{1,i}\rangle - s_1\langle n_{1,i}n_{1,i+1}(1-n_{2,i})\rangle, \end{aligned} \quad (4)$$

with an identical equation for $\langle n_{2,i}\rangle$ except with the exchange of the 1 and 2 labels [14]. Eq. 4 is difficult to solve exactly, because it requires us to compute two point correlation functions, which in turn depend on three point correlation functions, and so on. This forms an infinite set of equations called the BBGKY hierarchy. So, to avoid this messy hierarchy, we move to the mean field and assume that we have no correlations between different lattice sites. This allows us to expand our averages and approximate that, for example, $\langle n_{1,i}n_{1,i+1}(1-n_{2,i})\rangle \approx \langle n_{1,i}\rangle\langle n_{1,i+1}\rangle\langle 1-n_{2,i}\rangle$. We can also move to the continuum limit and let $n_{j,i} \to \rho_j(x,t)$ represent the coarse-grained density at lattice point i and lane j, where we define the rescaled, continuous variable $x = i/N$, which ranges from 0 to 1. Using this coarse graining, we can use a Taylor series to expand $n_{j,i\pm1} \to \rho_j(x,t) \pm N^{-1}\partial_x\rho_j(x,t) + (2N^2)^{-1}\partial_x^2\rho_j(x,t) + \mathcal{O}(N^{-3})$. Then, since we are interested in the steady state behavior of the system, we let the time derivative term vanish to get the solution $\rho_j(x, t\to\infty) = \rho_j^*(x)$ that is independent of time. The resulting differential equation for $p_j^*(x)$ is given by

$$\begin{aligned} 0 &= \frac{p_1\epsilon}{2}\frac{d^2\rho_1^*}{dx^2} + p_1\frac{d\rho_1^*}{dx}(2\rho_1^*(x)-1) \\ &+s_2(\rho_2^*(x))^2(1-\rho_1^*(x)) + a(1-\rho_1^*(x)) \\ &-d\rho_1^*(x) - s_1(\rho_1^*(x))^2(1-\rho_2^*(x)), \end{aligned} \quad (5)$$

where $\epsilon = 1/N$ is the *spacing* between adjacent values of $x = i/N$ which we took to zero in the continuum limit [14]. Thus, formally, we have that $\epsilon \to 0$. Also, we technically have two more differential equations which describe the particle entry and exit at the left and right boundaries. However, for simplicity, we shall approximate that N is sufficiently large that we can implement the boundary equations by just specifying the conditions $p_{1,2}^*(0) = \alpha$ and $p_{1,2}^*(1) = 1-\beta$ to take into account the particle incoming/outgoing rates at the boundary. Notice that we require ϵ to be non-zero to get a second order equation in each lane. This is necessary if we are to apply two boundary conditions per lane, as explained below.

We can test our mean field theory by again finding the coexisting phase regime. To make things more interesting, we also make use of the asymmetric lane switching rates. Namely, we will let one of the lanes have a high lane-switching rate $s_2 = 1$ and the other one have a slower rate $s_1 = 0.01$. We see in Fig. 3 that the asymmetric rates pushes the boundary between the high and low density phases to the left for the $s_1 = 0.01$ lane. This makes physical sense because we expect that particles are more likely to get jammed in this lane due to the slower lane switching rate. Also, in the $s_2 = 1$ lane, we see that the fast switching eliminates the two coexisting phases and pushes the system into a low density state.

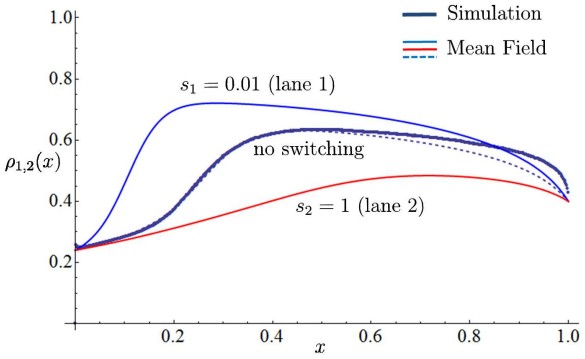

FIG. 3: A comparison of the solution to the mean field equations and a Monte Carlo simulation result with no lane switching. The mean field equations were also solved for asymmetric lane switching rates. We used $a = 0.3/(N-2)$ and $b = 0.1/(N-2)$ and the other parameter values from Fig. 2. The mean field equations were integrated numerically with Mathematica 7.0.

In Fig. 3, we see that the simulation solution does not exactly match the mean field. This is not surprising because our Monte Carlo simulations were run on limited computer resources which prevented us from getting the best statistics. Also, in general, it is difficult to integrate the mean field equations due to the non-linear terms and the small value of ϵ. Specifically, we know that ϵ is typically very small and in the thermodynamic limit we have to take $N \to \infty$ and $\epsilon \to 0$, correspondingly. This turns our mean field equations (Eq. 5) into *first order* equations. But, since we have two initial conditions for each of the lines, i.e. the rates α and β, the system becomes *over-determined*. In this case, we have to integrate the equations twice: once for the $\rho_{1,2}(0) = \alpha$ boundary conditions and once for the $\rho_{1,2}(1) = 1-\beta$ conditions. The whole solution is then achieved by matching the two solutions. In the case of our coexisting phases shown in Fig. 2, for example, we will get a discontinuity at the boundary between the phases. Such an infinitely sharp boundary is often called a "shock". It is the presence of this sharp boundary that makes our mean field equations difficult to integrate numerically. More powerful analytic methods that treat the $\epsilon \to 0$ are mentioned in [10] and are beyond the scope of this paper.

III. ADDING DEFECTS

We now want to add another aspect to our TASEP model. Namely, we modify the dynamics by adding a single "slow" lattice site in one of the lanes with a smaller probability q of moving a particle to the right at that site. Such a "defect" has biological significance in a couple of different systems. For example, it is known that

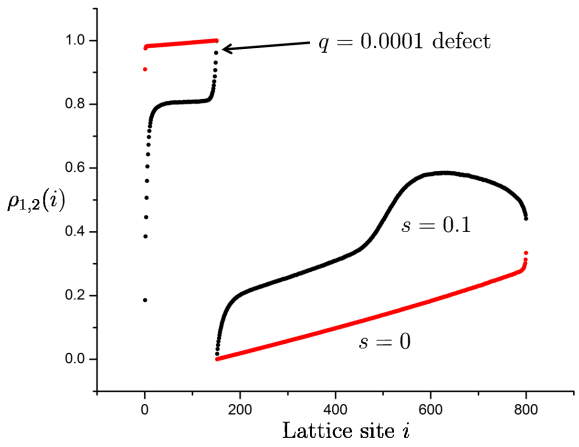

FIG. 4: Introducing a defect in the 150th lattice site in lane 1 disrupts the two coexisting phases in the case where we do not allow lane switching. The particle density in lane 1 is shown. The lane switching preserves the robustness of the phase and reduces the effects of the defect. In this case, we used symmetric lane switching rates $s_1 = s_2 = s$.

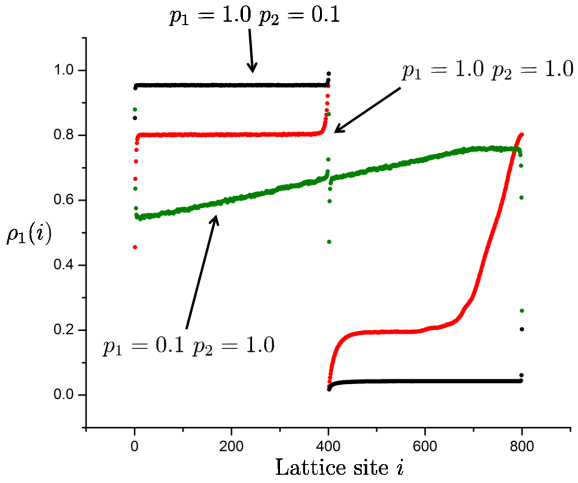

FIG. 5: A defect was added in the middle of the lattice at the $i = 400$ site in lane 1. The resulting particle density in lane 1 is shown. The parameters used are $q = 0.01$, $s_1 = s_2 = 0.1$, $a = b = 0$ (no Langmuir kinetics), $\alpha = 0.3$, and $\beta = 0.2$. The speeds of the two lanes were varied as shown which resulted in drastic changes in the density profiles.

strands of mRNA can contain rare codons which cause the ribosome to lag during the translation process. These "slow codons" often form localized clusters which serve to regulate translation in many organisms. A recent TASEP-like model developed by Tom Chou and Greg Lakatos [15] shows that only a single localized region of slow codons is necessary to reduce the ribosome current. Thus, we are motivated to include such a single defect in our model. Localized defects are also relevant for molecular motor movement. A study by D. Chrétien et al. showed that individual microtubules exhibit variation in protofilament number along the length of the microtubule [16]. The usual quoted number of 13 protofilaments per microtubule is in fact variable. The filaments exhibit sharp lattice defects which change the local protofilament number and will thus influence the molecular motor dynamics. By adding single defects to our model, we hope to make some qualitative predictions about the behavior of the motors.

Keeping all of the parameter values used in Fig. 2, we added a defect lattice site (with $q = 0.0001$) in one of the lanes in the $i = 150$ position. Then, we see in Fig. 4 that if we allow no lane switching ($s_1 = s_2 = 0$), this defects disrupts the phase coexistence and introduces a domain wall at the location of the defect in the lane. Conversely, if we allow for lane switching to occur even at a small rate, i.e. $s = 0.1$, we regain the interesting phase. So, we see that lane switching adds a robustness to the balancing act that occurs between TASEP and Langmuir dynamics in this phase. Our model confirms the Chou et al. result that a single defect, or localized cluster of defects, is sufficient to significantly modify the global dynamics of the motors *if we have no lane switching*. This makes sense for ribosomes because they walk along just one strand of mRNA. However, for molecular motors such as kinesin, it is possible that the ability of the motor to detach from its track and to switch lanes prevents the defects from significantly modifying the overall rate of motor procession.

Finally, we studied the effects of having a defect coupled with asymmetric processivity rates p_1 and p_2. As seen in Fig. 5, our model predicts that even without Langmuir kinetics, lane switching has the ability to eliminate effects of a defect, provided that the lane that does not have the defect is faster than the lane with the defect ($p_1 = 0.1$ and $p_2 = 1.0$ line in Fig. 5). Conversely, if the lane without the defect is *slow*, then the defect in the fast lane can effectively block the movement of motors in both lanes, as shown in the $p_1 = 1.0$ and $p_2 = 0.1$ line in Fig. 5. Also, for the case where we have equally fast lanes, we see that the lane switching allows motors to "leak through" the defect and eventually establish a high density phase toward the right end of the microtubule (i.e. the right side of the $p_1 = p_2 = 1.0$ line in Fig. 5). Interestingly, to the left of the defect, the expected behavior of the TASEP does not change for the $p_1 = p_2$ case! Specifically, for our parameter values $\alpha = 0.3$ and $\beta = 0.2$, the exact solution to the basic TASEP tells us that we should be in the high density phase, which is characterized by a uniform density $\rho = 1 - \beta$ in the bulk [11]. As we see in Fig. 5, the density to the left of the defect in the $p_1 = p_2 = 1.0$ case remains at 0.8. We suspect that this is due to the "leakage" of the particles past the defect, which lessens the jamming effect of the defect and allows the system to settle into its natural high density phase. Conversely, when the defect is in the fast lane, we see that the jamming effect is more pronounced and the density jumps to approximately 0.95.

IV. CONCLUSIONS

We have seen how the TASEP model can be used to model the movement of molecular motors along a track. The generality and simplicity of the model allows it to be applied to a variety of biological systems, such as kinesin, dyein, and myosin. The modified TASEP model presented in this paper predicts that the processive dynamics of molecular motors coupled with particular attachment and detachment rates can lead to a build-up of proteins near one end of the microtubule or actin filament, resulting in a low and high density coexisting phase regime, as discussed in [10]. We found that this phase is stabilized with respect to localized defects with the addition of symmetric lane switching dynamics. Also, by having asymmetric lane switching rates, we are able to either destroy the coexisting phase or move its boundary to the left or right. A subject of further study might be the computation of molecular motor *currents* in the model. We can measure the average speed of the motor to the right at a particular lattice site i in lane j by computing $J_{j,i} \equiv \langle n_{j,i}(1 - n_{j,i+1})\rangle$, where the average would be taken over many Monte Carlo simulation steps. We could even compute the rate at which the motors switch lanes, i.e. from lane 1 to lane 2, by calculating $K_{1,i} \equiv \langle n_{1,i}(1 - n_{2,i})\rangle$. These rates would allow us to make some qualitative predictions of the rate at which molecular motors move along a filament.

We were able to get more predictions by adding defects to our model. Our simulations have shown that lane-switching and Langmuir dynamics preserve the robustness of the bulk phase of the TASEP model and minimize the effects of localized defects. Given the available biological data on defects in microtubules, the analysis presented here suggests that an experiment measuring the motor density profile around a defect in a microtubule would be a good test of our model. Similarly, it would be interesting to see how modifying the concentrations of MAPs in a cell influences the motor density profiles on microtubules. Our analysis suggests that by varying the expression of proteins such as tau, we should be able to qualitatively change the distribution of molecular motors. In particular, we should be able to see the transition between the phase with two coexisting high and low density regimes and a single, homogeneous phase as shown in Fig. 2. Moreover, with the addition of a defect, we should be able to see the effects of lane switching in preserving the motor density profile. In particular, the model predicts that kinesin, which is more tightly bound to protofilaments, should be more susceptible to point-like defects, such as protofilament lattice dislocations, than a less tightly bound motor such as dynein.

Our study of asymmetric processivity rates p_1 and p_2 combined with lane switching suggest that these rates have a significant impact on the response of a system to a defect. A good experimental test of our model would be an analysis of the motion of heteromotor complexes that are able to move over a diverse cytoskeletal structure, as discussed in [7]. Since the processivity of such a complex will be different depending on the particular kind of "track" it is moving on, our model might be able to make qualitative predictions of how such structures respond to structural defects in filaments and MAPs that alter heteromotor binding rates. The results we presented on asymmetric lane switching rates also suggest that molecular motor density profiles will depend on how favorable a certain filament track is relative to another. So, we should see the effects of these lane switching dynamics in the density profiles of heteromotor complexes. Finally, we could enhance our model by including even more lanes, or internal states for the particles. The latter would be particularly relevant to biological applications because we know that molecular motors have to go through a series of internal states while moving along a filament [8].

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