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Lattice Boltzmann method for simulating Min protein dynamics incorporating the role of nucleoids

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Abstract

The dynamics of Min proteins plays a center role in accurate cell division. Although the nucleoids may presumably play an important role in prokaryotic cell division, there is a lack of models to account for its participation. In this work, we apply the lattice Boltzmann method to investigate protein oscillation based on a mesoscopic model that takes into account the nucleoid's role. We found that our numerical results are in reasonably good agreement with the previous experimental results on comparing with the other computational models without the presence of nucleoids, the highlight of our finding is that the local densities of MinD and MinE on the cytoplasmic membrane increases, especially along the cell width, when the size of the obstacle increases, leading to a more distinct cap-like structure at the poles. This feature indicated the realistic pattern and reflected the combination of Min protein dynamics and nucleoid's role.

Keywords: lattice Boltzmann method; cell division; Min proteins; protein oscillation; nucleoid; *E. coli*

1. Introduction

Cell division is one of the most important process in asexual reproduction, in order to separates a cell into two daughter cells. Within the process, the DNA has been duplicated and taken part into two regions. Experimentally, the Min proteins that control the placement of the division site are the MinC, the MinD, and the MinE proteins [1]. Experiments involving the use of modified proteins show that MinC is able to inhibit the formation of the FtsZ-ring [2]. MinD is an ATPase that is connected peripherally to the cytoplasmic membrane. It can bind to the MinC and activate the function of the MinC [3, 4]. Recent studies show that the MinD can also recruit the MinC to the membrane. This leads to suggest that the MinD collaborate with the MinC by concentrating the MinC near to its targeted site of activation [5, 6]. MinE provides topological specificity to the division inhibitor [7]. Its expression results in site-specific suppression of the MinC/MinD action by which the FtsZ assembly is allowed at the middle of the cell but

is prohibited at the other sites. Without the MinE, the MinC/MinD is distributed uniformly over the entire membrane. This results in a complete blockage of Z-ring formation. The long filamentous cells, which subsequently form, are not able to divide [5, 6, 8, 9].

The presence of MinE is not only required for MinC/MinD oscillation, it is also involved in setting the frequency of the oscillation cycles [8]. Several sets of evidence have been complied to show that the MinE localization cycle is strongly coupled to the oscillatory behavior of MinD. Recent microscopy of the fluorescent labeled proteins involved in the regulation of *E. coli* division have uncovered stable and coherent oscillations (both spatial and temporal) among these three proteins [10]. The proteins move between the cytoplasmic membrane and cytoplasm, and produce an oscillating pattern from one end to the other end of the bacterium. The detailed mechanism to describe such behavior of these proteins to determine the correct position of the division plane is yet unknown, but the observed pole-to-pole oscillations of the corresponding distribution are believed to be essential and not ignorable.

Several models for Min protein oscillation have been proposed and studied [11-15, 16 , 17]. These models are described as macroscopic nonlinear reaction-diffusion equations (RDE) and are solved numerically by using conventional finite difference schemes. Howard *et al.* (2001) [13] proposed an RDE model which is expressed by the dynamics properties of a protein's association and dissociation with the membrane. Meinhardt *et al.* (2001) [16] included the dynamics of FtsZ proteins in their model and suggested that the pattern formation of the Min system depends on the interaction of a self-enhancing component, as well as its long-ranging antagonists. Kruse *et al.* (2002) [15] found that pole-to-pole oscillation is related to a tendency of membrane bound MinD to cluster, attach, and detach from the cell wall. However, the model proposed by Kruse imposed rapid membrane diffusion of MinD which seems unrealistic. In 2004, Huang and Wingreen [14] proposed a model to reproduce the experimental oscillations in a number of cell shapes such as rod-shape, round and ellipsoidal. In 2005, Drew *et al.* [11] proposed a mathematical model to describe the polymerization and depolymerization behavior of MinD. Their results are that the MinD protein binds to the membrane, followed by the subsequent binding of MinE. Modchang *et al.* (2005) [17], who studied the effects of an external field, found that the concentrations of MinD and MinE are not symmetric about the middle of the long cell axis, nor are the minimum/maximum MinD and MinE concentrations at the middle of the long axis.

All aforementioned models deal only with the oscillation of Min proteins, yet nucleoid zones are also an important factor of cell division after the duplication phase has occurred. This present work therefore focuses on modeling of Min proteins oscillation taking into account the role of nucleoids as obstacles to Min proteins flow inside *E. coli* by using a mesoscopic LBM. Since this is the first investigation of this condition for Min protein movement, our main goal is to understand the pattern formation in the Min protein flow compared to that produced by

previous models which did not account for such obstacles. We expect that our findings could contribute to a better understanding of Min protein oscillation-mediated cell division.

2. MIN Protein Dynamics Model

As an insights into the nature of the dynamics of Min proteins is of great importance, we initially followed Howard *et al.*'s model [13], wherein a set of four non-linear coupled reaction diffusion equations in one spatial dimension was used to describe pole-to-pole oscillation of Min proteins. Their explanation of Min protein dynamics was restricted to only one spatial dimension and for that reason it may not be very realistic. Thus, we turned to Waipot *et al.* [18], who extended their model to two spatial dimensions and used the lattice Boltzmann method (LBM) to study the oscillation dynamics of the Min proteins in *E. coli*. Straightforward and relatively simple, the model gives the correct placement of the division septum in *E. coli*. This feature makes is easy to add the nucleoids as obstacles in our study. This condition showed allow a more realistic explanation of the mechanism of Min proteins during the cell division process. This mechanism is controlled by the rates of change of the protein densities which are a result of the diffusions of MinD and MinE and to the mass transfer between the cell membrane and the cytoplasm, as schematically shown in Fig. 1.

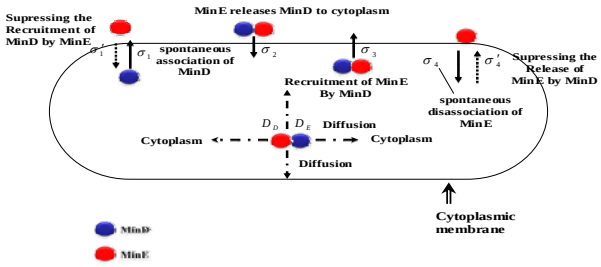


Fig. 1 A schematic diagram of the MinD/MinE dynamics which was proposed by Howard *et al.* (2001)

In dimensionless form, the dynamics are expressed by the following equations:

$$\frac{\partial \rho_D}{\partial t} - D_D \nabla^2 \rho_D = R_D = -\frac{\sigma_1 \rho_D}{1+\sigma_1' \rho_e} + \sigma_2 \rho_e \rho_d \quad (1)$$

$$\frac{\partial \rho_d}{\partial t} - D_d \nabla^2 \rho_d = R_d = \frac{\sigma_1 \rho_D}{1+\sigma_1' \rho_e} - \sigma_2 \rho_e \rho_d \quad (2)$$

$$\frac{\partial \rho_E}{\partial t} - D_E \nabla^2 \rho_E = R_E = \frac{\sigma_4 \rho_e}{1+\sigma_4' \rho_D} - \sigma_3 \rho_D \rho_E \quad (3)$$

$$\frac{\partial \rho_e}{\partial t} - D_e \nabla^2 \rho_e = R_e = -\frac{\sigma_4 \rho_e}{1+\sigma_4' \rho_D} + \sigma_3 \rho_D \rho_E \quad (4)$$

where ∇^2 is the two dimensional Laplacian operator. Subscription letters $s = \{D, d, E, e\}$ stand for the cytoplasmic MinD, the membrane bound MinD, the cytoplasmic MinE, and the membrane bound MinE, respectively. Here, ρ_s is the mass density of particles of species s which is a function time t and position (x, y). R_s is a reaction, D_s is the diffusion coefficient; σ_1 is the parameter related to the spontaneous association of MinD to the cytoplasmic membrane; σ_1' is that which is related to the suppression of MinD recruitment from the cytoplasm by the membrane bound MinE; and the σ_2 reflects the rate that MinE on the membrane drives the MinD on the membrane into the cytoplasm, σ_3 is the rate that cytoplasmic MinD recruits cytoplasmic MinE to the membrane, σ_4 describes the rate of

dissociation of MinE from the membrane to the cytoplasm, and σ_4' represents the cytoplasmic MinD suppression of the release of the membrane bound MinE. The diffusion on the membrane is assumed to be negligible as the all proteins are immobile in the cytoplasm [13] and it seems reasonable to set D_d and D_e equal to zero. In this dynamics, we allow the Min protein to bind/unbind from the membrane, but not be degraded in the process. Thus, the total amount for each type of Min protein and the total concentration of Min proteins are conserved.

In this work, we focus on the MinD/MinE dynamics with nucleoids as obstacles. These nucleoids appear typically around the central region of cell poles in an *E. coli* where the presence of bulk DNA has an inhibitory effect on FtsZ ring formation. Two complementary mechanisms have been proposed for localizing cell division in rod-shaped *E. coli* cells: one where nucleoid occlusion [19] prevents FtsZ ring assembly on membrane regions immediately surrounding nucleoids that are actively involved in transertion (transition) [20], and the other where the Min system [8] inhibits FtsZ ring assembly at cell poles, while allowing ring formation at midcell. Since the nucleoid and Min system appear to be independent of each other, our studies are simplified with the nucleoid zones used as obstacles in an *E. coli* cell during the accurate positioning process of the septum. The schematic diagram in our studies is shown in Fig. 3.

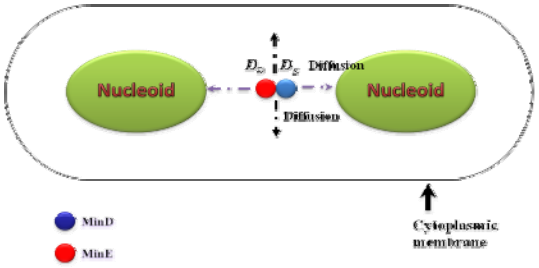


Fig. 3 A schematic diagram of the MinD/MinE dynamics with nucleoid zones

3. The Lattice Boltzmann Method and Simulation

In this section, we apply the lattice Boltzmann method (LBM) for obtaining reaction diffusion equations (RDE), as shown in Eqs. (1)-(4). The discrete form of the lattice Boltzmann equation can be written as

$$f_\alpha^s(\vec{x}+\vec{e}_\alpha\delta t, t+\delta t) - f_\alpha^s(\vec{x},t) = \Omega_\alpha^s(\vec{x},t)\,, \qquad (5)$$

where $f_\alpha^s(\vec{x},t)$ is the particle distribution function of species s with discrete velocity $\vec{e}_\alpha$ at space $\vec{x}$ and time t. The species $s=\{D,d,E,e\}$ represents the cytoplasmic MinD, membrane-bound MinD, cytoplasmic MinE and membrane-bound MinE, respectively. The collision operator Ω_α^s for species s can be separated into two terms [21]. The first term is the Boltzmann BGK approximation of the distribution function with a single relaxation time τ_s and the second term is a reactive collision term so that

$$\Omega_\alpha^s(\vec{x},t) = -\frac{1}{\tau_s}(f_\alpha^s(\vec{x},t) - f_\alpha^{(eq,s)}(\vec{x},t)) + \phi_\alpha^s\,, \qquad (6)$$

where $f_\alpha^{(eq,s)}$ is the equilibrium distribution. Here we use the simple equilibrium distribution function corresponding to a system with zero mean flow as

$$f_\alpha^{(eq,s)} = \omega_\alpha^s \rho_s\,, \qquad (7)$$

where ω_α^s is the weight function which depends on the lattice symmetry [22]. The density of

particle species s is denoted by ρ_s. For the reactive term ϕ_α^s, we use the simple isotropic form:

$$\phi_\alpha^s = \omega_\alpha^s R_s \quad (8)$$

The term R_s is the non-linear reaction term and depends on the density of the reacting species. By Chapman-Enskog expansion [23], the relation between the diffusion coefficient and relaxation time is

$$D_s = \frac{1}{3}(\tau_s - \frac{1}{2}) \quad (9)$$

The simulation process also consists of two steps which are governed by the following two equations:

Collision:

$$f_\alpha^{s,*}(\vec{x},t+\delta t) = f_\alpha^s(\vec{x},t) - \frac{1}{\tau}(f_\alpha^s(\vec{x},t) - f_\alpha^{(eq,s)}(\vec{x},t)) + \omega_\alpha^s R_s \quad (10)$$

Streaming:

$$f_\alpha^s(\vec{x}+\vec{e}_\alpha\delta t, t+\delta t) = f_\alpha^{s,*}(\vec{x},t+\delta t) \quad (11)$$

For the boundary condition, we use the mirror-image method suggested by Zhang *et al*. [24] as shown in Fig. 4.

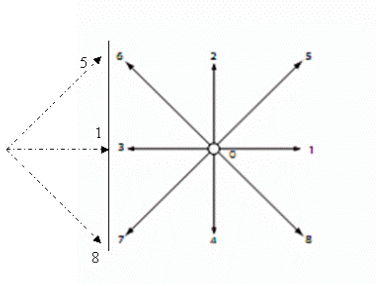


Fig. 4 Sketch of mirror-image boundary condition

If the node B is a boundary node, it will see its image in node I. The distribution functions are also specified at the image node, to serve as the missing distribution function to the real node. The exact form of the distribution function at the image cell is yet to be decided by the specific boundary. In this work, the impermeable boundary is applied and seems appropriate for the reaction-diffusion system. For the impermeable boundary, the distribution functions at the imaginary nodes use the mirrored distribution function at their real corresponding nodes. As in Fig. 4, the pro-collision and pre-streaming distribution functions at the imaginary node I are:

$$f_1(I,t) = f_3(B,t)$$
$$f_5(I,t) = f_6(B,t)$$
$$f_8(I,t) = f_7(B,t)$$

Such boundary conditions are considered suitable for a low speed flows and diffusion systems [24].

4. Results and Discussions

The LBM model given in section 3 is implemented to simulate the two-dimension (2D) model on a personal computer using C programming. To simplify the coding, the LBM algorithm needs all parameters to be dimensionless. So we transform the original parameters by letting

$n = \rho/\rho_0$, $\tilde{D}_D = D_D\delta t/(\delta\vec{x})^2$,
$\tilde{D}_E = D_E\delta t/(\delta\vec{x})^2$, $\tilde{\sigma}_1 = \sigma_1\delta t$, $\tilde{\sigma}_1' = \sigma_1'\rho_0$,
$\tilde{\sigma}_2 = \sigma_2\rho_0\delta t$, $\tilde{\sigma}_3 = \sigma_3\rho_0\delta t$, $\tilde{\sigma}_4 = \sigma_4\rho_0\delta t$,
$\tilde{\sigma}_4' = \sigma_4'\rho_0$,

where δt, $\delta\vec{x}$ and ρ_0 are the time step, grid spacing, and the unit of concentration, respectively. The relaxation time τ_s is calculated by Eq. (9). We assume that the *E. coli* cell is rectangular and the dimension is taken to be $1\times 2\ \mu m$. In the simulation, we choose discrete space steps $\delta x = \delta y = 2\times 10^{-2}\ \mu m$ and time step $\delta t = 4\times 10^{-4}\ s$, and set $\rho_0 = 1/\mu m^2$ as the concentration unit. In this study we place square obstacles at the central region of cell poles to

represent the nucleoid zones (see computational domain in Fig. 5). Infact, we have tested our model by placing one nucleoid in the middle of the cell. With this set-up, it appears that the system can not provide the division site around the middle of the cell, as normally occurs in nature for *E. coli.* This supports the biological fact that in the dividing process when the cell already has duplicated all contents to be ready for cell partitioning, the cell is composed of two nucleoid zones, with each being approximately in the middle of each half cell. This is the configuration that we use here in our simulation. We also assumed that each nucleoid has a square shape - mainly due to the lattice and cell characteristics that we imposed as a rectangular system, which has been found to work well in our previous investigations [18].

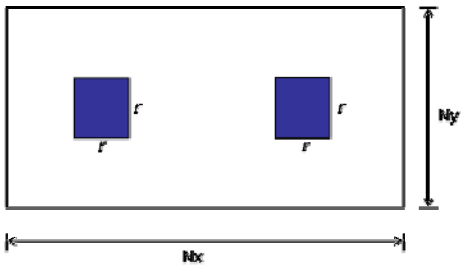


Fig. 5 The computational domain for the *E. coli* cell with nucleoids

In our simulations, the interaction between each nucleoid and the Min proteins is mainly presented in the form of a mirror-image boundary condition. This is to reflect a physical interaction in contrast to a chemical interaction. The main reason for this consideration is that we wanted to start our investigation with a simple model and take advantage of the LBM, which is a proven all-around computational method for complicated models [18]. Here, the dimension of obstacles are $r \times r$. Other parameters are given in [13], are summarized in Table 1.

From the simulation data of MinD and MinE, it was found that as a result of the increase in the size of each nucleoid from 0% and 9% of the cell size, the oscillation pattern of MinD and MinE proteins for an obstacle that is 9% of the cell size gives the best agreement with that in the experimental results, as shown in Fig. 6 and 7. The MinD appears to localize at the polar zones, while MinE prefers to localize around the middle of the cell. These results not only agree well with experimental data from the point of view of flow pattern foundation, but also with regards to the size of each nucleoid. Moreover, increasing the percentage of each nucleoid in the cell size (0,9 %), the MinD and MinE proteins show a localization pattern near the cytoplasmic membrane, which is relatively more intense than that found when no nucleoid is included. Once again, this characteristics of protein dynamics agrees well with the experimental results [25, 26] where MinD and MinE proteins are located at or near the cytoplasmic membrane and lesser appear in the cytoplasm.

With regards to the effect of the obstacles on the Min protein dynamics, we found that the local density of MinD and MinE on the cytoplasmic membrane increases, especially along the cell width, when the percentage of obstacle in space increases. In other words, the results indicated that the size of the obstacle (nucleoid) may induce higher local density of MinD and MinE at the cytoplasmic membrane.

These results agree with experimental data, as shown in Fig. 6b and Fig. 7b. The high fluorescent intensity of both proteins is located near the cytoplasmic membrane. One explanation for this may be the fact that added nucleoids establish a physical nucleoid occlusion constraint and force Min proteins to distribute more in the vicinity of the membrane leading to the cap-like membrane bound structure. In addition, we investigated the pattern formation of MinD and MinE proteins in terms of time-averaged density, as shown in Fig. 8. It is found that one again both proteins show different localizations normally seen in experiments. The density of MinD is maximum density at the cell poles and minimum at the midcell, while that of MinE is minimum at cell poles and maximum at midcell. This indicates that the division site is likely to occur at or near the midcell zone. Biologically, the MinD in our simulation is indeed the MinC/MinD complex. MinC is the division inhibitor in this system and it interacts with the division protein FtsZ to prevent formation of stable FtsZ rings, an essential first step in an assembly of the division machinery [27].

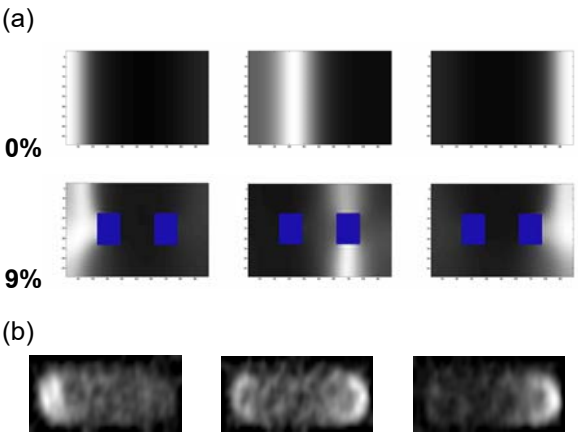


Fig. 6 MinD dynamics from LBM and experiment. (a) The numerical results of MinD dynamics for different conditions of obstacles. The obstacles are shown at 0,9 percents of cell size. (b) The experimental results of MinD protein are shown in term of fluorescent intensity [25, 26]. The MinD clusters are observed near the cytoplasmic membrane

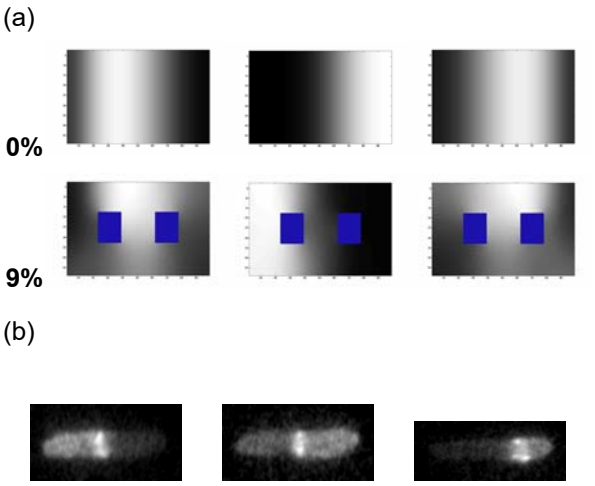
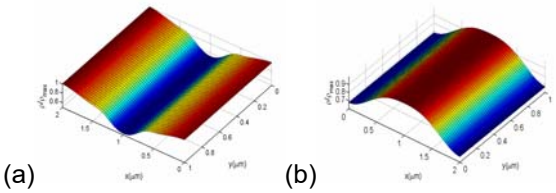
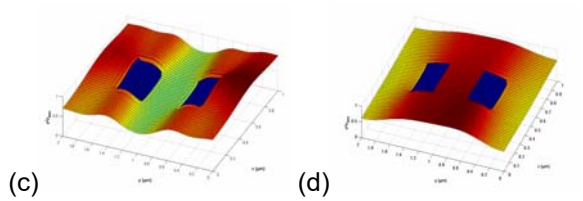


Fig. 7 MinE dynamics from the LBM and experiment. (a) The numerical results of MinE dynamics for different conditions of obstacles. The obstacles are shown at 0,9 percents of cell size. (b) The experimental results of MinE proteins are shown in terms of fluorescent intensity [25, 26]. The MinE clusters are observed near the cytoplasmic membrane





(c) (d)

Fig. 8 Time-average plots of MinD and MinE dynamics from the LBM with and without obstacles. The MinD protein is shown in terms of the time-averaged density for the obstacles 0,9 percents of cell size as shown in (a), (c), respectively. The MinE protein is shown in terms of time-averaged density for the obstacles 0,4,9 percents of cell size as shown in (b), (d), respectively

5. Concluding Remarks

To summarize, we have investigated the oscillatory movement of Min proteins in *E. coli* with nucleoids as obstacles. With an appropriate nucleoid size, the results are in reasonably good agreement with those from other experiments: namely MinD density is maximum density at the cell poles and minimum at the midcell, while MinE density is minimum density at the cell poles and maximum at midcell, as has been shown by previous models and experimental studies. Hence, our results indicate that the division site may occur at or near midcell zone. On comparing the results from this work with those done with other models with no nucleoid present, the highlight of our findings is that local densities of MinD and MinE on the cytoplasmic membrane (which are relatively higher than those found with no nucleoid present) increases, especially along cell width, when the percentage of obstacle space increases, leading to a more distinct cap-like structure at the poles. Finally, we would like to mention that the LBM approach is a useful scheme for simulating biological systems at the cellular and molecular level and which are governed by reaction-diffusion equations.

Acknowledgments

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