

I. INTRODUCTION

A. What is the MinD system and why is it important?

-System of proteins in E.Coli and other cells. -Theorized to be instrumental in cell citokenisis. Reference experiments

B. How proteins move in cell

-Reference experimental showing proteins oscillating - Reference theory showing difEQ model shows oscillations -Reference Mannik shoving into crevices. -Worthwhile studying effect of walls shape on the movement of cells (Sign post of what to expect from this paper)

II. METHODS AND INITIAL CONDITIONS

A. Mathematical Model

The model for the behavior of the MinD and MinE proteins inside the cell implemented the same set of 5 reaction-diffusion equations described in the paper by Huang et al (equations 1, 2, 3, 4, and 5). A 3d grid was constructed in cartesian coordinates with a grid spacing of $.05 \mu\text{m}$. From there, we were able to define a cell shape on the grid, and solve the reaction-diffusion equations numerically to observe the time evolution of the MinD and MinE concentrations inside the cell.

Our simulation used the same diffusion constants and reaction rates as Huang et al, which are

$$\begin{aligned} D_D &= D_E = 2.5 \mu\text{m}^2 / \text{sec}, \\ \sigma_D^{\text{ADP} \rightarrow \text{ATP}} &= 1/\text{sec}, \sigma_D = 0.025 \mu\text{m}/\text{sec}, \\ \sigma_{dD} &= 0.0015 \mu\text{m}^3/\text{sec}, \\ \sigma_{de} &= 0.7/\text{sec}, \sigma_E = 0.093 \mu\text{m}^3/\text{sec}. \end{aligned}$$

To test our computational model, we implemented a pill shaped cell, and tested using the same cell parameters as Huang et al, which were a radius of $0.5 \mu\text{m}$ in the middle and at the spherical endcaps, and two different cell lengths of $4 \mu\text{m}$ and $10 \mu\text{m}$. We found the same type of oscillations as in their paper using these initial conditions, verifying that our model works as intended. Below are snapshots of MinD and MinE concentrations at 5 second timestamps in the $4 \mu\text{m}$ cell:

[insert 5 second time stamps of $4 \mu\text{m}$ sim]

We then began to define other, non-traditional cell shapes for the purpose of modeling squished and perturbed E. coli cells, which were created experimentally in Mannick et al. To achieve this, we went with a cartesian

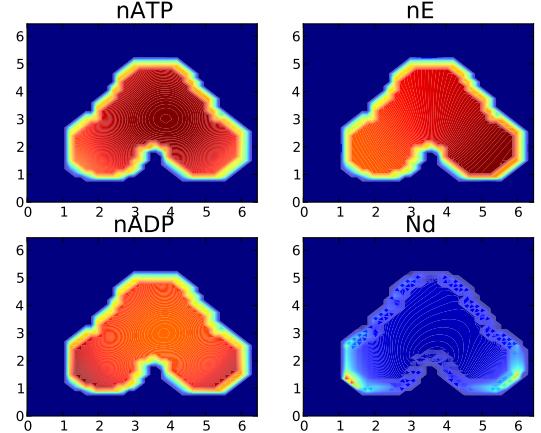


FIG. 1. randst 99

lattice rather than the cylindrical lattice used in Huang et al's simulations, as it allows for more flexibility in defining the cell shape. Some of the cell shape models included a flattened pill (stadium shape), an ellipsoid, a spherical cell, and various randomly generated smooth shapes, such as those in the figures below.

[insert memf print of 2-3 cell shapes]

To interpret the results, we generated several different plot views of the printed simulation data. These plots included a time averaged view of the protein densities in the cell; a plot tracking the location of protein concentrations that were global maxima in space and local maxima in time; and an animated view that showed the actual dispersion of protein concentrations in the cell over time.

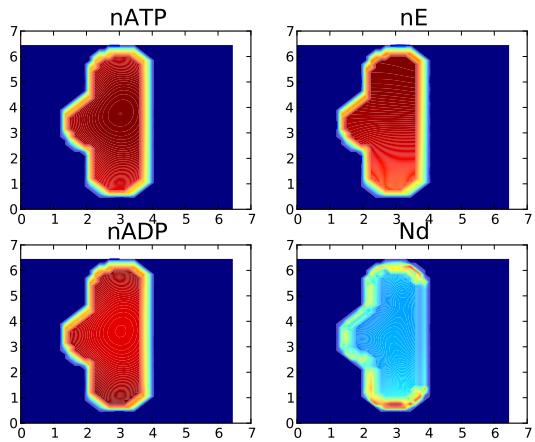
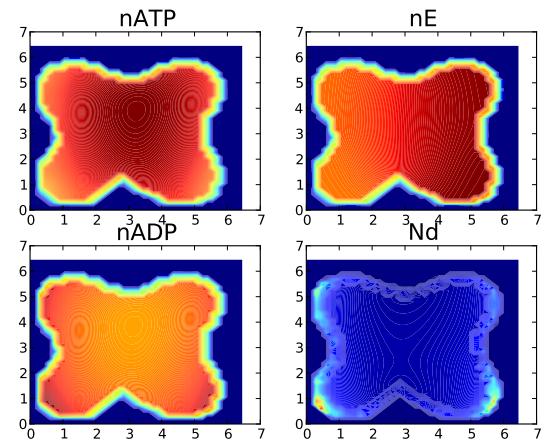
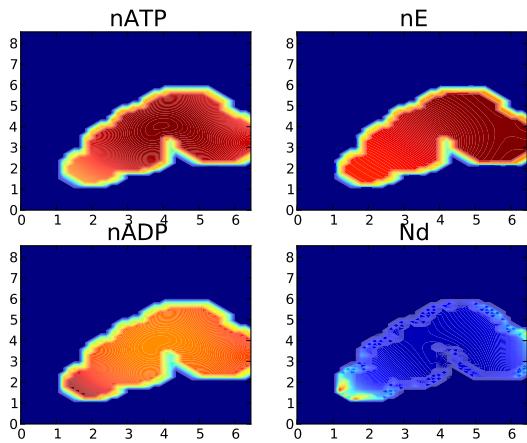
III. SPECIFIC RESULTS

Pill Normal section - Our goal with this project was to test whether or not the computational model developed by Huang et al was consistent with the newer experimental results (squishing E Coli) produced by Mannick et al.

Pill Short - Know how short is too short
 Randst 99 -
 Randst 98 -
 Randst 97 -
 Randst 96 -
 Triangle -

IV. INTERPRETATION OF DATA

-Discussion of conceptual reasons of why we see what we see -Plots that are more interpretive (area-rating) -



Some sort of predictive claim?

V. CONCLUSION

APPENDIX