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A model for exponential elongation in Escherichia coli and evidence of inert polar cellular regions

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A model for exponential elongation in *Escherichia coli*

and evidence of inert polar cellular regions

A Thesis submitted in partial satisfaction of the requirements for the degree

Master of Science

in

Biology

by

Joshua Aaron Kenchel

Committee in charge:

Professor Lin Chao, Chair
Professor Justin Meyer
Professor Scott Rifkin

2015
The Thesis of Joshua Aaron Kenchel is approved and it is acceptable in quality and form for publication on microfilm and electronically:

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Chair

University of California, San Diego

2015
DEDICATION

This work is dedicated to all those who inspired me to follow this path, in one way or another – in particular, my grandfather, Dr. Jonas Schultz, the first scientist I ever knew; Dr. Ryan Simkovsky, who taught me the practice of good scientific inquiry; my family and close friends, who have always offered encouragement in the face of doubt; Frank Joyce, whose passionate interest in the natural world personifies the nobility of this profession (and who never felt entitled to an honorific prefix); Dr. Scott Rifkin, who opened my mind with a hands-on introduction to quantitative and computational biology alongside his colleague and comrade: Dr. Lin Chao, who graciously took me in as an apprentice and gave me every opportunity to succeed at the next level of academia.
Nothing in Biology makes sense except in the Light of Evolution.

Theodosius Dobzhansky
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I would especially like to thank Audrey Proenca, who constructed the mother machine and performed the experiment that produced the video used for this research, and Dr. Camilla Rang for management of cell cultures and microscopy. I also thank them both for their feedback. In addition, they and the other members of the Chao Lab – Matt Metzger, Brock Wooldridge, Kevin Chi, Jin Jung, César Aguzzoli, and Sarah Schwartz, among others – made the lab a warm and welcoming place to learn and work.
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ABSTRACT OF THE THESIS

A model for exponential elongation in *Escherichia coli*

and evidence of inert polar cellular regions

by

Joshua Aaron Kenchel

Master of Science in Biology

University of California, San Diego, 2015

Professor Lin Chao, Chair

*Escherichia coli*, the most thoroughly studied model organism, is a rod-shaped bacterium that elongates at an exponential rate relative to its length. Recent studies have suggested that *E. coli* cells contain regions that are inert; that is, they do not contribute to the elongation of the cell. It is likely that these regions are mostly contained in the semispherical poles of the cell. Using the formulas for the idealized geometry of an *E. coli* cell (a cylinder plus two semispheres), I derived
models for the exponential elongation of the cell. To test the validity of each model, I collected length data over time from growing *E. coli* cells and fit the data to the models. The data best fit a model in which *E. coli* cells contain inert regions of length equal to two times the radius of the cell, which is the combined length of the poles of the cell. The data thus present evidence in support of the existence of inert polar regions of *E. coli* cells.
Introduction

Background

*Escherichia coli*, the most thoroughly studied model organism, is a rod-shaped Gram-negative bacterium. An *E. coli* cell consists of a cylindrical body capped by two semispheres\textsuperscript{1}. The radius of the cylinder equals that of the semispheres. As the cell grows, it elongates lengthwise along the cylindrical axis at an exponential rate\textsuperscript{2}, but the radius of the cell remains constant.

One of the first goals of the nascent field of microbiology was the identification, characterization, and classification of the myriad microbial morphologies. In the past couple of decades, there has been renewed interest in microbial shape\textsuperscript{3}, including that of *E. coli*, this time with an emphasis on discovering and describing the molecular structures and pathways that create and maintain morphology. While the basic structure and elongation rate of *E. coli* have been well characterized, the precise mechanisms of elongation and maintenance of cell shape remain unclear\textsuperscript{1}. It was once thought that turgor pressure alone might be the force that pushes the boundaries of the cell outward and causes it to grow\textsuperscript{4}. One could imagine a similar scenario in which elongation is dependent upon the quantity of some metabolite in the cell, in which case the exponential change in size of the cell might be a direct function of volume. Or, perhaps some lengthwise structure – say, an axial filament like the one found in *Mycoplasma pneumoniae*\textsuperscript{5} – pushes the ends of the cell outward; in this case, the exponential change in size would be a direct function of length.
A recent study found that the peptidoglycan in the cell wall of the semispherical poles remains inert after formation\textsuperscript{6}. Inert peptidoglycan (iPG) appears to be localized at the poles, while new peptidoglycan is synthesized and replaces or adds to the peptidoglycan in the cell wall of the cylindrical body of the cell. As suggested by Young\textsuperscript{1} in 2003, inert poles may form a kind of scaffold that forces cells to add new peptidoglycan only along the cylindrical body of the cell and thus maintain the rod shape. If the addition of peptidoglycan along the cell surface is the force that dictates elongation, the change in cell size might be a direct function of surface area.

Another recent study found that peptidoglycan synthesis along the cylindrical cell wall is directed by the helical filament protein MreB\textsuperscript{7}. If the synthesis of cell wall were directed by a helix that increased in length as the cell elongated, it could explain why the cell elongates exponentially. That is, if the helical MreB filament directs the synthesis of new peptidoglycan, the rate of cell elongation is directly proportional to peptidoglycan synthesis, and the length of the helical filament increases linearly with time, then the rate of cell elongation should be exponentially related to the length of the cell.

Furthermore, if the MreB helix spans the cylindrical portion of the cell, but does not extend to the spherical poles, then the exponential elongation of the E. coli cell might be a direct function of the cylindrical portion of the cell only, and not the entire mass of the cell. Such a structure could help explain the observation that the polar cell wall is inert, and does not grow.
Current studies use the model $L_t = L_0 e^{kt}$, in which $L$ is the length of the cell from tip to tip, in order to measure the elongation rate $k$ of a cell; however, this model does not account for possible inert regions of the cell, such as the poles. If the cell wall at the poles exclusively contains iPG, the poles might not contribute at all to exponential elongation. I approached this question from a mathematical perspective in order to test these recent observations and determine an accurate model for the exponential elongation of an *E. coli* cell.

**Elongation model**

Using the formulas for the idealized geometry of an *E. coli* cell, a cylinder plus a sphere, I derived multiple models for different possible scenarios and mechanisms of *E. coli* elongation. I considered whether elongation might be driven by the entire mass of the cell, or by the cylindrical body only. I also considered whether exponential elongation might be a direct function of the length, surface area, or volume of the cell; thus, I derived a model for each of six scenarios in total. Because the length of a cell is much more easily observable than the surface area or volume, I converted each model to a function of $L_t$, the length of the cell at time $t$.

As mentioned, the basic model for the elongation of an *E. coli* cell is

$$L_t = L_0 e^{kt}$$

(1)
If exponential elongation is a direct function of the surface area of the cell, (1) takes the form \( A_t = A_0 e^{kt} \); if it a direct function of volume, it takes the form \( V_t = V_0 e^{kt} \). In order to model exponential elongation while taking into account the possibility of inert poles, I derived a base model,\(^5\)

\[ L_t = g e^{kt} + c \]

(2)
in which \( L_t \) is the length at time \( t \), \( g \) is the (growing) portion of the cell that contributes to exponential elongation, \( k \) is an unspecified growth rate, and \( c \) is the constant (inert) portion of the cell that does not contribute to elongation. Equation (2) also takes forms analogous to those of (1) based on whether elongation is a direct function of length, surface area, or volume. Because the entire mass of the cell must be either growing or inert,

\[ L_0 = g + c \]

(3)
The values of \( g \) and \( c \) change according to which portions of the cell contribute to elongation. \( L \) is the length of the cylindrical portion of the cell only.

For example, let us assume that the entire mass of the cell contributes to elongation, and that elongation is a direct function of volume. Let us also maintain the assumption that the radius of the cylinder and semispheres of the cell are constant. In that case, the
formulas for the volume of a cylinder and a sphere plug into (2) with the volume of the cylinder as $g$ and the volume of the sphere as $c$. Doing so yields

$$\pi r^2(L_t - 2r) + \frac{4}{3}\pi r^3 = [\pi r^2(L_0 - 2r) + \frac{4}{3}\pi r^3]e^{kt}$$

(4)

in which $r$ is the radius of the cell and $L$ is the length of the entire cell from tip to tip. Note that because the axial length of each semisphere is equal to $r$, the length of the cylinder is $L - 2r$. Also, only $L$ changes with time; because $r$ is constant and the volume of the sphere is dependent on $r$ alone, the volume of the sphere is constant.

Equation (4) reduces to

$$L_t = (L_0 - \frac{2}{3}r)e^{kt} + \frac{2}{3}r$$

(5)

in which $c = \frac{2}{3}r$, where $r$ is the radius of the cylinder and sphere. In this scenario, $g = L_0 - \frac{2}{3}r$, so the values of $c$ and $g$ are consistent with (3). The structure of (5) is intuitive: when $t = 0$, $L_t = L_0$.

The other scenarios in which the entire cell contributes to elongation reduce to a form similar to that of (5), except $c = 0$ and $g = L_0$; thus, the models for those scenarios are identical to (1). Therefore, if the entire mass of the cell contributes to elongation, depending on whether elongation is a direct function of length, surface area, or volume,
\[ \frac{c}{r} = 0 \text{ or } \frac{2}{3} \] (6)

The three scenarios in which only the cylindrical body contributes to elongation all evaluate to a model with a form similar to that of (5), except \( c = 2r \) and \( g = L_0 - 2r \). Therefore, if the semispherical poles of the cell are inert and therefore do not contribute to elongation,

\[ c = 2r \] (7)

Every possible model given the possible conditions of the cylinder or the whole cell contributing to the elongation of the cell and the elongation being a direct function of the length, surface area, or volume of the cell is summarized in Table 1. Since in every hypothesized model, \( g \) is determined by \( L_0 \) and \( c \), and \( c \) is always directly proportional to \( r \), an efficient way to test the validity of the hypothesized models would be to gather length data from \( E. coli \) cells, estimate the value of the ratio \( c/r \) that best fits the data, and compare it to the ratio \( c/r \) in the hypothesized models. With this strategy in mind, I measured the lengths of \( E. coli \) cells over time to produce elongation curves, fit those curves to the base model (2), and recorded the values of the parameters estimated by the nonlinear best fit.
Table 1. Hypothesized models for E. coli cell elongation. I derived models for elongation as a function of volume surface area, or length, as well as for the cell having inert or growing poles, for a total of six scenarios. For each of the six scenarios, I plugged the formulas for the geometry of the cell into the base model and solved for $L_t$, the length at time $t$. This yielded three distinct models with a form similar to the base model, in which $c = 0$, $(2/3)r$, or 2.

<table>
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<td>$L_t = (L_0 - (2/3)r)e^{kt} + (2/3)r$</td>
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Materials and Methods

Growth conditions and microscopy

We grew *E. coli* in a “mother machine”\(^8\), a chip made of polydimethylsiloxane (PDMS) with channels and traps carved into it. The 16 channels in the chip allowed medium to flow through them. 2,000 traps in total, each 1.25 \(\mu\)m wide and 30 \(\mu\)m deep, were carved into the sides of the 16 channels. The traps are designed to be approximately the diameter of an *E. coli* cell, so that a single cell fits snugly and no two cells can sit side-by-side in a trap. A single mother cell can sit at the bottom of a trap growing and dividing indefinitely. The flow of medium through the channel pushes fresh nutrients into the traps and clears wastes. As the mother cell divides and creates new daughter cells, it pushes its daughter cells toward the opening. Approximately every fourth or fifth daughter cell is swept by the flowing medium out of the trap, into the channel, and away into a waste receptacle. The mother machine thus allows for the recording of many generations of cells without the issues of overcrowding and stacking posed by minicolonies.

We grew *E. coli* cells in this mother machine in Luria broth with a supplement of 0.75% Tween 20, a lubricant that prevents bacterial adhesion to the walls of the chip and subsequent biofilm formation.

The procedure for inoculating the mother machine was as follows. First, the chip was placed in a vacuum chamber for 15 minutes, and then removed. A drop of culture was added to the chip, and the chip was centrifuged in order to push *E. coli*
cells into the traps. Lastly, the chip was connected to syringes for the input and output of medium.

We recorded images of the cells every two minutes using a digital phase contrast microscope, at 100X magnification. No fluorescence imaging was used in this experiment.

While watching the digital videos of the dividing *E. coli* cells, I measured and recorded the length of each *E. coli* cell every frame using ImageJ\(^9\).

**Statistical analyses and nonlinear best fit**

Using ImageJ, I measured the lengths of individual cells over time. For each individual cell, I measured its length at each frame many times and averaged those replicates to yield one averaged elongation curve.

For each cell, I fit the averaged elongation curve\(^10\) to the base model (2) and recorded the best-fit values of \(g\), \(k\), and \(c\). Because the predicted values of \(c\) were always directly proportional to the radius \(r\), I calculated and recorded the ratio \(c/r\) for each curve.

In order to determine how many replicate elongation curves would yield averaged curves with precise measurements of the constants fit to the elongation model, I performed a bootstrap analysis. First, I measured 60 replicate curves of the same cell. Next, I sampled those curves, with replacement, into 20 sets of replicate curves. I repeated this for each one replicate per set through 60 replicates per set, incremented by one. I averaged each set of replicates to yield an averaged curve, fit
that curve to the base model, and recorded the estimated values of the constants g, k, and c. Lastly, I calculated the variance of c/r given each of 1 through 60 replicates per averaged curve.

I compared the marginal fractional decrease in variance of the predicted value of c to the marginal fractional increase in the amount of data collection required to achieve that variance. The intersection of those two curves represented the most efficient number of replicates per averaged curve relative to the amount of work required.

I used the two-tailed student’s t-test to determine whether the calculated mean value of c/r was significantly different than the values hypothesized by the models in Table 1.
Results

I gathered length data from digital video by hand-measuring 86 *E. coli* cells that were elongating and dividing. For each cell, these data produced an elongation curve consisting of the entire set of length measurements between formation and division. I fit each of these elongation curves to the base model, \( L_t = g e^{kt} + c \). The nonlinear best fit output estimates of the parameters \( g, k, \) and \( c \), which represent the size of the growing portion of the cell, the growth rate, and the size of the inert portion of the cell, respectively.

The nonlinear best fit produced widely deviating estimates of the parameters when each elongation curve was generated from only a single set of measurements per cell (Figure 1a). The elongation curve for a single set of measurements seemed to contain considerable measurement error, which propagated to even greater error in the parameter estimates. When I took multiple replicate measurements of the same cell and calculated the average of the length measurements at each point in time, the resulting averaged curve appeared much less noisy and yielded much less erratic parameters from the nonlinear best fit (Figure 1b).

In order to determine whether generating an averaged curve for each cell from multiple replicate sets of measurements would significantly reduce the error of the parameter estimates, I collected 60 replicate sets of measurements for a single cell and performed a bootstrap analysis. I sampled the replicate data to form simulated 60 datasets of 20 averaged elongation curves, in which each averaged curve was generated from between 1 and 60 replicate measurements. The bootstrap
Figure 1. Examples of elongation curves. (a) A series of curves from a single set of length data following one lineage of *E. coli* cells elongating and dividing. When fit to the base model, these curves yielded highly erratic estimates of the parameters $g$, $k$, and $c$. (b) Averaged elongation curves from the same lineage as the data in (a), generated by averaging three replicate sets of measurements, including the data shown in (a). The curves appear less noisy than the curves in (a), and yielded much less erratic parameter estimates.
a) 

![Graph showing length (µm) over time (min)]

b) 

![Graph showing mean of three replicate length measurements (µm) over time (min)]
analysis revealed that the margin of error in the parameters produced by the nonlinear best fit decreased substantially as the number of replicate measurements used to produce each average elongation curve increased (Figure 2a). The marginal fractional increase in data collection outpaced the marginal fractional decrease in error between 9 and 10 replicates per averaged curve (Figure 2b). This result indicated that 10 measurements per cell would yield growth curves with variance of c/r minimized relative to the data collection required to retrieve that variance.

I measured each of the 86 cells 10 times, generated an averaged elongation curve for each, and fit each curve to the base model. The nonlinear best fit for each curve estimated the value of c, which I divided by the radius r. The mean observed value for the ratio c/r for 86 averaged elongation curves, each generated by the average of 10 replicate curves, was 2.151 (95% confidence interval: (1.908, 2.409), Figure 3). The t-tests indicated that the mean measured value of c/r = 2.151 was not significantly different than 2 (p = 0.2354), which was the value hypothesized if the poles of the cell do not contribute to elongation, but was significantly different than 2/3 (p < 10⁻¹⁵, t = 11.736, df = 85) and 0 (p ≈ 0, t = 17.0064, df = 85), which were the values hypothesized if the entire cell contributes to elongation.
Figure 2. Analysis of error versus replicate collection of length measurements. I measured the length of the same cell from formation to division 60 times. I sampled the length data from the 60 elongation curves, with replacement, into 20 sets of replicate curves, and averaged each set of length data into a single averaged elongation curve. I repeated this process for one through 60 replicates per set, incremented by one. I fit each of the averaged elongation curves to the base model and recorded the value of the constant c. (a) Shown are the variance of the average value of the ratio c/r versus the number of replicates per averaged curve (solid circles). A best-fit curve is shown as a solid line. (b) Marginal fractional change curves are shown for the decrease in variance, drawn from the best-fit curve in (a) (solid line), and the increase in data collection required (dashed line) to achieve that variance. The curves intersect between 9 and 10 replicates, indicating that 10 replicates will minimize the error of the data relative to the effort required to record those data.
a) Variance of c/r vs. Replicates per averaged elongation curve.

b) Marginal fractional change vs. Replicates per averaged elongation curve.

Legend:
- Decrease in error
- Increase in data collection
Figure 3. Measurement of the inert portion of the cell. When fit to the base model, each of 86 elongation curves yielded a constant c, representing the portion of cell mass that does not contribute to elongation. Shown here is the average ratio of c to the radius of the cell, r. The error bar represents a 95% confidence interval. Shown also on the graph are the three hypothesized values for c/r, which were 0, 2/3, and 2. For c/r = 0 and c/r = 2/3, the entire cell contributes to elongation; for c/r = 2, only the cylindrical portion contributes.
Discussion

The observed mean value $c/r = 2.151$ is not significantly different from 2, which was the value hypothesized for the scenario in which only the cylindrical portion of the cell contributes to exponential elongation. The confidence interval of the observed data for $c/r$ includes $c/r = 2$. This observation indicates that there is a region of an *E. coli* cell of length $2r$ that is inert, which is to say that it does not contribute to elongation. While this inert region might be in any part of the cell, or dispersed in parts throughout the cell, it is most likely located in the semispherical poles of the cell, whose combined axial lengths equal $2r$. In other words, this result aligns with and supports the finding of de Pedro et al.\(^6\) that peptidoglycan synthesized at the poles is inert. The poles of an *E. coli* cell likely do not contribute to exponential elongation of the cell.

Because $c = 2r$, due to Equation (3),

$$g = L_0 - 2r$$

(8)

Therefore, a more appropriate model for exponential elongation in *E. coli* is

$$L_t = (L_0 - 2r)e^{kt} + 2r$$

(9)
This model indicates that the spherical poles of the rod-shaped *E. coli* cell are inert and do not contribute to elongation of the cell. Using this model rather than \( L_t = L_0e^{kt} \) should yield more accurate measurements of the growth rate \( k \).

It has not escaped notice that the midpoint of the confidence interval of \( c/r \) is greater than 2, and that the value \( c/r = 2 \) is near the lower end of the confidence interval. If a further study consisting of more precise measurements of cell length and/or a larger sample were to find that suggesting that the quantity of inert peptidoglycan in an *E. coli* cell may be greater than that in the poles alone. Indeed, this is what Young (2003) suggests in a review of studies of bacterial shape\(^1\): rod-shaped cells may contain fluid patches of inert peptidoglycan (iPG) that constantly shift and are prone to producing morphological mutants.

One additional factor for which I did not account was the change in geometry of *E. coli* cells as they prepare to divide. As an *E. coli* cell divides, it produces a furrow near the midpoint of its length, which changes the shape of the cell from the assumed cylinder-sphere geometry. Accounting for this change in geometry might alter the values of \( c \) and \( g \) assumed by the models presented in this thesis; if non-growing iPG is exclusively deposited at the cleavage furrow, it would increase the magnitude of \( c \), the constant portion of the cell. I did notice that the slope many of the elongation curves decreased slightly at the end of the curves, though I did not count how many curves had this characteristic or analyze its potential significance; that slight decrease in slope might be attributable to the cell devoting energy resources to division rather than elongation. I also noticed that
many of the curves had a similar decrease in slope near the middle of the curve; though I again did not perform any analysis of it, this fluctuation might be due to cell cycle effects such as the cell devoting energy resources to replication of the genome.

It may also be that the observed possible difference in the value of c from 2r is due to differences in the iPG content of old daughter cells versus new daughter cells, which were observed by Rang et al. The doubling time of the old daughter cell was found to be significantly greater than that of the new daughter cell. It might be the case that the old daughter cell wall consists of a greater proportion of iPG than that of the new daughter cell, which should result in a greater value of c.

Since *E. coli* is the most utilized model organism, understanding every facet of its biology will likely be a stepping-stone to further and greater discoveries. This model may also be applicable to many other species of rod-shaped bacteria. Rod-shaped bacteria are a polyphyletic clade, and while many have MreB or MreB-homologous helical structural fibers, not all rod-shaped bacteria use an MreB-analogous system. It would be interesting to see whether or the model supported in this thesis fits the elongation observed in a broad variety of rod-shaped bacterial species; such information might yield broad or specific insights into the nature of their structure and physiology.
References


