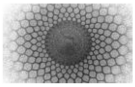
TTMP vol 3 (1), 2026
doi: 10.30511/TTMP.2026.2083409.1070

Transactions in Theoretical and Mathematical Physics



A Dynamical Physics Equation for Cell Proliferation

Javad Sharifi[a]

[1]Electrical and Computer Engineering Department, Qom University of Technology

Received: 07 January 2026 / Accepted: 20 February 2026 / Published: 23 February 2026

Abstract. A dynamical model for mitosis cell proliferation from a geometric viewpoint is derived. The derived kinetics are nonlinear partial differential equations of cell volume and mass density. We consider the process of inherent cell growth due to biological processes as the mass increase in the cell which exerts force inside of the cell. Likewise, the process of segregation is viewed as a mass decrease. We consider the physical aspects of inherent biological processes such as anabolism of structural proteins and lipids swelling due to mass influx and shrinking due to mass outflow. To this aim, by first and second equations of motion together with Fick's laws of diffusion for nutrition, we model this force and obtain the cell radius function and mass density evolution equations. The control action will appear in our dynamic equation.

Keywords: Dynamic Physics Equation; Cell Proliferation; Fick Laws

1 Introduction

We can model biological processes and bio-systems with the aim of physical laws and system insight. Furthermore, by control theory of biological and biophysical processes, a new field of sciences such as system biology, system biophysics, etc., has been created. This system's viewpoints opened new windows to physics and life science. From the biophysical and biological control side, it makes it possible to study many biological processes such as gene regulatory networks (GRN) [1–5], genetic switch [6, 7], and DNA computing [8–12], which is suited for technology developments.

Someone may view cell proliferation systematically to infer new sophisticated medical methods for stem cell differentiation technology, cancer cure, etc. The well-known cell cycle entails an ordered series of macromolecular events that lead to cell division and the production of two daughter cells, each containing chromosomes identical to those of the parental cell [13]. A brief illustration of cell mitosis is depicted in Fig. 1. In normal cells, several genes control the process of cell division. Normal growth requires a balance between the activity of genes, those that promote cell proliferation, and those that suppress it. It also relies on the activities of genes that signal when damaged cells should undergo apoptosis. Cells become cancerous after mutations accumulate in the various genes that control cell proliferation. Deregulated cell proliferation led to cancer and many diseases [14].

From a physical and geometrical viewpoint, cancer cells divide more rapidly than their progenitors and become less dependent on signals from other cells. Cancer cells even evade programmed cell death, even though their multiple abnormalities would normally make them prime targets for apoptosis. In the late stages of cancer, cells break through normal tissue boundaries and metastasize to new sites in the body. In other words, cancer can occur when cell division rates accelerate or by inhibition of normal controls on the cell cycle arrest or programmed cell death. From a control system viewpoint, cancer cell dynamics are different from normal cell dynamics. Hence, by dynamic modeling of normal cell proliferation and then by designing an appropriate control strategy it would be possible to prevent cells from becoming cancerous during cell proliferation and differentiation.

For the modeling aim, a novel scanning ion conductance microscopy technique has been reported in [15] for assessing the volume of living cells and the method gives a high-resolution characterization of dynamic changes in cell volume without change in cell functionality. Volume growth rate and division probability functions for mammalian cells have been determined as functions of cell volume [16], and

[a]e-mail: sharifi@qut.ac.ir

integral equations are derived from the distribution of birth volumes in successive generations [17]. Despite biological diversity, the dynamics of a single cell volume exhibit common basic features in various types of cells [18]. In some mathematical models [19], the surface S and volume V of a cell is considered as $S = \beta V^n$, $\beta > 0$, where β is a positive real number, and the main result is that the volume should grow at least with a third power of the time. In [20], a linear time increase for dry mass is found, while volume growth curves are either quadratic or exponential, depending on the relative contributions of metabolism and transport to cell water. An interesting side of cells' inherent biological processes is the beautiful physical geometry representations [21, 22]. Here, we will derive equations for cell radius more generally with physical insight and without assumption on the type of growth, i.e., spherical, linear, or exponential growth, and also the control action is taken into account.

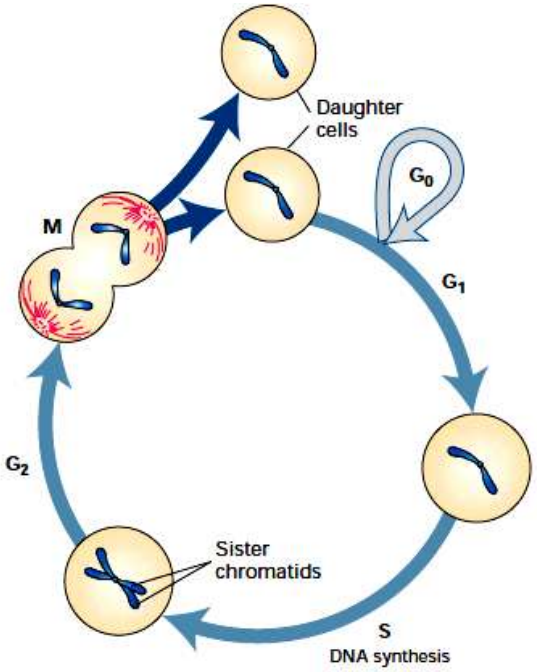


Fig. 1: Major phases of the cell cycle: G1 is the period between "birth" of a cell and the initiation of DNA synthesis. In the S phase, a replicated chromosome consists of two daughter DNA molecules, and associated chromosomal proteins (sister chromatid) will be produced. The end of G2 is marked by the onset of mitosis followed by the division of the cytoplasm to yield two daughter cells. The G1, S, and G2 phases are collectively referred to as interphase. The non-proliferating cells leave the cell cycle in G1 and enter the G0 state [13].

Assumptions and Notations

Let us define some notations used in this paper. We denote the mass and volume of a cell by m and V, respectively. The inner product between two vectors $\vec{A}$ and $\vec{B}$ is

$$\langle \vec{A}, \vec{B} \rangle = \vec{A} \cdot \vec{B}, \tag{1}$$

and $\|\vec{A}\| = \sqrt{\langle \vec{A}, \vec{A} \rangle}$ is the Euclidean norm. The vector $\vec{r}$ denotes the position vector. For instance, in spherical coordinates, $\vec{r} = (r, \theta, \varphi)$. For a scalar function $f(\vec{r}) = f(r, \theta, \varphi)$, one has

$$\nabla f = \partial_r f \, \hat{r} + \frac{1}{r} \partial_\theta f \, \hat{\theta} + \frac{1}{r \sin\theta} \partial_\varphi f \, \hat{\varphi}. \tag{2}$$

$$\nabla^2 f = \frac{1}{r^2} \partial_r \left(r^2 \partial_r f \right) + \frac{1}{r^2 \sin\theta} \partial_\theta \left(\sin\theta \, \partial_\theta f \right) + \frac{1}{r^2 \sin^2\theta} \partial_\varphi^2 f. \tag{3}$$

To develop a model for dynamic cell proliferation, we assume the following conditions:

1. The nutrition (mass) diffuses from a homogeneous medium into a cell and vice versa.
2. The temperature is constant and its variation is negligible.
3. The synthesis of cell mass involves the by-now-familiar processes of transcription and translation.
4. The mass and volume for some time interval are increasing, and in another interval are decreasing. The mass and volume are functions $m(t, \vec{r})$ and $V(t, \vec{r})$.

2 Proliferation Kinetics of Cell Mass Density and Radius

First of all, we visualize a single-parent cell with an arbitrary shape. It has a radius vector function $\vec{R}(t, \vec{r})$ and a mass density $\rho(t, \vec{r})$. The cell imports water and some chemical particles needed for growth and some biochemical reactions and other processes will occur. In physical view those processes have important consequences: the variation of volume and mass. Before the division starts time, the cell mass and volume increase. In the division phase, the parent cell growth will divide into daughter cells. To model this phase, we imagine that one daughter cell is a newly created parent cell that exports the second daughter cell like an ensemble of mass outside itself, see Fig. 2. Then with this idea, we can assume that we have two general physical phases in cell proliferation: first is the mass import and expansion, and the second is mass export and contraction.

Mass current and flow. The time rate of mass is called the mass current, i.e.

$$I_m = \frac{dm}{dt}. \tag{4}$$

The mass flux is defined as $J_m = I_m / A$, where A is the surface section of volume mass transit. If the direction of mass transit is denoted by $\vec{n}$, then $\vec{J}_m = J_m \vec{n}$ is the mass flow [23].

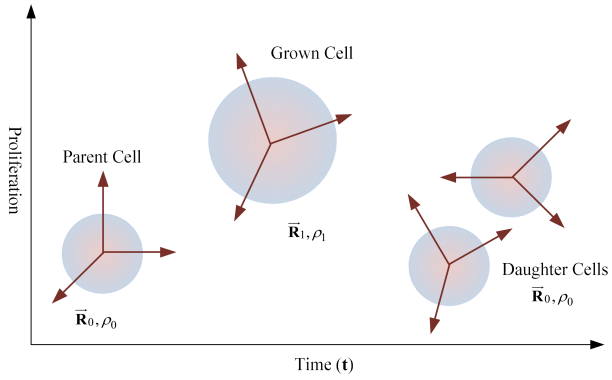


Fig. 2: A simple schematic that shows a mitosis cell division: in the initial time of growth the cell radius increases to a new radius, not equal to twice the initial radius, and then after some time when the division starts, the radius decreases to a new decreased radius for a cell.

Cell mass diffusion and Fick's laws. Molecules will move from where they are at a high concentration to where they are at a lower concentration. They diffuse down a concentration gradient. Fick's law is used to measure the rate of diffusion. The larger the area and difference in concentration cause thinner surface and quicker rate. By this law we have

$$\vec{J}_m + D\nabla\rho = 0, \tag{5}$$

where $\vec{J}_m$ is the mass current flow, D is the diffusion coefficient and $\rho(t,\vec{r})$ is the mass density. By using Fick's first law and the continuity equation, i.e.

$$\nabla\cdot\vec{J}_m + \partial_t\rho = 0, \tag{6}$$

one has the well-known Fick's second law of diffusion:

$$\partial_t\rho = -\nabla\cdot(D\nabla\rho). \tag{7}$$

For an isotropic diffusion coefficient, this equation reduces to

$$\partial_t\rho = -D\nabla^2\rho. \tag{8}$$

Force on the Cell Surface. Let $\vec{R} = \vec{R}(t,\vec{r})$ be the cell vector function at time t and in the direction $\vec{r}$. Let us define* $m_{\vec{R}} = c_1\rho\|\vec{R}\|$ as the mass distributed along the radius vector $\vec{R}$ in the cell, where c_1 is a dimensional correction constant. By Newton's second law, the force exerted along the vector $\vec{R}$ on the cell surface is

$$\vec{F} = \frac{d\vec{P}}{dt}, \tag{9}$$

*More generally, one may consider a nonlinear relation $m_{\vec{R}} = f(\rho)\|\vec{R}\|$, where $f(\rho)$ is a scalar nonlinear function. For simplicity, we restrict ourselves here to the linear case $f(\rho) = c_1\rho$.

where

$$\vec{P} = m_{\vec{R}}\partial_t\vec{R} = c_1\rho\|\vec{R}\|\partial_t\vec{R}. \tag{10}$$

Thus we arrive at†

$$\vec{F} = \partial_t\vec{P} = c_1\partial_t\left(\rho\|\vec{R}\|\partial_t\vec{R}\right). \tag{11}$$

Since the force $\vec{F}$ is proportional to the mass flow $\vec{J}_m$, then likewise by variation of mass, the force direction and amplitude will change. Hence, we postulate heuristically a law that relates the cell surface force with diffused mass‡.

The Cell Force–Mass Divergence Law: the force divergence is proportional to the intercellular mass density up to the transportability constant c:

$$\nabla\cdot\vec{F} = c\rho. \tag{12}$$

By using the force divergence law (12) together with Eq. (2), and the vector calculus identity [24]

$$\nabla\cdot(g\vec{A}) = \nabla g\cdot\vec{A} + g\nabla\cdot\vec{A}, \tag{13}$$

for the scalar $g = c_1\rho\|\vec{R}\|$ and the vector $\vec{A} = \partial_t\vec{R}$, we obtain

$$\begin{aligned}
&\partial_t\rho\,\|\vec{R}\|\nabla\cdot(\partial_t\vec{R}) + \rho\,\partial_t\|\vec{R}\|\nabla\cdot(\partial_t\vec{R})\\
&\quad+\rho\,\|\vec{R}\|\partial_t\nabla\cdot(\partial_t\vec{R})\\
&\quad+\nabla\left(\partial_t\rho\,\|\vec{R}\|\right)\cdot\partial_t\vec{R}\\
&\quad+\nabla\left(\rho\,\partial_t\|\vec{R}\|\right)\cdot\partial_t\vec{R} - k\rho = 0.
\end{aligned} \tag{14}$$

Here $k = c/c_1$. Equation (17) is a scalar equation; hence it does not directly determine the cell radius vector. The proof of this equation is as follows. From (11), one has

$$\vec{F} = \partial_t\vec{P} = \partial_t\left(c_1\rho\|\vec{R}\|\partial_t\vec{R}\right). \tag{15}$$

$$\begin{aligned}
\nabla\cdot\vec{F} &= \nabla\cdot\left(\partial_t\vec{R}\,\partial_t(c_1\rho\|\vec{R}\|) + c_1\rho\|\vec{R}\|\,\partial_t^2\vec{R}\right)\\
&= \left\langle\nabla(c_1\rho\|\vec{R}\|),\partial_t^2\vec{R}\right\rangle\\
&\quad+\left\langle\nabla\left(\partial_t(c_1\rho\|\vec{R}\|)\right),\partial_t\vec{R}\right\rangle\\
&\quad+\partial_t(c_1\rho\|\vec{R}\|)\nabla\cdot(\partial_t\vec{R})\\
&\quad+c_1\rho\|\vec{R}\|\nabla\cdot(\partial_t^2\vec{R}) = c\rho.
\end{aligned} \tag{16}$$

†Since $\vec{P} = c_1\rho(t,\vec{r})\,\|\vec{R}(t,\vec{r})\|\,\partial_t\vec{R}(t,\vec{r}) = \vec{P}(t,\vec{r})$ and $\vec{r}$ does not depend on time, $\partial_t\vec{r} = 0$, the total time derivative of $\vec{P}$ reduces to the partial time derivative:

$$\frac{d\vec{P}}{dt} = \frac{\partial\vec{P}}{\partial t} + \frac{\partial\vec{P}}{\partial\vec{r}}\cdot\frac{\partial\vec{r}}{\partial t} = \frac{\partial\vec{P}}{\partial t}.$$

‡This law is the analogue of Gauss' divergence law for the electric displacement field $\vec{D}$ and charge density ρ [24], namely $\nabla\cdot\vec{D} = \rho$.

Since $\nabla\cdot\vec{F}=c\rho$, one has

$$\left\langle\nabla\left(c_1\rho\|\vec{R}\|\right),\partial_t^2\vec{R}\right\rangle+\left\langle\nabla\left(\partial_t\rho\,\|\vec{R}\|\right),\partial_t\vec{R}\right\rangle + \partial_t\rho\,\|\vec{R}\|\nabla\cdot(\partial_t\vec{R})+\rho\|\vec{R}\|\nabla\cdot(\partial_t^2\vec{R})-k\rho=0. \tag{17}$$

where $k=c/c_1$, and the proof is complete. To obtain the radius vector, we have to extract a vector partial differential equation. Note that

$$\partial_t(\nabla\cdot\vec{F})=\nabla\cdot\partial_t\vec{F}=\nabla\cdot\partial_t^2\left(c_1\rho\|\vec{R}\|\partial_t\vec{R}\right)=c\,\partial_t\rho. \tag{18}$$

Since ∂_t and ∇ are continuous operators, and since ρ and $\vec{R}$ are continuous functions, the operators ∂_t and ∇ commute:

$$[\partial_t,\nabla]=\partial_t\nabla-\nabla\partial_t=0. \tag{19}$$

By substituting from Eq. (7) for $\partial_t\rho$, one finds

$$\nabla\cdot\partial_t^2\left(c_1\rho\|\vec{R}\|\partial_t\vec{R}\right)=-\nabla\cdot(cD\nabla\rho), \tag{20}$$

or equivalently

$$\nabla\cdot\left[\partial_t^2\left(c_1\rho\|\vec{R}\|\partial_t\vec{R}\right)+cD\nabla\rho\right]=0. \tag{21}$$

Thus, the expression inside the divergence is a vector independent of the spatial coordinates. Therefore,

$$\partial_t^2\left(\rho\|\vec{R}\|\partial_t\vec{R}\right)+(kD)\nabla\rho=\alpha\vec{u}(t)+\vec{v}. \tag{22}$$

For a general function $m_{\vec{R}}=f(\rho)\|\vec{R}\|$, Eq. (22) becomes

$$\partial_t^2\left(f(\rho)\|\vec{R}\|\partial_t\vec{R}\right)+(kD)\nabla\rho=\alpha\vec{u}(t)+\vec{v}. \tag{23}$$

The vector $\alpha\vec{u}(t)+\vec{v}$ is interpreted as a control action regulating cell proliferation. Here α and $\vec{v}$ are, respectively, a constant scalar and a constant vector. For instance, the control signals of these events are a small number of heterodimeric protein kinases containing a regulatory subunit, called cyclin, and a catalytic subunit, called cyclin-dependent kinase. These kinases regulate the activities of multiple proteins; for more details, see [13]. The solution of the partial differential equations (7) and (22) gives the time evolution of the cell radius and mass density. By appropriate control, the regulation of proliferation toward the true profile becomes possible.

3 Analysis of the Cell Mass-Volume Diffusion Dynamic

Consider the mass-volume relation $m=\rho V$ and the mass rate $\partial_t m$. By Eq. (7) for constant diffusion, we obtain

$$\frac{\partial_t m}{m}-\frac{\partial_t V}{V}=-D\left(\frac{\nabla^2V}{V}-2\frac{\|\nabla V\|^2}{V^2}\right), \tag{24}$$

where $\nabla^2V=\partial_x^2V+\partial_y^2V+\partial_z^2V$ is the Laplace operator in Cartesian coordinates and

$$\|\nabla V\|^2=(\nabla V,\nabla V)=(\partial_xV)^2+(\partial_yV)^2+(\partial_zV)^2. \tag{25}$$

Based on the last equation, we have these conditions.

(a) The first phase of proliferation, the cell grows. In this phase, both the mass amount and the volume are increasing and hence $\partial_tV,\partial_tm>0$, but evidence shows that the mass increase ratio is more than the volume increase ratio [23], i.e.

$$\frac{\partial_t m}{m}>\frac{\partial_t V}{V}>0. \tag{26}$$

Then one finds for volume the relation

$$V(\partial_xV)^2+V(\partial_yV)^2+V(\partial_zV)^2 - 2\partial_x^2V-2\partial_y^2V-2\partial_z^2V<0. \tag{27}$$

(b) At t_0,

$$\frac{\partial_t m}{m}=\frac{\partial_t V}{V}, \tag{28}$$

which means the cell stops growing. For $t>t_0$ the cell starts to divide, and in the division phase we have $V,m>0$ and $\partial_tV,\partial_tm<0$. Also

$$\frac{\partial_t m}{m}<\frac{\partial_t V}{V}<0, \tag{29}$$

which is equivalent to $\partial_t\rho<0$ or $\nabla\cdot\vec{J}_m>0$, which means that the mass flows outward the cell.

Remark. During a normal exponential-growth cell cycle, both DNA content N and the cytoplasmic mass C double, but asynchronously. In the cell cycle and after mitosis, the cells begin to increase C, thus late G1 cells have a low N/C ratio. In the subsequent step, S-phase, the nucleus content of the cells doubles, thus the $N/C\simeq 1$ is re-established [25]. According to our model, when the volume growth rate of the cell, increase in cytoplasm, is equal to its mass growth rate, increase in nuclear mass, the cell will divide.

4 Conclusion

Here, we derive some partial differential equations for radius vector and mass density of a cell. The systematic views of biological processes are still in their infancy; it is far from clear at this time what will be the ultimate impact of systematic view of cell growth, symmetric and asymmetric. As a future work, someone can model the asymmetric cell proliferation and differentiation.

References

1. M. B. Elowitz and S. Leibler, Nature, vol. 403, no. 20, pp. 335–338, 2000. DOI: https://doi.org/10.1038/35002125

2. L. Chen and K. Aihara, IEEE Transactions on Circuits and Systems, vol. 49, no. 10, 2002. DOI: https://doi.org/10.1109/TCSI.2002.803354
3. B. S. Chen and Y. C. Wang, BMC Bioinformatics, 7, 52, doi:10.1186/1471-2105-7-52, 2006. DOI: https://doi.org/10.1186/1471-2105-7-52
4. R. Jothi et al., Molecular Systems Biology, vol. 5, doi:10.1038/msb.2009.52, 2009. DOI: https://doi.org/10.1038/msb.2009.52
5. R. Siammas et al., Molecular Systems Biology, vol. 7, no. 25, 2011. DOI: https://doi.org/10.1038/msb.2011.25
6. T. S. Gardner, C. R. Cantor and J. J. Collins, Nature, vol. 403, no. 20, pp. 339–342, 2000. DOI: https://doi.org/10.1038/35002131
7. V. A. Rhodius et al., Molecular Systems Biology, vol. 9, doi:10.138/msb.2013.58, 2013. DOI: https://doi.org/10.1038/msb.2013.58
8. L. M. Adleman, Science, 266, 5187, pp. 1021–1024, 1994. DOI: https://doi.org/10.1126/science.7973651
9. Y. Benenson et al., Nature, 414, 6862, pp. 430–434, 2001. DOI: https://doi.org/10.1038/35106533
10. M. N. Stojanovic, T. E. Mitchell and D. Stefanovic, Journal of the American Chemical Society, 124, 14, pp. 3555–3561, 2002. DOI: https://doi.org/10.1021/ja016756v
11. J. I. Farren, J. Hasty, C. R. Cantor and J. J. Collins, PNAS, vol. 100, no. 13, pp. 7714–7719, 2003. DOI: https://doi.org/10.1073/pnas.1332628100
12. J. MacDonald et al., Nano Letters, vol. 6, no. 11, pp. 2598–2603, 2006. DOI: https://doi.org/10.1021/nl0620684
13. H. Lodish et al., *Molecular Cell Biology*, sixth edition, W. H. Freeman, 2007.
14. M. Gthocleous and W. Harris, Trends in Cell Biology, vol. 23, no. 10, pp. 484–491, 2013. DOI: https://doi.org/10.1016/j.tcb.2013.05.004
15. Y. E. Korchev et al., Biophysical Journal, 78, 1, pp. 451–457, 2000. DOI: https://doi.org/10.1016/S0006-3495(00)76605-7
16. W. B. Bonnor, Bulletin of Mathematical Biology, 29, 3, pp. 429–436, 1967. DOI: https://doi.org/10.1007/BF02476582
17. E. C. Anderson et al., Biophysical Journal, 9, 2, pp. 246–263, 1969. DOI: https://doi.org/10.1016/S0006-3495(69)86383-6
18. G. I. Bell, Biophysical Journal, 8, 4, pp. 431–444, 1968. DOI: https://doi.org/10.1016/S0006-3495(68)86498-7
19. U. C. Selhuber et al., Physical Biology, 6, 2, 025015, 2009. DOI: https://doi.org/10.1088/1478-3975/6/2/025015
20. J. A. Hernandez, Bulletin of Mathematical Biology, 69, 5, pp. 1631–1648, 2007. DOI: https://doi.org/10.1007/s11538-006-9181-5
21. M. Terasaka et al., Cell, vol. 154, no. 2, pp. 285–296, 2013. DOI: https://doi.org/10.1016/j.cell.2013.06.043
22. F. M. Wallas, Cell, vol. 154, issue 2, pp. 265–266, 2013. DOI: https://doi.org/10.1016/j.cell.2013.06.044
23. M. Potter and D. C. Wiggart, *Fluid Mechanics*, McGraw-Hill, 2008.
24. T. Chow, *Mathematical Methods for Physicists: A Concise Introduction*, Cambridge University Press, 2003.
25. D. H. Worthington, M. Salamone and D. S. Nachtwey, Cell Proliferation, vol. 9, 2, pp. 119–130, 1976. DOI: https://doi.org/10.1111/j.1365-2184.1976.tb01260.x