

A Mathematical Modeling of Cell Rolling and Division in Human Body by Artificial Intelligence

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Abstract

The present paper deals with regulation and cell division in a high-dimensional, nonlinear process governed by intricate biochemical signaling and physical constraints. Traditional mathematical models, primarily based on Ordinary Differential Equations often struggle to account for the inherent biological "noise" and the massive morphological datasets generated by modern microscopy. This study proposes a novel framework that integrates physics informed Neural Network with stochastic modeling to simulate the mitotic progression of human somatic cells in the present paper we utilize a hybrid approach combining Convolutional Neural Network for real-time automated segmentation of mitotic phases and Recurrent Neural Network to predict the temporal evolution of cyclin-dependent kinase concentrations. The mathematical foundation rests on a modified reaction-diffusion system.

Key Words: Morphological datasets, Automated Segmentation, Mitotic Progression

1. Introduction

In this field a lot of work has already been done we will like to explore the excellent work of some researchers here. KlainD., et al (2024) has clearly explained the multi-Omics single-cell optimal transport model for linking dynamics cellular processes in space and time Kennedy, P., et al

(2025) demonstrated how deep learning architectures track mitosis across diverse cell types and environments. Xie, et al (2025) focused on a clinical application on the title theme to detect aneuploidy and chromosomal errors during division. The present mathematical modelling is executed through Biological Informed Neural Network. This differs from standard AI because they are constrained by the physical laws of biology

2. Mathematical modeling and Governing Equations:

The rolling of blood cells specially leukocyte white blood cells along the vascular endothelium is critical biological process mediated by transient molecular bonds. This rolling behaviour occurs because the shear force of blood flow competes with adhesive forces of selection of proteins when a human cell divides, it produces two daughter cells. If we start with a certain number of cells and they all divide at a constant rate, the growth is exponential.

3. Cell Population Growth Model

The Mathematical formula for the number of cells N at time t will be given as

$$N(t) = N(0) + \sqrt{t} T \log 2$$

- $N(t)$: final number cell
- $N(0)$ initial number cell
- t : Total time elapsed

•T: Doubling time: The time it takes one cell to complete

4. Cell division model

Cell division completes in the following steps

1. G1: Growth
2. S(DNA Synthesis)
3. G2: Preparation
4. M : (Mitosis)

This will be governed by following differential equation and can be solved for M

$$dM/dt = KG2.G1 - (b+d)M$$

This is a first order differential equation which can be solved in a very easy way.

where KG2 : The rate at which cell transition from G2 to mitosis

b: The division rate

d: cell death rate

2. Momentum equation,

$$p = m v$$

$$F = dp/dt$$

$$F \rightarrow = d(p \rightarrow)/dt$$

For constant mass this becomes the familiar form:

$$F = m a \text{ (or } F \rightarrow = m a \rightarrow)$$

$$\int F dt = \Delta p = m(v_2 - v_1)$$

$$F = \rho Q (v_2 - v_1)$$

$$\text{(or in vector form: } F \rightarrow = \rho Q (v \rightarrow_2 - v \rightarrow_1))$$

where

F = force exerted by the fluid (opposite to force on the fluid)

ρ = fluid density

Q = volume flow rate

$$\rho (\partial v \rightarrow / \partial t + (v \rightarrow \cdot \nabla) v \rightarrow) = -\nabla p + \nabla \cdot \tau \rightarrow + \rho g \rightarrow + \text{other body forces}$$

1. Simple exponential growth

In exponentially growing cell populations or early embryonic development the number of cells N(t) follows the exponential model:

$$N(t) = N_0 \times 2^{(t / Td)}$$

or equivalently (continuous form):

$$dN/dt = r N \rightarrow N(t) = N_0 e^{(r t)}$$

where:

$r = \ln(2) / Td$ is the growth rate

$Td \approx$ cell doubling time typically 15–48 hours for many human cell types in culture

This assumes perfect synchrony is not present, every cell divides, and daughter cells immediately start cycling without loss. In reality, most adult human tissues do not grow exponentially forever due to spatial constraints,

differentiation, quiescence, apoptosis, and the Hayflick limit (cells typically stop dividing after ~40–60 divisions due to telomere shortening).

These equations track cell density $n(t, a)$ where a = age (time since last division or entry into a phase):

$$\partial n / \partial t + \partial n / \partial a = -\mu(a) n(t, a)$$

with boundary condition at $a=0$ (birth/division):

$$n(t, 0) = \int \beta(a) n(t, a) da$$

where:

$\mu(a)$ = age-dependent death/loss rate

$\beta(a)$ = age-dependent division (birth) rate

5. Zero Equation Turbulence Model

The zero equation model is a type of Reynolds-averaged Navier-Stokes or Large Eddy Simulation model that estimates turbulence properties based on flow characteristics without solving any set of PDEs for turbulent quantities such as turbulent kinetic energy or turbulent dissipation. This model can be combined with the Prandtl mixing length and Boussinesq approximation to model turbulence in simplified flow situations. These models rely on the same fundamental assumptions and simplifications, which makes them suitable for straightforward cases.

wall does not allow the flow of fluid, thereby preventing pressure from varying in a direction that is perpendicular to the wall.

6. constant Inlet velocity Model

The velocity magnitude and pressure profiles for Newtonian blood flow at the point of stenosis in a stenotic artery under laminar flow conditions are parabolic profile with a pronounced peak at the center. This is indicative of the highest velocity occurring at the site of the smallest cross-sectional area of the arterial lumen. As blood travels through the narrowed section of an artery, it speeds up to maintain a consistent flow rate. The velocity of the blood gradually decreases as it moves further away from the wall

7. Hybrid Composition Model of Cu and Au Particles in blood The combination of copper (Cu) and Au particles is highly regarded in the medical field, particularly for drug delivery systems. Their effectiveness stems from several key characteristics:

- **High Surface Area-to-Volume Ratio:** This allows for high drug loading capacity and precise control over dosage.

- **Biocompatibility:** They possess a low toxicity profile, making them safe for use within the human body.
- **Synergistic Effects:** By combining copper's natural antimicrobial properties with Au chemical stability and inertness, these hybrid particles protect encapsulated medications and allow for extended circulation in the bloodstream.

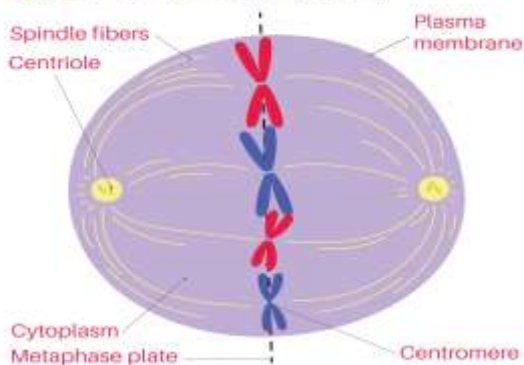
• Concentration and Volume Fractions

Determining the correct concentration of these particles is vital for safety and performance. If the concentration is too low, the treatment is ineffective; if it is too high, it may become toxic.

1. **General Range:** Studies typically utilize volume fractions between **0.1% and 10%**.
2. **Specific Ranges:** Copper particles usually stay between **0.1% and 5%**, while Au ranges from **0.1% up to .03%**

Current Study Parameters: For this specific analysis, a volume fraction of **0.025 (2.5%)** is used for both Cu and Au. This means that for every unit of blood (the base fluid), 0.025 units are occupied by the combined particle.

METAPHASE



Continuity of blood motion

$$\nabla \cdot \mathbf{u} = 0$$

Momentum equation:

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u} \cdot \nabla \mathbf{u} \right) = -\nabla p + \nabla \cdot \boldsymbol{\tau}$$

where \mathbf{u} is velocity, p is pressure, ρ is density, and $\boldsymbol{\tau}$ is the extra stress tensor. The non-Newtonian nature comes from a constitutive relation for $\boldsymbol{\tau} = 2\mu(\dot{\gamma}) \mathbf{D}$, where \mathbf{D} is the rate-of-deformation tensor and $\dot{\gamma} = \sqrt{2 \text{tr}(\mathbf{D}^2)}$ is the shear rate. $\sqrt{\boldsymbol{\tau}} = \sqrt{\boldsymbol{\tau}_0} + \sqrt{(\mu_\infty \dot{\gamma})}$

$$\rightarrow \text{apparent viscosity } \mu(\dot{\gamma}) = [\sqrt{\boldsymbol{\tau}_0/\dot{\gamma}} + \sqrt{\mu_\infty}]^2$$

$$\mu(\dot{\gamma}) = K \dot{\gamma}^{n-1} \quad (n < 1 \text{ for shear-thinning})$$

These are sufficient for many arterial flows but ignore RBC distribution.

Mass conservation (mixture):

$$\nabla \cdot \mathbf{u} = 0$$

Momentum equation.

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u} \cdot \nabla \mathbf{u} \right) = -\nabla p + \nabla \cdot \boldsymbol{\tau}$$

Hematocrit transport (RBC volume fraction ϕ or H):

$$\frac{\partial \phi}{\partial t} + \mathbf{u} \cdot \nabla \phi = \nabla \cdot \mathbf{N}$$

where \mathbf{N} is the diffusive flux of RBCs (from diffusive flux model — DFM):

$\mathbf{N} \approx -D(\phi) \nabla \phi + \text{shear-induced migration terms}$ (often $\sim \dot{\gamma} \phi^2$ terms)

The viscosity $\mu(\phi, \dot{\gamma})$ couples everything — it increases strongly with hematocrit ϕ and decreases with shear rate (shear-thinning)

our aim is to solve or balance this equation

$$F(\text{net}) = F(\text{motor}) + F(\text{drag}) + F(\text{stochastic})$$

Where AI is used to estimate the stochastic variable which represents the unpredictable tug of war during chromosome alignment. The present problem is solved by using the following steps

Multi Omics data: DNA, RNA, Proteins and time lapsed image

Mathematics contribution: Solution of ordinary differential equations for CDK-1/cyclin oscillator

AI Role: A gating mechanism that adjust the ODE parameters in real as changes the shape of the cells. Artificial intelligence analyzes the symmetry of the division of cells mathematical eccentricity of cleavage furrow exceeds a certain threshold, the AI predict a 90%+ probability of chromosomal misalignment. The integration of Artificial Intelligence into the mathematical modeling of human cell division represents a paradigm shift from descriptive biology to predictive engineering by synthesizing high-resolution amazing with stochastic differential equations but we have moved beyond simple clock model towards a holistic Digital Twin model. The cell division process itself doesn't have a single universal "mathematical formula", because it is a complex biological phenomenon involving many molecular steps. However, different aspects of cell division are described mathematically in several useful ways.

Mitosis (somatic cell division — growth, repair, asexual reproduction):

$$(2n \times 2) / 2 = 2n$$

→ One diploid cell (2n) duplicates DNA → divides once → produces 2 identical diploid cells (2n each)

Meiosis (gamete formation — sexual reproduction):

$$(2n \times 2) / 4 = n$$

→ One diploid cell (2n) duplicates DNA → divides twice → produces 4 haploid cells (n each)

These are not dynamic equations but very useful summaries of the final result.

Start with 1 cell → after 10 perfect divisions → $1 \times 2^{10} = 1,024$ cells

Start with 20 bacteria, doubles every 2 hours → after 24 hours (12 doublings):

$$20 \times 2^{12} = 20 \times 4096 = 81,920 \text{ bacteria}$$

Continuous time version (most realistic for large populations):

$$N(t) = N_0 \times e^{(rt)}$$

or equivalently

$$N(t) = N_0 \times 2^{(t / t_d)}$$

Where:

t = time

r = intrinsic growth rate (divisions per unit time)

t_d = doubling time (time for population to double)

e ≈ 2.718 (base of natural logarithm)

This is the classical exponential growth model used in biology, microbiology, cancer research, and cell culture studies.

Here are some visual how exponential cell population growth looks:

The curve starts slow, then accelerates dramatically — classic signature of exponential growth from cell division.)

For realistic cell populations, scientists use more sophisticated equations that include:

Cell cycle duration

Death rate

Unequal division

Size-dependent division probability

Age-structured PDEs → $\partial n / \partial t + \partial n / \partial a = -d(a)n$ (a = age)

Size-structured models → growth rate proportional to volume → $dV/dt = kV$

Stochastic models of division timing

But for most educational and practical purposes, the exponential model $N = N_0 \times 2^n$ or $N(t) = N_0$

$\times e^{(rt)}$ is the key mathematical representation of the cell division process at the population level chemotherapy agents target the mitotic spindle AI models can simulate how specific drugs will force balance the force balance in patient specific cancer cells line before the treatment is even administered. This personalized dosage that maximizes cell death in tumours while in tumours while minimizing damage to healthy dividing tissues

Applications

Application of this research is centred on these pillars

1. Early detection of chromosomal instability

By identifying suitable mathematical derivation in the leverage furrow or spindle symmetry often invisible to the human eye AI can predict the likelihood of abnormal/chromosome count. This has profound implementation for parental screening and understanding the origin of congenital disorder

2. Regenerative Medicine

Stem cell engineering controlling the rate and symmetry of division is critical AI-mathematical models allows researchers to program the environment to ensure spongitum-cells divide into the correct specialised tissues accelerating the development of lab grown tissue

Result and Discussion

As we look towards the Mathematics of life will not be confined to static equations on page. Instead, it will exist as a dynamic, AI-driven simulation capable of accounting for the complexity of human biology. The transition from observing division quantifying every movement ensures that we are not longer just spectator of the cell.

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