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Research Article

Partial Differential Equations Applied to Biological Systems in Healthcare

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Abstract
Partial differential equations are widely used in health-related modeling, especially in oncology, bioengineering, transport phenomena, and tissue dynamics. However, most studies in the literature are predominantly application-oriented and rarely present the full mathematical derivation of the governing models. In this context, the present work proposes a more rigorous approach by combining formal mathematical deduction with practical biomedical applications. The study revisits five classical PDE models—the diffusion equation, the reaction–diffusion equation, the Fisher–KPP equation, the Keller–Segel system, and the Cahn–Hilliard equation—showing how each one arises from conservation laws, constitutive principles, reaction mechanisms, or variational formulations. In addition, illustrative applications and graphical analyses are presented in order to connect the analytical development with health-related interpretations. The relevance of the work lies precisely in bridging the gap between formalism and application, offering a structured contribution to biomathematics, bioengineering, applied mathematics, and mathematical modeling in health.

Keywords: *partial differential equations; mathematical modeling; health applications; biomathematics; bioengineering.*

Introduction

Ordinary partial differential equations gave way to *partial* differential equations when mathematical analysis moved from variation in a single independent variable to phenomena depending on several variables, such as time and space. Their development is historically associated with eighteenth-century mathematical physics, especially the works of d'Alembert and Euler on vibrating strings and the later work of Fourier on heat conduction, which consolidated PDEs as a central language for mechanics, diffusion, elasticity, fluid flow, and other continuum phenomena [1], [2], [3], [11], [5]. In contrast to ordinary differential equations, PDEs involve partial derivatives with respect to two or more independent variables and are particularly useful when the phenomenon under study depends simultaneously on temporal and spatial variation. At present, PDE-based models are widely used in health-related problems, especially in oncology, epidemiology, medical imaging, tissue dynamics, drug diffusion, and bioengineering [25], [7], [8], [9]. However, the screened literature is overwhelmingly application-oriented: most papers focus on simulation, numerical treatment, parameter fitting, or computational performance, while full mathematical derivation of the classical governing models is rarely presented in detail. This article addresses that gap by revisiting the main PDE models applied to health, emphasizing their formal derivation and mathematical structure, while also presenting illustrative applications. The bibliographic survey carried out in ScienceDirect, PubMed, SciELO, and SpringerLink is summarized in the graphs below. The values shown in the following figures correspond to an exploratory bibliographic screening conducted for the period 2020–2025 in PubMed, ScienceDirect, SpringerLink, and SciELO. The purpose of this survey was not to produce an exhaustive bibliometric study, but rather to identify the approximate volume of publications involving partial differential equations in health-related contexts and to observe their predominant areas of application. In the screened literature, the dominant tendency was application-oriented research, whereas complete mathematical derivations of the governing PDE models were rarely presented in a systematic way.

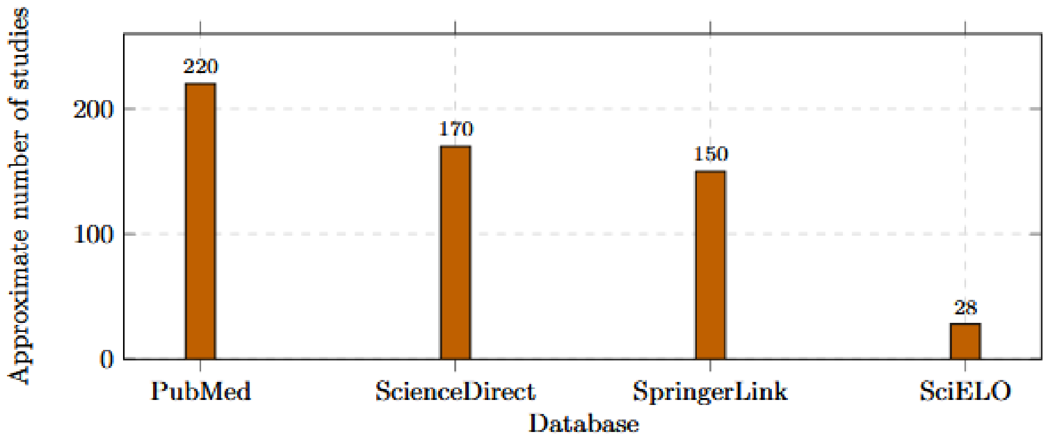

Figure 1: Approximate number of PDE-related health studies retrieved in the exploratory screening for the period 2020--2025.

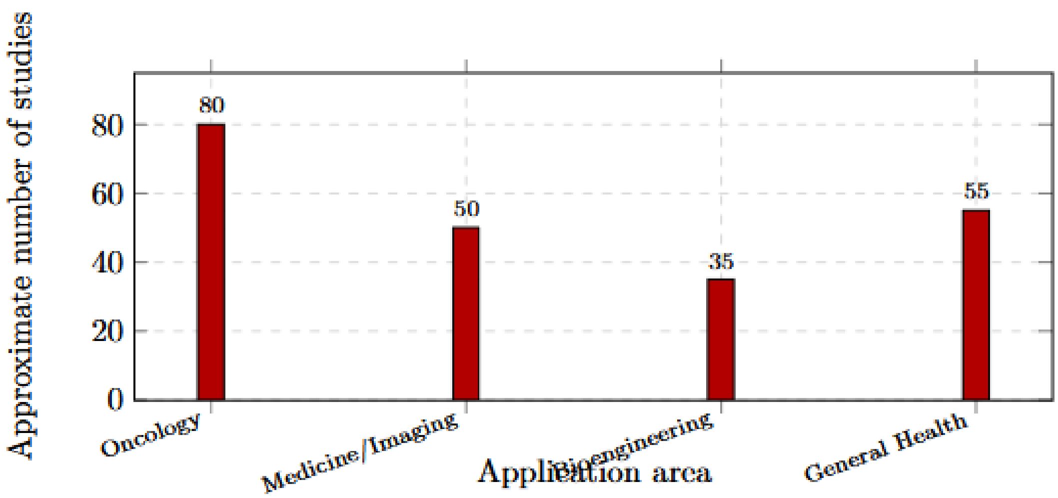

Figure 2: Approximate thematic distribution of PDE-based health studies indexed in PubMed (2020--2025).

In the exploratory PubMed screening, PDE-based studies appeared predominantly in oncology, followed by general health modeling, medical imaging, and bioengineering. This profile is compatible with the biomedical focus of the database and reinforces the strong presence of PDEs in cancer dynamics, diffusion-based imaging models, and physiological transport problems.

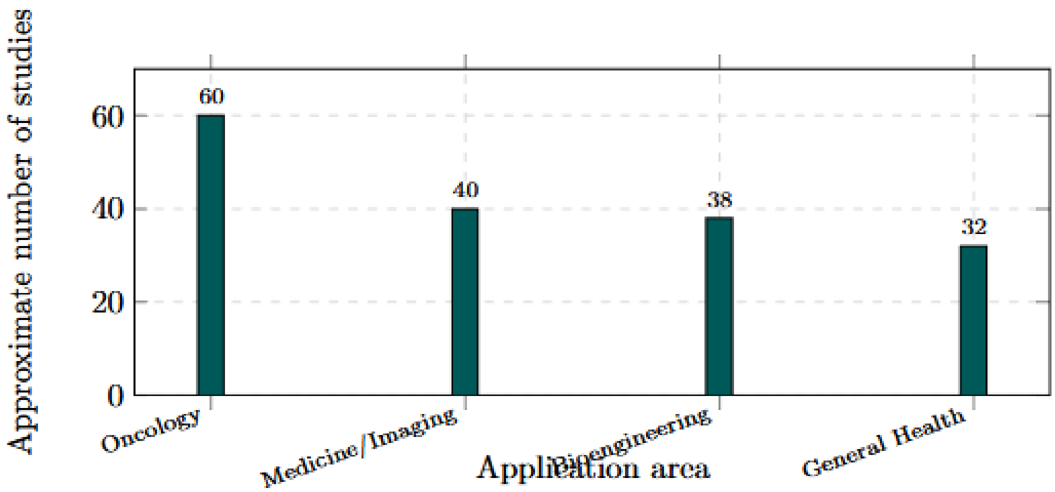

Figure 3: Approximate thematic distribution of PDE-based health studies indexed in ScienceDirect (2020--2025).

In ScienceDirect, the exploratory distribution suggests a strong concentration in oncology, with relevant participation of medical imaging and bioengineering. This indicates that PDEs are frequently employed not only in mechanistic biomedical models, but also in computational and image-based health applications.

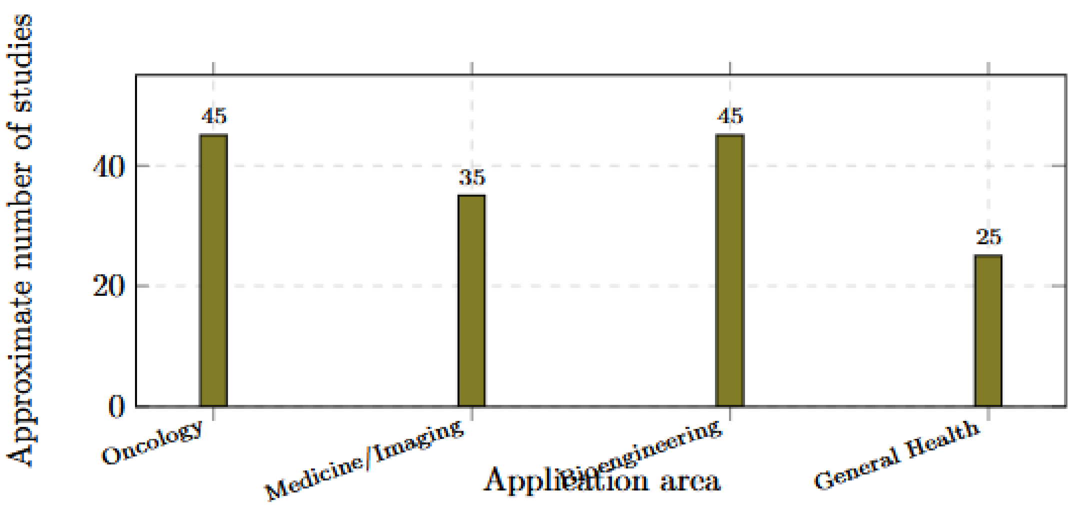

Figure 4: Approximate thematic distribution of PDE-based health studies indexed in SpringerLink (2020--2025).

The exploratory SpringerLink screening shows a more balanced distribution, with particularly strong presence in oncology and bioengineering. This pattern is consistent with the visibility of PDE-based modeling in tissue engineering, transport phenomena, tumor dynamics, and computational biomedical systems.

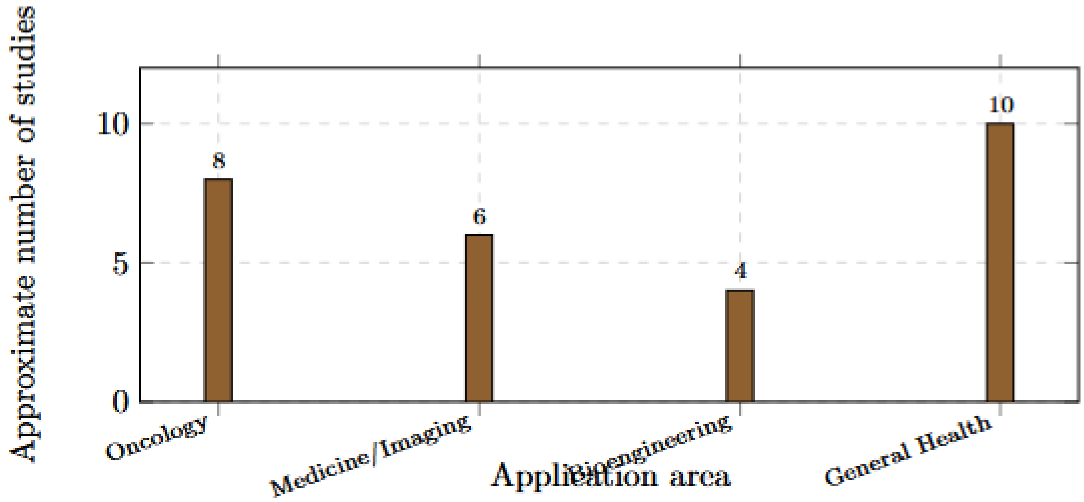

Figure 5: Approximate thematic distribution of PDE-based health studies indexed in SciELO (2020--2025).

In SciELO, the retrieved volume was smaller, with stronger concentration in general health and oncology-oriented applications. Even in this reduced corpus, the dominant tendency remained application-based, with limited emphasis on full mathematical derivation of the underlying PDE models.

Objective

The main objective of this study is to analyze classical partial differential equation models applied to biological systems in health, with emphasis on both their mathematical derivation and their practical interpretation. More specifically, the article seeks to revisit the main PDE formulations found in the literature and to present them in a more explicit and structured way, showing how these models arise from physical, biological, and mathematical assumptions. Unlike many application-oriented studies, this work aims to make the formal analytical construction of the equations more accessible to the reader. In addition, the study intends to relate the mathematical formalism to health-related applications, especially in areas such as oncology, bioengineering, diffusion processes, and medical modeling. Another objective is to organize the state of the art on PDE applications in health, identifying recurrent models and their main contexts of use. Thus, the article combines formal derivation, theoretical interpretation, and illustrative applications within a unified analytical framework.

Justification

The justification for this study lies in the fact that partial differential equations occupy a central role in contemporary mathematical modeling, especially in problems involving time-space dynamics, diffusion, transport, reaction, and biological interactions in health-related systems. Although there is a significant number of publications involving PDE-based applications in the scientific literature, most studies are predominantly focused on simulation, numerical implementation, or applied interpretation, with little emphasis on mathematical derivation and formal analytical development. As a consequence, many readers have access only to the final form of the equations, without a clear understanding of how these models are constructed or why their terms appear in a given formulation. This represents an important gap in the literature. In this sense, the present work is justified by the need to provide a more rigorous and didactic treatment of classical PDE models, combining the mathematical formalism behind their derivation with their practical use in health applications. Such an approach is relevant not only for mathematical comprehension, but also for the correct interpretation, adaptation, and critical use of these models in interdisciplinary research.

Methodology

The methodological structure adopted in this study is summarized in Table 1. The research is bibliographic, descriptive, and analytical, with emphasis on the formal derivation of classical partial differential equation models and their application to health-related problems.

Aspect	Description
Nature of the research	This study is bibliographic, descriptive, and analytical, with a qualitative approach focused on classical epidemiological models formulated through ordinary differential equations.
Literature survey	Books, scientific articles, theses, and dissertations will be consulted in order to identify the main epidemiological models established in the literature and their most common applications in health.
Theoretical and mathematical analysis	The selected models will be examined with emphasis on their assumptions, biological interpretation, and mathematical structure. A central part of the study consists of explicitly deriving the equations step by step, showing how the models emerge from biological hypotheses and balance relations.
Wolfram Mathematica	*Wolfram Mathematica* will be used as a symbolic computational tool to assist in algebraic manipulation, expansion of expressions, and verification of intermediate analytical steps.
MATLAB	*MATLAB* will be employed for graphical representations whenever necessary, especially to illustrate the behavior of the models and support the discussion of epidemiological applications.
Python	Python will be used for numerical simulations in selected examples involving specific parameter values, allowing the numerical solution of differential equation systems and the generation of illustrative plots.
Purpose of the methodology	The methodology combines bibliographic review, formal mathematical derivation, symbolic computation, and numerical illustration, providing a clearer understanding of both the theoretical foundation and the practical relevance of epidemiological models.

Table 1 - Methodological structure of the study

Results and discussion

The Diffusion Equation

Joseph Fourier (1768–1830) was a French mathematician and physicist born in Auxerre, France. He studied in the intellectual environment created by the French scientific schools of the late eighteenth century, taught at the *École Normale*, and later worked at the *École Polytechnique*. Fourier became one of the central figures in mathematical physics after formulating the analytical theory of heat conduction, published in 1822, where he introduced the differential model that later became known as the heat or diffusion equation [10], [11], [12]. Although originally proposed for thermal conduction, the same mathematical structure is now widely used in health-related problems such as drug diffusion in tissues, oxygen transport, heat propagation in biological media, and controlled release systems [12], [13], [14].

Formal derivation of the model

Let $u(x,t)$ denote the concentration of a diffusing substance at position x and time t. Consider a one-dimensional tissue segment $[x, x+\Delta x]$. The total amount of substance in this small interval is approximately

$$\int_{x}^{x+\Delta x} u\,(\xi,t)\, d\xi. \tag{1}$$

The rate of variation of this amount is given by

$$\frac{d}{dt}\int_{x}^{x+\Delta x} u\,(\xi,t)\, d\xi. \tag{2}$$

By conservation of mass, this variation must equal the net flux entering the interval. Let $J(x,t)$ be the flux. Then

$$\frac{d}{dt}\int_{x}^{x+\Delta x} u\,(\xi,t)\,d\xi = J(x,t) - J(x+\Delta x,t). \tag{3}$$

Using the Leibniz rule, since the interval endpoints are fixed,

$$\int_{x}^{x+\Delta x} \frac{\partial u}{\partial t}(\xi,t)\,d\xi = J(x,t) - J(x+\Delta x,t). \tag{4}$$

Now divide both sides by Δx:

$$\frac{1}{\Delta x}\int_{x}^{x+\Delta x} \frac{\partial u}{\partial t}(\xi,t)\,d\xi = \frac{J(x,t)-J(x+\Delta x,t)}{\Delta x}. \tag{5}$$

Taking the limit as $\Delta x \to 0$, the left-hand side becomes

$$\frac{\partial u}{\partial t}(x,t), \tag{6}$$

while the right-hand side becomes

$$-\frac{\partial J}{\partial x}(x,t). \tag{7}$$

Therefore,

$$\frac{\partial u}{\partial t} = -\frac{\partial J}{\partial x}. \tag{8}$$

Equation (8) is the local conservation law.

To close the model, we use Fourier–Fick's constitutive law: the flux is proportional to the negative gradient of concentration,

$$J(x,t) = -D\frac{\partial u}{\partial x}(x,t), \tag{9}$$

where $D > 0$ is the diffusion coefficient.

Differentiate (9) with respect to x:

$$\frac{\partial J}{\partial x} = -\frac{\partial}{\partial x}\left(D\frac{\partial u}{\partial x}\right). \tag{10}$$

If D is constant, then

$$\frac{\partial J}{\partial x} = -D\frac{\partial^2 u}{\partial x^2}. \tag{11}$$

Substituting (11) into (8), we obtain

$$\frac{\partial u}{\partial t} = -\left(-D\frac{\partial^2 u}{\partial x^2}\right), \tag{12}$$

that is,

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2}. \tag{13}$$

Equation (13) is the classical one-dimensional diffusion equation, also known historically as the heat equation.

Importance of the model

The importance of the diffusion equation lies in the fact that it provides the simplest continuum description of transport driven by gradients. In the health sciences, this equation is fundamental for describing the spread of drugs in tissue, oxygen transport, movement of nutrients, temperature diffusion in biological media, and diffusion-controlled release systems [13], [14]. Its mathematical simplicity also makes it an ideal starting point for more advanced PDE models involving reaction, advection, anisotropy, or multiphase biological media.

Application of the model

As an illustrative health-related application, consider the diffusion of a drug across a thin one-dimensional tissue slab of length L. Suppose the boundaries are absorbing, so that the concentration vanishes at both ends:

$$u(0,t) = 0, \qquad u(L,t) = 0. \tag{14}$$

Assume that the initial drug concentration is nonuniform and given by

$$u(x,0) = C_0 \sin\left(\frac{\pi x}{L}\right). \tag{15}$$

We now solve the problem

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2}, \qquad 0 < x < L,\ t > 0, \tag{16}$$

subject to (14) and (15).

Solution by separation of variables

Assume a separated solution of the form

$$u(x,t) = X(x)T(t). \tag{17}$$

Then

$$\frac{\partial u}{\partial t} = X(x)T'(t) \tag{18}$$

and

$$\frac{\partial^2 u}{\partial x^2} = X''(x)T(t). \tag{19}$$

Substituting into (16),

$$X(x)T'(t) = DX''(x)T(t). \tag{20}$$

Divide both sides by $DX(x)T(t)$:

$$\frac{T'(t)}{D\,T(t)} = \frac{X''(x)}{X(x)}. \tag{21}$$

The left-hand side depends only on t, and the right-hand side only on x, so both must be equal to a constant, say $-\lambda$:

$$\frac{T'}{DT} = -\lambda, \qquad \frac{X''}{X} = -\lambda. \tag{22}$$

Thus, we obtain the system

$$T'(t) + D\lambda T(t) = 0, \tag{23}$$

$$X''(x) + \lambda X(x) = 0. \tag{24}$$

The boundary conditions (14) imply

$$X(0) = 0, \qquad X(L) = 0. \tag{25}$$

The eigenvalue problem (24)–(25) has nontrivial solutions only for

$$\lambda_n = \left(\frac{n\pi}{L}\right)^2, \qquad n = 1,2,3,\ldots \tag{26}$$

with eigenfunctions

$$X_n(x) = \sin\left(\frac{n\pi x}{L}\right). \tag{27}$$

For each n, equation (23) becomes

$$T_n{}'(t) + D\left(\frac{n\pi}{L}\right)^2 T_n(t) = 0, \tag{28}$$

whose solution is

$$T_n(t) = A_n \exp\left[-D\left(\frac{n\pi}{L}\right)^2 t\right]. \tag{29}$$

Therefore, the general solution is

$$u(x,t) = \sum_{n=1}^{\infty} A_n \sin\left(\frac{n\pi x}{L}\right) \exp\left[-D\left(\frac{n\pi}{L}\right)^2 t\right]. \tag{30}$$

Now impose the initial condition (15):

$$u(x,0) = \sum_{n=1}^{\infty} A_n \sin\left(\frac{n\pi x}{L}\right) = C_0 \sin\left(\frac{\pi x}{L}\right). \tag{31}$$

By orthogonality of the sine basis, it follows that

$$A_1 = C_0 \qquad \text{and} \qquad A_n = 0 \text{ for } n \geq 2. \tag{32}$$

Hence the solution reduces to

$$u(x,t) = C_0 \sin\left(\frac{\pi x}{L}\right) \exp\left[-D\left(\frac{\pi}{L}\right)^2 t\right]. \tag{33}$$

Application exercise
Consider a drug diffusing through a tissue layer of length

$$L = 1 \text{ cm}, \tag{34}$$

with diffusion coefficient

$$D = 0.02 \text{ cm}^2/\text{h}, \tag{35}$$

and initial concentration amplitude

$$C_0 = 100 \text{ mg/cm}^3. \tag{36}$$

Using (33), the concentration is

$$u(x,t) = 100 \sin(\pi x) \exp[-0.02\pi^2 t]. \tag{37}$$

We evaluate the concentration at the midpoint $x = 0.5$, where $\sin(\pi/2) = 1$:

$$u(0.5,t) = 100 \exp[-0.02\pi^2 t]. \tag{38}$$

For $t = 0$,

$$u(0.5,0) = 100. \tag{39}$$

For $t = 5$,

$$u(0.5,5) = 100 \exp[-0.02\pi^2 \cdot 5]. \tag{40}$$

Since

$$0.02\pi^2 \cdot 5 \approx 0.98696, \tag{41}$$

we obtain

$$u(0.5,5) \approx 100e^{-0.98696} \approx 37.27. \quad (42)$$

For $t = 10$,

$$u(0.5,10) = 100\exp[-0.02\pi^2 \cdot 10]. \quad (43)$$

Since

$$0.02\pi^2 \cdot 10 \approx 1.97392, \quad (44)$$

it follows that

$$u(0.5,10) \approx 100e^{-1.97392} \approx 13.90. \quad (45)$$

For $t = 15$,

$$u(0.5,15) = 100\exp[-0.02\pi^2 \cdot 15]. \quad (46)$$

Since

$$0.02\pi^2 \cdot 15 \approx 2.96088, \quad (47)$$

we obtain

$$u(0.5,15) \approx 100e^{-2.96088} \approx 5.18. \quad (48)$$

Thus, the concentration at the center of the tissue decreases rapidly over time due to diffusion and boundary absorption.

Graphical representation.

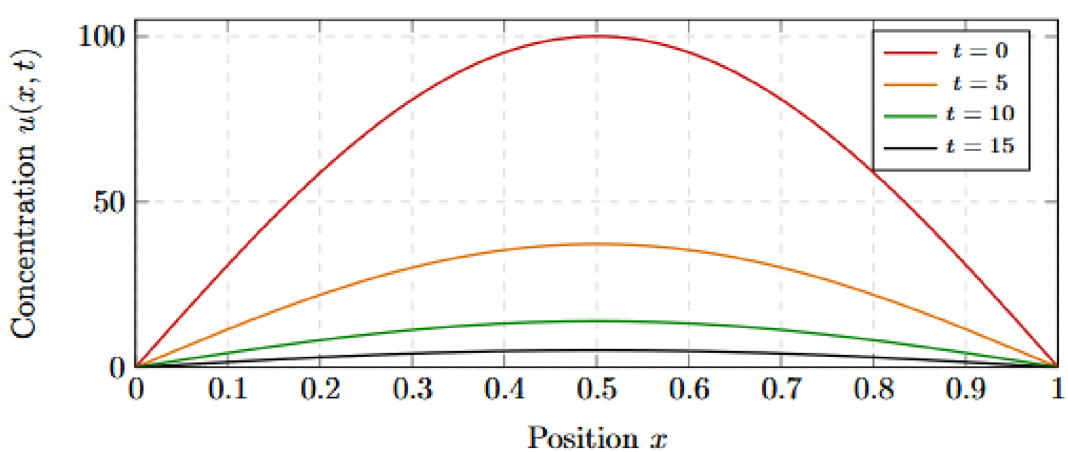

Figure 6: Drug concentration profiles in a one-dimensional tissue slab governed by the diffusion equation.

This application shows that the diffusion equation provides a clear and rigorous description of how a substance spreads and attenuates in tissue over time. From the health perspective, this type of model is relevant for drug delivery, transport in biological media, and diffusion-controlled therapeutic systems. From the mathematical perspective, its importance lies in the fact that it is derived directly from conservation principles and constitutive laws, making the origin of the model transparent. This is precisely the type of formal structure that is often omitted in application-oriented studies, but which is essential for a deeper understanding of PDE-based modeling in health.

The Reaction–Diffusion Equation

Alan Mathison Turing (1912–1954) was a British mathematician, logician, and theoretical scientist educated at King's College, Cambridge, and later at Princeton University. Although widely recognized for his foundational work in computation and logic, Turing also made a major contribution to mathematical biology through his 1952 paper on morphogenesis, in which he showed how diffusion combined with local reaction mechanisms can generate biological pattern formation [15], [16], [17]. In this sense, while the diffusion term has its historical roots in Fourier and Fick, the reaction–diffusion framework became a central mathematical model for biological and health-related systems through Turing's work. Today, reaction–diffusion equations are widely used in oncology, tissue dynamics, drug transport with consumption, epidemiology, wound healing, and biological pattern formation [17], [23], [25].

Formal derivation of the model

Let $u(x,t)$ denote the concentration of a biological substance, chemical agent, drug, nutrient, or cell density at position x and time t. Consider again a one-dimensional control interval $[x, x+\Delta x]$. The total amount of substance in this interval is

$$\int_x^{x+\Delta x} u\,(\xi,t)\,d\xi. \tag{49}$$

The rate of change of this amount is

$$\frac{d}{dt}\int_x^{x+\Delta x} u\,(\xi,t)\,d\xi. \tag{50}$$

In a reaction–diffusion system, the variation of mass is not explained only by flux across the boundaries. It also includes local production or consumption inside the interval. Let $J(x,t)$ denote the diffusive flux and let $R(u)$ denote the local reaction term. Then the mass balance law becomes

$$\frac{d}{dt}\int_x^{x+\Delta x} u\,(\xi,t)\,d\xi = J(x,t) - J(x+\Delta x,t) + \int_x^{x+\Delta x} R\left(u(\xi,t)\right)d\xi. \tag{51}$$

Using the Leibniz rule on the left-hand side,

$$\int_x^{x+\Delta x} \frac{\partial u}{\partial t}(\xi,t)\,d\xi = J(x,t) - J(x+\Delta x,t) + \int_x^{x+\Delta x} R\left(u(\xi,t)\right)d\xi. \tag{52}$$

Now divide both sides by Δx:

$$\frac{1}{\Delta x}\int_x^{x+\Delta x} \frac{\partial u}{\partial t}(\xi,t)\,d\xi = \frac{J(x,t)-J(x+\Delta x,t)}{\Delta x} + \frac{1}{\Delta x}\int_x^{x+\Delta x} R\left(u(\xi,t)\right)d\xi. \tag{53}$$

Take the limit as $\Delta x \to 0$. Then:

$$\frac{1}{\Delta x}\int_x^{x+\Delta x} \frac{\partial u}{\partial t}(\xi,t)\,d\xi \to \frac{\partial u}{\partial t}(x,t), \tag{54}$$

$$\frac{J(x,t)-J(x+\Delta x,t)}{\Delta x} \to -\frac{\partial J}{\partial x}(x,t), \tag{55}$$

and

$$\frac{1}{\Delta x}\int_{x}^{x+\Delta x} R\left(u(\xi,t)\right)d\xi \to R\big(u(x,t)\big). \tag{56}$$

Therefore, the local balance law is

$$\frac{\partial u}{\partial t} = -\frac{\partial J}{\partial x} + R(u). \tag{57}$$

Now impose the constitutive law of diffusion, namely Fourier–Fick law,

$$J(x,t) = -D\frac{\partial u}{\partial x}(x,t), \tag{58}$$

where $D > 0$ is the diffusion coefficient.
Differentiate (58) with respect to x:

$$\frac{\partial J}{\partial x} = -\frac{\partial}{\partial x}\left(D\frac{\partial u}{\partial x}\right). \tag{59}$$

If D is constant, then

$$\frac{\partial J}{\partial x} = -D\frac{\partial^2 u}{\partial x^2}. \tag{60}$$

Substituting (60) into (57), we obtain

$$\frac{\partial u}{\partial t} = -\left(-D\frac{\partial^2 u}{\partial x^2}\right) + R(u), \tag{61}$$

that is,

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + R(u). \tag{62}$$

Equation (62) is the general one-dimensional reaction–diffusion equation.

Interpretation of the terms
Equation (62) contains two distinct mechanisms:

$$D\frac{\partial^2 u}{\partial x^2}, \tag{63}$$

which models spatial spreading due to diffusion, and

$$R(u), \tag{64}$$

which models local kinetics, such as production, decay, proliferation, uptake, or chemical reaction.

Thus, the equation combines transport and local transformation. This is precisely why it is useful in health applications: many biological systems involve both movement in space and reaction in time.

A classical linear reactive case

To build an explicit analytical example, consider a first-order consumption mechanism. Suppose that the substance is locally removed at a rate proportional to its concentration. Then

$$R(u) = -ku, \tag{65}$$

where $k > 0$ is the reaction or decay constant.

Substituting (65) into (62), we obtain

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} - ku. \tag{66}$$

Equation (66) models a diffusing substance that is simultaneously consumed in the medium. In health-related contexts, this can represent, for example, the diffusion of a drug through tissue while the drug is metabolized or absorbed.

Importance of the model

The reaction–diffusion equation is one of the most important partial differential equations in mathematical biology and health modeling because it extends the pure diffusion law to situations in which the transported quantity also changes locally through biological or chemical processes. This makes it suitable for modeling drug diffusion with uptake, oxygen transport with consumption, nutrient diffusion in tumors, cell density with proliferation, wound healing dynamics, and spatial disease propagation [17], [23], [25]. Its importance lies in the fact that it connects conservation principles, constitutive transport laws, and local kinetics within a single formal mathematical framework.

Application of the model

Consider a one-dimensional tissue slab of length L, where a therapeutic substance diffuses and is simultaneously consumed by the tissue. Assume homogeneous absorbing boundaries:

$$u(0,t) = 0, \qquad u(L,t) = 0, \tag{67}$$

and initial concentration

$$u(x,0) = C_0 \sin\left(\frac{\pi x}{L}\right). \tag{68}$$

We now solve

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} - ku, \qquad 0 < x < L,\ t > 0. \tag{69}$$

Solution by separation of variables
Assume again a separated solution

$$u(x,t) = X(x)T(t). \tag{70}$$

Then

$$\frac{\partial u}{\partial t} = X(x)T'(t), \tag{71}$$

and

$$\frac{\partial^2 u}{\partial x^2} = X''(x)T(t). \tag{72}$$

Substituting (70)–(72) into (69),

$$X(x)T'(t) = DX''(x)T(t) - kX(x)T(t). \tag{73}$$

Now divide both sides by $X(x)T(t)$, assuming $X \neq 0$ and $T \neq 0$:

$$\frac{T'(t)}{T(t)} = D\frac{X''(x)}{X(x)} - k. \tag{74}$$

Bring the constant k to the left:

$$\frac{T'(t)}{T(t)} + k = D\frac{X''(x)}{X(x)}. \tag{75}$$

The left-hand side depends only on t, and the right-hand side only on x, so both sides must be equal to a constant. Let this constant be $-\lambda$:

$$\frac{T'}{T} + k = -\lambda, \qquad D\frac{X''}{X} = -\lambda. \tag{76}$$

Thus,

$$\frac{T'}{T} = -(\lambda + k), \tag{77}$$

and

$$X'' + \frac{\lambda}{D}X = 0. \tag{78}$$

So, we obtain the system

$$T'(t) + (\lambda + k)T(t) = 0, \tag{79}$$

$$X''(x) + \frac{\lambda}{D}X(x) = 0. \tag{80}$$

The boundary conditions imply

$$X(0) = 0, \qquad X(L) = 0. \tag{81}$$

The spatial eigenvalue problem (80)–(81) has nontrivial solutions for

$$\frac{\lambda_n}{D} = \left(\frac{n\pi}{L}\right)^2, \qquad n = 1,2,3, \dots \tag{82}$$

hence

$$\lambda_n = D\left(\frac{n\pi}{L}\right)^2. \tag{83}$$

The corresponding eigenfunctions are

$$X_n(x) = \sin\left(\frac{n\pi x}{L}\right). \tag{84}$$

For each n, the temporal equation (79) becomes

$$T_n{}'(t) + \left[D\left(\frac{n\pi}{L}\right)^2 + k\right]T_n(t) = 0. \tag{85}$$

This is a first-order linear ODE. Separating variables,

$$\frac{dT_n}{T_n} = -\left[D\left(\frac{n\pi}{L}\right)^2 + k\right]dt. \tag{86}$$

Integrating both sides,

$$\int \frac{dT_n}{T_n} = -\int \left[D\left(\frac{n\pi}{L}\right)^2 + k\right]dt. \tag{87}$$

Thus,

$$\ln|T_n| = -\left[D\left(\frac{n\pi}{L}\right)^2 + k\right]t + C_n. \tag{88}$$

Exponentiating,

$$T_n(t) = A_n \exp\left\{-\left[D\left(\frac{n\pi}{L}\right)^2 + k\right]t\right\}. \tag{89}$$

Therefore, the general series solution is

$$u(x,t) = \sum_{n=1}^{\infty} A_n \sin\left(\frac{n\pi x}{L}\right)\exp\left\{-\left[D\left(\frac{n\pi}{L}\right)^2 + k\right]t\right\}. \tag{90}$$

Now impose the initial condition (68):

$$u(x,0) = \sum_{n=1}^{\infty} A_n \sin\left(\frac{n\pi x}{L}\right) = C_0 \sin\left(\frac{\pi x}{L}\right). \tag{91}$$

By orthogonality,

$$A_1 = C_0, \qquad A_n = 0 \text{ for } n \geq 2. \tag{92}$$

Hence the solution reduces to

$$u(x,t) = C_0 \sin\left(\frac{\pi x}{L}\right) \exp\left\{-\left[D\left(\frac{\pi}{L}\right)^2 + k\right] t\right\}. \tag{93}$$

This is the explicit solution for the chosen reaction–diffusion problem.

Application exercise

Consider a drug diffusing through a tissue slab of length

$$L = 1 \text{ cm}, \tag{94}$$

with diffusion coefficient

$$D = 0.02 \text{ cm}^2/\text{h}, \tag{95}$$

reaction rate

$$k = 0.08 \text{ h}^{-1}, \tag{96}$$

and initial amplitude

$$C_0 = 100 \text{ mg/cm}^3. \tag{97}$$

Then, from (93),

$$u(x,t) = 100\sin(\pi x)\exp[-(0.02\pi^2 + 0.08)t]. \tag{98}$$

We now evaluate the concentration at the midpoint $x = 0.5$, where $\sin(\pi/2) = 1$:

$$u(0.5,t) = 100\exp[-(0.02\pi^2 + 0.08)t]. \tag{99}$$

First compute the exponential coefficient:

$$0.02\pi^2 + 0.08 \approx 0.02(9.8696) + 0.08 \approx 0.197392 + 0.08 \approx 0.277392. \tag{100}$$

Therefore,

$$u(0.5, t) = 100e^{-0.277392t}. \quad (101)$$

For $t = 0$,

$$u(0.5,0) = 100. \quad (102)$$

For $t = 5$,

$$u(0.5,5) = 100e^{-0.277392\cdot 5} = 100e^{-1.38696}. \quad (103)$$

Since

$$e^{-1.38696} \approx 0.24985, \quad (104)$$

we get

$$u(0.5,5) \approx 24.99. \quad (105)$$

For $t = 10$,

$$u(0.5,10) = 100e^{-0.277392\cdot 10} = 100e^{-2.77392}. \quad (106)$$

Since

$$e^{-2.77392} \approx 0.06236, \quad (107)$$

it follows that

$$u(0.5,10) \approx 6.24. \quad (108)$$

For $t = 15$,

$$u(0.5,15) = 100e^{-0.277392\cdot 15} = 100e^{-4.16088}. \quad (109)$$

Since

$$e^{-4.16088} \approx 0.01558, \quad (110)$$

we obtain

$$u(0.5,15) \approx 1.56. \quad (111)$$

Thus, compared with the pure diffusion case, the concentration decays much faster because the tissue not only spreads the substance but also consumes it.

Graphical representation.

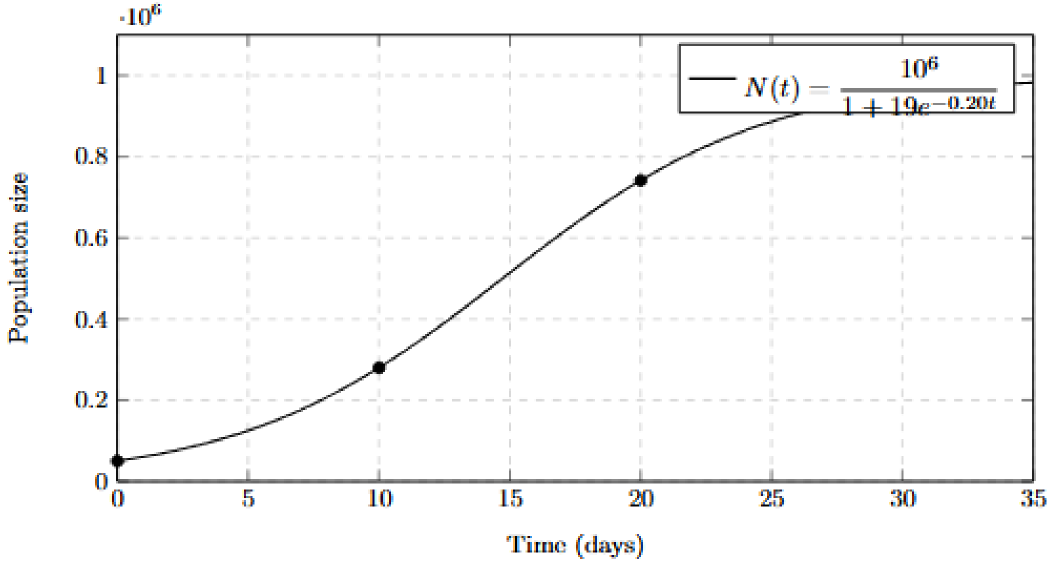

Figure 7: Drug concentration profiles governed by a reaction--diffusion equation with first-order consumption.

This application shows that the reaction–diffusion equation is more realistic than the pure diffusion equation when the transported substance is also transformed locally by the medium. In the present example, the therapeutic agent spreads spatially but is simultaneously consumed by the tissue, leading to a faster decrease in concentration than in the purely diffusive case. This type of model is highly relevant in medicine, pharmacokinetics, tissue engineering, and biomathematics, since many biomedical processes involve exactly this combination of spatial transport and local reaction. From the formal point of view, the model is also important because it arises directly from conservation laws plus a constitutive flux law and a reaction mechanism, making the mathematical origin of the PDE completely explicit.

The Fisher–KPP Equation

Ronald Aylmer Fisher (1890–1962) was a British statistician, geneticist, and mathematical biologist educated at Gonville and Caius College, Cambridge. He is one of the central figures in modern statistics and population genetics. In 1937, Fisher proposed a partial differential equation to describe the spatial spread of an advantageous gene in a population, combining diffusion with logistic growth [20], [21]. In the same year, Andrey Kolmogorov, Ivan Petrovsky, and Nikolai Piskunov independently studied the same class of equation and established important analytical results on traveling waves and propagation speed [22]. For this reason, the model is widely known as the Fisher–KPP equation. Although originally developed in population genetics, it is now broadly used in health-related applications such as tumor invasion, spatial epidemiology, tissue regeneration, and biological spread phenomena [23], [24], [25].

Formal derivation of the model

Let $u(x,t)$ denote the density of a biological population, cellular concentration, or infected fraction at position x and time t. The starting point is the reaction–diffusion balance law

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + R(u), \tag{112}$$

where $D > 0$ is the diffusion coefficient and $R(u)$ is the local reaction term.

The Fisher–KPP model is obtained when the local kinetics follow the logistic law. Recall first the classical Malthusian ODE

$$\frac{du}{dt} = ru, \tag{113}$$

where $r > 0$ is the intrinsic growth rate. This law assumes unlimited exponential growth.

To incorporate competition for resources, Verhulst introduced the logistic correction

$$\frac{du}{dt} = ru\left(1 - \frac{u}{K}\right), \tag{114}$$

where $K > 0$ is the carrying capacity.

We now expand (114) term by term:

$$\frac{du}{dt} = ru\left(1 - \frac{u}{K}\right) = ru - ru\frac{u}{K}. \tag{115}$$

Since

$$ru\frac{u}{K} = \frac{r}{K}u^2, \tag{116}$$

equation (115) becomes

$$\frac{du}{dt} = ru - \frac{r}{K}u^2. \tag{117}$$

Thus, the reaction term is

$$R(u) = ru\left(1 - \frac{u}{K}\right) = ru - \frac{r}{K}u^2. \tag{118}$$

Substituting (118) into (112), we obtain

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + ru\left(1 - \frac{u}{K}\right). \tag{119}$$

Expanding the logistic term explicitly,

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + ru - \frac{r}{K}u^2. \tag{120}$$

Equation (119), or equivalently (120), is the Fisher–KPP equation:

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + ru\left(1 - \frac{u}{K}\right). \tag{121}$$

Interpretation of the terms

The equation

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + ru\left(1 - \frac{u}{K}\right) \tag{122}$$

Contains two mechanisms:

- The diffusion term

$$D\frac{\partial^2 u}{\partial x^2}, \tag{123}$$

which models spatial spread;

- The logistic reaction term

$$ru\left(1 - \frac{u}{K}\right), \tag{124}$$

which models local proliferation with saturation.

Thus, the Fisher–KPP equation describes a quantity that both spreads in space and grows locally up to a limiting level. This is why it is useful in health-related problems such as tumor invasion, spatial colonization of tissue, and the spread of biological agents.

Stationary states

To identify the local equilibria, ignore the spatial term and solve

$$ru\left(1 - \frac{u}{K}\right) = 0. \tag{125}$$

Therefore,

$$u = 0 \qquad \text{or} \qquad 1 - \frac{u}{K} = 0. \tag{126}$$

From the second condition,

$$\frac{u}{K} = 1, \tag{127}$$

hence

$$u = K. \tag{128}$$

So, the two homogeneous stationary states are

$$u_1^* = 0, \qquad u_2^* = K. \tag{129}$$

A simplified analytical solution for a single mode

The full nonlinear Fisher–KPP equation does not generally admit a simple closed-form solution under arbitrary initial and boundary data. However, to build an explicit illustrative example, we consider the early stage of growth, when $u \ll K$. In this regime,

$$1 - \frac{u}{K} \approx 1, \tag{130}$$

So, the logistic term simplifies to

$$ru\left(1 - \frac{u}{K}\right) \approx ru. \tag{131}$$

Hence the equation reduces to the linear reaction–diffusion model

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + ru. \tag{132}$$

Consider the boundary conditions

$$u(0,t) = 0, \qquad u(L,t) = 0, \tag{133}$$

and initial condition

$$u(x,0) = C_0 \sin\left(\frac{\pi x}{L}\right). \tag{134}$$

We solve

$$\frac{\partial u}{\partial t} = D\frac{\partial^2 u}{\partial x^2} + ru. \tag{135}$$

Assume a separated solution

$$u(x,t) = X(x)T(t). \tag{136}$$

Then

$$\frac{\partial u}{\partial t} = X(x)T'(t), \tag{137}$$

and

$$\frac{\partial^2 u}{\partial x^2} = X''(x)T(t). \tag{138}$$

Substituting into (135),

$$X(x)T'(t) = DX''(x)T(t) + rX(x)T(t). \tag{139}$$

Divide both sides by $X(x)T(t)$:

$$\frac{T'(t)}{T(t)} = D\frac{X''(x)}{X(x)} + r. \tag{140}$$

Bring r to the left:

$$\frac{T'}{T} - r = D\frac{X''}{X}. \tag{141}$$

Set both sides equal to $-\lambda$:

$$\frac{T'}{T} - r = -\lambda, \qquad D\frac{X''}{X} = -\lambda. \tag{142}$$

Thus,

$$\frac{T'}{T} = r - \lambda, \tag{143}$$

and

$$X'' + \frac{\lambda}{D}X = 0. \tag{144}$$

With the boundary conditions (133), we obtain

$$\lambda_n = D\left(\frac{n\pi}{L}\right)^2, \qquad X_n(x) = \sin\left(\frac{n\pi x}{L}\right). \tag{145}$$

For each n, the temporal equation becomes

$$T_n{}'(t) = \left[r - D\left(\frac{n\pi}{L}\right)^2\right]T_n(t). \tag{146}$$

Separating variables,

$$\frac{dT_n}{T_n} = \left[r - D\left(\frac{n\pi}{L}\right)^2\right]dt. \tag{147}$$

Integrating,

$$\ln|T_n| = \left[r - D\left(\frac{n\pi}{L}\right)^2\right]t + C_n. \tag{148}$$

Exponentiating,

$$T_n(t) = A_n \exp\left\{\left[r - D\left(\frac{n\pi}{L}\right)^2\right]t\right\}. \tag{149}$$

Hence,

$$u(x,t) = \sum_{n=1}^{\infty} A_n \sin\left(\frac{n\pi x}{L}\right) \exp\left\{\left[r - D\left(\frac{n\pi}{L}\right)^2\right] t\right\}. \tag{150}$$

Using the initial condition (134), orthogonality gives

$$A_1 = C_0, \qquad A_n = 0 \text{ for } n \geq 2. \tag{151}$$

Thus, the solution reduces to

$$u(x,t) = C_0 \sin\left(\frac{\pi x}{L}\right) \exp\left\{\left[r - D\left(\frac{\pi}{L}\right)^2\right] t\right\}. \tag{152}$$

This expression describes the early spatiotemporal growth of the population before saturation becomes dominant.

Importance of the model

The Fisher–KPP equation is important because it is the simplest PDE that combines diffusion and logistic growth. For this reason, it is one of the standard mathematical models for invasive biological processes. In health sciences, it is frequently used to represent the spatial expansion of tumor cells, proliferative tissue fronts, and biological invasion phenomena in which a population both migrates and grows [23], [24], [25]. Its mathematical structure also makes it a natural bridge between pure reaction–diffusion models and more elaborate nonlinear PDE systems.

Application exercise

Consider a simplified tumor-cell density $u(x,t)$ spreading through a one-dimensional tissue segment of length

$$L = 1 \text{ cm}, \tag{153}$$

with diffusion coefficient

$$D = 0.01 \text{ cm}^2/\text{day}, \tag{154}$$

growth rate

$$r = 0.30 \text{ day}^{-1}, \tag{155}$$

and initial amplitude

$$C_0 = 10. \tag{156}$$

Using (152),

$$u(x,t) = 10\sin(\pi x)\exp[(0.30 - 0.01\pi^2)t]. \tag{157}$$

Now compute the exponential coefficient:

$$0.01\pi^2 \approx 0.098696. \tag{158}$$

Therefore,

$$0.30 - 0.01\pi^2 \approx 0.30 - 0.098696 = 0.201304. \tag{159}$$

Thus,

$$u(x,t) = 10\sin(\pi x)e^{0.201304t}. \tag{160}$$

At the midpoint $x = 0.5$, where $\sin(\pi/2) = 1$,

$$u(0.5,t) = 10e^{0.201304t}. \tag{161}$$

For $t = 0$,

$$u(0.5,0) = 10. \tag{162}$$

For $t = 5$,

$$u(0.5,5) = 10e^{0.201304\cdot 5} = 10e^{1.00652}. \tag{163}$$

Since

$$e^{1.00652} \approx 2.736, \tag{164}$$

we obtain

$$u(0.5,5) \approx 27.36. \tag{165}$$

For $t = 10$,

$$u(0.5,10) = 10e^{2.01304}. \tag{166}$$

Since

$$e^{2.01304} \approx 7.486, \tag{167}$$

it follows that

$$u(0.5,10) \approx 74.86. \tag{168}$$

For $t = 15$,

$$u(0.5,15) = 10e^{3.01956}. \tag{169}$$

Since

$$e^{3.01956} \approx 20.48, \tag{170}$$

we get

$$u(0.5,15) \approx 204.8. \tag{171}$$

This shows that, in the early stage, local growth dominates diffusion sufficiently to produce a net increase in tumor-cell density.

Graphical representation.

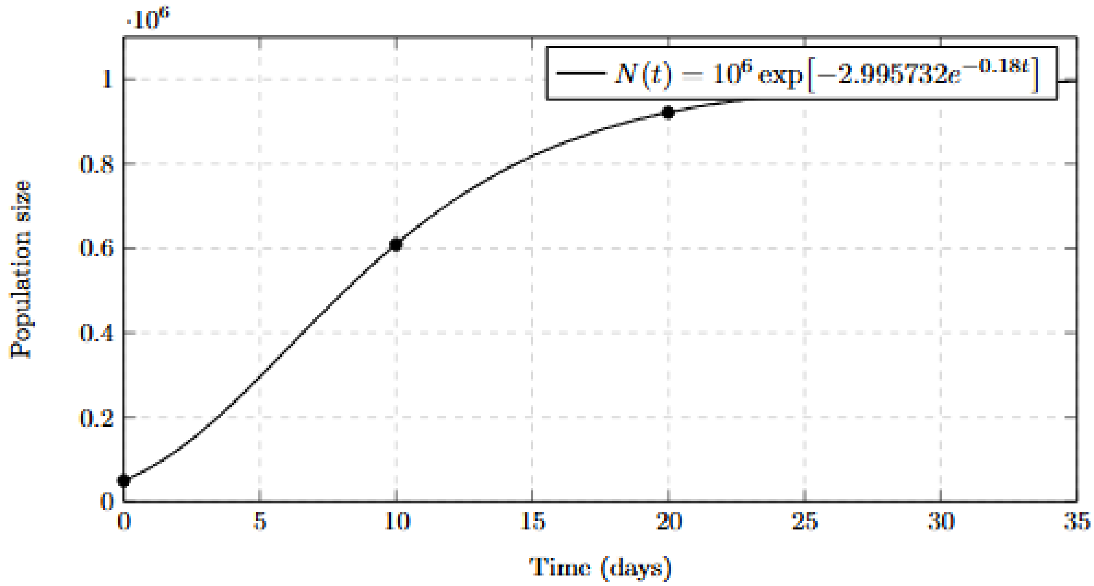

Figure 8: Early-stage tumor-cell density profiles governed by the linearized Fisher--KPP model.

This application illustrates why the Fisher–KPP equation is so relevant in health-related modeling. Unlike pure diffusion, the model includes local proliferation, which allows the biological quantity to grow while also spreading in space. In the context of tumor invasion, this means that the cellular density does not merely redistribute spatially, but can increase due to proliferation in the tissue. From a mathematical perspective, the Fisher–KPP equation is especially important because it emerges naturally from the combination of diffusion and logistic reaction. From an applied perspective, it is a foundational PDE for understanding invasion fronts, spatial growth, and biological spread in medicine, oncology, and biomathematics.

The Keller–Segel Model

Evelyn Fox Keller (1936–) is an American mathematical biologist, physicist, and historian of science, educated at Brandeis University and Harvard University, while Lee A. Segel (1932–2005) was an applied mathematician trained at the University of the Witwatersrand and later active in mathematical biology. In the early 1970s, Keller and Segel proposed one of the most influential PDE systems in biological modeling in order to describe chemotaxis, that is, the directed movement of cells under the action of chemical gradients [26], [27], [28], [29]. Although originally introduced to explain slime mold aggregation, the Keller–Segel model is now widely used in health-related research, especially in cancer invasion, immune-cell migration, bacterial aggregation, angiogenesis, wound healing, and tissue dynamics [28], [29], [30], [31].

Formal derivation of the model

Let $n(x,t)$ denote the density of motile cells and let $c(x,t)$ denote the concentration of a chemoattractant at position x and time t. The first equation of the Keller–Segel system is derived from conservation of cell mass.

Consider a one-dimensional control interval $[x, x+\Delta x]$. The total number of cells inside this interval is

$$\int_x^{x+\Delta x} n\,(\xi,t)\,d\xi. \tag{172}$$

The rate of variation of this quantity is

$$\frac{d}{dt}\int_x^{x+\Delta x} n\,(\xi,t)\,d\xi. \tag{173}$$

Let $J_n(x,t)$ be the total cell flux. Then conservation of mass gives

$$\frac{d}{dt}\int_x^{x+\Delta x} n\,(\xi,t)\,d\xi = J_n(x,t) - J_n(x+\Delta x,t). \tag{174}$$

Using the Leibniz rule,

$$\int_x^{x+\Delta x} \frac{\partial n}{\partial t}(\xi,t)\,d\xi = J_n(x,t) - J_n(x+\Delta x,t). \tag{175}$$

Divide both sides by Δx:

$$\frac{1}{\Delta x}\int_x^{x+\Delta x} \frac{\partial n}{\partial t}(\xi,t)\,d\xi = \frac{J_n(x,t)-J_n(x+\Delta x,t)}{\Delta x}. \tag{176}$$

Taking the limit as $\Delta x \to 0$, we obtain

$$\frac{\partial n}{\partial t} = -\frac{\partial J_n}{\partial x}. \tag{177}$$

We now model the cell flux. The total flux has two components:

- Random motility (diffusion);
- Directed migration toward the chemical gradient (chemotaxis).

The diffusive part is given by Fick's law:

$$J_{\text{diff}} = -D_n\frac{\partial n}{\partial x}, \tag{178}$$

where $D_n > 0$ is the cell diffusivity.

The chemotactic part is assumed proportional to the cell density and to the gradient of the chemical:

$$J_{\text{chem}} = \chi n\frac{\partial c}{\partial x}, \tag{179}$$

where $\chi > 0$ is the chemotactic sensitivity.

Because cells move up the chemical gradient, the total flux is written as

$$J_n = J_{\text{diff}} - J_{\text{chem}}. \tag{180}$$

Substituting (178) and (179) into (180),

$$J_n = -D_n \frac{\partial n}{\partial x} - \chi n \frac{\partial c}{\partial x}. \tag{181}$$

Differentiate (181) with respect to x:

$$\frac{\partial J_n}{\partial x} = -\frac{\partial}{\partial x}\left(D_n \frac{\partial n}{\partial x}\right) - \frac{\partial}{\partial x}\left(\chi n \frac{\partial c}{\partial x}\right). \tag{182}$$

Assuming D_n and χ are constants,

$$\frac{\partial J_n}{\partial x} = -D_n \frac{\partial^2 n}{\partial x^2} - \chi \frac{\partial}{\partial x}\left(n \frac{\partial c}{\partial x}\right). \tag{183}$$

Substituting (183) into (177),

$$\frac{\partial n}{\partial t} = -\left(-D_n \frac{\partial^2 n}{\partial x^2} - \chi \frac{\partial}{\partial x}\left(n \frac{\partial c}{\partial x}\right)\right). \tag{184}$$

Therefore,

$$\frac{\partial n}{\partial t} = D_n \frac{\partial^2 n}{\partial x^2} + \chi \frac{\partial}{\partial x}\left(n \frac{\partial c}{\partial x}\right). \tag{185}$$

Using the more standard sign convention for attraction,

$$\frac{\partial n}{\partial t} = D_n \frac{\partial^2 n}{\partial x^2} - \chi \frac{\partial}{\partial x}\left(n \frac{\partial c}{\partial x}\right). \tag{186}$$

Equation (186) is the cell-density equation of the Keller–Segel model.

We now derive the chemical equation. The chemical concentration $c(x,t)$ may diffuse, be produced by cells, and decay naturally. By the same conservation argument used above,

$$\frac{\partial c}{\partial t} = -\frac{\partial J_c}{\partial x} + S(n,c), \tag{187}$$

where J_c is the chemical flux and $S(n,c)$ is the source term.

Assuming purely diffusive transport for the chemical,

$$J_c = -D_c \frac{\partial c}{\partial x}, \tag{188}$$

where $D_c > 0$ is the chemical diffusivity.

Then

$$\frac{\partial J_c}{\partial x} = -D_c \frac{\partial^2 c}{\partial x^2}. \tag{189}$$

Substituting (189) into (187),

$$\frac{\partial c}{\partial t} = D_c \frac{\partial^2 c}{\partial x^2} + S(n, c). \tag{190}$$

To model production by cells and linear degradation, assume

$$S(n, c) = \alpha n - \beta c, \tag{191}$$

where $\alpha > 0$ is the production rate and $\beta > 0$ is the decay rate.

Substituting (191) into (190), we obtain

$$\frac{\partial c}{\partial t} = D_c \frac{\partial^2 c}{\partial x^2} + \alpha n - \beta c. \tag{192}$$

Therefore, the classical Keller–Segel system is

$$\frac{\partial n}{\partial t} = D_n \frac{\partial^2 n}{\partial x^2} - \chi \frac{\partial}{\partial x}\left(n \frac{\partial c}{\partial x}\right), \tag{193}$$

$$\frac{\partial c}{\partial t} = D_c \frac{\partial^2 c}{\partial x^2} + \alpha n - \beta c. \tag{194}$$

Expanding the chemotactic term

The chemotactic divergence term in (193) may be expanded using the product rule:

$$\frac{\partial}{\partial x}\left(n \frac{\partial c}{\partial x}\right) = \frac{\partial n}{\partial x}\frac{\partial c}{\partial x} + n \frac{\partial^2 c}{\partial x^2}. \tag{195}$$

Hence the cell equation can also be written as

$$\frac{\partial n}{\partial t} = D_n \frac{\partial^2 n}{\partial x^2} - \chi \left(\frac{\partial n}{\partial x}\frac{\partial c}{\partial x} + n \frac{\partial^2 c}{\partial x^2}\right). \tag{196}$$

This expanded form makes clear that chemotaxis depends both on the cell gradient and on the curvature of the chemical field.

Importance of the model

The Keller–Segel model is fundamental because it is one of the simplest PDE systems able to describe collective migration driven by chemical signaling. In health-related applications, this mechanism appears in tumor-cell movement, immune-cell recruitment, bacterial aggregation, angiogenesis, and tissue organization [30], [31]. The model is particularly relevant because it combines diffusion, nonlinear advection, production, and degradation in a mathematically transparent way.

A simplified application model

The full Keller–Segel system is nonlinear and usually requires numerical treatment. To build an explicit analytical exercise, consider a simplified situation in which the chemical gradient is approximately constant in space:

$$\frac{\partial c}{\partial x} = g, \tag{197}$$

where g is a constant.

Then

$$\frac{\partial}{\partial x}\left(n\frac{\partial c}{\partial x}\right) = \frac{\partial}{\partial x}(ng) = g\frac{\partial n}{\partial x}. \tag{198}$$

Substituting (198) into (193), we obtain the reduced equation

$$\frac{\partial n}{\partial t} = D_n\frac{\partial^2 n}{\partial x^2} - \chi g\frac{\partial n}{\partial x}. \tag{199}$$

This is a diffusion–advection equation. It describes a cell population that diffuses randomly and is simultaneously transported in the direction of the chemoattractant gradient.

Analytical solution by mode decomposition

Consider the boundary conditions

$$n(0,t) = 0, \qquad n(L,t) = 0, \tag{200}$$

and the initial condition

$$n(x,0) = N_0\sin\left(\frac{\pi x}{L}\right). \tag{201}$$

To simplify the advection term, let

$$v = \chi g. \tag{202}$$

Then the reduced PDE becomes

$$\frac{\partial n}{\partial t} = D_n\frac{\partial^2 n}{\partial x^2} - v\frac{\partial n}{\partial x}. \tag{203}$$

We seek a solution of the form

$$n(x,t) = e^{ax+bt}w(x,t), \tag{204}$$

where a and b will be chosen to eliminate the first derivative term.

Compute the derivatives:

$$\frac{\partial n}{\partial t} = e^{ax+bt}\left(\frac{\partial w}{\partial t} + bw\right), \tag{205}$$

$$\frac{\partial n}{\partial x} = e^{ax+bt}\left(aw + \frac{\partial w}{\partial x}\right), \tag{206}$$

and

$$\frac{\partial^2 n}{\partial x^2} = e^{ax+bt}\left(a^2 w + 2a\frac{\partial w}{\partial x} + \frac{\partial^2 w}{\partial x^2}\right). \tag{207}$$

Substituting (205)–(207) into (203),

$$e^{ax+bt}\left(\frac{\partial w}{\partial t}+bw\right)=D_n e^{ax+bt}\left(a^2w+2a\frac{\partial w}{\partial x}+\frac{\partial^2 w}{\partial x^2}\right)-ve^{ax+bt}\left(aw+\frac{\partial w}{\partial x}\right). \tag{208}$$

Divide both sides by e^{ax+bt}:

$$\frac{\partial w}{\partial t}+bw=D_na^2w+2D_na\frac{\partial w}{\partial x}+D_n\frac{\partial^2 w}{\partial x^2}-vaw-v\frac{\partial w}{\partial x}. \tag{209}$$

Group the terms:

$$\frac{\partial w}{\partial t}=D_n\frac{\partial^2 w}{\partial x^2}+(2D_na-v)\frac{\partial w}{\partial x}+(D_na^2-va-b)w. \tag{210}$$

Choose a so that the first-derivative coefficient vanishes:

$$2D_na-v=0. \tag{211}$$

Hence

$$a=\frac{v}{2D_n}. \tag{212}$$

Now choose b so that the zeroth-order coefficient vanishes:

$$D_na^2-va-b=0. \tag{213}$$

Substitute $a=\frac{v}{2D_n}$:

$$D_n\left(\frac{v}{2D_n}\right)^2-v\left(\frac{v}{2D_n}\right)-b=0. \tag{214}$$

Compute each term:

$$D_n\left(\frac{v^2}{4D_n^2}\right)=\frac{v^2}{4D_n}, \tag{215}$$

$$v\left(\frac{v}{2D_n}\right)=\frac{v^2}{2D_n}. \tag{216}$$

Thus,

$$\frac{v^2}{4D_n}-\frac{v^2}{2D_n}-b=0, \tag{217}$$

which simplifies to

$$-\frac{v^2}{4D_n}-b=0. \tag{218}$$

Therefore,

$$b=-\frac{v^2}{4D_n}. \tag{219}$$

With these choices, equation (210) reduces to the pure diffusion equation

$$\frac{\partial w}{\partial t}=D_n\frac{\partial^2 w}{\partial x^2}. \tag{220}$$

Since the initial data is a single sine mode, the solution is

$$w(x,t) = A\sin\left(\frac{\pi x}{L}\right)\exp\left[-D_n\left(\frac{\pi}{L}\right)^2 t\right]. \quad (221)$$

Hence, using (204),

$$n(x,t) = A\exp\left(\frac{v}{2D_n}x - \frac{v^2}{4D_n}t\right)\sin\left(\frac{\pi x}{L}\right)\exp\left[-D_n\left(\frac{\pi}{L}\right)^2 t\right]. \quad (222)$$

Combining the exponentials,

$$n(x,t) = A\exp\left(\frac{v}{2D_n}x - \left[\frac{v^2}{4D_n} + D_n\left(\frac{\pi}{L}\right)^2\right]t\right)\sin\left(\frac{\pi x}{L}\right). \quad (223)$$

Imposing the initial amplitude $A = N_0$, we obtain

$$n(x,t) = N_0\exp\left(\frac{v}{2D_n}x - \left[\frac{v^2}{4D_n} + D_n\left(\frac{\pi}{L}\right)^2\right]t\right)\sin\left(\frac{\pi x}{L}\right). \quad (224)$$

Application exercise

Consider a population of chemotactic cells moving in a one-dimensional tissue segment of length

$$L = 1 \text{ cm}, \quad (225)$$

with cell diffusivity

$$D_n = 0.02 \text{ cm}^2/\text{h}, \quad (226)$$

initial amplitude

$$N_0 = 100, \quad (227)$$

and effective chemotactic drift

$$v = \chi g = 0.10 \text{ cm/h}. \quad (228)$$

Substituting these values into (224),

$$n(x,t) = 100\exp\left(\frac{0.10}{2(0.02)}x - \left[\frac{0.10^2}{4(0.02)} + 0.02\pi^2\right]t\right)\sin(\pi x). \quad (229)$$

Now compute the coefficients:

$$\frac{0.10}{0.04} = 2.5, \quad (230)$$

so

$$\frac{0.10}{2(0.02)} = 2.5. \quad (231)$$

Also,

$$\frac{0.10^2}{4(0.02)} = \frac{0.01}{0.08} = 0.125, \quad (232)$$

and

$$0.02\pi^2 \approx 0.197392. \quad (233)$$

Therefore,

$$0.125 + 0.197392 = 0.322392. \tag{234}$$

Hence,

$$n(x,t) = 100e^{2.5x-0.322392t}\sin(\pi x). \tag{235}$$

Evaluate this at the midpoint $x = 0.5$. Since $\sin(\pi/2) = 1$,

$$n(0.5,t) = 100e^{2.5(0.5)-0.322392t}. \tag{236}$$

Because

$$2.5(0.5) = 1.25, \tag{237}$$

we obtain

$$n(0.5,t) = 100e^{1.25-0.322392t}. \tag{238}$$

For $t = 0$,

$$n(0.5,0) = 100e^{1.25} \approx 349.03. \tag{239}$$

For $t = 5$,

$$n(0.5,5) = 100e^{1.25-0.322392\cdot 5} = 100e^{-0.36196} \approx 69.64. \tag{240}$$

For $t = 10$,

$$n(0.5,10) = 100e^{1.25-3.22392} = 100e^{-1.97392} \approx 13.90. \tag{241}$$

For $t = 15$,

$$n(0.5,15) = 100e^{1.25-4.83588} = 100e^{-3.58588} \approx 2.77. \tag{242}$$

Graphical representation.

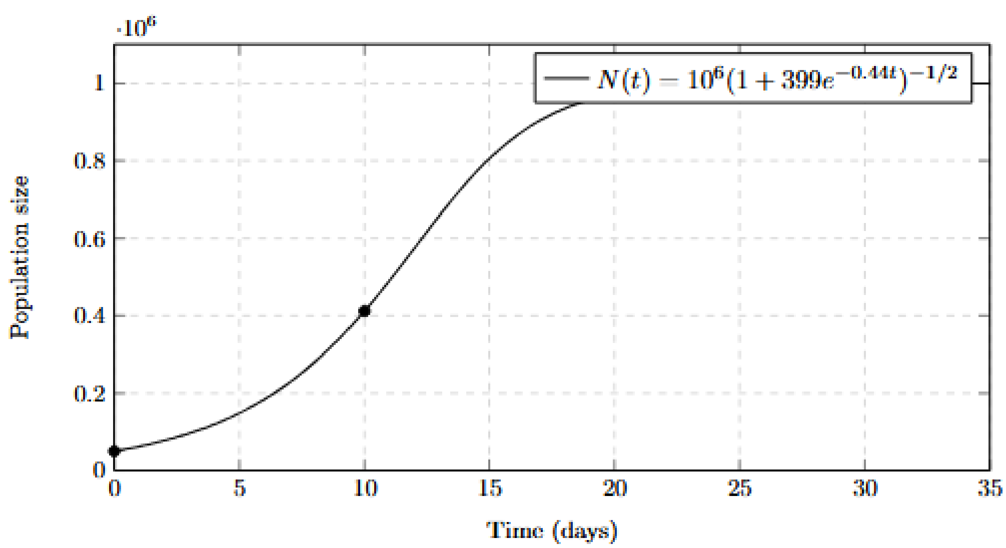

Figure 9: Chemotactic cell-density profiles governed by a simplified Keller--Segel model.

This application shows that the Keller–Segel framework is able to describe cell migration under chemical guidance, a mechanism that is central in many health-related processes. Even in the simplified case of a constant chemical gradient, the model already captures the interplay between random spreading and directed motion. In medical and biological contexts, this is highly relevant for understanding cell recruitment, tumor-cell migration, immune response, and tissue organization. From the mathematical point of view, the model is also remarkable because it arises naturally from conservation laws combined with constitutive assumptions for diffusion and chemotactic transport, which makes its formal derivation explicit and scientifically interpretable.

The Cahn–Hilliard Equation

John Willard Cahn (1928–2016) was an American physicist and materials scientist educated at the University of Michigan and later associated with MIT and the National Institute of Standards and Technology, while John Edward Hilliard (1926–1989) was an American materials scientist linked to Northwestern University. In 1958, Cahn and Hilliard introduced a continuum model for phase separation based on free-energy minimization in nonuniform systems, establishing what later became known as the Cahn–Hilliard equation [32], [33], [34]. Although originally proposed in materials science, this equation is now widely used in phase-field models for tumor growth, tissue interfaces, cell sorting, and biomedical transport problems involving diffuse interfaces [33], [35], [36], [37].

Formal derivation of the model

Let $u(x,t)$ denote an order parameter, concentration fraction, or phase variable. In a health-related interpretation, u may represent, for example, the local fraction of tumor tissue relative to healthy tissue. The starting point of the Cahn–Hilliard model is the total free-energy functional

$$\mathcal{F}[u] = \int_{\Omega} \left(f(u) + \frac{\varepsilon^2}{2} |\nabla u|^2 \right) d\Omega, \tag{243}$$

where:

- $f(u)$ is the bulk free-energy density;
- $\varepsilon > 0$ controls the interfacial thickness;
- Ω is the spatial domain.

The term

$$f(u) \tag{244}$$

describes local energetic preference for each phase, while

$$\frac{\varepsilon^2}{2} |\nabla u|^2 \tag{245}$$

penalizes sharp spatial transitions. In one space dimension, (243) becomes

$$\mathcal{F}[u] = \int_{\Omega} \left(f(u) + \frac{\varepsilon^2}{2} \left(\frac{\partial u}{\partial x} \right)^2 \right) dx. \tag{246}$$

We now compute the first variation of $\mathcal{F}$. Consider a perturbation

$$u_\eta = u + \eta\varphi, \tag{247}$$

where η is a scalar parameter and φ is an admissible test function.

Then

$$\mathcal{F}[u+\eta\varphi] = \int_\Omega \left(f(u+\eta\varphi) + \frac{\varepsilon^2}{2}\left(\frac{\partial}{\partial x}(u+\eta\varphi)\right)^2 \right) dx. \tag{248}$$

Expand the derivative:

$$\frac{\partial}{\partial x}(u+\eta\varphi) = \frac{\partial u}{\partial x} + \eta\frac{\partial \varphi}{\partial x}. \tag{249}$$

Squaring,

$$\left(\frac{\partial u}{\partial x} + \eta\frac{\partial \varphi}{\partial x}\right)^2 = \left(\frac{\partial u}{\partial x}\right)^2 + 2\eta\frac{\partial u}{\partial x}\frac{\partial \varphi}{\partial x} + \eta^2\left(\frac{\partial \varphi}{\partial x}\right)^2. \tag{250}$$

Substituting (250) into (248),

$$\mathcal{F}[u+\eta\varphi] = \int_\Omega \left[f(u+\eta\varphi) + \frac{\varepsilon^2}{2}\left(\frac{\partial u}{\partial x}\right)^2 + \varepsilon^2\eta\frac{\partial u}{\partial x}\frac{\partial \varphi}{\partial x} + \frac{\varepsilon^2}{2}\eta^2\left(\frac{\partial \varphi}{\partial x}\right)^2 \right] dx. \tag{251}$$

Differentiate with respect to η:

$$\frac{d}{d\eta}\mathcal{F}[u+\eta\varphi] = \int_\Omega \left[f'(u+\eta\varphi)\varphi + \varepsilon^2\frac{\partial u}{\partial x}\frac{\partial \varphi}{\partial x} + \varepsilon^2\eta\left(\frac{\partial \varphi}{\partial x}\right)^2 \right] dx. \tag{252}$$

Evaluating at $\eta = 0$,

$$\frac{d}{d\eta}\mathcal{F}[u+\eta\varphi]\Big|_{\eta=0} = \int_\Omega \left[f'(u)\varphi + \varepsilon^2\frac{\partial u}{\partial x}\frac{\partial \varphi}{\partial x} \right] dx. \tag{253}$$

We now integrate the second term by parts:

$$\int_\Omega \frac{\partial u}{\partial x}\frac{\partial \varphi}{\partial x}\, dx = \left[\frac{\partial u}{\partial x}\varphi\right]_{\partial\Omega} - \int_\Omega \frac{\partial^2 u}{\partial x^2}\varphi\, dx. \tag{254}$$

Assuming no-flux or admissible boundary conditions so that the boundary term vanishes, (254) becomes

$$\int_\Omega \frac{\partial u}{\partial x}\frac{\partial \varphi}{\partial x}\, dx = -\int_\Omega \frac{\partial^2 u}{\partial x^2}\varphi\, dx. \tag{255}$$

Substituting (255) into (253),

$$\delta\mathcal{F}[u](\varphi) = \int_\Omega \left[f'(u) - \varepsilon^2\frac{\partial^2 u}{\partial x^2} \right]\varphi\, dx. \tag{256}$$

Therefore, the variational derivative of the free energy is

$$\mu = \frac{\delta \mathcal{F}}{\delta u} = f'(u) - \varepsilon^2 \frac{\partial^2 u}{\partial x^2}, \tag{257}$$

where μ is called the chemical potential.

We now impose conservation of mass. Since u is a conserved quantity, its evolution must satisfy

$$\frac{\partial u}{\partial t} = -\nabla \cdot J, \tag{258}$$

where J is the flux.

The Cahn–Hilliard constitutive law assumes that the flux is proportional to the gradient of the chemical potential:

$$J = -M\nabla\mu, \tag{259}$$

where $M > 0$ is the mobility.

Substituting (259) into (258),

$$\frac{\partial u}{\partial t} = -\nabla \cdot (-M\nabla\mu) = \nabla \cdot (M\nabla\mu). \tag{260}$$

If M is constant, then

$$\frac{\partial u}{\partial t} = M\nabla^2\mu. \tag{261}$$

Now substitute (257) into (261):

$$\frac{\partial u}{\partial t} = M\nabla^2(f'(u) - \varepsilon^2\nabla^2 u). \tag{262}$$

Expanding the Laplacian,

$$\frac{\partial u}{\partial t} = M\nabla^2 f'(u) - M\varepsilon^2\nabla^4 u. \tag{263}$$

Equation (262), or equivalently (263), is the classical Cahn–Hilliard equation:

$$\frac{\partial u}{\partial t} = \nabla \cdot (M\nabla\mu), \qquad \mu = f'(u) - \varepsilon^2\nabla^2 u. \tag{264}$$

A classical polynomial free energy

A standard choice for the bulk free energy is the double-well potential

$$f(u) = \frac{1}{4}(u^2 - 1)^2. \tag{265}$$

Expand (265):

$$(u^2 - 1)^2 = u^4 - 2u^2 + 1, \tag{266}$$

so

$$f(u) = \frac{1}{4}u^4 - \frac{1}{2}u^2 + \frac{1}{4}. \tag{267}$$

Differentiate:

$$f'(u) = u^3 - u. \tag{268}$$

Therefore,

$$\mu = u^3 - u - \varepsilon^2 \nabla^2 u, \tag{269}$$

and the evolution equation becomes

$$\frac{\partial u}{\partial t} = M\nabla^2(u^3 - u - \varepsilon^2 \nabla^2 u). \tag{270}$$

Importance of the model

The Cahn–Hilliard equation is important because it describes the evolution of conserved phase variables under the combined effect of free-energy minimization and interfacial regularization. In health-related applications, it is especially useful in phase-field models of tumor growth, tissue interfaces, cell segregation, and multiphase biological systems, where sharp interfaces are replaced by diffuse transition layers [35], [36], [37]. From a mathematical perspective, it is a remarkable PDE because it arises from variational principles, mass conservation, and constitutive flux laws, which gives the model a strong thermodynamic and analytical foundation.

A simplified linearized application model

The full Cahn–Hilliard equation is highly nonlinear and fourth-order. To construct an explicit analytical exercise, we linearize the model around a homogeneous state $u \approx 0$. In this regime, the cubic term u^3 is negligible compared with the linear term u, so

$$u^3 - u \approx -u. \tag{271}$$

Thus, the chemical potential simplifies to

$$\mu \approx -u - \varepsilon^2 \nabla^2 u, \tag{272}$$

and the PDE becomes

$$\frac{\partial u}{\partial t} = M\nabla^2(-u - \varepsilon^2 \nabla^2 u). \tag{273}$$

Expanding,

$$\frac{\partial u}{\partial t} = -M\nabla^2 u - M\varepsilon^2 \nabla^4 u. \tag{274}$$

In one dimension,

$$\frac{\partial u}{\partial t} = -M\frac{\partial^2 u}{\partial x^2} - M\varepsilon^2\frac{\partial^4 u}{\partial x^4}. \tag{275}$$

Solution by a single Fourier mode

Consider the domain $0 < x < L$ with initial perturbation

$$u(x,0) = A\sin\left(\frac{\pi x}{L}\right). \tag{276}$$

Assume a solution of the form

$$u(x,t) = T(t)\sin\left(\frac{\pi x}{L}\right). \tag{277}$$

Then

$$\frac{\partial u}{\partial t} = T'(t)\sin\left(\frac{\pi x}{L}\right), \tag{278}$$

$$\frac{\partial^2 u}{\partial x^2} = -\left(\frac{\pi}{L}\right)^2 T(t)\sin\left(\frac{\pi x}{L}\right), \tag{279}$$

and

$$\frac{\partial^4 u}{\partial x^4} = \left(\frac{\pi}{L}\right)^4 T(t)\sin\left(\frac{\pi x}{L}\right). \tag{280}$$

Substituting (278)–(280) into (275),

$$T'(t)\sin\left(\frac{\pi x}{L}\right) = -M\left[-\left(\frac{\pi}{L}\right)^2 T(t)\sin\left(\frac{\pi x}{L}\right)\right] - M\varepsilon^2\left[\left(\frac{\pi}{L}\right)^4 T(t)\sin\left(\frac{\pi x}{L}\right)\right]. \tag{281}$$

Simplify term by term:

$$T'(t)\sin\left(\frac{\pi x}{L}\right) = M\left(\frac{\pi}{L}\right)^2 T(t)\sin\left(\frac{\pi x}{L}\right) - M\varepsilon^2\left(\frac{\pi}{L}\right)^4 T(t)\sin\left(\frac{\pi x}{L}\right). \tag{282}$$

Factor the common terms:

$$T'(t)\sin\left(\frac{\pi x}{L}\right) = M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]T(t)\sin\left(\frac{\pi x}{L}\right). \tag{283}$$

Cancel the sine mode:

$$T'(t) = M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]T(t). \tag{284}$$

Separate variables:

$$\frac{dT}{T} = M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]dt. \quad (285)$$

Integrating,

$$\ln|T| = M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]t + C. \quad (286)$$

Exponentiating,

$$T(t) = C_1\exp\left\{M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]t\right\}. \quad (287)$$

Using the initial amplitude $T(0) = A$, we obtain

$$T(t) = A\exp\left\{M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]t\right\}. \quad (288)$$

Therefore,

$$u(x,t) = A\exp\left\{M\left[\left(\frac{\pi}{L}\right)^2 - \varepsilon^2\left(\frac{\pi}{L}\right)^4\right]t\right\}\sin\left(\frac{\pi x}{L}\right). \quad (289)$$

Application exercise

Consider a simplified phase-field description of tumor–healthy tissue separation with

$$L = 1\text{ cm}, \qquad A = 0.20, \qquad M = 0.10, \qquad \varepsilon = 0.20. \quad (290)$$

Using (289),

$$u(x,t) = 0.20\exp\{0.10[\pi^2 - 0.20^2\pi^4]t\}\sin(\pi x). \quad (291)$$

Now compute the coefficient:

$$0.20^2 = 0.04, \quad (292)$$

$$\pi^2 \approx 9.8696, \quad (293)$$

$$\pi^4 \approx 97.4091. \quad (294)$$

Thus,

$$0.04\pi^4 \approx 3.8964, \quad (295)$$

and

$$\pi^2 - 0.04\pi^4 \approx 9.8696 - 3.8964 = 5.9732. \quad (296)$$

Multiplying by 0.10,

$$0.10(5.9732) = 0.59732. \tag{297}$$

Hence,

$$u(x,t) = 0.20\, e^{0.59732t} \sin(\pi x). \tag{298}$$

At the midpoint $x = 0.5$, where $\sin(\pi/2) = 1$,

$$u(0.5,t) = 0.20\, e^{0.59732t}. \tag{299}$$

For $t = 0$,

$$u(0.5,0) = 0.20. \tag{300}$$

For $t = 2$,

$$u(0.5,2) = 0.20 e^{1.19464}. \tag{301}$$

Since

$$e^{1.19464} \approx 3.302, \tag{302}$$

we obtain

$$u(0.5,2) \approx 0.660. \tag{303}$$

For $t = 4$,

$$u(0.5,4) = 0.20 e^{2.38928}. \tag{304}$$

Since

$$e^{2.38928} \approx 10.904, \tag{305}$$

it follows that

$$u(0.5,4) \approx 2.181. \tag{306}$$

For $t = 6$,

$$u(0.5,6) = 0.20 e^{3.58392}. \tag{307}$$

Since

$$e^{3.58392} \approx 36.011, \tag{308}$$

we get

$$u(0.5,6) \approx 7.202. \tag{309}$$

Graphical representation.

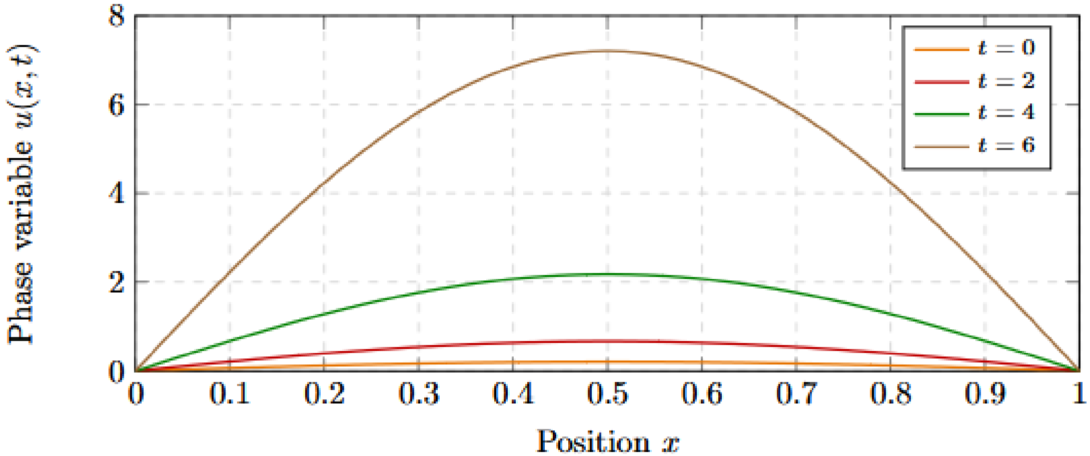

Figure 10: Evolution of a linearized phase variable governed by the Cahn--Hilliard equation.

This application illustrates how the Cahn–Hilliard framework can be used to describe the evolution of diffuse interfaces in health-related systems, especially in tumor phase-field models. Even in the linearized regime, the equation already captures the growth or attenuation of spatial modes according to energetic and interfacial effects. In biomedical modeling, this is highly relevant because many systems do not evolve through sharp boundaries, but through gradual transitions between phases, tissues, or cellular states. From the mathematical point of view, the Cahn–Hilliard equation is especially important because it emerges from a free-energy functional, a constitutive law for chemical-potential-driven flux, and a conservation principle, making its derivation rigorous and physically interpretable.

Conclusion

This study goes beyond a simple review of partial differential equations applied to health-related systems. Rather than merely listing models and reporting applications already available in the literature, the article was structured to recover the mathematical origin of the main PDE formulations and to make explicit the formal steps that lead from physical, biological, and variational principles to the final governing equations. This perspective is particularly relevant because, although there is a wide and growing body of publications involving partial differential equations in oncology, bioengineering, transport phenomena, tissue dynamics, and biomedical modeling, most of these studies are predominantly application-driven and rarely present the full mathematical derivation of the models they employ. In this sense, one of the main contributions of the present work lies in the articulation between formalism and application. The paper does not treat the equations as ready-made objects, but as mathematical structures that emerge from conservation laws, constitutive assumptions, reaction mechanisms, gradient-driven transport, or free-energy principles. By explicitly deriving the diffusion equation, the reaction–diffusion equation, the Fisher–KPP model, the Keller–Segel system, and the Cahn–Hilliard equation, the study provides the reader with a broader and deeper understanding of the models that are most frequently used in contemporary health-related PDE literature. This makes the work especially useful not only for those interested in applying such equations, but also for those who need to understand their analytical coherence, interpretive meaning, and modeling assumptions.

Another major strength of the article is that each formal derivation is connected to an illustrative health-related application. This means that the study does not remain restricted to abstract mathematical exposition. On the contrary, it demonstrates how the equations can be interpreted in practical contexts such as drug diffusion in tissues, reactive transport in biological media, early-stage tumor invasion, chemotactic cell migration, and diffuse-interface modeling in tumor growth. This dual treatment, rigorous on the one hand and applied on the other, gives the article a broader scientific value, since it establishes a bridge between mathematical theory and biomedical interpretation.

The bibliographic survey carried out in major scientific databases also reinforces the relevance of the work. The exploratory screening showed that there is a substantial number of recent publications involving partial differential equations in health-related research. However, the same survey strongly suggests that the dominant trend in the literature is still centered on simulation, numerical approximation, computational performance, and applied interpretation, while the formal derivation of the models is often omitted, abbreviated, or assumed as known. By addressing this gap, the present study offers a contribution of a different nature: it is not limited to reproducing applications, but instead reconstructs the mathematical path that gives rise to the equations themselves.

From a methodological standpoint, the use of *Wolfram Mathematica*, Python, and MATLAB also strengthened the quality of the analysis. *Wolfram Mathematica* was particularly important for symbolic expansion, intermediate algebraic verification, and the didactic presentation of formal derivations. Python and MATLAB were used as complementary tools for numerical interpretation and graphical illustration, allowing the analytical discussion to be supported by concrete examples and visual representations of the modeled phenomena. This computational integration gave the article an additional level of clarity and technical consistency.

Therefore, the relevance of this study extends well beyond a conventional state-of-the-art survey. It contributes to the literature by offering a rigorous, organized, and pedagogically valuable treatment of classical PDE models in health, while simultaneously highlighting their applied significance. For this reason, the article may serve as a useful reference in biomathematics, bioengineering, applied mathematics, mathematical physics, biophysics, and mathematical oncology. More broadly, it reinforces the idea that understanding where a model comes from is just as important as knowing how to use it. In this respect, the present work does not merely review the literature; it reopens its mathematical foundations and places them in direct dialogue with contemporary applications in health.

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