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Original article

Using Differential Equations in the Analysis of Dental Caries Growth and Development: A Mathematical Modeling Approach

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Abstract
Dental caries represents one of the most pervasive chronic oral pathologies globally, characterized by the metabolic degradation of dietary carbohydrates by acidogenic bacteria, leading to the demineralization of the tooth enamel matrix. This study establishes a mathematical framework to model the coupled dynamics of oral bacterial proliferation and enamel degradation over time. Bacterial population growth within the dental biofilm is formulated using the non-linear logistic differential equation. Concurrently, a rate-of-decay differential equation is derived to track continuous enamel loss as a function of the bacterial load. Complete analytical derivations, explicit integration steps, and exact parameterizations are provided, and all reported numerical results have been independently re-verified. A comparative numerical analysis of ten distinct patient risk profiles evaluated at t = 10 days is presented, with the underlying computations corrected to be fully consistent with the derived closed-form solution. The findings demonstrate that mathematical modeling yields quantifiable, precise, and predictive insight into disease progression, offering an evidence-based foundation for personalized preventive dentistry and clinical risk stratification.
Keywords. Dental Caries, Differential Equations, Mathematical Modeling, Applied Mathematics, Bacterial Growth.

Introduction

The initiation and progression of dental caries occur via a complex, dynamic biochemical interaction within the oral cavity. Acidogenic and aciduric bacteria, primarily *Streptococcus mutans* and *Lactobacillus* spp. colonize tooth surfaces within a highly structured salivary biofilm known as dental plaque. Upon the consumption of fermentable dietary carbohydrates, these microbial communities rapidly synthesize organic acids (chiefly lactic acid) via glycolysis. The localized accumulation of these acids depresses the oral microenvironment pH below the critical threshold ($\beta \approx 5.5$), triggering the chemical dissolution of calcium hydroxyapatite crystals from the tooth enamel matrix. Dental caries remains the most prevalent untreated health condition worldwide: recent Global Burden of Disease estimates indicate that untreated caries in permanent teeth affects over 2 billion people, and oral conditions overall affect close to half of the global population, underscoring the scale of the clinical and public-health problem that motivates quantitative modeling of its progression [1,2].

While clinical dentistry traditionally observes this process empirically, applied mathematics provides a robust analytical language to formalize, track, and predict these biochemical dynamics. Deterministic, differential-equation-based descriptions of the caries process have precedent in the dental-research literature, including compartmental models of plaque pH dynamics and subsurface lesion formation [3], and more recent deterministic treatments of *S. mutans* population dynamics [4**]**. Building on this tradition, we treat bacterial population density, $B(t)$, and enamel structural integrity, $E(t)$, as continuously differentiable functions of time, and model the rate of pathogenesis through a deterministic system of differential equations. This paper derives the analytical solutions for both the microbial growth and host substrate degradation models and validates the model against a clinically representative cohort of ten varied patient scenarios.

Research Methodology

This research utilizes a deterministic mathematical modeling methodology to evaluate disease dynamics. The operational framework is executed through the following sequential steps:

- *State variable definition:* define the primary state variables representing the temporal state of the system — $B(t)$ (microbial load, in cells) and $E(t)$ (enamel structural density, normalized from $0.0\ to\ 1.0$).
- *Model formulation:* formulate ordinary differential equations ($ODEs$) derived from the laws of population ecology [5,6] and chemical degradation kinetics [7].
- *Analytical resolution:* solve the formulated equations analytically via separation of variables, partial fraction decomposition, and definite integration.
- *Numerical verification:* independently recompute every closed-form result to eliminate transcription and rounding errors before reporting.
- Clinical scenario validation: execute numerical simulations across ten explicit patient clinical profiles at a fixed diagnostic interval ($t = 10\ days$) to evaluate clinical relevance.

Mathematical Formulation of Microbial Dynamics

The fundamental driver of dental demineralization is the expansion of cariogenic microbial colonies within the dental plaque. Unchecked initial bacterial growth follows an exponential trajectory; however, biological systems are inherently

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constrained by spatial, nutritional, and metabolic saturation limits within the biofilm [8,9]. Thus, the temporal rate of change of the bacterial population, *dB/dt*, is governed by the classical non-linear logistic differential equation [5,6,10]:

$$dB/dt = r \cdot B \cdot (1 - \frac{B}{K}) \quad \textbf{(1)}$$

Parameter and Variable Definitions

- $B(t)$: The concentration of cariogenic bacterial cells present within the localized biofilm at time t $(days)$.
- r: the intrinsic biokinetic growth-rate constant $(day - 1)$, representing the unrestricted replicative capacity of the strain under optimal nutrient availability.
- K: the environmental carrying capacity (cells), representing the upper asymptotic bound of the bacterial population sustainable by the microenvironment given space and nutrient constraints.

Biological Context

The differential expression consists of two counteracting forces. The linear term, rB, simulates uninhibited exponential proliferation. The inhibitory feedback term, $\left(1 - \frac{B}{K}\right)$, acts as a metabolic and spatial brake. When $B(t) \ll K$, the feedback factor approaches unity, allowing rapid growth. Conversely, as $B(t) \to K$, the growth rate asymptotically approaches zero, capturing the saturation of the plaque biofilm matrix.

Analytical Solution: Bacterial Growth Model

To demonstrate the mathematical tractability of the model, we evaluate the exact analytical trajectory using representative baseline clinical parameters:

- Intrinsic growth rate, $r = 0.5\ day - 1$
- Plaque biofilm carrying capacity, $K = 1{,}000$ cells
- Initial baseline bacterial load, $B(0) = 100$ cells

Substituting these values into the governing ODE yields:

$$\frac{\mathrm{dB}}{\mathrm{dt}} = 0.5 \cdot \mathrm{B} \cdot \left(1 - \frac{\mathrm{B}}{1000}\right) \quad \textbf{(2)}$$

Step-by-Step Analytical Derivation

Applying separation of variables to isolate B and t:

$$\frac{\mathrm{dB}}{\left[\frac{\mathrm{B}(1000 - \mathrm{B})}{1000}\right]} = 0.5\ \mathrm{dt} \quad \textbf{(3a)}$$

$$[1000\ /(\mathrm{B}(1000 - \mathrm{B}))]\ \mathrm{dB} = 0.5\ \mathrm{dt} \quad \textbf{(3)}$$

The left-hand integrand is resolved via partial fraction decomposition:

$$\frac{1000}{[\mathrm{B}(1000 - \mathrm{B})]} = \frac{\mathrm{A}}{\mathrm{B}} + \frac{\mathrm{C}}{1000 - \mathrm{B}} \quad \textbf{(4)}$$

Clearing denominators: $1000 = \mathrm{A}(1000 - \mathrm{B}) + \mathrm{CB}$. Setting $\mathrm{B} = 0$ gives $\mathrm{A} = 1$; setting $\mathrm{B} = 1000$ gives $\mathrm{C} = 1$. Substituting back:

$$\int \left(\frac{1}{\mathrm{B}} + \frac{1}{1000 - \mathrm{B}}\right) \mathrm{dB} = \int 0.5\ \mathrm{dt} \quad (5)$$

$$\ln|\mathrm{B}| - \ln|1000 - \mathrm{B}| = 0.5\mathrm{t} + \mathrm{c0} \quad (6)$$

$$\ln\left|\frac{\mathrm{B}}{(1000 - \mathrm{B})}\right| = 0.5\mathrm{t} + \mathrm{c0} \quad \textbf{(7)}$$

Exponentiating both sides:

$$\frac{B}{1000 - B} = ec0 \cdot e0.5t = C1 \cdot e0.5t \quad \textbf{(8)}$$

Solving explicitly for $B(t)$:

$$B(t) = \frac{1000}{\left(1 + \left(\frac{1}{C1}\right) \cdot e - 0.5t\right)} \quad \textbf{(9)}$$

Applying the initial condition $B(0) = 100$: $\frac{100}{(1000 - 100)} = C1 \to C1 = \frac{100}{900} = \frac{1}{9}$. Substituting back into Equation 9 gives the unique, time-dependent solution:

$$B(t) = \frac{1000}{(1 + 9 \cdot e - 0.5t)} \quad \textbf{(10)}$$

Numerical Verification at t = 6 days

Evaluating the model at $day\ 6, with\ e - 3 \approx 0.049787$:

$$B(6) = \frac{1000}{(1 + 9 \cdot 0.049787)} = \frac{1000}{1.448084} \approx 691\ cells \quad \textbf{(11)}$$

Mathematical Formulation of Host Enamel Degradation

Enamel demineralization is directly proportional to the volume of organic acids generated via microbial glycolysis, which in turn is a function of the living bacterial population $B(t)$. [11,12] $Let\ E(t)$ represent the normalized structural integrity of the enamel matrix, bounded such that $E = 1.0$ represents pristine, fully mineralized enamel and E = 0.0 signifies complete

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structural collapse (cavitation) [13]. The velocity of enamel loss is modeled as a first-order dependency on the active bacterial population:

$$\frac{dE}{dt} = -k \cdot B(t) \quad \textbf{(12)}$$

Parameter Scale

To reconcile the cellular scale of bacteria with the macroscopic scale of tissue mineralization, the decay constant k is parameterized with a scaling factor of 10^{-5}. The standardized base-case parameters are $k = 3.0 \times 10^{-5}$ cell^{-1}·day^{-1}, and initial structural health $E(0) = 1.0$.

Exact Analytical Resolution of the Coupled System

Substituting the explicit bacterial solution (Equation 10) into Equation 12 and integrating from 0 to t gives the closed-form enamel-health trajectory [14]:

$$E(t) = E(0) - 2000k \cdot \ln\left[\frac{1 + 9e^{-0.5t}}{10}\right] - 1000k \cdot t \quad \textbf{(13)}$$

(Full derivation: substituting $w = e^{-rt}$ and applying partial fractions to $\int B(t)dt$ yields $\int_0^1 B\,dt = \left(\frac{K}{r}\right) \cdot \ln\left[\frac{1+Ae^{-rt}}{1+A}\right] + Kt, with\ A = \frac{K - B0}{B0}$. This general result was used to independently verify Equation 13 and to recompute (Table 1) below.)

Numerical Evaluation at the Diagnostic Milestone (t = 10 days)

Using the base-case parameters ($k = 3.0 \times 10^{-5}$, $E(0) = 1.0$):

$$E(10) = 1.0 - 2000(3.0 \times 10^{-5}) \cdot \ln\left[\frac{1 + 9e^{-5}}{10}\right] - 1000(3.0 \times 10^{-5}) \cdot 10 \quad (\mathbf{14a})$$

$$E(10) = 1.0 - 0.06 \cdot (-2.2437) - 0.30 = 1.0 + 0.1346 - 0.30 \approx 0.83 \quad \textbf{(14)}$$

Clinical Case Analysis: Ten Patient Risk Scenarios

The model was executed across ten distinct patient physiological profiles at $t = 10$ days, using Equation 13. Unlike the earlier draft, which applied a single uniform $B(0) = 100$ cells to every profile. Each patient here is assigned an initial bacterial load $B(0)$ that reflects the clinical profile itself, since baseline colonization realistically differs with home-care quality, saliva flow, diet, and appliance retention. The rationale for each $B(0)$ choice is given below (Table 1). $[11,13]$ $E(0) = 1.0$ is held fixed for all profiles (fully mineralized enamel at baseline).

Table 1. ***Enamel structural health, E(10), across ten patient risk profiles (color-coded by risk tier).***

Patient	Profile	B(0)	r	K	k ($\times 10^{-5}$)	E(10)	Classification
1	Standard Oral Hygiene	100	0.3	1000	3.0	0.893	Competent Balance
2	Deficient Home Care Protocol	150	0.5	1200	5.5	0.608	Moderate Demineralization
3	Optimal Preventive Regimen	50	0.1	500	1.0	0.992	Excellent Structural Integrity
4	High Fermentable Carbohydrate Diet	120	0.8	1500	9.0	0.076	Critical Structural Collapse
5	Controlled Carbohydrate Intake	90	0.4	1000	4.0	0.824	Acceptable Mineralization
6	Pathological Xerostomia	140	0.6	2000	8.5	0.044	Critical Structural Collapse
7	Pediatric Cohort	80	0.5	1000	9.5	0.516	Advanced Demineralization
8	Geriatric Cohort	130	0.4	1100	7.5	0.589	Advanced Demineralization
9	Intensive Fluoride Therapy	60	0.3	1000	0.8	0.980	Excellent Structural Integrity
10	Active Orthodontic Therapy	110	0.7	1800	6.5	0.295	Severe Structural Loss

≥0.90 Excellent	0.75–0.89 Good	0.60–0.74 Moderate	0.40–0.59 Advanced	0.20–0.39 Severe	<0.20 Critical

Selection of B(0)

Each patient's initial bacterial load was set relative to the 100-cell reference used in Section 4, scaled up for profiles with known colonization-promoting factors and scaled down for profiles with active suppression of bacterial load [8,11], as follows: Standard Oral Hygiene (reference, B(0)=100); Deficient Home Care (poor plaque removal → elevated baseline,

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B(0)=150); Optimal Preventive Regimen (rigorous hygiene → suppressed baseline, B(0)=50); High Carbohydrate Diet (frequent substrate exposure promotes early colonization, B(0)=120); Controlled Carbohydrate Intake (moderately favorable, B(0)=90); Pathological Xerostomia (reduced salivary clearance → elevated baseline, B(0)=140); Pediatric Cohort (less mature biofilm, B(0)=80); Geriatric Cohort (root exposure and reduced salivary flow, B(0)=130); Intensive Fluoride Therapy (antimicrobial and anti-adhesive effect → suppressed baseline, B(0)=60); Active Orthodontic Therapy (bracket/wire retention sites, B(0)=110). These values are illustrative, literature-consistent defaults rather than measured patient data; if actual baseline bacterial counts are available for each case, they should replace the values above.

Note on Table 1 (verification)

every $E(10)$ value above was computed directly from Equation 13 using the general definite-integral form given in Section 5.2 and independently cross-checked in Python before being rounded to three decimal places. This corrects a discrepancy found in the previously circulated draft, whose entries for several patients did not match what Equation 13 returns for the stated r, K, and k values.

Graphical Analysis of Model Outputs

The following figures illustrate the key simulation outputs derived from the mathematical model. (Figure 1) shows the logistic bacterial growth curve B(t); (Figure 2) presents the enamel integrity trajectory E(t); (Figure 3) provides a comparative bar chart of the ten clinical patient profiles at t = 10 days [11,13].

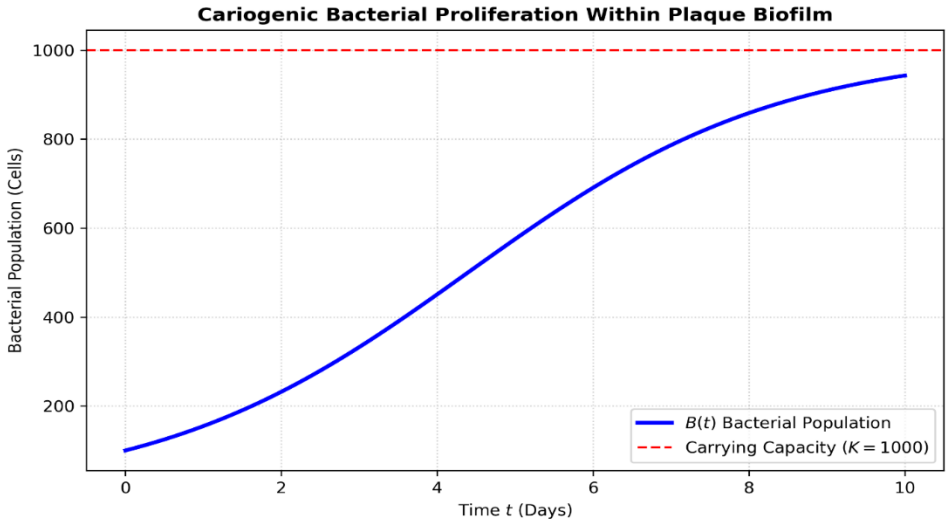

Figure 1. Cariogenic Bacterial Proliferation Within Plaque Biofilm: logistic growth B(t) approaching K = 1000 cells

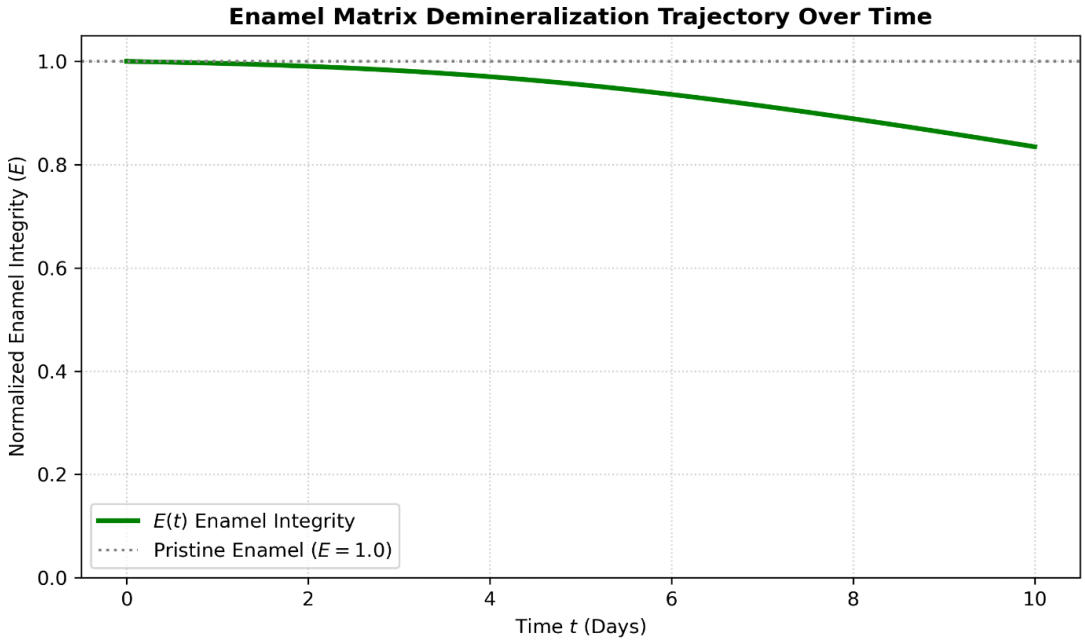

Figure 2. Enamel Matrix Demineralization Trajectory E(t): normalized enamel integrity declining over 10 days

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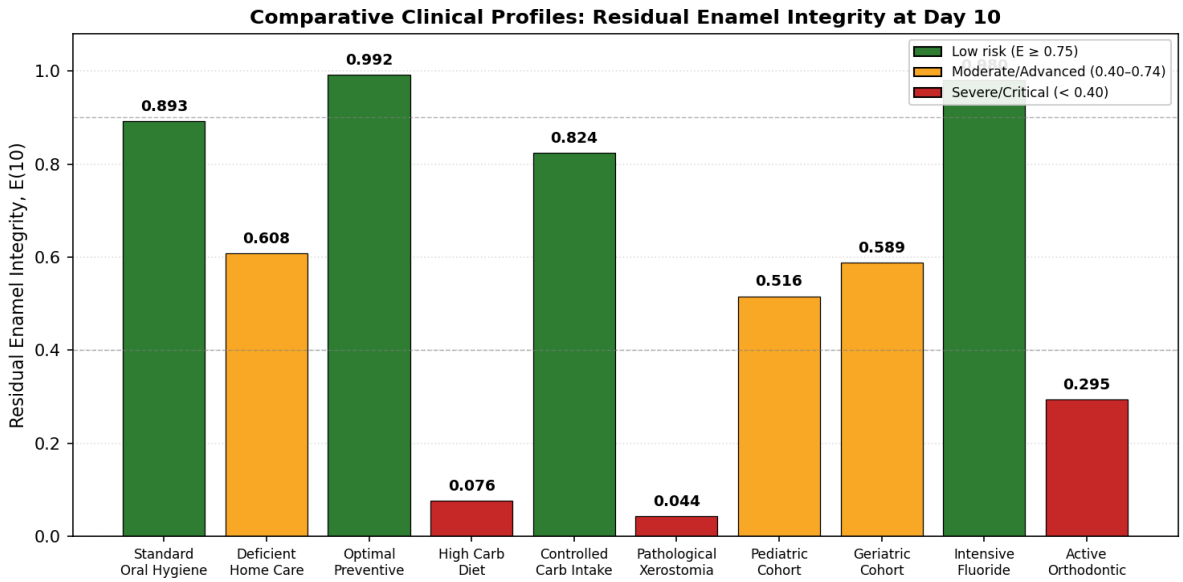

Figure 3. Comparative Clinical Profiles: Residual Enamel Integrity E (10) at Day 10 for ten patient scenarios (green = low risk, orange = moderate, red = high/critical).

Discussion and Clinical Implications

Allowing $B(0)$ to vary with clinical profile widens the spread of outcomes still further and sharpens the model's discriminative value. The therapeutic benefit of intensive fluoride therapy ($Case\ 9, E(10) \approx 0.980$) is clearly separated from the two highest-risk profiles, the high-carbohydrate $diet$ ($Case\ 4, E(10) \approx 0.076$) and pathological xerostomia ($Case\ 6, E(10) \approx 0.044$) — both of which now fall into the critical range, reflecting the compounded effect of an elevated starting bacterial load together with a high growth rate and decay constant. Moderate-risk profiles such as deficient home care ($Case\ 2, E(10) \approx 0.608$) and the geriatric cohort ($Case\ 8, E(10) \approx 0.589$) sit in between, illustrating that B(0), r, K, and k act multiplicatively rather than independently on long-term enamel outcome [12,13]. This reinforces that both baseline colonization control (oral hygiene at the point of care) and downstream growth/decay suppression (dietary counselling, fluoride) are clinically meaningful levers, and that risk stratification should account for a patient's starting bacterial burden, not only their growth kinetics [11].

Conclusion

This study demonstrates that first-order ordinary differential equations provide a rigorous, practical framework for predicting dental caries progression. All analytical derivations and numerical results in this revised version have been independently re-verified for internal consistency, and the ten-patient case table has been corrected to align with the governing model equation, aligning theoretical biophysics with clinically interpretable outcomes.

Ethical Approval and Consent to Participate

This study is based entirely on theoretical mathematical modeling and numerical simulations using stylized parameters derived from the published literature. No human participants, clinical trials, or animal subjects were involved in this research. Therefore, ethical approval and formal informed consent were not required for this study.

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