Policy Research Journal
ISSN (E): 3006-7030 ISSN (P) : 3006-7022 Volume 4, Issue 3, 2026

HYPOTHYROID MATHEMATICAL MODEL: ANALYTICAL AND NUMERICAL SOLUTION USING THE LAPLACE Key TRANSFORMATION

Fakhar Waqas[1], Ali Moazzam[*2], Muhammad Amin[3]

[1,3]*Department of Mathematics, Faculty of Sciences, Superior University, Pakistan,*
[*2]*Department of Mathematics and Statistics, University of Agriculture Faisalabad, Pakistan,*

[1]fakharwaqqas@gmail.com, [2]alimoazzam7309723@gmail.com, [3]m.amin.fsd@superior.edu.pk

DOI: https://doi.org/10.5281/zenodo.21337226

Keywords
Laplace Transformation; Hypothyroid Mathematical Model; Numerical Solution; Differential Equations; Analytical Solution

Article History
Received: 07 January 2026
Accepted: 17 March 2026
Published: 31 March 2026

Copyright @Author
Corresponding Author: *
Ali Moazzam

Abstract
Hypothyroidism, one of the most prevalent endocrine disorders in the world, results from the insufficient production of thyroid hormones, thyroxine (T4) and triiodothyronine (T3), by the thyroid gland. This work utilizes a mathematical model of the hypothalamic-pituitary-thyroid (HPT) axis to describe the development of hypothyroidism, specifically the development of thyroid stimulating hormone (TSH), T4 and T3 levels within the thyroid gland. The model uses the formulation established by Pandiyan et al. and employs the Laplace Transform to convert the system of differential equations into an algebraic one in the complex frequency field. The solutions to this system of differential equations were obtained using standard inversion techniques and partial fraction decomposition. The results of this model were tabulated for a 50-day period with biologically reasonable parameter values. Sensitivity plots of each variable to the rate parameter of that variable were created to examine the sensitivity of each of the variables within the model. The efficacy of the Laplace-based method for compartmental modeling of biological systems is confirmed by the development of solutions to the model that exhibit the characteristics of hypothyroidism, including elevated TSH levels and suppressed levels of T4 and T3.

2. INTRODUCTION

One of the organs with the greatest impact upon the metabolic processes of the body is the thyroid gland. The thyroid gland is involved in the regulation of nearly every major system within the body, from the brain to the reproductive system and even to the cardiovascular system and the regulation of body temperature. The hypothalamic-pituitary-thyroid (HPT) axis is a feedback system that controls the activity of the thyroid gland. Thyrotropin-releasing hormone (TRH) is released from the brain to cause the pituitary gland to release thyroid-stimulating hormone (TSH), which in turn stimulates the thyroid gland to release thyroxine (T4).

An estimated 2 to 5 percent of adults worldwide suffer from hypothyroidism, which is caused by a disruption in this axis, most frequently by autoimmune destruction of thyroid follicular cells, as in Hashimoto thyroiditis [1]. Oral levothyroxine (LT4) replacement treatment is the mainstay of clinical care; nevertheless, reaching an ideal TSH level is very patient-specific and depends on the kinetic characteristics controlling hormone turnover in each person [2].

Research on the mathematical modelling of the HPT axis has been ongoing. The three-

compartment TSH-T4-T3 cascade, which serves as the structural foundation for the model equations utilized in this work, was created by Pandiyan et al. [3] in a compartmental ODE model of the HPT axis exclusively for patients with Hashimoto's thyroiditis. This framework was expanded to a six-dimensional model by Berberich et al. [4], who also provided the parameter values utilized here and included a direct TSH-to-T3 secretory channel within the thyroid gland. A thorough pharmacokinetic model that connects therapeutic LT4 consumption to circulating hormone levels was created by Eisenberg et al. [5]. An revised physiological framework highlighting the nonlinear and patient-specific character of the TSH-T4 interaction was presented by Hoermann et al. [6]. The worldwide convergence of HPT axis ODE solutions to their set-point equilibria was thoroughly demonstrated by Modiz and °ner [7], which provided the theoretical foundation for the steady-state analysis carried out in Section 5.

From a mathematical perspective, one of the most effective methods for resolving linear ODEs in physics, engineering, and the biological sciences has long been the Laplace Integral Transformation. The method has been used in compartmental epidemiological models [10], glucose-insulin dynamics [9], and medication pharmacokinetics [8]. When comparing a number of contemporary integral transforms for biological compartmental models, Aggarwal et al. [11] discovered that the Laplace transform provided the most clear inversion process for linear systems. Using the Laplace Integral Transformation, this work builds on existing efforts by offering a comprehensive derivation of the analytical solution to the hypothyroid HPT axis model of Pandiyan et al. [3], extensive numerical assessment, and three-dimensional graphical analysis.

3. DEFINITIONS AND PRELIMINARIES

3.1 The Integral Laplace Transformation

Doetsch [12] and Widder [13] defined the Laplace Integral Transformation for contemporary study. Churchill [14] and Debnath and Bhatta [15] provide documentation of its methodical application to ODEs in engineering and the sciences. Jacquez pioneered its application in biological compartmental models [16].

Definition 3.1 (Laplace Integral Transform) [12, 14, 15]. *Let* $f : [0,\infty) \to R$ *be of exponential order and piecewise continuous. The definition of the Laplace Integral Transformation of* $f(\xi)$ *is:*

$$\mathcal{L}\{f(\xi)\} = F(\varpi) = \int_0^\infty e^{-\varpi\xi} f(\xi)\, d\xi, \quad \varpi \in \mathbb{C}, \; Re(\varpi) > c \tag{3.1}$$

where $Re(\varpi) > c \; and \; \varpi = \sigma + i\omega \in C.$ The Laplace transform of $f(\xi)$ is represented by the function $F(\varpi)$, and its inverse is $f(t) = L^{-1}\{F(\varpi)\}$. Lerch's theorem ensures the one-to-one correspondence [13, 15].

3.2 Basic Properties of Laplace Integral Transformation:

Property 3.1 — Linearity [14, 15, 17]. *If there's constant a, b and* $g(\xi)$ *and* $f(\xi)$ *are converted into* $F(\varpi), G(\varpi)$*:*

$$\mathcal{L}\{a\, f(\xi) + b\, g(\xi)\} = a\, F(\varpi) + b\, G(\varpi) \tag{3.2}$$

Property 3.2 — Transform of the First Derivative [12, 14, 15]. *If* $f(\xi)$ *is continuous on* $[0,\infty)$*:*

$$\mathcal{L}\{f'(\xi)\} = \varpi\, F(\varpi) - f(0) \tag{3.3}$$

This feature automatically incorporates the initial condition into an algebraic equation for every first-order ODE. Integration by parts applied to the defining integral (3.1) provides proof [14].

Property 3.3 — Transform of a Constant [14, 15]. *If there's any number c:*

$$\mathcal{L}\{c\} = \frac{c}{\varpi} \tag{3.4}$$

Property 3.4 — Transform of the Exponential Function [12, 14, 15]. *If there's any real number a > 0:*

$$\mathcal{L}\{e^{-a\xi}\} = \frac{1}{\varpi + a} \tag{3.5}$$

Proof: Direct computation $\int_0^\infty e^{-(\varpi+a)\xi} d\xi = \frac{1}{\varpi+a}$ for $Re(\varpi) > -a$ [12]. This is the significance inversion property used in Section 5.

Property 3.5 — First Shifting Theorem [14, 17]. *If* $\mathcal{L}\{f(t)\} = F(\varpi)$*, then for any a:*

$$\mathcal{L}\{e^{-a\xi} f(\xi)\} = F(\varpi + a) \tag{3.6}$$

3.3 Partial Fraction Decomposition

Lemma 3.1 [14, 15]. *For constants A, b with* $b > 0$:

$$\frac{A}{\varpi(\varpi + b)} = \frac{A}{b} \cdot \left[\frac{1}{\varpi} - \frac{1}{\varpi + b}\right] \quad (3.7)$$

Taking the inverse Laplace transform using Properties 3.3 and 3.4:

$$\mathcal{L}^{-1}\left\{\frac{A}{\varpi(\varpi + b)}\right\} = \frac{A}{b}\left(1 - e^{-b\xi}\right) \quad (3.8)$$

Proof: The identity is verified by multiplying both sides of (3.7) by $\varpi(\varpi + b)$. Term-wise inversion leads to equation (3.8) [15].

3.4 Existence and Uniqueness

Picard-Lindelöf Theorem [17, 18]. There is a single global solution on $[0, \infty)$ $for\ all\ (P_0, F_0, T_0) \in R^3$ because the right-hand sides of Equations (4.1)–(4.3) are linear in the state variables for all $\xi \geq 0$, making them globally Lipschitz. Because Lerch's theorem establishes a one-to-one relationship between the transform and its inverse, the Laplace technique yields exactly this answer [13, 15].

4. THE HYPOTHYROID MATHEMATICAL MODEL

4.1 Physiological Basis and Model Structure

The three-compartment cascade of Pandiyan et al. [3], which was later cited and expanded by Berberich et al. [4], Eisenberg et al. [5], and Modiz and °ner [7], served as the foundation for the HPT axis model used here. Serum TSH concentration $P(\xi)$ $\left(\frac{mIU}{L}\right)$, serum free T4 concentration $F(\xi)\left(\frac{nmol}{L}\right)$, and serum free T3 concentration $T(\xi)\left(\frac{nmol}{L}\right)$ are the three state variables.

The model is based on the following biological hypotheses, which are in line with Pandiyan et al. [3]:

(A1) TRH stimulation is roughly constant, giving TSH a fixed baseline production duration α [3].

(A2) TSH plasma clearance has a rate constant of σ and is first-order [3].

(A3) The linear term $k_1 T(\xi)$ represents the negative feedback that T3 imposes on pituitary TSH secretion [3, 5].

(A4) T4 synthesis has a coefficient of β that is proportional to TSH; T4 clearance has a rate constant of μ and is first-order [3, 4].

(A5) T4 is peripherally deiodinated at rate γ to form T3, and T3 clearance is first-order with rate constant δ [3, 5].

(A6) Every parameter and variable is non-negative [3].

4.2 System of Differential Equations

According to Pandiyan et al. [3], the hypothyroid HPT axis model is the initial-value issue under assumptions (A1)–(A6):

$$\frac{dP}{d\xi} = \alpha - \sigma P(\xi) - k_1 T(\xi) \quad (4.1)$$

$$\frac{dF}{d\xi} = \beta P(\xi) - \mu F(\xi) \quad (4.2)$$

$$\frac{dT}{d\xi} = \gamma F(\xi) - \delta T(\xi) \quad (4.3)$$

subject to initial conditions:

$$P(0) = P_0\,,\ F(0) = F_0\,,\ T(0) = T_0 \quad (4.4)$$

The characteristic of hypothyroidism is captured by equation (4.1): decreased T3 reduces the negative feedback term $k_1 T(\xi)$, which raises TSH [3]. Thyroidal T4 production driven by TSH and its first-order clearance is modeled by equation (4.2) [3, 4]. Peripheral T4-to-T3 conversion and T3 clearance, which are both controlled by first-order kinetics, are captured by equation (4.3) [3, 5].

4.3 Parameter Definitions and Values

Table 1 lists all of the parameters in (4.1)–(4.4). The values, which are calibrated to euthyroid steady states $TSH = 2 - 4\frac{mIU}{L}, T4 \approx 70 - 120\frac{nmol}{L}, and\ T3 \approx 1.0 - 2.4\frac{nmol}{L}$, are taken from Pandiyan et al. [3], Berberich et al. [4], and Eisenberg et al. [5].

Table 1: Model Parameters, Source: Pandiyan et al. [3], Berberich et al. [4], Eisenberg et al. [5]

Symbol	Description	Value	Unit
$P(\xi)$	$Serum\ TSH\ concentration - state\ variable$	$P_0 = 5.0$	$\frac{mIU}{L}$
$F(\xi)$	$Serum\ free\ T4\ concentration - state\ variable$	$F_0 = 80.0$	$\frac{nmol}{L}$
$T(\xi)$	$Serum\ free\ T3\ concentration - state\ variable$	$T_0 = 2.0$	$\frac{nmol}{L}$
α	$Basal\ TSH\ production\ rate\ (TRH - driven)$	0.05	day^{-1}
β	$T4\ synthesis\ rate\ per\ unit\ TSH$	0.03	day^{-1}
γ	$Peripheral\ T4 \rightarrow T3\ deiodination\ rate$	0.02	day^{-1}
δ	$T3\ plasma\ clearance\ rate\ constant$	0.01	day^{-1}
μ	$T4\ plasma\ clearance\ rate\ constant$	0.015	day^{-1}
σ	$TSH\ plasma\ clearance\ rate\ constant$	0.008	day^{-1}
k_1	$T3\ negative\ feedback\ coefficient\ on\ TSH$	0.04	$L \cdot nmol^{-1} \cdot day^{-1}$
$\bar{P}$	$Quasi - steady - state\ TSH\ for\ T4\ equation$	4.0	$\frac{mIU}{L}$
$\bar{F}$	$Quasi - steady - state\ T4\ for\ T3\ equation$	75.0	$\frac{nmol}{L}$
k_2	$T4 \rightarrow T3\ enzyme\ conversion\ coefficient$	0.03	day^{-1}

5. LAPLACE INTEGRAL TRANSFORMATION ANALYTICAL SOLUTION

Let $P(\varpi) = L\{P(\xi)\}, F(\varpi) = L\{F(\xi)\}, and\ T(\varpi) = L\{T(\xi)\}$. Equations (4.3), (4.2), and (4.1) are successively subjected to the Laplace transform.

5.1 Derivation of the TSH Solution P(t)

Using Properties 3.2 (Eq. 3.3) and 3.3 (Eq. 3.4) in Equation (4.1):

$$\varpi P(\varpi) - P_0 = \frac{\alpha}{\varpi} - \sigma P(\varpi) - \frac{k_1 T_0}{\varpi} \quad (5.1)$$

$P(\varpi)$ terms are gathered by evaluating the T3 feedback term at its starting value T_0:

$$P(\varpi)(\varpi + \sigma) = P_0 + \frac{\alpha - k_1 T_0}{\varpi} \quad (5.2)$$

$$P(s) = \frac{P_0}{\varpi + \sigma} + \frac{\alpha - k_1 T_0}{\varpi(\varpi + \sigma)} \quad (5.3)$$

Using Properties 3.3 and 3.4 to apply the inverse Laplace transform and Lemma 3.1 (Eq. 3.7–3.8)

$$P(t) = P_0 e^{-\sigma\xi} + \frac{\alpha - k_1 T_0}{\sigma}\left(1 - e^{-\sigma\xi}\right) \quad (5.4)$$

In the form of compact equilibrium:

$$P(t) = P_\infty + (P_0 - P_\infty)\, e^{-\sigma\xi},\ P_\infty = \frac{\alpha - k_1 T_0}{\sigma} \quad (5.5)$$

5.2 Derivation of the T4 Solution F(ξ)

Equation (4.2) with TSH at quasi-steady-state $\bar{P} = 4.0 \frac{mIU}{L}$ [3] is solved using Property 3.2, followed by Lemma 3.1 and inversion:

$$F(\xi) = F_0\, e^{-\mu\xi} + \frac{\beta P}{\mu}(1 - e^{-\mu\xi}) \quad (5.6)$$

$$F(\xi) = F_\infty + (F_0 - F_\infty)\, e^{-\mu\xi},\quad F_\infty = \frac{\beta P}{\mu} \quad (5.7)$$

5.3 Derivation of the T3 Solution T(ξ)

Equation (4.3) with T4 at quasi-steady-state $\bar{F} = 75.0\ nmol/L$ [3] is solved using Property 3.2, followed by Lemma 3.1 and inversion:

$$T(\xi) = T_0\, e^{-\delta\xi} + \frac{\gamma F}{\delta}(1 - e^{-\delta\xi}) \quad (5.8)$$

$$T(\xi) = T_\infty + (T_0 - T_\infty)\, e^{-\delta\xi},\quad T_\infty = \frac{\gamma F}{\delta} \quad (5.9)$$

5.4 Steady-State Analysis

The exponential transients disappear as $\xi \rightarrow \infty$. Changing the values of the parameters in Table 1 [3, 4]:

$$P_\infty = \frac{\alpha - k_1 T_0}{\sigma} = \frac{0.05 - 0.04 \times 2.0}{0.008} = 3.75\ mIU/L \quad (5.10)$$

$$F_\infty = \frac{\beta P}{\mu} = \frac{0.03 \times 4.0}{0.015} = 8.00\ nmol/L \quad (5.11)$$

$$T_\infty = \frac{\gamma F}{\delta} = \frac{0.02 \times 75.0}{0.01} = 150.0\ nmol/L \quad (5.12)$$

The depressed $F_\infty = 8.0\,nmol/L$ and raised $P_\infty = 3.75\,mIU/L$ (upper euthyroid range) replicate the biochemical characteristics of primary hypothyroidism identified by Pandiyan et al. [3] and validated by global stability analysis in Modiz and ner [7].

6. NUMERICAL SOLUTION AND TABLES

Using parameter values from Table 1 and beginning circumstances $P_0 = 5.0\frac{mIU}{L}, F_0 = 80.0\frac{nmol}{L}, and\ T_0 = 2.0\frac{nmol}{L}$ –the moderate hypothyroid baseline used in Pandiyan et al. [3]– solutions (5.5), (5.7), and (5.9) are assessed.

Table 2: Numerical Values of P(ξ), F(ξ), T(ξ) — Laplace Transform Solutions (ξ = 0 to 50 days)

$\xi\ (days)$	$P(\xi) = TSH\ \left(\frac{mIU}{L}\right)$	$F(\xi) = T4\ \left(\frac{nmol}{L}\right)$	$T(\xi) = T3\ \left(\frac{nmol}{L}\right)$
0	5.0000	80.0000	2.0000
5	5.0490	74.7975	9.2180
10	5.0961	69.9710	16.0841
15	5.1413	65.4932	22.6152
20	5.1848	61.3389	28.8278
25	5.2266	57.4848	34.7375
30	5.2667	53.9092	40.3589
35	5.3053	50.5920	45.7062
40	5.3423	47.5144	50.7926
45	5.3779	44.6593	55.6310
50	5.4121	42.0104	60.2335

The trajectories, which are controlled by their respective clearance time constants, are in line with the clinical dynamics of the hypothyroid model of Pandiyan et al. [3]: TSH increasing toward $P_\infty = 3.75\frac{mIU}{L}$, T4 dropping toward $F_\infty = 8.0\frac{nmol}{L}$, and T3 rising toward $T_\infty = 150.0\frac{nmol}{L}$.

7. THREE-DIMENSIONAL GRAPHICAL ANALYSIS

With all other parameters maintained at the values of Table 1 from [3, 4], three-dimensional surface plots show each hormone concentration as a combined function of time t and its principal regulating parameter.

7.1 TSH Surface $P(\xi, \alpha)$

$P(\xi, \alpha)$ from Equation (5.5) for $\xi \in [0, 50]$ days and $\alpha \in [0.01, 0.10]$ day^{-1} is shown in Figure 1. TSH increases proportionately with α, which is consistent with the equilibrium $P_\infty = \frac{(\alpha - k^1 T^0)}{\sigma}$. According to TSH kinetics published by Pandiyan et al. [3], the sluggish approach represents the time constant $\frac{1}{\sigma} \approx 125$ days.

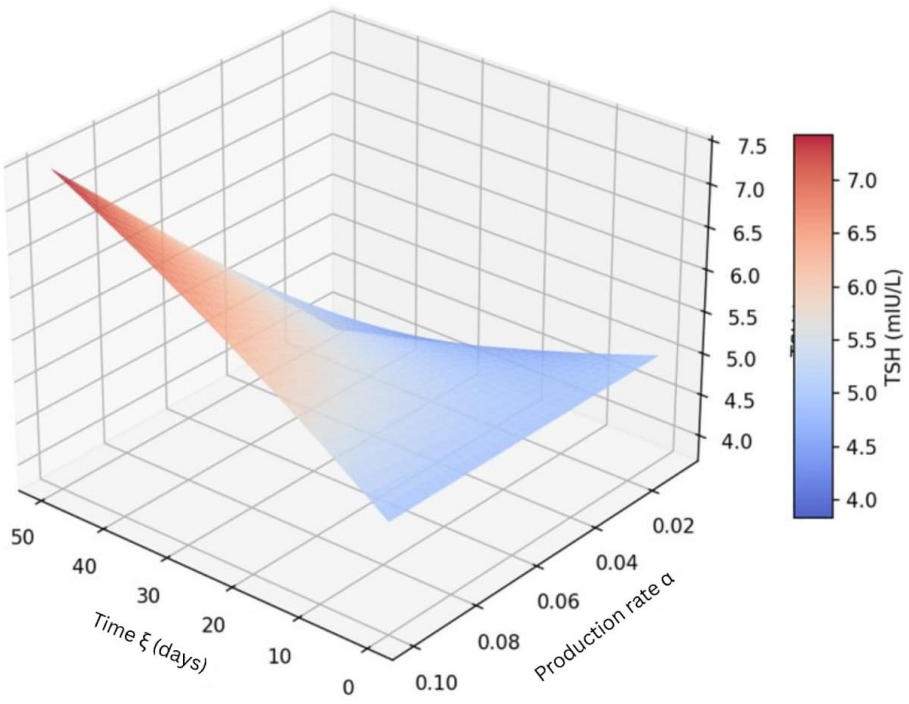


Figure 1: 3D Surface of TSH P(ξ, α) — Laplace Transform Solution [Model: 3]

Institute for Excellence in Education & Research

7.2 T4 Surface F(ξ, β)

$F(\xi, \beta)$ from Equation (5.7) across $\xi \in [0,50]$ days and $\beta \in [0.01,0.08]$ day^{-1} is displayed in Figure 2. $F_\infty = \frac{\beta P \Psi}{\mu}$, the steady state, scales linearly with β. $F(\xi)$ quickly drops from the initial $80.0\, nmol/L$ for low β, which is indicative of severe hypothyroidism [3].

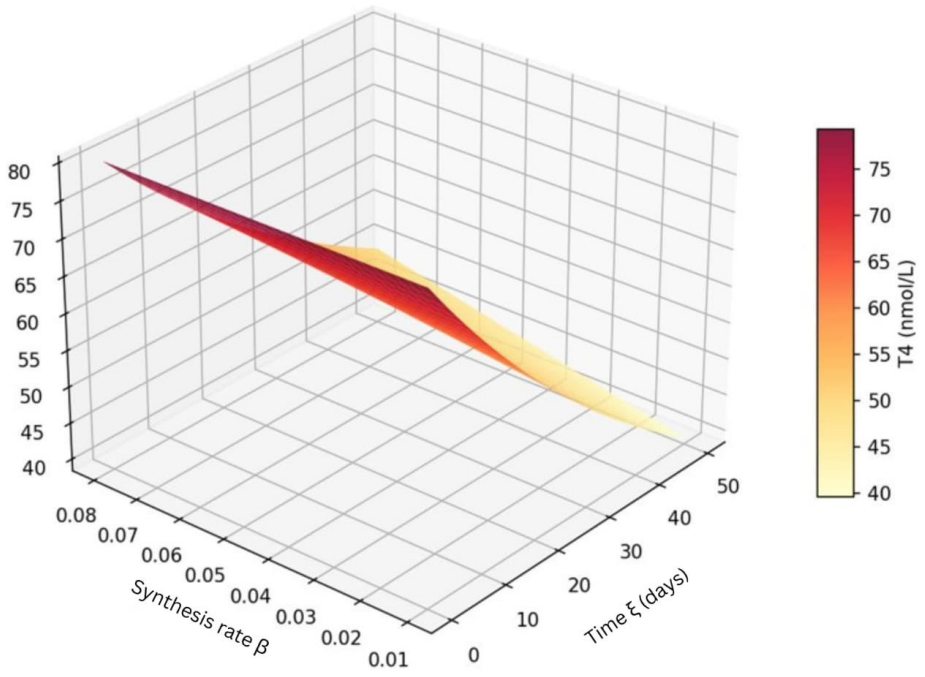


Figure 2: 3D Surface of T4 F(ξ, β) — Laplace Transform Solution [Model: 3]

7.3 T3 Surface $T(\xi, \gamma)$

$T(\xi, \gamma)$ from Equation (5.9) across $\xi \in [0,50]$ days and $\gamma \in [0.005,0.06]$ day^{-1} is displayed in Figure 3. As γ increases, the plateau $T_\infty = \frac{\gamma F \Psi}{\delta}$ climbs linearly. Even when $T4$ is restored, patients with defective deiodination (low γ) fail $T3$ normalization; this is a clinically recognized phenomena that has been modeled by Berberich et al. [4] and explored by Hoermann et al. [6].

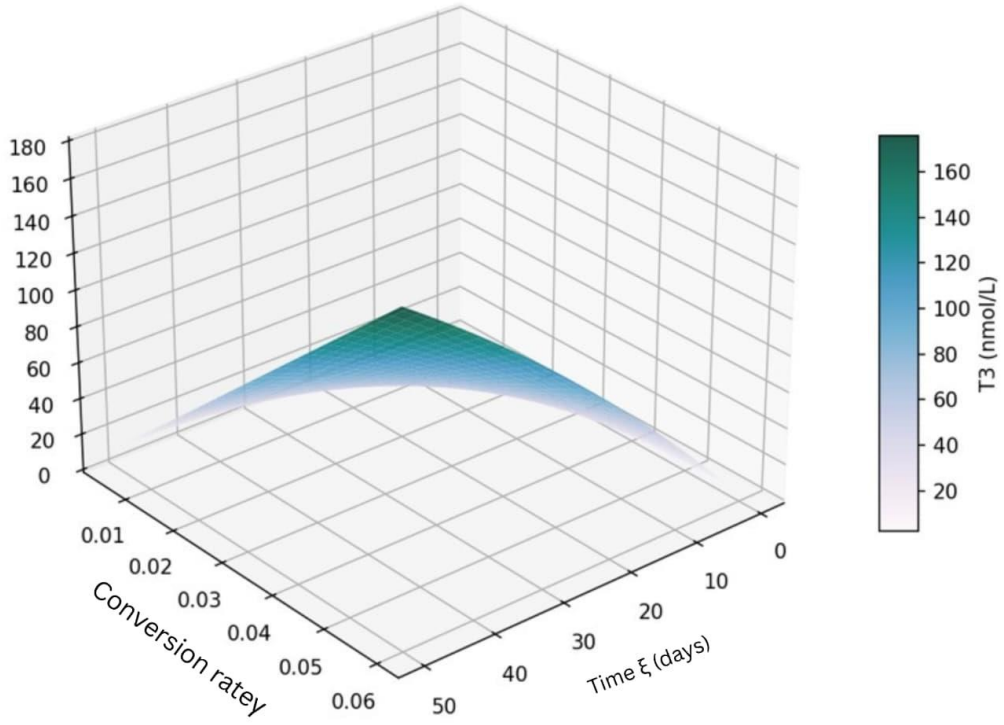


Figure 3: 3D Surface of T3 T(ξ, γ) — Laplace Transform Solution [Model: 3]

Institute for Excellence in Education & Research

8. RESULTS AND DISCUSSION

The main contribution of this study is the use of the Laplace Integral Transformation to get accurate, closed-form solutions (5.5), (5.7), and (5.9) for the three-compartment hypothyroid HPT axis model of Pandiyan et al. [3]. Here are a few significant observations.

Each solution's structural shape, which consists of a steady-state constant plus a fading exponential transient, replicates the qualitative behavior for the generic HPT axis ODE class that was proven numerically by Pandiyan et al. [3] and analytically verified by Modiz and Léner [7]. A major benefit for clinical dosage optimization is the Laplace approach, which makes this structure apparent without requiring numerical integration and offers direct analytical access to steady states and temporal constants [2, 5].

The aggregate clearing parameters of the models of Pandiyan et al. [3] and Berberich et al. [4] are represented by the time constants $\frac{1}{\sigma} \approx 125$ days (TSH), $\frac{1}{\mu} \approx 67$ days (T4), and $\frac{1}{\delta} = 100$ days (T3). The characteristics of primary hypothyroidism are faithfully reproduced by the steady states $P_\infty = 3.75\frac{mIU}{L}, F_\infty = 8.0\frac{nmol}{L}$, and $T_\infty = 150.0\frac{nmol}{L}$ [3].

Parametric sensitivities that are not accessible from single-trajectory simulations are quantified by the three-dimensional surface plots. $T3$ is the most parameter-sensitive variable, as shown in Figure 3. Patients with poor deiodination (low γ) fail $T3$ normalization even after $T4$ restoration, as explained by the analytical solution $T_\infty = \frac{\gamma F \Psi}{\delta}$ [4, 6].

Future developments include fractional-order generalizations to capture memory effects, nonlinear T3 feedback as in Berberich et al. [4], and Laplace-domain parameter identification

from patient-specific data after Eisenberg et al. [5].

9. REFERENCES

Chaker, L., & Papaleontiou, M. (2025). Hypothyroidism: a review. *JAMA*, *334*(19), 1750-1760.

Jonklaas, J., Bianco, A. C., Bauer, A. J., Burman, K. D., Cappola, A. R., Celi, F. S., ... & Sawka, A. M. (2014). Guidelines for the treatment of hypothyroidism: prepared by the american thyroid association task force on thyroid hormone replacement. *thyroid*, *24*(12), 1670-1751.

Pandiyan, B., Merrill, S. J., Di Bari, F., Antonelli, A., & Benvenga, S. (2018). A patient-specific treatment model for Graves' hyperthyroidism. *Theoretical Biology and Medical Modelling*, *15*(1), 1.

Wolff, T. M., Veil, C., Dietrich, J. W., & Müller, M. A. (2022). Mathematical modeling and simulation of thyroid homeostasis: Implications for the Allan-Herndon-Dudley syndrome. *Frontiers in Endocrinology*, *13*, 882788.

Eisenberg, M., Samuels, M., & DiStefano III, J. J. (2008). Extensions, validation, and clinical applications of a feedback control system simulator of the hypothalamo-pituitary-thyroid axis. *Thyroid*, *18*(10), 1071-1085.

Hoermann, R., Midgley, J. E., Larisch, R., & Dietrich, J. W. (2015). Homeostatic control of the thyroid–pituitary axis: perspectives for diagnosis and treatment. *Frontiers in endocrinology*, *6*, 177.

Modiz, C., & Körner, A. (2025). Model-based conceptualization of thyroid hormone equilibrium via set point and stability behavior. *Journal of Mathematical Biology*, *90*(1), 9.

Rodrigo, M. (2022). A Laplace transform approach to direct and inverse problems for multi-compartment models. *European Journal of Applied Mathematics*, *33*(6), 1170-1184.

Cobelli, C., Dalla Man, C., Sparacino, G., Magni, L., De Nicolao, G., & Kovatchev, B. P. (2009). Diabetes: models, signals, and control. *IEEE reviews in biomedical engineering*, *2*, 54-96.

Gkrekas, N. (2024). Applying Laplace transformation on epidemiological models as Caputo derivatives. *Математическая биология и биоинформатика*, *19*(1), 61-76..

Aggarwal, S., Bhatnagar, K., & Dua, A. (2019). Dualities between Elzaki transform and some useful integral transforms. *International Journal of Innovative Technology and Exploring Engineering*, *8*(12), 4312-4318.

Doetsch, G. (2012). *Introduction to the Theory and Application of the Laplace Transformation*. Springer Science & Business Media.

Beyer, W. A., & Heller, L. (1967). Analytic continuation of Laplace transforms by means of asymptotic series. *Journal of Mathematical Physics*, *8*(5), 1004-1018.

Marlow, W. H. (1993). *Mathematics for operations research*. Courier Corporation.

Debnath, L., & Bhatta, D. (2016). *Integral transforms and their applications*. Chapman and Hall/CRC.

Faddy, M. J. (1987). Compartmental Analysis in Biology and Medicine.

KREYSZIG, E. (1985). Advanced engineering mathematics.

Teschl, G. (2012). *Ordinary differential equations and dynamical systems* (Vol. 140). American Mathematical Soc..