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Modelling Vitamin D Concentration Dynamics in the Human Body Using Partial Differential Equation

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Abstract

Vitamin D is important for bone health, the immune system, regulating metabolic processes, and maintaining the calcium and phosphate balance. Many people, however, encounter vitamin D deficiencies, which can be attributed to insufficient sun exposure, a lack of vitamin D-rich food, and certain health issues. Most existing models have used ordinary differential equations (ODEs) to model vitamin D dynamics. The problem with the existing models, however, is that the models assume a uniform distribution, which does not account for the differing spatial levels of vitamin D in various body compartments. This research focuses on vitamin D dynamics in sunlight exposure, supplementation, and body mass index (BMI), and aims to apply it to an existing partial differential equation (PDE) model. The model is built using the Crank-Nicolson finite difference method to solve the equations and is validated with a synthetic dataset of 1000 people. The model shows that sunlight exposure and vitamin D supplementation positively contribute to serum 25-hydroxyvitamin D (25(OH)D) levels, and negatively impact it is BMI. Unlike traditional ODE models, the PDE model does a better job of mimicking the distribution, conversion, and destruction of vitamin D in principal body compartments. The study shows that, for simulating the dynamics of vitamin D, the PDE approach has more biological realism, which may be applied to future studies of vitamin D metabolism and public health.

1. Introduction

Vitamin D is responsible for the regulation of calcium and phosphate homeostasis in the body, a critical process that helps maintain skeletal integrity, immune function and numerous metabolic pathways. Although the deficiency of vitamin D is less common in sunny parts of the world, it continues to be an important public health issue, especially in people with reduced exposure to sunlight, poor dietary nutrient intake and physiological factors which also inhibit the synthesis or metabolism of vitamin D [1], [2]. Low levels of vitamin D have been linked to various health problems such as bone-related diseases, immune dysregulation and metabolic diseases [3], [4].

Historic mathematical descriptions of vitamin D metabolism have typically used ordinary differential equation (ODE) formulations, which are derived under the assumption that there is no spatial variation in the distribution of vitamin D within the body. However, this theory can be criticized for oversimplifying the underlying physiology, as vitamin D is produced, transported, stored and catabolized by a range of organs and tissues including the skin, liver, kidneys, blood stream and adipose tissue [5], [6]. As a result, ODE models are not

well suited to react to local spatial heterogeneity and specialized metabolic dynamics that underlie vitamin D regulation [7].

To overcome these limitations, the present work employs a partial differential equation (PDE) approach to describe vitamin D dynamics in the human organism. The governing PDE is adapted from the reaction – diffusion transport equations of [8], which was made to the model describing vitamin D_3 kinetics at the intracellular level. While [8] model concentrates on cellular transport and metabolism, its mathematical form–based on mass-transport theory, and Fick's law–offers a generalizable, scalable framework within which to simulate the transport, conversion, and degradation of vitamin D in physiological compartments. Here, this model is re-framed as a framework for whole-body vitamin D dynamics in which diffusion terms represent the movement of vitamin D between compartments, source terms represent solar exposure, dietary intake and supplementation and loss terms account for metabolic clearing/uptake.

Importantly, several key lifestyle and physiological factors such as sunlight exposure [6], vitamin D supplementation [9], body mass index (BMI) [10] with known effects on vitamin D synthesis, bioavailability, and metabolism are included in the PDE model. Notably, elevated BMI results in lowered circulating levels of vitamin D by sequestration in adipose tissue [11]. The core hypothesis of this study, therefore, is that both sun exposure and supplementation increase serum levels of 25-hydroxyvitamin D (25(OH)D) and a higher BMI decreases the vitamin D status.

To demonstrate the capability of the proposed model, a synthetic dataset including 1000 participants with different level of sunlight exposure, supplement use, and different BMI are created. It is anticipated that the resulting biologically informed and mathematically sound, 3D model will advance to a better understanding the relation between lifestyle and physiologic factors with vitamin D status. These results could also aid the development of public health recommendations on vitamin D supplementation based on evidence, especially for groups with an increased risk of deficiency.

2. Methodology

In this section, the dynamics of vitamin D in the body of a human being have been modelled by modifying an existing partial differential equation (PDE). This article is dedicated to the use of the given PDE model, which was used to simulate the transport, degradation, and transformation of vitamin D in the different body compartments. The model is used in terms of the accuracy, the computational efficiency and convergence behaviour compared to the available experimental and clinical results. It is a model that mimics the dynamics of the vitamin D concentration in the skin, liver, kidney, blood, and storage tissues which are utilized in determining the effect of various factors on vitamin D such as sunlight exposure, vitamin D intake and body mass index (BMI) on the body.

2.1 Formulation of the PDE model

The PDE model in this study also incorporates the dynamics of vitamin D in the human body from transport and conversion among various compartments such as skin, liver, kidneys, circulating blood, and storage tissues. The governing equation for this study was derived from the reaction–diffusion PDE by [8]. The general reaction-diffusion equation introduced by [8] is given by:

$$\frac{\partial D}{\partial t} = K\nabla^2 D + S - kD, \tag{1}$$

where:

- $D(x,t)$ represents the concentration of vitamin D at a spatial position x and time t,
- $\frac{\partial D}{\partial t}$ denotes the temporal rate of change of vitamin D concentration,
- K is the diffusion coefficient, describing the rate of vitamin D transport between compartments,
- $\nabla^2 D$ is the Laplacian operator, representing spatial diffusion of vitamin D within the biological system,
- S is the source term, accounting for vitamin D synthesis or input
- k is the first-order degradation or conversion rate constant, representing metabolic consumption and clearance of vitamin D.

which forms the basis of the present model.

To translate this equation to whole body vitamin D metabolism, Eq. (1) is expanded for every physiological compartment:

$$\frac{\partial C_i(x,t)}{\partial t} = D_i \frac{\partial^2 C_i(x,t)}{\partial x^2} + R_i(C_j) - k_i C_i(x,t) \tag{2}$$

where:

- $C_i(x,t)$ represents the concentration of vitamin D in the compartment i at position x and time t,
- D_i is the diffusion coefficient in the compartment i,
- $R_i(C_j)$ is the reaction term (which models synthesis, conversion, and interaction between compartments),
- k_i is the degradation rate for the compartment i.

In this model skin vitamin D synthesis is modelled as a source term by ultraviolet B (UVB) exposure. Transformation in the liver and kidneys is incorporated as reaction terms, and transport by blood is described via diffusion. Accumulation in adiposetissue is modelled as a sink, which is more pronounced for individuals with higher BMI. Dietary supplementation and sunlight exposure are accounted as external inputs via Dirac delta functions, which permits the inclusion of instantaneous changes in the blood vitamin D level. This formulation shares the PDE structure with that of [8] except that we replaced the biological interpretation to account for whole-body vitamin D dynamics.

2.2 Validation Using Synthetic Clinical Data

The PDE model is tested on a synthetic dataset generated to mimic observed clinical vitamin D behaviour. Validation is carried out based on comparison with known clinical trends rather than experimental measurements, as there is no real patient-level clinical data [1], [2], [3].

A simulated dataset with 1,000 observations that includes age, BMI, sunlight exposure, dietary intake, and vitamin D supplementation is created. The estimated serum levels of 25-hydroxyvitamin D (25(OH)D) and 25-dihydroxyvitamin D ($1,25(OH)_2D$) are compared with clinically recognized reference intervals and the physiologic metabolism [4], [5].

The synthetic dataset is created in MATLAB by random sampling, and parameter limits are selected based on data reported in the clinical/epidemiological literature. More precisely, the ranges of sunlight exposure are derived from synthesis studies [2], BMI-dependent effects on vitamin D bioavailability are informed by obesity research [3], and supplementation doses are based on existing pharmacokinetic work [7]. The robustness of the model is evaluated through simulations for different supplementing doses, lengths of sunlight exposure, and BMI classes.

2.3 Method of Solving the PDE Model

The Crank-Nicolson finite difference approach is applied for PDE model solving. This technique was chosen due to its impressive stability and accuracy concerning diffusion-reaction issues. Below is an example of a generalized discretized equation for the Crank-Nicolson method at each time step:

$$\frac{C^{n+1}-C^n}{\Delta t}=\frac{1}{2}(L(C^{n+1})+L(C^n))+\frac{1}{2}(S^{n+1}+S^n) \tag{2}$$

where:

- C^{n+1} and C^n are the concentrations of vitamin D at the next and current time steps, respectively,
- L is the spatial operator, which includes both diffusion and reaction terms,
- S represents the source term (inputs like supplementation and sunlight exposure).

The system of equations resulting from this discretization is solved iteratively using MATLAB's matrix solvers. The time steps are selected to ensure stability, and the model is run over multiple time steps to simulate vitamin D dynamics over time.

2.4 Implementation and Visualization Using Software

The PDE model was numerically implemented with the help of MATLAB R2024b, which is a well-known platform to solve complex differential equations and large-scale computations. In order to model real life physiological scenarios, a synthetic dataset was created with 1000 records and variables like age, BMI, exposure to sunlight and dose of vitamin D supplements. The PDE model was applied with the spatial domain being discretized to form compartments of the skin, liver, kidneys, bloodstream and storage tissues. MATLAB plotting routines were used to visualize the results of the simulation i.e, the temporal variation of the vitamin D concentration and space distribution between the individual compartments. Linear regression analysis and correlation analysis were conducted using Minitab to examine, verify, and quantify the relationships between serum 25(OH)D levels and contributing factors such as sunlight exposure, supplementation dose, supplementation frequency, and BMI. Such a combination of MATLAB and Minitab allowed us to effectively compute, visualize, and statistically interpret the effects of various physiological and lifestyle variables on vitamin D status.

3. Result and Discussion

3.1 Descriptive Analysis of the Synthetic Dataset

The dataset consisting of 1000 records was generated using MATLAB, incorporating factors such as age, body mass index (BMI), sunlight exposure, vitamin D supplementation, and dietary vitamin D intake, which are the factors that influence vitamin D status.

Table 1 Descriptive statistics (n = 1000)

Variable	Mean	Std Dev	Min	25%	Median	75%	Max
Age (years)	50.68	19.78	18.00	34.00	50.00	68.00	84.00
BMI (kg/m^2)	26.26	4.86	16.00	22.80	26.10	29.50	42.00
Sunlight exposure (hr/day)	1.84	0.95	0.20	1.01	1.85	2.67	3.50
Dietary vitamin D (IU/day)	297.66	121.20	0.00	212.00	299.45	381.50	689.20
Supplement dose (IU)	752.40	1023.28	0.00	0.00	400.00	1000.00	4000.00
Supplement frequency (per week)	2.40	2.49	0.00	0.00	2.00	5.00	7.00
25(OH)D (ng/mL)	21.18	7.88	5.00	15.40	20.60	26.00	58.50
$1,25(OH)_2D$ (pg/mL)	45.13	6.11	27.90	41.00	45.20	49.30	66.30

The mean age of the sample was 50.68 years, with BMI ranged from 16 (underweight category) to 42 (obese class II category). Respondents reported a median of 1.85 hours of sun exposure per day, which is relatively low and typically associated with insufficient vitamin D. A wide variety of supplementation practices, with reported dosages between 0 and 4000 IU per day, were captured in the dataset. Together, these variables illustrate the primary factors that affect vitamin D status, as well as the potential for the dataset to be used to analyse these factors.

3.2 Linear Regression and Correlation Analysis

Linear regression was evaluated to determine the relationships between serum 25-hydroxyvitamin D (25(OH)D) levels and vitamin D related factors such as sunlight exposure, supplement dosage, supplement frequency, and BMI. The strengths and direction of association were assessed based on the correlation coefficient (r), proportion of explained variance which was measured based on the coefficient of determination (R^2) and the p-value derived after the analysis of variance (ANOVA). The level of significance used here was 0.05. Each factor was tested on the hypothesis where H_0 was an indication of no linear relationship with serum 25(OH)D, versus the alternative hypothesis (H_1) being indicative of a significant linear relationship.

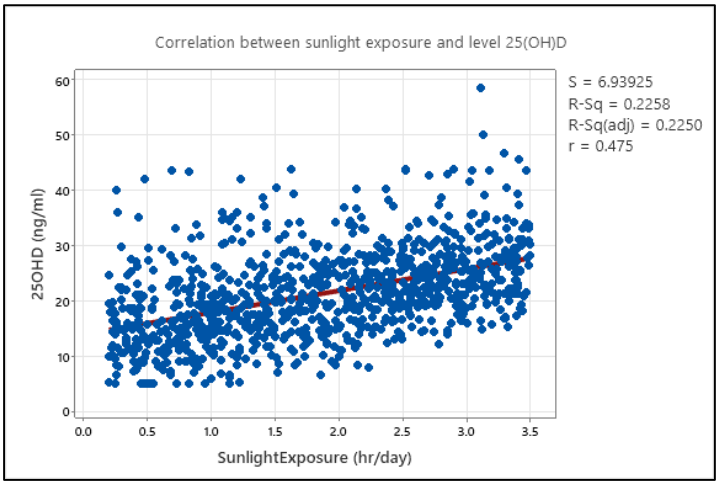

Fig. 1 Correlation between sunlight exposure per hour and level $25(OH)D$

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Fig. 1 shows the correlation between the exposure to sunlight and the serum 25(OH)D is moderate, with an *r*-value of about 0.475. The coefficient of determination is R^2 = 0.2258, which implies that 22.58% of the change in the levels of 25(OH)D can be attributed to sun exposure, whereas the rest of the change is attributed to other factors. The fact that the slope of the fitted line is positive supports the fact that high exposure to sunlight is related to high levels of vitamin D. The null hypothesis (H_0: no linear relationship between sunlight exposure and 25(OH)D) is rejected in favour of the alternative hypothesis (H_1: there is a linear relationship) as p = 0.000 which is less than the level of significance of 0.05, which proves that sunlight exposure is a statistically significant factor that can be considered in the determination of vitamin D status.

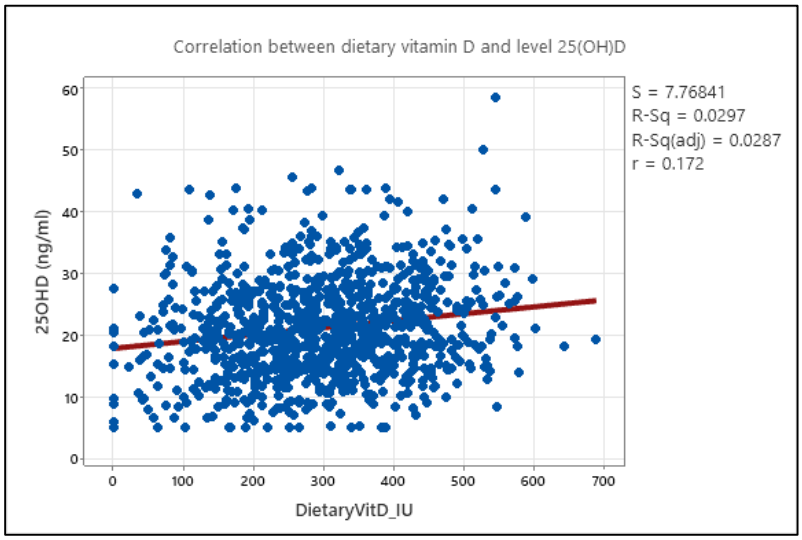

Fig. 2 Correlation between dietary vitamin D intake and level 25(OH)D

Based on Fig. 2 it shows that the dietary intake of Vitamin D has a weak positive association with the serum 25(OH)D with r = 0.172. The dietary intake has an insignificant contribution, with the R^2 = 0.0297 implying that dietary intake only explains a minor deviation in the 25(OH)D levels. Even though the relationship is statistically significant with p = 0.000, which is less than the significance level of 0.05, the modest value of both r and R^2 means that diet is not a powerful predictor of vitamin D levels. Thus, the null hypothesis is rejected, although the practical impact is insignificant.

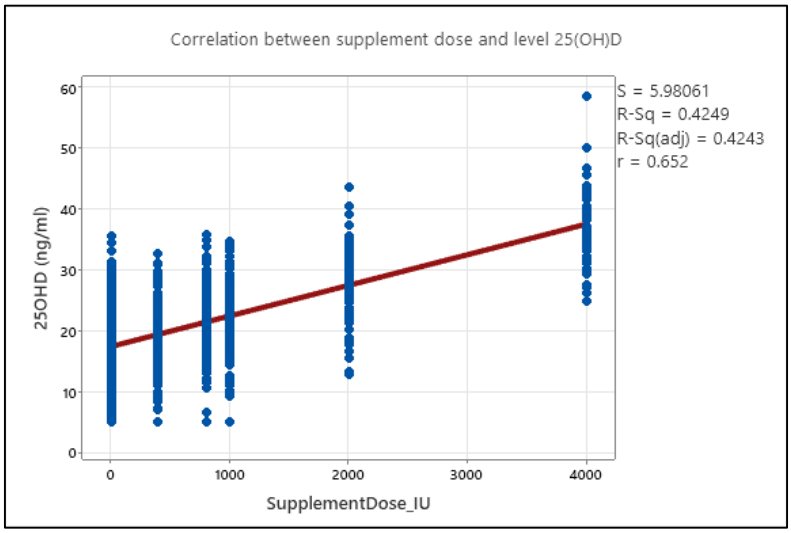

Fig. 3 Correlation between supplement dose and level 25(OH)D

According to Fig. 3, there is a moderately strong positive relationship between supplement dose and serum 25(OH)D, with r = 0.652. The R^2 = 0.4249, which implies that 42.49 percent of the change in 25(OH)D concentrations is determined by the dose of the supplement, which is why it is the most significant of the factors examined. The fitted line shows clearly that the greater the supplement doses, the greater the vitamin D levels. As p = 0.000 is less than the significance level of 0.05, the null hypothesis is rejected and the alternative hypothesis is accepted, which proves that supplement dose is a very strong determinant of vitamin D status.

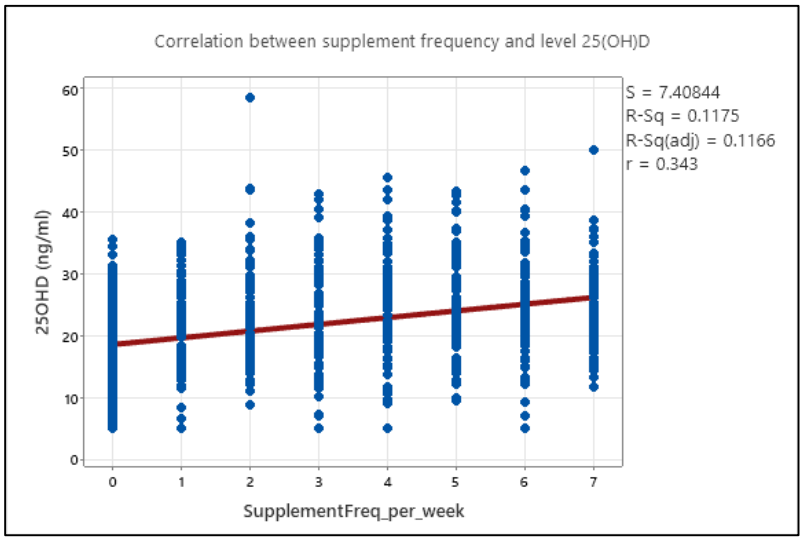

Fig. 4 Correlation between supplement frequency and level 25(OH)D

Fig. 4 shows that the frequency of supplements per week has a weak to moderate positive correlation with serum 25(OH)D, and the correlation coefficient is r = 0.343. The value 0.1175 of R^2 means that 11.75% of the change in the level of vitamin D is due to the frequency of supplementation. Although the relationship is statistically significant with p = 0.000, which is less than the significance value of 0.05, the relatively small value of R^2 indicates that frequency instead of dosage is not very crucial. Therefore, the null hypothesis is not accepted, and the effect of supplement frequency on the 25(OH)D level is moderate and insignificant.

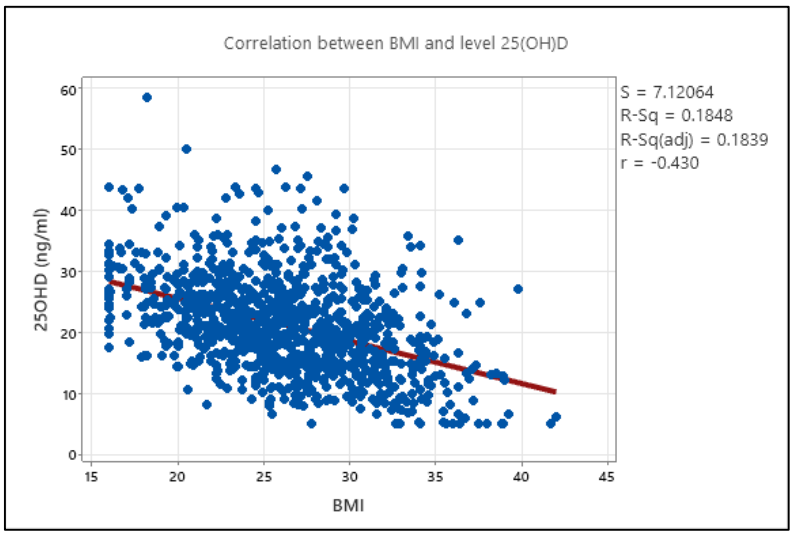

Fig. 5 Correlation between BMI and level 25(OH)D

From Fig. 5, a deduction was made that there exists a moderate negative association between BMI and serum 25(OH)D, with r = -0.430. The value of R^2 is 0.1848 which means that 25(OH)D levels explain 18.48 percent of the variability in BMI. This indicates that as vitamin D levels increase, BMI tends to decrease. This is supported by the negative slope of the regression line. Because p = 0.000, which is less than the significance level of 0.05, the null hypothesis is rejected, indicating that BMI is a statistically significant factor adversely related to vitamin D status.

Table 2 Root mean square error (RMSE) of vitamin D related factors

Factor	R^2	RMSE (ng/ml)	Predictive accuracy
Sunlight exposure	0.2258	5.98	High predictive accuracy
Dietary vitamin D intake	0.0297	6.94	Moderate predictive accuracy
Suplement dose	0.4249	7.12	Moderate predictive accuracy
Supplement frequency	0.1175	7.41	Low predictive accuracy
BMI	0.1848	7.77	Very low predictive accuracy

The predictive accuracy of every linear regression model was measured using the root mean square error (RMSE) analysis with smaller RMSE values showing better prediction of serum 25(OH)D levels. Supplement dose is the least affected variable with the lowest RMSE (5.98 ng/mL), which proves that it is the best predictor of vitamin D status. Sun exposure and BMI showed a moderate predictive validity as they had a strong but imprecise effect on the serum 25(OH)D content. On the other hand, supplement frequency and dietary vitamin D intake had a higher RMSE value, which implies a poor predictive ability. In general, the findings point to the fact that although a number of factors can be statistically significant, supplement dosage can be the most accurate and consistent predictor of vitamin D status and diet and supplementation frequency do not have much to offer when used separately.

3.3 PDE Model Simulation Result

The results of the PDE simulation prove a physiologically consistent behaviour of vitamin D transport and metabolism between compartments of the body.

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Initial Concentrations (C25) in compartments:
------------------------------------------
Skin: 0.74 ng/mL
Blood: 11.1 ng/mL
Liver: 0.74 ng/mL
Kidney: 1.184 ng/mL
Storage: 1.036 ng/mL
------------------------------------------
--------------------------
Updated Concentrations (C25) after Crank-Nicolson Step:
------------------------------------------
Skin: 5.8342 ng/mL
Blood: 9.4581 ng/mL
Liver: 3.2884 ng/mL
Kidney: 1.3022 ng/mL
Storage: 1.0469 ng/mL
------------------------------------------
--------------------------
Calculated Model Inputs:
--------------------------
Sunlight Exposure (hours/day): 2.33
Dietary Vitamin D (IU/day): 231.6
Supplement Dose (IU): 400
Supplement Frequency (per week): 6
--------------------------
Summary of Initial and Updated 25(OH)D Concentrations:
Initial Concentration of 25(OH)D (Blood): 11.1 ng/mL
Updated Concentration of 25(OH)D (Blood): 9.4581 ng/mL
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After Crank-Nicolson update, the vitamin D concentration in the skin rose significantly to 5.8342 ng/mL as compared to 0.74 ng/mL, which indicated that the cutaneous synthesis was improved by exposure to sunlight, which increased to 2.33 hours per day. Conversely, the plasma concentration of 25 (OH)D was lowered from 11.1 ng/mL to 9.4581 ng/mL, which means that the vitamin D was diverted out of circulation into metabolic and storage routes. This decrease implies that quite a large percentage of the synthesized vitamin D was transformed in the liver or delivered to storage tissues.

The liver level rose to 3.2884 ng/mL from 0.74 ng/mL, which proves the active metabolism of 25(OH)D to its biologically active metabolite $1,25(OH)_2D$ in the liver. The same was observed in the concentration of kidney which went up marginally, from 1.184 ng/mL to 1.3022 ng/mL, which is in accordance with the regulation of the kidney on the activation of vitamin D. Storage tissue concentration too increased marginally from 1.036 ng/mL to 1.0469 ng/mL, which implies that some of the vitamin D had been stored in adipose storage and as a result the immediate availability of the vitamin D in the bloodstream was diminished. It is more pronounced in people with a high BMI, which is a clinical definition of BMI 25 kg/m^2 (overweight) or BMI 30 kg/m^2 (obese) because the more adipose tissue there is, the higher the vitamin D sequestration of adipose tissue, and thus the lower the concentration of 25(OH)D in a person.

Moreover, the results show that supplementation affects the vitamin D metabolism since the model input directly states the supplement dose of 400 IU a single dosage of 400 IU 6 times per week, which shows that frequent supplementation is one of the factors contributing to improve conversion and metabolism of 25(OH)D to its active form. Comprehensively, the model is a good representation of the interplay between synthesis, metabolism, storage, and supplementation to establish the serum vitamin D levels. These are obtained by solving the governing reaction–diffusion PDE which is done numerically using the Crank–Nicolson finite difference method.

The physiological tissues (cutaneous, arterial and storage) are discretized spatially, while the system is marched in time with a fixed step. The initial concentrations of vitamin D are imposed in such a way that the preindustrial conditions correspond to physiological baseline values, whereas there is added solar exposure at a skin term equivalent to 2.33 hours per day. Supplementation is input as a separate external variable with Dirac delta functions modelling an intake of 400 IU six times per week. At each time point, the Crank–Nicolson method numerically propagates vitamin D concentrations in the body compartments, including diffusion between compartments, metabolic transformation, degradation and storage. The tabulated values are the concentrations after a whole Crank–Nicolson time step, that is after the redistribution of vitamin D over the body has taken place.

4. Conclusion

This study successfully applied a partial differential equation (PDE) model in estimating the dynamics of the concentration of vitamin D in various body compartments. The model incorporated vitamin D synthesis, metabolism, storage and transport, and, most importantly, the role of sunlight, supplements, and body mass index (BMI) on serum 25-hydroxyvitamin D (25(OH)D) levels. The result indicated that sunlight and supplements were the most important factors in increasing vitamin D levels, and the fact that a high BMI negatively impacted the availability of vitamin D due to fat tissue sequestration was also of great importance.

The model was able to demonstrate its spatial and temporal dynamics in the concentration of vitamin D in the skin, liver, kidney, blood, and storage compartments. This was more consistent with biological realities compared to older models that assume uniform distribution. The model was able to predict and was validated with a synthetic dataset, which, together with the existing clinical data, verified its predictions. Nonetheless, the validation of the model was carried out with a synthetic dataset, and it is recommended to use real clinical data and long-term simulations to improve the model and its predictive capability. Moreover, the model phenotype and usability for largely heterogeneous populations can be further enhanced by considering the impact of genetics and seasonal variations in sunlight exposure.

In summary, the PDE model contributes to a better understanding of the intricacies of vitamin D metabolism and illuminates how certain behaviours, such as taking supplements and spending time in the sun, affect vitamin D levels. This knowledge could be used to develop public health interventions to address vitamin D deficiency.

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Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of the paper.

Author Contribution

The authors confirm contribution to the paper as follows: ***study conception and design:*** *Nurul Haziqah Din;* ***data collection:*** *Nurul Haziqah Din;* ***analysis and interpretation of results*** *Nurul Haziqah Din, Cik Sri Mazzura Muhammad Basri* ***draft manuscript preparation:*** *Nurul Haziqah Din, Cik Sri Mazzura Muhammad Basri. All authors reviewed the results and approved the final version of the manuscript.*

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