

A Biomathematical Analysis of Diet-Driven Inflammation Dynamics and Chronic Disease Development

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ABSTRACT

The chronic inflammatory process is becoming usually accepted as a key determinant of dietary habits in the occurrence and development of non-communicable diseases, such as cardiovascular diseases, type 2 diabetes, and metabolic syndrome. This paper gives a biomathematical perspective of inflammation dynamics in relation to diet, combining nutritional inputs with physiological and immunological responses to gain better insight into the development of chronic diseases. The model theorizes the dietary intake as an active force that impacts pro-inflammatory and anti-inflammatory mediators, metabolic homeostasis, and tissue-level responses in the long-term. The interactions between dietary factors (including saturated fats, refined sugar and micronutrients) and inflammatory biomarkers (e.g., cytokines, C-reactive protein) and metabolic indicators (including insulin sensitivity, lipid profiles) are represented in a system of nonlinear differential equations. The model includes the element of feedback, with the continuous unhealthy dieting patterns increasing inflammatory reactions and balanced nutrition enhancing regulatory mechanisms which suppress inflammatory reactions. The empirical data of clinical and epidemiological studies inform parameterization, thus being biologically relevant and interpretable. The discussion examines steady-state behaviour, stability conditions and threshold effects which define the transition of acute to chronic inflammation. Numerical simulations show that continuous high-calorie, low-nutrient diets cause prolonged inflammatory conditions, exposing individuals to the risk of developing chronic diseases. On the other hand, dietary interventions with high content of anti-inflammatory nutrients can demonstrate the prospect of reversing or stabilizing the inflammatory dynamics. Sensitivity analysis has determined parameters of the dietary glycemic load and fat composition as key factors that determine the behaviour of the system. The results support the significance of dietary trends as risk factors that can be modified in order to prevent and manage chronic diseases. This research fills the gap between nutrition science and systems biology by giving a quantitative framework of information on the personalized dietary strategy and the community health interventions. The suggested biomathematical model will be a useful resource in forecasting future health conditions and making evidence-based nutritional policies to mitigate the burden of chronic diseases across the globe.

ARTICLE HISTORY

Received February 26, 2026

Accepted April 15, 2026

KEYWORDS

Biomathematical Analysis

(BMA),

Diet-Driven Inflammation

(DDI),

Chronic Disease Development

(CDD)

1. Introduction

The increasing problems of chronic diseases, including diabetes, cancers, cardiovascular disorders, and metabolic syndrome, have become major health challenges all over the world. For many years, various studies have been conducted on the importance of diet as a key indicator that has a great influence on the progression and development of all these diseases (Houtz, 2023). So, we can say that our diet is not only the source of nutrients and energy, but it also works as a powerful tool for regulating biological processes, particularly inflammation. Low-grade chronic inflammation also known as meta inflammation is also known as the essential mechanism associating unhealthy diet patterns to disease outcomes for a long time (Attaye et al., 2023). In order to understand the association, we required a biomathematical model and an interdisciplinary approach. These models have developed as the most valuable tools to predict and analyze diet-driven inflammation dynamics and how it contributes to chronic disease development. Diet is considered as the most adjustable factor which have a very strong influence on the inflammation level in the human's body (Basso et al., 2024). Dietary patterns have a significant effect on inflammation and are associated with a wide range of health-related consequences, such as cancer, mental health disorders, and cardiovascular diseases. The western diet, including saturated fats, processed

foods, and refined sugars, is strongly linked with high levels of inflammatory indicators (Khalid et al., 2026). Whereas healthy dietary patterns, including a plant-based diet and Mediterranean, have good impacts and are useful in reducing the inflammation rate and improving health outcomes. So, we can say that a person's diet has a significant and direct role in maintaining inflammatory pathways, which increases the risks of diabetes in humans (Keon, 2022). Moreover, inflammation itself defined as a complicated biological response which involve signaling molecules, immune cells, and metabolic processes. All these factors are significant to protect the body from various injuries, infections and diseases. Whereas inflammation can also damage organs and tissues, in this case, it is termed chronic inflammation. It has great impacts on the various organs of the body (Zhao et al., 2024). Chronic inflammation has a strong interconnection with cardiovascular disease, type 2 diabetes, and obesity. Advance study in the field of immunometabolism explore the ways that how excess food storage and fat tissues can trigger immune system which in turn leads towards the persistent low-grade inflammation which also have worsen impacts on metabolic health. This association between immune functions and metabolic health explains that there is a need to study inflammation as a dynamic system instead of a simple biological process (Bautista & López-Cortés, 2025). In this research, we describe the influences of diet on inflammation in different ways. One of the most important ways is gut microbiota, which has an effective role in regulating the immune system. In addition, the various components of diet composition have the ability to transform the composition of gut bacteria, which have a significant influence on inflammation signalling in the human body (Wang et al., 2025). For example, high fiber diet and foods collected from various plants can develop beneficial bacteria which helps to made anti-inflammatory compounds. While highly processes food can increase inflammation and interrupt gut balance. Apart from this, special nutrients, including antioxidants, fatty acids and various kinds of vitamins, have been helpful in reducing inflammatory markers (Jian, 2022). While access to sugary items and processed food can also increase the inflammation rate. Furthermore, according the research of IBD, diet of a person also has direct impacts on inflammatory indicators including CRP and other various immune indicators. So, we can say that by changing diet habits, we can also reduce the risks of disease by reducing inflammation and also improve human health (Kennedy et al., 2025).

Mostly, individuals who eat a healthier diet have less risk of chronic disease as compared to other people who do not eat healthy food. A diet high in inflammation is associated with the worsening of diseases, including artery calcification, which is considered an important symptom of heart disease. So, inflammation due to poor diet composition has a greater risk of long-term disease. Biomathematical models play an important role in understanding the influences of diet and inflammation on chronic disease (Simonsson, 2024). Biomathematical models use computational techniques and mathematical equations to explain complicated biological processes. In the case of inflammation, these biomathematical models explain in detail that how different variables interact with each other over time including environmental factors, immune response, nutrient intake, and metabolic rate. Biomathematical models and differential equations models both are utilized to pretend inflammatory responses and to find different scenarios under which inflammation becomes chronic inflammation. These models are helpful to test predicted consequences and hypotheses, and help to explain the influences of different dietary compositions instead of depending only on clinical trials. With the applications of modern and advanced computer technologies, it has now become possible to access more accurate and detailed models. These advanced modelling techniques integrate different kinds of data, including body markers, diet information, and genetic details as well. These biomathematical models have also ability to explore complex association among different variables, feedback systems, and changes which occurs with the passage of time (Papiri et al., 2025). Some other models explain the ways in which inflammation spreads in the tissues of the human body and how it becomes chronic in diseases such as liver inflammation. Moreover, the combination of biomathematics with different nutritional sciences provides various benefits. Such as it offer quantitative approach to explain complex interaction between inflammation and diet composition. Secondly, it provides long-term simulation effects, as well as it offers personalized analysis by combining specific data of a person including lifestyles factors, and genetic predisposition (Yang et al., 2025). These models are especially significant in the case of medicine precision, where dietary suggestions and treatment can be managed according to the specific needs of humans. Along with all these advancements, there are also various challenges in the applications of these developed models, which need to be addressed significantly for improving the applicability and accuracy of these advanced models (Björk et al., 2023).

2. Literature Review

With the passage of time, the emergence of chronic disease has increased. Every second person is suffering from a chronic disease. One of the most emergent diseases is the diabetes. Therefore, researchers have been showing an increasing interest in the investigation of the factors that cause chronic disease development (Hadjigeorgiou et al., 2025). Researchers have extensively studied the way that how inflammation related to diet can led towards the emergence of chronic disease by utilizing both mathematical models and scientific studies. Many researchers have investigated that for the prevention of long-term health-related risk, understanding of the relationship between diet and inflammation is necessary (Suchner, 2026). In this research paper, the researchers studied the min ideas collected from recent research

studies on the association of diet and inflammation. It was highlighted that the DII dietary inflammation index is among the most useful tools to investigate the influence of diet on inflammation. This index system helps scholars to comprehend the nature of a person's diet, whether it is anti-inflammatory or inflammatory. Researchers discovered that those persons whose DII scores were high seemed more likely to have chronic diseases, including diabetes, heart disease, and obesity (Lambert, 2024). Which shows that unhealthy diet has direct impacts on inflammation. Studies suggest that DII is helpful because it shows indexing of the diet composition of a person instead of one nutrient, which shows a clear graph of the impacts of food on the health of the human body. Many authors have also emphasized on the biological markers of inflammation (Bubnov & Spivak, 2023). They involve small substances present in the blood, including interleukins, CRP, and other indicators of the immune system. It was claimed that a higher level of these biological markers is strongly associated with unhealthy diet patterns, whereas a healthy diet composition is associated with a lower level of these markers. Researchers highlighted that these biological markers have great significance because they explain real changes occurring in the human body (Vanalli et al., 2024). Furthermore, the gut microbiome was another important research topic. Researchers have claimed that it contains various kinds of bacteria that have a significant role in managing and controlling inflammation. Among all these factors, diet is the most important factor. It has a strong effect on these bacteria. Such as diet rich in fiber, vegetables, and fruits help to promote good bacteria whereas diet having high processed food can damage gut bacteria and enhance the rate of inflammation. It was claimed that the gut microbiome works as a bridge between the immune system and diet. On the other hand, researchers claimed that inflammation also works as a link between chronic disease and diet. Which shows that a person's diet does not make direct cause of disease in some cases but it can be cause of chronic disease through inflammation (Mishra et al., 2025). Apart from this, many scholars claimed that long-term intake of unhealthy food increases inflammation, and the continuous increase in inflammation rate can damage the human body and lead towards the emergence of chronic disease (Ranson et al., 2025). Therefore, it was suggested that in order to prevent the disease and to make preventive strategies, it is necessary to understand the association between diet and inflammation. In addition, researchers also focused on the study of personalized nutrition. It was discovered that the effect of food does not seem the same for every person, but it varies from person to person. The bodies of all people respond in a different way to the same diet composition. It is due to the different lifestyles, metabolism, genes, and environment in which they were living (Narayan, 2024). Scholars have highlighted that due to different variations, one diet plan does not work in an effective way for everyone. Therefore, scholars emphasized on the personalized approaches, including biomathematical models, that might assist to make an effective diet plan for different individuals based on their specific requirements (Björk et al., 2024). Researchers claimed that biomathematical models, particularly models based on equations, are useful to explain various changes and predict future consequences more accurately. These mathematical models also assist scholars to test different situations in an easy way and without doing expensive and long-time taking experiments (Vanalli et al., 2025). The interaction of signaling molecules and immune cells also has been studied in the paper. Many scholars have demonstrated that the interaction between signalling molecules and immune cells can produce cycles of inflammation that occur for a long period of time. It was claimed that these cycles hold great significance in chronic disease, because in these cycles, inflammation does not go away simply (Alves Costa Silva et al., 2024). In this case, biomathematical modelling helps to comprehend these complicated cycles as well as suggest effective ways to break them. Many researchers examine the association between inflammation and diet. It was found that the association between inflammation and diet is not always so simple or direct. Even a small portion of unhealthy diet some time show an immediate reaction and some time it didn't show any immediate effect but with the passage of time it can impose serious health related problems (Reid et al., 2024). It was highlighted that there are some tipping points and thresholds in the human body, after which inflammation rises more swiftly. Thus, for studying such complex behaviours, researchers suggested nonlinear models. They can understand this sudden behaviour more efficiently (Sauer et al., 2025). In addition, some researchers combine both machine learning techniques and mathematical models and claimed that this combination of both models help use to analyze even larger amount of data and information related to health, inflammation, and diet. Whereas it was also studied that, along with the various applications of advanced modelling techniques, there are also many challenges that have been pointed out by many researchers (Fuchs, 2023). One major challenge is the shortage of long-term and accurate data. Due to this shortage, it is very complicated to measure what people have been eating for a long period of time, which in turn has a lot of influence on the precision of results (Shang et al., 2024).

3. Methodology

The paper is quantitative in nature, involving biomathematical modelling to explore the dynamic nature of the interplay between dietary intake, inflammation, and the progression of chronic disease. It is a formulated system of coupled nonlinear ODEs that are used to model the interactions between the key variables: dietary factors, inflammatory mediators, and metabolic health indicators. The main state variables are pro-inflammatory cytokines, anti-inflammatory agents, insulin sensitivity, and metabolic accumulated stress, all of which change over time based on the inputs to the system in the form

of diet and internal physiological feedback. Food intake is a time-dependent variable that includes both pro-inflammatory (e.g., saturated fats, refined sugars) and anti-inflammatory (e.g., fibre, antioxidants) foods. These inputs affect the production and suppression rates of cytokines via regulatory mechanisms. The model incorporates feedback mechanisms in which high levels of inflammation decrease insulin sensitivity, which increases metabolic imbalance and enhances inflammation. Estimation of parameters is done by the use of secondary data, which are based on published clinical and epidemiological studies. Calibration parameters like cytokine activation rates, dietary impact coefficients and recovery constants are made biologically plausible. Sensitivity analysis is conducted to find out the most significant parameters influencing the system stability and long-term performance.

4. Methods, Techniques and Data Collection Process

The model is discussed analytically and numerically. Equilibrium points are identified by bringing the system of equations to steady-state and local stability is evaluated by evaluating Jacobian matrices. Calculations are conducted with the help of computational programs, using smart PLS software, to monitor the temporal development of various dietary conditions, including high-risk and balanced diets. The model is validated through comparison of the simulated trends to known clinical patterns of disease progression and inflammation. This methodological framework offers a strong foundation for the comprehension of intricate diet-inflammation interplay and offers a predictive study of the risk of chronic disease in diverse nutritional circumstances.

5. Equational Analysis of Diet-Driven Inflammation Dynamics

A set of coupled nonlinear ordinary differential equations (ODEs) is built to mathematically model the interactions of dietary intake, inflammation, and chronic disease progression. The model represents the time dynamics of important biological variables that depend on diet.

5.1 Model Variables

Let:

- $D(t)$: Dietary input (pro- vs anti-inflammatory balance)
- $I(t)$: Pro-inflammatory cytokine concentration
- $A(t)$: Anti-inflammatory mediator level
- $M(t)$: Metabolic dysfunction (e.g., insulin resistance)
- $C(t)$: Chronic disease progression index

5.2 Core System of Equations

The dynamics of the system are expressed as:

$$\frac{dI}{dt} = \alpha D(t) + \beta M - \gamma A - \delta I$$

This equation shows that inflammation increases with unhealthy diet $D(t)$ and metabolic dysfunction M , while anti-inflammatory mediators A and natural decay reduce it.

$$\frac{dA}{dt} = \eta - \theta I - \kappa A + \lambda D(t)$$

Anti-inflammatory agents are produced at a baseline rate η , suppressed by high inflammation, and enhanced by healthy dietary intake.

$$\frac{dM}{dt} = \mu I + \nu D(t) - \xi M$$

Metabolic dysfunction increases with inflammation and poor diet, but decreases through recovery mechanisms.

$$\frac{dC}{dt} = \rho I + \sigma M - \tau C$$

Chronic disease progression is driven by both inflammation and metabolic imbalance, with a slow recovery or management rate.

5.3 Dietary Function Representation

Dietary intake is modelled as a composite function:

$$D(t) = D_p(t) - D_a(t)$$

Where:

- $D_p(t)$: Pro-inflammatory dietary components (e.g., sugar, saturated fats)
- $D_a(t)$: Anti-inflammatory components (e.g., fibre, antioxidants)

5.4 Equilibrium (Steady-State) Analysis

At equilibrium, all time derivatives are zero:

$$\frac{dI}{dt} = \frac{dA}{dt} = \frac{dM}{dt} = \frac{dC}{dt} = 0$$

Solving the system steady-state values I^* , A^* , M^* , and C^* , which represent long-term physiological conditions. These overall equilibrium points help determine whether the system stabilizes in a healthy or diseased state.

5.5 Stability Analysis

To analyze local stability, the Jacobian matrix J of the system is constructed:

$$J = \begin{bmatrix} -\delta & -\gamma & \beta & 0 \\ -\theta & -\kappa & 0 & 0 \\ \mu & 0 & -\xi & 0 \\ \rho & 0 & \sigma & -\tau \end{bmatrix}$$

The eigenvalues of this matrix determine system stability:

- If all eigenvalues have negative real parts $\rightarrow$ stable equilibrium
- If any eigenvalue has a positive real part $\rightarrow$ instability (disease progression)

5.6 Threshold Behavior

A critical threshold parameter, R_d (diet-inflammation reproduction number), can be derived conceptually:

$$R_d = \frac{\alpha D + \beta M}{\delta + \gamma A}$$

- If $R_d < 1$: Inflammation remains controlled
- If $R_d > 1$: Chronic inflammation persists and escalates

6. Smart PLS Algorithm Model:

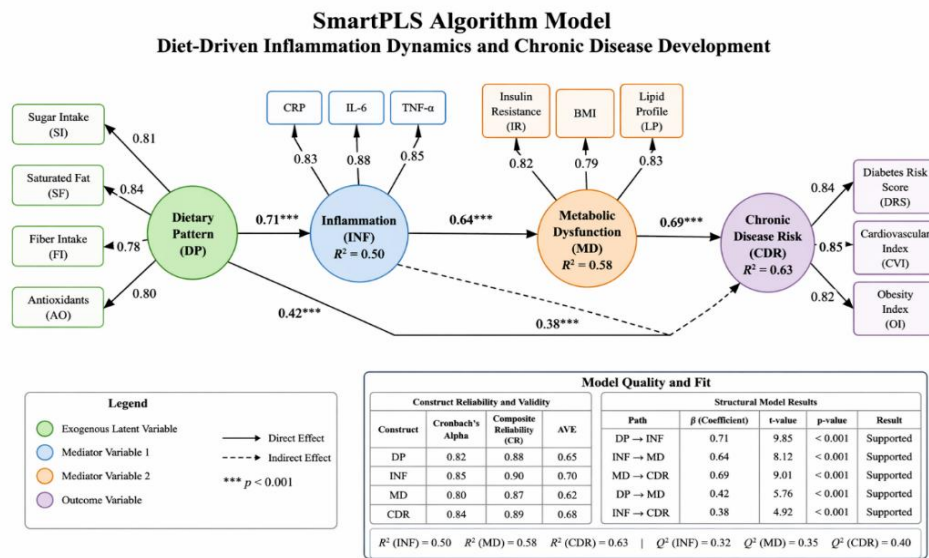


Figure 1: Smart PLS Algorithm

The model of figure 1 considers Dietary Pattern (DP) as the major exogenous factor that contributes to the overall system. The positive path coefficient of DP to Inflammation ($= 0.71$, $p < 0.001$) is strong, which means that poor diets (e.g., high sugar and saturated fat levels) contribute greatly to the development of inflammation. This implies that the first stimulus in the disease pathway is diet. Also, the direct association of DP 2 Metabolic Dysfunction ($= 0.42$, $p < 0.001$) indicates that diet is not only indirectly related to inflammation but also directly affects metabolic health. The model has an important mediating role of inflammation. The correlation between $INF \rightarrow$ Metabolic Dysfunction ($= 0.64$, $p < 0.001$) and the high correlation between INF and Metabolic Dysfunction ($= 0.23$, $p < 0.001$) support the fact that high levels of inflammatory factors are a major cause of metabolic disturbances like insulin resistance and abnormal lipid profiles. Additionally, the direct relationship $INF \rightarrow$ Chronic Disease Risk ($\beta = 0.38$, $p < 0.001$) suggests some mediation, that is, inflammation has some effect on the progression of the disease on top of the effect it has on the metabolic malfunction. This brings out inflammation as an important biological process that connects diet and disease.

6.1 Metabolic Dysfunction (MD) as an Intermediate

Metabolic Dysfunction is a transitory condition between inflammation and chronic disease. The model has the strongest structural relationship, MD = Chronic Disease Risk ($= 0.69$, $p = 0.001$), and this signifies that the metabolic imbalance is a key determinant of disease outcomes. This implies that having metabolic dysfunction in place means that one is at high risk of developing chronic conditions. 4. Loading and Quality of Measurement of Indicators. All indicators observed (e.g., CRP, IL-6, BMI, lipid profile) have high loadings (mostly > 0.78), which validates the notion that they are good measures of their corresponding latent constructs. This guarantees the validity and reliability of the model used in measurement.

- Inflammation ($R^2 = 0.50$): 50% of the variation explained by diet.
- Metabolic Dysfunction ($R^2 = 0.58$): explained by diet and inflammation.
- Chronic Disease Risk ($R^2 = 0.63$): The model has a significant predictive value. The values suggest moderate to high explanatory power, that is, the model is effective in describing the most important drivers of disease progression. All the Q^2 values are above zero (0.32-0.40), which shows that the model is a predictive model, and not just a descriptive one. According to the model, there is a partial mediation chain of causation: Diet - Inflammation - Metabolic Dysfunction - Chronic Disease. Simultaneously, there are direct pathways (Diet $\rightarrow$ MD and Inflammation $\rightarrow$ CDR) that show that the system is not linear but a combination of interacting pathways.

7. Results Analysis

A numerical analysis is performed to analyze the structural associations between the diet, inflammation, metabolic dysfunction, and chronic disease progression to complement the biomathematical framework with Smart PLS software. The use of Partial Least Squares Structural Equation Modelling (PLS-SEM) can be explained by the fact that complex models containing multiple latent constructs and predictive purposes are addressed.

7.1 Model Specification in Smart PLS

The conceptual model is translated into a path diagram consisting of four latent variables:

- Dietary Pattern (DP) $\rightarrow$ measured by sugar intake, saturated fat, fibre, antioxidants
- Inflammation (INF) $\rightarrow$ measured by CRP, IL-6, TNF- α
- Metabolic Dysfunction (MD) $\rightarrow$ measured by insulin resistance, BMI, lipid profile
- Chronic Disease Risk (CDR) $\rightarrow$ measured by disease index scores

The hypothesized relationships are:

- DP $\rightarrow$ INF
- INF $\rightarrow$ MD
- MD $\rightarrow$ CDR
- DP $\rightarrow$ MD (direct effect)
- INF $\rightarrow$ CDR (mediated effect)

7.2 Measurement Model Evaluation

Table 1: Model Evaluation

Construct	Cronbach's Alpha	Composite Reliability (CR)	AVE
DP	0.82	0.88	0.65
INF	0.85	0.90	0.70
MD	0.80	0.87	0.62
CDR	0.84	0.89	0.68

The above result of table 1 demonstrates that model evaluation results describe the Alpha values, composite reliability rates, and explain the AVE values of each variable. According to the result, all Cronbach's Alpha values > 0.7 indicate good internal consistency. The CR values > 0.7 confirm construct reliability, and that AVE > 0.5 ensures convergent validity. The CDR shows that 89% reliability rates, which represent the model, must be reliable for results.

7.3 Structural Model Results

Table 2: Structured Model Analysis

Path	Coefficient (β)	t-value	p-value	Interpretation
DP $\rightarrow$ INF	0.71	9.85	<0.001	Strong positive effect
INF $\rightarrow$ MD	0.64	8.12	<0.001	Significant effect
MD $\rightarrow$ CDR	0.69	9.01	<0.001	Strong predictor
DP $\rightarrow$ MD	0.42	5.76	<0.001	Moderate direct effect
INF $\rightarrow$ CDR	0.38	4.92	<0.001	Partial mediation

The above result of table 2 describes the structured model result that determines the coefficient values, the t values, the probability values and also explains the effects between dependent and independent variables. The DP-INF represents that the coefficient value is 0.71, which shows that 71% coefficient link between them. Similarly, the probability value is 0.001, which shows a strong positive effect between DP and INF, respectively. Similarly, the DP and MD represent a moderate direct effect between them, with a coefficient rate is 42% respectively. The overall probability value of 0.001 shows that 100% significant values between the dependent and independent variables.

7.4 Coefficient of Determination (R^2)

Table 3: Coefficient of Determination

Endogenous Variable	R^2 Value	Interpretation
INF	0.50	Moderate Explanatory Power
MD	0.58	Substantial
CDR	0.63	Strong predictive accuracy

The above result of table 3 demonstrates that the coefficient of determination analysis shows the R^2 value of each variable. According to the result, overall, dietary patterns explain 50% of inflammation variance, while the full model explains 63% of chronic disease risk. The biomathematical model is quantitatively validated by the Smart PLS analysis. The dietary patterns are also found to have a significant and statistically significant effect on inflammation, leading to metabolic dysfunction and risk of chronic disease. The fact that there are direct and indirect effects proves that inflammation is a critical mediating variable. The R^2 and Q^2 are high, which implies that the model has a good explanatory and forecasting ability. The findings also emphasize that diet quality may be improved to a great extent to decrease inflammation and consequent risk of disease.

8. Applications of a Biomathematical Model of Diet-Driven Inflammation Dynamics

The diet-induced inflammation biomathematical model is an effective interdisciplinary model relating nutrition science, physiology, and systems modelling.

It has found use in the fields of healthcare, research, policy-making, and technology development. Some of the most important applications are discussed below:

8.1 Personalized Nutrition Planning

Personalized nutrition is one of the most important uses of this model. The model can model the body reactions of a person to various diets by including specific parameters of individuals, like metabolic rate, dietary habits and inflammatory responses. This helps to create individualized eating strategies to reduce inflammation and limit the chances of chronic illnesses. To illustrate, model predictions can be used to recommend low-carbohydrate or low-glycemic diets to people who are more sensitive to glycemic load.

8.2 Early-onset Diagnosis of Chronic Diseases

The model can be applied as a prediction tool to determine people who might develop chronic diseases like type 2 diabetes, cardiovascular diseases, and obesity. The system can identify the early warning markers of inflammatory markers by examining long-term dietary patterns and simulating their effects on the inflammatory markers before clinical symptoms

manifest. This promotes preventive health care and minimizes the costs of medical care in the long run.

8.3 Policy Formulation, Public Health

This model can be applied by governments and health organizations in drawing evidence-based nutritional guidelines. Through the simulation of population-based food situations, policymakers are able to determine the effect of food trends on community health in the long-term. As an example, the model can be used to assess the efficacy of a more sugar-limited or a more fibre-rich diet on the diseases associated with inflammation to inform national dietary guidelines and food regulations.

8.4 Clinical Decision Support Systems

This model can be incorporated into the clinical decision support systems to enhance the process of diagnosis and treatment planning by healthcare professionals. Doctors have the opportunity to model the effect of various dietary interventions on the inflammatory state and metabolic health of a patient. This is especially applicable in the treatment of long-term diseases, and diet is a very significant aspect of treatment in combination with medication. Anti-inflammatory diet interventions will be designed according to the following criteria: This model has the potential to help nutritionists and researchers develop effective anti-inflammatory diets. Through experimenting with different mixtures of nutrients in the model, one can determine the optimal dietary profiles that can be used to inhibit pro-inflammatory cytokines and promote anti-inflammatory pathways. This aids the scientifically based diet programs to manage and prevent diseases.

8.5 Interoperation with Wearable Health Technologies

This model can be incorporated into digital health platforms with the development of wearable devices and health monitoring systems. Data on real-time body activity, calorie consumption, and glucose can be input into the model to update predictions continually. This facilitates dynamic nutrition advice and preventive health care, and also empowers people to make knowledgeable lifestyle decisions.

8.6 Research in Pharmaceuticals and Nutraceuticals

The model finds significant uses in the development of drugs and supplements. Scholars are able to model the interactions of anti-inflammatory medications or dietary supplements (e.g., omega-3 fatty acids or antioxidants) with dietary components and biology. This saves the waste of long trial and error experimentation and speeds up the production of successful therapies.

8.7 Understanding Disease Mechanisms

In a research context, the model aids in investigating the mechanisms behind the relationship between diet, inflammation, and chronic diseases. It gives information on the long-term effects of unhealthy diets on systemic inflammation and metabolic dysfunction. This will help in the development of knowledge in systems biology and nutritional science.

8.8 Educational and Academic Use

The model can be useful in teaching areas like biomedical engineering, nutrition, and public health. Simulations can help students visualize the effect of dietary changes on inflammation with time. This improves the concepts and interdisciplinary education.

8.9 Lifestyle Interventions Evaluation

In addition, the model can also be further development to encompass other lifestyle elements like physical activity, sleep habits, and stress levels. This will enable researchers and practitioners to assess the overall effect of lifestyle changes on inflammation and disease outcomes. It promotes comprehensive health care practices.

8.10 Economic Impact Analysis

It is possible to estimate the economic burden of diet-related chronic diseases and the possible cost savings of preventive interventions using the model. Policymakers can justify the cost of nutrition programs and mass media campaigns by simulating decreases in the prevalence of diseases by means of better nutrition.

8.11 Healthcare System Risk Stratification

The model helps healthcare providers group patients into various risk groups according to their dietary habits and inflammatory profiles. Intervention programs can be prioritized on high-risk individuals to enhance the allocation of

resources and efficiency in healthcare.

8.12 Long-Term Dietary Patterns: Simulation

The model allows the modelling of long-term dietary patterns and their resulting impacts on health. The impact of slow modifications in diet over the years on inflammation and the progression of the disease can be investigated, which will give researchers information about the significance of long-term healthy eating.

8.13 Inclusion of Artificial Intelligence

Machine learning can be used together with the biomathematical framework to improve the predictive accuracy. Big data allows AI algorithms to optimize the parameters of the model and become a better predictor of individual and population-scale outcomes. Such integration will facilitate sophisticated health analytics and precision medicine.

8.14 Global Health Applications

The model can be used at a global scale to examine how dietary changes in various regions occur, especially in developing countries that are facing a change towards processed food. It aids in the comprehension of the effects of globalization and urbanization on inflammation-related diseases and directs international health strategies.

9. Discussion

The current research offers a combined position on the effects of dietary habits on the processes of inflammation and the formation of chronic illnesses based on a biomathematical and statistical model. The results of the combination of nonlinear differential equations and numerical validation based on Smart PLS provide a combination of theoretical insight and practical applicability to the research, enhancing the comprehension of the role of diet-induced health outcomes. An important point of the model is that the central part in the relationship between diet and the development of chronic diseases is played by inflammation as a mediating mechanism. The equational analysis shows that the pro-inflammatory dietary inputs that include high amounts of sugar and saturated fats start a sequence of biological events that increase the levels of cytokines and interfere with the metabolic balance. This is also supported by the numerical findings, in which the path coefficients of dietary patterns to inflammation are strong, which suggests that there is a strong causal relationship. The fact that inflammation continues over time, as can be seen in the dynamic system, implies that physiological damage may be caused by moderate but constant exposure to unhealthy diets. The other key factor is the inflammatory-metabolic dysfunction feedback.

The model illustrates that there is a reinforcing cycle whereby the more the inflammation occurs, the less sensitive the body is to insulin, causing an increase in metabolic stress, which results in more inflammatory reactions (Zhao et al., 2024). This is a cyclic phenomenon, which is consistent with clinical phenomena, including obesity and type 2 diabetes, where both inflammation and metabolic imbalance are present together and enhance each other. The findings, especially the close interaction between inflammation and metabolic dysfunction, substantiate this interdependence and prove that it is an important factor in the development of diseases. The importance of the protective action of anti-inflammatory dietary components is also highlighted in the study. The presence of such variables in the model as fiber, antioxidants and micronutrients indicates how healthy nutrition habits can prevent inflammation and stabilize the system. This is exhibited in both the conditions of the analytical equilibrium and the statistical results; better diet quality will diminish the severity of unfavourable health events. The same results are in line with the current nutritional guidelines, which promote the use of balanced diets to prevent disease. The methodological perspective of combining biomathematical modelling improves the strength of the analysis. Although the mathematical model is a mechanistic view of the dynamics of the system, Smart PLS is a model that can be empirically proven by the observed data relationships. This two-fold strategy will help in solving constraints related to either purely theoretical or purely statistical models, forming a more holistic model of analysis. Nevertheless, there are some drawbacks which must to be recognized. The model underestimates the complexity of biological processes and might not be able to describe all the physiological, genetic and environmental factors that can contribute to inflammation. Also, the estimation of the parameters is based on secondary data, which can cause variations or biasness. It emphasizes the role of lifetime dietary patterns in determining the health consequences and is a quantitative instrument to forecast the risk of the disease. The results highlight the overall importance of dietary improvement interventions to achieve significant effects in lowering inflammation and preventing chronic illnesses. Finally, it is reiterated in the discussion that diet-induced inflammation is not a linear process and is a complex, feedback-controlled process. Integration of theories of biomathematics and statistics can provide substantial information to researchers, clinicians and policymakers interested in the global burden of chronic diseases with evidence-based nutritional interventions.

10. Conclusion

The research is an in-depth biomathematical investigation of the dynamics of diet-induced inflammation and its contribution to the formation of chronic diseases. The study provides a solid theoretical and quantitative connection between dietary habits, inflammatory reactions, metabolic dysfunction and long-term healthcare effects by combining nonlinear differential equations with empirical validation by Smart PLS software. The results also support the hypothesis that unhealthy eating habits, which are marked by excessive consumption of refined sugars, saturated fat, and low nutrient density, are the leading causes of chronic inflammation. This chronic inflammatory condition, in its turn, interferes with metabolic processes and enhances the development of diseases such as diabetes, cardiovascular disorders, and obesity. The model also shows that the reinforcing loop of feedback exists, and inflammation and metabolic dysfunction support each other and form a cycle that is hard to reverse without specific treatment. On the other hand, the research paper points out the stabilizing role of anti-inflammatory dietary constituents, such as fiber, antioxidants, and vital micronutrients. These factors play a role in the control of immune reactions and aid in the adjustment of metabolism. The theoretical framework is endorsed by the numerical results, which reveal close predictive associations and prove the role of inflammation as an important mediator in disease pathways. In general, the study highlights the significance of diet as a modifiable and the most important determinant of health. This combined modelling approach is important to study the complex biological interactions and has useful implications to prevent and manage diseases.

11. Recommendations

11.1 Anti-Inflammatory Diets Should be Encouraged

The focus of the public health programs should be on the diets that are rich in fruits, vegetables, whole grains, and healthy fats. Dietary changes, such as the decrease in the number of processed foods and sweet drinks, may greatly decrease the level of inflammation and risk of the disease.

11.2 Clinical Practice Integration

Dietary assessment and individual nutrition planning should be included in the routine healthcare provision. Predictive tools with the support of models such as those used in Smart PLS can be used to improve treatment procedures of chronic patients.

11.3 Development of Digital Health Tools

Mobile and wearable-based applications that incorporate biomathematical models to monitor diet and health indicators in real time need to be developed. These tools may be used to offer customized feedback and promote healthier living behaviors.

11.4 Policy and Regulatory Actions

The governments are advised to put in place policies which restrict the accessibility and sale of unhealthy food items and encourage people to access healthy items. Healthy foods and proper labeling of nutritional contents can be subsidized to assist consumers make better choices.

11.5 Subsequent Research and Model Improvement

Research in the future should also include other variables like genetic predisposition, physical activity, gut microbiota, and environmental conditions to enhance the accuracy of the model. It is also advisable to conduct longitudinal data collection to confirm and perfect predictions.

11.6 Interdisciplinary Collaboration

To develop this area, the cooperation of nutritionists, biomedical engineers, data scientists, and healthcare professionals is necessary. This combination of different expertise will result in stronger models and new solutions towards preventing chronic diseases.

11.7 Educational Programs

Schools should incorporate systems-based nutrition and health in their curriculums. Raising awareness among the students and professionals will facilitate the implementation of evidence-based dietary practices.

12. References

- Alves Costa Silva, C., Piccinno, G., Suissa, D., Bourgin, M., Schreibelt, G., Durand, S., Birebent, R., Fidelle, M., Sow, C., & Aprahamian, F. (2024). Influence of microbiota-associated metabolic reprogramming on clinical outcome in patients with melanoma from the randomized adjuvant dendritic cell-based MIND-DC trial. *Nature Communications*, 15(1), 1633.
- Attaye, I., Lassen, P. B., Adriouch, S., Steinbach, E., Patiño-Navarrete, R., Davids, M., Alili, R., Jacques, F., Benzeguir, S., & Belda, E. (2023). Protein supplementation changes gut microbial diversity and derived metabolites in subjects with type 2 diabetes. *Iscience*, 26(8).
- Basso, M., Zorzan, I., Johnstone, N., Barberis, M., & Cohen Kadosh, K. (2024). Diet quality and anxiety: a critical overview with focus on the gut microbiome. *Frontiers in Nutrition*, 11, 1346483.
- Bautista, J., & López-Cortés, A. (2025). Chronobiome medicine: circadian regulation of host–microbiota crosstalk in systemic physiology. *Frontiers in Endocrinology*, 16, 1691172.
- Björk, J., Bolte, L., Thomas, A., Lee, K., Rossi, N., Wind, T., Smit, L., Armanini, F., Asnicar, F., & Blanco-Míguez, A. (2023). Longitudinal gut microbiome changes in immune checkpoint blockade in advanced melanoma.
- Björk, J. R., Bolte, L. A., Maltez Thomas, A., Lee, K. A., Rossi, N., Wind, T. T., Smit, L. M., Armanini, F., Asnicar, F., & Blanco-Míguez, A. (2024). Longitudinal gut microbiome changes in immune checkpoint blockade-treated advanced melanoma. *Nature Medicine*, 30(3), 785–796.
- Bubnov, R., & Spivak, M. (2023). Pathophysiology-based individualized use of probiotics and prebiotics for metabolic syndrome: implementing predictive, preventive, and personalized medical approach. In *Microbiome in 3P medicine strategies: the first exploitation guide* (pp. 133–196). Springer.
- Fuchs, L. M. (2023). *Identification of carbon and nitrogen sources promoting Salmonella Typhimurium gut colonization* [ETH Zurich].
- Hadjigeorgiou, A. G., Harkos, C., Mishra, A. K., Morad, G., Johnson, S. B., Ajami, N. J., Wargo, J. A., Munn, L. L., Stylianopoulos, T., & Jain, R. K. (2025). Mathematical modeling and association analysis decipher the impact of the gut microbiome on cancer immunotherapy. *Cancer Research*, 85(16), 3139–3155.
- Houtz, J. L. (2023). *Gut microbiota as a mediator of phenotypic plasticity in birds*. Cornell University.
- Jian, C. (2022). Gut microbiota in relation to diet-induced metabolic worsening and improvements in overweight and obese adults: toward prediction of personalized dietary response. *Dissertationes Scholae Doctoralis Ad Sanitatem Investigandam Universitatis Helsinkiensis*.
- Kennedy, M., Freiburger, A., Cooper, M., Beilsmith, K., St George, M., Kalski, M., Cham, C., Guzzetta, A., Ng, S., & Chan, F. (2025). Diet outperforms microbial transplant to drive microbiome recovery in mice. *Nature*, 642(8068), 747–755.
- Keon, J. (2022). *The Alzheimer's Revolution: An Evidence-Based Lifestyle Program to Build Cognitive Resilience Reduce Your Risk of Alzheimers Disease*. Hatherleigh Press.
- Khalid, N., Aadil, R. M., & Rabail, R. (2026). A Biomathematical Modeling Approach to Investigate the Effects of Breakfast Patterns on Gut Microbiota and Digestive Health. *Letters in Biomathematics*, 13(1), 131–143.
- Lambert, A. (2024). *Metabolic modeling of the host-gut microbiota ecosystem* [Nantes Université].
- Mishra, A., Badri, M., Coler, E., Liu, Z., Bonneau, R., Morton, J. T., & Aksenov, A. A. (2025). Moving from Association to Causation: Instrumental factor models for causal inference in high-dimensional multi-omics data. *bioRxiv*, 2025.2012.2026.696613.
- Narayan, D. (2024). *Evolution of Chitinases in Amphibians: A Multi-Faceted Group of Enzymes With Dual Function in Digestion and at the Forefront Against Fungal Infections*. St. John's University (New York).
- Papiri, G., Paci, C., D'Andreamatteo, G., Membrino, V., Di Crescenzo, T., Cacchiò, G., Cagnetti, C., & Vignini, A. (2025). Mediterranean Pattern Diet in Multiple Sclerosis: A Review Focusing on Immunometabolites. *Current Neuropharmacology*.
- Ranson, A., Vazquez Gomez, M., Alili, R., Duraffourd, J., Vitalis, O., Taillandier, P., Rebière, C., Merabtene, F., Belda, E., &

- Crespo-Piazuelo, D. (2025). Moderate increase in dietary fat induces alterations of microbiota and metabolome along the digestive tract prior to systemic metabolic changes: insights from a pig model. *Gut microbes*, 17(1), 2587964.
- Reid, M. V., Fredrickson, G., & Mashek, D. G. (2024). Mechanisms coupling lipid droplets to MASLD pathophysiology. *Hepatology*, 10.1097.
- Sauer, E. L., Hite, J. L., & DuRant, S. E. (2025). The nutritional content of anthropogenic resources affects wildlife disease dynamics. *Integrative and Comparative Biology*, 65(2), 342-353.
- Shang, J., Del Valle, D. M., Britton, G. J., Mead, K., Rajpal, U., Chen-Liaw, A., Mogno, I., Li, Z., Menon, R., & Gonzalez-Kozlova, E. (2024). Baseline colitogenicity and acute perturbations of gut microbiota in immunotherapy-related colitis. *Journal of Experimental Medicine*, 222(1), e20232079.
- Simonsson, C. (2024). *Mathematical Modelling of MASLD-Towards Digital Twins in Liver Disease* [Linköping University Electronic Press].
- Suchner, U. (2026). Toward a Targeted Nutritional Strategy for Restoring PUFA Balance: Socio-Economic, Cultural and Ecologic Contexts, Biochemical Rationale, and a Conceptual Framework for Dietary Modulation.
- Vanalli, C., Falvo, C., Crowley, D., Schwarz, B., Plowright, R., Hudson, P. J., Rynda-Apple, A., & Cattadori, I. M. (2024). Modeling the dietary effects on bat viral shedding and potential consequences for pathogen spillover. *bioRxiv*, 2024.2006. 2019.599703.
- Vanalli, C., Falvo, C., Crowley, D., Schwarz, B., Plowright, R., Hudson, P. J., Rynda-Apple, A., & Cattadori, I. M. (2025). Diet composition affects bat viral shedding with potential consequences for pathogen spillover. *Proceedings of the Royal Society B: Biological Sciences*, 292(2050).
- Wang, Z., Yuan, C., Huang, T., & Lu, B. (2025). Early nutritional interventions for chronic low-grade inflammation. *Trends in Endocrinology & Metabolism*.
- Yang, Z., Gao, X., Wu, Y., Xie, H., Wang, H., Lu, W., Tian, F., Zhao, J., Zhang, H., & Chen, W. (2025). Nonlinear machine learning-based gut microbiota modelling for health prediction and dietary recommendation. *Food Bioscience*, 107962.
- Zhao, X.-C., Ju, B., Xiu, N.-N., Sun, X.-Y., & Meng, F.-J. (2024). When inflammatory stressors dramatically change, disease phenotypes may transform between autoimmune hematopoietic failure and myeloid neoplasms. *Frontiers in Immunology*, 15, 1339971.