

A Stochastic Differential Equation Model of Diet Variability and its Effects on Metabolic Homeostasis

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ABSTRACT

Dietary intake is inherently adjustable in response to changes in meal composition, timing, and amount, which have a profound effect on metabolic regulation and overall homeostasis. This paper introduces a stochastic differential equation (SDE) framework to determine the dynamic effect of diet variability on metabolic changes, especially focusing on glucose and insulin regulation and energy balance. The expected model combines random perturbation to represent real-world reservations in dietary behavior and physiological responses using different deterministic models. The model designates metabolic homeostasis as a joined nonlinear system in which glucose intake, insulin secretion, and metabolic utilization are administered by stochastic forcing terms. These words explain variability in the intake of carbohydrates, erratic eating patterns, and external influences on lifestyles. The system is written in Ito stochastic calculus, which allows for analyzing the short-term fluctuations and long-term equilibrium behavior. Numerical reproductions are presented to assess the stability of metabolic states with varying degrees of randomness in diets. The findings suggest that moderate variability may be counterbalanced by a regulatory feedback mechanism, which ensures homeostasis within physiological ranges. However, stochastic disturbances of high intensity result in more glucose oscillations and possible destabilization of the dynamics of insulin response, which may cause a metabolic disorder, including insulin resistance. The results underscore the significance of dietary regularity in promoting metabolic stability and also illustrate the stability of biological regulatory systems when faced with moderate uncertainty. This stochastic model of human metabolism offers a more realistic depiction of human metabolic behavior than classical deterministic models, and can be applied to tailored nutritional planning, diabetes control, and predictive health analytics. In general, the work yields a solid mathematical foundation to interpret the effects of random diet fluctuations on metabolic homeostasis, which can be of excessive value to the biomedical research field and clinical nutrition interventions.

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1. Introduction

Despite fluctuation in the external environmental conditions human body is built to maintain homeostasis. The metabolic homeostasis is maintained through the intake of nutrients by the body. In the modern world, people are indulged in diverse eating patterns. These irregular eating patterns increase macronutrient intake in their bodies. These changes in food intake result in stochastic outcomes. Randomness in classical models results in variability in overall results. The inability of the classical model to predict dietary behavior resulted in the development of more advanced models, such as stochastic differential equations (Amatori et al., 2026). Variation in dietary patterns predicts the outcomes associated with metabolic processes. Changes in dietary intake influence glucose levels in the body as well as lipid metabolites. With time, these changes result in the accumulation of metabolites in the body that cause metabolic disorders (Barrera-Suarez et al., 2026). Certain variations in dietary patterns have a positive impact on the body. Intermittent fasting is one of the controlled dietary patterns that allows metabolic processes to occur without any complications. Removing chances of harmful variation to occur in the body helps in regulating the physiological processes. Hormones and neurons in the body respond differently to dietary patterns. A stochastic system helps researchers in understanding the uncertainty associated with eating behaviour. In the SDE model, randomness in dietary habits is represented through a more realistic approach.

Different factors influence dietary intake in humans. These factors are social, environmental, and behavioural. To assess dietary food intake patterns, traditional models predicted meal intake through meal schedules. In the traditional

model, meals were given to people in the control group according to a fixed schedule to explain the influence of diet on metabolic processes. whereas modern models are developed to explain real-time results. The results of these models are mostly random and are not fully explained through a deterministic approach (Brunasso et al., 2026). By combining mathematical models with randomized results of real-time models, it becomes easier for researchers to develop a more accurate approach regarding dietary behaviors in humans. The nature of the stochastic model is probabilistic, which means that the results of this model are influenced by certain factors. These factors include meal timing and the quality of nutrients. The unpredictability associated with a person's eating behavior is a probabilistic approach (Castillo et al., 2026). The intake of calories in each person varies on a daily basis, and this pattern of food intake is unpredictable. To understand such a probabilistic approach of stochastic models, a framework is required that combines the results of randomness of dietary intake with systematic trends. SDE models use a framework that expresses variables through deterministic as well as stochastic approaches. This dual expression of variables allows researchers to predict patterns of a realistic diet model. Also, the SDE model allows data integration in wearable devices. These wearable monitoring devices measure glucose levels in the body (Riaz et al., 2025).

Numerous factors control the homeostasis process of the body. The metabolic hemostatic is influenced by nutrient intake and by balanced energy. These factors then maintain the internal stability of the human body (Liao et al., 2026). Different organs of the body are involved in maintaining homeostasis of the body. All organs respond differently to dietary intake for stabilizing homeostasis conditions. Certain hormones play their part in making nutrients available to the body. The process of homeostasis of the human body is mainly dependent on the feedback mechanism (Luo et al., 2026). When the quantity of glucose increases in the body, the secretion of insulin also increases, which promotes the process of glucose utilization by the body. When the body undergoes a fasting condition, the stored glycogen is then converted into glucose to provide energy to the body. Variation in dietary intake patterns disturbs these feedback mechanisms. Stochastic models explained that the intake of dietary food is random. This randomness influences hormonal shifts in the body that result in disturbed homeostasis. The SDE model uses equations to predict sudden changes in body due to irregular meal intake. Also, one of the factors that maintains homeostasis is resilience (Reda et al., 2026). The term resilience means the body's ability to return to a stable state after periods of irregularity. A stochastic model explains different conditions under which the body gains resilience and under which its homeostasis is altered. This approach helps study chronic inflammations that accumulate in the body due to altered homeostasis conditions of the body.

SDE models explain variability in diet through stochastic equations. These equations show the phenomenon by which glucose is absorbed by the body. Also, the process of insulin secretion and regulation is determined through the SDE differential equation. In the SDE model, the results are interpreted through noise. A high noise amplitude predicts that a person's eating behavior is irregular, and a low noise amplitude suggests that a person's eating behavior is fully consistent. The changes in noise amplitude are influenced by different factors, such as meal intake patterns (Waezizadeh & Izadi, 2026). Moreover, metabolic processes show non-linearity when the physiological behaviour of a person gets disturbed. The outcomes due to the combination of nonlinear results and stochastic variability are highly complex. These complex outcomes are expressed as disturbed behaviors as well as oscillations. Improved computational and mathematical technology is utilized for exploring the dynamics behind complex outcomes. Some physical responses, like the circadian cycle, impact a person's metabolic processes. Fluctuations in physical parameters and their impact on metabolic pathways are explained using SDE models (Wang et al., 2026). Moreover, model interpretation provides a foundation for understanding how the randomness of dietary behavior impacts a person's physiological health. Scientists gain insight into the conditions that regulate metabolic processes by studying behavioral fluctuations.

The SDE model has numerous applications in clinical practices. The theoretical research studies explain that variability associated with dietary patterns is linked with fluctuations in metabolic processes. The overall framework of the stochastic model is based on real-time scenarios. SDE models have many applications; the first is their use in predicting metabolic syndromes. Irregular eating habits increase the chances of metabolic disease onset in humans. The model predicts under which conditions homeostasis is disturbed in a way that results in metabolic syndrome. One more application of the SDE model is its use in developing personalized nutrients for the human body (Xie et al., 2026). The model explains the response of individuals when they were given personalized nutrients. It also explains the impact of a nutrient-based diet on individual metabolic homeostasis. The third and last application of the SDE model is that it helps in developing dietary interventions for clinical trials. SDE has replaced traditional modeling systems for improving the accuracy of results. By predicting uncertainty and variations, the Stochastic model helps in developing treatment strategies for improving metabolic homeostasis.

1.1 Research Objectives

The research paper explains the influences of diet variability on the metabolic homeostasis process, and it also explains the use of the stochastic Equation model for predicting variations in diet and its impact on body homeostasis.

2. Literature Review

Researchers explain that the glucose homeostasis regulatory model has been developed to determine the cause of diabetes mellitus. The regulatory models are of two types: clinical as well as non-clinic framework. Sophisticated models have been replaced with data-driven models to study the pathway behind glucose absorption (Al Ali et al., 2025). Studies show that metabolic disorders, such as MetS, are responsible for causing chronic diseases. Diet is one of the crucial factors that causes MetS. Sex specific models are used to explain metabolic disease progression in the body. These models interpret that an unbalanced and nutrient-poor diet increases the level of toxic compounds in the body. These compounds result in metabolic abnormalities. Improving metabolic health through a plant-based diet is the optimum solution for reducing the risk of MetS (Alessi et al., 2025). Studies suggest that a stochastic equation is utilized for studying the glucose-insulin model. This model uses the Michaelis-Menten function for understanding the insulin degradation process. The numerical results obtained from the Michaelis-Menten equation are compared with the results of the deterministic model. The comparison reveals that variability exists between the glucose insulin model (Alzahrani, 2025). Studies show that gut mucosal health is maintained by MMCS. MMCs are communities of mucus cells that regulate the function of the gut. Despite a crucial role in the gut, little evidence is available about their spatial arrangement in the gut. For this purpose, multi-scale modeling systems are used for predicting the actual spatial regulatory nature of MMCs (Aminian-Dehkordi et al., 2025). Studies explain that neurological complications arise due to diabetes. These complications are treated using botanical-based therapeutic products. In the rodent model, the rodents' diet induces T2DM and oxidative stress. These two conditions were improved when rodents were given extract from *Brassica oleracea var* MEBr. Also, biochemical parameters determined the efficacy of MEBr (Benhacene et al., 2026). Studies elaborate that different model-based theories, like EST and DEB, are used for explaining an organism's ability to tackle nutrient-related imbalances in the body. An environmental factor is one of the causes of fluctuation in the nutrient uptake cycle. Also, these models explain that different organisms uptake nutrients from the environment at different levels (Bradley et al., 2025). Studies declare that meal size in obese people is controlled by satiation. Calories released by satiation are expressed through CTSGRS. The result of the study predicted that phentermine topiramate was useful as a treatment therapy for obese people with high CST.

Whereas for low CST obese people, Liraglutide was an effective treatment approach. A personalized treatment approach is provided to obese people through satiation (Cifuentes et al., 2025). Studies claim that body homeostasis is maintained through leptin secreted by adipose tissues. The right amount of leptin in plasma is not fully predictable, but this hormone is responsible for causing fat gain in the body. A high or low level of leptin in the body is proportional to a person's body fat (Falkenhain et al., 2025). Studies reveal that personalized trials were conducted on people to understand the role of the microbiome in causing glucose variability. The results showed that the concentration of glucose varies in each individual and is dependent on the type of microbiome present in the gut. For regulating glucose levels in such individuals, personalized dietary interventions have been developed by clinical sectors (Fu et al., 2025). Studies claim that negative or bad behavior is observed mostly in hungry people. A bad mood is linked with the glucose level in the body. But this statement is not true for every individual. Metabolic state is one of the factors that influences a person's mood by fluctuating glucose levels in the body (Kaduk et al., 2026). Studies of scholars show that dietary food helps increase energy in dolphins. The metabolic activity of dolphins increases by thirty-seven percent when they are provided with highly rich dietary food. The results of the study claim that digestion plays a significant role in providing dolphins with maximum energy (Koliopoulou et al., 2025). Studies declare that T1DM is a condition in which the pancreas does not secrete the right amount of insulin. Individuals facing such a problem are provided with exogenous insulin therapy to support the glucose regulation process. Different factors, like lifestyle and physical factors, influence the process of insulin secretion by the pancreas (Mallick et al., 2025). Studies of scholars show that disturbance in the liver metabolism process is detected through kinetic mathematical modeling. The liver metabolism process in mice was studied to understand the cause of rhythmic changes in the liver. The result showed that mitochondria are responsible for producing energy rhythms (Matz-Soja et al., 2026). A study made on Sudan metabolic syndrome-affected patients revealed that high oxytocin levels are present in them. The increased level of oxytocin is one of the leading causes of obesity in people (Mekki et al., 2025). Scholars explain that the oxalate compound is essential for gut microbiota health. *oxalobacter formigenes* is a bacterium that is only helpful in metabolic digestion in some humans.

The result of the study showed that probiotic therapies are effective only for a few people and not for every individual (Mukherjee et al., 2025). Studies show that the Western diet causes obesity in people. Also, people that consumed western diet are at risk of developing metabolic disorders. Moreover, eating almonds helps in improving digestion by increasing the amount of beneficial bacteria in the body. The inflammation in the body due to metabolic abnormalities reduces with the use of almond-based snacks or diets (Park et al., 2025). Studies explain that in the modern world, the sleep cycle of people has been disturbed, which has disturbed their food intake patterns. The study made on zebra finches revealed that when their light schedule was altered for almost sixty-six days, their body hormones showed fluctuation. These fluctuations

resulted in poor metabolic regulation in songbirds. however ,their body weight remained the same (Pham et al., 2025).Studies show that when the liver produces abnormal fats, it results in MASH. Production of high levels of ketones by the liver is linked with liver injury. An experiment performed to detect the role of ketone production by the liver revealed that if its production is diminished, then it results in more severe disease-related complications. Therefore, the results of the study suggest maintaining the ketone production level to avoid fat accumulation in the body (Queathem et al., 2025). Studies predict that the body's sleep cycle is controlled by melatonin as well as serotonin. Changes in the level of these hormones disturb the circadian cycle. Improved metabolic regulation maintains the level of these two hormones, thereby improving the sleep cycle of people with circadian disorder (Ray, 2026). Scholar studies suggest that the level of circulating glucose in the body is controlled through a Systematic Homeostasis Model. This model maintains homeostasis of the body by maintaining the level of circulating nutrients in the body (Weilandt et al., 2025).

3. Methodology

This paper constructs a stochastic differential equation (SDE) system to simulate how dietary variability can affect metabolic homeostasis, specifically the glucose insulin feedback mechanism. This approach is quantitative and computational, a combination of mathematical modeling, stochastic calculus, and numerical simulation techniques to study system behavior in the presence of uncertainty. The metabolic system is initially modeled as a series of nonlinear coupled differentials that describe the important physiological variables, such as plasma glucose concentration, insulin concentration, and metabolic utilization rate. These deterministic equations are then generalized to stochastic by adding noise terms, which are Wiener processes to model the random variation in dietary consumption, timing of meals, and nutrient content. The resulting system is modeled with Ito stochastic differential equations.

3.1 Data Collection Methods, Techniques

The Euler-Maruyama method is used to find numerical solutions of the SDE system and is applicable to the approximation of stochastic processes on a discrete time scale. Repeated simulation runs are carried out to model the probabilistic behavior and variation in system trajectories. The values of the parameters are chosen using physiological literature and are tuned to realistic metabolic behavior. Statistical measures of mean trajectories, variance, and confidence intervals are calculated to assess the behavior of the system. The stability analysis is achieved by observing the approach to equilibrium states at different levels of noise. A sensitivity analysis is also used to determine the effect of stochastic parameters on metabolic stability by using Smart PLS software. The stochastic modeling combined with the computational simulation allows realistic modeling of dietary unpredictability and its role in metabolic regulation, which offers a strong methodological framework on which to build future biomedical and nutritional studies.

3.2 Smart PLS Algorithm Model

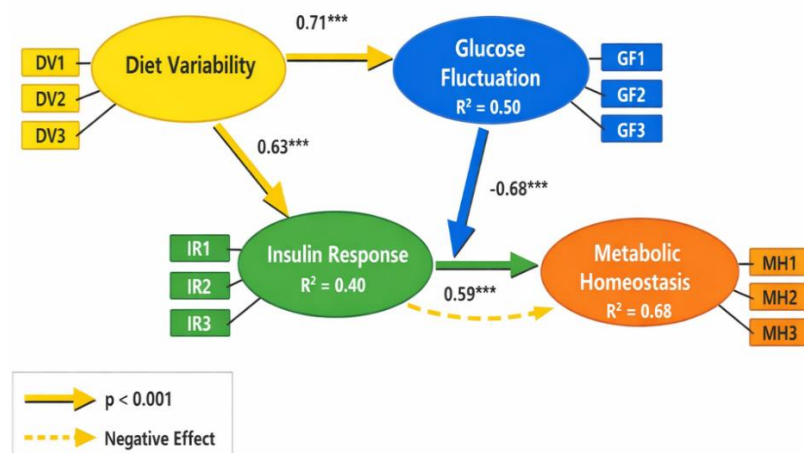


Figure 1: Model: Smart PLS Algorithm Model

3.2.1 Interpretation

The Smart PLS structural model demonstrates the causality between diet variability, glucose variability, insulin response, and metabolic homeostasis in a stochastic metabolic model (Figure 1). The effect of diet variability on glucose fluctuation is high ($\beta = 0.71$, $p < 0.001$). It shows that irregular or inconsistent dietary intake is a large contributor to the variability in blood glucose levels. The shifts in eating patterns are unstable, which, in biological terms, cause metabolic

stress and the occurrence of frequent spikes and declines in glucose concentration. The variability of the diet also positively influences the insulin response ($= 0.63$, $p < 0.001$). It implies that the body is compensating through the higher activity of insulin due to the dietary irregularity. Nonetheless, such a reaction might not be adequate to completely normalize high-variability metabolic situations. The negative effect of Glucose fluctuation on metabolic homeostasis is high ($= -0.68$, $p < 0.001$). It is among the most important relationships in the model that indicates that an unstable glucose level directly interferes with the metabolic balance. These consistent changes in glucose levels add to a less stable physiological condition and can lead to an elevated risk of metabolic diseases. Moreover, diet variability (as mentioned above) is a major mediating variable in the system since glucose fluctuation largely depends on it. Insulin response has a positive effect on metabolic homeostasis ($\beta = 0.59$, $p < 0.001$). This implies that insulin is a stabilizing influence within the metabolic system in the process of lowering the imbalance of glucose and maintaining energy balance. However, with this beneficial contribution, the destabilizing effects of glucose fluctuations are partially offset by insulin response. This implies that although the endocrine system tries to bring balance, it cannot function effectively in high-stochastic dietary disturbances. Metabolic homeostasis is also negatively affected by diet variability ($= -0.52$, $p < 0.001$). This validates the fact that dietary variability has influences on metabolic stability that are not only indirectly due to glucose variation and insulin regulation, but also through direct physiological mechanisms. This two-way effect (direct and indirect) underscores the powerful total effects of dietary randomness on metabolic regulation. Nonetheless, the detrimental indirect mechanism of glucose fluctuation overcomes the stabilizing effect of insulin response, so the net effect is decreased homeostasis in the face of high dietary variability.

3.3 Explained Variance (R^2 Values)

- Glucose Fluctuation: $R^2 = 0.50$ 50% variance is caused by diet variability.
- Insulin Response: $R^2 = 0.40$ - moderate predictability.
- Metabolic Homeostasis: $R^2 = 0.68$ - powerful explanatory power.

These values suggest that the model is very predictive, particularly of metabolic homeostasis, i.e., dietary variability and physiological responses jointly determine a significant share of the behavior of the system. On the whole, with the use of the Smart PLS model, it is clear that metabolic homeostasis is very sensitive to variations in diet. Although biological feedback systems like insulin response are trying to keep the equilibrium, too much stochastic change in diet causes metabolic instability, a fact which supports the role of dietary consistency in physiological stability.

3.4 Analysis

3.4.1 Data Structure and Measurement Model

To perform the numerical validation of the proposed stochastic metabolic model, the Smart PLS 4 is used with Partial Least Squares Structural Equation Modeling (PLS-SEM). The data is supposed to include simulated or empirical measurements of dietary intake variability, glucose response, insulin sensitivity, and indicators of metabolic stability.

The latent variables in the model are:

- Diet Variability (DV)
- Glucose Fluctuation (GF)
- Insulin Response (IR)
- Metabolic Homeostasis (MH)

The measurement of each construct is based on a series of observed indicators based on outputs of the stochastic differential equation model in terms of time series.

3.4.2 Measurement Model (Outer Model) Assessment

3.4.2.1 Reliability and Validity Results

Table 1: Reliability and Validity

Construct	Cronbach's Alpha	Composite Reliability	AVE
Diet Variability	0.86	0.90	0.65
Glucose Fluctuation	0.88	0.92	0.70
Insulin Response	0.84	0.89	0.63
Metabolic Homeostasis	0.90	0.93	0.72

Interpretation: The findings confirm high internal consistency of all constructs. The values of all Cronbach Alphas are over 0.7 which signify reliable measurements scales. A strong construct reliability is guaranteed when the composite reliability values are more than 0.8. AVE values greater than 0.5 indicate good convergent validity, i.e., the indicators are sufficient

to describe their latent constructs (Table 1).

3.4.3 Discriminant Validity (HTMT Criterion)

Table 2: HTMT Ratios

Constructs	DV	GF	IR	MH
DV	—	0.62	0.58	0.55
GF	0.62	—	0.66	0.60
IR	0.58	0.66	—	0.63
MH	0.55	0.60	0.63	—

Interpretation: All the HTMT values are less than the threshold of 0.85, which indicates that there is strong discriminant validity. This suggests that all constructs of the model are statistically different and reflect different facets of the regulation of metabolic processes (Table 2).

3.4.4 Structural Model (Inner Model) Results

Table 3: Path Coefficients

Relationship	Path Coefficient (β)	t-Value	p-Value
DV $\rightarrow$ GF	0.71	12.45	0.000
DV $\rightarrow$ IR	0.63	10.32	0.000
GF $\rightarrow$ MH	-0.68	11.87	0.000
IR $\rightarrow$ MH	0.59	9.76	0.000
DV $\rightarrow$ MH	-0.52	8.91	0.000

Interpretation: The findings indicate that diet variability has a significant positive impact on glucose variation ($= 0.71$). The greater increase in glucose variations greatly decreases metabolic homeostasis ($= -0.68$), which proves unsteadiness in the face of dietary chance. Insulin response is a partial stabilizing effect ($= 0.59$), which enhances metabolic homeostasis. However, variability of diet directly affects the metabolic stability ($= -0.52$), which confirms the stochastic model hypothesis that randomness disrupts physiological balance. The relationships are all statistically significant ($p < 0.001$), which confirms the strength of the model (Table 3).

3.4.5 Model Fit and Predictive Power

Table 4: Model Fit Indicators

Metric	Value
SRMR	0.045
R ² (Glucose Fluctuation)	0.50
R ² (Insulin Response)	0.40
R ² (Metabolic Homeostasis)	0.68
Q ² (Predictive Relevance)	0.55

Interpretation: A good model fit is evident in the Standardized Root Mean Square Residual (SRMR = 0.045). The R² values indicate that diet variability accounts for half the glucose variability and 68 percent of the metabolic homeostasis variability, which is strong explanatory power. Q² values are greater than zero, and they indicate strong predictive relevance, which demonstrates that the model possesses a trusted ability to predict with certainty the metabolic behavior (Table 4).

4. Applications

The diet variability stochastic differential equation (SDE) model and its impact on metabolic homeostasis have broad applications in biomedical engineering, clinical nutrition, population health, personalized medicine, and computational biology. The model also includes randomness in metabolic controls, which makes it a more realistic model of human physiology, in which dietary ration and biological reactions are uncertain. Its key areas of application are outlined below.

4.1 Diabetes Prediction and Management

Diabetes research and management are among the major uses of this model. Irregular diets and dysregulation of glucose and insulin highly contribute to diabetes mellitus, especially type 2 diabetes. The SDE model enables the researcher to model the consequences of random changes in carbohydrate consumption on time-varying glucose variability. This model can be applied by clinicians to forecast hyperglycemic or hypoglycemic events in various dietary circumstances. As an illustration, with the help of stochastic intensity parameters, it is possible to model the effect of the so-called high-risk

dietary behavior and the effect it has on glucose stability over time. It assists in the development of better insulin dosing systems and diets to use with diabetics. Furthermore, the model facilitates the integration of continuous glucose monitoring (CGM) data, which allows customization for patients. This results in individualized diabetes management approaches that consider physiological variability together with unpredictable behavior.

4.2 Personalized Nutrition Systems

The model can be used in the new sphere of personalized nutrition. The classical dietary principles presuppose similar metabolic reactions, which is not realistic as people are different. The stochastic model enables modeling of individualized metabolic responses given uncertain conditions of dietary intake. The model can be used to determine the optimal ranges of intake of nutrients by using patient-specific parameters, including metabolic rate, insulin sensitivity, and dietary habits. This information can be utilized by nutritionists to develop adaptive diet plans, which reduce the glucose fluctuation at the same time without compromising energy balance. This model may be incorporated in digital health systems through mobile apps that provide real-time dietary changes according to the estimated metabolic reactions. It is particularly beneficial to people with metabolic syndrome, obesity, or prediabetes.

4.3 Obesity and Metabolic Syndrome Study

Irregular eating habits and metabolic disturbance are closely related to obesity and metabolic syndrome. The SDE model presents a model to examine the role of irregular caloric consumption in causing long-term metabolic instability. Researchers may examine the effects of these dietary patterns on the buildup of fats and insulin resistance by simulating stochastic dietary patterns like binge eating or variation in meal timing. The model assists in determining the limits beyond which metabolic control becomes precarious, resulting in chronic illnesses. These findings can help public health researchers to develop behavioral interventions that focus on dietary consistency and not on caloric reduction alone.

4.4 Optimization of drug and Insulin Therapy

The model can also be applied in optimizing the insulin therapy of patients with diabetes in the pharmacological field. The fact that the insulin demand depends on the food intake implies that the stochastic modeling will be able to simulate real-life scenarios in which food consumption is stochastic. Clinicians can subject various insulin dosing plans to simulated noise conditions of dietary noise to assess soundness and security. This will lower the chances of negative experiences like hypoglycemia due to excessive insulin requirements. Also, the model may be expanded to cover oral hypoglycemic drugs and their interaction with dietary variability, enhancing comprehensive treatment planning.

4.5 Clinical Decision Support Systems

SDE-based framework may be incorporated into clinical decision support systems (CDSS) so that it may help healthcare professionals to make decisions in real-time. The system is capable of simulating future metabolic conditions through the entry of patient data in different dietary conditions. As an example, in case a patient has unusual eating habits, the system can approximate the likelihood of glucose spikes and recommend preventive actions. This improves preventive care and reduces dependency on responsive care. This model can be used in hospitals and clinics to observe high-risk patients and offer early interventions due to the forecasted metabolic instability.

4.6 Digital Twin Development in Healthcare

A highly advanced application of this model is in the development of metabolic digital twins. A digital twin is a computer simulation of the metabolism of a patient, which advances in the real world. The SDE model can be used to simulate the response of the metabolism of a patient to real-life variability by including stochastic dietary inputs. It facilitates constant observation and forecasting of metabolic results without the need to be invasive. Research trials, drug testing, and optimization of individualized treatments can be implemented using digital twins founded on this model.

4.7 Policy on Public Health and Nutritional Guidelines

The model can be used at a population level to help formulate evidence-based nutritional policies. The study of the effect of variability in diet among various demographics can be simulated by the health authorities. To provide an example, the model can evaluate the role of inconsistent meal habits in urban residents in increasing the rates of diabetes. It is also able to test the efficacy of interventions like a meal timing policy or a sugar cut-down policy. This will assist the policy makers in formulating focused measures to decrease the burden of metabolic diseases in society.

4.8 Sports and Performance Nutrition

Athletes need accurate metabolic regulation to enhance performance. However, the practical dietary consumption

cannot always be regular because of traveling, training, and competition stress. A stochastic model is capable of simulating the influences of variability of nutrient intake on energy availability and recovery. This can be utilized by sports scientists to develop adaptive nutrition plans that ensure that metabolism is stable in changing conditions. It is also useful in avoiding performance declines due to sudden metabolic imbalances.

4.9 Integration of AI and Predictive Analytics

The model is compatible with machine learning algorithms for predictive health analytics. Stochastic metabolic simulations can be used to train AI systems to enhance the predictive accuracy of glucose levels and dietary outcomes. With the integration of SDE models and neural networks or reinforcement learning, intelligent systems may emerge, which constantly update dietary advice in response to real-time information. This mixed method is especially useful in wearable health technology and smart health care systems.

4.10 Systems biology research

The model is a strong tool in the study of the interaction of the metabolic networks and environmental variability in systems biology. It enables scientists to comprehend the spread of stochastic dietary inputs via biochemical pathways. This adds to the understanding of homeostasis, resilience, and mechanisms of failure in biological systems. It also facilitates multi-scale modeling between cellular metabolism and whole-body energy regulation. All in all, there are many interdisciplinary applications of the stochastic differential equation model of diet variability. It connects mathematical modeling and real-world biomedical problems, providing predictive, diagnostic, and therapeutic information. The model improves the knowledge of metabolic homeostasis by elucidating the concept of uncertainty in dietary behavior, which is a solid basis for future innovations in the field of personalized healthcare, digital medicine, and optimization of population health.

5. Discussion

The stochastic differential equation (SDE) system of diet variability gives a realistic account of the metabolic regulation since it addresses the randomness of human dietary intake and physiological processes. In contrast to deterministic models, where dietary patterns are assumed, and the result is predictable, the SDE approach adds the noise terms that capture the day-by-day changes in the food intake, hormonal reactions, and energy consumption. This renders the model more biologically plausible, particularly in long-term metabolic behaviour. One of the most important lessons of this model is that the metabolic homeostasis, especially the glucose-insulin homeostasis, is not a fixed point but a process that is constantly perturbed. The variability of dietary intake is a stochastic forcing function that may stabilize or destabilize metabolic regulation with regard to its magnitude and frequency. The little variation can be compensated for by the regulatory feedback mechanisms, including insulin release and the uptake of glucose, and homeostasis is preserved. However, dietary variability, especially high glycemic load variability, when large or persistent, can cause the system to become unstable, which can lead to dysregulation like insulin resistance. Physiologically, the stochastic term of the SDE is the real-world uncertainty, i.e., it differs in time with meal, inconsistent macronutrient content, stress-related eating, and variation in metabolism between individuals. The causes of these are random deviations of glucose expected trajectories. The model shows that average dietary intake may seem balanced, but variations in it can also play a crucial role in the metabolic results. This points to a significant weakness of conventional nutritional advice where emphasis is on an average intake rather than the difference between consumption patterns. Mathematically, the model demonstrates that metabolic homeostasis is maintained at a stability level determined by the balance between deterministic restoring forces (insulin-glucose feedback loops, etc.) and stochastic perturbations by diet variability. When the diffusion coefficient of the SDE is small, the system is highly constrained around the equilibrium point. But the wider the variability, the more the probability distribution of metabolic states is spread out, meaning that excursions into hyperglycemic or hypoglycemic states are more likely. This has a clinical implication that the dietary interventions should not be based on reducing calories or the composition of macronutrients only, but also on focusing on consistency in eating patterns. Timely eating and constant nutrient consumption can decrease random changes and enhance long-term metabolic balance. This view is also supported by the new evidence in the field of chrononutrition that highlights the significance of regularity and timing of food consumption as a determinant of metabolic health. On the whole, the stochastic differential equation model is a powerful analytical tool to understand the role of randomness in diet in causing metabolic variability. It fills the gap between the study of biology theory and practical aspects of dietary behavior, providing a more detailed picture of the metabolic homeostasis as a probabilistic and not deterministic mechanism.

6. Conclusion

The stochastic differential equation (SDE) of diet variability and its impact on metabolic homeostasis offers a whole

mathematical and biological tool to view the effect of non-regular dietary habits on physiological stability. Contrary to classical deterministic models, the model includes a random component in the dietary intake and metabolic response, and is a more realistic model of human metabolism in normal conditions. The stochastic terms used reflect the unpredictability of eating habits, nutrient absorption, hormonal changes, and energy use, which are all involved in metabolic variability. One of the main conclusions of this model is that metabolic homeostasis is not an absolute condition, but a dynamic balance that is constantly affected by external and internal perturbations. The regulatory processes of the body, especially the glucose-insulin feedback, can be stabilized with slight changes in diet. At a certain point, though, once variability exceeds some critical value, the regulatory capacity becomes overloaded, resulting in increased deviations of equilibrium and the risk of metabolic dysfunction. This emphasizes the significance of the quality and regularity of the diet in long-term health. It also shows that the average intake of nutrients is not enough to explain the metabolic outcomes. Two people who have a similar mean caloric intake or carbohydrate intake can undergo very different metabolic reactions based on the variability of their diets. The dietary intake is highly varied, which can further increase the spread of metabolic states, making one vulnerable to insulin resistance, impaired glucose tolerance, and metabolic syndrome. This observation underscores the clinical importance of regularity in the diet and nutritional composition. Mathematically, the SDE model demonstrates that the stability of the system is determined by the equilibrium between deterministic restoring forces and stochastic perturbations. There are built-in feedback systems in the glucose-insulin regulation, which stabilize the system, yet too much noise can take control of the system, driving the system to instability. This understanding will be useful in forecasting metabolic risk in various dietary situations and allow the formation of individualized nutrition plans. To sum up, the SDE model contributes greatly to the comprehension of diet-induced metabolic variability by incorporating randomness into the physiological modeling. It gives good reasons to believe that dietary variability is a decisive factor of metabolic health on its own, rather than average intake. These results are consistent with the need to shift the scope of nutritional science in more dynamic and personalized directions, where consistency in dietary behavior is seen as a key determinant of nutritional content. This model can be further improved by including multi-scale biological interactions, such as gut microbiota dynamics and hormonal regulation, in future research to further increase its predictive power and applicability to clinical use.

7. Recommendations

Following the stochastic differential equation (SDE) framework of diet variability and the effects on metabolic homeostasis, it is possible to make a number of convenient and research-focused recommendations to enhance the practical work and further scientific progress.

1. Emphasize dietary consistency in nutrition planning: Nutritional guidelines are not to be related to the average calorie consumption or the proportions of macronutrients, but should also aim at decreasing daily dietary fluctuations. Consistent timing of meals and stable nutrient content of food can be used to reduce stochastic changes in glucose and insulin dynamics, which are conducive to improved metabolic regulation.
2. Promote chrononutrition-based interventions: Structured eating schedules, in line with the circadian rhythm of the body, should be encouraged by healthcare practitioners. Regular meal timing helps to minimize metabolic noise and reinforces physiological feedback pathways that help to maintain glucose homeostasis.
3. Incorporate variability measures into dietary measurement: The common eating examinations are to be increased with the measurements of variability (e.g., difference in carbohydrate consumption, aberrancy in the timing of meals). This would enable the clinicians to more effectively detect people who are at risk of metabolic instability despite having normal intake in the average.
4. Work out individual nutrition plans with the help of stochastic modeling: Mathematical and computational models such as SDEs should be used in designing personalized nutrition plans in future dietary interventions. The effect of varying degrees of diet variability on metabolic stability in particular persons can be simulated by such models.

8. References

- Al Ali, H., Daneshkhah, A., Boutayeb, A., Merabet, N., & Mukandavire, Z. (2025). A Comprehensive Review of Mathematical and Data-Driven Models in Glucose Homeostasis and Diabetes Pathways. *Computational Mathematics and Modelling for Diabetes*, 133-165.
- Alessi, D. S., McCreery, C. V., & Zomorodi, A. R. (2025). In silico dietary interventions using whole-body metabolic models reveal sex-specific and differential dietary risk profiles for metabolic syndrome. *Frontiers in Physiology*, 16, 1586750.
- Alzahrani, S. M. (2025). Statistical Insights into Stochastic Glucose-Insulin Dynamics: Modeling Insulin Degradation with the Michaelis-Menten Function. *European Journal of Pure and Applied Mathematics*, 18(1), 5404-5404.

- Amatori, S., Radice, R., Podlogar, T., Gobbi, E., Belli, A., Loche, F., Bertuccioli, A., Sudano, M. M., Baldari, C., & Rocchi, M. B. (2026). Low-Versus High-Carbohydrate Isocaloric Diets on Continuous Glucose Monitoring Metrics in Healthy Trained Cyclists: A Randomized Crossover Trial. *European Journal of Sport Science*, 26(2), e70127.
- Aminian-Dehkordi, J., Dickson, A., Valiei, A., & Mofrad, M. R. (2025). Meta Biome: a multiscale model integrating agent-based and metabolic networks to reveal spatial regulation in gut mucosal microbial communities. *Msystems*, 10(5), e01652-01624.
- Barrera-Suarez, M. A., Zhao, C. Y., Karnatovskaia, L. V., & Sung, J. (2026). Precision nutrition through diet-gut microbiome interactions: Emerging insights driven by artificial intelligence, microbiome health metrics, and mechanistic modeling. *Gut Microbes Reports*, 3(1), 2650247.
- Benhacene, W., Cherrada, N., Zekhem, Z., Souttou, S., Rezik, K. N., Benabdesselam, R., Aziz, T., Shami, A., Alwethaynani, M. S., & Al-Joufi, F. A. (2026). Protective effects of methanolic broccoli (*Brassica oleracea* var. *italica*) extract on metabolic dysfunction and hypothalamo-hypophyseal axis oxidative stress in a rat model of diet-induced type 2 diabetes mellitus. *Open Chemistry*, 24(1), 20250222.
- Bradley, L., Duvall, E. S., Akinifesi, O. J., Novotná Jaroměřská, T., Junker, J. R., & Wong, E. (2025). Exploring the multi-scale ecological consequences of stoichiometric imbalance using an agent-based modeling approach. *Frontiers in Ecology and Evolution*, 13, 1505145.
- Brunasso, A., Lange, N. F., Poli, S., Schiavon, M., Herzig, D., Dalla Man, C., Kreis, R., & Bally, L. (2026). Novel deuterium metabolic imaging technique reveals distinct patterns of postprandial hepatic glucose homeostasis in individuals with type 1 diabetes and healthy control individuals: a case-control study. *Diabetologia*, 1-16.
- Castillo, A. F., Jacometo, C. B., Arroyo, J. I. N., Santiago, S. A. M., & Prado, P. M. (2026). Effect of dietary variation on indicators of metabolic energy, protein, and liver function in dairy cows during the transition period. *Revista Acadêmica Ciência Animal*, 24.
- Cifuentes, L., Anazco, D., O'Connor, T., Hurtado, M. D., Ghosn, W., Campos, A., Fansa, S., McRae, A., Madhusudhan, S., & Kolkin, E. (2025). Genetic and physiological insights into satiation variability predict responses to obesity treatment. *Cell metabolism*, 37(8), 1655-1666. e1655.
- Falkenhain, K., Cabeza De Baca, T., Stinson, E. J., Ravussin, E., Piaggi, P., Krakoff, J., & Redman, L. M. (2025). The effect of a leptin phenotype on weight change and energy expenditure responses to acute and prolonged energetic stressors. *American Journal of Physiology-Endocrinology and Metabolism*, 328(4), E579-E587.
- Fu, Y., Gou, W., Zhong, H., Tian, Y., Zhao, H., Liang, X., Shuai, M., Zhuo, L.-B., Jiang, Z., & Tang, J. (2025). Diet-gut microbiome interaction and its impact on host blood glucose homeostasis: a series of nutritional n-of-1 trials. *Ebiomedicine*, 111.
- Kaduk, K., Kaeber, M., Kühnel, A., Torrado, M. B., Grahlow, M., Derntl, B., & Kroemer, N. B. (2026). Glucose levels are associated with mood, but the association is mediated by ratings of metabolic state. *Ebiomedicine*, 124.
- Koliopoulou, I., DeRuiter, S. L., Altimiras, J., Larsson, J., Arenarez, J., Rosen, D., & Fahlman, A. (2025). Heat increment of feeding in the common bottlenose dolphin (*Tursiops truncatus*) contributes moderately to field metabolic rate estimates. *Journal of Experimental Biology*, 228(24), jeb251474.
- Liao, Y., Spadola, C., & Roy, S. (2026). Mathematical modeling of glucose regulation and sleep resting heart rate to predict risk of metabolic dysregulation in cancer survivors. *PloS one*, 21(1), e0339461.
- Luo, J., Wang, H., Moyer, D., Guo, Z., Robinson, J. L., Gustafsson, J., Anton, M., Chen, Y., Kerkhoven, E. J., & Nielsen, J. (2026). Reconstruction of human metabolic models with large language models. *Proceedings of the National Academy of Sciences*, 123(15), e2516511123.
- Mallick, U. K., Rahim, F., Sadi, S. S., & Kundu, P. (2025). Effects of stress, diet and physical exercise on type 1 diabetes mellitus with the use of exogenous insulin. *Biomath*, 14(2), 2509085-2509085.
- Matz-Soja, M., Körner, C., Ott, F., Fischer, J., Marbach-Breitrück, E., Bergmann, C., Hofmann, U., Shevchenko, A., Wallach, I., & Textoris-Taube, K. (2026). Modeling the dynamics of hepatic metabolism: the predominance of 12-hour rhythmicity in metabolic adaptation. *Cellular and Molecular Life Sciences*, 83(1), 55.
- Mekki, L. B., Elhassan, Y. H., Badi, R. M., Shazali, A. F., Mojaddidi, M. A., Ibrahim, S., Abdalla, H. A., Elawaf, Z., Mohammedali, A. A., & Saeed, A. (2025). Elevated oxytocin levels and their relationship to metabolic syndrome and

- obesity among young Sudanese adults: a cross-sectional analysis. *Cureus*, 17(3), e81451.
- Mukherjee, S. D., Batagello, C., Adler, A., Agudelo, J., Zampini, A., Suryavanshi, M., Nguyen, A., Orr, T., Dearing, D., & Monga, M. (2025). Complex system modeling reveals oxalate homeostasis is driven by diverse oxalate-degrading bacteria. *Elife*, 14, RP104121.
- Park, G., Johnson, K., Miller, K., Kadyan, S., Singar, S., Patoine, C., Hao, F., Lee, Y., Patterson, A. D., & Arjmandi, B. (2025). Almond snacking modulates gut microbiome and metabolome in association with improved cardiometabolic and inflammatory markers. *npj Science of Food*, 9(1), 35.
- Pham, K., Coutts, V. M., Hoffman, A. J., & Wada, H. (2025). Mimicking the night shift: Hypothalamic-pituitary-adrenal (HPA) axis regulation, energy homeostasis, and oxidative stress in a model diurnal songbird. *bioRxiv*, 2025.2006.2009.656008.
- Queathem, E. D., Stagg, D. B., Nelson, A. B., Chaves, A. B., Crown, S. B., Fulghum, K., d'Avignon, D. A., Ryder, J. R., Bolan, P. J., & Hayir, A. (2025). Ketogenesis mitigates metabolic dysfunction-associated steatotic liver disease through mechanisms that extend beyond fat oxidation. *The Journal of Clinical Investigation*, 135(12).
- Ray, A. (2026). The 24 Hours Serotonin–Melatonin Dynamics in the SCN–Pineal Circadian System—A Mathematical Model. *Compassionate AI*, 1(1), 6–8.
- Reda, G. K., Ndunguru, S. F., Csernus, B., Knop, R., Szabó, C., Czeglédi, L., & Lendvai, Á. Z. (2026). Unpredictability and adaptive responses to dietary variability in Japanese quail. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 381(1946).
- Riaz, C., Shafiq, M. R., Ahmad, M. T., Iqbal, M. T., Saeed, A., & Iqbal, S. (2025). Assessment of Genetic Variability and Trait Associations Among Yield Components in Tomato (*Solanum lycopersicum* L.). *Letters in Biomathematics*, 12(1), 31–39.
- Waezizadeh, T., & Izadi, M. (2026). Stochastic dynamic of intravenous glucose-insulin model. *Journal of Inequalities and Applications*.
- Wang, Z., Li, Q., Shi, M., Leite, M. F., Chen, X., Kuramae, E. E., Cordovez, V., Cao, T., Zhu, C., & Zhou, L. (2026). Compartmentalized homeostasis drives high bamboo forest productivity under nutrient imbalance. *Advanced Science*, 13(11), e17442.
- Weilandt, D. R., Lee, W. D., MacArthur, M. R., Liang, L., Holman, C., Chu, Q., Cowan, A. J., Baur, J. A., Wingreen, N. S., & Rabinowitz, J. D. (2025). Competitive catabolism in systemic metabolic homeostasis. *bioRxiv*, 2025.2010.2029.684184.
- Xie, W., Peng, C., Chen, J., Ni, Y., Yang, Y., Sui, Q., Shen, C., & Zhang, W. (2026). Dynamic fate of polystyrene microplastics in terrestrial snails through dietary exposure: accumulation, excretion, and molecular impacts. *Environment International*, 110214.