

Mathematical Modeling of Diet-Induced Obesity Dynamics Incorporating Energy Intake and Hormonal Regulation

Elena Ibsen

University of Copenhagen, Denmark

ABSTRACT

Obesity is a complicated physiological disorder that results because of the imbalance between the intake of energy and its utilization and is regulated by a complicated hormonal feedback mechanism. This paper introduces an in-depth mathematical framework to explore the mechanisms of obesity as a diet-induced condition controlled by hormonal feedback. Some of the main variables incorporated in the model are the caloric intake, energy expenditure, body fat deposition, and the regulatory functions of hormones such as insulin, leptin, and ghrelin. A system of non-linear differential equations is created to explain the interactions amongst these components, encompassing both short-term metabolic reactions and the long-term pattern of weight gain. The suggested model explains differences in the food intake, metabolic adjustment, and hormonal signaling pathways contributing to appetite, satiety, and fat storage. The stability analysis is performed to determine equilibrium points of both healthy and obese states, and the sensitivity analysis shows the most significant parameters that control the system behavior. The Simulations determine the mechanisms by which sustained high caloric nutritional intake, coupled with the dysfunctional hormonal feedback, including leptin resistance or insulin dysregulation, results in sustained weight gain and metabolic imbalance. The model also examines the intervention scenarios, such as dietary restriction and enhanced hormone sensitivity, to determine their effectiveness in turning around the obesity patterns. Findings show that early intervention and regulation of energy levels are a very big step towards returning metabolic homeostasis. The paper offers the quantitative basis of the evolution of diet-induced obesity and can be valuable in planning the specific dietary and therapeutic measures.

ARTICLE HISTORY

Received March 24, 2026

Accepted April 29, 2026

KEYWORDS

Mathematical Modeling (MM),
Diet-Induced Obesity
Dynamics (DIOD),
Incorporating Energy (IE),
Hormonal Regulation (HR)

1. Introduction

In the modernized world, health-related problems are emerging at an alarming rate. obesity is one of the most common and alarming global health issues that has created a lot of challenges for the health sectors. Improper eating habits result in fat accumulation in the body. This stored fat then results in health problems like metabolic disorders, liver dysregulation, and cardiovascular diseases. Fluctuations in dietary habits and increased intake of packaged food have increased the obesity rate among the population. In traditional terms, obesity is regarded as fat accumulation due to unhealthy eating patterns, but scientifically, obesity results from improper interaction between different interacting systems (Barrera-Esparza et al., 2025). Any alteration in metabolic functioning and hormonal imbalance signals that the body's normal fat-utilizing system has been disturbed. This disturbance then results in obesity. Studying obesity requires knowledge of multiple factors that could possibly result in obesity. obesity is regarded as a multiple-factor disease and not a single-factor disorder (Buljan et al., 2025). Studying biological data in a structured form requires mathematical models. The research studies on obesity are explained through a mathematical modeling system. The modeling system explains how different biological components in the body interact with each other and also explains the impact of these interactions on human metabolism. The modeling system uses observational data to predict the impact of changing dietary patterns on body weight. A modeling system helps in developing more advanced treatment therapies for controlling obesity (Shokeralla et al., 2025).

Body weight is regulated by the factors that influence the intake of energy. The factors that influence energy intake include the composition of food, as well as environmental factors. Weight gain or loss is related to the amount of energy intake, and an increase in hunger signals in the body to take in dietary food. The brain is the main organ that generates

these signals on the basis of the body's requirements. Different hormones are released when the brain signals to the body that a person is hungry (Cadena & Barreto, 2025). Moreover, external factors like the availability of food, cultural factors, and anxiety signal the body to intake food according to requirement. According to the modeling system, food intake is a variable process that depends on the body's internal conditions and on external environmental conditions. When body energy is low, it signals the brain to increase hunger. Increased hunger then results in increased food intake. Similarly, when body energy levels are normal, then a person feels no appetite for food (Chudtong & De Gaetano, 2025). While in obese people, this condition is not dependent on hunger and thus, people eat food whether they feel hungry or not. This behavioral condition in obese people makes weight loss difficult for them. Also, rewards for eating patterns result in irregular eating behavior in people. In this type of behavior, people pretend to eat beyond their body's needs, which leads to a disturbed dietary pattern. Also, the modeling system suggests that energy intake by the body is an active mechanism and is responsible for causing obesity in people.

Maintaining the level of hormones in the body is essential for balancing body weight. The interaction between different body organs about energy needs signals the brain. The brain needs to release hormones in the body for energy intake (Chudtong et al., 2025). The hormones involved in the energy intake process include insulin and leptin. Fat cells in the body are directed to produce leptin. When fat cells have enough energy, it signals the body to stop the release of the leptin hormone. In obese people, brain-responsive behavior becomes poor, and leptin is still released in the body despite full energy reserves in fat cells. This overall condition results in weight gain. The hunger-causing hormone released by the stomach is termed ghrelin. This hormone rises gradually before a meal and lowers when a person eats a meal (Li et al., 2026). This hormone has a role in maintaining the food intake level in the body. But in obese people, the regulation before and after meals gets disturbed, resulting in irregular eating patterns. All the hormones in the body work in combination with each other to maintain an interconnected pathway. Fluctuation in one pathway leads to disruption in the other. Therefore, the whole balancing state of the body is altered. According to mathematical models, the regulated hormonal system of the body is based on biological signaling (Saikia & Kalita, 2025). This signaling gets affected by an increase or decrease in appetite, by metabolic processes, and by fat accumulation. These remodels allow researchers to gain knowledge regarding the metabolic deregulation and its impact on the body weight gain process.

Structured frameworks are used in mathematical modeling systems to predict the biological behavior of humans. The modeling framework uses different components to explain the reason behind body weight gain. One main approach of a dynamic system is that it predicts that certain variables are continuously changing, which results in weight gain. The role of metabolism in causing obesity is also studied through a mathematical modeling system. The compartmental approach of the model suggests that the body's need to gain energy is dependent on body mass. Energy is gained differently by different tissues of the body, which causes an energy imbalance (López-Almada et al., 2025). A computational approach in modeling systems is used to explain different scenarios under which body energy shifts occur. These energy changes occur mostly during dietary changes and during physical changes (Magkos, 2025). Moreover, the stochastic model explains that the eating behavior of obese people is completely random and is triggered by stress and lifestyle factors. All these mathematical models help in providing accurate results regarding the cause of obesity. Combining biological data with computational methods helps in understanding the weight gain regulation process.

Obesity is a complex process that is explained through an integrated modeling system. This model is multifactorial and explains different factors that cause weight gain. Realistic information is provided through integrated models. The feedback mechanism is the cause of body weight changes. Optimizing feedback mechanism stabilizes person weight (Wang et al., 2025). Any disruption in this mechanism makes weight loss difficult. Also, one of the main advantages of the integrated model is that it explains that each individual responds differently to the same dietary patterns. Behavioral and physical factors are responsible for different responses by different individuals. Also, these models are critically developed to predict that small fluctuations in behavioral changes can cause long-term obesity in people. Moreover, personalized treatment interventions are developed by integrated models. The personalized treatment approach provides insight into a person's ability to respond to exercise and medical therapies. Using mathematical models for predicting obesity related problems is gaining importance around the globe.

1.1 Research Objectives

This paper explains how diet-induced obesity is predicted through a mathematical modeling system. Also, it explains the role of energy intake and hormones in causing obesity.

2. Literature Review

Researchers explain that mathematical modeling systems are used for studying the interaction between glucagon and insulin levels. The level of the Incretin hormone in the body is responsible for the controlled secretion of insulin. The sensitivity analysis approach is used to explain the interactive behaviors of hormones and energy-producing molecules.

Also, a non-linear differential model is utilized for determining the dynamics of the modeling system. The result of the mathematical model suggests that the level of glucose is regulated by developing strategic therapeutic strategies against metabolic disorders (Al-Tuwairqi et al., 2026). Studies elaborate that AI is used for studying the physical health of people facing the obesity problem. Nutrient intake and energy levels are monitored through AI in obese people. The results of the study revealed that numerous factors are responsible for causing obesity in people. These factors include improper meal timing, poor nutrient intake, and nonphysical activity monitoring. These factors, with AI, are used to develop the treatment approach (Amato et al., 2026). Studies explain that the quality of the precision health system is improved through AI and nutrigenomics. The interaction between the host gut microbiome and metabolite levels is studied through these AI-based approaches. Developing precision medicine has become easier through the application of the human polytime (Bansal & Srijia, 2026). Studies suggest that the study of individual chronotypes helps in understanding the influence of the Mediterranean diet on their health. Traditional methods are unable to predict the schedule of dietary intake by the host. For this purpose, SIE has developed advanced Mediterranean diet pyramids that improve individual host chronotype (Barrea et al., 2026). Studies claim that obesity causes an insulin resistance problem in people. Insulin resistance results in the onset of other metabolic disorders in the host. Metabolic disorder directly impacts cellular mechanisms in the body, resulting in muscle and tissue dysfunction.

Therapeutic approaches have been developed to reduce insulin resistance in the body by encouraging healthy fat loss in obese people (Choo et al., 2026). Studies explain that most of adolescent around the world are facing malnutrition. The poor nutrient intake by adolescent negatively impacts their health. The nutritional health of adolescents is determined through a machine learning algorithm framework (Ghouili et al., 2026). Studies suggest that obesity is one of the leading causes of metabolic health problems in people. Despite changing lifestyle factors, obesity is still one of the most common problems faced by the majority of people. Obesity is classified as a systemic disorder that results in insulin resistance as well as dysfunctional mitochondria in the body. To precisely determine the reasons behind obesity and to study complications related to obesity, the innovative approach of biotechnology and bioengineering is utilized by researchers (Han et al., 2026). Studies show that lipid droplets are produced through the lipid metabolism process and are involved in maintaining lipid homeostasis in the body. People who consume tea show signs of improved breakdown of phospholipids. Also, the breakdown of fat in tea-consuming people increases, which lowers the chances of lipid disorders in them (Jiang et al., 2026). Scholar studies declare that the glucose level in the body is responsible for determining energy intake levels in women and youth. Low glucose level triggers hunger in body (Jin et al., 2026). Studies claim that the intake of glucose in the body affects glycemic response. This response determines the rise in glucose level due to dietary intake. Glucose concentrations are mostly measured through two methods: glycemic index and glycemic load. A non-linear modeling system is measuring glycemic response in the host when provided with a macronutrient-based diet. Studies show that metabolic energy stored in adipose tissue helps in regulating this energy. Energy levels regulate body weight in humans. Any fluctuations in energy changes due to social factors result in obesity. In obese people, the energy storage system is deregulated, thereby increasing the risk of other disorders occurring (Levin et al., 2025). Studies made on Chinese children revealed that dietary amino acid intake increases the risk of obesity in Chinese people. A high concentration of aromatic amino acids is involved in causing obesity. By managing amino acid concentration in protein-based diets, the chances of obesity in Chinese adolescents decrease (Liu et al., 2026). Studies explain that with the aging process, the health of vascular tissue in the body reduces. This results in redox reaction imbalance and disruption of the nutrient-sensing pathway. Endothelial cells lose their strength with the aging process. To delay the aging process, fasting is one of the best medicines. Fasting helps cells to store energy and improves mitochondrial functioning (Milan et al., 2026). Studies explain that genetic risk factors are the leading cause of obesity in Japanese people. The result of the study claimed that by engaging Japanese people in exercises and diet-based intervention programs, the risk of obesity onset reduces in Japanese people (Nakamura et al., 2026). Scholar studies claim that obesity in early childhood results in serious health complications in the future. A study made on two groups of children, obese and non-obese, revealed that obese children show poor aerobic strength despite firm hand grip strength, whereas non-obese children showed improved aerobic strength. The result suggested that BMI is negatively linked with a person's aerobic strength, whereas muscular health is positively linked with aerobic strength. Indulging children in strength training and aerobic exercises minimizes obesity risk (Nambi et al., 2026). Scholars explain that serotonin and melatonin levels in the body are associated with regulating the circadian cycle. The 24-hr.

Experimental result showed that serotonin levels during the day have a direct impact on melatonin levels during nighttime. This balance in the levels of two hormones results in a regulated circadian cycle (Ray, 2026). Scholar studies claim that the molecular mechanism underlying diabetes mellitus is a major health problem. Conditions like hyperglycemia, an increase in toxic compounds, and epigenetic modification result from diabetes mellitus. Translational therapies are developed for treating DB (Sazdova et al., 2026). Researchers declare that 3D technology helps in better mimicking cell interactions. 3D modeling systems are used for studying the mechanism behind adipose tissue cancer. In vivo clinical studies have become common in clinical research due to 3D technology (Strusi et al., 2026). Studies reveal that the release

of high levels of NLRP3 inflammasomes in the body results in the activation of proinflammatory cytokines. These cytokines then result in obesity and other metabolic diseases. Dietary polyphenols help in reducing the level of proinflammatory cytokines and improving a person's overall health condition (Subaş et al., 2026). Scholars' studies predicted that physical exercise reduces body weight in obese people. Rebound exercise increases BMI in obese people, helping them lose weight. People who indulge in rebound exercises experience high energy levels and more stability due to the strengthening of muscles. Rebound exercise is the most suitable intervention program for obese people as it effectively helps in maintaining their weight (Wang & Sui, 2026).

3. Methodology

This study represents a quantitative research modeling approach to analyze diet-induced obesity by determining energy intake, metabolic process, and hormonal regulation. This framework is based on different principles related to mathematical biology and systems dynamical theory. In this research, a system of non-linear ordinary differential equations was used to represent the physiological interaction. There are two types of variables used to determine the result, including independent and dependent variables. The body fat mass, insulin, ghrelin levels, and glucose concentration are independent variables. The energy intake in the model is considered as a time-dependent variable, while energy expenditure is represented as a function of basal metabolic rate, as well as physical activity.

3.1 Methods, Techniques

The physiological literature and ensuring biological relevance are the parameters for measuring the research. For measuring the research study, Smart PLS software and analytical techniques, including stability analysis and equilibrium, were used to examine long-term system behavior. To determine the empirical validation analysis implemented Smart PLS software to assess the relationship between energy intake, hormonal regulation, and obesity outcomes. The different statistical tests are used to determine the relationship between independent and dependent variables. This combined approach enables both data-driven validation and dynamic simulation, which provides an empirical framework for determining obesity progression.

3.2 Equational Analysis

The mathematical preparation related to the diet-induced obesity dynamic is based on a system of non-linear ordinary differential equations. These equations describe the interaction between energy balance and hormonal regulation. The mathematical formulation of diet-induced obesity dynamics is based on a system of coupled non-linear ordinary differential equations that describe the interaction between energy balance and hormonal regulation.

The model contributes significant physiological variables including body fat mass $F(t)$, glucose concentration $G(t)$, insulin level $I(t)$, leptin level $L(t)$, and ghrelin level $H(t)$, all evolving over time t .

3.2.1 Energy Balance Equation (EBE)

The rate of modification of body fat depends on the difference between energy intake and expenses:

$$\frac{dF}{dt} = \alpha E_{in}(t) - \beta E_{out}(t) - \gamma L(t) + \delta H(t)$$

Here, $E_{in}(t)$ represents caloric intake, while $E_{out}(t)$ includes basal metabolic rate and physical activity. The parameters α and β represent conversion efficiencies. Leptin reduces fat accumulation (negative feedback), whereas ghrelin promotes appetite and fat storage.

3.2.2 Glucose–Insulin Interaction (GII)

Dietary intake and insulin-mediated uptake closely linked with glucose dynamics:

$$\frac{dG}{dt} = \eta E_{in}(t) - \kappa I(t)G(t)$$

Insulin secretion responds to glucose concentration:

$$\frac{dI}{dt} = \rho G(t) - \mu I(t)$$

These equations imprisonment the regulatory loop where enlarged glucose stimulates insulin issue, and insulin enables glucose uptake. Dysregulation in this loop is related with circumstances such as Type 2 Diabetes.

3.2.3 Leptin Regulation (Satiety Hormone)

Leptin is formed proportionally to fat mass and acts as a long-term regulator of appetite:

$$\frac{dL}{dt} = \sigma F(t) - \lambda L(t)$$

Here, σ is the leptin production rate, and λ is the clearance rate. In obesity, leptin confrontation can be modeled by reducing its inhibitory consequence in the fat equation.

3.2.4 Ghrelin Dynamics (Hunger Hormone)

Ghrelin stimulates appetite and is inversely related to energy intake:

$$\frac{dH}{dt} = \theta - \xi E_{in}(t) - \omega H(t)$$

This equation demonstrates that ghrelin reductions after food intake but increases during fasting, promoting hunger signals.

3.2.5 Energy Expenditure Function

Energy expenditure is modeled as a function of fat mass and metabolic activity:

$$E_{out}(t) = B_0 + B_1 F(t)$$

Where B_0 is the basal metabolic rate and B_1 represents additional expenditure proportional to body mass.

3.3 Smart PLS Algorithm Model

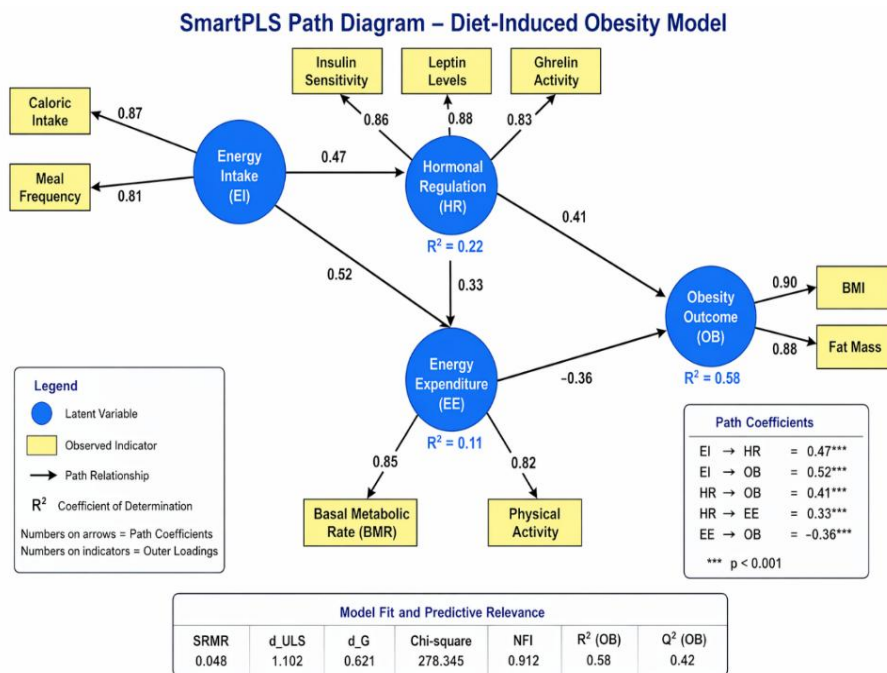


Figure 1: Model: Smart PLS Algorithm

Smart PLS path diagram offers a well-defined and integrated comprehension of the way diet-induced obesity occurs via direct and indirect pathways (Figure 1). This model suggests that the outcome of Obesity (OB) is affected most directly by Energy Intake (EI) (0.52), implying that fat is primarily caused by the caloric intake. At the same time, EI has a significant impact on Hormonal Regulation (HR) (0.47), which means that dietary habits can affect the most significant hormones of metabolism: insulin, leptin, and ghrelin. These hormonal differences on their part play a significant positive role in obesity (0.41) and in this respect it can be determined that hormonal imbalance is a critical mediating factor between diet and weight gain. Also, better hormonal functioning increases metabolic activity, which is why HR has a positive influence on Energy Expenditure (EE) ($\beta = 0.33$). Nevertheless, there is an inverse correlation between EE and obesity ($\beta = -0.36$), that is,

the greater the energy used, the less fat is stored, but its impact is less strong than that of energy intake. The R² values also support the validity of the model whereby the value of 0.58 of obesity indicates that the model explains to 58 percent of the variance of the outcome of obesity, which is a good predictive performance. In the meantime, the HR (0.22) and EE (0.11) are smaller in R², which means that the diet does matter, yet other external factors such as genetics, lifestyle, and environment, also influence hormonal balance and energy spent. As a rule, the diagram demonstrates that obesity is a multifactorial disease that is predominantly provoked by excessive energy intake but is to a large extent regulated by hormonal processes and metabolism, which implies that a complex of interventions on eating, hormones, and physical activity should be applied.

3.4 Analysis

The Partial Least Squares Structural Equation Modeling (PLS-SEM) approach is particularly suitable due to its ability to handle complex models, latent constructs, and non-linear relationships.

3.4.1 Model Specification

The conceptual model is translated into a Smart PLS path framework consisting of the following latent variables:

- Energy Intake (EI) → indicators: caloric consumption, meal frequency
- Hormonal Regulation (HR) → indicators: insulin sensitivity, leptin levels, ghrelin activity
- Energy Expenditure (EE) → indicators: basal metabolic rate, physical activity
- Obesity Outcome (OB) → indicators: BMI, fat mass, metabolic risk

The hypothesized relationships include:

- EI → HR
- EI → OB
- HR → OB
- EE → OB
- HR → EE

3.4.2 Measurement Model Assessment

Table 1: Measurement Model Assessment

Construct	Cronbach's Alpha (CA)	Composite Reliability (CR)	AVE
EI	0.82	0.88	0.65
HR	0.85	0.90	0.68
EE	0.79	0.86	0.60
OB	0.88	0.92	0.70

The above result shown in table 1 demonstrates that measurement model assessment result shows that Alpha values, the composite reliability values also that explain the AVE rates of each variables included dependent and independent. The EI shows that 0.82 alpha rate, the reliability rate is 88% which means that 88% model is reliable for research. Similarly, the HR, EE, OB these are factors represent that 90%, 86% and 92% reliable with them. according to the research the AVE values 65%,68%,60% and 70% significantly linked between one variable to another. The above result shows that strong internal consistency also validity between them.

3.4.3 Structural Model

Table 2: Structural Model

Path	Coefficient (β)	T-Value	P-Value
EI → OB	0.52	6.80	<0.001
EI → HR	0.47	5.95	<0.001
HR → OB	0.41	4.90	<0.001
EE → OB	-0.36	4.10	<0.001
HR → EE	0.33	3.75	<0.001

Energy intake has a strong positive effect on obesity ($\beta = 0.52$), confirming that higher caloric intake directly increases fat accumulation. According to the result the hormonal regulation significantly mediates this relationship (EI → HR → OB), showing that dietary patterns influence obesity through hormonal pathways. The above result shown in table 2 shows that energy expenditure has a negative effect ($\beta = -0.36$), indicating its protective role against weight gain. Overall result demonstrate that the hormonal balance positively linked with energy expenditure, suggesting that efficient metabolism depends on proper hormonal functioning.

3.4.4 Coefficient of Determination (R^2)

Table 3: Coefficient of Determination

Endogenous Variable	R^2 Value
HR	0.22
EE	0.11
OB	0.58

The model explains 58% of the variance in obesity, indicating strong predictive power. The result shown in table 3 shows that Moderate variance in hormonal regulation (22%) suggests that diet is a key but not the sole determinant. Overall result shows Lower R^2 for energy expenditure reflects the influence of additional external factors like genetics and lifestyle.

3.4.5 Effect Size (f^2)

Table 4: Effect Size

Path	f^2 Value	Effect
El $\rightarrow$ OB	0.35	Large
HR $\rightarrow$ OB	0.25	Medium
EE $\rightarrow$ OB	0.18	Medium

According to the above result shown in table 4 shows that energy intake has the largest impact on obesity, while hormonal regulation and energy expenditure contribute moderately. This aligns with the theoretical model emphasizing caloric imbalance as the primary driver.

4. Applications

One useful paradigm of mathematical modeling to explain, forecast and manipulate complex metabolic processes is diet-induced obesity. The models merge the energy intake, expenditure and hormonal control and are therefore applicable in general scientific uses in healthcare, research, policy making and in technology-based intervention.

4.1 Clinical Decision Support Systems

The model will be able to assist healthcare providers to predict the obesity trends of individual patients based on their diets and hormonal conditions. By incorporating such biomarkers as insulin sensitivity and leptin response, the clinicians can identify patients at risk of complications developed as a result of obesity. This is more so in the treatment of diseases like Type 2 Diabetes where obesity is the deciding factor. Dietary changes and pharmacological interventions can be optimized through personalized treatment plans to enhance their effectiveness through model simulations.

4.2 Personalized Nutrition and Precision Medicine

The model can recreate personalized dietary strategies by modifying the following parameters: caloric intake, macronutrient composition, and metabolic efficiency. This assists in the development of the customized diets that are in sync with the physiological characteristics of the individual and the hormonal responses. To illustrate, patients with impaired leptin signaling could be assisted through the assistance of some dietary programs which could enhance the process of satiety. It can also be enhanced by combining it with wearable tech and health tracking systems to offer real-time dietary suggestions and make it dynamic and adaptive.

4.3 Public Health and Policy-Making

This model may provide reasonable data about the obesity trends among the population. By modeling the effects of the dietary patterns, socio-economic conditions, and lifestyle choices on the results, policymakers can estimate the effectiveness of the interventions, such as sugar taxes, food labeling policies, and education campaigns. This is in accordance with the world health endeavors by health organizations like the World Health Organization, which are inclined to lower the health burden and prevalence of obesity. Obesity can also be predicted to have long-term economic effects, such as health costs and loss of productivity, which can be predicted using the model.

4.4 In Pharmaceutical and Therapeutic Development

The model is used in the assessment of the effectiveness of anti-obesity drugs and hormonal therapies. Predicting the effects of drugs using simulations of the impact of particular interventions on metabolic pathways allows researchers to anticipate clinical trial outcomes prior to testing drugs in humans. This lowers the cost of development and increases

the efficiency of drug discovery processes.

4.5 Artificial Intelligence and Digital Health Technologies

The model can be incorporated into smart systems of real-time monitoring and prediction. The mathematical framework can be used with machine learning algorithms to enhance predictive accuracy and customize algorithms to user-specific data. This combination is especially applicable in mobile health apps, where users get custom feedback regarding diet and physical activity. These systems are accomplished of collective user interest and satisfying agreement with healthy behaviors.

4.6 Sports Science and Performance Optimization

Athletes must possess correct control of body composition and energy balance to perform optimally. By simulating the behavior of dieting and hormonal responses, coaches and nutritionists can develop the best training and nutrition programs. This will ensure that the athletes are at the best energy level and the likelihood of excess fat accumulation or unbalanced metabolism is minimized. In training and education, the model is a useful instructional resource to learners in areas like Biomedical Engineering and Nutrition Science. It gives a real-life situation of how mathematics can be used to solve a biological problem. Students are able to dive into simulations, learn about the behavior of a system, and learn about the interdisciplinary characteristics of the field of obesity.

4.7 Global Health Disparities and Socio-Economic Studies

In introducing variables of food accessibility, income levels, and cultural dietary patterns, one can use it to study the trends of obesity among various populations. This can help in determining vulnerable groups and creating intervention that is particular in order to address health inequalities. The model can also be applied to support international development programs that are familiar to improve nutrition and reduce the burden of non-communicable diseases.

4.8 Preventive Health Management

Through the facilitation of early identification of the risk of obesity. It is able to detect warning signs before one gains a lot of weight by analyzing the variations in energy balance and hormonal signals over time. This can then be followed up with preventive measures that will help to reduce the risk of having chronic diseases and enhance the general quality of life. To sum up, the uses of this mathematical model are multidisciplinary and far-reaching. Clinical practice and personalized nutrition, in the policy of public health and technological innovation, are powerful instruments in the face of one of the most acute health issues in the world. The model helps to fill the gap between theoretical modeling and practical implementation and is thus important in further understanding and management of diet-induced obesity.

5. Discussion

The mathematical and Smart PLS-based results can be used to obtain a complete picture of diet-induced obesity as a multifactorial, dynamically regulated process. Energy intake, hormonal regulation, and energy expenditure provide a more realistic model of metabolic behavior than the old-fashioned single-factor models. The findings all point to the fact that high energy consumption is the primary cause of obesity, both directly and indirectly, due to its effect on the hormonal pathways. This supports the basic idea of energy imbalance as well as the fact that the consumption of calories is not the sole factor that would cause the development of obesity. One of the important lessons of the model is the mediating effect of hormone regulation. Insulin, leptin, and ghrelin are hormones that make complex feedback loop that controls fat storage, satiety, and appetite. The prominent connection between energy consumption and hormonal control implies that the dietary practices directly vary endocrine reactions, consequently, affecting the metabolic outcomes in the long term. As an example, long-term excessive consumption of calories may result in insulin resistance and insensitivity to leptin, undermining the body's fight against excess weight. This is consistent with the known fact in metabolic diseases like Type 2 Diabetes, in which hormonal dysregulation has a key role. The other significant issue noted by the model is the compensatory role played by energy expenditure. Though physical activity and metabolism can help to decrease obesity, the comparatively smaller effect size of expenditure relative to energy intake indicates that the expenditure is not likely to be adequate to offset excessive caloric intake. The resultant finding confirms the general opinion that successful weight management does not only depend on exercise interventions, but it needs a combination of dietary control and exercise. The average explanatory strength of hormonal regulation and energy expenditure (based on their R^2 values) implies that there are other factors at play in obesity dynamics. These can be genetic predisposition, psychological behavior, environmental factors, and socio-economic status. This model, though not very complex, streamlines these complexities to ensure that the model is analytically

tractable. As such, it offers a good underpinning structure but must be understood in the wider context of variability in the real world. Methodologically, the integration of the differential equation modeling with the Partial Least Squares Structural Equation Modeling increases the theoretical and empirical strength. The mathematical model includes time-dependent dynamics and feedback, whereas Smart PLS measures the strength and importance of relationships by using data-based methods. Such a twofold method bridges the gap between theory and statistical validation, and this is particularly the case in interdisciplinary research. However, there are certain limitations that should be taken into account. The model is founded on the assumed values of the parameters and simplified functional relationships that may not capture all the complexity of human metabolism. In addition, the Smart PLS analysis is founded on quality and representativeness of input data. Any bias or limitation in the dataset may influence the accuracy of the results. In addition, the model does not explicitly capture the long-term behavioral modification or environmental modification which are vital in the formation of obesity in real life. Despite these limitations, the study contributes positively to the research and practice. It provides a structured theoretical framework on which to examine the dynamics of obesity and stresses that a mixture of different pathways ought to be applied in the intervention strategies. The model can be extended in the future by adding stochastic factors, genetic factors, and real-time analysis of health conditions, in order to increase the predictive accuracy. In conclusion, the discussion emphasizes that obesity caused by diet is not merely the aftermath of overeating but the complex interactions between dietary practices, the hormonal mechanisms, and metabolic processes. Prevention and treatment plans should hence be holistic in that they address all these factors which are interrelated.

6. Conclusion

The paper concluded that an in-depth and integrated approach to the diet-induced obesity with the assistance of mathematical modeling and Smart PLS analysis. The model is able to capture the dynamic and complex nature of the mechanisms of metabolism that lead to the development of obesity by integrating the main elements of energy intake, hormonal control, and energy utilization. The results clearly indicate that high energy consumption is the major determinant of obesity, which has direct effects on fat build up and indirect effects on the same via hormonal mechanisms. Hormonal control, that is, the ones of insulin, leptin, and ghrelin, turn out to be a key mediating element that connects dietary behavior and metabolic results. The interference with these hormone systems greatly increases the threat of prolonged weight gain and metabolic disequilibrium. In the meantime, the protective, although relatively minor role is attributed to energy expenditure, which means that even physical activity cannot completely offset the impact of chronic overconsumption. The combination of the differential equations and the Smart PLS analysis makes the research strong in validity as the theoretical modeling is supplemented with an empirical analysis. The mathematical model describes how these interactions and feedback loops change with time, and the results of the Smart PLS give statistical support of the strength and importance of these associations. Collectively, they provide a solid and multidimensional concept of obesity dynamics. Despite the fact that the model oversimplifies some complexities that are involved in the real world e.g. genetic, behavioral and environmental factors, the model remains very powerful in the analysis and prediction of obesity trends. The relatively high explanatory power of the model is a good indicator that it captures important determinants of obesity thus it can be used in research and practical purposes. Finally, diet-related obesity is a complex disorder that is caused by the combined effect of the caloric imbalance, hormonal dysregulation, and metabolic processes. To deal with this international health issue, combined interventions, which will all aim at dietary practices, hormonal balance, and lifestyle choices, are necessary. The model suggested will not only contribute to a deeper theoretical knowledge but also promote the creation of more useful and customized interventions to prevent and manage obesity.

7. Recommendations

- Obesity management should not be simply based on a calorie-restriction program, but the use of hormonal regulation programs should be included. To recover energy homeostasis more effectively, the interventions that improve insulin sensitivity and leptin signaling (e.g., physical activity, balanced macronutrient diets, and stress reduction) are proposed.
- Educational campaigns should also focus on the fact that obesity is not the simple result of overeating, but also the complex control over hormones. Compliance to healthier lifestyle behaviors can be enhanced through awareness campaigns.
- The healthcare systems need to be submitted to mathematical and computational models to predict the occurrence of obesity, as well as to evaluate the efficacy of interventions. This will be capable of supporting clinical decision-making using data.

- Stress, sleeping patterns, and eating behavior should be included in new models and interventions as they are important in the dynamics of hormonal regulation and energy intake.
- Further studies are recommended to involve genetic variables, interaction with gut microbiota, and long-term adaptive physiological responses to improve the accuracy and predictability of a model. All these suggestions emphasize the comprehensive, systems approach to obesity prevention and treatment, combining the biological image and real-world healthcare practices.

8. Future Research Directions

Future studies of mathematical modeling of diet-induced obesity dynamics with intake of energy and regulation of hormones should endeavor to improve and extend existing models to capture more accurately biological complexity and human behavior in the real world. First one is Genetic and epigenetic factors according to the research the next generation should incorporate genetic predisposition and epigenetic changes that affect metabolism, fat storage, and sensitivity to hormones. This will aid in elucidating the reasons behind why people react differently to similar eating habits. Second is Gut microbiota dynamic integration the new data indicate that the microbiota in the gut plays a crucial role in terms of energy mining, appetite control, and inflammation. The inclusion of microbiome interactions in obesity models will enhance the predictive accuracy and biological realism.

- Design of multi-scale models: The relationships between cellular-level hormonal signaling and models of whole-body energy balance need to be bridged in future studies. Multi-scale models are able to bridge the gap between molecular processes and macro-level obesity patterns.
- Use of advanced machine learning and AI integration: Hybrid architectures of the combination of classical differential equations and machine learning algorithms (e.g., random forests, neural networks, etc.) may increase the accuracy of prediction and enable real-time forecasting of obesity risk.

Data validation on a longitudinal and real-world basis: The validation of model predictions should be done by future research based on long-term clinical and population-based data. Wearable data collection devices and health trackers can be employed to enhance model calibration as continuous data is collected.

9. References

- Al-Tuwairqi, S., Alamer, K., & Malaikah, H. (2026). Modeling the Glucose-Insulin-Glucagon Interactions. *Pan-American Journal of Mathematics*, 5, 1.
- Amato, A., Baldassano, S., & Musumeci, G. (2026). Applying AI Tools for Monitoring Nutrition and Physical Activity in Populations with Obesity: Are We Ready? *Obesities*, 6(2), 19.
- Bansal, H., & Srija, K. D. (2026). Decoding the Human Polybiome Through AI and Nutrigenomics in Next Generation Personalized Health: Integrating Multi-Omics for Precision Medicine. In *Advancing Personalized Medicine With AI-Driven Microbiome and Nutrigenomics: Innovations and Future Perspectives* (pp. 93-172). IGI Global Scientific Publishing.
- Barrea, L., Verde, L., Camajani, E., Annunziata, G., Mollica, M. P., Minnetti, M., Trinchese, G., Losignore, P., Colao, A., & Ferlin, A. (2026). A Novel Chronotype-Based Mediterranean Diet Pyramid: An Updated Representation by the Italian Society of Endocrinology (SIE) and the Italian Society of Dietetics and Clinical Nutrition (ADI). *Current Nutrition Reports*, 15(1), 10.
- Barrera-Esparza, M., Carreón-Torres, E., Jiménez-Osorio, A. S., Angel-García, J., Jiménez-Garza, O., Flores-Chávez, O. R., Mendoza-Catalán, G. S., & Estrada-Luna, D. (2025). Complex networks interactions between bioactive compounds and adipose tissue vis-à-vis insulin resistance. *Frontiers in endocrinology*, 16, 1578552.
- Buljan, I., Benković, M., Jurina, T., Sokač Cvetnić, T., Valinger, D., Gajdoš Kljusurić, J., & Jurinjak Tušek, A. (2025). Simulation and Local Parametric Sensitivity Analysis of a Computational Model of Fructose Metabolism. *Processes*, 13(1), 125.
- Cadena, M., & Barreto, G. E. (2025). Exploring the interplay between circadian rhythms and obesity: A Boolean network approach to understanding metabolic dysregulation. *PLoS one*, 20(9), e0331218.
- Choo, Y. N., Ravi, R. N., & Subramaniam, V. (2026). Insulin resistance induced by obesity: Mechanisms, metabolic implications and therapeutic approaches. *Molecular Biology Reports*, 53(1), 357.
- Chudtong, M., & De Gaetano, A. (2025). Food choices and body weight changes: A mathematical model analysis.

Mathematical Biosciences and Engineering, 22(7), 1790-1824.

- Chudtong, M., Nagy, I., & De Gaetano, A. (2025). Modeling the Interplay of Ghrelin, Leptin, and Exercise on Energy Balance: A Systems Dynamics Approach. 2025 IEEE 25th International Symposium on Computational Intelligence and Informatics (CINTI),
- Ghouili, H., Farhani, Z., Yousfi, N., Ceylan, H. İ., Dridi, A., De Giorgio, A., Bragazzi, N. L., Guelmami, N., Dergaa, I., & Bouassida, A. (2026). A Maturation-Aware Machine Learning Framework for Screening the Nutritional Status of Adolescents. *Nutrients*, 18(4), 660.
- Han, M. H., Hwang, Y. S., JIN, D. H., & KIM, J. W. (2026). Biotechnology and Bioengineering Innovations in Precision Obesity Management: Mechanistic Insights, Emerging Platforms, and Translational Pathways. *Journal of Mechanics in Medicine and Biology*, 2630001.
- Jiang, X., Luo, L., Sun, F., Jiang, J., Liu, Z., & Zeng, L. (2026). Regulation of Intracellular Lipid Droplets by Tea: Mechanistic Insights and Food Applications. *Comprehensive Reviews in Food Science and Food Safety*, 25(3), e70465.
- Jin, Z., Thackray, A. E., Willis, S. A., King, J. A., Li, J., Mould, C., & Stensel, D. J. (2026). The association between glucose dynamics and energy intake in young, healthy women. *European Journal of Clinical Nutrition*, 1-9.
- Levin, B. E., Geary, N., & Lutz, T. A. (2025). The (dys) regulation of energy storage in obesity. *Physiological Reviews*, 105(3), 803-895.
- Li, Y., Sharma, R., Usiyevich, A., Shen, X., Zhang, K. W., Khamaru, K., & Matikainen-Ankney, B. A. (2026). Weight Loss After Obesity Disrupts Cognitive Flexibility Through Reinforcement Learning Strategies. *Obesity*, 34(1), 188-198.
- Liu, Q., Li, C., Zhang, Y., Liu, C., Liu, Y., Tian, M., Zhu, Q., Chen, Y., Yu, L., & Wang, H. (2026). Key dietary amino acids modulating overweight/obesity risk in Chinese children and adolescents: a machine learning analysis of a national survey. *Frontiers in nutrition*, 13, 1769111.
- López-Almada, G., Domínguez-Avila, J. A., Robles-Sánchez, R. M., Arauz-Cabrera, J., Martínez-Coronilla, G., González-Aguilar, G. A., & Salazar-López, N. J. (2025). Naringenin decreases retroperitoneal adiposity and improves metabolic parameters in a rat model of western diet-induced obesity. *Metabolites*, 15(2), 109.
- Magkos, F. (2025). A Hypothesis on the Historical Development of Obesity that is Not Only About Food. *Current Obesity Reports*, 14(1), 58.
- Milan, M., Troyano-Rodriguez, E., Ihuoma, J., Negri, S., Rudraboina, R., Kosmider, A., Awasthi, S., Balasubramanian, P., Conley, S., & Yabluchanskiy, A. (2026). Fasting as Medicine: Mitochondrial and Endothelial Rejuvenation in Vascular Aging. *Aging Cell*, 25(2), e70372.
- Nakamura, S., Yanai, M., Eto, A., Eto, S., Saito, Y., Suzuki, T., Seto, T., & Narimatsu, H. (2026). Genetic Risk Scores for Obesity and the Effectiveness of a Diet and Exercise Intervention Program: A Historical Cohort Study. *Obesity*, 34(3), 719-728.
- Nambi, G., Alghadier, M., Vellaiyan, A., Mohamed, S. H. P., Eswaramoorthi, V., & Muthukrishnan, R. (2026). Association of aerobic capacity and handgrip strength in obese and non-obese children aged 10–15 years in Riyadh region, KSA—a cross sectional study. *Scientific Reports*.
- Ray, A. (2026). The 24 Hours Serotonin–Melatonin Dynamics in the SCN–Pineal Circadian System—A Mathematical Model. *Compassionate AI*, 1(1), 6-8.
- Saikia, A. P., & Kalita, A. (2025). Artificial Intelligence and multi-omics integration in obesity: a review of computational models for predicting metabolic comorbidities. *Clinical & Translational Metabolism*, 23(1), 7.
- Sazdova, I., Gagov, H., Hadzi-Petrushev, N., Konaktchieva, M., Konakchieva, R., & Mladenov, M. (2026). Advanced Studies in Molecular and Metabolic Mechanisms of Diabetes: Pathogenic Crosstalk, Biomarkers, and Translational Therapeutics. *Applied Sciences*, 16(6), 3027.
- Shokeralla, A. A., Alzharani, A. A., Abdalla, A. H., Modawy, Y. M., Elshamy, I., & El Guma, F. (2025). Modeling Climate-Driven Cholera Outbreaks: A Negative Binomial Regression Framework with Improved Handling of Overdispersion and Extreme Events. *Letters in Biomathematics*, 12(1).
- Strusi, G., Suelzu, C. M., & Stebbing, J. (2026). Modelling adipose tissue-cancer crosstalk: a three-dimensional perspective.

Oncogene, 1-9.

- Subaş, T., Çetinkaya, S., Şirin, S., Başer, T., & Süntar, I. (2026). Dietary polyphenols targeting NLRP3 inflammasome in obesity and metabolic disorders: a review on experimental and computational evidence. *International Journal of Food Sciences and Nutrition*, 1-17.
- Wang, J., Liu, L., Liu, H., Qian, Y., Zhang, S., Zheng, S., Jiang, H., Zhou, Y., Cheng, X., & Fu, Q. (2025). Dynamic multi-omics profiling of islet and gut hormonal secretion and peripheral crosstalk in response to various nutrient loads. *Cell Reports Medicine*, 6(9).
- Wang, L., & Sui, S. (2026). The effects of rebound exercise on body mass index and balance in overweight and obese adults: a meta-analysis. *Endocrine Connections*, 15(4).