A Novel Mathematical Model for Overweight, Obesity, and Their Impact on Diabetes and Hypertension

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Abstract

In this paper, we present a new mathematical model describing the dynamics of overweight and obesity and their impact on diabetes and hypertension. In constructing the model, we consider negative and positive interactions among individuals with normal weight, overweight, and obesity, as well as social factors influencing overweight and hypertension diagnoses. As a novel contribution to transmission dynamics, we interpret the basic reproduction number from two perspectives: negative and positive interactions. Focusing on parameters linked to social factors and their health impact, we present theoretical results characterizing their influence on the basic reproduction number and compute corresponding sensitivity indices. Additionally, we perform a global sensitivity analysis of model parameters using first- and total-order Sobol' indices with various methods and sampling techniques, concluding that parameters associated with social factors are among the most influential. We conduct computational simulations of the basic reproduction number and model's compartments to examine the influence of social-factor parameters on overweight and hypertension. Our findings indicate the need to explore strategies to prevent the rise of overweight, obesity, and diabetes in the population. Social factors associated with overweight and hypertension diagnosis have a substantial impact on the progression of these dynamics. Recognizing this influence enables the identification of the most vulnerable groups and the design of more precise and effective interventions.

Keywords: Diabetes, hypertension, mathematical model, obesity, overweight, social factors

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1. INTRODUCTION

The 2024 edition of the World Obesity Atlas, published by the World Obesity Federation (WOF), estimates that 42% of the world's adult population was overweight in 2020, approximately 1.39 billion individuals overweight and 810 million obese [1]. Body weight status is determined using the body mass index (BMI), defined as [2]:

$$\text{BMI} = \frac{\text{weight}}{\text{height}^2}.$$

Individuals are classified as normal weight when BMI $\in [18.5, 24.9]$, overweight when BMI $\in [25, 29.9]$, obese when BMI $\in [30, 40]$, and severely obese when BMI > 40. Although BMI can be elevated in individuals with high muscle mass, we assume such cases are excluded, as a preliminary analysis is performed before BMI calculation [1].

Diabetes is a disease caused by insufficient insulin production or poor absorption of this hormone, which regulates blood glucose and provides energy for the body. Several factors increase the risk of developing type 2 diabetes, including age, family history, smoking, excessive alcohol consumption, ethnicity, and certain medical conditions [3], [4]. For the purposes of this paper we will focus on type 2 diabetes. Approximately 90% of individuals with type 2 diabetes are overweight or obese, and obesity triples the risk of developing diabetes compared to non-obese individuals [5], [6].

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Hypertension, or high blood pressure, is a chronic condition characterized by persistently elevated arterial pressure [7]. It is closely linked to obesity, as both disorders can influence and exacerbate each other. An increased body mass index (BMI) is directly associated with higher blood pressure levels, reflecting a pathophysiological connection between excess adiposity and vascular dysfunction [8]. This condition is highly prevalent among individuals with type 2 diabetes mellitus and constitutes a major risk factor for the development of cardiovascular complications. Management in diabetic patients involves lifestyle and dietary modifications, pharmacological therapy, and controlling additional cardiovascular risk factors to prevent long-term morbidity and mortality [9]. Patients with type 2 diabetes are significantly more likely to have hypertension, and at any age, their average blood pressure exceeds that of non-diabetics. Type 2 diabetes is generally associated with central obesity, which further increases the likelihood of hypertension.

Different studies have been developed to address obesity and its effect on health and the population. Bernard et al. [10] proposed a new mathematical model for obesity and explored the impact of the media on the spreading of this phenomenon in a constant population. Moya et al. [11] presented a new mathematical model for the study of overweight and obesity in a population and its impact on the growth in the number of diabetics, considering the influence of human interactions and social factors, and demonstrated the need for a control strategy to reduce obesity and, consequently, diabetes. Moya et al. [12] introduced a variant of the model discussed in [11], incorporating the negative effect of human interactions and using Caputo's fractional derivatives as a modeling method, and demonstrated the need for effective control strategies to reduce overweight and obesity. Moya et al. [13] formulated an optimal control problem, using controls focused on the control of human interactions and social pressure, using the Caputo derivative model [12]. Keisuke et al. [14] suggested a model to describe the risk of obesity as a function of time and age, the hazard of which is treated as dependent and independent of the prevalence of obesity, compared the effectiveness of different types of interventions, including those targeting never obese individuals (primary prevention) and obese and formerly obese individuals (secondary prevention) and showed that the optimal choice of obesity interventions varies depending on the potential for transmission of obesity from one person to another. Salma M and Reem T [15] proposed two mathematical models to study the impact of fast food on obesity, analyzing separately the influence of peer pressure on fast food consumption and the role of exercise on weight gain. The models demonstrated the dynamics of individuals moving from one weight class to another as a function of their body mass index (BMI) and showed the importance of resisting peer pressure that drives individuals to consume fast food, as well as maximizing the role that quitters can play in convincing obese individuals to stop consuming fast food and also that physical activity plays an important role in weight reduction. Zina et al. [16] presented a mathematical model describing obesity and its complications with the aim of reducing the obese population and decreasing the prevalence of type 2 diabetes, cardiovascular disease and hypertension and demonstrated that the prevalence of obesity and its complications can be controlled and minimized by reducing the impact of social factors.

Our objective is to study the relationships among overweight, obesity, diabetes, and hypertension, and to assess how obesity and overweight influence diabetes and hypertension in terms of reducing or increasing the number of cases. To this end, we propose a new mathematical model that captures the dynamics of these diseases, allowing us to understand their interactions and the impact of each on the others, while also incorporating the influence of social factors. As an innovative aspect of this work, the basic reproduction number of positive interactions among individuals is defined and analyzed.

The article is organized as follows: Section 2 is devoted to model construction. Section 3 demonstrates the basic properties and defines the basic reproduction number for positive and negative interactions. Section 4 includes numerical results: computational simulations, study of the basic reproduction numbers, and presentation of global sensitivity analysis and compartment behavior. Section 5 presents the conclusions.

2. MODEL FORMULATION

A compartment represents a homogeneous subpopulation within the epidemiological system, where individuals share the same health or disease state and exhibit similar dynamic properties. Each compartment groups epidemiologically equivalent individuals, and transitions between compartments describe disease progression or state changes within the population [17]. The model includes the following compartments: N_w, containing normal weight individuals, O_w, overweight individuals, O_b, obese individuals, D, individuals with type 2 diabetes, H, individuals with hypertension, and C, individuals with concurrent diabetes and hypertension.

For the H and D compartments, we did not stratify by body weight because these chronic diseases are incurable but manageable. Hypertensive patients who fail to improve their lifestyle may die from blood pressure-related causes, develop diabetes, or remain in the same compartment. Similarly, diabetics may die from diabetes-related causes, develop hypertension, or remain in their compartment. The birth rate is Λ, and μ is the natural death rate, which is the same in all compartments and represents any death not associated with overweight, obesity, diabetes, or hypertension.

The transmission dynamics in this study focus on overweight and obesity, modeled as conditions spread through lifestyle influence, where one individual's behavior affects another's. We define three transmission rates based on the effect of negative or positive influence.

The negative effect transmission rate is

$$\lambda_N = \left(\frac{\alpha_N (O_w + O_b)}{N_T} \right), \tag{1}$$

the positive effect on overweight is

$$\lambda_{p1} = \left(\frac{\alpha_{p1} N_w}{N_T} \right), \tag{2}$$

and the positive effect on the obese is

$$\lambda_{p2} = \left(\frac{\alpha_{p2} N_w}{N_T} \right), \tag{3}$$

where β_N, α_{p1}, and α_{p2} are the effective contact rates in the negative and positive impact contacts, respectively, and N_T is the total population.

Social factors that can contribute to overweight and obesity include access to food, social networks, and stress. These factors can vary by neighborhood, zip code, and social class. A neighborhood's food environment is characterized by whether or not healthy foods are readily available and the types of food offered in schools and daycares. Another key factor is social networks. Chronic stress can increase appetite and cortisol levels, a hormone that regulates blood sugar; a restricted diet can also lead to weight gain, low mood, and body dissatisfaction [18], [19]. Parameter p_{s1} incorporates social factors into the model and how they influence whether a person of normal weight becomes overweight.

High blood pressure can be caused by various social factors, such as level of education, work environment, and alcohol consumption [20]. Other social factors, such as stress, isolation, and socioeconomic status can contribute to hypertension. These factors can affect the body's stress response system, which can lead to vascular damage and hypertension [21]. Then, parameter p_{s2} refers to the diagnosis of hypertension due to the influence of social factors.

The progression from overweight to obese is defined by γ. The parameters α_1 and β_1 represent the rate of developing diabetes due to causes other than overweight or obesity. The parameters α_2 and β_2 are the rates of development of diabetes and hypertension in overweight individuals, respectively, and α_3 and β_3 are the rates of diabetes and hypertension in obese individuals. We can assume that $\alpha_1 < \alpha_2$, $\alpha_1 < \alpha_3$ [22], [23], [24], $\beta_1 < \beta_2$ and $\beta_1 < \beta_3$ [25], [26].

We assume that in the progression from obese to overweight, due to the difficulties of the process because of the high body weight, it is necessary the interaction with other individuals generally of normal weight that can be medical personnel, physical trainers, among others that guide the process of weight loss. We define d_O as death associated with obesity, d_D as death associated with diabetes, d_H as death associated with hypertension, and d_C as death associated with hypertension-diabetes. In defining these parameters we must assume that in cases of obesity-associated death cannot be related to diabetes and/or hypertension, analogously it is assumed for d_D, d_H, and d_C. The parameters η_{DH} and η_{HD} are the rates of an individual with diabetes developing hypertension and a hypertensive developing diabetes, respectively.

Table 1 presents the definition of the model parameters and Figure 1 shows the flow diagram of the model dynamics. The following system of ordinary differential equations defines the dynamics of overweight and

obesity behavior and its impact on diabetes and hypertension:

$$\frac{dN_w}{dt} =\Lambda + \lambda_{p1}O_w - \Big(\mu + \lambda_N + p_{s1} + p_{s2} + \alpha_1 + \beta_1\Big)N_w, \tag{4}$$

$$\frac{dO_w}{dt} =\Big(\lambda_N + p_{s1}\Big)N_w + \lambda_{p2}O_b - \Big(\mu + \lambda_{p1} + \lambda_N + \gamma + \alpha_2 + \beta_2\Big)O_w, \tag{5}$$

$$\frac{dO_b}{dt} =(\gamma + \lambda_N)O_w - \Big(\mu + \lambda_{p2} + d_O + \alpha_3 + \beta_3\Big)O_b, \tag{6}$$

$$\frac{dD}{dt} =\alpha_1 N_w + \alpha_2 O_w + \alpha_3 O_b - (\mu + d_D + \eta_{DH})D, \tag{7}$$

$$\frac{dH}{dt} =(\beta_1 + p_{s2})N_w + \beta_2 O_w + \beta_3 O_b - (\mu + d_H + \eta_{HD})H, \tag{8}$$

$$\frac{dC}{dt} =\eta_{DH}D + \eta_{HD}H - (\mu + d_C)C, \tag{9}$$

with initial conditions: $N_w(0) = N_{w0} > 0, \quad O_w(0) = O_{w0} > 0, \quad O_b(0) = O_{b0} > 0, \quad D(0) = D_0 > 0, \quad H(0) = H_0 > 0, \quad C(0) = C_0 > 0.$

Table 1: Parameters description Values of the parameters of Model (4)-(9).

Parameter	Description	Value	Reference
Λ	Birth rate	667.685	[27], [28]
μ	Natural death rate	1/70.5	[28]
d_O	Death rate associated with obesity	0.07	[11], [12], [13]
d_D	Death rate associated with diabetes	0.013	[11], [12], [13], [29]
d_H	Death rate associated with hypertension	0.012	[30]
d_C	Death rate associated with diabetes-hypertension	0.005	Assumed
α_N	Negative effective contact rates	2	[11], [12]
α_{p1}	Positive effective contact rates associated with overweight	0.2	[12], [13]
α_{p2}	Positive effective contact rates associated with obesity	0.1	Assumed
p_{s1}	Rate of becoming overweight associated with social factors	0.25	[11]
p_{s2}	Rate of hypertension associated with social factors	0.291	[31]
γ	Rate of progression from overweight to obesity	0.002	[10], [11]
α_1	Rate at which individuals develop diabetes independent of body weight	0.1	[11]
α_2	Rate at which overweight individuals develop diabetes	0.35	[32], [11]
α_3	Rate of developing diabetes in obesity cases	0.4	[32], [11]
β_1	Rate at which individuals develop hypertension independent of body weight	0.05	Assumed
β_2	Rate at which overweight individuals develop hypertension	0.35	Assumed
β_3	Rate at which obese individuals develop hypertension	0.65	[33]
η_{DH}	Rate at which diabetic individuals develop hypertension	0.5	[34]
η_{HD}	Rate at which hypertensive individuals develop diabetes	0.223	[35]

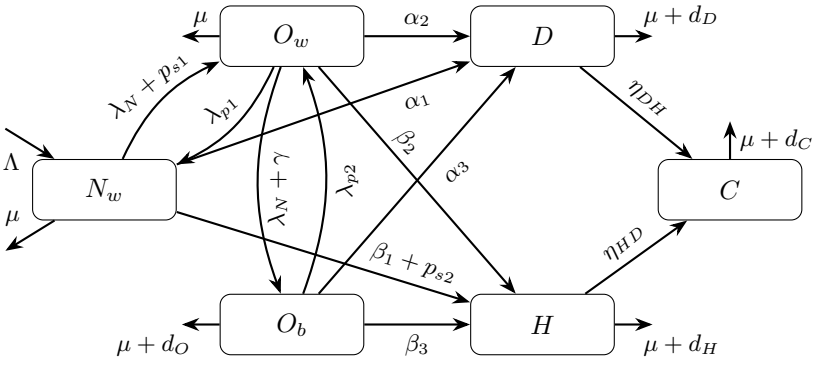


Figure 1: Flow chart of Model (4)-(9).

3. Model Analysis

3.1. Basic Properties

Model (4)-(9) represents the human population divided into compartments. By definition, all variables and parameters are positive. Solutions of Model (4)-(9) remain non-negative for positive initial conditions at any time $t > 0$ [36], [37].

A valid population model must satisfy logical requirements, such as positivity of solutions. Therefore, we define a biologically feasible region as the domain where the model is meaningful. Next, we determine this region for Model (4)-(9).

Lemma 3.1. *The closed set* $\Omega = \left\{ (N_w, O_w, O_b, D, H, C) \in \mathbb{R}^6_+ : N_T(t) \leq \dfrac{\Lambda}{\mu} \right\}$ *is positively-invariant and attracts all solutions of Model (4)-(9).*

Proof: The derivative of N_T (total population) is

$$\frac{dN_T}{dt} = \Lambda - \mu N_T - d_O O_b - d_D D - d_H H - d_C C. \tag{10}$$

Since $\dfrac{dN_T}{dt} \leq \Lambda - \mu N$, it follows that $\dfrac{dN_T}{dt} \leq 0$, if $N_T(t) \geq \dfrac{\Lambda}{\mu}$. Hence, the standard comparison theorem from [38] can be used to show that $N_T(t) \leq N_T(t_0)\exp\{-\mu t\} + \dfrac{\Lambda}{\mu}\left(1 - \exp\{-\mu t\}\right)$.

In particular, if $N_T(t_0) \leq \dfrac{\Lambda}{\mu}$, then $N_T(t) \leq \dfrac{\Lambda}{\mu}$ for all $t > 0$.

Hence, the domain Ω is positively invariant.

Furthermore, if $N_T(t_0) > \dfrac{\Lambda}{\mu}$, then either the solution enters the domain Ω in finite time or $N_T(t)$ approaches $\dfrac{\Lambda}{\mu}$ asymptotically as $t \to \infty$. Hence, the domain Ω attracts all solutions in $\mathbb{R}^6_+$. ■

3.2. Basic Reproduction Number

The basic reproduction number ($\Re_0$) represents the average number of secondary cases caused by an infected individual during the infectious period. It is a key public health indicator for estimating the speed of disease spread [39], [40]. If $0 < \Re_0 < 1$, the infection will eventually disappear; if $\Re_0 > 1$ it can spread within the population. A higher $\Re_0$ indicates greater difficulty in controlling the epidemic. Factors influencing $\Re_0$ include the duration of infectivity, pathogen transmissibility, and contact rates between susceptible and infected individuals.

Our model incorporates positive and negative interactions among normal weight, overweight, and obese individuals. Accordingly, we compute the basic reproduction number for negative interactions, treating overweight and obesity as epidemic-like phenomena [28], [41], [42], and define reproduction numbers for positive interactions, representing cases where normal weight individuals influence overweight or obese individuals to adopt healthier lifestyles and reduce body weight.

Another important element is that in constructing the basic reproduction number, we have incorporated the diabetes, hypertension, and hypertension and diabetes compartments, which are transitional but allow us to study the impact of the parameters associated with these chronic diseases on the basic reproduction number.

Let's define the basic reproduction number for negative interactions as $\Re_0^N$ and for positive interactions as $\Re_0^{p1}$ and $\Re_0^{p2}$. Two fundamental questions appear: what is the objective of separating the interactions? How are positive interactions treated in the construction of $\Re_0^N$ and vice versa?

In the analysis of the basic reproduction numbers we consider the dynamics of the full model (4)-(9). That is, for the dynamics $\Re_0^N$, the elements $\dfrac{\alpha_{p1} N_w}{N_T}$ and $\dfrac{\alpha_{p2} N_w}{N_T}$ are constants, in $\Re_0^{p1}$ the elements $\dfrac{\alpha_N N_w}{N_T}$ and $\dfrac{\alpha_{p2} N_w}{N_T}$ are constants and in $\Re_0^{P2}$ the elements $\dfrac{\alpha_N N_w}{N_T}$ and $\dfrac{\alpha_{p1} N_w}{N_T}$ are constant. This will help to answer the question: which has a greater impact on transmission according to the basic reproduction number: negative or positive, and among the positive ones, which one influences more: the interactions between a

normal weight individual and an overweight individual or an obese individual. These results may contribute to a better understanding of the dynamics, and to the construction of future control strategies.

The disease-free equilibrium point is when all variables in the model reach zero except for the one for those of normal weight. In this case, we do not have the diseases of overweight and obesity and the consequences of diabetes and hypertension. Therefore, the disease-free equilibrium point (ϵ_0) is:

$$\epsilon_0 = \left(\frac{\Lambda}{k_{nw}}, 0, 0, 0, 0, 0\right), \tag{11}$$

where $k_{nw} = \alpha_1 + \beta_1 + p_{s1} + p_{s2} + \mu$.

To find $\Re_0$'s we use the next-generation matrix, utilizing the methodology presented in [39], [40]. The transmission matrix captures the rates of new infections generated between the different compartments, while the transition matrix represents the rates of movement between compartments involved in the basic reproduction number. The transmission and transition matrices for the study of negative interactions are:

$$F_N = \begin{pmatrix} \frac{\alpha_N \Lambda}{N_T k_{nw}} & \frac{\alpha_N \Lambda}{N_T k_{nw}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{12}$$

$$V_N = \begin{pmatrix} k_w^N & -\frac{\alpha_{p2}\Lambda}{N_T k_{nw}} & 0 & 0 & 0 \\ -\gamma & k_b^N & 0 & 0 & 0 \\ -\alpha_2 & -\alpha_3 & k_D & 0 & 0 \\ -\beta_2 & -\beta_3 & 0 & k_H & 0 \\ 0 & 0 & -\eta_{DH} & -\eta_{HD} & k_C \end{pmatrix}, \tag{13}$$

where $k_w^N = \mu + \gamma + \alpha_2 + \beta_2 + \frac{\alpha_{p1}\Lambda}{N_T k_{nw}}$, $k_b^N = \mu + d_O + \alpha_3 + \beta_3 + \frac{\alpha_{p2}\Lambda}{N_T k_{nw}}$, $k_D = \mu + d_D + \eta_{DH}$, $k_H = \mu + d_H + \eta_{HD}$ and $k_C = \mu + d_C$.

Thus, the basic reproduction number is defined as the spectral radius (i.e., the absolute value of the largest eigenvalue) of the matrix representing the negative of the product of the transmission matrix and the inverse of the transition matrix, which in this case is:

$$\Re_0^N = \rho(F_N V_N^{-1}) = \frac{\Lambda \alpha_N (k_b^N + \gamma)}{N_T k_{nw} k_b^N k_w^N - \alpha_{p2}\gamma\Lambda}, \tag{14}$$

where $\rho(F_N V_N^{-1})$ is the spectral radius of the matrix $F_N V_N^{-1}$.

The transmission and transition matrices for the basic reproduction number associated with positive interactions between a normal weight individual and an overweight individual are:

$$F_{p1} = \begin{pmatrix} \frac{\alpha_{p1}\Lambda}{N_T k_{nw}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{15}$$

$$V_{p1} = \begin{pmatrix} k_w^{p1} - \frac{\alpha_N \Lambda}{N_T k_{nw}} & -\frac{\alpha_N \Lambda}{N_T k_{nw}} - \frac{\alpha_{p2}\Lambda}{N_T k_{nw}} & 0 & 0 & 0 \\ -\gamma & k_b^N & 0 & 0 & 0 \\ -\alpha_2 & -\alpha_3 & k_D & 0 & 0 \\ -\beta_2 & -\beta_3 & 0 & k_H & 0 \\ 0 & 0 & -\eta_{DH} & -\eta_{HD} & k_C \end{pmatrix}, \tag{16}$$

where $k_w^{p1} = \mu + \alpha_2 + \beta_2 + \gamma$. Then, we have that:

$$\Re_0^{p1} = \rho(F_{p1}V_{p1}^{-1}) = \frac{\Lambda\alpha_{p1}k_b^N}{\Lambda(\alpha_{p2}k_b^N + \alpha_N(k_b^N + \gamma)) - N_T k_{nw} k_b^N k_w^{p1}}, \tag{17}$$

where $\rho(F_{p1}V_{p1}^{-1})$ is the spectral radius of the matrix $F_{p1}V_{p1}^{-1}$.

For computing the basic reproduction number associated with the interactions between a normal weight individual and an obese individual with positive effect, the transmission and transition matrices are:

$$F_{p2} = \begin{pmatrix} 0 & \frac{\alpha_{p2}\Lambda}{N_T k_{nw}} & 0 & 0 & 0 \\ 0 & -\frac{\alpha_{p2}\Lambda}{N_T k_{nw}} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{18}$$

$$V_{p2} = \begin{pmatrix} k_w^N - \frac{\alpha_N\Lambda}{N_T k_{nw}} & -\frac{\alpha_N\Lambda}{N_T k_{nw}} & 0 & 0 & 0 \\ -\gamma & k_b^{p2} & 0 & 0 & 0 \\ -\alpha_2 & -\alpha_3 & k_D & 0 & 0 \\ -\beta_2 & -\beta_3 & 0 & k_H & 0 \\ 0 & 0 & -\eta_{DH} & -\eta_{HD} & k_C \end{pmatrix}, \tag{19}$$

where $k_b^{p2} = \mu + \alpha_3 + \beta_3 + d_O$. Then, we have that:

$$\Re_0^{p2} = \rho(F_{p2}V_{p2}^{-1}) = \frac{\alpha_{p2}\Lambda(\alpha_N\Lambda - (k_w^N - \gamma)N_T k_{nw})}{N_T k_{nw}(k_b^{p2}k_w^N N_T k_{nw} - \alpha_N\Lambda(k_b^{p2} + \gamma))}, \tag{20}$$

where $\rho(F_{p2}V_{p2}^{-1})$ is the spectral radius of the matrix $F_{p2}V_{p2}^{-1}$.

We will present results that relate the local and global stability of the disease-free equilibrium point with the basic reproduction number associated with interactions with negative effects, so, we will conduct the study for the $\Re_0^N$. When the $\Re_0^N$ is less than unity, these interactions are reduced, helping to reduce the epidemic. But, in the case of positive interactions with the basic reproduction number, the objective is for it to be greater than unity, and the greater the number, the greater the impact of these interactions, which help reduce overweight and obesity in the population. The results and their demonstrations are constructed without the presence of social factors and their impact on overweight and hypertension ($p_{s1} = p_{s2} = 0$) since the next objective is to directly study their impact on basic reproduction numbers.

Theorem 3.2. *The disease-free equilibrium point (ϵ_0) of Model (4)-(9), is locally asymptotically stable (LAS) if $\Re_0^N < 1$ and unstable if $\Re_0^N > 1$.*

The threshold quantity $\Re_0^N$ measures the average number of new diseases (overweight and obesity) generated by a single overweight or obese person in a completely normal weight population. Consequently, the disease-free equilibrium point of Model (4)-(9) is locally asymptotically stable (LAS) whenever $\Re_0^N < 1$ and unstable if $\Re_0^N > 1$. This means that overweight and obesity can be eliminated from the community (when $\Re_0^N < 1$) if the population sizes of Model (4)-(9) are in the basin of attraction of the disease-free equilibrium point ϵ_0.

Now, we prove the global stability of the disease-free equilibrium point. Following [43], we can rewrite Model (4)-(9) as

$$\frac{dS}{dt} = f(N_w, I), \tag{21}$$

$$\frac{dI}{dt} = g(N_w, I), \quad g(S, 0_{\mathbb{R}^4}) = 0, \tag{22}$$

where $N_w \in \mathbb{R}_+$ is the normal weight compartment and $I = (O_w, O_b, D, H, C) \in \mathbb{R}^5_+$ have the overweight, obese, diabetes, hypertension, diabetes-hypertension compartments. Moreover, throughout the paper, $0_{\mathbb{R}^n}$ denotes the null vector in $\mathbb{R}^n$.

The disease-free equilibrium point is now denoted by $E_0 = (N_w^0, 0_{\mathbb{R}^5})$ where $N_w^0 = \dfrac{\Lambda}{k_{nw}}$.

The conditions (H_1) and (H_2) below must be satisfied to guarantee the global asymptotic stability of E_0.

$$(H_1): \quad \text{For} \quad \frac{dN_w}{dt} = f(N_w, 0_{\mathbb{R}^5}), \quad N_w^0 \quad \text{is globally asymptotically stable,}$$
$$(H_2): \quad g(N_w, I) = AI^T - g^*(N_w, I), \quad g^*(N_w, I) \geq 0, \quad \text{for} \quad (N_w, I) \in \Omega,$$

where $A = D_I g(N_w^0, 0_{\mathbb{R}^5})$, $D_I g(N_w^0, 0_{\mathbb{R}^5})$ is the Jacobian of g at $(N_w^0, 0_{\mathbb{R}^5})$, and is a M-matrix (the off-diagonal elements of A are non-negative), I^T is the transpose of vector $I \in \mathbb{R}^5_+$, and Ω is the biologically feasible region.

The following theorem shows the global stability of the disease-free equilibrium point.

Theorem 3.3. *The fixed point E_0 is a globally asymptotically stable equilibrium (GAS) of Model (4)-(9) provided that $\Re_0^N < 1$ and that the conditions (H_1) and (H_2) are satisfied.*

Proof: Let

$$f(N_w, 0_{\mathbb{R}^5}) = \Lambda - (\mu + \alpha_1 + \beta_1 + p_{s1} + p_{s2})N_w. \tag{23}$$

As $f(N_w, 0_{\mathbb{R}^5})$ is linear, then N_w^0 is globally stable. Then, (H_1) is satisfied. Let

$$A = \begin{pmatrix} -k_w^N + \alpha_N & \alpha_N + \alpha_{p2} & 0 & 0 & 0 \\ \gamma & -k_b^N & 0 & 0 & 0 \\ \alpha_2 & \alpha_3 & -k_D & 0 & 0 \\ \beta_2 & \beta_3 & 0 & -k_H & 0 \\ 0 & 0 & \eta_{DH} & \eta_{HD} & -k_C \end{pmatrix}, \tag{24}$$

$$I = (O_w, O_b, D, H, C), \tag{25}$$

$$g^*(N_w, I) = AI^T - g(N_w, I), \tag{26}$$

$$g^*(N_w, I) = \begin{pmatrix} g_1^*(N_w, I) \\ g_2^*(N_w, I) \\ g_3^*(N_w, I) \\ g_4^*(N_w, I) \\ g_5^*(N_w, I) \end{pmatrix} = \begin{pmatrix} \alpha_N(O_w + O_b)\left(1 - \dfrac{N_w}{N_T}\right) + \alpha_{p2} O_b \left(1 - \dfrac{N_w}{N_T}\right) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}. \tag{27}$$

Since $\dfrac{N_w}{N_T} \leq 1$ then $1 - \dfrac{N_w}{N_T} \geq 0$. Thus $g^*(N_w, I) \geq 0$ for all $(N_w, I) \in \Omega$. Consequently, E_0 is a globally asymptotically stable point. ∎

Analogous proofs can be found in the bibliographical references [12], [27], [44].

An important question is: how does the joint variation of the parameters p_{s1} and p_{s2} impact the basic reproduction numbers? To answer this question we will use the limits and find the expressions that define the different variations of p_{s1} and p_{s2}. We have:

$$\lim_{(p_{s1}, p_{s2}) \to (1,0)} \Re_0^N = \lim_{(p_{s1}, p_{s2}) \to (0,1)} \Re_0^N = \frac{\alpha_N \Lambda (k_b^N + \gamma)}{(1 + \alpha_1 + \beta_1 + \mu) k_b^N k_w^N N_T - \alpha_{p2} \gamma \Lambda}, \tag{28}$$

$$\lim_{(p_{s1}, p_{s2}) \to (0,0)} \Re_0^N = \frac{\alpha_{p1} \Lambda (k_b^{p1} + \gamma)}{(\alpha_1 + \beta_1 + \mu) k_b^N k_w^N N_T - \alpha_{p2} \gamma \Lambda}, \tag{29}$$

$$\lim_{(p_{s1}, p_{s2}) \to (1,1)} \Re_0^N = \frac{\alpha_N \Lambda (k_b^N + \gamma)}{(2 + \alpha_1 + \beta_1 + \mu) k_b^N k_w^N N_T - \alpha_{p2} \gamma \Lambda}. \tag{30}$$

A new question arises: what conditions must these variations of p_{s1} and p_{s2} meet to have a positive impact on the basic reproduction numbers? In the case of $\Re_0^N$, for the impact to be positive, given that it refers to negative interactions, it must be less than unity, and in the case of $\Re_0^{p1}$ and $\Re_0^{p2}$, it must be greater than unity, since they are defined for positive interactions. The following results guarantee these conditions.

Lemma 3.4. *The expressions (28)-(30) are less than one when:*

$$\frac{\Lambda}{N_T} < \frac{(1+\alpha_1+\beta_1+\mu)k_b^N k_w^N}{\alpha_N(k_b^N+\gamma)-\alpha_{p2}\gamma}, \tag{31}$$

$$\frac{\Lambda}{N_T} < \frac{(\alpha_1+\beta_1+\mu)k_b^N k_w^N}{\alpha_N(k_b^N+\gamma)-\alpha_{p2}\gamma}, \tag{32}$$

$$\frac{\Lambda}{N_T} < \frac{(2+\alpha_1+\beta_1+\mu)k_b^N k_w^N}{\alpha_N(k_b^N+\gamma)-\alpha_{p2}\gamma}. \tag{33}$$

The previous Lemma shows conditions for $\Re_0^N < 1$ when the social factors that lead to overweight (p_{s1}) and hypertension (p_{s2}) tend to their lowest or highest values.

$$\lim_{(p_{s1},p_{s2})\to(1,0)} \Re_0^{p1} = \lim_{(p_{s1},p_{s2})\to(0,1)} \Re_0^{p1} = \frac{\alpha_{p1}k_b^N\Lambda}{\Lambda(\alpha_{p2}\gamma+\alpha_N(k_b^N+\gamma))-k_bk_wN_T(1+\alpha_1+\beta_1+\mu)}, \tag{34}$$

$$\lim_{(p_{s1},p_{s2})\to(0,0)} \Re_0^{p1} = \frac{\alpha_{p1}k_b^N\Lambda}{\Lambda(\alpha_{p2}\gamma+\alpha_N(k_b^N+\gamma))-k_bk_wN_T(\alpha_1+\beta_1+\mu)}, \tag{35}$$

$$\lim_{(p_{s1},p_{s2})\to(1,1)} \Re_0^{p1} = \frac{\alpha_{p1}k_b^N\Lambda}{\Lambda(\alpha_{p2}\gamma+\alpha_N(k_b^N+\gamma))-k_bk_wN_T(2+\alpha_1+\beta_1+\mu)}. \tag{36}$$

The following Lemma shows conditions for $\Re_0^{p1} > 1$, in the limits. These would imply positive interactions between individuals with normal weight and overweight.

Lemma 3.5. *The expressions (34)-(36) are greater than one when:*

$$\frac{\Lambda}{N_T} > \frac{-k_b^N k_w^{p1}(1+\alpha_1+\beta_1+\mu)}{\alpha_{p1}k_b^N-\alpha_{p2}\gamma-\alpha_N(k_b^N+\gamma)}, \tag{37}$$

$$\frac{\Lambda}{N_T} > \frac{-k_b^N k_w^{p1}(\alpha_1+\beta_1+\mu)}{\alpha_{p1}k_b^N-\alpha_{p2}\gamma-\alpha_N(k_b^N+\gamma)}, \tag{38}$$

$$\frac{\Lambda}{N_T} > \frac{-k_b^N k_w^{p1}(2+\alpha_1+\beta_1+\mu)}{\alpha_{p1}k_b^N-\alpha_{p2}\gamma-\alpha_N(k_b^N+\gamma)}. \tag{39}$$

The different joint behaviors of p_{s1} and p_{s2} in $\Re_0^{p2}$ using the limit definition are:

$$\lim_{(p_{s1},p_{s2})\to(1,0)} \Re_0^{p2} = \lim_{(p_{s1},p_{s2})\to(0,1)} \Re_0^{p2} =$$
$$\frac{\alpha_{p2}\Lambda(\alpha_N\Lambda+(1+\alpha_1+\beta_1+\mu))N_T(\gamma-k_w^N)}{N_T(1+\alpha_1+\beta_1+\mu)(k_b^{p2}k_w^NN_T(1+\alpha_1+\beta_1+\mu)-\alpha_N(k_b^{p2}+\gamma)\Lambda)}, \tag{40}$$

$$\lim_{(p_{s1},p_{s2})\to(0,0)} \Re_0^{p2} = \frac{\alpha_{p2}\Lambda(\alpha_N\Lambda+(\alpha_1+\beta_1+\mu))N_T(\gamma-k_w^N)}{N_T(1+\alpha_1+\beta_1+\mu)(k_b^{p2}k_w^NN_T(\alpha_1+\beta_1+\mu)-\alpha_N(k_b^{p2}+\gamma)\Lambda)}, \tag{41}$$

$$\lim_{(p_{s1},p_{s2})\to(1,1)} \Re_0^{p2} = \frac{\alpha_{p2}\Lambda(\alpha_N\Lambda+(2+\alpha_1+\beta_1+\mu))N_T(\gamma-k_w^N)}{N_T(2+\alpha_1+\beta_1+\mu)(k_b^{p2}k_w^NN_T(1+\alpha_1+\beta_1+\mu)-\alpha_N(k_b^{p2}+\gamma)\Lambda)}. \tag{42}$$

As above, the following Lemma provides conditions for $\Re_0^{p2} > 1$, in the limits, implying positive interactions between individuals with normal weight and obese.

Lemma 3.6. *The expressions (40)-(42) are less than one when:*

$$\frac{\Lambda}{N_T} > \frac{(1+\alpha_1+\beta_1+\mu)(k_b^{p2}k_w^N N_T(1+\alpha_1+\beta_1+\mu)-\alpha_N(k_b^{p2}+\gamma)\Lambda)}{\alpha_{p2}(\alpha_N\Lambda+(1+\alpha_1+\beta_1+\mu))N_T(\gamma-k_w^N)}, \tag{43}$$

$$\frac{\Lambda}{N_T} > \frac{(1+\alpha_1+\beta_1+\mu)(k_b^{p2}k_w^N N_T(1+\alpha_1+\beta_1+\mu)-\alpha_N(k_b^{p2}+\gamma)\Lambda)}{\alpha_{p2}(\alpha_N\Lambda+(1+\alpha_1+\beta_1+\mu))N_T(\gamma-k_w^N)}, \tag{44}$$

$$\frac{\Lambda}{N_T} > \frac{(1+\alpha_1+\beta_1+\mu)(k_b^{p2}k_w^N N_T(1+\alpha_1+\beta_1+\mu)-\alpha_N(k_b^{p2}+\gamma)\Lambda)}{\alpha_{p2}(\alpha_N\Lambda+(1+\alpha_1+\beta_1+\mu))N_T(\gamma-k_w^N)}. \tag{45}$$

Lemmas 3.4-3.6 are obtained directly by comparing expressions (28)-(42) with respect to unity depending on the basic reproduction number ($\Re_0^N < 1$, $\Re_0^{p1} > 1$ and $\Re_0^{p2} > 1$).

3.3. Sensitivity Indices Analysis

In this subsection, we present the study of the sensitivity indices with respect to the basic reproduction number. This allows us to characterize the impact of the parameters of interest associated with social factors and the rates of negative and positive effective contact on the basic reproduction number.

The sensitivity analysis of the basic reproduction number determines the relative importance of the parameters present in the basic reproduction number, such as the parameters of transmission, resistance, recovery, among others. The sensitivity index can be defined using the partial derivatives, provided that the variable is differentiable with respect to the parameter under study. Sensitivity analysis also helps to identify the vitality of the parameter values in the predictions using the model [45], [46].

Definition 1. *([46]) The normalized forward sensitivity index of a variable v that depends differentiably on a parameter p is defined as:*

$$\Upsilon_p^v := \frac{\partial v}{\partial p} \times \frac{p}{v}. \tag{46}$$

The sensitivity index of $\Re_0$ helps to determine the parameters that have an impact on it.

We can characterize the sensitivity index as follows:

- A positive value of the sensitivity index implies that an increase of the parameter value causes an increase of the basic reproduction number.
- A negative value of the sensitivity index implies that an increase of the parameter value causes a decrease of the basic reproduction number.

Furthermore, a highly sensitive parameter must be estimated carefully, since a small variation in it will cause large quantitative changes [45].

For the parameters associated with the impact of social factors on overweight and hypertension, the sensitivity indices with respect to the basic reproduction numbers are:

$$\Upsilon_{\Re_0^N}^{p_{s1}} = -\frac{k_b^N k_w^N N_T p_{s1}}{k_b^N k_w^N N_T k_{nw} - \alpha_{p2}\Lambda\gamma}, \tag{47}$$

$$\Upsilon_{\Re_0^N}^{p_{s2}} = -\frac{k_b^N k_w^N N_T p_{s2}}{k_b^N k_w^N N_T k_{nw} - \alpha_{p2}\Lambda\gamma}, \tag{48}$$

$$\Upsilon_{\Re_0^{p1}}^{p_{s1}} = \frac{k_b^N k_w^N N_T p_{s1}}{\Lambda(\alpha_{p2}k_b^N + \alpha_N(k_b^N+\gamma)) - k_b^N k_w^N N_T k_{nw}}, \tag{49}$$

$$\Upsilon_{\Re_0^{p1}}^{p_{s2}} = \frac{k_b^N k_w^N N_T p_{s2}}{\Lambda(\alpha_{p2}k_b^N + \alpha_N(k_b^N+\gamma)) - k_b^N k_w^N N_T k_{nw}}, \tag{50}$$

$$\Upsilon_{\Re_0^{p2}}^{p_{s1}} = p_{s1}\left(\frac{\alpha_N\Lambda}{k_{nw}\big((k_w^N-\gamma)N_T k_{nw} - \alpha_N\Lambda\big)} - \frac{k_b^N k_w^N N_T}{k_b^N k_w^N N_T k_{nw} - \alpha_N(k_b^N+\gamma)\Lambda}\right), \tag{51}$$

$$\Upsilon_{\Re_0^{p2}}^{p_{s2}} = p_{s2}\left(\frac{\alpha_N\Lambda}{k_{nw}\big((k_w^N-\gamma)N_T k_{nw} - \alpha_N\Lambda\big)} - \frac{k_b^{p2} k_w^N N_T}{k_b^N k_w^N N_T k_{nw} - \alpha_N(k_b^N+\gamma)\Lambda}\right). \tag{52}$$

From Equations (47)–(52), we obtain the following results.

Lemma 3.7. *Let $\Upsilon^{p_{s1}}_{\Re_0^N}$ and $\Upsilon^{p_{s2}}_{\Re_0^N}$ be the sensitivity indices associated with the impact of p_{s1} and p_{s2} on $\Re_0^N$, we have that:*

- *If $\dfrac{\Lambda}{N_T} > \dfrac{k_b^N k_w^N N_T k_{nw}}{\gamma\alpha_{p2}}$, then $\Upsilon^{p_{s1}}_{\Re_0^N} > 0$ and $\Upsilon^{p_{s2}}_{\Re_0^N} > 0$. This implies that an decrease in p_{s1} and p_{s2} causes a decrease in $\Re_0^N$.*

Proof: The numerator of (47) is negative by definition. For the denominator to be negative, the following must be true:

$$k_b^N k_w^N N_T k_{nw} - \alpha_{p2}\gamma\Lambda < 0, \tag{53}$$

$$\Rightarrow \frac{\Lambda}{N_T} > \frac{k_b^N k_w^N k_{nw}}{\alpha_{p2}\gamma}. \tag{54}$$

So if Inequality (54) holds, we have that (47) is positive.

The second part of the Lemma, which refers to p_{s2}, is an analogous proof using (48), and the same condition is reached. ■

Lemma 3.8. *Let $\Upsilon^{p_{s1}}_{\Re_0^{p1}}$ and $\Upsilon^{p_{s2}}_{\Re_0^{p1}}$ be the sensitivity indices associated with the impact of p_{s1} and p_{s2} on $\Re_0^{p1}$, we have that:*

- *If $\dfrac{\Lambda}{N_T} < \dfrac{k_b^N k_w^{p1} N_T k_{nw}}{\alpha_{p2}k_b^N + \alpha_N(k_b^N + \gamma)}$, then $\Upsilon^{p_{s1}}_{\Re_0^{p1}} < 0$ and $\Upsilon^{p_{s2}}_{\Re_0^{p1}} < 0$. This implies that an decrease in p_{s1} and p_{s2} causes a increase in $\Re_0^{p1}$.*

Proof: By definition, the product $k_b^N k_w^N N_T p_{s1}$ is positive, so the sign of (49) will depend on the sign of the denominator. For the expression to be negative, the following must be true:

$$\Lambda(\alpha_N k_b^N + \alpha_{p2}\gamma) - k_b^N k_w^N N_T k_{nw} < 0, \tag{55}$$

$$\Rightarrow \frac{\Lambda}{N_T} < \frac{k_b^N k_w^N k_{nw}}{\alpha_N k_b^N + \alpha_{p2}\gamma}. \tag{56}$$

Then, for (49) to be negative, Inequality (56) must be fulfilled. Similarly, we proceed using (50) and obtain the same condition. ■

Lemma 3.9. *Let $\Upsilon^{p_{s1}}_{\Re_0^{p2}}$ and $\Upsilon^{p_{s2}}_{\Re_0^{p2}}$ be the sensitivity indices associated with the impact of p_{s1} and p_{s2} on $\Re_0^{p2}$, we have that:*

- *If $\dfrac{\Lambda}{N_T} < \dfrac{k_b^{p2} k_w^N k_{nw}((k_w^{p2} - \gamma)N_T k_{nw} - \alpha_N\Lambda)}{\alpha_N(k_w^{p2} N_T k_{nw} - \alpha_N(k_b^N + \gamma)\Lambda}$, then $\Upsilon^{p_{s1}}_{\Re_0^{p2}} < 0$ and $\Upsilon^{p_{s2}}_{\Re_0^{p2}} < 0$. This implies that an decrease in p_{s1} and p_{s2} causes a increase in $\Re_0^{p2}$.*

Proof: For (51) to be negative it is necessary that:

$$p_{s1}\left(\frac{\alpha_N\Lambda}{k_{nw}\big((k_w^N - \gamma)N_T k_{nw} - \alpha_N\Lambda\big)} - \frac{k_b^N k_w^N N_T}{k_b^N k_w^N N_T k_{nw} - \alpha_N(k_b^N + \gamma)\Lambda}\right) < 0. \tag{57}$$

By definition p_{s1} is positive, then the negativity of $\Upsilon^{p_{s1}}_{\Re_0^{p2}}$ is summarized as:

$$\left(\frac{\alpha_N\Lambda}{k_{nw}\big((k_w^N - \gamma)N_T k_{nw} - \alpha_N\Lambda\big)} - \frac{k_b^N k_w^N N_T}{k_b^N k_w^N N_T k_{nw} - \alpha_N(k_b^N + \gamma)\Lambda}\right) < 0, \tag{58}$$

$$\Rightarrow \frac{\alpha_N\Lambda}{k_{nw}\big((k_w^N - \gamma)N_T k_{nw} - \alpha_N\Lambda\big)} < \frac{k_b^N k_w^N N_T}{k_b^N k_w^N N_T k_{nw} - \alpha_N(k_b^N + \gamma)\Lambda}, \tag{59}$$

$$\Rightarrow \frac{\Lambda}{N_T} < \frac{k_b^N k_w^N k_{nw}\big((k_w^N - \gamma)N_T k_{nw} - \alpha_N\Lambda\big)}{\alpha_N\big(k_b^N k_w^N N_T k_{nw} - \alpha_N(k_b^N + \gamma)\Lambda\big)}. \tag{60}$$

The proof for $\Upsilon^{p_{s2}}_{\Re_0^{p_2}}$ is analogous and the same condition is reached. ■

4. NUMERICAL RESULTS

This section presents computational simulations of Model (4)-(9) to analyze compartment behavior. Parameter values are listed in Table 1 and the initial conditions are: $N_w(0) = 874140$, $O_w(0) = 1200$, $O_b(0) = 1500$, $D(0) = 10000$, $H(0) = 12000$, and $C(0) = 1500$. The study period spans 10 years, with parameters expressed in yearly units.

We first perform a global sensitivity analysis of all model parameters, followed by an assessment of the impact of social-factor parameters (p_{s1} and p_{s2}) on basic reproduction numbers and compartment dynamics. All code was implemented in MATLAB R2024b, using the `ode45` solver for the system of ordinary differential equations (4)-(9).

4.1. Global Sensitivity Analysis

Global sensitivity analysis (GSA) evaluates how model outputs depend on input parameters [47]. Unlike local methods, GSA explores screening and variance decomposition to overcome their limitations. We employ Sobol' indices for GSA, a widely used approach in biological and epidemiological modeling [48], [49]. Sobol' indices quantify parameter influence at different orders: First-order indices measure the individual contribution of each parameter to output variance, ignoring interactions. Total-order indices capture both individual effects and all higher-order interactions involving the parameter. In this work, we study the first-order and total-order indices of the model parameters.

To calculate the Sobol' indices, we need the underlying variances and use a surrogate-model-based approach based on Polynomial Chaos Expansion (PCE). The PCE of the computational model response is a sum of orthogonal polynomials weighted by coefficients to be determined [50], [51]. Note that the quality of the PCE is directly dependent on the number of terms in the expansion. The family of orthonormal polynomials to be used is chosen according to the input distribution of the model, where the aim is to minimize the number of terms needed in the expansion to build a good computational representation of the model [49], [52].

There are several methods for calculating the coefficients of the polynomial chaos expansion for a given basis. Specifically, we will use the following methods:
Method I: Ordinary Least Squares (OLS). The main advantage of the least squares minimization method over the projection method is that an arbitrary number of points can be used to calculate the coefficients, as long as they are a representative sample of the random input vector. Theory, errors, methodology, and examples for this method can be found in [49], [53]. Method II: Least Angle Regression (LARS). A complementary strategy to favor high-dimensional sparsity is to directly modify the least-squares minimization problem. The LARS algorithm used in this work, together with its theoretical formulation, code, and examples, can be found in [54], [55]. In our method, we take into account a stopping criterion that prevents adding regressors after the error estimate is above its minimum value for at least 10% of the maximum number of possible iterations. For each method, we will experiment with different sampling techniques, comprising stochastic sampling methods and deterministic low-discrepancy sequences; Monte Carlo (MC), Latin hypercube sampling (LHS), Sobol' sequence sampling (Sobol'), and Halton sequence sampling (Halton) [48], [56], [57], [58].

Building a good surrogate model requires a rigorous validation process for the obtained response. Using a good error metric is essential to characterizing a good approximation. In this work, we use to estimate the surrogate error estimation the Leave-One-Out (ϵ_{LOO}) cross-validation error [49]. We used the UQLab library [59], [60], [61], as well as the code presented in [49]. We considered years as the time unit and the maximum time period 10 years.

We use independent uniform random variables for each parameter as a probabilistic input model [13], [49], with upper and lower bounds of $\pm 1.5\%$ dispersion around the mean, and consider the fixed nominal values presented in Table 1. To build the PCE surrogate model, 2000 samples of experimental design were taken for all the studied techniques with a maximum polynomial degree of 18 and the surrogate error estimation the Leave-One-Out (ϵ_{LOO}) error in all cases was of the order 10^{-8} so the quality of the surrogate approximation proved to be reasonable for the purpose of its use.

Figures 2-5 show first- and total-order Sobol' indices computed using OLS and LARS across multiple sampling techniques. Results were consistent across methods. The most influential parameters are α_N, p_{s1} and p_{s2}, with α_N exhibiting the highest Sobol' index. This indicates that the negative effective contact rate

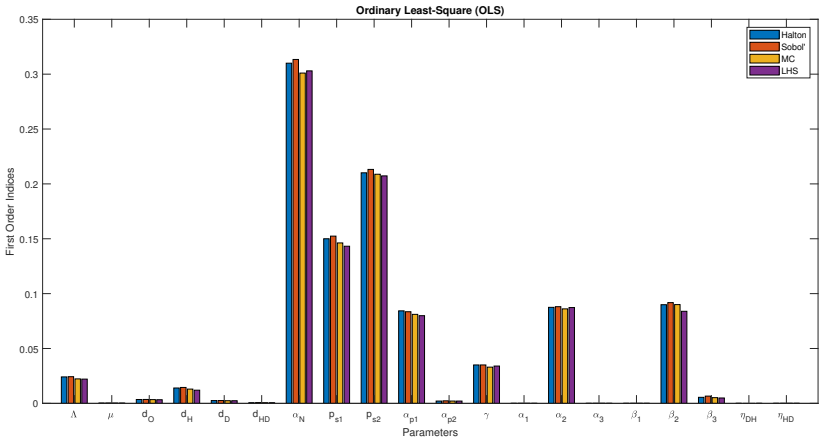


Figure 2: First order Sobol' indices using OLS with different sampling methods for the parameters of Model (4)-(9).

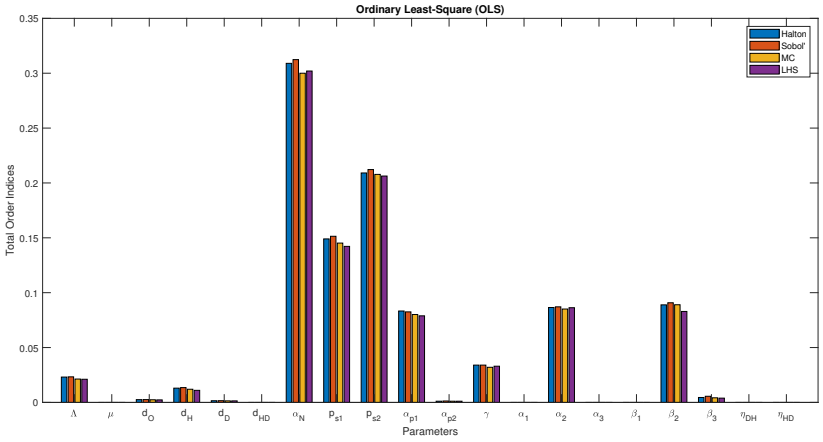


Figure 3: Total order Sobol' indices using OLS with different sampling methods for the parameters of Model (4)-(9).

(α_N) exerts greater influence than positive contact rates (α_{p1} and α_{p2}). Among social factors, (p_{s2}), related to hypertension, has a stronger impact than (p_{s1}), weight gain, suggesting that social determinants more strongly affect hypertension than overweight. These parameters have an index of order 10^{-1}.

In the following order are the parameters β_2, α_2, α_{p1}, γ, Λ and d_H which have values in order 10^{-2}. We have that the parameter associated with developing hypertension due to being overweight (β_2) exceeds in all the techniques used and with an average of 0.0918 the parameter associated with developing diabetes due to being overweight (α_2) which had an average of 0.0875.

Following these, we have the parameters with values in the order 10^{-3} in the following order, β_3, d_O, d_D and α_{p2}. We have that the death rate associated with obesity (d_O) has a greater influence than the death rate associated with diabetes (d_D), but the one with the greatest influence is the one associated with hypertension (d_H), which is important information for decision-making and control of these deaths. We also have that the parameter associated with developing hypertension being overweight (β_2) has a greater influence than this same situation but being obese (β_3), this can mean that in many cases being overweight is already a triggering factor for developing hypertension without the need to become obese, which is an element to be

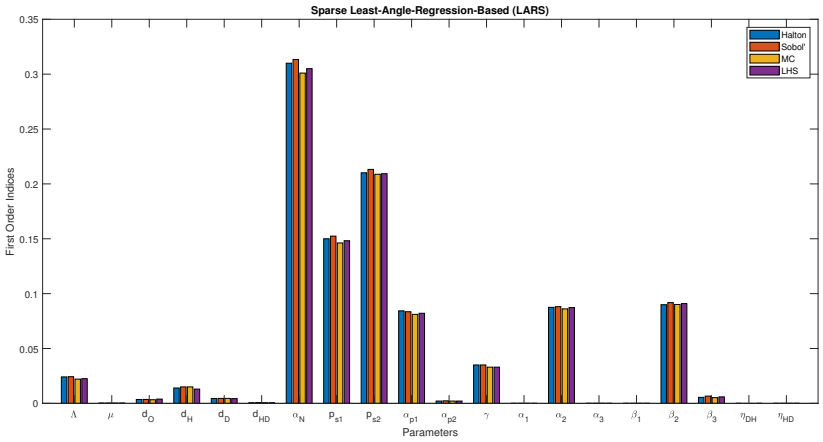


Figure 4: First order Sobol' indices using LARS with different sampling methods for the parameters of Model (4)-(9).

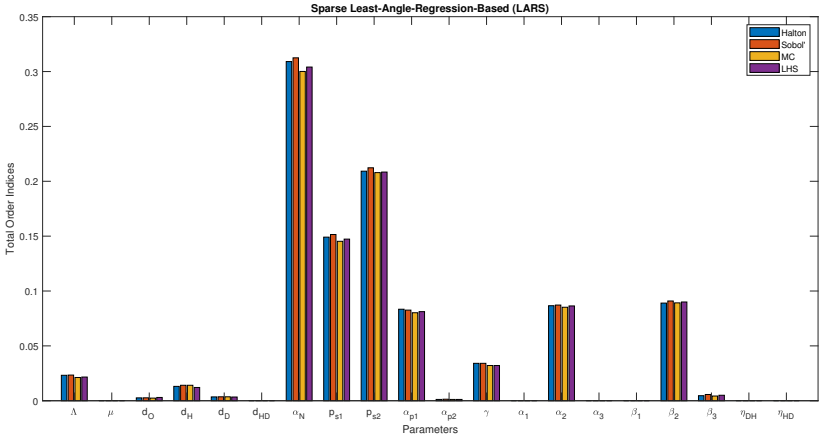
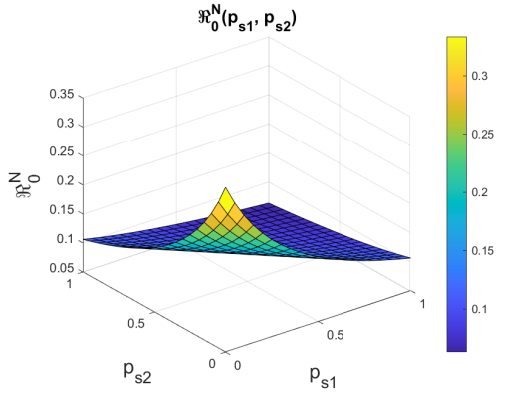


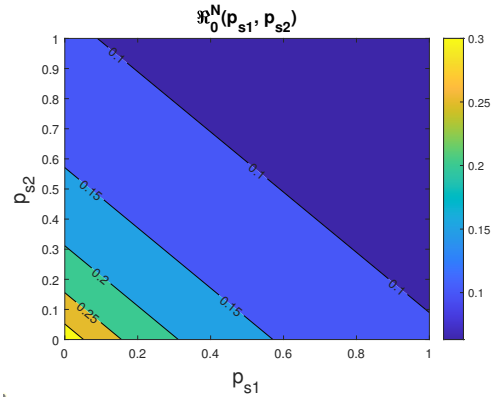
Figure 5: Total order Sobol' indices using LARS with different sampling methods for the parameters of Model (4)-(9).

taken into account for future proposals for controlling obesity and hypertension. An important factor is that in the positive rates of effective contact, the positive relationship between an overweight and a normal weight individual (α_{p1}) has greater influence, compared to the positive relationship between a normal weight and an obese individual (α_{p2}). When we refer to a positive relationship, we are referring to the motivation to improve one's lifestyle and other elements causing that individual to lose body weight. But we also have that since γ has a greater influence than α_{p2}, the awareness of an obese individual with which they improve their lifestyle and manage to lose weight and become overweight is more influential than the positive interactions between obese and normal weight individuals.

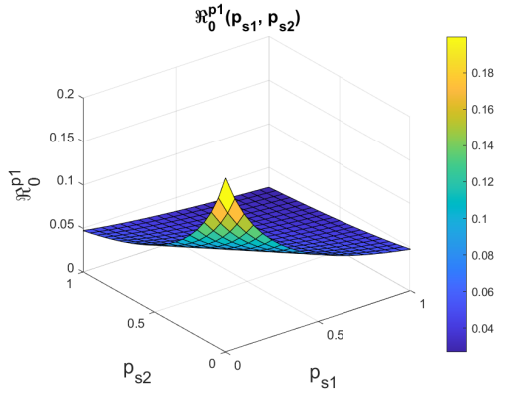
The other parameters have Sobol' indices in the order 10^{-n} with $n \geq 3$, so we assume that they do not have a considerable influence on Model (4)-(9). The information provided in the global sensitivity study can be used in parameter estimation, as can the medical/epidemiological information obtained from the order and relationship of parameters.



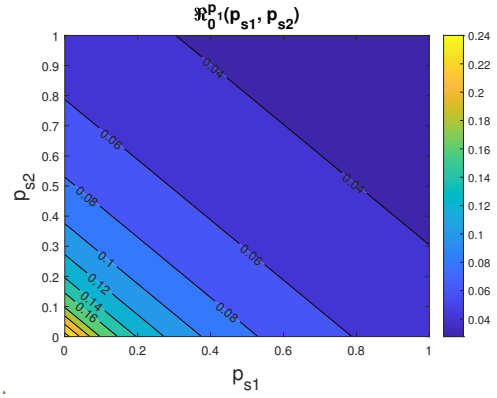
(a) Behavior of $\Re_0^N$ when p_{s1} and p_{s2} are varying in the closed unity interval.



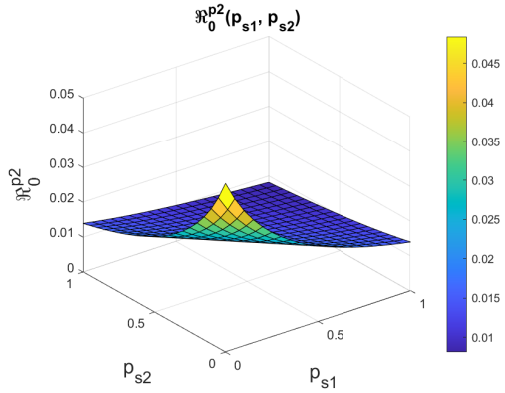
(b) Contour of the surface of figure 6(a)



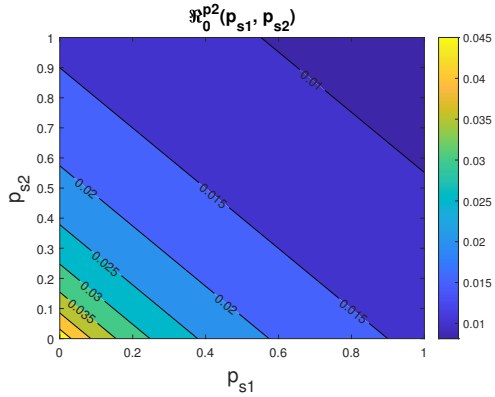
(c) Behavior of $\Re_0^{p1}$ when p_{s1} and p_{s2} are varying in the closed unity interval.



(d) Contour of the surface of figure 6(c)



(e) Behavior of $\Re_0^{p2}$ when p_{s1} and p_{s2} are varying in the closed unity interval.



(f) Contour of the surface of figure 6(e)

Figure 6: Joint variation of the parameters p_{s1} and p_{s2} in the $\Re_0^N$, $\Re_0^{p1}$ and $\Re_0^{p2}$.

4.2. Basic Reproduction Number Study

The sensitivity indices associated with p_{s1} and p_{s2} and with respect to the basic reproduction numbers using expressions (47)-(52) are less than zero, $\Upsilon^{p_{s1}}_{\Re^N_0} = -0.2932$, $\Upsilon^{p_{s2}}_{\Re^N_0} = -0.1398$, $\Upsilon^{p_{s1}}_{\Re^{p1}_0} = -0.3672$, $\Upsilon^{p_{s2}}_{\Re^{p1}_0} = -0.1748$, $\Upsilon^{p_{s1}}_{\Re^{p2}_0} = -0.2936$ and $\Upsilon^{p_{s2}}_{\Re^{p2}_0} = -0.1040$. This means that a decrease of p_{s1} and p_{s2} causes an increase of $\Re^N_0$, $\Re^{p1}_0$ and $\Re^{p2}_0$. These parameters define the impact of social factors on overweight and hypertension. A decrease in their effect in the case of positive interactions has a positive impact because $\Re^{p1}_0$ and $\Re^{p2}_0$ increase, indicating that these interactions are having a significant effect. However, in the case of negative interactions, a decrease in these parameters causes $\Re^N_0$ to increase, and therefore, the impact of negative interactions increases. Interpreting an increase in p_{s1} and p_{s2} is epidemiologically inconsistent, as adverse social determinants —including low education, socioeconomic inequality, and obesogenic environments— consistently act as risk factors rather than protective factors. Therefore, assuming that these parameters could increase contradicts well-established empirical evidence showing that poorer social conditions raise the risk of obesity and hypertension [62], [63], [64].

When we vary p_{s1} and p_{s2} jointly, we observe that all the basic reproduction numbers are less than unity so this variation does not negatively impact $\Re^N_0$ but in the case of $\Re^{p1}_0$ and $\Re^{p2}_0$ it does, see Figures 6(a)-6(f). In all cases the maximum value is reached when both parameters tend to zero, i.e. when the effect of social factors disappears, which means these parameters have a positive effect on $\Re^{p1}_0$ and $\Re^{p2}_0$.

When we studied the basic reproduction numbers defined for interactions with negative and positive effects, we obtained information on the parameters associated with the effect of social factors on overweight and hypertension, since the impact on the dynamics is not the same. In the case of the dynamics based on negative interactions, the growth of these parameters independently or jointly maintained the $\Re^N_0$ lower than unity, but it is a contradictory effect because if p_{s2} increases, for example, hypertension cases also increase. Moreover, in the dynamics of positive interactions based on the study of the sensitivity indices that were negative, a decrease of these parameters, i.e. reducing the impact of social factors increased the $\Re^{p1}_0$ and $\Re^{p2}_0$, which is a necessary behavior to reduce overweight and obesity in the community. Analysis of both negative and positive interactions highlights the roles of these parameters, as well as others, in shaping the dynamics of the system.

4.3. Model Simulations

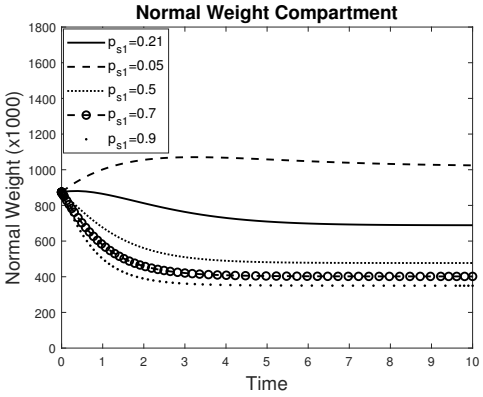
Now, we are going to study the impact of rates associated with social factors on overweight (a) and blood pressure and its consequences on hypertension (b) in the compartments. The purpose is to simulate different values of these parameters and obtain the behavior in the different compartments. For p_{s1}, we will study the values $0.05, 0.21, 0.5, 0.7, 0.9$, and for p_{s2}, $0.05, 0.1, 0.5, 0.7, 0.9$. These values were chosen at random to obtain information on the behavior of the compartments.

While studying the compartment of individuals with normal weight for different values of p_{s1} we have different asymptotic behaviors, for p_{s1} values of 0.05, and 0.21, it first grows, with the growth being larger for the smaller value, and then stabilizes and for values greater than 0.21 studied decreases and then stabilizes, with this decrease being stronger for larger values of p_{s1}, see Figure 7(a).

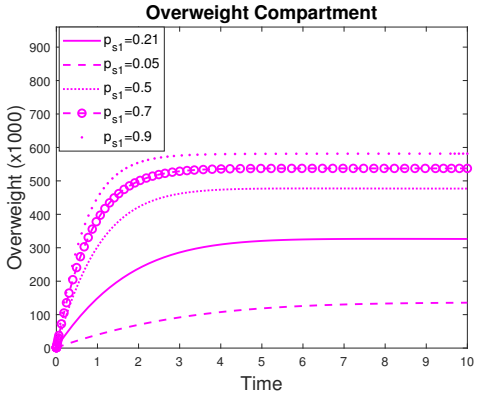
Since the asymptotic behavior varies for different values of p_{s1} we decided to increase the number of values of the rate under study, see Figure 8. We can observe that for values of $p_{s1} < 0.3$, it first grows and then stabilizes and that the smaller the value of p_{s1}, the higher the growth and for values of $p_{s1} \geq 0.3$ it decreases and then stabilizes, with the decrease being greater for higher values of p_{s1}. But at the end of the study we obtain that for higher values of p_{s1} the number of individuals classified with normal weight is lower. This information is relevant because if we reduce the rate of the effect of the social factors that lead to overweight we manage to maintain a greater number of individuals with normal weight, which is the desired effect.

In the case of the overweight compartment, we observe that for higher values of p_{s1}, a larger number of overweight individuals is reported at the end of the study period. Moreover, for all values considered, an increase is observed at the beginning of the simulation, see Figure 7(b). This behavior is logical because the higher p_{s1}, the greater the number of individuals reaching overweight.

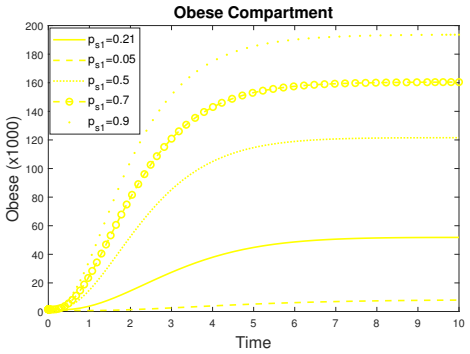
For the obese compartment at the beginning of the study for different values of p_{s1} we have a decrease that is more evident for lower values of p_{s1}, and then it grows and stabilizes being the highest reported values for



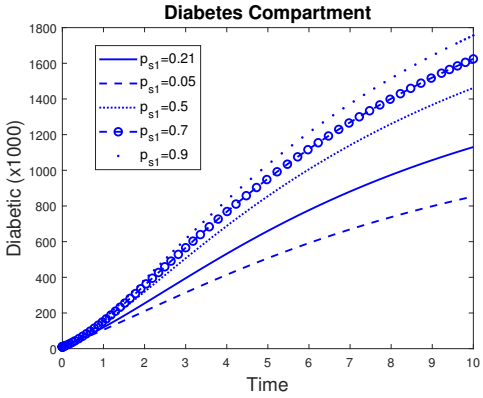
(a) Behavior of the Normal Weight (N_w) compartment over time for different values of p_{s1}.



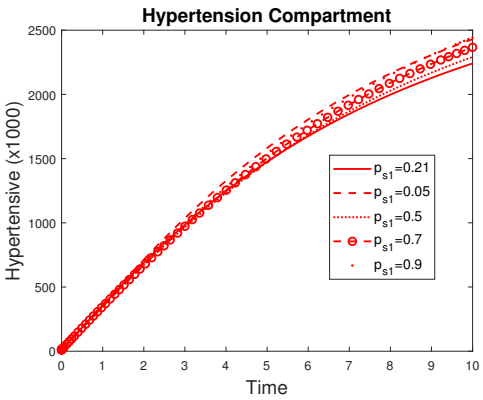
(b) Behavior of the Overweight (O_w) compartment over time for different values of p_{s1}.



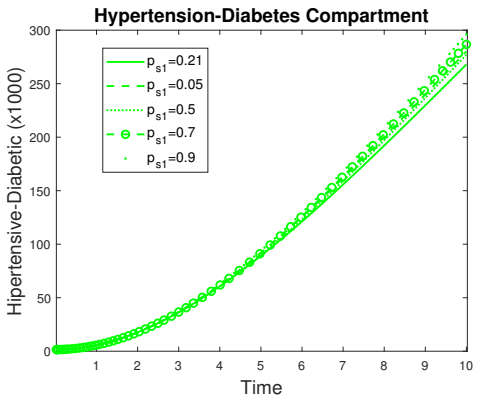
(c) Behavior of the Obese (O_b) compartment over time for different values of p_{s1}.



(d) Behavior of the Diabetes (D) compartment over time for different values of p_{s1}.



(e) Behavior of the Hypertesion (H) compartment over time for different values of p_{s1}.



(f) Behavior of the Diabetes-Hypertesion (C) compartment over time for different values of p_{s1}.

Figure 7: Numerical solution of the compartments of Model (4)-(9) for different values of p_{s1}, for a period of 10 years and with a scale of $\times 1000$ individuals.

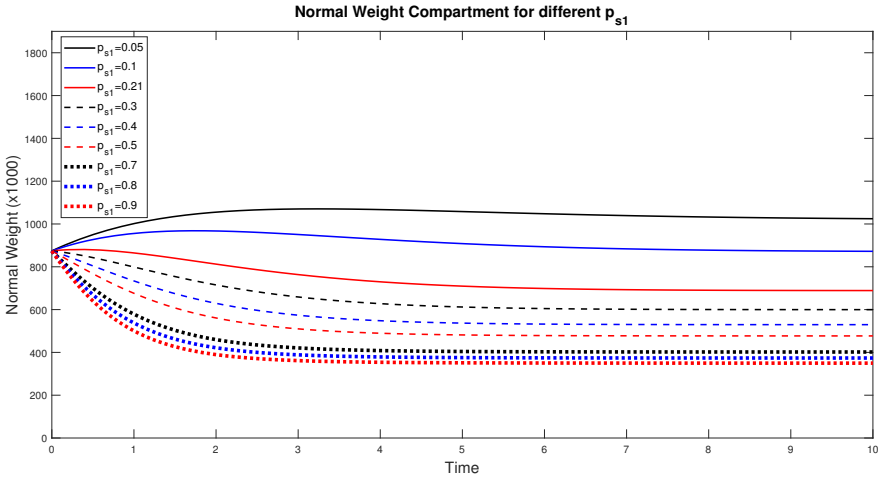


Figure 8: Behavior of the Normal Weight (N_w) compartment for different values of p_{s1}, in the interval $[0.05, 0.9]$ with the objective of observing the asymptotic behavior and delving into the results obtained in Figure 7(a).

higher values of p_{s1}, see Figure 7(c). Despite p_{s1} not having direct influence on the obese compartment, the results show that it has a significant impact on the behavior of the compartment. In the diabetes compartments throughout the study and the different values of p_{s1}, we have an increase, see Figure 7(d). This occurs despite p_{s1} not having direct influence on the diabetes compartment.

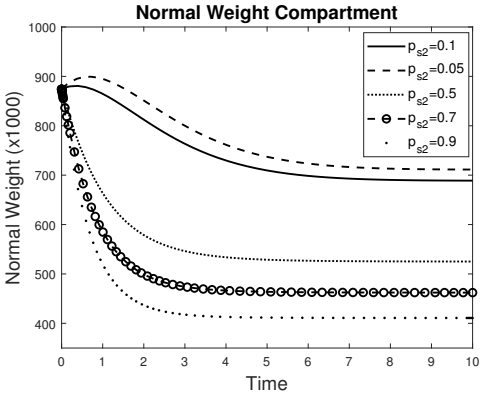
In the hypertension and hypertension-diabetes compartments, the behavior for different values of p_{s1} maintains a similar asymptotic behavior, and the differences between the reported cases do not present significant differences; see Figures 7(e) and 7(f). Therefore, the impact of the variation of the parameter p_{s1} on these behaviors has no significant effect.

We conclude from this study that the results provide relevant information that helps us understand how social factors leading to obesity directly affect the model's dynamics. A decrease in p_{s1} helps maintain and increase the number of individuals with normal weight, reduces the number of overweight individuals, and, despite not directly affecting the diabetic compartment, its growth contributes to the increase in diabetic diagnoses.

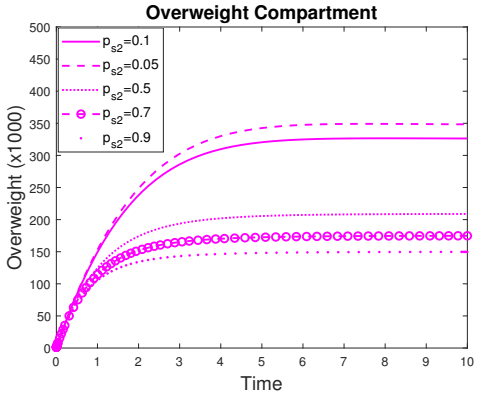
In the study of the normal weight compartment for different values of p_{s2}, we observe that for initially low values of p_{s2}, there is an increase that later stabilizes in the number of normal weight individuals, see Figure 9(a). Since this parameter directly impacts this compartment, we decided to simulate it for other values of p_{s2}, Figure 10. Thus, we identify that by reducing the value of p_{s2}, that is, the rate of social factors that lead to hypertension, we manage to maintain a greater number of normal weight individuals.

In the overweight and obesity compartments, we find that the higher the p_{s2}, the more cases of overweight and obesity are reported, and the asymptotic behavior is analogous to what occurs with p_{s1}, see Figures 9(b) and 9(c). However, p_{s1} has a greater impact since a greater number of overweight and obese individuals are reported compared to the variation in p_{s2}. In the case of diabetes, a similar long-term behavior is observed with respect to the variation of p_{s1}, see Figure 9(d). For the Hypertension and Hypertension-Diabetes compartments, it is observed that the higher the value of p_{s2}, the greater the number of individuals in these compartments, see Figures 9(e) and 9(f).

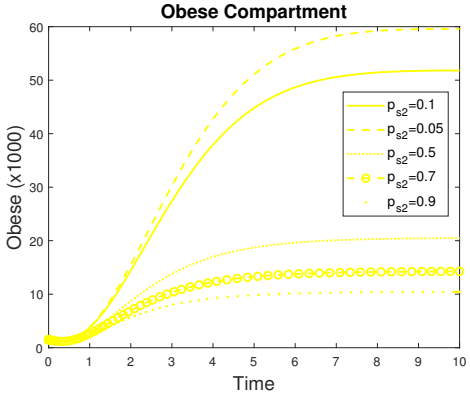
Using computational simulations of Model (4)-(9) with the parameter values in Table 1 and the initial conditions, we can conclude that the parameters p_{s1} and p_{s2} have a significant impact on the dynamics. Therefore, if we reduce the impact of social factors, we can reduce the number of individuals who are overweight and obese, as well as diabetic and hypertensive individuals. Interpreting the simulation results and basic reproduction numbers contributes to understanding how social factors affect the dynamics of overweight and obesity and can help develop control strategies.



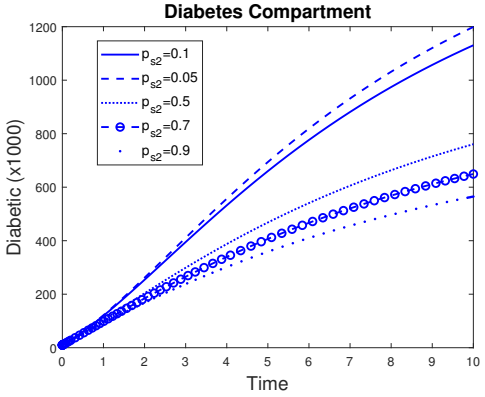
(a) Behavior of the Normal Weight (N_w) compartment over time



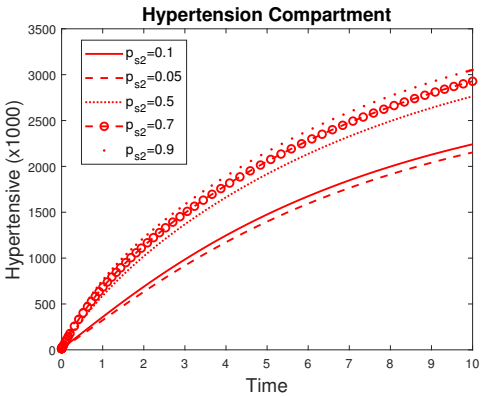
(b) Behavior of the Overweight (O_w) compartment over time



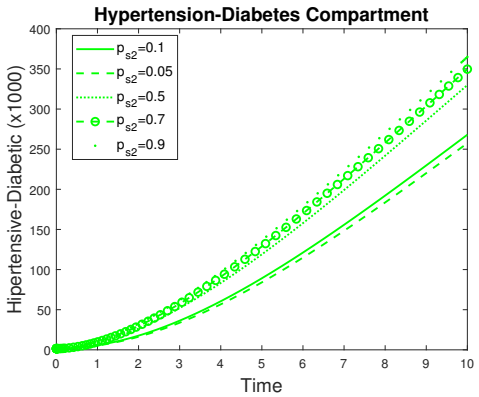
(c) Behavior of the Obese (O_b) compartment over time



(d) Behavior of the Diabetes (D) compartment over time



(e) Behavior of the Hypertesion (H) compartment over time



(f) Behavior of the Diabetes-Hypertesion (C) compartment over time

Figure 9: Numerical solution of the compartments of Model (4)-(9) for different values of p_{s2}, for a period of 10 years and with a scale of $\times 1000$ individuals.

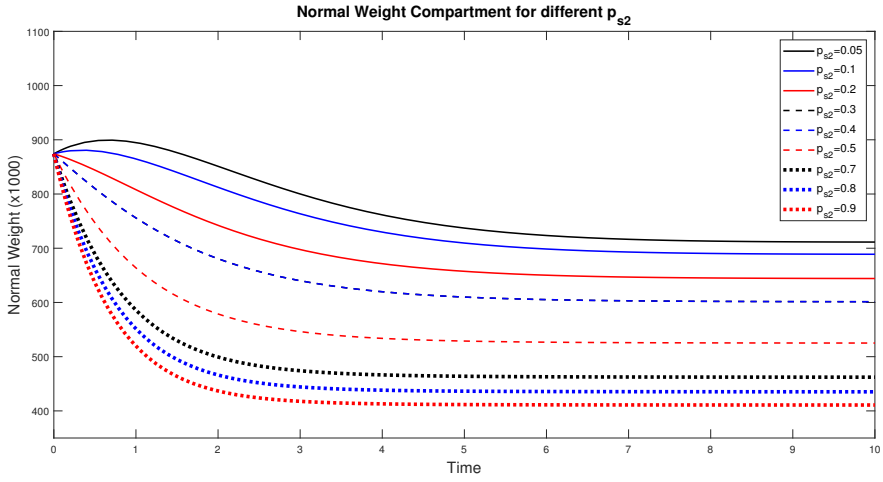


Figure 10: Behavior of the Normal Weight compartment (N_w) for different values of p_{s2} in the interval $[0.05, 0.9]$ with the objective of observing the asymptotic behavior and delving into the results obtained in Figure 9(a).

5. Conclusions

This paper introduced a novel mathematical model capturing the dynamics of overweight and obesity and their impact on diabetes and hypertension. The model incorporated both negative and positive interactions among normal weight, overweight, and obese individuals, as well as social factors influencing these conditions. Using the next-generation matrix method, we computed the basic reproduction number for negative interactions, representing the influence of overweight or obese individuals on normal weight individuals, and defined reproduction numbers for positive interactions, reflecting lifestyle improvements driven by normal weight individuals. We derived theoretical results characterizing the effect of social-factor parameters on reproduction numbers and performed sensitivity analyses using literature-based data. Our findings underscore the significant role of social determinants in shaping disease dynamics.

For the global sensitivity analysis, we used the Sobol' indices and, to obtain them, the chaos expansion polynomial. To obtain the polynomial coefficients, we employed two techniques: Ordinary Least-Squares (OLS) and Least Angle Regression (LARS) with different sampling methods: Markov Chain, Halton, Latin hypercube sampling, and Sobol'. We found that parameters associated with social factors and their influence on obesity and hypertension have high Sobol' indices and are among the most influential. This information is important for understanding the dynamics and for future work to estimate parameters.

The study of basic reproduction numbers focused on parameters associated with social factors. The sensitivity indices were negative, and we found that their increase causes a decrease in the basic reproduction numbers. However, they were always less than one, which for the dynamics of negative interactions is a positive effect, but not for positive dynamics. The joint variation of these parameters in the basic reproduction numbers was always less than one, and the greatest value was reached when both were equal to zero. Qualitatively, this is a positive effect for the dynamics of positive interactions and is significant because it represents a reduction in the impact of social factors on overweight and hypertension. These parameters were studied directly in the compartments, and among the results obtained, we found that lower values of the parameters manage to maintain a greater number of individuals with normal weight, reduce overweight and hypertensive problems, and that these parameters significantly affect the diabetic compartment despite not being directly linked. All the information provided by the study verifies the importance of these parameters in the dynamics and provides information for future control strategies to reduce overweight and obesity in the population.

The proposed model and study can be expanded and adapted to different population contexts, offering a significant contribution to public health and the strategic planning of health systems. By integrating social factors, this approach enables a more realistic quantification of the social and economic environment's

influence on obesity, diabetes, and hypertension, contributing to a more comprehensive understanding of these chronic diseases. The results derived from the model can serve as a valuable tool to support public policy development, prioritize preventive interventions, and optimize the allocation of healthcare resources.

Furthermore, the proposed framework facilitates the design of control strategies aimed at preventing disease progression and reducing associated complications, recognizing that these conditions can have irreversible effects on individuals' health and place a heavy burden on healthcare systems. In future research, we plan to address the optimal control problem based on the proposed model, with the goal of identifying the most effective strategies to reduce overweight and obesity and mitigate their impact on diabetes and hypertension. In addition, multi–population studies are planned to analyze the behavior of these diseases according to variables such as sex, age, presence of comorbidities, socioeconomic status, and other social determinants of health, thereby strengthening the capacity of health systems to respond to these growing epidemiological challenges.

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