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Mathematical Model for the Study of Obesity in a Population and its Impact on the Growth of Diabetes

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Abstract. In this paper, we present a deterministic mathematical model for the study of overweight, and obesity in a population and its impact on the growth of the number of diabetics. For the construction of the model, we take into account social factors and the interactions between the elements of society. We find the basic reproduction number and prove the global stability of the disease-free equilibrium point. We present theoretical results and find the sensitivity indices to characterize the impact of parameters associated with overweight, obesity and diagnosed diabetes on the basic reproduction number. To validate the model, we perform computational simulations and study the basic reproduction number and compartments. We present the behavior of the compartments for a scenario and study the impact of the variation of parameters associated with overweight by social pressure and diabetes due to causes other than obesity.

Keywords: diabetes, obesity, overweight, mathematical model, ordinary differential equation.

AMS Subject Classification: 34A12; 92B05; 92D30.

1 Introduction

A large part of the world population lives in countries where overweight and obesity account [21] for more deaths than malnutrition. Current data reports that obesity has tripled since 1975 and in 2016, more than 1.9 billion adults were overweight and of those, more than 650 million were obese [21]. We defined the body mass index (BMI) as [20]: $BMI = \frac{\text{weight}}{\text{height}^2}$. Then, normal-weight individual is when $BMI \in [18.6, 24.9]$, overweight individual is when $BMI \in [25, 29.9]$, obese individual is when $BMI \in [30, 40]$ and in complicated situations over 40.

The root cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. Changes in diet and physical activity may be related to environmental and social changes associated with development and lack of supportive policies in sectors such as health, agriculture, transportation, urban planning, environment, food processing, distribution, marketing and education [21]. We know that overweight and obesity can be prevented.

There are two types of diabetes and the main difference between the two types of diabetes is that type 1 diabetes is a genetic disorder that often shows up early in life, and type 2 is largely diet-related and develops over time [1,16].

There is a relationship between obesity and type 2 diabetes. The likelihood and severity of type 2 diabetes are related to body mass index (BMI). The risk of diabetes is seven times higher in obese people than in those with a healthy weight, and the risk triples in overweight people [16,27].

Ejima et al. [13] presented a mathematical model of the genetic and non-genetic effects of obesity and showed that homozygous individuals are more susceptible to both the risk of social contagion and the risk of spontaneous weight gain. Kim and So-Yeun Kim [17] proposed a mathematical model for the dynamic of obesity with the presence of psychological and social factors. Paudel [22] proposed a SIR model for the dynamic of obesity in the southeaster region of the United States, and discussed the effect of social network on the spread of obesity among friends and family members. Wang [26] proposed a mathematical model to simulate the dynamic of social obesity by incorporating the structures of individual heterogeneity and overeating behaviors. Al-Tuwairqi and Matbouli [2] proposed two mathematical models to study the impact of fast food on obesity and the role that exercise plays in weight gain separately and showed that physical activity has a significant role in reducing weight. These works contributed to the construction of others models in particular, Bernard et al. [6] developed a deterministic compartmental model for the dynamic of obesity and explored the impact of the media on the propagation of this phenomenon in a constant population. In the modeling of diabetes, we find works such as the one of Dubey and Goswami [12] who presented a model of diabetes and its complications involving the fractional operator with exponential kernel. Moreover, Sandhya and Kumar [25] proposed a mathematical model for the study of diabetes mellitus with presence of plasma glucose concentration, generalized insulin and plasma insulin concentration. Ali et al. [3] adapted two mathematical models, one with β-cells and one without a β-cell component, to determine their ability to predict glucose concentration and determine the pathways of type 1 diabetes using published data on glucose concentration in four groups of experimental mice. Anusha and Athithan [4] presented a mathematical model of type 2 diabetes and divided the population into susceptible, unbalanced glucose level (IGL), treatment and restriction populations. Banzi et al. [5] presented a mathematical model of glucose-insulin dynamic for type 2 diabetes and allowed to investigate the behavior of glucose, insulin, glucagon, stored insulin and labile insulin in diabetics.

The aim of this paper is to present a mathematical model with ordinary differential equations that study overweight and obesity in a population and how it contributes to new cases of diabetes. This paper is organized as follows. In Section 2, we introduce the model and show the basic properties. In Section 3, we study the basic reproduction number. Section 4 is devoted to numerical experimentation. We finish the paper with some conclusions in Section 5.

2 Model formulation

Based on the body mass index, we define different compartments: normal-weight individuals, S, overweight individuals, O_w, obese individuals, O_b and diabetic individuals, D. It is important to highlight that there are individuals overweight or obese and it is due to the muscular weight due to sports. In these specific cases, we assume the weight status to be ideal and the definition of BMI will be centred on the healthy or unhealthy life of the individual. When the patient is diagnosed with diabetes, we assume that he improves his lifestyle to avoid the consequences of this disease, so we do not differentiate in body weight and there is no cure for diabetes.

Based on the influence that an overweight and obese person can have on a person who is currently of normal weight (i.e., is in compartment S) and increase his IBM, we define the transmission rate as:

$$\lambda_o = \alpha^*(O_w + \epsilon O_b)/N,$$

where α^* effective contact rate is defined as the contact between an individual from the O_w and O_b compartments and an individual from S which will exert a negative effect by changing the lifestyle of the individual from S and N is the total population. The modification parameter ϵ, i.e., it would be the influence of obese people, in this case we assume that it is not the same and that an obese person can influence negatively more than an overweight person. To find the value of this parameter a study can be made in the population with the overweight and obese and how they can influence a person who has normal weight. The rate M_S and M_D represent the recruitment rate of individuals with normal-weight and diabetes respectively, that not only take into account births but also other factors such as immigration. The rate β_1 represents the social pressure that leads an individual of S to an unhealthy lifestyle, this include excessive consumption of fast food, no physical exercise, sedentary lifestyle, stress, among others. In order to obtain a value for this parameter β_1, we must carry out a study in the population and in time, because the time to reach the overlap may vary from person to person. Let δ be the rate of overweight individuals who move into the S group (lowering the IBM), which may be related to a healthy lifestyle or disease. Analogously η will be individuals who go from obese to overweight. Moreover, γ rate is the rate of individuals who go from overweight to obese by continuing an unhealthy lifestyle. Parameters δ, η, γ can be obtained from population studies showing the evolution of overweight and obesity and in the reverse direction until a normal weight is achieved. The death rate from natural causes is μ and is the same from any compartment and we define d as the death rate associated with underweight diseases, mainly cardiac, and t_D represents the modification parameter associated with d for the obese state. The parameter t_D will contain the impact that obesity specifically has on this mortality with respect to overweight mortality. The parameters α_2, and α_3 represent the cases that become diabetics from the compartments that are linked to the weight of the individuals. The parameter α_1 is related to new cases of diabetics not related to weight, including genetic, racial, hereditary and other factors and μ_d is the death rate associated with diabetes. Parameter α_1 can be obtained from population-based studies showing the impact of diabetes where the causative factor is not related to body weight.

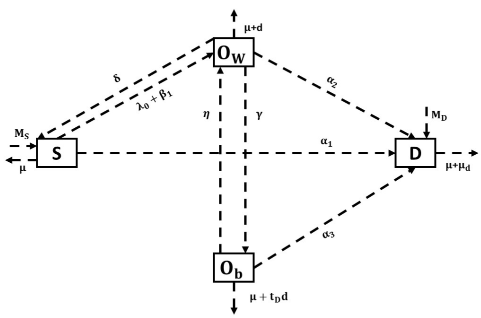


Figure 1. Flow diagram of model (2.1)

Figure 1 shows the dynamic diagram of the model and Table 1 the description of the variables and parameters of the model. This dynamic can be described by the following system of ordinary differential equations:

$$
\begin{aligned}
\frac{dS}{dt} &= M_S + \delta O_w - (\lambda_o + \mu + \alpha_1 + \beta_1)S, \\
\frac{dO_w}{dt} &= (\lambda_o + \beta_1)S + \eta O_b - (\gamma + \mu + d + \delta + \alpha_2)O_w, \\
\frac{dO_b}{dt} &= \gamma O_w - (\eta + \mu + t_D d + \alpha_3)O_b, \\
\frac{dD}{dt} &= M_D + \alpha_1 S + \alpha_2 O_w + \alpha_3 O_b - (\mu_d + \mu)D,
\end{aligned} \tag{2.1}
$$

with the initial conditions

$$S_0 > 0, \quad O_{w0} \geq 0, \quad O_{b0} \geq 0, \quad D_0 \geq 0.$$

Table 1. Description of the parameters used in the construction of model (2.1).

Param.	Description	Value	Reference
α_2	Development rate of diabetes in overweight people	0.35	[14, 15]
α_3	Development rate of diabetes in obese people	0.4	[14, 15]
M_S	Recruitment rate	667.685	[23]
M_D	Recruitment rate of diabetics	4.1	Assumed
α^*	Effective contact rate	2	Assumed
ϵ, t_D	Modification parameters	1.17, 1.02	Assumed
γ	Transition rate from overweight to obese	0.0015	[6]
η	Transition rate from obese to overweight	0.1	[6]
α_1	Rate of development of diabetes not associated with body weight	0.1	Assumed
β_1	Rate of social pressure leading to body weight gain	0.25	Assumed
μ	Natural death rate	1/70.5	[18]
d	Death rate associated with body weight	0.07	Assumed
μ_d	Death rate associated with diabetes	0.013	Assumed
δ	Transition rate from overweight to normal-weight	0.002	[6]

2.1 Positivity and boundedness of solutions

Now, let us prove the existence and non-negativity of solution of model (2.1) and find the biologically feasible region.

Existence and non-negativity of solutions

Theorem 1. *Let initial data be $S(0) = S_0 > 0, O_w(0) = O_{w0} \geq 0$, $O_b(0) = O_{b0} \geq 0$ and $D(0) = D_0 \geq 0$. Then, the solutions S, O_w, O_b and D of model (2.1) are positive for all $t > 0$. Furthermore,*

$$\lim_{t\to\infty} \sup N(t) \leq (M_S + M_D)/\mu.$$

Proof. The first equation of model (2.1) is

$$\frac{dS}{dt} = M_S + \delta O_w - (\mu + \alpha_1 + \beta_1 + \lambda_o)S.$$

Consequently,

$$\frac{dS}{dt} \geq M_S - (\mu + \alpha_1 + \beta_1 + \lambda_o)S. \tag{2.2}$$

The inequality (2.2) can be expressed as

$$\frac{d}{dt}\Big[S(t)\exp\Big\{(\mu+\alpha_1+\beta_1)t+\int_0^t\lambda_o(s)ds\Big\}\Big] \geq M_S\exp\Big\{(\mu+\alpha_1+\beta_1)t+\int_0^t\lambda_o(s)ds\Big\}.$$

Hence, for $t^*>0$,

$$S(t^*)\exp\Big\{(\mu+\alpha_1+\beta_1)t^*+\int_0^{t^*}\lambda_o(s)ds\Big\}-S(0) \geq \int_0^{t^*} M_S\exp\Big\{(\mu+\alpha_1+\beta_1)u+\int_0^u\lambda_o(w)dw\Big\}du.$$

So that,

$$S(t^*)\geq S(0)\exp\Big\{-\Big((\mu+\alpha_1+\beta_1)t^*+\int_0^{t^*}\lambda_o(s)ds\Big)\Big\}+\exp\Big\{-\Big((\mu+\alpha_1+\beta_1)t^* +\int_0^{t^*}\lambda_o(s)ds\Big)\Big\}\int_0^{t^*}M_S\exp\Big\{\Big((\mu+\alpha_1+\beta_1)u+\int_0^u\lambda_o(w)dw\Big)du\Big)\Big\}>0.$$

Similarly, it can be shown that $O_w(t)\geq 0$, $O_b(t)\geq 0$ and $D(t)\geq 0$ for all $t>0$. Moreover, we have

$$\frac{dN}{dt}=M_S+M_D-\mu N-\mu_d D-d(O_w+t_D O_b).$$

Then,

$$M_S+M_D-(\mu+d+\mu_d)N\leq\frac{dN}{dt}\leq M_S+M_D-\mu N,$$

which gives

$$\frac{M_S+M_D}{\mu+d+\mu_d}\leq\lim_{t\to\infty}\inf N(t)\leq\lim_{t\to\infty}\sup N(t)\leq(M_S+M_D)/\mu.$$

So, we have that

$$\lim_{t\to\infty}\sup N(t)\leq(M_S+M_D)/\mu.$$

□

The closed set $\Omega=\big\{(S,O_w,O_b,D)\in\mathbb{R}_+^4 : N(t)\leq(M_S+M_D)/\mu\big\}$ is the biologically feasible region for model (2.1).

Theorem 2. *The solutions of model (2.1) with non-negative initial conditions exists for all time.*

Proof. The right-hand side of model (2.1) is locally Lipschitz continuous, and this proves the local existence of the solution. The global existence of the solution follows from priori bound (see Theorem 1). □

3 Basic reproduction number

In a population composed only with susceptible individuals, the average number of infections caused by an infected individual is defined as basic reproduction number $\Re_0$. In our study, the susceptible compartment is composed of individuals who are currently at a normal weight and we define infected individuals as overweight and obese individuals, since an interaction with them may provoke an increase in IBM and S-compartment outflow. If $0 < \Re_0 < 1$ the infection will die out in the long run and if $\Re_0 > 1$ the infection will be able to spread in a population [10]. The higher the $\Re_0$ the more difficult it is to control the epidemic. The $\Re_0$ can be affected by several factors, such as the duration of infectivity of the affected patients, the infectivity of the organism and the degree of contact between the susceptible and infected populations.

The basic reproduction number study is focused on assessing the influence of a person with an unhealthy weight on a person with a healthy weight. Obesity in our study can lead to diabetes but diabetes is not curable, it is controllable. Our interest is to study the disease-free equilibrium point because of its relation with the basic reproduction number. The disease-free equilibrium point (DFE) is

$$\epsilon_0 = \big(M_S/(\mu + \alpha_1 + \beta_1), 0, 0, 0\big) \tag{3.1}$$

which is the point where we have only a number of individuals with normal weight. To find the basic reproduction number, we use the new generation matrix method presented in [9, 10, 11] where

$$F = \begin{pmatrix} \alpha^* M_S/Nk_{11} & \alpha^* \epsilon M_S/Nk_{11} \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} k_{12} & -\eta \\ -\gamma & k_{13} \end{pmatrix},$$

are the matrices of the term related to the new cases of overweight or obesity and transition terms, respectively. Then, for model (2.1) the basic reproduction number is:

$$\Re_0 = \rho(FV^{-1}) = \frac{\alpha^* M_S (k_{13} + \epsilon\gamma)}{Nk_{11}(k_{12}k_{13} - \eta\gamma)}, \tag{3.2}$$

where $k_{11} = \mu + \alpha_1 + \beta_1$, $k_{12} = \gamma + \mu + d + \alpha_2 + \delta$, $k_{13} = \eta + \mu + t_D d + \alpha_3$ and $k_{14} = \mu + \mu_D$. Now, we study the local and global stability of the infection-free equilibrium point when we do not have the effect of social pressure ($\beta_1 = 0$). This parameter will be studied for its effect on the basic reproduction number and in the different compartments.

The result presented below (Theorem 3) is obtained using Theorem 2 of [11].

Theorem 3. *The DFE (ϵ_0) of model (2.1), given by (3.1), is locally asymptotically stable (LAS) if $\Re_0 < 1$ and unstable if $R_0 > 1$.*

The threshold quantity $\Re_0$ is the basic reproduction number of the overweight and obese disease. It measures the average number of new disease generated by a single infectious agent in a fully normal-weight (susceptible) population. Consequently, the disease-free equilibrium of model (2.1) is locally asymptotically stable (LAS) whenever $\Re_0 < 1$ and unstable if $\Re_0 > 1$. This means that overweight and obesity can be removed from the community (when $\Re_0 < 1$) if the population sizes of model (2.1) are in the basin of attraction of the disease-free equilibrium ϵ_0.

Now, we prove the global stability of the disease-free equilibrium point. Following [7], we can rewrite model (2.1) as

$$\frac{dX}{dt} = f(X, I), \quad \frac{dI}{dt} = G(X, I), \quad G(X, 0_{\mathbb{R}^2}) = 0,$$

where $X \in \mathbb{R}^2_+$ is the vector with diabetics and normal-weight individuals and $I \in \mathbb{R}^2_+$ have the compartment related overweight and obese of model (2.1).

The disease-free equilibrium point is now denoted by $E_0 = (X_0, 0_{\mathbb{R}^2})$, where $X_0 = \big(M_S/(\mu + \alpha_1), 0\big)$. The conditions (H_1) and (H_2) below must be satisfied to guarantee the global asymptotic stability of E_0.

$$\begin{aligned}
&(H_1): \quad \text{For} \quad \frac{dX}{dt} = f(X, 0_{\mathbb{R}^2}), \quad X_0 \quad \text{is globally asymptotically stable,} \\
&(H_2): \quad G(X, I) = AI^T - G^*(X, I), \quad G^*(X, I) \geq 0, \quad \text{for} \quad (X, I) \in \Omega,
\end{aligned}$$

where $A = D_I G(X_0, 0_{\mathbb{R}^2})$ ($D_I G(X_0, 0_{\mathbb{R}^2})$ is the Jacobian of G at $(X_0, 0_{\mathbb{R}^2})$) is a M-matrix (the off-diagonal elements of A are non-negative) and Ω is the biologically feasible region.

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

Theorem 4. *The fix point E_0 is a globally asymptotically stable equilibrium (G.A.S) of model (2.1) provided that $\Re_0 < 1$ and that the conditions (H_1) and (H_2) are satisfied.*

Proof. Let $f(X, 0_{\mathbb{R}^2}) = \big(M_S - k_{11}S, M_D + \alpha_1 S\big)^T$. As $f(X, 0)$ is linear, then X_0 is globally stable. Then, (H_1) is satisfied. Let

$$\begin{aligned}
&A = \begin{pmatrix} -k_{12} + \alpha^* & \eta + \epsilon\alpha^* \\ \gamma & -k_{13} \end{pmatrix}, \\
&I = (O_w, O_b), \quad G^*(X, I) = AI^T - G(X, I), \\
&G^*(X, I) = \begin{pmatrix} G_1^*(X, I) \\ G_2^*(X, I) \end{pmatrix} = \begin{pmatrix} \alpha^*(O_w + \epsilon O_b)\big(1 - S/N\big) \\ 0 \end{pmatrix}.
\end{aligned}$$

Since $\frac{S}{N} \leq 1$, then $1 - \frac{S}{N} \geq 0$. Thus, $G^*(X, I) \geq 0$ for all $(X, I) \in \Omega$. Consequently, E_0 is a globally asymptotically stable point. □

Analogous proofs can be found in the bibliographical references [18, 23, 24].

We will study the joint influence of selected parameters on the basic reproduction number. The selected parameters are α_1, β_1, α_2, α_3, η, γ and δ which are associated with the transition between compartments and the diagnosis of diabetes. These parameters are defined in the interval $[0, 1]$ and we want to study the joint behavior when they lie at the extreme values of the interval. At the extremes of the interval are the critical behaviors due to which can represent all individuals or none and the interesting thing is to study this epidemiological situation together.

Using the threshold quantity $\Re_0$, in (3.2), we want to study the impact of parameters related to the diagnosis of diabetes in overweight (α_2) and obese (α_3) individuals on the dynamic of a population and find the conditions that characterize these effects. Now, we are going to study the possible combinations in the behavior of these parameters based on the limits. We have

$$\lim_{\substack{\alpha_2\to 1\\ \alpha_3\to 0}} \Re_0 = \frac{\alpha^* M_S\big(\eta+\mu+t_D d+\epsilon\gamma\big)}{Nk_{11}\big((\gamma+\mu+d+1+\delta)(\eta+\mu+t_D d)-\eta\gamma\big)}. \tag{3.3}$$

If the limit (3.3) is greater than unity, then when $\alpha_2 \to 1$ and $\alpha_3 \to 0$ it has a negative impact, that is if

$$\frac{\alpha^* M_S}{Nk_{11}} > \frac{(\gamma+\mu+d+1+\delta)(\eta+\mu+t_D d)-\eta\gamma}{\eta+\mu+t_D d+\epsilon\gamma}.$$

Now, we study the case when $\alpha_2 \to 0$ and $\alpha_3 \to 1$. We have

$$\lim_{\substack{\alpha_2\to 0\\ \alpha_3\to 1}} \Re_0 = \frac{\alpha^* M_S(\eta+\mu+t_D d+1+\epsilon\gamma)}{Nk_{11}\big((\gamma+\mu+d+\delta)(1+\eta+\mu+t_D d)-\eta\gamma\big)}. \tag{3.4}$$

Then, if the limit (3.4) is greater than unity, then when $\alpha_2 \to 0$ and $\alpha_3 \to 1$, that is when

$$\frac{\alpha^* M_S}{Nk_{11}} > \frac{(\gamma+\mu+d+\delta)(1+\eta+\mu+t_D d)-\eta\gamma}{\eta+\mu+d+1+\epsilon\gamma}.$$

In the case of $\alpha_1 \to 1$ and $\alpha_3 \to 1$, follows that

$$\lim_{\substack{\alpha_2\to 1\\ \alpha_3\to 1}} \Re_0 = \frac{\alpha^* M_S\big(\eta+\mu+t_D d+1+\epsilon\gamma\big)}{Nk_{11}\big((\gamma+\mu+d+1+\delta)(\eta+\mu+t_D d+1)-\gamma\eta\big)}. \tag{3.5}$$

If the limit (3.5) is greater than unity, then when $\alpha_2 \to 1$ and $\alpha_3 \to 1$ it has a negative impact, that is if

$$\frac{\alpha^* M_S}{Nk_{11}} > \frac{(\gamma+\mu+d+1+\delta)(\eta+\mu+t_D d+1)-\gamma\eta}{\eta+\mu+t_D d+1+\epsilon\gamma}.$$

For $\alpha_2 \to 0$ and $\alpha_3 \to 0$, we have

$$\lim_{\substack{\alpha_2\to 0\\ \alpha_3\to 0}} \Re_0^T = \frac{\alpha^* M_S\big(\eta+\mu+t_D d+\epsilon\gamma\big)}{Nk_{11}\big((\gamma+\mu+d+\delta)(\eta+\mu+t_D d)-\gamma\eta\big)}. \tag{3.6}$$

If the limit (3.6) is greater than unity, then when $\alpha_2 \to 0$ and $\alpha_3 \to 0$ it has a negative impact, that is when

$$\frac{\alpha^* M_S}{Nk_{11}} > \frac{(\gamma+\mu+d+\delta)(\eta+\mu+t_D d)-\gamma\eta}{\eta+\mu+t_D d+\epsilon\gamma}.$$

Let us define the following expressions:

$$\begin{aligned}
\Delta_D =& \frac{\alpha^* M_S}{N k_{11}}, \\
\Delta_{D_1} =& \frac{(\gamma+\mu+d+1+\delta)(\eta+\mu+t_D d)-\eta\gamma}{\eta+\mu+t_D d+\epsilon\gamma}, && (3.7)\\
\Delta_{D_2} =& \frac{(\gamma+\mu+d+\delta)(1+\eta+\mu+t_D d)-\eta\gamma}{\gamma+\mu+t_D d+1+\epsilon\gamma}, && (3.8)\\
\Delta_{D_3} =& \frac{(\gamma+\mu+d+1+\delta)(\eta+\mu+t_D d+1)-\gamma\eta}{\eta+\mu+t_D d+1+\epsilon\gamma}, && (3.9)\\
\Delta_{D_4} =& \frac{(\gamma+\mu+d+\delta)(\eta+\mu+t_D d)-\gamma\eta}{\eta+\mu+t_D d+\epsilon\gamma}. && (3.10)
\end{aligned}$$

The following lemma characterizes the impact on the basic reproduction number of the parameters associated with the diagnosis of diabetes in obese and overweight individuals.

Lemma 1. *1. The impact when $\alpha_2 \to 1$ and $\alpha_3 \to 0$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{D_1}$, no impact if $\Delta_D = \Delta_{D_1}$ and negative if $\Delta_D > \Delta_{D_1}$.*

2. The impact when $\alpha_2 \to 0$ and $\alpha_3 \to 1$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{D_2}$, no impact if $\Delta_D = \Delta_{D_2}$ and negative if $\Delta_D > \Delta_{D_2}$.

3. The impact when $\alpha_2 \to 1$ and $\alpha_3 \to 1$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{D_3}$, no impact if $\Delta_D = \Delta_{D_3}$ and negative if $\Delta_D > \Delta_{D_3}$.

4. The impact when $\alpha_2 \to 0$ and $\alpha_3 \to 0$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{D_4}$, no impact if $\Delta_D = \Delta_{D_4}$ and negative if $\Delta_D > \Delta_{D_4}$.

Analogously, we perform the same procedure for η and γ. These parameters are associated with the passage from obese to overweight, a positive situation and the passage from overweight to obese which means a negative condition respectively. With the study of these parameters together, we are assessing a positive and negative condition of exit from the obese compartment. We obtain the following expressions:

$$\begin{aligned}
\Delta_{O_1} =& \frac{(\mu+d+\alpha_2+\delta)(\alpha_3+\mu+t_D d+1)}{1+\mu+t_D d+\alpha_3}, && (3.11)\\
\Delta_{O_2} =& (1+\alpha_2+\mu+d+\delta)(\mu+t_D d+\alpha_3)/(\alpha_3+\mu+t_D d+\epsilon), && (3.12)\\
\Delta_{O_3} =& \frac{(1+\delta+\alpha_2+\mu+d)(\mu+t_D d+\alpha_3+1)-1}{1+\mu+t_D d+\alpha_3+\epsilon}, && (3.13)\\
\Delta_{O_4} =& (\delta+\alpha_2+\mu+d)(\alpha_3+\mu+t_D d)/(\mu+t_D d+\alpha_3), && (3.14)
\end{aligned}$$

and obtain the following lemma.

Lemma 2. *1. The impact when $\eta \to 1$ and $\gamma \to 0$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{O_1}$, no impact if $\Delta_D = \Delta_{O_1}$ and negative if $\Delta_D > \Delta_{O_1}$.*

2. *The impact when* $\eta \to 0$ *and* $\gamma \to 1$ *is positive in reducing overweight and obesity if* $\Delta_D < \Delta_{O_2}$*, no impact if* $\Delta_D = \Delta_{O_2}$ *and negative if* $\Delta_D > \Delta_{O_2}$*.*

3. *The impact when* $\eta \to 1$ *and* $\gamma \to 1$ *is positive in reducing overweight and obesity if*$\Delta_D < \Delta_{O_3}$*, no impact if* $\Delta_D = \Delta_{O_3}$ *and negative if* $\Delta_D > \Delta_{O_3}$*.*

4. *The impact when* $\delta \to 0$ *and* $\gamma \to 0$ *is positive in reducing overweight and obesity if* $\Delta_D < \Delta_{O_4}$*, no impact if* $\Delta_D = \Delta_{O_4}$ *and negative if* $\Delta_D > \Delta_{O_4}$*.*

Analogously, the process is carried out for γ and δ representing the cases reaching overweight and those reaching normal-weight, which represents a positive (reaching normal-weight) and a negative (reaching obesity) output from the overweight individuals compartment and we define the following expressions:

$$\Delta_{O_{11}} = \frac{k_{13}(1+\alpha_2+\mu+d)-\eta}{k_{13}+\epsilon}, \tag{3.15}$$

$$\Delta_{O_{12}} = \frac{k_{13}(1+\alpha_2+\mu+d)}{k_{13}}, \tag{3.16}$$

$$\Delta_{O_{13}} = \frac{k_{13}(2+d+\alpha_2+\mu)-\eta}{k_{13}+\epsilon}, \tag{3.17}$$

$$\Delta_{O_{14}} = \frac{k_{13}(d+\alpha_2+\mu)}{k_{13}}, \tag{3.18}$$

and obtain the following lemma.

Lemma 3. 1. *The impact when* $\gamma \to 1$ *and* $\delta \to 0$ *is positive in reducing overweight and obesity if* $\Delta_D < \Delta_{O_{11}}$*, no impact if* $\Delta_D = \Delta_{O_{11}}$ *and negative if* $\Delta_D > \Delta_{O_{11}}$*.*

2. *The impact when* $\gamma_2 \to 0$ *and* $\delta \to 1$ *is positive in reducing overweight and obesity if* $\Delta_D < \Delta_{O_{12}}$*, no impact if* $\Delta_D = \Delta_{O_{12}}$ *and negative if* $\Delta_D > \Delta_{O_{12}}$*.*

3. *The impact when* $\gamma \to 1$ *and* $\delta \to 1$ *is positive in reducing overweight and obesity if* $\Delta_D < \Delta_{O_{13}}$*, no impact if* $\Delta_D = \Delta_{O_{13}}$ *and negative if* $\Delta_D > \Delta_{O_{13}}$*.*

4. *The impact when* $\gamma \to 0$ *and* $\delta \to 0$ *is positive in reducing overweight and obesity if* $\Delta_D < \Delta_{O_{14}}$*, no impact if* $\Delta_D = \Delta_{O_{14}}$ *and negative if* $\Delta_D > \Delta_{O_{14}}$*.*

Similarly, the process is carried out for η and δ. These parameters represent positive conditions in our dynamic as they will represent the cases coming out of obesity and into overweight and those coming out of overweight and reaching normal-weight respectively. We define the expressions:

$$\Delta_{O_{21}} = \frac{(\gamma+\mu+d+\alpha_2)(\alpha_3+\mu+t_D d+1)-\gamma}{1+\mu+t_D d+\alpha_3+\epsilon\gamma}, \tag{3.19}$$

$$\Delta_{O_{22}} = \frac{(\gamma+1+\alpha_2+\mu+d)(\mu+t_D d+\alpha_3)}{\alpha_3+\mu+t_D d+\epsilon\gamma}, \tag{3.20}$$

$$\Delta_{O_{23}} = \frac{(\gamma+d+\alpha_2+\mu+1)(\mu+t_D d+\alpha_3+1)-\gamma}{1+\mu+t_D d+\alpha_3+\epsilon\gamma}, \tag{3.21}$$

$$\Delta_{O_{24}} = \frac{(\gamma+\alpha_2+\mu+d)(\alpha_3+\mu+t_D d)}{\mu+t_D d+\epsilon\gamma+\alpha_3}, \tag{3.22}$$

and have the following lemma.

Lemma 4. *1. The impact when $\eta \to 1$ and $\delta \to 0$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{O_{21}}$, no impact if $\Delta_D = \Delta_{O_{21}}$ and negative if $\Delta_D > \Delta_{O_{21}}$.*

2. The impact if $\eta \to 0$ and $\delta \to 1$ is positive in reducing overweight and obesity only if $\Delta_D < \Delta_{O_{22}}$, no impact if $\Delta_D = \Delta_{O_{22}}$ and negative if $\Delta_D > \Delta_{O_{22}}$.

3. The impact when $\eta \to 1$ and $\delta \to 1$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{O_{23}}$, no impact if $\Delta_D = \Delta_{O_{23}}$ and negative if $\Delta_D > \Delta_{O_{23}}$.

4. The impact when $\eta \to 0$ and $\delta \to 0$ is positive in reducing overweight and obesity if $\Delta_D < \Delta_{O_{24}}$, no impact if $\Delta_D = \Delta_{O_{24}}$ and negative if $\Delta_D > \Delta_{O_{24}}$.

Now, we study the impact of the parameter that reports new cases of diabetes and is not associated with body weight and the parameter associated with weight gain due to social pressure. For the different variations of these parameters. We define the following expressions:

$$\Delta_{O_{31}} = (1+\mu)(k_{12}k_{13} - \eta\gamma)/(k_{13} + \epsilon\gamma), \tag{3.23}$$

$$\Delta_{O_{32}} = (2+\mu)(k_{12}k_{13} - \eta\gamma)/(k_{13} + \epsilon\gamma), \tag{3.24}$$

$$\Delta_{O_{33}} = \mu(k_{12}k_{13} - \eta\gamma)/(k_{13} + \epsilon\gamma), \tag{3.25}$$

and have the following lemma.

Lemma 5. *1. The impact when $\alpha_1 \to 1$ and $\beta_1 \to 0$ and $\alpha_1 \to 0$ and $\beta_1 \to 1$ is positive in reducing overweight and obesity if $\Delta_D k_{11} < \Delta_{O_{31}}$, no impact if $\Delta_D k_{11} = \Delta_{O_{31}}$ and negative if $\Delta_D k_{11} > \Delta_{O_{31}}$.*

2. The impact when $\alpha_1 \to 1$ and $\beta_1 \to 1$ is positive in reducing overweight and obesity if $\Delta_D k_{11} < \Delta_{O_{32}}$, no impact if $\Delta_D k_{11} = \Delta_{O_{32}}$ and negative if $\Delta_D k_{11} > \Delta_{O_{32}}$.

3. The impact when $\alpha_1 \to 0$ and $\beta_1 \to 0$ is positive in reducing overweight and obesity if$\Delta_D k_{11} < \Delta_{O_{33}}$, no impact if $\Delta_D k_{11} = \Delta_{O_{33}}$ and negative if $\Delta_D k_{11} > \Delta_{O_{33}}$.

We study the asymptotic behavior of the parameters $\alpha_1, \alpha_2, \alpha_3, \beta_1, \eta, \gamma$ and δ using the partial derivatives of $\Re_0$ with respect to these parameters.

$$\frac{\partial \Re_0}{\partial \alpha_1} = \frac{\partial \Re_0}{\partial \beta_1} = -\frac{\alpha^* M_S (k_{13} + \epsilon\gamma)}{N k_{11}^2 (k_{12}k_{13} - \eta\gamma)}, \tag{3.26}$$

$$\frac{\partial \Re_0}{\partial \alpha_2} = -\frac{\alpha^* M_S (k_{13}(k_{13} + \epsilon\gamma))}{N k_{11} (k_{12}k_{13} - \eta\gamma)^2}, \tag{3.27}$$

$$\frac{\partial \Re_0}{\partial \alpha_3} = -\frac{\alpha^* M_S (\eta\gamma + \epsilon k_{13})}{N k_{11} (k_{12}k_{13} - \eta\gamma)^2}, \tag{3.28}$$

$$\frac{\partial \Re_0}{\partial \eta} = \frac{\alpha^* M_S ((k_{12}k_{13} - \eta\gamma) - (k_{12} - \gamma)(k_{13} + \epsilon\gamma))}{N k_{11} (k_{12}k_{13} - \eta\gamma)^2}, \tag{3.29}$$

$$\frac{\partial \Re_0}{\partial \gamma} = \frac{\alpha^* M_S (\epsilon(k_{12}k_{13} - \gamma\eta) - (k_{13} - \eta)(k_{13} + \epsilon\gamma))}{N k_{11} (k_{12}k_{13} - \eta\gamma)^2}, \tag{3.30}$$

$$\frac{\partial \Re_0}{\partial \delta} = -\frac{\alpha^* M_S (k_{13}(k_{13} + \epsilon\gamma))}{N k_{11} (k_{12}k_{13} - \eta\gamma)^2}. \tag{3.31}$$

The partial derivatives with respect to α_1, α_2, α_3, β_1 and δ are unconditionally less than zero. This will mean that any outflow from the weight-related compartment of individuals to the diabetes compartment will go a long way in reducing the burden of obesity in the population, as we assume that these individuals with diabetes will change their lifestyle and control their weight. The same applies for δ, which represents the improvement in the lives of overweight people that brings them to a state of normal-weight. In the case of β_1, we will study the impact directly on the compartments in Section 4 to validate this result.
We can characterize the behavior of the partial derivatives with respect to the $\Re_0$ of the η and γ parameters with the following lemma.

Lemma 6. *The parameter associated with the transition from obese to overweight, η (overweight to obese, γ) have a positive impact on the basic reproduction number ($\Re_0$) in the population (respectively) if*

$$\frac{(k_{12}k_{13}-\eta\gamma)}{(k_{12}-\gamma)(k_{13}+\epsilon\gamma)}<1 \qquad \left(\frac{\epsilon(k_{12}k_{13}-\eta\gamma)}{(k_{13}-\eta)(k_{13}+\epsilon\gamma)}<1\right),$$

no impact if

$$\frac{(k_{12}k_{13}-\eta\gamma)}{(k_{12}-\gamma)(k_{13}+\epsilon\gamma)}=1 \qquad \left(\frac{\epsilon(k_{12}k_{13}-\eta\gamma)}{(k_{13}-\eta)(k_{13}+\epsilon\gamma)}=1\right),$$

and a detrimental impact if

$$\frac{(k_{12}k_{13}-\eta\gamma)}{(k_{12}-\gamma)(k_{13}+\epsilon\gamma)}<1 \qquad \left(\frac{\epsilon(k_{12}k_{13}-\eta\gamma)}{(k_{13}-\eta)(k_{13}+\epsilon\gamma)}>1\right).$$

Disease equilibrium points

Model (2.1) can be written in matrix form as:

$$\begin{pmatrix} -(\lambda_o+k_{11}) & \delta & 0 & 0 \\ \lambda_o+\beta_1 & -k_{12} & \eta & 0 \\ 0 & \gamma & -k_{13} & 0 \\ \alpha_1 & \alpha_2 & \alpha_3 & -k_{14} \end{pmatrix} \begin{pmatrix} S \\ O_w \\ O_b \\ D \end{pmatrix} = \begin{pmatrix} -M_S \\ 0 \\ 0 \\ -M_D \end{pmatrix}. \tag{3.32}$$

To find the disease equilibrium points, we solve the system (3.32). Then, the solution of system is (S^*, O_w^*, O_b^*, D^*), where:

$$\begin{aligned} S^* =& \frac{M_S(k_{12}k_{13}-\eta\gamma)}{A_1}, \quad O_w^* = \frac{k_{13}(\beta_1+\lambda_o)M_S}{A_1}, \quad O_b^* = \frac{\gamma(\beta_1+\lambda_o)M_S}{A_1}, \\ D^* =& \frac{k_{13}(k_{11}k_{12}+k_{12}\lambda_o-\delta(\beta_1+\lambda_o))M_D + k_{13}(\alpha_1 k_{12}+\alpha_2(\beta_1+\lambda_o))M_S}{k_{14}A_1} \\ &- \frac{((k_{11}+\lambda_o)\eta M_D - \alpha_3(\beta_1+\lambda_o)M_S + \alpha_1\eta M_S)\gamma}{k_{14}A_1}, \end{aligned}$$

with $A_1 = k_{13}(k_{11}k_{12}+k_{12}\lambda_o-\delta(\beta_1+\lambda_o)) - (k_{11}+\lambda_o)\eta\gamma$.

3.1 Sensitivity index

In this section, we study the impact of the parameters on the threshold quantity, $\Re_0$. 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 be 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 [8, 19, 28].

Definition 1. ([28]) The normalized forward sensitivity index of a variable, v, that depends differentiability on a parameter p is defined as:

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

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 in the parameter value causes an increase in 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.

We selected to find the sensitivity index the parameters associated with diabetes in overweight and obese (α_2 and α_3), the parameters γ, η and δ that will represent the transition in the weight-associated compartments, α_1 which is associated with the diagnosis of diabetes due to factors unrelated to weight and β_1 which is associated with weight gain due to social pressure.

The expressions of the sensitivity indices of the selected parameters are:

$$\Upsilon_{\alpha_1}^{\Re_0} = -\alpha_1/k_{11}, \tag{3.33}$$

$$\Upsilon_{\beta_1}^{\Re_0} = -\beta_1/k_{11}, \tag{3.34}$$

$$\Upsilon_{\alpha_2}^{\Re_0} = -\alpha_2 k_{13}/(k_{12}k_{13} - \eta\gamma), \tag{3.35}$$

$$\Upsilon_{\alpha_3}^{\Re_0} = -\alpha_3(\gamma\eta + \epsilon k_{13})/(k_{12}k_{13} - \eta\gamma)(k_{13} + \epsilon\gamma), \tag{3.36}$$

$$\Upsilon_{\eta}^{\Re_0} = \frac{\eta((k_{12}k_{13} - \eta\gamma) - (k_{12} - \gamma)(k_{13} + \epsilon\gamma))}{(k_{12}k_{13} - \eta\gamma)(k_{13} + \epsilon\gamma)}, \tag{3.37}$$

$$\Upsilon_{\gamma}^{\Re_0} = \frac{\gamma(\epsilon(k_{12}k_{13} - \gamma\eta) - (k_{13} - \eta)(k_{13} + \epsilon\gamma))}{(k_{12}k_{13} - \eta\gamma)(k_{13} + \epsilon\gamma)}, \tag{3.38}$$

$$\Upsilon_{\delta}^{\Re_0} = -\delta k_{13}/(k_{12}k_{13} - \eta\gamma). \tag{3.39}$$

Parameters α_1, β_1, α_2, α_3 and δ have a negative sensitivity index with respect to $\Re_0$, which implies that an increase in these parameters will mean a decrease in $\Re_0$. Epidemiologically for parameters α_2, and α_3 the overweight and obese are diagnosed with diabetes, here they will require continuous care so that a successful control will mean among other factors to be a normal-weight. In the case of δ this behavior is evident in $\Re_0$ because it will mean the cases that are overweight that reach a normal-weight.

The value of the other sensitivity indices studied will depend on the scenario under study. We use the expressions (3.37) and (3.38) to determine when the sensitivity indices $\Upsilon_{\eta}^{\Re_0}$ and $\Upsilon_{\gamma}^{\Re_0}$ are negative and obtain the following lemma.

Lemma 7. *The sensitivity indices* $\Upsilon_{\eta}^{\Re_0}$ *and* $\Upsilon_{\gamma}^{\Re_0}$ *are less than zero, when* $\frac{k_{12}k_{13}-\eta\gamma}{(k_{12}-\gamma)(k_{13}+\epsilon\gamma)} < 1$ *and* $\frac{\epsilon(k_{12}k_{13}-\eta\gamma)}{(k_{13}-\eta)(k_{13}+\epsilon\gamma)} < 1$, *respectively.*

4 Numerical simulations

In this section, we perform computational simulations to validate the proposed model and make a study of the basic reproduction number and compartments. We use the fourth-order Runge–Kutta numerical scheme coded in MATLAB programming language. The data used for the computational simulations are extracted from the literature or assumed. The values for the parameters used in the computational simulations are listed in Table 1. The initial conditions are $S_0 = 874.1400$, $O_{w0} = 1.2000$, $O_{b0} = 1.5000$ and $D_0 = 100.0000$. The assumed values for the parameters and initial conditions do not represent a real scenario.

4.1 Basic reproduction number

First, let's apply Lemmas 1–5 to this scenario. Table 2 shows the values of the expressions associated with these results for this scenario. For the variations

Table 2. Values of expressions (3.7)–(3.10), (3.11)–(3.14), (3.15)–(3.18), (3.19)–(3.22) and (3.23)–(3.25).

	Values		Values
$\Delta_D = 0.3667$	$\Delta_{D_1} = 1.0767$ $\Delta_{D_2} = 0.0874$ $\Delta_{D_3} = 1.0860$ $\Delta_{D_4} = 0.0861$	$\Delta_D = 0.3667$	$\Delta_{O_1} = 0.9646$ $\Delta_{O_2} = 0.1429$ $\Delta_{O_3} = 0.5062$ $\Delta_{O_4} = 0.9646$
$\Delta_D = 0.3667$	$\Delta_{O_{11}} = 0.5977$ $\Delta_{O_{12}} = 1.9626$ $\Delta_{O_{13}} = 0.9312$ $\Delta_{O_{14}} = 0.9626$	$\Delta_D k_{11} = 0.0486$	$\Delta_{O_{31}} = 0.4423$ $\Delta_{O_{32}} = 0.8784$ $\Delta_{O_{33}} = 0.062$
$\Delta_D = 0.3667$	$\Delta_{O_{21}} = 0.9612$ $\Delta_{O_{12}} = 1.9273$ $\Delta_{O_{13}} = 1.9596$ $\Delta_{O_{14}} = 0.9460$		

of the parameters α_2 and α_3 using Lemma 1, we have that when α_2 tends to unity and α_3 tends to zero and when both parameters are tending to unity the impact is positive, in the other variations studied it is negative. This shows that the growth of the parameter α_2 associated with the diagnosis of diabetes in overweight has a positive impact when varied together with α_3 associated with the diagnosis of diabetes in obese.

We will study what happens when we vary the parameter associated with obese people who improve their lifestyle and become overweight (η) and the parameter associated with overweight people who worsen their condition and become obese (γ).

In the different variations and using Lemma 2, we have that when η tends to zero and γ tends to unity the impact is negative, the other variants the impact is positive. This means that a growth in the overweight becoming obese that is not equilibrium with the behavior of the obese improving and becoming overweight has a negative effect on the dynamic.

Using Lemmas 3–4 characterizing the variations of the parameters η and γ with respect to δ always has a positive impact. The interpretation is that the parameter δ which is associated with overweight individuals who reach normal-weight has a positive impact on the dynamic when it is varied jointly with individuals who become obese and others who become obese to overweight.

Regarding the study of the variation of α_1 and β_1, and using Lemma 5 we have that when these parameters tend to zero the impact is negative, in the other variants it is positive. By decreasing the number of cases diagnosed with diabetes due to other causes and the impact of social pressure together we have that the number of overweight and obese people may increase.

We can conclude that the parameter δ has a positive influence on the dynamic in this study when varied with the other parameters and these results help in the design of a control strategy because it shows the joint strength of the parameters under study.

Table 3 shows the derivatives (3.26)–(3.31) and sensitivity indices (3.33)–(3.39) with respect to the basic reproduction number for this scenario.

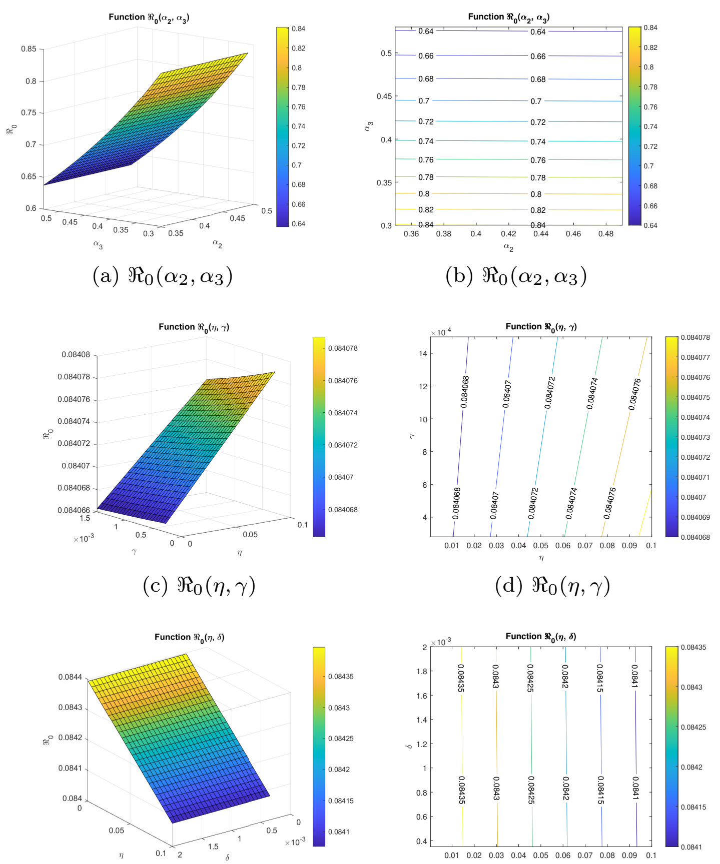
Table 3. Values of derivatives (3.26)–(3.31) and sensitivity indices (3.33)–(3.39) with respect to $\Re_0$ for the scenario under study.

Parameters	Derivative	Sensitivity Index
α_1	-0.23086	-0.2743
β_1	-0.2386	-0.6865
α_2	-1.9221	-0.8001
α_3	-0.0051	-0.0024
γ	0.0810	1.4451e-04
η	-2.0748e-04	-2.4678e-05
δ	-0.1921	-0.0046

We can conclude that parameters α_1, α_2, α_3, β_1, η and δ have a negative sensitivity index with respect to the basic reproduction number, which means that an independent increase in them will result in a decrease of $\Re_0$. In the case of η the opposite happens, the sensitivity index is positive and an increase in this parameter causes an increase in the $\Re_0$ and for higher $\alpha-$values the impact is greater.

Now, we are going to study graphically the behavior of the basic reproduction number when we vary these parameters together. The intervals of the parameters are $\eta \in [0.00028, 0.1]$, $\gamma \in [0.00028, 0.0015]$, $\delta \in [0.00035, 0.002]$ (extracted from [6]), $\alpha_2 \in [0.35, 0.49]$ $\alpha_3 \in [0.3, 0.53]$ (extracted from [14, 15]), $\alpha_1 \in [0.05, 0.7]$ (assumed) and $\beta_1 \in [0.1, 0.8]$ (assumed). The joint variation of the selected parameters provides us with information on how these parameters varying together influence the basic reproduction number. Remember that here Lemmas 1–4 go to the extremes of the interval and now we define intervals for the parameters discussed with the specialists.

In the joint variation α_2 and α_3 we have that the highest value is reached when α_2 and α_3 reach the lowest values in the intervals under study and whenever α_3 is taking the lowest value in that interval and α_2 growing. The smallest value is reached when α_3 reaches the largest value in the interval and α_2 is varying in its respective interval. This is evidence that α_3 has a strong influence on the dynamic of the model so that obese cases diagnosed with diabetes require differentiated attention although this variation keeps the $\Re_0$ less than unity (see Figures 2a–2b).



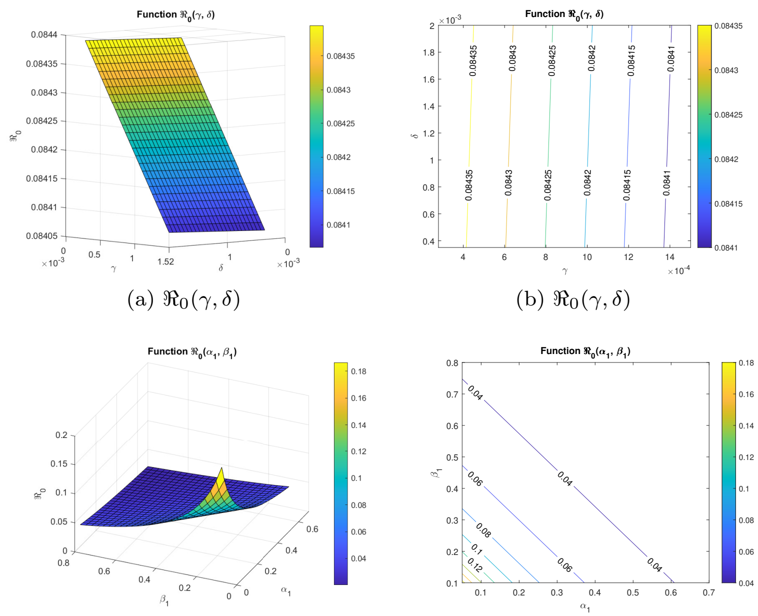
(a) $\Re_0(\alpha_2, \alpha_3)$ (b) $\Re_0(\alpha_2, \alpha_3)$

(c) $\Re_0(\eta, \gamma)$ (d) $\Re_0(\eta, \gamma)$

(e) $\Re_0(\eta, \delta)$ (f) $\Re_0(\eta, \delta)$

Figure 2. Joint variation of parameters on $\Re_0$, surfaces and curves.

For the other variations, the $\Re_0$ of the parameters under study is always less than unity, so the variations studied graphically in the scenario under study do not have a strong influence on $\Re_0$ (see Figures 3a–3d). We can observe that when α_1 and β_1 are taking the lowest value in the intervals studied, it is when $\Re_0$ reaches its highest value, which proves Lemma 5, that in this scenario α_1 and β_1 are tending to zero negatively affects (see Figures 3c–3d). We can observe that the variations α_2 and α_3 are the ones that report the highest basic reproduction number and close to the unit, so the diagnosis of diabetes in overweight and obese people must be studied in depth to design some control strategy.



(a) $\Re_0(\gamma, \delta)$ (b) $\Re_0(\gamma, \delta)$

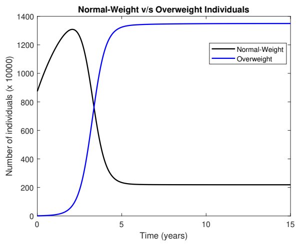
(c) $\Re_0(\alpha_1, \beta_1)$ (d) $\Re_0(\alpha_1, \beta_1)$

Figure 3. Joint variation of parameters on $\Re_0$, surfaces and curves.

4.2 Compartmental study

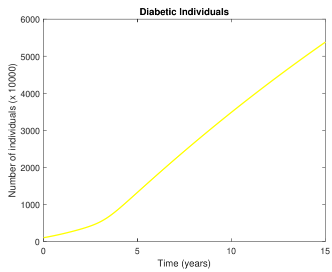
We studied a 15-year period to investigate the evolution of diabetes in the population. Normal-Weight individuals compartment has initially a growth and then decrease and stabilizes at end of the study. The difference between the initial value ($t = 0$) and final ($t = 15$) is 655.453 (in ten thousand people) less individuals were reported in this compartment (see Figure 4a).

This will mean that the output of this compartment at the end of the study is greater and this is negative because we have a reduction in the number of individuals who will have an adequate weight and who do not have diabetes. The maximum value reached in this compartment is approximately two years into the study and was 1308.38.



(a) Normal-Weight v/s Overweight Individuals

(b) Obese Individuals



(c) Diabetic Individuals

Figure 4. Behavior of the compartments of model (2.1) for the scenario under study and the different alpha-values studied.

In the compartment of overweight individuals we have growth until it stabilizes for this scenario. This means that mainly a large number of individuals changing their lifestyle are leaving the compartment of normal-weight individuals. This has great consequences on the population because overweight and obesity not only have an impact on diabetes but also on other diseases such as coronary heart disease. In this compartment the difference between the number of individuals at the final and initial time point was 1348.45 (see Figure 4a). The maximum number of individuals reported in this compartment is 1349.65 at the end of the study.

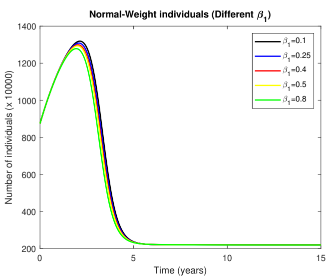
In individuals with obesity we have at the beginning a decrease in the number reported and then it starts to grow. This compartment is the one with the lowest number of cases reported in the study but also the most dangerous because of the harmfulness of this condition for the health of the individual. In this case we need to apply strategies to control its growth, since the difference between the final which represents the largest number of reported cases and initial value is 2.5961 (see Figure 4b). The greater the weight of the individual, the greater the probability of suffering from diabetes and other diseases and the more difficult it is to lose weight.

In the diabetic compartment, we have a growth during the whole study, this indicates that diabetes has a strong impact on the population. The difference between the final value which is the highest of the study and the initial value is 5279.01 and it is a significant amount (see Figure 4c). We can conclude that to reduce the impact of diabetes in the population, we must promote a lifestyle that avoids overweight and obesity.

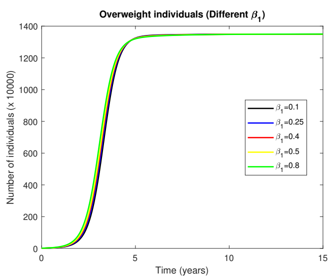
Diabetes is a disease with great consequences for health and that has no cure and an important factor is a good lifestyle. In this scenario, the number of individuals with normal-weight is reduced and the number of overweight, obese and diabetics increases, this shows that we need strategies to control this progress as it is detrimental to the individual and to the health system.

We studied the influence directly in the compartments of parameters α_1 and β_1 that are associated with cases that develop diabetes due to factors not associated with body weight and social pressure.

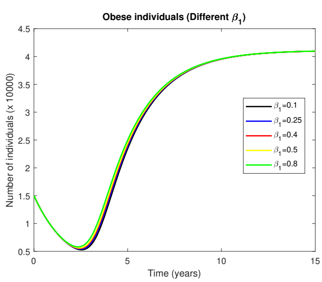
In the case of β_1, we see that at the beginning and at the end of the study for the different values studied the impact was not significant mainly in diabetics. At the intermediate time points we see that the higher β_1 results are worse because normal-weight individuals are reduced (for higher values of β_1 the reduction in those of normal-weight is greater), and overweight, obese and diabetics individuals increase (for higher values of β_1 the increase is more significant) (see Figures 5a–5d).



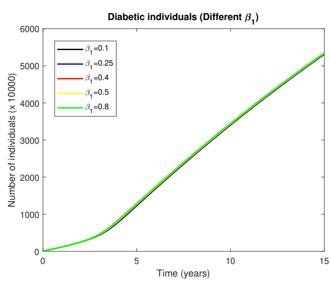
(a) Variation β_1, Normal-Weight individuals



(b) Variation β_1, Overweight individuals



(c) Variation β_1, Obese individuals



(d) Variation β_1, Diabetic individuals

Figure 5. Behavior when $\beta_1 = 0.1, 0.25, 0.4, 0.5, 0.8$.

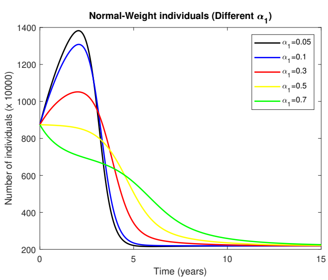
Table 4 shows the cumulative number of cases by compartment for the different β_1 at the end of the study. We show that despite the differences not being significant, the number of individuals with normal-weight decreases and those who are overweight, obese and diabetic increase as β_1 increases.

When we vary α_1, we have that in the compartment of individuals with

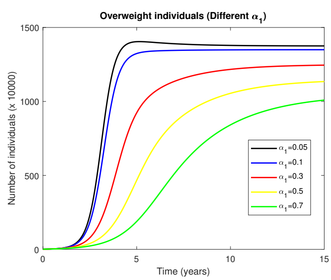
Table 4. Number of cases reported by compartments for the different values of β_1 at the end of the study period (15 years).

β_1−values	Normal-Weight individuals	Overweight individuals	Obese individuals	Diabetic individuals
0.1	218.781	1349.65	4.09601	5308.57
0.25	218.687	1349.6	4.09616	5319.15
0.4	218.593	1349.71	4.09632	5520.35
0.5	218.529	1349.75	4.09643	5333.89
0.8	218.338	1349.87	4.09679	5348.33

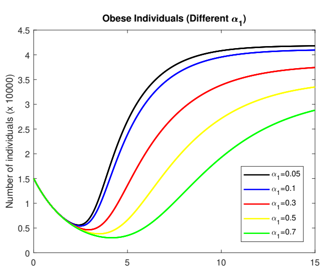
normal-weight, we have that initially the lower α_1 values report a higher number of individuals with that weight, then decreases starting to decrease for the lower α_1 values and at the end of the study the higher α_1 values report a higher number of cases but without a significant difference. It is important to highlight the initial behavior for the values of $\alpha_1 = 0.7$ (highest of the values studied) that has a decrease unlike the behavior of the other values of α_1 studied (see Figure 6a). These results may provide information to reduce the impact of diabetes in the population, since hereditary, racial and genetic factors also greatly influence the reporting of new cases of diabetes.



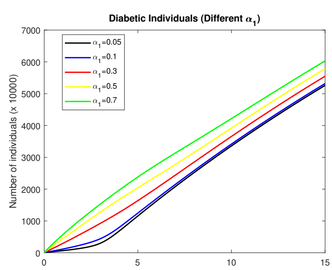
(a) Variation of α_1, Normal-Weight individuals



(b) Variation of α_1, Overweight individuals



(c) Variation of α_1, Obese individuals



(d) Variation of α_1, Diabetic individuals

Figure 6. Behavior when $\alpha_1 = 0.05, 0.1, 0.3, 0.5, 0.7$.

In the case of diabetics, it is important to highlight that the asymptotic behavior changes at the beginning of the study, for higher values we have a convex behavior and for the lower ones slightly concave, and for higher than α_1 a lower number of diabetic cases are reported at the beginning. After 5 years of study, the behavior of diabetics tends to increase the number of cases (see Figure 6d).

In the case of the overweight, the behavior tends to grow and then stabilizes, reporting a greater number of overweight cases for values less than α_1. In the obese initially we see a decrease and then we have a growth behavior, where for values less than α_1 the number of reported cases with obesity is greater.

Table 5 shows the values reported by each compartment for the different values of α_1. In this case we have that for values greater than α_1 the number of cases with normal-weight is greater, the number of overweight and obese is reduced and the number of diabetes increases significantly.

Table 5. Number of cases reported by compartments for the different values of α_1 at the end of the study period (15 years).

α_1−values	Normal-Weight individuals	Overweight individuals	Obese individuals	Diabetic individuals
0.05	218.601	1375.26	4.18056	5261.64
0.1	218.687	1349.65	4.09616	5319.15
0.3	219.358	1245.21	3.74334	5520.21
0.5	221.071	1134.37	3.34948	5785.53
0.7	225.227	1008.93	2.88069	6031.67

We can conclude that both diagnosis of diabetes and the influence of social pressure on overweight and obesity have an influence on the dynamic. We must take into account that the behavior for the different values of α_1 was more significant in the compartments. To achieve a control strategy with the aim of reducing diabetes in the population, we recommend paying attention to parameter α_1, because we show that factors other than obesity can report significant numbers of diabetics.

5 Conclusions

In this paper, we presented a mathematical model to study obesity and overweight and their relationship with diabetes. The model allows to study the behavior of normal-weight, overweight and obese individuals and their interactions and the influence of social factors (social pressure). Using the next generation matrix method we found the basic reproduction number and showed the global stability of the disease-free equilibrium point. We calculated the sensitivity indices with respect to the basic reproduction number for the parameters associated with the transition between the normal-weight, overweight and obese compartments and those associated with diabetes. We presented theoretical results to characterize the joint variation of these parameters and identified the sign of the sensitivity index on $\Re_0$. To validate the model, we performed computational simulations with data from the literature and other assumed data validated by specialists. We checked the theoretical results and found that the parameter associated with the transition from overweight to obese has a positive sensitivity index, which means that a growth in this parameter causes a growth in $\Re_0$.

In the study of the compartments, we found that the normal-weight compartment has a significant decrease and the overweight, obese and diabetic compartments have an increase. These results show the need for a control strategy to control obesity and consequently diabetes.

We studied the impact on the dynamics of the parameters β_1 and α_1 and found that the parameter α_1 gives a volatile behavior to the compartments of individuals with normal weight and its variation causes a more significant effect on the compartments compared to the variation of β_1. In particular, the growth of parameter α_1 will increase the number of diabetic cases. Thus, factors other than obesity and overweight also have an impact on the number of diabetic cases.

In our work, we limit ourselves to the study of the negative impact of overweight and obesity on people of normal weight and define the transmission rate. We must take into account that the opposite effect (i.e., those of normal weight may contribute to help the obese and overweight) may also have an influence but this is not the current focus of our work. To obtain some parameters such as those related to transitions, development of diabetes and the influence of social factors on overweight require population-based studies.

These results provide information for the construction of a control strategy with the aim of reducing obesity and overweight and diabetes. In future work, we will use the model to study scenarios with different demographic and industrial characteristics, we will propose an optimal control model with the objective of reducing overweight and obesity in the population and consequently the impact of diabetes.

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