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A mathematical model with fractional order for obesity with positive and negative interactions and its impact on the diagnosis of diabetes

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Abstract

Overweight and obesity are current problems humankind faces and have serious health consequences because they contribute to diseases such as heart diseases and diabetes. In this paper, we present a 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 different elements of society. We use fractional-order derivatives in the Caputo sense because of the advantages of this type of technique with respect to the memory effect, and it shows different behaviors depending on the fractional order. We find the basic reproduction number and prove the local and global stability of the disease-free equilibrium points. We study the sensitivity index with respect to the basic reproduction number for parameters associated with weight gain due to social pressure and the rate of diagnosis of diabetes not associated with body weight. To validate the model, we perform computational simulations with data extracted from the literature. We conclude that for higher fractional orders a higher value of the basic reproduction number was reached. We show that at the end of the study for different fractional orders that normal-weight individuals are decreasing, and overweight, obese, and diabetic people are increasing.

1. Introduction

In 2016, more than 1.9 billion adults were overweight, and among them, more than 650 million were obese worldwide. As a result, diseases associated with overweight and obesity have also been on the rise globally [1].
To determine body weight state, we use the body mass index (BMI), which is defined as [2]:

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

Then, individuals are considered of normal-weight when $BMI \in [18.6, 24.9]$, overweight individuals are when $BMI \in [25, 29.9]$, obese individuals are when $BMI \in [30, 40]$, and in complicated situations over 40. We know that the body mass index can be high in people with high muscle mass but we assume that these cases are not included because before calculating the BMI we do a preliminary analysis. The root cause of obesity and overweight is an energy imbalance between calories consumed and calories expended.
A poorly balanced diet, stress, sedentary lifestyle, and factors such as environmental changes, and lack of sanitation in sectors such as agriculture, environment, processing, distribution, and marketing of food cause an increase in the number of cases of overweight and obesity in the population [1]. We have two types of diabetes and among their differences is that type I diabetes is primarily diagnosed by genetic disorders and usually manifests itself at an early age and type 2 diabetes is mainly related to lifestyle and develops over time. We also have

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cases where the continuous use of medications can develop diabetes [3, 4]. Among the factors that lead to type 2 diabetes is obesity. The likelihood of developing type 2 diabetes as well as its severity increases with obesity. The risk of developing type 2 diabetes in people with obesity is 7 times higher and in overweight people 3 times higher than in people with normal-weight and a healthy lifestyle [4, 5].
In recent years, the use of mathematical models to study the dynamics of obesity and overweight has increased [6]-[10]. Ejima et al. [6] presented a mathematical model that studies the genetic and non-genetic effects that lead to obesity and among the results they obtained was 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 [7] with the inclusion of psychological and social factors proposed a mathematical model for the dynamics of obesity. Paudel [8] proposed a model for the dynamics of obesity with a SIR structure and analyzed the effect of the social network on the spread of obesity. Al-Tuwairqi and Matbouli [9] proposed two mathematical models to study the impact of fast food on the increase in obesity and the role of physical activity. Pietrus et al. [10] developed a mathematical model to study the impact of media on the dynamics of obesity in a population. Model proposals for the study of the impact of diabetes on a population have increased in recent decades [11]-[15]. For example, Dubey and Goswami [11] presented a model of diabetes and its complications using a fractional operator. Sandhya and Kumar [12] proposed a mathematical model for the study of diabetes mellitus with the presence of plasma glucose concentration, generalized insulin, and plasma insulin concentration. Anusha and Athithan [13] proposed a mathematical model for type 2 diabetes and stratified the population into susceptible, unbalanced glucose level (IGL), treatment, and restriction. Banzi et al. [14] presented a mathematical model to investigate the behavior of glucose, insulin, glucagon, stored insulin, and labile insulin in diabetics. Delgado et al. [15] presented a deterministic compartmental model that studies overweight, obesity, and diabetes in a population, taking into account the negative impact of an overweight or obese individual on a normal-weight individual, and also the impact of social pressure on the increase of overweight and obese cases and consequently the increase of diabetes diagnosis.
The aim of this work is to present a mathematical model using fractional-order derivatives in the Caputo sense for the study of obesity and, as a consequence, diabetes in a population. This model has as its main center the positive impact of the contact of an individual with normal-weight with an overweight and the inverse negative impact. It also studies the impact of pressure on the increase in cases of overweight and obesity. It allows us to quantify in the dynamics of how overweight and obesity increase the diagnosis of diabetes, also take into account that other factors not associated with body weight can cause new cases of diabetes.
This paper is organized as follows: In Section 2, we present the mathematical definitions used in the paper. In Section 3, we introduce the model, show the basic properties and study the basic reproduction number. Section 4 is devoted to numerical experimentation. We finish the paper with some conclusions in Section 5.

2. Theorical Background

The following definitions are used to formulate and study the fractional-order model.
We assume that $\alpha \in \mathbb{R}_+$, $b > 0$, $f \in AC^n[a,b]$ (absolutely continuous), and $n = [\alpha]$ (entire part of α). We define the left-sided and right-sided fractional integral Riemann-Louville for $f : \mathbb{R}^+ \longrightarrow \mathbb{R}$ and $\alpha > 0$ are:

$$ {}_a\mathbb{I}_t^\alpha f(t) := \frac{1}{\Gamma(\alpha)} \int_a^t \frac{f(s)ds}{(t-s)^{1-\alpha}}, \qquad \text{(Left)} $$

$$ {}_t\mathbb{I}_b^\alpha f(t) := \frac{1}{\Gamma(\alpha)} \int_t^b \frac{f(s)ds}{(s-t)^{1-\alpha}} \qquad \text{(Right)} $$

where Γ is Gamma function, and we define $\mathbb{I}_t^\alpha f(t) = {}_0\mathbb{I}_t^\alpha f(t)$.
The left-sided and right-sided Riemann–Liouville fractional derivatives are defined as [16, 17]:

$$ {}_a\mathbf{D}_t^\alpha f(t) = \frac{d^n}{dt^n}\left(\frac{1}{\Gamma(n-\alpha)} \int_a^t (t-s)^{n-\alpha-1} f(s)ds\right), \qquad \text{(Left)} $$

$$ {}_t\mathbf{D}_b^\alpha f(t) = \frac{d^n}{dt^n}\left(\frac{(-1)^n}{\Gamma(n-\alpha)} \int_t^b (s-t)^{n-\alpha-1} f(s)ds\right) \qquad \text{(Right)} $$

and we denote $\mathbf{D}_t^\alpha f(t) = {}_0\mathbf{D}_t^\alpha f(t)$.
The left-sided and right-sided fractional derivatives proposed by Caputo are given by [16, 17]:

$$ {}_a^c\mathbb{D}_t^\alpha f(t) = \frac{1}{\Gamma(n-\alpha)} \int_a^t (t-s)^{n-1-\alpha} f^{(n)}(s)ds, \qquad \text{(Left)} $$

$$ {}_t^c\mathbb{D}_b^\alpha f(t) = \frac{(-1)^n}{\Gamma(n-\alpha)} \int_t^b (s-t)^{n-1-\alpha} f^{(n)}(s)ds \qquad \text{(Right)} $$

and we define ${}^c\mathbb{D}_t^\alpha f(t) = {}_0^c\mathbb{D}_t^\alpha f(t)$.
In the order-fractional derivatives, we find the memory effect which is an important factor in epidemic modeling [18]-[20].
In recent decades, works have been presented where models with fractional orders are used and compared with real data and it has been obtained that they can capture real behaviors, see [21]-[24].

3. Model Formulation

The body mass index is a hasty diagnosis of overweight and obesity but we assume that we have a previous evaluation for the diagnosis of patients with these physical conditions. Then, based on the body mass index, we define the compartments of our model as: normal-weight individuals, S, overweight individuals, O_w, obese individuals, O_b and diabetic individuals, D.
We define the negative impact rate as

$$ \lambda_O = (\alpha^*)^\alpha \frac{(O_w + \varepsilon O_b)}{N}, $$

and positive impact rate as

$$\lambda_S = \frac{(\beta^*)^\alpha S}{N},$$

where α^* and β^* are the effective contact rate and N is the total population ($N = S + O_w + O_b + D$). These rates will represent the negative influence that an overweight or obese person has on a person with normal-weight, leading this person to increase his body weight and leave the compartment, and the positive influence that a person with normal-weight has on an overweight or obese person, who with an improvement in his lifestyle, decreases his body weight and passes through the different compartments. Let us assume that the effective contact rate in the negative effect (α^*) is greater than the effective contact rate in the positive effect (β^*). This means that leading a nutritionally disordered and/or sedentary life is easier than leading a nutritionally healthy life in addition to other elements such as physical exercise.

We assume that the impact of overweight and obese on a person with normal-weight is different, for this, we use the modification parameter ε such that $\varepsilon > 1$. The rates M_S and M_D represent the entry of individuals with normal-weight and diabetes respectively.

The mortality rate from natural causes in the population is defined as μ and we assume that it is the same from any compartment. We define d as the mortality rate associated with diseases related to increased body weight, mainly cardiac ones, and t_H represents the modification parameter for the obese and t_D for diabetics.

The rate α_1 characterizes cases diagnosed with diabetes due to causes that are not directly associated with weight gain including genetic, racial, hereditary, and other factors. Parameters α_2 and α_3 are the rates of diabetes diagnoses associated with overweight and obesity, respectively. The rate of death associated with diabetes is denoted by μ_D.

The rate δ characterize individuals who are overweight but improve to normal-weight and η is for obese individuals who become overweight. These two parameters are not related to the interaction with a normal-weight individual. Individuals who increase in body weight from overweight to obese are defined by the rate γ. The rate β_1 represents the social pressure that causes an individual with normal-weight to become obese. This rate is characterized by stress, lack of time for healthy eating and physical exercise, sedentary lifestyle, etc. The fractional derivative operator in the Caputo sense, ${}^c\mathbb{D}_t^\alpha$ has a dimension α, then on the right-hand side of the model all parameters will have power dimension α except the modification parameter [25]. So, the model that relates obesity to diabetes in a population using fractional derivatives in the Caputo sense is:

$$\begin{aligned}
{}^c\mathbb{D}_t^\alpha S &= M_S^\alpha + (\delta^\alpha + \lambda_S)O_w - (\mu^\alpha + \alpha_1^\alpha + \beta_1^\alpha + \lambda_O)S,\\
{}^c\mathbb{D}_t^\alpha O_w &= (\lambda_O + \beta_1^\alpha)S + \eta^\alpha O_b - (\lambda_S + \gamma^\alpha + \mu^\alpha + d^\alpha + \delta^\alpha + \alpha_2^\alpha)O_w,\\
{}^c\mathbb{D}_t^\alpha O_b &= \gamma^\alpha O_w - (\eta^\alpha + \mu^\alpha + t_H d^\alpha + \alpha_3^\alpha)O_b,\\
{}^c\mathbb{D}_t^\alpha D &= M_D^\alpha + \alpha_1^\alpha S + \alpha_2^\alpha O_w + \alpha_3^\alpha O_b - (\mu_d^\alpha + \mu^\alpha + t_D d^\alpha)D,
\end{aligned} \tag{3.1}$$

with initial condition

$$S(0) = S_0 \geq 0, O_w(0) = O_{w0} \geq 0, O_b(0) = O_{b0} \geq 0, D(0) = D_0 \geq 0 \text{ and with } \alpha \in (0,1].$$

Variable	Description
S	normal-weight individuals
O_W	Overweight individuals
O_b	Obese individuals
D	Diabetic individuals
Parameter	**Description**
M_S, M_D	Recruitment rates for normal-weight and diabetic individuals
α^*, β^*	Effective contact rates
d	death rate associated with weight gain
μ	Natural death rate
μ_d	Diabetes death rate
η	Rate of weight reduction from obese to overweight
γ	Rate of weight gain from overweight to obese
δ	Rate of weight reduction from overweight to normal-weight
α_1	Rate of diagnosis of diabetes not associated with body weight
α_2	Rate of diabetes diagnosis in overweight individuals
α_3	Rate of diagnosis of diabetes in obese individuals
β_1	Rate of weight gain associated with social factors
ε, t_D, t_H	Modification parameters

Table 3.1: Description of variables and parameters of model (3.1).

Basic properties of model (3.1)

Now, let us prove the existence and positivity of the solution of Model (3.1), and let's find the biologically feasible region. The following results and their proofs can be found in [26].

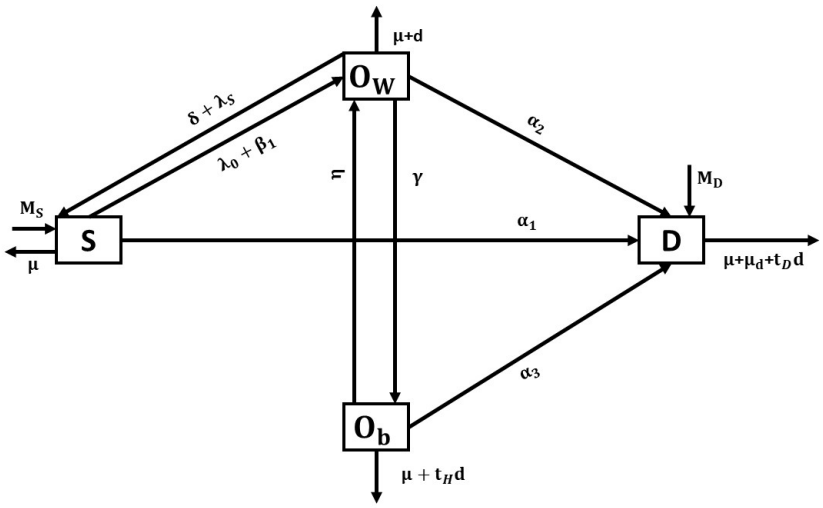


Figure 3.1: Flow chart of Model (3.1).

Existence and non-negativity of solutions

Let's denominate

$$\Omega = \{x = (S, O_w, O_b, D) : S, O_w, O_b, D \geq 0\}.$$

The following lemma and corollary will be used in the proof of Theorem (3.3) and can be found in [27]-[30].

Lemma 3.1. *(Generalized mean value theorem) Suppose that $f \in C[a,b]$ ($C[a,b]$ is the set of continuous functions on the interval $[a,b]$) and ${}^c\mathbb{D}_t^\alpha f \in C[a,b]$, for $\alpha \in (0,1]$. Then, for all $t \in (a,b]$, it exists $\varepsilon \in [a,t]$, such that*

$$f(t) = f(a) + \frac{1}{\Gamma(\alpha)}({}^c\mathbb{D}_t^\alpha f)(\varepsilon)(t-a)^\alpha.$$

Corollary 3.2. *Consider that $f \in C[a,b]$ and ${}^c\mathbb{D}_t^\alpha f \in C[a,b]$, for $\alpha \in (0,1]$. Then if*

- ${}^c\mathbb{D}_t^\alpha f(t) \geq 0$, $\forall t \in (a,b)$, *then $f(t)$ is non-decreasing for each $t \in [a,b]$,*
- ${}^c\mathbb{D}_t^\alpha f(t) \leq 0$, $\forall t \in (a,b)$, *then $f(t)$ is non-increasing for each $t \in [a,b]$.*

Theorem 3.3. *There is an unique solution $x = (S, O_w, O_b, D)^T$ of Model (3.1) for $t \geq 0$ and the solution will remain in Ω.*

Proof. By Theorem 3.1 and Remark 3.2 of [30], we have that the solution in $(0,\infty)$ of the initial value problem (3.1) exists and is unique. Now, we will prove the positivity of the solution of Model (3.1). In order to do this, we need to prove that for every hyperplane bounding the nonnegative orthant, the vector field points to Ω. From Model (3.1), we have:

$$\begin{aligned}
{}^c\mathbb{D}_t^\alpha S\big|_{S=0} &= M_S^\alpha + \delta^\alpha O_w > 0,\\
{}^c\mathbb{D}_t^\alpha O_w\big|_{O_w=0} &= \left(\frac{\varepsilon(\alpha^*)^\alpha O_b}{N} + \beta_1^\alpha\right)S + \eta^\alpha O_b \geq 0,\\
{}^c\mathbb{D}_t^\alpha O_b\big|_{O_b=0} &= \gamma^\alpha O_w \geq 0,\\
{}^c\mathbb{D}_t^\alpha D\big|_{D=0} &= M_D^\alpha + \alpha_1^\alpha S + \alpha_2^\alpha O_w + \alpha_3^\alpha O_b > 0.
\end{aligned}$$

Using Corollary 3.2, we have that the solution will remain in Ω. □

Biologically feasible region

Now, let's prove that Ω_α is the biologically feasible region for Model (3.1).

Lemma 3.4. *The closed set $\Omega_\alpha = \left\{(S, O_w, O_b, D) \in \mathbb{R}_+^4 : N \leq \frac{M_S^\alpha + M_D^\alpha}{\mu^\alpha}\right\}$ is positively invariant with respect to model (3.1).*

Proof. The fractional derivative in the Caputo sense of the total population is

$${}^c\mathbb{D}_t^\alpha N(t) = {}^c\mathbb{D}_t^\alpha S(t) + {}^c\mathbb{D}_t^\alpha O_w(t) + {}^c\mathbb{D}_t^\alpha O_b(t) + {}^c\mathbb{D}_t^\alpha D(t) = M_S^\alpha + M_D^\alpha - \mu^\alpha N(t) - d^\alpha(O_w(t) + t_H O_b(t) + t_D D(t)) - \mu_d^\alpha D(t),$$

and we have

$${}^c\mathbb{D}_t^\alpha N(t) + \mu^\alpha N(t) \leq M_S^\alpha + M_D^\alpha.$$

To continue the proof we use the following definitions:

Definition 3.5. *The Laplace transform of the Caputo fractional derivatives of the function $\phi(t)$ with order $\alpha > 0$ is defined as*

$$\mathscr{L}\left[{}^{c}\mathbb{D}_{t}^{\alpha}\phi(t)\right] = s^{\alpha}\phi(s) - \sum_{v=0}^{n-1}\phi^{(v)}(0)s^{\alpha-v-1}. \tag{3.2}$$

Definition 3.6. *The Laplace transform of the function $t^{\alpha_1-1}\mathbb{E}_{\alpha,\alpha_1}(\pm\lambda t^{\alpha})$ is defined as*

$$\mathscr{L}\left[t^{\alpha_1-1}\mathbb{E}_{\alpha,\alpha_1}(\pm\lambda t^{\alpha})\right] = \frac{s^{\alpha-\alpha_1}}{s^{\alpha}\mp\lambda}, \tag{3.3}$$

where $\mathbb{E}_{\alpha,\alpha_1}$ is two-parameters Mittag-Leffler function $\alpha,\alpha_1 > 0$. Further, the Mittag-Leffler function satisfies the following equation:

$$\mathbb{E}_{\alpha,\alpha_1}(f) = f\mathbb{E}_{\alpha,\alpha+1}(f) + \frac{1}{\Gamma(\alpha_1)}. \tag{3.4}$$

Applying the Laplace transform to (3.2), we have

$$s^{\alpha}\phi(N) - s^{\alpha-1}\phi(0) \leq \frac{M_S^{\alpha}+M_D^{\alpha}}{s} - \mu^{\alpha}\phi(N),$$

which further gives

$$N(t) \leq \frac{M_S^{\alpha}+M_D^{\alpha}}{s(s^{\alpha}+\mu^{\alpha})} + \frac{s^{\alpha-1}}{s^{\alpha}+\mu^{\alpha}}N(0).$$

Using the equations (3.2)-(3.4), we assumed that $(S(0),O_w(0),O_b(0),D(0)) \in \mathbb{R}_+^4$, then

$$N(t) \leq (M_S^{\alpha}+M_D^{\alpha})t^{\alpha}\mathbb{E}_{\alpha,\alpha+1}(-\mu^{\alpha}t^{\alpha}) + N(0)\mathbb{E}_{\alpha,1}(-\mu t^{\alpha}).$$

Using the asymptotic behavior of the Mittag-Leffler function, we can observe that $N(t) \to \dfrac{M_S^{\alpha}+M_D^{\alpha}}{\mu^{\alpha}}$ as $t \to \infty$.

The Ω_{α} region is well established and the solution with initial value that belongs to Ω_{α} remains in Ω_{α} for each time $t > 0$. □

4. Basic Reproduction Number Study

The objective of this section is to calculate the basic reproduction number, using the next-generation matrix method that uses the equilibrium point without obesity, overweight, and diabetes. As a consequence of using this equilibrium as part of the method used, we study its stability and the direct effect on the basic reproduction number of the parameter associated with weight gain due to social pressure.

In a population composed only of susceptible individuals, the average number of infections caused by an infected individual is defined as basic reproduction number $\Re_0$. 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 [31]. 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 study of our basic reproduction number is centered on the impact of overweight and obesity, so these compartments are the ones that will be analyzed. We recall that diabetes is included in our study as a consequence of weight gain and furthermore, the body weight of individuals is not differentiated within this compartment. To find the basic reproduction number, we use the next generation-matrix method presented in [31]-[33], where the disease-free equilibrium point of Model (3.1) is $\varepsilon_0 = \left(\dfrac{M_S^{\alpha}}{k_{11}}, 0, 0, 0\right)$ where $k_{11} = \mu^{\alpha} + \alpha_1^{\alpha} + \beta_1^{\alpha}$. For our study, the disease-free equilibrium point refers to overweight and obesity, and then we will have people with normal-weight and diabetics since we may have diagnoses of diabetes that are not associated with body weight.

The transmission and transition matrices are:

$$F = \begin{pmatrix} \dfrac{M_S^{\alpha}\left((\alpha^*)^{\alpha}-(\beta^*)^{\alpha}\right)}{Nk_{11}} & \dfrac{M_S^{\alpha}\varepsilon(\alpha^*)^{\alpha}}{Nk_{11}} \\ 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} k_{12} & -\eta^{\alpha} \\ -\gamma^{\alpha} & k_{13} \end{pmatrix},$$

respectively. Then, for Model (3.1) the basic reproduction number is:

$$\Re_0 = \rho(FV^{-1}) = \frac{M_S^{\alpha}\left(k_{13}\left((\alpha^*)^{\alpha}-(\beta^*)^{\alpha}\right)+\varepsilon(\alpha^*)^{\alpha}\gamma^{\alpha}\right)}{Nk_{11}(k_{12}k_{13}-\eta^{\alpha}\gamma^{\alpha})},$$

where $k_{12} = \gamma^{\alpha}+\mu^{\alpha}+d^{\alpha}+\delta^{\alpha}+\alpha_2^{\alpha}$, $k_{13} = \eta^{\alpha}+\mu^{\alpha}+t_H d^{\alpha}+\alpha_3^{\alpha}$, $k_{14} = \mu_d^{\alpha}+\mu^{\alpha}+t_D d^{\alpha}$, and $\rho(FV^{-1})$ is the spectral radius of the matrix FV^{-1}.

We will study the local and global stability of the equilibrium point without obesity, overweight, and diabetes and without the impact of social pressure ($\beta_1 = 0$). For this parameter, we study its impact on the basic reproduction number and on the compartments.

The local stability of ε_0 can be determined using the following theorem:

Theorem 4.1. *Let $\alpha = \frac{p}{q}$ where $p,q \in \mathbb{Z}_+$ and $gdc(p,q) = 1$. Define $M = q(p = M\alpha)$, then the disease-free equilibrium point ε_0 of model (3.1) is asymptotically stable if $[arg(\lambda)] > \frac{\pi}{2M}$ for all roots λ of the following equation*

$$\det(\lambda^p \mathbb{I}_4 - M_1) = 0,$$

where $\mathbb{I}_4$ is the identity matrix of order 4×4, M_1 is the matrix of the linearization of model (3.1) at ε_0, $arg(\lambda)$ is the argument of λ and $gdc(p,q)$ is greatest common divisor of p and q.

Proof.

$$\det(\lambda^p \mathbb{I}_4 - M_1) = (\lambda^p + k_{11})(\lambda^p + k_{14})(\lambda^{2p} + b\lambda^p + c),$$

where $b = \frac{M_S^\alpha\left((\beta^*)^\alpha - (\alpha^*)^\alpha\right)}{Nk_{11}} + k_{12} + k_{13}$, $c = \frac{M_S^\alpha\left(k_{13}\left((\beta^*)^\alpha - (\alpha^*)^\alpha\right) - \varepsilon(\alpha^*)^\alpha\gamma^\alpha\right)}{Nk_{11}} + k_{12}k_{13} - \eta^\alpha\gamma^\alpha$.
Let's study the case when $c > 0$. Then, $c > 0$ implies that:

$$\frac{M_S^\alpha\left(k_{13}\left((\beta^*)^\alpha - (\alpha^*)^\alpha\right) - \varepsilon(\alpha^*)^\alpha\gamma^\alpha\right)}{Nk_{11}} + k_{12}k_{13} - \eta^\alpha\gamma^\alpha > 0. \tag{4.1}$$

Developing the expression (4.1), we have that $c > 0$ when,

$$\frac{M_S^\alpha\left((k_{13}\left((\alpha^*)^\alpha - (\beta^*)^\alpha\right) + \varepsilon(\alpha^*)^\alpha\gamma^\alpha\right)}{Nk_{11}(k_{12}k_{13} - \eta^\alpha\gamma^\alpha)} = \Re_0 < 1.$$

Using that $c > 0$ if $\Re_0 < 1$, we will prove that $b > 0$. We have:

$$\frac{M_S^\alpha\left((k_{13}\left((\alpha^*)^\alpha - (\beta^*)^\alpha\right) + \varepsilon(\alpha^*)^\alpha\gamma^\alpha\right)}{Nk_{11}} < k_{12}k_{13} - \eta^\alpha\gamma^\alpha,$$

$$\frac{M_S^\alpha(k_{13}\left((\alpha^*)^\alpha - (\beta^*)^\alpha\right)}{Nk_{11}} < k_{12}k_{13} - \eta^\alpha\gamma^\alpha - \frac{\varepsilon(\alpha^*)^\alpha\gamma^\alpha}{Nk_{11}}. \tag{4.2}$$

The elements of the right-hand side of inequality (4.2) are positive, we have that:

$$\frac{M_S^\alpha\left((k_{13}\left((\alpha^*)^\alpha - (\beta^*)^\alpha\right)\right)}{Nk_{11}} < k_{12}k_{13}.$$

First, we divide by k_{13}, which by definition is nonzero (it suffices that μ is nonzero which is the death rate from causes not associated with obesity), and we have that:

$$\frac{M_S^\alpha\left((\alpha^*)^\alpha - (\beta^*)^\alpha\right)}{Nk_{11}} < k_{12}.$$

Now, we add k_{13} which is positive, and obtain the inequality:

$$\frac{M_S^\alpha\left((\alpha^*)^\alpha - (\beta^*)^\alpha\right)}{Nk_{11}} < k_{12} + k_{13}.$$

We can conclude that:

$$b = \frac{M_S^\alpha\left((\beta^*)^\alpha - (\alpha^*)^\alpha\right)}{Nk_{11}} + k_{12} + k_{13} > 0.$$

Authors in [34], showed that the Routh-Hurwitz criteria, $b, c > 0$, are necessary and sufficient for $[arg(\lambda)] > \frac{\pi}{2}$. It is clear that all of the eigenvalues are negative ($[arg(\lambda)] > \frac{\pi}{2}$) if $\Re_0 < 1$. Hence, the disease-free equilibrium ε_0 is locally asymptotically stable for $\alpha \in (0,1]$ if $\Re_0 < 1$. □

This theorem was proved with analogous ideas in references [35]-[37]. From Theorem 2 of [32, 37], we have the following lemma characterizing the instability with the $\Re_0$:

Lemma 4.2. *The disease-free equilibrium point ε_0 of model (3.1) is unstable if $\Re_0 > 1$.*

Now, using an analogous method applied to Model (3.1), we prove the global stability of the disease-free equilibrium point. Following [38], we can rewrite the model (3.1) as

$$\begin{aligned} {}^c\mathbb{D}_t^\alpha X &= F(X,I), \\ {}^c\mathbb{D}_t^\alpha I &= G(X,I), \quad G(X,0) = 0, \end{aligned}$$

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 (3.1).

The disease-free equilibrium point is now denoted by $E_0^\alpha = (X_0, 0)$, $X_0 = \left(\frac{M_S^\alpha}{\mu^\alpha}, 0\right)$.

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

$$\begin{aligned} (H_1^\alpha): &\quad \text{For} \quad {}^c\mathbb{D}_t^\alpha X = F(X,0), \quad X_0 \quad \text{is globally asymptotically stable,} \\ (H_2^\alpha): &\quad G(X,I) = AI - G^*(X,I), \quad G^*(X,I) \geq 0, \quad \text{for} \quad (X,I) \in \Omega_\alpha, \end{aligned}$$

where $A = D_I G(X_0, 0)$, ($D_I G(X_0,0)$ is the Jacobian of G with respect to I at $(X_0,0)$) is a M-matrix (the off-diagonal elements of A are non-negative) and Ω_α is the biologically feasible region.
The following result shows the global stability of the disease-free equilibrium point.

Theorem 4.3. *The fixed point E_0^α is a globally asymptotically stable equilibrium (G.A.S) of Model (3.1) provided that $\Re_0 < 1$ and that the conditions (H_1^α) and (H_2^α) are satisfied.*

Proof. Let

$$F(X,0) = \begin{pmatrix} M_S^\alpha - k_{11}S \\ M_D^\alpha + \alpha_1^\alpha S - k_{14}D \end{pmatrix}.$$

As $F(X,0)$ is linear, then X_0 is globally stable and (H_1^α) is satisfied. Let

$$A = \begin{pmatrix} -k_{12} + (\alpha^*)^\alpha + \lambda_S & \eta^\alpha + \varepsilon(\alpha^*)^\alpha \\ \gamma^\alpha & -k_{13} \end{pmatrix},$$

$$I = (O_w, O_b),$$

$$G^*(X,I) = AI^T - G(X,I),$$

$$G^*(X,I) = \begin{pmatrix} G_1^*(X,I) \\ G_2^*(X,I) \end{pmatrix} = \begin{pmatrix} (\alpha^*)^\alpha (O_w + \varepsilon O_b)\left(1 - \dfrac{S}{N}\right) \\ 0 \end{pmatrix}.$$

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

Analogous proofs can be found in the bibliographical references [26, 28, 29].

4.1. Sensitive index

In this section, we performed a sensitivity analysis of the $\Re_0$ with respect to the parameters β_1 and α_1, which are related to weight gain associated with social pressure and the diagnosis of diabetes that is associated with factors other than body weight. 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 [39]-[41].

Definition 4.4. *([41]) 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 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.

Since the $\Re_0$ was calculated with the parameters at the $\alpha-$level, the sensitivity analysis is also performed at this level for consistency of the study. The expressions of the sensitivity indices of the selected parameters are as follows:

$$\Upsilon_{\alpha_1}^{\Re_0} = -\frac{\alpha\alpha_1^\alpha}{k_{11}},$$

$$\Upsilon_{\beta_1}^{\Re_0} = -\frac{\alpha\beta_1^\alpha}{k_{11}}.$$

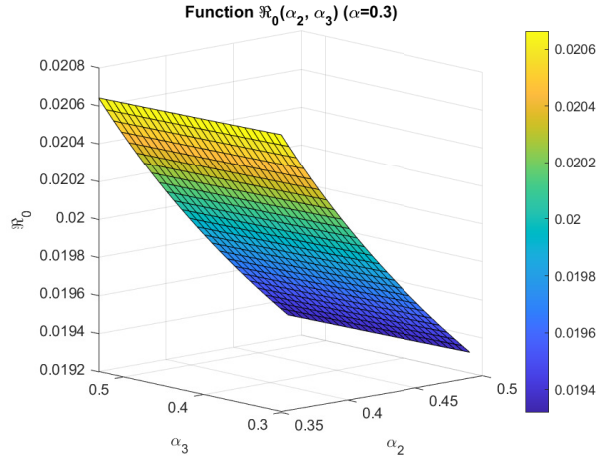
Parameters α_1 and β_1 have a negative sensitivity index with respect to $\Re_0$, which implies that an increase of these parameters will mean a decrease of $\Re_0$. The social pressure exerted on an individual and the diagnosis of diabetes for causes not associated with body weight independently, a growth in their respective parameters will mean a decrease on $\Re_0$.

5. Numerical Simulations

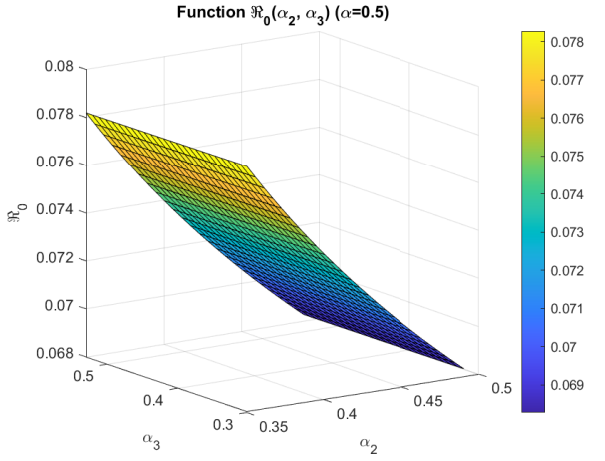
In this section, we perform computational simulations to validate the proposed model and make a study of the basic reproduction number. The algorithm used to numerically solve nonlinear differential equations of fractional-order system (3.1) is in [42]-[44]. The algorithm has the structure of a PECE (Predict-Evaluate-Correct-Evaluate) method and combines a fractional-order algorithm with a classical method. The approach chosen is Adams-Bashforth-Moulton for both integrators. The key to deriving the method in the fractional variant is to use the trapezoidal quadrature product formula. This algorithm is independent of the $\alpha-$value and behaves very similarly to the classical Adams-Bashforth-Moulton method. The stability properties do not change in the fractional version compared to the classical algorithm. The programming was carried out in Matlab software. The data used for the computational simulations are extracted from the literature or assumed. The parameter values for the computational simulations are $(\alpha^*)^\alpha = 2$ (assumed), $(\beta^*)^\alpha = 0.2$ (assumed), $M_S^\alpha = 667.685$ [28], $M_D^\alpha = 4.1$ (assumed), $\beta_1^\alpha = 0.25$ (assumed), $\varepsilon = 0.007$ [10], $\eta^\alpha = 0.1$ [10], $\gamma^\alpha = 0.0015$ [10], $\delta^\alpha = 0.002$ [10], $\mu^\alpha = \frac{1}{70.5}$ [26], $d^\alpha = 0.07$ (assumed), $t_H = 1.02$ (assumed), $t_D = 1.03$ (assumed), $\mu_d^\alpha = 0.013$ (assumed), $\alpha_1^\alpha = 0.1$ (assumed), $\alpha_2^\alpha = 0.35$ [45, 46], $\alpha_3^\alpha = 0.4$ [45, 46], and $\alpha = 0.3, 0.5, 0.7, 0.9, 1$. The initial conditions are $S = 874.1400$, $O_w = 1.2000$, $O_b = 1.5000$ and $D = 100.0000$ on the scale of 10000 individuals.

5.1. Basic reproduction number

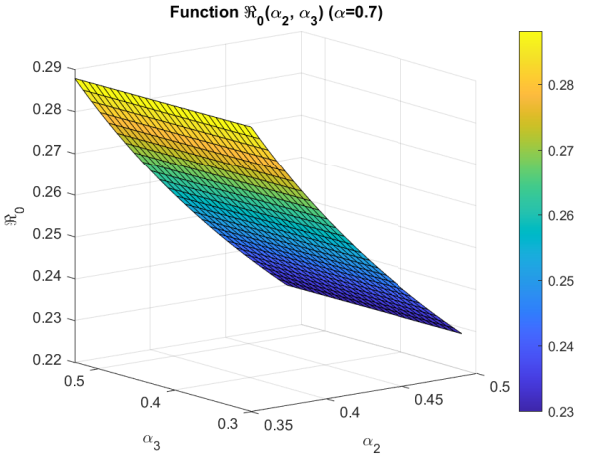
In this section, we study the impact on the basic reproduction number of the parameters $\eta, \gamma, \delta, \beta_1$, and $\alpha_i, i = 1,2,3$ and interpret how joint variations of them affect or not the behavior of $\Re_0$ for different fractional orders. We will study the joint variations of the parameters associated with transitions through the compartments associated with body weight and diabetes as a consequence of weight. 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 [10]), $\alpha_2 \in [0.35, 0.49]$ and $\alpha_3 \in [0.3, 0.53]$ (extracted from [45, 46]). The joint variation of the selected parameters provides us with information on how they will influence the $\Re_0$, for different fractional orders. It will allow us to obtain information not only about how they may or may not affect $\Re_0$, but also what happens when we vary the fractional order. This information will help us create future strategies to reduce overweight and obesity.
In the study of the variation of the parameters α_2 and α_3 associated with suffering from diabetes as a consequence of overweight and obesity, we observed that $\Re_0$ increases in value as α increases in the study, see Figures 5.1a-5.1e. For $\alpha = 0.9$, we find values greater and less than unity and for $\alpha = 1$, $\Re_0$ is greater than unity, see Figures 5.1d and 5.1e. This information gives us different possibilities of behavior for different $\alpha-$values which helps in the control proposals. We can notice that when α_3 increases it has a strong influence on the basic reproduction number.
In the joint variation of η and γ that are associated with the cases that go from obese to overweight (positive situation) and those that go from overweight to obese (adverse situation), it happens that $\Re_0$ increases as $\alpha-$values increases, see Figures 5.2a-5.2e. For $\alpha = 0.9$, we have that $\Re_0$ remains close to unity (≤ 0.95) and exceeds it, see Figure 5.2d. For $\alpha = 1$, $\Re_0$ exceeds unity reaching values greater than two, see Figure 5.2e. The higher $\Re_0$ the more difficult is the control.
An analogous behavior occurs for the variation of η and δ (positive situation because these are the ones that go from overweight to reach a normal-weight), see Figures 5.3a-5.3e. These variations show the influence that N has on the dynamics.
In the variation of γ and δ together in $\Re_0$, unlike the two previous ones, the unit is only exceeded when $\alpha = 1$ (for the $\alpha-$values studied) and it is lower than the variations of η and γ, and η and δ, see Figures 5.4a-5.4e.
We can conclude that with greater fractional order the $\Re_0$ in all the variations studied will be greater, even reaching values greater than unity. The higher the $\Re_0$, the more difficult the design of strategies to control overweight and obesity and, as a consequence, diabetes. Furthermore, with selected parameters, it shows us that we must control more than one compartment in the dynamics.



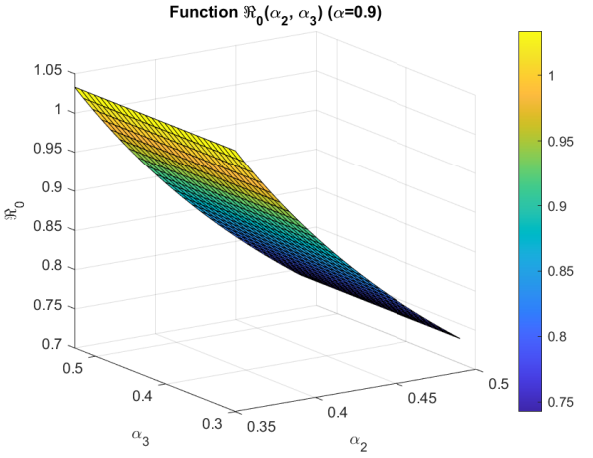
(a) $\Re_0(\alpha_2, \alpha_3)$ for $\alpha = 0.3$



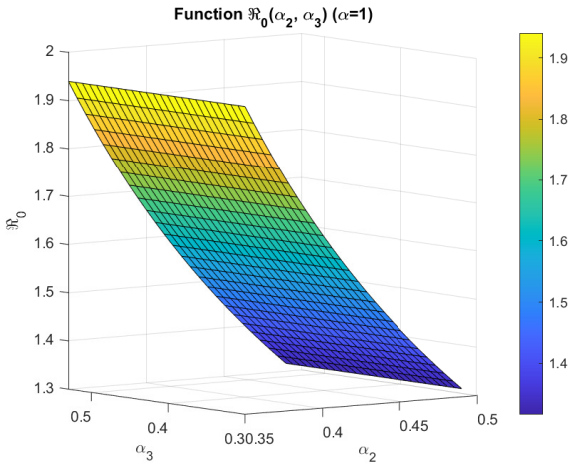
(b) $\Re_0(\alpha_2, \alpha_3)$ for $\alpha = 0.5$



(c) $\Re_0(\alpha_2, \alpha_3)$ for $\alpha = 0.7$

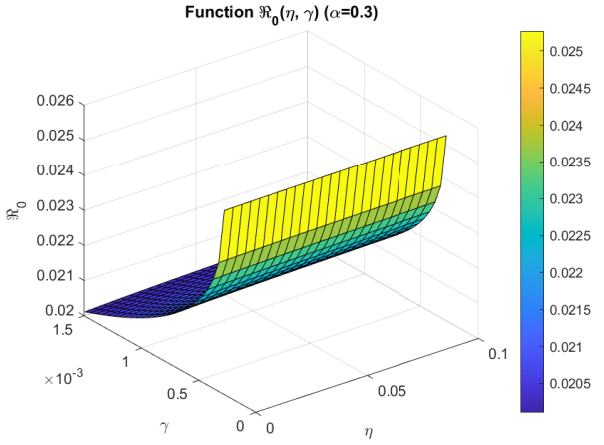


(d) $\Re_0(\alpha_2, \alpha_3)$ for $\alpha = 0.9$

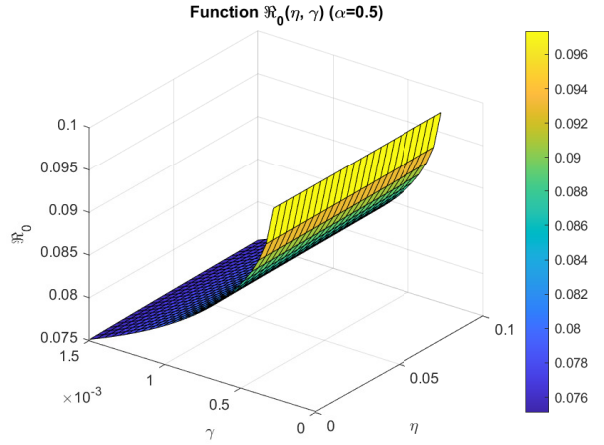


(e) $\Re_0(\alpha_2, \alpha_3)$ for $\alpha = 1$

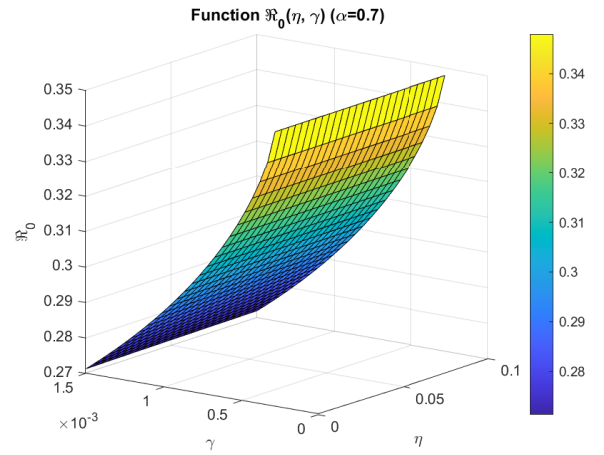
Figure 5.1: Joint variation of α_2^α and α_3^α in $\Re_0$ for different $\alpha-$values.



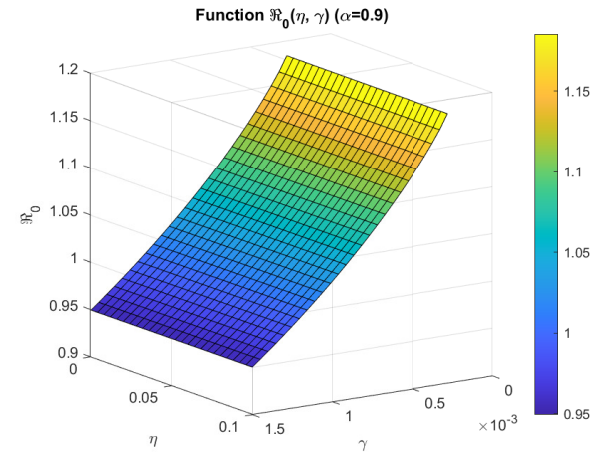
(a) $\Re_0(\eta, \gamma)$ for $\alpha = 0.3$



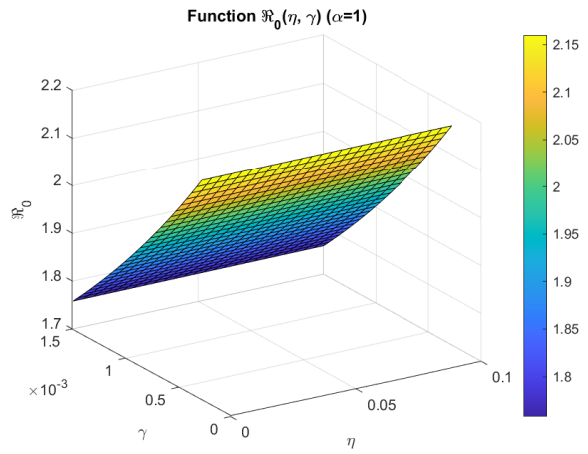
(b) $\Re_0(\eta, \gamma)$ for $\alpha = 0.5$



(c) $\Re_0(\eta, \gamma)$ for $\alpha = 0.7$

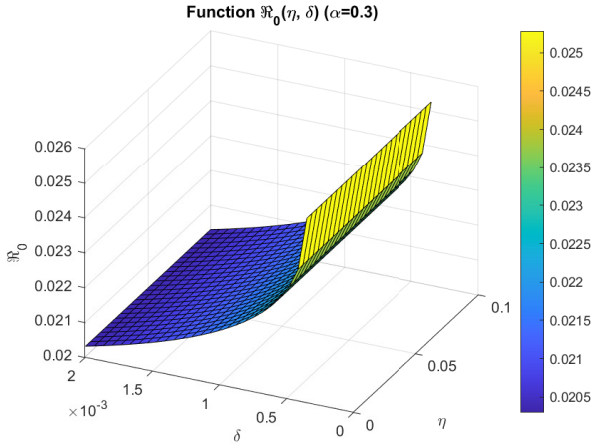


(d) $\Re_0(\eta, \gamma)$ for $\alpha = 0.9$

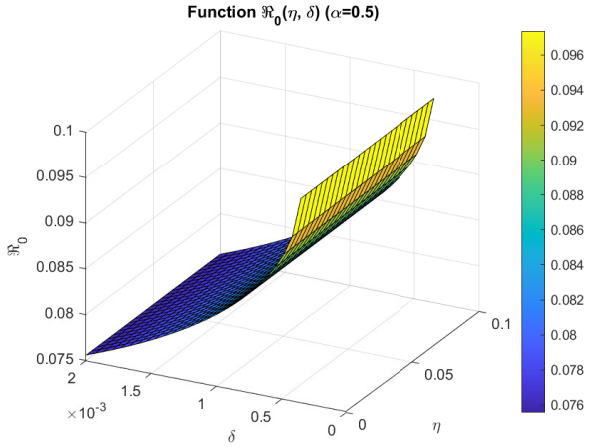


(e) $\Re_0(\eta, \gamma)$ for $\alpha = 1$

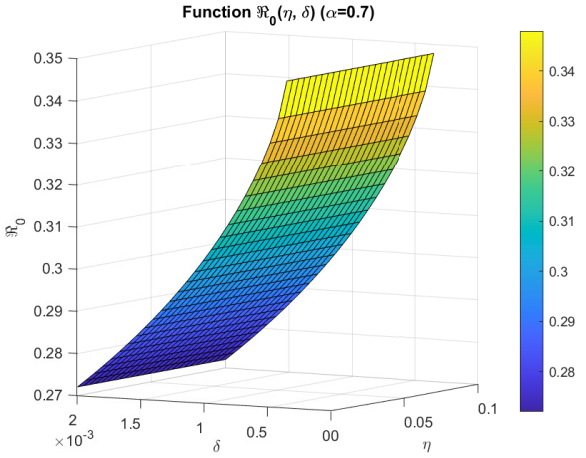
Figure 5.2: Joint variation of η^α and γ^α in $\Re_0$ for different $\alpha-$values.



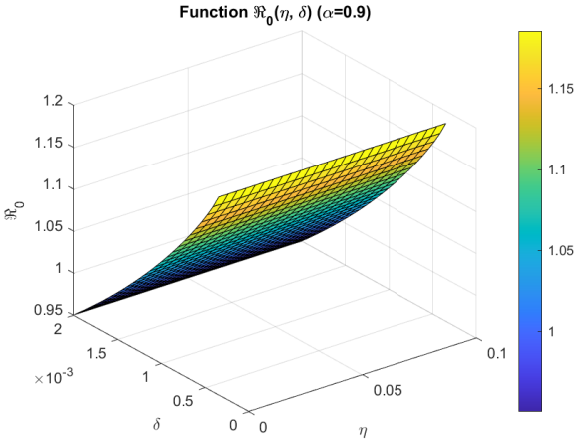
(a) $\Re_0(\eta, \delta)$ for $\alpha = 0.3$



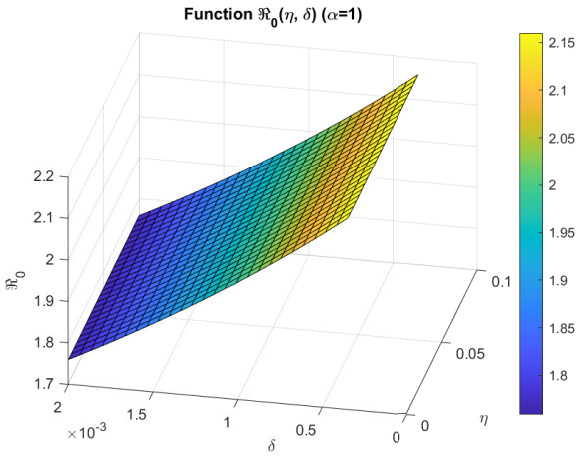
(b) $\Re_0(\eta, \delta)$ for $\alpha = 0.5$



(c) $\Re_0(\eta, \delta)$ for $\alpha = 0.7$

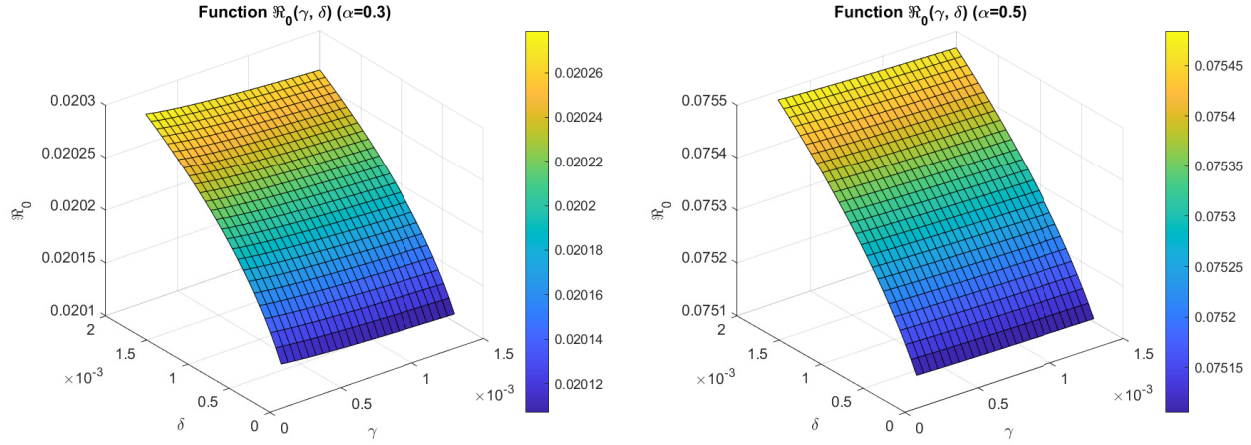


(d) $\Re_0(\eta, \delta)$ for $\alpha = 0.9$



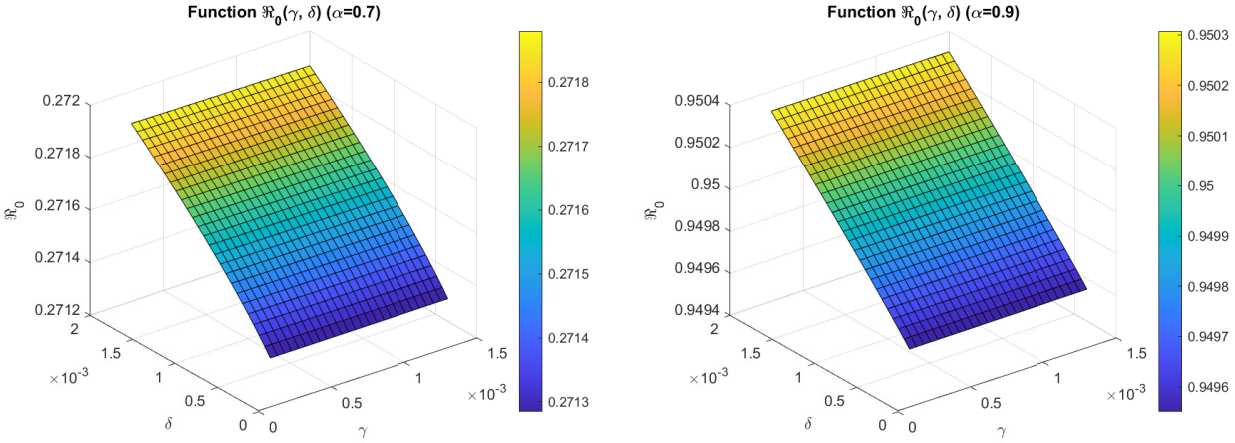
(e) $\Re_0(\eta, \delta)$ for $\alpha = 1$

Figure 5.3: Joint variation of η^α and δ^α in $\Re_0$ for different $\alpha-$values.



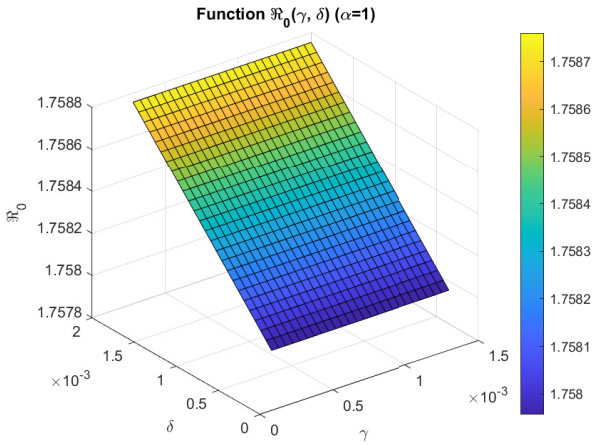
(a) $\Re_0(\gamma, \delta)$ for $\alpha = 0.3$

(b) $\Re_0(\gamma, \delta)$ for $\alpha = 0.5$



(c) $\Re_0(\gamma, \delta)$ for $\alpha = 0.7$

(d) $\Re_0(\gamma, \delta)$ for $\alpha = 0.9$

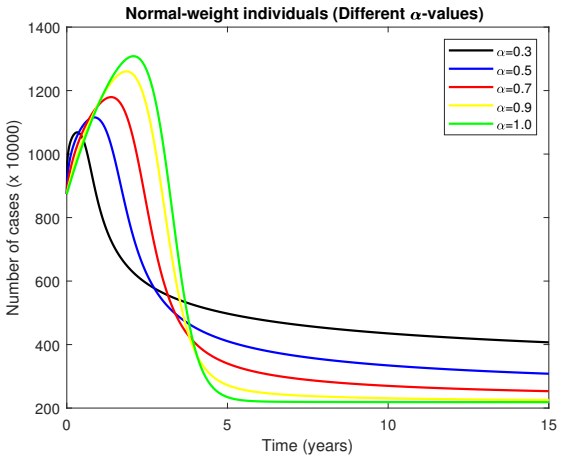


(e) $\Re_0(\gamma, \delta)$ for $\alpha = 1$

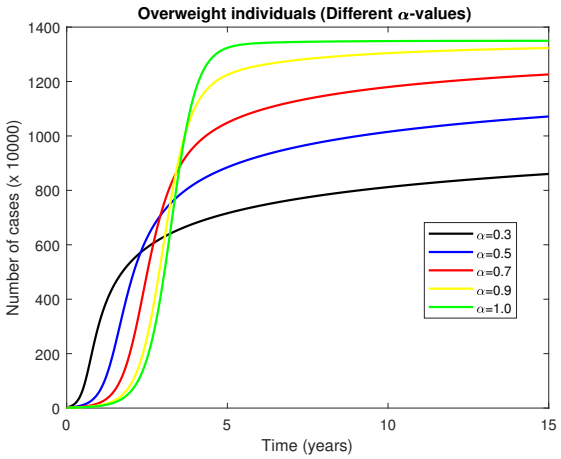
Figure 5.4: Joint variation of δ^α and γ^α in $\Re_0$ for different $\alpha-$values.

5.2. Compartmental study

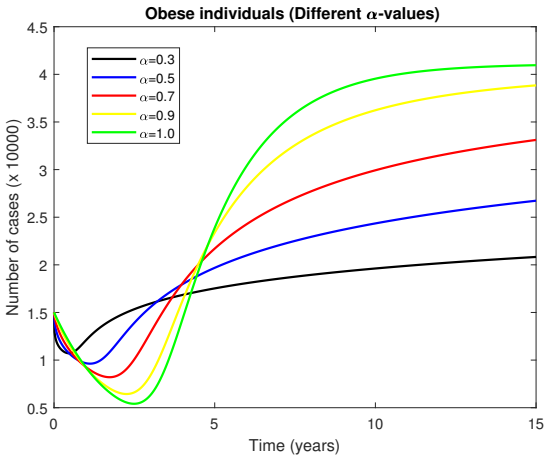
In the compartment of people with normal-weight for the different $\alpha-$values under study, we have a growth in this compartment, where for higher α's a greater number of cases are reported. Then, begins a decrease where the lowest values are reached for the higher $\alpha-$values. This means that for higher $\alpha-$values the output of this compartment is higher, which is the opposite objective because the idea is to maintain a higher number of people with ideal weight, see Figure 5.5a. Quantitatively, we started in this compartment with 874.1400, and at the end of the study (15 years) for $\alpha = 0.3$ which reported the highest number of cases was 407.016 which means a reduction of 467.124 (more than half with respect to the initial value). For $\alpha = 1$, at the end of the study reported 218.687 meaning a reduction from the initial value, this means a reduction of 655.453. Observing Figure 5.5, the other behavior for $\alpha-$values studied is between 0.3 and 1, (see Figure 5.5a), so we can conclude that in this compartment at the end of the study, the outputs are significant and the objective is to achieve the highest number of healthy people.
In the compartment of overweight individuals at the beginning of the study, there is a growth that then stabilizes, where the final value is significantly higher than the initial value for all the fractional orders studied. During the longer time of the study and at the end of the study, higher $\alpha-$values report higher number of overweight individuals, see Figure 5.5b. At the end of the study for $\alpha = 0.3$ (lowest $\alpha-$value that reported the lowest reported number of cases) was 860.304 and for $\alpha = 1$ (highest $\alpha-$value that reported the highest number of cases) was 1,349.65 which represents a high number of reported cases considering that we started with 1.2000. All these cases will become obese or diabetic if they do not improve their lifestyle, which affects their health significantly.
For the compartment of obese individuals at the beginning of the study there is a decrease in the number of cases for all the $\alpha-$values under study, see Figure 5.5c. Approximately the number of cases reported with obesity starts to increase for the different $\alpha-$values reaching the highest values reported for the highest $\alpha-$values. We can observe that for $\alpha = 1$ it was reported 4.09616, and for $\alpha = 0.3$ it was 2.08284, the other fractional orders studied the reported values are between $\alpha = 0.3$ and $\alpha = 1$. Remembering that we started with 1.5000, the increase of individuals in this compartment is qualitatively significant. In comparison with the overweight compartment in the obese, a lower number was reported, but if the person does not change lifestyle from overweight to obese, will become obese, see Figures 5.5c and 5.5d.
In the diabetics compartment during the whole study there is a growth of the reported cases with respect to the initial value 100.0000, see Figure 5.5d. We can observe that overweight and obesity have a strong impact on diabetes. At the beginning lower $\alpha-$values report a higher number of cases but approximately at 3 years of study the opposite process happens and at the end of the study higher $\alpha-$values report a higher number of diabetics. Quantitatively, we have at the end of the study that for $\alpha = 0.3$ it was reported 874.534, and for $\alpha = 1$ it was 5,379.01 and as the other $\alpha-$values studied the values reported are between those reported for $\alpha = 0.3$ and $\alpha = 1.0$ then the study at the end reports that the diabetic cases are in the interval $[874.534; 5,379.01]$ which is a significant increase with respect to the initial value 100.0000. This evidences the need to apply control in overweight and obese cases due to the impact they have on diabetes and that upon reaching the diabetic state the situation is irreversible because diabetes has no cure at present.
All the results obtained contribute to providing information on the behavior of overweight and obesity obtained from the model. By studying different $\alpha-$values, it allows us to observe different behaviors and with them design different, more effective strategies for controlling overweight and obesity, taking into account several possible scenarios (depending on the $\alpha-$value) and appropriate time. In this case, we show the need for control strategies due to the growth of diabetics in the population coming from the increase in overweight and obese people, because this disease has a strong influence on the increase in diabetes in the population.
The variation of the parameter β_1 in the model was studied; this is directly associated with the social impact that leads a person with normal-weight to become overweight. We only studied the overweight compartment because the construction of the model will directly affect that compartment. The variation of this parameter independently will show us if only social pressure will affect the dynamics. We can observe that when we increase the $\alpha-$value for all the different values of β_1 studied ($\beta_1 = 0.1, 0.25, 0.40, 0.50, 0.70$) we have an increase in the number of overweight people. The number of overweight people in the population increases over time and the greater β_1 the number reported is higher. This increase for these variations of the parameter and the fractional order is not quantitatively or qualitatively significant, since the variation in the number of individuals is not impactful and the tendency of overweight behavior does not change for the variations of the parameter, see Figures 5.6a-5.6e.



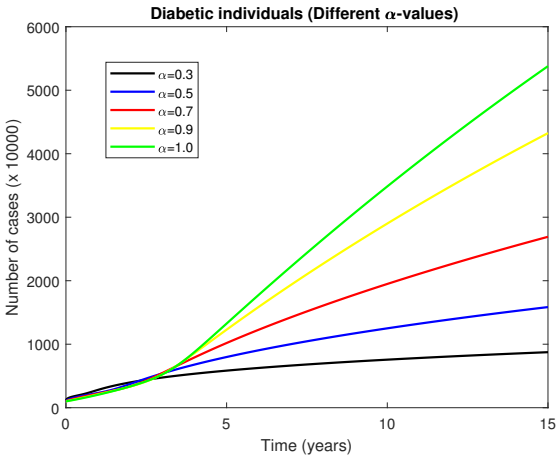
(a) Normal-Weight Individuals



(b) Overweight Individuals

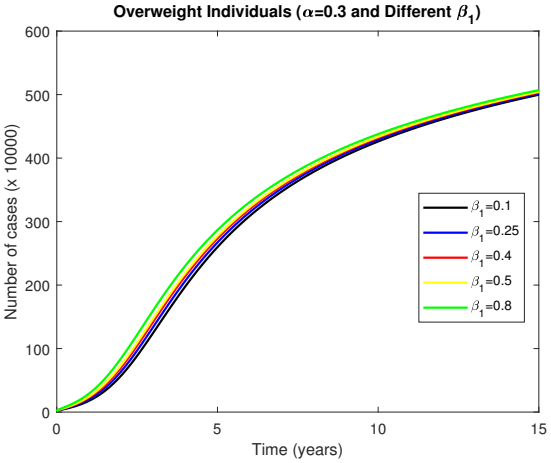


(c) Obese Individuals

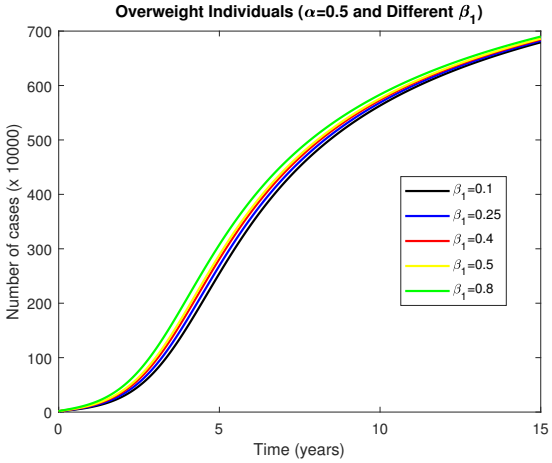


(d) Diabetic Individuals

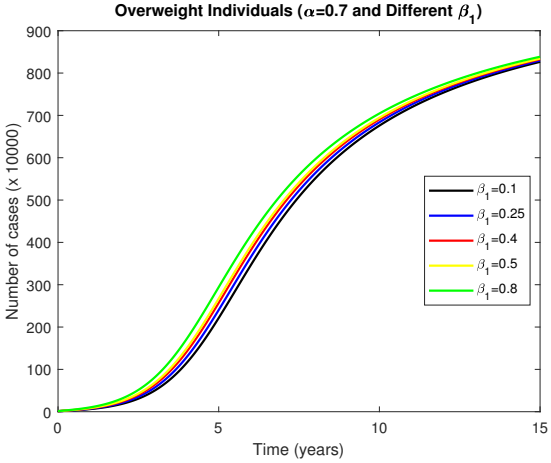
Figure 5.5: Behavior of the compartments of Model (3.1) for the scenario under study and different $\alpha-$values.



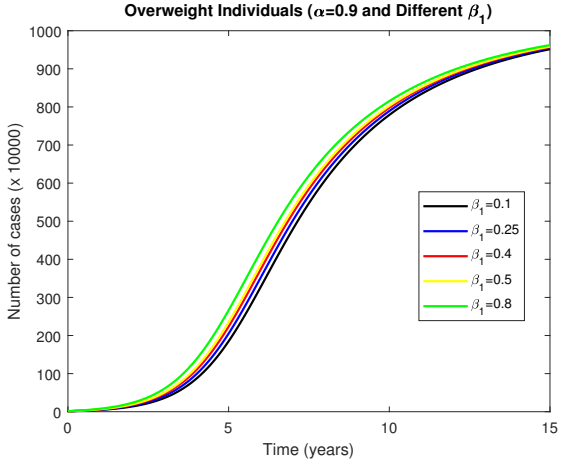
(a) Variation of β_1 for $\alpha = 0.3$



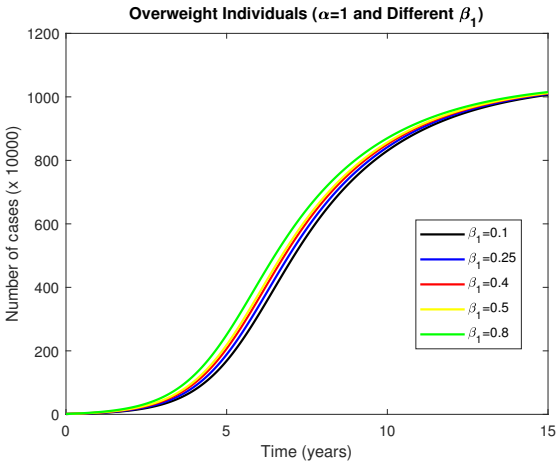
(b) Variation of β_1 for $\alpha = 0.5$



(c) Variation of β_1 for $\alpha = 0.7$



(d) Variation of β_1 for $\alpha = 0.9$



(e) Variation of β_1 for $\alpha = 1$, overweight individuals

Figure 5.6: Behavior of the overweight compartment when $\beta_1 = 0.05, 0.1, 0.3, 0.5, 0.7$ for different $\alpha-$values, overweight compartment.

6. Conclusion

In this paper, we presented a mathematical model to study obesity and overweight, and their relationship with diabetes. The model enables us to study the behavior of normal-weight, overweight, and obese individuals, as well as their interactions and the influence of social factors (social pressure). For the construction of the model, we used fractional order derivatives in the Caputo sense taking advantage of this modeling technique with respect to the memory effect. Using the next-generation matrix method, we find the basic reproduction number and show the local and global stability of the disease-free equilibrium point. Sensitivity indices were calculated with respect to the basic reproduction number for the parameters associated with weight gain due to social pressure and the diagnosis of diabetes not associated with body weight. The sensitivity index is negative for both, which means that an increase in these parameters will mean a decrease in the $\Re_0$. This means that as people are diagnosed with diabetes for reasons other than body weight, they enter the diabetic compartment and do not actively influence other individuals and also that social pressure alone will not significantly influence the increase in overweight and obesity. To validate the model, we performed computational simulations with data from the literature and other assumed data validated by specialists. In the study of the weight and diabetic groups, the normal-weight population decreased significantly, the overweight and obese increased and the diabetic group grew throughout the study. We can observe that the higher the $\alpha-$values, the higher the number of cases reported for the overweight, obese, and diabetic groups, and the opposite occurs for the normal-weight groups. This behavior shows the need to strategies with the objective of ensuring that people achieve a healthy lifestyle and a normal-weight and consequently reduce the impact of diabetes due to causes associated with body weight. We study the variation in the simulations of the parameter β_1, since this parameter is related to weight gain due to social pressure (which can lead to an unhealthy lifestyle). The study of this parameter will allow us to know if social pressure is an important factor in the increase in overweight people and, as a consequence, in the increase in cases of diabetes. This variation in the impact of social pressure (β_1) in our study did not cause significant effects on the dynamics (it was checked directly in the $\Re_0$ with the sensitivity analysis), particularly in the increase in overweight individuals. We can conclude that for our study, intervening only in the social pressure exerted on the individual will not be able to reduce the number of overweight individuals, so we have to intervene in human interactions. In future work, we intend to study different real scenarios and estimate parameters for them and propose, based on the results obtained regarding the behavior of the system, the problem of optimal control of reducing overweight and obesity and, as consequences, diabetes.

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