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Research Article

Analysis of the mathematical model's stability describing the prevalence of obesity and its role in diabetes

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Abstract

We propose a continuous-time dynamic epidemiological model that describes the prevalence of obesity in a population and examines the relationship between weight gain and the development of diabetes, particularly type 2 diabetes. We investigate both the local and global stability of the disease-free and endemic equilibrium points using the Routh–Hurwitz criteria and the construction of appropriate Lyapunov functions. Furthermore, a sensitivity analysis of the

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basic reproduction number R_0 is carried out to determine the relative impact of each model parameter. Numerical simulations performed using MATLAB are presented to illustrate the dynamical behavior of the proposed model. The simulation results are consistent with and support the theoretical findings.

AMS subject classifications (2020): Primary 92D30; Secondary 34A34, 92C50, 37N25.

Keywords: Mathematical model; Stability; Sensitivity; Reproduction number; Asymptotically stable.

1 Introduction

An abnormal or excessive accumulation of body fat that has a detrimental effect on health is referred to as overweight or obesity [7]. This accumulation occurs when a person consumes more dietary energy than their body requires [41]. A common tool used to diagnose obesity is the body mass index (BMI), which is calculated using an individual's height and weight. According to the values of this index (expressed in kilograms per square meter), the World Health Organization (WHO) has established thresholds for overweight, obesity, and morbid obesity: From 25 to 30, from 30 to 40, and above 40, respectively [47].

Numerous factors, including genetic predisposition [43], unhealthy dietary habits, and sedentary lifestyles [37], may contribute to obesity. According to the WHO, approximately 13% of the global population is obese [47]. A report by this organization [48] indicates that obesity has reached nearly pandemic proportions in Europe, with 59% of adults and 8% of children under five years old being overweight. Moreover, one in three school-aged children is affected by this condition. The same study [48] also revealed a link between obesity and a variety of diseases, such as diabetes, cardiovascular diseases, and at least 13 different types of cancer, with an estimated 200,000 new cases each year. Obesity can also lead to disability and may result in premature death.

Diabetes is a chronic metabolic disease characterized by high blood glucose levels and is primarily associated with aging and obesity [2]. Approximately 422 million people worldwide live with diabetes, and about one in ten adults in the UK has type 2 diabetes (T2D) [32, 45]. According to the WHO [44], between 2000 and 2019 the number of premature deaths related to diabetes increased by 3%. In particular, obesity represents one of the most significant risk factors for T2D [35, 2]. A recent US Census report and estimates from the CDC indicate that about 11% of Americans have diabetes and approximately 14% are obese [28, 27].

Our study focuses exclusively on **T2D**, which is the most prevalent form globally, accounting for approximately 90–95% of all diabetes cases [46, 3]. Type 1 and gestational diabetes are much less frequent, and their etiology and risk factors differ significantly. Therefore, T2D is the most relevant for modeling the link with obesity.

A substantial body of clinical and epidemiological evidence has established a strong relationship between obesity and T2D. Excess body fat, particularly visceral adiposity, plays a central role in the development of insulin resistance, which is one of the primary mechanisms underlying T2D [17]. Adipose tissue is an active endocrine organ that secretes adipokines and pro-inflammatory cytokines such as TNF-α and IL-6. These mediators contribute to chronic low-grade inflammation and interfere with insulin signaling pathways in peripheral tissues.

Obesity is also closely associated with metabolic syndrome, a cluster of metabolic abnormalities including abdominal obesity, dyslipidemia, hypertension, and impaired glucose metabolism, which significantly increases the risk of developing T2D [14]. Behavioral and environmental factors, such as unhealthy diets, physical inactivity, and sedentary lifestyles, further exacerbate these metabolic disturbances and contribute to the growing prevalence of both obesity and diabetes worldwide. The main biological pathway linking obesity to T2D is summarized in Figure 1.

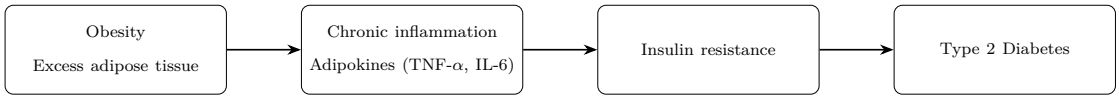

Figure 1: Conceptual biological pathway linking obesity to T2D. Excess adipose tissue promotes chronic low-grade inflammation and the secretion of adipokines and pro-inflammatory cytokines. These processes impair insulin signaling, leading to insulin resistance and ultimately the development of T2D.

Numerous academic studies have investigated the dynamics of various epidemiological phenomena in the context of combating different diseases (such as alcoholism, smoking, COVID-19, and monkeypox) [13, 25, 26, 9, 33, 12, 18, 22, 5, 21, 38, 39, 42, 15, 6, 24, 31, 8, 36, 11].

Since obesity can spread through social interactions and behavioral influence [4], the dynamics of this condition have been analyzed using several epidemiological models, for instance [16, 34, 19, 1, 9]. The authors in [16] investigated childhood obesity and, through numerical simulations, showed that eating habits are a major contributing factor. By performing a numerical covariate analysis of an SIS epidemic model, the authors in [34] demonstrated that preventive measures become more effective when adult obesity is considered. Similar results were obtained by the authors in [19] using an SIS difference equation model. However, these studies considered continuous control strategies, which may be inefficient or impractical in certain contexts. In [1], a deterministic model incorporating time-dependent control strategies was proposed, including dietary programs, healthy living campaigns, and therapeutic interventions. In our previous work

[9], we studied a discrete mathematical model for the prevalence of obesity and applied four control strategies to reduce its spread: Educational programs, dietary and exercise interventions, drug-based medical treatment, and surgical intervention. The optimal control strategies were determined using the discrete-time Pontryagin maximum principle.

In previous work [10], we also investigated the strong relationship between obesity and diabetes using a continuous mathematical model, in which optimal strategies were proposed to limit their spread.

Building upon previous models, our proposed continuous-time model synthesizes key elements from both works:

- From [9], we adopt the modeling framework and the control strategies for obesity prevalence.
- From [20], we incorporate components describing the dynamics of T2D in the population.

Importantly, our model extends these previous works [9], [20], by integrating the dynamics of obesity and T2D simultaneously in a unified continuous-time framework, allowing for joint sensitivity analysis and optimal control strategies that target both conditions. This novel contribution provides a more comprehensive understanding of the interaction between obesity and diabetes, which was not addressed in the previous studies.

In the present article, we introduce the following contributions:

- We employ the next-generation matrix method to determine the basic reproduction number (R_0).
- Local and global stability analyses are performed.
- A sensitivity analysis is conducted to determine the relative importance of the model parameters in the transmission dynamics of these two diseases.

The remainder of this paper is organized as follows: Section 2 presents the formulation of the mathematical model. Section 3 is devoted to the computation of the basic reproduction number. Section 4 deals with the stability analysis of the model. Section 5 presents the numerical simulations and discussion of the results. Section 6 outlines the perspectives and possible extensions of the model. Finally, Section 7 concludes the paper.

2 The mathematical model and its fundamental characteristics

2.1 The mathematical model

We propose an $SWRIEDC$ model that highlights the detrimental role of overweight in the development of diabetes, particularly T2D, and describes the dynamics of obesity and diabetes epidemics within a given population. The total population is divided into seven compartments. A schematic representation of the proposed model is illustrated in Figure 2.

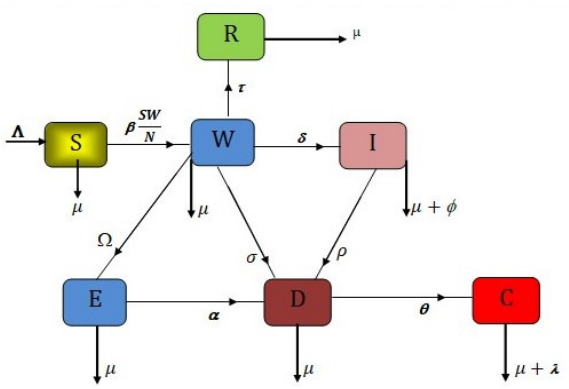

Figure 2: Flowchart of the proposed model.

Table 1 describes the different population classes considered at time t.

Table 1: Definitions of the state variables

Variable	Meaning
$S(t)$	Individuals susceptible to obesity
$W(t)$	Overweight or moderately obese individuals
$I(t)$	Obese individuals
$R(t)$	Recovered individuals
$E(t)$	Pre-diabetic individuals
$D(t)$	Diabetic individuals without complications
$C(t)$	Diabetic individuals with complications

Let

$$N(t) = S(t) + W(t) + I(t) + R(t) + E(t) + D(t) + C(t)$$

denote the total population at time t.

The dynamics of the model are described by the following system of seven nonlinear differential equations:

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda - \mu S(t) - \beta \frac{S(t)}{N} W(t), \\ \frac{dW(t)}{dt} = \beta \frac{S(t)}{N} W(t) - (\tau + \sigma + \mu + \delta + \Omega) W(t), \\ \frac{dR(t)}{dt} = \tau W(t) - \mu R(t), \\ \frac{dI(t)}{dt} = \delta W(t) - (\phi + \rho + \mu) I(t), \\ \frac{dE(t)}{dt} = \Omega W(t) - (\mu + \alpha) E(t), \\ \frac{dD(t)}{dt} = \sigma W(t) + \rho I(t) + \alpha E(t) - (\mu + \theta) D(t), \\ \frac{dC(t)}{dt} = \theta D(t) - (\mu + \lambda) C(t), \end{cases} \tag{1}$$

subject to the initial conditions

$$S(0), W(0), R(0), I(0), E(0), D(0), C(0) > 0.$$

The model parameters are described in Table 2.

Table 2: Definitions and values of the parameters

Parameter	Definition	Value	Reference
Λ	Recruitment rate of individuals susceptible to obesity	4×10^6	Assumed
μ	Natural death rate	0.02	[9]
β	Rate at which susceptible individuals become overweight due to social influence	0.45	[9]
τ	Recovery rate of overweight individuals	0.07	Assumed
δ	Rate at which overweight individuals become obese	0.05	[9]
λ	Mortality rate due to diabetes complications	0.001	[20]
ϕ	Obesity-related mortality rate	0.03	Assumed
ρ	Rate at which obese individuals develop diabetes without complications	0.06	Assumed
Ω	Rate at which overweight individuals become pre-diabetic	0.03	Assumed
α	Rate at which pre-diabetic individuals develop diabetes without complications	0.06	[20]
σ	Rate at which overweight individuals develop diabetes without complications	0.025	Assumed
θ	Rate at which diabetic individuals develop complications	0.08	[20]

2.2 Fundamental properties of the model

2.2.1 Invariant region

It must be shown that all solutions of system (1) with positive initial conditions remain positive for all $t > 0$. This result is established by the following lemma.

Lemma 1. All feasible solutions $S(t), W(t), R(t), I(t), E(t), D(t)$, and $C(t)$ of system (1) are bounded in the region

$$\Delta = \left\{ (S, W, R, I, E, D, C) \in \mathbb{R}_+^7 : S + W + R + I + E + D + C \leq \frac{\Lambda}{\mu} \right\}. \tag{2}$$

Proof. From system (1), we have

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dW}{dt} + \frac{dR}{dt} + \frac{dI}{dt} + \frac{dE}{dt} + \frac{dD}{dt} + \frac{dC}{dt},$$

which yields

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - \phi I(t) - \lambda C(t).$$

Hence,

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N(t).$$

Solving this differential inequality gives

$$N(t) \leq \frac{\Lambda}{\mu} + N(0)e^{-\mu t}.$$

Taking the limit as $t \to +\infty$ yields

$$\limsup_{t \to +\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Therefore,

$$S(t) + W(t) + R(t) + I(t) + E(t) + D(t) + C(t) \leq \frac{\Lambda}{\mu}.$$

Thus, the region Δ defined in (2) is positively invariant for system (1). Consequently, it is sufficient to study the dynamics of system (1) in the region Δ of nonnegative solutions.

□

2.2.2 Positivity of solutions

We establish the following theorem.

Theorem 1. If $S(0), W(0), R(0), I(0), E(0), D(0)$, and $C(0)$ are positive, then the solutions $S(t), W(t), R(t), I(t), E(t), D(t)$, and $C(t)$ of system (1) remain nonnegative for all $t > 0$.

Proof. From the first equation of system (1), we have

$$\frac{dS(t)}{dt} = \Lambda - A(t)S(t),$$

where

$$A(t) = \beta \frac{W(t)}{N} + \mu.$$

Multiplying both sides by $\exp\left(\int_0^t A(s)ds\right)$ gives

$$\frac{d}{dt}\left[S(t)\exp\left(\int_0^t A(s)ds\right)\right] = \Lambda \exp\left(\int_0^t A(s)ds\right).$$

Integrating from 0 to t yields

$$S(t) = S(0)e^{-\int_0^t A(s)ds} + \Lambda e^{-\int_0^t A(s)ds}\int_0^t e^{\int_0^w A(s)ds}dw \geq 0.$$

Thus, $S(t) \geq 0$.

Similarly, using the remaining equations of system (1), we obtain

$$W(t) \geq W(0)\exp\left(-\int_0^t (\beta\frac{S(s)}{N} - (\mu + \tau + \sigma + \delta + \Omega))ds\right) \geq 0, \tag{3}$$

$$R(t) \geq R(0)e^{-\mu t} \geq 0, \tag{4}$$

$$I(t) \geq I(0)e^{-(\mu+\phi+\rho)t} \geq 0, \tag{5}$$

$$E(t) \geq E(0)e^{-(\mu+\alpha)t} \geq 0, \tag{6}$$

$$D(t) \geq D(0)e^{-(\mu+\theta)t} \geq 0,$$

$$C(t) \geq C(0)e^{-(\mu+\lambda)t} \geq 0.$$

Therefore, all solutions of system (1) remain positive for all $t \geq 0$.

□

3 Model stability and sensitivity analysis

3.1 Disease-free equilibrium point

The disease-free equilibrium (DFE) of system (1), corresponding to the absence of obesity in the population, is easily obtained. At this equilibrium we have

$$W^0 = 0, \quad R^0 = 0, \quad I^0 = 0, \quad E^0 = 0, \quad D^0 = 0, \quad C^0 = 0.$$

From the first equation of system (1), we obtain

$$\frac{dS(t)}{dt}(S^0, 0, 0, 0, 0, 0, 0) = \Lambda - \mu S^0 = 0, \tag{7}$$

which yields

$$S^0 = \frac{\Lambda}{\mu}. \tag{8}$$

Therefore, the DFE point is given by

$$E^0_{eq} = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\right). \tag{9}$$

3.2 The basic reproduction number (R_0)

The basic reproduction number is a threshold parameter that governs the overall dynamics of the disease. It represents the average number of secondary infections produced by a single infected individual during its entire infectious period in a fully susceptible population.

To compute R_0, we use the next generation matrix method [40, 29, 30].

Considering the infected compartments, system (1) can be rewritten as

$$\begin{cases} \frac{dW(t)}{dt} = \beta\frac{S(t)}{N}W(t) - (\tau+\sigma+\mu+\delta+\Omega)W(t) = \beta\frac{S(t)}{N}W(t) - K_1W(t), \\ \frac{dR(t)}{dt} = \tau W(t) - \mu R(t), \\ \frac{dI(t)}{dt} = \delta W(t) - (\phi+\rho+\mu)I(t) = \delta W(t) - K_2I(t), \\ \frac{dE(t)}{dt} = \Omega W(t) - (\mu+\alpha)E(t) = \Omega W(t) - K_3E(t), \\ \frac{dD(t)}{dt} = \sigma W(t) + \rho I(t) + \alpha E(t) - (\mu+\theta)D(t) = \sigma W(t) + \rho I(t) + \alpha E(t) - K_4D(t), \\ \frac{dC(t)}{dt} = \theta D(t) - (\mu+\lambda)C(t) = \theta D(t) - K_5C(t), \end{cases} \tag{10}$$

where

$$K_1 = \tau+\sigma+\mu+\delta+\Omega, \quad K_2 = \phi+\rho+\mu, \quad K_3 = \mu+\alpha, \quad K_4 = \mu+\theta, \quad K_5 = \mu+\lambda. \tag{11}$$

The system can be written in matrix form as

$$\frac{dX}{dt} = F(X) - V(X),$$

where F represents the new infection terms and V represents the transition terms.

At the DFE E^0_{eq} we have $S = \frac{\Lambda}{\mu}$. The matrices F and V are therefore given by

$$F = \begin{bmatrix} \beta\frac{\Lambda}{N\mu} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 & 0 \\ -\tau & \mu & 0 & 0 & 0 & 0 \\ -\delta & 0 & K_2 & 0 & 0 & 0 \\ -\Omega & 0 & 0 & K_3 & 0 & 0 \\ -\sigma & 0 & -\rho & -\alpha & K_4 & 0 \\ 0 & 0 & 0 & 0 & -\theta & K_5 \end{bmatrix}.$$

The next-generation matrix is given by FV^{-1}. The spectral radius of this matrix yields the basic reproduction number. At the DFE we have $N_0 = \frac{\Lambda}{\mu}$. Substituting this value into the expression of R_0 gives

$$R_0 = \frac{\beta\Lambda}{N_0\mu(\tau+\sigma+\mu+\delta+\Omega)} = \frac{\beta}{\tau+\sigma+\mu+\delta+\Omega} = \frac{\beta}{K_1}. \tag{12}$$

3.3 Endemic equilibrium point

The endemic equilibrium represents a steady state in which the disease persists in the population.

Let

$$E^*_{eq} = (S^*, W^*, R^*, I^*, E^*, D^*, C^*)$$

denote the endemic equilibrium point obtained by setting the right-hand side of system (1) equal to zero.

The equilibrium values are given by

$$\begin{cases} S^* &= \frac{NK_1}{\beta}, \\ W^* &= \frac{N\mu}{\beta}(R_0-1), \\ R^* &= \frac{N\tau}{\beta}(R_0-1), \\ I^* &= \frac{N\delta\mu}{\beta K_2}(R_0-1), \\ E^* &= \frac{N\Omega\mu}{\beta K_3}(R_0-1), \\ D^* &= \frac{N\mu}{\beta K_4}\left(\sigma+\frac{\delta\rho}{K_2}+\frac{\alpha\Omega}{K_3}\right)(R_0-1), \\ C^* &= \frac{\theta N\mu}{\beta K_4 K_5}\left(\sigma+\frac{\delta\rho}{K_2}+\frac{\alpha\Omega}{K_3}\right)(R_0-1). \end{cases} \tag{13}$$

3.4 Local stability analysis

We analyze the local stability of the equilibrium points E^0_{eq} and E^*_{eq}.

3.4.1 Local stability of the DFE

Theorem 2. The DFE E^0_{eq} is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The Jacobian matrix of system (1) is given by

$$J = \begin{bmatrix} -\beta\frac{W}{N} - \mu & -\beta\frac{S}{N} & 0 & 0 & 0 & 0 & 0 \\ \beta\frac{W}{N} & \beta\frac{S}{N} - K_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & -\mu & 0 & 0 & 0 & 0 \\ 0 & \delta & 0 & -K_2 & 0 & 0 & 0 \\ 0 & \Omega & 0 & 0 & -K_3 & 0 & 0 \\ 0 & \sigma & 0 & \rho & \alpha & -K_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & -K_5 \end{bmatrix}.$$

Evaluating this matrix at E_{eq}^0 yields

$$J(E_{eq}^0) = \begin{bmatrix} -\mu & -\beta\frac{S^0}{N} & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta\frac{S^0}{N} - K_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & -\mu & 0 & 0 & 0 & 0 \\ 0 & \delta & 0 & -K_2 & 0 & 0 & 0 \\ 0 & \Omega & 0 & 0 & -K_3 & 0 & 0 \\ 0 & \sigma & 0 & \rho & \alpha & -K_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & -K_5 \end{bmatrix}.$$

The eigenvalues are

$$-\mu, \quad K_1(R_0 - 1), \quad -K_2, \quad -K_3, \quad -K_4, \quad -K_5.$$

Thus, if $R_0 < 1$, all eigenvalues have negative real parts. Therefore, the DFE is locally asymptotically stable. Conversely, if $R_0 > 1$, the equilibrium is unstable.

□

3.4.2 Bifurcation behavior at $R_0 = 1$

The change in stability of the DFE at $R_0 = 1$ indicates the occurrence of a transcritical bifurcation. This behavior is consistent with the structure of system (1), where the transmission process is governed by the bilinear incidence term $\beta\frac{S}{N}W$.

In epidemic models with bilinear incidence and without nonlinear feedback mechanisms, the disease dynamics are typically characterized by a single threshold determined by the basic reproduction number R_0. In this case, the DFE is stable when $R_0 < 1$ and loses stability when $R_0 > 1$, while an endemic equilibrium emerges.

From the analytical expressions of the endemic equilibrium given in (13), all endemic state variables are proportional to $(R_0 - 1)$. Therefore, the endemic equilibrium exists only when $R_0 > 1$, which rules out the possibility of backward bifurcation in the present model.

3.4.3 Local stability of the endemic equilibrium

Theorem 3. For system (1), the endemic equilibrium E^*_{eq} is unstable if $R_0 \leq 1$ and locally asymptotically stable if $R_0 > 1$.

The proof of Theorem 3 is provided in Appendix A.

3.5 Global stability analysis

To establish the global asymptotic stability of system (1), we use Lyapunov's method. We first study the global stability of the DFE E^0_{eq}.

3.5.1 Global stability of the DFE

Theorem 4. The DFE E^0_{eq} is globally asymptotically stable if $R_0 \leq 1$ and unstable otherwise.

Proof. Consider the Lyapunov function

$$V(S, W) = \frac{1}{2}\left[(S - S^0) + W\right]^2 + \frac{N}{\beta}(\mu + K_1)W.$$

Differentiating with respect to time yields

$$\frac{dV}{dt} = -\mu(S - S^0)^2 - K_1 W^2 - \frac{N}{\beta}(\mu + K_1)K_1(1 - R_0)W.$$

Thus, $\frac{dV}{dt} \leq 0$ for $R_0 \leq 1$. Moreover, $\frac{dV}{dt} = 0$ if and only if $S = S^0$ and $W = 0$.

Furthermore, when $W = 0$, the remaining state variables satisfy linear differential equations with negative coefficients. Hence $R(t)$, $I(t)$, $E(t)$, $D(t)$ and $C(t)$ converge exponentially to zero. Therefore, the largest invariant set contained in $\{\dot{V} = 0\}$ reduces to the DFE E^0_{eq}.

According to the LaSalle's invariance principle [23], the equilibrium E^0_{eq} is globally asymptotically stable when $R_0 \leq 1$.

□

3.5.2 Global stability of the endemic equilibrium

Theorem 5. If $R_0 > 1$, then the endemic equilibrium E^*_{eq} of system (1) is globally asymptotically stable.

The proof of Theorem 5 is provided in Appendix B.

4 Sensitivity analysis of R_0

Understanding the relative importance of the parameters involved in the spread of obesity and diabetes is essential. In this context, we perform a sensitivity analysis of the basic reproduction number R_0 in order to evaluate how variations in the parameters of the $SWRIEDC$ model affect its value.

This analysis is carried out by computing the sensitivity indices of R_0 with respect to the model parameters. These indices allow us to determine the relative contribution of each parameter to the transmission dynamics of the disease.

Using the approach proposed by Chitnis, Hyman, and Cushing [5], the normalized forward sensitivity index of R_0 with respect to a parameter n is defined as

$$\Upsilon_n^{R_0} = \frac{\partial R_0}{\partial n} \times \frac{n}{R_0}. \tag{14}$$

If $\Upsilon_n^{R_0} < 0$, then the parameter contributes to disease control. Conversely, if $\Upsilon_n^{R_0} > 0$, the parameter contributes to the spread of the disease.

Recall that the basic reproduction number is given by

$$R_0 = \frac{\beta}{K_1}, \tag{15}$$

where

$$K_1 = \mu + \delta + \tau + \sigma + \Omega.$$

Table 3 presents the sensitivity indices corresponding to the parameters β, μ, δ, σ, τ, and Ω.

The result $\Upsilon_\beta^{R_0} = 1$ indicates that increasing (decreasing) the transmission rate β by a given percentage leads to an increase (decrease) of the same percentage in the basic reproduction number R_0.

On the other hand, the parameters μ, σ, δ, τ, and Ω have negative sensitivity indices, which means that increasing their values contributes to reducing the spread of the disease.

Table 3: Sensitivity indices of the basic reproduction number R_0 with respect to selected parameters

Parameter	Sensitivity index
β	$\Upsilon_{\beta}^{R_0} = 1 > 0$
μ	$\Upsilon_{\mu}^{R_0} = -1 - \frac{\mu}{K_1} < 0$
δ	$\Upsilon_{\delta}^{R_0} = -\frac{\delta}{K_1} < 0$
σ	$\Upsilon_{\sigma}^{R_0} = -\frac{\sigma}{K_1} < 0$
τ	$\Upsilon_{\tau}^{R_0} = -\frac{\tau}{K_1} < 0$
Ω	$\Upsilon_{\Omega}^{R_0} = -\frac{\Omega}{K_1} < 0$

Therefore, strengthening prevention and intervention strategies associated with these parameters may significantly reduce the transmission dynamics of obesity and diabetes.

5 Numerical simulations

In this section, we present numerical simulations of system (1) using different initial conditions. We assume that at each time t the total population satisfies

$$S(t) + W(t) + R(t) + I(t) + E(t) + D(t) + C(t) = N.$$

The parameter values used in the simulations are listed in Table 2. For illustration purposes, we consider

$$N = 1.16 \times 10^7.$$

Case $R_0 < 1$

For the DFE we have

$$E_{eq}^0 = (S^0, W^0, R^0, I^0, E^0, D^0, C^0) = (2 \times 10^8, 0, 0, 0, 0, 0, 0)$$

with

$$R_0 = 0.88 < 1.$$

Starting from different initial values of the state variables, Theorem 4 implies that the DFE E_{eq}^0 of system (1) is globally asymptotically stable in the feasible region Δ(2).

The following curves are obtained for different initial values $S_0, W_0, R_0, I_0, E_0, D_0$, and C_0 (Figures 3–9).

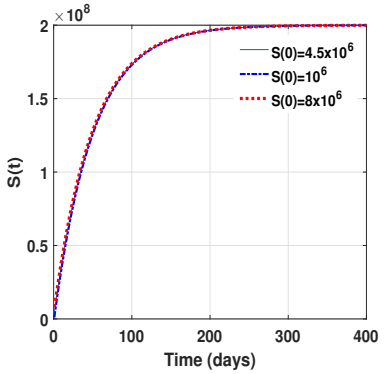

Figure 3: Evolution of $S(t)$ for $R_0 = 0.88$

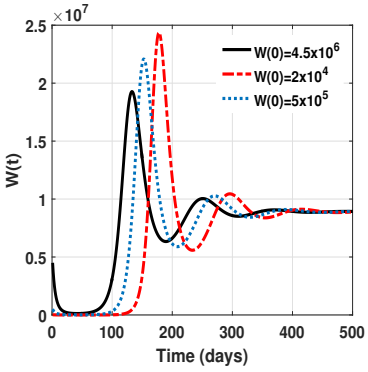

Figure 6: Evolution of $W(t)$ for $R_0 = 0.88$

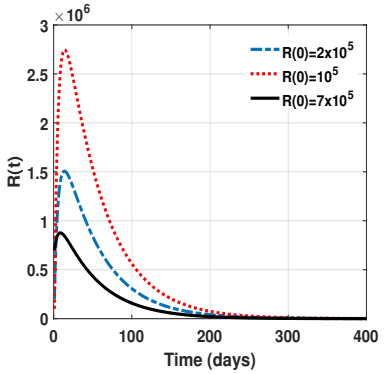

Figure 4: Evolution of $R(t)$ for $R_0 = 0.88$

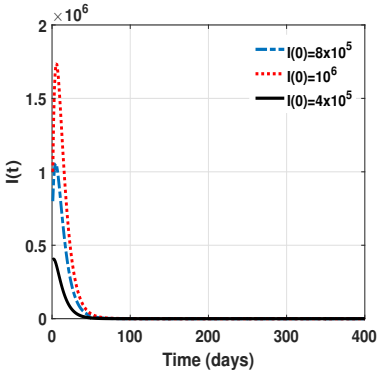

Figure 7: Evolution of $I(t)$ for $R_0 = 0.88$

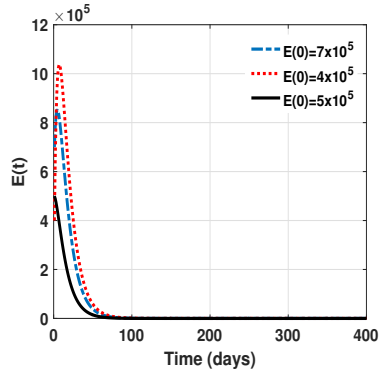

Figure 5: Evolution of $E(t)$ for $R_0 = 0.88$

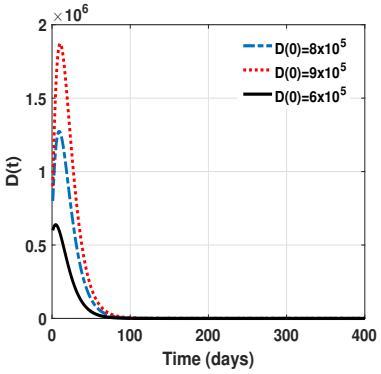

Figure 8: Evolution of $D(t)$ for $R_0 = 0.88$

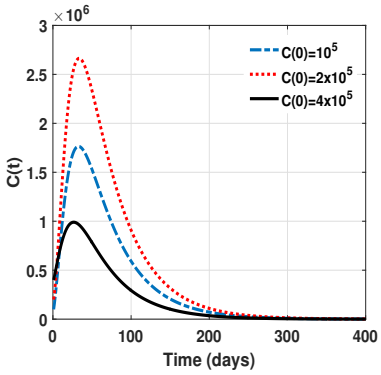

Figure 9: Evolution of $C(t)$ for $R_0 = 0.88$

Figures 3–9 show that the number of susceptible individuals increases and approaches approximately $S_0 = 2 \times 10^8$ (Figure 3). In addition, the number of individuals exposed to diabetes (E) decreases and approaches zero (Figure 5). Similarly, the number of overweight individuals (W), obese individuals (I), diabetic individuals without complications (D), and diabetic individuals with complications (C) gradually decreases and converges to zero (Figures 6–9).

The number of recovered individuals (R) initially increases, then decreases and eventually approaches zero (Figure 4). Therefore, when $R_0 = 0.88 < 1$, the solution converges to the DFE point

$$E^0_{eq} = (S_0, 0, 0, 0, 0, 0, 0).$$

Consequently, system (1) is globally asymptotically stable in this case.

Case $R_0 > 1$

Keeping the same parameter values given in Table 2, we obtain the endemic equilibrium

$$E^*_{eq} = (S^*, W^*, R^*, I^*, E^*, D^*, C^*)$$

with

$$(1.13 \times 10^8, 8.9 \times 10^6, 3.12 \times 10^7, 4 \times 10^6, 3.34 \times 10^6, 6.67 \times 10^6, 2.53 \times 10^7)$$

and

$$R_0 = 1.77 > 1.$$

According to Theorem 5, the endemic equilibrium E^*_{eq} of system (1) is globally asymptotically stable in the feasible region $\Delta(2)$. The corresponding numerical simulations are shown in Figures 10–16.

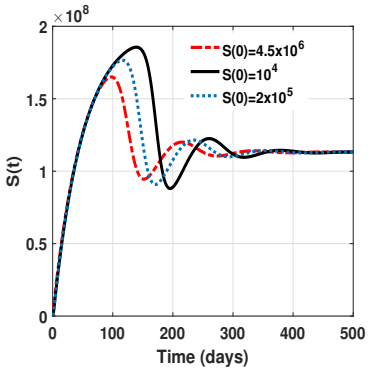

Figure 10: Evolution of $S(t)$ for $R_0 = 1.77$

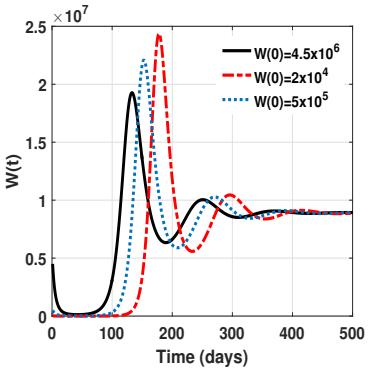

Figure 13: Evolution of $W(t)$ for $R_0 = 1.77$

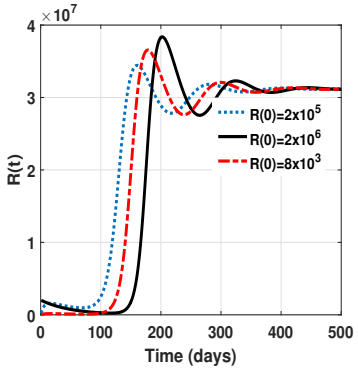

Figure 11: Evolution of $R(t)$ for $R_0 = 1.77$

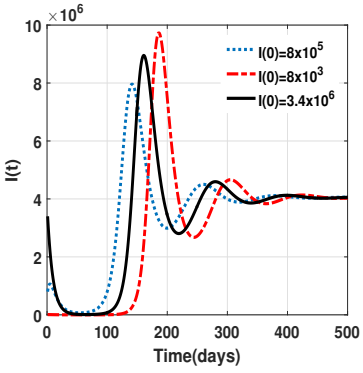

Figure 14: Evolution of $I(t)$ for $R_0 = 1.77$

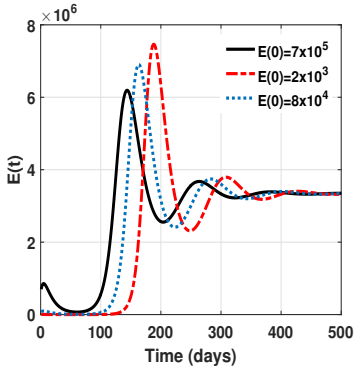

Figure 12: Evolution of $E(t)$ for $R_0 = 1.77$

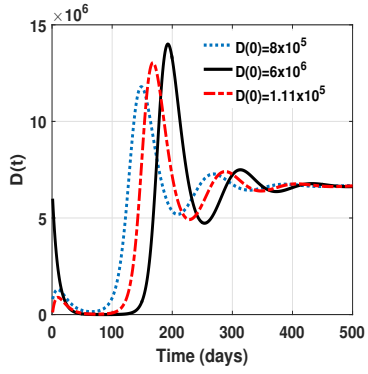

Figure 15: Evolution of $D(t)$ for $R_0 = 1.77$

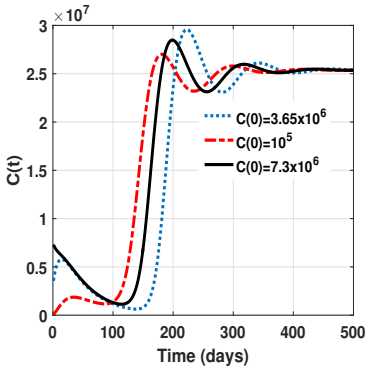

Figure 16: Evolution of $C(t)$ for $R_0 = 1.77$

From these figures we observe that the number of susceptible individuals initially increases and then gradually decreases until it stabilizes at approximately

$$S^* \approx 1 \times 10^8.$$

The number of overweight (W) and obese (I) individuals increases initially, reaches a peak after approximately five months, and then stabilizes after about one year at

$$W^* \approx 9 \times 10^6, \qquad I^* \approx 4 \times 10^6.$$

The number of individuals exposed to diabetes (E) and diabetic patients without complications (D) follows a similar trend: The number remains relatively low during the first 100 days, then increases rapidly before stabilizing after about one year at

$$E^* \approx 3 \times 10^6, \qquad D^* \approx 6.6 \times 10^6.$$

According to Figure 16, the number of diabetic individuals with complications (C) increases after approximately 100 days, reaches a maximum around day 200, and then gradually decreases until stabilizing at

$$C^* \approx 2.5 \times 10^7$$

around day 300.

Finally, the number of recovered individuals (R) initially increases rapidly to a maximum value and then decreases slightly before stabilizing after approximately one year at

$$R^* \approx 3 \times 10^7.$$

Therefore, when $R_0 > 1$, the solution converges to the endemic equilibrium

$$E^*_{eq}(S^*, W^*, R^*, I^*, E^*, D^*, C^*).$$

Hence, the proposed model is globally asymptotically stable in the endemic case.

6 Perspectives and future work

While the present model provides valuable insights into the interaction between obesity and T2D, several limitations should be noted:

- The model focuses on T2D and does not include type 1 or gestational diabetes.
- It does not account for the fact that some obese individuals may never develop diabetes, nor that some nonobese individuals may develop it.
- Age, demographic, and other individual-specific risk factors are not explicitly incorporated.
- Intermediate conditions such as metabolic syndrome are not included.

Addressing these limitations will be the focus of future research. Possible extensions include as follows:

- Expanding the model to include additional compartments representing different risk profiles.
- Incorporating age, gender, and other demographic factors to improve realism.
- Including metabolic syndrome and other intermediate risk factors for both obesity and T2D.
- Exploring interactions between T2D and other chronic diseases for more comprehensive public health insights.

These extensions aim to enhance the model's applicability and provide a more detailed understanding of population-level dynamics and targeted intervention strategies.

7 Conclusion

In this paper, we have studied a continuous mathematical model, $SWRIEDC$, for obesity-related diabetes, which describes the dynamics of individuals affected by these two diseases. We derived the basic reproduction number of the system (1) as

$$R_0 = \frac{\Lambda\beta}{\mu N(\mu+\delta+\tau+\sigma+\Omega)}.$$

To identify the parameters that most significantly influence R_0, we performed a sensitivity analysis. Using the theory of nonlinear system stability, we analyzed the local and global behavior of the model. The DFE E^0_{eq} is locally asymptotically stable if $R_0 \leq 1$, whereas the endemic equilibrium E^*_{eq} is locally asymptotically stable when $R_0 > 1$.

Furthermore, by constructing appropriate Lyapunov functions, we showed that E^0_{eq} is globally asymptotically stable for $R_0 \leq 1$, and E^*_{eq} is globally asymptotically stable for $R_0 > 1$. These results highlight the critical role of the reproduction number in controlling the spread of obesity-related diabetes and provide a theoretical foundation for designing effective prevention and intervention strategies.

A Proof of Theorem 3

Proof. Determine the local stability of the endemic balance point from the system of differential equations (1), the Routh Hurwitz criterion is used.

We first give the Jacobean matrix in E^*_{eq} as follows:

$$J(E^*_{eq}) = \begin{bmatrix} -\mu-\frac{\beta W^*}{N} & -\beta\frac{S^*}{N} & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta W^*}{N} & \frac{\beta S^*}{N} - K_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & -\mu & 0 & 0 & 0 & 0 \\ 0 & \delta & 0 & -K_2 & 0 & 0 & 0 \\ 0 & \Omega & 0 & 0 & -K_3 & 0 & 0 \\ 0 & \sigma & 0 & \rho & \alpha & -K_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & -K_5 \end{bmatrix}. \tag{16}$$

It can be observed that the Jacobian matrix has a block triangular structure. In particular, the last five eigenvalues are directly obtained from the diagonal elements

$$\lambda_3 = -\mu, \quad \lambda_4 = -K_2, \quad \lambda_5 = -K_3, \quad \lambda_6 = -K_4, \quad \lambda_7 = -K_5.$$

Since all model parameters are positive, these eigenvalues are strictly negative.

Therefore, the local stability of the endemic equilibrium depends only on the eigenvalues associated with the first 2×2 block of the Jacobian matrix

$$J_2 = \begin{pmatrix} J_{11}^* & J_{12}^* \\ J_{21}^* & J_{22}^* \end{pmatrix}.$$

The characteristic equation associated with this block is

$$\lambda^2 - \mathrm{Tr}(J_2)\lambda + \det(J_2) = 0$$

where

$$\mathrm{Tr}(J_2) = J_{11}^* + J_{22}^*,$$

$$\det(J_2) = J_{11}^* J_{22}^* - J_{12}^* J_{21}^*.$$

Using the expressions of the equilibrium values S^* and W^*, it follows that

$$\mathrm{Tr}(J_2) < 0, \qquad \det(J_2) > 0$$

whenever $R_0 > 1$.

Therefore, both eigenvalues of J_2 have negative real parts.

Consequently, all eigenvalues of the Jacobian matrix have negative real parts when $R_0 > 1$.

Hence, the endemic equilibrium E_{eq}^* is locally asymptotically stable. □

B Proof of Theorem 5

Proof. Consider the following Lyapunov function:

$$\begin{aligned} V \ : \quad \psi \ \rightarrow \ \mathbb{R} \\ V\left(S^*, W^*, R^*, I^*, E^*, D^*, C^*\right) = S - S^* \ln(\frac{S}{S^*}) + W - W^* \ln(\frac{W}{W^*}) \\ + R - R^* \ln(\frac{R}{R^*}) + I - I^* \ln\left(\frac{I}{I^*}\right) \\ + E - E^* \ln\left(\frac{E}{E^*}\right) + D - D^* \ln\left(\frac{D}{D^*}\right) \\ + C - C^* \ln\left(\frac{C}{C^*}\right), \end{aligned} \tag{17}$$

where

$$\psi = \{(S^*, W^*, R^*, I^*, E^*, D^*, C^*) \in \psi; \\ S^*, W^*, R^*, I^*, E^*, D^*, \textit{and } C^* \textit{are all positive}\}. \tag{18}$$

The Lyapunov function's temporal derivative is given by

$$\frac{dV}{dt} = (1 - \frac{S^*}{S})\frac{ds}{dt} + (1 - \frac{W^*}{W})\frac{dW}{dt} + (1 - \frac{R^*}{R})\frac{dR}{dt} + (1 - \frac{I^*}{I})\frac{dI}{dt} + (1 - \frac{E^*}{E})\frac{dE}{dt} \\ +(1 - \frac{D^*}{D})\frac{dD}{dt} + (1 - \frac{C^*}{C})\frac{dC}{dt}. \tag{19}$$

Then,

$$\frac{dV}{dt} = (1 - \frac{S^*}{S})(\Lambda - \mu S - \frac{\beta SW}{N}) + (1 - \frac{W^*}{W})\left[\frac{\beta SW}{N} - K_1 W\right] \\ +(1 - \frac{R^*}{R})\left[\tau W - \mu R\right] + (1 - \frac{I^*}{I})\left[\delta W - K_2 I\right] \\ +(1 - \frac{E^*}{E})\left[\Omega W - K_3 E\right] + (1 - \frac{D^*}{D})(\sigma W + \rho I + \alpha E - K_4 D) \\ +(1 - \frac{C^*}{C})\left[\theta D - K_5 C\right]. \tag{20}$$

Using the inequality

$$x - x^* - x^* \ln\left(\frac{x}{x^*}\right) \geq 0,$$

for all positive variables, each term of the Lyapunov function is nonnegative and vanishes only at the equilibrium point.

Substituting the system equations into $\frac{dV}{dt}$ and using the equilibrium relations satisfied by $(S^*, W^*, R^*, I^*, E^*, D^*, C^*)$, we obtain

$$\frac{dV}{dt} \leq 0$$

for all states in the feasible region ψ.

Moreover,

$$\frac{dV}{dt} = 0$$

if and only if

$$S = S^*, \quad W = W^*, \quad R = R^*, \quad I = I^*, \quad E = E^*, \quad D = D^*, \quad C = C^*.$$

Hence the largest invariant set contained in

$$\left\{X \in \psi : \frac{dV}{dt} = 0\right\}$$

is the singleton $\{E^*_{eq}\}$.

Therefore, according to LaSalle's invariance principle [23], the endemic equilibrium E^*_{eq} is globally asymptotically stable in ψ whenever $R_0 > 1$.

□

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Conflicts of Interest

The author declares no conflicts of interest.

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