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## OPTIMAL CONTROL IN A MATHEMATICAL MODEL OF A SPREAD OF THE OBESITY EPIDEMIC AND ITS IMPACT ON DIABETES

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**Abstract.** The obesity epidemic is associated with several cardiovascular risk factors, including diabetes mellitus, dyslipidemia, and hypertension. Insulin resistance is believed to play a crucial role in the emergence of various health problems. Apart from genetic factors, obesity, particularly abdominal obesity, is a major contributor to the onset of diabetes. In this study, we present a mathematical model that captures the interplay between the prevalence of obesity and diabetes, and we investigate the adverse effects of excess weight on the well-being of diabetic individuals. Our approach employs Pontryagin's maximum principle to determine the optimal control strategies, and we solve the resulting system through iterative methods. Finally, we perform numerical simulations using Matlab to validate our theoretical analysis.

**Keywords:** obesity; diabetes; continuous-time mathematical model.

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## 1. INTRODUCTION

Overweight and obesity refer to an unusual or extreme buildup of body fat that negatively impacts health [5]. This buildup occurs when the consumption of dietary energy exceeds an individual's needs [28]. The body mass index (BMI) is a widely-used measure for assessing obesity, which takes into account a person's weight and height to determine their body fatness. The World Health Organization (WHO) has set thresholds for overweight, obesity, and morbid obesity based on BMI values in kilograms per square meter respectively: from 25 to 30, from 30 to 40 and exceeding 40 [30].

As per the WHO, 40% of the global population is considered overweight, and 13% are suffering from obesity [30]. A WHO report [31] revealed that obesity in Europe has reached alarming levels, labeled as "near epidemic rates," and causing 1.2 million annual deaths and 200000 new cases of cancer. The report [31] highlights that the countries in the region have failed to meet the WHO's targets for reducing obesity by 2025. Instead, the percentage of obese adults has climbed to 59%, with 8% of children under five years old also affected. Furthermore, this epidemic affects one in three school-aged children.

Numerous illnesses, including diabetes, heart disease and many different forms of cancer, are associated with excessive obesity. Additionally, having too much body fat can increase the likelihood of disability and premature death, as per the report [31]. The latest statistics presented by the WHO [32] on the global spread of the obesity epidemic during the International Day against Obesity (4 March 2022) are shocking and terrifying, indicating that obesity affects more than a billion people globally, including 39 million children, 650 million adults, 340 million youths, and a growing population of 39 million adults. By 2025, the WHO estimates that over 170 million adults and children would have health issues due to being overweight or obese [32]. Among the chronic metabolic diseases is diabetes, brought on by elevated blood sugar, mainly linked to aging and obesity [1]. It can result in serious health complications, including renal illness, heart disease and eye problems. Globally, type 2 diabetes is the most prevalent, affecting more than 420 million individuals worldwide [24, 33]. Between 2000 and 2019, premature death rates due to diabetes increased by 3% and diabetes mellitus and related kidney disease are estimated to have caused about two million deaths in 2019 [36], according

to the WHO [36]. Obesity is a significant risk factor for diabetes, particularly type 2 diabetes [26, 1], and these two conditions are often referred to as “twin epidemics”.

Newly released census data and estimates from the Centers for Disease Control say that around fourteen percent of the American population is severely obese, while nearly 11% have diabetes [20, 4]. Latest investigations have revealed that individuals with obesity have fat cells that release substances and enzymes that prevent the body from properly using insulin, resulting in insulin resistance [27]. This, in turn, raises the likelihood of developing diabetes by 80 – 85% [33, 38].

Obesity is caused by a variety of factors, including genetics to a lesser degree [34], but the most significant causes are unhealthy lifestyle choices like poor diets and a lack of physical activity [27]. Social factors also play a role in obesity. According to studies, the risk of obesity rises by nearly 40% if a sibling or spouse is overweight, and by about 60% if a close friend is obese [35]. In order to prevent overweight and decrease the likelihood of diabetes, it is crucial to adopt healthy eating habits [8, 9, 17], that focus on balanced, nutritious meals while avoiding fast food and resisting temptation from advertisements promoting unhealthy foods and sweets commonly found on social media.

Practicing physical activity plays a key role in treating obesity, maintaining a healthy weight, and preventing diabetes [36]. Experts recommend adopting regular physical activity to protect against cardiovascular disease risk, reduce the stroke risk, and improve blood glucose levels [2, 12, 19], which should be adopted as a lifelong habit rather than a temporary measure, even for obese individuals. If the option of healthy nutrition and exercise fails for this category of obese people, they should consider alternative procedures and solutions to diet, such as the surgical procedure. This article’s goal is to shed light on the negative impact of obesity and diabetes on health, and to propose methods and strategies to reduce the spread of these two epidemics, including encouraging diabetics to follow medical advice to prevent complications from diabetes. Infectious disease researchers have cited the beneficial role of mathematics and mathematical models in providing a clear framework for revealing how infectious diseases spread. To explain the transmission of the epidemic, they built mathematical models that contain vital biological data [25, 13, 16, 37]. In this context, we mention some works that have studied

the dynamics of various epidemiological phenomena such as alcoholism, smoking, Covid-19, and obesity [6, 7, 15, 23, 29, 39].

In a previous article [6], we proposed an ideal control method to combat the spread of obesity using a discrete time mathematical model. In this present study, we present the following important additions:

An optimal control modeling of the transmission of the obesity epidemic and its impact on Diabetes.

A Continuous-time mathematical modeling.

An analytical study of the presented model.

A suggested optimal control strategy that can limit the spread of the obesity epidemic and help reduce the prevalence of diabetes.

The present work aims to suggest a compartmental model that uses differential equations to explain how obesity spreads through the population, considering the contribution of this epidemic to the incidence of diabetes. The objective is contain the propagation of the epidemic, and to achieve this, we present effective methods to reduce the number of sick individuals by suggesting an optimal control in the used model. Thus, we propose three controls: the first one represents an awareness program through media and education. The second control refers to the encouragement of overweight people to adopt a balanced and healthy diet. The third control is to put people with diabetes without complications under medical supervision, or even treatment if necessary.

The following is how the paper is set up: in Section 2, we provide our SWRIEDC mathematical model, which depicts the obesity's dynamics. with comorbidity of diabetes. We analyze the fundamental characteristics and positivity of solutions in Section 3. In Section 4, we formulate the optimal control problems for the proposed model, where we provide results on the existence of optimal controls and characterize them using Pontryagin's maximum principle [11]. We also present numerical simulations using Matlab in Section 5. Finally, in Section 6, we wrap up the findings of our suggested study.

## 2. FORMULATION OF THE MODEL

We study the *SWRIEDC* model, which describes the dynamics of the obesity epidemic in a specific population by highlighting the negative role of overweight in the type's 2 diabetes's progression. The population indicated by  $N$  is divided into seven compartments. A schematical representation of the proposed model is shown in Figure (1).

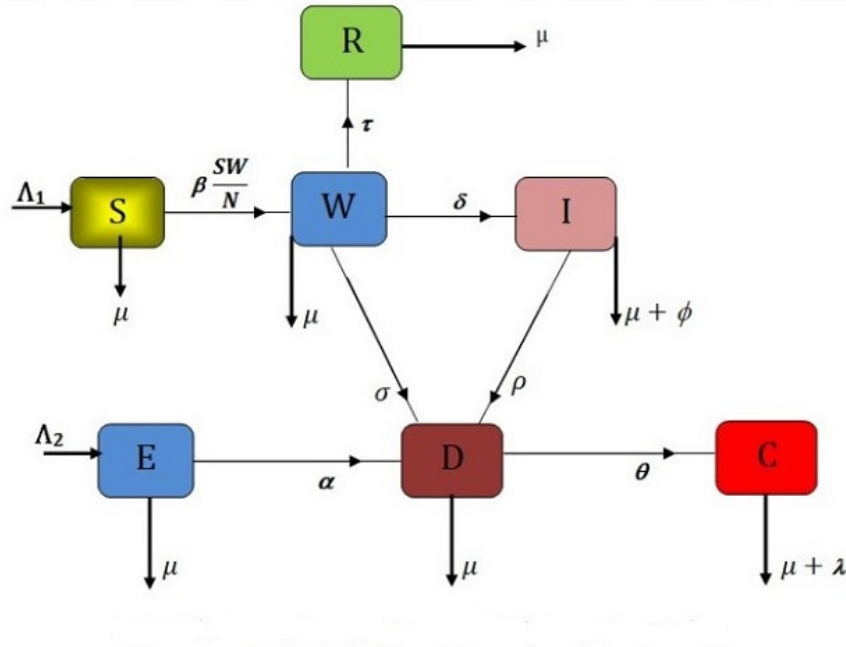


FIGURE 1. Schematic illustration of obesity-diabetes model

The table 1 describes the different classes of the population studied:

TABLE 1. State variables and their meanings

Variable	meaning
S	Obesity-prone individuals
W	People overweight or moderately obese
I	Obese people
R	People who have made a full recovery
E	Pre-diabetic people
D	Diabetics who are complications-free
C	Diabetics with significant health issues

Let  $N(t)=S(t)+W(t)+I(t)+R(t)+E(t)+D(t)+C(t)$ : The total number at date  $t$  of individuals in the model population. The model is described by the following ordinary differential equations:

$$(1) \quad \begin{cases} \frac{dS(t)}{dt} = \Lambda_1 - \mu S(t) - \beta \frac{S(t)}{N} W(t) \\ \frac{dW(t)}{dt} = \beta \frac{S(t)}{N} W(t) - (\tau + \sigma + \mu + \delta) W(t) \\ \frac{dR(t)}{dt} = \tau W(t) - \mu R(t) \\ \frac{dI(t)}{dt} = \delta W(t) - (\phi + \rho + \mu) I(t) \\ \frac{dE(t)}{dt} = \Lambda_2 - (\mu + \alpha) E(t) \\ \frac{dD(t)}{dt} = \sigma W(t) + \rho I(t) + \alpha E(t) - (\mu + \theta) D(t) \\ \frac{dC(t)}{dt} = \theta D(t) - (\mu + \lambda) C(t) \end{cases}$$

With initial conditions,  $S(0) \geq 0$ ,  $W(0) \geq 0$ ,  $R(0) \geq 0$ ,  $I(0) \geq 0$ ,  $E(0) \geq 0$ ,  $D(0) \geq 0$ , and  $C(0) \geq 0$ .

The model parameters are as follows;

TABLE 2. Values and description of the parameters.

Parameter	Description	Value	Source
$\Lambda_1$	The recruitment rate of the exposed to obesity	$4 \times 10^6$	Assumed
$\mu$	Rate of natural death	0.02	[6]
$\beta$	The obesity rate from contact with infected individuals	0.45	[6]
$\tau$	The rate which overweight people recover	0.07	Assumed
$\delta$	The proportion of the overweight that joined the compartment I	0.05	[6]
$\lambda$	Death rate as a result of diabetes with difficulties	0.001	[14]
$\phi$	Mortality rate due to obesity.	0.03	Assumed
$\rho$	Rate of uncomplicated diabetes in obese individuals	0.06	Assumed
$\Lambda_2$	The recruitment rate of pre-diabetes without complications	$5 \times 10^5$	Assumed
$\alpha$	Rate of uncomplicated diabetes in pre-diabetic patients	0.06	[14]
$\sigma$	The rate of overweight who develop diabetes without complications	0.025	Assumed
$\theta$	The rate of diabetics who will evolve complications of diabetes	0.08	[14]

### 3. QUALITATIVE ANALYSIS OF THE PROPOSED MODEL

#### 3.1. Solutions positivity.

**Theorem 1.** *If  $S(0) \geq 0$ ,  $W(0) \geq 0$ ,  $R(0) \geq 0$ ,  $I(0) \geq 0$ ,  $E(0) \geq 0$ ,  $D(0) \geq 0$ , and  $C(0) \geq 0$ , the solutions  $S(t)$ ,  $W(t)$ ,  $R(t)$ ,  $I(t)$ ,  $E(t)$ ,  $D(t)$  and  $C(t)$  of system (1) are positive for all  $t \geq 0$ .*

*Proof.* We have according to the first equation of the system (1) that

$$(2) \quad \frac{dS(t)}{dt} = \Lambda_1 - \mu S - \beta \frac{S}{N} W$$

$$(3) \quad \frac{dS(t)}{dt} + \left( \mu + \beta \frac{W(t)}{N} \right) S(t) \geq 0$$

or

$$\frac{dS(t)}{dt} + F(t)S(t) \geq 0$$

where

$$F(t) = \mu + \beta \frac{W(t)}{N} \exp\left(\int_0^t F(x)dx\right)$$

The last inequality's two sides are multiplied by

$$\exp\left(\int_0^t F(x)dx\right)$$

We obtain:

$$\frac{dS(t)}{dt} \cdot \exp\left(\int_0^t F(x)dx\right) + F(t) \cdot \exp\left(\int_0^t F(x)dx\right) \cdot S(t) \geq 0$$

then

$$\frac{d}{dt} \left( S(t) \cdot \exp\left(\int_0^t F(x)dx\right) \right) \geq 0$$

This inequality can be integrated from 0 to  $t$  to produce:

$$\int_0^t \frac{d}{dz} \left( S(z) \cdot \exp\left(\int_0^z F(x)dx\right) \right) dz \geq 0$$

then

$$(4) \quad S(t) \cdot \exp\left(\int_0^t F(x)dx\right) - S(0) \geq 0$$

then

$$(5) \quad S(t) \geq S(0) \cdot \exp\left(-\int_0^t F(x)dx\right)$$

which implies finally:

$$(6) \quad S(t) \geq 0$$

Likewise, we demonstrate  $W(t) \geq 0, R(t) \geq 0, I(t) \geq 0, E(t) \geq 0, D(t) \geq 0$  and  $C(t) \geq 0$ .  $\square$

### 3.2. Invariant region.

**Theorem 2.** *There exists a domain  $\Omega$  in which the solution set  $(S, W, R, I, E, D, C)$  is contained and bounded.*

*Proof.* Given the solution set  $(S, W, R, I, E, D, C)$  with positive initial conditions:

$$S(0) \geq 0, W(0) \geq 0, R(0) \geq 0, I(0) \geq 0, E(0) \geq 0, D(0) \geq 0, \text{ and } C(0) \geq 0$$

The equations of system (1) are added, and the result is:

$$(7) \quad \frac{dN}{dt} = \Lambda - \mu N - \phi I - \lambda C \leq \Lambda - \mu N.$$

So,

$$\frac{1}{\mu} \cdot \frac{dN}{dt} + N \leq \frac{\Lambda}{\mu}$$

then,

$$N(t) \leq N_0 \cdot \exp(-\mu t) + \frac{\Lambda}{\mu}$$

where  $\Lambda = \Lambda_1 + \Lambda_2$  that  $\Lambda$  is the sum of the recruitment rates  $\Lambda_1$  and  $\Lambda_2$ , and  $N_0$  refers to the initial value of the population. And therefore,

$$(8) \quad \lim_{t \rightarrow \infty} (\sup N(t)) = \frac{\Lambda}{\mu}$$

The region  $\Omega$  is implied to be a positively invariant set for system (1). As a result, we just need to think about the system's dynamics on the set  $\Omega$ .  $\square$

### 3.3. Global solution's existence and uniqueness.

**Theorem 3.** *There is only one solution for the system (1) that meets the provided initial condition  $(S(0), W(0), R(0), I(0), E(0), D(0), C(0))$*



*Proof.* Let's put:  $Y(t) = \begin{pmatrix} S(t) \\ W(t) \\ R(t) \\ I(t) \\ E(t) \\ D(t) \\ C(t) \end{pmatrix}$  and  $F(Y) = \begin{pmatrix} \frac{dS(t)}{dt} \\ \frac{dW(t)}{dt} \\ \frac{dR(t)}{dt} \\ \frac{dI(t)}{dt} \\ \frac{dE(t)}{dt} \\ \frac{dD(t)}{dt} \\ \frac{dC(t)}{dt} \end{pmatrix}$

Consequently, the system (1) is recast as follows:

$$F(Y) = \bar{a} + (WA_1 + A_2)Y \text{ where } \bar{a} = \begin{pmatrix} \Lambda_1 \\ 0 \\ 0 \\ 0 \\ \Lambda_2 \\ 0 \\ 0 \end{pmatrix}, A_1 = \begin{pmatrix} -\frac{\beta}{N} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta}{N} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and}$$

$$A_2 = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -k_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau & -\mu & 0 & 0 & 0 & 0 \\ 0 & \delta & 0 & -k_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\alpha + \mu) & 0 & 0 \\ 0 & \sigma & 0 & \rho & \alpha & -k_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & -k_4 \end{pmatrix}$$

such as:

$$(9) \quad \begin{cases} k_1 = \tau + \mu + \delta + \sigma \\ k_2 = \rho + \mu + \phi \\ k_3 = \mu + \theta \\ k_4 = \mu + \lambda \end{cases}$$

So,

$$F(Y) = \bar{a} + (WA_1 + A_2)Y,$$

It follows that there exist  $U = \|\bar{a}\|$  and  $\Delta = |W| \|A_1\| + \|A_2\|$  such that

$$(10) \quad \|F(Y)\| \leq U + \Delta \cdot \|Y\|$$

The system(1) therefore offers a one global solution on  $\Omega$ . □

## 4. THE PROBLEM OF OPTIMAL CONTROL

**4.1. Model with controls.** Our suggested control plan seeks to reduce the proportion of overweight persons  $W(t)$  and diabetics who are uncomplicated. In model (1) we include three controls  $u_1(t), u_2(t)$  and  $u_3(t)$  for  $t \in [0, T]$ . The first control  $u_1(t)$  represents the awareness campaign for those at risk at time  $t$ , i.e.  $S(t)$ , through media and education aimed at highlighting the importance of maintaining a fit body on the one hand and sensitizing to the danger of obesity and its negative impact on health by causing several diseases including type 2 diabetes, high blood pressure and heart disease.

The second control  $u_2(t)$  designates the urging overweight people to adopt a balanced diet that is free from harmful fats and sugars and rich in healthy fats, with fiber and whole grains. This control aims to prevent excessive weight gain or excess obesity.

The third control  $u_3(t)$  is putting people with diabetes without complications under medical supervision through periodic monitoring of the blood's amount of glucose, and drug treatment intervention in case of necessity.

$$(11) \quad \left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Lambda_1 - \mu S(t) - \beta(1 - u_1(t)) \frac{S(t)}{N} W(t) \\ \frac{dW(t)}{dt} = \beta(1 - u_1(t)) \frac{S(t)}{N} W(t) - k_1 W(t) - u_2(t) W(t) \\ \frac{dR(t)}{dt} = \tau W(t) - \mu R(t) + u_2(t) W(t) \\ \frac{dI(t)}{dt} = \delta W(t) - k_2 I(t) \\ \frac{dE(t)}{dt} = \Lambda_2 - \mu E(t) - \alpha(1 - u_3(t)) E(t) \\ \frac{dD(t)}{dt} = \sigma W(t) + \rho I(t) + \alpha(1 - u_3(t)) E(t) - k_3 D(t) \\ \frac{dC(t)}{dt} = \theta D(t) - k_4 C(t) \end{array} \right.$$

The issue is reducing the objective functional:

$$(12) \quad J(u_1, u_2, u_3) = W(T) + D(T) + \int_0^{T-1} \left[ W(t) + D(t) + \frac{A}{2} u_1^2(t) + \frac{B}{2} u_2^2(t) + \frac{G}{2} u_3^2(t) \right] dt$$

Where the positive parameters  $A, B$  and  $G$  serve as coefficients that help to evaluate the relative importance of  $u_1(t), u_2(t)$  and  $u_3(t)$  at time  $t$ .  $T$  refers to the final time.

Our goal is to determine the ideal controls  $u_1^*, u_2^*$  and  $u_3^*$  that satisfy certain conditions:

$$(13) \quad J(u_1^*, u_2^*, u_3^*) = \min_{(u_1, u_2, u_3) \in U_{ad}} J(u_1, u_2, u_3)$$

Where  $U_{ad}$  is the set of admissible controls defined by:

$$U_{ad} = \{(u_1, u_2, u_3) / u_{1 \min} \leq u_1(t) \leq u_{1 \max} ; u_{2 \min} \leq u_2(t) \leq u_{2 \max}$$

$$\text{and } u_{3 \min} \leq u_3(t) \leq u_{3 \max} / 0 \leq t \leq T\}$$

## 4.2. Existence and characterization of the ideal control.

### 4.2.1. Existence of an ideal control.

**Theorem 4.** Consider the system's control issue (11). There exist the best controls  $(u_1^*, u_2^*, u_3^*) \in U_{ad}^3$  such that:

$$(14) \quad J(u_1^*, u_2^*, u_3^*) = \min_{(u_1, u_2, u_3) \in U_{ad}} J(u_1, u_2, u_3)$$

*Proof.* The following steps can be checked to see if the optimal control exists using a result from Fleming and Rishel's [10]. To demonstrate that there exists a nonempty set of controls and corresponding state variables, we employ a simplified version of the results presented by Boyce and Deprima [3]: We put  $X'_i = F_{X_i}(t, X_1, X_2, X_3, \dots, X_7)$  with  $i = 1; \dots; 7$ ,  $(X_1, X_2, X_3, \dots, X_7) = (S, W, R, I, E, D, C)$  where  $X'_1, X'_2, X'_3, X'_4, X'_5, X'_6$  and  $X'_7$  from the right of equations(11). Let  $u_1, u_2$  and  $u_3$  for some constants and since all parameters are constants and  $X_1, X_2, X_3, X_4, X_5, X_6$  and  $X_7$  are continuous, then  $F_S, F_W, F_R, F_I, F_E, F_D$  and  $F_C$  are also continuous. Additionally, the partial derivatives  $\frac{\partial F_{X_i}}{\partial X_i}$  are all continuous, hence there must be a single solution  $(S, W, R, I, E, D, C)$  that meets the basic requirements. When condition 1 is met in  $U_{ad}$ , the set of controls and associated state variables are not empty.

– The control space  $U_{ad} = \{(u_1, u_2, u_3) / u_{1 \min} \leq u_1(t) \leq u_{1 \max} ; u_{2 \min} \leq u_2(t) \leq u_{2 \max} ; u_{3 \min} \leq u_3(t) \leq u_{3 \max}\}$  is convex and closed by definition.

– A linear function of  $u_1$ ,  $u_2$  and  $u_3$  with time- and state-dependent coefficients can be used to define all of the right-hand sides of system equations, which are continuous, bounded above by the addition of bounded control and state.

– The integrand in the objective functional  $W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2}u_3^2(t)$  is clearly convex on  $U_{ad}$ .

–It rests to show that there exist constants  $\Gamma_1, \Gamma_2, \Gamma_3, \Gamma_4 > 0$ , and  $\Gamma$  such that  $W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2}u_3^2(t)$  satisfies

$$W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2}u_3^2(t) \geq \Gamma_1 + \Gamma_2 |u_1|^\Gamma + \Gamma_3 |u_2|^\Gamma + \Gamma_4 |u_3|^\Gamma.$$

The state variables being bounded, let  $\Gamma_1 = \frac{1}{2} \inf_{t \in [0, T]} [W(t) + D(t)]$ ,  $\Gamma_2 = A$ ,  $\Gamma_3 = B$ ,  $\Gamma_4 = G$  and  $\Gamma = 2$  then it follows that:

$W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2}u_3^2(t) \geq \Gamma_1 + \Gamma_2 |u_1|^\Gamma + \Gamma_3 |u_2|^\Gamma + \Gamma_4 |u_3|^\Gamma$ . consequently, we draw the conclusion that there is an ideal control from Fleming and Rishel[10].  $\square$

**4.2.2. Characterization of the ideal control.** We use Pontryagin's maximal principle [21, 22] to the Hamiltonian  $H$  in order to obtain the prerequisites for the ideal control. This Hamiltonian is defined at time  $t$  by

$$(15) \quad H(t) = W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2} + \sum_{i=1}^7 \varepsilon_i(t) \cdot f_i(S, W, R, I, E, D, C, u_1, u_2, u_3, t)$$

Where the adjoint variables are  $\varepsilon_i(t)$ , and  $f_i$  is the right side of the differential equation of the  $i$ th state variable at time  $t$ .

**Theorem 5.** *Considering the ideal controls  $(u_1^*, u_2^*, u_3^*)$  and the solutions  $S^*, W^*, R^*, I^*, E^*, D^*$  and  $C^*$  of the associated state system (2), the following adjoint variables  $\varepsilon_1(t), \varepsilon_2(t), \varepsilon_3(t), \varepsilon_4(t), \varepsilon_5(t), \varepsilon_6(t)$  and  $\varepsilon_7(t)$  are present and satisfying:*

$$\varepsilon_1'(t) = -\frac{\partial H(t)}{\partial S(t)} = \varepsilon_1(t) \left[ \mu + \beta(1 - u_1(t)) \frac{W(t)}{N} \right] - \varepsilon_2(t) \beta(1 - u_1(t)) \frac{W(t)}{N};$$

$$\begin{aligned} \varepsilon_2'(t) = -\frac{\partial H(t)}{\partial W(t)} = & -1 + \varepsilon_1(t) \beta(1 - u_1(t)) \frac{S(t)}{N} - \varepsilon_2(t) \left[ \beta(1 - u_1(t)) \frac{S(t)}{N} - k_1 - u_2(t) \right] \\ & - \varepsilon_3(t) (\tau + u_2(t)) - \varepsilon_4(t) \delta - \varepsilon_6(t) \sigma; \end{aligned}$$

$$\varepsilon_3'(t) = -\frac{\partial H(t)}{\partial R(t)} = \varepsilon_3(t) \mu;$$

$$\begin{aligned}\varepsilon_4^i(t) &= -\frac{\partial H(t)}{\partial I(t)} = \varepsilon_4(t)k_2 - \varepsilon_6(t)\rho; \\ \varepsilon_5^i(t) &= -\frac{\partial H(t)}{\partial E(t)} = \varepsilon_5(t)\mu + (\varepsilon_5(t) - \varepsilon_6(t))\alpha(1 - u_3(t)); \\ \varepsilon_6^i(t) &= -\frac{\partial H(t)}{\partial D(t)} = -1 + \varepsilon_6(t)k_3 - \varepsilon_7(t)\theta; \\ \varepsilon_7^i(t) &= -\frac{\partial H(t)}{\partial C(t)} = \varepsilon_7(t)k_4.\end{aligned}$$

Considering the transversality circumstances at time  $T$  :  $\varepsilon_1(T) = \varepsilon_3(T) = \varepsilon_4(T) = \varepsilon_5(T) = \varepsilon_7(T) = 0$  and  $\varepsilon_2(T) = \varepsilon_6(T) = 1$ . Additionally, for  $t$  such that  $0 \leq t \leq T$ , the optimal controls  $u_1^*$ ,  $u_2^*$ , and  $u_3^*$  are given by

$$(16) \quad \begin{cases} u_1^* = \min \left[ u_{1\max}; \max \left( u_{1\min}, \frac{\beta W(t)}{AN} (\varepsilon_2(t) - \varepsilon_1(t)) S(t) \right) \right] \\ u_2^* = \min \left[ u_{2\max}; \max \left( u_{2\min}, \frac{1}{B} (\varepsilon_2(t) - \varepsilon_3(t)) W(t) \right) \right] \\ u_3^* = \min \left[ u_{3\max}; \max \left( u_{3\min}, \frac{\alpha}{G} (\varepsilon_6(t) - \varepsilon_5(t)) E(t) \right) \right] \end{cases}$$

*Proof.* We use the Pontryagin's maximum principle[6, 21, 22], for defining the ideal controls. So, the following is how we defined the Hamiltonian H:

$$\begin{aligned}H(t) &= W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2}u_3^2(t) \\ &\quad + \sum_{i=1}^7 \varepsilon_i(t) \cdot f_i(S, W, R, I, E, D, C, u_1, u_2, u_3, t)\end{aligned}$$

$$\begin{aligned}H(t) &= W(t) + D(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{G}{2}u_3^2(t) + \varepsilon_1(t) \cdot \left[ \Lambda_1 - \mu S(t) - \beta(1 - u_1(t)) \frac{S(t)}{N} W(t) \right] \\ &\quad + \varepsilon_2(t) \cdot \left[ \beta(1 - u_1(t)) \frac{S(t)}{N} W(t) - k_1 W(t) - u_2(t) W(t) \right] + \varepsilon_3(t) \cdot [\tau W(t) - \mu R(t) + u_2(t) W(t)] \\ &\quad + \varepsilon_4(t) \cdot [\delta W(t) - k_2 I(t)] + \varepsilon_5(t) \cdot [\Lambda_2 - \mu E(t) - \alpha(1 - u_3(t)) E(t)] \\ &\quad + \varepsilon_6(t) \cdot [\sigma W(t) + \rho I(t) + \alpha(1 - u_3(t)) E(t) - k_3 D(t)] + \varepsilon_7(t) \cdot [\theta D(t) - k_4 C(t)]\end{aligned}$$

With the constants  $k_1$ ,  $k_2$ ,  $k_3$  and  $k_4$ , are given in (9).

In this situation, N is regarded as constant.

For  $t$  such that  $0 \leq t \leq T$ , the ideal controls  $u_1$ ,  $u_2$  and  $u_3$  is amenable to resolution based on the optimality criteria:

$$-\frac{\partial H(t)}{\partial u_1} = 0 \implies -Au_1 - \varepsilon_1\beta \frac{SW}{N} + \varepsilon_2\beta \frac{SW}{N} = 0.$$

$$-\frac{\partial H(t)}{\partial u_2} = 0 \implies -Bu_2 + \varepsilon_2 W - \varepsilon_3 W = 0.$$

$$-\frac{\partial H(t)}{\partial u_3} = 0 \implies -Gu_3 - \alpha\varepsilon_5 E + \alpha\varepsilon_6 E = 0.$$

We have

$$u_1(t) = \frac{\beta W(t)}{AN} (\varepsilon_2(t) - \varepsilon_1(t)) S(t)$$

$$u_2(t) = \frac{1}{B} (\varepsilon_2(t) - \varepsilon_3(t)) W(t)$$

$$u_3(t) = \frac{\alpha}{G} (\varepsilon_6(t) - \varepsilon_5(t)) E(t)$$

By the bounds in  $U_{ad}$  of the controls, it is easy to obtain  $u_1^*$ ,  $u_2^*$  and  $u_3^*$  in the form of (16)  $\square$

## 5. NUMERICAL SIMULATION

In this paragraph, we aim to solve the optimal control problem numerically for our *SWRIEDC* model. The model's optimality framework includes the state system, the adjoint system, the starting and ending time conditions, as well as a general description. By using an iterative process, we move from forward resolution of the state system to resolve the system by backtracking the resolution of the adjacent system. For the first iteration, we start with an initial estimate of the controls, and we update the control based on the characterization before proceeding to the next iteration. We continue this process until we achieve convergence for subsequent iterations. The initial value of compartments is chosen as follows :  $S(0) = 4.5 \times 10^6$  ,  $W(0) = 4.5 \times 10^6$  ,  $I(0) = 8 \times 10^6$  ,  $R(0) = 2 \times 10^5$  ,  $E(0) = 7 \times 10^5$  ,  $D(0) = 8 \times 10^5$  and  $C(0) = 1 \times 10^5$

**5.1. Effect of each control on the evolution of infected persons (W,I,D and C).** In this first part of the simulation, we track the evolution of the number of cases of different infections : heavy weight (W : figure 2a), obese ( I : figure 2b ), diabetic patients with and without complications (C and D : figures 2c and 2d respectively), where we highlight the effectiveness of each of the three controls separately, including:

- The  $u_1$  control aims to raise awareness among people likely to be overweight (S) through the media and education, of the harmful effects of obesity on health.

- The  $u_2$  control is the encouragement of overweight people (W) to adopt a healthy and balanced diet.

- The third  $u_3$  control consists of putting diabetics (D) under periodic medical supervision.

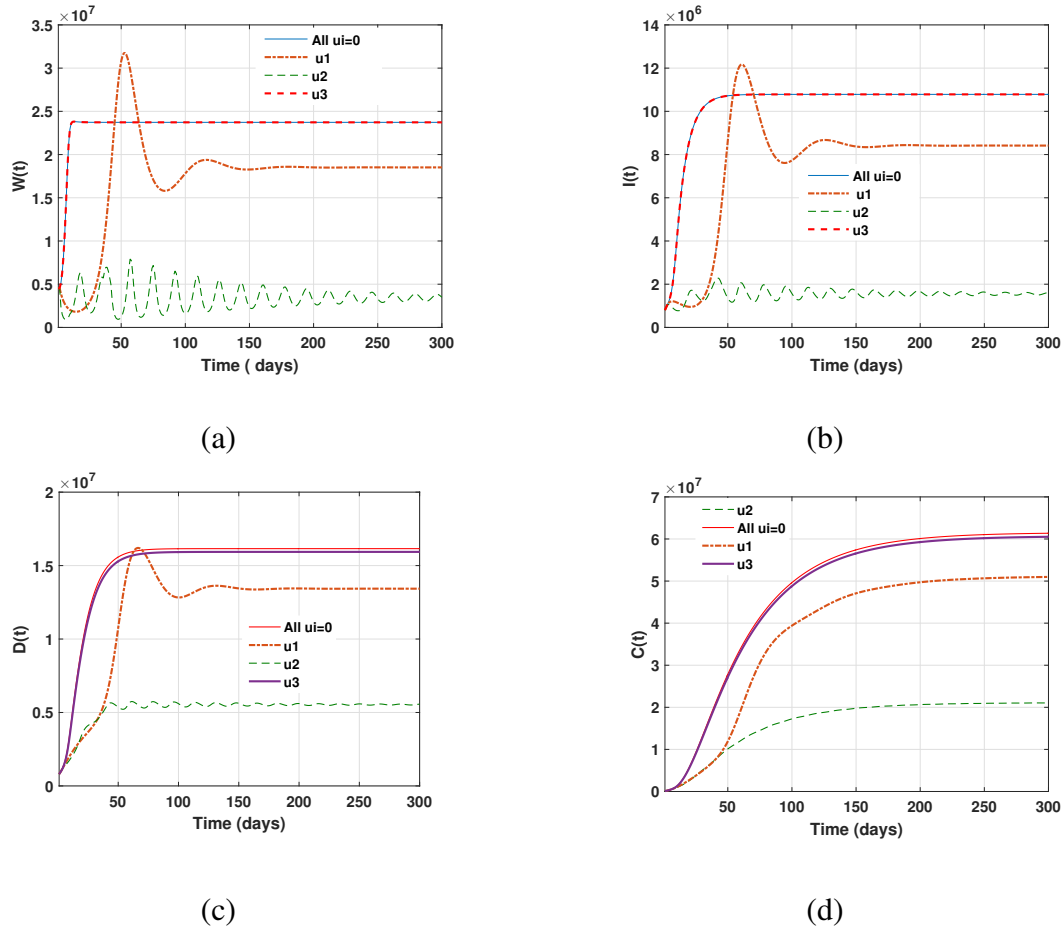


FIGURE 2. Evolution of the number of infected persons before and after the application of various controls separately

Figure 2a demonstrates that the control  $u_1$  lowers the number of ( $W$ ), whereas the control  $u_2$  reduces the number of these populations efficiently but with little disruption because the body is still getting used to the new diet at the time of adoption of this control. Due to the fact that control  $u_3$  is for diabetic patients ( $D$ ), it is obvious that it has no impact on population growth ( $W$ ). According to Figure 2b, the population ( $I$ ) decreases significantly when the  $u_2$  control is adopted and just little when the  $u_1$  control is applied, while the  $u_3$  control is inactive in terms of reducing the cases of obese people ( $I$ ).

Figure (2c) depicts a large reduction in diabetes (D) cases due to control  $u_2$ , and a weaker reduction due to control  $u_1$ . However, the impact of  $u_3$  control on population evolution (D) is essentially nonexistent.

The qualitative effects of the three controls  $u_1$ ,  $u_2$  and  $u_3$  on the evolution of cases of diabetes (C) (Fig. 2d) and cases of diabetes (D) (Fig. 2c) are the same.

The table (3) gives the percentage decrease  $p_i$  in the number of different patients : those with heavy weight (W), obese (I), patients with problems from diabetes (C) and those without complications (D), after 200 days of adopting the three controls separately : awareness ( $u_1$ ), urging on healthy nutrition ( $u_2$ ) and medical supervision ( $u_3$ ).

TABLE 3. Number and the percentage  $p_i$  of the reduction in the number of people with different infections before and after 200 days have passed since the control  $u_i$  was implemented

The type of infected	W	I	D	C
Figure	(2a)	(2b)	(2c)	(2d)
All $u_i = 0 ; (\times 10^7)$	$W_0 = 2.37$	$I_0 = 1.08$	$D_0 = 1.61$	$C_0 = 6$
$p_0(\%)$	---	---	---	---
$u_1 \neq 0 ; (\times 10^6)$	$W_1 = 18.5$	$I_1 = 8.42$	$D_1 = 13.4$	$C_1 = 49.7$
$p_1(\%)$	21.94	22	16.77	17.16
$u_2 \neq 0 ; (\times 10^6)$	$W_2 = 2.95$	$I_2 = 1.66$	$D_2 = 5.6$	$C_2 = 20.6$
$p_2(\%)$	87.55	84.62	65.21	65.66
$u_3 \neq 0 ; (\times 10^7)$	$W_3 = 2.37$	$I_3 = 1.08$	$D_3 = 1.59$	$C_3 = 5.92$
$p_3(\%)$	0	0	90.12	1.33

We read the data of Table (3) as follows : Balanced healthy nutrition ( control  $u_2$ ) contributes to reducing the rates of various types of infections: overweight (W), obesity ( I ) and diabetes mellitus with complications (C) or without (D), as after adopting this control for a period of two hundred days, the decrease rates are respectively 87,55%, 84,62%, 65,66% and 65,21%. Awareness of the danger of obesity ( control  $u_1$ ) has the same role, but with a lower decrease rate: approx., 22% to avoid obesity and about 17% for preventing diabetes. It is obvious that



control  $u_3$  which represents medical monitoring has no effect on reducing the number of cases of obesity or overweight, because it contributes to reducing the possibility of developing diabetes without complications by about 90%.

**5.2. Impact of the combination of two controls on the evolution of infected persons (W,I,D and C).** At this stage, we are interested in studying the effect of adopting each pair of the three controls on the dynamics of obesity and diabetes ( See figures 3 ).

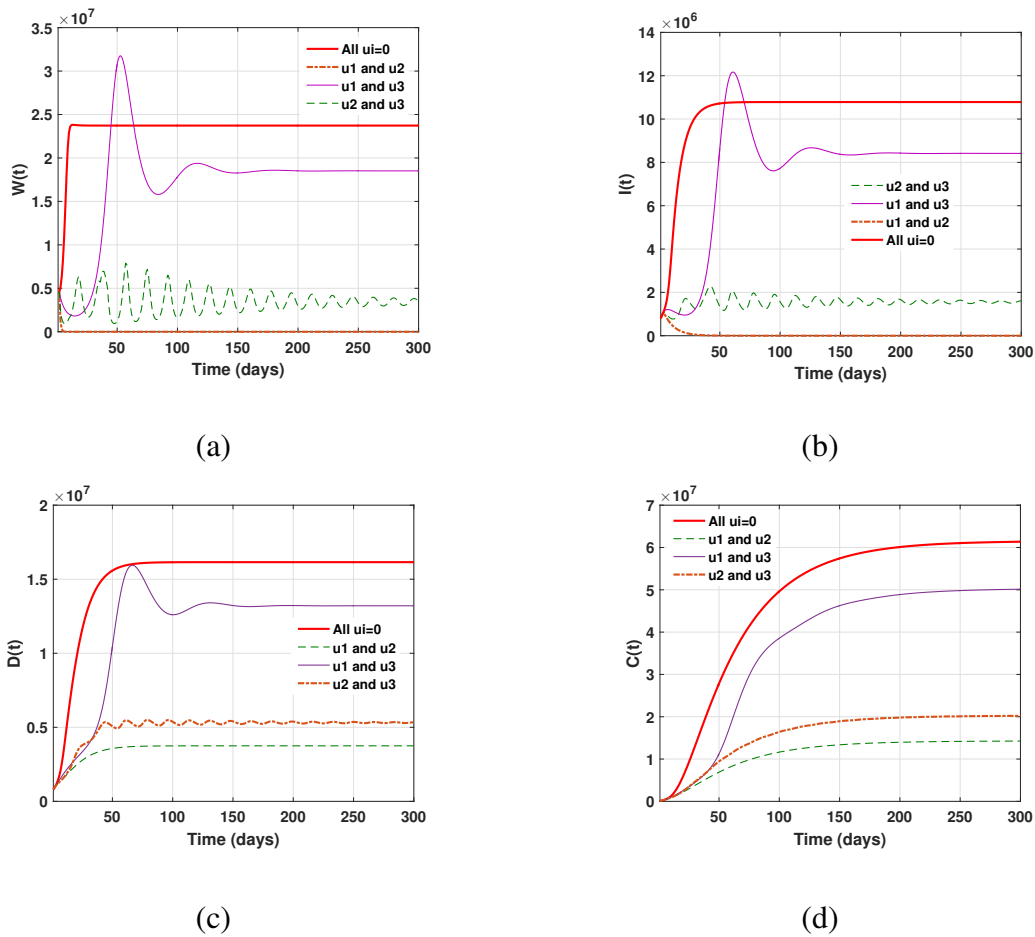


FIGURE 3. The development of the number of patients before and after applying a pair of two different controls

The pair of controls  $(u_1, u_2)$  has an immediate effect to get rid of obesity. About a week after adopting them, the cases of overweight disappear (figure 3a), and within one month obesity is eliminated (figure 3b). In addition, the application of these two controls reduces the possibility

of developing diabetes, because the decrease in cases of excess weight reduces the number of cases of diabetes resulting from obesity, and indeed, after the passage of about two hundred days, the number of diabetics without complications and the diabetics who do are is, respectively 3.75 million ( figure 3c ) and 14 million (figure 3d ), which is a much smaller number of recorded cases if these two controls were adopted separately ( figures 2c and 2d ).

After 100 days of applying the two controls  $u_2$  and  $u_3$ , the number of cases of diabetes without complications stabilizes in the 5 million ( figure 3c ), which represents a decrease of about 11% compared to the case approach the two controls individually ( figure 2c ). The pair of controls  $(u_1, u_3)$  reduces the prevalence of both comorbid and uncomorbid diabetes: C and D ( figures 3c and 3d ). This effect is larger compared to the case of adopting these controls separately ( figures 2c and 2d ). This control pair has the same degree of importance as the control  $u_1$  (awareness) in the fight against overweight and obesity ( figures 2a, 2b, 3a and 3b ).

**5.3. The effectiveness of the three controls combined in reducing the prevalence of obesity and diabetes.** In the following, we examine the role of the three controls combined in the development of the population of various compartments in our suggested model, so we obtain figures (4).

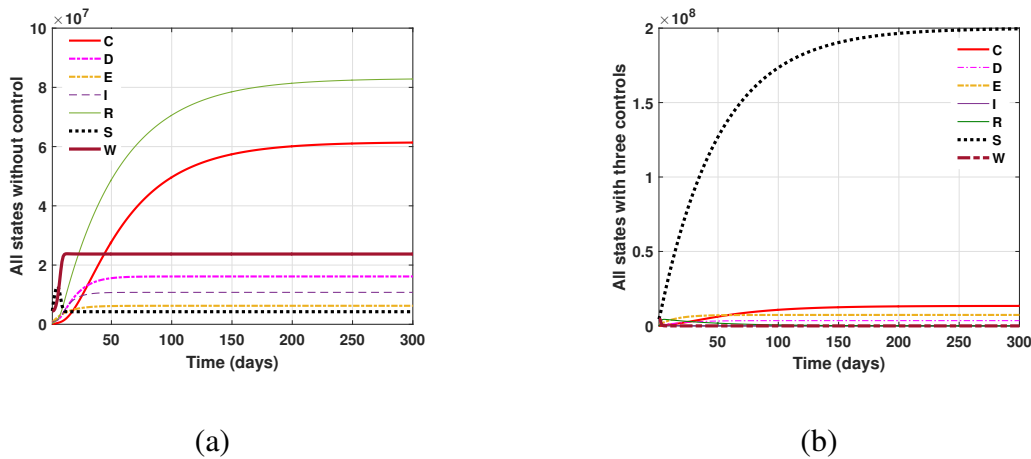


FIGURE 4. Evolution of all states with and without control

It is clear that the three controls, awareness ( $u_1$ ), healthy nutrition ( $u_2$ ) and medical monitoring ( $u_3$ ) contribute significantly to the prevention of obesity, and thus protect against the onset

of diabetes. Although the figure (4b) shows that we have an increased amount of persons at risk of obesity (S) compared to the state of lack of controls ( figure 4a), the three proposed measures significantly reduce the chances of overweight developing into obesity or diabetes with or without complications. It is obvious that the decrease in infected individuals leads to a decrease in the number of recovered cases (figure 4b) the opposite of what it used to be without controls ( figure 4a).

## 6. CONCLUSION

In this article, we propose a mathematical model to study the dynamics of obesity and its potential for progression to diabetes mellitus. We link our model to three control measures: awareness of the seriousness of obesity and its complications through the media and civil society, encouragement to eat healthy balanced foods, and periodic medical monitoring of diabetic patients without complications. The problem of optimal control is formulated and analyzed, and thus the best control strategies are found by reducing the number of overweight individuals and preventing the development of diabetes complications, using the maximum Pontryagin principle. A comparison of the evolution of all cases with and without controls is also shown, we try to study all possible combinations between controls and analyze all scenarios. All methods work well in lowering the prevalence of either obesity, diabetes, or both.

## CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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