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RESEARCH PAPER

Optimal control of diabetes model with the impact of endocrine-disrupting chemical: an emerging increased diabetes risk factor

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Abstract

Diabetes, a persistent pathological condition characterized by disruptions in insulin hormone regulation, has exhibited a noteworthy escalation in its prevalence over recent decades. The surge in incidence is notably associated with the proliferation of endocrine-disrupting chemicals (EDCs), which have emerged as primary contributors to the manifestation of insulin resistance and the consequent disruption of beta cell function, ultimately culminating in the onset of diabetes. Consequently, this study endeavors to introduce a model for diabetes that aims to elucidate the ramifications of exposure to EDCs within the diabetic population. In the pursuit of mitigating the deleterious effects of EDC-induced diabetes, we propose a framework for optimal control strategies. The utilization of Pontryagin's maximum principle serves to explicate the principles governing the optimal control mechanisms within the proposed model. Our findings underscore that heightened concentrations of EDCs play a pivotal role in exacerbating the prevalence of diabetes. To substantiate our model, we employ parameter estimation techniques utilizing a diabetes dataset specific to the demographic context of India. This research contributes valuable insights into the imperative need for proactive measures to regulate and diminish EDC exposure, thereby mitigating the escalating diabetes epidemic.

Keywords: Diabetes; endocrine-disrupting chemical; mathematical model; optimal control; simulation

AMS 2020 Classification: 37M05; 37N25; 49K10; 92C60

1 Introduction

Disease has always been a part of human life. Malaria, tuberculosis, plague, and other infectious diseases have decimated human life. The researcher is beginning to predict how the disease will progress and understand how interventions will affect its spread. The mechanisms and kinds of interaction terms vary depending on the disease. Diabetes (a chronic disease) has become

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a significant burden for individuals, leading to a variety of health problems in recent years [1]. Diabetes and its consequences have increased globally, likely because of the increasing diabetes risk factors, particularly population aging and obesity. It is a disorder characterized by insulin hormone problems, according to the World Health Organisation (WHO) [2]. According to the American Diabetes Association (ADA) [3], it is a group of metabolic disorders characterized by hyperglycemia secondary to diabetes. Factors that increase one's likelihood of developing diabetes include getting older, leading an unhealthy life, not getting enough exercise, eating a high-calorie diet, having stress, being overweight, and so on [4].

Despite incredible advances in biomedical sciences, diabetes remains an irreversible lifetime disease. Over the past 30 years, the number of people with diabetes has risen quickly in all age and gender groups, as well as in developing and developed countries. According to the International Diabetes Federation (IDF) [5], the prevalence of diabetes has risen even more by over 40 million people over the past quarter century. More than 540 million people had diabetes in 2021. If the current growth rate continues, this number will reach 780 million by 2045. According to the WHO [2], 1.6 million people died of diabetes in 2016, making it the seventh leading cause of death. In 2015, the Malaysian National Health Movement Survey (NHMS) found that 17.5% of adults over the age of 18 had diabetes [6]. Following that, the Malaysian province predicted a 10-year diabetes prevalence project and estimated that the diabetic population will increase by 31.3 percent by 2025 [7]. In 2021, diabetes caused the deaths of 6.7 million people worldwide [5]. It is associated with a 75 percent increase in adult mortality [8]. Hyperglycemia can lead to complications. Retinopathy, nephropathy, neuropathy, and an increased incidence of heart disease and stroke are other complications [9].

During this time of rising diabetes rates, humanity has witnessed large production and release of Endocrine-disrupting chemicals. Endocrine-disrupting chemicals (EDCs) can be either man-made or natural. Because their structure is nearly identical to steroid hormones, they could perhaps interact with hormones, androgen, and progesterone receptors, interfering with any aspect of endogenous hormone function, including biosynthesis, metabolism, transport, elimination, or receptor binding of endogenous hormones, increasing the risk of endocrine and metabolic diseases in humans and animals [10]. An endocrine disruptor is any chemical or chemical mixture from the outside that can interfere with hormones work [11]. According to the European Union, 147 of the 564 chemicals proposed by various organizations as potential EDC in scientific research or reports remain in the ecosystem or are produced in large quantities [12]. Plasticizers (Phthalates and Bisphenol A (BPA) or its derivative bisphenol S (BPS)) and pesticides such as dichlorodiphenyltrichloroethane (DDT), etc. are the most dangerous hazards to human health [13]. Prolonged repeated exposure to EDC compounds with concentrations even lower than the human body's established tolerance threshold for individual substances will also significantly increase the risk of hormonal and metabolic diseases such as diabetes both in men and women [14].

In addition, the development of modern civilization and the growing demand for new chemicals have raised our vulnerability to EDC. The release of these chemicals from everyday objects like food packaging, plastic water bottles, makeup, cash register receipts, clothing, food, contact lenses and dental sealants increases exposure [15]. Some EDCs may be more common in babies and young children than adults due to increased consumption of specific foods and water [16]. Researchers discovered that higher plasma concentrations of perfluorooctanesulfonic acid (PFOS) and perfluorooctanoic acid (PFOA) were associated with an increased risk of Type 2 diabetes (T2D) after controlling for common T2D risk factors such as BMI, family history and physical activity [17].

Prolonged repeated exposure to EDC compounds with concentrations even lower than the human body's established tolerance threshold for individual substances will also significantly increase the risk of hormonal and metabolic diseases such as diabetes both in men and women [14].

Researchers discovered that exposure to any pesticide was associated with a 61% increased risk of T2D in a meta-analysis of 21 prior studies involving over 66,000 people, with some pesticides appearing riskier than others [18].

In many models, authors have tried to describe how diabetes increases among people. Boutayeb et al. [19, 20] introduced a diabetic model, demonstrating the incidence of diabetes and its complications. Derouich et al. [21] proposed an optimal control approach to model the progression of diabetes from prediabetes, with or without control. Widyaningsih et al. [22] analyzed a mathematical model of diabetes with lifestyle and genetic factors. Bassey [23] analyzed the optimal control model for dual treatment of delayed type-II diabetes. Jajarmi et al. [24] created a new and efficient numerical method for the fractional modeling of diabetes and tuberculosis co-existence. Akinsola et al. [25] executed a mathematical analysis with numerical solutions of the diabetes mellitus model with optimal control. Ndii et al. [26] have tried to control the effect of hard water. Anusha et al. [27] studied mathematical modelling co-existence of diabetes and COVID-19 in deterministic and stochastic Approaches. Özköse et al. [28] investigated the interaction between COVID-19 and diabetes using real data. Agwu et al. [29] also analyzed the diabetes and tuberculosis co-existence model. Mollah et al. [30] studied the Optimal control for the diabetes model with an awareness program and treatment. Singh et al. [31] investigated the calcium distribution in the alpha-cell. Balakrishnan et al. [32] created a fractional-order control model for diabetes. A growing body of evidence suggests that environmental chemicals are linked to the rising prevalence of T2D. Therefore, We used the basic diabetes model [19, 21] to develop the model. Our primary goal in this paper is to reduce EDC exposure to reduce diabetes prevalence. The novelty of the proposed model is outlined by the following points:

- A new model was developed to determine the impact of EDC exposure on the diabetes population.
- A food population which gives a more realistic insist for the prevalence of diabetes.
- An optimal control problem is introduced with Possible control variables to reduce the effect of EDC and the prevalence of diabetes.
- The results for simulating different compartments of the model for the parameters b and r describe the effect of EDC Exposure.
- The proposed model provides some new ideas about the dynamic behavior of diabetes.

In Section 2, the model's formulation is built and briefly discussed. In Section 3, an optimal control problem is proposed. Furthermore, we established some results for the existence and characterization of optimal control. The numerical simulation is performed to validate the theoretical results discussed in Section 4.

2 Model formulation

We construct a diabetes model predicting the growing diabetic population, which suggests that higher EDC concentration levels in our daily routine (food, water, etc.) may be linked to the prevalence of diabetes. The impact of EDC usage is a chief concern since a growing body of evidence from studies has also shown a link between early EDC exposure and the prevalence of T2D late in life. Thus, we have developed a class F to describe the level of EDC present in the usual diet and lifestyle. The concentration of EDC intake increases at rate b and is limited by carrying capacity K, which equals the maximum solubility of each compound in food, air, soil, water and so on. When consuming EDC-exposed products at a rate of $\beta_H \frac{F}{F+K}$, people become exposed. β_H represents the rate at which healthy individuals consume EDC daily. The probability of individuals exposed to EDC is determined by the equation $\frac{F}{F+K}$, where K is the maximum concentration of EDC in a food product. The maximum chance of developing diabetes is set at 0.5.

Therefore, the maximum EDC concentration in a food product was equal to its carrying capacity K. It is also feasible to transition back to a normal lifestyle at a rate of α_3, provided that one is cognizant of EDC and adopts a health-conscious way of living. The variables and parameters of our model are outlined in Table 1 and Table 2.

Table 1. Model variables and their descriptions

Variables	Description
P	Healthy popoulation
S	Pre-diabetes population
D	Diabetes population
C	Diabetes population with complication
E	Exposed population
F	Food exposed with EDC

Table 2. Model parameters, their descriptions and values

Parameters	Description	Values	Source
Λ	Recruitment rate	$\frac{10^6}{365}$	[33]
β_H, β_1	Rate of ingesting of EDC	0.2	Assumed
μ	Natural death rate	$\frac{1}{365*65}$	[33]
α_1	Rate of healthy persons to become pre-diabetic	0.1	[34]
α_2	Rate at which a pre-diabetic person becomes healthy	0.02	[34]
α_3	Rate at which a exposed person becomes healthy	0.05	Assumed
ϵ	Probability of people to have complication	0.3	Assumed
γ_1	Probability of a pre-diabetic to become diabetic	0.1	[35]
γ_2	Probability of a diabetic developing a complications	0.1	[35]
γ_3	Probability of a pre-diabetic developing a complication	0.1	[35]
θ_1	Probability of a Exposed to become diabetic	0.05	Assumed
θ_2	Probability of a Exposed developing a complication	0.033	Assumed
b	Rate at which concentration of EDC increase	0.3	Assumed
r	Rate at which concentration of EDC decrease by control	0.1	Assumed
δ	Disease induced death rate	$\frac{1}{365*40}$	[33]

By taking into account the model parameters description and flow diagram given in Figure 1, the system of equations is provided as follows:

$$\begin{aligned}
\frac{dP}{dt} &= \Lambda - (\alpha_1 + \beta_H \frac{F}{F+K} + \mu)P + \alpha_2 S + \alpha_3 E, \\
\frac{dS}{dt} &= \alpha_1 P - (\gamma_1 + \gamma_3 + \alpha_2 + \beta_1 \frac{F}{F+K} + \mu)S, \\
\frac{dD}{dt} &= \left(\gamma_1 + (1-\epsilon)\beta_1 \frac{F}{F+K}\right) S - (\gamma_2 + \mu)D + \theta_1 E, \\
\frac{dC}{dt} &= \left(\gamma_3 + \epsilon\beta_1 \frac{F}{F+K}\right) S + \gamma_2 D + \theta_2 E - (\mu + \delta)C, \\
\frac{dE}{dt} &= \beta_H \frac{F}{F+K} P - (\theta_1 + \theta_2 + \alpha_3 + \mu)E, \\
\frac{dF}{dt} &= bF\left((1 - \frac{F}{K}\right) - rF,
\end{aligned} \tag{1}$$

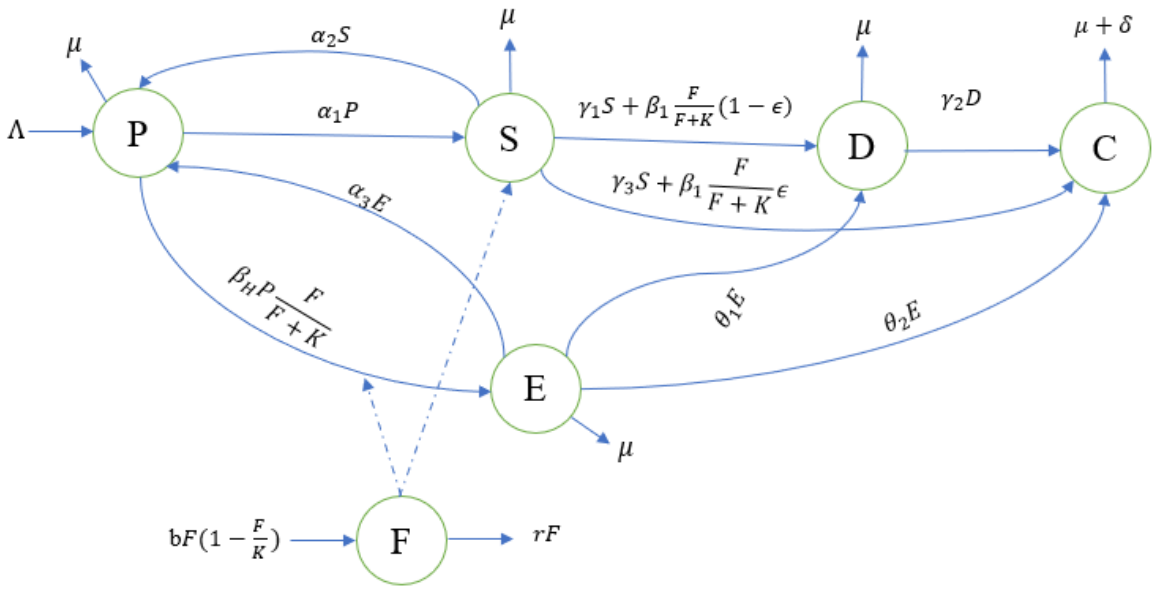


Figure 1. A flow diagram for diabetes model with the effects of EDC

with initial conditions

$$P(0) \geq 0,\ \ S(0) \geq 0,\ \ D(0) \geq 0,\ \ C(0) \geq 0,\ \ E(0) \geq 0, \text{ and } F(0) \geq 0. \tag{2}$$

For the diabetes model (1), it is needed to show that its state variables are non-negative for all time $t > 0$ and that the feasible region is bounded is studied in the following theorems:

Theorem 1 *Suppose that the initial condition (2) of system (1) be non-negative, then the solution $P(t) \geq 0$, $S(t) \geq 0$, $D(t) \geq 0$, $C(t) \geq 0$, $E(t) \geq 0$ and $F(t) \geq 0$ are also non-negative $\forall t > 0$.*

Proof Now, let us take the first equation of system (1) as follows

$$\begin{aligned} \frac{dP}{dt} &= \Lambda - (\alpha_1 + \beta_H \frac{F}{F+K} + \mu)P + \alpha_2 S + \alpha_3 E \\ &\geq -(\alpha_1 + \beta_H \frac{F}{F+K} + \mu)P, \end{aligned}$$

$$\frac{dP}{dt} + \{\alpha_1 + \beta_H \frac{F}{F+K} + \mu\}P \geq 0.$$

Then we obtain, $\frac{d}{dt}P(t)\exp(\int_0^t(\alpha_1 + \beta_H \frac{F}{F+K} + \mu)ds) \geq 0$. Integrating from 0 to t,

$$\int_0^t \frac{d}{dt}\left(P(s)\exp\left(\int_0^t\left(\alpha_1 + \beta_H \frac{F}{F+K} + \mu\right)ds\right)\right)ds \geq 0,$$

then

$$P(t) \geq P(0)\exp\left(\int_0^t\left(\alpha_1 + \beta_H \frac{F}{F+K} + \mu\right)ds\right) \implies P(t) \geq 0.$$

This shows that $P(t) \geq 0$ for all $t > 0$. Similarly, we can show for all other classes. ■

Theorem 2 *Let*

$$\omega_H = \left\{(P,S,D,C,E) \in \mathbb{R}^5_+, 0 \leq P+S+D+C+E \leq \frac{\Lambda}{\mu}\right\}, \tag{3}$$

and

$$\omega_F = \left\{F \in \mathbb{R}_+, 0 \leq F \leq K(1-\frac{r}{b})\right\}. \tag{4}$$

Define $\omega = \omega_H \times \omega_F$. *If* $N(0) \leq \frac{\Lambda}{\mu}$ *and* $F(0) \leq K(1-\frac{r}{b})$, *then the region* ω *is positively invariant under system (1) with initial condition (2) in* $\mathbb{R}^6+$.

Proof Let us consider system (1), we have human population $N = P + S + D + C + E$ and Food compartment F exposed with concentration of EDC. From adding first five equation of system (1), we have

$$\frac{dN}{dt} = \Lambda - \mu P - \mu S - \mu E - \mu D - \mu C - \delta C \leq \Lambda - \mu N,$$

which yields that

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

where Λ be the recruitment rate and $N(0)$ represents initial values of total population.

$$\lim_{t\to\infty} \sup N(t) = \frac{\Lambda}{\mu} = N_\infty.$$

Assuming $0 \leq N(0) \leq N_\infty$, we obtain that $0 \leq N(t) \leq N_\infty$, for all $t > 0$. For this reason, we define a separate feasible region ω_H for the human population as in (3). For the food compartment, it follows that

$$\frac{dF}{dt} = bF\left(1-\frac{F}{K}\right) - rF.$$

Let

$$F_\infty = K(1-\frac{r}{b}).$$

Note that F_∞ is the stable equilibrium point of the above differential equation. Assuming $0 \leq F(0) \leq F_\infty$. We obtain that $0 \leq F(t) \leq F_\infty$. Our compartment F doesn't exceed F_∞. We get feasible region ω_F for the Food compartment as in (3). Therefore, $N(t)$ and $F(t)$ are bounded for all $t > 0$, respectively. Hence every solution of system (1) with initial condition (2) in ω are remains in ω. ■

3 Optimal control problem

In this section, we used an optimal control approach to reduce the consumption of EDC-exposed food products by individuals at higher risk of T2D. In our model (1), we have included the following controls to reduce the impact of EDC among Healthy people as well as Diabetes people.

- u_1 be the percentage of healthy people prevented from pre-diabetes.
- u_2 be the people prevented from consumption of EDC.
- u_3 be a treatment for exposed.
- u_4 be the control implemented to decrease the level of EDC.

The optimal control problem for the system (1) is given in the following system of equation.

$$\begin{aligned}
\frac{dP}{dt} &= \Lambda - (\alpha_1(1-u_1) + \beta_P(1-u_2)\frac{F}{F+K} + \mu)H + \alpha_2 S + (\alpha_3 + pu_3)E, \\
\frac{dS}{dt} &= \alpha_1(1-u_1)H - (\gamma_1 + \gamma_3 + \alpha_2 + \beta_1(1-u_2)\frac{F}{F+K} + \mu)S, \\
\frac{dD}{dt} &= (\gamma_1 + (1-\epsilon)\beta_1(1-u_2)\frac{F}{F+K})S - (\gamma_2 + \mu)D + \theta_1 E, \\
\frac{dC}{dt} &= (\gamma_3 + \epsilon\beta_1(1-u_2)\frac{F}{F+K})S + \theta_2 E + \gamma_2 D - (\mu + \delta)C, \\
\frac{dE}{dt} &= \beta_P(1-u_2)\frac{F}{F+K}P - (\theta_1 + \theta_2 + \alpha_3 + \mu + pu_3)E, \\
\frac{dF}{dt} &= bF(1-\frac{F}{K}) - u_4 F.
\end{aligned} \tag{5}$$

The problem is to minimize the objective functional J defined as.

$$J(u_1(t), u_2(t), u_3(t), u_4(t)) = \int_0^T \left(A_1 S + A_2 C + A_3 D + A_4 E + A_5 F + \frac{B_1 u_1^2}{2} + \frac{B_2 u_2^2}{2} + \frac{B_3 u_3^2}{2} + \frac{B_4 u_4^2}{2} \right) dt, \tag{6}$$

where A_i, B_i, $i = 1$ to 4 are cost coefficients. They are selected to weigh the relative importance of u_i, $i = 1$ to 4 at time t, T is the final time. In other words, we seek the optimal controls u_i^*, $i = 1$ to 4 such that

$$J(u_1^*, u_2^*, u_3^*, u_4^*) = \min_{u_i \in U} J(u_1, u_2, u_3, u_4), \tag{7}$$

where U is the set of admissible controls defined by

$$\begin{aligned}
U = \{(u_i)/0 \le u_{1_{\min}} \le u_1(t) \le u_{1_{\max}} \le 1, 0 \le u_{2_{\min}} \le u_2(t) \le u_{2_{\max}} \le 1, \\
0 \le u_{3_{\min}} \le u_3(t) \le u_{3_{\max}} \le 1, 0 \le u_{4_{\min}} \le u_4(t) \le u_{4_{\max}} \le 1, t \in [0, T]\}.
\end{aligned}$$

$$H(t) = (A_1 S + A_2 C + A_3 D + A_4 E + A_5 F) + \left(\frac{B_1 u_1^2}{2} + \frac{B_2 u_2^2}{2} + \frac{B_3 u_3^2}{2} + \frac{B_4 u_4^2}{2} \right) + \sum_1^{11} \lambda_i f_i(P, S, D, C, E, F), \tag{8}$$

where f_i is the R.H.S of differential equation (5) of i^{th} state variable.

Existence of the optimal control

Using the result of Fleming and Rishel [36], we can prove the existence of optimal control. It follows that the set of controls and corresponding state variables is non-empty. Also, the control space U is convex and closed by definition. All the R.H.S of equation (5) is continuous, bounded above by a sum of bounded control and state and can be written as a linear function of u_i with a coefficient depending on the time and state. The integrant in the objective function is convex on U.

$$L(y, u_i, t) \geq -\eth_1 + \eth_2|u_1|^{\eth} + \eth_3|u_2|^{\eth} + \eth_4|u_3|^{\eth} + \eth_5|u_4|^{\eth}.$$

Thus, the results satisfy all the conditions mentioned in Fleming and Rishel's work [36]. Therefore, we establish the following theorem:

Theorem 3 *Consider the control problem with the system (5). There exists an optimal control u_i, $i = 1$ to $4 \in U^4$ such that*

$$J(u_1^*, u_2^*, u_3^*, u_4^*) = \min_{u_i \in U} J(u_1, u_2, u_3, u_4). \tag{9}$$

Proof The existence of the optimal control obtained using the result of Fleming and Rishel [36], checking the following steps:

- It follows that the controls and corresponding state variables are non-empty. We will use a simplified version of an existence result.
- $J(u_1(t), u_2(t), u_3(t), u_4(t))$ is convex in U.
- The control space $U = (u_i)/u_i$, $i = 1$ to 4 is measurable. $0 \leq u_{1_{\min}} \leq u_1(t) \leq u_{1_{\max}} \leq 1$, $0 \leq u_{2_{\min}} \leq u_2(t) \leq u_{2_{\max}} \leq 1$,$0 \leq u_{3_{\min}} \leq u_3(t) \leq u_{3_{\max}} \leq 1$, $0 \leq u_{4_{\min}} \leq u_4(t) \leq u_{4_{\max}} \leq 1$, $t \in [0, T]$ is convex and closed by definition.
- All the R.H.S of equation (5) is continuous, bounded above by a sum of bounded control and state and can be written as a linear function of u_i with a coefficient depending on the time and state.
- The integrant in the objective functional $\left(\frac{B_1 u_1^2}{2} + \frac{B_2 u_2^2}{2} + \frac{B_3 u_3^2}{2} + \frac{B_4 u_4^2}{2}\right)$ is clearly convex on U.
- Since the solution of system (5) is bounded, the system satisfies the Lipshitz property with respect to the variables P, S, D, C, E and F. Therefore, there exists an optimal control.

Hence, from Fleming and Rishel [36], we conclude that there exists an optimal control. ■

Characterization of the optimal control

To derive the necessary conditions for the optimal control, we apply Pontryagin's maximum principle to the Hamiltonian H given by equation (8) at time t.

Theorem 4 *Given the optimal control (u_1, u_2, u_3, u_4) and the solution P^*,S^*,D^*,C^*,E^*,F^* of the corresponding state system (5), there exists adjoint variable λ_i, for i = 1 to 6 satsifying*

$$-\frac{d\lambda_p}{dt} = \frac{\partial H}{\partial P}, -\frac{d\lambda_s}{dt} = \frac{\partial H}{\partial S}, -\frac{d\lambda_d}{dt} = \frac{\partial H}{\partial D}, -\frac{d\lambda_c}{dt} = \frac{\partial H}{\partial C}, -\frac{d\lambda_e}{dt} = \frac{\partial H}{\partial E}, -\frac{d\lambda_f}{dt} = \frac{\partial H}{\partial F},$$

with the transversality conditions at time $T, \lambda_j(T) = 0$, $j = p, s, d, c, e, f$. Furthermore, for t ∈ [0,T], the

optimal controls $u_1^*, u_2^*, u_3^*, u_4^*$ *are given by*

$$u_1^*(t) = \max\left\{0, \min\left\{1-\varepsilon, \frac{(\lambda_2-\lambda_1)\alpha_1 P}{B_1}\right\}\right\},$$
$$u_2^*(t) = \max\left\{0, \min\left\{1-\varepsilon, \frac{(\lambda_1-\lambda_5)\beta_P P}{B_2}\frac{F}{F+K} + \frac{(\lambda_3-\lambda_2)\beta_1 S}{B_2}\frac{F}{F+K} + \frac{(\lambda_4-\lambda_3)\epsilon\beta_1 S}{B_2}\frac{F}{F+K}\right\}\right\},$$
$$u_3^*(t) = \max\left\{0, \min\left\{1-\varepsilon, \frac{(\lambda_5-\lambda_1)p_1 E}{B_3}\right\}\right\},$$
$$u_4^*(t) = \max\left\{0, \min\left\{1-\varepsilon, \frac{\lambda_6 F}{B_4}\right\}\right\}.$$

Proof For $t \in [0, T]$, the adjoint equation and transversality conditions obtained by using Pontryagin's principle such that

$$\begin{aligned}
\lambda_1' &= \lambda_1\left(\alpha_1(1-u_1) - \beta_P(1-u_2)\frac{F}{F+K} + \mu\right) - \lambda_2\alpha_1(1-u_1) - \lambda_5(1-u_2)\beta_P\frac{F}{F+K},\\
\lambda_2' &= -A_1 - \lambda_1\alpha_2 + \lambda_2(\gamma_1+\gamma_3+\alpha_2+\beta_1(1-u_2)\frac{F}{F+K}+\mu) - \lambda_3(\beta_1(1-\epsilon)(1-u_2)\frac{F}{F+K}+\gamma_1)\\
&\quad -\lambda_4(\epsilon\beta_1(1-u_2)\frac{F}{F+K}+\gamma_3),\\
\lambda_3' &= -A_2 + \lambda_3(\gamma_2+\mu) - \lambda_4\gamma_2,\\
\lambda_4' &= -A_3 + \lambda_4(\mu+\delta),\\
\lambda_5' &= -A_4 - \lambda_1(\alpha_3+p_1u_3) - \lambda_3\theta_1 - \lambda_4\theta_2 + \lambda_5(\theta_1+\theta_2+\alpha_3+p_1u_3+\mu),\\
\lambda_6' &= -A_5 + \beta_P P(\lambda_1-\lambda_5)(1-u_2)\frac{K}{(K+F)^2} + \beta_1 S(\lambda_2-\lambda_3(1-\epsilon)-\lambda_4\epsilon)(1-u_2)\frac{K}{(K+F)^2}\\
&\quad -\lambda_6\left(b(1-\frac{F}{K}) - b\frac{F}{K} - u_4\right),
\end{aligned}$$

with transversality conditions $\lambda_i = 0$, $i = 1$ to 11. For $t \in [0, T]$, the optimal controls $u_1^*, u_2^*, u_3^*, u_4^*$ can be solved by the optimality conditions $\dfrac{\partial H}{\partial u_i}$.

$$\begin{aligned}
u_1^*(t) &= \frac{(\lambda_2-\lambda_1)\alpha_1 P}{B_1},\\
u_2^*(t) &= \frac{(\lambda_1-\lambda_5)\beta_P P}{B_2}\frac{F}{F+K} + \frac{(\lambda_3-\lambda_2)\beta_1 S}{B_2}\frac{F}{F+K} + \frac{(\lambda_4-\lambda_3)\epsilon\beta_1 S}{B_2}\frac{F}{F+K},\\
u_3^*(t) &= \frac{(\lambda_5-\lambda_1)p_1 E}{B_3},\\
u_4^*(t) &= \frac{\lambda_6 F}{B_4}.
\end{aligned}$$

By the bounds in U of the controls, it is easy to obtain the optimal controls. ■

4 Numerical simulation and discussion

Simulation is required to understand the reasoning behind theoretical findings. It changes according to the values assigned to the parameters. We stimulate the diabetes model using Euler's method. The optimal control problem is solved using the Forward-backward sweep method.

Initial and final conditions exist for state and adjacent systems, respectively. The weight constants and initial conditions are $A_1 = A_2 = A_3 = A_4 = A_5 = 1$, $B_1 = B_2 = B_3 = B_4 = 2000$.
The parameter values described in Table 2 are applied to simulate the diabetes model using the Matlab program. We have used diabetes data for India from 1980 to 2015. The diabetes dataset is available on the NCD-RisC website (https://ncdrisc.org/index.html). Then, using manual calibration, we fitted each parameter to get the best fit to our proposed model (1). Figure 2 shows that our model fits almost to the dataset. The range of parameter values used in calibration are from the literature. The parameters b and r are essential for regulating the EDC density of the food.

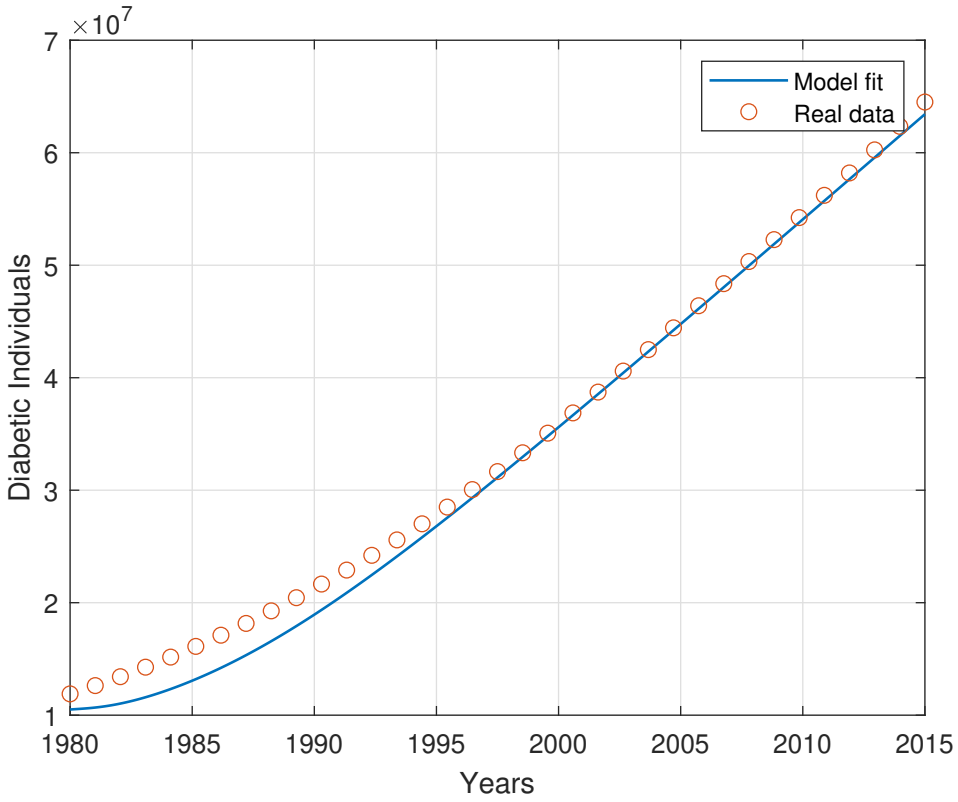
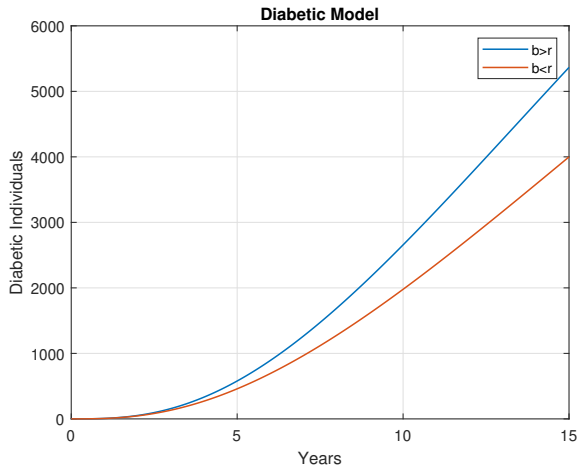


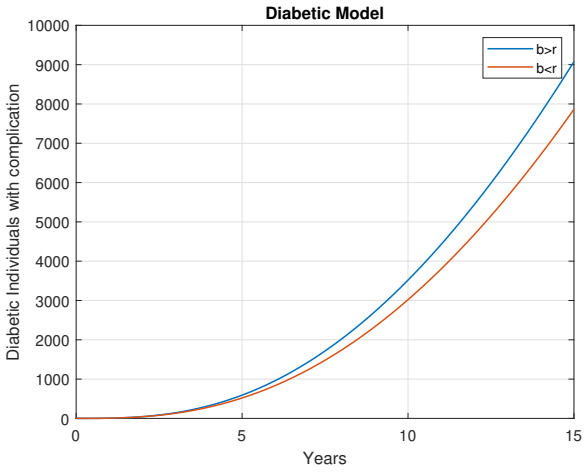
Figure 2. The diabetes population data from 1980 to 2015 in India and best curve fit of the proposed model

As a basic guideline, r must be higher than b. It means that r is the controlling parameter of EDC in any product. Parameter b is higher than parameter r. It represents that higher concentrations of EDC in food may affect humans. Every population with $b < c$ and $b > c$ is depicted in Figure 3.

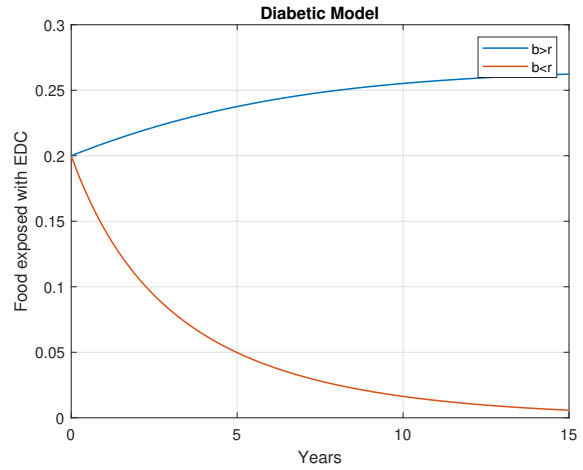
It noted that whenever the control parameter r fails to control the level of Endocrine, the diabetes prevalence increases. Figure 4 depicts each compartment with and without control. Diabetes is largely preventable by taking the proposed control variable. Figure 5 illustrates the control profile with $B_4 = 20$ and $B_4 = 2000$. The graph indicates that if control costs are low, people can afford them for a long time. If the control cost is reasonable, then more individuals will be able to get better. According to the findings in Figure 6, the concentration of EDC in food products is reduced more effectively over time if the cost of control is affordable and the exposed population seems minimized. The graph indicates that lowering the concentration of EDC impacts $T2D$, although other regulations are applied to reduce diabetes incidence. The prevalence of diabetes has decreased after implementing the necessary controls. The graph clearly shows that the lower the control costs, the higher the likelihood of recovery.



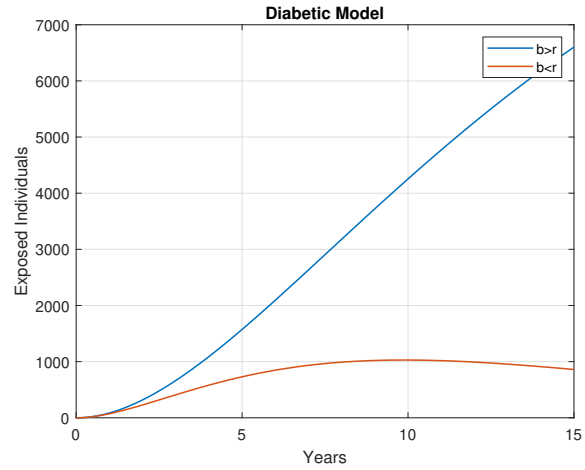
(a) Profile of diabetes population for $b > r$ and $b < r$



(b) Profile of diabetes population with complication for $b > r$ and $b < r$

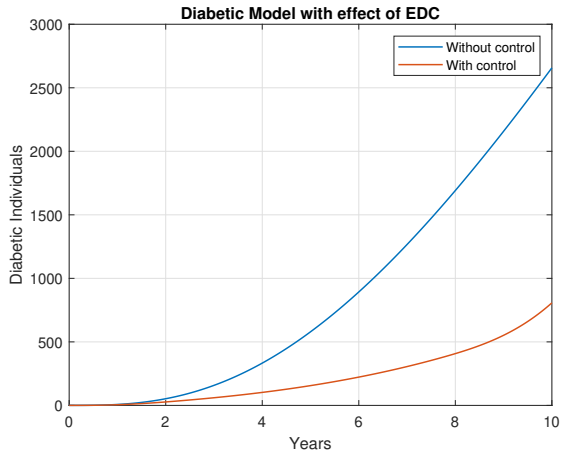


(c) Profile of food exposed with EDC compartment for $b > r$ and $b < r$

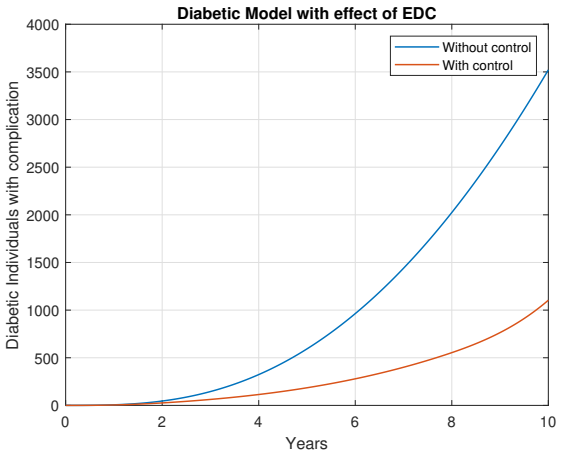


(d) Profile of exposed population with EDC for $b > r$ and $b < r$

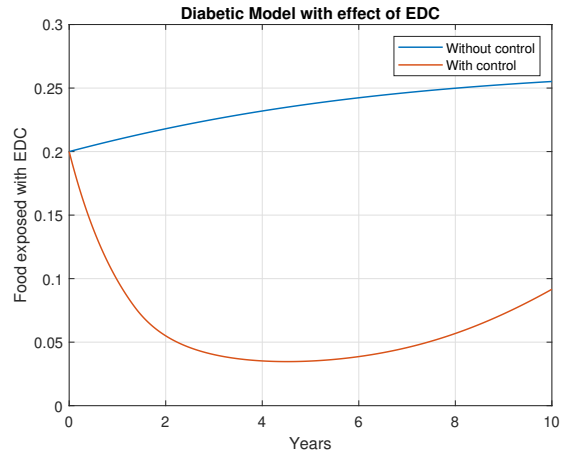
Figure 3. The dynamic of variables D, C, E, F for $b > r$ and $b < r$



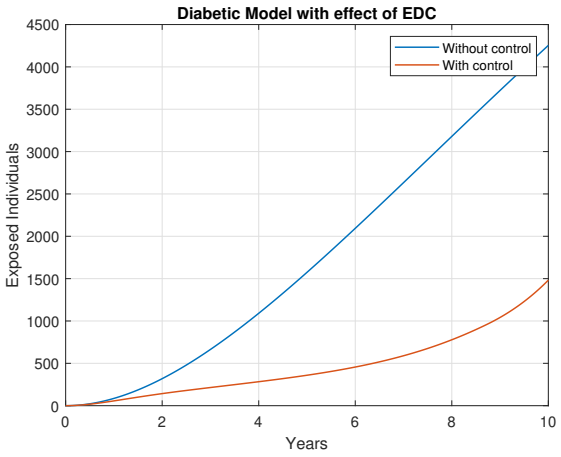
(a) Profile of diabetes population without control and with control



(b) Profile of diabetes population with complication without control and with control

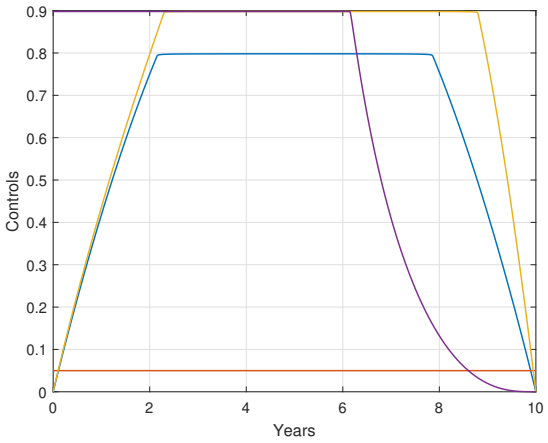


(c) Profile of food exposed with EDC compartment without control and with control

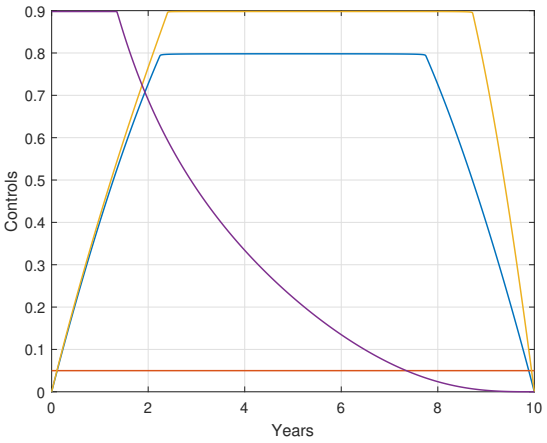


(d) Profile of exposed population with EDC without control and with control

Figure 4. The dynamic of variables with and without control

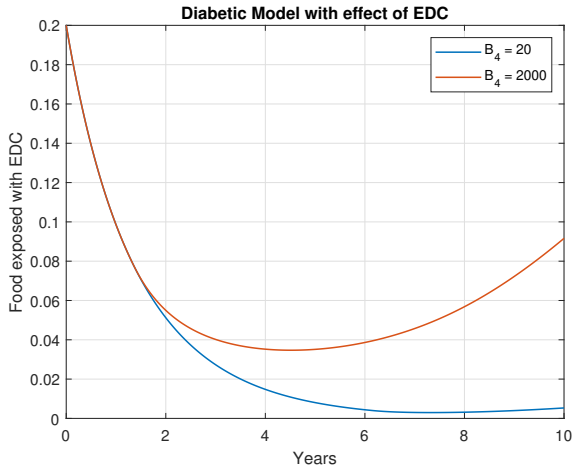


(a) Profile of control with $B4 = 50$

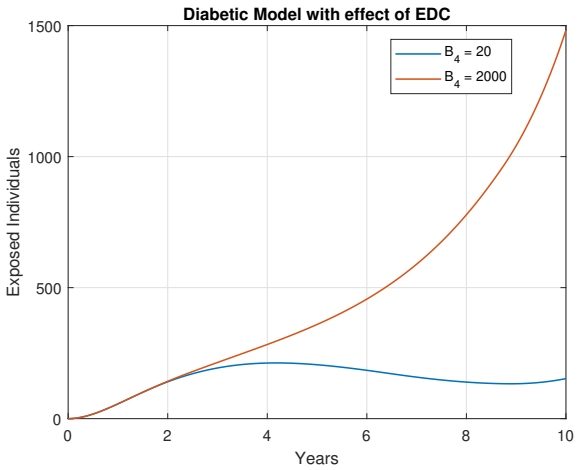


(b) Profile of control with $B4 = 5000$

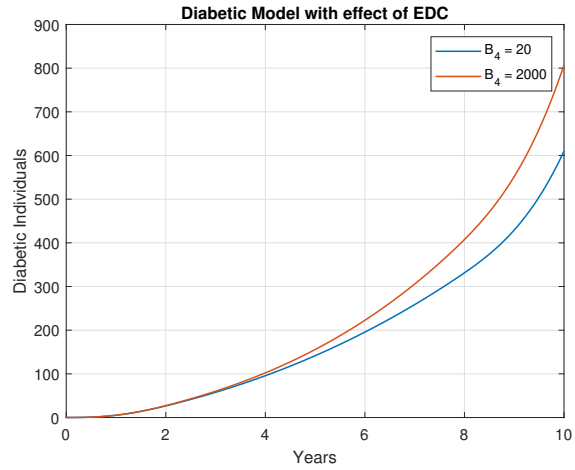
Figure 5. The control profile with different values of cost of controls



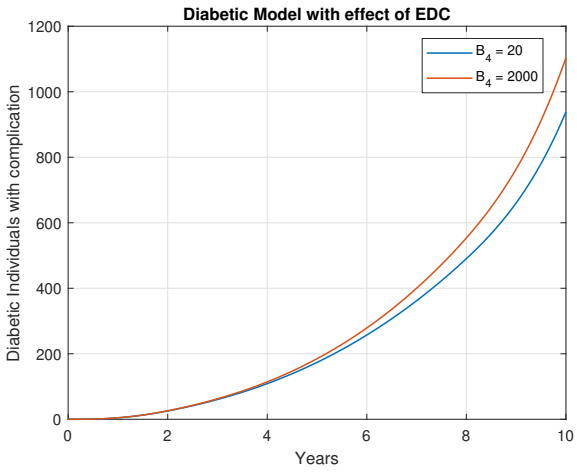
(a) Profile of food exposed with EDC with B4 = 20 and 2000



(b) Profile of exposed with EDC with B4 = 20 and 2000



(c) Profile of diabetes population with B4 = 20 and 2000



(d) Profile of diabetes population with complication with B4 = 20 and 2000

Figure 6. The profile of E, D, C and F with different values of the cost of controls

5 Conclusion

In this paper, we have developed a mathematical model of the diabetic population with the effect of EDC. This proposed model offers a different approach to understanding the prevalence of diabetes, particularly when the daily consumption of food is exposed to some harmful chemicals that lead to health problems. A suitable control strategy discussed includes intervention for exposed people, diabetes prevention, control of EDC concentration on daily consumption, and prevention of consuming EDC. We have found the optimal control strategies that are more effective in controlling the prevalence of diabetes. The findings demonstrate the efficacy of the proposed control strategies. The results show that less EDC exposure is better for diabetes control. In the future, one can try to incorporate other sources of T2D with fractional-order differential equations and cost-effective analysis to improve the effective way of controlling diabetes. Also, studying the nature of equilibrium and stability analysis can be considered.

Declarations

List of abbreviations

Not applicable.

Ethical approval

The authors state that this research complies with ethical standards. This research does not involve either human participants or animals.

Consent for publication

Not applicable.

Conflicts of interest

The authors confirm that there is no competing interest in this study.

Data availability statement

Data availability is not applicable to this article as no new data were created or analyzed in this study.

Funding

Not applicable.

Author's contributions

L.P.: Conceptualization, Methodology, Software, Validation, Data Curation, Writing - Original Draft. M.C.: Writing - Review & Editing, Supervision. All authors have read and agreed to the published version of the manuscript.

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