The Mathematical Modeling of Diabetic Population with the Formulation of Optimal Control Strategies

Abstract

In this research, a robust mathematical model is formulated to capture both type-1 and type-2 diabetes progression among children and adults, accounting for cases with and without control measures. The effects of these control interventions on diabetic dynamics are explored, and the efficacy of the outlined strategies is assessed. In the optimal control formulation, Pontryagin's maximum (or minimum) principle is employed to derive optimal control characterizations, while the Runge-Kutta forward-backward sweep algorithm is applied for numerical simulations of state and adjoint variables. This study demonstrates that the number of advanced-stage diabetic patients and the inherent costs of managing diabetic progression can be minimized. The findings of this research offer valuable insights for policymakers and healthcare professionals in improving diabetes management.

Keywords Diabetes . Mathematical model . Simulation . Optimal control

1 *Introduction*

In diabetes research, mathematical modeling has emerged as an invaluable tool for understanding the complex mechanisms involved in blood glucose regulation. Diabetes, as defined by the World Health Organization [22], is a disorder resulting from insulin-related issues in the pancreas, leading to elevated blood sugar levels or hyperglycemia. This condition disrupts the normal metabolism of carbohydrates, fats, and proteins, causing various complications.

Globally, diabetes is a widespread condition affecting millions and poses significant challenges to health and well-being. The sustained high blood sugar levels characteristic of diabetes lead to a range of symptoms and complications, including cardiovascular disease, neuropathy, retinopathy, and nephropathy. These complications underscore the critical need for effective management and prevention strategies.

The disease is categorized mainly into two types: Type 1 diabetes, an autoimmune condition often diagnosed in childhood or adolescence [23], and Type 2 diabetes, which typically affects adults and is associated with insulin resistance or inadequate insulin production [3]. The causes of diabetes are diverse, ranging from genetic and environmental factors to lifestyle influences such as diet and physical activity [23].

In Nigeria, diabetes prevalence was historically low, but recent trends show an increase, particularly in urban areas. Misconceptions about the disease, such as the belief that it is caused by carbohydrate consumption, have led to inadequate dietary practices and poor glycemic control among patients[1][14]. National bodies like the Diabetes Association of Nigeria and the Endocrine and Metabolic Society of Nigeria are actively working to address these issues by promoting guidelines and public awareness.

Mathematical modeling, with its ability to capture complex systems through equations, provides a powerful framework for understanding diabetes. It allows for the simulation of disease progression, evaluation of treatment strategies, and exploration of various scenarios, thus aiding in the development of effective interventions.
In more than a decade, significant mathematical models for diabetes have been developed to simulate, analyze, and gain insights into the dynamics within diabetic populations. In related research, Boutayeb et al. (2014) [6] presented a strategy to control and model the progression of diabetes from the pre-diabetes stage to more advanced stages, including those with and without complications. They demonstrated that

an optimal control strategy exists to manage this progression. To do this, they used a numerical method called the implicit finite-difference method to track the number of people in each stage of diabetes over time. The drawback of this [6] research is that it does not consider the early stages and disabilities due to diabetes compartments in their model. Boutayeb et al. (2015) [7] built upon the mathematical model created by Boutayeb A, et al (2014), expanding it to encompass the dynamics of healthy individuals alongside those with pre-diabetes and diabetes. They introduce an optimal control approach aimed at reducing the overall burden of pre-diabetes and diabetes, including its associated complications. The model demonstrates that by implementing effective control measures, it is possible to significantly limit the numbers of individuals who develop pre-diabetes and diabetes, both with and without complications. Through this extended framework, the authors analyze the interactions within a population comprising healthy individuals, pre-diabetics, and diabetics over a 10-year period. They evaluate the outcomes of these populations under two scenarios: one without any control measures and another where optimal control strategies are actively applied. The results underscore the potential of targeted interventions to improve health outcomes, highlighting the importance of managing the progression of diabetes effectively to alleviate its impact on individuals and the healthcare system. This research not only adds depth to the existing model but also provides valuable insights for public health initiatives aimed at diabetes prevention and management. The drawback of this [7] research is that it does not consider the early stages and disabilities due to diabetes compartments in the model. Permatasari et al. (2018) [17] developed an optimal control mathematical model to manage the progression of diabetes. Their model includes the dynamics of individuals who become disabled due to diabetes. They proposed an optimal control approach to reduce the burden of pre-diabetes by preventing its progression to diabetes with or without complications. They discussed the existence and characterization of this optimal control using the Pontryagin minimum principle. The results show that an optimal control strategy exists within this mathematical model of the diabetic population. The findings indicate that the effectiveness of the control variable (prevention) is significantly influenced by the number of healthy people. By implementing this strategy, they aimed to lessen the impact of diabetes on individuals and society. The drawback of this [17] research is that it does not consider type-1 diabetes in the model. Kouidere et al. (2020) [13] reported that the rise in diabetes cases is closely linked to the increase in endocrine-disrupting chemicals (EDCs), which they identified as major contributors to insulin resistance and beta cell dysfunction, ultimately leading to the development of diabetes. They introduced a model to explain the impact of EDC exposure on the diabetic population and suggested optimal control strategies to reduce the harmful effects of EDC-induced diabetes. Utilizing Pontryagin's maximum principle, they explained how these optimal control methods operate within their model. Their findings indicated that high levels of EDCs significantly worsen the prevalence of diabetes. They substantiated their model with parameter estimation techniques using a diabetes dataset from India. The researchers emphasized the urgent need for proactive measures to control and reduce exposure to EDCs to help mitigate the growing diabetes epidemic. The drawback of the [13] research is that it does not consider diabetes with disabilities in the model.

Following previous mathematical models on diabetes, in this paper we present an optimal control model of diabetic population by considering the Type-1 class, Pancretic Problem class and the Insulin Resistance class. This paper focuses on developing a detailed mathematical model to study the progression of diabetes. The model aims to provide insights into optimizing treatment timing and improving public health strategies for diabetes management.

2 *Methodology*

2.1 *Model formulation*

The Model divides the total Population at any time t, denoted as $N(t)$, into eight mutually exclusive compartments, namely; Population of Suceptible Healthy Humans $H_s(t)$,Population of Humans with Type 1 Diabetes $T_1(t)$, Population of Humans with Pancreatic Problem $P_p(t)$, Population of Humans with Diabetes with insulin resistance $I_r(t)$, Population of Prediabetic Humans $P_d(t)$, Population of Humans with diabetes without complications $D_w(t)$, Population of Human with complication $D_c(t)$ and the Population of Humans with disabilities due to Diabetes D_d. Thus,

$$N(t) = H_s(t) + T_1(t) + P_p(t) + I_r(t) + P_d(t) + D_w(t) + D_c(t) + D_d(t) \quad (1)$$

Table 1: The Model variables

Variables	Description
$H_s(t)$	Susceptible Healthy Humans Population
$T_1(t)$	Humans with Type 1 Diabetes Population
$P_p(t)$	Humans with Pancreatic Problem Population
$I_r(t)$	Human with Insulin resistance Population
$P_d(t)$	Prediabetic Human Population
$D_w(t)$	Human with Diabetes Without complications Population
$D_c(t)$	Human with Diabetes with Complications Population
$D_d(t)$	Humans with Disabilities due to Diabetes Population

Description of the models variables and parameters

Table 2: The Model Parameters

Parameters	Description
ρ	Recruitment rate into Susceptible Human Population
θ_1	Proportion of Healthy Humans that became Prediabetic
θ_2	Proportion of Prediabetic Humans that became Diabetic with complications
θ_3	Prop. of Insulin Resistance pop. that became Diabetic without Compli.
θ_4	Proportion of Humans without Complicated Diabetes that became Disabled
σ_1	Rate of Progression into the Type 1 Diabetes Population
σ_2	Rate of Progression into Pancreatic Problem Population
σ_3	Rate of Progression into Insulin resistance Population
σ_4	Rate of Progression from Type 1 Diabetes to Prediabetic Human Population
σ_5	Rate of Progression from Pancreatic Problem Population to Prediabetic Human Population
σ_6	Rate of Progression from Insulin resistance population to Prediabetic Human Population
σ_7	Rate of Progression from the Prediabetic Population to Diabetes without Complications Human Population
σ_8	Rate of Progression from the Diabetes without Complications Population to Complicated Diabetes Human Population
σ_9	Rate of Progression from the Complicated Diabetes to Disabled Human Population
δ_1	Recovery rate of Type 1 diabetic Population to Susceptible human Population
δ_2	Recovery rate of Prediabetic Population to Susceptible Humans Population
δ_3	Recovery rate of Diabetes without Complications to Prediabetic Population
δ_4	Recovery rate of popopulation with complications to population without complications
μ	Natural Death rate
α	Mortality rate due to Diabetic Complications
ζ	Mortality Rate due to Diabetic Disabilities

2.2 *Assumptions for the model*

The proposed model assumes the following:

i Each compartment represents a stage in the diabetic progression.

ii A diabetic patient can move from a stage to another except for the I_r and the D_d stages.

iii There is a natural death rate in each compartment.

iv A patient can detriorate to a higher stage.

v The recovery for diabetes is progressive in nature.

vi Mortality can also occur as a result of diabetes in the stages (compartments).

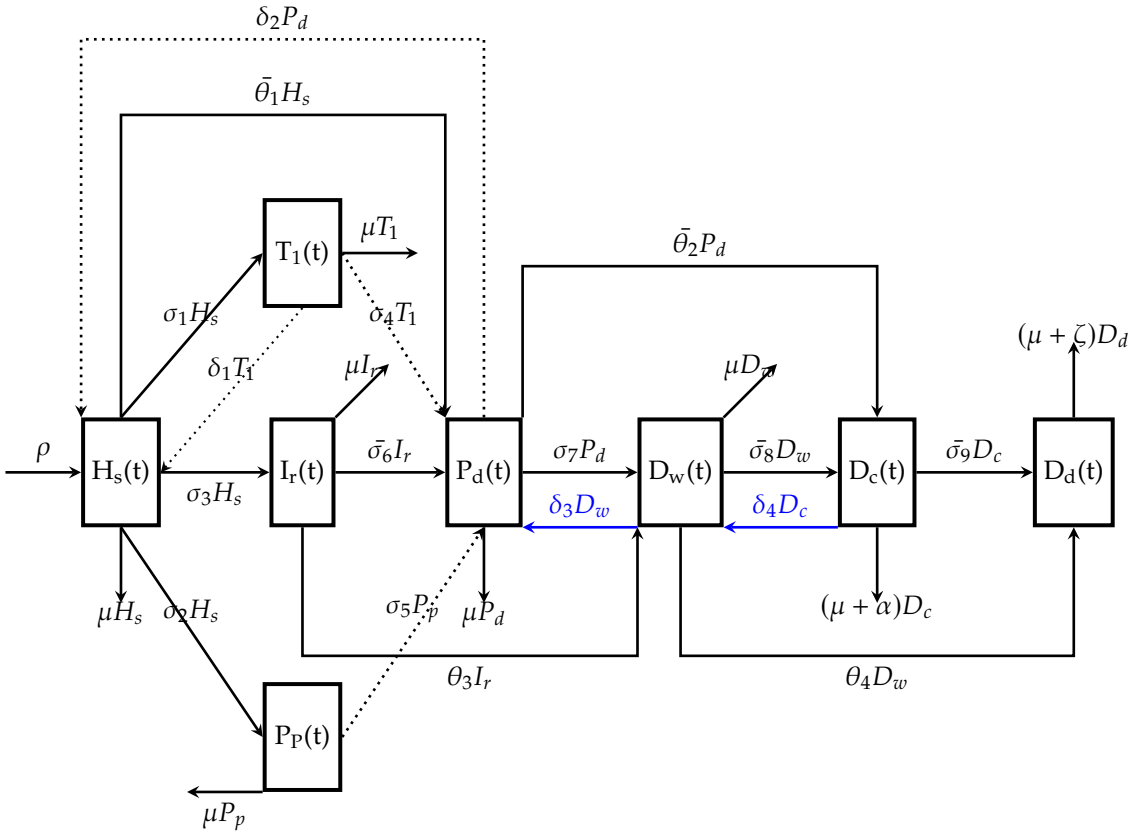

Figure 1: *Schematic diagram of Model of Diabetes with control*

where $\bar{\theta}_1 = \theta_1(1-u_1)$, $\bar{\theta}_2 = \theta_2(1-u_1)$, $\bar{\sigma_8} = \sigma_8(1-u_2)$ and $\bar{\sigma_9} = \sigma_9(1-u_2)$.
In the graphical representation of the proposed model shown in Figure 1 above, the compartment $H_s(t)$ of the healthy people is increased by ρ (which is the recruitment rate of healthy people), increased by $\delta_1 T_1$ (which is the recovery rate of Type-1 diabetes patients), decreased by $\theta_1 H_s$ (the number of prediabetic population), decreased by $\sigma_1 H_s$ (the number of Type-1 population), decreased by $\sigma_2 H_s$ (the number of Humans with Pancreatic problem population), decreased by $\sigma_3 H_s$ (the number of Insulin resistance population) and also decreased by μH_s (population due to natural mortality) as indicated in eqn. (2) below.
The compartment $T_1(t)$ comprises of population who are Type-1 diabetic. The compartment is increased by $\sigma_1 H_s$ (the population of healthy people), decreased by $\sigma_4 T_1$ (which is the proportion of population that becomes prediabetic), decreased by μT_1 (which is the proportion of Type-1 diabetic population due to natural mortality) and decreased by $\delta_1 T_1$ (which is the proportion of Type-1 diabetic population that recovered to the susceptible human population) as expressed in eqn. (3) below.
The compartment $P_p(t)$ comprises of population who have Pancreatic problem. The compartment is increased by $\sigma_2 H_s$ (the population of healthy people), deceased by $\sigma_5 P_p$ (which is the proportion of population that becomes prediabetic), decreased by μP_p (which is the proportion of Pancreatic problem population due to natural mortality) as expressed in eqn. (4) below.
The compartment $I_r(t)$ comprises of population who are resistant to insulin. The compartment is increased by $\sigma_3 H_s$ (the population of healthy people), decreased by $\sigma_6 I_r$ (which is the proportion of insulin resistant population that becomes prediabetic), decreased by μI_r (which is the proportion of insulin resistant population due to natural mortality), decreased by $\theta_3 I_r$ (which is the proportion of insulin resistant population that becomes diabetic without complications) and increased by $\delta_2 D_w$ (which is the proportion of prediabetic population that recovered to the insulin resistant population) as expressed in eqn. (5) below. The compartment $P_d(t)$ comprises of population who are prediabetic. The compartment is increased by $\sigma_6 I_r$ (which is the proportion of insulin resistant population that becomes prediabetic), increased by $\sigma_4 T_1$ (which is the proportion of population that becomes prediabetic), increased by $\sigma_5 P_p$ (which is the proportion of population that becomes prediabetic), increased by $\theta_1 H_s$ (which is the proportion of healthy population that becomes prediabetic), increased by $\delta_3 D_w$ (which is the proportion of diabetic without complications that recovers to prediabetic population), decreased by μP_d (which is the proportion of prediabetic population subject to natural mortality) and decreased by $\sigma_7 P_d$ (which is the proportion of prediabetic population that becomes diabetic without complications) as indicated in eqn. (6). The compartment $D_w(t)$ comprises of population who are diabetic without complications. The com-

partment is increased by $\sigma_7 P_d$ (which is the proportion of prediabetic population that becomes diabetic without complications), increased by $\theta_3 I_r$ (which is the proportion of insulin-resistance population that becomes diabetic without complications), increased by $\delta_4 D_c(t)$ (which is the proportion of diabetic with complications that recovers to population of diabetic without complications), deceased by μD_w (which is the proportion of diabetic without complications subject to natural mortality), decreased by $\sigma_8 D_w$ (which is the proportion of population of diabetic without complications that becomes diabetic with complications), decreased by $\theta_4 D_w$ (which is the proportion of population of diabetic without complications that becomes diabetic with disabilities population) and decreased by $\delta_3 D_w$ (which is the proportion of population of diabetic without complications that becomes prediabetic population) as indicated in eqn. (7). The compartment $D_c(t)$ comprises of population who are diabetic with complications. The compartment is increased by $\sigma_8 D_w$ (which is the proportion of diabetic without complications population that becomes diabetic with complications), increased by $\theta_2 P_d$ (which is the proportion of prediabetic population that becomes diabetic with complications), decreased by $\delta_4 D_c(t)$ (which is the proportion of diabetic with complications that recovers to population of diabetic without complications), decreased by μD_c (which is the proportion of diabetic with complications subject to natural mortality), decreased by $\sigma_9 D_c$ (which is the proportion of population of diabetic with complications that becomes diabetic with disabilities)and deceased by αD_c (which is the proportion of diabetic with complications subject to death due to diabetic complications) as indicated in eqn. (8).
Finally, the compartment with disabilities $D_d(t)$ is increased by $\sigma_9 D_c$ (which is the proportion of diabetic with complications population that becomes diabetic with disabilities), increased by $\theta_4 D_w$ (which is the proportion of diabetic without complications population that becomes diabetic with disabilities), decreased by $\mu D_d(t)$ (which is the proportion of population of diabetic with disabilities that die natural death) and decreased by $\zeta D_d(t)$ (which is the proportion of population of diabetic with disabilities that died as a result of the condition) as indicated in eqn. (9).

However the model reduces to the case of non control strategy if the control variables in the model is equated to zero (i.e $u_1(t), u_2(t) = 0$), and they are defined as: The initial control, $0 \leq u_1 \leq 1$, denotes the prevention of diabetes from the Type 1 diabetic stage (H_s) and Prediabetic stage (P_d) to the Prediabetic stage (P_d) and Diabetes with complications (D_c) respectively. The second control, $0 \leq u_2 \leq 1$, denotes the treatment of diabetes from the diabetes without complications(D_w) and Diabetes with complications (D_c) to Diabetes with complications (D_c) and Diaebetes with disabilities (D_d) respectively.

The dynamical equations representing the figure above, the description and the optimal control model for the diabetics progression are represented below.

$$\frac{dH_s}{dt} = \rho - (\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)H_s + \delta_1 T_1 + \delta_2 P_d \tag{2}$$

$$\frac{dT_1}{dt} = \sigma_1 H_s - (\delta_1 + \sigma_4 + \mu)T_1 \tag{3}$$

$$\frac{dP_p}{dt} = \sigma_2 H_s - (\sigma_5 + \mu)P_p \tag{4}$$

$$\frac{dI_r}{dt} = \sigma_3 H_s - (\sigma_6 + \theta_3 + \mu)I_r \tag{5}$$

$$\frac{dP_d}{dt} = \theta_1(1-u_1)H_s + \sigma_6 I_r + \sigma_4 T_1 + \sigma_5 P_p - (\theta_2(1-u_1) + \delta_2 + \sigma_7 + \mu)P_d + \delta_3 D_w \tag{6}$$

$$\frac{dD_w}{dt} = \theta_3 I_r + \sigma_7 P_d - (\mu + \delta_3 + \sigma_8(1-u_2) + \theta_4)D_w + \delta_4 D_c \tag{7}$$

$$\frac{dD_c}{dt} = \theta_2(1-u_1)P_d + \sigma_8(1-u_2)D_w - (\sigma_9(1-u_2) + \mu + \delta_4 + \alpha)D_c \tag{8}$$

$$\frac{dD_d}{dt} = \theta_4 D_w + \sigma_9(1-u_2)D_c - (\mu + \zeta)D_d \tag{9}$$

with initial conditions; $H_s(0) > 0$, $T_1(0) \geq 0$, $P_p(0) \geq 0$, $I_r(0) \geq 0$, $P_d(0) \geq 0, D_w(0) \geq 0$, $D_c(0) \geq 0$ and $D_d(0) \geq 0$.

2.3 *Analysis of the Model*

2.3.1 *Invariant Region*

Let the feasible region ϕ, of model (1), be defined by

$$\phi = \left\{(H_s(t), T_1(t), P_p(t), I_r(t), P_d(t), D_w(t), D_c(t), D_d(t)) \in \mathbb{R}^8 : N(t) \leq \frac{\rho}{\mu}\right\}$$

Then, the following theorem can be established.

Theorem 2.1. *The feasible region ϕ of the Diabetic Model as defined above is positively Invariant.*

Proof. Showing that ϕ is positively invariant, the total Population gotten by adding the corresponding components of the Model equations (2 - 9) per unit time is

$$N(t) = H_s(t) + T_1(t) + P_p(t) + I_r(t) + P_d(t) + D_w(t) + D_c(t) + D_d(t) \tag{10}$$

Hence, we have,

$$\frac{dN(t)}{dt} = \frac{dH_s(t)}{dt} + \frac{dT_1(t)}{dt} + \frac{dP_p(t)}{dt} + \frac{dI_r(t)}{dt} + \frac{dP_d(t)}{dt} + \frac{dD_w(t)}{dt} + \frac{dD_c}{dt} + \frac{dD_d}{dt}$$

From Equation (2 - 9), we have,

$$\frac{dN(t)}{dt} = \rho - \mu N(t) - \alpha D_c(t) - \zeta D_d(t), \tag{11}$$

Since $\alpha D_c(t) + \zeta D_d(t) \geq 0$, it then implies that

$$\frac{dN(t)}{dt} \leq \rho - \mu N(t). \tag{12}$$

integrating both sides,

$$\int \frac{dN(t)}{\rho - \mu N(t)} \leq \int dt$$

$$\frac{-1}{\mu} \ln(\rho - \mu N(t)) \leq t + c$$

where c is the constant of integration. Hence,

$$\ln(\rho - \mu N(t)) \geq -\mu t - \bar{c}$$

, where $\bar{c} = c\mu$. And multiplying both sides by exponential yields,

$$\rho - \mu N(t) \geq A \exp^{-\mu t}$$

where $A = exp^{-\bar{c}}$ is a constant of integration. Let $N(0) = N_0$, then

$$\rho - \mu N_0 \geq A$$

. Therefore,

$$\rho - \mu N(t) \geq (\rho - \mu N_0) \exp^{-\mu t}$$

$$N(t) \leq \frac{\rho}{\mu} - \frac{(\rho - \mu N_0)}{\mu} \exp^{-\mu t}$$

$$\lim_{t \to 0} N(t) \leq \lim_{t \to 0} \left[\frac{\rho}{\mu} - \frac{(\rho - \mu N_0)}{\mu} \exp^{-\mu t}\right]$$

$$N(t) \leq \frac{\rho}{\mu} \ \ as \ \ t \to \infty$$

where $N(t)$ exists within the feasible region $\phi = \left[0, \frac{\rho}{\mu}\right]$ of the diabetic model for $H_s(t) \geq 0$, $T_1(t) \geq 0$, $P_p(t) \geq 0$, $I_r(t) \geq 0$, $P_d(t) \geq 0$, $D_w(t) \geq 0$, $D_c(t) \geq 0$, $D_d(t) \geq 0$ and hence, it's positively Invariant. □

2.3.2 *The Positivity Theorem*

Theorem 2.2. *Let $H_s(0) \geq 0$, $T_1(0) \geq 0$, $P_p(0) \geq 0$, $I_r(0) \geq 0$, $P_d(0) \geq 0$, $D_w(0) \geq 0$, $D_c(0) \geq 0$ and $D_d(0) \geq 0$ be the initial conditions of the system ((2) - (9)) then the solutions H_s, T_1, P_p, I_r, P_d, D_w, D_c, D_d remain positive for all time $t > 0$*

Proof. Considering the first model equation (2) given by

$$\begin{aligned} \frac{dH_s}{dt} &= \rho - (\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)H_s + \delta_1 T_1 \\ &\geq -(\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)H_s \end{aligned}$$

So, integrating both sides over the time interval $0 \leq t \leq \infty$ yields

$$\int_0^t \frac{dH_s(t)}{H_s(t)} \geq -\int_0^t (\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)dt \;\; \forall\, t \in [0, \infty)$$

It then follows that

$$H_s(t) = H_s(0)\exp^{-(\sigma_1+\sigma_2+\sigma_3+\theta_1(1-u_1)+\mu)t} \geq 0 \;\; \forall\, t > 0 \tag{13}$$

Considering the second model equation (3) given by

$$\begin{aligned} \frac{dT_1}{dt} &= \sigma_1 H_s - (\delta_1 + \sigma_4 + \mu)T_1(t) \\ &\geq -(\delta_1 + \sigma_4 + \mu)T_1(t) \end{aligned}$$

So, integrating both sides over the time interval $0 \leq t \leq \infty$, we have

$$\int_0^t \frac{dT_1(t)}{T_1(t)} \geq -\int_0^t (\delta_1 + \sigma_4 + \mu)dt$$

It then follows that

$$T_1(t) \geq T_1(0)\exp^{-(\delta_1+\sigma_4+\mu)t} \geq 0 \quad for\ all\ t > 0 \tag{14}$$

The derived positivity equations for the model equations are stated below for all $t > 0$:

$$\begin{cases} P_p(t) \geq P_p(0)\exp^{-(\sigma_5+\mu)t} \geq 0; & P_p(0) > 0, \\ I_r(t) \geq I_r(0)\exp^{-(\sigma_6+\theta_3+\mu)t} \geq 0 & I_r(0) > 0, \\ P_d(t) \geq P_d(0)\exp^{-(\theta_2(1-u_1)+\delta_2+\sigma_7+\mu)t} \geq 0 & P_d(0) > 0, \\ D_w(t) \geq D_w(0)\exp^{-(\mu+\delta_3+\sigma_8(1-u_2)+\theta_4)t} \geq 0 & D_w(0) > 0, \\ D_c(t) \geq D_c(0)\exp^{-(\sigma_9(1-u_2)+\mu+\delta_4+\alpha)t} \geq 0 & D_c(0) > 0, \\ D_d(t) \geq D_d(0)\exp^{-(\mu+\zeta)t} \geq 0 & D_d(0) > 0. \end{cases}$$

This completes the proof.
Therefore, the solution $H_s(0) > 0$, $T_1 > 0$, $P_p > 0$, $I_r > 0$, $P_d > 0$, $D_w > 0$, $D_c > 0$, $D_d > 0$ is non-negative for all time $t > 0$.

The optimal control problem is formulated to obtain the minimum number of diabetic populations D_c and D_d under minimum cost. The terms $A_1{u_1}^2$ represents the cost of preventive measures taken, $A_2{u_2}^2$ represents the cost of the curative measures with A_1 and A_2 being the positive balancing coefficients (weights) that regularize the optimal control. The coefficients, ω_1, ω_2 represents the cost measures on the Diabetes with complications and Diabetes with disabilities respectively. Quadratic expressions of the controls are included to indicate nonlinear costs potentially arising at high intervention levels. Therefore the objective function in (16) is minimized subject to the model equations. We seek the optimal controls u_1^* and u_2^* such that.

$$J(u_1^*, u_2^*) = min\ J\{(u_1, u_2)|u_i \in U\ for\ i = 1, 2\} \tag{15}$$

The problem is then to minimize the objective functional defined as:

$$\min_{u\in U} J(u) = \min_{u\in U} \int_0^T \left\{\omega_1 D_c(t) + \omega_2 D_d(t) + \frac{1}{2}[A_1 u_1^2(t) + A_2 u_2^2(t)]\right\} dt \tag{16}$$

subject to the system of equations. □

2.3.3 *Existence Of Solutions*

Theorem 2.3. *A controlled system that satisfies a given initial condition* $H_s(0) \geq 0$, $T_1(0) \geq 0$, $P_p(0) \geq 0$, $I_r(0) \geq 0$, $P_d(0) \geq 0$, $D_w(0) \geq 0$, $D_c(0) \geq 0$, $D_d(0) \geq 0$ *has a unique solution.*

Proof. The dynamical equations can be expressed as follows:

$$\begin{bmatrix} H_s^*(t) \\ T_1^*(t) \\ P_p^*(t) \\ I_r^*(t) \\ P_d^*(t) \\ D_w^*(t) \\ D_c^*(t) \\ D_d^*(t) \end{bmatrix} = \begin{bmatrix} A_{11} & \delta_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ \sigma_1 & A_{22} & 0 & 0 & 0 & 0 & 0 & 0 \\ \sigma_2 & 0 & A_{33} & 0 & 0 & 0 & 0 & 0 \\ \sigma_3 & 0 & 0 & A_{44} & \delta_2 & 0 & 0 & 0 \\ \theta_1(1-u_1) & \sigma_4 & \sigma_5 & \sigma_6 & A_{55} & \delta_3(u_3) & 0 & 0 \\ 0 & 0 & 0 & \theta_3 & \sigma_7 & A_{66} & \delta_4 & 0 \\ 0 & 0 & 0 & 0 & \theta_2(1-u_1) & \sigma_8(1-u_2) & A_{77} & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta_4 & \sigma_9(1-u_2) & A_{88} \end{bmatrix} \begin{bmatrix} H_s(t) \\ T_1(t) \\ P_p(t) \\ I_r(t) \\ P_d(t) \\ D_w(t) \\ D_c(t) \\ D_d(t) \end{bmatrix} , + \begin{bmatrix} \rho \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

where $A_{11} = -(\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)$, $A_{22} = -(\delta_1 + \sigma_4 + \mu)$, $A_{33} = -(\sigma_5 + \mu)$, $A_{44} = -(\sigma_6 + \theta_3 + \mu)$, $A_{55} = -(\theta_2(1-u_1) + \delta_2 + \sigma_7 + \mu)$, $A_{66} = -(\mu + \delta_3 + \sigma_8(1-u_2) + \theta_4)$, $A_{77} = -(\sigma_9(1-u_2) + \mu + \delta_4 + \alpha)$ and $A_{88} = -(\mu + \zeta)$; and can be compactly written as a linear system below;

$$\varphi(X) = AX + B, \tag{17}$$

$$\begin{aligned} \varphi(X_1) &= AX_1 + B \\ \varphi(X_2) &= AX_2 + B \\ ||\varphi(X_1) - \varphi(X_2)|| &= |A|.||X_1 - X_2|| \\ &\leq K.||X_1 - X_2|| \end{aligned}$$

where $K = |A| < \infty$ and its positive definite.
Hence

$$\|\varphi(X_1) - \varphi(X_2)\| \leq K.\|X_1 - X_2\| \tag{18}$$

It then follows that the control function φ is Lipschitz continuous function. It can be concluded that a solution for the system of equation (2) to (9) exists and its unique. □

2.3.4 *Existence of an Optimal Control*

Theorem 2.4. *There exists an optimal control* $u^* \in U$ *such that*

$$J(u^*) = \min_{u \in U} J(u)$$

Proof. To prove the existence of the optimal control, we applied the result from Fleming and Rishel (1975) [11] by verifying the following conditions:

1. **Convexity and Closure of the Control Set:** The control set $U = \{u : 0 \leq u \leq 1, t \in [0, T]\}$ is convex and closed by definition. Convexity ensures that any weighted combination of controls is still a valid control, and closure implies that the limit of any sequence of controls remains within the set.

2. **Boundedness and Continuity of the System:** The right-hand side of equation (16) is bounded and continuous, as it is a sum of bounded controls and state variables. This can be written as a linear function of u with coefficients depending on time and state, ensuring that the system remains well-defined.

3. **Convexity of the Objective Functional:** The integrand of the objective functional,

$$\omega_1 D_c(t) + \omega_2 D_d(t) + \frac{1}{2}\left[A_1 u_1^2(t) + A_2 u_2^2(t)\right]$$

is convex in U. Convexity ensures that any local minimum is also a global minimum.

4. **Coercivity of the Objective Functional:** There exist constants $\gamma_1, \gamma_2 > 0$ and $\gamma > 1$ such that:

$$\omega_1 D_c(t) + \omega_2 D_d(t) + \frac{1}{2}\left[A_1 u_1^2(t) + A_2 u_2^2(t)\right] > \gamma_1 + \gamma_2 |u|^\gamma.$$

   This inequality shows that the objective functional grows at least quadratically with respect to the control variables, ensuring coercivity.

5. **Boundedness of State Variables:** The state variables are assumed to be bounded, which guarantees the stability and well-posedness of the optimal control problem.

Hence, we conclude that there exists an optimal control u^* that minimizes the objective functional $J(u)$ within the closed and convex control set U. Therefore, the existence of an optimal control is established. □

2.3.5 *Characteristics of the Optimal Control*

In order to derive the necessary conditions for the optimal control, we apply Pontryagin's [18] maximum (minimum) principle to the Hamiltonian H.

Theorem 2.5. *Given an optimal control u^* and solutions H_s^*, T_1^*, P_p^*, I_r^*, P_d^*, D_w^*, D_c^* and D_d^* of the corresponding equations (2 - 9), there exist adjoint variables λ_i for $i = 1, 2, \cdots 8$ satisfying*

$$\begin{aligned}
\dot{\lambda_1} &= (\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)\lambda_1 - \sigma_1\lambda_2 - \sigma_2\lambda_3 - \sigma_3\lambda_4 - \theta_1(1-u_1)\lambda_5 \\
\dot{\lambda_2} &= -\delta_1\lambda_1 + (\delta_1 + \sigma_4 + \mu)\lambda_2 - \sigma_4\lambda_5 \\
\dot{\lambda_3} &= (\sigma_5 + \mu)\lambda_3 - \sigma_5\lambda_5 \\
\dot{\lambda_4} &= (\sigma_6 + \theta_3 + \mu)\lambda_4 - \sigma_6\lambda_5 - \theta_3\lambda_6 \\
\dot{\lambda_5} &= -\delta_2\lambda_4 + (\theta_2(1-u_1) + \mu + \delta_2 + \sigma_7)\lambda_5 - \sigma_7\lambda_6 - \theta_2(1-u_1)\lambda_7 \\
\dot{\lambda_6} &= -\delta_3\lambda_5 + (\delta_3 + \sigma_8(1-u_2) + \theta_4 + \mu)\lambda_6 - \sigma_8(1-u_2)\lambda_7 - \theta_4\lambda_8 \\
\dot{\lambda_7} &= -\omega_1 - \delta_4\lambda_6 + (\sigma_9(1-u_2) + \delta_4 + \mu + \alpha)\lambda_7 - \sigma_9(1-u_2)\lambda_8 \\
\dot{\lambda_8} &= -\omega_2 + (\mu + \zeta)\lambda_8
\end{aligned}$$

with transversality conditions $\lambda_i(T) = 0$ for $i = 1, 2 \cdots, 8$ and the control variables (u_1^*, u_2^*, u_3^*) satisfy the following optimality conditions:

$$\begin{aligned}
u_1^* &= \min\left\{\max\left\{0, \frac{(\lambda_5 - \lambda_1)\theta_1 H_s + (\lambda_7 - \lambda_5)\theta_2 P_d}{A_1}\right\}, 1\right\} \\
u_2^* &= \min\left\{\max\left\{0, \frac{(\lambda_7 - \lambda_6)\sigma_8 D_w + (\lambda_8 - \lambda_7)\sigma_9 D_c}{A_2}\right\}, 1\right\}
\end{aligned}$$

Proof. The Hamiltonian is defined as

$$\begin{aligned}
H \quad = \quad & \omega_1 D_c + \omega_2 D_d + \frac{1}{2}(A_1 u_1^2 + A_2 u_2^2) \\
& + \lambda_1\left(\rho - (\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)H_s + \delta_1 T_1 + \delta_2 P_d\right) \\
& + \lambda_2\left(\sigma_1 H_s - (\delta_1 + \sigma_4 + \mu)T_1\right) \\
& + \lambda_3\left(\sigma_2 H_s - (\sigma_5 + \mu)P_p\right) \\
& + \lambda_4\left(\sigma_3 H_s - (\sigma_6 + \theta_3 + \mu)I_r\right) \\
& + \lambda_5\left(\theta_1(1-u_1)H_s + \sigma_6 I_r + \sigma_4 T_1 + \sigma_5 P_p - (\theta_2(1-u_1) + \delta_2 + \sigma_7 + \mu)P_d + \delta_3 D_w\right) \\
& + \lambda_6\left(\theta_3 I_r + \sigma_7 P_d - (\mu + \delta_3 + \sigma_8(1-u_2) + \theta_4)D_w + \delta_4 D_c\right) \\
& + \lambda_7\left(\theta_2(1-u_1)P_d + \sigma_8(1-u_2)D_w - (\sigma_9(1-u_2) + \mu + \delta_4 + \alpha)D_c\right) \\
& + \lambda_8\left(\theta_4 D_w + \sigma_9(1-u_2)D_c - (\mu + \zeta)D_d\right)
\end{aligned} \tag{19}$$

The adjoint equations can be easily computed by

$$\begin{cases}
\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial H_s}, \quad \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial T_1}, \quad \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial P_p}, \quad \frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial I_r}, \\
\frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial P_d}, \quad \frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial D_w}, \quad \frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial D_c}, \quad \frac{d\lambda_8}{dt} = -\frac{\partial H}{\partial D_d}
\end{cases} \tag{20}$$

The adjoint system eveluated at optimal controls u_1^* and u_2^* and the corresponding model state variables H_s, T_1, P_p, I_r, P_d, D_w, D_c, D_d is given by

- $\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial H_s} = (\sigma_1 + \sigma_2 + \sigma_3 + \theta_1(1-u_1) + \mu)\lambda_1 - \sigma_1\lambda_2 - \sigma_2\lambda_3 - \sigma_3\lambda_4 - \theta_1(1-u_1)\lambda_5$
- $\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial T_1} = -\delta_1\lambda_1 + (\delta_1 + \sigma_4 + \mu)\lambda_2 - \sigma_4\lambda_5$
- $\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial P_p} = (\sigma_5 + \mu)\lambda_3 - \sigma_5\lambda_5$
- $\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial I_r} = (\sigma_6 + \theta_3 + \mu)\lambda_4 - \sigma_6\lambda_5 - \theta_3\lambda_6$
- $\frac{d\lambda_5}{dt} = -\frac{\partial H}{\partial P_d} = -\delta_2\lambda_4 + (\theta_2(1-u_1) + \mu + \delta_2 + \sigma_7)\lambda_5 - \sigma_7\lambda_6 - \theta_2(1-u_1)\lambda_7$
- $\frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial D_w} = -\delta_3\lambda_5 + (\delta_3 + \sigma_8(1-u_2) + \theta_4 + \mu)\lambda_6 - \sigma_8(1-u_2)\lambda_7 - \theta_4\lambda_8$
- $\frac{d\lambda_7}{dt} = -\frac{\partial H}{\partial D_c} = -\omega_1 - \delta_4\lambda_6 + (\sigma_9(1-u_2) + \delta_4 + \mu + \alpha)\lambda_7 - \sigma_9(1-u_2)\lambda_8$
- $\frac{d\lambda_6}{dt} = -\frac{\partial H}{\partial D_d} = -\omega_2 + (\mu + \zeta)\lambda_8$

with transversality conditions (or final time conditions): $\lambda_i(T) = 0$ for $i = 1, 2, \cdots, 8$. The characterizations of the optimal controls, $u_1^*(t)$ and $u_2^*(t)$ are based on the conditions

$$\frac{\partial H}{\partial u_1} = 0, \quad \frac{\partial H}{\partial u_2} = 0 \tag{21}$$

respectively, subject to the conditions given the lebesque measurable control set $\xi = \left\{u_1, u_2\ 0 \le u_i \le 1, \text{for } i = 1, 2 \text{ and } \forall\, t \in [0, T]\right\}$, and the computations of the control variables u_1 and u_2 are measurable functions are summarily given below by

$$u_1 = \frac{(\lambda_5 - \lambda_1)\theta_1 H_s + (\lambda_7 - \lambda_5)\theta_2 P_d}{A_1}, \; u_2 = \frac{(\lambda_7 - \lambda_6)\sigma_8 D_w + (\lambda_8 - \lambda_7)\sigma_9 D_c}{A_2}$$

Subjecting the control variables to the bounds yield

$$u_1^* = \min\left\{\max\left\{0, \frac{(\lambda_5 - \lambda_1)\theta_1 H_s + (\lambda_7 - \lambda_5)\theta_2 P_d}{A_1}\right\}, 1\right\},$$
$$u_2^* = \min\left\{\max\left\{0, \frac{(\lambda_7 - \lambda_6)\sigma_8 D_w + (\lambda_8 - \lambda_7)\sigma_9 D_c}{A_2}\right\}, 1\right\}$$

and this completes the proof. □

3 *Simulation of Results and Discussion*

Numerical simulations to study how different control strategies affect the dynamics of diabetes was conducted. These simulations are performed using MATLAB, and the time is set in years. The initial values for the model state variables are set as follows: $H_s(0) = 1000$, $T_1(0) = 30$, $P_p(0) = 10$, $I_r(0) = 70$, $P_d(0) = 15$, $D_w(0) = 5$, $D_c(0) = 5$, $D_d(0) = 2$.
For the adjoint system, the terminal conditions are set to zero for all variables, with $t = 5$ years. The cost coefficients for the state variables are: $\omega_1 = 0.01$ and $\omega_2 = 0.1$. The quadratic cost coefficients for the control measures are: $A_1 = 1$ and $A_2 = 2$. Parameter values from literature and our own estimates were used, as shown in Table 3 below. Graphs illustrating the effects of the control measures under different combinations was the plotted. There are three control strategies with different combinations of measures. The outcomes of having different control measures with scenarios where no interventions are applied was compared. This helps to show how effective these strategies can be in reducing the spread of diabetes. The findings provide valuable insights into the best ways to control the disease and help understand how to manage and reduce its impact. The collated and assumed parameters are tabulated below;

Table 3: Parameter and values used for numerical simulation

Parameter	Values	Source
ρ	100	Estimated
σ_1	0.05	[16]
σ_2	0.03	[2]
σ_3	0.04	[9]
σ_4	0.02	Estimated
σ_5	0.02	Estimated
σ_6	0.03	[24]
σ_7	0.1	Estimated
σ_8	0.07	[4]
σ_9	0.05	Estimated
δ_1	0.05	[19]
δ_2	0.1	[10]
δ_3	0.07	Estimated
δ_4	0.06	[10]
θ_1	0.2	[20]
θ_2	0.1	[12]
θ_3	0.15	Estimated
θ_4	0.05	[5]
μ	0.005	[21]
α	0.05	[15]
ζ	0.03	[15]

To investigate the optimal control strategies in reducing the spread of diabetes among the given control strategies, the non-delay model will be simulated for each of the strategies as stated below.

i. *Strategy 1*
(S1): Control with prevention of diabetes ($u_1 \neq 0$, $u_2 = 0$**)**

ii. *Strategy 2*
(S2): Control with Treatment of diabetes ($u_1 = 0$, $u_2 \neq 0$**)**

iii. *Strategy 3*
(S3): Control with prevention and treatment of diabetes ($u_1 \neq 0$, $u_2 \neq 0$**)**

3.1 *Strategy 1*

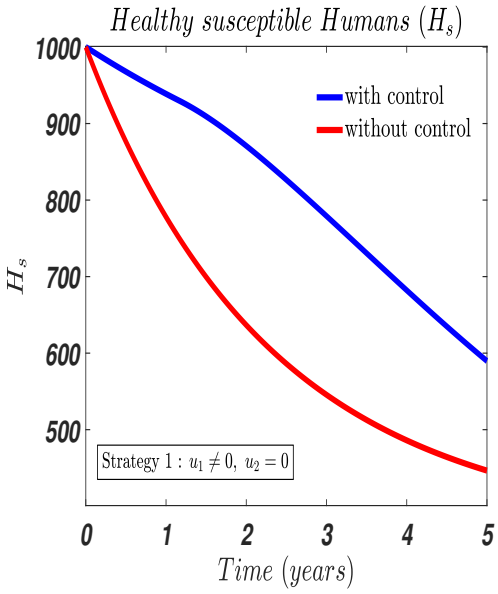

Figure 2: Susceptible Humans Population

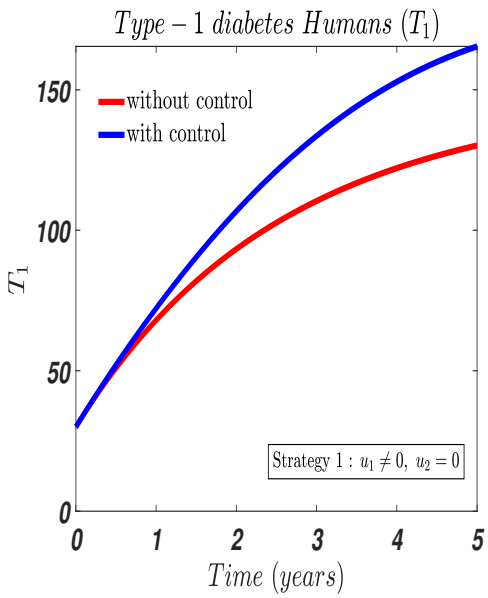

Figure 3: Human with Type-1 Diabetes Population

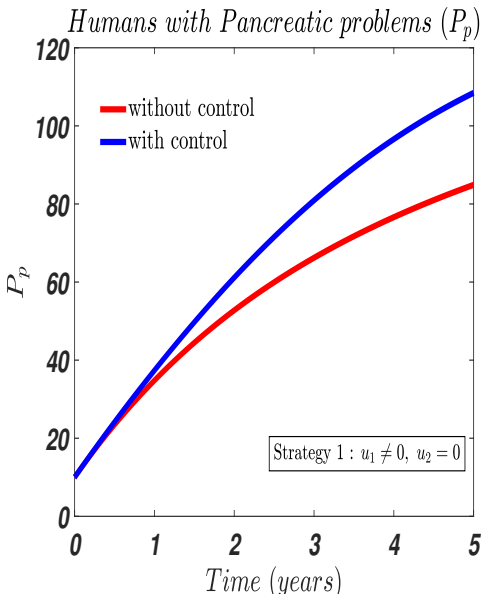

Figure 4: Humans with Pancreatic Problems

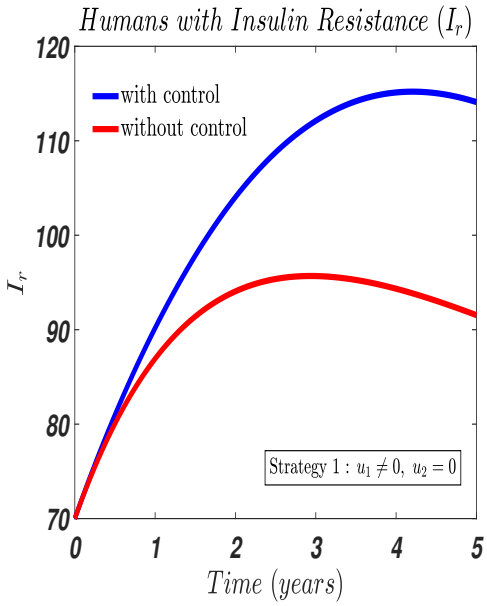

Figure 5: Humans with Insulin Resistance

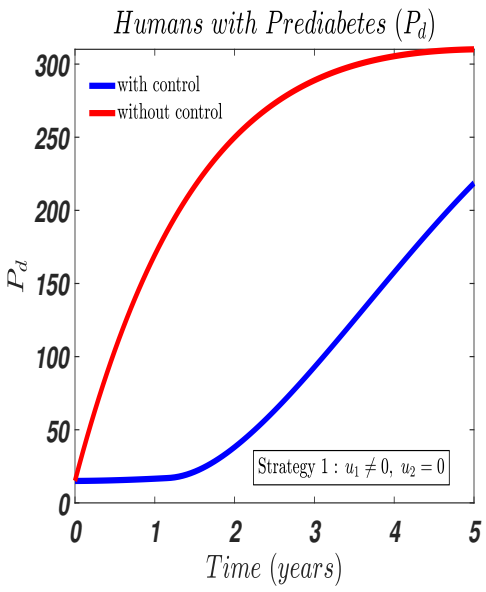

Figure 6: Humans with Prediabetes Population

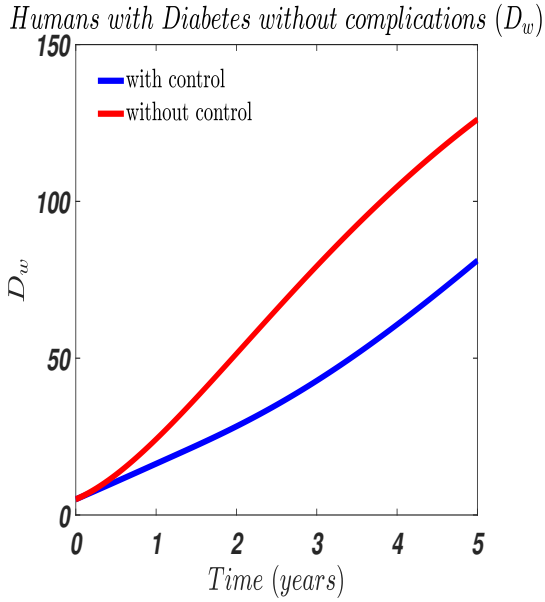

Figure 7: Humans with Diabetes without complications Population

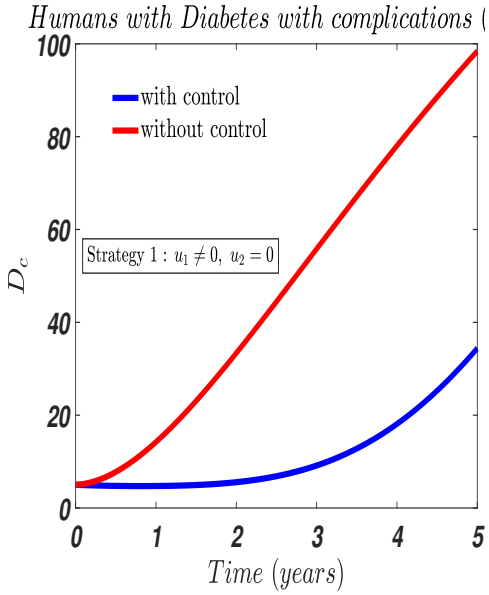

Figure 8: Humans with Diabetes with complications Population

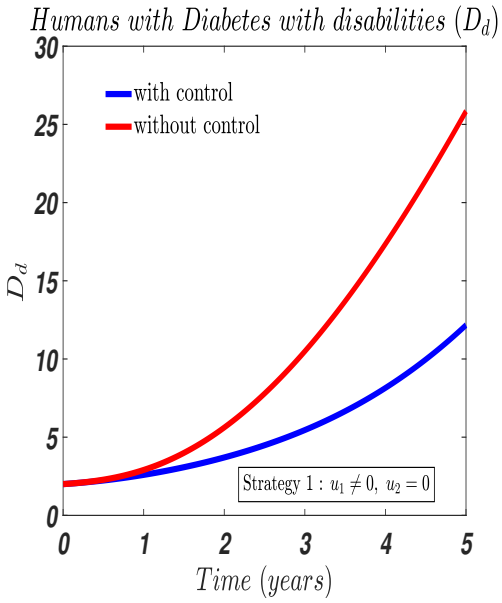

Figure 9: Humans with Diabetes with Disabilities Population

Table 4: Evolution of number of diabetics with strategy 1 after 5 years.

	Initial	Without Control	With Strategy 1	% Difference
H_s	1000	457.4	589.1	28.8
T_1	30	132	165.3	25.2
P_p	10	86.08	108.4	25.8
I_r	70	92.71	114	23.0
P_d	15	301.5	219.1	-27.3
D_w	5	123.7	81.34	-34.2
D_c	5	95.48	34.58	-63.8
D_d	2	25.25	12.2	-51.8

In this strategy, the optimal control u_1 is being activated. In Figure 2 to 9 using Table 4, the number of susceptible population is critical in identifying potential diabetes cases was observed. Initially, there are 1000 healthy individuals, and the figures indicate various diabetic states emerging from this group. A positive increase of 28.8% in maintaining a larger healthy population, highlighting its success in preventive measures. The population with Pancreatic problems and Type 1 diabetes increases from 10 and 30 respectively. Strategy 1 shows a 25.2% and 25.8% increase in both class, showing limited impact on Type-1 diabetes and pancreatic complications, as these are not directly influenced by preventive controls, which is expected since Pancreatic issues and Type 1 diabetes are not typically influenced by lifestyle factors targeted in this strategy. The population with insulin resistance shows a 23.0% increase, Strategy 1 shows moderate effectiveness in reducing insulin resistance with Strategy, indicating that the control measures help to some extent in managing insulin resistance, which is crucial for preventing Type 2 diabetes. Decrease in prediabetic (-27.3%) and uncomplicated diabetes populations (-34.2%) illustrates some success in managing these stages, slowing progression. in the advanced Stages (Diabetes with Complications and Disabilities), with reductions of -63.8% and -51.8%, Strategy 1 significantly lowers the populations experiencing severe complications and disabilities due to diabetes. This confirms the strategy's strength in delaying or even preventing later-stage complications in individuals at risk. In conclusion, strategy 1 is highly effective in maintaining a healthier population, particularly in slowing progression from early diabetes stages to more severe complications. However, its impact on Type-1 diabetes and pancreatic complications remains limited.

3.2 *Strategy 2*

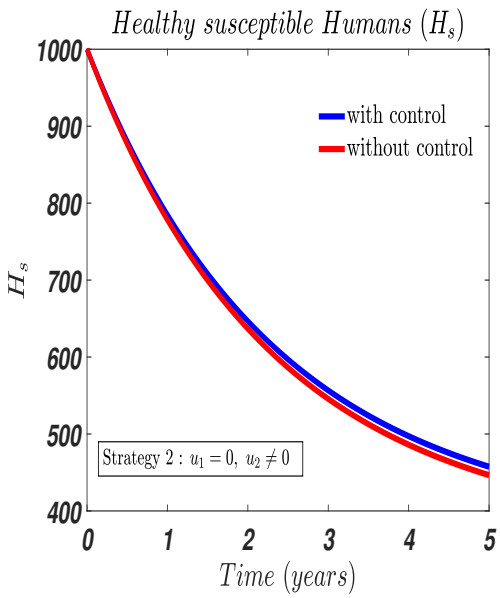

Figure 10: Susceptible Humans Population

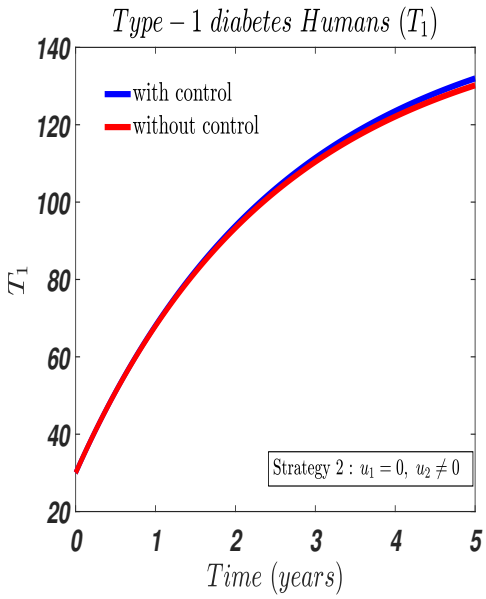

Figure 11: Human with Type-1 Diabetes Population

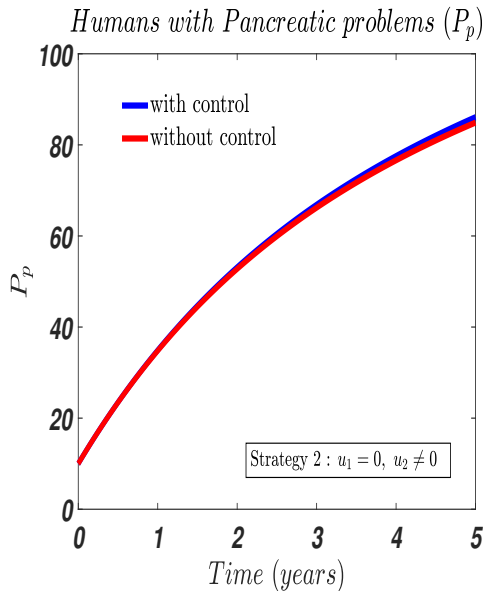

Figure 12: Humans with Pancreatic Problems

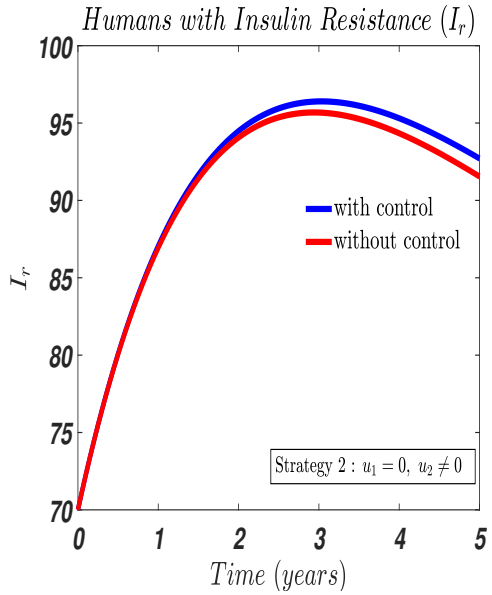

Figure 13: Humans with Insulin Resistance

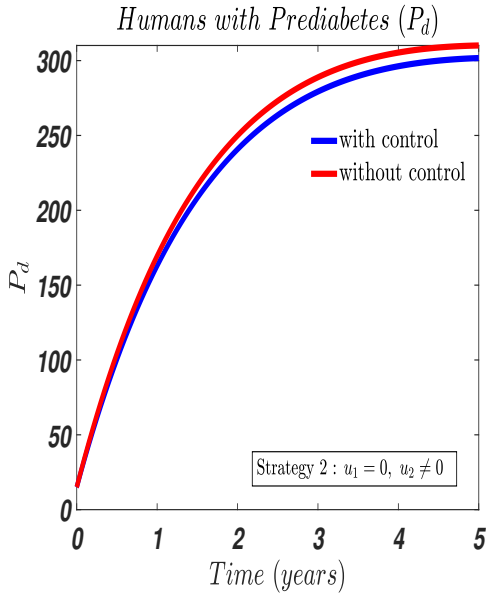

Figure 14: Humans with Prediabetes Population

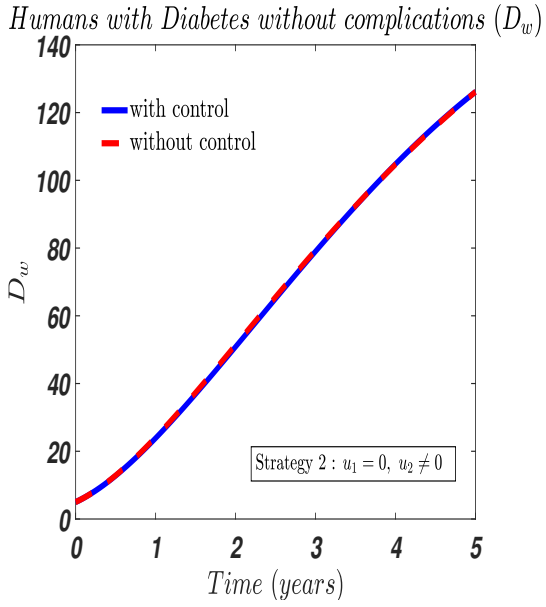

Figure 15: Humans with Diabetes without complications Population

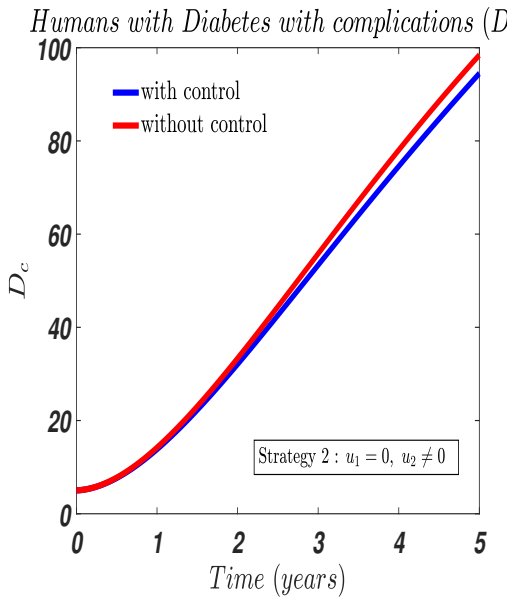

Figure 16: Humans with Diabetes with complications Population

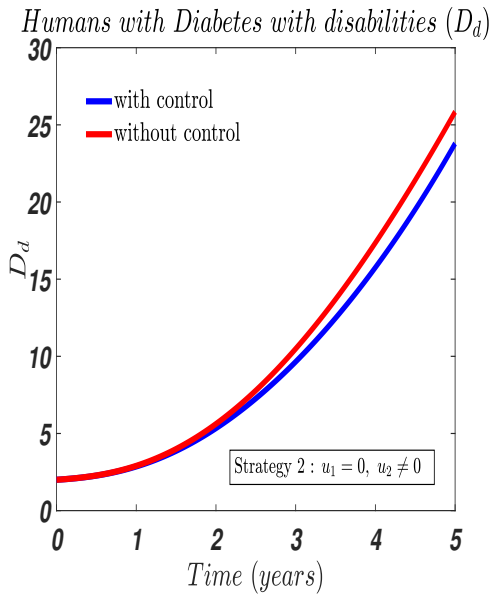

Figure 17: Humans with Diabetes with Disabilities Population

Table 5: Evolution of number of diabetics with strategy 2 after 5 years.

	Initial	Without Control	With Strategy 2	% Difference
H_s	1000	457.4	457.4	0.00
T_1	30	132	132	0.00
P_p	10	86.08	86.08	0.00
I_r	70	92.71	92.71	0.00
P_d	15	301.5	301.5	0.00
D_w	5	123.7	126.1	1.9
D_c	5	95.48	94.47	-1.1
D_d	2	25.25	23.8	-5.8

In this strategy, the optimal controls u_2 was activated. Figure 10 shows population remains constant at 457.4 with or without Strategy 2. With no change observed, Strategy 2 lacks a preventive effect on the healthy population. Its focus is on controlling progression within the diabetic population, thus it doesn't reduce the number of new cases. In figure 11, the population with Type 1 diabetes also remains unchanged at 132, reflecting that this strategy does not influence Type 1 diabetes cases, with no change in the population over five years. In figure 12, the number of individuals with pancreatic problems shows no change (86.08), suggesting Strategy 2 has no effect on this risk factor. In figure 13, Insulin resistance is a key factor in the development of Type 2 diabetes. numbers remain constant (92.71). This shows that Strategy 2 does not impact the population at risk of developing insulin resistance, a precursor for Type 2 diabetes. From figure 14, The prediabetes population stays the same (301.5) with or without the strategy, indicating no effect in preventing progression to diabetes. Figure 15, the population with diabetes but without complications shows a slight increase in diabetes without complications (1.9%). This suggests that the strategy isn't as effective in managing early-stage diabetes, showing mixed results in preventing further progression. In advanced Stages (Diabetes with Complications and Disabilities) figure 16 and figure 17 shows that the strategy's impact is minor, with only slight reductions in diabetes with complications (-1.1%) and disabilities (-5.8%). This indicates a limited ability to reduce the most severe diabetes outcomes. In conclusion, Strategy 2 appears to have no significant effect on the diabetic populations.

3.3 *Strategy 3*

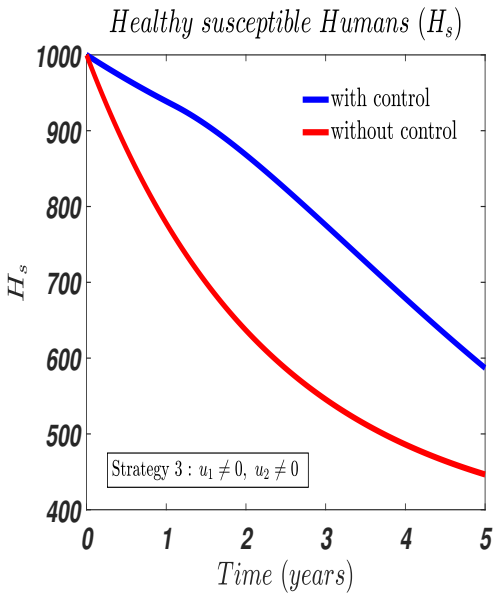

Figure 18: Susceptible Humans Population

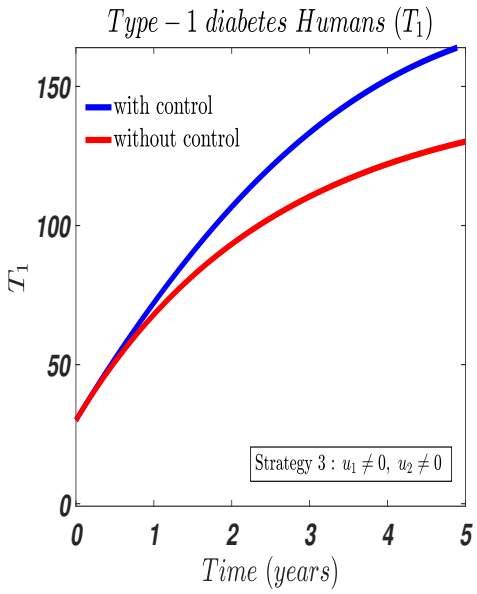

Figure 19: Human with Type-1 Diabetes Population

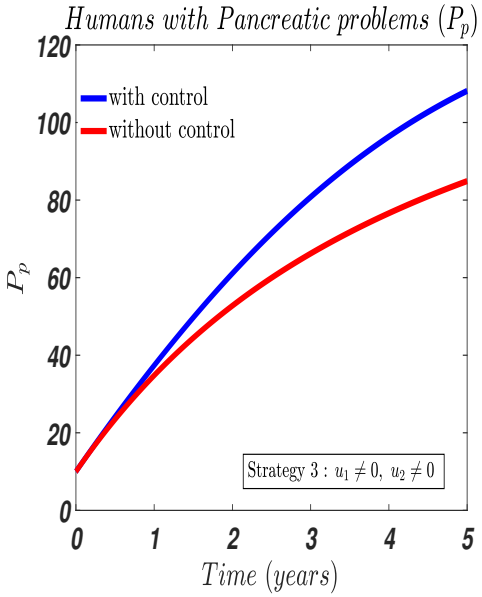

Figure 20: Humans with Pancreatic Problems

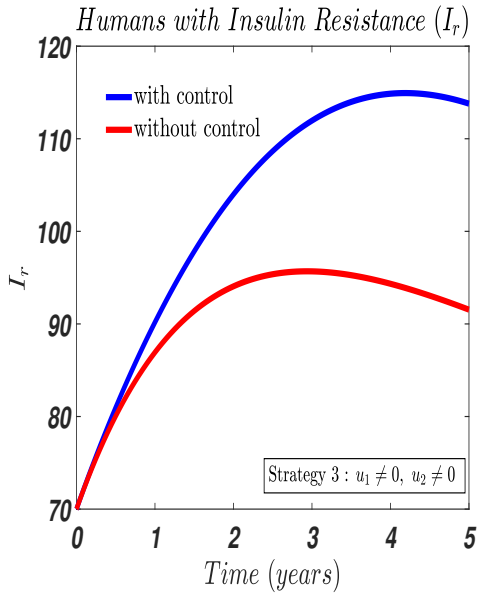

Figure 21: Humans with Insulin Resistance

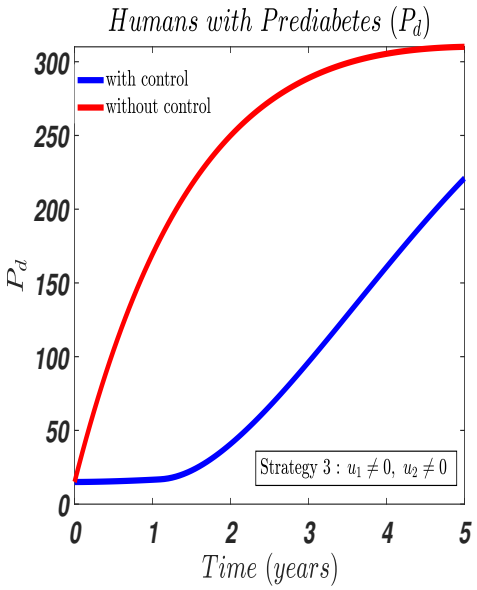

Figure 22: Humans with Prediabetes Population

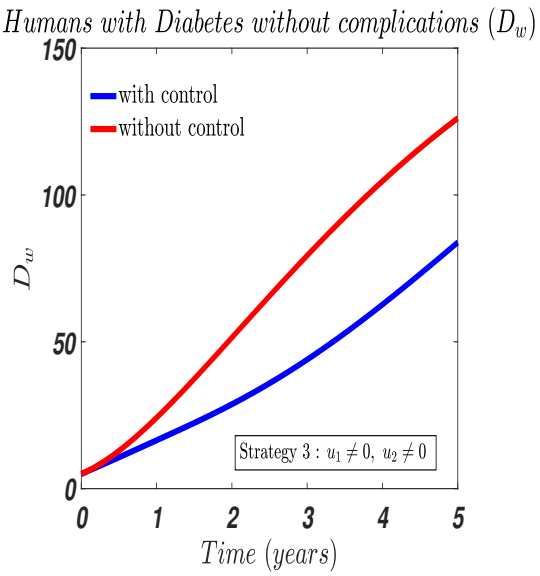

Figure 23: Humans with Diabetes without complications Population

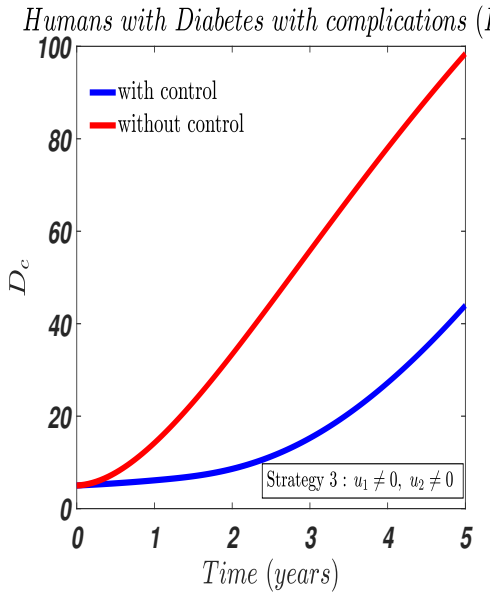

Figure 24: Humans with Diabetes with complications Population

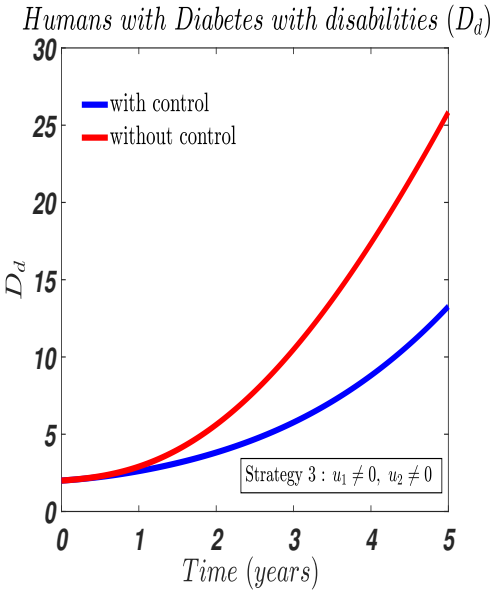

Figure 25: Humans with Diabetes with Disabilities Population

Table 6: Evolution of number of diabetics with strategy 3 after 5 years.

	Initial	Without Control	With Strategy 3	% Difference
H_s	1000	457.4	586.4	28.2
T_1	30	132	164.8	24.84
P_p	10	86.08	108.1	25.58
I_r	70	92.71	113.7	22.6
P_d	15	301.5	221.5	-26.5
D_w	5	123.7	83.47	-32.5
D_c	5	95.48	44.15	-53.8
D_d	2	25.25	13.27	-47.6

Strategy 3 incorporates both prevention and treatment controls (u_1 and u_2) providing a comprehensive approach aimed at both halting disease onset and controlling progression. The figures illustrate the changes in various diabetes-related populations, while the table provides data on how these populations evolve over five years with and without the implementation of Strategy 3. Figures (18 to 25) and the table 6 are interpreted to evaluate the effectiveness of Strategy 3 in combating the diabetes epidemic. Figure 18 shows the susceptible population with a 28.2% increase, this approach effectively prevents new diabetes cases. The strong increase indicates that Strategy 3's combined controls help sustain a larger non-diabetic population over time. Figure 19 and figure 20 indicates that Type-1 diabetes cases increased by 24.84%, and pancreatic problems by 25.58%. These increases show that even with a combined approach, Strategy 3 has limited impact on managing Type-1 diabetes (which is largely genetic) and pancreatic problems. These categories remain challenging to address solely through preventive and treatment-focused strategies. In figure 21, with a 22.6% increase, indicates that despite the combined strategy, managing insulin resistance remains challenging. This increase suggests a need for more targeted interventions to address this precursor to Type-2 diabetes. In figure 22 and figure 23, strategy 3 shows success in reducing the prediabetes population by -26.5% and those with diabetes without complications by -32.5%. This reduction reflects the strategy's effectiveness in managing early-stage diabetes and preventing progression. In dvanced Stages (Diabetes with Complications figure 24 and Disabilities figure 25), there are significant reductions of -53.8% in diabetes with complications and -47.6% in disabilities, showing that Strategy 3 is highly effective in managing later stages of diabetes and reducing severe health impacts. In conclusion, strategy 3 is the most comprehensive and effective among the three strategies. It reduces the overall diabetic population, minimizes complications, and provides an optimal approach for managing diabetes progression.

3.4 *Control profiles*

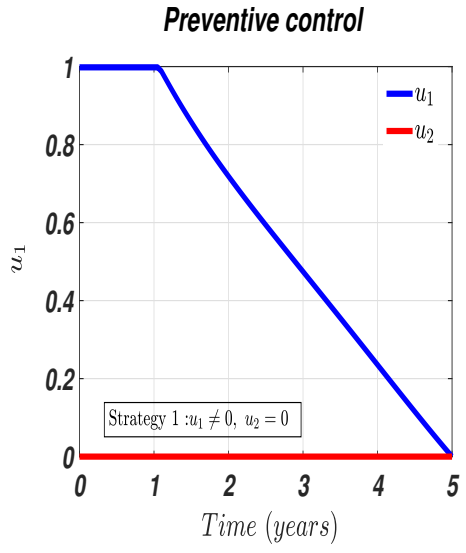

Figure 26: Prevention control profile (u_1)

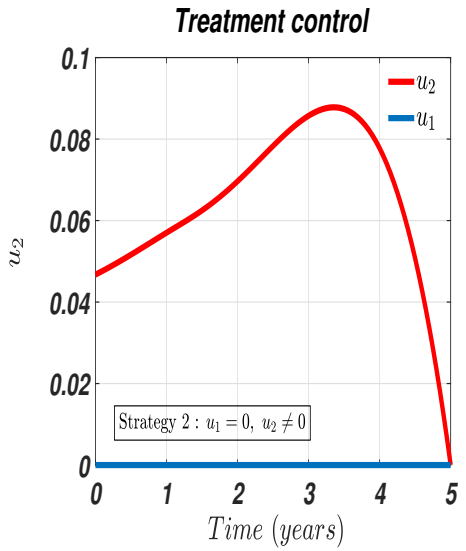

Figure 27: Treatment control profile (u_2)

The **Prevention control** (u_1) function as shown in figure 26 shows a steady decline from its maximum value of 1 at the start to 0 over a 5-year period. The high initial value indicates that the model emphasizes prevention strongly at the beginning. This corresponds to an aggressive campaign focused on lifestyle changes, public health awareness, or early screening for diabetes. This aligns with the strategies used to reduce risk factors in a population, such as dietary interventions, exercise programs, or public health messaging to prevent the onset of diabetes, particularly Type 2. Also, the steady decrease suggests a reduction in the emphasis on prevention over time. As prevention efforts take effect, there might be fewer new cases due to lifestyle changes and early intervention, reducing the need for intensive preventive action. This decrease also represent resource reallocation, where efforts shift away from prevention as initial high-risk groups stabilize and fewer individuals are at immediate risk. By the end of the period, prevention efforts may not be as necessary or are expected to be maintained by the population independently, hence

reaching zero in the model.
Treatment Control (u_2) function as shown in figure 27 follows a different pattern, starting low, rising to a peak at around 2.5 years, and then decreasing to zero by the end of the period. This trend suggests a targeted approach to treatment based on anticipated disease progression within the population. The initial low level of u_2 suggests that treatment efforts are minimal initially, likely because prevention is prioritized and fewer individuals require medical intervention at the beginning. As time progresses, treatment control gradually ramps up, in response to individuals who have developed diabetes or prediabetes despite preventive measures. By the midpoint (2.5 years), the treatment control reaches its peak, which could indicate the point at which the prevalence of diabetes is highest in the modeled population. After the peak, the treatment control gradually declines. This decrease implies that effective treatment is reducing the number of active cases needing intensive medical intervention. Alternatively, it also indicates that those initially diagnosed have stabilized through ongoing treatment, and new cases are fewer due to the earlier prevention efforts. The declining need for treatment also reflect a successful transition to disease management, where patients no longer require intensive control as they manage their condition through lifestyle adjustments and lower-level care. By the end of the 5-year period, treatment control reaches zero, potentially suggesting a stabilized population where diabetes management no longer requires significant active treatment input, or the intervention period is complete.

The combination of these control profiles reflect a dynamic approach to diabetes intervention, where prevention is front-loaded and treatment peaks as the effects of prevention stabilize the population. The interplay between u_1 and u_2 is used to assess the effectiveness of early prevention on reducing the need for later treatment, a critical factor in long-term diabetes management strategies.
By modeling these controls, this work illustrates optimal allocation of healthcare resources over time maximizing prevention initially to reduce the long-term treatment burden. This approach could serve as a guide for public health policymakers looking to minimize the economic and societal impact of diabetes through phased intervention strategies.

4 *Conclusion*

This study analyzed the effectiveness of three different strategies for managing and controlling the spread of diabetes over a five-year period. By examining various populations, including susceptible humans, those with Type-1 diabetes, pancreatic problems, insulin resistance, prediabetes, diabetes without complications, diabetes with complications, and diabetes with disabilities, the impact of each strategy was assessed. Strategy 1 emphasizing preventive controls, effectively sustains a healthier population and reduces progression from early to advanced diabetic stages, though it has limited impact on Type-1 diabetes and pancreatic issues. Strategy 2 focused on managing the diabetic population with a different approach. It demonstrated limited impact in maintaining the healthy susceptible population and showed minimal improvements in the populations. Strategy 3 aimed at controlling the spread and management of diabetes with another distinct approach. It combines both prevention and treatment controls, proves to be the most comprehensive approach, achieving reductions across all diabetic stages and significantly minimizing the most severe complications. However, an increase in insulin resistance under Strategy 3 suggests a need for enhanced focus on early-stage management.

In summary, Strategy 3 offers the most balanced and effective approach for overall diabetes management, combining preventive and treatment measures to control both new cases and disease progression effectively. It addresses multiple stages of diabetes, reducing the population at risk while minimizing the transition to severe complications and disabilities.

5 *Conflict of Interest*

Conflict of interest is not relevant to the context of this article. (NOT APPLICABLE).

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