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Research Article

Impact of lifestyle interventions and adequate medical infrastructure on diabetes prevention: Dynamics analysis and optimal control

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Abstract

Diabetes mellitus is a chronic metabolic disorder that affects the ability to regulate blood glucose levels in the human body. In recent decades, the rapid increase in the number of people living with diabetes around the world has made it one of the most pressing public health concerns and has placed a growing burden on healthcare systems. The onset of diabetes is driven by a combination of genetic predisposition and lifestyle factors, including poor diet, physical inactivity, and obesity, emphasizing the importance of early prevention and effects lifestyle interventions. In this study, we formulate and analyze a nonlinear mathematical model to

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study the effect of lifestyle intervention and proper medical infrastructure on diabetes prevention. The model incorporates the interactions between five state variables related to diabetes mellitus, namely, healthy population, pre-diabetic population due to unhealthy lifestyle, pre-diabetic population due to genetic factors, diabetic population without complications, and diabetic population with complications. We study the basic properties of the model system, such as existence, nonnegativity, and boundedness of solutions. Furthermore, a nontrivial equilibrium point for the model system is identified, and local and global stability of the equilibrium points are established. Additionally, we propose an optimal control problem by introducing two control variables that prevent healthy people from becoming pre-diabetic due to poor lifestyle through awareness campaigns promoting healthy lifestyle and clinical interventions to improve treatment rates for the diabetic population with complications. The optimal control problem is further solved to reduce the burden of diabetes with and without complications. Finally, our findings suggest that lifestyle interventions aimed at raising awareness among the healthy population about unhealthy lifestyles, together with adequate medical infrastructure to improve treatment rates among diabetic individuals with complications, can substantially ease the growing strain imposed by diabetes on healthcare systems.

AMS subject classifications (2020): Primary 37M05, 34D23, 37N25; Secondary 92B05, 49K15.

Keywords: Diabetes; Awareness; Lifestyle intervention; Medical infrastructure; Simulation; Optimal control.

1 Introduction

Diabetes mellitus, popularly known as diabetes, is a chronic and progressive disease that has become a major challenge to the world's healthcare system [23]. It has become one of the leading causes of mortality and morbidity around the world, contributing to the increasing cost of the healthcare system worldwide [14]. Diabetes-related health costs were projected to reach USD 1,028 billion by 2030, up from an anticipated USD 966 billion in 2021 [14]. Diabetes can arise due to a variety of factors, including genetic predisposition and irregular lifestyle habits such as an unhealthy diet and physical inactivity. Another common cause is the inability of the pancreas to produce sufficient insulin, a hormone that enables glucose from food to enter the body's cells and be used for energy [36]. Type 1 diabetes occurs when the immune system repeatedly attacks and damages the pancreatic cells responsible for regulating blood glucose levels, resulting in a build-up of glucose in the bloodstream, whereas type 2 diabetes occurs when the body may not produce sufficient insulin or cannot effectively use the insulin it produces, resulting in higher than normal blood glucose levels. Pre-diabetes is a health condition when the blood sugar level

is elevated but not high enough to be considered diabetes [36]. Individuals with pre-diabetes are more likely to acquire type 2 diabetes if the condition is not treated. Early diagnosis is crucial as untreated pre-diabetes can progress to type 2 diabetes, which in turn can lead to serious health complications such as heart disease and even death. Blood tests are available to detect diabetes and pre-diabetes, facilitating timely intervention and management.

In 2021, a study on global health found that 529 million people around the world were living with diabetes [30], indicating that approximately 6.1% of the population had diabetes. The highest rates were in the Middle East and North Africa (9.3%), and in Oceania (12.3%). Qatar had the highest rate for people aged $75-79$, at 76.1%. Total diabetes prevalence, particularly among older adults, predominantly reflects type 2 diabetes, which in 2021 accounted for 96.0% of all diabetes cases and 95.4% of diabetes-related DALYs worldwide. In the year 2021, more than half of the health problems from type 2 diabetes were linked to high body mass index (BMI), which has increased significantly since 1990. By 2051, it is expected that over 1.31 billion people will have diabetes, with rates above 10% in regions like the Middle East, North Africa (16.8%), and Latin America (11.3%). Out of 204 countries, 89 are projected to have diabetes rates over 10%.

As per the report of the World Health Organization (WHO), the incidence of diabetes is not stable but is rising each year [37]. This increase is largely attributed to changes in lifestyle among the global population. Moreover, social interaction is an important factor influencing lifestyle, and it plays a significant role in lifestyle changes that may increase an individual's risk of developing diabetes. Furthermore, genetic factors are responsible for the incidence of diabetes, particularly in individuals with a family history of the disease [15]. Therefore, the rise in diabetes cases is linked to both genetic factors and unhealthy lifestyles, including an unhealthy level of inactivity, poor dietary choices, and other destructive routines. Currently, countries around the world are facing a significant increase in the number of people with diabetes. If not properly treated, all forms of diabetes can result in severe complications. When individuals with diabetes learn to effectively control their blood glucose levels, this awareness is crucial in reducing the risk of severe complications. Another well-established method to mitigate the long-term effects of diabetes by controlling blood glucose levels is to perform [13]. Recent studies show that lifestyle intervention is an effective method for diabetes remission [12, 18]. Diabetes commonly presents with symptoms such as excessive thirst, frequent urination, heightened hunger, exhaustion, weight loss, and blurred vision. Some complications of diabetes are heart disease, kidney failure, nerve damage, coma and eventually, death. On the other hand, diabetes without complications made it difficult to lead a healthy lifestyle, especially when it remains undiagnosed.

In recent years, mathematical modeling has been widely used to study the dynamics of various diseases and extensive research has been carried out by many researchers to better un-

derstand their transmission, progression, and control [28, 29, 1, 2, 38, 4, 27]. In recent decades, numerous mathematicians have proposed and studied different mathematical models related to population dynamics [5, 33, 8, 16, 25, 11, 21, 22, 20, 9, 7, 31, 32, 35] and glucose-insulin dynamics [24, 19, 17, 3] of diabetes. In the majority of diabetes studies, researchers have primarily concentrated on continuous and discrete time models, which are governed by ordinary differential equations. Also, mathematical analyses such as stability around the equilibrium points, the formulation and solution of optimal control problems to find different optimal strategies are performed in these studies, like other disease-related works [6, 7, 28, 29]. A basic mathematical model to illustrate the dynamics of a population affected by diabetes, which was proposed by Boutayeb et al. [5] in 2016, can be expressed by the following system of differential equations:

$$\begin{aligned}
\frac{dP}{dt} &= n - (I_1 + I_2 + I_3 + \mu)P + \gamma_1 E, \\
\frac{dE}{dt} &= I_1 P - (\gamma_1 + \beta_1 + \beta_3 + \mu)E, \\
\frac{dD}{dt} &= I_2 P + \beta_1 E + \gamma_2 C - (\beta_2 + \mu)D, \\
\frac{dC}{dt} &= I_3 P + \beta_2 D + \beta_3 E - (\gamma_2 + \mu + \delta)C,
\end{aligned}$$

where P, E, D, and C represent healthy, pre-diabetic, diabetic with and without complications, respectively. The parameters n, I_1, I_2, I_3, μ, β_1, β_2, β_3, γ_1, γ_2, δ recruitment rate of adult population, rate of healthy people become pre-diabetic, rate of healthy people become diabetic, rate of healthy people developing complication, probability of pre-diabetic people become diabetic, probability of diabetic people developing complication, probability of pre-diabetic people developing complication, rate of pre-diabetic people become healthy, rate of complication that are cured, and mortality rate due to complications, respectively. The authors introduced an optimal control approach with an objective to reduce the burden of diabetes. In 2016, Derouich et al. [8] published a paper, where they suggested an optimal control technique modeling the progression from pre-diabetes to diabetes with and without complications and found that utilizing that optimal control, the number of diabetes cases with and without complications may be greatly decreased over 10 years. In 2018, Permatasari, Tjahjana, and Udjiani [33] constructed an optimal control model with diabetes caused disabled population to prevent adults from diabetes by minimizing the number of pre-diabetics at minimum cost. In 2020, Kouidere et al. [16] proposed an optimal control model to find a strategy that can minimize treatable diabetics through awareness campaigns about the complications of diabetes, the bad impacts of an irregular lifestyle and the surrounding environment, along with treatment. Expanding the work by W. Boutayeb et al. [5], A. Kouidere et al. [15] proposed a novel model in 2021 by introducing two

new state variables: pre-diabetic individuals due to genetic factors and those affected by negative socio-environmental influences. They also formulated an optimal control problem to reduce the number of diabetics without complications and the vaccination cost by conducting awareness campaigns about the complications of diabetes, the bad impact of an irregular lifestyle, along with the surrounding environment. In 2022, Nasir [25] developed a five-compartmental model of the diabetes population and also proposed an optimal control problem to minimize the pre-diabetic population with minimum cost. It was found that the total number of people with diabetes can be suppressed effectively by the awareness of a healthy diet and regular lifestyle. In 2021, Gupta and Kumar [11] proposed a mathematical model considering that pre-diabetic individuals went into remission due to lifestyle changes, including a healthy diet and physical exercise.

Using both deterministic and stochastic modeling approaches, Mollah and Biswas [21] assessed the effectiveness of diabetes awareness programs and found that increased population-level awareness significantly lowers the risk of diabetes. Furthermore, Mollah and Biswas [22] proposed a population model for Type 2 diabetes to study the combined effects of awareness programs and treatment. Also, they proposed an optimal control problem and found that both awareness programs and treatment are effective in reducing the prevalence of Type 2 diabetes. Mishra and Kumari [20] first proposed a model incorporating the effect of awareness through word-of-mouth communication only and then extended the model to incorporate the effect of awareness through social media and TV advertisements. Their study suggested that social media and television advertisements are more effective in increasing awareness among individuals and in reducing the risk of diabetes mellitus. In 2025, Pal et al. [31] proposed a mathematical model to study the prevalence of diabetes and kidney infection, and then formulated and solved an optimal control problem with two controls, representing the impacts of awareness at the local and global levels, by enhancing information through media. In another study, Pal et al. [32] proposed a mathematical model to study the effects of awareness campaigns on the prevention of diabetes and viral infections. Then, they proposed an optimal control problem considering the same controls as in [31]. Their findings indicate that interventions delivered through social media and television advertisements are significantly more effective in enhancing awareness and reducing the propagation of diabetes risk compared to reliance on word-of-mouth communication alone.

From the above studies, it has been established that awareness among the population plays a crucial role in the management of diabetes. Nowadays, the widespread availability of mobile phones and internet-based platforms such as YouTube has made health-related information accessible to a large segment of the population. Even illiterate individuals can acquire knowledge through audio-visual content, while those without direct access to mobile phones often become informed through interpersonal communication within families and communities, as younger

members typically have access to internet-enabled devices. This indirect diffusion of information ensures that awareness regarding the benefits of lifestyle interventions reaches almost the entire population. In addition, government-led awareness initiatives and public health campaigns further strengthen the dissemination of information related to diabetes prevention. Along with awareness, the availability of adequate and efficient treatment facilities plays a crucial role in diabetes control, as early diagnosis, timely medical intervention, and proper management help prevent disease progression and reduce the severity of diabetes-related complications. Keeping these in mind, we propose a nonlinear model incorporating the effect of lifestyle interventions among the healthy population through awareness, along with the role of proper treatment facilities for timely diagnosis of the complicated diabetic population.

The rest of this article is structured as follows: In Section 2, we present the continuous differential equations model that describes the dynamics of a population of diabetics. In Section 3, we establish some basic properties of the model, including the existence, nonnegativity, and boundedness of solutions. In this section, the stability analysis is conducted around the model's equilibrium point. We discuss the optimal control problem for the proposed model in Section 4, providing findings indicating the existence of the optimal control and characterizing it using Pontryagin's minimum principle. Section 5 contains numerical simulations which were done using MATLAB software. Finally, in Section 6, we summarize the findings of our study.

2 Model formulation

To formulate the model, we first introduce the notation and nomenclature associated with diabetes mellitus. The total population at any given time $t > 0$ is denoted by $N(t)$, and is divided into five subclasses based on health status, genetic predisposition, lifestyle, and medical condition. Individuals who are currently healthy but may develop pre-diabetes or diabetes over time due to genetic factors or unhealthy lifestyle habits are included in the healthy class, denoted by $H(t)$. The pre-diabetic population is further divided into two subclasses: $G(t)$, representing individuals who become pre-diabetic primarily due to genetic factors, and $B(t)$, representing individuals who become pre-diabetic mainly due to unhealthy lifestyle behaviors. Similarly, the diabetic population is divided into two subclasses: $D(t)$, denoting diabetic individuals without complications, and $C(t)$, denoting diabetic individuals with complications. The progression to complications is assumed to occur because of poor glycemic control, delayed diagnosis, or inadequate treatment. Therefore, the total population $N(t)$ at any time t is given by $N(t) = H(t) + G(t) + B(t) + D(t) + C(t)$. The formulation of our model is based on the following assumptions:

- Let n be the constant growth rate of the healthy adult population, who may later become pre-diabetic or diabetic [5]. We assume that technological advancement has increased public awareness regarding the role of lifestyle interventions in the prevention of diabetes.

- We assume that healthy adults may progress to a pre-diabetic state either due to genetic predisposition or unhealthy lifestyle behaviors, and may eventually develop diabetes if timely preventive measures are not taken [16]. For the model formulation, we assume that healthy individuals enter the pre-diabetic stage due to genetic factors at the rate v and due to an unhealthy lifestyle at the rate λ. Furthermore, γ_1 and γ_2 denote the rates at which pre-diabetic individuals caused by genetic factors progress to diabetes without complications and with complications, respectively. Similarly, δ represents the rate at which pre-diabetes caused by an unhealthy lifestyle progresses to diabetes due to the continued effects of irregular and unhealthy habits.

- We assume that a fraction of healthy individuals may progress directly to diabetes, with or without complications, without passing through the pre-diabetic stage [16]. This may occur due to factors such as late diagnosis, genetic susceptibility, lack of regular glucose monitoring, and other related causes. In the model, the parameters α_1 and α_2 represent the rates at which healthy individuals become diabetic without complications and with complications, respectively.

- We assume that individuals in the pre-diabetic class caused by an unhealthy lifestyle may return to the healthy class through lifestyle modification, regular physical activity, and improved dietary habits. It is also assumed that diabetic individuals without complications may achieve remission and return to the healthy class through early diagnosis, sustained lifestyle intervention, and appropriate medical treatment [11]. In the model, r_1 and r_2 represent the remission rates of the pre-diabetic class due to unhealthy lifestyle and the diabetic class without complications, respectively.

- In the absence of adequate treatment or regular monitoring, diabetic individuals without complications are assumed to progress to diabetes-related complications at the rate η.

- We assume that diabetic individuals with complications may return to the diabetic class without complications through effective treatment, whose success depends on the availability and quality of medical infrastructure, such as timely access to specialists, diagnostic facilities, and continuous care. Therefore, a saturated treatment function $\frac{\kappa C}{1+lC}$ [39, 26] is used to represent the recovery rate from complications. Here, κ denotes the

treatment rate, while l is a nonnegative parameter representing limitations in medical resources. Thus, when healthcare resources are limited, the treatment rate initially increases with the number of diabetic individuals with complications, but eventually approaches a maximum saturation level as healthcare capacity becomes fully utilized. Furthermore, β represents the mortality rate due to diabetes-related complications.

The above assumptions form the basis for the differential equation model, which captures the dynamics of healthy, pre-diabetic and diabetic populations with the impact of lifestyle intervention through awareness and the adequate medical facilities. The flowchart in Figure 1 illustrates the interactions between different populations of the model. Hence, the governing system of differential equations for the model is derived as follows:

$$\begin{aligned}
\frac{dH(t)}{dt} &= n - (\mu + \alpha_1 + \alpha_2 + v + \lambda)H(t) + r_1 B(t) + r_2 D(t),\\
\frac{dG(t)}{dt} &= vH(t) - (\gamma_1 + \gamma_2 + \mu)G(t),\\
\frac{dB(t)}{dt} &= \lambda H(t) - (\delta + \mu + r_1)B(t),\\
\frac{dD(t)}{dt} &= \alpha_1 H(t) + \gamma_1 G(t) + \delta B(t) - (\mu + \eta + r_2)D(t) + \frac{\kappa C}{1 + lC},\\
\frac{dC(t)}{dt} &= \alpha_2 H + \gamma_2 G(t) + \eta D(t) - \frac{\kappa C}{1 + lC} - (\mu + \beta)C(t).
\end{aligned} \tag{1}$$

We analyse the system with the initial conditions

$$H(0) \geq 0, \quad G(0) \geq 0, \quad B(0) \geq 0, \quad D(0) \geq 0, \quad C(0) \geq 0. \tag{2}$$

Moreover, all the parameters used in the model (1) are considered to be positive, and their

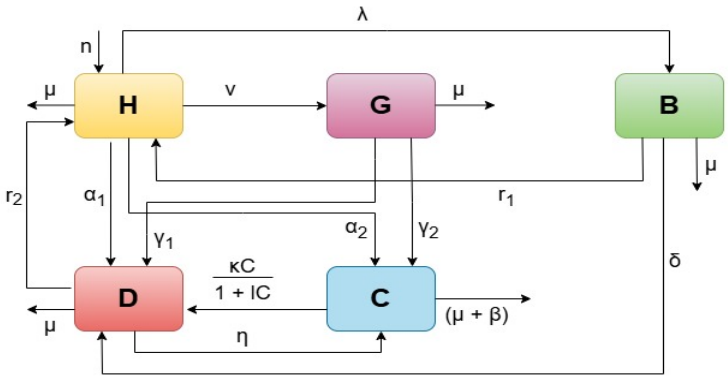

Figure 1: The schematic diagram for the model (1).

detailed descriptions and values are given in Table 1.

Table 1: Meanings of parameters for the model (1) along with their values.

Parameter(s)	Description	Units	Values
n	Constant growth rate of Healthy population	person day^{-1}	600000
μ	Natural death rate of healthy population	day^{-1}	0.02
α_1	Rate at which the healthy population becomes diabetic without complications	day^{-1}	0.3
α_2	Rate at which the healthy population becomes diabetic with complications.	day^{-1}	0.1
v	Rate at which the healthy population enters the pre-diabetic stage due to genetic factors.	day^{-1}	0.1
λ	Rate at which the healthy population enters the pre-diabetic stage due to a bad lifestyle	day^{-1}	0.2
γ_1	Rate at which the pre-diabetic population due to genetic factors become diabetic patients without complications	day^{-1}	0.2
γ_2	Rate at which the pre-diabetic population due to genetic factors become diabetic patients with complications	day^{-1}	0.1
δ	Rate at which the pre-diabetic population, due to a bad lifestyle, becomes diabetic patients without complications	day^{-1}	0.8
η	Rate at which the diabetic population without complication propagates to the complicated stage	day^{-1}	0.5
β	Represents the mortality rate of the diabetic population with complications.	day^{-1}	0.05
r_1	Rate at which the pre-diabetic population, due to a bad lifestyle, moves to a healthy population	day^{-1}	0.08
r_2	Rate at which the diabetic population without complications enters into a healthy category	day^{-1}	0.08
κ	The treatment rate	day^{-1}	0.65
l	The limitations of medical resources	person^{-1}	0.0003

3 Mathematical analysis of the model (1)

3.1 Basic properties of the model (1)

In this part, we demonstrate many fundamental aspects of the model (1), such as existence, nonnegativity, and boundedness of the model solutions. The verification of these basic properties plays a significant role in establishing the biological feasibility of the model (1).

Theorem 1. The dynamical system (1) with the initial condition (2) has a unique solution.

The proof of the theorem is given in *Appendix A*.

Theorem 2. Under the initial conditions (2), the solutions $H(t)$, $G(t)$, $B(t)$, $D(t)$ and $C(t)$ of the system (1) are nonnegative for all $t \geq 0$.

The proof of the theorem is given in *Appendix B*.

Theorem 3. The set

$$\Omega = \Big\{(H(t), G(t), B(t), D(t), C(t)) \in \mathbb{R}^5_+ \\ |H(t) + G(t) + B(t) + D(t) + C(t) \leq \frac{n}{\mu}\Big\},$$

is positively invariant under system (1) with initial conditions (2).

The proof of the theorem is given in *Appendix C*.

3.2 Calculation of equilibrium point

Generally, the solutions of $\frac{dH(t)}{dt} = \frac{dG(t)}{dt} = \frac{dB(t)}{dt} = \frac{dD(t)}{dt} = \frac{dC(t)}{dt} = 0$ are the equilibrium points of the model (1). Let the equilibrium point $T^*(H^*, G^*, B^*, D^*, C^*)$ be a solution of the following system:

$$n - (\mu + \alpha_1 + \alpha_2 + v + \lambda)H^*(t) + r_1 B^*(t) + r_2 D^*(t) = 0, \tag{3}$$

$$vH^*(t) - (\gamma_1 + \gamma_2 + \mu)G^*(t) = 0, \tag{4}$$

$$\lambda H^*(t) - (\delta + \mu + r_1)B^*(t) = 0, \tag{5}$$

$$\alpha_1 H^*(t) + \gamma_1 G^*(t) + \delta B^*(t) - (\mu + \eta + r_2)D^*(t) + \frac{\kappa C^*}{1 + lC^*} = 0, \tag{6}$$

$$\alpha_2 H^* + \gamma_2 G^*(t) + \eta D^*(t) - \frac{\kappa C^*}{1 + lC^*} - (\mu + \beta)C^*(t) = 0. \tag{7}$$

Since the state variable represents the individual population, only a positive solution is the biologically feasible equilibrium point. From (3), (4) and (5) we have

$$G^* = \frac{vH^*}{\gamma_1 + \gamma_2 + \mu}, \tag{8}$$

$$B^* = \frac{\lambda H^*}{\delta + \mu + r_1}, \tag{9}$$

$$D^* = \frac{-n}{r_2} + \frac{(\mu + \alpha_1 + \alpha_2 + v + \lambda)H^*}{r_2} - \frac{r_1 \lambda H^*}{r_2(\delta + \mu + r_1)}. \tag{10}$$

By substituting the expression of G^*, B^*, and D^* in (6) we obtain

$$(\mu + \beta)l(C^*)^2 - (Xl - \kappa - (\mu + \beta))C^* - X = 0, \tag{11}$$

where

$$\begin{aligned} X =& \alpha_2 H^* + \frac{\gamma_2 v}{\gamma_1 + \gamma_2 + \mu}H^* + \frac{\eta(\mu + \alpha_1 + \alpha_2 + v + \lambda)}{r_2}H^* \\ &- \frac{\lambda r_1}{r_2(\delta + \mu + r_1)}H^* - \frac{\eta n}{r_2}. \end{aligned}$$

The roots of the equation are as follows:

$$C^* = \frac{(Xl - \kappa - \mu - \beta) \pm \sqrt{(Xl - \kappa - \mu - \beta)^2 + 4X(\mu + \beta)l}}{2l(\mu + \beta)}. \tag{12}$$

Since

$$\sqrt{(Xl-\kappa-\mu-\beta)^2+4X(\mu+\beta)l} > \sqrt{(Xl-\kappa-\mu-\beta)^2} = |Xl-\kappa-\mu-\beta|,$$

therefore, irrespective of the sign of the term $(Xl-\kappa-\mu-\beta)$, the root

$$C^* = \frac{(Xl-\kappa-\mu-\beta)+\sqrt{(Xl-\kappa-\mu-\beta)^2+4X(\mu+\beta)l}}{2l(\mu+\beta)},$$

is always positive.

Again, from (6) we obtain

$$H^* = -\frac{n(\mu+\eta+r_2)}{A} - \frac{\kappa C^*}{A(1+lC^*)}, \tag{13}$$

where

$$A = \alpha_1 + \frac{\gamma_1 v}{\gamma_1+\gamma_2+\mu} + \frac{\delta\lambda}{\delta+\mu+r_1} + \frac{\lambda r_1(\mu+\eta+r_2)}{r_2(\delta+\mu+r_1)} - \frac{(\mu+\eta+r_2)(\mu+\alpha_1+\alpha_2+v+\lambda)}{r_2}.$$

If

$$A = \alpha_1 + \frac{\gamma_1 v}{\gamma_1+\gamma_2+\mu} + \frac{\delta\lambda}{\delta+\mu+r_1} + \frac{\lambda r_1(\mu+\eta+r_2)}{r_2(\delta+\mu+r_1)} < \frac{(\mu+\eta+r_2)(\mu+\alpha_1+\alpha_2+v+\lambda)}{r_2},$$

then H^* is positive, along with G^* and B^*. From (10), if

$$\frac{(\mu+\alpha_1+\alpha_2+v+\lambda)H^*}{r_2} > \frac{n}{r_2} + \frac{\lambda r_1 H^*}{r_2(\delta+\mu+r_1)},$$

then D^* is positive.

3.3 Local stability of the equilibrium point T^*

Theorem 4. The equilibrium point T^* is locally asymptotically stable under the following conditions:

$$r_1 + r_2 < \mu + \alpha_1 + \alpha_2 + v + \lambda,$$

$$
\begin{aligned}
&v < \gamma_1 + \gamma_2 + \mu,\\
&\lambda < \delta + \mu + r_1,\\
&\alpha_1 + \gamma_1 + \delta + \frac{\kappa}{(1+lC)^2} < \mu + \eta + r_2,\\
&\alpha_2 + \gamma_2 + \eta < \mu + \beta + \frac{\kappa}{(1+lC)^2}.
\end{aligned}
$$

Proof. The Jacobian matrix around the interior equilibrium point T^* for the model (1) is given by

$$
J(T^*) = \begin{bmatrix}
-(\mu+\alpha_1+\alpha_2+v+\lambda) & 0 & r_1 & r_2 & 0\\
v & -(\gamma_1+\gamma_2+\mu) & 0 & 0 & 0\\
\lambda & 0 & -(\delta+\mu+r_1) & 0 & 0\\
\alpha_1 & \gamma_1 & \delta & -(\mu+\eta+r_2) & \frac{\kappa}{(1+lC)^2}\\
\alpha_2 & \gamma_2 & \eta & -\frac{\kappa}{(1+lC)^2} & -(\mu+\beta)
\end{bmatrix}.
$$

Here, all the diagonal entries of the matrix $J(T^*)$ are negative. Also, it is not always easy to find eigenvalues for a large matrix. Thus, by Gersgorin's theorem, the interior equilibrium T^* is locally asymptotically stable provided the following condition holds:

$$
\begin{aligned}
&r_1 + r_2 < \mu + \alpha_1 + \alpha_2 + v + \lambda,\\
&v < \gamma_1 + \gamma_2 + \mu,\\
&\lambda < \delta + \mu + r_1,\\
&\alpha_1 + \gamma_1 + \delta + \frac{\kappa}{(1+lC)^2} < \mu + \eta + r_2,\\
&\alpha_2 + \gamma_2 + \eta < \mu + \beta + \frac{\kappa}{(1+lC)^2}.
\end{aligned}
$$

□

3.4 Global Stability of the equilibrium point T^*

In this section, we have shown the global stability of the model (1) around the equilibrium point T^* with the help of suitable Lyapunov function. Consider the following Lyapunov positive definite function:

$$
L = \frac{1}{2}(H-H^*)^2 + \frac{1}{2}(G-G^*)^2 + \frac{1}{2}(B-B^*)^2 + \frac{1}{2}(D-D^*)^2 + \frac{1}{2}(C-C^*)^2. \tag{14}
$$

By differentiating L with respect to t, we have

$$\frac{dL}{dt} =(H-H^*)\frac{dH}{dt}+(G-G^*)\frac{dG}{dt}+(B-B^*)\frac{dB}{dt}+(D-D^*)\frac{dD}{dt} \tag{15}$$

$$+(C-C^*)\frac{dC}{dt}. \tag{16}$$

Using (3)–(7), we have

$$\begin{aligned}\frac{dL}{dt} =& -(\mu+\alpha_1+\alpha_2+v+\lambda)(H-H^*)^2-(\gamma_1+\gamma_2+\mu)(G-G^*)^2\\ &-(\delta+\mu+r_1)(B-B^*)^2-(\mu+\eta+r_2)(D-D^*)^2\\ &-\left(\frac{\kappa}{(1+lC^*)^2}+\mu+\beta\right)(C-C^*)^2\\ &+(r_1+\lambda)(B-B^*)(H-H^*)\\ &+(r_2+\alpha_1)(D-D^*)(H-H^*)+v(G-G^*)(H-H^*)\\ &+\gamma_1(D-D^*)(B-B^*)+\alpha_2(C-C^*)(H-H^*)\\ &+\gamma_2(C-C^*)(G-G^*)+\left(\frac{\kappa}{(1+lC^*)^2}+\eta\right)(D-D^*)(C-C^*)\\ &+\frac{\kappa C^*}{1+lC^*}[(D-D^*)-(C-C^*)].\end{aligned}$$

From the above equation, using Young's inequality, we have $\frac{dL}{dt}$ is negative definite provided the following conditions hold:

$$(\delta+\mu+r_1)(\mu+\alpha_1+\alpha_2+v+\lambda)>\left(\frac{r_1+\lambda}{2}\right)^2, \tag{17}$$

$$(\mu+\eta+r_2)(\mu+\alpha_1+\alpha_2+v+\lambda)>\left(\frac{r_2+\lambda}{2}\right)^2, \tag{18}$$

$$(\gamma_1+\gamma_2+\mu)(\mu+\alpha_1+\alpha_2+v+\lambda)>\frac{v^2}{4}, \tag{19}$$

$$(\mu+\eta+r_2)(\gamma_1+\gamma_2+\mu)>\frac{\gamma^2}{4}, \tag{20}$$

$$(\mu+\eta+r_2)(\delta+\mu+r_1)>\frac{\delta^2}{4}, \tag{21}$$

$$(\mu+\eta+r_2)\left(\frac{\kappa}{(1+lC^*)^2}+\mu+\beta\right)>\frac{\left(\frac{\kappa}{(1+lC^*)^2}+\eta\right)^2}{4}, \tag{22}$$

$$\left(\frac{\kappa}{(1+lC^*)^2}+\mu+\beta\right)(\mu+\alpha_1+\alpha_2+v+\lambda)>\frac{\alpha_2^2}{4}, \tag{23}$$

$$\left(\frac{\kappa}{(1+lC^*)^2}+\mu+\beta\right)(\gamma_1+\gamma_2+\mu)>\frac{\gamma_2^2}{4}, \tag{24}$$

$$(C - C^*) > (D - D^*). \tag{25}$$

So, by Lyapunov's Direct method, we can conclude that our model (1) shows global stability around the interior equilibrium point T^* under the above conditions. Thus, we have the following theorem.

Theorem 5. The equilibrium point T^* is globally asymptotically stable under the conditions (17)–(25)

4 Optimal control problem

The optimal control problem aims to identify time-dependent intervention strategies that minimize the diabetic population and related complications at the population level, subject to the underlying disease transmission dynamics and control costs. In general, an optimal control problem for a controlled model consists of three main parts: an objective function, a set of state variables and controls. To formulate the optimal control problem associated with system (1), we introduce two time-dependent controls $u_1(t)$ and $u_2(t)$, representing the lifestyle intervention due to awareness about healthy lifestyle and the proper medical infrastructure, respectively. The detailed description and impact of these controls are given below:

1. **Lifestyle intervention control:** The control $u_1(t)$ represents the intensity of the behavioral modification as a result of awareness to maintain a proactive and healthy lifestyle, which includes performing regular physical activities, maintaining a healthy weight, and following a balanced diet, where $0 \leq u_1(t) \leq 1$. An increase in $u_1(t)$ reduces the rate at which individuals transition from the healthy class to pre-diabetic and diabetic classes by discouraging unhealthy lifestyle choices. Moreover, the control $u_1(t)$ enhances behavioral change among individuals in the pre-diabetic class due to bad lifestyle, thereby increasing the remission rate from this class back to the healthy population as $u_1(t)$ increases. Similarly, higher levels of $u_1(t)$ improve self-management and lifestyle adherence among diabetic individuals without complications, leading to an increased remission rate from the diabetic without complications class to the healthy class.

2. **Enhancing medical infrastructure:** The control $u_2(t)$ represents the intensity of clinical interventions aimed at improving the treatment outcomes of diabetic individuals with complications, where $0 \leq u_2(t) \leq 1$. This control captures the effect of expanding and strengthening medical resources, including improved healthcare infrastructure, availability of specialized medical personnel, access to diagnostic facilities, and timely thera-

peutic management. An increase in $u_2(t)$ enhances the treatment rate of individuals in the diabetic-with-complications compartment, thereby facilitating their transition to the diabetic-without-complications class and reducing the overall burden of severe diabetes related outcomes in the population.

Now, the controlled model based on (1) is stated below:

$$\begin{aligned}
\frac{dH(t)}{dt} =& n - (\mu + \alpha_2 + v)H(t) - \alpha_1(1 - u_1(t))H(t) \\
& - \lambda(1 - u_1(t))H(t) + r_1 u_1(t)B(t) + r_2 u_1(t)D(t), \\
\frac{dG(t)}{dt} =& vH(t) - (\gamma_1 + \gamma_2 + \mu)G(t), \\
\frac{dB(t)}{dt} =& \lambda(1 - u_1(t))H(t) - \delta B(t) - (\mu + r_1 u_1(t))B(t), \\
\frac{dD(t)}{dt} =& \alpha_1(1 - u_1(t))H(t) + \gamma_1 G(t) + \delta B(t) + \frac{\kappa u_2(t)C(t)}{1 + lC(t)} \\
& - \eta D(t) - (\mu + r_2 u_1(t))D(t), \\
\frac{dC(t)}{dt} =& \alpha_2 H(t) + \gamma_2 G(t) - \frac{\kappa u_2(t)C(t)}{1 + lC(t)} - (\mu + \beta)C(t) + \eta D(t),
\end{aligned} \tag{26}$$

Moreover, the objective functional is

$$\mathcal{J}(B, u) = \int_0^T \{C(t) + D(t) + \frac{1}{2}A_1 u_1^2(t) + \frac{1}{2}A_2 u_2^2(t)\}dt, \tag{27}$$

which has to be minimized. Here, the positive constants A_1 and A_2, referred to as weighting constants, are related to costs of following a healthy diet, engaging in regular physical activities, organizing awareness programs, expanding medical resources, etc. It will also be kept in mind that these costs should also be minimized. Hence, our main objective is to find an optimal control set (u_1^*, u_2^*) such that

$$\mathcal{J}(u_1^*, u_2^*) = \min_{(u_1^*, u_2^*) \in \Gamma} J(u_1(t), u_2(t)), \tag{28}$$

subject to the state constraints (26) and initial conditions (2). The control set Γ is defined as follows:

$$\Gamma = \{(u_1, u_2) | u_i\text{'s are measurable}, 0 \leq u_i \leq 1, \text{ for all } i = 1, 2 \text{ and } t \in [0, t_f]\}. \tag{29}$$

4.1 Existence of the optimal control problem

Now, the first target is to show the existence of an optimal control set (u_1^*, u_2^*) that can minimize the objective function (27). In order to establish the existence of optimal control, methodologies from several previous research works [10, 31, 32, 28, 29] were applied, resulting in the following results.

Theorem 6. For the control model (26) with the controls (u_1, u_2), there exists an optimal control set (u_1^*, u_2^*) such that

$$\mathcal{J}(u_1^*, u_2^*) = \min_{(u_1^*, u_2^*) \in \Gamma} J(u_1(t), u_2(t)).$$

Proof. It can be easily shown that the set of state variables under the controls (u_1, u_2) in (26) is bounded, which confirms the existence of solutions. Hence, the set Γ is nonempty. From the definition, the control space Γ is convex and closed. Consider the integrand of the objective functional as

$$\mathcal{L} = C(t) + D(t) + \frac{1}{2}A_1 u_1^2(t) + \frac{1}{2}A_2 u_2^2(t). \tag{30}$$

The Hessian matrix for the integrand $\mathcal{L}$ is given by

$$H_{\mathcal{L}} = \begin{bmatrix} \mathcal{L}_{u_1,u_1} & \mathcal{L}_{u_1,u_2} \\ \mathcal{L}_{u_2,u_1} & \mathcal{L}_{u_2,u_2} \end{bmatrix} = \begin{bmatrix} A_1 & 0 \\ 0 & A_2 \end{bmatrix}. \tag{31}$$

Clearly, the Hessian matrix $H_{\mathcal{L}}$ is positive definite, which indicates that the integrand function $\mathcal{L}$ is convex for all $u_i \in \Gamma$, $i = 1, 2$. Since the state variables $D(t)$ and $C(t)$ are bounded, so by order completeness property, they have supremum, let $\xi_1 = \sup_{t\in[0,t_f]} D(t)$ and $\xi_2 = \sup_{t\in[0,t_f]} C(t)$, then we have

$$C(t) + D(t) + A_1 u_1^2(t) + A_2 u_2^2(t) \geq \xi_3 |u_1|^\alpha + \xi_4 |u_2|^\alpha - \xi_1 - \xi_2,$$

choosing $\xi_3 = A_1$, $\xi_4 = A_2$, and $\alpha = 2$. So, the integrand $\mathcal{L}$ is bounded below.

From Fleming and Rishel [10], thus, the existence of the optimal control set (u_1, u_2) can be concluded, and for this, the objective functional $\mathcal{J}(u_1^*, u_2^*)$ is minimized. □

4.2 Characterization of the optimal control

Now, we apply Pontryagin's minimum principle [34] to find the necessary conditions for the optimal control problem. For this, the Hamiltonian $\mathcal{H}$ related to state system (26) is considered as below:

$$\mathcal{H} = C(t) + D(t) + \frac{1}{2}A_1u_1^2(t) + \frac{1}{2}A_2u_2^2(t) + \sum_{i=1}^{5}\theta_i f_i(H, G, B, D, C), \tag{32}$$

where

$$\begin{aligned}
f_1(H,G,B,D,C) &= n - (\mu+\alpha_2+v)H(t) - \alpha_1(1-u_1(t))H(t) - \lambda(1-u_1(t))H(t)\\
&\quad + r_1u_1(t)B(t) + r_2u_1(t)D(t),\\
f_2(H,G,B,D,C) &= vH(t) - (\gamma_1+\gamma_2+\mu)G(t),\\
f_3(H,G,B,D,C) &= \lambda(1-u_1(t))H(t) - \delta B(t) - (\mu + r_1u_1(t))B(t),\\
f_4(H,G,B,D,C) &= \alpha_1(1-u_1(t))H(t) + \gamma_1G(t) + \delta B(t) + \frac{\kappa u_2(t)C(t)}{1+lC(t)}\\
&\quad - \eta D(t) - (\mu + r_2u_1(t))D(t),\\
f_5(H,G,B,D,C) &= \alpha_2H(t) + \gamma_2G(t) - \frac{\kappa u_2(t)C(t)}{1+lC(t)} - (\mu+\beta)C(t) + \eta D(t),
\end{aligned}$$

and θ_1, θ_2, θ_3, θ_4, and θ_5 are adjoint functions that need to be determined. Applying Pontryagin's maximum principle [34], we have the following theorem.

Theorem 7. For a given set of optimal controls (u_1^*, u_2^*) with associated optimal solutions H^*, G^*, B^*, D^*, and C^* to the model (26), there exist adjoint variables θ_1, θ_2, θ_3, θ_4, and θ_5 which satisfy the equations

$$\begin{aligned}
\theta_1'(t) &= \theta_1\mu + (\theta_1-\theta_2)v + (1-u_1)(\theta_1-\theta_4)\alpha_1 + (\theta_1-\theta_5)\alpha_2\\
&\quad + (1-u_1)(\theta_1-\theta_3)\lambda,\\
\theta_2'(t) &= \theta_2\mu + (\theta_2-\theta_4)\gamma_1 + (\theta_2-\theta_5)\gamma_2,\\
\theta_3'(t) &= \theta_3\mu + (\theta_3-\theta_1)u_1r_1 + (\theta_3-\theta_4)\delta,\\
\theta_4'(t) &= -1 + \theta_4\mu + (\theta_4-\theta_5)\eta + (\theta_4-\theta_1)u_2r_2,\\
\theta_5'(t) &= -1 + (\theta_5-\theta_4)\frac{\kappa u_2}{(1+lC)^2} + \theta_5(\mu+\beta),
\end{aligned}$$

with the tranversality conditions

$$\theta_1(t_f) = 0, \quad \theta_2(t_f) = 0, \quad \theta_3(t_f) = 0, \quad \theta_4(t_f) = 0, \quad \theta(t_f) = 0. \tag{33}$$

The optimal controls are given by

$$u_1^* = \min\left\{\max\left\{\frac{(\theta_3-\theta_1)(\lambda H + r_1 B) + (\theta_4-\theta_1)(\alpha_1 H + r_2 D)}{A_1}, 0\right\}, 1\right\}, \tag{34}$$

$$u_2^* = \min\left\{\max\left\{\frac{(\theta_5-\theta_4)\kappa C}{A_2(1+lC)}, 0\right\}, 1\right\}. \tag{35}$$

Proof. We can find the adjoint functions using the following equations:

$$\begin{aligned}
\theta_1^{'}(t) &= -\frac{\partial \mathcal{H}}{\partial H} \\
&= \theta_1\mu + (\theta_1-\theta_2)v + (1-u_1)(\theta_1-\theta_4)\alpha_1 \\
&\quad + (\theta_1-\theta_5)\alpha_2 + (1-u_1)(\theta_1-\theta_3)\lambda, \\
\theta_2^{'}(t) &= -\frac{\partial \mathcal{H}}{\partial G} \\
&= \theta_2\mu + (\theta_2-\theta_4)\gamma_1 + (\theta_2-\theta_5)\gamma_2, \\
\theta_3^{'}(t) &= -\frac{\partial \mathcal{H}}{\partial B} \\
&= \theta_3\mu + (\theta_3-\theta_1)u_1 r_1 + (\theta_3-\theta_4)\delta, \\
\theta_4^{'}(t) &= -\frac{\partial \mathcal{H}}{\partial D} \\
&= -1 + \theta_4\mu + (\theta_4-\theta_5)\eta + (\theta_4-\theta_1)u_2 r_2, \\
\theta_5^{'}(t) &= -\frac{\partial \mathcal{H}}{\partial C} \\
&= -1 + (\theta_5-\theta_4)\frac{\kappa u_2}{(1+lC)^2} + \theta_5(\mu+\beta).
\end{aligned}$$

The transversality conditions are

$$\theta_1(t_f) = 0, \;\; \theta_2(t_f) = 0, \;\; \theta_3(t_f) = 0, \;\; \theta_4(t_f) = 0, \;\; \theta(t_f) = 0.$$

Moreover, the optimal controls u_1^* and u_2^* can be calculated using the following equations:

$$\begin{aligned}
\frac{\partial \mathcal{H}}{\partial u_1} &= A_1 u_1 + (\theta_1-\theta_2)(\lambda H + r_1 B) + (\theta_1-\theta_4)(\alpha_1 H + r_2 D) = 0, \\
\frac{\partial \mathcal{H}}{\partial u_2} &= A_2 u_2 + (\theta_4-\theta_5)\frac{\kappa C}{1+lC} = 0.
\end{aligned}$$

which give

$$u_1 = \frac{(\theta_3-\theta_1)(\lambda H + r_1 B) + (\theta_4-\theta_1)(\alpha_1 H + r_2 D)}{A_1},$$

$$u_2 = \frac{(\theta_5 - \theta_4)\kappa C}{A_2(1 + lC)}.$$

Using the bounds of the controls u_1 and u_2, we get the optimal controls as follows:

$$u_1^* = \min\left\{\max\left\{\frac{(\theta_3 - \theta_1)(\lambda H + r_1 B) + (\theta_4 - \theta_1)(\alpha_1 H + r_2 D)}{A_1}, 0\right\}, 1\right\},$$
$$u_2^* = \min\left\{\max\left\{\frac{(\theta_5 - \theta_4)\kappa C}{A_2(1 + lC)}, 0\right\}, 1\right\}.$$

□

5 Numerical simulations

In this section, we perform a series of numerical simulations for the model (1) to validate the analytical results and study the effects of different model parameters on the model's dynamics using MATLAB. To write and compile code in MATLAB, we used the values of the model parameters (1) as given in Table 2. For these parameter values, the components of the equilibrium point T^* are calculated as $H^* = 956600$, $G^* = 298964$, $B^* = 212580$, $D^* = 897372$, and $C^* = 7900630$. For the Jacobian matrix J^* calculated at the equilibrium point T^*, the eigenvalues are obtained as $-0.9002 + 0.1297i$, $-0.9002 - 0.1297i$, 0.0700, -0.4413 and -0.2983, although one real part of eigenvalue is reported as positive, indicating the local asymptotic stability of the equilibrium point T^*. Figures 2 and 3 demonstrate that the solution trajectories of the system (1) start from different initial points in $H-B-C$ and $H-D-C$ space, respectively. All trajectories are observed to converge to the equilibrium point (H^*, B^*, C^*) irrespective of the initial points, indicating the global stability of T^*. Since these parameter values satisfy conditions (17)–(25), it follows that diabetes persists whenever these conditions hold.

Table 2: Values of parameters for the model (1).

Parameters	Values	Source	Parameters	Values	Source
n	600000	[5]	δ	0.8	[15]
μ	0.02	[5]	η	0.5	[5]
α_1	0.3	[5]	β	0.05	[11]
α_2	0.1	[5]	r_1	0.08	[11]
v	0.1	[5]	r_2	0.08	[11]
λ	0.2	[16]	κ	0.65	[26]
γ_1	0.2	[15]	l	0.0003	[26]
γ_2	0.1	[15]			

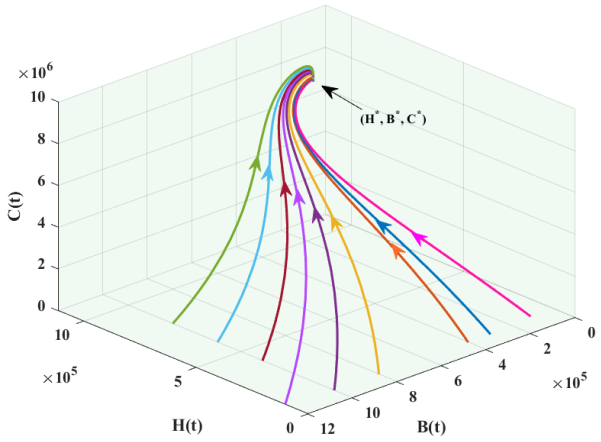

Figure 2: Global stability of (H^*, B^*, C^*) in $H - B - C$ plane

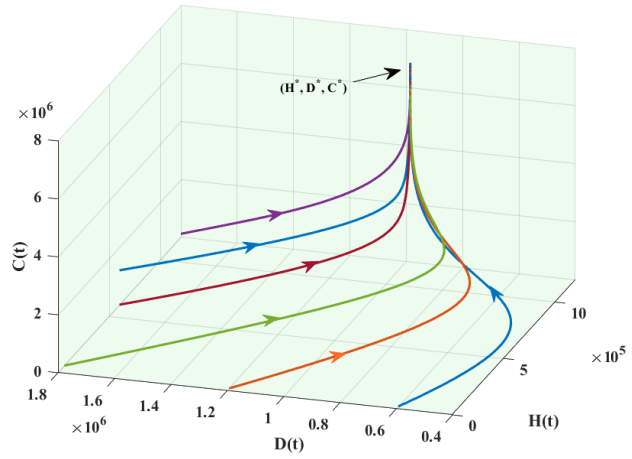

Figure 3: Global stability of (H^*, D^*, C^*) in $H - D - C$ plane

5.1 Effect of some key parameters on dynamics of the model (1)

We now examine the effects of key parameters α_1, λ, r_1, r_2, and κ on the dynamics of the model (1). Figure 4 shows the effects on the model system (1) as the parameter α_1 is varied, which denotes the rate at which the healthy population becomes diabetic without complications. On increasing the value of α_1, the healthy population (H), pre-diabetic population due to genetic

factors (G), and pre-diabetic population due to bad lifestyle (B) decrease, while the diabetic population without complications (D) and the diabetic population with complications increase (C). Thus, an increase in α_1 indicates a higher rate at which healthy individuals develop diabetes directly, bypassing the pre-diabetic stage. This leads to a decline in the healthy and pre-diabetic populations, while increasing the diabetic populations with and without complications. Biologically, this reflects poor early detection and prevention, resulting in faster disease progression and a higher overall diabetes burden.

In Figure 5, it can be seen that as the parameter value of λ increases, which represents the rate at which the healthy population become pre-diabetic due to bad lifestyles, the healthy population decreases. However, the pre-diabetic population due to a bad lifestyle, the diabetic population without complications and the diabetic population with complications increase. Biologically, an increase in λ signifies a higher influence of unhealthy lifestyle behaviors, causing more healthy individuals to transition into the pre-diabetic state due to poor lifestyle choices. As a result, the healthy population declines, while the pre-diabetic population due to bad lifestyle increases and subsequently contributes to higher diabetic populations with and without complications. This highlights the critical role of lifestyle-related risk factors in driving diabetes progression and complications.

Figure 6 shows the dynamics of the five population compartments of the model (1) for different values of the parameter r_1, which denotes the rate at which pre-diabetic individuals who adopt a healthy lifestyle return to the healthy class. The equilibrium level of the healthy population (H) is observed to increase, while the equilibrium level of the pre-diabetic population due to a poor lifestyle (B), the populations of diabetic individuals without complication (D) and diabetic individuals with complication (C) decrease with increasing value of r_1. Biologically, these results indicate that effective awareness-driven lifestyle interventions not only promote remission from pre-diabetes but also interrupt the disease progression pathway, increasing the healthy population and reducing diabetes-related complications.

The dynamics of the five population compartments of the model (1) for different values of the parameter r_2, representing the remission rate from diabetics without complication (D) to healthy (H), achieved through lifestyle interventions taking a high-protein diet and regular exercises, is shown in Figure 7. It is observed that the equilibrium level of the healthy class (H) increases, and the equilibrium levels of diabetics without complications (D) and with complications (C) decrease as the value of r_2 increases. The increase in the healthy class is more distinct compared to both diabetics classes. Furthermore, the equilibrium levels of pre-diabetic populations G and B increase with increasing value of r_2. This behavior suggests that an increase in r_2, which represents the rate of effective medical management or early detection, promotes recovery or stabilization of diabetic individuals, thereby reducing the burden of advanced disease. However,

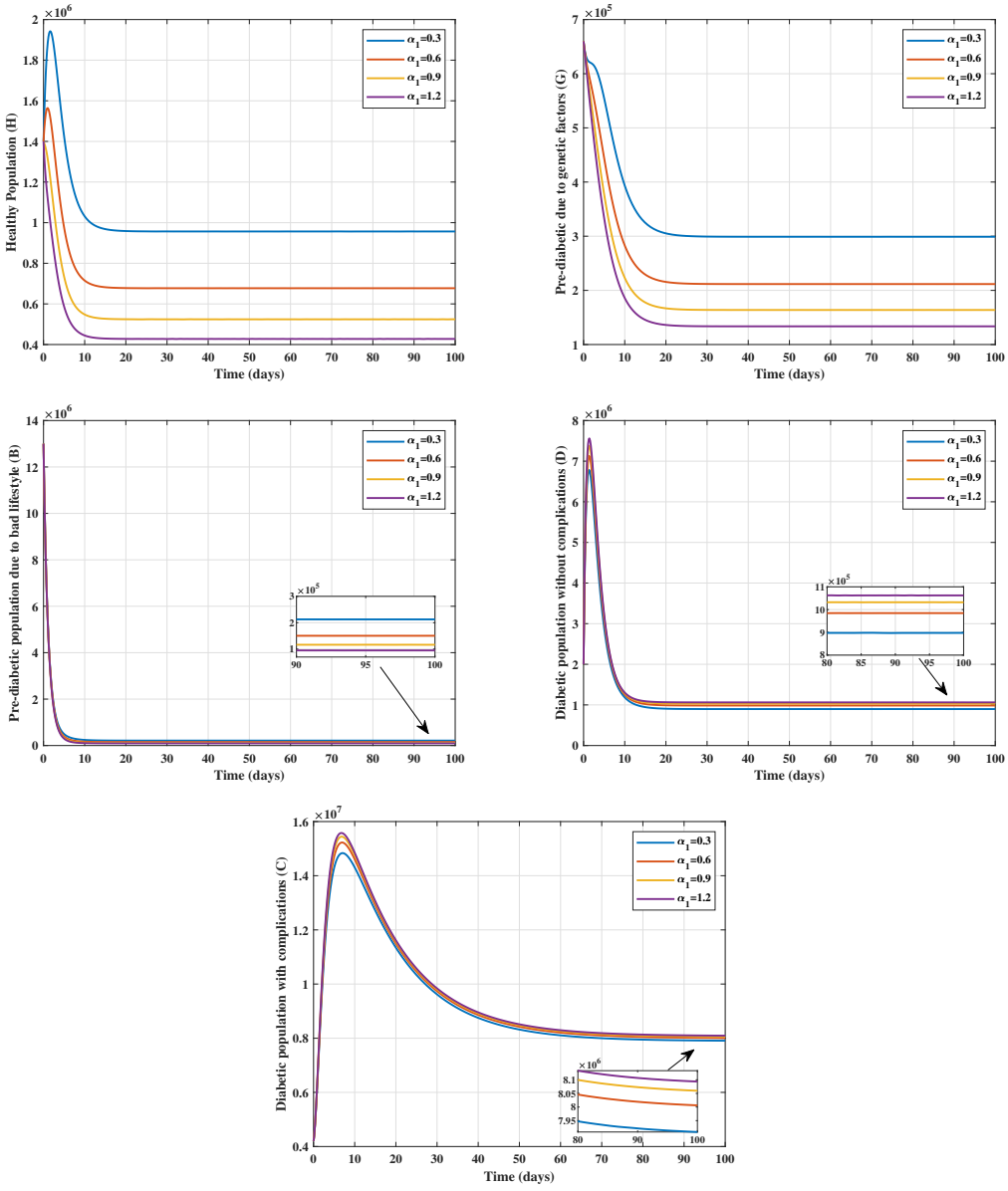

Figure 4: Dynamics of the populations of model (1) while varying α_1.

enhanced screening and prolonged survival may result in a temporary accumulation of individuals in the pre-diabetic classes G and B, even as the overall diabetic population declines.

Figure 8 shows the effect of the parameter κ, which represents the treatment rate for the diabetic population, on the model (1). As the parameter value κ increases, the healthy population (H), the pre-diabetic population due to a bad lifestyle (B), and the diabetic population without

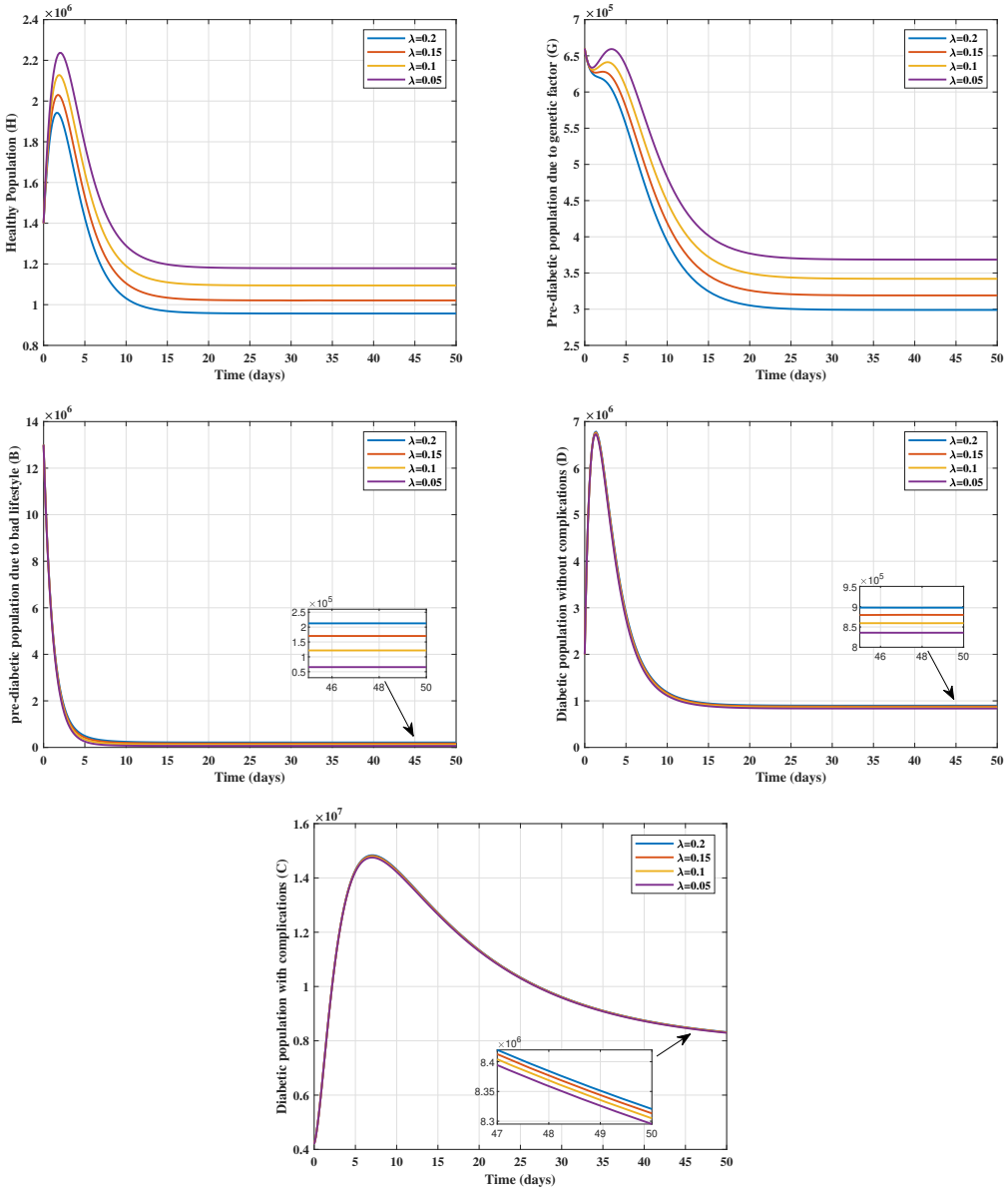

Figure 5: Dynamics of the populations of model (1) while varying λ.

complications (D) increase, while the diabetic population with complications (C) decreases significantly. Thus, an increase in the treatment rate κ reflects improved medical care and access to effective treatment for diabetic individuals. This leads to a significant reduction in the diabetic population with complications (C), as timely treatment prevents disease progression and promotes recovery. Consequently, the populations of healthy individuals (H), pre-diabetics due

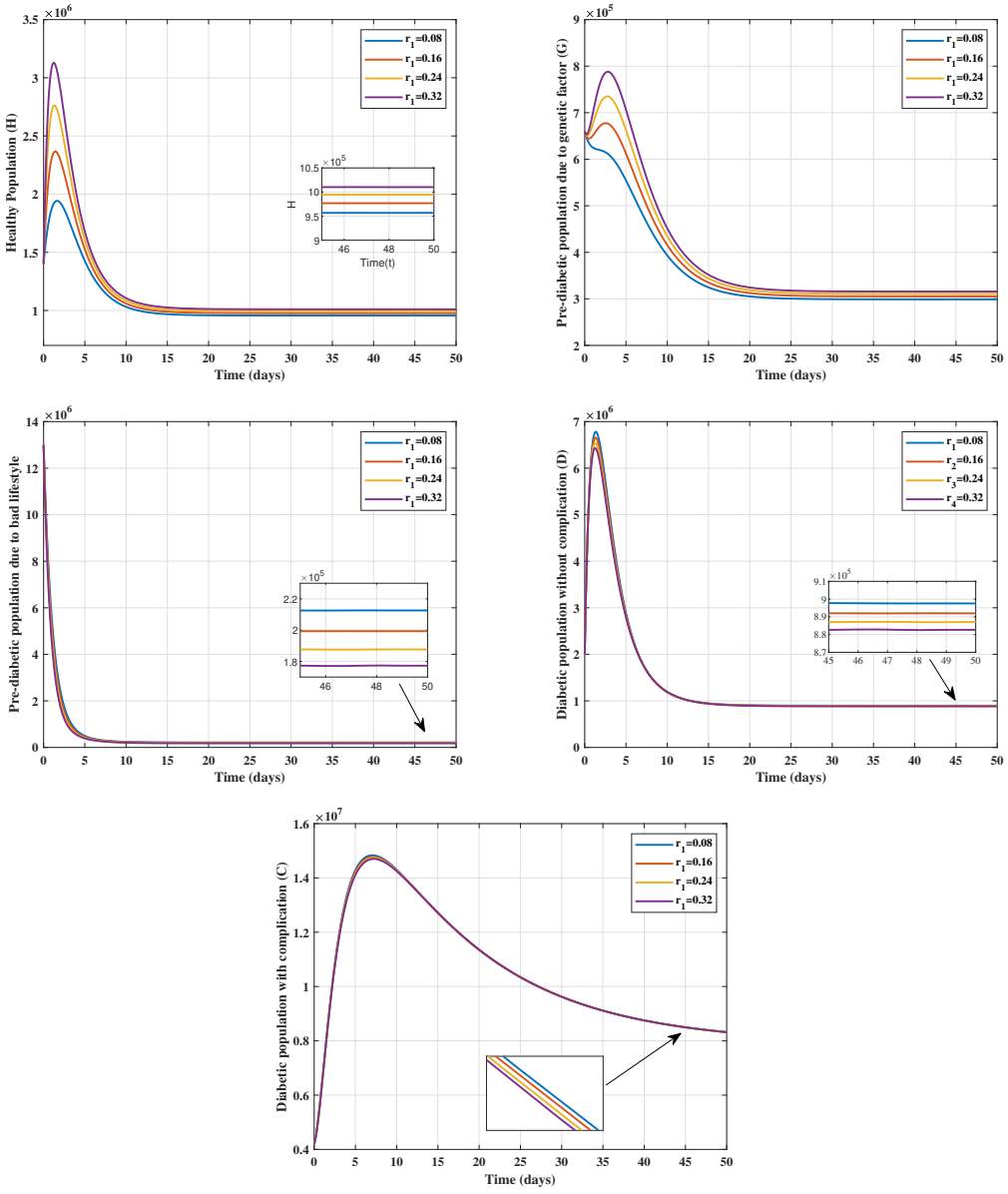

Figure 6: Dynamics of the populations of model (1) while varying r_1.

to bad lifestyle (B), and diabetics without complications (D) increase, indicating better disease management and overall improvement in population health.

Overall, the results indicate that unhealthy lifestyle adoption significantly alters the transient dynamics of the population and reduces the long-term healthy population, while increasing the disease burden at early stages. These findings highlight the critical biological role of lifestyle

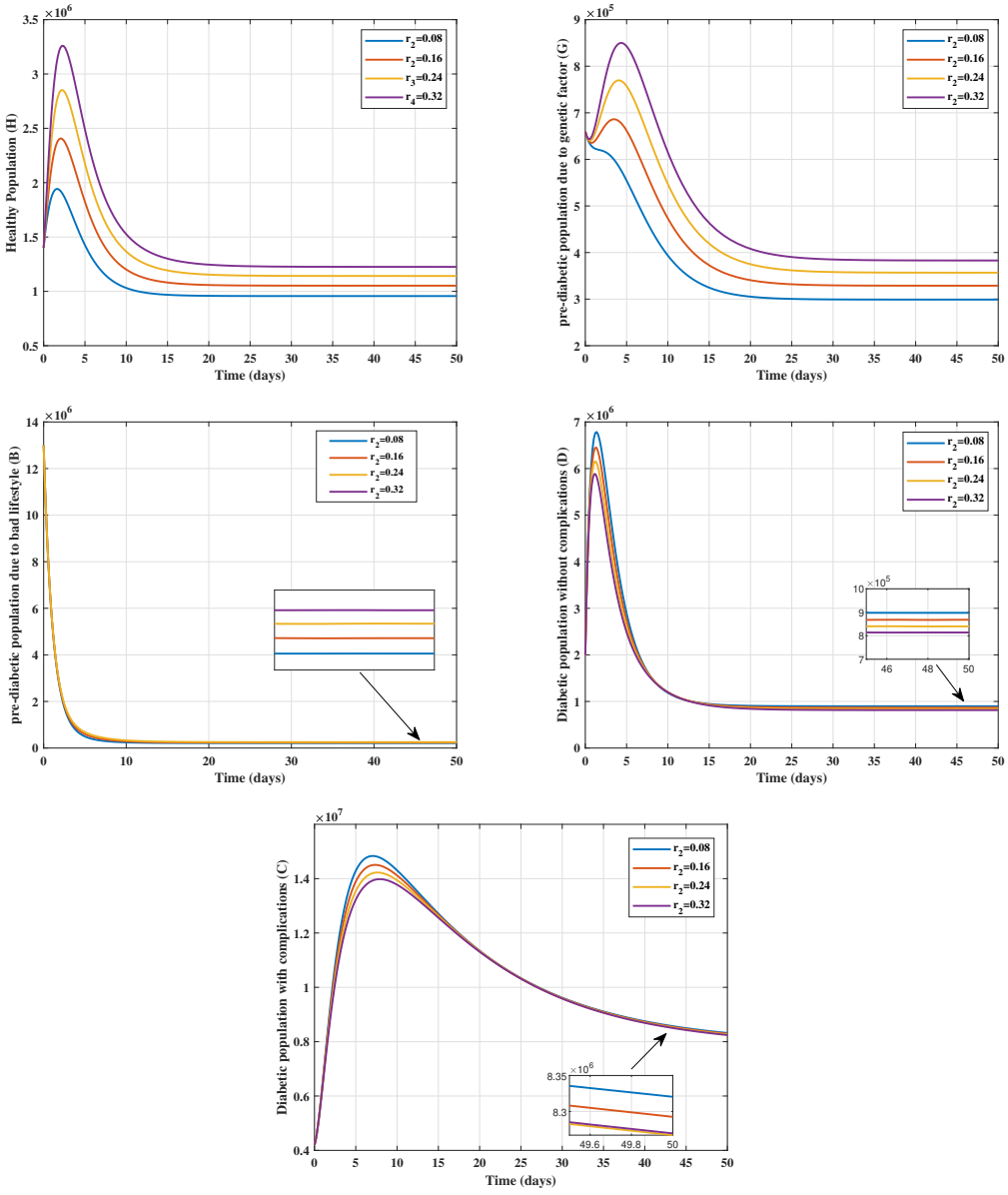

Figure 7: Dynamics of the populations of model (1) while varying r_2.

modification and preventive strategies in controlling the onset and progression of diabetes mellitus at the population level.

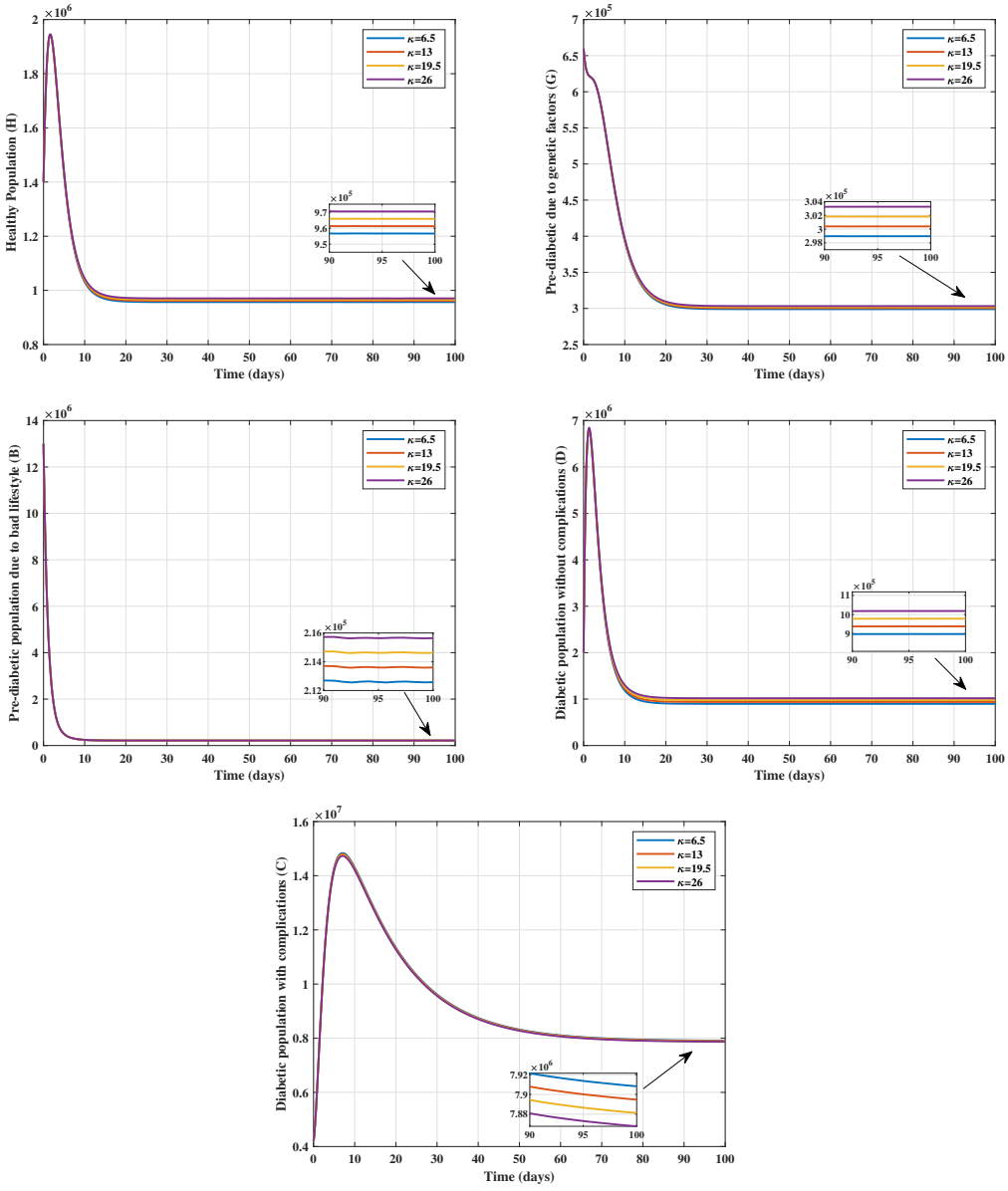

Figure 8: Dynamics of the populations of model (1) while varying κ.

5.2 Numerical simulations of the optimal control problem

In this part, we solve the optimality system by means of numerical simulations. For this purpose, we use an iterative method that involves forward solving the state system and backwards solving the adjoint system. In the first iteration, we make an initial guess for the controls. Before the

next iteration, we use the characterization to update the controls accordingly. This process is continued until successive iterations have converged. For solving the optimal control problem, we choose the weight functions as: $A_1 = 1$ and $A_2 = 400$ and the initial values for the model populations are considered as: $H(0) = 1400000$, $G(0) = 660000$, $B(0) = 13000000$, $D(0) = 2000000$, and $C(0) = 4200000$. The number of model populations after 100 months in both situations with and without controls is shown in Table 3. The values of all other parameters are considered as given in Table 2,

After 100 Months	Without control	With control
Healthy population(H)	956678	2835390
Pre-Diabetic due to bad lifestyle population(B)	212580	11.1205
Diabetics without complications(D)	897597	296860
Diabetics with complications(C)	7908450	7434080

Table 3: Evolution of the different diabetic populations after 100 months

Figure 9 illustrates the time evolution of the different population compartments of the diabetes model, comparing scenarios without control and with optimal control over the time interval $0 \leq t \leq 100$ days. It is clear from Figure 9(a) that the healthy population increases over time and eventually reaches a steady state. Under optimal control, the healthy population attains a higher equilibrium level compared to the scenario without any controlled measures. This suggests that awareness programs, lifestyle interventions, preventive strategies, and improving healthcare infrastructure are effective in sustaining individuals in the healthy class. Optimal control reduces the progression of healthy individuals toward pre-diabetes, thereby bolstering overall population health. From Figure 9(b), the results indicate that the pre-diabetic population is initially high and gradually declines as time progresses. Under optimal control, the population size reduced significantly throughout the simulation period. This indicates that interventions such as lifestyle modification, physical exercise, and dietary awareness effectively limit the growth of pre-diabetes caused by unhealthy behaviour. Optimal control reduces the burden of pre-diabetes, thereby preventing its progression to diabetes. Figure 9(c) shows that the diabetic population without complications rises quickly and reaches a stable level. Under optimal control, this compartment remains at a lower equilibrium compared to the uncontrolled scenario. This indicates that early awareness, lifestyle interventions, timely detection, continuous monitoring, improved healthcare and diagnostic facilities reduce the number of individuals progressing to the complicated diabetic stage. The implementation of the optimal control delays disease progression and improves glycaemic regulation, leading to a decrease in the diabetic population without complications. Figure 9(d) illustrates that, under optimal control, the population of diabetics with complications initially increases, reaches a peak, and then gradually declines, approaching a lower steady-state

level. Without any control measures, this population remains relatively higher. This highlights the effectiveness of clinical interventions, treatment compliance, and strengthened healthcare resources in reducing severe diabetic complications. Optimal control plays a crucial role in preventing the worsening of diabetes, reducing long-term health risks, mortality, and healthcare costs. The comparison clearly shows that optimal control strategies:

- Increase the number of healthy individuals,
- Reduce the number of pre-diabetic cases due to an unhealthy lifestyle,
- Limit the progression to diabetes,
- Effectively minimize diabetic-related complications.

6 Conclusions

In this manuscript, we have formulated a mathematical model of populations of Diabetes Mellitus with five compartments: Healthy population, pre-diabetics due to the genetic factors, pre-diabetics due to bad lifestyle, diabetic population without complications, and diabetics with complications. We established some basic properties of the model, like existence, nonnegativity and boundedness of the solutions, which are necessary for biological feasibility of the model. Furthermore, various conditions for local and global stability of a nonnegative equilibrium point have been derived. Then, to minimize the number of diabetic population and pre-diabetic populations due to a bad lifestyle, an optimal control problem is formulated using two control measures, which involve lifestyle intervention such as regular exercise, healthy diet, etc. and clinical intervention such as improving the healthcare infrastructure, enhanced diagnostic centre, etc. Then, we employed different results of control theory and obtained the characterizations of the optimal control. The problem is solved numerically using a forward-backwards iterative technique, resulting in an optimal control strategy which can maximize healthy population and minimizes the pre-diabetic population due to a bad lifestyle and the diabetic population to a significantly low level with proper diet, routine exercise, and enhanced medical facilities at minimum cost. The following conclusion can be drawn from this study:

- A mathematical model is formulated to study the dynamical relationship between the healthy population, the pre-diabetic population due to bad lifestyle and genetic factors, and the diabetic population with and without complications.

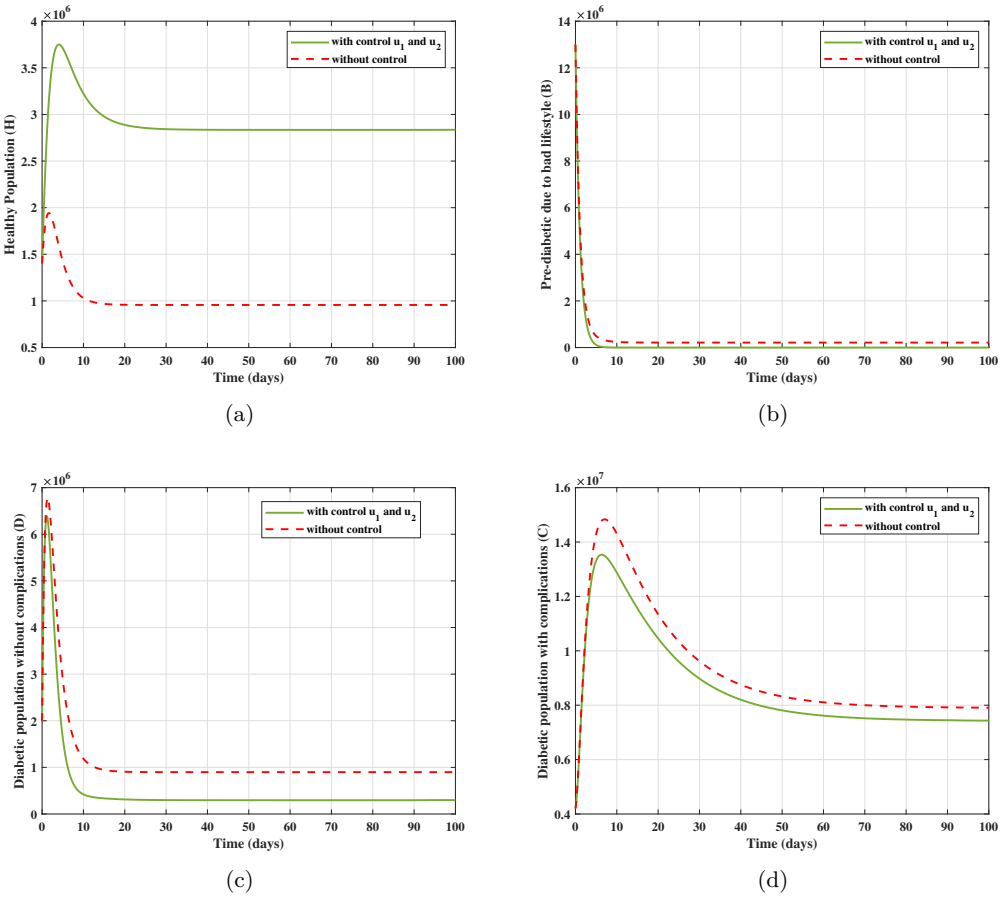

Figure 9: Evolution of model populations without and with optimal control u^*.

- Optimal control study reveals that increasing awareness among the pre-diabetic population, along with clinical interventions to improve the treatment rate of diabetics with complications, leads to an increase in the number of healthy individuals.

Although this study provides us with an optimal strategy to control the number of pre-diabetics due to a bad or irregular lifestyle via lifestyle intervention and adequate treatment facility, the model studied here can be further extended through the consideration of some other factors like delays caused when a healthy man becomes pre-diabetic, or the delay caused when a diabetic without complication becomes diabetic with complications, and so on, which will become the model more realistic. Also, this model can be extended by incorporating the memory effect through fractional order modeling. These improvements are left for future work.

Appendix A

Let $X = \begin{pmatrix} H(t) \\ G(t) \\ B(t) \\ D(t) \\ C(t) \end{pmatrix}$ and $\phi(X) = \begin{pmatrix} \frac{dH(t)}{dt} \\ \frac{dG(t)}{dt} \\ \frac{dB(t)}{dt} \\ \frac{dD(t)}{dt} \\ \frac{dC(t)}{dt} \end{pmatrix}$. The system (1) can be rewritten in the following form:

$$\phi(X) = \dot{A} = AX + B(X), \tag{36}$$

where

$$A = \begin{pmatrix} -(\mu+\alpha_1+\alpha_2+v+\lambda) & 0 & r_1 & r_2 & 0 \\ v & -(\gamma_1+\gamma_2+\mu) & 0 & 0 & 0 \\ \lambda & 0 & -(\delta+\mu+r_1) & 0 & 0 \\ \alpha_1 & \gamma_1 & \delta & -(\mu+\eta+r_2) & \beta_2 \\ \alpha_2 & \gamma_2 & 0 & \eta & -(\mu+\beta_1+\beta_2) \end{pmatrix}$$

and

$$B(X) = \begin{pmatrix} n \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Then

$$||\phi(X_1) - \phi(X_2)|| \leq ||A||.||(X_1 - X_2||.$$

Thus, it is obtained that the function Φ is uniformly Lipschitz continuous with the restrictions $H(0) \geq 0$, $G(0) \geq 0$, $B(0) \geq 0$, $D(0) \geq 0$, and $C(0) \geq 0$. Hence, a unique solution of the system (1) exists.

Appendix B

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

$$\begin{aligned} \frac{dH(t)}{dt} &= n - (\mu+\alpha_1+\alpha_2+v+\lambda)H(t) + r_1B + r_2D, \\ \implies \frac{dH(t)}{dt} &\geq -(\mu+\alpha_1+\alpha_2+v+\lambda)H(t), \end{aligned}$$

$$\implies \frac{dH(t)}{dt} + kH(t) \geq 0,$$

where $k = \mu + \alpha_1 + \alpha_2 + v + \lambda \geq 0$.

Multiplying by the integrating factor $e^{kt} \geq 0$ and solving the corresponding differential equation we have,

$$\begin{aligned} &\frac{d}{dt}\{H(t)e^{kt}\} = 0 \\ \implies &H(t) = H(0)e^{-kt}. \end{aligned}$$

Using the comparison theorem, we obtain $H(t) \geq 0$ for all t.

Similarly, we prove that $G(t) \geq 0$, $B(t) \geq 0$, $D(t) \geq 0$, and $C(t) \geq 0$ for all $t \geq 0$.

Appendix C

Since

$$N(t) = H(t) + G(t) + B(t) + D(t) + C(t),$$

by differentiating with respect to t, we get

$$\begin{aligned} &\frac{dN(t)}{dt} = \frac{dH(t)}{dt} + \frac{dG(t)}{dt} + \frac{dB(t)}{dt} + \frac{dD(t)}{dt} + \frac{dC(t)}{dt} \\ \implies &\frac{dN(t)}{dt} = n - \mu N(t) - \beta_1 C(t) \\ \implies &\frac{dN(t)}{dt} \leq n - \mu N(t) \\ \implies &N(t) \leq \frac{n}{\mu} - (\frac{n}{\mu} - N(0))e^{-\mu t}, \end{aligned}$$

where $N(0)$ represents the initial values of the population. Thus, $\lim\limits_{t\to\infty} \sup N(t) = \frac{n}{\mu}$, which implies that the region Ω is a positively invariant set for the system (1).

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Conflicts of Interest

All authors jointly worked on the results and they read and approved the final manuscript, and also declare that they have no competing interests.

References

[1] Aljohani, A., Shokri, A. and Mukalazi, H. *Analyzing the dynamic patterns of COVID-19 through nonstandard finite difference scheme*, Sci. Rep., 14(1) (2024), 8466.

[2] Aljohani, A., Hussain, A. and Shokri, A. *Mathematical modeling and the spreading of the cholera epidemic through numerical methods*, Comput. Syst. Oncol., 5(2) (2025), e70006.

[3] Banzi, W., Kambutse, I., Dusabejambo, V., Rutaganda, E., Minani, F., Niyobuhungiro, J., Mpinganzima, L. and Ntaganda, J.M. *Mathematical modelling of glucose–insulin system and test of abnormalities of type 2 diabetic patients*, Int. J. Math. Math. Sci., 2021(1) (2021), 6660177.

[4] Borah, P.B., Nath, B.J., Nath, K.C. and Sarmah, H.K. *Stability analysis of a nonlinear mathematical model for COVID-19 transmission dynamics*, Commun. Math. Biol. Neurosci., (2023), Article ID 26.

[5] Boutayeb, W., Lamlili, M.E., Boutayeb, A. and Derouich, M. *The dynamics of a population of healthy people, pre-diabetics and diabetics with and without complications with optimal control*, In: Proceedings of the Mediterranean Conference on Information & Communication Technologies 2015 (MedCT 2015), Vol. 1, Springer Int. Publ., Cham (2016), 463–471.

[6] Chen, Y. and Zhang, J. *Dynamic analysis and optimal control of influenza model with discrete age structure*, Math. Methods Appl. Sci., 47(6) (2024), 4260–4282.

[7] Das, P., Das, S., Das, P., Rihan, F.A., Uzuntarla, M. and Ghosh, D. *Optimal control strategy for cancer remission using combinatorial therapy: a mathematical model-based approach*, Chaos Solitons Fractals, 145 (2021), 110789.

[8] Derouich, M., Boutayeb, A., Boutayeb, W. and Lamlili, M. *Optimal control approach to the dynamics of a population of diabetics*, Appl. Math. Sci., 8(56) (2014), 2773–2782.

[9] El Mansouri, A., Smouni, I., Khajji, B., Labzai, A., Belam, M. and Tidli, Y. *Optimal control in a mathematical model of a spread of the obesity epidemic and its impact on diabetes*, Commun. Math. Biol. Neurosci., (2023), Article ID 74.

[10] Fleming, W.H. and Rishel, R.W. *Deterministic and stochastic optimal control*, Springer Science & Business Media, 2012.

[11] Gupta, R. and Kumar, S. *Analysis of fractional-order population model of diabetes and effect of remission through lifestyle intervention*, Int. J. Appl. Comput. Math., 7(2) (2021), 53.

[12] Hallberg, S.J., Gershuni, V.M., Hazbun, T.L. and Athinarayanan, S.J. *Reversing type 2 diabetes: a narrative review of the evidence*, Nutrients, 11(4) (2019), 766.

[13] Hayes, C. and Kriska, A. *Role of physical activity in diabetes management and prevention*, J. Am. Diet. Assoc., 108(4) (2008), S19–S23.

[14] International Diabetes Federation. *IDF diabetes atlas*, 10th ed., International Diabetes Federation, Brussels (2021). Available at: https://www.idf.org.

[15] Kouidere, A., Khajji, B., Balatif, O. and Rachik, M. *A multi-age mathematical modeling of the dynamics of population diabetics with effect of lifestyle using optimal control*, J. Appl. Math. Comput., 67(1) (2021), 375–403.

[16] Kouidere, A., Labzai, A., Ferjouchia, H., Balatif, O. and Rachik, M. *A new mathematical modeling with optimal control strategy for the dynamics of population of diabetics and its complications with effect of behavioral factors*, J. Appl. Math., 2020(1) (2020), 1943410.

[17] Kumari, P., Singh, S. and Singh, H.P. *Bifurcation and stability analysis of glucose–insulin regulatory system in the presence of β-cells*, Iran. J. Sci. Technol. Trans. A Sci., 45(5) (2021), 1743–1756.

[18] Marín-Peñalver, J.J., Martín-Timón, I., Sevillano-Collantes, C. and del Cañizo-Gómez, F.J. *Update on the treatment of type 2 diabetes mellitus*, World J. Diabetes, 7(17) (2016), 354–395.

[19] Ma, M. and Li, J. *Dynamics of a glucose–insulin model*, J. Biol. Dyn., 16(1) (2022), 733–745.

[20] Misra, A.K. and Kumari, M. *Modeling the effects of TV and social media advertisements on diabetes*, J. Biol. Syst., 31(3) (2023), 1099–1124.

[21] Mollah, S. and Biswas, S. *Effect of awareness program on diabetes mellitus: deterministic and stochastic approach*, J. Appl. Math. Comput., 66(1) (2021), 61–86.

[22] Mollah, S. and Biswas, S. *Optimal control for the complication of type 2 diabetes: the role of awareness programs by media and treatment*, Int. J. Dyn. Control, 11(2) (2023), 877–891.

[23] Mutyambizi, C., Pavlova, M., Chola, L., Hongoro, C. and Groot, W. *Cost of diabetes mellitus in Africa: a systematic review of existing literature*, Glob. Health, 14(1) (2018), 3.

[24] Naik, P.A., Yeolekar, B.M., Owolabi, K.M. and Yeolekar, M. *Modeling and analyzing glucose–insulin interactions during diabetes through fractional dynamics in presence of glucagon*, J. Adv. Res. (2026), accepted.

[25] Nasir, H. *Stability analysis and optimal control of a five-state diabetic population model*, J. Stat. Manag. Syst., 25(1) (2022), 245–267.

[26] Nasir, H. *Hopf bifurcation analysis for a diabetic population model with two delays and saturated treatment*, Phys. Scr., 96(12) (2021), 125013.

[27] Nath, B.J., Dehingia, K., Mishra, V.N., Chu, Y.M. and Sarmah, H.K. *Mathematical analysis of a within-host model of SARS-CoV-2*, Adv. Differ. Equ., 2021(1) (2021), 113.

[28] Nath, B.J., Sarmah, H.K. and Maurer, H. *An optimal control strategy for antiretroviral treatment of HIV infection in presence of immunotherapy*, Qual. Theory Dyn. Syst., 21(2) (2022), 30.

[29] Nath, B.J., Sadri, K., Sarmah, H.K. and Hosseini, K. *An optimal combination of antiretroviral treatment and immunotherapy for controlling HIV infection*, Math. Comput. Simul., 217 (2024), 226–243.

[30] Ong, K.L., Stafford, L.K., McLaughlin, S.A., Boyko, E.J., Vollset, S.E., Smith, A.E., Dalton, B.E., Duprey, J., Cruz, J.A., Hagins, H. and Lindstedt, P.A. *Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021*, Lancet, 402 (2023), 203–234.

[31] Pal, K.K., Rai, R.K., Tiwari, P.K. and Misra, A.K. *Mathematical modeling of control measures for preventing kidney failure and managing diabetes: sensitivity analysis and optimal strategies*, J. Appl. Math. Comput., 71(5) (2025), 6455–6487.

[32] Pal, K.K., Rai, R.K., Tiwari, P.K. and Misra, A.K. *Role of awareness programs on diabetes prevention and control of viral infection: a study of optimal control*, Eur. Phys. J. Plus, 140(2) (2025), 150.

[33] Permatasari, A.H., Tjahjana, R.H. and Udjiani, T. *Existence and characterization of optimal control in mathematics model of diabetics population*, J. Phys. Conf. Ser., 983(1) (2018), 012069.

[34] Pontryagin, L.S., Boltyanskii, V.G., Gamkrelidze, R.V. and Mishchenko, E.F. *The mathematical theory of optimal processes*, John Wiley & Sons, London, UK (1962).

[35] Rai, R.K., Pal, K.K., Tiwari, P.K., Martcheva, M. and Misra, A.K. *Impact of social media and word-of-mouth on the transmission dynamics of communicable and non-communicable diseases*, Int. J. Biomath., 16 (2024), 2450094.

[36] Urbina, G., Riahi, D.N. and Bhatta, D. *Mathematical modeling of nonlinear blood glucose–insulin dynamics with beta cells effect*, Appl. Appl. Math., 15(1) (2020), 10.

[37] World Health Organization. *Report of a WHO/IDF consultation*, World Health Organization (2016).

[38] Xu, H.Y., Khalsaraei, M.M., Shokri, A., Ramos, H. and Molayi, M. *Nonstandard finite difference schemes with positivity property with application to a mathematical model of HIV infection*, Appl. Comput. Math., 23(4) (2024), 541–557.

[39] Zhonghua, Z. and Yaohong, S. *Qualitative analysis of a SIR epidemic model with saturated treatment rate*, J. Appl. Math. Comput., 34(1) (2010), 177–194.