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A MULTI-AGE MATHEMATICAL MODEL OF PSYCHOLOGICAL STRESS, HBA1C DYNAMICS, AND METABOLIC DISORDERS WITH CONTROL STRATEGIES

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Abstract. This article investigates how psychological stress-social, economic, and intellectual-affects HbA1c dynamics and related health outcomes across different age groups. The population is divided into six compartments: healthy, stressed, elevated HbA1c, prediabetic, diabetic, and individuals with cardiovascular complications. Age-specific analysis includes young adults (18–25), middle-aged adults (45–66), and older adults (> 70), emphasizing differences in stress response and metabolic regulation. Two intervention strategies are proposed: $u(t)$, targeting stress reduction through family support and yoga practice, and $v(t)$, promoting healthy diet and lifestyle habits. Numerical simulations demonstrate that combining both strategies effectively lowers HbA1c levels, slows diabetes progression, and reduces cardiovascular risks across all age groups. The optimal control problem is formulated to minimize the number of stressed and metabolically dysregulated individuals as well as the associated intervention costs, and Pontryagin's Maximum Principle is applied to determine the optimal strategies.

Keywords: mathematical modelling; diabetes; stress; multi age; optimal control; numerical simulation.

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1. INTRODUCTION

Glycated hemoglobin, or HbA1c, is a vital biomarker for assessing long-term blood glucose control. It measures the percentage of hemoglobin in red blood cells chemically bonded with circulating glucose. By measuring HbA1c, we gain insight into average blood glucose levels over the past two to three months, providing a reliable indicator of an individual's glucose regulation [1]. Elevated HbA1c levels indicate poor glucose management, which is commonly associated with diabetes-related complications, such as cardiovascular disease, neuropathy, and retinopathy [2], in non-diabetic individuals, elevated HbA1c levels can reflect high baseline glucose levels, which over time, can contribute to increased blood vessel and heart strain. This chronic strain is associated with a greater likelihood of hypertension, atherosclerosis, and other cardiovascular conditions [3]. Additionally, individuals with higher HbA1c levels who have not yet developed diabetes may be at increased risk of progressing to prediabetes . This stage is characterized by blood glucose levels that are above normal but not high enough to be diagnosed as diabetes. Prediabetes is a significant risk factor for developing type 2 diabetes and further cardiovascular complications if left unmanaged [4]. Stress is a biological response triggered by internal or external stimuli [5]. The ability to adapt to stress has evolutionary benefits, supporting survival through mechanisms known as the "fight or flight" response, which temporarily suppresses growth, reproduction, and immunity [6, 7, 8, 9]. However, when stress becomes chronic, these effects may persist, contributing to various health issues. Chronic stress often leads to behaviors such as inactivity and poor dietary choices (e.g., larger portions, comfort eating, and increased alcohol consumption), which can result in weight gain and disrupt glucose and lipid metabolism [9]. The stress response involves a complex neuroendocrine system, including both the central nervous system and peripheral pathways [6]. Glucocorticoids (GCs) and catecholamines are key hormonal mediators in this system, produced by the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system, respectively. Acute stress does not typically disrupt overall metabolism; however, chronic stress elevates stress hormones, which can impair glucose regulation. In an acute stress event, blood glucose and insulin levels rise briefly to maintain normoglycemia. But during chronic stress, the body undergoes "allostasis," or stability through change, where glucose levels remain elevated to signal ongoing insulin

resistance to the beta cells, which can eventually lead to type 2 diabetes (T2D), even if beta-cell function is initially normal [10]. This prolonged elevation in glucose due to chronic stress can also cause an increase in HbA1c levels , a marker for long-term glucose control. Elevated HbA1c is a sign of poor glucose regulation and is associated with greater risks for cardiovascular complications and other metabolic health issues, further complicating the impact of chronic stress on overall health. The connection between psychological stress, type 2 diabetes, and cardiovascular complications is well-documented, stress promotes chronic inflammation and insulin resistance, both of which increase diabetes risk , for individuals with diabetes, stress worsens cardiovascular complications by increasing blood pressure, cholesterol, and inflammation, creating a feedback loop of worsening health [8]. This interaction is particularly complex in older adults [11], as aging intensifies the effects of stress on metabolic regulation, increasing vulnerability to complications. Younger adults, often managing academic and career-related stress, show metabolic responses to stress that affect their health, while middle-aged adults, balancing work and family responsibilities, experience pronounced impacts on metabolic health [12]. Finally, older adults, often facing life transitions and chronic health issues, are especially vulnerable to stress-induced cardiovascular complications in the context of diabetes [13]. Several studies in medical literature have explored the relationship between chronic stress and metabolic dysfunction, including elevated HbA1c levels. Research has shown that individuals experiencing long-term stress tend to have higher HbA1c values, indicating poor glucose control and an increased risk of developing type 2 diabetes. A study by Stojanovic et al. (2016) demonstrated that chronic stress is linked to increased HbA1c levels in both diabetic and non-diabetic individuals, highlighting the broader implications of stress on glucose metabolism [14]. Similarly, Lazzarino et al. (2013) showed that stress-induced increases in cortisol and insulin resistance can lead to long-term elevations in blood glucose, contributing to diabetes and complications like cardiovascular disease and neuropathy [15]. Recent studies expand on these findings, such as a large-scale population study of 608,474 Canadian adults, which found that even slight increases in HbA1c among non-diabetics were associated with heightened cardiovascular risks, emphasizing the importance of HbA1c as a marker for broader health risks [16]. Additionally, research on age and gender effects on HbA1c in non-diabetic adults revealed that

HbA1c levels tend to rise with age, and gender differences also contribute to HbA1c variability, further underscoring the value of demographic and stress-related considerations in HbA1c management [17]. Several mathematical studies have explored aspects of glucose-insulin dynamics, diabetes progression, and general metabolic regulation, yet few have directly examined the influence of stress on HbA1c levels. For example, Ackerman et al. (1965) pioneered one of the earliest mathematical models of glucose-insulin interaction, creating a framework for subsequent studies on glucose homeostasis [18]. Since then, other researchers, like Bergman et al. (1985), have expanded on this by developing models of insulin sensitivity, such as the Minimal Model, which has been instrumental in analyzing glucose tolerance and the risk of diabetes [19]. Moreover, recent studies by Boutayeb et al. (2004) have applied compartmental modeling to evaluate the epidemiology of diabetes and its complications across populations, highlighting the broader public health impact of diabetes but not directly incorporating stress factors [20].

While these studies have been foundational, there is limited mathematical work specifically modeling the relationship between chronic stress, HbA1c, and subsequent cardiovascular risks. Our work aims to fill this gap by incorporating stress as a variable influencing HbA1c levels, informed by recent medical findings on stress-related glucose dysregulation. By doing so, this study extends existing models of glucose metabolism and HbA1c regulation to better reflect real-world scenarios where stress is a known factor in both diabetic and non-diabetic health outcomes.

Given this context, our study has three primary objectives. First, we aim to analyze how psychological stress impacts HbA1c levels and metabolic complications across various age groups and health statuses. Second, we investigate how age influences stress sensitivity and health effects to identify at-risk subgroups for developing diabetes or cardiovascular issues. Third, we design optimal control strategies to reduce stress impacts and enhance glucose regulation in diabetic and prediabetic populations.

To achieve these objectives, we utilize optimal control theory, particularly Pontryagin's Maximum Principle. This theorem enables the optimization of intervention strategies, such as reducing stress through family support and yoga, which promote emotional well-being and relaxation [21]. Additionally, we emphasize healthier dietary and lifestyle habits to manage behavioral

risks optimally [22]. This approach allows us to improve HbA1c levels and reduce metabolic and cardiovascular complications. Using Pontryagin's theorem and optimal control, we develop strategies to better manage psychological stress impacts on metabolic and cardiovascular health in diabetes-prone individuals, marking a novel contribution that bridges mathematical modeling with medical insights on stress and glucose regulation. To validate our theoretical results, we apply numerical simulations with and without optimal control interventions. In scenarios without control, stress and unhealthy habits negatively impact HbA1c levels and exacerbate diabetes-related complications. By contrast, in simulations implementing optimal control strategies, we observe a notable improvement in HbA1c levels and a reduction in cardiovascular risk. These numerical applications confirm the theoretical predictions, demonstrating the effectiveness of optimal control in managing stress and improving metabolic health outcomes

2. Model Formulation

In this section, we formulate a model that includes six compartments to capture the interactions between psychological stress, HbA1c levels, diabetes, and cardiovascular complications across different age groups. The model compartments are described as follows:

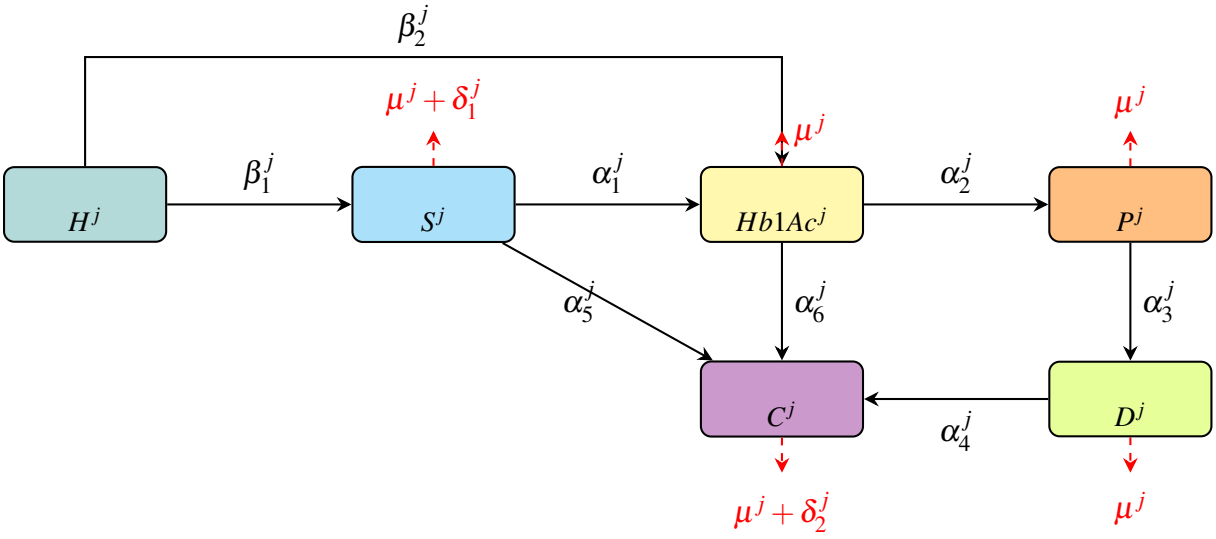

Figure 1. Compartments Model

- **Healthy Population ($H^j(t)$):** This compartment represents individuals without significant metabolic disorders.
- **Stressed Population ($S^j(t)$):** This compartment includes individuals experiencing significant psychological stress due to social, economic, or intellectual factors.
- **Population with Elevated HbA1c Levels ($Hb^j(t)$):** This group consists of individuals with high HbA1c levels, indicative of poor long-term glucose control.
- **Prediabetic Population ($P^j(t)$):** This compartment includes individuals with elevated glucose levels that are not yet classified as diabetes.
- **Diabetic Population ($D^j(t)$):** This group consists of individuals diagnosed with diabetes.
- **Population with Cardiovascular Complications ($C^j(t)$):** This compartment includes individuals with cardiovascular complications associated with high HbA1c levels or diabetes.

  Elevated HbA1c and uncontrolled diabetes are significant risk factors for cardiovascular diseases. Stress, both psychological and physiological, can exacerbate these complications by impacting glucose control and vascular health. The model explores the interplay between stress, HbA1c levels, diabetes, and cardiovascular health, and assesses the potential for interventions to reduce cardiovascular risks.

  Each compartment in this model reflects the impact of psychological stress and metabolic health across different age groups, providing insights into how stress influences HbA1c levels and related health outcomes. The model aims to identify effective strategies for managing stress and improving health in various populations. Here, j denotes age groups: $j = 1$ for ages 18-25, $j = 2$ for ages 45-66, and $j = 3$ for ages over of 70

2.1. Model Equations. The dynamics of the model are governed by the following system (1) of differential equations:

$$
(1)\qquad \begin{cases} \dfrac{dH^j}{dt} = I^j - (\beta_1^j + \beta_2^j + \mu^j)H^j, \\ \dfrac{dS^j}{dt} = \beta_1^j H^j - (\mu^j + \delta_1^j + \alpha_1^j + \alpha_5^j)S^j, \\ \dfrac{dHb^j}{dt} = \beta_2^j H^j + \alpha_1^j S^j - (\mu^j + \alpha_2^j + \alpha_6^j)Hb^j, \\ \dfrac{dP^j}{dt} = \alpha_2^j Hb^j - (\mu^j + \alpha_3^j)P^j, \\ \dfrac{dD^j}{dt} = \alpha_3^j P^j - (\mu^j + \alpha_4^j)D^j, \\ \dfrac{dC^j}{dt} = \alpha_6^j Hb^j + \alpha_5^j S^j + \alpha_4^j D^j - (\mu^j + \delta_2^j)C^j. \end{cases}
$$

2.2. Explanation of Terms.

- α_1^j: Represents the direct impact of psychological stress on increasing HbA1c levels. Stress disrupts metabolism, leading to higher blood glucose over time, captured by the transition from stressed individuals to those with elevated HbA1c [23].
- α_2^j: Describes the progression from elevated HbA1c to a prediabetic state. Chronically high HbA1c signals poor glucose regulation, increasing the risk of prediabetes [24].
- α_3^j: Represents the progression from prediabetes to type 2 diabetes. As glucose regulation worsens due to insulin resistance or beta cell dysfunction, the likelihood of developing diabetes increases.

- α_4^j: Models the transition from diabetes to cardiovascular complications. Poorly managed diabetes with high glucose levels can damage blood vessels, leading to heart disease or stroke [25].
- α_5^j: Represents the direct effect of stress on cardiovascular complications. Chronic stress negatively impacts cardiovascular health by increasing blood pressure and inflammation.[Stress and cardiovascular disease: an update [26].
- α_6^j: Describes the transition from elevated HbA1c to cardiovascular complications. High HbA1c damages blood vessels, contributing to diseases like atherosclerosis [27].
- β_1^j: Represents how healthy individuals become stressed due to psychological factors such as social and economic pressures, leading to chronic stress [28].
- β_2^j: Reflects how healthy individuals can develop elevated HbA1c due to unhealthy lifestyle factors, such as poor diet or lack of exercise, leading to poor glucose control [23].
- δ_1^j: Represents the mortality rate due to stress-related causes such as suicide. High stress levels can lead to mental health crises, resulting in increased risk of self-harm [29].
- δ_2^j: Represents the mortality rate due to cardiovascular complications such as stroke. Poor glucose regulation, along with stress, significantly increases the risk of cardiovascular events, leading to death [30, 31, 32, 33].
- μ^j: Represents natural mortality in each population group, accounting for deaths not specifically related to stress, metabolic diseases, or cardiovascular complications.

3. Basic Properties

3.1. Positivity of Solutions of the Stress Model. To establish that all solutions of the stress model remain positive for all $t \geq 0$, we present the following theorem.

Theorem 1. *If $H^j(0) \geq 0$ $S^j(0) \geq 0$, $Hb^j(0) \geq 0$, $P^j(0) \geq 0$, $D^j(0) \geq 0$, and $C^j(0) \geq 0$, then the solutions of the system of equations $H^j(t) \geq 0$ $S^j(t) \geq 0$, $Hb^j(t) \geq 0$, $P^j(t) \geq 0$, $D^j(t) \geq 0$, and $C^j(t) \geq 0$ for all $t \geq 0$.*

Proof. Consider the differential equation for $S^j(t)$:

$$\frac{dS^j(t)}{dt} = I^j - (\mu^j + \delta_1^j + \alpha_1^j + \alpha_5^j)S^j(t).$$

Define:

$$A^j(t) = \mu^j + \delta_1^j + \alpha_1^j + \alpha_5^j.$$

Then the differential equation can be rewritten as:

$$\frac{dS^j(t)}{dt} + A^j(t)S^j(t) = I^j.$$

To solve this, multiply both sides by the integrating factor:

$$\exp\left(\int_0^t A^j(s)\,ds\right).$$

We obtain:

$$\frac{d}{dt}\left(S^j(t)\exp\left(\int_0^t A^j(s)\,ds\right)\right) = I^j\exp\left(\int_0^t A^j(s)\,ds\right).$$

Integrate both sides from 0 to t:

$$S^j(t)\exp\left(\int_0^t A^j(s)\,ds\right) = S^j(0) + \int_0^t I^j\exp\left(\int_0^s A^j(u)\,du\right)ds.$$

Thus:

$$S^j(t) = \exp\left(-\int_0^t A^j(s)\,ds\right)\left[S^j(0) + \int_0^t I^j\exp\left(\int_0^s A^j(u)\,du\right)ds\right].$$

Since $S^j(0) \geq 0$ and $I^j \geq 0$, it follows that $S^j(t) \geq 0$ for all $t \geq 0$.

Similarly, consider the differential equation for $Hb^j(t)$:

$$\frac{dHb^j(t)}{dt} = \alpha_1^j S^j(t) - (\mu^j + \alpha_2^j + \alpha_6^j)Hb^j(t).$$

Define:

$$B^j(t) = \mu^j + \alpha_2^j + \alpha_6^j.$$

Thus:

$$\frac{dHb^j(t)}{dt} + B^j(t)Hb^j(t) = \alpha_1^j S^j(t).$$

Multiply both sides by:

$$\exp\left(\int_0^t B^j(s)\,ds\right).$$

We get:

$$\frac{d}{dt}\left(Hb^j(t)\exp\left(\int_0^t B^j(s)\,ds\right)\right) = \alpha_1^j S^j(t)\exp\left(\int_0^t B^j(s)\,ds\right).$$

Integrate from 0 to t:

$$Hb^j(t)\exp\left(\int_0^t B^j(s)\,ds\right) = Hb^j(0) + \int_0^t \alpha_1^j S^j(s)\exp\left(\int_0^s B^j(u)\,du\right)ds.$$

Thus:

$$Hb^j(t) = \exp\left(-\int_0^t B^j(s)\,ds\right)\left[Hb^j(0) + \int_0^t \alpha_1^j S^j(s)\exp\left(\int_0^s B^j(u)\,du\right)ds\right].$$

Since $Hb^j(0) \geq 0$ and $S^j \geq 0$, it follows that $Hb^j(t) \geq 0$ for all $t \geq 0$.

Proceed similarly for $P^j(t)$, $D^j(t)$, and $C^j(t)$ using their respective differential equations:

$$\frac{dP^j(t)}{dt} = \alpha_2^j Hb^j(t) - (\mu^j + \alpha_3^j)P^j(t),$$

$$\frac{dD^j(t)}{dt} = \alpha_3^j P^j(t) - (\mu^j + \alpha_4^j)D^j(t),$$

$$\frac{dC^j(t)}{dt} = \alpha_6^j Hb^j(t) + \alpha_5^j S^j(t) + \alpha_4^j D^j(t) - (\mu^j + \delta_2^j)C^j(t).$$

In each case, the solution is positive due to the positivity of initial conditions and the non-negativity of the source terms I^j, α_i^j, and δ_i^j.

Thus, the solutions $S^j(t)$, $Hb^j(t)$, $P^j(t)$, $D^j(t)$, and $C^j(t)$ are positive for all $t \geq 0$.

□

4. Boundedness of Solutions

Theorem 2. *If I^j is bounded and $\mu^j > 0$, then the solution $N^j(t)$ of the system*

$$\frac{dN^j}{dt} \leq I^j - \mu^j N^j$$

is bounded above by $\frac{I^j}{\mu^j}$ for all $t \geq 0$.

Proof. Consider the differential inequality:

$$\frac{dN^j}{dt} \leq I^j - \mu^j N$$

Rewrite it as:

$$\frac{dN^j}{dt} + \mu^j N^j \leq I^j$$

To solve this, consider the associated homogeneous differential equation:

$$\frac{dN^j}{dt} + \mu^j N^j = 0$$

The general solution of this homogeneous equation is:

$$N_h^j(t) = N^j(0)\exp(-\mu^j t)$$

For the non-homogeneous equation:

$$\frac{dN^j}{dt} + \mu^j N^j = I^j$$

A particular solution can be found by setting:

$$N_p^j(t) = \frac{I^j}{\mu^j}$$

Substitute $N_p^j(t) = \frac{I^j}{\mu^j}$ into the non-homogeneous differential equation:

$$\frac{d}{dt}\left(\frac{I^j}{\mu^j}\right) + \mu^j\left(\frac{I^j}{\mu^j}\right) = I^j$$

which simplifies to:

$$0 + I^j = I^j$$

Thus, $N_p^j(t) = \frac{I^j}{\mu^j}$ is a particular solution.

The general solution to the differential equation is:

$$N^j(t) = N_h^j(t) + N_p^j(t) = N^j(0)\exp(-\mu^j t) + \frac{I^j}{\mu^j}$$

To bound $N(t)$, observe:

$$N^j(t) \leq N^j(0)\exp(-\mu^j t) + \frac{I^j}{\mu^j}$$

As $t \to \infty$, $N^j(0)\exp(-\mu^j t) \to 0$, hence:

$$\lim_{t\to\infty} N^j(t) \leq \frac{I^j}{\mu^j}$$

Therefore, $N^j(t)$ is bounded above by $\frac{I^j}{\mu^j}$ for all $t \geq 0$. □

5. Existence and Uniqueness of Solutions

Theorem 3. *Consider the initial value problem for a system of first-order differential equations*

$$\frac{d\mathbf{N}(t)}{dt} = \mathbf{A}(t)\mathbf{N}(t) + \mathbf{b}(t),$$

with initial condition $\mathbf{N}(0) = \mathbf{N}_0$. *If* $\mathbf{A}(t)$ *and* $\mathbf{b}(t)$ *are continuous functions in* t *on some interval* I *containing* $t = 0$*, then there exists a unique solution* $\mathbf{N}(t)$ *to the initial value problem on that interval.*

6. Optimal Control

In this section, we address the minimization of S^j (stressed population) and Hb^j (HbA1c levels) in the context of managing prediabetic risk and preventing complications related to diabetes. Elevated stress levels and high HbA1c levels are associated with deteriorating metabolic health and increased predisposition to chronic diseases. Therefore, reducing these levels is crucial for improving overall health and preventing serious conditions.

To achieve this minimization, we employ two distinct controls: u_1 and u_2.
The control u_1 specifically targets stress management. This control includes interventions such as relaxation techniques, psychological support, and stress management strategies aimed at reducing the stressed population (S^j). Effective stress management is essential for mitigating its negative effects on metabolic health and thus reducing HbA1c levels[34].

The control u_2 directly targets the reduction of HbA1c levels. This involves interventions that improve glucose metabolism and regulation, such as specific dietary modifications, physical exercise programs, and appropriate medical treatments. These measures aim to lower HbA1c (Hb^j) by enhancing glucose management and promoting better metabolic health[35].

By combining these controls, we aim to optimize the reduction of both stress levels and HbA1c. Stress management through u_1 helps to mitigate the negative impacts of chronic stress, while u_2 directly improves metabolism and glucose levels. This integrated approach supports optimal health and reduces the risk of metabolic complications, facilitating effective prevention of diabetes-related disorders. Thus, the controlled mathematical system is given by the

following system (2) of differential equations:

$$
(2)\qquad
\begin{cases}
\dfrac{dH^j}{dt} = I^j - (\beta_1^j + \beta_2^j + \mu^j)H^j + u_1^j S^j + v^j Hb^j, \\[2ex]
\dfrac{dS^j}{dt} = \beta_1^j H^j - (\mu^j + \delta_1^j + \alpha_1^j + \alpha_5^j)S^j - \varepsilon_1^j u_1^j S^j, \\[2ex]
\dfrac{dHb^j}{dt} = \beta_2^j H^j + \alpha_1^j S^j - (\mu^j + \alpha_2^j + \alpha_6^j)Hb^j - \varepsilon_2^j v^j Hb^j, \\[2ex]
\dfrac{dP^j}{dt} = \alpha_2^j Hb^j - (\mu^j + \alpha_3^j)P^j, \\[2ex]
\dfrac{dD^j}{dt} = \alpha_3^j P^j - (\mu^j + \alpha_4^j)D^j, \\[2ex]
\dfrac{dC^j}{dt} = \alpha_6^j Hb^j + \alpha_5^j S^j + \alpha_4^j D^j - (\mu^j + \delta_2^j)C^j.
\end{cases}
$$

where $Hb^j(0) \geq 0$ $S^j(0) \geq 0$, $Hb^j(0) \geq 0$, $P^j(0) \geq 0$, $D^j(0) \geq 0$, $C^j(0) \geq 0$ are given initial states, and $N^j = H^j + S^j + Hb^j + P^j + D^j + C^j$ for all $j \in \{1,2,3\}$.

The objective is to compare the costs of these interventions and their effectiveness in managing stress. To do this, we need to determine the optimal level of effort required to control stress effectively. For this purpose, we use the objective function.

$$
J(u^j, v^j) = S^j(T) + Hb^j(T) - H^j(T) + \int_0^T \left(S^j(t) + Hb^j(t) - H^j(t) + E^j \frac{1}{2}(u^j(t))^2 + F^j \frac{1}{2}(v^j(t))^2 \right) dt
$$

where the parameters $E^j > 0$ and $F^j > 0$, for all $j \in \{1,2,3\}$, are the cost coefficients at time t, and T is the final time. In other words, we seek the optimal controls u^j* and v^j* such that:

$$
J(u^j*, v^j*) = \min_{(u^j, v^j) \in U_{ad}} J(u^j, v^j)
$$

where U_{ad} is the set of admissible controls defined by

$$
U_{ad} = \{(u^j, v^j) : 0 \leq u^j(t) \leq 1; 0 \leq v^j(t) \leq 1, t \in [0,T]\}.
$$

6.1. The Optimal Control Existence and Characterization. We first show the existence of solutions to the system (2). After that, we will prove the existence of the optimal control [36].

6.1.1. *Existence of an Optimal Control.*

Theorem 4. *Subject to the control system* (2) *with initial conditions, there exist optimal controls* u^j*, v^j* *such that*

$$J(u^j*, v^j*) = \min_{(u^j, v^j) \in U_{ad}} J(u^j, v^j)$$

if the following conditions are met:

(1) The set of controls and corresponding state variables is nonempty.

(2) The control set U_{ad} *is convex and closed.*

(3) The right-hand side of the state system is bounded by a linear function in the state and control variables.

(4) The integrand,

$$L(S^j, Hb^j, H^j, u^j, v^j) = S^j(t) + Hb^j(t) - H^j(t)E^j\frac{1}{2}(u^j(t))^2 + F^j\frac{1}{2}(v^j(t))^2$$

of the objective functional is convex on U_{ad}*, and there exist constants* κ_1 *and* κ_2 *such that*

$$L(S^j, Hb^j, H^j, u^j, v^j) \geq -\kappa_1 + \kappa_2(|u^j|^2 + |v^j|^2).$$

Proof. The existence of the optimal control can be obtained using a result by Fleming and Rishel [37], checking the following steps:

Step 1: It follows that the set of controls and corresponding state variables is nonempty. In Diprima and Elementary [38], to prove that the set of controls and the corresponding state variables is nonempty, we will use a simplified version of an existence result [38].

Let $X_i = F_{X_i}(t, X_1, X_2, X_3, X_4, X_5, X_6)$ with $i = 1, 2, 3, 4, 5, 6$, where $(X_1, X_2, X_3, X_4, X_5, X_6) = (H^j, S^j, Hb^j, P^j, D^j, C^j)$, where X_1, X_2, X_3, X_4, X_5 and X_6 are from the right-hand side of the equations of system (2). Let u and v be some constants, and since all parameters are constants and X_1, X_2, X_3, X_4, X_5 and X_6 are continuous, then F_H, F_S, F_Hb, F_P, F_D, and F_C are also continuous.

Additionally, the partial derivatives $\frac{\partial F_{X_i}}{\partial X_i}$ with $i = 1, 2, 3, 4, 5, 6$ are all continuous. Therefore,

there exists a unique solution $(H^j, S^j, Hb^j, P^j, D^j, C^j)$ that satisfies the initial conditions. Hence, the set of controls and the corresponding state variables is nonempty, and the condition is satisfied.

Step 2: U_{ad} is convex and closed by definition. Take any controls (u_1^j, u_2^j) and $(v_1^j, v_2^j) \in U_{ad}$ and $\lambda \in [0,1]$. Then $0 \leq \lambda u_i^j + (1-\lambda)v_i^j$. Additionally, we observe that $\lambda u_i^j \leq \lambda$ and $(1-\lambda)v_i^j \leq (1-\lambda)$. Then $\lambda u_i^j + (1-\lambda)v_i^j \leq 1$, hence $0 \leq \lambda u_i^j + (1-\lambda)v_i^j \leq 1$, for $i = 1,2$. The control space $U_{ad} = \{(u_j, v_j) : 0 \leq u_j(t) \leq 1; 0 \leq v_j(t) \leq 1, t \in [0,T]\}$ is measurable.

Step 3: All the right-hand sides of the equations of system (2) are continuous, bounded above by a sum of bounded control and state, and can be written as a linear function of u and v with coefficients depending on time and state. From the system of differential equations (2),

$$\frac{dN^j}{dt} \leq I^j - \mu^j N^j \implies \limsup_{t\to\infty} N^j(t) \leq \frac{I^j}{\mu^j}$$

Therefore, all solutions of the system (2) are bounded. So, there exist positive constants $Z_1, Z_2, Z_3, Z_4, Z_5 Z_6$ such that $\forall t \in [t_0, t_f]$:

$$H^j(t) \leq Z_1, \quad S^j(t) \leq Z_2, \quad Hb^j(t) \leq Z_3, \quad P^j(t) \leq Z_4, \quad D^j(t) \leq Z_5, \quad \text{and} \quad C^j(t) \leq Z_6.$$

We consider

$$\begin{aligned}
F_H &\leq I^j + u^j S^j + v^j Hb^j \\
F_S &\leq \beta_1^j H^j - \varepsilon_1^j u^j S^j \\
F_{Hb} &\leq \beta_2^j H^j + \alpha_1^j S^j - \varepsilon_2^j v^j Hb^j \\
F_P &\leq \alpha_2^j Hb^j \\
F_D &\leq \alpha_3^j P^j \\
F_C &\leq \alpha_6^j Hb^j + \alpha_5^j S^j + \alpha_4^j D^j
\end{aligned}$$

So we can rewrite system (2) in matrix form as

$$F(t, H^j, S^j, Hb^j P^j, D^j, C^j) \leq I^j + A_j X_j(t) - B_j U_j(t)$$

where

$$F(t,H^j,S^j,Hb^j,P^j,D^j,C^j) = \begin{bmatrix} F_H \\ F_S \\ F_H b \\ F_P \\ F_D \\ F_C \end{bmatrix}^T, \quad I^j = \begin{bmatrix} I^j \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}^T,$$

$$X_j(t) = \begin{bmatrix} H^j \\ S^j \\ Hb^j \\ P^j \\ D^j \\ C^j \end{bmatrix}^T, \quad U_j(t) = \begin{bmatrix} u^j \\ v^j \end{bmatrix}^T,$$

$$A_j = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_1^j & 0 & 0 & 0 & 0 & \\ \beta_2^j & \alpha_1^j & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_2^j & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_3^j & 0 & 0 \\ 0 & \alpha_5^j & \alpha_6^j & 0 & \alpha_4^j & 0 \end{pmatrix},$$

and

$$B_j = \begin{pmatrix} S^j & Hb^j \\ -\varepsilon_1^j S^j & 0 \\ 0 & -\varepsilon_2^j Hb^j \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{pmatrix}.$$

It gives a linear function of the control vector and state variable vector. Therefore, we can write

$$F(t,H^j,S^j,Hb^j,P^j,D^j,C^j) \leq I^j + A^j X^j(t) + B^j U^j(t) \leq \phi^j + \psi^j(X^j(t) + U^j(t)),$$

where $\Sigma_j \leq \phi_j$ and $\psi_j = \max(A_j, B_j)$.

Hence, we see the right-hand side is bounded by a sum of state and control vectors. Therefore, condition 3 is satisfied.

Step 4:

$$J(u_j, v_j) = S^j(T) + Hb^j(T) - H^j(T) + \int_0^T \left(S^j(t) + Hb^j(t) - H^j(t) + E_j(u_j(t))^2 + F_j(v_j(t))^2\right) dt$$

is clearly connex in U_{ad}.

We conclude that there exists an optimal control For more details, see [39].

6.2. Characterization of the Optimal Control. In order to derive the necessary condition for the optimal control, we apply Pontryagin's Maximum Principle [36].

The idea is to introduce the adjoint function to attach the system of differential equations to the objective functional, resulting in the formation of a function called the Hamiltonian. This principle converts into a problem of minimizing the Hamiltonian $HA^j(t)$ at time t, defined by:

$$HA^j(t) = S^j(t) + Hb^j(t) - H^j(t) + E_j \frac{1}{2}(u_j(t))^2 + F_{2j}(v_j(t))^2 + \sum_{k=1}^{6} \lambda_k^j(t) f_k(H^j, S^j, Hb^j, P^j, D^j, C^j),$$

where f_k is the right side of the system of differential equations (2) for the k-th state variable at time t.

Theorem 5. Given the optimal controls u^j*, v^j* and the solutions $H^j* S^j*$, Hb^j*, P^j*, D^j*, and C^j* of the corresponding state system (2), there exist adjoint variables λ_1^j, λ_2^j, λ_3^j, λ_4^j, and λ_5^j, λ_6^j satisfying:

$$\begin{aligned}
\dot{\lambda}_1^j &= -\frac{\partial HA^j}{\partial H^j} = \lambda_1^j(\beta_1 + \beta_2^j + \mu) - \lambda_2^j \beta_1^j - \beta_2^j \lambda_3^j, \\
\dot{\lambda}_2^j &= -\frac{\partial HA^j}{\partial S^j} = (\mu^j + \delta_1^j + \alpha_1^j + \alpha_5^j)\lambda_2^j + \varepsilon_1^j u^j \lambda_2^j - u^j \lambda_1^j - \alpha_1^j \lambda_3^j - \alpha_5 \lambda_6^j, \\
\dot{\lambda}_3^j &= -\frac{\partial HA^j}{\partial Hb^j} = (\mu^j + \alpha_2^j + \alpha_6^j)\lambda_3^j + \varepsilon_2^j v^j \lambda_3^j - \alpha_2^j \lambda_4^j - \lambda_1^j v^j - \lambda_6^j \alpha_6^j, \\
\dot{\lambda}_4^j &= -\frac{\partial HA^j}{\partial P^j} = (\mu^j + \alpha_3^j)\lambda_4^j - \alpha_3^j \lambda_5^j, \\
\dot{\lambda}_5^j &= -\frac{\partial HA^j}{\partial D^j} = (\mu^j + \alpha_4^j)\lambda_5^j - \alpha_4 \lambda_6,
\end{aligned}$$

$$\dot{\lambda}_6^j = -\frac{\partial HA^j}{\partial C^j} = (\mu^j + \delta_2^j)\lambda_6^j,$$

with the transversality conditions at time T:

$$\lambda_1^j(T) = -1,$$

$$\lambda_2^j(T) = 1,$$

$$\lambda_3^j(T) = 1,$$

$$\lambda_4^j(T) = 0,$$

$$\lambda_5^j(T) = 0,$$

$$\lambda_6^j(T) = 0,$$

Furthermore, for $t \in [0,T]$, the optimal controls u^j* and v^j* are given by:

$$u^{j*}(t) = \min\left(1, \max\left(0, \frac{\lambda_1^j - \varepsilon_1^j \lambda_2^j}{E^j}\right)\right) \tag{3}$$

$$v^{j*}(t) = \min\left(1, \max\left(0, \frac{\lambda_1^j - \varepsilon_2^j \lambda_3^j}{F^j}\right)\right) \tag{4}$$

Proof

The Hamiltonian at time t is given by:

$$HA^j(t) = S^j(t) + Hb^j(t) - H^j(t) + \frac{E^j}{2}(u^j(t))^2 + \frac{F^j}{2}(v^j(t))^2 + \sum_{k=1}^{6} \lambda_k^j(t) f_k(H^j, S^j, Hb^j, P^j, D^j, C^j)$$

Where

$$f_1(H^j, S^j, Hb^j, P^j, D^j, C^j) = I^j - (\beta_1^j + \beta_2^j + \mu^j)H^j,$$

$$f_2(H^j, S^j, Hb^j, P^j, D^j, C^j) = \beta_1^j H^j - (\mu^j + \delta_1^j + \alpha_1^j + \alpha_5^j)S^j,$$

$$f_3(H^j, S^j, Hb^j, P^j, D^j, C^j) = \beta_2^j H^j + \alpha_1^j S^j - (\mu^j + \alpha_2^j + \alpha_6^j)Hb^j,$$

$$f_4(H^j, S^j, Hb^j, P^j, D^j, C^j) = \alpha_2^j Hb^j - (\mu^j + \alpha_3^j)P^j,$$

$$f_5(H^j, S^j, Hb^j, P^j, D^j, C^j) = \alpha_3^j P^j - (\mu^j + \alpha_4^j)D^j,$$

$$f_6(H^j, S^j, Hb^j, P^j, D^j, C^j) = \alpha_6^j Hb^j + \alpha_5^j S^j + \alpha_4^j D^j - (\mu^j + \delta_2^j)C^j.$$

For $t \in [0,T]$, the adjoint equations and transversality conditions can be obtained by using Pontryagin's Maximum Principle, such that:

$$\frac{d\lambda_i(t)}{dt} = -\frac{\partial H}{\partial x_i}, \quad i = 1,2,\ldots,6,$$

with the transversality conditions, for more details, see [40, 41].

$$\lambda_i(T) = \frac{\partial \phi}{\partial x_i}(x(T)), \quad i = 1,2,\ldots,6.$$

For $t \in [0,T]$, the optimal controls u_j^* and v_j^* can be solved from the optimality condition:

$$\frac{dH_j}{du_j} = 0 \quad \text{and} \quad \frac{dH_j}{dv_j} = 0,$$

So we have

$$u^j(t) = \frac{\lambda_1{}^j - \varepsilon_1^j \lambda_2{}^j}{E^j}.$$

$$v^j(t) = \frac{\lambda_1{}^j - \varepsilon_2^j \lambda_3{}^j}{F^j}$$

by the bounds in U_{ad} of the controls, it easy to obtain u^j* and v^j* in the form (3), (4).

7. NUMERICAL SIMULATION

7.1. Algorithm. In this formulation, there were initial conditions for the state variables and terminal conditions for the adjoints. That is, the optimality system is a two-point boundary value problem with separated boundary conditions at time steps $t = 0$ and $t = T$. We solve the optimality system by an iterative method with forward solving of the state system followed by backward solving of the adjoint system. We start with an initial guess for the controls at the first iteration and then, before the next iteration, we update the controls by using the characterization. We continue until convergence of successive iterates is achieved. A code is written and compiled in MATLAB using the following data (Tables 1–3).

I^1	H_0^1	S_0^1	Hb_0^1	P_0^1	D_0^1	C_0^1
1000	2000	900	230	120	90	50

α_3^1	μ^1	δ_1^1	α_4^1	α_5^1	α_6^1	β_1^1	β_2^1	δ_2^1	α_1^1	α_2^1
0.006	0.3	0.01	0.07	0.02	0.1	0.5	0.4	0.01	0.5	0.4

TABLE 1. The description of the parameters used for the definition of discrete-time systems of age group between 18-25 years.

I^2	H_0^2	S_0^2	Hb_0^2	P_0^2	D_0^2	C_0^2	α_1^2	α_2^2
1100	6000	2050	1000	300	2000	150	30	0.01

α_3^2	μ^2	δ_1^2	α_4^2	α_5^2	α_6^2	β_1^2	β_2^2	δ_2^2
0.001	0.3	0.01	0.3	0.044	0.035	0.5	0.4	0.22

TABLE 2. The description of the parameters used for the definition of discrete-time systems of age group 45-66 years.

I^3	H_0^3	S_0^3	Hb_0^3	P_0^3	D_0^3	C_0^3	α_1^3	α_2^3
1000	4000	3200	2200	1400	1000	900	0.004	0.01

α_3^3	μ^3	δ_1^3	α_4^3	α_5^3	α_6^3	β_1^3	β_2^3	δ_2^3
0.001	0.3	0.01	0.41	0.0043	0.012	0.5	0.4	0.66

TABLE 3. The description of the parameters used for the definition of discrete-time systems of age group uper of 70 years.

7.2. Discussion.

7.2.1. *The result without control.* The results of our compartmental model, analyzed over a three-month period, show distinct changes in different populations based on stress, HbA1c levels, prediabetes, diabetes, and cardiovascular complications.

Stressed Population (S): An increase in stress is clearly observed in individuals over the age of 70. This increase is directly linked to factors such as social isolation, increased financial concerns, and physical and cognitive health problems that often accompany aging. The age

group of 45 to 66 also shows a slight increase in stress, caused by significant professional and personal responsibilities, as well as life transitions, such as preparing for retirement. In contrast, young adults (18-25 years) show a decrease in stress, which can be attributed to greater flexibility in their lifestyle, higher resilience, and active social networks that facilitate stress management [42].

Populations with Elevated HbA1c Levels, Prediabetics, and Diabetics: There is a marked increase in HbA1c levels, cases of prediabetes, and diabetes, particularly among individuals over the age of 70 [24]. This increase is the direct result of a combination of chronic stress, age-related metabolic changes, and reduced physical activity, which together promote the development of metabolic disorders[42]. For individuals aged 45 to 66, the increase is also significant and is directly associated with increased intellectual stress and less healthy lifestyles, including poor diet and a sedentary lifestyle. Among young adults (18-25 years), although the increase in cases of prediabetes and diabetes is less pronounced, it is clearly linked to poor dietary habits, such as frequent consumption of fast foods and unbalanced nutrition[43].

Population with Cardiovascular Complications: The increase in cardiovascular complications is particularly notable in individuals over the age of 70. This rise is caused by the accumulation of risk factors such as hypertension, long-standing diabetes, and the effects of chronic stress on the cardiovascular system [44]. In people aged 45 to 66, cardiovascular complications also increase, influenced by stress, diabetes, and a less active lifestyle. In contrast, among young adults, the increase in cardiovascular complications is very limited, which can be explained by overall better cardiovascular health and shorter exposure to risk factors[44].

Conclusion: These results clearly indicate that stress, HbA1c levels, prediabetes, diabetes, and cardiovascular complications vary significantly based on age and health behaviors. Older individuals are the most affected by these risk factors, highlighting the importance of targeted prevention strategies for this age group. While young adults are less affected by these complications in the short term, they are already showing signs of increased risk due to poor dietary choices. Therefore, early interventions are necessary to promote healthy lifestyle behaviors and prevent progression to pathological states. Public health efforts must be tailored to the specific needs of each age group to maximize their effectiveness.

Population / Age	**H**	**S**	**Hb1aC**	**Pr**	**D**	**C**
18-25	370	1636,21	1454	1653	1090	3719
45-66	222,22	3810,82	2414	1964	1658	10644
$>$70	799,22	4210,33	2867	3391	2260	13579

TABLE 4. Without Control

7.2.2. ***The result with control***. After obtaining the initial results from our compartmental model, we developed two intervention strategies to improve the health of the different populations identified in the study. The first strategy u focuses on stress management through family support and yoga practice, while the second strategy v aims to promote healthy eating and good hygiene. Here is the analysis of the results following the implementation of these strategies across different populations:

- **Increase in the Healthy Population:**[45, 46] Strategies u and v led to a notable increase in the healthy population. This increase is the most pronounced among young adults (18-25 years), followed by the 45-66 age group, and finally, individuals over 70. The rapid improvement in young adults can be attributed to their greater adaptability and ability to integrate positive lifestyle changes. The 45-66 age group also shows a significant increase in health, likely due to their willingness to adopt stress management practices like yoga and improve their diet. Older adults, while showing improvements, exhibit a lesser increase in the healthy population, possibly due to physical limitations and chronic illnesses associated with aging.
- **Decrease in the Stressed Population:**[44, 45] A significant reduction in stress is observed across all age groups, with the most substantial decrease among young adults, followed by the 45-66 age group, and finally, individuals over 70. The reduction in stress among young adults and middle-aged individuals is largely due to the effectiveness of stress management practices such as yoga and increased social support. Among older adults, the reduction in stress is less pronounced but still notable, which can be attributed to improved stress management through family support.

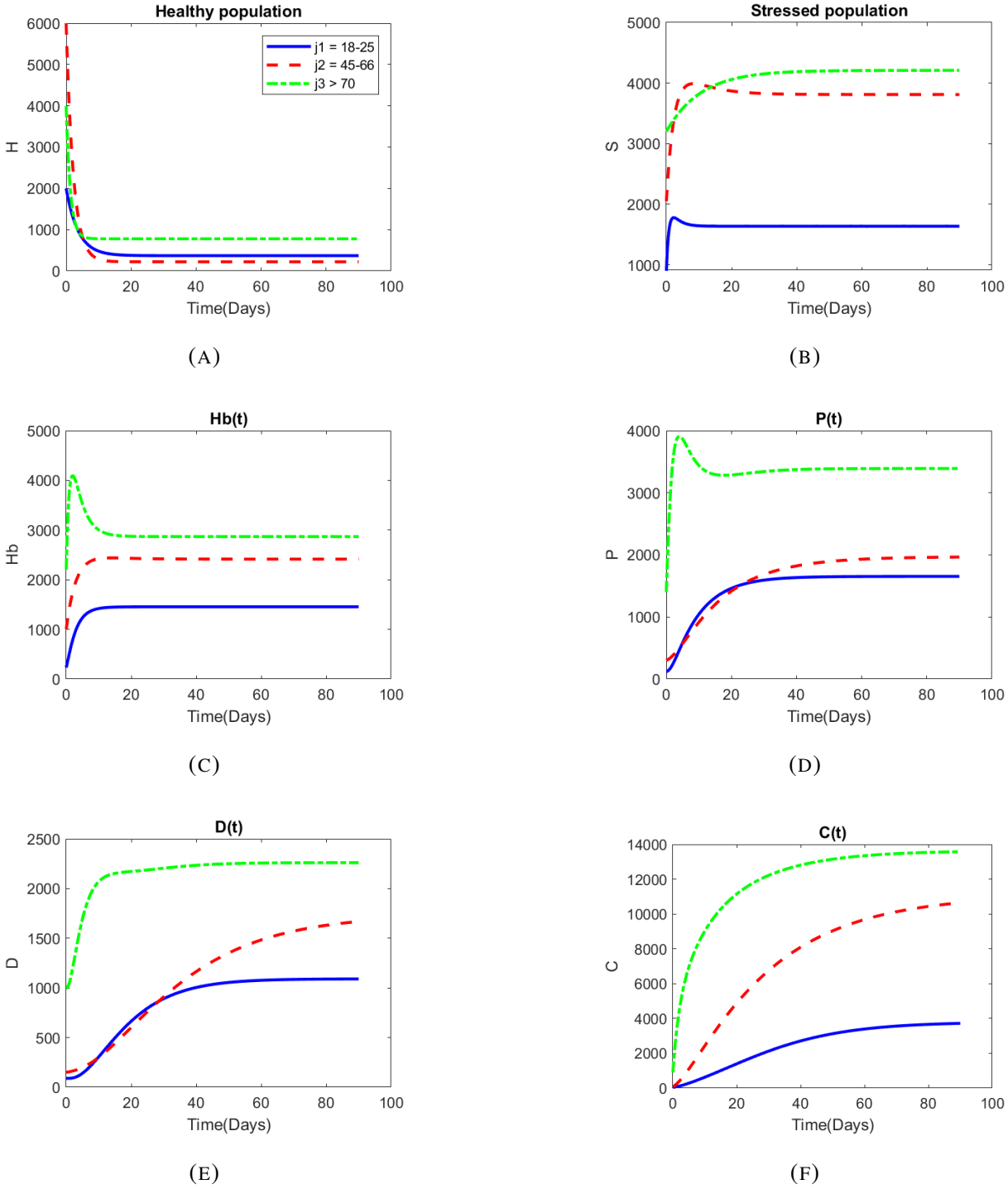

FIGURE 2. The evolution of the population for all age groups without control

- **Decrease in Elevated HbA1c Levels and Prediabetes Cases:**[47, 48, 49] HbA1c levels decreased in all age categories, with a more marked reduction among young adults and middle-aged individuals (45-66 years). This suggests that improvements in diet

and the adoption of a healthier lifestyle have had a positive impact on blood glucose regulation. Among older adults, the decrease in HbA1c levels and prediabetes cases is less pronounced, possibly due to age-related factors such as reduced physical activity and the presence of chronic diseases. However, a slight improvement is still observable, indicating that the intervention strategies have a beneficial effect even in this age group.

- **Reduction in Diabetes Cases:**[50] Diabetes cases have decreased across all age categories, particularly among young adults and middle-aged individuals. The significant reduction in these groups may be linked to better stress management and improvements in diet and lifestyle. For older adults, while the decrease is more moderate, it remains significant, demonstrating that even interventions at an advanced age can have positive effects on diabetes management.
- **Reduction in Cardiovascular Complications:**[51, 52, 53, 54] Cardiovascular complications have also decreased, especially among young adults and middle-aged individuals, due to better diet, increased physical activity, and reduced stress. Older adults show a more modest reduction in cardiovascular complications, which may be due to existing comorbidities and the natural progression of cardiovascular diseases with age. Nonetheless, even a modest reduction indicates that the intervention strategies are beneficial for improving cardiovascular health across all age groups.

Population / Age	**H**	**S**	**Hb**	**Pr**	**D**	**C**
18-25	4166,67	150,37	89,04	271,06	92,51	498,03
45-66	3030,31	384,72	125,42	200,69	403,44	438,38
>70	2222	649,35	763,27	1696,76	426,05	2214,57

TABLE 5. with control

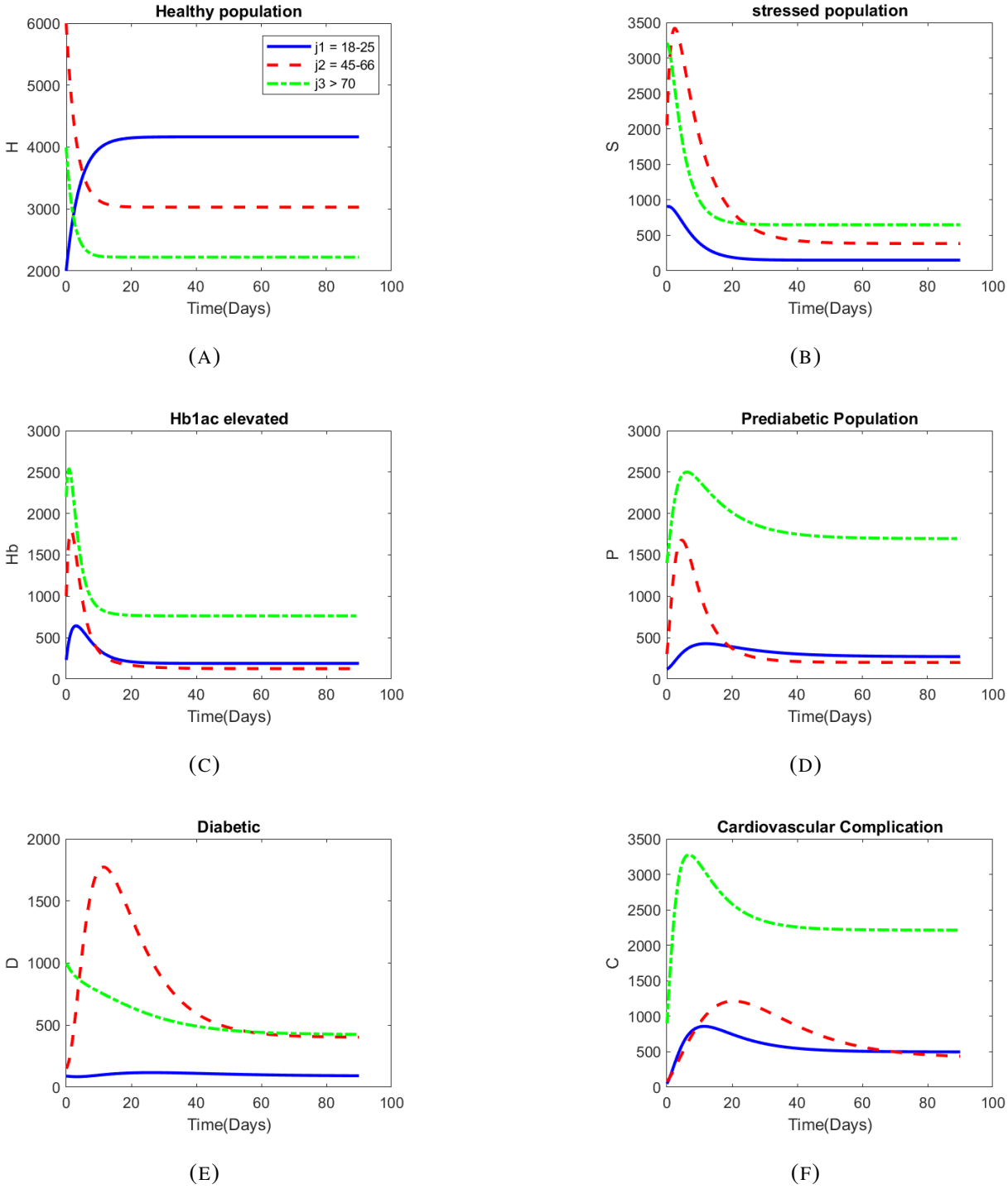

FIGURE 3. The evolution of the population of for all age groups with control u and v

8. Conclusion

Age has a significant impact on several major aspects of physical and psychological health. This work focuses specifically on how aging influences stress, glucose metabolism, and cardiovascular complications.

Regarding stress, aging is associated with complex psychological and biological changes that may influence emotional regulation and resilience. For the biological system, various changes occur with aging, particularly concerning cortisol, adrenaline, and noradrenaline. Cortisol dysregulation generally includes reduced stability of the circadian system; baseline cortisol levels may become higher and, most importantly, recovery after stress becomes slower. These different changes mean that the body remains in a prolonged "state of alert", which leads to chronic fatigue, sleep disturbances, irritability, and chronic inflammation. All these troubles lead undoubtedly to an increase in chronic stress. Psychologically, while some older adults develop better coping mechanisms through life experience, the majority faces increased vulnerability to chronic stress due to factors such as social isolation, loss of loved ones, reduced physical autonomy, financial concerns, and chronic illness.

At the same time, age is strongly associated with progressive alterations in glucose metabolism. Glycated hemoglobin (HbA1c) levels tend to increase with age, even in non-diabetic individuals, due to decreased insulin sensitivity, pancreatic beta-cell dysfunction characterized by a reduction in the mass of these cells, an altered insulin secretory response to glucose, oxidative stress, and amyloid deposits in the islets of Langerhans. Furthermore, the adipose tissue of older individuals exhibits increased secretion of pro-inflammatory cytokines such as tumor necrosis factor α (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP), which disrupt insulin signaling pathways by inhibiting the activity of the insulin receptor substrate and reducing glucose uptake by the GLUT-4 transporter in skeletal muscle. Age is therefore a factor in the chronic increase in blood glucose levels leading to prediabetes and then diabetes.

At the cardiac level, aging is associated with progressive arterial stiffening due to collagen deposition, elastin fragmentation, vascular calcification, and endothelial dysfunction, leading to increased systolic blood pressure and cardiac afterload. Endothelial nitric oxide production declines with age, impairing vasodilation and promoting a pro-inflammatory and pro-thrombotic

vascular environment. Concurrently, chronic low-grade inflammation (inflammaging), oxidative stress, and mitochondrial dysfunction contribute to the acceleration of atherosclerosis and vascular damage. At the myocardial level, aging induces progressive left ventricular hypertrophy, increased myocardial fibrosis, decreased diastolic relaxation, and reduced cardiac compliance. Furthermore, coronary artery disease becomes increasingly common with age due to cumulative endothelial damage, lipid deposition, chronic inflammation, and plaque formation in coronary vessels.

In summary, aging significantly influences psychological, metabolic, and cardiovascular health, collectively contributing to increased morbidity in older adults. This effect underscores the importance of comprehensive medical and psychosocial care for aging populations.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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