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Optimal Control Strategies for Mitigating Cardiovascular and Type 2 Diabetes Risks in Psoriasis and Psoriatic Arthritis: A Mathematical Model and Numerical Simulations

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ABSTRACT: In this study, we investigate a novel mathematical model that describes the dynamics of psoriasis and psoriatic arthritis. The goal of this paper is to mitigate the risks associated with psoriasis and psoriatic arthritis on cardiovascular health and type 2 diabetes through control strategies involving three variables: awareness programs for patients with psoriasis or psoriatic arthritis, medical follow-up, and promoting a healthy and regular lifestyle. We demonstrate the existence of optimal controls and describe them using states and adjoint functions, primarily based on Pontryagin's maximum principle. Numerical simulations for various scenarios validate the effectiveness of the optimization approach.

Key Words: Psoriasis, psoriatic arthritis, type 2 diabetes, cardiovascular health, mathematical model, optimal controls, pontryagin maximum principle, numerical simulations.

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1. Introduction

Psoriasis is a chronic autoimmune skin disease that manifests as thick, red, scaly patches [1,2]. These lesions, often itchy, can appear on various parts of the body, including the elbows, knees, scalp, and lower back [3]. The exact causes of psoriasis are not fully understood, but they involve a combination of genetic and environmental factors, as well as an abnormal immune response. Triggers can include stress, infections, certain medications, and skin trauma. The consequences of psoriasis go beyond skin symptoms, potentially affecting patients' quality of life, causing pain, and being associated with other conditions such as psoriatic arthritis, cardiovascular diseases, and mental health disorders [4]. The World Health Organization (WHO) [1] estimates that approximately $2-3\%$ of the global population is affected by psoriasis, amounting to over 125 million people.

Psoriatic arthritis is a type of inflammatory arthritis [1]. This condition is characterized by joint inflammation, causing pain, stiffness, and swelling, mainly in the fingers, toes, knees, and ankles [5]. Similar to psoriasis, an abnormal immune response is central to the development of psoriatic arthritis. The consequences of this disease can be severe, including permanent joint damage, reduced mobility, and

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significant impairment of quality of life [3]. Around 30% of individuals with psoriasis will go on to develop psoriatic arthritis [1]. According to the National Psoriasis Foundation [1], this disease affects about 2% of the global population, corresponding to around 30 million people.

Type 2 diabetes is a chronic disease characterized by insulin resistance and insufficient insulin production by the pancreas, leading to persistent hyperglycemia. The main causes include a sedentary lifestyle, a diet high in calories and carbohydrates, obesity, and genetic factors. The consequences of this disease can be severe, including cardiovascular complications, nerve damage (neuropathy), kidney problems (nephropathy), and vision disorders (retinopathy) [6]. The World Health Organization (WHO) reports that over 420 million people globally are living with diabetes, with the vast majority of cases being type 2 diabetes [7].

Cardiovascular diseases (CVD) include a variety of conditions that impact the heart and circulatory system, including coronary artery disease, strokes, hypertension, and heart failure. The main causes of these diseases include an unbalanced diet, smoking, heavy alcohol use and insufficient physical activity, as well as genetic factors and underlying medical conditions like diabetes and hypercholesterolemia [8]. The consequences of CVD can be severe, including heart attacks, strokes, decreased quality of life, and an increased risk of premature death. The World Health Organization (WHO) states that cardiovascular diseases are the leading cause of death worldwide, responsible for about 17.9 million fatalities annually [9].

People with psoriasis and psoriatic arthritis often exhibit elevated levels of pro-inflammatory cytokines, which significantly contribute to the development of cardiovascular diseases [10,11]. Additionally, patients with severe psoriasis face a 57% higher risk of cardiovascular mortality compared to the general population [12]. This correlation underscores the importance of holistic and proactive management of patients with psoriasis and psoriatic arthritis to reduce their cardiovascular risk [13,14].

There is a significant relationship between psoriasis, psoriatic arthritis, and type 2 diabetes, mainly due to systemic inflammation, common risk factors, and metabolic mechanisms [15]. Patients with psoriatic arthritis (PsA) face a significantly greater risk of developing type 2 diabetes compared to both the general population and those with psoriasis. The adjusted relative risk was 1.40 compared to the general population and 1.53 compared to patients with psoriasis [16,17].

Recent studies on the mathematical modeling of psoriasis have provided significant insights into the disease's pathogenesis and potential treatment strategies. One notable approach is the development of models that simulate the interactions between various immune cells and keratinocytes, which are central to the formation and progression of psoriatic lesions. For instance, a study published in Frontiers in Immunology [18] focuses on the role of IL-36 cytokines, using a network model to depict interactions among T-lymphocytes, dendritic cells, macrophages, and keratinocytes. This model employs ordinary differential equations (ODEs) to capture the dynamics of cell populations and their responses to IL-36, providing a framework to understand cytokine-driven inflammation in psoriasis.

A study by P. K. Roy and A. Datta [19] explored the control of cytokine release in the treatment of psoriasis. They developed a model that incorporates the effects of cytokines on keratinocyte proliferation and used optimal control theory to determine the best strategies for drug administration. Their model shows how negative feedback can be used to regulate the effects of cytokines, potentially leading to more effective treatments.

This article presents a detailed mathematical model to examine the effects of psoriasis and psoriatic arthritis on cardiovascular risk and type 2 diabetes. We design strategies to minimize these associated complications using advanced optimal control techniques. This work offers a new perspective and practical tools for clinicians and researchers seeking to improve the management of these complex patients. This article is structured as detailed below :

• Section 2: We introduce our mathematical model $SP_sP_{sa}D_2C_aR$, which outlines the interactions between the different variables and the underlying mechanisms influencing the development of cardiovascular complications and type 2 diabetes among individuals with psoriasis and psoriatic arthritis.

• Section 3: Devoted to the discussion of fundamental properties, positivity and boundedness of solution, and the existence and uniqueness of solution.

• Section 4: We establish the optimal control problems for the proposed model, setting clear objectives to minimize cardiovascular complications and type 2 diabetes in patients. We present findings on the existence of optimal controls and describe them using the Pontryagin maximum principle.

• Section 5: Includes numerical simulations performed with Matlab, illustrating the effectiveness of the suggested control strategies. We analyze the simulation results to evaluate the impact of the optimal controls on the trajectories of the model variables and on the reduction of complications.

• Section 6: The conclusion summarizes the main contributions of the article and discusses the practical implications of the results obtained. We highlight the benefits of optimal control strategies for the care of individuals with psoriasis and psoriatic arthritis.

2. The Mathematical Model Formulation

The presented model aims to describe the dynamics of psoriasis and arthritis psoriasis (AP), as well as their associated complications, including cardiovascular problems and type 2 diabetes. The model uses a compartmental structure inspired by the classical epidemiological SEIR to capture transitions between different health conditions within the population.
Let N represent the size of the population. The compartment diagram is shown in 1:

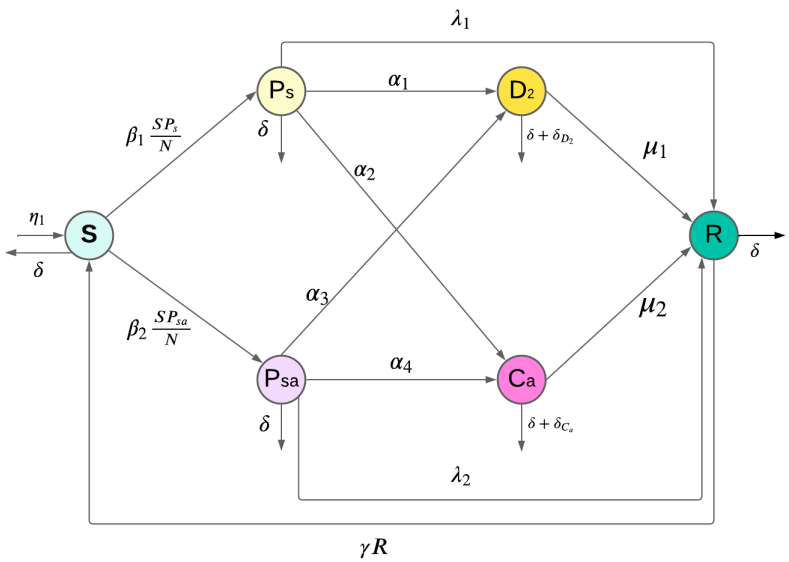

Figure 1: Schematic diagram for Psoriasis, Psoriatic Arthritis, and their effects on Cardiovascular Health and Type 2 Diabetes

• **Compartment** S : This compartment represents the population susceptible to developing psoriasis or psoriatic arthritis. The number of susceptible individuals increases due to the recruitment rate of new births η_1 and the return of recovered individuals $\gamma R(t)$. Conversely, the susceptible population decreases due to infection rates: $\beta_1 \frac{S(t)P_s(t)}{N(t)}$ for psoriasis and $\beta_2 \frac{S(t)P_{sa}(t)}{N(t)}$ for psoriatic arthritis. Moreover, individuals leave this compartment at a constant mortality rate δ, adding to the overall natural mortality rate δS.
The equation that represents this compartment is as follows:

$$\frac{dS(t)}{dt} = \eta_1 - \beta_1 \frac{S(t)P_s(t)}{N(t)} - \beta_2 \frac{S(t)P_{sa}(t)}{N(t)} - \delta S(t) + \gamma R(t)$$

• **Compartment** P_s : This compartment quantifies the number of individuals who have developed psoriasis. The population in this compartment grows due to the infection rate $\beta_1 \frac{S(t)P_s(t)}{N(t)}$. It diminishes through various rates: δP_s for natural mortality, $\alpha_1 P_s$ the rate at which people infected with psoriasis develop cardiovascular complications, $\alpha_2 P_s$ the rate at which people infected with psoriasis develop type 2 diabetes complications, and $\lambda_1 P_s$ the recovery rate of those infected with psoriasis.
Hence, the equation that represents this compartment is as follows:

$$\frac{dP_s(t)}{dt} = \beta_1 \frac{S(t)P_s(t)}{N(t)} - (\alpha_1 + \alpha_2 + \lambda_1 + \delta)P_s(t)$$

.

• **Compartment** P_{sa} : This compartment represents the population of individuals who have developed psoriatic arthritis. The number of people in this compartment increases through the infection rate $\beta_2 \frac{S(t)P_{sa}(t)}{N(t)}$. Conversely, it decreases due to several factors: natural mortality δP_{sa}, the rate at which people infected with psoriatic arthritis develop cardiovascular complications $\alpha_3 P_{sa}$, the rate at which people infected with psoriatic arthritis develop type 2 diabetes complications $\alpha_3 P_{sa}$, and the recovery rate of those infected with psoriatic arthritis $\lambda_2 P_{sa}$.
Hence, the equation that represents this compartment is as follows:

$$\frac{dP_{sa}(t)}{dt} = \beta_2 \frac{S(t)P_{sa}(t)}{N(t)} - (\alpha_3 + \alpha_4 + \lambda_2 + \delta)P_{sa}(t)$$

• **Compartment** D_2 : This compartment captures the number of individuals who have developed cardiovascular complications as a result of psoriasis or psoriatic arthritis. The population in this compartment increases through the rates $\alpha_1 P_s$ and $\alpha_3 P_{sa}$, while it decreases due to the recovery rate of individuals with cardiovascular complications $\mu_1 D_2$, natural mortality δ, and mortality specifically due to cardiovascular problems δ_{D_2}.
So, the equation that represents this compartment is as follows:

$$\frac{dD_2(t)}{dt} = \alpha_1 P_s(t) + \alpha_3 P_{sa}(t) - (\mu_1 + \delta + \delta_{D_2})D_2(t)$$

.

• **Compartment** C_a : This compartment accounts for the individuals who have developed type 2 diabetes complications as a consequence of psoriasis or psoriatic arthritis. The population in this compartment increases due to the rates $\alpha_2 P_s$ and $\alpha_4 P_{sa}$, while it decreases due to the recovery rate of individuals with type 2 diabetes complications $\mu_2 C_a$, natural mortality δ, and mortality specifically due to type 2 diabetes problems δ_{C_a}.
So, the equation that represents this compartment is as follows:

$$\frac{dC_a(t)}{dt} = \alpha_2 P_s(t) + \alpha_4 P_{sa}(t) - (\mu_2 + \delta + \delta_{C_a})C_a(t)$$

.

• **Compartment** R : This compartment represents the count of individuals who have achieved recovery. The population in this compartment increases through recovery rates $\lambda_1 P_s$, $\lambda_2 P_{sa}$, $\mu_1 D_2$ and $\mu_2 C_a$. It decreases due to the return rate γR and natural mortality rate δR.
Thus, the equation that represents this compartment is as follows:

$$\frac{dR(t)}{dt} = \lambda_1 P_s(t) + \mu_1 D_2(t) + \mu_2 C_a(t) + \lambda_2 P_{sa}(t) - (\gamma + \delta)R(t)$$

.

Lastly, we introduce the Prosiaris mathematical model, which is described using this system of differ-

ential equations:

$$\begin{cases} \dfrac{dS(t)}{dt} = \eta_1 - \beta_1 \dfrac{S(t)P_s(t)}{N(t)} - \beta_2 \dfrac{S(t)P_{sa}(t)}{N(t)} - \delta S(t) + \gamma R(t) \\ \dfrac{dP_s(t)}{dt} = \beta_1 \dfrac{S(t)P_s(t)}{N(t)} - (\alpha_1 + \alpha_2 + \lambda_1 + \delta)P_s(t) \\ \dfrac{dP_{sa}(t)}{dt} = \beta_2 \dfrac{S(t)P_{sa}(t)}{N(t)} - (\alpha_3 + \alpha_4 + \lambda_2 + \delta)P_{sa}(t) \\ \dfrac{dD_2(t)}{dt} = \alpha_1 P_s(t) + \alpha_3 P_{sa}(t) - (\mu_1 + \delta + \delta_{D_2})D_2(t) \\ \dfrac{dC_a(t)}{dt} = \alpha_2 P_s(t) + \alpha_4 P_{sa}(t) - (\mu_2 + \delta + \delta_{C_a})C_a(t) \\ \dfrac{dR(t)}{dt} = \lambda_1 P_s(t) + \mu_1 D_2(t) + \mu_2 C_a(t) + \lambda_2 P_{sa}(t) - (\gamma + \delta)R(t) \end{cases} \tag{2.1}$$

With

$$S(0) > 0, P_s(0) > 0, P_{sa}(0) > 0, D_2(0) > 0, C_a(0) > 0, R(0) > 0$$

$$S(t) + P_s(t) + P_{sa}(t) + D_2(t) + C_a(t) + R(t) = N(t) \qquad , \qquad t \in [0, T]$$

The details of parameters are shown in table 1 and table 2 :

Table 1: Variables and description

Variable	description
$S(t)$	Number of susceptible
$P_s(t)$	Number of affected by psoriasis
$P_{sa}(t)$	Number of affected by psoriatic arthritis
$D_2(t)$	Number of developed cardiovascular problems
$C_a(t)$	Number of developed type 2 diabetes complications
$R(t)$	Number of recovered

Table 2: Parameters and description

Parameters	description
β_1	Rate of progression of those susceptible to psoriasis
β_2	Rate of progression of those susceptible to psoriasis arthritis
α_1	Rate of cardiovascular complications for those infected with psoriasis
α_2	Rate of diabetic complications for those infected with psoriasis
α_3	Rates of cardiovascular complications for those infected with psoriasis and arthritis
α_4	Rates of diabetic complications for those infected with psoriasis arthritis
λ_1	Recovery rate of those infected with psoriasis
λ_2	Recovery rate of those infected with psoriasis arthritis
μ_1	Recovery rate of individuals with cardiovascular complications
μ_2	Recovery rate of individuals with complications related to type 2 diabetes
δ	Natural mortality rate
γ	Rate of loss of immunity of those who have recovered
η_1	New births
$N(t)$	Total population at time t

3. Model Analysis

3.1. The solution's positivity

Theorem 3.1 *If $S(0)$, $P_s(0)$, $P_{sa}(0)$, $D_2(0)$, $C_a(0)$ and $R(0)$ are positive. Then $S(t)$, $P_s(t)$, $P_{sa}(t)$, $D_2(t)$, $C_a(t)$ and $R(t)$ are positive $\forall t > 0$.*

Proof:

Let us define

$$t' = sup\,\{t \geq 0, S(0) > 0, P_s(0) > 0, P_{sa}(0) > 0, D_2(0) > 0, C_a(0) > 0, R(0) > 0\}$$

The positivity of the initial condition and the continuity of the state variable guarantee that $t' > 0$.
• If $t' = +\infty$ then the positivity hold.
• If $0 < t' < +\infty$ then $S(t') = 0$ or $P_s(t') = 0$ or $P_{sa}(t') = 0$ or $D_2(t') = 0$ or
$C_a(t') = 0$ or $R(t') = 0$

We have

$$\frac{dS(t)}{dt} = -\beta_1 \frac{S(t)P_s(t)}{N(t)} - \beta_2 \frac{S(t)P_{sa}(t)}{N(t)} - \delta S(t) + \gamma R(t) + \eta_1$$

$$\frac{dS(t)}{dt} + \left(\beta_1 \frac{P_s(t)}{N(t)} + \beta_2 \frac{P_{sa}(t)}{N(t)} + \delta\right) S(t) = \eta_1 + \gamma R(t)$$

$$\frac{dS(t)}{dt} + \left(\beta_1 \frac{P_s(t)}{N(t)} + \beta_2 \frac{P_{sa}(t)}{N(t)} + \delta\right) S(t) \geq \gamma R(t)$$

Let

$$L(t) = \beta_1 \frac{P_s(t)}{N(t)} + \beta_2 \frac{P_{sa}(t)}{N(t)}$$

We multiply by

$$\exp\left(\int_0^{t'} L(s)ds\right)$$

We achieve

$$\frac{dS(t)}{dt} \times \exp\left(\int_0^{t'} L(s)ds\right) + L(t) \times \exp\left(\int_0^{t'} L(s)ds\right) \times S(t) \geq \gamma R(t) \times \exp\left(\int_0^{t'} L(s)ds\right)$$

$$\frac{d}{dt}\left[S(t) \times \exp\left(\int_0^{t'} L(s)ds\right)\right] \geq \gamma R(t) \times \exp\left(\int_0^{t'} L(s)ds\right)$$

Let integrate this inequality from 0 to t' , we obtain

$$S(t') \times \exp\left(\int_0^{t'} L(s)ds\right) - S(0) \geq \int_0^{t'} \gamma R(t) \times \exp\left(\int_0^{t'} L(s)ds\right)$$

$$S(t') \times \exp\left(\int_0^{t'} L(s)ds\right) \geq S(0) + \int_0^{t'} \gamma R(t) \times \exp\left(\int_0^{t'} L(s)ds\right)$$

$$S(t') \geq S(0) \times \exp\left(\int_0^{t'} L(s)ds\right) + \exp\left(\int_0^{t'} L(s)ds\right) \times \int_0^{t'} \gamma R(t) \times \exp\left(\int_0^{t'} L(s)ds\right)$$

for the meaning of t', $R(0) > 0$ for all $t \in [0, t']$. Then $S(t') > 0$, hence $S(t') \neq 0$.
So $S(t)$ is positive.

We have

$$\frac{dP_s(t)}{dt} = (\beta_1 \frac{S(t)}{N(t)} - \alpha_1 - \alpha_2 - \lambda_1 - \delta)P_s(t)$$

Let

$$L_1(t) = -\beta_1 \frac{S(t)P_s(t)}{N(t)} + \alpha_1 + \alpha_2 + \lambda_1 + \delta$$

Then

$$\frac{dP_s(t)}{dt} = -L_1(t)P_s(t)$$

By multiplying with

$$\exp\left(\int_0^t L_1(s)ds\right)$$

We obtain

$$\frac{dP_s(t)}{dt} \times \exp\left(\int_0^t L_1(s)ds\right) = -L_1(t) \times \exp\left(\int_0^t L_1(s)ds\right) \times P_s(t)$$

$$\frac{dP_s(t)}{dt} \times \exp\left(\int_0^t L_1(s)ds\right) + L_1(t) \times \exp\left(\int_0^t L_1(s)ds\right) \times P_s(t) = 0$$

$$\frac{d}{dt}\left[P_s(t) \times \exp\left(\int_0^t L_1(s)ds\right)\right] = 0$$

$$P_s(t) \times \exp\left(\int_0^t L_1(s)ds\right) - P_s(0) = 0$$

$$P_s(t) = P_s(0) \times \exp\left(\int_0^t -L_1(s)ds\right) \geq 0$$

So $P_s(t)$ is positive.

We have

$$\frac{dD_2(t)}{dt} = \alpha_1 P_s(t) + \alpha_3 P_{sa}(t) - (\mu_1 + \delta + \delta_{D_2})D_2(t)$$

Let

$$\alpha(t) = \alpha_1 P_s(t) + \alpha_3 P_{sa}(t) \qquad \text{and} \qquad L_2(t) = \mu_1 + \delta + \delta_{D_2}$$

Then

$$\frac{dD_2(t)}{dt} = \alpha(t) - L_2(t)D_2(t)$$

By multiplying with

$$\exp\left(\int_0^t L_2(s)ds\right)$$

We obtain

$$\frac{dD_2(t)}{dt} \times \exp\left(\int_0^t L_2(s)ds\right) = \alpha(t) \times \exp\left(\int_0^t L_2(s)ds\right) - L_2(s) \times \exp\left(\int_0^t L_2(s)ds\right) \times D_2(t)$$

$$\frac{dD_2(t)}{dt} \times \exp\left(\int_0^t L_2(s)ds\right) + L_2(s) \times \exp\left(\int_0^t L_2(s)ds\right) \times D_2(t) = \alpha(t) \times \exp\left(\int_0^t L_2(s)ds\right)$$

$$\frac{d}{dt}\left[D_2(t) \times \exp\left(\int_0^t L_2(s)ds\right)\right] = \alpha(t) \times \int_0^t \left(\exp\left(\int_0^w L_2(s)ds\right)\right) dw$$

$$D_2(t) \times \exp\left(\int_0^t L_2(s)ds\right) - D_2(0) = \alpha(t) \times \int_0^t \left(\exp\left(\int_0^w L_2(s)ds\right)\right) dw$$

$$D_2(t) = \left[D_2(0) + \alpha(t) \int_0^t \left(\exp\left(\int_0^w L_2(s)ds\right)\right) dw\right] \times \exp\left(\int_0^t -L_2(s)ds\right) \geq 0$$

So $D_2(t)$ is positive.

By processing in the same way, we find that P_{sa} and C_a are positive.
Therefore, we can see that $S(0)$, $P_s(0)$, $P_{sa}(0)$, $D_2(0)$, $C_a(0)$ et $R(0)$ are positive, for all $t > 0$ and this concludes the proof. □

3.2. Boundedness of the solution

Lemma 3.1 *All solutions of the system equations are bounded by:*

$$\left\{\Omega = (S, P_s, P_{sa}, D_2, C_a, R)) \in \mathbb{R}_+^6; S + P_s + P_{sa} + D_2 + C_a + R \leq \frac{\eta_1}{\delta}\right\}$$

Proof: We have

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dP_s(t)}{dt} + \frac{dP_{sa}(t)}{dt} + \frac{dD_2(t)}{dt} + \frac{dC_a(t)}{dt} + \frac{dR(t)}{dt}$$

$$\frac{dN(t)}{dt} = \eta_1 - \delta S(t) - \delta P_s(t) - \delta P_{sa}(t) - \delta D_2(t) - \delta C_a(t) - \delta R(t)$$

$$\frac{dN(t)}{dt} = \eta_1 - \delta N(t)$$

$$\frac{dN(t)}{\eta_1 - \delta N(t)} = dt \Longrightarrow \int_0^t \frac{dN(t)}{\eta_1 - \delta N(t)} = \int_0^t dt$$

$$\Longrightarrow -\left[\frac{-1}{\delta}\log\left(\eta_1 - \delta N(t)\right)\right]_0^t = t$$

$$\Longrightarrow \frac{-1}{\delta}\left[\log(\eta_1 - \delta N(t)) - \log(\eta_1 - \delta N(0))\right] = t$$

$$\Longrightarrow \log\left(\frac{\eta_1 - \delta N(t)}{\eta_1 - \delta N(0)}\right) = -\delta t$$

$$\Longrightarrow \frac{\eta_1 - \delta N(t)}{\eta_1 - \delta N(0)} = \exp(-\delta t)$$

$$\Longrightarrow \eta_1 - \delta N(t) = (\eta_1 - \delta N(0))\exp(-\delta t)$$

$$\Longrightarrow N(t) = \frac{\eta_1}{\delta} + \left(N(0) - \frac{\eta_1}{\delta}\right)\exp(-\delta t)$$

$$\Longrightarrow \limsup_{t\to+\infty} N(t) = \frac{\eta_1}{\delta}$$

$$\Longrightarrow N(t) \leq \frac{\eta_1}{\delta}$$

Then

$$S(t) + P_s(t) + P_{sa}(t) + D_2(t) + C_a(t) + R(t) \leq \frac{\eta_1}{\delta}$$

□

3.3. The existence and uniqueness of the solution

Theorem 3.2 *For all $S(0), P_s(0), P_{sa}(0), D_2(0), C_a(0), R(0)$ there exist a unique solution of the system given.*

Proof: Let $X(t) = \begin{pmatrix} S(t) \\ P_s(t) \\ P_{sa}(t) \\ D_2(t) \\ C_a(t) \\ R(t) \end{pmatrix}$ and $\psi(X) = \begin{pmatrix} \frac{dS(t)}{dt} \\ \frac{dP_s(t)}{dt} \\ \frac{dP_{sa}(t)}{dt} \\ \frac{dD_2(t)}{dt} \\ \frac{dC_a(t)}{dt} \\ \frac{dR(t)}{dt} \end{pmatrix}$

The system can be writing as follow

$$\psi(X) = MX + J(X)$$

Where

$$M = \begin{pmatrix} \delta & 0 & 0 & 0 & 0 & \gamma \\ 0 & -(\alpha_1+\alpha_2+\lambda_1+\delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\alpha_3+\alpha_4+\lambda_2+\delta) & 0 & 0 & 0 \\ 0 & \alpha_1 & \alpha_3 & -(\mu_1+\delta_{D_2}+\delta) & 0 & 0 \\ 0 & \alpha_2 & \alpha_4 & 0 & -(\mu_2+\delta_{C_a}+\delta) & 0 \\ 0 & \lambda_1 & \lambda_2 & \mu_1 & \mu_2 & -(\gamma+\delta) \end{pmatrix}$$

and

$$J(X) = \begin{pmatrix} \eta_1 - \beta_1 \frac{S(t)P_s(t)}{N(t)} - \beta_2 \frac{S(t)P_{sa}(t)}{N(t)} \\ \beta_1 \frac{S(t)P_s(t)}{N(t)} \\ \beta_2 \frac{S(t)P_{sa}(t)}{N(t)} \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

On the other hand, we have

$$|J(X_1) - J(X_2)| = 2\left|\beta_1 \frac{S_1(t)P_{s1}(t)}{N} + \beta_2 \frac{S_2(t)P_{sa1}(t)}{N} - \beta_1 \frac{S_2(t)P_{s2}(t)}{N} - \beta_2 \frac{S_2(t)P_{sa2}(t)}{N}\right|$$

$$= 2\left|\beta_1 \frac{S_1(t)P_{s1}(t)}{N} - \beta_1 \frac{S_2(t)P_{s1}(t)}{N} + \beta_1 \frac{S_2(t)P_{s1}(t)}{N} + \beta_2 \frac{S_1(t)P_{sa1}(t)}{N}\right.$$
$$\left. + \beta_2 \frac{S_1(t)P_{sa1}(t)}{N} - \beta_2 \frac{S_2(t)P_{sa1}(t)}{N} + \beta_2 \frac{S_2(t)P_{sa1}(t)}{N} - \beta_1 \frac{S_2(t)P_{s2}(t)}{N} - \beta_2 \frac{S_2(t)P_{sa2}(t)}{N}\right|$$

$$\leq 2\left(\left|\beta_1 \frac{P_{s1}(t)}{N}\right| |S_1(t) - S_2(t)| + \left|\beta_2 \frac{P_{sa1}(t)}{N}\right| |S_1(t) - S_2(t)| + \left|\beta_1 \frac{S_2(t)}{N}\right| |P_{s1}(t) - P_{s2}(t)|\right.$$
$$\left. + \left|\beta_2 \frac{S_2(t)}{N}\right| |P_{sa1}(t) - P_{sa2}(t)|\right)$$

$$\leq 2\left(\frac{\eta_1}{\delta}\left(\left|\frac{\beta_1}{N}\right| + \left|\frac{\beta_2}{N}\right|\right) |S_1(t) - S_2(t)| + \left|\frac{\beta_1}{N}\right| |P_{s1}(t) - P_{s2}(t)| + \left|\frac{\beta_2}{N}\right| |P_{sa1}(t) - P_{sa2}(t)|\right)$$

$$\leq K\,|X_1 - X_2|$$

Where

$$K = 2\frac{\eta_1}{\delta}\left(\left|\frac{\beta_1}{N}\right| + \left|\frac{\beta_2}{N}\right|, \left|\frac{\beta_1}{N}\right|, \left|\frac{\beta_2}{N}\right|\right)$$

Then

$$\|J(X_1) - J(X_2)\| \leq C\|X_1 - X_2\|$$

Where

$$C = \max(K, \|M\|) < \infty$$

Thus, it follows that the function ψ is a lipschitz continous. So the system admit a unique solution. □

4. The Problem of Optimal Control

In this section, we outline our optimal control problem, examine the existence of the optimal control, and subsequently identify the control terms.
To minimize the impact of cardiovascular disease and diabetic complications in people who have psoriasis and arthrite psoriasic, we propose three control strategies:

• First, (control u_1) patient education and awareness will help to recognize early signs of complications and adopt appropriate preventive measures, thereby enhancing the overall effectiveness of interventions.

• Second, (control u_2) regular medical follow-up will allow proactive management of psoriasis, arthritis prosiasic and early treatment of their effects such as cardiovascular problems and diabetes 2 complications, facilitating continuous monitoring and treatment adjustments based on individual needs.

• Third, (control u_3) embracing a wellness-oriented lifestyle, which involves a nutritious eating plan, consistent physical activity, and effective stress alleviation techniques, will contribute to better overall health and mitigate risk factors for cardiovascular disease and type 2 diabete.

$$\begin{cases} \dfrac{dS(t)}{dt} = \eta_1 - \beta_1(1-u_1(t))\dfrac{S(t)P_s(t)}{N(t)} - \beta_2(1-u_1(t))\dfrac{S(t)P_{sa}(t)}{N(t)} - \delta S(t) + \gamma R(t) \\ \dfrac{dP_s(t)}{dt} = \beta_1(1-u_1(t))\dfrac{S(t)P_s(t)}{N(t)} - (\lambda_1+\delta)P_s(t) - \alpha_1(1+u_2(t))P_s(t) - \alpha_2(1+u_2(t))P_s(t) \\ \dfrac{dP_{sa}(t)}{dt} = \beta_2(1-u_1(t))\dfrac{S(t)P_{sa}(t)}{N(t)} - (\lambda_2+\delta)P_{sa}(t) - \alpha_3(1+u_2(t))P_{sa}(t) - \alpha_4(1+u_2(t))P_{sa}(t) \\ \dfrac{dD_2(t)}{dt} = \alpha_1(1+u_2(t))P_s(t) + \alpha_3(1+u_2(t))P_{sa}(t) - (\mu_1+\delta+\delta_{D_2})D_2(t) - u_3(t)D_2(t) \\ \dfrac{dC_a(t)}{dt} = \alpha_2(1+u_2(t))P_s(t) + \alpha_4(1+u_2(t))P_{sa}(t) - (\mu_1+\delta+\delta_{C_a})C_a(t) - u_3(t)C_a(t) \\ \dfrac{dR(t)}{dt} = \lambda_1 P_s(t) + (\mu_1+u_3(t))D_2(t) + (\mu_2+u_3(t))C_a(t) + \lambda_2 P_{sa}(t) - (\gamma+\delta)R(t) \end{cases} \tag{4.1}$$

4.1. The optimal control problem

To achieve our purpose of reducing the impact of psoriasis, psoriasis arthritis on health, we must optimize the following objective function :

$$J(u_1,u_2,u_3) = D_2(T) + C_a(T) + \int_0^T \left(D_2(t) + C_a(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{C}{2}u_3^2(t) \right) dt$$

Where $A \geq 0, B \geq 0, C \geq 0$ are the cost coefficient chosen to prioritize the relative importance of $u_1(t), u_2(t)$ and $u_3(t)$ at time t; T is the final time.
In simpler terms, we aim to determine the optimal controls $u_1^*(t), u_2^*(t)$ and $u_3^*(t)$ such that

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

where U is the set of admissible control defined by

$$U = \left\{ (u_1,u_2,u_3) \;\middle|\; \begin{array}{l} 0 \leq u_{1,\min}(t) \leq u_1(t) \leq u_{1,\max}(t) \leq 1, \\ 0 \leq u_{2,\min}(t) \leq u_2(t) \leq u_{2,\max}(t) \leq 1, \\ 0 \leq u_{3,\min}(t) \leq u_3(t) \leq u_{3,\max}(t) \leq 1, \\ t \in [0,T] \end{array} \right\}$$

4.2. Existence of the optimal control

Theorem 4.1 *Considered the control problem with system given, there exist an optimal control* $(u_1^*,u_2^*,u_3^*) \in U^3$ *such that* $J(u_1^*,u_2^*,u_3^*) = \min_{u_1,u_2,u_3 \in U} J(u_1,u_2,u_3)$.

Proof: The existence of the optimal control can be confirmed using a methodology inspired by Fleming and Rishel [20]. The procedure is outlined as follows:

• The control and state variable set is nonempty. To verify this, we employ a simplified approach derived from the work of Boyce and DiPrima [21].
Consider the system: $Z_i' = F_{Zi}(t, Z_1, Z_2, Z_3, Z_4, Z_5, Z_6)$ with $i = 1, ..., 6$, where

$$((Z_1', Z_2', Z_3', Z_4', Z_5', Z_6') = (S, P_s, P_{sa}, D_2, C_a, R))$$

$Z_1, Z_2, Z_3, Z_4, Z_5, Z_6$ represent the expressions on the right-hand side of the system of equations.
Let u_1, u_2, u_3 be constants. Since all parameters are constants and $Z_1, Z_2, Z_3, Z_4, Z_5, Z_6$ are continuous, it follows that $F_S, F_{P_s}, F_{P_{sa}}, F_{D_2}, F_{C_a}$ and F_R are also continuous.
Moreover, the partial derivatives $\frac{\partial F_{Zi}}{\partial Z_i}$ are all continuous. Thus, there exist a unique solution $(S, P_s, P_{sa}, D_2, C_a, R)$ that satisfies the initial conditions. Therefore, the set is nonempty and the condition is met.

• The control space $U = \{(u_1, u_2, u_3) \mid 0 \leq u_{1,\min}(t) \leq u_1(t) \leq u_{1,\max}(t) \leq 1,\ 0 \leq u_{2,\min}(t) \leq u_2(t) \leq u_{2,\max}(t) \leq 1, 0 \leq u_{3,\min}(t) \leq u_3(t) \leq u_{3,\max}(t) \leq 1, t \in [0, T]\}$ is, by definition, a convex and closed set.
• All the right-hand sides of the system of equations are continuous and bounded above by a sum involving bounded control and state variables. They can be expressed as linear functions of $u_1, u_2,$ and u_3 with coefficients that depend on time and state.
• The objective functional integrand $D_2(t)+C_a(t)+\frac{A}{2}u_1^2(t)+\frac{B}{2}u_2^2(t)+\frac{C}{2}u_3^2(t)$ is evidently convex on U.

• It remains to demonstrate that there exist constants $\xi_1, \xi_2, \xi_3, \xi_4 > 0$ and ξ such that $D_2(t)+C_a(t)+\frac{A}{2}u_1^2(t)+\frac{B}{2}u_2^2(t)+\frac{C}{2}u_3^2(t)$ fulfills

$$D_2(t) + C_a(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{C}{2}u_3^2(t) \geq \xi_1 + \xi_2|u|^\xi + \xi_3|v|^\xi + \xi_4|w|^\xi$$

Therefore, we conclude that an optimal control exists, as established by Fleming and Rishel [20]. □

4.3. Characterization of the optimal control

Prior to characterizing the optimal control, we define the Hamiltonian for the optimal control problem as follows:

$$H(t) = D_2(t) + C_a(t) + \frac{A}{2}u_1^2(t) + \frac{B}{2}u_2^2(t) + \frac{C}{2}u_3^2(t) + \sum_{i=1}^{6}\theta_i(t)f_i(S, P_s, P_{sa}, D_2, C_a, R)$$

Where f_i represents the right-hand side of the difference equation for the i^{th} state variable at time t.

$$f_1(S, P_s, P_{sa}, D_2, C_a, R) = \eta_1 - \beta_1(1 - u_1(t))\frac{S(t)P_s(t)}{N(t)} - \beta_2(1 - u_1(t))\frac{S(t)P_{sa}(t)}{N(t)} - \delta S(t) + \gamma R(t),$$

$$f_2(S, P_s, P_{sa}, D_2, C_a, R) = \beta_1(1 - u_1(t))\frac{S(t)P_s(t)}{N(t)} - (\lambda_1 + \delta)P_s(t) - \alpha_1(1 + u_2(t))P_s(t) + \alpha_2(1 + u_2(t))P_s(t),$$

$$f_3(S, P_s, P_{sa}, D_2, C_a, R) = \beta_2(1 - u_1(t))\frac{S(t)P_{sa}(t)}{N(t)} - (\lambda_2 + \delta)P_{sa}(t) - \alpha_3(1 + u_2(t))P_{sa}(t) + \alpha_4(1 + u_2(t))P_{sa}(t),$$

$$f_4(S, P_s, P_{sa}, D_2, C_a, R) = \alpha_1(1 + u_2(t))P_s(t) + \alpha_3(1 + u_2(t))P_{sa}(t) - (\mu_1 + \delta + \delta_{D_2})D_2(t) - u_3(t)D_2(t),$$

$$f_5(S, P_s, P_{sa}, D_2, C_a, R) = \alpha_2(1 + u_2(t))P_s(t) + \alpha_4(1 + u_2(t))P_{sa}(t) - (\mu_1 + \delta + \delta_{C_a})C_a(t) - u_3(t)C_a(t),$$

$$f_6(S, P_s, P_{sa}, D_2, C_a, R) = \lambda_1 P_s(t) + (\mu_1 + u_3(t))D_2(t) + (\mu_2 + u_3(t))C_a(t) + \lambda_2 P_{sa}(t) - (\gamma + \delta)R(t).$$

Then by applying Pontryagin's maximum principle [22] we obtain

$$\begin{cases} \theta_1'(t) &= -\dfrac{\partial H}{\partial S} \\ \theta_2'(t) &= -\dfrac{\partial H}{\partial P_s} \\ \theta_3'(t) &= -\dfrac{\partial H}{\partial P_{sa}} \\ \theta_4'(t) &= -\dfrac{\partial H}{\partial D_2} \\ \theta_5'(t) &= -\dfrac{\partial H}{\partial C_a} \\ \theta_6'(t) &= -\dfrac{\partial H}{\partial R} \end{cases}$$

$$\begin{cases} \theta_1'(t) &= \theta_1(t)\left[\beta_1\dfrac{P_s}{N}(1-u_1)+\beta_2\dfrac{P_{sa}}{N}(1-u_1)+\delta\right]-\theta_2(t)\left[\beta_1\dfrac{P_s}{N}(1-u_1)\right]-\theta_3(t)\left[\beta_2\dfrac{P_{sa}}{N}(1-u_1)\right] \\ \theta_2'(t) &= \theta_1(t)\left[\beta_1\dfrac{S}{N}(1-u_1)\right]-\theta_2(t)\left[\beta_1\tfrac{S}{N}(1-u_1)-(\lambda_1+\delta)-\alpha_1(1+u_1)-\alpha_2(1+u_2)\right] \\ & \quad -\theta_4(t)\left[\alpha_1(1+u_2)\right]-\theta_5(t)\left[\alpha_2(1+u_2)\right]-\lambda_1\theta_6(t) \\ \theta_3'(t) &= \theta_1(t)\left[\beta_2\tfrac{S}{N}(1-u_1)\right]-\theta_3(t)\left[\beta_2\dfrac{S}{N}(1-u_1)-(\lambda_2+\delta)-\alpha_3(1+u_2)-\alpha_4(1+u_2)\right] \\ & \quad -\theta_4(t)\left[\alpha_3(1+u_2)\right]+\theta_5(t)\left[\alpha_4(1+u_2)\right]-\lambda_2\theta_6(t) \\ \theta_4'(t) &= -1+\theta_4(t)\left[\mu_1+\delta+\delta_{D_2}+u_3\right]-\theta_6(t)\left[\mu_1+u_3\right] \\ \theta_5'(t) &= -1+\theta_5(t)\left[\mu_1+\delta+\delta_{C_a}+u_3\right]-\theta_6(t)\left[\mu_2+u_3\right] \\ \theta_6'(t) &= -\gamma\theta_1(t)+\left[\gamma+\delta\right]\theta_6(t) \end{cases}$$

The transversality conditions at time T

$$\begin{cases} \theta_1(T) &= 0 \\ \theta_2(T) &= 0 \\ \theta_3(T) &= 0 \\ \theta_4(T) &= 1 \\ \theta_5(T) &= 1 \\ \theta_6(T) &= 0 \end{cases}$$

For $t \in [0,T]$, the optimal control u_1^*, u_2^* and u_3^* can be derived from the optimality conditions, which are :

$$\frac{\partial H}{\partial u_1} = Au_1^* + \theta_1(t)\left[\beta_1\frac{SP_s}{N} + \beta_2\frac{SP_{sa}}{N}\right] + \theta_2(t)\left[-\beta_1\frac{SP_s}{N}\right] + \theta_3(t)\left[-\beta_2\frac{SP_{sa}}{N}\right] = 0$$

$$\frac{\partial H}{\partial u_2} = Bu_2^* + \theta_2(t)\left[-\alpha_1 P_s - \alpha_2 P_s\right] + \theta_3(t)\left[-\alpha_3 P_{sa} - \alpha_4 P_{sa}\right] + \theta_4(t)\left[\alpha_1 P_s + \alpha_3 P_{sa}\right] + \theta_5(t)\left[\alpha_2 P_s + \alpha_4 P_{sa}\right] = 0$$

$$\frac{\partial H}{\partial u_3} = Cu_3^* - \theta_4(t)D_2 - \theta_5(t)C_a + \theta_6(t)\left[D_2 + C_a\right] = 0$$

Then we have

$$\begin{cases} u_1^* &= \left(\dfrac{\theta_2 - \theta_1}{A}\right)\beta_1\dfrac{SP_s}{N} + \left(\dfrac{\theta_3 - \theta_1}{A}\right)\beta_2\dfrac{SP_{sa}}{N} \\ u_2^* &= \left(\dfrac{\theta_2 - \theta_4}{B}\right)\alpha_1 P_s + \left(\dfrac{\theta_2 - \theta_5}{B}\right)\alpha_2 P_s + \left(\dfrac{\theta_3 - \theta_4}{B}\right)\alpha_3 P_{sa} + \left(\dfrac{\theta_3 - \theta_5}{B}\right)\alpha_4 P_{sa} \\ u_3^* &= \left(\dfrac{\theta_4 - \theta_6}{C}\right)D_2 + \left(\dfrac{\theta_5 - \theta_6}{C}\right)C_a \end{cases}$$

5. Numerical Simulation

In this section, we address the numerical resolution of the optimal control problem associated with our $SP_sP_{sa}D_2C_aR$ model. The process begins with the derivation of the optimality system, which is based on the state and adjoint equations. Our approach to optimal control is realized by solving this system, which consists of six differential equations coupled with boundary conditions. The solution is obtained through an iterative procedure.

Starting with an initial estimate for the control variables $u_1(t)$, $u_2(t)$, and $u_3(t)$, we first solve the state variables forward in time. Concurrently, we solve the adjoint variables θ_i (for $i = 1, 2, 3, 4, 5$) in reverse, from the initial time t_0 to the final time t_f. If the new state and adjoint variables differ from the previous iteration, the control variables $u_1(t)$, $u_2(t)$, and $u_3(t)$ are updated accordingly. This iterative process continues until the system converges.

Finally, we provide a series of numerical simulations to demonstrate our theoretical findings, utilizing the controlled system with the given parameter values.

Table 3: Parameter values

Parameter	**Value**	**Parameter**	**Value**
β_1	0.8	β_2	0.6
α_1	0.8	α_2	0.7
α_3	0.4	α_4	0.2
λ_1	0.1	λ_2	0.1
μ_1	0.1	μ_2	0.1
δ	0.1	δ_{D2}	0.1
δ_{Ca}	0.1	γ	0.1
η_1	0.1		

The control strategies outlined in this study are designed to accomplish multiple objectives:

First strategy : Raising awareness and sensitizing people with psoriasis and prosiasis arthris about the risk of developing type 2 diabetes or cardiovascular diseases.
To implement this strategy, we apply only control u_1, which involves the implementation of educational and awareness initiatives for individuals with psoriasis and psoriatic arthritis to inform them about the health risks associated with these conditions.

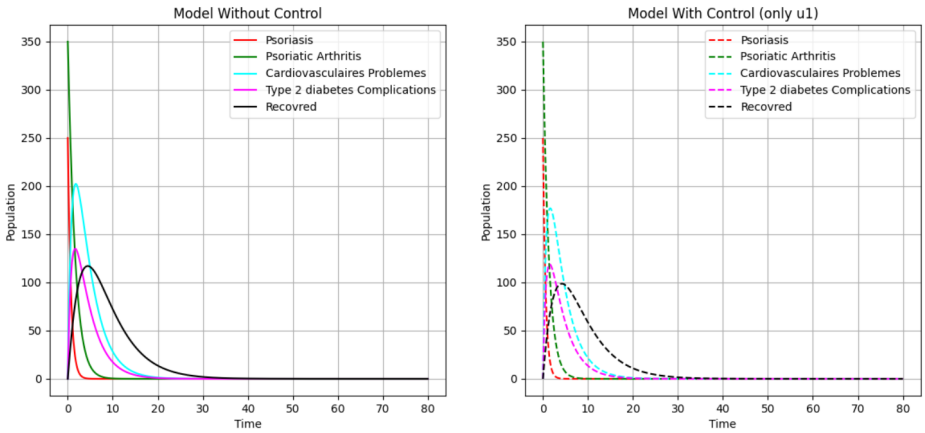

Figure 2: Evaluation of the Model with Control u_1 Versus Without Control

Cases of cardiovascular problems and type 2 diabetes complications reach around 200 and 130 respectively without control, but with control, they remain below 175 and 120 respectively. These figures clearly demonstrate the positive impact of control u_1 on reducing infections and progression to more severe states.

Second strategy : Reducing the risk of type 2 diabetes complication in people with psoriasis and psoriasis arthris by a regular medical follow-up Strategy 2 involves applying only control u_2, which focuses on regular medical follow-up. This strategy aims to proactively manage psoriasis and psoriatic arthritis by monitoring patients consistently and treating any emerging complications, such as cardiovascular issues and type 2 diabetes, at an early stage. Regular medical check-ups allow for continuous assessment and timely adjustments to treatment plans based on individual patient needs, enhancing overall disease management and reducing the severity of related health complications.

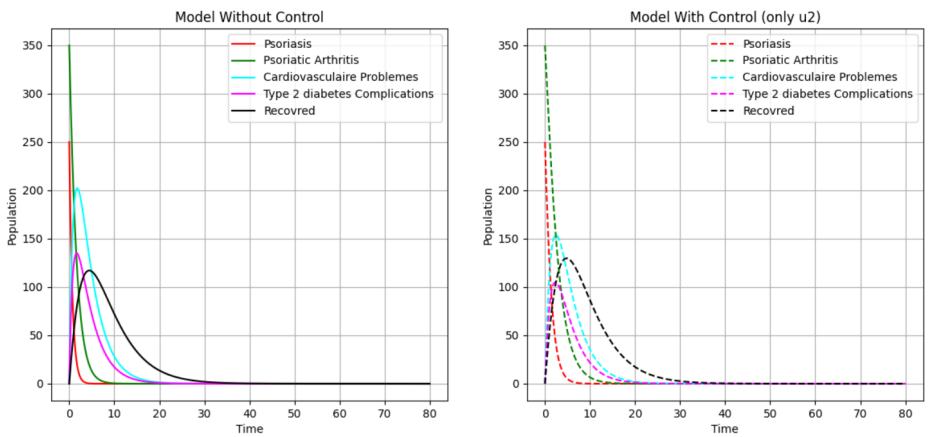

Figure 3: Evaluation of the Model with Control u_2 Versus Without Control

Before Control, the number of individuals experiencing cardiovascular problems increases to approximately 200. Similarly, Type 2 diabetes complications grow from 0 to about 140. After implementing only the medical follow-up control u_2, the increase in cardiovascular problems slows down, reaching around 150, thus reducing the severity compared to the no-control scenario. Concurrently, Type 2 diabetes complications back to approximately 100, demonstrating a marked decrease in complications relative to the situation without any controls.

Third strategy : Mitigating Type 2 Diabetes Complications and cardiovasculare disease in Individuals with Psoriasis and Psoriatic Arthritis through a Wellness-Oriented Lifestyle Applying only the control u_3 to adopt a healthy lifestyle involves motivating individuals to adopt a balanced diet and participate in regular physical activity and practice effective stress management. This strategy aims to improve overall health and well-being, potentially reducing the risk and impact of chronic conditions such as cardiovascular problems and Type 2 diabetes. By focusing solely on promoting a wellness-oriented lifestyle, the strategy seeks to enhance individuals' resilience and reduce the long-term burden of these health issues.

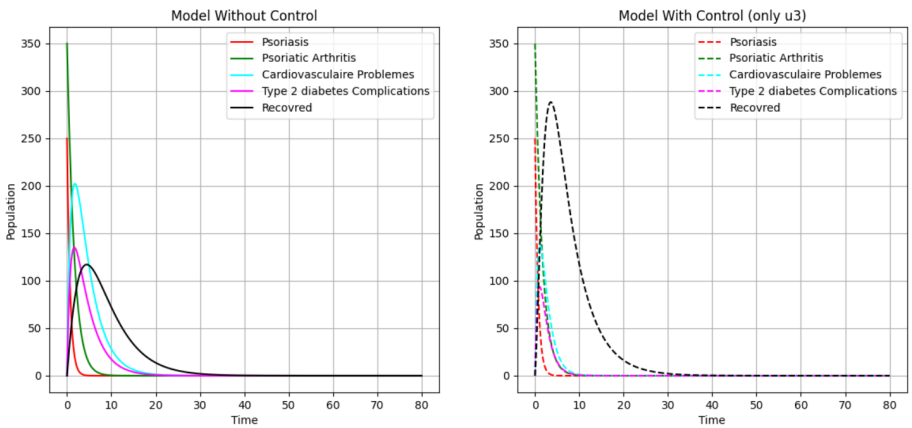

Figure 4: Evaluation of the Model with Control u_3 Versus Without Control

Before adopting a wellness-oriented lifestyle, the number of individuals experiencing cardiovascular problems steadily increased, reaching about 200. Similarly, the population with Type 2 diabetes complications grew significantly, reaching approximately 140 individuals. However, after implementing a wellness-oriented lifestyle, the number of individuals with cardiovascular problems was reduced to around 140, and those with Type 2 diabetes complications decreased to about 90. This comparison highlights the significant effectiveness of the wellness-oriented lifestyle in mitigating both cardiovascular issues and Type 2 diabetes complications.

Forth strategy : Mitigating Type 2 Diabetes Complications and cardiovasculare disease in Individuals with Psoriasis and Psoriatic Arthritis by combining all control methods Strategy 4 involves implementing a comprehensive approach by applying all three control measures simultaneously: awareness programs for psoriasis and psoriatic arthritis, enhanced medical follow-up, and promoting a wellness-oriented lifestyle. This integrated strategy aims to maximize the impact on health outcomes by addressing multiple aspects of disease management and prevention. By combining efforts in awareness, continuous medical care, and lifestyle improvements, this approach seeks to significantly reduce the incidence and severity of psoriasis-related complications, enhance general well-being and improve the standard of living for those affected.

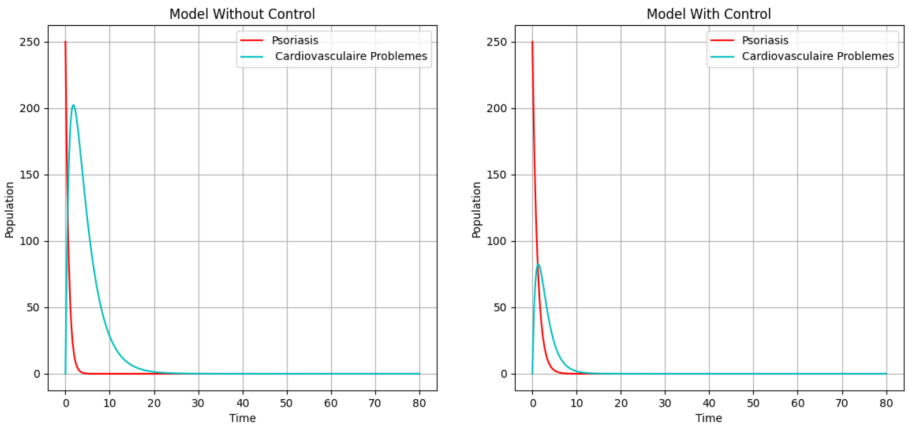

Figure 5: The Incidence of Cardiovascular Issues in People with Psoriasis Before and After Implementing All Controls

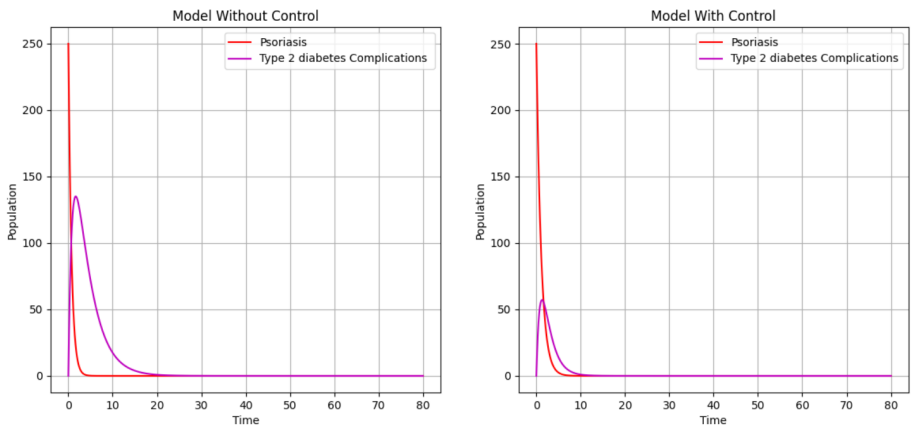

Figure 6: The Incidence of Type 2 Diabetes Complications in Psoriasis Patients Before and After Implementing All Controls

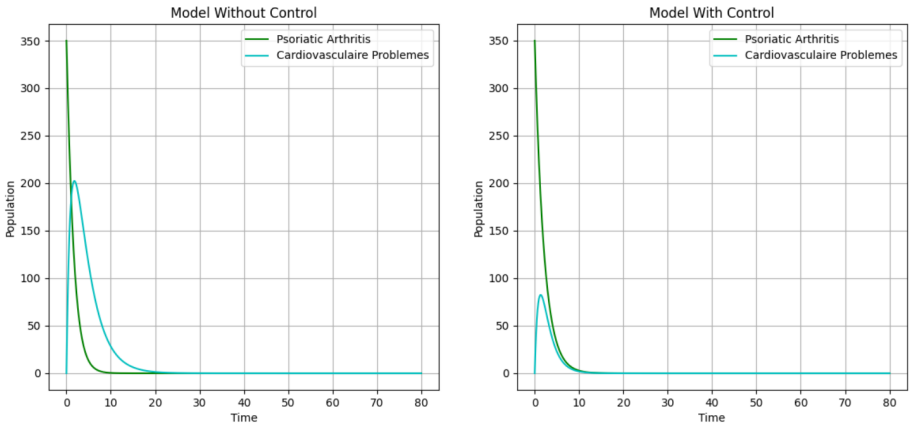

Figure 7: The Incidence of Cardiovascular Issues in People with Psoriasis Arthritis Before and After Implementing All Controls

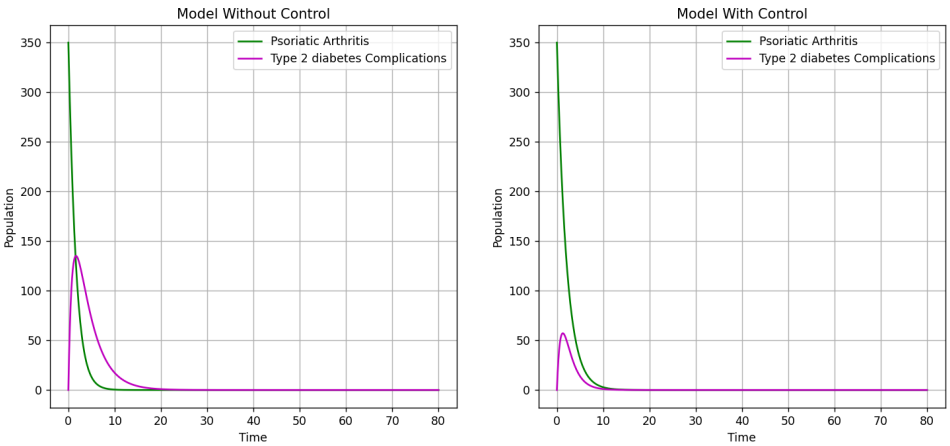

Figure 8: The Incidence of Type 2 Diabetes Complications in Psoriasis Arthritis Patients Before and After Implementing All Controls

The integrated use of these strategies leads to a notable decrease in complications from type 2 diabetes and cardiovascular issues. Data show that the population suffering from these complications decreases markedly when these controls are applied together, illustrating a notable improvement in overall health outcomes. In summary, Strategy 4 optimizes interventions to reduce the negative impacts of chronic diseases and improve patients' quality of life.

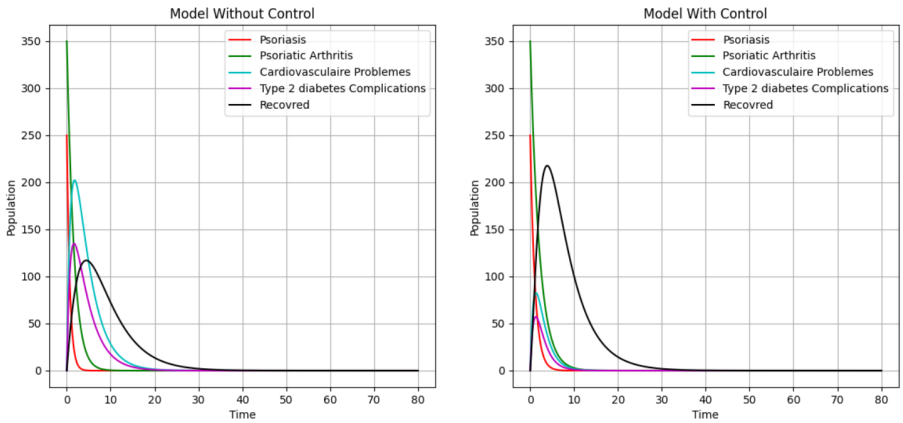

Figure 9: Evaluation of the Model with and Without Control

6. Conclusion

This article has explored in depth the complex interactions between psoriasis, psoriatic arthritis, and their associated complications, including cardiovascular problems and type 2 diabetes. Through a rigorous analysis of the effects of these conditions on overall health, it has become clear that psoriasis and psoriatic arthritis are not just skin or joint disorders, but systemic diseases that also predispose to serious complications such as cardiovascular problems and type 2 diabetes.

The study highlighted that cardiovascular and metabolic complications can significantly worsen patients' quality of life, leading to a progressive deterioration in their health status. In response to this challenge, we examined various controls and interventions to mitigate these adverse effects. The strategies implemented include patient education, regular medical follow-up and promotion of a healthy lifestyle, each offering distinct but complementary benefits.

The results show that the simultaneous application of these controls, i.e. Strategy 4, is particularly effective. By combining patient education, rigorous medical monitoring and healthy lifestyle habits, we observe a significant reduction in complications related to type 2 diabetes and cardiovascular problems. These integrated approaches not only allow us to manage symptoms more effectively, but also prevent the worsening of conditions.

In conclusion, the management of psoriasis and psoriatic arthritis, as well as their associated complications, requires a comprehensive and multifaceted approach. The adoption of combined controls represents a promising strategy to enhance well-being and patient health status, highlighting the importance of early and continuous intervention in the management of these chronic diseases.

Conflict of Interests

The authors declare that there is no conflict of interests.

References

1. National Psoriasis Foundation, *Psoriasis Foundation Website*, https://www.psoriasis.org/, (2022).
2. National Psoriasis Foundation, *Psoriasis Causes, Triggers, and Treatments: What Is Psoriasis?*, https://www.psoriasis.org/about-psoriasis/, (2024).
3. Mayo Clinic, *Psoriatic Arthritis - Symptoms and Causes*, https://www.mayoclinic.org/diseases-conditions/psoriatic-arthritis/symptoms-causes/syc-20354076, (2021).
4. World Health Organization, *Global Report on Psoriasis*, https://www.who.int/publications/i/item/9789241565189, (2024).
5. National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS), *Official Website*, https://www.niams.nih.gov/, (2024).

6. World Health Organization, *Cardiovascular Diseases (CVDs)*, https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds), (2021).
7. Verywell Health, *How Prevalent Is Type 2 Diabetes? Statistics and Facts*, https://www.verywellhealth.com/type-2-diabetes-statistics-5214216, (2023).
8. Centers for Disease Control and Prevention (CDC), *About Type 2 Diabetes*, https://www.cdc.gov/diabetes/about/about-type-2-diabetes.html, (2024).
9. Dattani, S., Samborska, V., Ritchie, H., and Roser, M., *Cardiovascular Diseases*, Our World in Data, https://ourworldindata.org/cardiovascular-diseases, (2023).
10. Berna-Rico, E., Abbad-Jaime De Aragon, C., Garcia-Aparicio, A., Palacios-Martinez, D., Ballester-Martinez, A., Carrascosa, J. M., De La Cueva, P., Anton, C., Azcarraga-Llobet, C., Garcia-Mouronte, E., De Nicolas-Ruanes, B., Puig, L., Jaen, P., Mehta, N. N., Gelfand, J. M., and Gonzalez-Cantero, A., *Cardiovascular Screening Practices and Statin Prescription Habits in Patients with Psoriasis*, Acta Dermato-Venereologica, 103, adv5087, (2023).
11. Dermatology Times, *Addressing Cardiovascular Risk Factors in Psoriasis Patients*, https://www.dermatologytimes.com/view/addressing-cardiovascular-risk-factors-in-psoriasis-patients, (2024).
12. Garshick, M. S., Ward, N. L., Krueger, J. G., and Berger, J. S., *Cardiovascular Risk in Patients With Psoriasis: JACC Review Topic of the Week*, Journal of the American College of Cardiology, 77, 1670–1680, (2021).
13. Kampe, T., Baloghová, J., Kolarčik, P., Rimárová, K., and Dorko, E., *SCORE2 Screening Tool for Cardiovascular Risk Assessment in Psoriasis*, Journal of Clinical Medicine, 13(11), 3237, (2024).
14. Eder, L., Dey, A., Joshi, A. A., Boehncke, W.-H., Mehta, N. N., and Szentpetery, A., *Psoriasis, Psoriatic Arthritis and Cardiometabolic Disease*, The Journal of Rheumatology Supplement, (2019).
15. Holm, J. G., and Thomsen, S. F., *Type 2 Diabetes and Psoriasis: Links and Risks*, Psoriasis (Auckland), (2019).
16. National Psoriasis Foundation, *Type 2 Diabetes Risk Associated With Psoriatic Arthritis*, https://www.psoriasis.org/advance/type-2-diabetes-risk-associated-with-psa/, (2020).
17. Charlton, R., Green, A., Shaddick, G., Snowball, J., and Nightingale, A., *Risk of Type 2 Diabetes and Cardiovascular Disease in an Incident Cohort of People With Psoriatic Arthritis*, Rheumatology, 58, 144–148, (2019).
18. Pandey, S., Tiwari, S., Basu, S., Mishra, R. K., and Pandey, R., *Dynamics of a Network Mediated by IL-36 and Involved in the Pathogenesis of Psoriasis*, Frontiers in Network Physiology, (2024).
19. Grigorenko, N. L., Grigorieva, É. V., Roi, P. K., and Khailov, E. N., *Optimal Control Problems for a Mathematical Model of the Treatment of Psoriasis*, Computational Mathematics and Modeling, 30, 352–363, (2019).
20. Fleming, W. H., and Rishel, R. W., *Deterministic and Stochastic Optimal Control*, Springer-Verlag, (1975).
21. Boyce, W. E., and DiPrima, R. C., *Elementary Differential Equations and Boundary Value Problems*, Wiley, (2020).
22. Pontryagin, L. S., Boltyanskii, V. G., Gamkrelidze, R. V., and Mishechenko, E. F., *The Mathematical Theory of Optimal Processes*, Wiley, (1962).
23. Lions, J. L., *Exact Controllability, Stabilizability, and Perturbations for Distributed Systems*, Siam Rev. 30, 1-68, (1988).
24. C. M. Dafermos, C. M., *An abstract Volterra equation with application to linear viscoelasticity.* J. Differential Equations 7, 554-589, (1970).

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