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A STAGE-STRUCTURED DIABETES PROGRESSION MODEL WITH CONTROL-RELAPSE DYNAMICS: WELL-POSEDNESS, PERSISTENCE, AND STABILITY

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Abstract. We develop and analyze a nonlinear stage-structured model for diabetes progression that distinguishes uncontrolled and controlled diabetes and incorporates bidirectional management–relapse transitions together with bounded prevalence-weighted deterioration effects. We establish local and global well-posedness, positivity, boundedness, and the existence of a positively invariant feasible region. We also characterize the equilibrium structure, derive conditions for the existence of a positive equilibrium, and prove persistence of the downstream disease classes. A central analytical feature is the decomposition of the system into an upstream linear subsystem and a downstream nonlinear subsystem, which clarifies the mechanism governing the long-term dynamics. Local asymptotic stability of the positive equilibrium is established through Jacobian analysis and the Routh–Hurwitz criterion, while a Lyapunov–LaSalle argument yields a sufficient condition for global asymptotic stability. As an extension, we formulate an optimal-control version of the model with prevention, management, and relapse-prevention interventions and solve the resulting system numerically over a 10-year horizon. The simulations show

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that combining all three controls yields the largest reduction in uncontrolled diabetes and severe-complication burden, while sensitivity analysis identifies the progression-related parameters with the strongest influence on severe outcomes. These results show that the proposed framework is mathematically tractable and biologically relevant for studying long-term diabetes complication dynamics.

Keywords: diabetes progression; well-posedness; positive equilibrium; persistence; optimal control.

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1. INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder whose burden extends far beyond elevated blood glucose. Poor glycemic control may lead over time to cardiovascular disease, renal damage, disability, and premature death, making diabetes a major noncommunicable disease of global public-health concern [1, 2]. From a population perspective, the burden of diabetes depends not only on the incidence of disease, but also on how individuals move through different stages of progression, how effectively control is maintained, and how rapidly complications develop when control is poor.

A clinically important feature of diabetes is that glycemic control is often temporary. Some individuals achieve improved control through treatment, education, and follow-up, whereas others lose control because of nonadherence, interrupted care, financial barriers, or persistent lifestyle pressures [3]. This makes it natural, at the modeling level, to distinguish between uncontrolled and controlled diabetes while allowing bidirectional movement between these states. Such a distinction is clinically meaningful because complication risk depends strongly on whether control is sustained over time [2]. At the same time, lifestyle and environmental factors continue to shape the pressure toward deterioration, especially in low-resource settings [4, 5].

Compartmental models based on systems of ordinary differential equations provide a useful framework for studying diabetes progression at the population level, since each compartment and transition has a clear interpretation and the resulting system remains amenable to rigorous analysis [6, 7]. Although mathematical models have been developed for diabetes prevalence, complications, and intervention strategies [8, 9], many formulations treat diabetes without complications as a single class. As a result, they do not explicitly capture the interaction between

uncontrolled disease, temporary control, relapse, and lifestyle-related deterioration within a single analytically tractable framework.

To address this gap, we develop a stage-structured diabetes progression model with recruitment and mortality, comprising healthy individuals, prediabetes, lifestyle-related risk exposure, uncontrolled diabetes, controlled diabetes, treatable complications, and severe complications. The model incorporates management–relapse dynamics, prevalence-dependent deterioration, and reduced complication risk under temporary control. This structure makes it possible to examine how progression, control, relapse, and complication development interact to shape long-term disease burden.

The main contributions of this paper are as follows. First, we formulate a biologically interpretable nonlinear model for diabetes progression that distinguishes uncontrolled and controlled diabetes while incorporating relapse and lifestyle-related deterioration. Second, we establish the fundamental qualitative properties of the system, including local and global well-posedness, positivity, boundedness, and the existence of a positively invariant feasible region. Third, we characterize the equilibrium structure, derive conditions for the existence of a positive equilibrium, and study persistence and stability properties of the downstream disease classes. Finally, we extend the model to an optimal-control framework in order to examine how prevention, management, and relapse-prevention interventions affect the long-term burden of severe complications. The remainder of the paper is organized as follows. Section 2 presents the model formulation and parameter definitions. Section 3 develops the qualitative analysis of the model. Section 4 introduces the optimal-control problem and derives the necessary conditions for optimality. Section 5 contains the numerical illustrations, and the final section concludes the paper.

2. Model Formulation

We formulate a deterministic stage-structured compartmental model for diabetes progression based on balance-law principles. At time t, the total population $N(t)$ is partitioned into mutually exclusive health states representing clinically meaningful stages of disease development and complication progression. Specifically, we consider the following seven compartments:

- $H(t)$: healthy individuals without diabetes,

- $P(t)$: individuals with prediabetes,
- $E(t)$: individuals exposed to lifestyle or behavioral risk factors,
- $D_u(t)$: individuals with diabetes without complications and poor glycemic control,
- $D_c(t)$: individuals with diabetes without complications but under glycemic control,
- $C_T(t)$: individuals with diabetes and treatable complications,
- $C_S(t)$: individuals with diabetes and severe complications.

Accordingly, the total population is given by

$$N(t) = H(t) + P(t) + E(t) + D_u(t) + D_c(t) + C_T(t) + C_S(t). \tag{1}$$

Model assumptions. The model is built under the following assumptions:

(A1) New individuals enter the population through the healthy class H at a constant recruitment rate I. All compartments are subject to natural mortality at rate μ.

(A2) Healthy individuals progress to the prediabetes class P at rate θ_1 and to the lifestyle-related risk class E at rate θ_2. Individuals in P progress to uncontrolled diabetes at rate β_1, while individuals in E progress to uncontrolled diabetes at rate γ.

(A3) Individuals with uncontrolled diabetes D_u may achieve glycemic control and move to the controlled class D_c at rate τ. Conversely, loss of adherence, interruption of care, or treatment failure may lead to relapse from D_c back to D_u at rate ρ.

(A4) Controlled diabetes provides partial protection against progression to complications. In particular, individuals in D_c progress to treatable and severe complications at reduced rates $\kappa\beta_3$ and $\kappa\eta_3$, respectively, where $\kappa \in (0,1)$ measures the protective effect of sustained glycemic control.

(A5) Individuals in the uncontrolled diabetes class D_u progress to treatable complications C_T at rate β_2 and to severe complications C_S at rate η_2. Individuals in C_T may further deteriorate to C_S at rate η_1.

(A6) Lifestyle-related exposure contributes additionally to deterioration through the prevalence-weighted terms $\alpha_1 \frac{D_u E}{N}$ and $\alpha_2 \frac{C_T E}{N}$. These terms represent exposure-amplified progression effects rather than direct transfers out of the exposure class E.

(A7) In addition to natural mortality, uncontrolled diabetes, treatable complications, and severe complications experience disease-induced mortality at rates δ_u, δ_T, and δ, respectively.

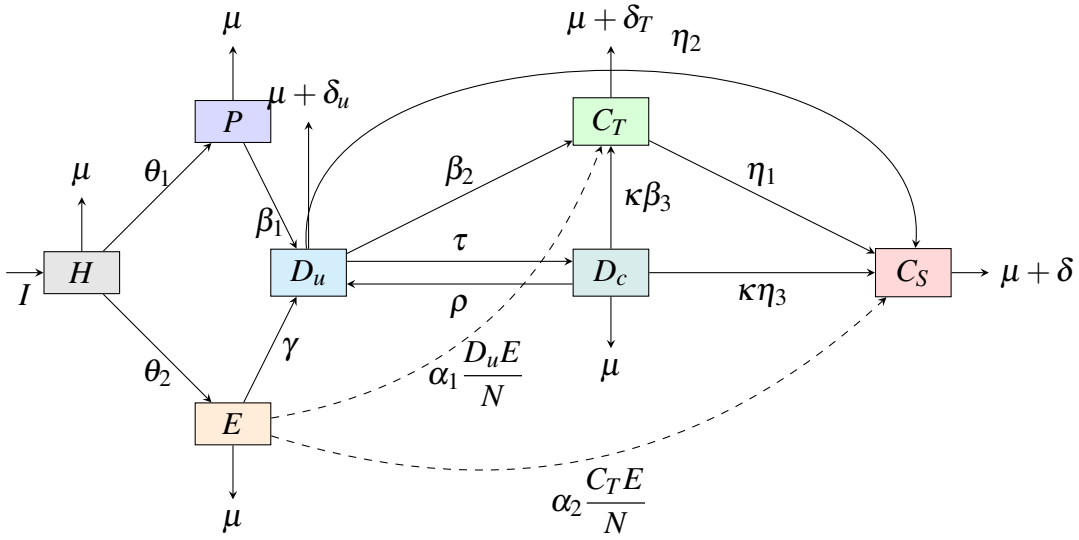

FIGURE 1. Compartmental structure of the diabetes progression model. The upstream subsystem consists of H, P, and E, while the downstream subsystem consists of D_u, D_c, C_T, and C_S. Dashed arrows denote exposure-amplified deterioration effects rather than direct population transfers.

Figure 1 summarizes the compartmental structure of the model. The classes H, P, and E form the upstream subsystem, while D_u, D_c, C_T, and C_S form the downstream diabetes–complication subsystem. The prevalence-weighted terms $\alpha_1 \frac{D_u E}{N}$ and $\alpha_2 \frac{C_T E}{N}$ represent exposure-amplified deterioration. Guided by this structure, the governing system is given by

$$\frac{dH}{dt} = I - (\mu + \theta_1 + \theta_2)H, \tag{2}$$

$$\frac{dP}{dt} = \theta_1 H - (\mu + \beta_1)P, \tag{3}$$

$$\frac{dE}{dt} = \theta_2 H - (\mu + \gamma)E, \tag{4}$$

$$\frac{dD_u}{dt} = \beta_1 P + \gamma E + \rho D_c - \tau D_u - \alpha_1 \frac{D_u E}{N} - (\mu + \delta_u + \beta_2 + \eta_2)D_u, \tag{5}$$

$$\frac{dD_c}{dt} = \tau D_u - \rho D_c - (\mu + \kappa\beta_3 + \kappa\eta_3)D_c, \tag{6}$$

$$\frac{dC_T}{dt} = \beta_2 D_u + \kappa\beta_3 D_c + \alpha_1 \frac{D_u E}{N} - \alpha_2 \frac{C_T E}{N} - (\mu + \delta_T + \eta_1) C_T, \tag{7}$$

$$\frac{dC_S}{dt} = \eta_2 D_u + \kappa\eta_3 D_c + \eta_1 C_T + \alpha_2 \frac{C_T E}{N} - (\mu + \delta) C_S. \tag{8}$$

The baseline parameter values used in the analytical and numerical investigations are summarized in Table 1. Whenever possible, these values were drawn from Ethiopia-based technical reports and peer-reviewed studies. When reported quantities appeared in different forms, they were converted to consistent annual-rate units. In the numerical simulations, population variables are reported in millions, so the recruitment rate is expressed in million persons per year. The numerical results should therefore be interpreted as Ethiopia-informed and scenario-based rather than as the outcome of a formal statistical calibration procedure.

TABLE 1. Baseline parameter values used in the analytical and numerical investigations.

Parameter	Meaning	Units	Value	Source
I	Recruitment into H	million persons year^{-1}	1.026	[11]
μ	Natural mortality rate	year^{-1}	0.015	[11]
δ_u	Disease-induced mortality in D_u	year^{-1}	0.10	[10, 17]
δ_T	Disease-induced mortality in C_T	year^{-1}	0.12	[10, 17]
δ	Disease-induced mortality in C_S	year^{-1}	0.25	[10, 12]
θ_1	Transition rate $H \to P$	year^{-1}	0.030	[14]
θ_2	Transition rate $H \to E$	year^{-1}	0.010	[13, 19]
β_1	Progression rate $P \to D_u$	year^{-1}	0.030	[23, 14]
γ	Progression rate $E \to D_u$	year^{-1}	0.050	[13, 24]
β_2	Progression rate $D_u \to C_T$	year^{-1}	0.10	[11, 20]
η_2	Progression rate $D_u \to C_S$	year^{-1}	0.040	[17]
β_3	Baseline progression rate $D_c \to C_T$ before reduction	year^{-1}	0.105	[17, 15]
η_3	Baseline progression rate $D_c \to C_S$ before reduction	year^{-1}	0.015	[18, 17]
η_1	Progression rate $C_T \to C_S$	year^{-1}	0.10	[18, 17]
α_1	Exposure-amplified deterioration toward C_T	year^{-1}	0.002	[21, 22]
α_2	Exposure-amplified deterioration toward C_S	year^{-1}	0.0015	[23, 24]
τ	Management rate $D_u \to D_c$	year^{-1}	0.35	[12, 13]
ρ	Relapse rate $D_c \to D_u$	year^{-1}	0.20	[12]
κ	Risk-reduction factor under control $(0 < \kappa < 1)$	–	0.55	[10]

3. WELL-POSEDNESS, EQUILIBRIA, AND STABILITY ANALYSIS

In this section, we investigate the qualitative properties of system (2)–(8). We begin by establishing the basic well-posedness of the model, including local existence and uniqueness,

positivity, positive invariance, boundedness, and global existence of solutions. We then turn to the equilibrium structure, persistence properties, and stability analysis. These results show that the proposed diabetes progression framework defines a mathematically consistent nonlinear dynamical system whose long-term behavior can be studied rigorously.

3.1. Well-posedness and invariant-region properties. Throughout this section, all parameters are assumed to be nonnegative, and the recruitment term satisfies $I \geq 0$. Let

$$X(t) = \big(H(t), P(t), E(t), D_u(t), D_c(t), C_T(t), C_S(t)\big)$$

denote the state vector, and let

$$N(t) = H(t) + P(t) + E(t) + D_u(t) + D_c(t) + C_T(t) + C_S(t)$$

be the total population. We assume that the initial condition satisfies $X(0) \in \mathbb{R}^7_+$ and $N(0) > 0$, so that the prevalence-weighted terms

$$\frac{D_u E}{N} \qquad \text{and} \qquad \frac{C_T E}{N}$$

are well defined.

Theorem 1 (Local existence and uniqueness)**.** *Let*

$$\mathscr{U} = \left\{ X \in \mathbb{R}^7 : \; H + P + E + D_u + D_c + C_T + C_S > 0 \right\}.$$

For every initial condition $X(0) \in \mathscr{U}$, system (2)–(8) *admits a unique local solution on a maximal interval* $[0, t_{\max})$.

Proof. Write the system in vector form as

$$\dot{X} = f(X).$$

The only nonpolynomial terms in the vector field are $\frac{D_u E}{N}$ and $\frac{C_T E}{N}$. Since $N > 0$ on $\mathscr{U}$, these terms are continuously differentiable on $\mathscr{U}$. Hence $f \in C^1(\mathscr{U}, \mathbb{R}^7)$, and in particular f is locally Lipschitz on $\mathscr{U}$. The conclusion therefore follows from the Picard–Lindelöf theorem [25, 26].

Theorem 2 (Positivity of solutions)**.** *If $X(0) \in \mathbb{R}^7_+$ and $N(0) > 0$, then the unique local solution of system* (2)–(8) *satisfies*

$$H(t), P(t), E(t), D_u(t), D_c(t), C_T(t), C_S(t) \geq 0 \qquad \text{for all } t \in [0, t_{\max}).$$

Proof. We verify that the vector field is inward-pointing on each boundary hyperplane of the nonnegative orthant.

If $H = 0$, then

$$\frac{dH}{dt} = I \geq 0.$$

If $P = 0$, then

$$\frac{dP}{dt} = \theta_1 H \geq 0.$$

If $E = 0$, then

$$\frac{dE}{dt} = \theta_2 H \geq 0.$$

If $D_u = 0$, then $\alpha_1 \frac{D_u E}{N} = 0$ and

$$\frac{dD_u}{dt} = \beta_1 P + \gamma E + \rho D_c \geq 0.$$

If $D_c = 0$, then

$$\frac{dD_c}{dt} = \tau D_u \geq 0.$$

If $C_T = 0$, then $\alpha_2 \frac{C_T E}{N} = 0$ and

$$\frac{dC_T}{dt} = \beta_2 D_u + \kappa \beta_3 D_c + \alpha_1 \frac{D_u E}{N} \geq 0.$$

If $C_S = 0$, then

$$\frac{dC_S}{dt} = \eta_2 D_u + \kappa \eta_3 D_c + \eta_1 C_T + \alpha_2 \frac{C_T E}{N} \geq 0.$$

Hence solutions with nonnegative initial data cannot leave the nonnegative orthant. Therefore $\mathbb{R}^7_+$ is forward invariant, and every solution remains nonnegative for as long as it exists.

Theorem 3 (Positive invariant region and boundedness)**.** *Assume that $\mu > 0$, and define*

$$\Omega = \left\{ X \in \mathbb{R}^7_+ : \, N \leq \frac{I}{\mu} \right\}.$$

Then Ω *is positively invariant for system* (2)–(8). *More generally, for every nonnegative initial condition, the set*

$$\Omega^* = \left\{ X \in \mathbb{R}^7_+ : \, N \leq \max\left(N(0), \frac{I}{\mu} \right) \right\}$$

is positively invariant. In particular,

$$N(t) \leq \max\left(N(0), \frac{I}{\mu} \right) \qquad \textit{for all } t \in [0, t_{\max}),$$

and hence each state variable remains bounded on $[0, t_{\max})$.

Proof. By Theorem 2, all solution components remain nonnegative. Summing equations (2)–(8) yields

$$\frac{dN}{dt} = I - \mu N - \delta_u D_u - \delta_T C_T - \delta C_S.$$

Since $D_u, C_T, C_S \geq 0$, it follows that

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

On the boundary $N = \frac{I}{\mu}$, we have

$$\frac{dN}{dt} = I - \mu \frac{I}{\mu} - \delta_u D_u - \delta_T C_T - \delta C_S = -(\delta_u D_u + \delta_T C_T + \delta C_S) \leq 0.$$

Hence trajectories cannot leave the set $\{N \leq I/\mu\}$ through this boundary, and therefore Ω is positively invariant.

Moreover, whenever $N(t) > \frac{I}{\mu}$, the inequality $\dot{N} \leq I - \mu N < 0$ shows that the total population is decreasing. Thus,

$$N(t) \leq \max\left(N(0), \frac{I}{\mu} \right) \qquad \text{for all } t \in [0, t_{\max}),$$

which proves the positive invariance of Ω^*.

Finally, since each component of $X(t)$ is nonnegative and bounded above by $N(t)$, all state variables remain bounded on the maximal interval of existence.

Remark 1 (Well-defined prevalence-weighted terms)**.** *If* $N(0) > 0$*, then* $N(t) > 0$ *for all* $t \in [0, t_{\max})$. *Indeed, if*

$$\delta_{\max} = \max\{\delta_u, \delta_T, \delta\},$$

then

$$\dot{N} = I - \mu N - \delta_u D_u - \delta_T C_T - \delta C_S \geq -\mu N - \delta_{\max}(D_u + C_T + C_S) \geq -(\mu + \delta_{\max})N.$$

By comparison,

$$N(t) \geq N(0)e^{-(\mu+\delta_{\max})t} > 0 \qquad \textit{for all } t \in [0, t_{\max}).$$

Hence the terms $\frac{D_u E}{N}$ *and* $\frac{C_T E}{N}$ *remain well defined along all solution trajectories.*

Corollary 1 (Global existence and uniqueness)**.** *For every initial condition* $X(0) \in \mathbb{R}^7_+$ *with* $N(0) > 0$*, system* (2)–(8) *admits a unique global solution*

$$X(t) \in \mathbb{R}^7_+ \qquad \textit{for all } t \geq 0.$$

Proof. By Theorem 1, there exists a unique local solution on a maximal interval $[0, t_{\max})$. By Theorem 2, the solution remains in $\mathbb{R}^7_+$, and by Theorem 3, it remains bounded on every finite time interval. Remark 1 shows that $N(t)$ stays strictly positive, so the solution never approaches the singular set $\{N = 0\}$. Standard continuation results for ordinary differential equations therefore imply that $t_{\max} = \infty$ [25, 26].

Remark 2. *Combining Theorem 3 with Corollary 1, we obtain*

$$\limsup_{t\to\infty} N(t) \leq \frac{I}{\mu}.$$

3.2. Equilibrium analysis. Under the present parameter assumptions, system (2)–(8) does not admit a disease-absent equilibrium. Since the recruitment term maintains positive upstream classes and the transitions $P \to D_u$ and $E \to D_u$ generate persistent inflow into uncontrolled diabetes, the relevant steady state is a positive equilibrium.

An equilibrium of system (2)–(8) is a vector

$$x^* = (H^*, P^*, E^*, D_u^*, D_c^*, C_T^*, C_S^*) \in \mathbb{R}^7_+$$

for which all derivatives vanish. Setting equations (2)–(4) equal to zero gives

$$H^* = \frac{I}{\mu + \theta_1 + \theta_2}, \tag{9}$$

$$P^* = \frac{\theta_1}{\mu + \beta_1} H^*, \tag{10}$$

$$E^* = \frac{\theta_2}{\mu+\gamma} H^*. \tag{11}$$

In particular, if $I > 0$, then $H^* > 0$, and if $\theta_1 > 0$ and $\theta_2 > 0$, then also $P^* > 0$ and $E^* > 0$.

For the remaining components, the steady-state relations yield

$$D_u^* = \frac{\beta_1 P^* + \gamma E^*}{\tau + (\mu + \delta_u + \beta_2 + \eta_2) + \alpha_1 \frac{E^*}{N^*} - \frac{\rho\tau}{\rho+\mu+\kappa\beta_3+\kappa\eta_3}},$$

$$D_c^* = \frac{\tau}{\rho + \mu + \kappa\beta_3 + \kappa\eta_3} D_u^*,$$

$$C_T^* = \frac{\beta_2 D_u^* + \kappa\beta_3 D_c^* + \alpha_1 \frac{D_u^* E^*}{N^*}}{(\mu + \delta_T + \eta_1) + \alpha_2 \frac{E^*}{N^*}},$$

and

$$C_S^* = \frac{\eta_2 D_u^* + \kappa\eta_3 D_c^* + \eta_1 C_T^* + \alpha_2 \frac{C_T^* E^*}{N^*}}{\mu + \delta}.$$

The ratio

$$\frac{D_c^*}{D_u^*} = \frac{\tau}{\rho + \mu + \kappa\beta_3 + \kappa\eta_3} \tag{12}$$

characterizes the equilibrium balance between controlled and uncontrolled diabetes in the no-complication subsystem.

Proposition 4 (Asymptotic behavior of the upstream subsystem)**.** *For every solution of system* (2)–(8)*, the upstream variables $H(t)$, $P(t)$, and $E(t)$ satisfy*

$$H(t) \to H^*, \qquad P(t) \to P^*, \qquad E(t) \to E^* \qquad \text{as } t \to \infty,$$

where

$$H^* = \frac{I}{\mu + \theta_1 + \theta_2}, \qquad P^* = \frac{\theta_1}{\mu + \beta_1} H^*, \qquad E^* = \frac{\theta_2}{\mu + \gamma} H^*.$$

Proof. The equations for H, P, and E form a linear triangular subsystem:

$$\dot{H} = I - (\mu + \theta_1 + \theta_2)H, \qquad \dot{P} = \theta_1 H - (\mu + \beta_1)P, \qquad \dot{E} = \theta_2 H - (\mu + \gamma)E.$$

The first equation is a scalar linear equation whose solution converges to

$$H^* = \frac{I}{\mu + \theta_1 + \theta_2}.$$

Substituting this into the equations for P and E and applying standard linear ODE theory yields

$$P(t) \to P^* = \frac{\theta_1}{\mu+\beta_1} H^*, \qquad E(t) \to E^* = \frac{\theta_2}{\mu+\gamma} H^*,$$

as $t \to \infty$ [25, 26].

Proposition 5 (Asymptotically autonomous reduction of the downstream subsystem)**.** *Let*

$$Y(t) = \big(D_u(t), D_c(t), C_T(t), C_S(t)\big)^\top,$$

and write the downstream equations of (2)–(8) *in the form*

$$\dot{Y} = F(t,Y).$$

Define

$$N_*(Y) = H^* + P^* + E^* + D_u + D_c + C_T + C_S,$$

where H^*, P^*, and E^* are given by (9)–(11)*. Then*

$$F(t,Y) = G(Y) + R(t,Y),$$

where the limiting vector field G is given by

$$G_1(Y) = \beta_1 P^* + \gamma E^* + \rho D_c - \tau D_u - \alpha_1 \frac{D_u E^*}{N_*(Y)} - (\mu + \delta_u + \beta_2 + \eta_2) D_u,$$

$$G_2(Y) = \tau D_u - \rho D_c - (\mu + \kappa\beta_3 + \kappa\eta_3) D_c,$$

$$G_3(Y) = \beta_2 D_u + \kappa\beta_3 D_c + \alpha_1 \frac{D_u E^*}{N_*(Y)} - \alpha_2 \frac{C_T E^*}{N_*(Y)} - (\mu + \delta_T + \eta_1) C_T,$$

$$G_4(Y) = \eta_2 D_u + \kappa\eta_3 D_c + \eta_1 C_T + \alpha_2 \frac{C_T E^*}{N_*(Y)} - (\mu + \delta) C_S,$$

and the remainder $R(t,Y)$ satisfies

$$\sup_{Y \in K} \|R(t,Y)\| \to 0 \qquad \text{as } t \to \infty$$

for every compact set $K \subset \mathbb{R}^4_+$.

Proof. By Proposition 4,

$$H(t) \to H^*, \qquad P(t) \to P^*, \qquad E(t) \to E^* \qquad \text{as } t \to \infty.$$

Write the downstream equations as

$$\dot{Y} = G(Y) + R(t,Y),$$

where $G(Y)$ is obtained by replacing $P(t)$ and $E(t)$ with their limits P^* and E^*, and by replacing

$$N(t) = H(t) + P(t) + E(t) + D_u + D_c + C_T + C_S$$

with

$$N_*(Y) = H^* + P^* + E^* + D_u + D_c + C_T + C_S.$$

Thus $R(t,Y)$ consists of terms involving the differences

$$H(t) - H^*, \qquad P(t) - P^*, \qquad E(t) - E^*.$$

Let $K \subset \mathbb{R}^4_+$ be compact. Since $H^* + P^* + E^* > 0$, there exists $c_K > 0$ such that

$$N_*(Y) \geq c_K > 0 \qquad \text{for all } Y \in K.$$

Moreover, for all sufficiently large t, the convergence of $H(t)$, $P(t)$, and $E(t)$ implies that

$$N(t) \geq \frac{c_K}{2} > 0 \qquad \text{uniformly for } Y \in K.$$

Since the maps

$$(P,E,Y) \mapsto \beta_1 P + \gamma E, \qquad (E,Y) \mapsto \frac{D_u E}{N}, \qquad (E,Y) \mapsto \frac{C_T E}{N}$$

are continuous on sets where the denominator is bounded away from zero, it follows that

$$\sup_{Y \in K} \|R(t,Y)\| \to 0 \qquad \text{as } t \to \infty.$$

This proves the claim.

Remark 3. *Proposition 5 shows that the long-term dynamics of the full system are determined by a limiting nonlinear subsystem for the downstream variables. In particular, the positive upstream limit acts as an asymptotic forcing term for the diabetes and complication classes.*

3.2.1. *Existence of a positive equilibrium.* To establish the existence of a positive equilibrium, define

$$m = \frac{E^*}{N^*} \in (0,1].$$

Then

$$\alpha_1 \frac{D_u^* E^*}{N^*} = \alpha_1 m D_u^*, \qquad \alpha_2 \frac{C_T^* E^*}{N^*} = \alpha_2 m C_T^*.$$

Using (12), the steady-state version of equation (5) becomes

$$\left[(\tau + \mu + \delta_u + \beta_2 + \eta_2) + \alpha_1 m - \frac{\rho\tau}{\rho + \mu + \kappa\beta_3 + \kappa\eta_3} \right] D_u^* = \beta_1 P^* + \gamma E^*.$$

Hence

$$D_u^*(m) = \frac{\beta_1 P^* + \gamma E^*}{a_0 + \alpha_1 m}, \qquad a_0 = (\tau + \mu + \delta_u + \beta_2 + \eta_2) - \frac{\rho\tau}{\rho + \mu + \kappa\beta_3 + \kappa\eta_3}. \tag{13}$$

Moreover,

$$a_0 = (\mu + \delta_u + \beta_2 + \eta_2) + \tau \left(1 - \frac{\rho}{\rho + \mu + \kappa\beta_3 + \kappa\eta_3} \right) > 0,$$

and therefore $D_u^*(m) > 0$ whenever $\beta_1 P^* + \gamma E^* > 0$. In addition,

$$D_c^*(m) = \frac{\tau}{\rho + \mu + \kappa\beta_3 + \kappa\eta_3} D_u^*(m). \tag{14}$$

From equations (7) and (8), we obtain

$$C_T^*(m) = \frac{(\beta_2 + \alpha_1 m) D_u^*(m) + \kappa\beta_3 D_c^*(m)}{\mu + \delta_T + \eta_1 + \alpha_2 m}, \tag{15}$$

and

$$C_S^*(m) = \frac{\eta_2 D_u^*(m) + \kappa\eta_3 D_c^*(m) + (\eta_1 + \alpha_2 m) C_T^*(m)}{\mu + \delta}. \tag{16}$$

Since $m = E^*/N^*$, we also have $N^* = E^*/m$, while

$$N^* = H^* + P^* + E^* + D_u^*(m) + D_c^*(m) + C_T^*(m) + C_S^*(m).$$

Thus m must satisfy the scalar equation

$$\frac{E^*}{m} = H^* + P^* + E^* + D_u^*(m) + D_c^*(m) + C_T^*(m) + C_S^*(m), \qquad m \in (0,1]. \tag{17}$$

Theorem 6 (Existence of a positive equilibrium)**.** *Assume that $I > 0$, $\mu > 0$, and $\theta_2 > 0$, so that $E^* > 0$. If*

$$\beta_1 P^* + \gamma E^* > 0,$$

then system (2)–(8) *admits at least one positive equilibrium*

$$x^* = (H^*, P^*, E^*, D_u^*, D_c^*, C_T^*, C_S^*) \in \mathbb{R}_+^7.$$

Proof. Define

$$F(m) = \frac{E^*}{m} - \Big(H^* + P^* + E^* + D_u^*(m) + D_c^*(m) + C_T^*(m) + C_S^*(m)\Big), \qquad m \in (0,1].$$

By (13)–(16), the function F is continuous on $(0,1]$.

As $m \to 0^+$, one has $\frac{E^*}{m} \to +\infty$, whereas the remaining terms remain finite. Hence

$$\lim_{m \to 0^+} F(m) = +\infty.$$

On the other hand,

$$F(1) = E^* - \Big(H^* + P^* + E^* + D_u^*(1) + D_c^*(1) + C_T^*(1) + C_S^*(1)\Big) < 0,$$

since $H^* > 0$ and all other terms are nonnegative. Therefore, by the Intermediate Value Theorem, there exists $m^* \in (0,1)$ such that $F(m^*) = 0$.

Set

$$N^* = \frac{E^*}{m^*}, \qquad D_u^* = D_u^*(m^*), \qquad D_c^* = D_c^*(m^*), \qquad C_T^* = C_T^*(m^*), \qquad C_S^* = C_S^*(m^*).$$

Then

$$x^* = (H^*, P^*, E^*, D_u^*, D_c^*, C_T^*, C_S^*)$$

satisfies the steady-state equations. Positivity of D_u^* follows from (13), and then (14)–(16) imply positivity of D_c^*, C_T^*, and C_S^*. Hence x^* is a positive equilibrium.

3.3. Uniform persistence of the downstream classes.

We now establish uniform persistence of the downstream disease classes under natural positivity assumptions on the upstream inflow and downstream progression terms.

Theorem 7 (Uniform persistence of the downstream classes)**.** *Assume that*

$$I > 0, \qquad \mu > 0, \qquad \theta_1 > 0, \qquad \theta_2 > 0, \qquad \beta_1 > 0, \qquad \gamma > 0, \qquad \tau > 0, \qquad \beta_2 > 0,$$

and

$$\eta_1 + \eta_2 > 0.$$

Let

$$X(0) \in (0,\infty)^7.$$

Then there exist constants

$$m_u, \quad m_c, \quad m_T, \quad m_S > 0$$

such that every solution of system (2)–(8) *satisfies*

$$\liminf_{t\to\infty} D_u(t) \geq m_u, \qquad \liminf_{t\to\infty} D_c(t) \geq m_c,$$

$$\liminf_{t\to\infty} C_T(t) \geq m_T, \qquad \liminf_{t\to\infty} C_S(t) \geq m_S.$$

Hence the classes D_u, D_c, C_T, and C_S are uniformly persistent.

Proof. By Proposition 4,

$$H(t) \to H^*, \qquad P(t) \to P^*, \qquad E(t) \to E^* \qquad \text{as } t \to \infty.$$

Since $I > 0$, $\theta_1 > 0$, and $\theta_2 > 0$, it follows from (9)–(11) that

$$H^* > 0, \qquad P^* > 0, \qquad E^* > 0.$$

Fix $\varepsilon \in (0,1)$. Then there exists $T_0 > 0$ such that, for all $t \geq T_0$,

$$P(t) \geq (1-\varepsilon)P^*, \qquad E(t) \geq (1-\varepsilon)E^*.$$

We first estimate D_u. Since $0 \leq E/N \leq 1$ and $D_c \geq 0$, equation (5) yields, for $t \geq T_0$,

$$\frac{dD_u}{dt} \geq \beta_1 P(t) + \gamma E(t) - \big(\tau + \mu + \delta_u + \beta_2 + \eta_2 + \alpha_1\big) D_u.$$

Hence

$$\frac{dD_u}{dt} \geq b_u - a_u D_u,$$

where

$$a_u := \tau + \mu + \delta_u + \beta_2 + \eta_2 + \alpha_1, \qquad b_u := (1-\varepsilon)(\beta_1 P^* + \gamma E^*) > 0.$$

Comparison with the scalar equation $y' = b_u - a_u y$ gives

$$\liminf_{t\to\infty} D_u(t) \geq \frac{b_u}{a_u}.$$

Choose

$$m_u := \frac{b_u}{2a_u} > 0.$$

Then there exists $T_1 \geq T_0$ such that

$$D_u(t) \geq m_u \qquad \text{for all } t \geq T_1.$$

Next, from equation (6), for $t \geq T_1$,

$$\frac{dD_c}{dt} = \tau D_u - \big(\rho + \mu + \kappa\beta_3 + \kappa\eta_3\big)D_c \geq \tau m_u - a_c D_c,$$

where

$$a_c := \rho + \mu + \kappa\beta_3 + \kappa\eta_3.$$

Therefore,

$$\liminf_{t\to\infty} D_c(t) \geq \frac{\tau m_u}{a_c}.$$

Choose

$$m_c := \frac{\tau m_u}{2a_c} > 0.$$

Then there exists $T_2 \geq T_1$ such that

$$D_c(t) \geq m_c \qquad \text{for all } t \geq T_2.$$

For C_T, equation (7) implies, for $t \geq T_2$,

$$\frac{dC_T}{dt} \geq \beta_2 D_u + \kappa\beta_3 D_c - \big(\mu + \delta_T + \eta_1 + \alpha_2\big)C_T,$$

since $\alpha_1 \frac{D_u E}{N} \geq 0$ and $0 \leq E/N \leq 1$. Hence

$$\frac{dC_T}{dt} \geq b_T - a_T C_T,$$

where

$$a_T := \mu + \delta_T + \eta_1 + \alpha_2, \qquad b_T := \beta_2 m_u + \kappa\beta_3 m_c.$$

Thus

$$\liminf_{t\to\infty} C_T(t) \geq \frac{b_T}{a_T}.$$

Choose

$$m_T := \frac{b_T}{2a_T} > 0.$$

Then there exists $T_3 \geq T_2$ such that

$$C_T(t) \geq m_T \qquad \text{for all } t \geq T_3.$$

Finally, equation (8) yields, for $t \geq T_3$,

$$\frac{dC_S}{dt} \geq \eta_2 D_u + \kappa\eta_3 D_c + \eta_1 C_T - (\mu + \delta)C_S,$$

since $\alpha_2 \frac{C_T E}{N} \geq 0$. Therefore

$$\frac{dC_S}{dt} \geq b_S - a_S C_S,$$

where

$$a_S := \mu + \delta, \qquad b_S := \eta_2 m_u + \kappa\eta_3 m_c + \eta_1 m_T.$$

Because $\eta_1 + \eta_2 > 0$, one has $b_S > 0$, and hence

$$\liminf_{t\to\infty} C_S(t) \geq \frac{b_S}{a_S}.$$

Choose

$$m_S := \frac{b_S}{2a_S} > 0.$$

Then, for all sufficiently large t,

$$C_S(t) \geq m_S.$$

Combining the above estimates, we obtain

$$\liminf_{t\to\infty} D_u(t) \geq m_u, \qquad \liminf_{t\to\infty} D_c(t) \geq m_c, \qquad \liminf_{t\to\infty} C_T(t) \geq m_T, \qquad \liminf_{t\to\infty} C_S(t) \geq m_S.$$

This proves uniform persistence of the downstream classes.

Remark 4. *Theorem 7 shows that the downstream disease classes are sustained dynamically in the long run and do not approach extinction under the stated assumptions.*

3.4. Stability of the positive equilibrium. We now study the stability properties of the positive equilibrium. Owing to the triangular structure of the model, the local stability problem for the full system reduces to the downstream subsystem.

Let

$$x^* = (H^*, P^*, E^*, D_u^*, D_c^*, C_T^*, C_S^*)$$

be a positive equilibrium of system (2)–(8). Write the system in vector form as

$$\dot{X} = f(X), \qquad X = (H, P, E, D_u, D_c, C_T, C_S)^\top,$$

and let $J(X) = Df(X)$ denote the Jacobian matrix. Since $N^* > 0$, the vector field is C^1 in a neighborhood of x^*. The Jacobian at equilibrium has the block lower-triangular form

$$J(x^*) = \begin{pmatrix} J_{HPE} & 0 \\ J_{21} & J_{\text{down}} \end{pmatrix},$$

with respect to the decomposition (H, P, E) and (D_u, D_c, C_T, C_S), where

$$J_{HPE} = \begin{pmatrix} -(\mu + \theta_1 + \theta_2) & 0 & 0 \\ \theta_1 & -(\mu + \beta_1) & 0 \\ \theta_2 & 0 & -(\mu + \gamma) \end{pmatrix}.$$

The eigenvalues of J_{HPE} are

$$-(\mu + \theta_1 + \theta_2), \qquad -(\mu + \beta_1), \qquad -(\mu + \gamma),$$

and are therefore strictly negative. Hence

$$\sigma\big(J(x^*)\big) = \sigma(J_{HPE}) \cup \sigma(J_{\text{down}}),$$

so the local stability of x^* is determined entirely by the downstream block.

Let

$$A = J_{\text{down}}(x^*),$$

and let its characteristic polynomial be

$$p(\lambda) = \det(\lambda I - A) = \lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4. \tag{18}$$

Theorem 8 (Local asymptotic stability of the positive equilibrium)**.** *Let x^* be a positive equilibrium of system* (2)–(8) *with $N^* > 0$, and let $A = J_{\text{down}}(x^*)$. Suppose the characteristic polynomial of A is given by* (18)*. If*

$$b_1 > 0, \qquad b_2 > 0, \qquad b_3 > 0, \qquad b_4 > 0,$$

and

$$b_1 b_2 - b_3 > 0, \qquad b_1 b_2 b_3 - b_3^2 - b_1^2 b_4 > 0,$$

then the positive equilibrium x^* is locally asymptotically stable.

Proof. Because $J(x^*)$ is block lower triangular,

$$\sigma\big(J(x^*)\big) = \sigma(J_{HPE}) \cup \sigma(A).$$

Since the eigenvalues of J_{HPE} are strictly negative, it remains to determine when A is Hurwitz. By the Routh–Hurwitz criterion for quartic polynomials [27, 28], all roots of

$$p(\lambda) = \lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4$$

lie in the open left half-plane provided that

$$b_1 > 0, \qquad b_2 > 0, \qquad b_3 > 0, \qquad b_4 > 0,$$

and

$$b_1 b_2 - b_3 > 0, \qquad b_1 b_2 b_3 - b_3^2 - b_1^2 b_4 > 0.$$

Hence A is Hurwitz, and therefore so is $J(x^*)$. It follows that x^* is locally asymptotically stable.

We next state a sufficient condition for global asymptotic stability.

Proposition 9 (A sufficient condition for global asymptotic stability)**.** *Let $\Omega \subset (0,\infty)^7$ be a compact positively invariant set for system* (2)–(8)*, and let*

$$X^* = (H^*, P^*, E^*, D_u^*, D_c^*, C_T^*, C_S^*) \in \Omega$$

be a positive equilibrium. Define

$$W(X) = \operatorname{diag}\left(\frac{1}{H}, \frac{1}{P}, \frac{1}{E}, \frac{1}{D_u}, \frac{1}{D_c}, \frac{1}{C_T}, \frac{1}{C_S}\right),$$

and

$$\bar{J}(X) = \int_0^1 J\big(X^* + s(X - X^*)\big)\, ds.$$

If there exists $\varepsilon > 0$ *such that*

$$\frac{W(X)\bar{J}(X) + \bar{J}(X)^\top W(X)}{2} \preceq -\varepsilon W(X) \qquad \text{for all } X \in \Omega,$$

then X^* *is globally asymptotically stable in* Ω.

Proof. Consider the Lyapunov function

$$\begin{aligned} V(X) = &\left(H - H^* - H^* \ln \frac{H}{H^*}\right) + \left(P - P^* - P^* \ln \frac{P}{P^*}\right) + \left(E - E^* - E^* \ln \frac{E}{E^*}\right) \\ &+ \left(D_u - D_u^* - D_u^* \ln \frac{D_u}{D_u^*}\right) + \left(D_c - D_c^* - D_c^* \ln \frac{D_c}{D_c^*}\right) \\ &+ \left(C_T - C_T^* - C_T^* \ln \frac{C_T}{C_T^*}\right) + \left(C_S - C_S^* - C_S^* \ln \frac{C_S}{C_S^*}\right). \end{aligned}$$

Since $\Omega \subset (0,\infty)^7$, all logarithms are well defined. Moreover,

$$r - 1 - \ln r \geq 0 \qquad (r > 0),$$

so $V(X) \geq 0$, with equality if and only if $X = X^*$.

Let

$$z = X - X^*.$$

Then

$$\dot{V}(X) = w(X)^\top \big(f(X) - f(X^*)\big),$$

where

$$w(X) = \left(\frac{H - H^*}{H}, \frac{P - P^*}{P}, \frac{E - E^*}{E}, \frac{D_u - D_u^*}{D_u}, \frac{D_c - D_c^*}{D_c}, \frac{C_T - C_T^*}{C_T}, \frac{C_S - C_S^*}{C_S}\right)^\top.$$

Since f is continuously differentiable,

$$f(X) - f(X^*) = \left(\int_0^1 J\big(X^* + s(X - X^*)\big)\, ds\right)(X - X^*) = \bar{J}(X) z.$$

Therefore,

$$\dot{V}(X) = w(X)^\top \bar{J}(X) z = \frac{1}{2} z^\top \big(\bar{J}(X)^\top W(X) + W(X)\bar{J}(X)\big) z.$$

By hypothesis,

$$\dot{V}(X) \leq -\varepsilon z^{\top} W(X) z \leq 0 \qquad \text{for all } X \in \Omega.$$

Moreover, $\dot{V}(X) = 0$ implies $z = 0$, hence $X = X^*$.

Since Ω is compact and positively invariant, every solution starting in Ω remains in Ω for all $t \geq 0$. The largest invariant set contained in

$$\{X \in \Omega : \dot{V}(X) = 0\}$$

is therefore the singleton $\{X^*\}$. By LaSalle's invariance principle, every solution with initial condition in Ω converges to X^* as $t \to \infty$ [27, 29].

Remark 5. *Theorem 8 reduces the local stability problem for the full system to the downstream Jacobian block. Proposition 9 provides a sufficient Lyapunov-type criterion for global asymptotic stability.*

4. An Optimal Control Extension

We extend system (2)–(8) by introducing time-dependent intervention controls acting over a finite horizon. The aim is to examine how prevention, management, and relapse-prevention modify the dynamics within a standard optimal-control framework.

We consider three controls:

- **Prevention control** $u_1(t)$, which reduces the effective transition from the healthy class H to the lifestyle-risk class E;
- **Management control** $u_2(t)$, which increases movement from uncontrolled to controlled diabetes;
- **Relapse-prevention control** $u_3(t)$, which reduces movement from controlled back to uncontrolled diabetes.

Each control satisfies

$$0 \leq u_i(t) \leq 1, \qquad i = 1,2,3,$$

where $u_i(t) = 0$ represents baseline practice and $u_i(t) = 1$ represents maximal intervention effort at time t.

Let

$$N(t) = H(t) + P(t) + E(t) + D_u(t) + D_c(t) + C_T(t) + C_S(t) \tag{19}$$

denote the total population. The controls are incorporated through the rate modifications

$$\theta_2 \mapsto \theta_2(1-u_1(t)), \qquad \rho \mapsto \rho(1-u_3(t)), \qquad \tau \mapsto \tau(u_2(t)). \tag{20}$$

To reflect improved management, we assume that $\tau(u_2)$ is a bounded increasing function of u_2, namely

$$\tau(u_2) = \tau_{\min} + (\tau_{\max} - \tau_{\min})u_2, \qquad \Delta\tau := \tau_{\max} - \tau_{\min} > 0. \tag{21}$$

The resulting controlled system is

$$\frac{dH}{dt} = I - \Big(\mu + \theta_1 + \theta_2(1-u_1)\Big)H, \tag{22}$$

$$\frac{dP}{dt} = \theta_1 H - (\mu + \beta_1)P, \tag{23}$$

$$\frac{dE}{dt} = \theta_2(1-u_1)H - (\mu + \gamma)E, \tag{24}$$

$$\frac{dD_u}{dt} = \beta_1 P + \gamma E + \rho(1-u_3)D_c - \tau(u_2)D_u - \alpha_1\frac{D_u E}{N} - (\mu + \delta_u + \beta_2 + \eta_2)D_u, \tag{25}$$

$$\frac{dD_c}{dt} = \tau(u_2)D_u - \rho(1-u_3)D_c - (\mu + \kappa\beta_3 + \kappa\eta_3)D_c, \tag{26}$$

$$\frac{dC_T}{dt} = \beta_2 D_u + \kappa\beta_3 D_c + \alpha_1\frac{D_u E}{N} - \alpha_2\frac{C_T E}{N} - (\mu + \delta_T + \eta_1)C_T, \tag{27}$$

$$\frac{dC_S}{dt} = \eta_2 D_u + \kappa\eta_3 D_c + \eta_1 C_T + \alpha_2\frac{C_T E}{N} - (\mu + \delta)C_S. \tag{28}$$

We consider the finite-horizon objective functional

$$J(u_1,u_2,u_3) = \int_0^T \left(w_1 D_u + w_2 C_T + w_3 C_S + \frac{1}{2}\left(A_1 u_1^2 + A_2 u_2^2 + A_3 u_3^2\right)\right) dt, \tag{29}$$

where $w_i > 0$ penalize uncontrolled diabetes and complication burden, while $A_i > 0$ weight control effort.

The admissible control set is

$$\mathscr{U} := \left\{(u_1,u_2,u_3) \mid u_i : [0,T] \to \mathbb{R} \text{ measurable},\ 0 \le u_i(t) \le 1 \text{ for a.e. } t \in [0,T],\ i = 1,2,3\right\}. \tag{30}$$

The optimal control problem is to determine $(u_1^*, u_2^*, u_3^*) \in \mathscr{U}$ such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{(u_1,u_2,u_3)\in\mathscr{U}} J(u_1, u_2, u_3), \tag{31}$$

subject to the controlled system (22)–(28) and nonnegative initial data.

4.1. Positivity and boundedness of the controlled system. Throughout this section, assume that the parameters are nonnegative, with $I > 0$, $\mu > 0$, and $\kappa \in (0,1)$. Assume also that the initial data are nonnegative and satisfy $N(0) > 0$.

Corollary 2 (Positivity and boundedness of the controlled system)**.** *For any admissible control triple $(u_1, u_2, u_3) \in \mathscr{U}$ and any nonnegative initial condition, the solution of the controlled system* (22)–(28) *remains in $\mathbb{R}^7_+$ for all $t \in [0,T]$. Moreover,*

$$0 \le N(t) \le \max\left\{N(0), \frac{I}{\mu}\right\} \qquad \text{for all } t \in [0,T],$$

and, in particular, $N(t) > 0$ on $[0,T]$.

Proof. Since $0 \le u_1, u_2, u_3 \le 1$, the controlled rates satisfy

$$\theta_2(1-u_1) \ge 0, \qquad \rho(1-u_3) \ge 0, \qquad \tau(u_2) \ge 0.$$

Hence the same boundary-invariance argument used for the uncontrolled system applies and yields positivity of the state variables.

Summing equations (22)–(28) gives

$$\dot{N} = I - \mu N - \delta_u D_u - \delta_T C_T - \delta C_S,$$

which is identical to the total-population equation in the uncontrolled case. The stated bound for $N(t)$ and the strict positivity of $N(t)$ therefore follow exactly as in Section 3.

4.2. Existence of an optimal control.

Theorem 10 (Existence of an optimal control)**.** *There exists at least one optimal control triple $(u_1^*, u_2^*, u_3^*) \in \mathscr{U}$ that minimizes the objective functional* (29) *subject to the controlled system* (22)–(28).

Proof. The admissible control set $\mathscr{U}$ is nonempty, closed, bounded, and convex. By Corollary 2, for each admissible control triple the corresponding state solution remains nonnegative and bounded on $[0,T]$. Since $N(t) > 0$ on $[0,T]$, the prevalence-weighted terms are well defined, and the right-hand side of the controlled system is continuous in the controls and locally Lipschitz in the state variables on the relevant bounded region. Hence the state system admits a unique solution on $[0,T]$ for each admissible control.

Moreover, the integrand

$$w_1 D_u + w_2 C_T + w_3 C_S + \frac{1}{2}\left(A_1 u_1^2 + A_2 u_2^2 + A_3 u_3^2\right)$$

is measurable in t, continuous in the state and control variables, nonnegative, and convex in (u_1,u_2,u_3) because $A_1, A_2, A_3 > 0$. Therefore, by the standard existence theorem for deterministic optimal control problems, an optimal control triple exists [30, 31].

4.3. Necessary conditions for optimality. We now derive first-order necessary conditions for optimality by Pontryagin's Maximum Principle. Define

$$x(t) = \big(H, P, E, D_u, D_c, C_T, C_S\big)(t), \qquad u(t) = \big(u_1, u_2, u_3\big)(t) \in [0,1]^3,$$

and let

$$N(t) = H(t) + P(t) + E(t) + D_u(t) + D_c(t) + C_T(t) + C_S(t).$$

The running cost is

$$L(x,u) = w_1 D_u + w_2 C_T + w_3 C_S + \frac{A_1}{2} u_1^2 + \frac{A_2}{2} u_2^2 + \frac{A_3}{2} u_3^2, \tag{32}$$

so that

$$J(u) = \int_0^T L(x(t), u(t))\, dt.$$

The Hamiltonian is defined by

$$\mathscr{H}(x,u,\lambda) = L(x,u) + \lambda^\top f(x,u), \tag{33}$$

where $f(x,u)$ denotes the right-hand side of the controlled system and

$$\lambda(t) = \big(\lambda_H, \lambda_P, \lambda_E, \lambda_{Du}, \lambda_{Dc}, \lambda_{CT}, \lambda_{CS}\big)(t)$$

is the adjoint vector.

Theorem 11 (Necessary conditions for optimality)**.** *Let $(u_1^*, u_2^*, u_3^*) \in \mathscr{U}$ be an optimal control for problem* (31)*, with corresponding state trajectory $x^*(t)$. Then there exists an absolutely continuous adjoint vector*

$$\lambda(t) = \big(\lambda_H, \lambda_P, \lambda_E, \lambda_{Du}, \lambda_{Dc}, \lambda_{CT}, \lambda_{CS}\big)(t)$$

such that, for almost every $t \in [0,T]$,

$$\dot{\lambda}(t) = -\nabla_x \mathscr{H}\big(x^*(t), u^*(t), \lambda(t)\big), \tag{34}$$

with transversality conditions

$$\lambda_H(T) = \lambda_P(T) = \lambda_E(T) = \lambda_{Du}(T) = \lambda_{Dc}(T) = \lambda_{CT}(T) = \lambda_{CS}(T) = 0, \tag{35}$$

and the pointwise minimization condition

$$\mathscr{H}\big(x^*(t), u^*(t), \lambda(t)\big) = \min_{(u_1,u_2,u_3)\in[0,1]^3} \mathscr{H}\big(x^*(t), (u_1,u_2,u_3), \lambda(t)\big) \quad \text{for a.e. } t \in [0,T]. \tag{36}$$

Proof. The result follows from Pontryagin's Maximum Principle for fixed final time and free terminal state, applied to the controlled system (22)–(28) and the objective functional (29) [30, 31].

4.4. Characterization of the optimal controls. Since the controls are constrained to lie in $[0,1]$, the minimizing controls are obtained pointwise from the stationarity conditions and then projected onto the admissible interval. Define

$$\Pi_{[0,1]}(z) = \min\{1, \max\{0, z\}\}.$$

Proposition 12 (Characterization of the optimal controls)**.** *Let $(x^*(t), \lambda(t))$ satisfy the controlled state system and adjoint system associated with the optimal control problem, and let*

$$\Delta\tau = \tau_{\max} - \tau_{\min} > 0.$$

Then, for almost every $t \in [0,T]$, the optimal controls are given by

$$u_1^*(t) = \Pi_{[0,1]}\left(\frac{\theta_2 H^*(t)\big(\lambda_E(t) - \lambda_H(t)\big)}{A_1}\right), \tag{37}$$

$$u_2^*(t) = \Pi_{[0,1]}\left(\frac{\Delta\tau\, D_u^*(t)\big(\lambda_{Du}(t) - \lambda_{Dc}(t)\big)}{A_2}\right), \tag{38}$$

$$u_3^*(t) = \Pi_{[0,1]}\left(\frac{\rho\, D_c^*(t)\big(\lambda_{Du}(t) - \lambda_{Dc}(t)\big)}{A_3}\right). \tag{39}$$

Proof. Differentiating the Hamiltonian with respect to the controls gives

$$\frac{\partial \mathscr{H}}{\partial u_1} = A_1 u_1 + \theta_2 H(\lambda_H - \lambda_E),$$

$$\frac{\partial \mathscr{H}}{\partial u_2} = A_2 u_2 + \Delta\tau\, D_u(\lambda_{Dc} - \lambda_{Du}),$$

and

$$\frac{\partial \mathscr{H}}{\partial u_3} = A_3 u_3 + \rho\, D_c(\lambda_{Dc} - \lambda_{Du}).$$

Setting these derivatives equal to zero yields the unconstrained minimizers,

$$u_1 = \frac{\theta_2 H(\lambda_E - \lambda_H)}{A_1}, \qquad u_2 = \frac{\Delta\tau\, D_u(\lambda_{Du} - \lambda_{Dc})}{A_2}, \qquad u_3 = \frac{\rho\, D_c(\lambda_{Du} - \lambda_{Dc})}{A_3}.$$

Projection onto the interval $[0,1]$ gives (37)–(39).

5. Numerical Results and Discussion

In this section, we present numerical computations illustrating the analytical results for the uncontrolled system and the effect of the optimal-control extension introduced in Section 4. The simulations are intended to support the qualitative structure of the model rather than to provide a formal calibration study. For the uncontrolled system, we use a 30-year simulation horizon in order to display the long-term behavior predicted by the analysis, in particular the approach toward the positive equilibrium and the persistence of the downstream classes after transient dynamics. By contrast, the optimal-control simulations are carried out over a 10-year horizon, consistent with the finite-horizon formulation in Section 4. All state variables are interpreted as population counts in millions, and the total adult population is taken as

$$N_0 = 68.4 \text{ million}.$$

The initial conditions are chosen as

$$H(0) = 60.5904, \qquad P(0) = 3.200, \qquad E(0) = 1.600,$$

$$D_u(0) = 2.350, \qquad D_c(0) = 0.350, \qquad C_T(0) = 0.200, \qquad C_S(0) = 0.1096,$$

which satisfy

$$H(0)+P(0)+E(0)+D_u(0)+D_c(0)+C_T(0)+C_S(0)=N_0$$

up to rounding. Unless otherwise stated, all simulations use the baseline parameter values listed in Table 1.

5.1. Baseline long-term dynamics. We first consider the uncontrolled system in order to illustrate boundedness of solutions and approach toward the positive equilibrium identified in Section 3. Figure 2 displays representative trajectories of the seven state variables under the baseline parameter set.

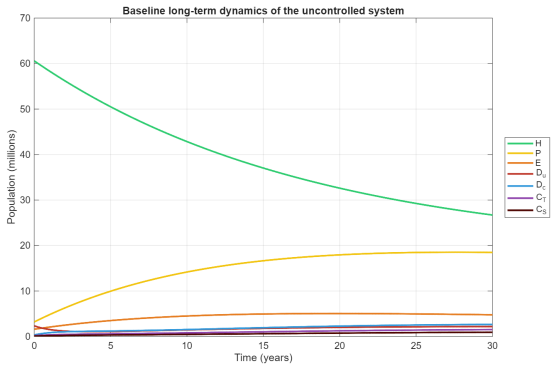

FIGURE 2. Baseline trajectories of the uncontrolled system under the parameter values in Table 1. The simulations illustrate positivity, boundedness, and approach toward the long-term positive equilibrium.

Figure 2 shows that all solution components remain nonnegative and bounded over the simulation horizon, in agreement with the analytical results of Section 3. The upstream classes approach long-term levels, while the downstream classes D_u, D_c, C_T, and C_S remain strictly positive and evolve toward sustained asymptotic values. Thus, the numerical behavior is consistent with a biologically meaningful long-term positive regime.

5.2. Persistence of the downstream classes. We next illustrate the persistence result for the downstream subsystem. Figure 3 displays the trajectories of D_u, D_c, C_T, and C_S over the extended simulation horizon.

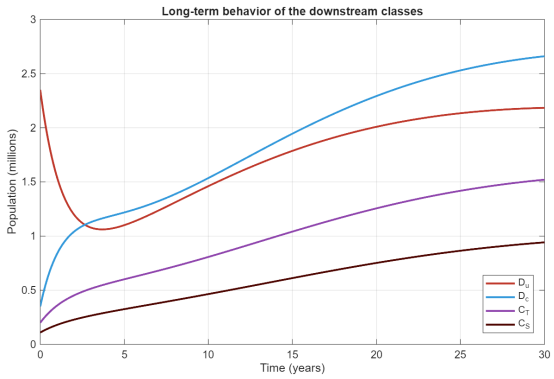

FIGURE 3. Long-term trajectories of the downstream classes D_u, D_c, C_T, and C_S in the uncontrolled system. The simulations show that all downstream classes remain strictly positive after transients, consistent with uniform persistence.

Figure 3 shows that the downstream classes D_u, D_c, C_T, and C_S remain strictly positive after the initial transient phase and continue toward sustained long-term levels. In particular, none of these classes tends toward extinction over the simulation horizon. This is consistent with Theorem 7 and confirms that, under the baseline parameter regime, the downstream disease and complication classes are dynamically maintained in the long run.

5.3. Sensitivity analysis of the severe-complication burden. To complement the baseline simulations, we examine the sensitivity of the 10-year severe-complication burden

$$B_S(p) := \int_0^T C_S(t;p)\,dt, \qquad T = 10 \text{ years}, \tag{40}$$

where p denotes the parameter vector and $C_S(t;p)$ is the corresponding solution of the uncontrolled system, that is,

$$u_1 = u_2 = u_3 \equiv 0,$$

with the baseline initial condition.

For each parameter p_i, define the elasticity index

$$\mathscr{E}_i := \frac{p_i}{B_S(p)} \frac{\partial B_S(p)}{\partial p_i}, \tag{41}$$

which measures the relative change in B_S induced by a relative change in p_i. We approximate (41) by the central finite-difference formula

$$\mathscr{E}_i \approx \frac{p_i}{B_S(p)} \frac{B_S\big(p_i(1+\varepsilon)\big) - B_S\big(p_i(1-\varepsilon)\big)}{2\varepsilon p_i}, \tag{42}$$

with all other parameters fixed at baseline. Throughout, we take $\varepsilon = 0.1$ and rank parameters by $|\mathscr{E}_i^{C}|$. Positive values indicate that increasing the parameter increases the burden functional B_S, whereas negative values indicate the opposite effect.

TABLE 2. Top drivers of the 10-year cumulative severe-complication burden $B_S = \int_0^T C_S(t)\,dt$ for the uncontrolled system. Parameters are ranked by $|\mathscr{E}_i^{C}|$.

Parameter	Elasticity $\mathscr{E}_i^{C}$	Effect on B_S
δ	-0.5629	decreases
η_2	$+0.3213$	increases
η_1	$+0.3165$	increases
β_2	$+0.1816$	increases
δ_u	-0.1159	decreases
γ	$+0.1115$	increases
δ_T	-0.1083	decreases
β_1	$+0.0997$	increases
κ	$+0.0989$	increases
μ	-0.0805	decreases

For the baseline initial condition and $T = 10$ years, the cumulative severe-complication burden is

$$B_S = \int_0^{10} C_S(t)\,dt = 3.1598$$

million person-years.

Table 2 shows that the strongest positive drivers of B_S are the progression parameters η_2, η_1, and β_2, while the strongest negative effect is associated with the severe-complication mortality rate δ. Thus, in the present parameter regime, the severe-complication burden is governed primarily by the direct and indirect progression pathways leading into C_S.

5.4. Optimal-control strategies. We solve the optimal control problem numerically by a forward–backward sweep method, using fourth-order Runge–Kutta integration for the state

equations together with the adjoint system derived from Pontryagin's Maximum Principle, under the bounds

$$0 \le u_i(t) \le 1, \qquad i = 1,2,3.$$

The three controls correspond to prevention (u_1), management (u_2), and relapse-prevention (u_3). To illustrate their respective roles, we consider four strategies: S1 activates (u_1,u_2), S2 activates (u_1,u_3), S3 activates (u_2,u_3), and S4 activates all three controls. For each strategy, we compare the baseline and controlled trajectories of D_u, D_c, C_T, and C_S, and summarize performance using the total objective value J together with the cumulative severe-complication burden

$$\int_0^T C_S(t)\,dt.$$

5.4.1. *Strategy 1: prevention and management* (u_1,u_2). In this case, prevention and management are active, while relapse-prevention is set to zero:

$$u_3(t) \equiv 0.$$

Figure 4 compares the baseline and controlled trajectories.

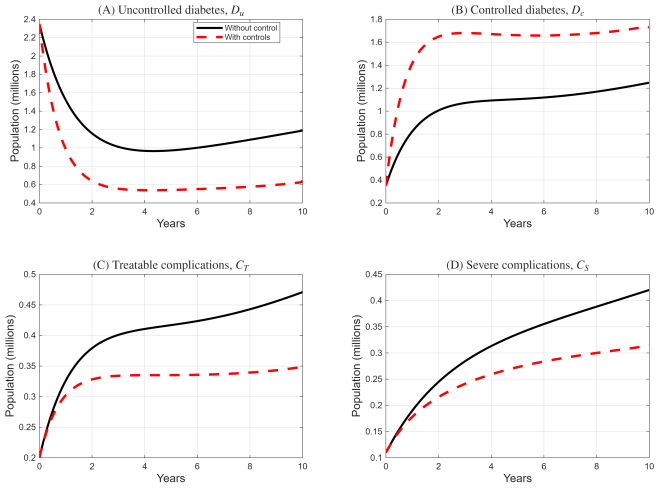

FIGURE 4. Strategy 1 (controls u_1,u_2; $u_3 \equiv 0$): baseline (solid) versus controlled (dashed) trajectories of (A) D_u, (B) D_c, (C) C_T, and (D) C_S over $T = 10$ years.

Figure 4 shows that Strategy 1 reduces the uncontrolled-diabetes class D_u and increases the controlled class D_c relative to baseline. The complication classes C_T and C_S are also lowered over the intervention horizon. Thus, the combined action of prevention and management decreases the uncontrolled burden and yields a corresponding downstream reduction in complications.

5.4.2. *Strategy 2: prevention and relapse-prevention* (u_1, u_3). In this case, prevention and relapse-prevention are active, while management remains at baseline:

$$u_2(t) \equiv 0.$$

Figure 5 shows the corresponding trajectories.

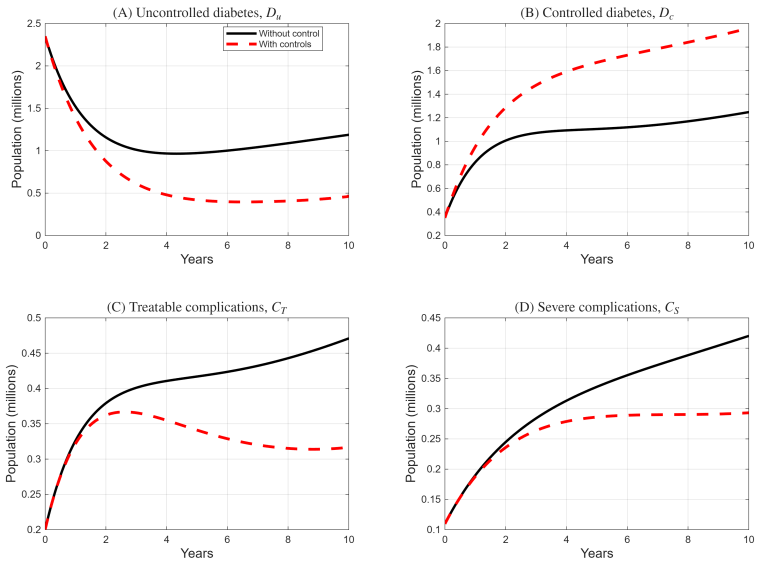

FIGURE 5. Strategy 2 (controls u_1, u_3; $u_2 \equiv 0$): baseline (solid) versus controlled (dashed) trajectories of (A) D_u, (B) D_c, (C) C_T, and (D) C_S over $T = 10$ years.

Figure 5 shows that, under Strategy 2, the uncontrolled-diabetes class D_u decreases while the controlled class D_c increases throughout the intervention horizon. This reflects the effect of relapse-prevention, which weakens the return flow from D_c to D_u. The classes C_T and C_S remain below their baseline trajectories, indicating a reduction in complication burden.

5.4.3. *Strategy 3: management and relapse-prevention* (u_2, u_3). We next consider the management–relapse loop by activating management and relapse-prevention while setting prevention to zero:

$$u_1(t) \equiv 0.$$

The corresponding trajectories are shown in Figure 6.

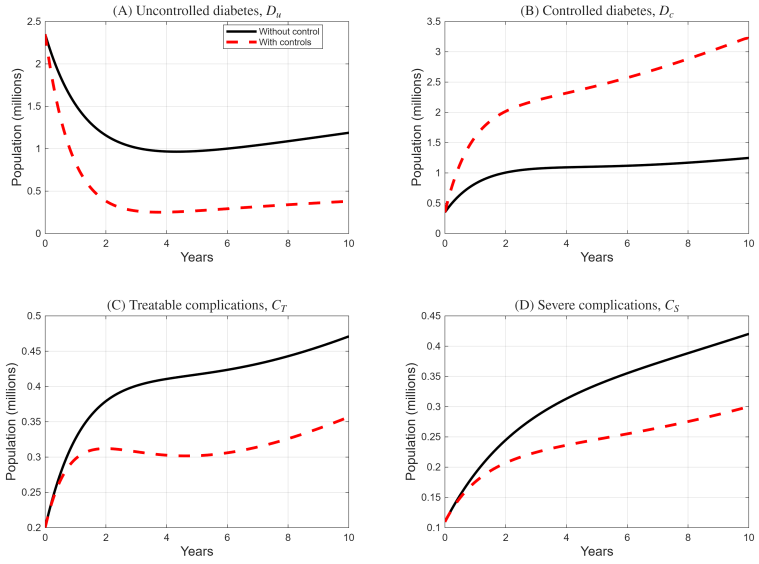

FIGURE 6. Strategy 3 (controls u_2, u_3; $u_1 \equiv 0$): baseline (solid) versus controlled (dashed) trajectories of (A) D_u, (B) D_c, (C) C_T, and (D) C_S over $T = 10$ years.

Figure 6 shows that Strategy 3 produces the strongest redistribution between uncontrolled and controlled diabetes among the two-control interventions. The class D_u decreases sharply, whereas D_c increases markedly. The complication classes C_T and C_S are likewise reduced relative to baseline. This indicates that direct intervention on the management–relapse loop has a particularly strong effect on the severe-complication burden.

5.4.4. *Strategy 4: prevention, management, and relapse-prevention* (u_1, u_2, u_3). Finally, all three controls are activated simultaneously. Figure 7 shows the resulting trajectories.

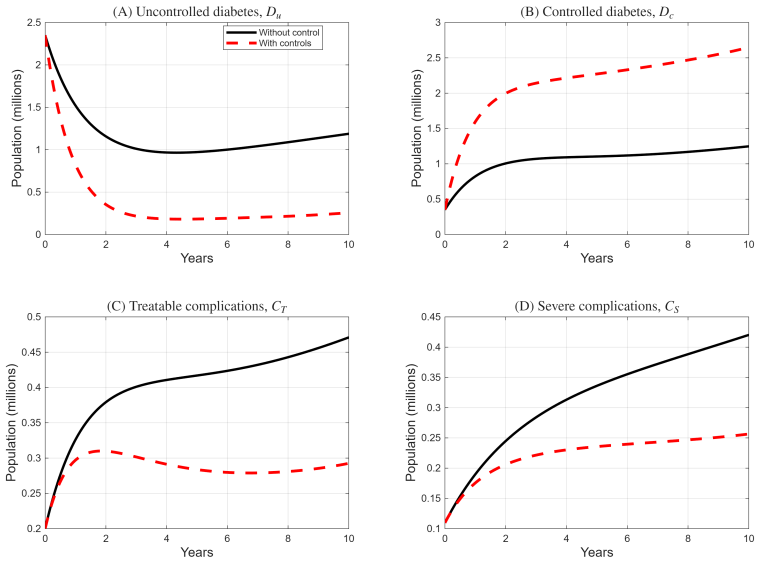

FIGURE 7. Strategy 4 (controls u_1, u_2, u_3): baseline (solid) versus controlled (dashed) trajectories of (A) D_u, (B) D_c, (C) C_T, and (D) C_S over $T = 10$ years.

Figure 7 shows that Strategy 4 produces the strongest overall effect among the four intervention scenarios. The uncontrolled-diabetes class D_u is reduced most substantially, the controlled class D_c increases most strongly, and the complication classes C_T and C_S remain furthest below baseline. Hence the simultaneous use of prevention, management, and relapse-prevention yields the largest reduction in severe outcomes over the study horizon.

TABLE 3. Comparison of optimal-control strategies over $T = 10$ years.

Strategy	J_{total}	J_{health}	J_{ctrl}	$\int_0^T C_S(t)\,dt$
S1: (u_1, u_2)	5.1986×10^3	5.0896×10^3	1.0904×10^2	2.5695
S2: (u_1, u_3)	5.1979×10^3	5.0969×10^3	1.0097×10^2	2.6223
S3: (u_2, u_3)	4.2666×10^3	4.1772×10^3	8.9352×10^1	2.3835
S4: (u_1, u_2, u_3)	3.9097×10^3	3.7619×10^3	1.4784×10^2	2.2402

5.4.5. *Comparison of strategies.* Table 3 shows that Strategy 4 yields the smallest total objective value and the lowest cumulative severe-complication burden, although at the highest implementation cost. Among the two-control strategies, Strategy 3 performs best overall. Strategies 1

and 2 are comparatively close, with Strategy 2 giving a slightly smaller total objective value but Strategy 1 yielding a lower cumulative severe-complication burden. Thus, in the present parameter regime, direct intervention on the management–relapse loop has a stronger influence on severe outcomes than prevention alone, while the combined intervention remains the most effective strategy.

6. CONCLUSION

In this paper, we developed and analyzed a nonlinear stage-structured model for diabetes progression that distinguishes between uncontrolled and controlled diabetes and incorporates relapse, complication development, and bounded lifestyle-related amplification. The analysis shows that the model is mathematically well posed: solutions exist globally, remain nonnegative, and evolve in a bounded positively invariant region. We also established the existence of a positive equilibrium, proved uniform persistence of the downstream disease classes, and derived conditions for local asymptotic stability together with a sufficient condition for global asymptotic stability.

A central conclusion of the study is that the distinction between uncontrolled and controlled diabetes is not only clinically relevant but also mathematically decisive for the long-term dynamics. In particular, the balance between management and relapse strongly influences the distribution of individuals across diabetes states and the resulting burden of treatable and severe complications. The upstream–downstream decomposition further clarifies how sustained inflow from prediabetes and lifestyle-related risk exposure drives the asymptotic behavior of the complication classes.

The numerical results support the analytical findings by showing bounded long-term dynamics, persistence of the downstream classes, and the dominant influence of progression-related parameters on severe-complication burden. In the optimal-control extension, the simultaneous use of prevention, management, and relapse-prevention produced the strongest reduction in uncontrolled diabetes and cumulative severe complications over the study horizon.

Overall, the work provides a mathematically tractable and biologically meaningful framework for studying diabetes progression with temporary control and relapse. Its relevance lies in showing how qualitative dynamical analysis and optimal-control methods can be combined

to identify mechanisms that shape long-term complication burden. Future work may extend the framework to coupled cardiometabolic systems, particularly diabetes–hypertension interactions, or to models with additional heterogeneity and data-informed parameter estimation.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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