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Modeling the dynamics of type 2 diabetes with modifiable lifestyle influence

Opeyemi Odetunde[a,*], Tunde Alesinloye[b], Omoniyi Francis[c]

[a]Department of Mathematics, University of Ilorin, Ilorin, Nigeria
[b]Department of Mathematics, University of Wyoming, Laramie, WY 82072, USA
[c]Department of Mathematics, University of Alabama at Birmingham, Birmingham, AL 35294, USA

Abstract

Type 2 diabetes mellitus (T2DM) is a multifactorial metabolic disorder driven by the interaction of insulin resistance, β-cell dysfunction, and modifiable lifestyle and environmental factors. Despite substantial advances in treatment, the global burden of T2DM continues to increase, highlighting the need for integrative frameworks that capture both biological and behavioral drivers of disease progression. In this study, we develop and analyze a deterministic compartmental model that incorporates genetic susceptibility, environmental exposure, diagnosis, treatment, and a modifiable lifestyle pressure variable representing socioeconomic and behavioral influences. The model is formulated as a system of nonlinear ordinary differential equations and analyzed using standard qualitative techniques, including positivity, boundedness, and stability analysis. We establish key dynamical properties and show that a disease-free equilibrium exists only under restrictive conditions, whereas an endemic equilibrium persists when environmental pressure and disease conversion are active. A semi-analytical solution obtained via the differential transformation method (DTM) is used to explore system dynamics and intervention scenarios. The results indicate that reducing lifestyle pressure, improving awareness, and increasing treatment initiation significantly reduce disease prevalence. Specifically, lowering lifestyle pressure or enhancing its dissipation reduces the equilibrium burden, while increasing awareness limits transitions into disease states and reduces overall disease spread.

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1. Introduction

Diabetes comprises three principal categories: type 1 diabetes (T1DM), type 2 diabetes mellitus (T2DM), and gestational diabetes (GDM). T2DM is characterised by chronic hyperglycaemia resulting from insulin resistance and progressive β-cell dysfunction [1, 2]. Unlike T1DM, which is autoimmune in origin, T2DM is strongly associated with lifestyle and social determinants such as diet quality, physical inactivity, and adiposity, although genetic predisposition and ageing also contribute. If poorly managed, T2DM leads to severe complications, including cardiovascular disease, nephropathy, neuropathy, and retinopathy. It remains a leading cause of mortality in the United States [3]. Globally, approximately 537 million adults were living with diabetes in 2021, with projections rising to 783 million by 2045 due to urbanization, dietary transitions, and declining physical activity levels according to Refs. [4–6].

Pharmacological management of T2DM has evolved significantly over the past several decades. Early treatments included sulfonylureas, which stimulate insulin secretion but are associated with risks such as weight gain and hypoglycaemia, and

*Corresponding author Tel. No.: +17013147828
Email address: talesin1@uwyo.edu (Opeyemi Odetunde)

metformin, which remains a first-line therapy due to its efficacy and favourable safety profile according to Ref. [7]. Insulin analogs improved basal and prandial profiles compared to human insulin. Since the 2000s, newer classes have expanded treatment options. More recently, additional drug classes have been introduced, including thiazolidinediones as reported by Ref. [8], DPP-4 inhibitors according to Ref. [9], GLP-1 receptor agonists [10], and SGLT2 inhibitors [11], many of which provide cardiovascular and renal benefits. Machine and deep learning algorithm have also been developed by several authors, including Fregoso-Aparicio *et al.*, [12], Kavakiotis *et al.*, [13] to understand the dynamics of T2DM. Dada *et al.*, [14] also considered the application of machine learning as cost-effective option for diabetes detection.

In addition to pharmacological interventions, lifestyle factors play a critical role in the development and progression of T2DM. These factors include dietary habits, physical activity, stress levels, and broader socioeconomic conditions [15–17]. Although both preventive and treatment strategies exist, the population at risk continues to expand. This trend underscores the importance of examining lifestyle and environmental influences alongside biological mechanisms. In this study, we focus on identifying factors that undermine the effectiveness of interventions and contribute to the persistence of T2DM. Specifically, we investigate the roles of genetic predisposition and modifiable lifestyle influences within a mathematical modeling framework.

Mathematical modeling provides a systematic approach for integrating biological processes, clinical outcomes, and population dynamics within a unified analytical framework [18–20]. Differential equation models have been widely used to describe glucose–insulin interactions, β-cell dynamics, and disease progression pathways [10]. Beyond physiological modeling, such approaches enable the evaluation of hypothetical scenarios, optimization of intervention strategies, and assessment of public health trade-offs in situations where randomized trials are impractical according to De Paola *et al.*, [21]. In this work, we adopt a compartmental modeling approach that distinguishes between genetically and non-genetically susceptible populations and explicitly incorporates exposure, diagnosis, treatment, and a modifiable lifestyle "pressure" variable.

Recent studies emphasize the importance of incorporating population structure and behavioral factors into models of noncommunicable diseases. Global trends indicate a sustained increase in diabetes prevalence over several decades, driven by demographic shifts, urbanization, and lifestyle changes [22–24]. Although adherence to healthy behaviors significantly reduces disease risk, such dynamics remain underrepresented in many mathematical models [18, 21, 25]. While diabetes is not infectious, compartmental modeling frameworks can still provide valuable insights into system stability and sensitivity to intervention parameters. These tools allow researchers to assess whether small perturbations in key variables lead to amplification or decay of disease burden [21, 26].

Existing studies have examined individual factors such as heredity, physical activity, and dietary behavior [18, 21]. However, to the best of our knowledge, no deterministic mathematical model has simultaneously integrated genetic susceptibility and a comprehensive set of lifestyle-related pressures into a unified analytical framework. This study addresses this gap by representing heredity as a risk factor and aggregating lifestyle influences, such as physical inactivity, stress, and dietary patterns, into a measurable "pressure" variable. This formulation provides a tractable and policy-relevant tool for analyzing screening, prevention, and treatment strategies.

Building on this framework, we develop a compartmental model that explicitly captures the interaction between biological susceptibility and modifiable environmental pressures. The model is designed to address policy-relevant questions, including how reductions in environmental pressure and improvements in awareness influence equilibrium behavior, system stability, and long-term disease outcomes.

2. Model formulation

In this section, we present a deterministic compartmental model for the progression of type 2 diabetes mellitus (T2DM) that incorporates genetic susceptibility, lifestyle behavior, treatment dynamics, and socioeconomic influences. Unlike classical SEIR-type models designed for infectious diseases, the proposed framework is adapted to capture the chronic and noncommunicable nature of T2DM. In particular, the model integrates a modifiable lifestyle "pressure" variable to represent environmental and behavioral drivers of disease progression.

2.1. The states and transitions in the diabetes model

The total population is partitioned into six mutually exclusive compartments representing distinct biological and behavioral states:

- $S_d(t)$: Genetically susceptible individuals with a family history or inherited predisposition to T2DM.
- $S_u(t)$: Non-genetically susceptible individuals without a family history, but still at risk due to environmental exposure.
- $E(t)$: Exposed individuals who have encountered risk factors but have not yet developed T2DM.
- $I(t)$: Individuals diagnosed with T2DM who are not currently receiving treatment.
- $T(t)$: Individuals undergoing treatment (e.g., pharmacological interventions such as tirzepatide).
- $P(t)$: Lifestyle pressure variable representing environmental and socioeconomic influences, including diet, stress, and physical inactivity.

Every individual in the population is assumed to be in one of these compartments at any time t, and transitions occur due to various biological and behavioral influences.

2.2. Assumptions of the model

The key assumptions are as follows:

1. Individuals in S_d and S_u become exposed through interaction with the lifestyle pressure variable P.
2. Exposed individuals progress to the diabetic state at a rate influenced by both lifestyle pressure and awareness/compliance level τ.
3. A fraction of individuals in the infected class I initiates treatment at rate η, while the remainder remains untreated.
4. Treatment reduces disease severity but does not guarantee full recovery.
5. Lifestyle pressure P increases due to adverse socioeconomic conditions and decreases through behavioral improvements and natural dissipation.
6. Natural mortality μ applies to all compartments, while additional mortality rates are associated with untreated (δ_1) and treated (δ_2) individuals with $\delta_2 < \delta_1$.

2.3. Model equations

Let

$$N(t) = S_d(t) + S_u(t) + E(t) + I(t) + T(t). \tag{1}$$

denote the total population at time t. The dynamics of the compartments are governed by the following system of nonlinear differential equations:

$$\begin{cases} \dfrac{dS_d}{dt} = \Lambda_1 - \beta P S_d - \mu S_d, \\ \dfrac{dS_u}{dt} = \Lambda_2 - \sigma\beta P S_u - \mu S_u, \\ \dfrac{dE}{dt} = \beta P S_d + \sigma\beta P S_u - P(\omega - \tau)E - \mu E, \\ \dfrac{dI}{dt} = P(\omega - \tau)E - (\eta + \mu + \delta_1)I, \\ \dfrac{dT}{dt} = \eta I - \delta_2 T - \mu T, \\ \dfrac{dP}{dt} = \phi N - (\gamma_1 + \gamma_2)P. \end{cases} \tag{2}$$

Here, all parameters are assumed to be non-negative constants. The condition $\delta_2 < \delta_1$ reflects the reduced mortality risk among treated individuals compared to untreated individuals.

2.4. Model parameters

The model parameters and their interpretations are summarized in Table 1. Each parameter represents a biologically or socially meaningful rate governing transitions between compartments.

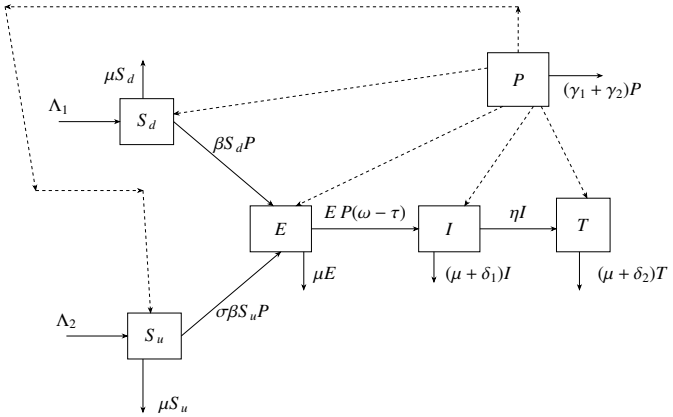


Figure 1: Compartmental flow diagram with labelled processes. Solid arrows denote population flows; dashed arrows denote influences proportional to P.

Table 1: Model parameters for the six-compartment system.

Symbol	Name	Value
Λ_1	Inflow to S_d	0.0044 [15]
Λ_2	Inflow to S_u	0.0066 [15]
μ	Natural mortality rate	0.011 [17]
β	Exposure rate (pressure-modulated)	0.00936 [27]
σ	Relative susceptibility of S_u	0.800 [28]
ω	Pressure effect on $E \to I$	0.0800 [5]
η	Treatment initiation rate	0.4500 [5]
τ	Awareness/compliance level	0.0700 [6]
ϕ	Lifestyle & physical activity effect on T2DM	0.0006 [16]
γ_1	Influence of socioeconomic status	0.0030 [Assumed]
γ_2	Influence of physical activity	0.0010 [Assumed]
δ_1	Death from diabetes-related complications from I	0.0040 [3]
δ_2	Death from diabetes-related complications from T	0.0020 [3]

Table 2: Initial conditions (sum to 980).

Compartment	Symbol	Initial Value
Genetically susceptible	$S_d(0)$	500
Non-genetically susceptible	$S_u(0)$	385
Exposed (pre-diagnosis)	$E(0)$	50
Diagnosed/infected	$I(0)$	30
In treatment	$T(0)$	25
Lifestyle pressure impact	$P(0)$	10%
Total		980

3. Model analysis

In this section, we investigate the qualitative properties of the proposed model. Specifically, we establish the positivity and boundedness of solutions, characterize the disease-free equilibrium (DFE), and analyze its feasibility and stability. We

also derive conditions for the existence of an endemic equilibrium.

3.1. Positivity and boundedness of solutions

Lemma [Positivity and Boundedness of Solutions]: Let the initial conditions satisfy

$$S_d(0),\ S_u(0),\ E(0),\ I(0),\ T(0) \geq 0,$$

and define the total population as in (1). Then the solutions $S_d(t), S_u(t), E(t), I(t), T(t)$ remain non-negative for all $t > 0$. Moreover, if $\mu > \phi$, the total population satisfies

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda_1 + \Lambda_2}{\mu - \phi}.$$

Proof. (i) Positivity: On each coordinate hyperplane, the vector field points inward:

$$\begin{aligned}
S_d = 0 &\Rightarrow \frac{dS_d}{dt} = \Lambda_1 \geq 0,\\
S_u = 0 &\Rightarrow \frac{dS_u}{dt} = \Lambda_2 \geq 0,\\
E = 0 &\Rightarrow \frac{dE}{dt} = \beta P S_d + \sigma\beta P S_u \geq 0,\\
I = 0 &\Rightarrow \frac{dI}{dt} = P(\omega - \tau)E \geq 0,\\
T = 0 &\Rightarrow \frac{dT}{dt} = \eta I \geq 0.
\end{aligned}$$

Thus, by standard invariance results, all components remain non-negative.

(ii) Boundedness: Summing all six equations in (2) gives:

$$\frac{dN}{dt} = \Lambda_1 + \Lambda_2 + \phi N - \mu N - \delta_1 I - \delta_2 T.$$

Since $\delta_1 I \geq 0$, $\delta_2 T \geq 0$, we have:

$$\frac{dN}{dt} \leq \Lambda_1 + \Lambda_2 - (\mu - \phi)N.$$

If $\mu > \phi$, solving this linear inequality gives:

$$N(t) \leq \frac{\Lambda_1 + \Lambda_2}{\mu - \phi} + \left(N(0) - \frac{\Lambda_1 + \Lambda_2}{\mu - \phi}\right) e^{-(\mu-\phi)t}.$$

Therefore:

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda_1 + \Lambda_2}{\mu - \phi}.$$

Hence, the feasible region

$$\Omega = \left\{ (S_d, S_u, E, I, T) \in \mathbb{R}^6_+ \;\middle|\; 0 \leq N(t) \leq \frac{\Lambda_1 + \Lambda_2}{\mu - \phi} \right\}.$$

is positively invariant whenever $\mu > \phi$. □

3.2. Disease-free equilibrium (DFE) state

We call a steady state "disease-free" if $E^* = I^* = T^* = 0$. Equating the left-hand side of (2) to zero, we have:

$$\begin{aligned}
0 &= \Lambda_1 - \beta P S_d - \mu S_d,\\
0 &= \Lambda_2 - \sigma\beta P S_u - \mu S_u,\\
0 &= \beta P S_d + \sigma\beta P S_u - \mu E - EP(\omega - \tau),\\
0 &= EP(\omega - \tau) - (\mu + \delta_1 + \eta)I,\\
0 &= \eta I - (\mu + \delta_2)T,\\
0 &= \phi N - (\gamma_1 + \gamma_2)P.
\end{aligned}$$

3.3. DFE feasibility

At equilibrium, if $P^* > 0$ (which typically holds when $dP/dt = \phi N - (\gamma_1 + \gamma_2)P$ with $\phi > 0$ and $N^* > 0$), then

$$\beta P^*(S_d^* + \sigma S_u^*) > 0.$$

unless $S_d^* = S_u^* = 0$. Consequently, to admit a DFE with $E^* = I^* = 0$, one needs either

$$P^* = 0 \quad \text{(e.g. } \phi = 0\text{)}.$$

or

$$\omega \leq \tau \text{ (no net progression } E \to I\text{)}.$$

Otherwise, the system generically supports an endemic equilibrium with $E^*, I^* > 0$.

3.4. Non-existence of a DFE when $\phi > 0$

If $\phi > 0$ and $N^* > 0$, the P-equation at steady state gives

$$P^* = \frac{\phi}{\gamma_1 + \gamma_2} N^* > 0.$$

Then the inflow into E at $E^* = 0$ is

$$\beta P^*(S_d^* + \sigma S_u^*) > 0.$$

since $S_d^*, S_u^* > 0$. Hence $\dot{E} \neq 0$ at $(E^*, I^*, T^*) = (0, 0, 0)$, which contradicts the DFE condition. Therefore, with $\phi > 0$ (endogenous pressure generation), a genuine DFE does *not* exist.

3.5. DFE under special cases

A DFE exists under either of the following conditions:

1. No pressure generation: $\phi = 0$ (and thus $P^* = 0$ at steady state), or
2. No incidence: $\beta = 0$ (no exposure from pressure).

In both cases, we have

$$S_d^* = \frac{\Lambda_1}{\mu}, \qquad S_u^* = \frac{\Lambda_2}{\mu}, \qquad E^* = I^* = T^* = 0,$$

$$P^* = \begin{cases} 0, & \phi = 0, \\ \dfrac{\phi}{\gamma_1 + \gamma_2} \dfrac{\Lambda_1 + \Lambda_2}{\mu}, & \beta = 0. \end{cases} \tag{3}$$

Note that when $\beta = 0$ and $\phi > 0$, $P^* > 0$ but there is no disease inflow (since incidence is off), so $(E^*, I^*, T^*) = (0, 0, 0)$ is a valid disease-free steady state.

$$\Phi(S_d^*, S_u^*, E^*, I^*, T^*) = \Phi\Big(\frac{\Lambda_1}{\mu}, \frac{\Lambda_2}{\mu}, 0, 0, 0\Big).$$

3.6. Local stability of the disease-free equilibrium

Let the state vector be

$$\mathbf{x} = (S_d, S_u, E, I, T, P)^\top,$$

so that the model system can be written in the compact form

$$\dot{\mathbf{x}} = \mathbf{F}(\mathbf{x}).$$

The Jacobian matrix of the system is

$$J(\mathbf{x}) = \left[\frac{\partial F_i}{\partial x_j}\right]_{i,j=1}^{6}.$$

For the model in (2), the Jacobian is given explicitly by

$$J(\mathbf{x}) = \begin{pmatrix} -(\beta P+\mu) & 0 & 0 & 0 & 0 & -\beta S_d \\ 0 & -(\sigma\beta P+\mu) & 0 & 0 & 0 & -\sigma\beta S_u \\ \beta P & \sigma\beta P & \frac{-(\mu+}{P(\omega-\tau))} & 0 & 0 & \frac{\beta S_d+\sigma\beta S_u}{-(\omega-\tau)E} \\ 0 & 0 & P(\omega-\tau) & -(\mu+\delta_1+\eta) & 0 & (\omega-\tau)E \\ 0 & 0 & 0 & \eta & -(\mu+\delta_2) & 0 \\ \phi & \phi & \phi & \phi & \phi & -(\gamma_1+\gamma_2) \end{pmatrix}. \quad (4)$$

As shown above, a genuine disease-free equilibrium does not exist when $\phi > 0$ and $\beta > 0$. Therefore, the local stability analysis of the disease-free equilibrium is carried out only in the two feasible special cases:

(A) $\phi = 0$, and

(B) $\beta = 0$.

Case (A): $\phi = 0$

When $\phi = 0$, the disease-free equilibrium is

$$E_0^{(A)} = \left(\frac{\Lambda_1}{\mu}, \frac{\Lambda_2}{\mu}, 0, 0, 0, 0\right).$$

Evaluating (4) at $E_0^{(A)}$ gives

$$J(E_0^{(A)}) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & -\frac{\beta\Lambda_1}{\mu} \\ 0 & -\mu & 0 & 0 & 0 & -\frac{\sigma\beta\Lambda_2}{\mu} \\ 0 & 0 & -\mu & 0 & 0 & \beta\left(\frac{\Lambda_1+\sigma\Lambda_2}{\mu}\right) \\ 0 & 0 & 0 & -(\mu+\delta_1+\eta) & 0 & 0 \\ 0 & 0 & 0 & \eta & -(\mu+\delta_2) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\gamma_1+\gamma_2) \end{pmatrix}. \quad (5)$$

The matrix in (5) is block upper triangular. Hence, its eigenvalues are the diagonal entries:

$$\lambda_1 = \lambda_2 = \lambda_3 = -\mu\,, \lambda_4 = -(\mu+\delta_1+\eta),$$

$$\lambda_5 = -(\mu+\delta_2), \lambda_6 = -(\gamma_1+\gamma_2),$$

Since all eigenvalues are strictly negative, it follows that $E_0^{(A)}$ is locally asymptotically stable when $\phi = 0$.

Case (B): $\beta = 0$

When $\beta = 0$, the disease-free equilibrium is

$$E_0^{(B)} = \left(\frac{\Lambda_1}{\mu}, \frac{\Lambda_2}{\mu}, 0, 0, 0, \frac{\phi}{\gamma_1+\gamma_2}\cdot\frac{\Lambda_1+\Lambda_2}{\mu}\right).$$

Let

$$P^* = \frac{\phi}{\gamma_1+\gamma_2}\cdot\frac{\Lambda_1+\Lambda_2}{\mu}.$$

Then the Jacobian at $E_0^{(B)}$ becomes

$$J(E_0^{(B)}) = \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu+P^*(\omega-\tau)) & 0 & 0 & 0 \\ 0 & 0 & (\omega-\tau)P^* & -(\mu+\delta_1+\eta) & 0 & 0 \\ 0 & 0 & 0 & \eta & -(\mu+\delta_2) & 0 \\ \phi & \phi & \phi & \phi & \phi & -(\gamma_1+\gamma_2) \end{pmatrix}. \quad (6)$$

Again, (6) is block triangular, so the eigenvalues are

$$\lambda_1 = \lambda_2 = -\mu, \qquad \lambda_3 = -(\mu+\delta_1+\eta),$$

$$\lambda_4 = -(\mu+P^*(\omega-\tau)), \qquad \lambda_5 = -(\mu+\delta_2), \quad \lambda_6 = -(\gamma_1+\gamma_2).$$

Therefore, provided $\mu+P^*(\omega-\tau) > 0$, all eigenvalues have negative real parts, and hence $E_0^{(B)}$ is locally asymptotically stable when $\beta = 0$. In particular, this condition is automatically satisfied when $\omega \geq \tau$, and more generally whenever $P^*(\tau-\omega) < \mu$.

Interpretation:

The parameter β measures how efficiently environmental pressure P *converts* into new diabetes cases. In this case, people are recruited into the P-compartment under the assumption that they are well-to-do and cognizant of the importance of physical activity for DM. If $\beta = 0$, no interaction within the population (contact = 0), pressure cannot generate new cases, even if P persists (any $\phi \geq 0$: for $\phi \neq 0$, population is aware of the importance of physical activity and other socioeconomic status on DM). The DFE is therefore *locally asymptotically stable*: small numbers of cases tend to fade rather than grow.

The analysis shows that, when risk is prevented from progressing to new diabetes cases through prevention programs, effective medications, and strong follow-up, the community exhibits increased resilience. Even if unhealthy pressures persist, they do not translate into new cases. This study, therefore, emphasizes the importance of screening, lifestyle interventions, and access to proven treatments.

3.7. Recommendation: How to push β toward zero (practical levers)

Clinical & behavioral prevention (most direct effect on β)..

- Scale intensive lifestyle programs (DPP-style): diet quality, physical activity, and weight loss.
- Treat pre-diabetes/obesity effectively: evidence-based anti-obesity medications (e.g., GLP-1/GIP agents such as tirzepatide), metformin in appropriate pre-diabetes groups, and bariatric/metabolic surgery when indicated.

- Tighten cardio-metabolic risk management: blood pressure, lipids, sleep, smoking cessation, and stress reduction.
- Improve screening & follow-up: systematic A1c/FPG screening, fast-track referral into prevention clinics, and culturally tailored coaching.

Systems & access (enable low β at scale)..

- Coverage and affordability for prevention programs and effective medicines.
- Closed-loop care pathways from screening → referral → retention.
- Community partnerships (Community centres, employers, schools) to boost retention and adherence.

3.8. Endemic equilibrium (EE) state

Let the endemic equilibrium be denoted by

$$E^* = (S_d^*, S_u^*, E^*, I^*, T^*, P^*).$$

Define

$$r_1 := \mu + \beta P^*, \quad r_2 := \mu + \sigma\beta P^*, \quad r_3 := \mu + P^*(\omega - \tau),$$
$$r_4 := \mu + \delta_1 + \eta, \quad r_5 := \mu + \delta_2, \quad r_6 := \gamma_1 + \gamma_2.$$

$$\begin{cases} 0 = \Lambda_1 - r_1 S_d^*, \\ 0 = \Lambda_2 - r_2 S_u^*, \\ 0 = \beta P^* S_d^* + \sigma\beta P^* S_u^* - r_3 E^*, \\ 0 = (\omega - \tau) P^* E^* - r_4 I^*, \\ 0 = \eta I^* - r_5 T^*, \\ 0 = \phi(S_d^* + S_u^* + E^* + I^* + T^*) - r_6 P^*. \end{cases} \tag{7}$$

Then the steady states can be written as

$$\begin{aligned} S_d^* &= \frac{\Lambda_1}{r_1}, \\ S_u^* &= \frac{\Lambda_2}{r_2}, \\ S^* &= S_d^* + S_u^* = \frac{\Lambda_1 r_2 + \Lambda_2 r_1}{r_1 r_2}, \\ E^* &= \frac{\beta P^*}{r_3}\left(\frac{\Lambda_1}{r_1} + \frac{\sigma\Lambda_2}{r_2}\right) = \frac{\beta P^*(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3}, \\ I^* &= \frac{(\omega - \tau)P^*}{r_4} E^* = \frac{\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4}, \\ T^* &= \frac{\eta}{r_5} I^* = \frac{\eta\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4 r_5}. \end{aligned} \tag{8}$$

The total headcount (excluding P) is

$$\begin{aligned} N^*(P^*) &= S^* + E^* + I^* + T^* \\ &= \frac{\Lambda_1 r_2 + \Lambda_2 r_1}{r_1 r_2} + \frac{\beta P^*(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3} \\ &\quad + \frac{\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4} \\ &\quad + \frac{\eta\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4 r_5}. \end{aligned}$$

Finally,

$$P^* = \frac{\phi}{r_6} N^*(P^*).$$

is a scalar fixed-point equation determining P^*; once P^* is found, all other components follow the formulas above.

$$\Phi(S, E, I, T, P) \equiv \Phi\Big(\tfrac{\Lambda_1 r_2 + \Lambda_2 r_1}{r_1 r_2}, \tfrac{\beta P^*(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3}, \tfrac{\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4}, \tfrac{\eta\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4 r_5}, \tfrac{\phi}{r_6} N^*(P^*)\Big).$$

Existence of P^* (Impact of Community rate on T2DM in the EE state)

From the steady-state equation for the pressure variable P, we have

$$(\gamma_1 + \gamma_2)P^* = \phi N^*(P^*).$$

Define

$$F(P) := P - \frac{\phi}{\gamma_1 + \gamma_2} N^*(P).$$

Observe that

$$F(0) = -\frac{\phi}{\gamma_1 + \gamma_2}\frac{\Lambda_1 + \Lambda_2}{\mu} < 0,$$

and since $N^*(P)$ is bounded for all $P \geq 0$, it follows that

$$F(P) \to +\infty \quad \text{as} \quad P \to \infty.$$

By continuity of $F(P)$, there exists at least one $P^* > 0$ such that $F(P^*) = 0$. Substituting P^* into the equilibrium expressions yields a biologically feasible endemic equilibrium

$$(S_d^*, S_u^*, E^*, I^*, T^*).$$

3.9. Jacobian at the EE state

Evaluating (4) at E^* gives

$$J(E^*) = \begin{pmatrix} -r_1 & 0 & 0 & 0 & 0 & a_{16} \\ 0 & -r_2 & 0 & 0 & 0 & a_{26} \\ a_{31} & a_{32} & -r_3 & 0 & 0 & a_{36} \\ 0 & 0 & a_{43} & -r_4 & 0 & a_{46} \\ 0 & 0 & 0 & \eta & -r_5 & 0 \\ \phi & \phi & \phi & \phi & \phi & -r_6 \end{pmatrix}. \tag{9}$$

The characteristic polynomial associated with $J(E^*)$ is

$$\chi(\lambda) = \lambda^6 + A_1\lambda^5 + A_2\lambda^4 + A_3\lambda^3 + A_4\lambda^2 + A_5\lambda + A_6.$$

Local stability of the endemic equilibrium

The endemic equilibrium E^* is locally asymptotically stable if the Routh-Hurwitz conditions associated with the above characteristic polynomial are satisfied. Due to the complexity of these conditions for a sixth-degree polynomial, the explicit inequalities are omitted here and provided in Appendix A. The coefficients $A_1, \ldots, A_6$ are functions of the model parameters and equilibrium values, obtained from the Jacobian matrix $J(E^*)$.

Interpretation:

Local stability implies that all eigenvalues of the Jacobian have negative real parts, so small perturbations around the endemic equilibrium decay over time. Epidemiologically, this indicates that the system returns to a persistent level of disease burden unless key parameters, particularly those related to lifestyle pressure or treatment are significantly altered.

3.10. Threshold analysis via the endemic equilibrium (special case)

In classical epidemiological models, the basic reproduction number R_0 serves as a threshold parameter that determines whether a disease invades or is eliminated. However, for the present system, which describes a non-communicable disease driven by endogenous lifestyle pressure $P(t)$, a disease-free equilibrium does not generally exist when $\phi > 0$. The exposed class $E(t)$ represents individuals at elevated risk who have not yet developed diabetes and are therefore not included in the disease burden. In the present model, exposure is assumed to be irreversible, so individuals in $E(t)$ either progress to diabetes or exit through natural mortality. Consequently, we formulate a threshold condition based on the system's endemic state.

Let the total disease burden be defined as

$$D(t) = I(t) + T(t).$$

Differentiating and using the governing system yields

$$\frac{dD}{dt} = \frac{dI}{dt} + \frac{dT}{dt} = P(t)(\omega - \tau)E(t) - (\mu + \delta_1)I(t) - (\mu + \delta_2)T(t).$$

This expression separates the inflow into the disease classes from the outflow due to mortality and treatment-related removal.

Endemic threshold quantity

We define the endemic threshold quantity $\mathcal{R}_D$ as

$$\mathcal{R}_D = \frac{P^*(\omega - \tau)E^*}{(\mu + \delta_1)I^* + (\mu + \delta_2)T^*},$$

where $(S_d^*, S_u^*, E^*, I^*, T^*, P^*)$ denotes the endemic equilibrium of the system.

Threshold condition

The sign of $\frac{dD}{dt}$ determines the evolution of the disease burden. In particular,

$$\frac{dD}{dt} > 0 \iff P(t)(\omega - \tau)E(t) > (\mu + \delta_1)I(t) + (\mu + \delta_2)T(t),$$

$$\frac{dD}{dt} < 0 \iff P(t)(\omega - \tau)E(t) < (\mu + \delta_1)I(t) + (\mu + \delta_2)T(t).$$

At equilibrium, the balance condition is given by

$$P^*(\omega - \tau)E^* = (\mu + \delta_1)I^* + (\mu + \delta_2)T^*,$$

which corresponds to

$$\mathcal{R}_D = 1.$$

Thus, $\mathcal{R}_D$ acts as a threshold parameter governing whether the disease burden will persist in the immediate generation after the disease carrier. In the absence of an improved modifiable lifestyle, the next generation will certainly carry the disease.

Explicit form of the threshold

Using the equilibrium expressions derived previously, we substitute

$$E^* = \frac{\beta P^*(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3},$$

$$I^* = \frac{\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4},$$

$$T^* = \frac{\eta\beta(\omega - \tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4 r_5}.$$

Substituting into $\mathcal{R}_D$, we have

$$\mathcal{R}_D = \frac{P^*(\omega - \tau)^{\frac{\beta P^*(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3}}}{(\mu + \delta_1)^{\frac{\beta(\omega-\tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4}}} + \frac{P^*(\omega - \tau)^{\frac{\beta P^*(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3}}}{(\mu + \delta_2)^{\frac{\eta\beta(\omega-\tau)P^{*2}(\Lambda_1 r_2 + \sigma\Lambda_2 r_1)}{r_1 r_2 r_3 r_4 r_5}}}.$$

Simplifying, we obtain

$$\mathcal{R}_D = \frac{r_4 r_5}{(\mu + \delta_1)r_5 + (\mu + \delta_2)\eta}.$$

3.10.1. Parameter dependence

Although the simplified threshold quantity

$$\mathcal{R}_D = \frac{r_4 r_5}{(\mu + \delta_1)r_5 + (\mu + \delta_2)\eta},$$

depends explicitly only on the removal-related parameters μ, δ_1, δ_2, and η, several epidemiological parameters continue to influence the endemic equilibrium levels and therefore affect the overall disease burden.

- The The lifestyle-pressure parameter ϕ increases the steady-state pressure level P^* through

$$P^* = \frac{\phi}{r_6} N^*(P^*),$$

which increases the equilibrium values E^*, I^*, and T^*, thereby increasing the endemic burden.

- The exposure rate β directly increases the generation of the exposed class E^* and consequently raises the equilibrium levels of the infectious and treated classes I^* and T^*.
- The progression parameter $(\omega - \tau)$ governs the transition from exposure to infection. An increase in ω or a decrease in τ accelerates progression into the infectious class, leading to largr equilibrium values of I^* and T^*.Conversely increased awareness (larger τ) reduces disease progression.

Interpretation

The quantity $\mathcal{R}_D$ serves as an endemic threshold parameter analogous to the basic reproduction number in infectious disease models. Specifically,

$$\mathcal{R}_D > 1 \Rightarrow \text{increasing disease burden},$$

$$\mathcal{R}_D < 1 \Rightarrow \text{declining disease burden},$$

$$\mathcal{R}_D = 1 \Rightarrow \text{endemic equilibrium}.$$

3.10.2. Recommendation

- Upstream environment (push $\phi \downarrow$, $r_6 \uparrow$): improve healthy food access, active-transport and park infrastructure, and economic security; reduce exposure to obesogenic products; Invest in neighborhood safety. Track proxies for ϕ (food insecurity, healthy retail density, walkability, stress indices).
- Prevention and behavior (push $\beta \downarrow$, $(\omega - \tau) \downarrow$): scale Diabetes Prevention Program–style interventions; support weight management; intensify risk-factor control; expand culturally tailored screening, coaching, and follow-up to lift τ.
- Care delivery (push $\eta \uparrow$): close loops from screening $\rightarrow$ referral $\rightarrow$ retention; ensure coverage and access to evidence-based therapies; monitor time-to-treatment and retention as system metrics.

3.11. Semi-analytic solution of the model: DTM-transformed system

The Differential Transformation Method (DTM) was applied to the diabetes mellitus (DM) model developed in this work. This semi-analytic method efficiently approximates ordinary differential equations by constructing a rapidly converging power-series solution without the need for discretization or linearization.

DTM is a numerical and semi-analytical approach that was initially presented by Zhou in the context of electrical circuit analysis [29]; and widely adopted by several authors including Odetunde *et al.*, [30] The technique is particularly useful for initial value problems since it converts differential equations into recursive algebraic relations for the series coefficients.

Let the differential transforms be:

$$S_d(t) \rightarrow S_d(k), \quad S_u(t) \rightarrow S_u(k), \quad E(t) \rightarrow E(k), \quad I(t) \rightarrow I(k),$$
$$T(t) \rightarrow T(k), \quad P(t) \rightarrow P(k).$$

By applying the DTM on the model equation, the following recurrence relations are obtained:

$$(k+1)S_d(k+1) = \Lambda_1 \cdot \delta(k) - \beta \sum_{m=0}^{k} P(m)S_d(k-m) - \mu S_d(k),$$

$$(k+1)S_u(k+1) = \Lambda_2 \cdot \delta(k) - \sigma\beta \sum_{m=0}^{k} P(m)S_u(k-m) - \mu S_u(k),$$

$$(k+1)E(k+1) = \beta \sum_{m=0}^{k} P(m)S_d(k-m) + \sigma\beta \sum_{m=0}^{k} P(m)S_u(k-m) - (\omega - \tau) \sum_{m=0}^{k} P(m)E(k-m) - \mu E(k),$$

$$(k+1)I(k+1) = (\omega - \tau) \sum_{m=0}^{k} P(m)E(k-m) - (\eta + \mu + \delta_1)I(k),$$

$$(k+1)T(k+1) = \eta I(k) - (\delta_2 + \mu)T(k),$$

$$(k+1)P(k+1) = \phi N(k) - (\gamma_1 + \gamma_2)P(k),$$

where,

$$N(k) = S_d(k) + S_u(k) + E(k) + I(k) + T(k) + P(k).$$

In fractional form:

$$S_d(k+1) = \frac{1}{k+1}\left[\Lambda_1 \cdot \delta(k) - \beta \sum_{m=0}^{k} P(m)S_d(k-m) - \mu S_d(k)\right],$$

$$S_u(k+1) = \frac{1}{k+1}\left[\Lambda_2 \cdot \delta(k) - \sigma\beta \sum_{m=0}^{k} P(m)S_u(k-m) - \mu S_u(k)\right],$$

$$E(k+1) = \frac{1}{k+1}\left[\beta \sum_{m=0}^{k} P(m)S_d(k-m) + \sigma\beta \sum_{m=0}^{k} P(m)S_u(k-m) - (\omega - \tau) \sum_{m=0}^{k} P(m)E(k-m) - \mu E(k)\right], \quad (10)$$

$$I(k+1) = \frac{1}{k+1}\left[(\omega - \tau) \sum_{m=0}^{k} P(m)E(k-m) - (\eta + \mu + \delta_1)I(k)\right],$$

$$T(k+1) = \frac{1}{k+1}\left[\eta I(k) - (\delta_2 + \mu)T(k)\right],$$

$$P(k+1) = \frac{1}{k+1}\left[\phi N(k) - (\gamma_1 + \gamma_2)P(k)\right].$$

DTM series solutions up to t^{10}

$$S_d(t) = \sum_{k=0}^{10} S_d(k)t^k = 500.000000 - 52.295600\,t + 1.42465988\,t^2 + 0.04835378\,t^3 - 0.00367443\,t^4 + 4.02369\times10^{-5}\,t^5 + 2.66013\times10^{-6}\,t^6 - 8.30677\times10^{-8}\,t^7 - 8.16997\times10^{-10}\,t^8 + 8.99447\times10^{-11}\,t^9 - 1.96565\times10^{-12}\,t^{10},$$

$$S_u(t) = \sum_{k=0}^{10} S_u(k)t^k = 385.000000 - 33.057200\,t + 0.612269768\,t^2 + 0.03277892\,t^3 - 0.00162258\,t^4 + 5.74804\times10^{-6}\,t^5 + 1.14112\times10^{-6}\,t^6 - 2.09790\times10^{-8}\,t^7 - 5.41655\times10^{-10}\,t^8 + 2.99760\times10^{-11}\,t^9 - 6.85937\times10^{-13}\,t^{10},$$

$$E(t) = \sum_{k=0}^{10} E(k)t^k = 50.000000 + 70.078800\,t - 5.596862648\,t^2 - 0.01162017\,t^3 + 0.01397724\,t^4 - 0.000381787\,t^5 - 9.67339\times10^{-6}\,t^6 + 6.28371\times10^{-7}\,t^7 - 2.03974\times10^{-9}\,t^8 - 5.49435\times10^{-10}\,t^9 + 1.38135\times10^{-11}\,t^{10},$$

$$I(t) = \sum_{k=0}^{10} I(k)t^k = 30.000000 - 8.950000\,t + 5.724815\,t^2 - 0.94380577\,t^3 + 0.10084604\,t^4 - 0.00906198\,t^5 + 7.08791\times10^{-4}\,t^6 - 4.75991\times10^{-5}\,t^7 + 2.76937\times10^{-6}\,t^8 - 1.42651\times10^{-7}\,t^9 + 6.62256\times10^{-9}\,t^{10},$$

$$T(t) = \sum_{k=0}^{10} T(k)t^k = 25.000000 + 13.175000\,t - 2.0993875\,t^2 + 0.867819596\,t^3 - 0.108999\,t^4 + 0.00935954\,t^5 - 6.99927\times10^{-4}\,t^6 + 4.68650\times10^{-5}\,t^7 - 2.75360\times10^{-6}\,t^8 + 1.42446\times10^{-7}\,t^9 - 6.60446\times10^{-9}\,t^{10},$$

$$P(t) = \sum_{k=0}^{10} P(k)t^k = 10.000000 + 0.560000\,t - 0.00426670\,t^2 + 1.79345\times10^{-5}\,t^3 - 9.86291\times10^{-7}\,t^4 + 6.39945\times10^{-8}\,t^5 - 3.86010\times10^{-9}\,t^6 + 2.58296\times10^{-10}\,t^7 - 1.58407\times10^{-11}\,t^8 + 8.30763\times10^{-13}\,t^9 - 3.83250\times10^{-14}\,t^{10}.$$

A decrease in the number of infected individuals with higher intervention and treatment rates aligns with findings from the Diabetes Prevention Program Outcomes Study. This study showed that intensive lifestyle changes lowered diabetes incidence by 58% and metformin by 31% during the initial trial, with ongoing reductions of 34% and 18%, respectively, over a decade of follow-up [31]. These results offer robust empirical support for the model's projection that stronger intervention and treatment approaches significantly reduce both immediate and long-term disease impact.

3.12. Validation of DTM solution via RK4 method

To assess the accuracy of the Differential Transformation Method (DTM), the truncated DTM series solution (up to order t^{10}) is compared with a numerical solution obtained using the classical fourth-order Runge–Kutta (RK4) method. Figure 6 presents the trajectories of all model compartments over the interval $t \in [0, 10]$ showing close agreement between the DTM approximation and the RK4 solution. Minor deviations are observed in the infected $I(t)$ and treated $T(t)$ compartments over time. This behavior is expected, since the DTM solution is expressed as a truncated power series, which provides a local approximation and may lose accuracy outside its convergence region.

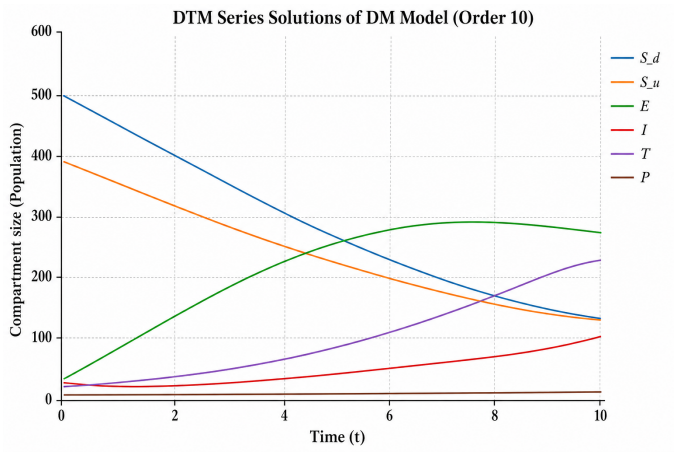


Figure 2: DTM series solutions (order t^{10}) for all compartments $S_d(t)$, $S_u(t)$, $E(t)$, $I(t)$, $T(t)$, and $P(t)$ over the interval $t \in [0, 10]$. The plot illustrates the overall temporal dynamics of the model, including the decline in susceptible populations and the evolution of exposed, infected, and treated individuals.

3.13. Error analysis of DTM approximation

To quantify the accuracy of the DTM solution, we computed the maximum absolute error and root mean square error (RMSE) between the DTM approximation and the RK4 numerical solution over $t \in [0, 10]$.

Table 5: Error metrics between DTM and RK4 solutions.

Variable	Maximum error	RMSE
S_d	1.80×10^{-3}	3.73×10^{-4}
S_u	1.28×10^{-3}	2.70×10^{-4}
E	2.28×10^{-3}	4.14×10^{-4}
I	2.00×10^{1}	4.25
T	1.99×10^{1}	4.24
P	1.16×10^{-4}	2.39×10^{-5}

The error metrics indicate that the DTM approximation closely matches the RK4 solution for the susceptible and exposed compartments. However, larger deviations are observed for the infected and treated compartments. This discrepancy is attributable to the nonlinear accumulation of approximation errors in higher-order terms, which is a known limitation of truncated series methods. Despite this, the overall agreement remains sufficiently accurate for qualitative and short-term quantitative analysis. These results confirm that the DTM provides a computationally efficient and reasonably accurate approach to analyzing system dynamics over a finite time horizon.

3.14. Interpretation of numerical results

The numerical simulations provide insight into the dynamic behavior of the model under baseline and intervention scenarios.

The results indicate that the susceptible populations $S_d(t)$ and $S_u(t)$ gradually decline over time, reflecting continuous exposure to lifestyle-related risk factors. Simultaneously, the ex-

Table 3: DTM coefficients for the new model (up to t^{10}).

k	$S_d(k)$	$S_u(k)$	$E(k)$	$I(k)$	$T(k)$	$P(k)$
0	500.000000	385.000000	50.000000	30.000000	25.000000	10.000000
1	-52.295600	-33.057200	70.078800	-8.950000	13.175000	0.560000
2	1.424660	0.612270	-5.596863	5.724815	-2.099388	-0.0042667
3	0.0483538	0.0327789	-0.0116202	-0.943806	0.867820	1.79345×10^{-5}
4	-0.00367443	-0.00162258	0.0139772	0.100846	-0.108999	-9.86291×10^{-7}
5	4.02369×10^{-5}	5.74804×10^{-6}	-0.000381787	-0.00906198	0.00935954	6.39945×10^{-8}
6	2.66013×10^{-6}	1.14112×10^{-6}	-9.67339×10^{-6}	7.08791×10^{-4}	-6.99927×10^{-4}	-3.86010×10^{-9}
7	-8.30677×10^{-8}	-2.09790×10^{-8}	6.28371×10^{-7}	-4.75991×10^{-5}	4.68650×10^{-5}	2.58296×10^{-10}
8	-8.16997×10^{-10}	-5.41655×10^{-10}	-2.03974×10^{-9}	2.76937×10^{-6}	-2.75360×10^{-6}	-1.58407×10^{-11}
9	8.99447×10^{-11}	2.99760×10^{-11}	-5.49435×10^{-10}	-1.42651×10^{-7}	1.42446×10^{-7}	8.30763×10^{-13}
10	-1.96565×10^{-12}	-6.85937×10^{-13}	1.38135×10^{-11}	6.62256×10^{-9}	-6.60446×10^{-9}	-3.83250×10^{-14}

Table 4: DTM series solutions evaluated at $t = 0, 0.01, \ldots, 0.1$.

t	$S_d(t)$	$S_u(t)$	$E(t)$	$I(t)$	$T(t)$	$P(t)$
0.00	500.000000	385.000000	50.000000	30.000000	25.000000	10.000000
0.01	499.477187	384.669489	50.700228	29.911072	25.131541	10.005600
0.02	498.954658	384.339101	51.399337	29.823282	25.262667	10.011198
0.03	498.432415	384.008836	52.097327	29.736627	25.393384	10.016796
0.04	497.910459	383.678694	52.794196	29.651100	25.523696	10.022393
0.05	497.388788	383.348675	53.489946	29.566695	25.653609	10.027989
0.06	496.867403	383.018779	54.184577	29.483407	25.783128	10.033585
0.07	496.346305	382.689007	54.878088	29.401230	25.912258	10.039179
0.08	495.825494	382.359359	55.570479	29.320160	26.041004	10.044773
0.09	495.304971	382.029835	56.261750	29.240190	26.169371	10.050365
0.10	494.784735	381.700435	56.951901	29.161314	26.297363	10.055957

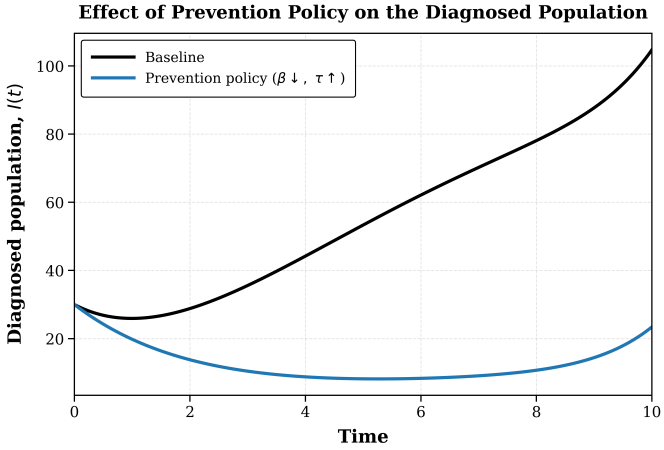


Figure 3: Impact of intervention policies on the diagnosed/infected population $I(t)$ over $t \in [0, 10]$. The policy scenario (modified β, ϕ, τ) shows a substantial reduction in infections compared to the baseline.

posed class $E(t)$ increases initially before stabilizing, indicating the accumulation of individuals at elevated risk.

The infected population $I(t)$ exhibits a decreasing trend under intervention scenarios, while the treated population $T(t)$ increases correspondingly. This behavior highlights the effectiveness of treatment initiation and intervention strategies in reducing disease burden.

The pressure variable $P(t)$ evolves dynamically and stabilizes at a positive level, reinforcing the conclusion that persistent environmental and socioeconomic factors sustain the disease in the population.

Overall, the simulations demonstrate that combined interventions targeting lifestyle pressure, awareness, and treatment significantly reduce the long-term prevalence of T2DM.

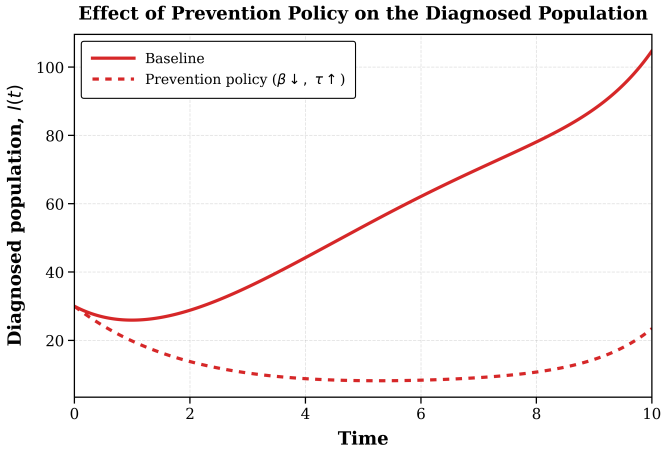


Figure 4: Impact of prevention and awareness interventions on $I(t)$ over $t \in [0, 10]$. The policy scenario (modified β, ϕ, τ) significantly reduces diagnosed/infected levels compared to the solution obtained using the original parameter values.

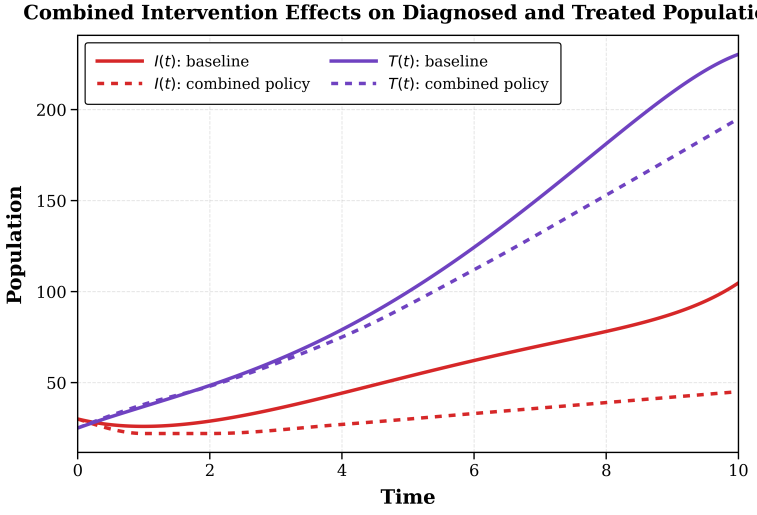


Figure 5: Joint effect of upstream, prevention, and clinical interventions on the infected $I(t)$ and treated $T(t)$ populations over $t \in [0, 10]$. The intervention scenario (modified $\beta, \phi, \tau, \gamma$) leads to a noticeable reduction in infection levels and a corresponding increase in treatment uptake compared to the model under original parameter values, highlighting the combined effectiveness of integrated control strategies.

4. Results

The proposed model was analyzed using standard qualitative techniques from mathematical analysis. The results confirm that the model provides a consistent and biologically meaningful representation of T2DM dynamics. In particular, the equilibrium structure captures transitional phases between increasing and decreasing trends in disease prevalence.

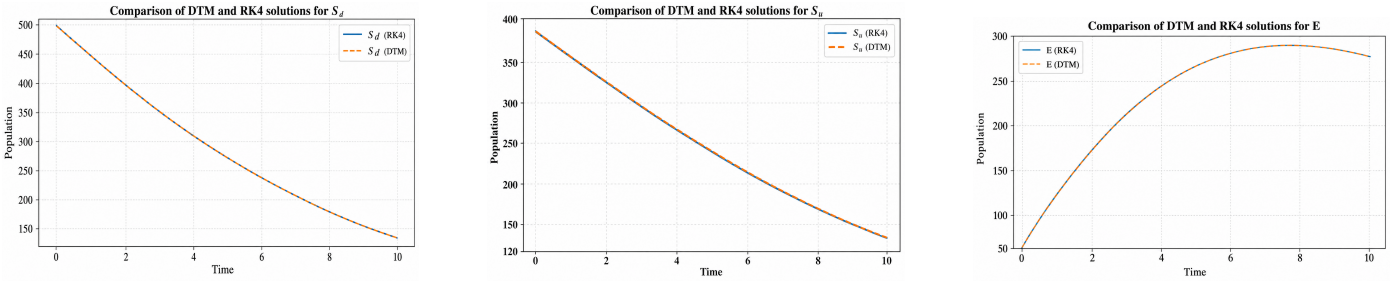
Due to the nonlinear nature of the system, obtaining a closed-form analytical solution is intractable. Therefore, the Differential Transformation Method (DTM) was employed to obtain a semi-analytical approximation of the system. The temporal evolution of each compartment was approximated using a truncated DTM series expansion up to order 10.

The solution trajectories for $t \in [0, 10]$ exhibit distinct and biologically consistent patterns (Figure 2). The genetically susceptible (S_d) and non-genetically susceptible (S_u) populations decrease gradually over time, reflecting natural mortality and transitions into the exposed class driven by lifestyle pressure.

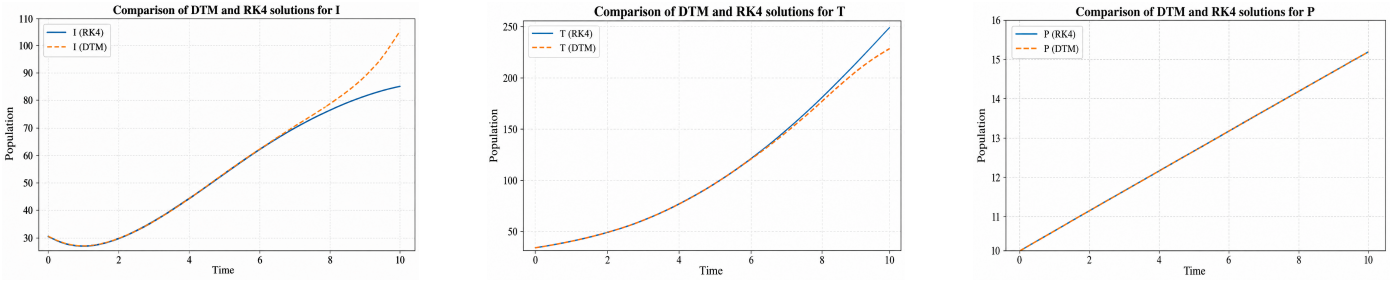
The exposed population (E) initially increases rapidly, reaches a peak, and subsequently stabilizes. This behavior indicates that exposure rises as susceptible individuals are influenced by lifestyle factors, but eventually levels off as the susceptible pool diminishes.

The diagnosed population (I) shows a modest initial increase before stabilizing at an intermediate level. This suggests that progression from exposure to diagnosis is balanced by treatment initiation and mortality effects. In contrast, the treatment class (T) increases steadily over time, with accelerated growth in later stages, reflecting increased treatment uptake in response to rising diagnoses.

The lifestyle pressure variable (P) remains relatively low

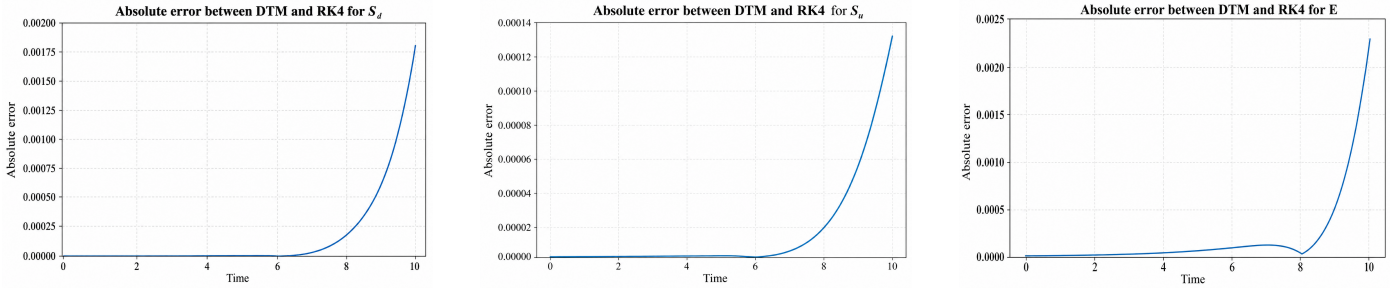


(a) $S_d(t)$ (b) $S_u(t)$ (c) $E(t)$

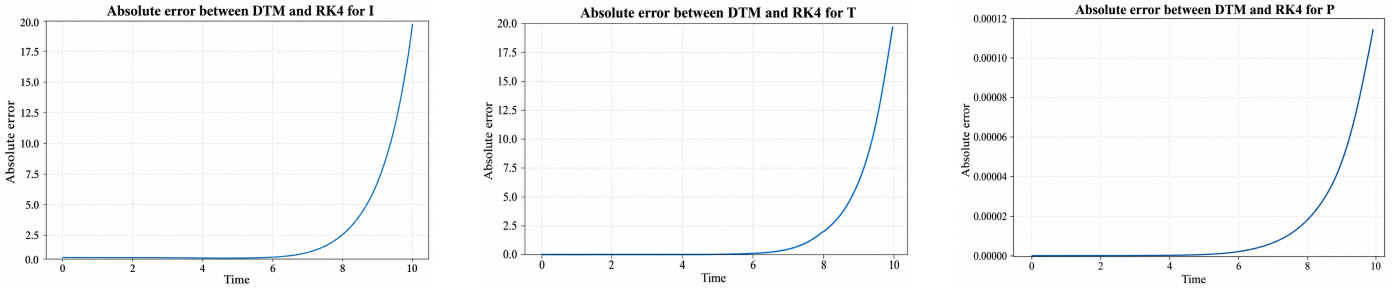


(d) $I(t)$ (e) $T(t)$ (f) $P(t)$

Figure 6: Comparison between the truncated DTM solution (order t^{10}) and the RK4 numerical solution for all compartments over the interval $t \in [0, 10]$. The simulations were performed using the parameter values listed in Table 1 and initial conditions $(S_d(0), S_u(0), E(0), I(0), T(0), P(0)) = (500, 385, 50, 30, 25, 10)$. The results show strong agreement between both methods, with slight deviations observed in the infected and treated compartments at later times.



(a) $S_d(t)$ (b) $S_u(t)$ (c) $E(t)$



(d) $I(t)$ (e) $T(t)$ (f) $P(t)$

Figure 7: Absolute error between the truncated DTM solution (order t^{10}) and the RK4 solution for all compartments over $t \in [0, 10]$, using parameter values from Table 1. The error remains small for most compartments but increases at later times for $I(t)$ and $T(t)$.

and nearly constant throughout the simulation. This indicates that only a small fraction of the population effectively mitigates

risk through improved awareness or favorable socioeconomic conditions.

Overall, the DTM approximation captures the expected system dynamics: declining susceptible populations, transient growth in exposure, gradual stabilization of infection levels, and sustained increases in treatment (Figure 2). The system remains bounded and converges toward a stable regime without exhibiting unbounded growth.

To evaluate the impact of intervention strategies, several policy scenarios were simulated. Figure 3 compares the baseline trajectory of the diagnosed population $I(t)$ with a prevention-focused intervention characterized by reduced transmission ($\beta \downarrow$) and increased awareness ($\tau \uparrow$). The intervention results in a sustained reduction in diagnosed cases, highlighting the effectiveness of combined prevention and awareness strategies.

A more comprehensive intervention scenario is presented in Figure 5, where upstream lifestyle risk reduction, prevention measures, and enhanced clinical treatment are implemented simultaneously. Under this scenario, both the diagnosed and treated populations grow more slowly compared to the baseline, demonstrating the synergistic benefits of integrated intervention approaches.

An alternative visualization of the prevention-focused scenario is shown in Figure 4, further illustrating the divergence between baseline dynamics and intervention outcomes.

Overall, these simulations indicate that while baseline dynamics limit uncontrolled growth through natural attrition, sustained reductions in disease burden require proactive intervention strategies that combine lifestyle modification, awareness, and improved clinical care.

5. Conclusion

In this study, we developed and analyzed a six-compartment deterministic model for type 2 diabetes mellitus (T2DM). The model distinguishes between genetically and non-genetically susceptible populations and incorporates exposure, diagnosis, treatment, and a modifiable lifestyle pressure variable P.

The analysis shows that a disease-free equilibrium (DFE) exists only under two restrictive conditions: (i) in the absence of pressure generation ($\phi = 0$), or (ii) when pressure does not induce disease progression ($\beta = 0$). In the latter case, the pressure variable may persist, but it does not generate new cases.

When both pressure generation and disease conversion are active ($\phi > 0$ and $\beta > 0$), the system admits an endemic equilibrium (EE). This result implies that T2DM persists in the population under sustained lifestyle and environmental influences. The local stability analysis indicates that the endemic equilibrium is robust to small perturbations, meaning that the long-term disease burden remains stable unless key parameters are modified.

The model identifies several parameters that strongly influence disease dynamics, including ϕ, ω, γ_1, and γ_2. These parameters govern lifestyle pressure, progression dynamics, and its dissipation. Their interaction determines whether the disease burden increases, stabilizes, or declines over time.

Further analysis highlights the role of the transmission parameter β in driving new cases arising from lifestyle-related risk factors. The threshold analysis also underscores the importance of γ_1 and γ_2 in regulating the long-term burden through pressure dissipation mechanisms.

Three primary intervention pathways emerge from the analysis. First, reducing lifestyle pressure ($\phi \downarrow$) or increasing its dissipation ($(\gamma_1 + \gamma_2) \uparrow$) lowers the equilibrium level of P and weakens its impact on disease progression. Second, decreasing the transition into disease states ($\beta \downarrow$ and $(\omega - \tau) \downarrow$), particularly through increased awareness ($\tau \uparrow$), reduces the inflow into the infected class. Third, increasing treatment initiation ($\eta \uparrow$) enhances recovery dynamics and reduces the steady-state size of the diagnosed population.

These findings correspond to practical public health interventions, including improved access to healthy food, promotion of physical activity, stress reduction, preventive screening programs, and enhanced healthcare delivery systems.

Overall, the results emphasize that sustained reductions in T2DM prevalence require coordinated strategies targeting lifestyle behavior, environmental conditions, and clinical care. Improving quality of life, encouraging healthy dietary practices, increasing physical activity, and reducing stress are critical components in mitigating the long-term burden of T2DM.

Data availability

No primary datasets were generated or collected in this study. The data used were parameter values obtained from published literature and assumptions, as stated in Table 1.

Declaration of competing interest

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