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Mathematical Modelling of Lifestyle Behavior and Diabetes Progression Dynamics

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ABSTRACT
This study develops an eight-compartmental deterministic mathematical model to examine how the social adoption of unhealthy lifestyle behaviour influences the progression of diabetes mellitus in a population. Diabetes itself is not transmitted; the transmissible process in the model is the adoption of unhealthy lifestyle behaviour. The model comprises susceptible, unhealthy-lifestyle, healthy-lifestyle, prediabetic, undiagnosed, controlled, complicated and recovered classes. Positivity and boundedness of solutions are established, and the Disease-Free and Endemic Equilibria are obtained. The Basic Reproduction Number, interpreted as a behavioural-adoption threshold, is derived using the Next Generation Matrix, and the local asymptotic stability of the Disease-Free Equilibrium is established by Jacobian analysis when $R_0 < 1$. The numerical parameter values are assumed illustrative values and are not estimates from a specific population. At the baseline parameter set, $R_0 = 2.833$, indicating persistence of unhealthy-lifestyle adoption in the model. Sensitivity analysis identifies β as the strongest positive driver, followed by μ, while γ, α and ρ reduce R_0. MATLAB simulations illustrate a larger unhealthy-lifestyle burden at higher adoption rates and a substantial reduction under increased awareness and lifestyle modification. The results are qualitative and theoretical, although it supports prevention, awareness, early diagnosis and sustained healthy-lifestyle promotion as important components of diabetes control but calibration and validation against observed data are needed before they can inform public-health practice.

Keywords: Diabetes mellitus, Lifestyle behaviour adoption, Mathematical modelling, Basic Reproduction Number, Local stability, Sensitivity analysis, MATLAB

INTRODUCTION

Diabetes mellitus is a major non-communicable disease whose increasing prevalence has become an important public-health concern. It is a chronic metabolic disorder associated with persistent elevated blood glucose and is strongly linked to modifiable lifestyle factors such as physical inactivity, poor diet and excess body weight (American Diabetes Association Professional Practice Committee, 2023; Hu, 2011). Type 2 diabetes in particular can often be prevented or delayed through a healthy diet, regular physical activity and a normal body weight (World Health Organization, 2023).

The International Diabetes Federation estimated that 537 million adults aged 20–79 years were living with diabetes worldwide in 2021, and projected 783 million by 2045 (International Diabetes Federation, 2021). The burden of undiagnosed diabetes is large: almost one in two adults with diabetes (44.7%) were unaware of their status, and the proportion was highest in the Africa region (53.6%), which increases the likelihood of prolonged hyperglycaemia and complications (Ogurtsova *et al*., 2022). Prediabetes is an important stage because lifestyle intervention can delay or reverse progression to diabetes (Tabák *et al.*, 2012).

Although diabetes itself is not an infectious disease, unhealthy lifestyle behaviour may be adopted through social interaction, peer influence and shared environments. Christakis and Fowler (2007) reported that obesity spreads through ties in a large social network, and Ejima *et al.* (2013) modelled obesity as a social-contagion process that also includes non-contagious hazards. These studies motivate the use of transmission-type formulations for lifestyle behaviour, while the disease itself remains non-infectious.

Compartmental and optimal-control models of diabetes have been developed previously. Boutayeb and Chetouani (2006) reviewed the mathematical models and data used in diabetology, and Kouidere *et al*. (2020, 2021) formulated optimal-control models in which awareness-based intervention acts on diabetic populations, including those with complications, under the influence of lifestyle and behavioural factors. The large share of people unaware of their diabetes (Ogurtsova *et al.,* 2022) motivates models that separate undiagnosed from diagnosed and controlled states, as is done here. In the studies cited above, however, lifestyle enters mainly as an external factor or as a control acting on diabetes-related states. To the best of the authors' knowledge, a model in which the social adoption of unhealthy lifestyle is itself a dynamical process with its own reproduction number, upstream of prediabetes, undiagnosed, controlled and complicated diabetes, has not been analysed in this way; this is the contribution of the present work.

The model treats the lifestyle transmission term as a representation of behavioural adoption rather than direct transmission of diabetes. Consequently, the Basic Reproduction Number R_0 derived below is a behavioural-adoption threshold and not a disease reproduction number, and the terms "Disease-Free Equilibrium" and "endemic" refer to the absence and persistence of unhealthy-lifestyle adoption together with its downstream diabetes-related states. The aim of this study is to formulate and analyse an eight-compartment deterministic model in which the social adoption of unhealthy lifestyle behaviour drives progression through prediabetes, diabetes, complications and recovery, and to determine, analytically and by MATLAB simulation,

how awareness and lifestyle modification alter the behavioural-adoption threshold R_0.

MATERIALS AND METHODS

Model Formulation

A deterministic compartmental model based on ordinary differential equations is used. At time t, the population is divided into eight mutually exclusive compartments: Susceptible (S), Unhealthy Lifestyle (L_u), Healthy Lifestyle (L_h), Prediabetic (P), Undiagnosed Diabetic (D_u), Controlled/Treated Diabetic (Dc), Complicated Diabetic (C), and Recovered (R). Thus,

$$N(t) = S(t) + Lu(t) + Lh(t) + P(t) + Du(t) + Dc(t) + C(t) + R(t) \quad (1)$$

Unhealthy lifestyle adoption is represented by the standard incidence $\lambda = \beta L_u/N$, so that β is the rate of adoption of unhealthy lifestyle per contact with an unhealthy-lifestyle individual and not a rate of transmission of diabetes. The model assumes homogeneous mixing, constant natural mortality μ in all compartments, recruitment into S at rate Λ, transition of unhealthy-lifestyle individuals to healthy lifestyle at rate ρ, awareness-driven movement from S to L_h at rate γ, progression from L_u to P at rate α, progression from P to D_u at rate δ, diagnosis/control at rate ξ, development of complications at rate κ, recovery at rates θ (controlled) and π (complicated), and diabetes-related mortality at rate ω in the D_u and C compartments only (equations (6) and (8)). The parameters, their units and their baseline values are given in Table 1.

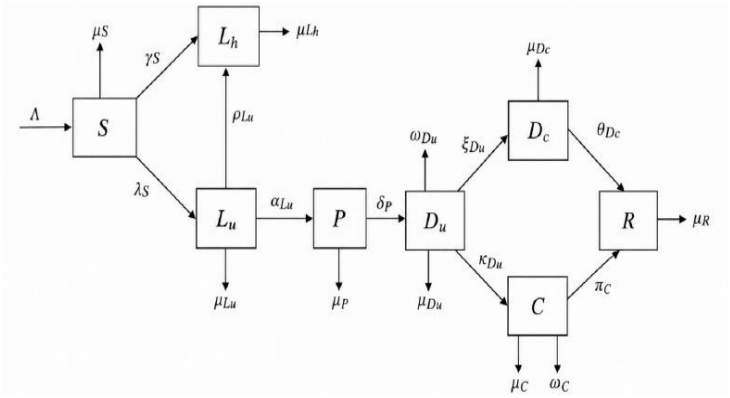


Figure 1: The Schematic diagram of the model

$$\frac{dS}{dt} = \Lambda - \lambda S - (\gamma + \mu)S \quad (2)$$

$$\frac{dL_u}{dt} = \lambda S - (\rho + \alpha + \mu)L_u \quad (3)$$

$$\frac{dL_h}{dt} = \gamma S + \rho L_u - \mu L_h \quad (4)$$

$$\frac{dP}{dt} = \alpha L_u - (\delta + \mu)P \quad (5)$$

$$\frac{dD_u}{dt} = \delta P - (\xi + \kappa + \mu + \omega)D_u \quad (6)$$

$$\frac{dD_c}{dt} = \xi D_u - (\theta + \mu)D_c \quad (7)$$

$$\frac{dC}{dt} = \kappa D_u - (\pi + \mu + \omega)C \quad (8)$$

$$\frac{dR}{dt} = \theta D_c + \pi C - \mu R \quad (9)$$

subject to non-negative initial conditions:

$$S(0), L_u(0), L_h(0), P(0), D_u(0), D_c(0), C(0), R(0) \geq 0$$

Summing equations (2)–(9):

$$\frac{dN}{dt} = \Lambda - \mu N - \omega D_u - \omega C \quad (10)$$

In the absence of disease-induced mortality($\omega = 0$), the total population approaches the demographic carrying capacity $N = \frac{\Lambda}{\mu}$ as$t \to \infty$.

Take:

$A_1 = (\gamma + \mu)$,
$A_2 = (\rho + \alpha + \mu)$,
$A_3 = (\delta + \mu)$,
$A_4 = (\xi + \kappa + \mu + \omega)$,
$A_5 = (\theta + \mu)$,
$A_6 = (\pi + \mu + \omega)$

Model Analysis

Positivity and Boundedness of Solutions

Because the variables are population sizes, solutions must remain non-negative and bounded. Adding equations (2)–(9) gives (10), and since $\omega(D_u + C) \geq 0$ we have $dN/dt \leq \Lambda - \mu N$. The standard comparison theorem then gives $N(t) \leq \Lambda/\mu + (N(0) - \Lambda/\mu)e^{-\mu t}$, so that lim sup $N(t) \leq \Lambda/\mu$ and $N(t) \leq \max\{N(0), \Lambda/\mu\}$ for all $t \geq 0$.

Theorem 1: Positivity and Boundedness of Solutions

For non-negative initial conditions, the solution of system (2)–(9) is non-negative for all $t \geq 0$, and the region $\Omega = \{(S, L_u, L_h, P, D_u, D_c, C, R) \in \mathbb{R}^8_+ : N \leq \Lambda/\mu\}$ is positively invariant.

Proof. Positivity: consider each face of the non-negative orthant.

If $S = 0$ then $dS/dt = \Lambda > 0$;
if $L_u = 0$ then $dL_u/dt = 0$;
if $L_h = 0$ then $dL_h/dt = \gamma S + \rho L_u \geq 0$;
if $P = 0$ then $dP/dt = \alpha L_u \geq 0$;
if $D_u = 0$ then $dD_u/dt = \delta P \geq 0$;
if $D_c = 0$ then $dD_c/dt = \xi D_u \geq 0$;
if $C = 0$ then $dC/dt = \kappa D_u \geq 0$; and
if $R = 0$ then $dR/dt = \theta D_c + \pi C \geq 0$.

Hence no solution can leave the non-negative orthant. Equivalently, each equation has the form $x' = f(t) - a(t)x$ with $f(t) \geq 0$, and the integrating-factor solution $x(t) = x(0)\exp(-\int_0^t a) + \int_0^t f(s)\exp(-\int_s^t a)ds$ is non-negative whenever $x(0) \geq 0$. For L_u we write $dL_u/dt = (\beta S/N - A_2)L_u$, so that $L_u(t) = L_u(0)\exp\int_0^t(\beta S/N - A_2)ds \geq 0$; here $N(t) > 0$ because $S(t) > 0$ for $t > 0$, since $dS/dt \geq \Lambda - (\beta + \gamma + \mu)S$.

Boundedness: by the bound obtained above, every compartment satisfies $0 \leq x(t) \leq N(t) \leq \max\{N(0), \Lambda/\mu\}$, and Ω is positively invariant because $N' < 0$ whenever $N > \Lambda/\mu$. Therefore, the model solutions remain biologically feasible for all $t \geq 0$.

Equilibrium Points of the Model

The model has two equilibrium states: the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). These

are obtained by setting the derivatives in equations (2)–(9) equal to zero and solving the resulting algebraic system.

Disease-Free Equilibrium (DFE)
At the disease-free state, the unhealthy-lifestyle and all diabetes-related compartments are set to zero ($L_u = P = D_u = D_c = C = R = 0$). The first equation then gives $S^0 = \Lambda/(\gamma + \mu)$, the equation for L_h gives $L_h^0 = \gamma S^0/\mu = \gamma\Lambda/[\mu(\gamma + \mu)]$, and $N^0 = S^0 + L_h^0 = \Lambda/\mu$. The disease-free equilibrium is therefore represented by the following vector:

$$E_0 = (S^\circ, L_u^\circ, L_h^\circ, P^\circ, D_u^\circ, D_c^\circ, C^\circ, R^\circ) = \left(\frac{\Lambda}{(\gamma+\mu)}, 0, \frac{\gamma\Lambda}{[\mu(\gamma+\mu)]}, 0,0,0,0,0\right) \quad (11)$$

This equilibrium represents the state in which unhealthy-lifestyle adoption and the diabetes-related compartments are absent, while the population is maintained by demographic recruitment and natural mortality.

Endemic Equilibrium (EE)
For the endemic state, all derivatives are set to zero with $L_u \neq 0$. Equation (3) then gives $\beta S/N = A_2$, that is $S^* = A_2N^*/\beta$. Equation (2) gives $L_u^* = (\Lambda - A_1S^*)/A_2$, and equations (4)–(9) are then solved successively for L_h^*, P^*, D_u^*, D_c^*, C^* and R^*.
With $Q = \beta\Lambda - A_1A_2N^*$, the endemic equilibrium is

$$E^* = (S^*, L_u^*, L_h^*, P^*, D_u^*, D_c^*, C^*, R^*) \quad (12)$$

$$Q = \beta\Lambda - A_1A_2N^*$$

$$S^* = \frac{A_2N^*}{\beta}, \qquad L_u^* = \frac{Q}{\beta A_2}$$

$$L_h^* = \frac{\gamma A_2^2 N^* + \rho Q}{\beta\mu A_2}$$

$$P^* = \frac{\alpha Q}{\beta A_2 A_3}, \qquad D_u^* = \frac{\delta\alpha Q}{\beta A_2 A_3 A_4}$$

$$D_c^* = \frac{\xi\delta\alpha Q}{\beta A_2 A_3 A_4 A_5}, \qquad C^* = \frac{\kappa\delta\alpha Q}{\beta A_2 A_3 A_4 A_6}$$

$$R^* = \frac{\delta\alpha Q\,(\theta\xi A_6 + \pi\kappa A_5)}{\mu\beta A_2 A_3 A_4 A_5 A_6}$$

Every component of (12) is a non-negative combination of N^* and Q, so the endemic equilibrium is feasible if and only if $N^* > 0$ and $Q > 0$, that is $\beta\Lambda > A_1A_2N^*$, which is equivalent to $L_u^* > 0$. Because every component of (12) is linear in N^*, substituting (12) into (1) gives a linear equation that determines N^*. At equilibrium, (10) gives $\mu N^* = \Lambda - \omega(D_u^* + C^*) \leq \Lambda$, so $N^* \leq \Lambda/\mu$, and therefore $\beta\Lambda > A_1A_2N^*$ holds whenever $\beta\mu > A_1A_2$, that is whenever $R_0 > 1$. The endemic equilibrium therefore exists and is feasible when $R_0 > 1$; its existence for $R_0 \leq 1$ in the presence of disease-induced mortality is not examined here. The numerical results in the study illustrate persistence of the endemic state when the reproduction number is above the threshold value.

Basic Reproduction Number
The Basic Reproduction Number, denoted by R_0, is the expected number of new unhealthy-lifestyle adoptions generated by one unhealthy-lifestyle individual introduced into an otherwise unhealthy-lifestyle-free population. Because diabetes is not infectious, R_0 is a behavioural-adoption threshold and not a disease reproduction number. It is obtained by the Next Generation Matrix (NGM) method (van den Driessche & Watmough, 2002).
Identification of the behaviour-transmitting subsystem. The "infected" compartments at the DFE are L_u, D_u, and C. However, only L_u generates new "infections" (new unhealthy lifestyle adopters), as D_u and C arise solely from progression within the system. Hence, the three compartments are considered in obtaining the Reproductive number(R_0) and the resulting threshold quantity is given by:

F, V, V^{-1} and FV^{-1} used in the NGM calculation:

$$F_j = \begin{pmatrix} \frac{\beta\mu}{(\gamma+\mu)} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

also,

$$V_j = \begin{pmatrix} A_2 & 0 & 0 \\ 0 & A_4 & 0 \\ 0 & -\kappa & A_6 \end{pmatrix},$$

then V^{-1} gives,

$$V^{-1} = \begin{pmatrix} \frac{1}{A_2} & 0 & 0 \\ 0 & \frac{1}{A_6} & 0 \\ 0 & \frac{\kappa}{A_4A_6} & \frac{1}{A_6} \end{pmatrix}$$

Therefore, FV^{-1} gives,

$$FV^{-1} = \begin{pmatrix} \frac{\beta\mu}{A_1} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{A_2} & 0 & 0 \\ 0 & \frac{1}{A_6} & 0 \\ 0 & \frac{\kappa}{A_4A_6} & \frac{1}{A_6} \end{pmatrix}$$

$$= \begin{pmatrix} \frac{\beta\mu}{A_1A_2} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (13)$$

Computing the R_0, gives:

$$R_0 = |FV^{-1} - \lambda I| = 0, i.e,$$

$$\left| \begin{pmatrix} \frac{\beta\mu}{A_1A_2} - \lambda & 0 & 0 \\ 0 & -\lambda & 0 \\ 0 & 0 & -\lambda \end{pmatrix} \right| = 0$$

$$\left(\frac{\beta\mu}{A_1A_2} - \lambda\right)(\lambda)^2 = 0$$

$$i.e,$$

$$\lambda^2 = 0 : \lambda_1 = 0, \lambda_2 = 0$$

$$\frac{\beta\mu}{A_1A_2} - \lambda = 0$$

$$\lambda_3 = \frac{\beta\mu}{A_1A_2} \quad (14)$$

Hence, R_0 becomes:

$$R_0 = \frac{\beta\mu}{(\gamma+\mu)(\rho+\alpha+\mu)} \quad (15)$$

Thus, the model threshold is determined by R_0. Values of $R_0 < 1$ indicate that the adoption of unhealthy lifestyle cannot be sustained under the model conditions, whereas $R_0 > 1$ is associated with persistence of unhealthy-lifestyle adoption and the existence of the endemic state described above. The critical case $R_0 = 1$ is treated separately below.
The expression for R_0 is also used in the local stability analysis of the Disease-Free Equilibrium below.

Local Stability of the DFE
Theorem 2: Local Stability of the Disease-Free Equilibrium
The DFE is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.
Proof. The local stability of the DFE is determined using the Jacobian matrix and the signs of its eigenvalues. The Jacobian matrix of the model is first formed and then evaluated at the DFE. The terms that arise from the

dependence of N on the state variables are all multiples of L_u, so they vanish at the DFE and are omitted from (16), which is written in the form needed at the DFE.

$$J=\begin{pmatrix} \frac{-\beta L_u}{N}-(\gamma+\mu) & \frac{-\beta S}{N} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta L_u}{N} & \frac{\beta S}{N}-A_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ \gamma & \rho & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -A_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \xi & -A_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \kappa & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & \pi & -\mu \end{pmatrix} \quad (16)$$

At the DFE, the Jacobian reduces to the matrix in (17), and with $S^0/N^0 = \mu/(\gamma + \mu)$ to the matrix in (18). This matrix is block triangular: the first two rows involve only S and L_u, and the remaining rows are lower triangular. Its eigenvalues are therefore the diagonal entries, and the characteristic equation is (19)–(20).

$$J(E_0)=\begin{pmatrix} -(\gamma+\mu) & \frac{-\beta S}{N} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta S}{N}-A_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ \gamma & \rho & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -A_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \xi & -A_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \kappa & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & \pi & -\mu \end{pmatrix} \quad (17)$$

$$J(E_1)=\begin{pmatrix} -(\gamma+\mu) & \frac{-\beta\mu}{(\gamma+\mu)} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta\mu}{(\gamma+\mu)}-A_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ \gamma & \rho & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & \alpha & 0 & -A_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta & -A_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \xi & -A_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \kappa & 0 & -A_6 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta & \pi & -\mu \end{pmatrix} \quad (18)$$

$$J(E)=\begin{pmatrix} -(\gamma+\mu)-\lambda & \frac{-\beta\mu}{(\gamma+\mu)} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta\mu}{(\gamma+\mu)}-A_2-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -A_3-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -A_4-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -A_5-\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -A_6-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu-\lambda \end{pmatrix} \quad (19)$$

$$(-(\gamma+\mu)-\lambda)\left(\frac{\beta\mu}{(\gamma+\mu)}-A_2-\lambda\right)(-\mu-\lambda)(-A_3-\lambda)(-A_4-\lambda)(-A_5-\lambda)$$
$$(-A_6-\lambda)(-\mu-\lambda)=0 \quad (20)$$

$$\lambda_1 = -(\gamma+\mu)$$
$$\lambda_2 = \frac{\beta\mu}{(\gamma+\mu)} - A_2$$
$$\lambda_3 = -\mu$$
$$\lambda_4 = -A_3$$
$$\lambda_5 = -A_4$$
$$\lambda_6 = -A_5$$
$$\lambda_7 = -A_6$$
$$\lambda_8 = -\mu \quad (21)$$
$$\frac{\beta\mu}{(\gamma+\mu)} < A_2, \quad (22)$$
$$\frac{\beta\mu}{(\gamma+\mu)A_2} < 1. \quad (23)$$

Putting the value of $A_2 = (\rho + \alpha + \mu)$, we have;

$$R_0 = \frac{\beta\mu}{(\gamma+\mu)(\rho+\alpha+\mu)} < 1 \quad (24)$$

The DFE is locally asymptotically stable when all eigenvalues of the Jacobian evaluated at the DFE have negative real parts, and it is unstable when at least one eigenvalue has a positive real part. All eigenvalues in (21) except λ_2 are negative, because $\gamma + \mu$, μ, A_3, A_4, A_5 and A_6 are positive. The remaining eigenvalue $\lambda_2 = \beta\mu/(\gamma + \mu) - A_2$ is negative if and only if $R_0 < 1$ and positive if $R_0 > 1$. Hence the DFE is locally asymptotically stable whenever $R_0 < 1$ and unstable whenever $R_0 > 1$.

Local Stability of the Endemic Equilibrium (EE)

For consistency with the Jacobian/eigenvalue approach, the local stability of the endemic equilibrium would be determined by evaluating the Jacobian at the endemic equilibrium, J(EE), and examining its eigenvalues. If every eigenvalue λ_i of J(EE) satisfies $\mathrm{Re}(\lambda_i) < 0$, the endemic equilibrium is locally asymptotically stable; if at least one eigenvalue has $\mathrm{Re}(\lambda_i) > 0$, it is unstable. At the endemic equilibrium the terms arising from the dependence of N on the state variables no longer vanish and must be retained. A formal analytical proof of the local stability of the EE is not given here; the baseline simulations (Figure 2) are consistent with convergence towards an endemic state.

Numerical Simulation

Numerical simulations were carried out in MATLAB using the ode45 solver (explicit Runge–Kutta (4,5) pair) over a 50-year period, with time in years. The baseline parameter values are given in Table 1, the initial conditions in Table 2, and the exact parameter changes used for every scenario in Table 3. All parameter values and initial conditions are assumed illustrative values chosen to demonstrate the behaviour of the model; they are not estimates for a specific population, including the Nigerian population, and are not fitted to data. With $\Lambda = 200$ and $\mu = 0.02$, the demographic carrying capacity is $\Lambda/\mu = 10\,000$, and the mean time spent in a compartment is the reciprocal of its total exit rate. In the scenarios examined, the computed trajectories are non-negative to within solver round-off (of order 10^{-7} or smaller at default tolerances), consistent with Theorem 1.

Table 1: Parameters of the Model, their Meaning, Units and Baseline Values (All Values Assumed for Illustration)

Par.	Meaning	Value	Basis
Λ	Recruitment into S	200	Assumed
μ	Natural mortality rate	0.02	Assumed
β	Unhealthy-lifestyle adoption rate	0.85	Assumed
γ	Awareness-driven uptake of healthy lifestyle ($S \rightarrow L_h$)	0.03	Assumed
ρ	Conversion $L_u \rightarrow L_h$	0.02	Assumed
α	Progression $L_u \rightarrow P$	0.08	Assumed
δ	Progression $P \rightarrow D_u$	0.08	Assumed

Par.	Meaning	Value	Basis
ξ	Diagnosis/control $D_u \rightarrow D_c$	0.06	Assumed
κ	Complications $D_u \rightarrow C$	0.12	Assumed
θ	Recovery $D_c \rightarrow R$	0.08	Assumed
π	Recovery $C \rightarrow R$	0.04	Assumed
ω	Diabetes-related mortality (D_u and C only)	0.03	Assumed

Table 2: Initial Conditions Used in the Simulations (All Values Assumed for Illustration)

Variable	Description	Value (persons)
S(0)	Susceptible	8000
$L_u(0)$	Unhealthy lifestyle	2000
$L_h(0)$	Healthy lifestyle	3000
P(0)	Prediabetic	1000
$D_u(0)$	Undiagnosed diabetic	500
$D_c(0)$	Controlled/treated diabetic	700
C(0)	Complicated diabetic	450
R(0)	Recovered	350
N(0)	Total population	16 000

Table 3: Parameter Values used in each Simulation Scenario (All other Parameters Take the Values in Table 1)

Scenario (Figure)	Parameter Values	R_0
Baseline, endemic (Fig. 2)	None changed	2.833
Intervention (Fig. 3)	$\beta = 0.45$, $\gamma = 0.18$, $\rho = 0.10$	0.225
Varying β (Fig. 4)	$\beta = 0.30, 0.50, 0.80, 1.00$	1.000, 1.667, 2.667, 3.333
Varying ρ (Fig. 5)	$\rho = 0.02, 0.20, 0.50, 0.80$	2.833, 1.133, 0.567, 0.378
Varying α (Fig. 6)	$\alpha = 0.08, 0.35, 0.65, 0.95$	2.833, 0.872, 0.493, 0.343
Varying γ (Fig. 7)	$\gamma = 0.03, 0.25, 0.55, 0.85$	2.833, 0.525, 0.249, 0.163

The case $\beta = 0.30$ gives $R_0 = 1$ exactly (the critical case).

RESULTS AND DISCUSSION

Analytical Results

For the baseline values in Table 1, the threshold expression gives $R_0 = (0.85 \times 0.02)/[(0.03 + 0.02)(0.02 + 0.08 + 0.02)] = 0.017/0.006 = 2.833$. Since this value exceeds unity, the model predicts persistence of unhealthy-lifestyle adoption and the associated diabetes-related states (Figure 2). In the intervention scenario of Table 3 ($\beta = 0.45$, $\gamma = 0.18$, $\rho = 0.10$), $R_0 = (0.45 \times 0.02)/[(0.18 + 0.02)(0.10 + 0.08 + 0.02)] = 0.009/0.04 = 0.225 < 1$, and unhealthy-lifestyle individuals decline towards zero (Figure 3). Analytically, the Disease-Free Equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$; the critical case $R_0 = 1$ is not decided by the linearisation.

The equilibrium and stability analysis therefore provide the threshold interpretation used for the numerical experiments: values of R_0 above unity are associated with endemic persistence, while values below unity correspond to local stability of the disease-free regime. These are properties of the model under assumed parameter values and are not empirical findings about any population.

Sensitivity analysis of R_0

The normalised forward sensitivity index of R_0 with respect to a parameter p is defined as

$$\Gamma_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0} \qquad (25)$$

Applying (25) to (15) gives the analytical expressions in Table 4, evaluated at the baseline values in Table 1. For μ, writing $R_0 = \beta\mu/[(\gamma + \mu)(\rho + \alpha + \mu)]$ gives $\Gamma_\mu = 1 - \mu/(\gamma + \mu) - \mu/(\rho + \alpha + \mu) = 1 - 0.400 - 0.167 = +0.433$.

Table 4: Normalized Sensitivity Indices of R_0 at the Baseline Parameter Values

Par.	Index expression	Index	Interpretation
β	1	+1.000	Direct proportional effect on R_0
μ	$1 - \mu/(\gamma+\mu) - \mu/(\rho+\alpha+\mu)$	+0.433	Higher natural mortality raises R_0
γ	$-\gamma/(\gamma+\mu)$	−0.600	Awareness lowers R_0
α	$-\alpha/(\rho+\alpha+\mu)$	−0.667	Faster progression out of L_u lowers R_0
ρ	$-\rho/(\rho+\alpha+\mu)$	−0.167	Lifestyle conversion lowers R_0

The sensitivity indices show that β has the strongest positive influence on R_0, followed by μ, whose index is positive because natural turnover raises the susceptible fraction $\mu/(\gamma + \mu)$ at the disease-free state by more than it shortens the stay in L_u at the baseline values. Among the parameters that public-health action can target, γ has a larger absolute index than ρ, indicating a stronger direct effect on R_0 under the baseline parameterisation. The α index is larger in magnitude than that of ρ, but α is a disease-progression parameter and μ is a demographic parameter, so neither is an intervention lever; a higher α lowers R_0 only because individuals leave the unhealthy-lifestyle class sooner, towards prediabetes, which is not a recommended strategy. Figure 2 shows the baseline endemic scenario and Figure 3 the intervention scenario defined in Table 3; Figures 4–7 show the effect of varying β, ρ, α and γ one at a time. All vertical axes start at zero.

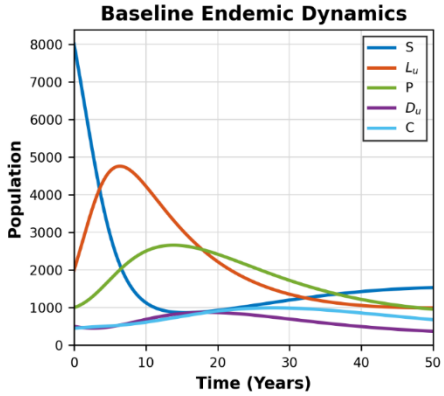


Figure 2: Baseline endemic dynamics of the model (parameters in Table 1, $R_0 = 2.833$).

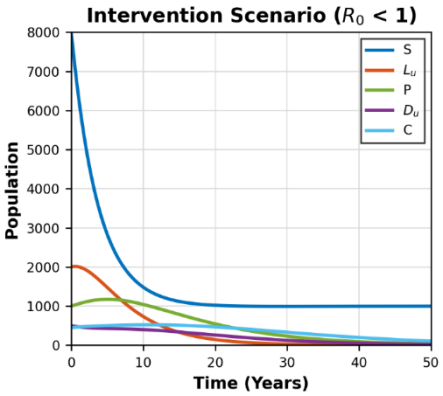


Figure 3: Intervention dynamics of the model with $\beta = 0.45$, $\gamma = 0.18$ and $\rho = 0.10$ ($R_0 = 0.225$); other parameters as in Table 1.

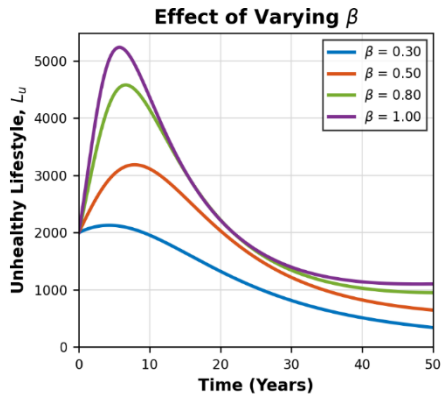


Figure 4: Effect of varying the unhealthy-lifestyle adoption rate β on the unhealthy-lifestyle population.

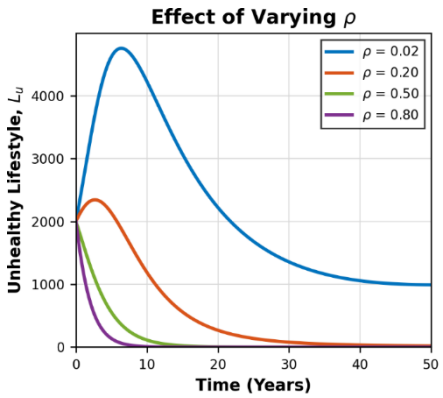


Figure 5: Effect of varying the conversion rate ρ on the unhealthy-lifestyle population.

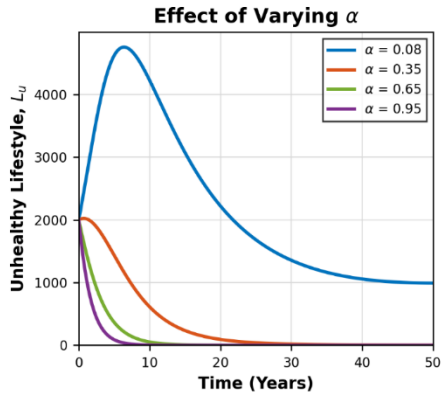


Figure 6: Effect of varying the progression rate α on the unhealthy-lifestyle population.

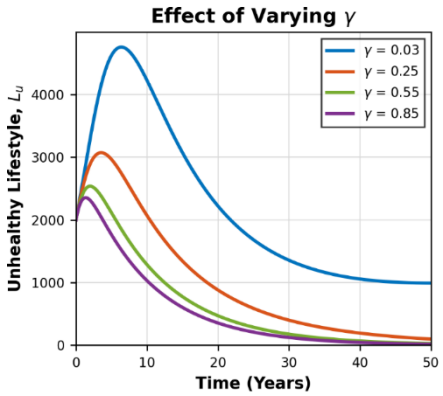


Figure 7: Effect of varying the awareness rate γ on the unhealthy-lifestyle population.

Discussion

The numerical results agree with the analytical threshold structure of the model. The baseline simulation ($R_0 = 2.833 > 1$; Figure 2) shows the unhealthy-lifestyle population rising and then declining slowly towards an endemic level, followed by changes in the prediabetic, undiagnosed and complicated diabetic states that illustrate how behavioural adoption propagates through the disease pathway. In the intervention scenario ($R_0 = 0.225 < 1$; Figure 3), the unhealthy-lifestyle and downstream compartments decline, in line with the local stability of the Disease-Free Equilibrium. This agreement supports the internal consistency of the mathematical analysis and the MATLAB simulations; it is not an empirical validation.

Increasing β increases the magnitude and persistence of the unhealthy-lifestyle population (Figure 4); at $\beta = 0.30$, $R_0 = 1$ exactly, and the class declines only slowly after its peak. The simulations therefore identify the rate of social adoption of unhealthy lifestyle behaviour as a major driver of the modelled burden. Higher ρ moves individuals out of the unhealthy-lifestyle class more rapidly and reduces its long-term size, whereas low values allow a large unhealthy-lifestyle population to persist for long (Figure 5). Variation in α changes the timing and magnitude of the peak (Figure 6); larger α removes individuals from L_u sooner, but into the

prediabetic class, so the reduction in R_0 reflects faster progression rather than a benefit. Increasing γ produces lower and shorter peaks because more susceptible individuals move directly towards the healthy-lifestyle class (Figure 7).

The positive sensitivity of β confirms the central role assigned to unhealthy-lifestyle adoption in the model. Because β controls the rate at which susceptible individuals adopt unhealthy lifestyle behaviour, increasing it enlarges the population entering the subsequent prediabetes and diabetes pathway. This is consistent with the social-contagion basis of the formulation (Christakis & Fowler, 2007; Ejima *et al.,* 2013).

The negative sensitivity indices for γ and ρ show that awareness and lifestyle modification reduce the threshold quantity. In particular, γ has a stronger absolute sensitivity than ρ under the baseline values (-0.600 against -0.167), because R_0 contains the factor $\mu/(\gamma + \mu)$, whereas ρ acts only through the denominator $\rho + \alpha + \mu$. The simulation results show the same qualitative pattern, with higher γ or ρ reducing the unhealthy-lifestyle population. Because R_0 depends only on β, μ, γ, ρ and α, the diagnosis, treatment and complication parameters (ξ, κ, θ, π, ω) affect the size of the diabetes burden but not the threshold itself.

The model extends earlier diabetes frameworks by bringing unhealthy-lifestyle adoption, prediabetes, undiagnosed and controlled diabetes, complications and recovery into one system, while retaining the standard compartmental threshold approach (Brauer et al., 2019; van den Driessche & Watmough, 2002). The optimal-control studies of Kouidere *et al*. (2020, 2021) likewise place awareness-oriented intervention at the centre of diabetes control; the present model differs in representing awareness (γ) and lifestyle conversion (ρ) as rates acting on the behavioural stage upstream of prediabetes and in supplying an analytical threshold for that stage, and it differs from the age-structured formulation of Kouidere *et al*. (2021) in using a single age class. Compared with the social-contagion model of obesity of Ejima *et al.* (2013), which accounts for contagious and non-contagious hazards, the present model is purely theoretical and adds the downstream diabetes pathway. The undiagnosed and controlled states reflect the large undiagnosed fraction reported by Ogurtsova *et al*. (2022), although screening effects are not examined here because R_0 does not depend on ξ.

Several limitations apply. The standard-incidence term $\lambda = \beta L_u/N$ assumes homogeneous mixing, so every individual is equally likely to meet an unhealthy-lifestyle individual; real behaviour spreads through structured networks, peer groups and households with heterogeneous contact and social influence (Christakis & Fowler, 2007), and homogeneous mass-action or standard-incidence mixing can over- or under-state adoption in clustered settings. The model also uses a single age class, constant parameters, no return from healthy to unhealthy lifestyle and no stochasticity. Most importantly, the numerical values are assumed baseline values and initial conditions, not parameter estimates from a specific population, so the simulations demonstrate qualitative model behaviour and must not be read as quantitative evidence about the Nigerian or any other diabetes population. Real-world application would require local epidemiological data, parameter estimation and validation.

Conclusion

An eight-compartment deterministic mathematical model was formulated and analysed to study how the social adoption of unhealthy lifestyle behaviour influences diabetes progression. The model links unhealthy-lifestyle adoption with healthy-lifestyle uptake, prediabetes, diabetes progression, diagnosis/control, complications and recovery. Positivity and boundedness of solutions were established, and the Basic Reproduction Number, interpreted as a behavioural-adoption threshold, was derived as $R_0 = \beta\mu/[(\gamma + \mu)(\rho + \alpha + \mu)]$. The Disease-Free Equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$, and an endemic state exists when $R_0 > 1$; the critical case $R_0 = 1$ and global stability were not established. At the assumed baseline parameter set, $R_0 = 2.833$. Sensitivity analysis identifies β as the strongest positive driver ($+1.000$), with μ also positive ($+0.433$), while γ, α and ρ reduce R_0 (-0.600, -0.667 and -0.167). MATLAB simulations reproduce these threshold behaviours and show that, in the model, increasing awareness and lifestyle modification reduces the unhealthy-lifestyle and related disease burden.

Because the parameter values were assumed rather than fitted to observed data, the model is theoretical and illustrative, and its results are qualitative. They suggest that sustained public awareness, promotion of healthy lifestyles, early diagnosis and appropriate diabetes management are model-relevant intervention areas, but any public-health recommendation requires calibration and validation against observed data. Future work may include calibration and validation, a Lyapunov-function proof of global stability using the invariance principle (LaSalle, 1976), and extensions to age structure, network-based contact, stochastic effects, optimal control, comorbidities and spatial heterogeneity.

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