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# Global Stability Analysis of the Mathematical Modelling for Heart Disease Transmission and Prevention Dynamics

***Agbo, C.E., Abah, R.T., & Ogunfiditimi, F.O.**
Department of Mathematics, University of Abuja, Nigeria

***Corresponding author email:** agbo.ene@uniabuja.edu.ng

**Abstract**
Heart disease remains a leading cause of global mortality, underscoring the urgent need for effective prevention and control strategies. This study analyses a compartmental mathematical model that capture the transmission and prevention dynamics of heart disease, incorporating both lifestyle-related and genetic risk factors. The model consists of six compartments—susceptible, two exposed classes (lifestyle and genetic), infected, treated, and recovered populations. Analytical methods are employed to establish the positivity and boundedness of solutions, determine the disease-free equilibrium (DFE), and compute the basic reproduction number $R_0$ using the next-generation matrix approach. Global stability of the DFE is proven via LaSalle's Invariance Principle and an appropriately constructed Lyapunov function. Numerical simulations implemented in R programming validate the theoretical results, demonstrating that when $R_0 < 1$, the disease can be eradicated. Findings highlight the pivotal role of primary prevention, lifestyle modification, early detection, and treatment in reducing disease prevalence. This framework offers valuable insights for designing public health interventions and provides a basis for future model extensions incorporating demographic and spatial complexities.

**Keywords:** Heart Disease, Mathematical Model, Basic Reproduction Number, Global Stability, Lyapunov Function

## Introduction

Heart disease includes any condition that influences the heart and vascular system, such as coronary artery disease (CAD), arrhythmias, and heart failure. These disorders may result in myocardial damage, presenting symptoms like angina pectoralis and arrhythmias (National Heart, Lung, and Blood Institute [NHLBI], 2020). According to reports from the World Health Organisation, cardiovascular disease is the main cause of death worldwide. Moreover, for nearly all nations, the upward trend observed during the past thirty years persists (Cai et al., 2023). In 2019, cardiovascular illnesses was responsible for nearly 18 million deaths worldwide, constituting 32% of total mortality; myocardial infarctions and cerebrovascular accidents represented 85% of these deaths (World Health Organization [WHO], 2021). This phenomenon has prompted investigations into models of heart disease and the cardiovascular system (CVS), where research utilising mathematical and computational models is much more feasible and cost-effective than in vivo or in vitro studies. The elementary functions of the cardiovascular system are to deliver oxygen and nutrients to the body and to eliminate metabolic waste products. The pulmonary circulation, responsible for transporting blood through the lungs, and the systemic circulation, which distributes blood throughout the body, are the two vascular circuits through which the heart pumps blood to fulfill their roles (Batzel et al., 2007). Given that heart diseases constitute a relatively intricate system necessitating expertise from diverse disciplines of physics and chemistry to fully grasp its dynamics, models or simulators of varying detail have been created to offer a thorough understanding of the system's operation. Diverse models have been proposed and analysed from multiple perspectives, including gas exchange, neuro-regulation mechanisms, and cardiac haemodynamics; however, the latter has consistently garnered greater attention due to its potential physiological or clinical applications (Simaan, 2009). The two principal methodologies used for this type of analysis are (1) lumped-parameter models, which encapsulate the principal behaviours of each cardiac component in a simplified manner (Ortiz-Rangel et al., 2022). The distributed parameter models, employing finite element software to characterise the cardiovascular system (CVS) in one, two, or three dimensions as described by (Alastruey et al., 2007), and the use a hydraulic modelling technique as proposed by (Rosalia et al., 2021).

When assessing and contrasting the effectiveness of various strategies for the prevention of heart disease, mathematical modeling is of the greatest importance. A multitude of studies has investigated the influence of preventive interventions on heart disease outcomes utilizing diverse modeling methodologies. The comparative effects of advanced medical treatments, secondary prevention via statin utilization, and primary prevention through sodium intake reduction were analyzed in a comprehensive Markov model specially designed for the Tunisian demographic (Saidi et al., 2019). The model suggests that the reduction of sodium intake could potentially lead to a 27% decrease in mortality attributable to ischemic heart disease and stroke, significantly exceeding the impact of secondary prevention (3%) and pharmacological interventions (1%). This underscores the substantial efficacy of primary prevention strategies in mitigating cardiovascular mortality. According to (Agbo et al., 2024), the model integrates agent-based modeling to reproduce individual-level modifications in lifestyle and environmental exposures, along with interconnected ordinary differential equations to elucidate population-level dynamics in disease transmission. The model is governed by the assumptions that risk factors are linearly additive, that populations exhibit homogeneity, and that parameters remain invariant over time (Agbo et al., 2024).

Numerous academic publications elucidate the utilization of Lyapunov functions and Volterra-Lyapunov matrices within the domain of global stability analysis. For instance, (Masoumnezhad et al., 2020) provide a comprehensive refinement of the Volterra-Lyapunov matrix methodology aimed at establishing the global stability of endemic equilibria in infectious disease models. In a parallel context (Sadki et al., 2023) apply the Lyapunov-LaSalle invariance principle to execute a global stability analysis pertaining to the dynamics of hepatitis C virus infection (Sadki et al., 2023). Such methodologies possess the potential for adaptation in the examination of global stability characteristics within heart disease models. Notably, a plethora of scholarly articles also introduces alternative methodologies for conducting global stability analysis. (Cardoso et al., 2021) expand upon Barbalat's Lemma to encompass fractional-order systems, thereby facilitating asymptotic analysis (Cardoso et al., 2021), while (Kifle & Obsu, 2022) apply the Castillo-Chavez and Song framework to substantiate the global stability of the disease free equilibrium point (Kifle & Obsu, 2022). The diversity inherent in these methodologies accentuates the importance of judiciously selecting the appropriate techniques predicated upon the unique attributes of the heart disease model under scrutiny.

The existing corpus of literature provides a substantial framework for understanding this epidemiology of heart disease, the associated risk factors, and the importance of employing mathematical modeling techniques. However, there is a notable shortfall in the application of the global stability analysis to heart disease model. By employing global stability analysis techniques, including LaSalle's Invariance Principle and linear stability analysis, this research endeavor aims to formulate a model based on the reproduction number, endemic equilibrium, and disease free equilibrium.

## Materials and Methods

This section discusses the global stability of the heart disease model developed by (Agbo et al., 2025).The model consists of six compartments: the population of susceptible healthy individuals to the disease, denoted as S, population of individual who are exposed to the disease by lifestyle and genetic disposition is denoted by E1 and E2, the population of infected individuals I, population of individual receiving treatment is denoted by T and the population of recovered individuals R and the model also consider the interaction between the compartment denoted by different parameter. Figure1 shows the schematic diagram of the heart disease model and Table1 shows the parameters with its description.


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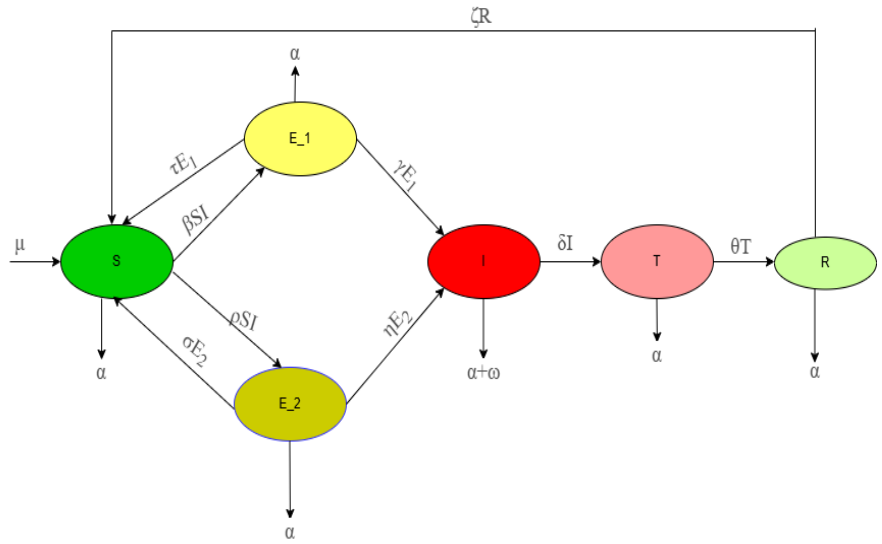


**Fig1: Schematic diagram of heart disease model (Agbo et al., 2025)**

The model equations are as follows

$$\frac{dS}{dt} = \mu - (\beta + \rho)SI - \alpha S + \tau E_1 + \sigma E_2 + \zeta R \quad (1)$$

$$\frac{dE_1}{dt} = \beta SI - (\gamma + \tau + \alpha)E_1 \quad (2)$$

$$\frac{dE_2}{dt} = \rho SI - (\eta + \sigma + \alpha)E_2 \quad (3)$$

$$\frac{dI}{dt} = \gamma E_1 + \eta E_2 - (\alpha + \omega + \delta)I \quad (4)$$

$$\frac{dT}{dt} = \delta I - (\alpha + \theta)T \quad (5)$$

$$\frac{dR}{dt} = \theta T - (\alpha + \zeta)R \quad (6)$$

**Table1: Model parameters and description**

| S/NO | PARAMETERS | DESCRIPTION |
|---|---|---|
| 1 | $\mu$ | Birth rate |
| 2 | $\alpha$ | Natural death rate |
| 3 | $\beta$ | Transmission rate by unhealthy lifestyles |
| 4 | $\rho$ | Transmission rate of heart diseases through genetic disposition |
| 5 | $\gamma$ | Rate of infection due to unhealthy dietary habit |
| 6 | $\tau$ | Rate of effective change in lifestyle |
| 7 | $\sigma$ | Rate of control on genetic transmission |
| 8 | $\eta$ | Infection transmission rate through to genetic |
| 9 | $\omega$ | Mortality rate cause by heart diseases |
| 10 | $\delta$ | Treatment rate |
| 11 | $\theta$ | Recovery rate |
| 12 | $\zeta$ | Loss of immunity |


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## Results

This section provide the analytical solution to the heart disease model finding the positivity and boundedness of solution and the disease free equilibrium. We then compute the basic reproduction number and analyse the stability of the disease-free equilibrium point.

### Positivity and boundedness

For equation (1)-(3), let $S(0) = S_0 \geq 0$, $\mathrm{E}_1(0) = E_{1_0} \geq 0$, $\mathrm{E}_2(0) = E_{2_0} \geq 0$, $I(0) = I_0 \geq 0$, $T(0) = T_0 \geq 0, R(0) = R_0 \geq 0$ as initial condition. To show the positivity and boundedness of the model let $\Pi = (\beta + \rho)I$ from equation(1) then

$$\dot{S} = \mu - \Pi \mathrm{S} - \alpha \mathrm{S} \quad (7)$$

$$\frac{dS}{dt} = -(\Pi + \alpha)S \equiv \frac{dS}{dt} = -(\Pi + \alpha)dt \quad (8)$$

Integrating (8)

$$\int_0^S \frac{dt}{S} \geq -\int_0^t (\Pi + \alpha)Sdt \quad (9)$$

$$\ln|S(t)| \geq -(\Pi + \alpha)S(t) + C \quad (10)$$

Hence

$$\mathrm{S(t)} \geq \mathrm{Ce}^{-(\Pi+\alpha)S(t)} \quad (11)$$

At time $\mathrm{t} = 0, \Rightarrow \mathrm{S(t)} \geq \mathrm{Ce}^{-(\Pi+\alpha)S(t)} \geq 0$ Since $-(\Pi + \alpha) \geq 0$. (12)

Similarly, it can be shown that $S_0 > 0, E_{1_0} > 0, E_{2_0} > 0, I_0 > 0, T_0 > 0, R_0 > 0$.

hence all the solutions of the model Equation (1-6) remain positive for all non-negative initial conditions as required at all time $\mathrm{t} > 0$. hence prove completed.

### Stability analysis

Stability analysis is carried out to determine the disease-free equilibrium point and endemic equilibrium point. To determine the two equilibrium points, each equation (1-6) must be equal to zero(0) or $\frac{dS}{dt} = 0$, $\frac{dE_1}{dt} = 0$, $\frac{dE_2}{dt} = 0$, $\frac{dI}{dt} = 0, \frac{dT}{dt} = 0, \frac{dR}{dt} = 0$ thus obtained

$$0 = \mu - (\beta + \rho)SI - \alpha S + \tau E_1 + \sigma E_2 + \zeta R \quad (13)$$

$$0 = \beta SI - (\gamma + \tau + \alpha)E_1 \quad (14)$$

$$0 = \rho SI - (\eta + \sigma + \alpha)E_2 \quad (15)$$

$$0 = \gamma E_1 + \eta E_2 - (\alpha + \omega + \delta)I \quad (16)$$

$$0 = \delta I - (\alpha + \theta)T \quad (17)$$

$$0 = \theta T - (\alpha + \zeta)R \quad (18)$$

Which is the stable state of $S, E_1, E_2, I, T \; and \; R$ .

### Disease free equilibrium

Equilibrium point for disease-free are conditions where there is no disease which implies

$$E_1 = E_2 = I = T = R = 0 \quad (19)$$

then from eqn (13)


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$$S = \frac{\mu}{\alpha} \tag{20}$$

Then, the equilibrium point of disease free for the heart disease model are

$$K_0 = (S, E_1, E_2, I, T, R) = (\frac{\mu}{\alpha}, 0,0,0,0,0) \tag{21}$$

**Basic reproduction number**

The basic reproduction number represents the quantity of secondary infections generated by a single infected individual within a completely susceptible community. This is acquired with next-generation matrix (Alsulami et al., 2024). Initially, we define the matrices F and V in the following manner:

$$F = \begin{pmatrix} \beta SI \\ \rho SI \end{pmatrix} \quad V = \begin{pmatrix} (\gamma + \tau + \alpha)E_1 \\ (\eta + \sigma + \alpha)E_2 \\ (\alpha + \delta + \omega)\, I - \gamma E_1 - \eta E_2 \\ (\alpha + \theta)T - \delta I \end{pmatrix} \tag{22}$$

The Jacobian matrices of F and V at $K_0$ when $S = \frac{\mu}{\beta}$ where the transition and transmission matrix respectively gives

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta\mu}{\alpha} & 0 \\ 0 & 0 & \frac{\rho\mu}{\alpha} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} (\gamma + \tau + \alpha) & 0 & 0 & 0 \\ 0 & (\eta + \sigma + \alpha) & 0 & 0 \\ \gamma & \eta & (\alpha + \omega + \delta) & 0 \\ 0 & 0 & \delta & (\alpha + \theta) \end{pmatrix} \tag{23}$$

$FV^{-1} =$

$$\begin{pmatrix} \frac{\beta\gamma\mu}{\alpha(\alpha^2 + \alpha\delta + \alpha\gamma + \alpha\omega + \alpha\tau + \delta\gamma + \delta\tau + \gamma\omega + \omega\tau)} & \frac{\beta\eta\mu}{\alpha(\alpha^2 + \alpha\delta + \alpha\eta + \alpha\omega + \alpha\sigma + \delta\eta + \delta\eta + \delta\sigma + \eta\omega + \omega\sigma)} & \frac{\beta\mu}{\alpha(\alpha + \delta + \omega)} & 0 \\ \frac{\gamma\rho\mu}{\alpha(\alpha^2 + \alpha\delta + \alpha\gamma + \alpha\omega + \alpha\tau + \delta\gamma + \delta\tau + \gamma\omega + \omega\tau)} & \frac{\eta\rho\mu}{\alpha(\alpha^2 + \alpha\delta + \alpha\eta + +\alpha\omega + \alpha\sigma + \delta\eta + \delta\sigma + \eta\omega + \omega\sigma)} & \frac{\rho\mu}{\alpha(\alpha + \delta + \omega)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

It follows that the basic reproduction number, denoted by $R_0$ given by $\sigma(FV^{-1})$ where $\sigma$ denoted the spectral radius is

$$R_0 = \frac{\mu(\alpha\beta\gamma + \alpha\eta\rho + \beta\eta\gamma + \beta\gamma\sigma + \eta\gamma\rho + \eta\rho\tau)}{\alpha(\alpha+\delta+\omega)(\alpha+\eta+\sigma)(\alpha+\gamma+\tau)} \tag{24}$$

**Global stability analysis**

Stability analysis is fundamental to dynamical analysis. Only stable solutions can be observed empirically. In this part, we analyse the global asymptotic stability of all equilibrium point by constructing appropriate Lyapunov functions and utilising the Lyapunov-LaSalle asymptotic stability theorem (L-LAST).

**Global stability of the disease-free equilibrium**

Global stability of the disease-free equilibrium when $R_0 \leq 1$ using Lasalle's Invariance Principle.

**Theorem1**


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The disease-free equilibrium $K_0 = (S, E_1, E_2, I, T, R) = (\frac{\mu}{\alpha}, 0,0,0,0,0)$ of model equation is globally asymptotically stable if $R_0 > 0$ and $R_0 \leq 1$.

**Proof**

Using the method used in (Oladipupo et al., 2023). Let the Lyapunov function be $\nabla(S, E_1, E_2, I, T, R)$ according to the approach in then

$$\nabla = (S, E_1, E_2, I, T, R) = (S - S_0 - S_0 \ln \frac{S}{S_0}) + E_1' + E_2' + I' + T' + R' \quad (25)$$

Differentiating $\nabla(S, E_1, E_2, I, T, R)$ with respect to time gives

$$\nabla' = (1 - \frac{S}{S_0})S' + E_1' + E_2' + I' + T' + R' \quad (26)$$

Substituting equation (1-6) into (33) and $S_0 = \frac{\mu}{\alpha}$ to give

$$= (1 - \frac{S}{S_0})(\mu - (\beta + \rho)IS - \alpha S + \tau E_1 + \sigma E_2 + \zeta R) + \beta IS - (\gamma + \tau + \alpha)E_1 + \rho IS - (\eta + \sigma + \alpha)E_2 + \gamma E_1 + \eta E_2 - (\alpha + \omega + \delta)I + \delta I - (\alpha + \theta)T + \theta T - (\alpha + \zeta)R \quad (27)$$

$$= (\mu - (\beta + \rho)IS - \alpha S + \tau E_1 + \sigma E_2 + \zeta R) - \mu \frac{S_0}{S} + (\beta + \rho)IS \frac{S_0}{S} + \alpha S \frac{S_0}{S} - \tau E_1 \frac{S_0}{S} - \sigma E_2 \frac{S_0}{S} - \zeta R \frac{S_0}{S} - \alpha(S, E_1, E_2, I, T, R) \quad (28)$$

Simplifying

$$\mu - \mu \frac{S}{S_0} - \frac{\mu S_0}{S} + (\beta + \rho)IS_0 + \mu - (\tau E_1 + \sigma E_2 + \zeta R)\frac{S_0}{S} - \alpha(S, E_1, E_2, I, T, R) \quad (29)$$

$$= \mu(2 - \frac{S}{S_0} - \frac{S_0}{S}) + (\beta + \rho)IS_0 - \alpha(S, E_1, E_2, I, T, R) \quad (30)$$

From equation (1) $(\beta + \rho)IS$ and $S_0 = \frac{\mu}{\alpha}$ are non-negatives hence

$$\nabla' \leq \mu(2 - \frac{S}{S_0} - \frac{S_0}{S}) + (\beta + \rho)IS_0 - \alpha(S, E_1, E_2, I, T, R) \quad (31)$$

By the inequality of arithmetic and geometric means

$$\frac{\mu(2SS_0 - (S_0^2 + S^2))}{SS_0} - \alpha(S, E_1, E_2, I, T, R) \leq 0 \quad (32)$$

This proved that $\nabla$ is the lyapunov function of $\nabla' = 0$ which implies that $E_1 = E_2 = I = T = R = 0$. Therefore, it follows that the larges invariant set in

$(S + E_1 + E_2 + I + T + R) \in \Psi: \nabla' = 0$ is

$$K_0 = (\frac{\mu}{\alpha}, 0,0,0,0,0) \quad (33)$$

Hence, by Lasalle's invariance principle the disease free equilibrium is globally asymptotically stable.

**Numerical simulation**

This section will quantitatively illustrate the global stability of the disease free equilibrium. The simulations were conducted utilising the relevant commands and packages in R programming. Table2 shows the parameters value and source utilise and setting the initial condition of $(S, E_1, E_2, I, T, R) = (1000, 200,100,40,28,16)$.

**Table2: Value of the model parameters corresponding to heart disease case**


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| Parameter | Value/day | Sources |
|---|---|---|
| $\mu$ | 0.02 | numerical estimate |
| $\alpha$ | 0.3 | numerical estimate |
| $\beta$ | 0.003 | (Yang et al., 2016) |
| $\rho$ | 0.002 | (Yang et al., 2016) |
| $\gamma$ | 0.003 | (Jibril & Odetunde 2020) |
| $\tau$ | 0.02 | numerical estimate |
| $\sigma$ | 0.009 | numerical estimate |
| $\eta$ | 0.001 | numerical estimate |
| $\omega$ | 0.55 | numerical estimate |
| $\delta$ | 0.95 | (McBryde et al., 2017) |
| $\theta$ | 0.2 | numerical estimate |
| Z | 0.3 | (Jibril & Odetunde 2020) |

**Discussion**

The formulated compartmental model for heart disease transmission and prevention dynamics was analyzed to determine its key epidemiological properties. The **positivity and boundedness** of solutions were first established, confirming that all state variables remain non-negative and bounded for all non-negative initial conditions, thus ensuring the model's biological feasibility. The **disease-free equilibrium (DFE)** was derived by setting all infected and exposed compartments to zero, yielding the steady-state values for the susceptible population in the absence of disease. The **basic reproduction number ($R_0$)** was computed using the next-generation matrix approach. This threshold parameter quantifies the expected number of secondary cases generated by one infected individual in a fully susceptible population. A **global stability analysis** of the DFE was performed using LaSalle's Invariance Principle, supported by the construction of an appropriate **Lyapunov function**. The results show that if $R_0 < 1$ the DFE is **globally asymptotically stable**; that is, the system will return to a disease-free state regardless of the initial distribution of the population across compartments. Conversely, if $R_0 > 1$**,** the disease persists and the system may approach an endemic equilibrium.

Numerical simulations were carried out in **R** using the parameter values provided in Table 2, with initial conditions designed to reflect a population containing a mix of susceptible, exposed1 and 2, infected, treated, and recovered individuals. The simulations aimed to validate the theoretical prediction of global stability for the DFE when $R_0 < 1$ **shown in figure2.**


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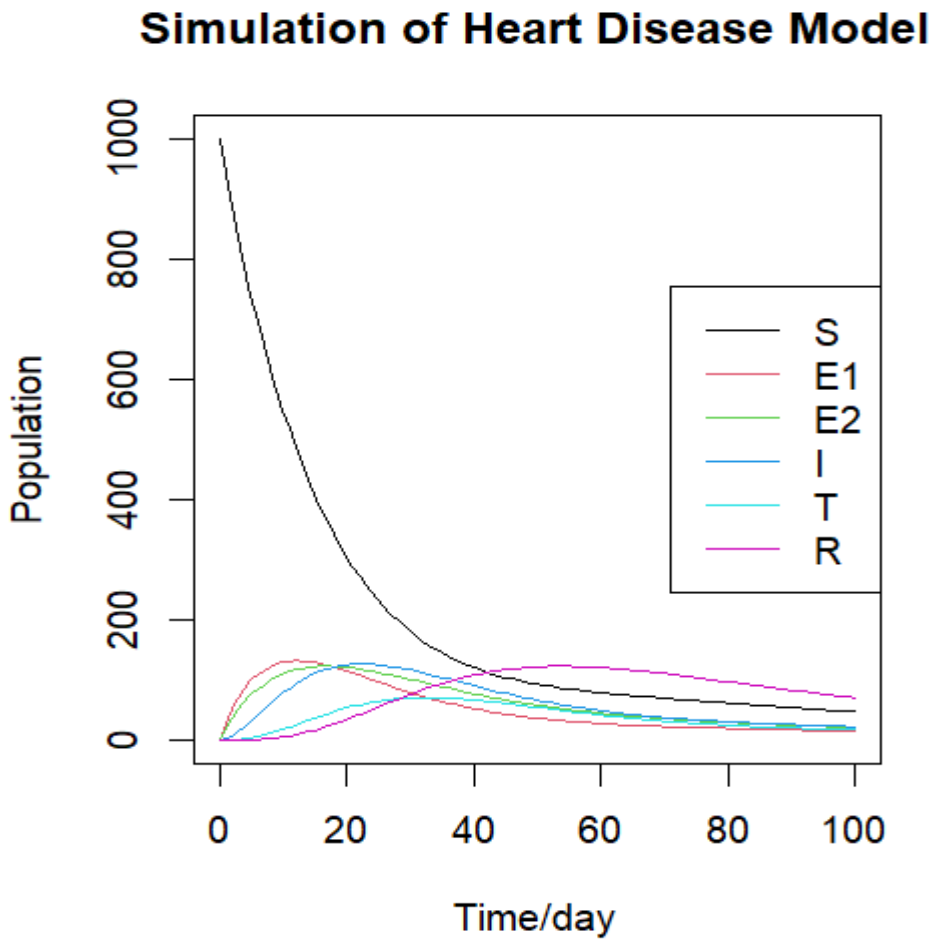


**Fig2: Simulation of the Heart Disease model at disease free equilibruim**

The trajectories of the model variables, illustrated in Figure 2**,** reveal a consistent decline of the exposed and infected compartments toward zero, alongside stabilization of the susceptible population at its disease-free value. The approach to equilibrium is smooth and monotonic, without oscillations, confirming the absence of complex dynamics such as sustained periodicity or chaos under the tested parameter regime. The numerical outcomes are in strong agreement with the analytical findings, thereby providing mutual validation between theory and simulation specifically in the Role of Risk Factors and Public Health Relevance **s**ince $R_0$ encapsulates parameters representing lifestyle-related and genetic transmission pathways, targeted interventions such as promoting healthier diets, increasing physical activity, controlling genetic predisposition through early screening, and enhancing treatment efficacy are pivotal for disease control. The model's behavior underscores the primacy of primary prevention measures. Reducing lifestyle-related risk transmission rates has a disproportionately large impact on $R_0$ compared to reactive measures alone. This aligns with real-world epidemiological evidence favoring preventive health strategies over purely treatment-based approaches.

**Conclusion**

This paper offers a comprehensive mathematical framework for analyzing the transmission and prevention dynamics of heart disease, incorporating both lifestyle-related and genetic risk factors. Analytical investigations established the positivity and boundedness of solutions, determined the disease-free equilibrium, and derived the basic reproduction number $R_0$ using the next-generation matrix method. Global stability analysis, grounded in LaSalle's Invariance Principle and Lyapunov function construction, demonstrated that the disease-free equilibrium is globally asymptotically stable when $R_0<1$. Numerical simulations in **R** confirmed the theoretical predictions, showing that, under suitable intervention strategies, disease prevalence declines monotonically to


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zero across a broad range of initial conditions. The findings highlight the critical importance of reducing $R_0$ through sustained **primary prevention measures**, such as promoting healthy lifestyles, early diagnosis, and effective treatment interventions. By providing both a robust theoretical foundation and empirical validation, this work offers valuable guidance for public health decision-making. Future research could enhance the model's applicability by incorporating demographic heterogeneity, spatial effects, and stochastic influences, thereby improving predictive accuracy and policy relevance in real-world settings.

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