A Mathematical Model of Cardiovascular Disease Dynamics Incorporating Personal Risk Factors

Abstract

Cardiovascular disease pose a significant global health challenge, demanding innovative approaches for prevention and control. Cardiovascular disease is the leading cause of death worldwide as it affects people of all ages, sexes, ethnicities and socioeconomic levels. However, the disease can largely be prevented by leading a healthy lifestyle. In this study, a deterministic mathematical model for cardiovascular disease incorporating personal risk factors was developed and analyzed. The model incorporates lifestyle modifications as key components, recognizing their impact on Cardiovascular disease risk. Stability analysis of the model showed that the disease free equilibrium point is locally asymptotically stable whenever $R_0 < 1$. The endemic equilibrium exist and is locally asymptotically stable whenever $R_0 > 1$. Numerical simulation of the model highlights a critical threshold behavior, when τ crosses approximately 0.63 the point where $R_0 = 1$, the system transitions from disease elimination to endemic persistence. This threshold is derived from the model's parameters, emphasizing that, controlling personal risk factors below this critical value can prevent the disease from becoming endemic.

Keywords: Cardiovascular disease, Personal risk factors, Stability analysis .

1 Introduction

Cardiovascular disease (CVD), a group of diseases affecting the heart and blood vessels has emerged as a significant global epidemic, imposing a substantial burden on health, society, and the economy. These diseases can affect one or many parts of your heart and/or blood vessels. A person may be symptomatic, physically experiencing the disease or asymptomatic, not feeling anything at all. Cardiovascular disease symptoms can vary depending on the cause. Older adults and women may have more subtle symptoms. However,

they can still have serious cardiovascular disease. Symptoms of cardiovascular disease include; Chest pain, Chest pressure, heaviness or discomfort, shortness of breath, dizziness or fainting and fatigue or exhaustion [4, 6].

The global crude prevalence of cardiovascular disease is expected to almost double from 598 million in 2025 to 1.14 billion in 2050, corresponding to a 3.6 percent year-on-year increase while the global mortality is projected to increase from 20.5 million deaths in 2025 to 35.6 million deaths in 2050 representing a 73.4 percent. Metabolic risk factors remain the leading factor underlying cardiovascular mortality accounting for 51.7 million, followed by behavioural accounting 18.8 million and environmental risk factors accounting 9.5 million [2].

A number of mathematical models have been developed and analyzed to explain the dynamics of diseases in humans. Sagar Gupta [4] modelled the impact of community awareness on the dynamics of cardiovascular infection. Aware people change their lifestyle that includes daily physical exercises, taking healthy food timely and live stress-free life etc. thereby preventing the onset of cardiovascular disease and its complications. A model incorporating the reproduction number, endemic equilibrium and disease-free equilibrium was developed utilizing stability analysis methods, including the Hurwitz stability criterion and linear stability analysis [1]. The model assumes linear additive of risk factors, homogeneity within populations and stable parameters over time. It incorporates lifestyle modifications, genetic predispositions, and environmental exposures as key components, recognizing their impact on heart disease risk.

A mathematical model that describes the population dynamics of heart failure and examines its stability was developed in [11]. The model consist of three nonlinear ordinary differential equations that describe the interaction between individuals at risk of heart failure, heart failure patients, and heart transplant patients. The findings of the study show that the parameters that should be controlled are the rate of acquired risk factors later in life, the probability of reversing modifiable risk factors, the progression rate from at-risk individuals to heart failure patients, the availability of heart transplant resources, the success rate of transplants, the rate of failed transplants and the saturation factor. Control measures include implementing educational and vaccination programs, promoting lifestyle changes, conducting regular screenings, and expanding heart transplant resources.

Personal risk factors for cardiovascular disease (CVD) are individual characteristics that increase the likelihood of developing heart disease, stroke or

other cardiovascular conditions. These factors can be changed through lifestyle choices. Lately, Public health campaigns against cardiovascular disease (CVD) have increasingly focused on preventing the prevalence of the disease by addressing these modifiable risk factors such as unhealthy diet, physical inactivity, tobacco use and harmful use of alcohol. The effects of these risk factors may show up in individuals as raised blood pressure, raised blood glucose, raised blood lipids, and overweight and obesity. Thus, in the phase of diminishing donor support for health programmes in the developing countries, prevention against the personal risk factors to cardiovascular disease is an option worth exploring [7].

2 The model

We formulate a model in which the total human population at any time t denoted by N is subdivided into classes, $S(t)$ the class of individuals susceptible to Cardiovascular disease. Recruitment into susceptible class is done at a rate Λ. The class $E(t)$ consist of individuals exposed to Cardiovascular disease. This infection occurs at the rate λ. ρ is the rate of progression to the $I(t)$ class after experiencing changes in cardiovascular health. The class $R(t)$ consist of individuals who have recovered from the Cardiovascular disease impact. This compartment considers lifestyle modifications, medical interventions, or other factors influencing recovery. Mortality occurs among the cardiovascular patients at the rate ν while natural death is assumed to occur in all classes at the rate μ.

The rate at which the susceptible individuals acquire Cardiovascular disease is defined as

$$\lambda = \frac{\theta\tau I(t)}{N(t)} \tag{1}$$

Where θ is the probability that susceptible individuals will be exposed to the Cardiovascular disease which is linked to an increased risk of the disease events in the short and long term. This increased risk is particularly pronounced in individuals with pre-existing high CVD risk making them more susceptible to cardiovascular issues. This study sought to investigate the role of personal risk factors on the dynamics and management of Cardiovascular disease. Personal risk factors that can contribute to high disease prevalence include unhealthy diet, physical inactivity, tobacco use and harmful use of alcohol among others. Therefore, τ denotes the personal risk factors.

From the above definitions, the resulting diagram for the model is given below.

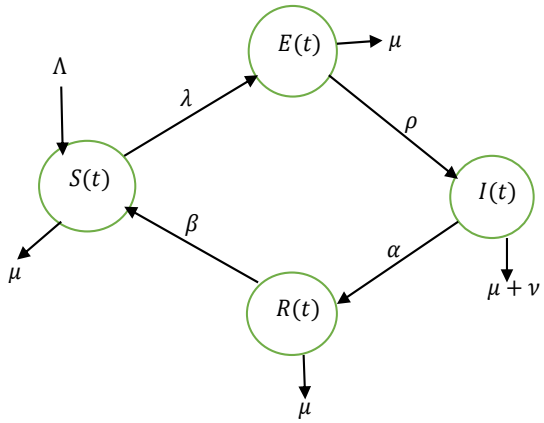


Figure 1: Model flow diagram

The dynamics described can be represented mathematically as:

$$\begin{aligned}
\dot{S}(t) &= \Lambda + \beta R(t) - \frac{\theta\tau I(t)}{N(t)} S(t) - \mu S(t) \\
\dot{E}(t) &= \frac{\theta\tau I(t)}{N(t)} S(t) - (\rho + \mu) E(t) \\
\dot{I}(t) &= \rho E(t) - (\nu + \alpha + \mu) I(t) \\
\dot{R}(t) &= \alpha I(t) - (\beta + \mu) R(t) \qquad (2)
\end{aligned}$$

3 Analysis of the Model

Since the model describes a human population, all the state variables and parameters will be non negative $t > 0$ in the feasible region Ω where $S(t), E(t), I(t), R(t) \in \Omega \subset R^4_+$. It can be shown that all the solutions are bounded in Ω, $\forall t > 0$ such that $0 \leq N \leq \frac{\Lambda}{\mu}$. Thus the model is epidemiologically well posed in the region Ω and can be analysed.

4 The basic reproduction number

The dynamics of the model are highly dependant on the basic reproduction number. The basic reproduction R_0 is the number of newly infected people followed by only one infected individual in a totally susceptible population. The basic reproduction number, R_0, for model (2) computed using the next

generation matrix method as used in [3]

$$R_0 = \frac{\theta\tau\rho}{(\mu+\alpha+\nu)(\mu+\rho)} \tag{3}$$

5 Disease-free Equilibrium point (DFE)

The disease-free equilibrium point, denoted by E_o is a steady-state solution for which there is no disease in the population [8]. To obtain the disease-free equilibrium point we set the normalized model system (2) equal to zero. Since there are no disease in the human populations, we set $E(t) = I(t) = 0$. This implies that $E_0 = \{S_0, E_0, I_0, R_0\} = \{\frac{\Lambda}{\mu}, 0, 0, 0\}$

6 Existence of Endemic Equilibrium

At the Endemic equilibrium point, persistence of the disease occurs and thus, at least one of the infected classes is greater than zero. The endemic equilibrium of model (2) is denoted by

$$E_e(S^*(t), E^*(t), I^*(t), R^*(t)). \tag{4}$$

Theorem 6.1. *Cardiovascular disease exist and persist in the population if one of the infected classes E^* and I^* is greater than zero whenever $R_0 > 1$*

Proof. Using mathematica software, the endemic state for $I^*(t)$ was given as

$$I^*(t) = -\frac{(\beta+\mu)N\mu(1-R_0)\Lambda(\rho+\mu)(\nu+\alpha+\mu)}{\theta(\beta+\mu)(\mu+\nu)(\mu+\rho)+\alpha\mu(\beta+\mu+\rho)\tau} \tag{5}$$

From Equation (5), $I^*(t) > 0$ whenever $R_0 > 1$ and this leads to the disease invading the susceptible population. □

7 Local Stability Analysis of the Disease Free Equilibrium (DFE)

The model in Equation (2) has disease free equilibrium given by $E_0 = \{S_0, E_0, I_0, R_0\} = \{\frac{\Lambda}{\mu}, 0, 0, 0\}$

Theorem 7.1. *If $R_0 < 1$ then $E_0 = \{S_0, E_0, I_0, R_0\} = \{\frac{\Lambda}{\mu}, 0, 0, 0\}$ is an equilibrium state in Ω and is locally asymptotically stable otherwise unstable.*

Proof. Consider the Jacobian matrix of Equation (2) given by

$$J = \begin{pmatrix} -\frac{\theta\tau I(t)}{N(t)} - \mu & 0 & -\frac{\theta\tau S(t)}{N(t)} & \beta \\ \frac{\theta\tau I(t)}{N(t)} & -(\rho+\mu) & \frac{\theta\tau S(t)}{N(t)} & 0 \\ 0 & \rho & -(\nu+\alpha+\mu) & 0 \\ 0 & 0 & \alpha & -(\mu+\beta) \end{pmatrix} \tag{6}$$

The Jacobian matrix of Equation (6) at DFE is given by

$$J_{E_0} = \begin{pmatrix} -\mu & 0 & -\theta\tau & \beta \\ 0 & -(\rho+\mu) & \theta\tau & 0 \\ 0 & \rho & -(\nu+\alpha+\mu) & 0 \\ 0 & 0 & \alpha & -(\mu+\beta) \end{pmatrix} \tag{7}$$

Clearly $-\mu$ and $-(\mu+\beta)$ are eigenvalues. We analyse the reduced matrix

$$J_{E_0} = \begin{pmatrix} -(\rho+\mu) & \theta\tau \\ \rho & -(\nu+\alpha+\mu) \end{pmatrix} \tag{8}$$

Using Routh-Hurwitz criterion [10], analyzing the stability of the Jacobian at DFE, the trace of Equation (8) is negative and the determinant is given by

$$Det(J_{E_0}) = (\rho+\mu)(\nu+\alpha+\mu) - \rho\theta\tau$$

Which simplifies to;

$$Det(J_{E_0}) = (1-R_0)(\rho+\mu)(\nu+\alpha+\mu) > 0 \tag{9}$$

The determinant of the Jacobian matrix at DFE given by equation (9) remains positive provide that $R_0 < 1$. Therefore, by Routh-Hurwitz criterion [10], the disease-free equilibrium of model (2) is locally asymptotically stable. This implies that, small perturbations from the DFE will decay over time, leading the system back to the disease-free state if $R_0 < 1$. □

7.1 Local stability of endemic equilibrium point

Theorem 7.2. *If $R_0 > 1$, then the endemic equilibrium $E_e\{S^*(t), (E^*(t), I^*(t), R^*(t)\}$, is locally asymptotically stable*

Proof. The Jacobian of Equation (2) at endemic state is given by

$$J = \begin{pmatrix} -\frac{\theta\tau I^*}{N^*} - \mu & 0 & -\frac{\theta\tau S^*}{N^*} & \beta \\ \frac{\theta\tau I^*}{N^*} & -(\rho+\mu) & \frac{\theta\tau S^*}{N^*} & 0 \\ 0 & \rho & -(\nu+\alpha+\mu) & 0 \\ 0 & 0 & \alpha & -(\mu+\beta) \end{pmatrix} \tag{10}$$

Where $I^*(t) = -\frac{(\beta+\mu)N\mu(1-R_0)\Lambda(\rho+\mu)(\nu+\alpha+\mu)}{\theta(\beta+\mu)(\mu+\nu)(\mu+\rho)+\alpha\mu(\beta+\mu+\rho)\tau}$ and $S^*(t) = \frac{N(\alpha+\mu+\nu)(\mu+\rho)}{\theta\tau\rho}$

Using Routh-Hurwitz criterion [10], analyzing the stability of the Jacobian at EE where $R_0 > 1$, then trace of Equation (13) is negative and the determinant computed using Mathematica gives

$$Det(J_e) = -\frac{I^*\alpha\beta\tau\theta\rho}{N}+(-\beta-\mu)\{\frac{S^*\theta\tau\mu\rho}{N}+(-\alpha-\mu-\tau)(\frac{I^*\tau\theta\mu}{N}+\mu^2+\frac{I^*\gamma\theta\rho}{N}+\mu\rho)\}$$

The determinant $DetJ_e(E^*) > 0$ provided that;

$$(-\beta-\mu)\{\frac{S^*\theta\tau\mu\rho}{N}+(-\alpha-\mu-\tau)(\frac{I^*\tau\theta\mu}{N}+\mu^2+\frac{I^*\gamma\theta\rho}{N}+\mu\rho)\} > 0 \quad (11)$$

and

$$(-\beta-\mu)\{\frac{S^*\theta\tau\mu\rho}{N}+(-\alpha-\mu-\tau)(\frac{I^*\tau\theta\mu}{N}+\mu^2+\frac{I^*\gamma\theta\rho}{N}+\mu\rho)\} > \frac{I^*\alpha\beta\tau\theta\rho}{N} \quad (12)$$

Thus, by Routh-Hurwitz criterion, the endemic state $E^*\{S^*(t), E^*(t), I^*(t), R^*(t)\}$ is locally asymptotically stable provided that inequality (11) and (12) holds. Therefore if $R_0 > 1$ the disease will persist in the population.

□

8 Sensitivity Analysis

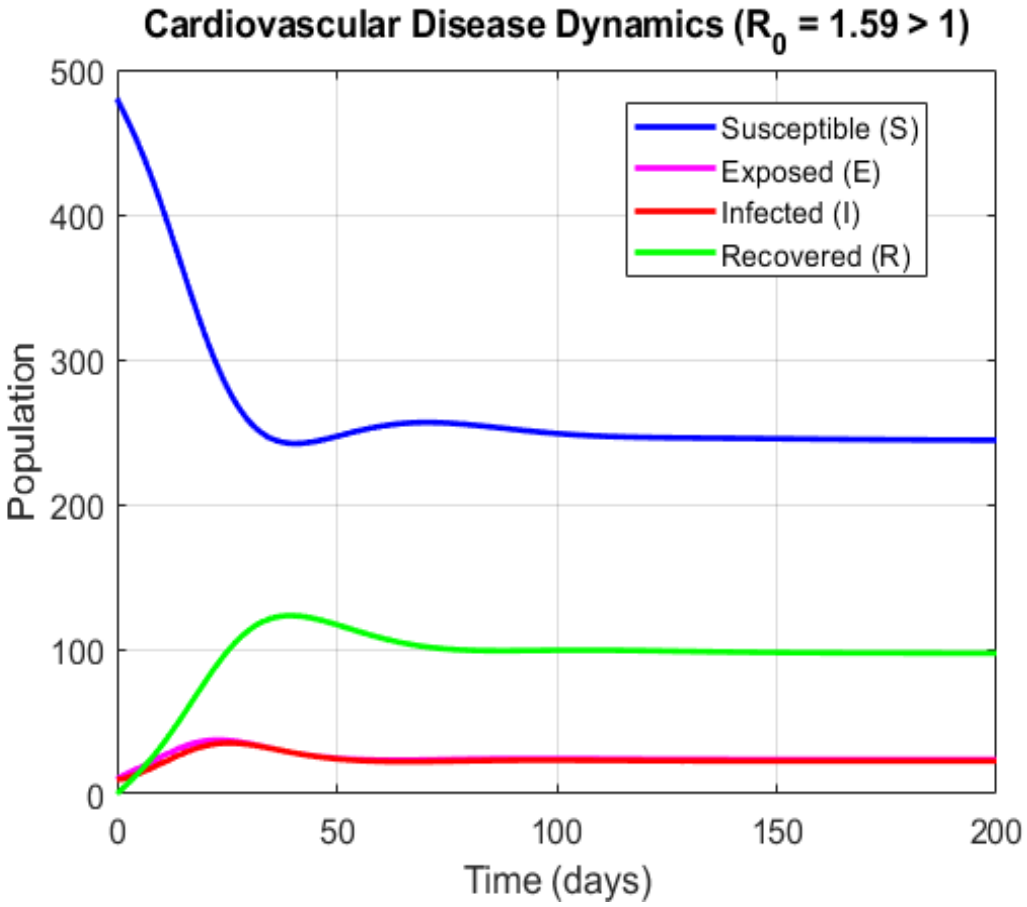
Sensitivity analysis of R_0 with respect to the model parameters was carried out to determine the role of risk factors on the dynamics and management of cardiovascular disease. To perform sensitivity analysis, the normalised forward sensitivity index also known as elasticity was used [5]. The normalised forward sensitivity index of the reproduction number R_0 in Equation (3) with respect to risk factors parameter τ is given by;

$$\Gamma_\tau^{R_0} = \frac{\partial R_0}{\tau} \times \frac{\tau}{R_0} \quad (13)$$

This implies that, the higher exposure to risk factors the higher the prevalence of cardiovascular disease.

9 Numerical simulation

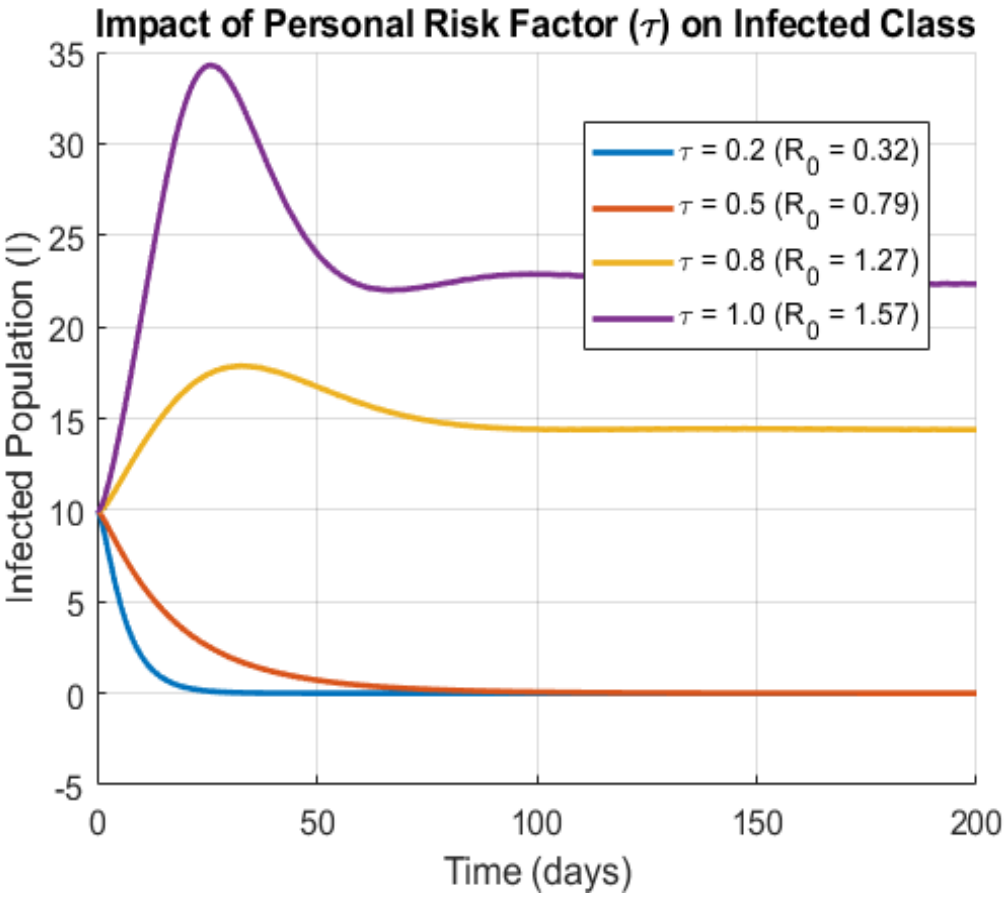
Numerical simulations are carried out to graphically illustrate the long term effect of risk factors on the dynamics of cardiovascular disease.



The graph depicts the progression of cardiovascular disease (CVD) through a population over time, with $R_0 = 1.59$ indicating an endemic scenario where the disease persists. Initially, the susceptible population $S(t)$ declines sharply as individuals become exposed $E(t)$ and then infected $I(t)$. The exposed population rises quickly before falling as people transition to the infected class, which peaks and then decreases due to recovery $R(t)$ or disease-induced mortality. Eventually, all compartments stabilize at non-zero values, reflecting an endemic equilibrium where the disease remains in the population at a steady level. The total population $N(t)$ remains relatively stable, with recruitment balancing natural deaths, though disease related mortality slightly reduces its size.

The dynamics highlight key epidemiological insights. The initial outbreak phase shows rapid transmission due to high R_0, leading to a significant peak

on disease prevalence. Over time, the depletion of susceptible individuals and the growth of the recovered population slow the spread, but the disease does not disappear entirely. Instead, it reaches an endemic state where new infections balance recoveries and deaths. This equilibrium is influenced by τ , where higher values correlate with more severe outbreaks and higher long-term disease burdens.



The graph illustrates how different levels of personal risk factors τ influence the dynamics of the infected population $I(t)$ in the cardiovascular disease model. When τ is low $(0.14$ and $0.5)$, corresponding to R_0 values of 0.16 and 0.79 which are both below 1, the infected population declines rapidly and eventually reaches zero. This occurs because the disease fails to sustain itself when $R_0 < 1$, leading to the eventual disappearance of the disease. The decline is slower for $\tau = 0.5$ compared to $\tau = 0.1$, reflecting the higher but still sub-critical prevalence potential.

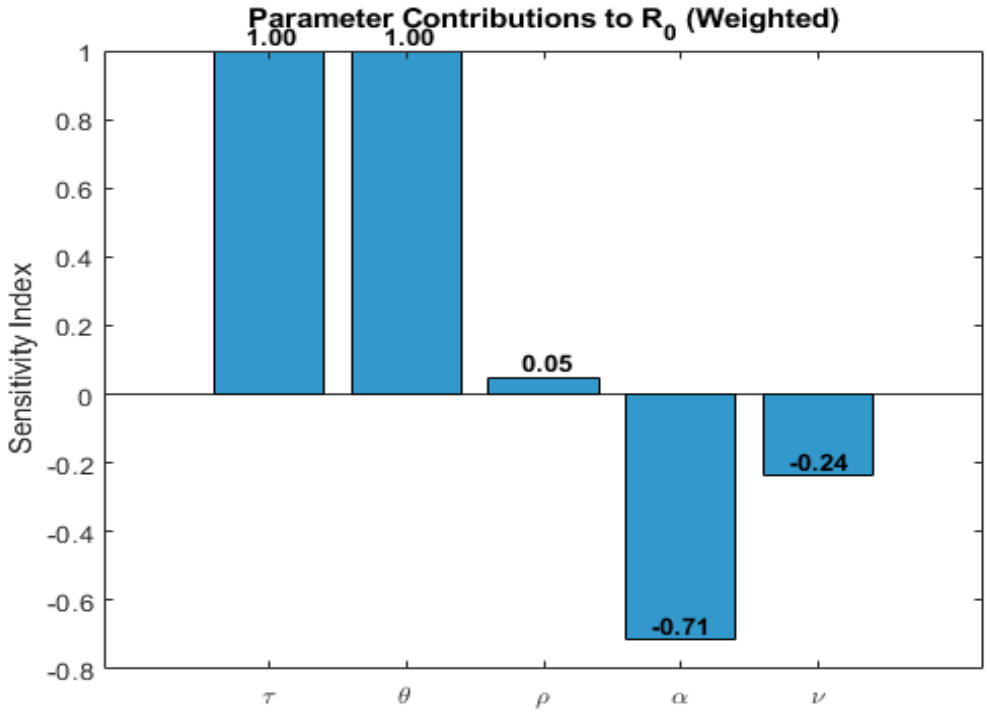
For higher τ values 0.8 and 1.0, where $R_0 > 1$, the infected population exhibits an initial outbreak, peaking at around 15 individuals for $\tau = 0.8$ and 30 individuals for $\tau = 1.0$. After the peak, the curves decline and stabilize at

endemic levels approximately 5 and 10 individuals, respectively. This stabilization happens because the system reaches an equilibrium where prevalence balance recoveries and deaths. The higher the τ , the greater the peak and the endemic level, demonstrating that increased personal risk factors lead to more severe outbreaks and a higher long-term disease burden.

The graph highlights a critical threshold behavior: when τ crosses approximately 0.63 the point where $R_0 = 1$, the system transitions from disease elimination to endemic persistence. This threshold is derived from the model's parameters, emphasizing that controlling personal risk factors below this critical value can prevent the disease from becoming endemic. Public health strategies targeting modifiable risk factors such as adopting a heart-healthy diet, regular physical activity, managing weight, quitting smoking and managing stress can effectively lower τ, thereby reducing $R_0 < 1$ and eliminating the disease. The results underscore the importance of preventive measures in mitigating cardiovascular disease prevalence, as even small reductions in τ can significantly alter the disease trajectory, moving the population from endemic persistence to complete eradication.

Generally, the graph not only demonstrates the direct relationship between personal risk factors and disease dynamics but also provides actionable insights for policymakers. By focusing on reducing τ, interventions can shift the system from an endemic state to one where the disease dies out, ultimately reducing the overall burden of cardiovascular disease in the population.

The sensitivity analysis graph clearly demonstrates that personal risk factors, τ exerts a substantially greater influence on cardiovascular disease prevalence than both θ and other model parameters. While τ and θ appear mathematically equivalent in the R_0 formula, τ real world impact dwarfs θ due to its greater modifiability and wider range of variability. Personal risk factors like unhealthy diet, physical inactivity, tobacco use and harmful use of alcohol represented by τ can be dramatically altered through public health interventions for instance, comprehensive anti-smoking programs, adopting a heart-healthy diet, regular physical activity, managing weight and managing stress might reduce τ by a given percentage leading to a proportional decline in the disease prevalence. In contrast, θ reflects more fixed biological factors like genetic susceptibility that might only change by a smaller percentage even with intensive interventions.



10 Conclusion

Prevention against the risk factors is considered one of the promising interventions against cardiovascular disease. Despite the advocacy of behaviour change and treatment, the prevalence and mortality rate of cardiovascular disease continues to be a problem. Thus, adopting a heart-healthy diet, regular physical activity, managing weight, quitting smoking and managing stress will help reduce and manage the disease prevalence. Moreover, Health education on the personal risk factors to Cardiovascular disease is paramount to raise public awareness and induce behavior change.

References

[1] **Agbo, C., Abah, R., and Abdullahi, A. M. (2024)**. A Mathematical Modeling on the Stability Analysis of Heart Disease Dynamics: Mathematical Modeling on the Stability Analysis of Heart Disease. Journal of Institutional Research, Big Data Analytics and Innovation, 1(1).

[2] **Chong, B., Jayabaskaran, J., Jauhari, S. M., Chan, S. P., Goh, R., Kueh, M. T. W., and Chan, M. Y. (2024)**. Global burden of cardiovascular diseases: projections from 2025 to 2050. European Journal of Preventive Cardiology, zwae281.

[3] **Castillo-Chavez, Feng Z. and Huang W.(2001)**. On the Computation of R0 and its Role on Global Stability, M-15553.

[4] **Gupta Sagar. (2023)**. Modeling the Impact of Community Awareness on the Dynamics of Cardiovascular Diseases. Butwal Campus Journal, 6(1), 88-95.

[5] **Mikucki, M. A. (2012)**. Sensistivity analysis of the basic reproduction number and other quantities for infectious disease models (Master's thesis, colorado state university).

[6] **Ng'ambi, W. F., Estill, J., Merzouki, F. A., Zyambo, C., Beran, D., and Keiser, O. (2025)**. The Prevalence, Prevention, and Treatment of Cardiovascular Diseases in Twelve African Countries (2014-2019): An Analysis of the World Health Organisation STEPwise Approach to Chronic Disease Risk Factor Surveillance. medRxiv, 2025-02.

[7] **Norsuhaimi Husna Norsuhaimi and Noraslinda Mohamed Ismail (2023)**. Survival Analysis and Factors of Heart Failure Disease. Survival, 18, 76-81.

[8] **Olaniyi S. and Obabiyi O. S.(2014)**. Qualitative analysis of malaria dynamics with nonlinear incidence function. *Applied Mathematical Sciences*, 8(78):3889-3904.

[9] **Prajapati, A. K., and Singh, U. K. (2022)**. An empirical analysis of ML techniques and/or algorithms for disease diagnosis prediction from the perspective of cardiovascular disease (CVD). International Journal of Computing Algorithm, 11(2), 6-16.

[10] **Routh E.,(1977)** A Treatise on the Stability of a Given State of Motion: Particularly Steady Motion, *Macmillan Publishers.*

[11] **TEE, S., and DAUD, A. A. M. (2025)**. Mathematical model and analysis of population dynamics for heart failure transplant. Journal of Quality Measurement and Analysis JQMA, 21(1), 69-85.