A cardiovascular disease mathematical model 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 often largely be prevented by leading a healthy lifestyle. In this study,a deterministic mathematical model for Cardiovascular disease incorporating personal risk factors and prevention measures is developed and analyzed. The model incorporates lifestyle modifications and environmental exposures as key components, recognizing their impact on Cardiovascular disease risk. The positivity and boundedness of the solution was proven. Using the next generation matrix approach, the reproduction number which is an essential parameter in analyzing the equilibrium for the model is determined and the respective disease free equilibrium points are shown to be locally and globally asymptotically stable. The endemic state is shown to exist provided that the reproduction number is greater than unity. By use of Routh-Hurwitz criterion and suitable Lyapunov functions, the endemic states are shown to be locally and globally asymptotically stable. Numerical simulations of the model using MATLAB are provided to verify and illustrate the analytical results. The findings of the study show that the parameters that should be controlled are the personal risk factors. Controlling these parameters can reduce the at-risk population thereby contributing to a lower overall prevalence of Cardiovascular disease.

Keywords: Cardiovascular disease, Personal risk factors, prevention measures.

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 heart issues include; Chest pain, Chest pressure, heaviness or discomfort, shortness of breath, dizziness or fainting and fatigue or exhaustion. Symptoms of blockages in blood vessels throughout your body include; pain or cramps in your legs when you walk, leg sores that are not healing, cool or red skin on your legs, swelling in your legs, numbness in your face or a limb [4].

The global crude prevalence of cardiovascular infection 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 [2].

A number of mathematical models have been developed and analyzed to explain the dynamics of infectious 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 [10]. 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.

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]. Lately, public heath campaigns against cardiovascular disease have focused on prevention against the risk factors. These Risk factors include; High Blood Pressure, high Cholesterol, smoking, diabetes, obesity, lack of Physical Activity and unhealthy diet. Thus, in the phase of diminishing donor support for health programmes in the developing countries, prevention against the risk factors to cardiovascular disease is an option worth exploring [5].

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 infection. Recruitment into susceptible class is done at a rate Λ. The class $E(t)$ consist of individuals exposed to Cardiovascular infection. 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 infection 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 infection is defined as

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

Where θ is the probability that susceptible individuals will acquire Cardiovascular infection. This study sought to investigate the role of personal risk factors on the dynamics and management of Cardiovascular infection. Personal risk factors which lead to infection and interfere with the disease management. They include High Blood Pressure, high Cholesterol, smoking, diabetes, obesity, lack of Physical Activity and unhealthy diet among others. Therefore, τ denotes the risk factors.

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

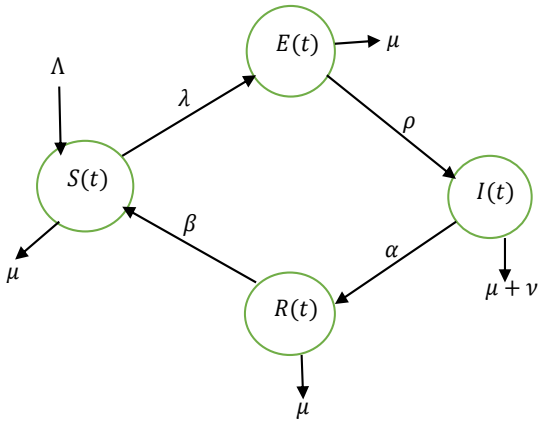


Figure 1: Model flow diagram

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, 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 or infection in the population [7]. To obtain the disease-free equilibrium point we set the normalized model system (2) equal to zero. Since there are no infections 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 infection 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 infection 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

The model in Equation (2) has disease free equilibrium (DFE) 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 [9], 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 [9], the disease-free equilibrium of model (2) is locally asymptotically stable. Given a small initial infective population, each infected individual in the entire period of infectivity will produce less than one infected individual on average 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 [9], 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;

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

and

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

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 inequality (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 effect of risk factors on the dynamics and prevention of cardiovascular infection [12]. To perform sensitivity analysis, the normalised forward sensitivity index also known as elasticity was used [6]. The normalised forward sensitivity index of the reproduction number R_0 in Equation (3) with respect to risk factors parameter τ is given by;

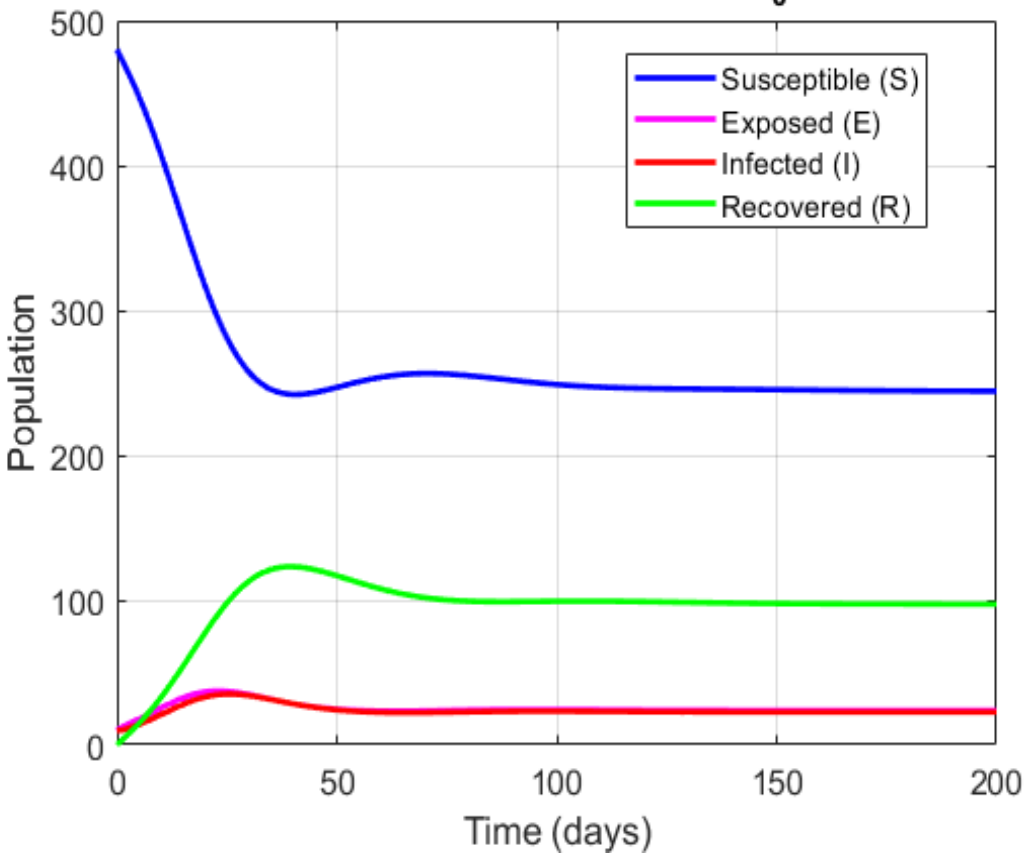
$$\Gamma_\tau^{R_0} = \frac{\partial R_0}{\tau} \times \frac{\tau}{R_0} \tag{13}$$

This implies that, the higher exposure to risk factors the higher the rate of infection

9 Numerical simulation

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

FIGURE 2. **Cardiovascular Disease Dynamics (R_0 = 1.59 > 1)**

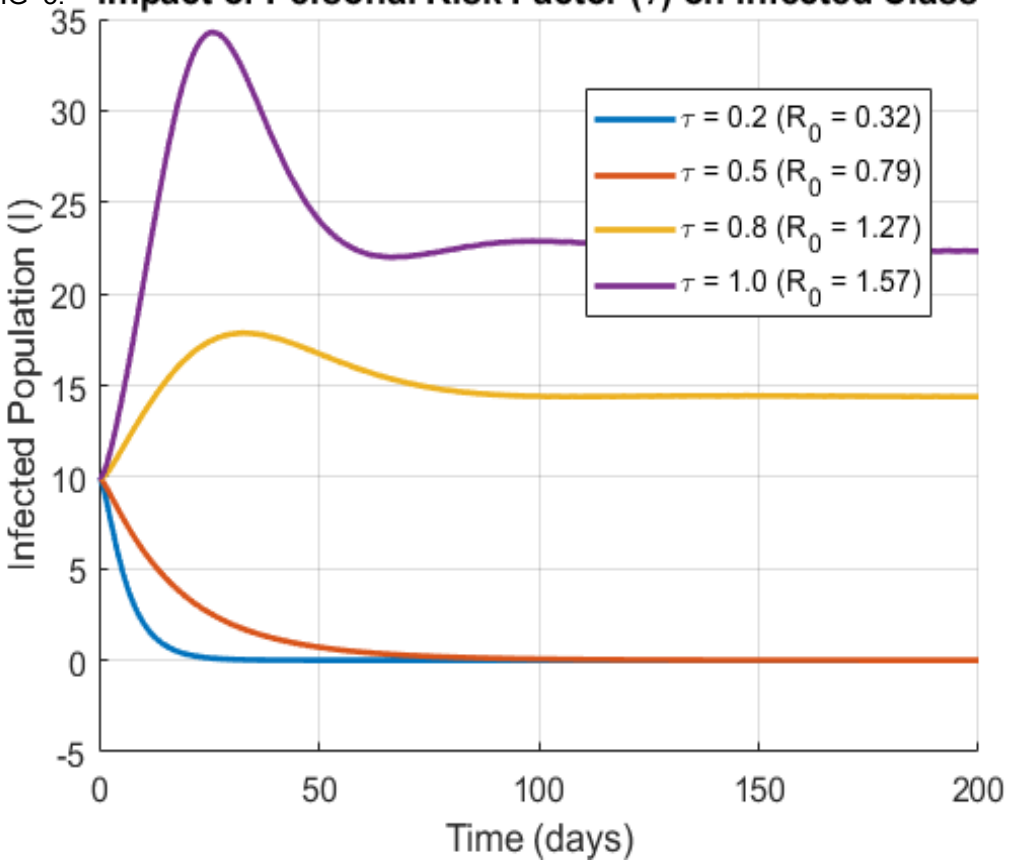


The graph depicts the progression of cardiovascular disease (CVD) through a population over time, with $R_0 = 1.59$ indicating an endemic scenario where the disease persists. Initially, the susceptible population (S) declines sharply as individuals become exposed (E) and then infected (I). The exposed population rises quickly before falling as people transition to the infected class, which peaks and then decreases due to recovery (R) 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) 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 in infections. 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.

FIG 3. **Impact of Personal Risk Factor (τ) on Infected Class**



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 (0.1 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 each infected individual transmits the infection to fewer than one person on average, 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 transmission potential.

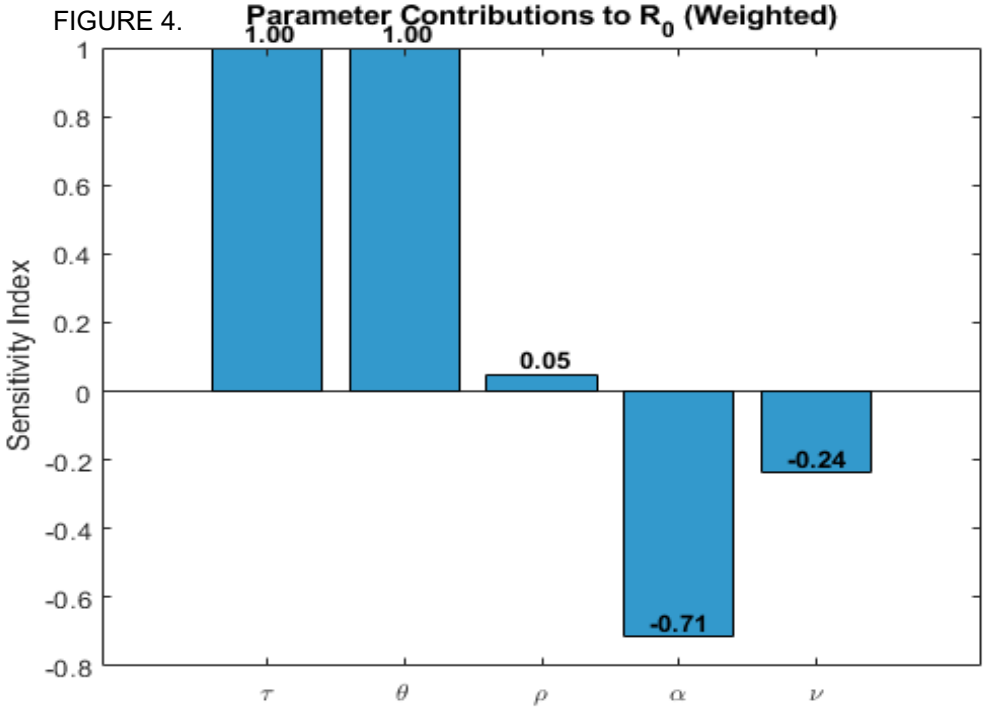
For higher τ values 0.8 and 1.0, where R_0 exceeds 1, the infected population exhibits an initial outbreak, peaking at around 15 individuals for $\tau = 0.8$ and 30 individuals for $\tau = 1.0$. After the peak, the curves decline and stabilize at endemic levels approximately 5 and 10 individuals, respectively. This stabiliza-

tion happens because the system reaches an equilibrium where new infections 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 reducing smoking, improving diet, or managing blood pressure 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 spread, 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.

FIGURE 4.



The sensitivity analysis graph clearly demonstrates that personal risk factors, τ exerts a substantially greater influence on cardiovascular disease transmission 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 smoking, diet, and physical inactivity represented by τ can be dramatically altered through public health interventions for instance, comprehensive anti-smoking programs, managing other health conditions, achieving and maintaining a healthy weight, eating a diet low in saturated fat and sodium, exercising at least 30 to 60 minutes per day on most days and managing stress might reduce τ by a given percentage leading to proportional decreases in disease spread. 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 infection. Despite the advocacy of behaviour change and treatment, the prevalence and mortality rate of Cardiovascular infection continues to be a problem. Thus, the importance of a combined prevention approaches against risk factors. Cardiovascular disease can be prevented by; avoiding all tobacco products, managing other health conditions, such as Type 2 diabetes, high cholesterol or high blood pressure, achieving and maintaining a healthy weight, eating a diet low in saturated fat and sodium, exercising at least 30 to 60 minutes per day on most days, and reducing and managing stress. Thus, Health education on the risk factors to this infection is paramount to raise public awareness and induce behavior change.

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