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Mathematical Modeling and Stability Analysis of the Disease-free Equilibrium of Heart Disease Transmission and Prevention Dynamics

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Abstract
Heart disease, commonly referred to as Cardiovascular disease (CVD), encompasses a diverse array of disorders that impact the heart and vascular system, resulting in significant health consequences, including myocardial infarctions, cerebrovascular accidents, and cardiac insufficiency. This condition may present without symptoms during its initial phases, thereby rendering early identification and preventive measures essential. This research aims to formulate a mathematical model to analyze the dynamics associated with heart disease, with a particular emphasis on the disease-free equilibrium (DFE) and its stability criteria. The model employs a compartmental structure to represent the population dynamics of heart disease. The eigenvalues exhibited negativity, which signifies that the DFE possesses local asymptotic stability. The DFE signifies the condition in which heart disease is eradicated from the population. It supports the notion that lowering the prevalence of heart disease requires early intervention and risk factor management.

Keywords: Heart Disease, Mathematical Modeling, Stability Analysis, Disease-Free Equilibrium (DFE), Epidemiology

Introduction

Heart disease, commonly referred to as Cardiovascular disease (CVD), encompasses a diverse array of disorders that impact the heart and vascular system, resulting in significant health consequences, including myocardial infarctions, cerebrovascular accidents, and cardiac insufficiency (World Health Organization [WHO], 2021). The primary case of this condition is attributed to the constriction or occlusion of blood vessels attributable to the accumulation of atheromatous plaques, a pathological state identified as atherosclerosis, which impairs perfusion to essential organs (Benjamin et al., 2019). Additional classifications of heart disease encompass arrhythmias (irregularities in cardiac rhythm), congenital heart anomalies (defects present at birth), cardiomyopathy (deteriorated myocardial function), and valvular heart disease (compromised cardiac valves) (National Heart, Lung, and Blood Institute [NHLBI], 2020).

Heart disease constitutes a significant public health challenge and persists as the foremost cause of mortality globally, being responsible for approximately 17.9 million fatalities each year (WHO, 2021). This condition may present without symptoms during its initial phases, thereby rendering early identification and preventive measures essential. Numerous modifiable risk determinants, including hypertension, tobacco use, excessive body weight, diabetes mellitus, hyperlipidemia, and detrimental lifestyle choices, considerably facilitate its advancement (Roth et al., 2017). Contemporary investigations have transitioned towards elucidating the transmission dynamics of cardiovascular disease through the application of mathematical models, which facilitate the examination of how risk factors disseminate within populations and influence disease prevalence. Although traditionally classified as a non-communicable disease (NCD), cardiovascular disease demonstrates attributes of social transmission, wherein detrimental behaviors and hereditary vulnerabilities may affect the development of the disease within communities (Qu et al., 2024). When evaluating and contrasting the efficacy of various heart disease prevention strategies, mathematical modelling is essential. Numerous studies have assessed how preventative efforts affect the outcomes of heart disease using a variety of modelling techniques
.
The impacts of better medical treatments, secondary prevention through statin uptake, and primary prevention through salt reduction were compared in a thorough Markov model created for the Tunisian population (Saidi et al., 2019). According to the model, cutting salt might potentially reduce fatalities from ischemic heart disease and stroke by 27%, greatly surpassing secondary prevention (3%), as well as medicinal measures (1%). This demonstrates how effective primary prevention strategies are at lowering cardiovascular mortality. According to

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(Agbo et al., 2024), The model combines agent-based modelling to simulate individual-level changes in lifestyle, and environmental exposures with interconnected ordinary differential equations to clarify population-level dynamics in disease transmission. The model makes the assumptions that risk factors are linearly additive, that populations are homogeneous, and that parameters remain constant across time.

Mathematical modeling has been extensively employed to investigate the transmission and management of infectious diseases; however, its utilization in the context of non-communicable diseases such as cardiovascular disease remains relatively nascent (Brauer et al., 2019). Mathematical models furnish a quantitative framework for examining disease progression, evaluating the efficacy of preventive measures, and forecasting prospective trends (Diekmann et al., 2020).

This research formulates a mathematical model to analyze the dynamics associated with heart disease, with a particular emphasis on the disease-free equilibrium (DFE) and its stability criteria. The DFE signifies a condition in which heart disease is eradicated from the population, thereby assisting policymakers in comprehending the thresholds necessary for effective disease management. Through the application of differential equations, analysis of the Jacobian matrix, and eigenvalue methodologies, this investigation delves into the stability of the DFE and its ramifications for public health interventions.

Method Formulation

The model employs a compartmental structure to represent the population dynamics in relation to heart disease. The population is stratified into distinct compartments, each capturing a specific aspect of the individual's health status.

- Susceptible (S): Individuals who do not have heart diseases.
- Exposed due to lifestyle (E_1): This compartment reflects people who have been exposed to varying degrees of heart disease. Based on lifestyle and dietary patterns, exposure levels are calculated, taking into account things like smoking, salt intake, processed food consumption, and long-term dietary habits.
  - Genetically Exposed (E_2): individuals who are genetically inherited and hence exposed to varying degrees of heart disease.
  - Infected (I): This compartment accounts for individuals with elevated heart disease over time.
  - Treatment(T): This compartment represents individuals undergoing different types of heart disease treatment, considering factors medication and therapies.
  - Recovered (R): Individuals who have recovered the heart disease impact. This compartment considers lifestyle modifications, medical interventions, or other factors influencing recovery.

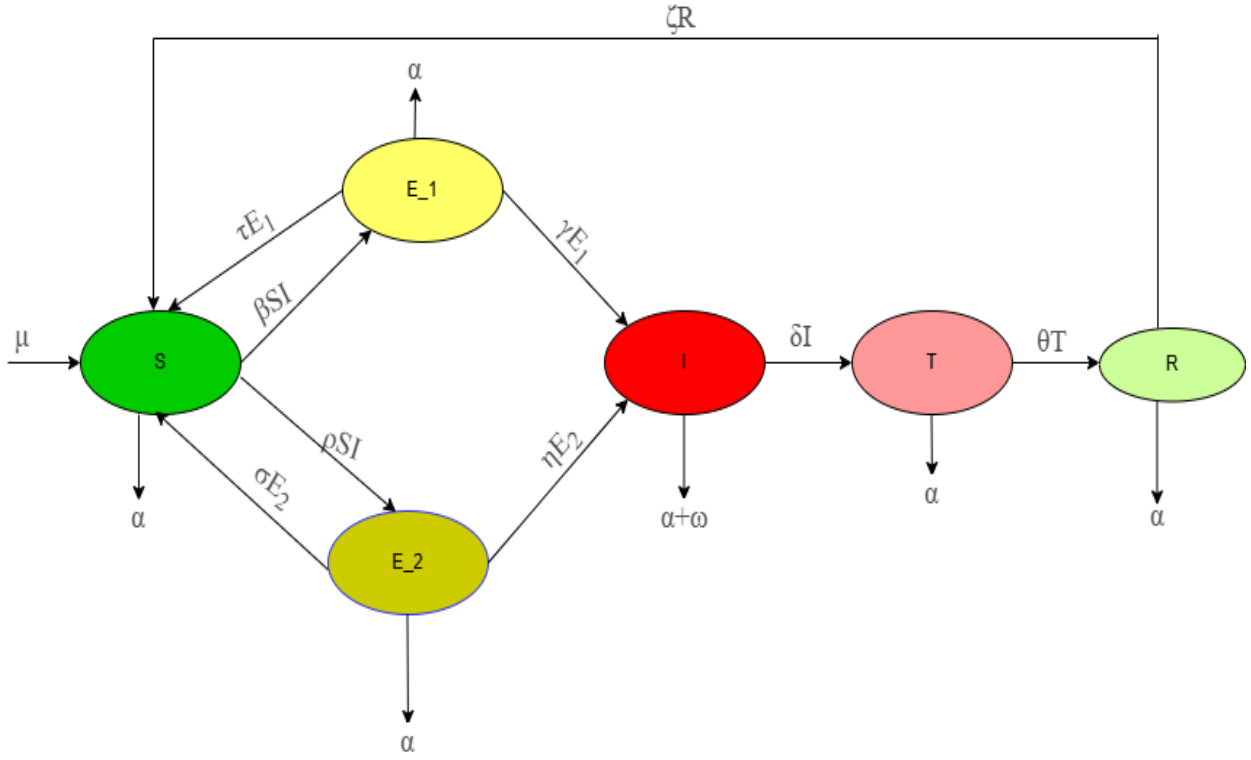

Fig1: Model Design

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Model Equations

The transitions between these compartments are dictated by a set of ordinary differential equations, encapsulating the rates of change within each compartment over time. Model formulation factor in the natural evolution of heart health, adaptation or recovery, and mortality, acting as the propelling force influencing these transitions. The model equations are as follows:

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

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

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

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

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

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

Where

S/NO	Variables And Parameters	Description
1	μ	Natural birth rate.
2	α	Natural death rate.
3	β	Transmission rate of heart diseases due to lifestyles such as unhealthy dietary habit.
4	ρ	Transmission rate of heart diseases due to inheritance. Infected rate due to unhealthy dietary habit.
5	γ	Rate of change in lifestyle.
6	τ	Rate of prevention on genetic disposition.
7	σ	Infected rate due to genetic.
8	η	Mortality rate due to heart diseases.
9	ω	Treatment rate due to medication and therapies.
10	δ	Recovery due to healthy lifestyle and medication.
11	θ	Loss of immunity.
12	ζ	

as

$$N(t) = S(t) + E_1(t) + E_2(t) + I(t) + T(t) + R(t) \quad (7)$$

Basic Properties of the Model

Invariant and Feasibility Analysis

Since the model formulation deals with the living population, it is assume that the variables and the parameters used are positive such that

$$t \to \infty \; N(t) \leq \frac{\mu}{\alpha} \quad (8)$$

Then

$$\dot{N}(t) = S'(t) + E'_1(t) + E'_2(t) + I'(t) + T'(t) + R'(t) \quad (9)$$

From Equation (3.9). The rate of change in total population at initial time $t = 0$ is,

$$\dot{N} = \mu - \alpha(S + E_1 + E_2 + I + T + R) \quad (10)$$

Substituting equation (1-6) into equation (10) gives

$$\dot{N} = \mu - \alpha(N) \quad (11)$$

Hence the positive invariant region for model (1)-(6) is

$$\Psi = (S + E_1 + E_2 + I + T + R) \in \mathbb{R}^6 \leq \frac{\mu}{\alpha} \quad (12)$$

Therfore, if $N_0 > \frac{\mu}{\alpha}$ then either the solution of equation (1-6) enter Ψ of $N(t) \to \frac{\mu}{\alpha}$ asymptotically. Hence the region Ψ attract solution of model (1-6) in $\mathbb{R}^6$.

Model Equilibrium Point

The equilibrium state is carried out to determine the disease-free equilibrium point the model. At the equilibrium Point $\frac{dS}{dt} = 0, \frac{dE_1}{dt} = 0, \frac{dE_2}{dt} = 0, \frac{dI}{dt} = 0, \frac{dT}{dt} = 0, \frac{dR}{dt} = 0$ thus obtained

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

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

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$\rho SI - (\eta + \sigma + \alpha)E_2 = 0$ (15)
$\gamma E_1 + \eta E_2 - (\alpha + \omega + \delta)I = 0$ (16)
$\delta I - (\alpha + \theta)T = 0$ (17)
$\theta T - (\alpha + \zeta)R = 0$ (18)
Then the equilibrium point of $S, E_1, E_2, I, T \; and \; R$ is found.

Disease Free equilibrium

Equilibrium point for disease-free is a point where there is no disease which implies
E_1=$E_2 = I \; = T = R = 0$ (19)
From (18)
$\mathrm{R} = \frac{\theta \mathrm{T}}{\alpha+\zeta}$ (20)
From (17)
$\mathrm{T} = \frac{\delta \mathrm{I}}{\alpha+\theta}$ (21)
From (16)
$\mathrm{I} = \frac{\gamma \mathrm{E}_1 + \eta \mathrm{E}_2}{\alpha+\omega+\delta}$ (22)
Let $\mathrm{E}_2 = 0$ and substitute 0 for E_2 in (22) which gives
$\mathrm{I} = \frac{\gamma \mathrm{E}_1}{\alpha+\omega+\delta}$ (23)
Substitute (23) into (14) yeld
$\beta \mathrm{S} \frac{\gamma \mathrm{E}_1}{(\alpha+\omega+\delta)} - (\gamma + \tau + \alpha)\mathrm{E}_1 = 0$ (24)
$E_1[\frac{\beta S\gamma - (\gamma+\tau+\alpha)(\alpha+\omega+\delta)}{(\alpha+\omega+\delta)}] = 0$ (25)
From (25) either $E_1 = 0$ or $\mathrm{S} = \frac{(\gamma+\tau+\alpha)}{\beta\gamma}$
For $E_1 = 0$
Substitute $E_1 = 0$ into (23) gives
$\mathrm{I} = 0$ (26)
Substitute (26) into (21) we have
$\mathrm{T} = 0$ (27)
Substitute (27) into (20) gives
$\mathrm{R} = 0$ (28)
Substitute (26) into (15) gives
$E_2 = 0$ (29)
Then substitute $E_1 = 0, \; E_2 = 0, \; \mathrm{I} = 0$, and $\mathrm{R} = 0$ into (13) which gives
$\mu - \alpha \mathrm{S} = 0$
$\mathrm{S} = \frac{\mu}{\alpha}$ (30)
Then, the equilibrium point of disease free for the heart disease model are
$K_0 = (S, E_1, E_2, I, T, R) = (\frac{\mu}{\alpha}, 0,0,0,0,0)$ (31)
The Jacobian matrix for (1-6) is obtained as

$$J(K_0) = \begin{bmatrix} -(\beta+\rho)I-\alpha & \tau & \sigma & (\beta+\rho)S & 0 & \zeta \\ I\beta & -(\gamma+\tau+\alpha) & 0 & \beta S & 0 & 0 \\ I\rho & 0 & -(\eta+\sigma+\alpha) & \rho S & 0 & 0 \\ 0 & \gamma & \eta & -(\alpha+\delta+\omega) & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\alpha+\theta) & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\alpha+\zeta) \end{bmatrix} \quad (32)$$

Local Stability Analysis

In the previous section, the number of the disease free equilibrium in the model equation (3.1- 3.6) is determined. In this section, To ascertain the system's local stability, we applied the Jacobian stability technique. Examine the Jacobian matrix (32) by substituting the value $S = \frac{\mu}{\alpha}$ and $I = 0$ gives

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$$J(K_0)=\begin{bmatrix} -\alpha & \tau & \sigma & (\beta+\rho)\frac{\mu}{\alpha} & 0 & \zeta \\ 0 & -(\gamma+\tau+\alpha) & 0 & \beta\frac{\mu}{\alpha} & 0 & 0 \\ 0 & 0 & -(\eta+\sigma+\alpha) & \rho\frac{\mu}{\alpha} & 0 & 0 \\ 0 & \gamma & \eta & -(\alpha+\delta+\omega) & 0 & 0 \\ 0 & 0 & 0 & \delta & -(\alpha+\theta) & 0 \\ 0 & 0 & 0 & 0 & \theta & -(\alpha+\zeta) \end{bmatrix} \quad (33)$$

For simplicity, let $n_1=(\beta+\rho)$, $n_2=(\alpha+\gamma+\tau)$, $n_3=(\alpha+\eta+\sigma)$, $n_4=(\alpha+\delta+\omega)$,$n_5=(\alpha+\theta)$, $n_6=(\alpha+\zeta)$

Then (33) become

$$J(K_0)=\begin{bmatrix} -\alpha & \tau & \sigma & \frac{\mu n_1}{\alpha} & 0 & \zeta \\ 0 & -n_2 & 0 & \beta\frac{\mu}{\alpha} & 0 & 0 \\ 0 & 0 & -n_3 & \rho\frac{\mu}{\alpha} & 0 & 0 \\ 0 & \gamma & \eta & -n_4 & 0 & 0 \\ 0 & 0 & 0 & \delta & -n_5 & 0 \\ 0 & 0 & 0 & 0 & \theta & -n_6 \end{bmatrix} \quad (34)$$

Using echelon row reduced method gives

$$J(K_{0_{ref}})=\begin{bmatrix} -\alpha & \tau & \sigma & \frac{\mu n_1}{\alpha} & 0 & \zeta \\ 0 & -n_2 & 0 & \beta\frac{\mu}{\alpha} & 0 & 0 \\ 0 & 0 & -n_3 & \rho\frac{\mu}{\alpha} & 0 & 0 \\ 0 & \gamma & \eta & -n_4-\frac{\gamma\beta\mu}{\alpha n_2} & 0 & 0 \\ 0 & 0 & 0 & \delta & -n_5 & 0 \\ 0 & 0 & 0 & 0 & \theta & -n_6 \end{bmatrix} \quad (35)$$

Using the method in (Abah et al., 2015) to find the eigenvalues of the row reduce matrix $J(K_{0ref})$ the characteristics equation $|J(K_{0ref})-\lambda|=0$ is expanded and simplified as follow

$$\left|J(K_{0_{ref}})-\lambda\right|=\begin{vmatrix} -\alpha-\lambda & \tau & \sigma & \frac{\mu n_1}{\alpha} & 0 & \zeta \\ 0 & -n_2-\lambda & 0 & \beta\frac{\mu}{\alpha} & 0 & 0 \\ 0 & 0 & -n_3-\lambda & \rho\frac{\mu}{\alpha} & 0 & 0 \\ 0 & \gamma & \eta & -n_4-\frac{\gamma\beta\mu}{\alpha n_2}-\lambda & 0 & 0 \\ 0 & 0 & 0 & \delta & -n_5-\lambda & 0 \\ 0 & 0 & 0 & 0 & \theta & -n_6-\lambda \end{vmatrix}=0 \quad (36)$$

The determinant of an upper triangular matrix is the product of its diagonal elements.
Therefore:

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$$\left|J(K_{0ref}) - \lambda\right| = (-\alpha - \lambda)(-n_2 - \lambda)(-n_3 - \lambda)(-n_4 - \frac{\gamma\beta\mu}{\alpha n_2} - \lambda)(-n_5 - \lambda)(-n_6 - \lambda) \quad (37)$$

From (37) the characteristics polynomial is obtained as

$$(-\alpha - \lambda)(-n_2 - \lambda)(-n_3 - \lambda)(-n_4 - \frac{\gamma\beta\mu}{\alpha n_2} - \lambda)(-n_5 - \lambda)(-n_6 - \lambda) = 0 \quad (38)$$

Each factor corresponds to an eigenvalue:

$$-\alpha - \lambda = 0 \Longrightarrow \lambda = -\alpha \quad (39)$$

$$-n_2 - \lambda = 0 \Longrightarrow \lambda = -n_2 \quad (40)$$

$$-n_3 - \lambda = 0 \Longrightarrow \lambda = -n_3 < 0 \quad (41)$$

$$-n_4 - \frac{\gamma\beta\mu}{\alpha n_2} - \lambda = 0 \Longrightarrow \lambda = -n_4 - \frac{\gamma\beta\mu}{\alpha n_2} < 0 \quad (42)$$

$$-n_5 - \lambda = 0 \Longrightarrow \lambda = -n_5 < 0 \quad (43)$$

$$-n_6 - \lambda = 0 \Longrightarrow \lambda = -n_6 < 0 \quad (44)$$

Hence the eigenvalues are

Results

The eigenvalues $\lambda_1 = -\alpha$, $\lambda_2 = -n_2$, $\lambda_3 = -n_3$, $\lambda_4 = -n_4 - \frac{\gamma\beta\mu}{\alpha n_2}$, $\lambda_5 = -n_5$, $\lambda_6 = -n_6$ exhibit negativity, which signifies that the disease-free equilibrium (DFE) possesses local asymptotic stability. This implies that should the system initiate in proximity to the DFE, it will asymptotically approach the DFE over time. In pragmatic terms, this suggests that the disease will extinguish if the initial count of infected individuals remains minimal and the system is situated near the DFE.

Discussion

The eigenvalues are dependent on the parameters of the model, and their respective values yield valuable insights into the manner in which various factors affect the stability of the system:

$\lambda_1 = -\alpha$

This eigenvalue is influenced by the intrinsic mortality rate of susceptible individuals. An elevated death rate accelerates the stability of the system.

$\lambda_2 = -n_2$

This eigenvalue pertains to the rate at which exposed individuals(E_1) transition to the subsequent stage by becoming infected. An increased rate of progression facilitates the stabilization of the system.

$\lambda_3 = -n_3$

This eigenvalue is connected to the velocity at which exposed individuals(E_2) advance to the next phase. Analogous to the preceding rate, an enhanced value contributes to a more rapid stabilization.

$\lambda_4 = -n_4 - \frac{\gamma\beta\mu}{\alpha n_2}$

This eigenvalue is contingent upon the rate of lifestyle change of infected individuals(I) and the transmission coefficient. An elevated recovery rate coupled with a diminished transmission coefficient promotes system stability.

$\lambda_5 = -n_5$

This eigenvalue is related to the rate at which individuals receiving treatment(T) recover or are eliminated. An increased recovery rate aids in stabilizing the system.

$\lambda_6 = -n_6$

This eigenvalue is associated with the rate at which recovered individuals(R) experience a loss of immunity or succumb. An accelerated loss of immunity contributes positively to system stability.

Conclusion

The study offers a mathematical framework for comprehending the prevention and transmission of heart disease. It supports the notion that lowering the prevalence of heart disease requires early intervention and risk factor management by validating the diseas-free equilibrium(DFE) local stability. This model could be expanded in future studies by adding stochastic components, demographic changes, or outside impacts like socioeconomic variables.

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