

MODEL FORMULATION AND COMPUTATION FOR FACTORS INFLUENCING MYOCARDIAL INFARCTION IN HUMANS

Malini Devi Ramanathan* and Vaishnavi Kalirajan

Received: December 12, 2023; Revised: March 08, 2024; Accepted: March 27, 2024

Abstract

Myocardial infarction commonly known as heart attack, is a medical emergency caused by other diseases in humans. In this paper, a mathematical model based on the factors influencing myocardial infarction is generated. The solution of the model is obtained with the help of the Homotopy Perturbation Method. The analytical results are verified with the exact solution by using MATLAB. The behavior of each parameter is discussed. The main aim of this paper is to develop a model for myocardial infarction and discuss the parameters that help in reducing the rate of myocardial infarction.

Keywords: Analytical expression, blockage rate, exact solution, homotopy perturbation mathematical model, method, myocardial infarction

Introduction

A medical emergency that develops as a result of any other disease is myocardial infarction, heart attack, or coronary artery disease. A heart attack happens when a blood vessel becomes clogged, which interferes with the heart's ability to pump blood. Fatty, cholesterol-filled deposits that create blood clots are the cause of these blood artery blockages. There are two different kinds of blockages: acute total obstruction and partial blockage. Heart attacks can also be caused by coronary artery spasms, certain viral infections that weaken the heart muscle, and spontaneous coronary artery dissection, in addition to blockages. The heart

muscle's tissue dies when there is insufficient blood flow. Depending on the severity of the medical emergency, the patients may require aortic valve surgery, arrhythmia surgery, congenital heart surgery, or coronary artery bypass graft (CABG) surgery. By this operation, the blood flow around constricted arteries is redirected to enhance the heart's pumping function. After surgery, the majority of patients recover completely in 12 weeks. Severe chest pain, shoulder pain, cold sweats, exhaustion, heartburn, abrupt dizziness, nausea, and shortness of breath are some of the signs and symptoms of a myocardial infarction.

PG and Research Department of Mathematics, The Standard Fireworks Rajaratnam College for Women, Thiruthangal Road, Sivakasi-626123, Tamilnadu, India. E-mail: malinidevi-mat@sfrcollege.edu.in; 18vaishnavik-pgmat@sfrcollege.edu.in

** Corresponding author*

DOI: <https://doi.org/10.55766/sujst-2024-02-e03011>

Suranaree J. Sci. Technol. 31(2):030179(1-9)

Age, cigarette use, high blood pressure, high cholesterol, obesity, diabetes, metabolic syndrome, family history of heart attacks, insufficient exercise, stress, use of illegal drugs, and so forth are risk factors for myocardial infarction. Maintaining a balanced diet, treating other medical illnesses, and taking prescription drugs as prescribed are among the strategies to prevent heart attacks (Avis *et al.*, 1989; Chow *et al.*, 2011; Ranthe *et al.*, 2015).

The mechanism of left ventricular remodeling following myocardial infarction was previously outlined and examined by Wang *et al.* (2012). A system of partial differential equations makes up the mathematical model of immunomodulatory therapy devised by Moise and Friedman (2022). This model lessens the damage brought on by inflammation in the area of the left ventricle wall surrounding the infarct area (Moise and Friedman, 2022). A mathematical model of blunt injury to the vascular wall was created by Ismailov *et al.* (2005). Sanjay Basu and colleagues created a microsimulation model to examine the differences in the effects of pharmaceutical treatments and alternative tobacco control strategies on myocardial infarction (Basu *et al.*, 2013). A three-dimensional cardiac magnetic resonance imaging model of a pig myocardial infarction has been constructed by Jason Ng and colleagues (Ng *et al.*, 2012).

In our paper, we underscore the pivotal role of blood vessel blockage rate within our model, recognizing various researchers' insights in elucidating blood flow dynamics within the human cardiovascular system. To understand the phenomena of blockage formation and blood hydrodynamics of the human cardiovascular system, Tentner *et al.* (2010) developed a computational model. The blockage rate of blood vessels is studied using angiography images and computational fluid dynamics by Kumar *et al.* (2020). Promit *et al.* (2022) proposed a deep learning technique to detect coronary artery block at an earlier stage. Dijkstra's algorithm was implemented by Maqbool and Kumar (2018) to study the vein blockage rate in human hearts. A numerical simulation was carried out by Saha *et al.* (2021) to detect blocked blood vessels earlier. Notably, our approach distinguishes itself by prioritizing analytical and mathematical methodologies over experimental techniques employed in prior studies, offering a novel perspective on this critical aspect of cardiovascular health.

In this study, we have built a model based on variables such as fat deposits in the body causing myocardial infarction, genetic transmission, and other illnesses. Our framed model consists of a set of ordinary differential equations. This model gives an understanding of the underlying mathematical

properties. A mathematical model has been developed considering the risk variables leading to myocardial infarction. Every parameter is defined for our model, along with a description and schematic diagram. The manually-obtained solution for our model using the Homotopy Perturbation Method is presented. The graphical depiction enhances understanding of the behavior of each parameter and demonstrates the connection between the precise MATLAB solution and the analytical solution. Furthermore, our model can be expanded to encompass additional factors recognized to impact myocardial infarction risk, including genetic predisposition, environmental exposures, and psychosocial factors. This augmentation with a broader array of variables will enable a more nuanced comprehension of the intricate interactions among multiple risk factors and their collective influence on disease outcomes. Additionally, fostering interdisciplinary collaboration among researchers from diverse fields such as epidemiology, cardiology, biostatistics, and health economics will facilitate our understanding of myocardial infarction risk factors. This collaborative effort will culminate in developing comprehensive models that integrate various perspectives and expertise, thereby enhancing the robustness and applicability of our research findings.

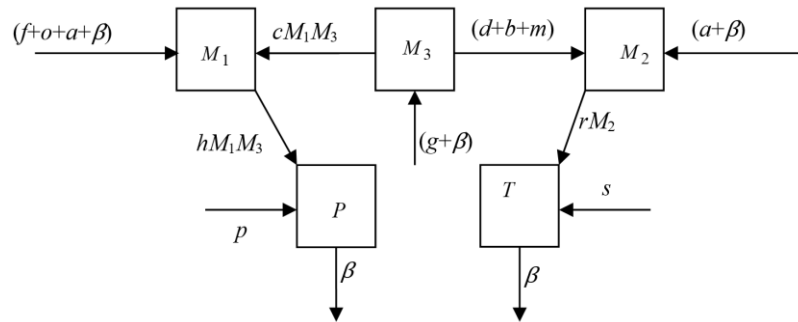
Materials and Methods

A mathematical model that causes myocardial infarction is developed. The factors that cause myocardial infarction are divided into three compartments, namely, the rate of myocardial infarction due to fat deposits in the body $M_1(t)$, other diseases $M_2(t)$, and heredity transmission $M_3(t)$. Further, the prevention rate of myocardial infarction $P(t)$ and treatment rate of the $T(t)$ class are also developed. The fat deposits in the body are due to the consumption rate of a fatty diet f , overweight rate of the human body o , cholesterol level in the body c and age of the person a . The other diseases are represented by the parameters rate of diabetes d , blood pressure b , and heart muscle diseases m . All these factors lead to blood vessel blockage, represented as β . The gene-wise transmission rate is denoted as g . The rest of the parameters and their description can be found in Table 1 and Figure 1.

$$\begin{aligned}\frac{dM_1}{dt} &= (f + o + a + \beta)M_1 + (c - h)M_1M_3 \\ \frac{dM_2}{dt} &= (a + \beta)M_2 + (d + b + m)M_2M_3 - rM_2T \\ \frac{dM_3}{dt} &= (g + \beta)M_3 - hM_1M_3 \\ \frac{dP}{dt} &= hM_1M_3 + p - \beta P\end{aligned}$$

Table 1. List of parameters

Symbol	Meaning	Units
M_1	Myocardial infarction rate due to fat deposits in body	%
M_2	Myocardial infarction rate due to other diseases	%
M_3	Myocardial infarction rate due to heredity transmission	%
P	Prevention rate of Myocardial infarction	%
T	Treatment rate of Myocardial infarction	%
f	Consumption rate of fatty diet	%
o	Overweight rate of human body	kg/m ²
a	Age of the person	Z+
β	Blockage rate in blood vessels	%
c	Cholesterol level in body	mg/dL
h	Healthy lifestyle rate	%
d	Rate of diabetes	mg/dL
b	Rate of blood pressure	mm/Hg
m	Rate of heart muscle diseases	%
r	Treatment rate of other diseases	%
g	Gene wise transmission rate	%
p	Prevention rate by consuming prescribed medicines	%
s	Success rate of surgery	%

**Figure 1. Schematic diagram representing the mathematical model of factors influencing myocardial infarction**

$$\frac{dT}{dt} = rM_2T + s - \beta T \quad (1)$$

The Homotopy Perturbation Method (HPM) stands out as a reliable mathematical approach extensively utilized in tackling non-linear differential equations, particularly in the realm of disease modeling within biological sciences.

By employing HPM, scientists can derive a series of solutions that rapidly converge towards the exact solution. This characteristic facilitates a deeper understanding of the dynamics inherent in disease models, moving beyond dependence solely on numerical simulations (Celestine *et al.*, 2021; Ganeshan, 2022; Vijayalakshmi and Senthamarai, 2022). Consequently, researchers gain invaluable insights into the intricate behavior of these models, enriching our comprehension of disease spread and containment strategies. The analytical expression for the rate of myocardial infarction due to fat deposits in the body M_1 , other diseases M_2 , heredity transmission M_3 and prevention and treatment rate of myocardial infarction (P , T) is derived using the Homotopy Perturbation Method (He, 2003; Rajabi

et al., 2007; Ayoade *et al.*, 2020). Thus, the step-by-step procedure for the application of the Homotopy Perturbation Method (HPM) for the system of equation (1) is explained in the following steps.

Now, we construct the homotopy as follows, $(1 - p) \times (L(v) + L(u_0)) + p \times (L(u) + N(u) - f(r)) = 0$ where L , N , f represents the linear term, non-linear term and unknown function of our system. Then our system of equations becomes,

$$\begin{aligned} (1-p) \left[\frac{dM_1}{dt} - (f+o+a+\beta)M_1 \right] &+ p \left[\frac{dM_1}{dt} - (f+o+a+\beta)M_1 - (c-h)M_1M_3 \right] = 0 \\ (1-p) \left[\frac{dM_2}{dt} - (a+\beta)M_2 \right] &+ p \left[\frac{dM_2}{dt} - (a+\beta)M_2 - (d+b+m)M_2M_3 - rM_2T \right] = 0 \\ (1-p) \left[\frac{dM_3}{dt} - (g+\beta)M_3 \right] &+ p \left[\frac{dM_3}{dt} - (g+\beta)M_3 + hM_1M_3 \right] = 0 \end{aligned}$$

$$(1-p) \left[\frac{dP}{dt} - p + \beta P \right] + p \left[\frac{dP}{dt} - hM_1M_3 - p + \beta P \right] = 0$$

$$(1-p) \left[\frac{dT}{dt} - s + \beta T \right] + p \left[\frac{dT}{dt} - rM_2T - s + \beta T \right] = 0 \quad (2)$$

Then we replace each compartment of our system in the form of a series,

$$\begin{aligned} M_1 &= p^0 M_{10} + p^1 M_{11} + \dots \\ M_2 &= p^0 M_{20} + p^1 M_{21} + \dots \\ M_3 &= p^0 M_{30} + p^1 M_{31} + \dots \\ P &= p^0 P_0 + p^1 P_1 + \dots \\ T &= p^0 T_0 + p^1 T_1 + \dots \end{aligned} \quad (3)$$

By using (3) in (2) and by comparing the coefficients of p^0 and p^1 , we obtain,

$$\begin{aligned} p^0: \frac{dM_{10}}{dt} - (f + o + a + \beta)M_{10} &= 0 \\ p^0: \frac{dM_{20}}{dt} - (a + \beta)M_{20} &= 0 \\ p^0: \frac{dM_{30}}{dt} - (g + \beta)M_{30} &= 0 \\ p^0: \frac{dP_0}{dt} - p + \beta P_0 &= 0 \\ p^0: \frac{dT_0}{dt} - s + \beta T_0 &= 0 \end{aligned} \quad (4)$$

$$\begin{aligned} p^1: \frac{dM_{11}}{dt} - (f + o + a + \beta)M_{11} - (c - h)M_{10}M_{30} &= 0 \\ p^1: \frac{dM_{21}}{dt} - (a + \beta)M_{21} - (d + b + m)M_{20}M_{30} + rM_{20}T_0 &= 0 \\ p^1: \frac{dM_{31}}{dt} - (g + \beta)M_{31} + hM_{10}M_{30} &= 0 \\ p^1: \frac{dP_1}{dt} + \beta P_1 - hM_{10}M_{30} &= 0 \\ p^1: \frac{dT_1}{dt} + \beta T_1 - rM_{20}T_0 &= 0 \end{aligned} \quad (5)$$

Solving (4) and (5) by using variable separable method or by bringing them into linear form $\frac{dy}{dx} + Py = Q$ and adding them, we get the required solution as follows,

$$\begin{aligned} M_1(t) &= M_{1i} e^{(f+o+a+\beta)t} + \frac{-(c-h)M_{10}M_{30}}{f+o+a+\beta} \\ &\quad + \left(M_{1i} + \frac{(c-h)M_{10}M_{30}}{f+o+a+\beta} \right) e^{(f+o+a+\beta)t} \\ M_2(t) &= M_{2i} e^{(a+\beta)t} + \frac{-((d+b+m)M_{20}M_{30} - rM_{20}T_0)}{a+\beta} \\ &\quad + \left(M_{2i} + \frac{((d+b+m)M_{20}M_{30} - rM_{20}T_0)}{a+\beta} \right) e^{(a+\beta)t} \\ M_3(t) &= M_{3i} e^{(g+\beta)t} + \frac{hM_{10}M_{30}}{g+\beta} \\ &\quad + \left(M_{3i} + \frac{-(hM_{10}M_{30})}{g+\beta} \right) e^{(g+\beta)t} \\ P(t) &= \frac{p}{\beta} + \left(P_i - \frac{p}{\beta} \right) e^{-\beta t} + \frac{hM_{10}M_{30}}{\beta} + \left(P_i - \frac{hM_{10}M_{30}}{\beta} \right) e^{-\beta t} \\ T(t) &= \frac{s}{\beta} + \left(T_i - \frac{s}{\beta} \right) e^{-\beta t} + \frac{rM_{20}T_0}{\beta} + \left(T_i - \frac{rM_{20}T_0}{\beta} \right) e^{-\beta t} \end{aligned} \quad (6)$$

where,

$$\begin{aligned} M_{10} &= M_{1i} e^{(f+o+a+\beta)t}, M_{20} = M_{2i} e^{(a+\beta)t}, M_{30} \\ &= M_{3i} e^{(g+\beta)t}, P_0 \\ &= \frac{p}{\beta} + \left(P_i - \frac{p}{\beta} \right) e^{-\beta t}, T_0 \\ &= \frac{s}{\beta} + \left(T_i - \frac{s}{\beta} \right) e^{-\beta t} \end{aligned}$$

Table 2. Table illustrating the accuracy between the analytical and numerical solution for different parameters at different points in time

Parameter	Value	$t = 0.0004$		$t = 0.0006$		$t = 0.0008$	
		HPM	Numerical	HPM	Numerical	HPM	Numerical
M_1	f	60	1.058	1.064	1.0833	1.0834	1.19
M_2	a	35	2.045	2.046	2.07	2.07	2.091
		$t = 2.5 \times 10^{-5}$		$t = 5 \times 10^{-5}$		$t = 0.0001$	
M_3	h	1	3.001	3.001	3.001	3.001	3.003
P	h	1	0.599	0.6	0.599	0.6	0.599
T	r	1	0.4999	0.5	0.4999	0.4999	0.4999

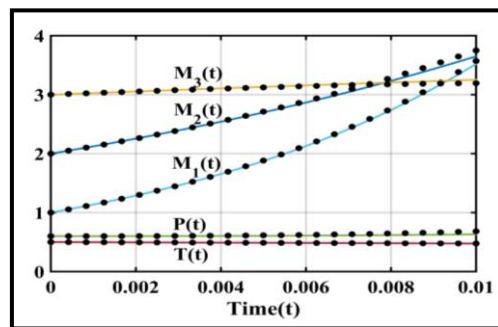


Figure 2. Factors Influencing Myocardial Infarction with respect to time

Results and Discussion

The solution (6) that is obtained through Homotopy Perturbation Method is verified for its accuracy with the exact solution. The behavior of each parameter is displayed in the form of a graph. The initial values for the system of equations are chosen to be $M_1 = 1$, $M_2 = 2$, $M_3 = 3$, $P_0 = 0.6$ and $T_0 = 0.5$. By setting the numerical values of the parameter as $f = 60$, $o = 25$, $a = 35$, $\beta = 5$, $c = 1.35$, $h = 1$, $g = 5$, $p = 0$, $s = 0$, $t = 0$, $d = 2.3$, $b = 2$, $m = 2$, we can see the behavior of each system M_1 , M_2 , M_3 , P , T in Figure 2. In Table 2, the accuracy between the manually derived analytical solution of our model in equation (2), using Homotopy Perturbation Method (HPM) and the numerical solution achieved using computational tool like MATLAB for different parameters at different point of time is displayed. In all the graphical representations, the solid line (—) represents the analytical solution derived using Homotopy Perturbation Method and the dotted line (...) represents the numerical solution.

As the consumption rate of fatty diet f (depicted in Figure 3(a)) rises, along with an increase in the overweight rate of the human body o

(Figure 3(b)), the age of the individual a (Figure 3(c)), and the cholesterol level in the body c (Figure 3(d)), there is a corresponding escalation in the rate of myocardial infarction attributed to fat deposits in the body $M_1(t)$.

In other words, higher intake of fatty foods, increased body weight, older age, and elevated cholesterol levels are all factors that contribute to an augmented risk of myocardial infarction stemming from fat accumulation within the body.

As depicted in Figures 4(a) and 4(b), there is an observable trend in the rate of myocardial infarction attributed to other diseases $M_2(t)$ over time. When considering the parameters involved, specifically the age of the individual a (illustrated in Figure 4(a)) and the rate of diabetes d (depicted in Figure 4(b)), it becomes evident that an increase in these factors correlates with a higher incidence of myocardial infarction stemming from other underlying health conditions. In simpler terms, as individuals grow older and the prevalence of diabetes rises, there is a corresponding escalation in the risk of experiencing myocardial infarction due to conditions other than fat deposits in the body.

In Figure 5, the rate of myocardial infarction attributed to hereditary factors $M_3(t)$ is depicted

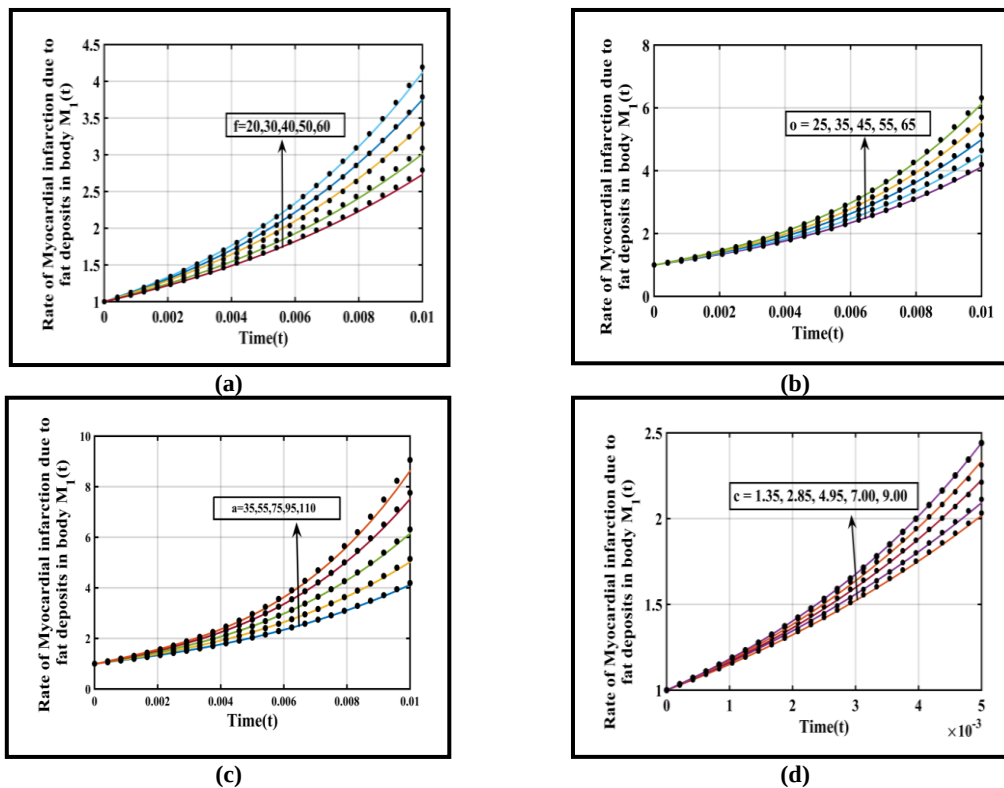


Figure 3. The rate of myocardial infarction due to fat deposits in body $M_1(t)$ with time. (a) consumption rate of fatty diet f , (b) overweight rate of human body o , (c) age of the person a , (d) cholesterol level in body c

over time. It is observed that as the adoption of a healthy lifestyle h increases, there is a gradual decrease in the rate of myocardial infarction caused by hereditary transmission. This suggests that lifestyle choices play a significant role in mitigating the risk associated with inherited predispositions to heart-related conditions. In essence, individuals

who adhere to healthier lifestyles tend to experience a lower incidence of myocardial infarction stemming from genetic factors passed down through family lines.

Figure 6 displays the trend in the prevention rate of myocardial infarction $P(t)$ over time. It is noticeable that when the adoption of a healthy

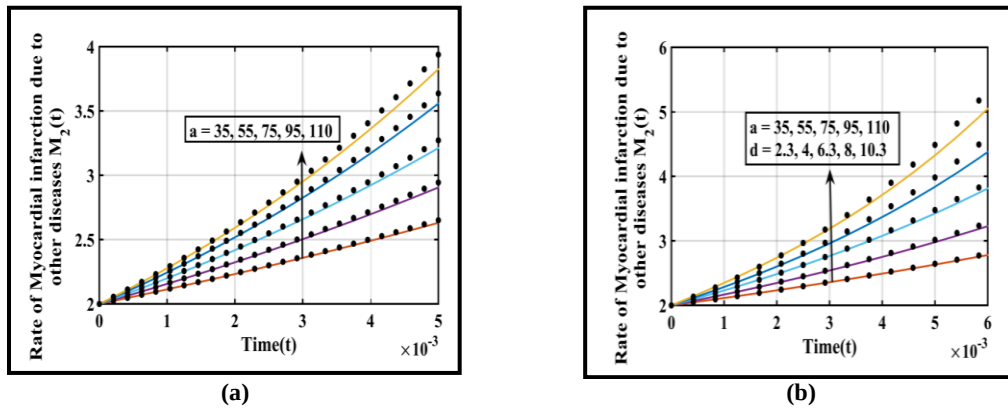


Figure 4. The rate of myocardial infarction due to other diseases $M_2(t)$ with time. (a) age of the person a , (b) rate of diabetes d

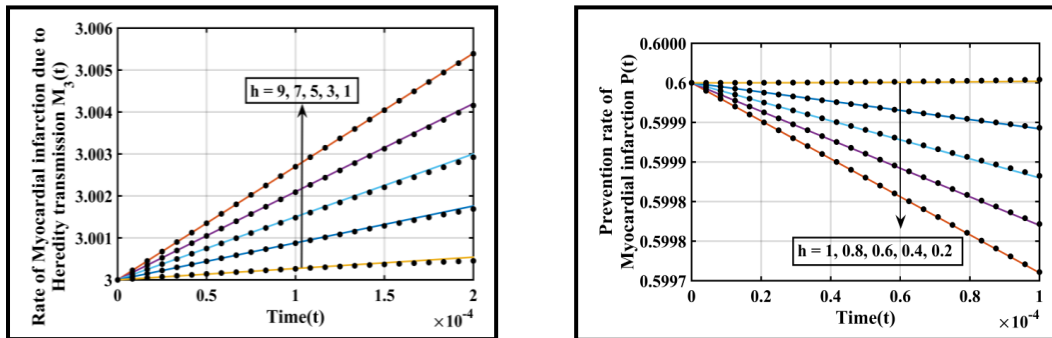


Figure 5. The rate of myocardial infarction due to heredity transmission $M_3(t)$ with time and the parameter healthy lifestyle rate h

Figure 6. The prevention rate of myocardial infarction $P(t)$ concerning time and the parameter healthy lifestyle rate h

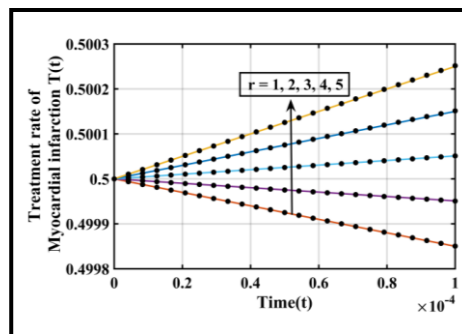


Figure 7. The treatment rate of myocardial infarction $T(t)$ with time and the parameter, the treatment rate of other diseases r

lifestyle h is more prevalent, there is a gradual increase in the prevention rate of myocardial infarction $P(t)$. This suggests a positive correlation between the prevalence of healthy lifestyle choices and the effectiveness of preventing myocardial infarction. In simpler terms, as more people embrace healthy habits, there is a corresponding rise in the effectiveness of measures aimed at preventing heart attacks.

In Figure 7, the treatment rate of myocardial infarction $T(t)$ is illustrated over time. It is evident that as the treatment rate for other diseases r increases, there is a corresponding rise in the treatment rate of myocardial infarction $T(t)$. This suggests interdependence between the treatment

approaches for various health conditions, indicating that advancements in treating other diseases may also positively impact the treatment of myocardial infarction. In essence, improvements in healthcare and treatment strategies across a range of illnesses contribute to better outcomes for myocardial infarction patients as well.

Through the analysis of the key parameter, blockage rate in blood vessels β , which is a primary factor contributing to myocardial infarction in humans, we examine Figures 8(a) to 8(e). It becomes evident that as the parameter range spans from $\beta \in [5, 100]$ there is a concurrent increase in the rates of myocardial infarction attributed to fat deposits in the body $M_1(t)$, other diseases $M_2(t)$,

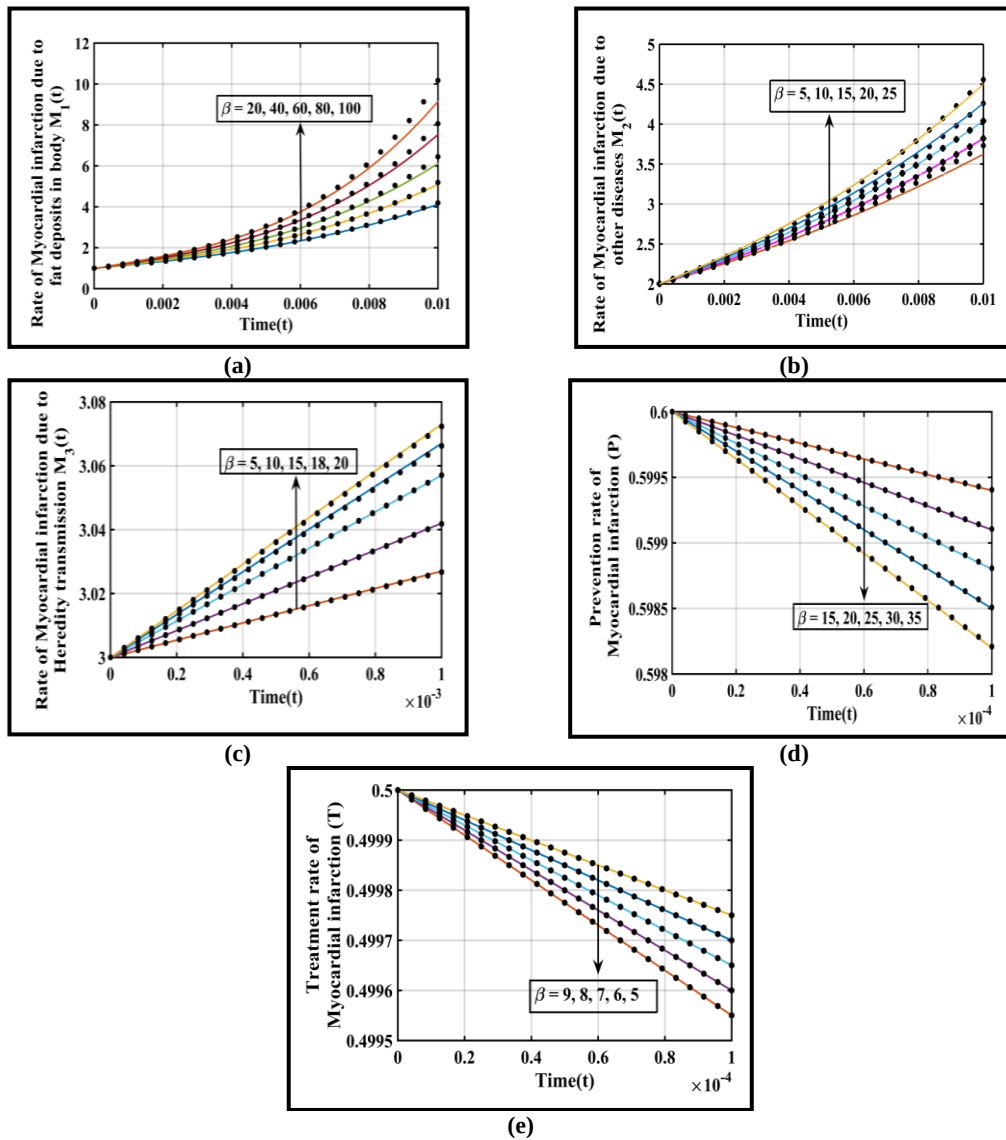


Figure 8. Analysis of blockage rate in blood vessels β parameter with time

and hereditary transmission $M_3(t)$. Simultaneously, the rates of prevention and treatment $P(t)$, $T(t)$ decreases. This indicates a negative correlation between the blockage rate and the effectiveness of preventative and treatment measures, while also highlighting its significant impact on the occurrence of myocardial infarction from various causes. Furthermore, the analysis suggests that when the blockage rate β falls below 5, myocardial infarction may be minimized through appropriate treatment strategies. In simpler terms, keeping the blockage rate within a lower range can potentially reduce the incidence of heart attacks, emphasizing the importance of managing this parameter effectively in cardiovascular health management.

Conclusions

Thus the mathematical model concerning all the factors responsible for myocardial infarction has been developed successfully. Homotopy Perturbation Method has been implemented to derive the analytical expression for the rate of myocardial infarction due to fat deposits in body $M_1(t)$, other diseases $M_2(t)$, heredity transmission $M_3(t)$, prevention rate of myocardial infarction $P(t)$ and treatment rate of myocardial infarction $T(t)$. The analytical expressions are verified for their accuracy with the numerical solution. Also, the key parameter, the blockage rate of blood vessels β is analyzed in order to minimize the rate of myocardial infarction.

Acknowledgement

The author(s) express deep gratitude to the institution for its unwavering support throughout the research endeavor. This study was made possible through funding received from the Seed Money Project of The Standard Fireworks Rajaratnam College for Women (Autonomous), Sivakasi. The author(s) extend heartfelt appreciation to the management and Principal of The Standard Fireworks Rajaratnam College for Women (Autonomous), Sivakasi, for their invaluable assistance and encouragement throughout the project.

References

Avis, N.E., Smith, K.W., and McKinlay, J.B. (1989). Accuracy of perceptions of heart attack risk: what influences perceptions and can they be changed?. *American Journal of Public Health*, 79(12):1608-1612. <https://doi.org/10.2105/AJPH.79.12.1608>

Ayoade, A.A., Peter, O.J., Abioye, A.I., Aminu, T.F., and Uwaheren, O.A. (2020). Application of homotopy perturbation method to an SIR mumps model. *Advances in Mathematics: Scientific Journal*, 9(3):329-1340. <https://doi.org/10.37418/amsj.9.3.57>

Basu, S., Glantz, S., Bitton, A., and Millett, C. (2013). The effect of tobacco control measures during a period of rising cardiovascular disease risk in India: a mathematical model of myocardial infarction and stroke. *PLoS Medicine*, 10(7):e1001480. <https://doi.org/10.1371/journal.pmed.1001480>

Celestine, A.B., Shior, M.M., Olorunnishola, O.A., Ezugorie, I.G., and Obeng-Denteh, W. (2021). Analysis of homotopy perturbation method (HPM) and its application for solving infectious disease models. *International Journal of Mathematics and Statistics Studies*, 9(4):27-38.

Chow, C.K., Islam, S., Bautista, L., Rumboldt, Z., Yusufali, A., Xie, C., Anand, S.S., Engert, J.C., Rangarajan, S., and Yusuf, S. (2011). Parental history and myocardial infarction risk across the world: the INTERHEART Study. *Journal of the American College of Cardiology*, 57(5):619-627. <https://doi.org/10.1016/j.jacc.2010.07.054>

Ganeshan, D. (2022). Analytical Solution of the Effect of Awareness Program by Media on the Spread of an Infectious Disease by Homotopy Perturbation Method. *International Journal of Mathematical Modelling and Computations*, 12(2):131-142. <https://doi.org/10.30495/ijm2c.2022.1937350.1228>

He, J.H. (2003). Homotopy perturbation method: a new nonlinear analytical technique. *Applied Mathematics and computation*, 135(1):73-79. [https://doi.org/10.1016/S0096-3003\(01\)00312-5](https://doi.org/10.1016/S0096-3003(01)00312-5)

Ismailov, R.M. (2005). Mathematical model of blunt injury to the vascular wall via formation of rouleaux and changes in local hemodynamic and rheological factors. Implications for the mechanism of traumatic myocardial infarction. *Theoretical Biology and Medical Modelling*, 2(1):1-10. <https://doi.org/10.1186/1742-4682-2-13>

Kumar, A., Kumar, V., Kumar, M., Kumar, A., Srivastav, V.K., Pandey, R., and Kumar, M. (2020). Numerical studies of blockage in human heart. In: Bhattacharyya, S., Kumar, J., and Ghoshal, K. (eds). *Mathematical Modeling and Computational Tools*. ICACM 2018. Springer Proceedings in Mathematics & Statistics, Springer, Singapore, vol 320. https://doi.org/10.1007/978-981-15-3615-1_17

Maqbool, A. and Kumar, M. (2018). Study and implementation of Dijkstra's algorithm in angiography process to analysis veins blockage in Human's Heart. *International Journal of Advanced Engineering Science and Technological Research*, 6(3):29-37.

Moise, N. and Friedman, A. (2022). A mathematical model of immunomodulatory treatment in myocardial infarction. *Journal of Theoretical Biology*, 544:111122. <https://doi.org/10.1016/j.jtbi.2022.111122>

Ng, J., Jacobson, J.T., Ng, J.K., Gordon, D., Lee, D.C., Carr, J.C., and Goldberger, J.J. (2012). Virtual electrophysiological study in a 3-dimensional cardiac magnetic resonance imaging model of porcine myocardial infarction. *Journal of the American College of Cardiology*, 60(5):423-430. <https://doi.org/10.1016/j.jacc.2012.03.029>

Promit, T.A., Khan, M.A.R., Arnob, N., Rashid, R.N., Reza, A., Alam, M.A., and Dipto, S.M. (2022). Detection of coronary artery blockage at an early stage using effective deep learning technique. In: 2022 25th International Conference on Computer and Information Technology (ICCIT), Cox's Bazar, Bangladesh, 2022, p. 523-528. <https://doi.org/10.1109/ICCIT57492.2022.10055691>

Rajabi, A., Ganji, D.D., and Taherian, H. (2007). Application of homotopy perturbation method in nonlinear heat conduction and convection equations. *Physics Letters A*,

- 360(4-5):570-573. <https://doi.org/10.1016/j.physleta.2006.08.079>
- Ranthe, M.F., Petersen, J.A., Bundgaard, H., Wohlfahrt, J., Melbye, M., and Boyd, H.A. (2015). A detailed family history of myocardial infarction and risk of myocardial infarction-a nationwide cohort study. *PLoS One*, 10(5):e0125896. <https://doi.org/10.1371/journal.pone.0125896>
- Saha, S., Biswas, P., and Nath, S. (2021). Numerical simulation of blocked blood vessel for early diagnosis of coronary artery disease. In: Das, B., Patgiri, R., Bandyopadhyay, S., and Balas, V.E. (eds). *Modeling, Simulation and Optimization. Smart Innovation, Systems and Technologies*, Springer, Singapore, vol 206. https://doi.org/10.1007/978-981-15-9829-6_56
- Tentner, A., Guria, G., Ioilev, A., Lo, S., Onoufriou, A., and Samigulin, M. (2010). Computational Modeling of Blood Hydrodynamics and Blockage Formation Phenomena in the Human Cardiovascular System. *Journal of Medical Devices*, 4(2):027504. <https://doi.org/10.1115/1.3439651>
- Vijayalakshmi, T. and Senthamarai, R. (2022). Application of homotopy perturbation and variational iteration methods for nonlinear imprecise prey-predator model with stability analysis. *The Journal of Supercomputing*, 78(2):2477-2502. <https://doi.org/10.1007/s11227-021-03956-5>
- Wang, Y., Yang, T., Ma, Y., Halade, G.V., Zhang, J., Lindsey, M.L., and Jin, Y.F. (2012). Mathematical modeling and stability analysis of macrophage activation in left ventricular remodeling post-myocardial infarction. *BMC Genomics*, 13(Suppl 6):S21 (2012). <https://doi.org/10.1186/1471-2164-13-S6-S21>