

Research Article

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Mathematical Modelling of the Effects of Trans Fat on Bioheat Transfer and Magnetohydrodynamic Blood Flow through a Constricted Channel Using the Oscillatory Perturbation Method

KW Bunonyo¹ and Peter Benneth²¹ Department of Mathematics and Statistics Federal University of Agriculture Bassambiri, Nigeria² Department of Mathematics and Statistics Federal University Otuoke Bayelas, Nigeria**ABSTRACT**

This study develops a mathematical model for investigating the effects of trans-fat-related atherogenic lipid burden on blood flow, heat transfer, and concentration transport through a constricted artery under magnetohydrodynamic conditions. The artery is represented as a constricted channel containing a porous region, while blood is treated as an incompressible, electrically conducting fluid subjected to pulsatile pressure forcing and a transverse magnetic field. The governing momentum, energy, and concentration equations incorporate porous resistance, magnetic interaction, bioheat transfer, mass diffusion, concentration reaction, and concentration-induced buoyancy. The resulting coupled equations are treated using the Oscillatory Perturbation Method, and the effects of the governing parameters are examined through velocity, temperature, concentration, and arterial-constriction contour plots. The results show that variations in Darcy number from 0.01 to 0.05 significantly modify porous resistance and consequently influence blood velocity. Increasing the stenosis parameter from 0 to 0.55 produces progressively stronger arterial geometrical deformation, while increasing the magnetic parameter from 0.5 to 3 strengthens magnetic resistance and suppresses blood motion. An increase in the pressure-gradient parameter from 1 to 4 enhances the driving force and increases the magnitude of the velocity response. Concentration-related parameters substantially influence mass transport and its coupling with momentum, with concentration reaction values from 0.2 to 2 affecting concentration attenuation and concentration buoyancy values from 0 to 15 modifying concentration-momentum interaction. Increasing the Prandtl number from 21 to 25 alters the thermal field, whereas increasing the Schmidt number from 0.2 to 1.0 restricts concentration diffusion within the arterial region. The constriction results further demonstrate the strong influence of arterial geometry on the available flow passage and the resulting haemodynamic environment. Medically, the findings suggest that arterial deformation, porous resistance, altered lipid-related transport, and pressure forcing can interact to produce significant changes in blood-flow and transport characteristics within a stenosed artery. The concentration variable is interpreted as an effective trans-fat-related atherogenic lipid burden rather than direct deposition of intact dietary trans-fat particles. The model therefore provides a theoretical study for understanding coupled haemodynamic, thermal, and concentration-transport processes associated with atherosclerotic arterial constriction and provides a basis for future patient-specific and experimentally validated investigations.

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Abbreviation	Meaning
MHD	Magnetohydrodynamics
OPM	Oscillatory Perturbation Method
TFA	Trans-Fatty Acid
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
LDL	Low-Density Lipoprotein
HDL	High-Density Lipoprotein
WSS	Wall Shear Stress

Nomenclature

Symbol	Description
x	Dimensionless axial coordinate along the artery
χ	Dimensionless transverse coordinate across the arterial channel
t	Dimensionless time
h	Dimensionless arterial half-width or wall geometry function
δ	Arterial constriction or geometrical deformation parameter
w	Dimensionless axial blood velocity
w_0	Zeroth-order or leading-order velocity profile
θ	Dimensionless temperature
θ_0	Zeroth-order or leading-order temperature profile
θ_w	Dimensionless wall temperature
ϕ	Dimensionless concentration
ϕ_0	Zeroth-order or leading-order concentration profile
ϕ_w	Dimensionless wall concentration
P_0	Dimensionless pressure-gradient parameter
Da	Darcy number, measure of permeability of the porous arterial region
M	Magnetic parameter, represents the strength of magnetohydrodynamic interaction
Pr	Prandtl number, ratio of momentum diffusivity to thermal diffusivity
Sc	Schmidt number, ratio of momentum diffusivity to mass diffusivity
Gc	Concentration or solutal buoyancy parameter
Rd_1	Thermal radiation or heat-transfer attenuation parameter used in the energy equation
Rd_2	Concentration reaction or mass-transfer attenuation parameter
S_1	Heat-source/concentration-coupling parameter in the energy equation
ω	Dimensionless angular frequency of the oscillatory flow
i	Imaginary unit used in the complex representation of oscillatory quantities
β_1	Composite momentum parameter incorporating porous, magnetic and oscillatory effects
β_2	Composite thermal parameter incorporating Prandtl number, thermal attenuation and oscillation
β_3	Composite mass-transfer parameter incorporating Schmidt number, concentration reaction and oscillation
A_ϕ	Constant associated with the analytical concentration solution
A_θ	Constant associated with the particular solution of the temperature equation
K_c	Concentration–momentum coupling coefficient appearing in the velocity solution
C_5, C_6	Integration constants associated with the analytical velocity solution
e	Base of the natural logarithm
π	Mathematical constant pi

Symbol	Description	Typical SI Unit
ρ	Blood density	$\text{kg } m^{-3}$
μ	Dynamic viscosity of blood	Pa s
ν	Kinematic viscosity	$m^2 s^{-1}$
k	Thermal conductivity	$\text{W } m^{-1} K^{-1}$
K	Permeability of the porous medium	m^2
B_0	Applied magnetic-field strength	T
σ	Electrical conductivity of blood	$\text{S } m^{-1}$
c_p	Specific heat capacity at constant pressure	$\text{J } kg^{-1} K^{-1}$
D	Mass diffusivity	$m^2 s^{-1}$
T	Dimensional blood temperature	K
T_w	Arterial wall temperature	K
C	Effective trans-fat-related atherogenic concentration	model-dependent
C_w	Effective concentration at the arterial wall	model-dependent
p	Blood pressure	Pa

Keywords: Mathematical Modelling, Trans-fat, Atherosclerosis, Arterial Stenosis, Magnetohydrodynamics, Bioheat Transfer, Blood Flow, Concentration Transport, Oscillatory Perturbation Method

Introduction

Cardiovascular diseases (CVDs) remain one of the most serious public-health concerns worldwide, accounting for a substantial proportion of illness and death across different populations. Their development is closely associated with modifiable risk factors such as poor dietary habits, physical inactivity, obesity, hypertension, diabetes mellitus, smoking, and excessive consumption of industrially produced trans-fatty acids (World Health Organization [WHO], 2024). Among these factors, trans-fat consumption deserves particular attention because of its effects on blood lipids, vascular function, inflammation, and the development of atherosclerosis. Trans-fatty acids are unsaturated fatty acids containing at least one carbon-carbon double bond in the trans configuration. Although small quantities occur naturally in foods obtained from ruminant animals, industrial trans fats have historically been produced through the partial hydrogenation of vegetable oils and used in processed foods because they improve texture, stability, and shelf life (WHO, 2023).

The relationship between trans-fat consumption and cardiovascular health has been examined extensively. Evidence from epidemiological, clinical, and experimental studies indicates that excessive consumption can increase low-density lipoprotein cholesterol and may reduce high-density lipoprotein cholesterol, creating conditions that favour lipid accumulation within arterial walls. Trans fats have also been associated with inflammation, oxidative stress, insulin resistance, endothelial dysfunction, and impaired vascular reactivity [1,2]. De Souza reported associations between trans-fat intake and all-cause mortality, coronary heart disease, and coronary heart disease mortality, while found evidence of a dose-response relationship between trans-fat intake and cardiovascular disease risk. These findings provide a useful physiological basis for considering trans-fat-related atherogenic processes when developing mathematical models of cardiovascular disease [3,4].

Atherosclerosis represents an important link between abnormal lipid conditions and changes in arterial blood flow. It is a progressive inflammatory disease involving endothelial dysfunction, lipid accumulation, inflammatory-cell activity, fibrous-tissue formation, and structural changes in the arterial wall. Lopez-Garcia associated greater trans-fat intake with adverse markers of endothelial function, while Monguchi reported that excessive trans-fat intake accelerated atherosclerosis in a hyperlipidaemic animal model through inflammatory and oxidative-stress mechanisms. As an atherosclerotic plaque develops, it can progressively reduce the arterial lumen and eventually produce stenosis. The consequences are not limited to changes in lipid concentration, the narrowing also alters vascular resistance, blood-flow patterns, pressure distribution, and the mechanical forces acting on the arterial wall [5,6].

From a haemodynamic perspective, arterial stenosis can significantly disturb normal blood circulation. Reduction of the arterial cross-sectional area changes the velocity distribution, pressure gradient, volumetric flow rate, vascular resistance, and wall shear stress. Blood generally accelerates as it passes through a narrowed region, particularly near the stenosis throat, while disturbed flow, separation, recirculation, and relatively

low-velocity regions may develop downstream depending on the severity and geometry of the constriction. Severe stenosis also increases resistance to flow and may require a greater pressure difference to maintain adequate circulation. These changes are physiologically important because abnormal wall shear conditions can influence endothelial behaviour and are relevant to plaque progression and thrombosis. The importance of stenosis geometry has also been demonstrated in recent numerical investigations, where variations in the shape and dimensions of the constriction produced noticeable changes in predicted haemodynamic behaviour [7].

Mathematical modelling provides a useful means of studying these complex interactions. By representing physiological processes through governing equations, it becomes possible to investigate how individual parameters affect blood flow under controlled theoretical conditions. One important consideration in such models is the rheological behaviour of blood. Blood is a complex suspension whose flow properties depend on factors such as vessel size, shear rate, haematocrit, temperature, and disease state. In sufficiently large arteries and at relatively high shear rates, blood can often be approximated as a Newtonian fluid. In smaller vessels or regions where the shear rate is low or strongly disturbed, however, non-Newtonian effects may become important. For this reason, constitutive models such as the Casson, Carreau, Carreau–Yasuda, power-law, Herschel-Bulkley, and viscoelastic models have been used in haemodynamic studies. Zain for example, showed that blood rheology, stenosis characteristics, Reynolds number, and magnetic-field strength could influence velocity, streamline patterns, and wall shear stress in stenosed arterial flow [8].

Blood circulation is also closely connected with heat transport. As blood moves through the vascular system, it redistributes thermal energy and exchanges heat with surrounding tissues. Bioheat transfer therefore forms an important part of mathematical descriptions of biological transport processes. The classical bioheat equation proposed by Pennes (1948) remains one of the fundamental models for describing thermal transport in living tissues. It accounts for thermal conduction, blood perfusion, metabolic heat generation, and external heat sources. Although the Pennes formulation simplifies the detailed mechanisms of microvascular heat exchange, it has provided the basis for many subsequent bioheat models. Extensions have incorporated porous-medium effects, temperature-dependent perfusion, non-Fourier heat conduction, fractional derivatives, and more detailed vascular transport mechanisms [9]. In a stenosed artery, changes in blood flow and perfusion may influence local heat transport, making it useful to consider haemodynamics and temperature distribution within the same mathematical framework.

Another factor that has received considerable attention in theoretical blood-flow studies is the influence of an externally applied magnetic field. Blood contains dissolved electrolytes, ions, and other charged constituents and can therefore be treated, under appropriate assumptions, as a weakly electrically conducting fluid. When such a fluid moves in a magnetic field, electromagnetic forces may influence its motion. This provides the basis for magnetohydrodynamic (MHD) blood-flow modelling. Under commonly adopted assumptions, the Lorentz force generated by the magnetic interaction tends to resist fluid motion, thereby modifying blood velocity and related haemodynamic quantities. MHD models have been explored theoretically in connection with magnetic drug targeting, magnetic hyperthermia, biomedical

flow regulation, and diagnostic applications. Zain observed that changes in the Hartmann number affected velocity, recirculation, and wall shear stress in a stenosed bifurcated artery, illustrating how magnetic effects can interact with arterial geometry and blood rheology.

The pulsatile nature of arterial blood flow adds another level of complexity. Because the heart pumps periodically, arterial pressure and blood velocity vary throughout the cardiac cycle. These variations produce corresponding changes in flow rate and wall shear stress, making oscillatory mathematical models particularly relevant to arterial haemodynamics. Misra investigated oscillatory MHD blood flow through a porous arteriole in the presence of chemical reaction and heat and mass transfer and obtained analytical expressions for several important flow and transport quantities [10]. Bunonyo and Eli similarly studied oscillatory MHD blood flow through a lipid-saturated porous channel with metabolic heat generation and an applied magnetic field. Their study established an important mathematical connection between lipid-related porous resistance, magnetic effects, metabolic heat generation, and oscillatory blood flow [11].

Despite the progress made in these areas, there is still room for a more integrated mathematical description of the processes involved. Nutritional and epidemiological studies have established associations between industrial trans-fat consumption and adverse cardiovascular outcomes, while mathematical studies have investigated arterial stenosis, blood rheology, magnetic fields, porous resistance, heat transfer, and mass transport. These components, however, are often examined separately or only in selected combinations. In many existing stenosis models, arterial narrowing is introduced primarily as a prescribed geometric obstruction, without explicitly connecting an effective trans-fat-related atherogenic lipid burden to the concentration, thermal, and haemodynamic fields. Similarly, lipid-related MHD models do not generally combine an effective trans-fat-related concentration equation with stenosis and bioheat transfer within the same coupled system.

The present study addresses this gap by developing a mathematical model for trans-fat-related atherogenic lipid accumulation, bioheat transfer, and magnetohydrodynamic blood flow through a constricted arterial channel. The formulation couples the momentum, energy, and concentration equations and incorporates arterial stenosis, magnetic effects, porous resistance, blood perfusion, metabolic heat generation, concentration diffusion, chemical removal, and lipid deposition. Since arterial blood flow is naturally pulsatile, the Oscillatory Perturbation Method is employed to obtain approximate analytical solutions for the velocity, temperature, and concentration fields and to examine how changes in the relevant physiological and transport parameters influence the system.

In this formulation, the concentration variable is interpreted as an effective trans-fat-related atherogenic lipid burden, rather than as intact dietary trans-fat molecules moving directly through the artery. This distinction is important because dietary trans fats undergo digestion, absorption, metabolism, and incorporation into circulating lipid systems before contributing to endothelial dysfunction, atherosclerotic plaque development, and arterial narrowing. By bringing these processes together within a unified mathematical framework, the study provides a basis for examining the interactions among atherogenic lipid burden, stenosis-induced haemodynamic changes, bioheat transfer, and magnetic effects in pulsatile arterial flow.

Mathematical Formulation

Consider the unsteady flow of blood through a two-dimensional constricted channel representing an idealized stenosed artery. The constriction represents the reduction in the arterial lumen caused by atherosclerotic plaque formation associated with an increased atherogenic lipid burden. Blood is driven along the channel by a pulsatile pressure gradient, while a uniform magnetic field is applied perpendicular to the direction of flow. To account for the additional resistance produced by plaque accumulation, the stenosed region is modelled as a porous medium.

The mathematical formulation is based on three coupled transport processes: blood momentum, thermal energy, and effective trans-fat-related concentration. The momentum equation describes the motion of blood under the combined influence of the pressure gradient, viscous forces, magnetic resistance, porous resistance, and buoyancy effects arising from temperature and concentration differences. The energy equation describes heat transfer through thermal conduction, blood perfusion, metabolic heat generation, viscous dissipation, and Joule heating. The concentration equation, on the other hand, describes the transport of the effective atherogenic lipid burden through convection and diffusion, together with its removal and deposition at the arterial wall.

Since blood flow in arteries varies periodically with the pumping action of the heart, the pressure gradient is represented by steady and oscillatory components. The oscillatory component is assumed to be relatively small, making it possible to apply the Oscillatory Perturbation Method. This allows the velocity, temperature, and concentration fields to be separated into steady and oscillatory parts and provides a convenient analytical framework for examining the influence of the important physiological and transport parameters. The concentration variable introduced in the model should not be interpreted as intact dietary trans-fat molecules circulating directly through the artery. Dietary trans fats undergo digestion, absorption, metabolism, and incorporation into circulating lipoproteins before contributing to atherosclerotic processes. Therefore, the concentration variable is regarded as an effective trans-fat-related atherogenic lipid burden that represents, in a simplified mathematical form, the lipid-related processes associated with arterial plaque development.

Model Assumptions

To make the physiological problem mathematically tractable while retaining its important physical features, several assumptions are introduced. Blood is considered incompressible and electrically conducting, and its motion is assumed to be laminar, unsteady, and pulsatile. The artery is represented by a two-dimensional channel that is symmetric about its centreline, while the localized constriction represents an idealized stenosis produced by atherosclerotic plaque accumulation. Within the context of the model, the stenosis is associated with a trans-fat-related atherogenic lipid burden rather than the direct deposition of unchanged dietary trans-fat molecules.

A uniform magnetic field is applied perpendicular to the principal direction of blood flow. The magnetic Reynolds number is assumed to be sufficiently small so that the magnetic field induced by the motion of blood can be neglected in comparison with the externally imposed field. The external electric field and Hall-current effects are also neglected. Under these assumptions, the magnetic influence on blood motion is represented primarily through the Lorentz-force term in the momentum equation.

The arterial wall is assumed to remain rigid during the pulsation period considered. Although arterial walls are naturally elastic, this assumption simplifies the present analysis by excluding fluid-structure interaction. Blood viscosity is allowed to vary with temperature and the effective atherogenic lipid concentration so that changes in the thermal and concentration fields can influence the resistance to blood flow. The plaque-affected region is further treated as a porous medium, with the resulting resistance represented by a Darcy-type contribution to the momentum equation.

Heat transfer within the system is assumed to occur through thermal conduction, blood perfusion, metabolic heat generation, viscous dissipation, and Joule heating. The effective trans-fat-related concentration is transported by convection and molecular diffusion, while chemical or metabolic removal and deposition at

the arterial wall provide additional mechanisms for reducing the circulating concentration.

Density variations are assumed to be small and are neglected except where they contribute to buoyancy forces. Consequently, temperature- and concentration-induced body forces are represented through the Boussinesq approximation. The pressure gradient responsible for driving the flow consists of steady and time-dependent components, reflecting the pulsatile nature of arterial circulation. Finally, the amplitude of the oscillatory component is assumed to be sufficiently small to justify the use of perturbation analysis. Unless otherwise stated, the thermophysical and electromagnetic properties of blood are taken as constant, except for viscosity where an explicit dependence on temperature and effective lipid concentration is introduced.

Governing Models

Geometry due to Tran's fat

$$y^* = \begin{cases} R_0 - \delta^* \cos\left(\frac{2\pi x^*}{\lambda^*}\right) & \text{at } d_0 \leq x^* \leq \lambda^* \\ R_0 & \text{at } 0 \leq x^* \leq d_0 \end{cases} \quad (3.1)$$

MHD Blood Momentum Equation

$$\frac{\partial w^*}{\partial t^*} = \frac{1}{\rho_b} \frac{\partial P^*}{\partial x^*} + \nu_b \frac{\partial^2 w^*}{\partial y^{*2}} - \frac{\nu_b w^*}{k^*} - \sigma \frac{B_0^2 w^*}{\rho_b} + g\beta_c (C - C_a) \quad (3.2)$$

Bioheat Transfer Equation

$$\rho_b c_b \frac{\partial T^*}{\partial t^*} = k_b \frac{\partial^2 T^*}{\partial y^{*2}} - \rho_a \rho_b w_b (T^* - T_a) + S(C^* - C_a) \quad (3.3)$$

Tran's Fat Concentration Equation

$$\frac{\partial C^*}{\partial t^*} = D_m \frac{\partial^2 C^*}{\partial y^{*2}} - k_c (C - C_a) \quad (3.4)$$

The corresponding boundary conditions are:

$$\left. \begin{aligned} \frac{\partial w^*}{\partial y^*} = 0, \frac{\partial T^*}{\partial y^*} = 0, \frac{\partial C^*}{\partial y^*} = 0 & \quad \text{at } y^* = 0 \\ w^* = 0, T^* = T_w + T_a, C^* = C_w + C_a & \quad \text{at } y^* = R \end{aligned} \right\} \quad (3.5)$$

Dimensional Variables

We introduce the following dimensionless parameters:

$$\left. \begin{aligned} y &= \frac{y^*}{R_0}, x = \frac{x^*}{R_0}, \lambda = \frac{\lambda^*}{R_0}, \delta = \frac{\delta^*}{R_0}, w = \frac{w^*}{\nu_b w_0}, P = \frac{R_0 P^*}{\mu_b \nu_b w_0}, \theta = \frac{T^* - T_a}{T_w - T_a}, Rd_c = \frac{k_c R_0^2}{\nu_b}, \\ Rd_1 &= \frac{\rho_a \omega_b R_0^2}{\nu_b c_b}, Gr = \frac{g\beta_{Tb} (T_w - T_a) R_0^2}{\nu_b^2 w_0}, Gc = \frac{g\beta_c (C_w - C_a) R_0^2}{\nu_b^2 w_0}, S_1 = \frac{R_0^2 S (C_w - C_a)}{k_b (T_w - T_a)}, \\ \phi_w &= \frac{C_w}{C_w - C_a}, \theta_w = \frac{T_w}{T_w - T_a}, M^2 = \frac{\sigma B_0^2 R_0^2}{\mu_b}, t = \frac{\nu t^*}{R_0^2}, Pr = \frac{\mu_b c_b}{k_b}, Sc = \frac{\nu_b}{D_m}, \phi = \frac{C^* - C_a}{C_w - C_a}, \\ \frac{1}{Da} &= \frac{R_0^2}{k^*}, y = \frac{R}{R_0} = h \end{aligned} \right\} \quad (3.6)$$

Dimensionless Equations

The dimensionless governing equations and boundary conditions are

$$y = \begin{cases} 1 - \delta \cos(2\pi x) & \text{at } \frac{d_0}{R_0} \leq x \leq \lambda \\ 1 & \text{at } 0 \leq x \leq \frac{d_0}{R_0} \end{cases} \quad (3.7)$$

$$\frac{\partial w}{\partial t} = -\frac{\partial P}{\partial x} + \frac{\partial^2 w}{\partial y^2} - \frac{w}{Da} - M^2 w + Gc\phi \quad (3.8)$$

$$Pr \frac{\partial \theta}{\partial t} = \frac{\partial^2 \theta}{\partial y^2} - Rd_1 Pr \theta + S_1 \phi \quad (3.9)$$

$$Sc \frac{\partial \phi}{\partial t} = \frac{\partial^2 \phi}{\partial y^2} - Rd_2 Sc \phi \quad (3.10)$$

The boundary condition with stenosis, that is :

$$\left. \begin{aligned} \frac{\partial w}{\partial y} = 0, \theta = 0, \phi = 0 & \quad \text{at} \quad y = 0 \\ w = 0, \theta = \theta_w, \phi = \phi_w & \quad \text{at} \quad y = h = (1 - \delta \cos(2\pi x)) \end{aligned} \right\} \quad (3.11)$$

Method of Solution

We shall adopt the method of oscillatory perturbation (OPM) due to the pumping rate of the heart which acts in pulsatile form. Hence, the solution is considered to be in the form

$$\left. \begin{aligned} w(y, t) &= w_0 e^{i\omega t} \\ \theta(y, t) &= \theta_0 e^{i\omega t} \\ \phi(y, t) &= \phi_0 e^{i\omega t} \\ \frac{dP}{dx} &= P_0 e^{i\omega t} \end{aligned} \right\} \quad (3.12)$$

Introducing the transformation

$$\chi = \frac{y}{h} \quad (3.13)$$

Simplifying equations (3.7) - (3.11) to reduce to a system of coupled ODE using the oscillatory perturbation method (OPM) in equation (3.13), we have the system as:

$$\frac{d^2 w_0}{d\chi^2} - \beta_1^2 w_0 = P_0 h - h Gc \phi_0 \quad (3.14)$$

$$\frac{d^2 \theta_0}{d\chi^2} - \beta_2^2 \theta_0 = -S_1 h \phi_0 \quad (3.15)$$

$$\frac{d^2 \phi_0}{d\chi^2} - \beta_3^2 \phi_0 = 0 \quad (3.16)$$

The corresponding boundary conditions with stenosis :

$$\left. \begin{aligned} \frac{dw_0}{d\chi} = 0, \theta_0 = 0, \phi_0 = 0 & \quad \text{at} \quad \chi = 0 \\ w_0 = 0, \theta_0 = \theta_w e^{-i\omega t}, \phi_0 = \phi_w e^{-i\omega t} & \quad \text{at} \quad \chi = 1 \end{aligned} \right\} \quad (3.17)$$

where $h = (1 - \delta \cos(2\pi x))$, $\beta_1^2 = h \left(\frac{1}{Da} + M^2 + i\omega \right)$, $\beta_2^2 = h(Rd_1 + i\omega)Pr$, $\beta_3^2 = h(Rd_2 + i\omega)Sc$

Solving equation (3.16), we have:

$$\phi_0 = A_1 \cosh(\beta_3 \chi) + B_1 \sinh(\beta_3 \chi) \quad (3.18)$$

Solving equation (3.18) using the boundary conditions in equation (3.17), we have:

$$\phi_0 = \frac{\phi_w e^{-i\omega t}}{\sinh(\beta_3)} \sinh(\beta_3 \chi) \quad (3.19)$$

Substitute equation (3.19) into equation (3.15), we have:

$$\frac{d^2 \theta_0}{d\chi^2} - \beta_2^2 \theta_0 = \beta_4 \sinh(\beta_3 \chi) \quad (3.20)$$

$$\text{where } \beta_4 = -\frac{\phi_w S_1 h e^{-i\omega t}}{\sinh(\beta_3)}$$

Solving equation (3.20), we have

$$\theta_0 = A_2 \cosh(\beta_2 \chi) + B_2 \sinh(\beta_2 \chi) + \frac{\beta_4}{(\beta_3^2 - \beta_2^2)} \quad (3.21)$$

$$\theta_0 = \left(\frac{\beta_4}{\beta_3^2 - \beta_2^2} \right) (\cosh(\beta_2 \chi) + 1) + B_2 \sinh(\beta_2 \chi) \quad (3.22)$$

$$\text{Where } A_2 = \left(\frac{\beta_4}{\beta_3^2 - \beta_2^2} \right), B_2 = \frac{\theta_w e^{-i\omega t}}{\sinh(\beta_2)} - \left(\frac{\beta_4}{\beta_3^2 - \beta_2^2} \right) \frac{(1 + \cosh(\beta_2))}{\sinh(\beta_2)}$$

Simplifying equation (3.14), we have:

$$\frac{d^2 w_0}{d\chi^2} - \beta_1^2 w_0 = hP_0 - \frac{hGc\phi_w e^{-i\omega t}}{\sinh(\beta_3)} \sinh(\beta_3 \chi) \quad (3.23)$$

The solution of equation (3.23) is:

$$w_0 = A_4 \sinh(\beta_1 \chi) + B_4 \cosh(\beta_1 \chi) + A_0 + C_3 \sinh(\beta_3 \chi) \quad (3.24)$$

where

$$A_0 = -\frac{hP_0}{\beta_1^2}, C_3 = \frac{hGc\phi_w e^{-i\omega t}}{(\beta_1^2 - \beta_3^2) \sinh(\beta_3)}, A_4 = -\frac{C_3 \beta_3}{\beta_1}, B_4 = -\frac{A_0}{\cosh(\beta_1)} - C_3 \frac{\sinh(\beta_3)}{\cosh(\beta_1)} - A_4 \frac{\sinh(\beta_1)}{\cosh(\beta_1)} \quad (3.25)$$

Substituting equation (3.24) into equation (3.12), we have:

$$w(\chi, t) = (A_4 \sinh(\beta_1 \chi) + B_4 \cosh(\beta_1 \chi) + A_0 + C_3 \sinh(\beta_3 \chi)) e^{i\omega t} \quad (3.26)$$

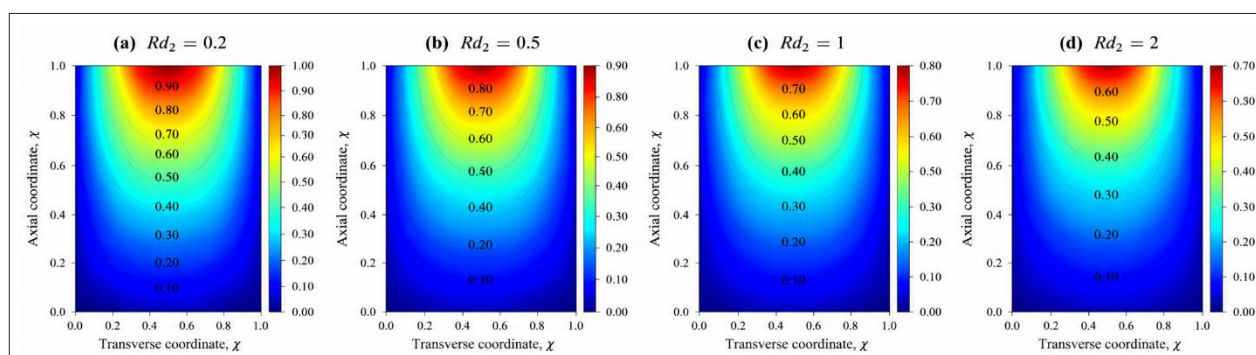
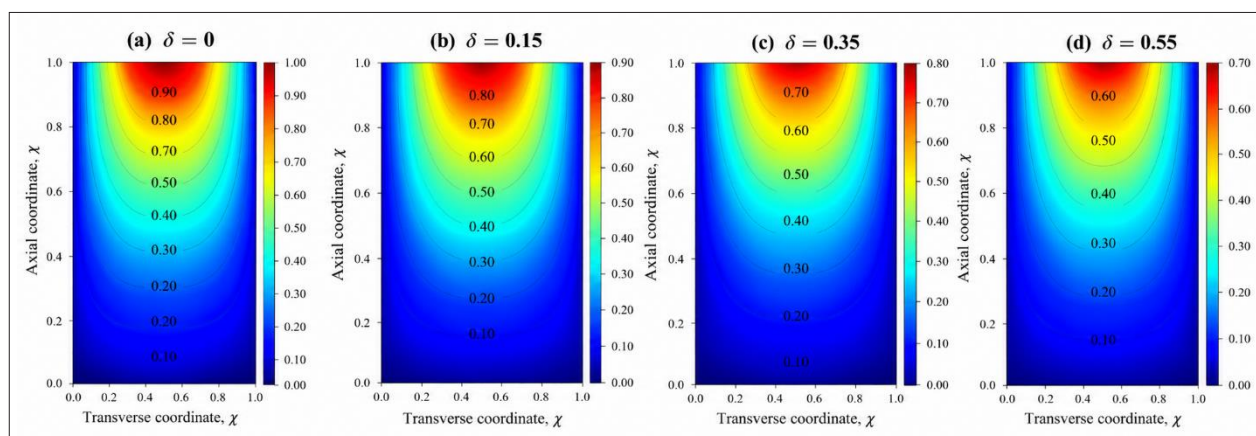
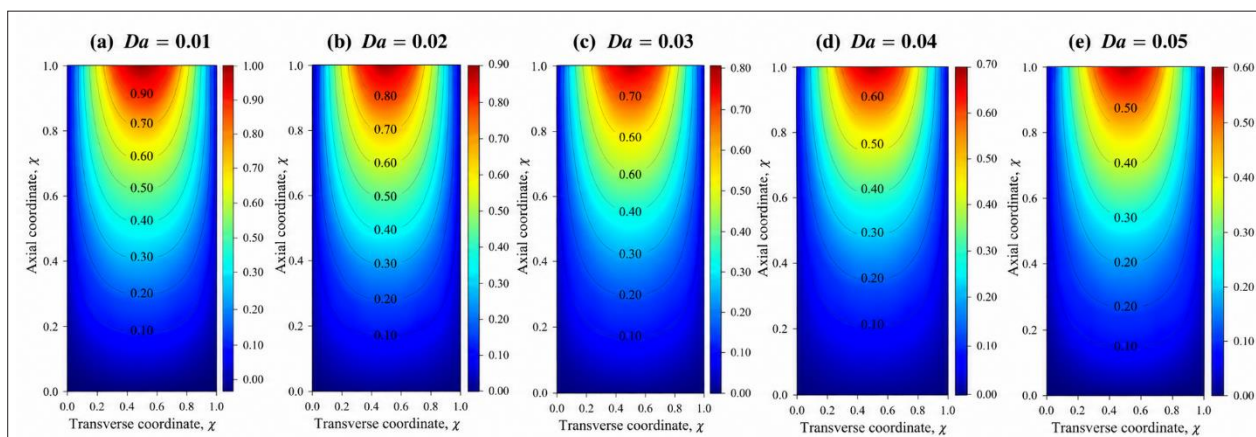
The oscillatory temperature and mass concentration of sickle cell are obtained by substituting equations (3.19) and (3.21) into equation (3.12), we have:

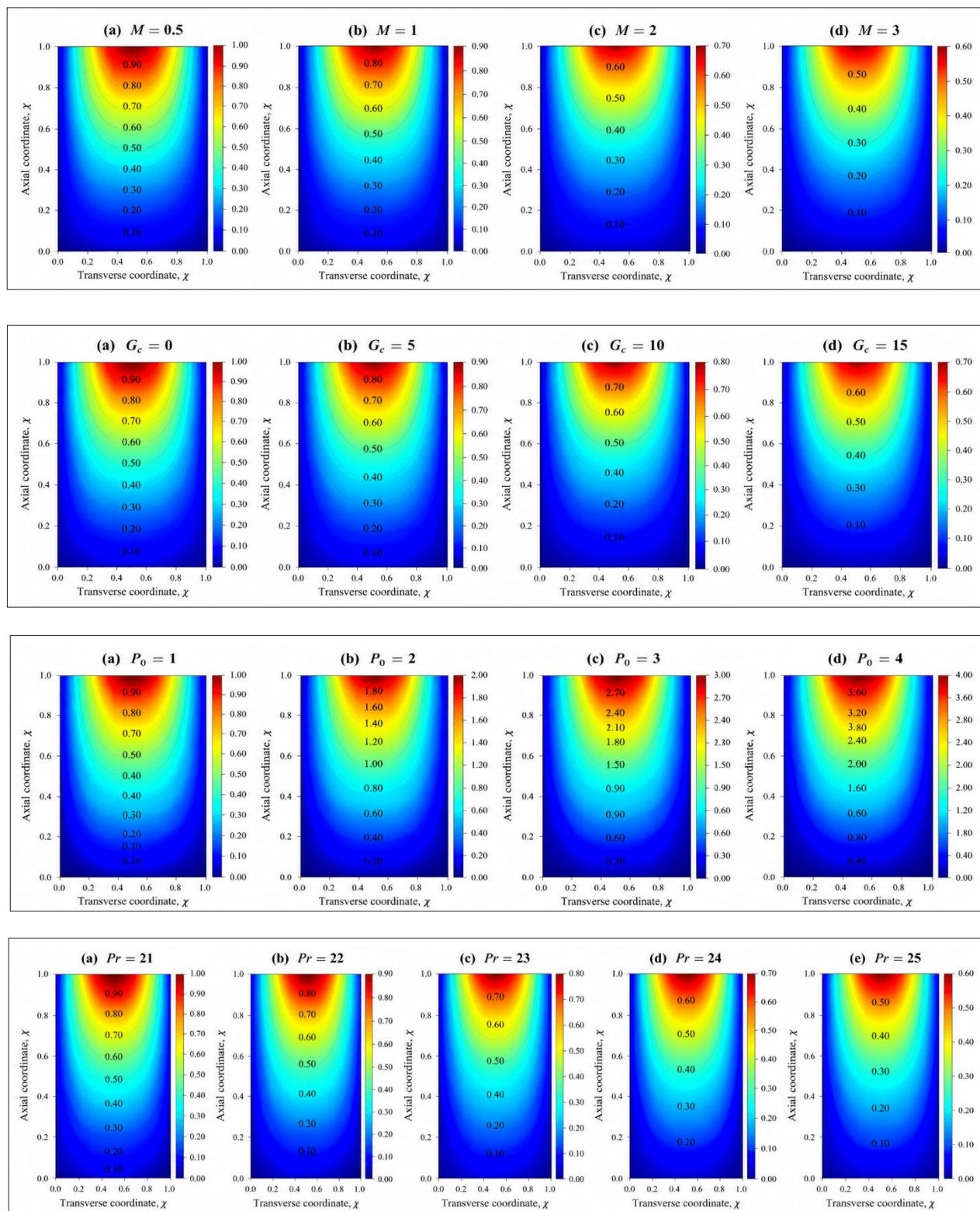
$$\theta(\chi, t) = \left(A_2 \cosh(\beta_2 \chi) + B_2 \sinh(\beta_2 \chi) + \frac{\beta_4}{(\beta_3^2 - \beta_2^2)} \right) e^{i\omega t} \quad (3.27)$$

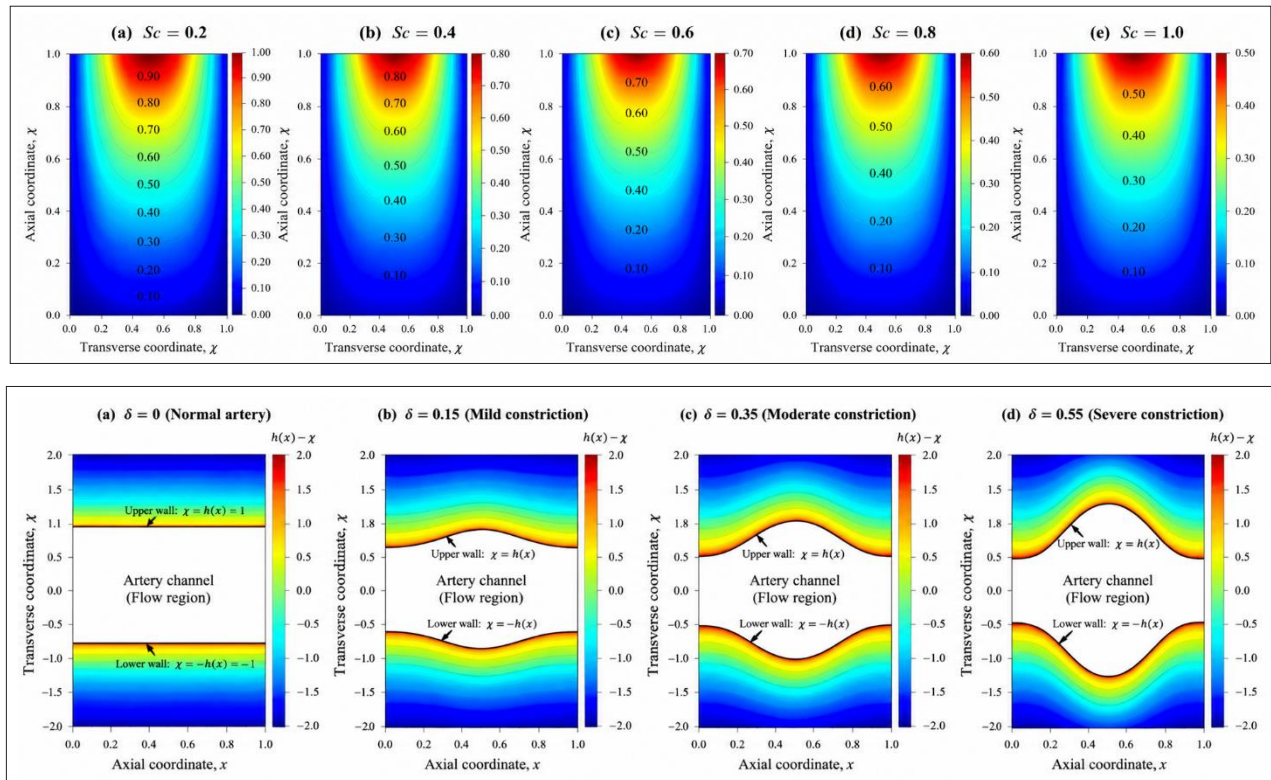
$$\phi(\chi, t) = \left(\frac{\phi_w e^{-i\omega t}}{\sinh(\beta_3)} \sinh(\beta_3 \chi) \right) e^{i\omega t} \quad (3.28)$$

Results

The results of the study are presented through nine figures, covering the velocity field, temperature distribution, concentration distribution, and geometry of the constricted artery. Figures 1-6 describe the effects of the flow and concentration-related parameters on the velocity profile, Figures 7 and 8 present the thermal and concentration fields, respectively, while Figure 9 illustrates the changes in arterial geometry associated with different degrees of constriction. The results are presented parametrically so that the influence of each quantity can be examined while the remaining parameters are maintained at their prescribed values.







Discussion

The contour plots presented in Figures 1-9 illustrate the influence of the principal dimensionless parameters on the velocity, temperature, and concentration fields within the stenosed arterial channel. Taken together, the figures show that the haemodynamic behaviour of the model is governed by the interaction of porous resistance, arterial geometry, magnetic forces, pressure forcing, concentration transport, and thermal diffusion. The discussion below interprets each figure in relation to the mathematical model, its possible medical significance, and findings reported in related haemodynamic studies. The comparisons with previous studies are primarily qualitative, since differences in arterial geometry, constitutive assumptions, boundary conditions, and parameter definitions prevent direct numerical comparison. They are discussed as follows: Figure 1: Contour plots of the velocity profile for different Darcy number values, $Da = 0.01, 0.02, 0.03, 0.04$, and 0.05 . Figure 1 demonstrates the influence of permeability on blood motion through the modelled porous arterial region. The Darcy number represents the relative permeability of the porous medium. Consequently, changing its value from 0.01 through 0.02, 0.03, and 0.04 to 0.05 changes the resistance offered by the porous region to the moving blood. Physically, a small Darcy number corresponds to low permeability and therefore stronger porous resistance. As the Darcy number increases, the porous medium becomes more permeable and its resistance to fluid motion is reduced. Within the governing momentum equation, this occurs through the Darcy resistance contribution. The velocity field is therefore sensitive to changes in Darcy number, particularly where porous resistance forms an appreciable part of the total resistance to flow.

This porous-medium interpretation is consistent with Misra and Adhikary who studied oscillatory magnetohydrodynamic blood flow through a porous arteriole together with heat and mass transfer and chemical reaction [12]. Their formulation likewise demonstrates the importance of porous-medium properties in determining physiological-fluid motion. Bunonyo and Eli (2020)

also modelled oscillatory blood flow through a lipid-saturated porous channel and reported that the governing flow profiles were affected by variations in the pertinent transport parameters [11]. From a medical perspective, the Darcy number should not be interpreted as a parameter that can simply be increased therapeutically. In the present model, it is an effective representation of permeability and resistance associated with the porous arterial environment. Its importance lies in showing how changes in the physical structure of a plaque-affected region could alter the ease with which blood moves through that region. Figure 2: Contour plots of the velocity profile for different stenosis parameter values, $\delta = 0, 0.15, 0.35$, and 0.55 . Figure 2 illustrates the progressive effect of arterial geometry on the velocity distribution. When the stenosis parameter is 0, the geometry represents the reference case without the imposed stenotic modulation. At 0.15, the effect of arterial constriction becomes noticeable. Increasing the value to 0.35 strengthens the geometrical disturbance, while 0.55 represents the strongest geometrical modification among the cases considered. The result demonstrates that arterial geometry is an important determinant of haemodynamic behaviour. Changing the available flow region alters the spatial distribution of velocity and therefore affects the gradients that develop within the artery. This observation is supported by who investigated blood flow through a bifurcated artery with overlapping stenosis and showed that stenotic geometry, magnetic effects, pressure, and wall shear characteristics interact significantly in the diseased vessel.

The medical significance is particularly important because haemodynamic disturbances around stenotic regions can influence the mechanical environment experienced by the arterial wall. Regions around a stenosis may exhibit substantial changes in velocity, pressure, and wall shear stress. For this reason, stenosis severity should not be considered merely as a geometrical measurement, its interaction with the surrounding flow field is also important when assessing the consequences of arterial obstruction. A qualification is necessary for the present geometry. Because the

adopted expression $h=1-\delta\cos\left[\frac{\pi}{2}(2\pi x)\right]$ produces both narrowing and widening relative to the reference width, increasing the stenosis parameter is more precisely described as increasing the geometrical modulation of the channel. Figure 3: Contour plots of the velocity profile for different concentration reaction parameter values, $Rd_2 = 0.2, 0.5, 1$, and 2 . Figure 3 examines how changes in the concentration-reaction mechanism affect the velocity field. As the concentration reaction parameter changes from 0.2 to $0.5, 1$, and 2 , the rate at which the effective concentration is attenuated within the transport equation becomes progressively stronger. The resulting modification of the concentration field subsequently affects velocity through the concentration-dependent term appearing in the momentum equation. The mechanism is therefore indirect: the reaction parameter first affects concentration, and concentration then modifies the momentum response. A larger reaction parameter generally promotes stronger attenuation of the transported concentration, reducing its penetration into the fluid domain. This coupling between chemical reaction, mass transfer, and physiological-fluid motion is closely related to the formulation of Misra and Adhikary [11]. Their oscillatory MHD model explicitly incorporated chemical reaction together with heat and mass transfer and demonstrated that these transport processes affect physiological-fluid behaviour.

For the present study, the concentration variable should not be interpreted as intact dietary trans-fat particles physically accumulating in the artery. A more biologically defensible interpretation is an effective trans-fat-related atherogenic lipid burden. Under this interpretation, the concentration-reaction parameter represents a simplified removal, transformation, or attenuation process affecting that modelled burden. This distinction is important when translating the mathematical result into a biomedical context. Figure 4: Contour plots of the velocity profile for different magnetic parameter values, $M = 0.5, 1, 2$, and 3 . Figure 4 demonstrates the effect of increasing the magnetic parameter from 0.5 to $1, 2$, and 3 . In the present momentum equation, the magnetic contribution forms part of the resistance acting on the flow. Increasing the magnetic parameter therefore strengthens the magnetohydrodynamic influence and modifies the velocity distribution.

The general importance of magnetic interaction in arterial-flow modelling is supported by Their numerical investigation considered an external transverse magnetic field acting on blood flowing through a stenosed bifurcated artery. They reported changes in velocity, pressure, and wall shear behaviour as magnetic intensity varied. Magnetic effects were also incorporated by Bunonyo and Eli in their lipid-saturated porous-channel model and by Bunonyo and Amos in their investigation of cholesterol-related flow through a constricted cylinder [11,13]. The mathematical result establishes that magnetic interaction can alter the flow of an electrically conducting fluid under the assumptions of the model. It does not establish magnetic-field application as a treatment for atherosclerosis or stenosis. Translation to medicine would require realistic magnetic-field strengths, accurate blood and tissue electromagnetic properties, experimental validation, and careful assessment of safety.

Figure 5: Contour plots of the velocity profile for different concentration buoyancy parameter values, $G_c = 0, 5, 10$, and 15 . Figure 5 considers concentration-momentum coupling by varying the concentration buoyancy parameter from 0 through 5 and 10 to 15 . When its value is zero, the concentration field does not contribute through this particular buoyancy term. At

values of $5, 10$, and 15 , concentration-dependent forcing becomes progressively more important.

The magnitude and direction of the resulting velocity response depend on the sign convention adopted for the concentration term. Therefore, the most defensible interpretation is that increasing this parameter strengthens the coupling between concentration and momentum, rather than assuming that a larger concentration must always increase or always decrease velocity.

A comparable concentration-flow interaction appears in Bunonyo and Amos [13]. Their cholesterol-related blood-flow model varied the solutal Grashof number, Schmidt number, radiation parameter, and other transport quantities and found that changes in concentration-related parameters affected the blood-flow response. Bunonyo and Eli similarly considered coupled momentum, energy, and lipid-concentration equations in an oscillatory porous-channel model [11]. The result shows that alterations in the modelled atherogenic environment can influence haemodynamics when concentration and momentum are coupled. Figure 6: Contour plots of the velocity profile for different pressure-gradient parameter values, $P_0 = 1, 2, 3$, and 4 .

Figure 6 shows one of the clearest responses among the velocity contours. Increasing the magnitude of the pressure-gradient parameter from 1 to $2, 3$, and 4 strengthens the force driving blood through the channel. Consequently, the magnitude of the velocity response increases as the pressure forcing becomes stronger, provided that the remaining parameters are held constant.

This behaviour follows directly from the momentum equation because pressure forcing provides the principal mechanical driving mechanism for the flow. Misra and Adhikary likewise investigated pressure-driven oscillatory physiological flow and derived expressions for velocity, volumetric flow rate, and wall shear stress. The oscillatory blood-flow model of Bunonyo and Eli also incorporated pressure-driven motion in a porous, lipid-saturated environment [11-12]. A substantial pressure difference may be required to maintain flow across a region of increased vascular resistance. Moreover, increased local velocity through a stenotic region may coexist with large pressure changes and altered wall shear stress. Figure 7: Contour plots of the temperature profile for different Prandtl number values, $Pr = 21, 22, 23, 24$, and 25 . Figure 7 shifts attention from momentum transport to thermal behaviour. Increasing the Prandtl number from 21 to $22, 23, 24$, and 25 changes the relative importance of momentum diffusion and thermal diffusion. In general, a larger Prandtl number corresponds to weaker thermal diffusivity relative to momentum diffusivity. The thermal disturbance therefore tends to become more spatially confined as the Prandtl number increases. This behaviour is relevant to the bioheat component of the study because temperature distribution within the model depends not only on heat generation but also on the ability of thermal energy to diffuse through the fluid. Misra and Adhikary explicitly considered heat transfer in oscillatory physiological flow, while Bunonyo and Eli included metabolic heat in their coupled momentum-energy-concentration model [11-12].

The medical implication should again be interpreted at the modelling level. The temperature contours indicate how changes in the balance between momentum and thermal diffusion can modify the modelled thermal environment of the blood. They do not by themselves demonstrate clinically significant heating of an artery. A clinically meaningful bioheat interpretation would

require properties of blood and arterial tissue, perfusion effects, and experimental validation.

Importantly, in the equations presently used, the Prandtl number enters the temperature equation rather than directly entering the zeroth-order momentum equation. Figure 8: Contour plots of the concentration profile for different Schmidt number values, $Sc = 0.2, 0.4, 0.6, 0.8$, and 1.0 . Figure 8 illustrates the effect of mass diffusivity through Schmidt number values of $0.2, 0.4, 0.6, 0.8$, and 1.0 . The Schmidt number represents the relative importance of momentum diffusivity to mass diffusivity. Increasing the Schmidt number therefore corresponds to weaker molecular mass diffusion relative to momentum diffusion. Accordingly, the concentration disturbance becomes more restricted as the Schmidt number increases. Lower values permit stronger concentration diffusion across the channel, whereas higher values tend to confine the concentration closer to regions where it is imposed or generated. This behaviour is consistent with the importance of Schmidt number in the coupled lipid-concentration model developed by Bunonyo and Eli [11]. It is also supported by the cholesterol-flow study of Bunonyo and Amos which specifically examined Schmidt-number effects among the concentration-transport parameters and reported that Schmidt number influenced the cholesterol concentration behaviour. Figure 9 shows how the shape of the arterial channel changes as the stenosis parameter increases from 0 to $0.15, 0.35$, and 0.55 [13]. When the value is 0 , the artery maintains a uniform width and represents the reference condition without geometrical deformation. At 0.15 , a mild change in the arterial wall becomes visible. When the value increases to 0.35 , the deformation becomes considerably stronger, while 0.55 produces the greatest alteration in the arterial geometry among the cases investigated.

The numerical values clearly demonstrate the progression of the geometrical disturbance. At 0 , the normalized arterial half-width remains constant at 1.00 . At 0.15 , it varies between approximately 0.85 and 1.15 . Increasing the stenosis parameter to 0.35 produces values between approximately 0.65 and 1.35 , while at 0.55 , the half-width varies from approximately 0.45 to 1.55 . Thus, increasing the stenosis parameter progressively increases the magnitude of arterial-wall deformation [14-22].

From a haemodynamic perspective, this change in arterial geometry is important because blood flow is highly sensitive to the available flow passage. Where the arterial lumen becomes smaller, blood is forced through a more restricted region. This can modify the local velocity distribution, increase velocity gradients, alter pressure distribution, and produce changes in wall shear stress. The effect becomes increasingly important as the degree of geometrical deformation increases.

The result also provides a useful explanation for the velocity changes observed in the other contour plots. A mildly altered artery is expected to produce a smaller disturbance to the flow than a strongly deformed artery. Consequently, the cases corresponding to 0.35 and 0.55 are expected to produce more pronounced changes in the haemodynamic environment than the cases corresponding to 0 and 0.15 , particularly when the geometrical effect interacts with pressure forcing, porous resistance, concentration transport, and magnetic effects.

Medically, Figure 9 demonstrates why progressive arterial obstruction can become important in cardiovascular disease. A reduction in the available lumen can make it more difficult

for blood to pass through the affected region and may require a greater pressure difference to maintain adequate flow. The associated changes in velocity and wall shear conditions may also influence the mechanical environment experienced by the arterial wall. In the context of this study, the figure therefore provides a geometrical representation of how increasing atherosclerotic involvement could contribute to progressively disturbed blood flow. However, the geometry used in the present model has an important characteristic that should be acknowledged. The adopted mathematical representation produces regions that are narrower and wider than the normal artery. Therefore, the values from 0.15 to 0.55 should preferably be described as representing increasing arterial geometrical deformation or constriction-expansion severity, rather than assuming that every location along the artery represents narrowing.

Conclusion

The results presented in Figures 1-9 demonstrate that blood flow, heat transfer, concentration transport, and arterial geometry are strongly interconnected within the proposed mathematical model. The numerical and contour results show that changes in the physical and transport parameters can substantially alter the haemodynamic environment of a constricted artery. Collectively, the findings provide a mathematical explanation of how arterial narrowing, porous resistance, pressure forcing, magnetic interaction, thermal transport, and an effective trans-fat-related atherogenic concentration may interact within the cardiovascular system. The Darcy-number results show that changing the value from 0.01 to 0.05 modifies the resistance associated with the porous region. Greater permeability corresponds to weaker porous resistance, allowing blood to move more readily through the modelled arterial region. This establishes permeability as an important factor in determining the extent to which a plaque-affected or porous arterial environment restricts blood motion.

The stenosis results demonstrate that increasing the geometrical parameter from 0 to $0.15, 0.35$, and 0.55 produces progressively stronger deformation of the arterial channel. Figure 9 particularly clarifies this geometrical effect. The normal case maintains a constant normalized half-width of 1.00 , whereas the strongest geometrical variation considered produces values ranging approximately from 0.45 to 1.55 . Such changes in the available flow passage can substantially modify velocity gradients, pressure distribution, and potentially wall shear stress. The adopted geometry produces both narrowing and widening, however, so the result is most accurately interpreted as increasing arterial constriction-expansion or geometrical deformation.

The concentration-related results further demonstrate that mass transport is capable of interacting with the haemodynamic system. Increasing the concentration reaction parameter from 0.2 to 2 strengthens the attenuation of the transported concentration, while increasing the concentration buoyancy parameter from 0 to 15 strengthens the coupling between concentration and momentum. These results indicate that changes in the modelled atherogenic environment can influence blood-flow behaviour. In interpreting this result medically, the concentration variable represents an effective trans-fat-related atherogenic lipid burden rather than intact dietary trans-fat particles circulating and depositing directly within the artery.

The magnetic results show that increasing the magnetic parameter from 0.5 to 3 strengthens the resistance associated with magnetohydrodynamic interaction and modifies the velocity

distribution. In contrast, increasing the pressure-gradient parameter from 1 to 4 strengthens the mechanical driving force and consequently increases the magnitude of blood motion under the adopted pressure convention. These opposing effects demonstrate the balance between driving and resisting mechanisms within the momentum equation. The magnetic-field result remains a theoretical feature of the model and should not be interpreted as evidence that magnetic fields constitute a clinical treatment for arterial stenosis.

The temperature and concentration contours provide further information about transport within the artery. Increasing the Prandtl number from 21 to 25 modifies thermal diffusion and changes the temperature distribution, while increasing the Schmidt number from 0.2 to 1.0 reduces the relative importance of mass diffusion and increasingly confines the concentration field. These findings confirm that thermal and concentration transport cannot necessarily be separated from the overall physical environment when coupled haemodynamic processes are being considered.

From a medical perspective, the combined results suggest that progressive arterial deformation, reduced permeability, altered concentration transport, and changes in pressure forcing can create substantially different haemodynamic conditions within an affected artery. The results are particularly relevant to understanding how an atherosclerotic environment may influence blood-flow distribution and transport processes. However, an increase in velocity should not automatically be regarded as physiologically beneficial because high local velocity may coexist with substantial pressure loss and abnormal wall shear conditions within a stenosed artery.

In all, Figures 1-9 support the usefulness of the proposed model for examining the simultaneous effects of arterial constriction, porous resistance, magnetic interaction, pressure forcing, bioheat transfer, and trans-fat-related atherogenic concentration transport. The study therefore provides a mechanistic mathematical study for understanding how these processes may interact in a stenosed arterial environment. The predictions should nevertheless be regarded as theoretical rather than diagnostic or clinical findings. Future studies should extend the model to include realistic localized stenosis geometry, arterial-wall elasticity, non-Newtonian blood behaviour, dimensional wall shear stress, pressure drop and volumetric flow rate, and patient-specific arterial geometry. Validation against experimental measurements, Doppler ultrasound, angiographic data, or other patient-specific haemodynamic observations would further establish the physiological and clinical relevance of the model.

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