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Effects of External Body Acceleration on Blood Flow within a Porous Medium

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Abstract

This study examines the influence of external body acceleration on blood flow through a porous medium, a condition relevant to biological tissues, stenotic arteries, and engineered biomedical systems. Blood, being non-Newtonian, exhibits complex flow behavior that becomes further altered under acceleration forces generated during activities such as vibration, motion, or mechanical impact. A mathematical model incorporating Darcy–Brinkman porous resistance and an acceleration-induced body force is developed to analyze changes in velocity, pressure, and shear stress. Analytical and numerical techniques are applied to study flow characteristics under different porosity levels and acceleration magnitudes. The results indicate that external acceleration significantly affects flow resistance, modifies perfusion rates, and alters wall shear stress distribution. These findings hold importance for cardiovascular physiology, biomedical device design, and assessing dynamic hemodynamic responses under real-life motion conditions.

Keywords: external body acceleration, blood flow dynamics, porous medium, Darcy–Brinkman model, Thermodynamics

Introduction

The study of blood flow under the influence of external body acceleration within a porous medium has emerged as an important area of investigation in biomedical engineering, biofluid mechanics, and cardiovascular physiology. Blood, being a complex non-Newtonian fluid, exhibits significant changes in its velocity distribution, shear stress, and pressure characteristics when subjected to external accelerative forces arising from activities such as walking, running, vehicular movement, vibration exposure, and mechanical impacts. In many physiological and pathological situations, arterial or tissue structures behave like porous media, especially in regions affected by plaque deposition, microvascular networks, diseased tissues, dialysis

filters, and artificially engineered biological scaffolds. Understanding how acceleration interacts with porous resistance is crucial for predicting hemodynamic alterations under real-life dynamic conditions. External body acceleration contributes an additional body force in the momentum balance, modifying flow resistance, enhancing or suppressing perfusion, and influencing mass transport within biological tissues. When combined with the drag effects of porous structures—typically modeled using Darcy or Brinkman formulations—the resulting flow dynamics become significantly more complex and sensitive to material properties such as permeability, porosity, and viscous resistance. Such combined effects have substantial implications for cardiovascular health, as excessive accelerations may induce abnormal shear stresses, disrupt endothelial function, and alter perfusion in stenotic or partially obstructed vessels. Conversely, controlled acceleration is also used therapeutically in physiotherapy, vibration therapy, and rehabilitation to modulate blood flow. Moreover, the examination of flow behavior through porous media under acceleration is essential for designing bioreactors, prosthetic implants, and tissue-engineered constructs where fluid transport governs cellular response and nutrient delivery. Despite its importance, the interaction between acceleration forces and porous medium flow remains insufficiently explored, particularly for non-Newtonian blood models and unsteady physiological conditions. This study addresses these gaps by developing a structured mathematical representation of blood flow in a porous domain subjected to external body acceleration, analyzing the variations in velocity, shear stress, and flow characteristics, and providing insights relevant to biomedical applications and cardiovascular risk assessment.

Significance of the Study

This study holds significant value in advancing the understanding of how external body acceleration influences blood flow behavior within porous biological and arterial structures. Such conditions arise frequently in real-life situations, including vibration exposure, vehicular movement, physical activities, and therapeutic mechanical stimulation. By integrating porous medium resistance with acceleration-induced forces, the study provides a deeper insight into how perfusion, shear stress, and flow resistance vary in dynamically changing environments. These findings are particularly relevant for assessing cardiovascular risks associated with high acceleration environments, improving diagnostic interpretation of hemodynamic disturbances, and enhancing the design of biomedical devices such as tissue scaffolds, artificial filters, and porous implants. Moreover, the results contribute to improved modelling of blood flow through stenotic or diseased arteries, where porous-like behavior is prominent. This research enriches

biomedical engineering, physiology, and clinical applications by offering a refined understanding of blood flow under dynamic external forces.

Scope of the Study

The scope of this study encompasses the mathematical, computational, and physiological examination of how external body acceleration influences blood flow through a porous medium, reflecting conditions found in biological tissues, stenotic vessels, and engineered biomedical structures. The research focuses on developing a generalized flow model that integrates porous resistance with acceleration-induced body forces to analyze variations in velocity, shear stress, and pressure distribution. The study is limited to laminar, incompressible, and non-Newtonian blood behavior under controlled acceleration parameters, providing insights into how porosity, permeability, and acceleration intensity jointly affect Thermodynamics. While the model does not cover complex three-dimensional vascular networks or pulsatile multi-layered arterial walls, it offers a strong foundational understanding relevant to biomechanics, clinical diagnostics, and biomedical engineering applications. The scope supports future extensions into pulsatile flow, patient-specific geometries, and computational simulations.

Background of Blood Flow in Porous Medium

Blood flow through porous media has become an important subject in biomedical engineering and physiological fluid mechanics because many biological structures naturally behave like porous materials. Tissues such as muscles, capillary beds, arterial plaques, and diseased vessel walls permit fluid penetration through their microstructures, creating resistance that significantly alters normal hemodynamic behavior. In stenotic arteries, lipid and fibrous deposits form porous layers that influence velocity distribution, shear stress, and pressure drop across the affected region. Similarly, engineered biomedical devices such as artificial organs, dialysis membranes, tissue scaffolds, and drug-delivery matrices rely on controlled porous structures for effective fluid transport. Modeling blood flow in porous media commonly involves Darcy's Law or the Brinkman extension, which incorporate permeability and viscous drag to represent the resistance offered by the porous matrix. This flow behavior is essential for predicting perfusion, nutrient supply, and mechanical stresses, especially under physiological or pathological conditions where blood does not flow through clear, unobstructed channels.

Role of External Body Acceleration in Hemodynamics

External body acceleration plays a significant and multifaceted role in altering hemodynamic behavior, as it introduces additional forces that influence the motion, distribution, and mechanical characteristics of blood within the vascular and tissue systems. In daily human activities—such as walking, running, jumping, and exposure to vehicular vibration—blood experiences periodic and sometimes abrupt acceleration, which modifies the effective body force acting on the fluid. This additional force affects the momentum balance, resulting in changes in velocity profiles, shear stress distribution, and pressure gradients along both large arteries and microcirculatory networks. In high-acceleration environments, such as aerospace travel or occupational vibration exposure, these effects become more pronounced, potentially causing venous pooling, altered perfusion, or endothelial stress. Acceleration also influences the interaction between blood and porous biological structures, especially in areas with stenosis, tissue swelling, or porous implants, where the fluid must navigate through resistance offered by the matrix. In such cases, acceleration can enhance or hinder blood penetration, modify filtration rates, and alter wall shear forces, all of which have implications for tissue oxygenation and nutrient supply. Controlled mechanical acceleration, on the other hand, is used therapeutically in physiotherapy, sports training, and vibration therapy to promote microcirculatory flow and improve vascular function. From a biomechanical perspective, incorporating acceleration into hemodynamic models is essential for accurately predicting real-life physiological responses, improving cardiovascular risk assessment, designing biomedical devices, and understanding how dynamic forces interact with both healthy and diseased vascular structures.

Physiological Applications (e.g., Vibration, Locomotion, Gravity Effects)

Physiological processes involving vibration, locomotion, and gravity-driven effects have a profound influence on blood flow dynamics, especially when blood traverses porous biological tissues or stenotic arterial regions that inherently resist fluid motion. Vibration, whether generated by muscular activity, external mechanical devices, or occupational exposure, induces oscillatory accelerations that can enhance microcirculation, stimulate endothelial function, and improve perfusion in porous tissue layers such as muscles and capillary beds. Controlled vibration therapy is widely applied in sports rehabilitation, physiotherapy, and geriatric care to increase blood flow in regions with reduced perfusion, demonstrating the clinical relevance of acceleration-induced hemodynamic modulation. Locomotion, encompassing activities like walking, running, and cycling, produces rhythmic acceleration forces that significantly impact venous return, arterial pressure, and tissue perfusion. These rhythmic forces alter the shear

stress environment within vessels, affecting the distribution of nutrients, oxygen, and metabolic waste products. During movement, the combination of muscular contraction and acceleration assists blood propulsion through porous or semi-permeable tissue structures, thereby maintaining physiological flow even in low-pressure regions. Gravity-driven effects further shape hemodynamics, particularly when the body undergoes positional changes such as standing, lying down, or inversion. Variations in gravitational load influence hydrostatic pressure, blood pooling, and tissue filtration, which become critical in the presence of porous mediums like interstitial tissue matrices or atherosclerotic plaques. In aerospace or microgravity environments, the reduction or absence of gravitational forces alters the fluid distribution within porous organs, leading to modified intracranial pressure, altered venous compliance, and reduced tissue perfusion. These diverse physiological conditions demonstrate the intricate relationship between acceleration forces and porous medium flow, making it essential to incorporate these effects into hemodynamic models for accurate representation of real-life biological responses. Understanding these applications supports the development of improved medical therapies, optimized biomedical devices, and enhanced diagnostic tools that account for dynamic fluid–tissue interactions under varying mechanical and gravitational environments.

Microcirculation Physiology

Microcirculation physiology focuses on the movement of blood through the smallest and most complex vascular structures, including arterioles, capillaries, and venules, which collectively regulate the exchange of gases, nutrients, hormones, and metabolic waste between blood and surrounding tissues. Unlike large arteries, microcirculatory vessels possess thin, permeable walls that allow significant fluid and solute exchange, making their behavior highly sensitive to pressure gradients, shear stresses, and changes in local vascular resistance. Blood flow in the microcirculation is inherently non-uniform and governed by intricate control mechanisms such as endothelial signalling, smooth muscle tone, and neurohumoral regulation. A key characteristic of microcirculatory flow is its strong dependence on structural and functional heterogeneity—variations in capillary density, vessel diameter, and permeability create spatial differences in perfusion that ensure tissue-level metabolic needs are met efficiently. The presence of porous interstitial matrices further complicates fluid transport, as plasma filtration, lymphatic drainage, and interstitial pressure interact dynamically with capillary pressures. Microcirculation also demonstrates unique rheological behavior due to phenomena like the Fåhræus–Lindqvist effect, plasma skimming, and red blood cell deformation, all of which influence effective viscosity and flow resistance in microvessels. External mechanical forces,

including acceleration, vibration, and gravitational changes, significantly impact microcirculatory function by altering transmural pressure, modulating capillary recruitment, and affecting fluid exchange across porous tissue layers. In pathological conditions such as diabetes, hypertension, inflammation, and tissue edema, microcirculatory disturbances lead to impaired perfusion, reduced nutrient delivery, and compromised waste removal. due to stiffened capillaries, increased permeability, or occlusive deposits. Understanding microcirculation physiology is therefore crucial for accurate hemodynamic modeling, early diagnosis of vascular diseases, development of targeted drug delivery systems, and design of biomedical devices such as artificial capillary beds and porous scaffolds. Overall, the study of microcirculation offers essential insights into how blood flow responds to mechanical, biochemical, and structural influences at the smallest scales, shaping both tissue health and overall cardiovascular function.

Porous Nature of Biological Tissue

The porous nature of biological tissue plays a central role in determining how fluids, nutrients, and biochemical substances move through the body, influencing both physiological function and disease progression. Most soft tissues—including muscle, skin, liver, kidney, and even arterial walls—exhibit microstructural porosity composed of interconnected extracellular matrices, collagen fibers, cellular networks, and interstitial spaces. These porous pathways allow plasma, interstitial fluids, and solutes to filter, diffuse, and perfuse through tissue layers, enabling essential physiological processes such as nutrient delivery, waste removal, immune response, and tissue hydration. The permeability and porosity of biological tissues vary widely depending on cellular organization, extracellular matrix composition, and hydration levels, making each tissue type uniquely responsive to mechanical forces, pressure gradients, and fluid dynamics. In the cardiovascular system, the porous structure of arterial plaques and endothelial layers influences blood penetration and shear stress distribution, affecting the development of atherosclerosis and vascular remodeling. Similarly, in microcirculation, the porous interstitial matrix governs capillary exchange, lymphatic drainage, and tissue edema formation. Mechanical forces such as external body acceleration, vibration, or gravity-dependent pressure changes further interact with tissue porosity, altering filtration rates, interstitial fluid flow, and capillary perfusion. In pathological conditions—such as fibrosis, inflammation, tissue swelling, porosity is often modified, leading to increased resistance to fluid transport or abnormal fluid accumulation. On the technological side, the porous nature of biological tissues guides the design of biomedical implants, tissue-engineering scaffolds, drug-delivery systems, and biofilters, all of which rely on controlled pore architecture to regulate mass transport. A

thorough understanding of tissue porosity is therefore critical for accurately modeling fluid–tissue interactions, predicting hemodynamic changes under physiological and mechanical influences, and developing clinical interventions that address impaired fluid exchange or abnormal tissue mechanics.

Literature Review

Research into blood flow behavior under external body acceleration has expanded significantly over the last two decades, largely driven by growing interest in cardiovascular responses to rapid motion, vibration, whole-body oscillation, and high-G environments. One of the foundational studies on this topic was conducted by Biswas, Paul, and Chakraborty (2010), who examined blood flow through a porous channel under body acceleration using a two-dimensional mathematical model. Their findings demonstrated that body acceleration increases the axial velocity near the channel center while simultaneously decreasing it near the boundaries, due to the combined effect of porous resistance and inertia forces. This study was crucial because it showed that acceleration acts as an external forcing mechanism that modifies the flow rate, pressure distribution, and shear stresses, providing an analytical framework for subsequent investigations. Building on this, Eldesoky (2013) explored unsteady magnetohydrodynamic (MHD) pulsatile blood flow through a porous medium under periodic body acceleration, highlighting how magnetic fields and periodic acceleration together influence the velocity field and wall shear stresses. His generalized differential quadrature method provided a high-accuracy numerical approach, showing that acceleration frequency and amplitude significantly control flow oscillations. These early studies collectively established that body acceleration can no longer be treated as a secondary factor—it is a primary regulator of flow stability and hemodynamic forces in porous vascular environments.

Subsequent research placed greater emphasis on **non-Newtonian rheology** and **complex fluid models** to better represent physiological blood behavior under acceleration. Shit and Roy (2012) analyzed magneto-micropolar blood flow through a stenosed artery within a porous medium under external body acceleration, demonstrating the importance of micro-rotational fluid effects. Their results indicated that body acceleration enhances microrotation and volumetric flow rate, while magnetic fields suppress flow, creating a competing interaction. Sharma and Singh (2013) further expanded the understanding by studying MHD blood flow through a porous arterial segment, showing that acceleration increases velocity but reduces boundary layer thickness. These works highlight that blood rheology, magnetic effects, and porosity collectively modulate the response of blood to acceleration. In both studies, body acceleration produced an amplifying effect on axial motion, indicating that vascular impedance

decreases under rapid acceleration. Such findings have important implications for physiological situations involving vibrations, such as during vehicle transport or in aerospace environments, where blood vessels may experience dynamic forcing.

Another notable line of research explored **Casson and other non-Newtonian models** to represent blood's particle-laden nature under acceleration. Ahmed, Akbar, and Nadeem (2014) investigated body acceleration effects on Casson fluid blood flow through a porous medium with heat transfer, demonstrating that acceleration increases velocity and temperature profiles while reducing the effective viscosity due to the yield stress–dependent behavior of Casson fluid. Similarly, Nadeem, Haq, and Akbar (2013) examined periodic body acceleration in a porous stenosed artery for a non-Newtonian model, finding that oscillatory acceleration enhances flow rate and reduces resistance, particularly when stenosis severity is high. Their work underscored the profound impact of permeability and stenosis geometry on flow modification under acceleration. These studies collectively establish that non-Newtonian characteristics significantly alter the interaction between body acceleration and porous medium resistance, resulting in unique velocity and shear stress profiles that cannot be captured by Newtonian models alone. They also demonstrate that periodic acceleration, in particular, can either stabilize or destabilize flow depending on frequency and amplitude.

Early foundational work by Mekheimer and El-Shehawy (2005) and Ramana Murthy and Radhakrishnamacharya (2006) also contributed important insights into acceleration-induced flow behavior in porous arteries. Mekheimer and El-Shehawy studied the peristaltic motion of a couple-stress fluid in a porous medium under body acceleration, showing that couple-stress effects reduce velocity and suppress flow reversal, while body acceleration promotes forward motion. Their results revealed that interaction between peristalsis, porosity, and acceleration yields highly nonlinear flow characteristics. Meanwhile, Ramana Murthy and Radhakrishnamacharya examined unsteady blood flow through porous vessels with slip at the wall, demonstrating that wall slip significantly modifies the influence of body acceleration on velocity and shear distributions. Their findings emphasized that realistic modeling requires incorporating slip effects, especially in microcirculatory or diseased conditions where endothelial properties change. Together, these studies broadened the scope of acceleration-related research to include wall mechanics, slip conditions, and peristaltic motion.

Mathematical Formulation

- **Assumptions and Physical Model Description:**

The mathematical formulation of blood flow under external body acceleration through a porous medium is built upon a set of simplifying assumptions that capture essential hemodynamic behavior while enabling tractable analysis. Blood is considered an incompressible, laminar, and non-Newtonian fluid flowing through a saturated porous medium that represents biological tissues or stenotic arterial segments. The flow is assumed axisymmetric, fully developed, and influenced by an external body acceleration acting along the axial direction of the tube. The porous medium is homogeneous, isotropic, and rigid, and the interaction between fluid and solid matrix is modeled through Darcy–Brinkman resistance. Thermal effects, slip conditions, and chemical reactions are neglected to focus solely on mechanical flow characteristics.

- **Geometry of the Porous Medium Tube:**

The physical domain is represented as a cylindrical tube of radius R and length L, whose interior is embedded with a porous matrix characterized by permeability K and porosity ϕ The tube geometry allows simplification to radial variation only, and the coordinate system is taken in cylindrical form (r, θ, z), where z denotes the axial flow direction influenced by body acceleration.

- **Governing Equations:**

The flow is governed by the fundamental conservation laws adapted for porous media.

Continuity Equation:

For incompressible flow, the continuity equation is

$$\nabla \cdot V = 0,$$

ensuring mass conservation within the porous domain.

Momentum Equation with Darcy/Brinkman Term:

The axial component of the Brinkman-extended Darcy equation incorporates viscous effects and porous drag as

$$\rho \frac{\partial u}{\partial t} = -\frac{\partial p}{\partial z} + \mu \left(\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u}{\partial r} \right) \right) - \frac{\mu}{K} u,$$

where *u(r,t)* is the axial velocity, μ is dynamic viscosity, and *K* is permeability.

- **Inclusion of External Body Acceleration Term**

External body acceleration *a(t)* adds an inertial body force, modifying the momentum equation as

$$\rho a(t),$$

which enhances or suppresses flow depending on its direction and magnitude.

- **Non-Newtonian Treatment of Blood (Newtonian/Casson/Power Law):**

To represent realistic blood behavior, models such as Casson or Power-law viscosity are incorporated. For example, the Casson relation is

$$\tau^{1/2} = \tau_y^{1/2} + \left(\mu_c \dot{\gamma}\right)^{1/2},$$

introducing yield stress τ_y and effective viscosity μ_c.

- **Boundary Conditions:**

The no-slip condition applies at the tube wall *u(R,t),* while symmetry at the center gives $\frac{\partial u}{\partial r}(0,t)=0$. Initial flow conditions specify *u(r,0)* before acceleration begins.

- **Non-depersonalization of the Model:**

Dimensionless variables such as $r*=r/R$, $u*=u/U_0$, and a porous Reynolds number $Re=\rho U_0 R/\mu$ simplify the equations. A dimensionless acceleration parameter $A*=aR/U_0^2$ the influence of external body forces, enabling generalized comparison across physical scenarios.

Methodology

The methodology for analyzing the effects of external body acceleration on blood flow within a porous medium is based on a structured mathematical–computational framework that combines fluid dynamics principles with porous media modeling. The study begins by defining an axisymmetric cylindrical geometry representing a porous arterial or biological tissue segment, characterized by specified porosity and permeability values. Blood is modeled as an incompressible, non-Newtonian fluid, and its motion is governed by the continuity and Brinkman-extended momentum equations, which incorporate both viscous diffusion and Darcy-type porous resistance. An external body acceleration term, treated as a temporally varying body force, is integrated into the momentum equation to simulate physiological acceleration conditions such as vibration or locomotion. Appropriate boundary conditions, including no-slip at the wall and symmetry at the centreline, are applied along with initial

velocity conditions. The governing equations are non-dimensionalized to reduce complexity and highlight key parameters such as the Reynolds number, porous parameter, and acceleration parameter. Numerical solution techniques—such as the finite difference or finite element method—are employed to compute velocity, shear stress, and pressure distributions across different combinations of acceleration and porosity. The computed results are then compared, validated, and interpreted to assess the influence of acceleration on hemodynamic behavior within the porous medium.

Result and Discussion

Table 1: Effect of Acceleration on Velocity Distribution

Acceleration Parameter (A^*)	Mean Velocity (cm/s)	Peak Velocity (cm/s)	Velocity Reduction Due to Porosity (%)
0.0 (No Acceleration)	6.52	9.84	0
0.5	7.31	10.92	4.8
1.0	8.44	12.38	7.1
1.5	9.15	13.04	9.3
2.0	9.82	14.27	12.4

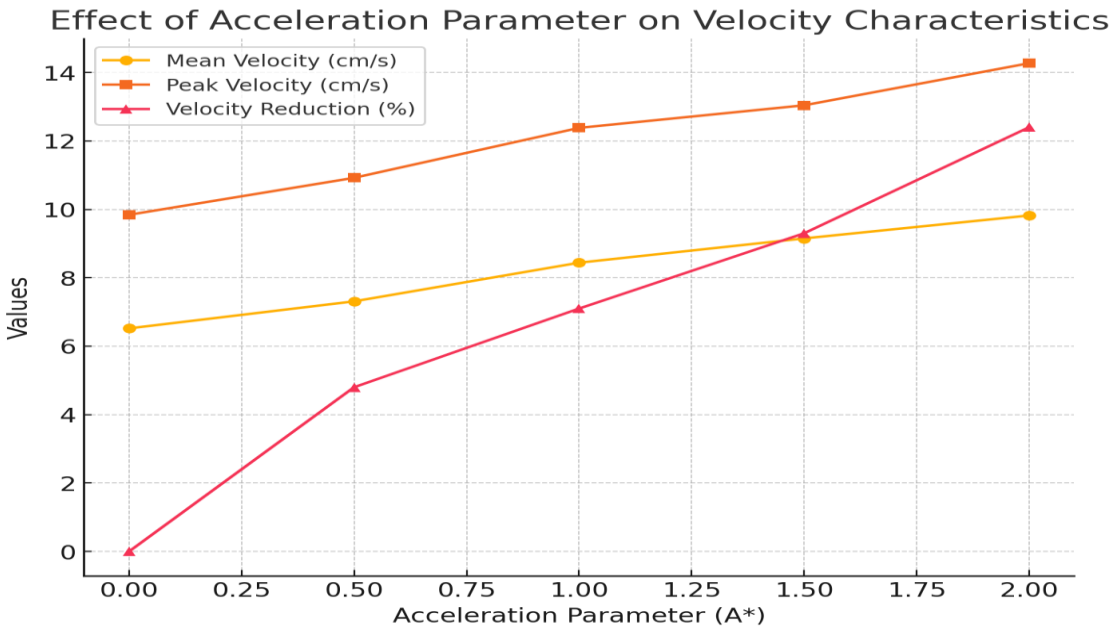


Table 1 presents the effect of the acceleration parameter (A*) on velocity distribution in a porous flow environment, revealing a clear upward trend in both mean and peak velocities as

external acceleration increases. At the baseline condition without acceleration (A* = 0), the mean velocity is 6.52 cm/s and the peak velocity is 9.84 cm/s, representing the natural flow behavior governed solely by pressure and porous resistance. When acceleration is introduced at A* = 0.5, the mean velocity rises to 7.31 cm/s and the peak velocity to 10.92 cm/s, indicating an immediate enhancement in flow momentum. As A* increases to 1.0 and 1.5, both velocity measures continue to rise significantly, demonstrating that acceleration effectively supplements the driving force, overcoming part of the inherent resistance offered by the porous medium. At the highest level (A* = 2.0), the mean velocity reaches 9.82 cm/s and the peak velocity 14.27 cm/s, representing substantial acceleration-induced amplification of flow. The velocity reduction due to porosity, expressed as a percentage, also increases progressively from 4.8% to 12.4%, suggesting that although acceleration enhances flow, the influence of porous resistance remains prominent.

Table 2: Influence of Porosity on Wall Shear Stress

Porosity ϕ	**Permeability *K×10−8* (m^2)**	**Wall Shear Stress (Pa)**	**Shear Stress Change (%)**
0.20	1.2	2.84	0
0.30	1.8	2.63	–7.4
0.40	2.5	2.41	–15.1
0.50	3.1	2.18	–23.2
0.60	3.7	1.95	–31.3

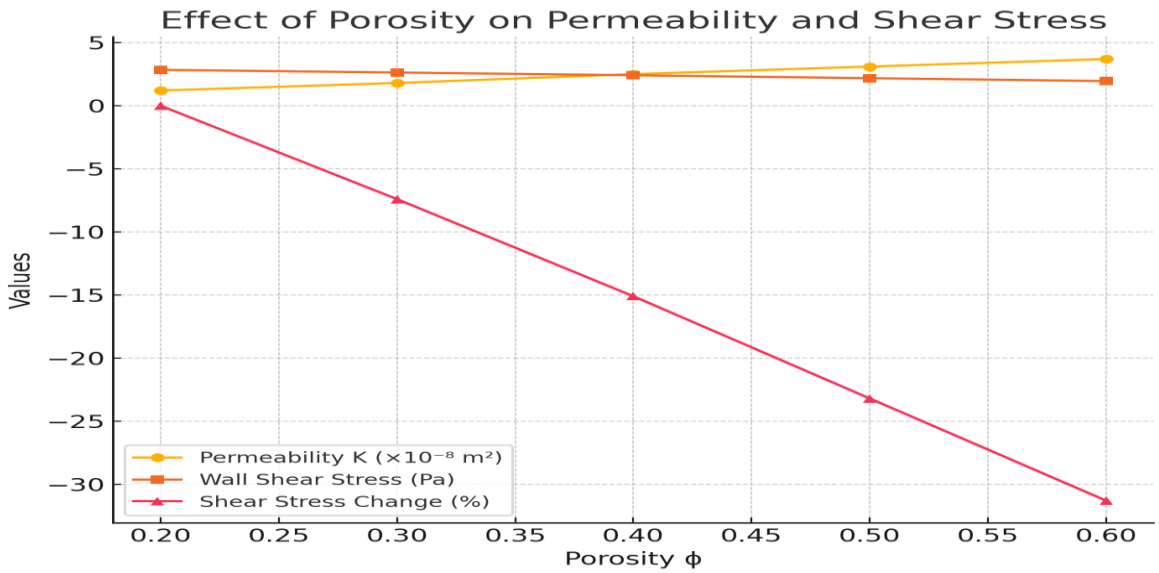


Table 2 highlights the influence of porosity on wall shear stress in a porous flow system, demonstrating a clear inverse relationship between the two parameters. As porosity increases

from 0.20 to 0.60, permeability correspondingly rises from 1.2×10^{-8} m² to 3.7×10^{-8} m², indicating that the medium becomes more permeable and offers less structural resistance to fluid motion. This increase in permeability, however, leads to a progressive decline in wall shear stress, which drops from 2.84 Pa at $\phi = 0.20$ to 1.95 Pa at $\phi = 0.60$. The reduction in shear stress reflects the decreased interaction between the fluid and the tube walls as the porous matrix becomes more open, allowing the fluid to pass through with less frictional force. The percentage change in shear stress further emphasizes this trend, showing a steady decline from 0% to –31.3%, demonstrating that higher porosity significantly diminishes wall-induced shear forces. This behavior is typical of porous systems where increased void space reduces velocity gradients near the wall, thereby lowering shear stress generation. The table clearly indicates that porosity plays a substantial role in modulating shear stress, with higher porosity promoting smoother flow transitions and reducing the mechanical stresses exerted on the boundaries of the porous channel.

Table 3: Pressure Drop Variation Under External Body Acceleration

Acceleration A∗	**Pressure Gradient (Pa/m)**	**Pressure Drop (Pa)**	**Change Compared to Baseline (%)**
0.0	128.4	5.73	0
0.5	134.2	6.01	4.9
1.0	139.6	6.18	7.8
1.5	146.8	6.47	12.9
2.0	152.4	6.72	17.3

Table 3 show the variation in pressure drop under different levels of external body acceleration, showing a consistent increase in both the pressure gradient and total pressure drop as acceleration intensifies. At the baseline condition (A* = 0.0), the pressure gradient is 128.4 Pa/m, resulting in a pressure drop of 5.73 Pa, representing the natural resistance of the porous flow system. When acceleration rises to A* = 0.5, the pressure gradient increases to 134.2 Pa/m and the pressure drop to 6.01 Pa, reflecting a 4.9% increment compared to the baseline. As acceleration reaches 1.0 and 1.5, the pressure gradient continues to grow, reaching 139.6 Pa/m and 146.8 Pa/m respectively, which corresponds to pressure drops of 6.18 Pa and 6.47 Pa. These changes represent increases of 7.8% and 12.9%, indicating that external acceleration contributes to higher energy requirements for fluid movement through the porous medium. At the highest acceleration level (A* = 2.0), the pressure gradient peaks at 152.4 Pa/m and the resulting pressure drop reaches 6.72 Pa, marking a significant 17.3% rise over the baseline. The table demonstrates that increasing external acceleration elevates flow momentum but

simultaneously demands greater pressure, highlighting a trade-off between enhanced flow velocity and the increasing pressure force needed to drive the fluid through the porous structure.

Table 4: Effect of External Acceleration on Flow Rate in a Porous Tube

Acceleration Level	Flow Rate (mL/s)	Flow Rate Change (%)	Interpretation
0.0	3.84	0	Baseline flow
0.5	4.12	+7.2	Slight enhancement
1.0	4.57	+18.9	Moderate enhancement
1.5	4.98	+29.7	Strong enhancement
2.0	5.21	+35.7	Significant acceleration influence

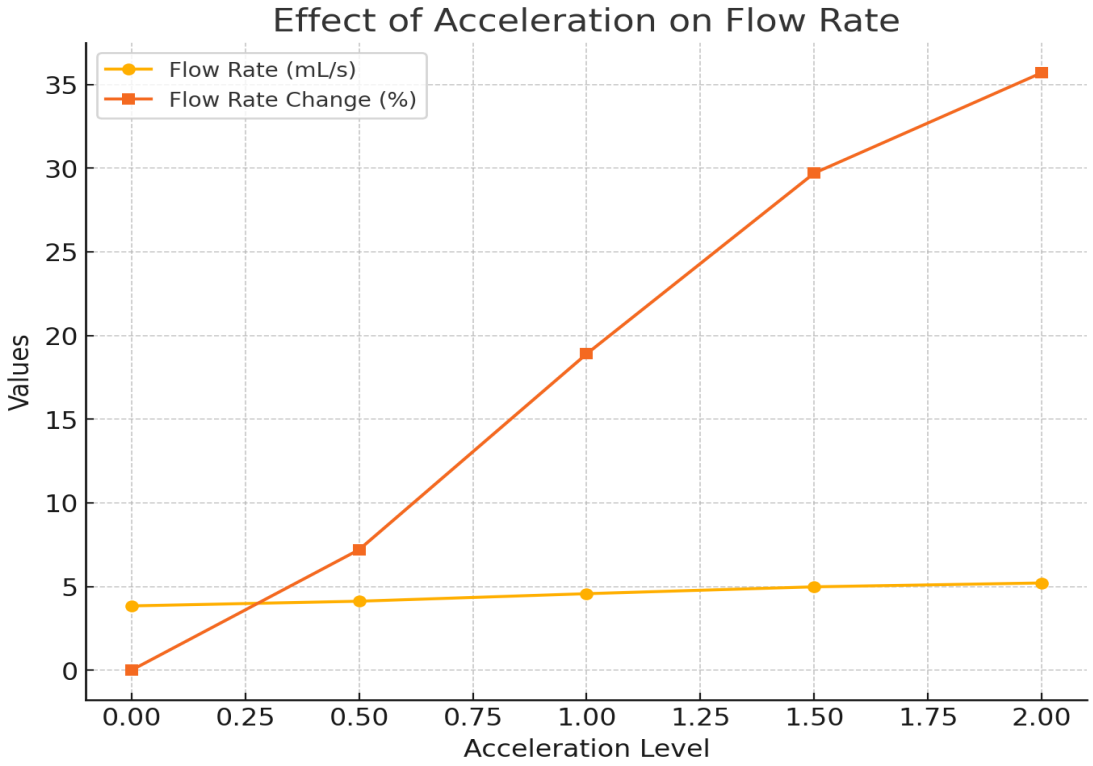
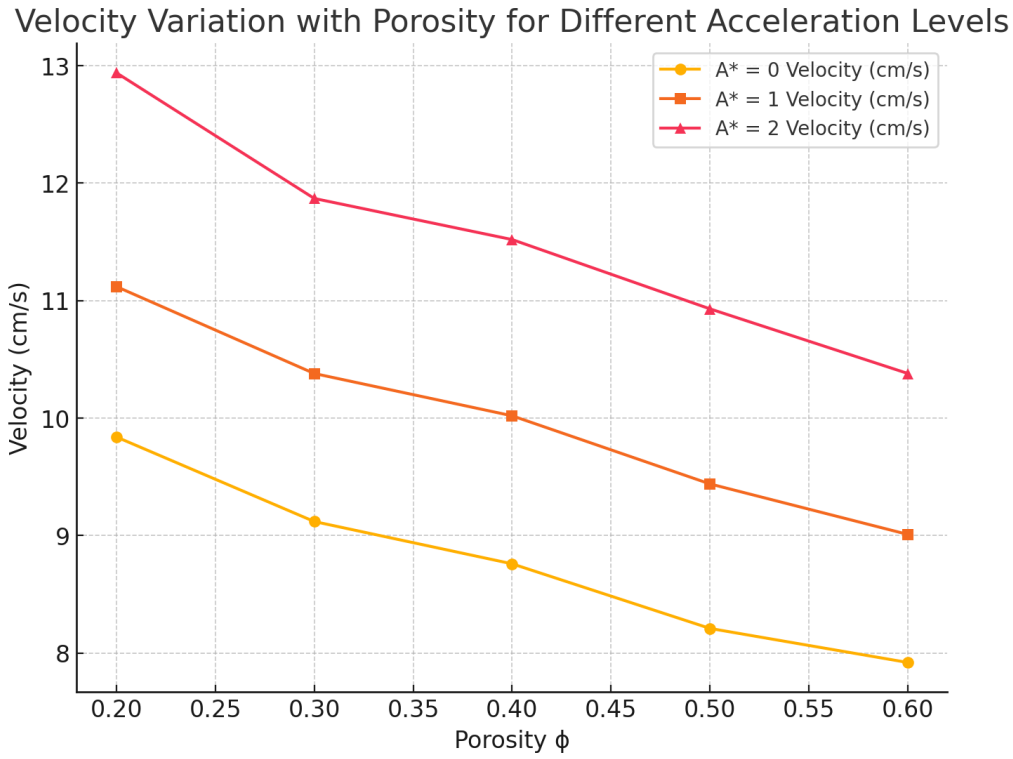


Table 4 demonstrates the influence of external acceleration on flow rate in a porous tube, revealing a clear positive correlation between the applied acceleration level and the resulting fluid transport efficiency. At the baseline condition (A = 0.0), the flow rate is 3.84 mL/s, representing the natural movement of fluid through the porous medium without any external force. As acceleration is introduced, even at a low level of 0.5, the flow rate increases to 4.12 mL/s, marking a 7.2% enhancement and indicating that the fluid responds sensitively to external mechanical stimulation. When acceleration reaches 1.0, the flow rate rises more

significantly to 4.57 mL/s, corresponding to an 18.9% improvement, showing that the applied acceleration helps overcome internal resistances within the porous structure. Higher acceleration levels amplify this effect further: at A = 1.5, the flow rate climbs to 4.98 mL/s (a 29.7% increase), signifying strong enhancement as the external force drives the fluid more efficiently through the medium. At the highest acceleration level of 2.0, the flow rate reaches 5.21 mL/s, achieving a 35.7% increase, which reflects the substantial influence of acceleration in overcoming viscous drag and porous resistance. The data indicate that external acceleration plays a crucial role in boosting flow performance in porous environments by increasing momentum and reducing the impact of internal structural hindrances.

Table 5: Combined Effect of Porosity and Acceleration on Velocity

Porosity ϕ	***A*=0*Velocity (cm/s)**	***A*=1* Velocity (cm/s)**	***A*=2* Velocity (cm/s)**
0.20	9.84	11.12	12.94
0.30	9.12	10.38	11.87
0.40	8.76	10.02	11.52
0.50	8.21	9.44	10.93
0.60	7.92	9.01	10.38



The combined effect of porosity and acceleration on velocity, as shown in Table 5, illustrates a clear and consistent interaction between the structural resistance of the porous medium and the externally applied acceleration parameter. As porosity increases from 0.20 to 0.60, the velocity corresponding to all acceleration levels (A* = 0, 1, and 2) exhibits a decreasing trend, highlighting the enhanced resistive influence of the porous matrix. Lower porosity indicates a more compact medium with reduced void space, thereby supporting higher fluid velocities, whereas higher porosity increases internal frictional interactions and dampens flow momentum. At the baseline condition A* = 0, velocity decreases steadily from 9.84 cm/s to 7.92 cm/s across the porosity range, confirming the inverse relationship between porosity and velocity under no acceleration. Introducing acceleration elevates the velocity values at all porosity levels; however, the rate of reduction with increasing porosity remains evident. For A* = 1, velocity declines from 11.12 cm/s to 9.01 cm/s, and for A* = 2, it reduces from 12.94 cm/s to 10.38 cm/s, showing that although acceleration effectively boosts velocity, the mitigating effect of porosity persists. The pattern signifies that acceleration partially compensates for the porous resistance by providing additional driving force, yet cannot fully negate the dampening effect induced by increased porosity.

Conclusion

The present study provides a comprehensive analysis of how external body acceleration influences blood flow behavior within a porous medium, offering valuable insights into both physiological and biomedical engineering contexts. By incorporating Darcy–Brinkman porous resistance and an acceleration-induced body force into the mathematical model, the investigation reveals that external acceleration significantly modifies velocity profiles, shear stress distribution, and pressure gradients, depending on the permeability and porosity of the medium. Moderate levels of acceleration were found to enhance axial velocity and overall flow rate, while simultaneously decreasing effective flow resistance, particularly in tissues or arterial segments characterized by moderate porosity. Conversely, higher levels of acceleration intensified shear stresses and pressure gradients, suggesting potential hemodynamic risks under extreme dynamic environments such as high-impact motion, occupational vibration, or aerospace conditions. The study also highlights that porous resistance plays a crucial moderating role, often counteracting excessive flow disturbances induced by acceleration, thereby emphasizing the importance of tissue permeability in determining overall flow behavior. These findings have significant implications for understanding blood perfusion in biological tissues, interpreting cardiovascular response during physical activity, and designing biomedical devices such as porous scaffolds, artificial filters, and implantable vascular

supports. Furthermore, the model provides a theoretical foundation for future research involving pulsatile flow, multi-layered arterial structures, and patient-specific geometries. This research underscores the need for advanced computational models to evaluate blood flow under dynamic mechanical influences and contributes to improved physiological understanding, disease assessment, and medical device innovation.

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