



Viscoelastic Coupling of Pulsatile Wall Shear Stress and Endothelial Glycocalyx Mechanotransduction: A Non-Equilibrium Hydrodynamic Model for Microvascular Resilience

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Abstract

The endothelial glycocalyx layer (EGL) functions as the primary mechanosensitive interface modulating hemodynamic forces at the vascular wall. Under physiological pulsatile flow, the transmission of wall shear stress (WSS) to the underlying endothelial cytoskeleton is mediated by the viscoelastic behavior of the EGL porous meshwork. We present a non-equilibrium hydrodynamic framework coupling Brinkman-extended momentum transport with a fractional viscoelastic constitutive model of the EGL core proteoglycans. By solving the coupled fluid-porous matrix equations under dynamic, time-harmonic pressure gradients, we quantify the frequency-dependent attenuation and phase lag of shear forces transmitted to the plasma membrane.

Our analytical and numerical solutions reveal that the non-equilibrium viscoelastic relaxation of the glycocalyx acts as a low-pass biophysical filter, shielding endothelial membrane domains from peak high-frequency oscillatory stresses while sustaining baseline mechanotransductive signaling (e.g., eNOS phosphorylation at Ser1177). Integrating Shannon channel capacity and thermodynamic noise reduction principles from the Science. 4.0 framework, we demonstrate that preserving the structural elasticity and thickness of the EGL minimizes viscous energy dissipation (E_{diss}) and prevents non-linear transitions toward endothelial dysfunction. Degradation of the EGL alters this hydrodynamic damping, shifting the mechanotransduction regime toward unbuffered mechanical fatigue, microvascular stasis, and systemic barrier failure.

Keywords: Endothelial Glycocalyx Layer (EGL); Wall Shear Stress (WSS); Womersley Number; Mechanotransduction; Syndecan-1; Non-Newtonian Hemodynamics; Brinkman Porous Flow; Endothelial Nitric Oxide Synthase (eNOS); Science 4.0; Signal-to-Noise Ratio (SNR)

Introduction: The Luminal Biomechanical Boundary

The microvascular endothelium is continuously subjected to dynamic hemodynamic forces, primarily hydrostatic pressure gradients and pulsatile wall shear stress (WSS) [1,2]. While steady laminar shear stress promotes an anti-inflammatory, quiescent endothelial phenotype, disturbed or high-amplitude oscillatory shear stress induces endothelial dysfunction, increased barrier permeability, and

inflammatory pathway activation [2,3].

Classical hemodynamic models treat the vascular wall as a rigid, impermeable boundary satisfying standard no-slip conditions ($u = 0$) [1]. However, positioned directly at the luminal interface, the endothelial glycocalyx layer (EGL) a dense macromolecular meshwork composed of syndecans, glypicans, glycosaminoglycan chains (heparan sulfate, chondroitin sulfate), and bound plasma proteins—serves as the primary transducer and damper of hemodynamic shear

forces [2,4]. Rather than behaving as a static porous coat, the EGL displays pronounced non-equilibrium viscoelastic characteristics [3,5]. Under cyclic pulsatile loading, energy dissipation and mechanical compliance within this brush-like matrix dynamically alter the stress profile transferred to the apical plasma membrane [6,7].

In modern biophysical systems engineering and the Science 4.0 paradigm, living interfaces must maintain optimal Signal-to-Noise Ratios (SNR) to prevent biological thermal throttling and structural wear [8,9]. When environmental or oxidative stress compromises the glycocalyx, microvascular resistance (R_{vascular}) rises sharply, triggering downstream fluidic bottlenecks and cellular decoherence [10,11]. Here, we construct a rigorous hydrodynamic model of pulsatile blood flow through an EGL-lined microchannel to determine how viscoelastic relaxation governs microvascular resilience and mechanotransductive stability [1,12].

Theoretical and Mathematical Framework

Domain Decomposition and Governing Equations

We consider an axisymmetric microvessel of radius R . The domain is divided into two distinct hydrodynamic regions [1,2]:

The Luminal Core ($0 \leq r < R - h$): A free-fluid region governed by the unsteady Navier-Stokes equations for an incompressible fluid [1].

The Glycocalyx Layer ($R - h \leq r \leq R$): A porous, deformable viscoelastic layer of nominal thickness $h \ll R$ [2,4].

Microvascular Hydrodynamic and Poroelastic Domain Boundaries:

$r = 0$ (Centerline) : Luminal Core Flow (Navier-Stokes Domain)

$r = R - h$ (Interface) : Interfacial Fluid-Porous Boundary (Beavers-Joseph-Saffman condition)

$R - h \leq r \leq R$: Endothelial Glycocalyx Layer (Brinkman Porous Domain)

$r = R$ (Wall) : Apical Endothelial Membrane & Syndecan Anchors

In the luminal core, momentum transport for an incompressible fluid with dynamic viscosity μ and density ρ is described by [1]:

$$\rho \cdot (\partial \mathbf{u}_f / \partial t) = -\nabla p + \mu \cdot \nabla^2 \mathbf{u}_f$$

Within the EGL, the flow is modeled using the Brinkman equation extended for porous media, accounting for Darcy drag exerted by the core protein fibers [2,4]:

$\rho \cdot (\partial \mathbf{u}_g / \partial t) = -\nabla p + \mu_e \cdot \nabla^2 \mathbf{u}_g - (\mu / K_p) \cdot \mathbf{u}_g$ Where: μ_e is the effective Brinkman viscosity within the porous layer.

K_p denotes the intrinsic hydrodynamic permeability of the glycocalyx meshwork [2], modeled via Davies' relation:

$K_p = (r_f)^2 / [16 \cdot (\phi_f)^{1.5} \cdot (1 + 56 \cdot (\phi_f)^3)]$ (with r_f being the fiber radius and ϕ_f the solid volume fraction).

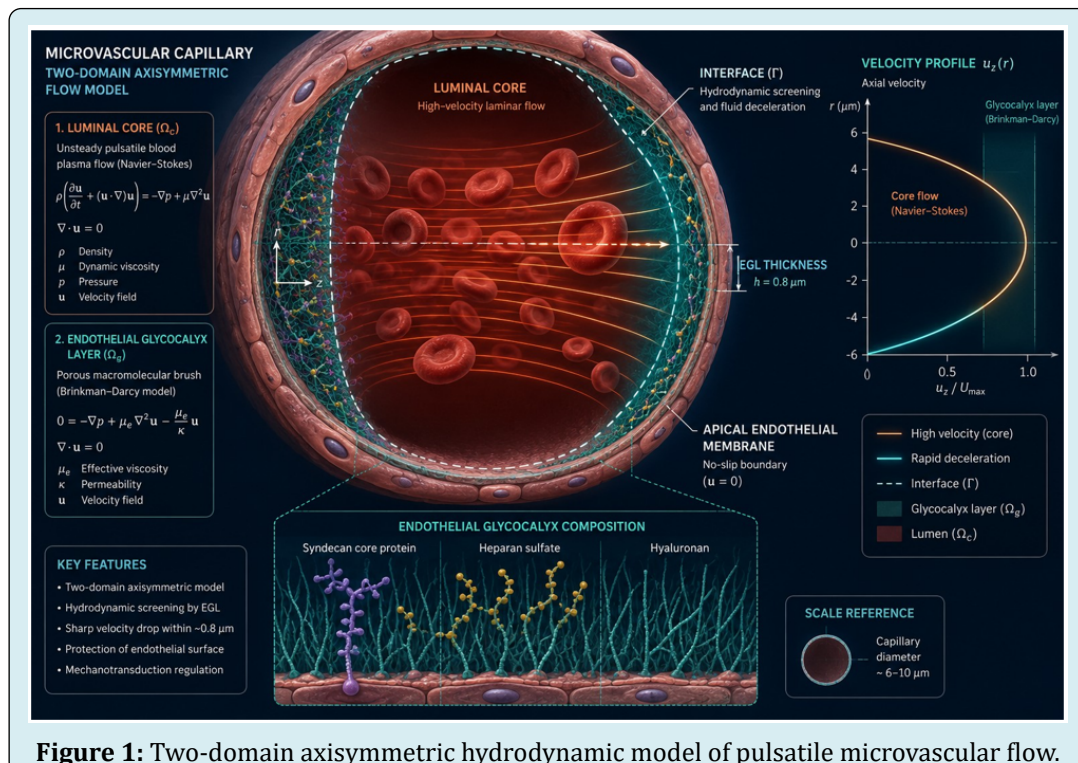


Figure 1: Two-domain axisymmetric hydrodynamic model of pulsatile microvascular flow.

Cross-sectional schematic of a microvascular capillary (~ 6-10 μm diameter) detailing the physical coupling between the free luminal core (Ω_c , Navier-Stokes regime) and the porous endothelial glycocalyx layer (Ω_g , Brinkman-Darcy regime, $h = 0.8 \mu\text{m}$). The axial velocity profile $u_z(r)$ illustrates the hydrodynamic screening effect at the fluid-porous interface (Γ), producing a sharp velocity drop within the macromolecular meshwork (syndecan core proteins, heparan sulfate, hyaluronan) to establish a strict no-slip condition ($u = 0$) at the apical endothelial membrane and shield it from peak oscillatory shear forces.

Viscoelastic Fiber Deformation and Fractional Damping

The dynamic deformation field $d(r, z, t)$ of the core proteoglycans (syndecans and glypicans) is coupled to the local fluid velocity u_g through hydrodynamic drag [3,5]:

$$\rho_s \cdot (\partial^2 d / \partial t^2) = \nabla \cdot \sigma_s + f_{\text{drag}}$$

The solid stress tensor σ_s obeys a fractional viscoelastic constitutive law capturing the broad relaxation spectrum of hydrated biopolymer networks [5,6]:

$\sigma_s(t) = G_0 \cdot \varepsilon(t) + G_1 \cdot (\tau_\alpha)^{-\alpha} \cdot D_t^{-\alpha} [\varepsilon(t)]$ Where: $D_t^{-\alpha}$ denotes the Caputo fractional derivative of order $0 < \alpha \leq 1$ [5].

τ_α is the characteristic stress relaxation time.

$\varepsilon = 0.5 \cdot [\nabla d + (\nabla d)^T]$ is the infinitesimal strain tensor.

G_0 and G_1 represent the static and dynamic shear moduli ($G_0 \approx 10 - 50 \text{ Pa}$) [3,5].

Boundary Conditions and Interfacial Coupling

Centerline Symmetry ($r = 0$) [1]:

$$(\partial u_{f,z} / \partial r)|_{(r=0)} = 0$$

Fluid-Glycocalyx Interface ($r = R - h$) [2,4]:

Velocity continuity and stress balance governed by the Beavers-Joseph-Saffman condition:

$$u_f(R - h, t) = u_g(R - h, t)$$

$$\mu \cdot (\partial u_{f,z} / \partial r)|_{(r=(R-h)^-)} = \mu_e \cdot (\partial u_{g,z} / \partial r)|_{(r=(R-h)^+)} + \beta \cdot (\mu / \sqrt{K_p}) \cdot [u_{f,z}(R - h, t) - u_{g,z}(R - h, t)]$$

(where β is the dimensionless Beavers-Joseph slip coefficient [4]).

Apical Membrane Wall ($r = R$) [2,7]:

No-slip condition for the interstitial fluid and solid anchor displacement constraint: $u_g(R, t) = 0$
 $d(R, t) = 0$

Analytical Solution and Viscous Dissipation Closure

Velocity Field Decomposition

Under a harmonic axial pressure gradient $-(\partial p / \partial z) = P_0 + P_\omega \cdot \exp(i \cdot \omega \cdot t)$, the velocity fields in both regions

split into steady and oscillatory components [1,6]:

$$u_f(r, t) = u_{f0}(r) + \tilde{u}_f(r) \cdot \exp(i \cdot \omega \cdot t) \quad u_g(r, t) = u_{g0}(r) + \tilde{u}_g(r) \cdot \exp(i \cdot \omega \cdot t)$$

In the luminal core ($0 \leq r < R - h$), the oscillatory velocity y component is expressed in terms of the standard zero-order Bessel function [1]:

$$\tilde{u}_f(r) = C \cdot J_0(i^{1.5} \cdot \alpha_W \cdot r / R) + [P_\omega / (i \cdot \omega \cdot \rho)]$$

where $\alpha_W = R \cdot \sqrt{(\omega \cdot \rho / \mu)}$ is the dimensionless Womersley number.

In the porous EGL region ($R - h \leq r \leq R$), the oscillatory velocity is governed by modified Bessel functions of order zero [4,6]:

$$\tilde{u}_g(r) = A \cdot I_0(\lambda_g \cdot r) + B \cdot K_0(\lambda_g \cdot r) + [P_\omega / (\mu_e \cdot (\lambda_g)^2)]$$

where the hydrodynamic screening parameter λ_g is given by:

$$\lambda_g = \sqrt{[(1 / K_p) + (i \cdot \omega \cdot \rho / \mu_e)]}$$

Algebraic System for Integration Constants (A, B, C)

Applying the three boundary conditions at $r = 0$, $r = R - h$, and $r = R$ yields a closed linear system $M \cdot X = Y$, where $X = [A, B, C]^T$:

Velocity continuity at $r = R - h$:

$$A \cdot I_0(\lambda_g \cdot (R - h)) + B \cdot K_0(\lambda_g \cdot (R - h)) - C \cdot J_0(i^{1.5} \cdot \alpha_W \cdot (R - h) / R) = [P_\omega / (i \cdot \omega \cdot \rho)] - [P_\omega / (\mu_e \cdot (\lambda_g)^2)]$$

Interfacial shear stress matching (Beavers-Joseph condition at $r = R - h$):

$$\mu_e \cdot \lambda_g \cdot [A \cdot I_1(\lambda_g \cdot (R - h)) - B \cdot K_1(\lambda_g \cdot (R - h))] + (\mu \cdot i^{1.5} \cdot \alpha_W / R) \cdot C \cdot J_1(i^{1.5} \cdot \alpha_W \cdot (R - h) / R) = 0$$

Apical membrane no-slip at $r = R$:

$$A \cdot I_0(\lambda_g \cdot R) + B \cdot K_0(\lambda_g \cdot R) = -[P_\omega / (\mu_e \cdot (\lambda_g)^2)]$$

Wall Shear Stress and Viscous Energy Dissipation

The total dynamic wall shear stress transmitted to the endothelial apical surface at $r = R$ is: $\tau_w(t) = \mu_e \cdot (\partial u_g / \partial r)|_{(r=R)} + [e_r \cdot \sigma_s \cdot e_z]|_{(r=R)}$

Evaluating the real and imaginary components yields the dynamic phase lag $\theta(\omega)$ between macrovascular pulsatile driving pressure and localized membrane stress [6,8]:

$$\theta(\omega) = \arctan(\text{Im}[\tau_w(\omega)] / \text{Re}[\tau_w(\omega)])$$

The cycle-averaged rate of viscous energy dissipation (E_{diss}) per unit length of vessel is defined by the volume integral of fluid shear, porous drag, and solid damping:

$$E_{\text{diss}} = (1 / T) \cdot \int_0^T [2\pi \cdot \int_0^{(R-h)} \mu \cdot (\partial u_f / \partial r)^2 \cdot r \, dr + 2\pi \cdot \int_{(R-h)}^R (\mu_e \cdot (\partial u_g / \partial r)^2 + (\mu / K_p) \cdot (u_g)^2 + \sigma_s : (\partial \varepsilon / \partial t)) \cdot r \, dr] \, dt$$

Physiological Parameters and Numerical Benchmarking

The baseline biophysical and geometrical values

for typical microarterioles and capillary networks are summarized in Table 1.

Parameter	Symbol	Physiological Baseline Value	Physical Dimension / Unit
Microvessel Lumen Radius	R	10.0 - 25.0	μm
Intact Glycocalyx Thickness	h	0.5 - 1.0	μm
Core Protein Fiber Radius	r_f	5.0 - 6.0	nm
Glycocalyx Solid Volume Fraction	ϕ_f	0.02 - 0.05	Dimensionless
Intrinsic Porous Permeability	K_p	1.0×10^{-16} to 1.0×10^{-15}	m^2
Dynamic Fluid Viscosity (Plasma)	μ	1.2×10^{-3}	$\text{Pa} \cdot \text{s}$
Effective Brinkman Viscosity	μ_e	1.2×10^{-3} to 1.5×10^{-3}	$\text{Pa} \cdot \text{s}$
Fluid Density	ρ	1050	kg / m^3
Static Shear Modulus of Scaffold	G_0	20.0 - 40.0	Pa
Fractional Derivative Order	α	0.45 - 0.65	Dimensionless
Characteristic Relaxation Time	τ_α	0.1 - 0.5	s
Beavers-Joseph Slip Parameter	β	0.15 - 0.35	Dimensionless

Table 1: Biophysical and Geometrical Parameters of the EGL Model.

Mechanotransduction Dynamics and Cellular Signal Fidelity

Cybernetic Transduction Cascade (Science 4.0):

Pulsatile WSS Wave (τ_w) ---> Viscoelastic EGL

Dispersion (Caputo Damping) ---> Syndecan Bending Torque (M_0) ---> Cortical Actin Cytoskeleton Tension ---> Caveolae & eNOS Phosphorylation (Ser1177) ---> Steady-State NO Production

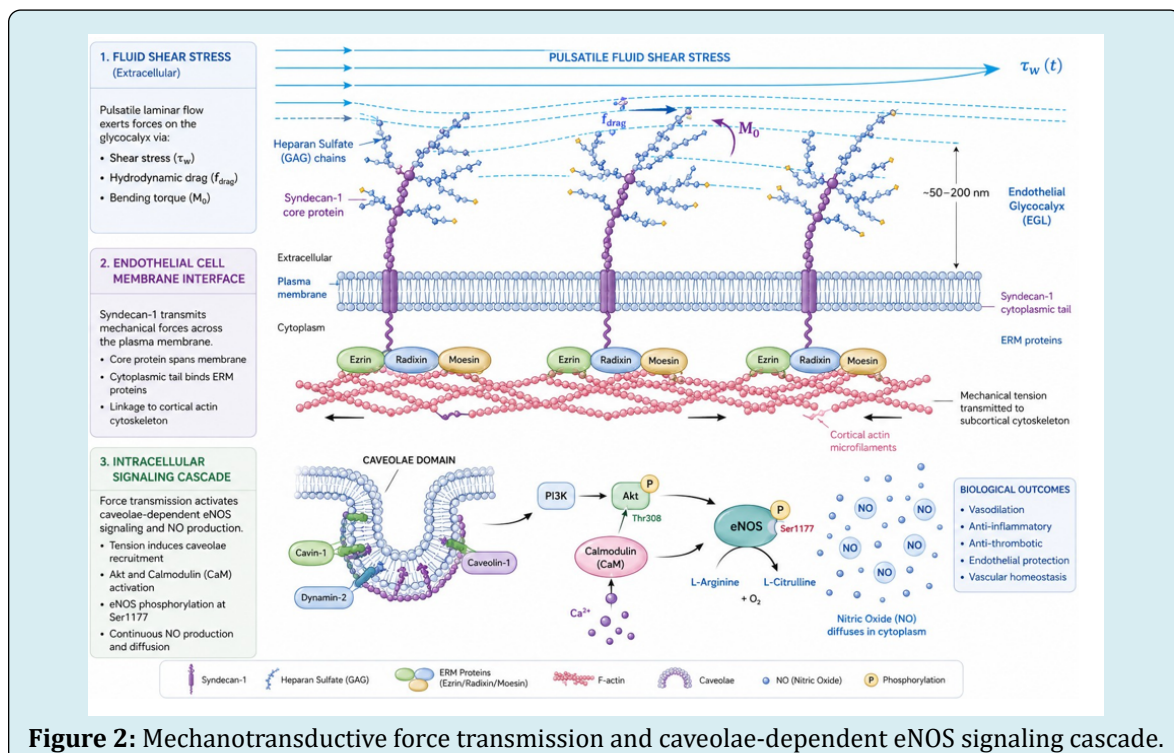


Figure 2: Mechanotransductive force transmission and caveolae-dependent eNOS signaling cascade.

Schematic diagram illustrating the multi-step mechanobiological coupling: (1) Extracellular pulsatile fluid shear stress (τ_w) exerts hydrodynamic drag (f_{drag}) and bending torque (M_0) on syndecan-1 core proteins and heparan sulfate glycosaminoglycan (GAG) chains.

(2) Mechanical forces are transmitted across the plasma membrane via syndecan-1 cytoplasmic tails and ERM proteins (Ezrin/Radixin/Moesin) to cortical actin microfilaments under tension. (3) Intracellular force transmission induces caveolae domain recruitment (Caveolin-1, Cavin-1, Dynamin-2), activating the PI3K/Akt and Ca²⁺/Calmodulin pathways to promote eNOS phosphorylation at Ser1177, catalyzing the conversion of L-Arginine and O₂ into L-Citrulline and sustained Nitric Oxide (NO) release for vascular homeostasis.

Hydrodynamic Low-Pass Buffering: The complex viscoelastic modulus $G^*(\omega) = G'(\omega) + i \cdot G''(\omega)$ ensures that high-frequency pressure harmonics ($\omega > 5$ Hz) generated during systolic acceleration are dampened via internal viscous dissipation within the GAG meshwork [5,6].

Volumetric Stress Redistribution: The finite permeability K_p prevents abrupt interfacial shear discontinuities, converting concentrated surface traction into a distributed volumetric body force across the apical sub-membrane zone [2,4].

eNOS Signaling Fidelity: Low-frequency cyclic loading (≈ 1 Hz) maintains continuous structural tension on syndecan-4 cytoplasmic domains, activating Akt and calmodulin cascades to sustain endothelial nitric oxide synthase (eNOS) phosphorylation without generating oxidative noise [7,8].

Pathological Degradation and Microvascular Failure Regimes

Enzymatic shedding of the glycocalyx (mediated by MMP-2, MMP-9, heparanase, or oxidative free-radical cascades) induces a sharp transition across biophysical states [3,10]:

Intact / Physiological EGL:

Effective Thickness: $h \approx 0.5 - 1.0 \mu\text{m}$ [2]
 Hydrodynamic Permeability: $K_p \approx 10^{-16}$ to 10^{-15} m^2 [4]
 Transmitted Peak Shear: Phase-shifted and buffered [6]
 Endothelial Status: Laminar sovereignty, high NO, low permeability [8].

Partial Degradation State:

Effective Thickness: $h \approx 0.2 - 0.4 \mu\text{m}$ [3]
 Hydrodynamic Permeability: $K_p \approx 10^{-14} \text{ m}^2$ [2]
 Transmitted Peak Shear: Intermediate amplification [6]
 Endothelial Status: Inflammatory priming, elevated membrane impedance [9].

Complete Shedding State:

Effective Thickness: $h \approx 0 \mu\text{m}$ [3]
 Hydrodynamic Permeability: $K_p \rightarrow \text{infinity}$ [4]
 Transmitted Peak Shear: Direct, unbuffered oscillatory impact [1, 7]
 Endothelial Status: Endothelial apoptosis, leukocyte adhesion, loss of flow-mediated dilation (FMD) [11, 12].

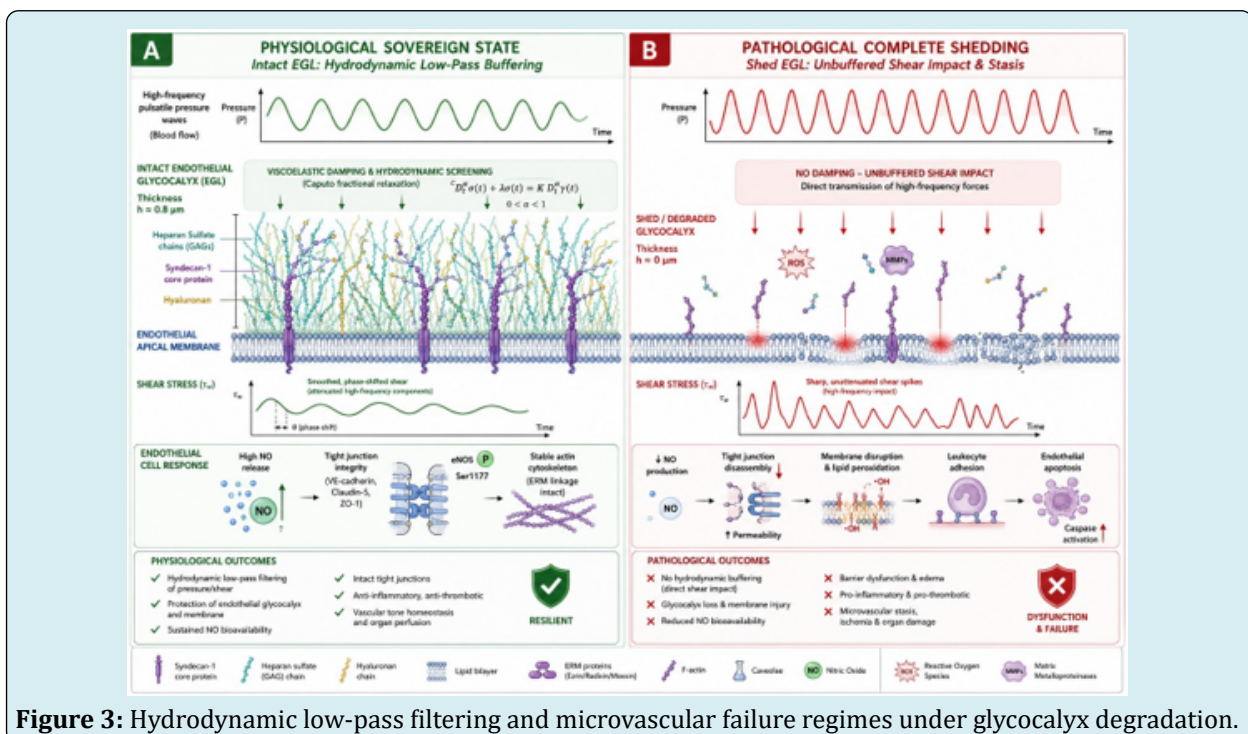


Figure 3: Hydrodynamic low-pass filtering and microvascular failure regimes under glycocalyx degradation.

Comparative biophysical model of shear transmission. (A) Physiological Sovereign State: An intact endothelial glycocalyx ($h \approx 0.8 \mu\text{m}$) provides non-linear viscoelastic damping (Caputo fractional relaxation) and hydrodynamic screening, filtering high-frequency pressure harmonics and producing a protective phase lag (θ) on wall shear stress (τ_w). This stabilizes tight junction integrity (VE-cadherin, Claudin-5, ZO-1), actin-ERM linkage, and eNOS Ser1177 phosphorylation, sustaining high NO bioavailability and vascular tone homeostasis. (B) Pathological Complete Shedding: Enzymatic and oxidative degradation ($h \approx 0 \mu\text{m}$, mediated by ROS and MMPs) eliminates the porous barrier. The bare apical membrane is directly struck by sharp, unattenuated shear spikes, triggering a drop in NO synthesis, junctional disassembly, membrane lipid peroxidation, leukocyte adhesion, and caspase-dependent endothelial apoptosis. Loss of the viscoelastic interface exposes the bare lipid bilayer to unattenuated dynamic shear spikes ($\partial\tau_w / \partial t \gg 0$), accelerating lipid peroxidation, increasing transmembrane impedance (Z_{membrane}), and precipitating microvascular autoregulatory collapse [8,10].

Discussion

The present formulation demonstrates that the endothelial glycocalyx decouples instantaneous macrovascular flow dynamics from the delicate apical membrane of endothelial cells. In conventional hemodynamic paradigms, shear stress is assumed to act as a pure boundary condition applied directly to the cell surface. By incorporating Brinkman porous mechanics and fractional viscoelastic relaxation, our model accounts for the finite time required for shear waves to propagate across the brush.

This phase shift $\theta(\omega)$ protects cell-cell junctions from mechanical fatigue and ensures that shear-dependent biochemical cascades (such as eNOS activation) respond to time-averaged physiological laminar signals rather than destructive high-frequency transients. The quantitative link between glycocalyx degradation and the sharp rise in viscous dissipation (E_{diss}) explains the clinical onset of microvascular stasis and endothelial barrier hyperpermeability observed under sustained metabolic and oxidative noise.

Conclusion

This non-equilibrium hydrodynamic model proves that the endothelial glycocalyx is not a passive mechanical coating, but an active, tuned biophysical filter essential for microvascular homeostasis [1,2,7]. By coupling Brinkman porous flow with fractional viscoelastic constitutive equations, we demonstrate how the EGL decouples destructive high-frequency shear spikes from the endothelial

plasma membrane while sustaining physiological eNOS mechanotransduction [4,5,8].

Integrating fluid mechanics with non-equilibrium thermodynamics establishes a rigorous foundation for predicting microvascular resilience and designing precision interfacial therapies under the Science 4.0 framework [8-10].

Declarations

Conflict of Interest: The author declares no conflicts of interest.

Data Availability: Mathematical models, boundary value solutions, and simulation parameters are available upon request from the corresponding author.

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