

UC Riverside

UC Riverside Undergraduate Research Journal

Title

Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

Permalink

<https://escholarship.org/uc/item/55s1x7zd>

Journal

UC Riverside Undergraduate Research Journal, 20(1)

Author

Pinzon, Keyanna (Keys) Milenia Lalo

Publication Date

2026-10-09

DOI

10.5070/RJ5.64259

Copyright Information

This work is made available under the terms of a Creative Commons Attribution-NonCommercial License, available at <https://creativecommons.org/licenses/by-nc/4.0/>

Peer reviewed

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

Keyanna-Milenia Pinzon, Department of Bioengineering

Ethan Liu, Department of Bioengineering

Kai Lin, Department of Bioengineering

Adarsh Mattappally, Department of Bioengineering

Victor G Rodgers, Ph.D.; Department of Bioengineering

ABSTRACT

Endothelial cells are continuously exposed to shear stress from blood flow, which plays a critical role in mechanotransduction and the regulation of cellular behavior. These forces induce cellular deformation and transmit stress to intracellular structures, including the nucleus. Cellular senescence is associated with increased stiffness, which alters how mechanical forces are distributed within the cell. However, there is a lack of computational models that directly compare healthy and senescent endothelial cells under identical flow conditions.

In this study, a computational model was developed using COMSOL Multiphysics® to evaluate the mechanical responses of healthy and senescent endothelial cells under steady-state, incompressible laminar flow. Material properties were defined using atomic force microscopy measurements, and both the cell body and nucleus were modeled as isotropic, linearly elastic materials.

Results showed that healthy cells exhibit approximately 20-40 times greater deformation than senescent cells across all regions. Additionally, Von Mises stress analysis revealed a reduction of approximately 29.5% in stress transmission to the nucleus in senescent cells. These findings indicate that increased cellular stiffness significantly reduces both deformation and internal force propagation.

Overall, this study demonstrates that senescence impairs the mechanical responsiveness of endothelial cells, which may contribute to altered mechanotransduction and dysfunction in vascular systems.

FACULTY MENTOR - Dr. Victor G. Rodgers, Department of Bioengineering



Dr. Rodgers applies transport phenomena, mathematical modeling, thermodynamics, and kinetics to biomedical engineering problems. The Rodgers Lab develops medical devices to reduce edema and deliver drugs for severe stroke, spinal cord injury, and TBI patients, and studies the thermodynamics of crowded protein systems.



**KEYANNA-MILENIA
PINZON**

Keyanna-Milenia Pinzon is a third-year Bioengineering honors student. She is a Chancellor's Research Fellow and Bilderback Bioengineering Scholar, and plans to pursue a Ph.D. in biomedical engineering focused on biological therapeutics.



ETHAN LIU

Ethan Liu is a third-year bioengineering major. He plans to pursue a career developing engineered biological components for medicine.



KAI LIN

Kai Lin is a third-year Bioengineering major. He plans to pursue a Ph.D. in biophysics and medical device development.



**ADARSH
MATTAPPALLY**

Adarsh Mattappally is a third-year Bioengineering major. He is president of the Undergraduate Research Club and plans to pursue an M.D.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

INTRODUCTION

Endothelial cells form a monolayer lining on the interior surface of blood vessels and play a critical role in maintaining vascular homeostasis via signaling pathways (Figure 1). These cells regulate processes such as vascular permeability, inflammation, blood clotting, and vascular flow (Bloom et al., 2024). As blood moves through vessels, it exerts fluid shear stress on the endothelial cell membrane, creating mechanical forces that influence cell behavior and function.

An important property of cells is their ability to transform mechanical stimuli into biochemical or electrical signals, a process known as mechanotransduction. Shear stress applied at the membrane is transmitted through the cytoskeleton and reaches the nucleus. Previous studies have shown that the nuclear-to-cytoplasmic (cell body) stiffness ratio determines how mechanical stress is distributed throughout the cell (Angom et al., 2022).

Cellular senescence occurs when the DNA of a cell is damaged, causing cell cycle arrest, but the cell continues to be metabolically active. Senescence naturally occurs in our bodies as we age and is an entry path to apoptosis, or cell death. It is believed to be a cause of many age-related diseases, including cardiovascular disease. Senescent cells exhibit higher stiffness, reflected by a higher Young's Modulus (Angom et al., 2022). These mechanical changes affect how cells respond to forces and the permeability of the membrane. Unfortunately, there is a lack of models that simulate senescent conditions in endothelial cells. Having a model to understand the behavior in relation to fluid flow can offer a useful tool to researchers.

In order to investigate these mechanical effects, a computational model was developed to simulate healthy and senescent endothelial cells under fluid flow and to quantify the forces acting on the cells. In this study, COMSOL Multiphysics® (Version 6.2, Burlington, MA, USA; referred to as COMSOL) is used due to the program's ability to couple fluid dynamics and solid mechanics, enabling analysis of shear stress, cell deformation, and stress gradients that are transmitted to the nucleus. Governing equations from fluid mechanics and linear elasticity were used to capture the interaction between fluid flow and cellular response, allowing deformation magnitude and stress distributions to

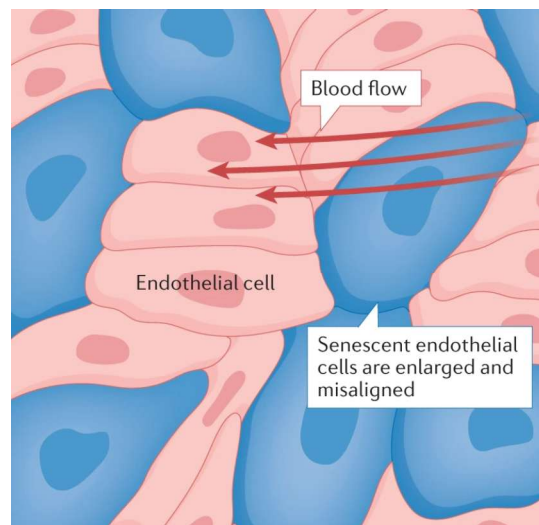


Figure 1. Morphological differences in senescent and healthy endothelial cells. Senescent cells (blue) become enlarged and misaligned to the blood flow. They exhibit higher stiffness and less deformation. Bloom et al., Mechanisms and consequences of endothelial cell senescence, 2022.

be quantified. The parameters were chosen to evaluate how differences in cellular stiffness influence the mechanical response of endothelial cells to shear flow.

THEORY

To model healthy and senescent endothelial cells under identical shear flow conditions, simulations were conducted under the Navier-Stokes equation implemented in COMSOL. The Navier-Stokes equation governs fluid motion and incorporates parameters that determine shear stress, including fluid density, dynamic viscosity, and the velocity field. The model will show how fluid flow generates shear forces at the cell surface, enabling direct comparison of mechanical responses between healthy and senescent cells.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

$$\rho (u \cdot \nabla)u = \nabla \cdot [-pI + K] + F$$

ρ = fluid density

u = fluid velocity vector

p = pressure

I = identity tensor

K = viscous stress tensor

F = external force

In addition to the conservation of momentum described by the Navier-Stokes equation, the conservation of mass within the fluid domain is enforced by the continuity equation:

$$\nabla \cdot u = 0$$

Both governing equations describe an incompressible fluid flow within the system. To solve the equations, the flow was assumed to be a fully developed laminar flow; the velocity profile remains constant along the direction of flow with no acceleration. This assumption simplifies the governing equations by making the spatial derivative of velocity equal to zero. Incompressible flow was assumed, and the density remained constant throughout the model. The cell body and the nucleus were linearly elastic, allowing deformation to occur using the constant material properties.

In the model, the fixed constraint was where the cell adheres to the endothelial wall at $z = 0$. The fluid was given the same properties as water. The model was unable to support the dynamic viscosity of blood, but rather than modeling blood rheology, our aim was to compare the difference in shear stress while keeping fluid type constant. The cells were simplified into a hemisphere. The cell was split in the y-direction, assuming symmetry.

METHODS

Assumptions - To enable tractable computation and isolate the effect of cellular stiffness, multiple assumptions were made to ensure efficiency of COMSOL simulations and the model within the scope of this analysis. The model was a stationary model; the fluid flow was fully developed and could be

characterized by laminar flow, allowing for a constant profile along the direction of flow. Additionally, the material of the cell body and nucleus was modeled as an isotropic linearly elastic material, enabling deformation to be calculated using constant material properties.

Additionally, the fluid was treated as incompressible with constant density, which is appropriate for biological fluids under low-speed conditions where density variations are negligible.

Geometry - A three-dimensional model was constructed to represent a single endothelial cell within a surrounding fluid domain. The fluid domain was defined as a cube with side lengths of $5.4 \mu\text{m}$. The cell was modeled as a quarter sphere positioned on the bottom surface of the domain, representing adhesion to the vessel wall. For the healthy cell, the radius of the sphere was set to $1.35 \mu\text{m}$ to reflect the measured cell height from the literature (Bloom et al., 2024). The nucleus was modeled as a hemisphere with a radius of $0.25 \mu\text{m}$, positioned concentrically within the cell body and elevated $0.5 \mu\text{m}$ above the base surface. The final geometry for the healthy cell is shown in Figure 2. For the senescent cell model shown in Figure 3, only the radius of the cell body was modified to $0.89 \mu\text{m}$ while all other geometric parameters were held constant to allow for direct comparison.

Subdomain - Because the primary focus of this study was the relative difference in mechanical responses between healthy and senescent endothelial cells, rather than precise replication of blood rheology, the fluid was modeled using properties equivalent to water. This simplification was necessary due to limitations in representing the dynamic viscosity of blood within the model. While this assumption did not capture the full complexity of physiological flow, it enabled consistent comparison of shear stress and deformation between the two cell conditions under identical fluid properties. The fluid was modeled using properties equivalent to water, with a density of 1000 kg/m^3 , along with the dynamic viscosity of $0.001 \text{ Pa}\cdot\text{s}$.

The mechanical properties of the cell body were assigned based on literature values, with Young's modulus set to 0.81 kPa for the healthy cell and 4.52 kPa for the senescent cell. The nucleus was assigned a constant Young's modulus of

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

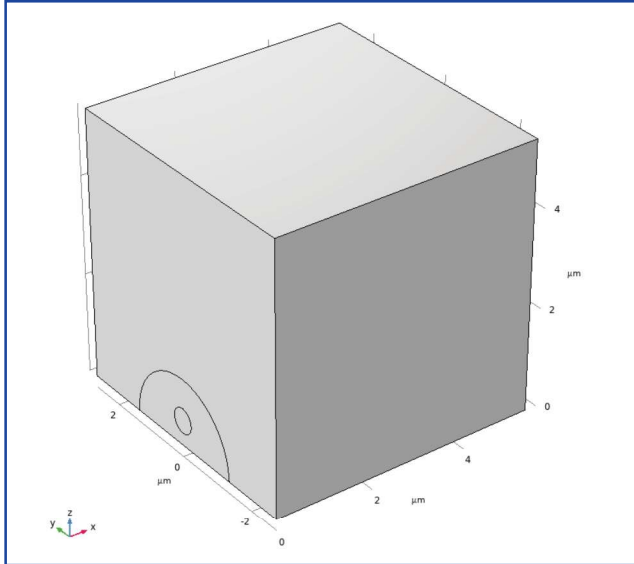


Figure 2: Fluid with velocity over a healthy endothelial cell. Fluid is represented by the cube. The cell membrane is shown as a hemisphere and the nucleus is represented as a sphere. Both are split in the y-direction assuming symmetry.

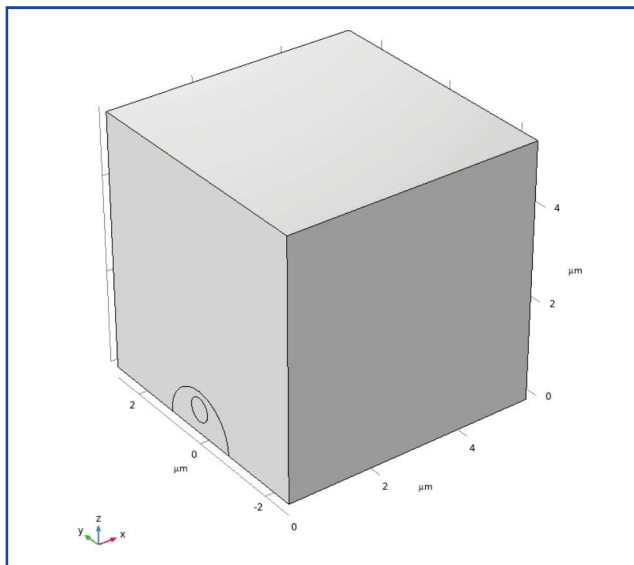


Figure 3: Fluid with velocity over a senescent endothelial cell. Compared to Fig. 2, the distance between the highest y-value of the cell and the nucleus is smaller due to senescence.

5 kPa for both cases. The Young's modulus stiffness values were taken from atomic force microscopy techniques on endothelial cells (Angom et al., 2022). A Poisson's ratio of 0.3 was assumed for both the cell body and the nucleus, consistent with literature values (Angom et al., 2022).

Boundary - A symmetry boundary condition was applied along one face of the fluid domain. The inlet and outlet were defined along the y-direction, with the inlet assigned a uniform velocity of 0.015 m/s and the outlet set to a reference pressure of 0 Pa. A fixed constraint was applied at the base of the cell. An attachment was set to the surfaces between the nucleus and the cell body. The remaining boundaries were set to no-slip. The shear stress transmitted from the cell body to the cell nucleus was analyzed. The cell was divided into three regions to quantify deformation.

The three regions characterized the spatial distribution of deformation throughout the cell. Shear stress was evaluated at the cell surface and around the nucleus, enabling comparison between membrane stress and internally propagated stress toward the nucleus.

RESULTS & DISCUSSION

The stress and deformation values were averaged across 25 sample points within each region. To quantify the mechanical response of healthy and senescent cells, we analyzed deformation and Von Mises stress distributions resulting from fluid flow. Cell deformation represented how the cell changes shape under shear stress, providing insight into how forces propagate through the structure. Displacement magnitudes were evaluated across three regions: the upper region exposed to flow, the middle region near the nucleus, and the adhesion site where the cell was fixed to the vessel wall.

The healthy cell exhibited much greater deformation across all regions compared to the senescent model (Figure 4). In the upper region, the healthy cell deformed by $1.12 \pm 0.06 \mu\text{m}$, compared to $0.03 \pm 0.01 \mu\text{m}$ by the senescent cell. In the middle region around the nucleus, deformation dropped from $0.53 \pm 0.06 \mu\text{m}$ in the healthy cell to $0.02 \pm 0.01 \mu\text{m}$ in the senescent, and from $0.13 \pm 0.02 \mu\text{m}$ to $0.01 \pm 0.01 \mu\text{m}$ at the adhesion site.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

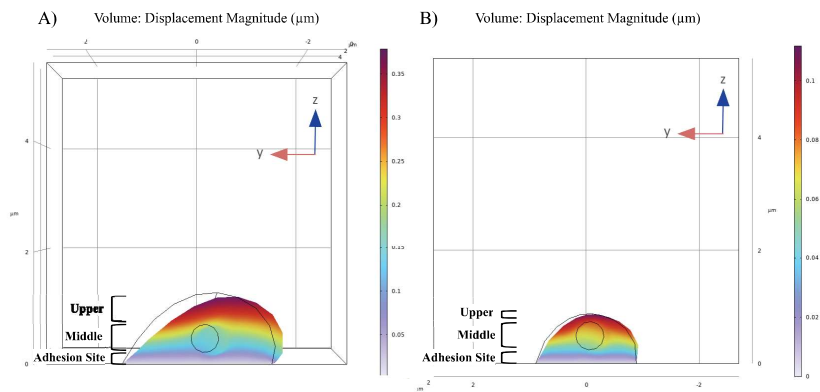


Figure 4. Magnitude of the volume of displacement of a healthy cell (A) and a senescent cell (B).

The upper region is classified the distance between the nucleus and the surface of the cell. The middle region is the diameter of the nucleus. The adhesion site is located below the nucleus. The healthy cell model faces a noticeably increased deformation compared to the senescent cell model.

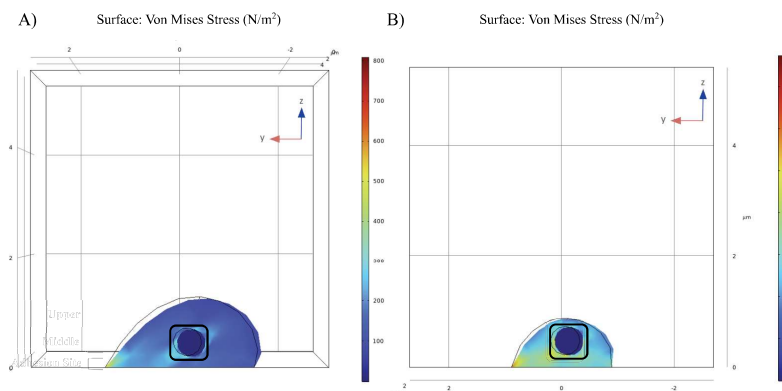


Figure 5. Distribution of stress in the membrane of a healthy cell (A) and a senescent cell (B).

The boxed region indicates the area averaged to calculate stress around the nucleus. Referencing the scale, the healthy cell model has a noticeably increased internal stress distribution compared to the senescent cell.

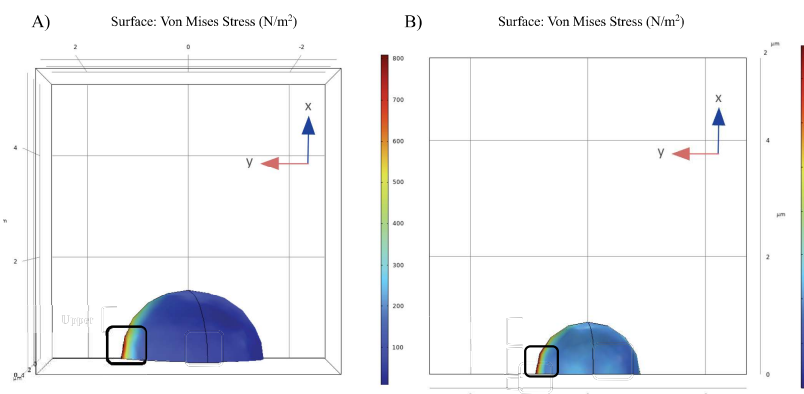


Figure 6. The distribution of stress on the surface of a healthy cell (A) and a senescent cell (B).

The boxed region indicates the area averaged to calculate shear surface force. The healthy cell has a noticeably greater surface shear stress compared to the senescent cell.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

This difference in deformation behavior directly influences the internal stress distribution, which was evaluated using Von Mises stress. Stress was evaluated in two locations, the outer cell surface and inner body just around the nucleus.

Like the deformation models, at the surface of the cell, the healthy cell experienced $763.42 \pm 17.91 \text{ N/m}^2$ stress compared to the senescent model's $284.23 \pm 13.72 \text{ N/m}^2$ (Figure 5).

Around the nucleus, the Von Mises stress decreased from $325.59 \pm 17.72 \text{ N/m}^2$ in the healthy cell model to $229.35 \pm 9.33 \text{ N/m}^2$ in the senescent cell model.

The results presented by the COMSOL simulation indicate that senescent endothelial cells experienced significantly lower Von Mises stress at both the cell membrane and around the nucleus compared to healthy cells.

This reduction reflected a decreased transmission of mechanical stress through the cellular structure. These findings are consistent with literature reports, which observed an approximate $\sim 56\%$ reduction in shear stress transmitted to the nucleus from healthy Human Umbilical Vein Endothelial Cells (HUVEC) to aged senescent HUVEC cells (Walther et al., 2023). In comparison, the present model predicted a reduction of approximately $\sim 29.5\%$ when observing the Von Mises transmitted to the nucleus. While the magnitude differs, both computational results and experimental findings agree on the trend of cellular senescence reducing nuclear mechanosensing. Reduced deformation and stress transmission observed in the senescent cell model suggest that increased cellular stiffness limits the cell's ability to mechanically respond to shear loading. This diminished mechanical responsiveness may impair mechanotransduction, as lower deformation reduces the propagation of forces to intracellular structures such as the nucleus, potentially altering shear-sensitive signaling pathways.

The discrepancy between the simulations and the experimental results (Walther et al., 2023) can be attributed to several simplifying assumptions in the model. First, the use of an idealized hemispherical geometry does not capture the complex morphology of endothelial cells, which may influence local stress distributions, force transmission

pathways, and assumptions within the Navier-Stokes equation. Additionally, modeling the cell body and nucleus as linearly elastic materials neglects the viscoelastic behavior of biological tissues, potentially affecting the magnitude and temporal response of deformation. The use of water properties instead of blood further simplifies the fluid environment, omitting non-Newtonian effects that can alter shear stress profiles. Among these assumptions, geometric simplification was expected to have the most direct impact on spatial stress distribution, while the material and fluid assumptions primarily influenced the magnitude of the predicted responses.

To enable tractable computation and to isolate the effect of cellular stiffness, several simplifying assumptions were applied to the Navier–Stokes formulation. The flow was assumed to be steady-state, eliminating time-dependent terms and allowing the velocity field to remain constant over time. This assumption was reasonable because the study focused on the comparative mechanical response of cells under constant shear conditions, rather than transient pulsatile blood flow.

The flow was also assumed to be fully developed and laminar, meaning the velocity profile did not change along the direction of flow, and inertial effects were minimal. This is consistent with microscale flow conditions typical of small vessels and ensures that shear stress remains uniform and comparable across simulations.

Additionally, the fluid was treated as incompressible with constant density, which is appropriate for biological fluids under low-speed conditions where density variations are negligible. Together, these assumptions simplified the governing equations while preserving the dominant shear-driven forces acting on the cell surface.

Although these simplifications did not capture the full complexity of physiological blood flow, they provided a controlled framework for consistently evaluating how differences in cellular stiffness influenced deformation and stress transmission under identical flow conditions. These findings suggest that increased cellular stiffness alters how mechanical forces are distributed within

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

the cell, which may influence how external stimuli are experienced at a structural level. The presented model offers a framework for understanding how changes in mechanical properties affect cellular response to fluid flow. In vascular systems, disrupted mechanotransduction has been associated with endothelial dysfunction, increased inflammation, and the progression of cardiovascular diseases such as atherosclerosis. The presented model offers a simplified yet informative framework for understanding how age-related mechanical changes may contribute to vascular pathology under fluid-flow conditions.

Future work should extend this model by incorporating viscoelastic material properties to better capture time-dependent cellular deformation. Additionally, implementing more realistic endothelial cell geometries would improve the accuracy of predictions of stress distribution and force transmission. Incorporating non-Newtonian fluid properties would further enhance physiological relevance by accounting for shear-dependent viscosity effects in blood flow.

CONCLUSION

This study used computational modeling to investigate how differences in cellular stiffness influenced the mechanical response of endothelial cells under fluid shear stress. Using coupled fluid dynamics and solid mechanics in COMSOL, healthy and senescent cells were analyzed under identical laminar flow conditions to quantify deformation and stress transmission.

Results show that cellular stiffness plays a critical role in how mechanical forces are distributed within the cell. Healthy cells, characterized by a lower Young's modulus, exhibited ~20–40 times greater deformation than did senescent cells. In contrast, senescent cells showed minimal deformation and a more uniform stress distribution. Analysis of Von Mises stress further revealed differences in force transmission to the nucleus, with healthy cells exhibiting localized stress concentrations, while senescent cells distributed stress more broadly. These findings indicate that increased stiffness reduces the cell's ability to mechanically adapt to external flow.

The computational trends observed in this study are consistent with findings reported in the literature (Walther et al., 2023), which demonstrated a substantial reduction in shear stress transmitted to the nucleus in senescent endothelial cells compared to healthy cells. While this model predicts a ~29.5% reduction compared to ~56% experimentally, both demonstrate the same overall trend of decreased stress transmission with increased stiffness. These results highlight the role of cellular stiffness in regulating how forces are experienced within the cell.

Location of Deformation	Average Deformation of Healthy Cell	Average Deformation of Senescent Cell
Upper Region	$1.12 \pm 0.06 \mu\text{m}$	$0.03 \pm 0.01 \mu\text{m}$
Middle Region	$0.53 \pm 0.06 \mu\text{m}$	$0.02 \pm 0.01 \mu\text{m}$
Adhesion Site	$0.13 \pm 0.02 \mu\text{m}$	$0.01 \pm 0.01 \mu\text{m}$

Table 1. Average deformation values of the healthy and senescent cell models from three distinct regions. Values shown were averaged from 20 random points along the corresponding region. Standard deviation calculated through Google Sheets.

Location of Shear	Healthy Cell	Senescent Cell
Cell Membrane	$764.07 \pm 18.01 \text{ N/m}^2$	$284.23 \pm 13.72 \text{ N/m}^2$
Around the Nucleus	$325.59 \pm 17.72 \text{ N/m}^2$	$229.35 \pm 9.33 \text{ N/m}^2$

Table 2. Average Von Mises stress values of the healthy and senescent cell models from both the outer cell membrane and inner area around the nucleus. Values shown were averaged from 25 random points along the corresponding region. Standard deviation calculated through Google Sheets.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

APPENDIX

Healthy Cell Membrane			
X	Y	Z	Value [N/m ²]
0.0058452	1.3472	0.0032849	753.39
0.033107	1.3408	0.0068658	751.98
0.058435	1.3388	0.0034157	771.08
0.080632	1.3329	0.0076866	765.86
0.10775	1.329	0.0069109	777.52
0.13139	1.3271	0.0036908	795.34
0.15503	1.3252	4.73 × 10 ⁻⁴	806.18
0.19267	1.3155	0.0070636	782.97
0.23845	1.3084	0.0066602	784.78
0.29291	1.299	0.0080607	766.25
0.32336	1.2949	0.0068125	750.51
0.28293	1.2982	0.012364	758.18
0.2508	1.3008	0.016718	748.69
0.20333	1.3079	0.017353	751.23
0.15416	1.3152	0.018211	752.94
0.10968	1.3247	0.013942	757.01
0.075909	1.3274	0.018542	731.55
0.051926	1.3352	0.011597	744.21
0.019648	1.3411	0.010158	737.44
0.20467	1.312	0.009824	773.6
0.26435	1.2988	0.016329	748.59
0.29486	1.2963	0.012229	754.37
0.18424	1.3161	0.0082195	780.31
0.28918	1.3027	0.0026576	782.4
0.30486	1.2964	0.0093682	755.47
0.24196	1.3065	0.0091169	772.23
0.17237	1.3179	0.0083781	780.94
0.13348	1.3203	0.015084	761.37
0.1657	1.3176	0.010762	774.44
0.18957	1.3124	0.013364	764.44
0.22483	1.3112	0.0055905	784.37
0.12999	1.3222	0.012634	767.94
0.081073	1.3253	0.020757	726.66

Healthy Cell Nucleus			
X	Y	Z	Value [N/m ²]
1.91 × 10 ⁻¹⁸	0.08796	0.23065	280.33
1.42 × 10 ⁻¹⁷	-0.094474	0.21152	341.08
1.33 × 10 ⁻¹⁷	-0.082884	0.21304	335.23
1.30 × 10 ⁻¹⁷	-0.10036	0.2006	306.68
1.45 × 10 ⁻¹⁷	-0.097919	0.21138	342.99
1.30 × 10 ⁻¹⁷	-0.079965	0.21173	332.87
1.35 × 10 ⁻¹⁷	-0.088432	0.20711	335.34
1.20 × 10 ⁻¹⁷	-0.064265	0.21986	327.8
1.33 × 10 ⁻¹⁷	-0.084665	0.21047	334.92
1.20 × 10 ⁻¹⁷	-0.063703	0.22134	327.75
1.40 × 10 ⁻¹⁷	-0.096499	0.2071	335.06
1.20 × 10 ⁻¹⁷	-0.0689	0.21181	326.57
1.43 × 10 ⁻¹⁷	-0.10281	0.20785	331.69
1.41 × 10 ⁻¹⁷	-0.091437	0.21521	341.25
1.43 × 10 ⁻¹⁷	-0.095204	0.21185	341.67
1.16 × 10 ⁻¹⁷	-0.059871	0.21867	322.27
1.36 × 10 ⁻¹⁷	-0.08198	0.22075	338.69
1.17 × 10 ⁻¹⁷	-0.054792	0.23058	320.31
1.13 × 10 ⁻¹⁷	-0.052543	0.22581	316.45
1.34 × 10 ⁻¹⁷	-0.083571	0.21424	336.24
1.39 × 10 ⁻¹⁷	-0.090737	0.21116	338.75
1.29 × 10 ⁻¹⁷	0.12171	0.4816	308.31
1.25 × 10 ⁻¹⁷	0.11654	0.50583	266.63
1.30 × 10 ⁻¹⁷	0.11441	0.47788	320.14
1.31 × 10 ⁻¹⁷	0.10696	0.45176	293.48
1.34 × 10 ⁻¹⁷	-0.080122	0.21961	337.04
1.43 × 10 ⁻¹⁷	-0.094454	0.21345	342.07
1.25 × 10 ⁻¹⁷	-0.070964	0.21771	330.8
1.19 × 10 ⁻¹⁷	-0.058378	0.22904	324.38
1.34 × 10 ⁻¹⁷	-0.085296	0.21155	335.84
1.23 × 10 ⁻¹⁷	-0.11633	0.21427	319.68
1.33 × 10 ⁻¹⁷	-0.097832	0.20281	317.51
1.32 × 10 ⁻¹⁷	-0.081713	0.21309	334.58

Table 3. Points used to calculate average Von Mises stress of the healthy endothelial cell in the membrane and around the nucleus.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

APPENDIX

Senescent Cell Membrane			
X	Y	Z	Value
4.99×10^{-1}	7.09×10^{-1}	2.66×10^{-2}	2.67×10^2
4.54×10^{-1}	7.38×10^{-1}	3.13×10^{-2}	2.72×10^2
4.16×10^{-1}	7.60×10^{-1}	4.63×10^{-2}	2.67×10^2
3.75×10^{-1}	7.90×10^{-1}	3.70×10^{-2}	2.82×10^2
3.32×10^{-1}	8.14×10^{-1}	4.37×10^{-2}	2.84×10^2
3.03×10^{-1}	8.20×10^{-1}	4.08×10^{-2}	2.88×10^2
2.83×10^{-1}	8.24×10^{-1}	4.14×10^{-2}	2.88×10^2
2.73×10^{-1}	8.26×10^{-1}	4.17×10^{-2}	2.88×10^2
2.47×10^{-1}	8.32×10^{-1}	3.87×10^{-2}	2.92×10^2
2.17×10^{-1}	8.38×10^{-1}	3.96×10^{-2}	2.92×10^2
1.97×10^{-1}	8.42×10^{-1}	4.02×10^{-2}	2.93×10^2
1.85×10^{-1}	8.44×10^{-1}	4.06×10^{-2}	2.93×10^2
1.35×10^{-1}	8.53×10^{-1}	4.21×10^{-2}	2.93×10^2
1.18×10^{-1}	8.57×10^{-1}	4.27×10^{-2}	2.93×10^2
1.05×10^{-1}	8.59×10^{-1}	4.31×10^{-2}	2.93×10^2
9.54×10^{-2}	8.61×10^{-1}	4.34×10^{-2}	2.93×10^2
8.30×10^{-2}	8.63×10^{-1}	4.37×10^{-2}	2.92×10^2
7.31×10^{-2}	8.65×10^{-1}	4.40×10^{-2}	2.92×10^2
4.94×10^{-2}	8.71×10^{-1}	4.09×10^{-2}	2.94×10^2
3.48×10^{-2}	8.73×10^{-1}	4.05×10^{-2}	2.95×10^2
2.54×10^{-2}	8.66×10^{-1}	7.20×10^{-2}	2.72×10^2
6.19×10^{-2}	8.60×10^{-1}	7.35×10^{-2}	2.69×10^2
9.49×10^{-2}	8.55×10^{-1}	7.03×10^{-2}	2.71×10^2
1.58×10^{-1}	8.42×10^{-1}	7.22×10^{-2}	2.69×10^2
2.14×10^{-1}	8.32×10^{-1}	6.66×10^{-2}	2.72×10^2
2.34×10^{-1}	8.28×10^{-1}	6.60×10^{-2}	2.71×10^2
2.34×10^{-1}	8.28×10^{-1}	6.60×10^{-2}	2.71×10^2
2.46×10^{-1}	8.26×10^{-1}	6.56×10^{-2}	2.71×10^2
2.85×10^{-1}	8.16×10^{-1}	7.60×10^{-2}	2.61×10^2
4.87×10^{-2}	8.61×10^{-1}	7.65×10^{-2}	2.68×10^2
1.81×10^{-2}	8.67×10^{-1}	7.17×10^{-2}	2.73×10^2
2.12×10^{-2}	8.76×10^{-1}	3.64×10^{-2}	2.99×10^2
1.22×10^{-2}	8.81×10^{-1}	2.55×10^{-2}	3.07×10^2
6.97×10^{-3}	8.84×10^{-1}	1.82×10^{-2}	3.13×10^2
2.65×10^{-2}	8.80×10^{-1}	1.86×10^{-2}	3.12×10^2

Senescent Cell Nucleus			
X	Y	Z	Value
1.34×10^{-17}	-0.0130835862	0.2342515173	234.8014022
1.22×10^{-17}	0.0148688136	0.2409398488	226.2279059
1.17×10^{-17}	0.0123754513	0.2308238869	222.8892878
1.24×10^{-17}	0.00436739726	0.2314481258	228.1496562
1.32×10^{-17}	-0.01070637129	0.23259773	233.3058142
1.32×10^{-1}	-0.01779303865	0.233123154	232.9608628
1.32×10^{-1}	-0.02346240121	0.233543392	232.6847876
1.34×10^{-1}	-0.02583957016	0.2351973981	234.1806201
1.08×10^{-1}	-0.03617604262	0.2358975266	225.9071143
8.68×10^{-18}	-0.042762473	0.2392747713	221.0481733
5.66×10^{-18}	-0.05316858733	0.2399658664	211.4774932
3.12×10^{-18}	-0.06111734304	0.2433383257	208.1069221
5.24×10^{-18}	-0.06034842854	0.2196827981	201.6473199
8.62×10^{-18}	-0.04784334948	0.2219726332	209.1591788
8.69×10^{-18}	-0.04148901545	0.2435971162	224.0976488
1.17×10^{-17}	-0.03065476471	0.2443448008	234.7633915
1.36×10^{-17}	-0.02254758501	0.2364306125	235.951897
1.36×10^{-17}	-0.01546099362	0.2359051693	236.2968177
1.38×10^{-17}	-0.01358618521	0.2372440346	237.9997419
1.34×10^{-17}	0.001522183738	0.2419798131	234.9948985
1.30×10^{-17}	0.009264056951	0.245795138	232.0422036
1.39×10^{-17}	0.004015666365	0.2520953662	238.3332448
1.41×10^{-17}	-7.01×10^{-4}	0.2495170897	240.0094979
1.40×10^{-17}	-0.00105699276	0.2480718138	239.5324973
1.32×10^{-17}	0.002500538635	0.2404303872	233.6411832
1.27×10^{-17}	0.007839126498	0.2400143903	230.1344204
1.25×10^{-17}	0.0105085473	0.239806403	228.3809658
1.27×10^{-17}	0.007839126498	0.2400143903	230.1344204
1.25×10^{-17}	0.002676435139	0.2301069683	228.5494668
1.32×10^{-17}	-0.007871662035	0.2323875545	233.4437938
1.32×10^{-17}	-0.01212359971	0.2327028008	233.2368222
1.32×10^{-17}	-0.01212359971	0.2327028008	233.2368222
1.34×10^{-17}	-0.02017024809	0.234777171	234.4567019
1.36×10^{-17}	-0.01687835898	0.2360102479	236.2278165
1.36×10^{-17}	-0.001147101267	0.2421881197	236.7484231

Table 4. Points used to calculate average Von Mises stress of the senescent endothelial cell in the membrane and around the nucleus.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

APPENDIX

Upper			
X	Y	Z	Value [μm]
0	-0.1736334	1.167815	0.3541
0	0.08118816	1.156571	0.3621063
0	-0.5673889	1.19391	0.3549113
0	-0.2726956	1.246524	0.3712426
0	0.2049733	1.1523419	0.3699916
0	0.2854135	1.105685	0.3636345
0	-0.0365238	1.117433	0.3460238
0	-0.3420992	1.149606	0.3463025
0	-0.6727333	1.069807	0.3242520
0	-0.936547	1.054061	0.3239967
0	-0.7661938	1.045624	0.31891
0	-0.405778	1.023230	0.3130519
0	-0.0211170	1.014048	0.318149
0	0.2810838	1.091508	0.3593633
0	0.1545570	1.139602	0.3630868
0	0.4126326	1.056818	0.3605648
0	0.206336	1.05135410	0.3427521
0	-0.1330747	1.151077	0.3510638
0	-0.0757103	1.1408	0.3502866
0	-0.3149755	1.198453	0.3584111

Middle			
X	Y	Z	Value [μm]
0	0.9488621	0.4442371	0.1994230
0	0.66276296	0.46484002	0.18437065
0	0.49813228	0.51297236	0.18982360
0	0.35642295	0.52152677	0.18303225
0	0.33507972	0.56507038	0.19630346
-2.40×10^{-17}	-0.3088916	0.64252747	0.18709257
-1.44×10^{-17}	-0.4322415	0.62069455	0.19360741
0	-0.5456737	0.56656564	0.18179239
0	-0.6801116	0.56487681	0.18499485
0	-0.8092071	0.54305043	0.18341194
0	-1.0239740	0.53651058	0.19238764
0	-1.1562063	0.44474696	0.17568859
0	-1.1714071	0.44247577	0.17659052
0	-0.9715200	0.49761262	0.17725606
0	-0.8278222	0.48865987	0.16805285
0	-0.7210485	0.53579703	0.17832296
0	-0.6167453	0.53464955	0.17473135
0	-0.5277893	0.57997368	0.18479075
-7.57×10^{-19}	-0.4868377	0.63933871	0.19985351
-8.09×10^{-19}	-0.4948364	0.5273310	0.17152950

Adhesion Site			
X	Y	Z	Value [μm]
0	1.21483428	0.12453770	0.06614605
0	0.99095860	0.09890715	0.04182225
0	0.88117104	0.10922278	0.04479769
0	0.64035193	0.15258422	0.05991435
0	0.34775256	0.12135022	0.04778366
0	0.05284874	0.14425678	0.05590380
7.99×10^{-19}	-0.0814964	0.13256072	0.05427280
0	-0.1975314	0.11180923	0.04736328
0	-0.3119772	0.17683210	0.07286809
0	-0.4656101	0.14919623	0.05493930
0	-0.8240378	0.09525245	0.03318312
0	-1.0217821	0.11613717	0.04555904
0	-1.1573642	0.09686997	0.04187022
0	-1.2153930	0.16448396	0.07666321
0	-1.2636392	0.09537088	0.04771613
0	-1.0975083	0.14220523	0.05931331
0	-0.8965323	0.14724106	0.05351260
0	-0.7207538	0.11899181	0.04146544
0	-0.5957637	0.12825113	0.04525249
0	-0.4790656	0.09922869	0.03593792

Table 5. Points used to calculate average deformation values of a healthy endothelial cell in three sites.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

APPENDIX

Upper			
X	Y	Z	Value [μm]
0	0.39340771	0.72742507	0.03484600
0	0.32732432	0.73892209	0.03432316
0	0.25174556	0.79303362	0.03584859
0	0.20121915	0.79330064	0.03542962
4.15×10^{-18}	0.11969325	0.79431210	0.03481885
6.98×10^{-18}	0.07697428	0.79533169	0.03448685
8.78×10^{-18}	0.04978384	0.79599680	0.03428128
7.75×10^{-18}	0.01610702	0.81239134	0.03533840
7.95×10^{-18}	-0.0148320	0.81325501	0.03527222
5.21×10^{-18}	-0.0610775	0.81459949	0.03538612
3.53×10^{-18}	-0.0764758	0.81505900	0.03545325
1.52×10^{-19}	-0.1112090	0.81219678	0.03554190
0	-0.1351765	0.79738302	0.03480342
0	-0.1392450	0.79363191	0.03460718
0	-0.1430975	0.79372475	0.03462429
0	-0.1552736	0.78245188	0.03406822
0	-0.1783114	0.78295856	0.03425576
0	-0.2013638	0.78343513	0.03442453
-2.34E-18	-0.2456950	0.74166809	0.03268597
-5.92E-18	-0.2540424	0.72635460	0.03209320

Middle			
X	Y	Z	Value [μm]
0	0.73816335	0.34276547	0.02273521
0	0.67887371	0.34447022	0.02176854
0	0.60531037	0.40394414	0.02398639
0	0.44612070	0.42612535	0.02342833
0	0.42209540	0.43311827	0.02355787
0	0.39071489	0.43311603	0.02324127
0	0.34220373	0.43643918	0.02305835
0	0.32483640	0.43639699	0.02295104
3.33×10^{-19}	0.31789352	0.43633853	0.02291588
6.08×10^{-18}	0.27303655	0.44589383	0.02299578
1.27×10^{-17}	0.22786636	0.45474817	0.02291163
-4.08×10^{-17}	-0.2790138	0.47353178	0.02444622
-2.00×10^{-17}	-0.3306844	0.47186522	0.02403718
-6.15×10^{-18}	-0.3652000	0.47134210	0.02390351
0	-0.3963241	0.47101453	0.02376260
0	-0.4513632	0.47097834	0.02393357
0	-0.4656326	0.45701592	0.02344653
0	-0.5069523	0.45718915	0.02358272
0	-0.5799972	0.43682266	0.02312439
0	-0.6872570	0.41735359	0.02321695

Adhesion Site			
X	Y	Z	Value [μm]
0	0.80468886	0.03708650	0.00232567
0	0.75700163	0.04733361	0.00296826
0	0.70520342	0.04741652	0.00293428
0	0.64988782	0.04770941	0.00281425
0	0.63241315	0.04431309	0.00259210
0	0.55993208	0.04431309	0.00259210
0	0.38736082	0.04432918	0.00259460
0	0.29415769	0.04428549	0.00258244
0	0.22504593	0.04421839	0.00250379
0	0.12827590	0.04431754	0.00237261
0	0.05911139	0.04450486	0.00223866
0	-0.0264253	0.05870181	0.00303432
0	-0.1125988	0.05894690	0.00319386
0	-0.3260567	0.06593845	0.00373093
0	-0.3918838	0.06241597	0.00347236
0	-0.4402725	0.06238468	0.00339915
0	-0.4989793	0.06237566	0.00336635
0	-0.5612335	0.06238458	0.00324383
0	-0.6026514	0.06238458	0.00324383
0	-0.7474746	0.06309619	0.00360418

Table 6. Points used to calculate average deformation values of a senescent endothelial cell in three sites.

Impact of Fluid Flow on Mechanical Stress and Deformation in Healthy and Senescent Endothelial Cells

REFERENCES

- Angom RS, Kulkarni T, Wang E, Kumar Dutta S, Bhattacharya S, Das P, et al. Vascular endothelial growth factor receptor-1 modulates hypoxia-mediated endothelial senescence and cellular membrane stiffness via YAP-1 pathways. *Front Cell Dev Biol.* 2022;10.
- Bloom SI, Islam MT, Lesniewski LA, Donato AJ. Mechanisms and consequences of endothelial cell senescence. *Nat Rev Cardiol.* 2022;20(1).
- Cabrera AP, Bhaskaran A, Xu J, Yang X, Scott HA, Mohideen U, et al. Senescence increases choroidal endothelial stiffness and susceptibility to complement injury: Implications for choriocapillaris loss in AMD. *Invest Ophthalmol Vis Sci.* 2016;57(14):5910–5918.
- Chala N, Moimas S, Giampietro C, Zhang X, Zambelli T, Exarchos V, et al. Mechanical fingerprint of senescence in endothelial cells. *Nano Lett.* 2021;21(12):4911–4920.
- Cheng CK, Wang N, Wang L, Huang Y. Biophysical and biochemical roles of shear stress on endothelium: A revisit and new insights. *Circ Res.* 2025;136(7):752–772.
- Keshavanarayana P, Spill F. A mechanical modeling framework to study endothelial permeability. *Biophys J.* 2024;123(3):334–348.
- Kliche K, Jeggle P, Pavenstädt H, Oberleithner H. Role of cellular mechanics in the function and life span of vascular endothelium. *Pflügers Arch.* 2011;462(2):209–217.
- Ma D, Ren J, Qian X. Enhanced viscoelasticity as a biomechanical signature in senescent vascular endothelial cells. *Cell Rep Phys Sci.* 2025;6(9):102844.
- Silvani G, Romanov V, Cox CD, Martinac B. Biomechanical characterization of endothelial cells exposed to shear stress using acoustic force spectroscopy. *Front Bioeng Biotechnol.* 2021;9.
- Singam A, Bhattacharya C, Park S. Aging-related changes in the mechanical properties of single cells. *Heliyon.* 2024;10(12):e32974.
- Walther BK, Sears AP, Mojiri A, Avazmohammadi R, Gu J, Chumakova OV, et al. Disrupted stiffness ratio alters nuclear mechanosensing. *Matter.* 2023;6(10):3608–3620.
- World Health Organization. Cardiovascular diseases.
- Zhai Y, Colmenarez JA, Mendoza VO, Dong P, Nunes K, Suh D, et al. Multiscale mechanical characterization of cornea with AFM, SEM, and uniaxial tensile test. *Proc ASME Int Mech Eng Congr Expo.* 2023;5.