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Erratum

Finite-Element Stress Analysis of a Multicomponent Model of Sheared and Focally-Adhered Endothelial Cells

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Correction to Figure 5: Figure 5B illustrates e_{yy} strain and Figure 5C illustrates e_{zz} strain. Caption should denote that e_{yy} strain is compressive upstream and tensile downstream of focal adhesions. e_{zz} strain is tensile upstream and compressive downstream of focal adhesions. Text referring to figure 5 should reflect this correction also.

Modeling of the Surface Glycocalyx

Endothelial cells are known to have a carbohydrate and plasma protein-rich surface glycocalyx the dimensions of which depend on media composition.[1] This layer may affect some aspects of mechanosensing by modulating the transfer of shear stress to the cell membrane or cytoplasm.[36,48,49] Although the presence or properties of the glycocalyx were not experimentally-assessed in this study, we wished to determine the role of the glycocalyx in modulating surface (e.g. membrane) and cytoplasmic stresses. Fluid–glycocalyx interaction was determined by finite element analysis of a 0.4 μm thick poroelastic surface layer, mechanically coupled to the cell surface. Inlet and exit pressures in the simulation space were identical to the model solved without the glycocalyx. Velocity profiles in the layer were computed using Brinkman theory and fluid–structure interaction was computed through a modification of a model proposed by Weinbaum et al.[55] Briefly, the Brinkman velocity, $\mathbf{u}_b$, was determined from:

$$\eta\nabla^2\mathbf{u}_b = \left(\frac{\eta}{K_p}\mathbf{u}_b + \nabla p\right)$$
$$\nabla\cdot\mathbf{u} = 0 \tag{7}$$

where the second equation is the continuity equation. NS and Brinkman velocities were matched at the upper boundary of the Brinkman layer. Fluid drag on the solid portion of the Brinkman layer was estimated by adding the drag force due to shear stress at the top of the brinkman layer to the force per unit volume due to drag on idealized vertical strands with N strands/unit area (Eq. 3 from [55]):

$$F(r) = \frac{\pi\eta U(r)r^2}{cK_p}$$
$$c = \frac{2\pi r_f^2}{\sqrt{3}(2r_f + \Delta)} \tag{8}$$

where $F(r)$ is force on a strand as a function of distance, r, from the membrane, $U(r)$ is the local (Brinkman) velocity, r_f is the radius of a strand (6 nm), c is the fiber volume fraction (calculated as 0.326), K_p is the Brinkman permeability (3.157×10^{-18} m^2), and Δ is the inter-strand spacing (8 nm). If each strand is centered on a vertex of an equilateral triangle with sides of length $2r_f + \Delta$, the number of strands per area (strand density) is 2.89×10^{15} strands/m^2 (1/2 strand/triangle*1/area of triangle). The effective force per unit volume was computed using the strand density and Eq. 8. When integrated over the simulated monolayer surface, y-direction force on the cell surface corresponding to the membrane was 626 pN when the glycocalyx was considered and 338 pN when it was absent. Thus the drag enhancement (ratio of integrated force with and without glycocalyx) is approximately 1.85. On the abluminal side the enhanced drag is approximately 1.5. (Drag enhancement reduces to 1.67 and 1.38 on the apical and basal sides of cell, respectively, if shear stresses at the top surface of Brinkman layer are neglected and only Eq. 8. is used to compute drag on the Brinkman layer.) If the top surface of the

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glycocalyx was subjected to all of the shear stress, then mechanical equilibrium would dictate that the overall drag on the cell would be the same with or without the glycocalyx. However, because of increased surface area contributed by vertical strands, there exists transmission of flow from the free stream to the upper surface of the Brinkman layer resulting in enhanced drag. This analysis suggests that the presence of a glycocalyx constitutes an additional mechanism for stress amplification in vascular endothelial cells. It should be pointed out, however, that this analysis only applies to studies of cells in flow chambers or in large arteries where the presence of the glycocalyx does not appreciably alter the pressure gradient (in contrast to capillaries). Also, much about the glycocalyx is unknown and its role in mechanotransduction is controversial. For example, recent studies suggest that the glycocalyx is important for some aspects of mechanotransduction and not for others.[16,42] However, from our analysis, it is suggested that experiments which denude endothelial cells of the glycocalyx or experiments which fail to use plasma components in the perfusates, may unintentionally alter the stress distribution in the cell and at FAs.