

Abstract Submitted
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The wall traction induced by flowing red blood cells in model microvessels and its potential mechanotransduction¹ JONATHAN FREUND, University of Illinois at Urbana-Champaign, JULIEN VERMOT, IGBMC, CNRS/INSERM/UdS — There is evidence in early embryonic development, even well before advective oxygen transport is important, that the presence of red blood cells *per se* trigger essential steps of normal vascular development. For example, Lucitti *et al.* [*Development* **134**, 3317 (2007)] showed that sequestration of blood cells early in the development of a mouse, such that the hematocrit is reduced, suppresses normal vascular network development. Vascular development also provides a model for remodeling and angiogenesis. We consider the transient stresses associated with blood cells flowing in model microvessels of comparable diameter to those at early stages of development ($6\mu\text{m}$ to $12\mu\text{m}$). A detailed simulation tool is used to show that passing blood cells present a significant fluctuating traction signature on the vessel wall, well above the mean stresses. This is particularly pronounced for slow flows ($\lesssim 50\mu\text{m/s}$) or small diameters ($\lesssim 7\mu\text{m}$), for which root-mean-square wall traction fluctuations can exceed their mean. These events potentially present mechanotransduction triggers that direct development or remodeling. Attenuation of such fluctuating tractions by a viscoelastic endothelial glycocalyx layer is also considered.

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