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Research Interests

In the cardiovascular system, mechanical forces play a significant role in regulating many biological and physiological functions. Vascular endothelial cells reside on the interior wall of blood vessels where they form a continuous lining of the cardiovascular system. As a result of the pulsatile nature of blood flow, these cells continuously encounter a complex distribution of forces (e.g., shear, hoop, axial, and normal stress) that are variable throughout the circulation and depend on the local geometry, elasticity of the vessel wall, peripheral resistance, and heart rate. Under physiological conditions, the endothelium is responsive to this diverse mechanical environment and plays an active role in the regulation of acute vascular responses (e.g., vascular reactivity, inflammation, coagulation) and the chronic maintenance of the homeostatic environment for resident tissues.

Our laboratory is interested in identifying molecular mechanisms by which biomechanical forces, generated within the cardiovascular system, are sensed by vascular endothelial cells, and how this sensing regulates endothelial cell activity and phenotype. The importance of this research is motivated, in part, by achieving a basic understanding of how mechanical stresses exerted on the endothelium can modulate the cell's ability to adapt to local hemodynamic and biochemical stimuli under physiological and pathological conditions (e.g., heart disease, stroke, vascular remodeling).

Current research projects are focused on identifying the basic cellular mechanisms of mechanotransduction (i.e., how an externally applied mechanical force activates intracellular biochemical signaling events) and understanding how the cell utilizes these mechanisms to sense distinct aspects of the flow environment.

To investigate our hypotheses the lab uses several interdisciplinary approaches and tools in cell and molecular biology and engineering. We currently are using novel in vitro flow models capable of reproducing the dynamic flow conditions experienced in the human arterial and venous circulation and exposing them to vascular cells in culture. A microscope mounted model allows us to monitor these cells in real-time (phase/DIC/fluorescence) under unique and well-defined flow paradigms.

Recent Publications

Hastings NE, Feaver RE, Lee MY, Wamhoff BR, Blackman BR
Human IL-8 regulates smooth muscle cell VCAM-1 expression in response to endothelial cells exposed to atheroprone flow.

Jin L, Hastings NE, Blackman BR, Somlyo AV
Mechanical properties of the extracellular matrix alter expression of smooth muscle protein LPP and its partner palladin: relationship to early atherosclerosis and vascular injury.

Thomas JA, Deaton RA, Hastings NE, Shang Y, Moehle CW, Eriksson U, Topouzis S, Wamhoff BR, Blackman BR, Owens GK
PDGF-DD, a novel mediator of smooth muscle cell phenotypic modulation, is upregulated in endothelial cells exposed to atherosclerosis-prone flow patterns.

Anderson CR, Hastings NE, Blackman BR, Price RJ
Capillary sprout endothelial cells exhibit a CD36 low phenotype: regulation by shear stress and vascular endothelial growth factor-induced mechanism for attenuating anti-proliferative thrombospondin-1 signaling.

Harry BL, Sanders JM, Feaver RE, Lansey M, Deem TL, Zarbock A, Bruce AC, Pryor AW, Gelfand BD, Blackman BR, Schwartz MA, Ley K
Endothelial cell PECAM-1 promotes atherosclerotic lesions in areas of disturbed flow in ApoE-deficient mice.

More Publications