**Sample poster abstract:**

**Tension on PECAM-1 Elicits Global Mechanotransduction**

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Mechanical forces regulate cell behavior and function during development, differentiation, and tissue morphogenesis. In the vascular system, forces produced by blood flow are critical determinants not only of morphogenesis and function, but also pathological states such as atherosclerosis. Endothelial cells (ECs) have numerous mechanotransducers, including platelet endothelial cell adhesion molecule-1 (PECAM-1) at cell-cell junctions and integrins at cell-matrix adhesions. However, the processes by which forces are transduced to biochemical signals and subsequently translated into downstream effects are poorly understood. Here, we examine mechanochemical signaling in response to direct force application on PECAM-1. We demonstrate that localized tensional forces on PECAM-1 result in, surprisingly, global signaling responses. Specifically, force-dependent activation of phosphatidylinositol 3-kinase (PI3K) downstream of PECAM-1 promotes cell-wide activation of integrins and the small GTPase RhoA. These signaling events facilitate changes in cytoskeletal architecture, including growth of focal adhesions and adaptive cytoskeletal stiffening. Taken together, our work provides the first evidence of a global signaling event in response to a localized mechanical stress. In addition, these data provide a possible mechanism for the differential stiffness of vessels exposed to distinct hemodynamic force patterns *in vivo*.

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