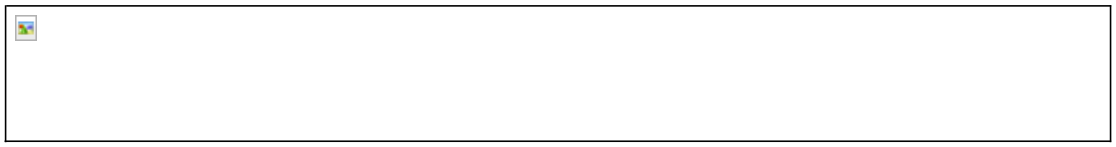

9 Flow Mechanotransduction Regulates Traction Forces, Intercellular Forces, and Adherens Junctions.

Ting LH, Jahn JR, Jung JI, Shuman BR, Feghhi S, Han SJ, Rodriguez ML, Sniadecki NJ.
Am J Physiol Heart Circ Physiol.2012 Mar 23; 302(11):H2220-9

Deborah Leckband, University of Illinois at Urbana-Champaign, IL, USA. F1000 Cell Biology

8 MUST READ

23 Apr 2012 | New Finding, Technical Advance

This interesting study demonstrates how flow modulates not only cell-cell junctional forces and consequent morphology but also the global distribution of stress in endothelial cells. Prior studies of stress distributions in endothelial cells considered how the balance of traction forces affected cell-cell tugging forces and junction size, but in the absence of fluid flow. Other studies considered how flow modulated junctional tension and signaling, but did not investigate changes in traction forces. This paper is interesting because these authors combined traction force measurements with fluid flow to assess how flow properties regulate the balance of stress in endothelial cells.

The findings demonstrate that flow alters endothelial cell contractility, as measured from traction force maps, and that this intracellular stress distribution in turn regulates cell-cell tension and endothelial barrier properties.

Competing interests
None declared

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George Truskey, Duke University, NC, USA. F1000 Physiology

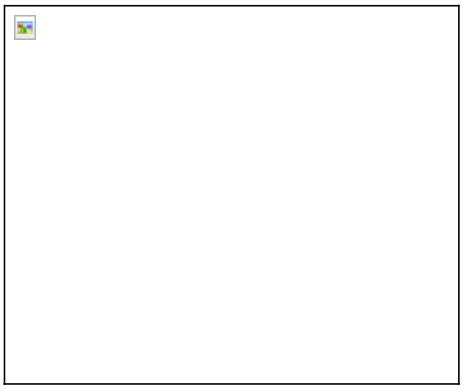
6 RECOMMENDED

02 Jul 2012 | New Finding

This is the first study to report the traction forces and intercellular forces for confluent endothelial monolayers exposed to different flow fields for longer than 12 hours. The results show that steady laminar flow causes a 20-25% increase in the intercellular force with corresponding increases in the junction proteins beta-catenin and zonula occludens (ZO)-1, whereas a recirculating flow causes a decrease in the intercellular force and beta-catenin and ZO-1 present at the junctions. While treatments known to increase transendothelial permeability produce a significant elevation of intercellular forces {1}, the current results suggest that some increase in intracellular force is associated with an increase in adherens and tight junction proteins.

Cells exert stress on the substrate and on neighboring cells. Stresses between individual cells and substrate depend upon the type and amount of ligand and the elasticity of the substrate. While the details regulating the traction forces between individual cells and substrates are well established, less is known about the stresses exerted between cells in a monolayer, and the interaction between cell-cell stress and cell-substrate stresses. While several different methods are available to measure the traction forces (e.g. traction force microscopy, substrates with elastic pillars), the forces between cells cannot, now, be directly measured and the intercellular forces must be determined from a force balance. For two cells, the calculation of the intercellular force is straightforward; however, for a cluster of connected cells or a monolayer of cells, only the net intercellular force among all cells can be measured.

Ting et al. use elastomeric micropost array substrates developed by Chris Chen's group {2} to examine the traction forces and intercellular forces of porcine aortic endothelial cells exposed to different flow fields for 16 hours. The force on microposts is slightly greater for cells exposed to steady laminar flow than for cells under static conditions or exposed to a recirculating flow. Steady laminar flow also causes the force vectors on individual microposts to align in the direction of flow, whereas no preferred direction for the force vectors on microposts is found for cells under static conditions or exposed to the recirculating flow. These differences in the traction forces lead to greater intercellular forces that are associated with increased levels of the junction proteins beta-catenin and ZO-1 at the endothelial cell junctions. For cells exposed to steady laminar flow, the levels of beta-catenin in the junctions can be reduced by inhibiting Rho kinase, suggesting that the presence of junction proteins permits linkages with the actin cytoskeleton, thereby increasing intercellular tension.



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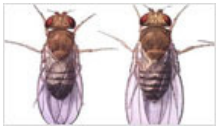

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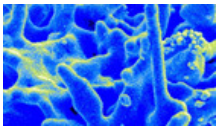

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Ruoning Wang, Douglas R. Green

This study is a first effort to understand how shear stresses influence intercellular force balances and thereby regulate the presence of junction proteins and transendothelial permeability. Future studies should examine the relationship between intercellular forces and permeability, and the relative contribution of different junction proteins to the intercellular force.

Competing interests
None declared

Cite this evaluation

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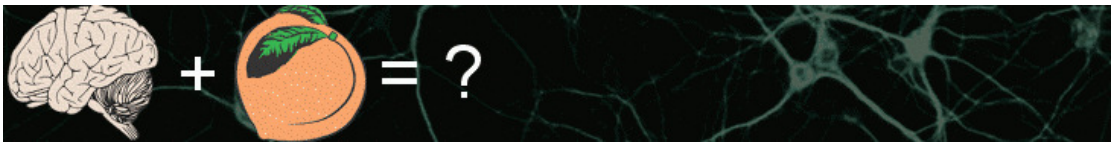

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