

Master Thesis: Morphology and elasticity of wild type endothelial cells and endothelial cells harboring BMPR2 mutation

Collaboration with MHH @ AG Olmers

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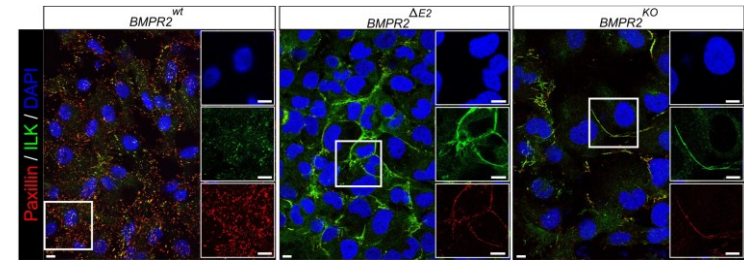
Institute of Cell Biology and Biophysics

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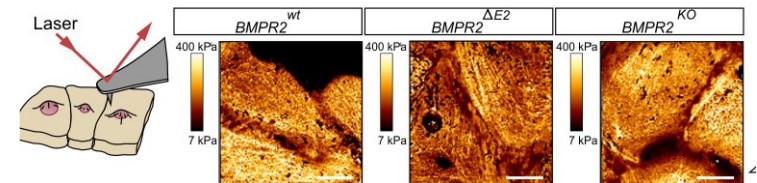
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Introduction

- Balanced transforming growth factor-beta (TGF β)/bone morphogenetic protein (BMP)-signaling is essential for tissue formation and homeostasis.
- BMP type 2 (BMP2) serves as a central gatekeeper of this balance highlighted by its deregulation in diseases such as pulmonary arterial hypertension (PAH).
- BMP2-deficient endothelial cells (ECs) exert increased matrix remodeling particularly at cell junctions, re-localization of mechano-complexes to cell junctions, and junctional stiffening.



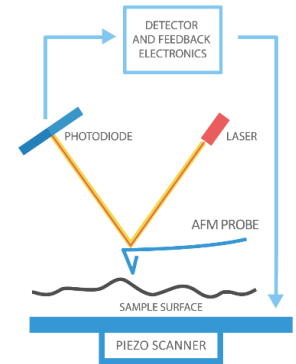
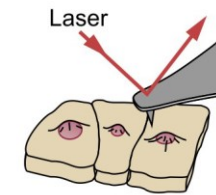
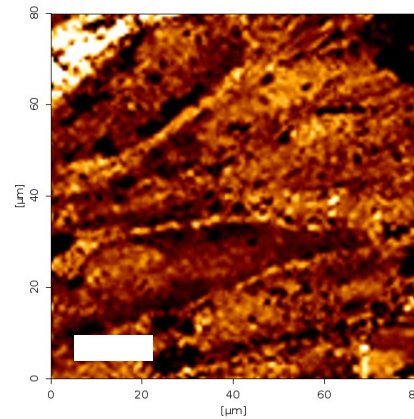
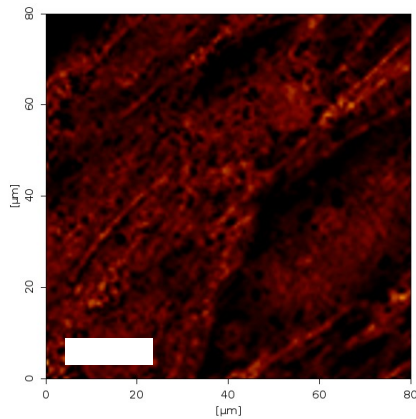
Mechano-complexes comprising β 1-integrin and ILK localize at cell junctions of BMP2-deficient ECs



EndMT and alterations in F-actin organization induce subcellular stiffening

Preliminary results

- Atomic Force Microscopy (AFM) – Quantitative imaging (QI) show differences in mechanical properties of wild type (WT) endothelial cells (iECs) and endothelial cells harboring BMPR2 mutation (PAH).



<https://www.nanoandmore.com/what-is-atomic-force-microscopy/>