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Stretch-induced actomyosin contraction in epithelial tubes: mechanotransduction pathways for tubular homeostasis

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Abstract

Many tissues in our body have a tubular shape and are constantly exposed to various stresses. Luminal pressure imposes tension on the epithelial and myoepithelial or smooth muscle cells surrounding the lumen of the tubes. Contractile forces generated by actomyosin assemblies within these cells oppose the luminal pressure and must be calibrated to maintain tube diameter homeostasis and tissue integrity. In this review, we discuss mechanotransduction pathways that can lead from sensation of cell stretch to activation of actomyosin contractility, providing rapid mechanochemical feedback for proper tubular tissue function.

Abbreviations

ATP, adenosine triphosphate; DAG, diacylglycerol; ECM, extracellular matrix; FAK, focal adhesion kinase; GAP, GTPase activating protein; GDI, guanine dissociation factor; GEF, guanine nucleotide exchange factor; GPCR, G protein-coupled receptors ; HUVEC, Human Umbilical Vein Endothelial Cells; IP_3, inositol 1,4,5-trisphosphate; MLC, myosin light chain; MLCK, myosin light chain kinase; MLCP, myosin light chain phosphatase; PIP_2, phosphatidylinositol 4,5-bisphosphate; PLC, phospholipase C; ROCK, Rho-associated protein kinase; TRPV, transient receptor potential vanilloid.