Intra- and intercellular signaling pathways in hypoxic pulmonary vasoconstriction

Hypoxic pulmonary vasoconstriction (HPV) is an evolutionary highly conserved physiological mechanism of vasotonus regulation in the pulmonary circulation by which the lung redistributes blood flow from poorly ventilated to better aerated areas in order to optimize ventilation-perfusion matching and thus, oxygenation. Although initially described more than 70 years ago, the intra- and intercellular signaling pathways that mediate HPV are still far from clear. Recent work in Dr. Kuebler’s laboratory has identified critical roles for a series of new signaling molecules including connexins, pannexins, cystic fibrosis transmembrane conductance regulator (CFTR), transient receptor potential (TRP) channels, and phosphatase and tensin homolog (PTEN) in the context of HPV. Intriguingly, these findings challenge the classic concept of the pulmonary artery smooth muscle cell as both sensor of hypoxia and effector in HPV, and provide novel mechanistic insights into the clinically detrimental loss of HPV in lung inflammation and infection.

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