Redox Signaling pathways in cardiovascular pathophysiology: hypoxia, HIF and novel therapeutic avenues

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Inadequate oxygen supply is a common characteristic for adaptation to high altitudes and various cardiovascular diseases. Manipulation of the cellular oxygen sensors, i.e. prolyl hydroxylase domain (PHD) enzymes, are thought to support the adaptation of the cardiac tissue towards hypoxia and ischemia. We analyzed mice, which mimic PHD inhibitor treatment for the potential of tissue protection (doi: 10.1161/CIRCRESAHA.120.318216). Aside from adaptation to the lack of oxygen, cardiac cells undergo striking changes that are redox-dependent modifications to regulatory proteins. An imbalance in the production of reactive oxygen species and an altered "redox state" is involved in many cardiac stress reactions that are involved in the development of heart failure. To further characterize the molecular consequences, we apply genetically encoded biosensors and characterize proteins in regard to specific redox modification as well as the consequences for cardiomyocyte metabolism (doi: 10.1038/s41467-023-37744-x.).