Commentary

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# Mitochondrial Dynamics and Oxidative Stress: Important Role in Chronic Hypoxia-Induced Pulmonary Hypertension from Adult to Neonates

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## Description

Pulmonary Hypertension (PH) presents a significant challenge across age groups, especially in connection to chronic hypoxia. Chronic Hypoxia-Induced Pulmonary Hypertension (CHPH), seen in both newborns and adults, leads to elevated pulmonary artery pressure and vascular remodeling, often associated with underlying lung diseases. Without treatment, CHPH can progress to right heart failure and mortality. In newborns, chronic hypoxia disrupts vital cardiovascular and respiratory adjustments, while in adults, conditions like chronic respiratory diseases or high-altitude living predispose to PH, highlighting the complexity of its pathogenesis. In this survey, we examine different parts of CHPH, zeroing in on mitochondrial brokenness, oxidative pressure, Hypoxia-Inducible Variables (HIFs), particle channels, aggravation, and miRNA dysregulation. The explanation of these systems not just gives experiences into the intricacies of CHPH, yet additionally sets out open doors for the advancement of creative medication procedures. The intricate network of pathways that play a role in the development of pH in both newborns and adults is deciphered in this section. By understanding the sub-atomic and cell systems at play, analysts plan to recognize novel focuses for drug improvement that could mediate at significant places in the movement of this condition.

### Disruptions in mitochondrial activity

Mitochondria are cell forces to be reckoned with vital in keeping up with ordinary cell homeostasis, and are basic for cell function. Disturbances in mitochondrial capabilities can bring about critical neurotic outcome. Long-term oxygen deficiency can disrupt the normal balance of mitochondrial fusion and fission, leading to endothelial dysfunction and structural changes in blood vessels associated with pulmonary hypertension.

In Pulmonary Hypertension (PH) research, the role of mitochondrial dysfunction in adults with Chronic Hypoxia-Induced PH (CHPH) is complex. Mitochondria, essential for producing Reactive Oxygen Species (ROS), are implicated in CHPH development. Studies show that mitochondrial ROS play a significant role, and interventions targeting them, like MitoQ, show promise in alleviating symptoms. However, there's debate-while acute hypoxia increases mtROS, chronic hypoxia may not, suggesting adaptation or methodological issues. Understanding mitochondrial energy metabolism in the lung under chronic hypoxia is vital. Lung mitochondria may function differently due to their high oxygen exposure, potentially leading to reduced energy function. Research indicates a drop in mitochondrial complex I activity in lung tissues after chronic hypoxia, suggesting reduced respiration.

More studies, including Seahorse flux analysis, are needed to fully grasp lung mitochondrial response to chronic hypoxia. Mitochondrial dysfunction plays a vital role in the development of chronic hypoxia-induced pulmonary hypertension in newborns. This condition is marked by increased reactivity in lung blood vessels, contributing to neonatal PH. Chronic hypoxia boosts ROS production by mitochondria, damaging endothelial cells and raising pulmonary vascular resistance, particularly observed in neonatal rats. Studies highlight the significance of Mitochondria-Associated Membranes(MAMs) in regulating mitochondria during hypoxia, suggesting targeting MAMs as a promising treatment approach for hypoxia-induced PH. These findings underscore the importance of mitochondrial dynamics and offer a potential therapeutic avenue for managing hypoxic PH in neonates.

### Oxidative stress

Increased oxidative stress, frequently induced by chronic hypoxia, plays an important role in the development of pulmonary hypertension. Reactive Oxygen Species (ROS), primarily from mitochondrial electron transport chains and NADPH oxidase enzymes, contribute to pulmonary vascular remodeling, endothelial dysfunction, and inflammation. Uncoupled Endothelial Nitric Oxide Synthase (eNOS) also generates ROS under hypoxia, worsening endothelial dysfunction. This oxidative imbalance leads to increased pulmonary vascular resistance, a hallmark of PH pathology. In adults with PH, chronic hypoxia-induced ROS, mainly from increased NADPH oxidase expression, drive disease progression by promoting vascular remodeling. ROS-targeted therapies, like 2-Hydroxybenzylamine, show promise in preclinical

models and are undergoing Phase II trials for PH treatment, aiming for potential commercialization. Neonatal lungs face heightened ROS risk due to circulatory changes at birth, leading to oxidative stress and endothelial dysfunction in newborn Pulmonary Hypertension (PH). ROS-induced vascular abnormalities in neonates, exacerbated by eNOS uncoupling, can result in long-term structural changes. Targeted antioxidant therapies, like SOD and superoxide scavengers, show promise in correcting vascular dysfunction and improving outcomes in neonatal PH.