Autophagy: Chapter 21. Autophagy and NADPH Oxidase Activity Tend to Regulate Angiogenesis in Pulmonary Artery Endothelial Cells with Pulmonary Hypertension

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Persistent pulmonary hypertension of the newborn (PPHN) occurs in one in 500 live births, with a high mortality rate. Inadequate pulmonary artery relaxation and decreased blood vessel density in the lungs are the primary causes of this type of pulmonary hypertension. Intrauterine ductus arteriosus ligation of fetal lambs during late gestation is the most commonly used animal model to study PPHN. Increased reactive oxygen species (ROS) formation in lungs, pulmonary arteries, and pulmonary artery endothelial cells (PAECs) is a common observation in this model. Uncoupled endothelial nitric oxide synthase, increased NADPH oxidase activity, decreased MnSOD expression and activity, and increased mitochondrial superoxide formation contribute to the increased ROS and decreased nitric oxide (NO) bioavailability in this model. Nitric oxide is an important mediator for angiogenic factor-mediated blood vessel formation; thus decreased NO formation, at least in part, explains the impaired angiogensis in PPHN. Pulmonary artery endothelial cells from PPHN sheep demonstrate a phenotype of increased autophagy and impaired angiogenesis. Antioxidants, or knocking down either Beclin 1 or p47phox, mitigate the impaired angiogenesis. Chemical inhibition of autophagy in PPHN PAECs decreases the association between gp91phox and p47phox, with improvement in angiogenesis. Data indicate a cross-talk between autophagy and NADPH oxidase activity in the developing lungs with PPHN, which plays an important role in regulating angiogenesis.

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