Lung tissue expression of angiotensinogen in ren-2 rats
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Pulmonary hypertension (PH) is a rapidly progressive disease that results in death. PH is characterized by increased vascular resistance, vascular remodeling, and right ventricular hypertrophy. Increased levels of ANG II lead to increased vasoconstriction, an increase in the number of vascular smooth muscle cells, hypertrophy, fibrosis, and vascular inflammation. A major contributor to PH could be lung tissue overexpression of the RAS because the RAS is a cascade of enzymes and peptides that ultimately for ANG II. Angiotensinogen is an inactive peptide produced by the liver and circulated systemically. ANG II is produced through the enzymatic reaction of renin cleaving angiotensinogen forming angiotensin I which is then converted into ANG II by angiotensin converting enzyme (ACE). In this study, we will look at lung tissue levels of angiotensinogen and where it is primarily found in the lung using indirect immunofluorescence microscopy. The lung sections will be from transgenic (TGR (mREN-2)27 (Ren-2) rats which overexpress ANG II and have been found to exhibit PH at 8-9 weeks of age. We hypothesize that angiotensinogen will be expressed throughout the lung particularly in the vascular wall, the endothelial cells and smooth muscle cells, in increased levels in Ren-2 rats compared to age matched Sprague-Dawley controls in which angiotensinogen is expressed at normal levels.