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Lung tissue expression of renin in ren-2 rats

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Pulmonary hypertension (PH), a rapidly progressive disease, is characterized by vasoconstriction, vascular remodeling, and right ventricular hypertrophy, leading to right ventricular failure and ultimately death. It is well known that locally increased production of angiotensin-II (ANG II) increases vasoconstriction, mitogenesis, fibrosis, inflammation and thrombosis during hypertension. Renin, a precursor enzyme in ANG II production, acts on the peptide angiotensinogen to form angiotensin-I (ANG I). ANG I is cleaved by angiotensin converting enzyme (ACE) to form ANG II. The Ren-2 rat is a transgenic strain that overexpresses mouse renin in a number of tissues. We recently determined that the Ren-2 rat exhibits PH, presumably as a consequence of transgene expression in the lungs, which could lead to elevated levels of ANG II in the lung. Overexpression of ACE in the intracinar arteries of patients with PH supports the hypothesis that locally increased production of ANG II leads to PH. We hypothesize that mouse renin will be expressed in the lung vasculature of Ren-2 rats, specifically in endothelial and smooth muscle cells. Confirmation of this hypothesis would support the presence of the mouse renin transgene in the Ren-2 rat and suggests the presence of a local RAS in the lungs.

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