Opposing Roles of Endoglin and Soluble Endoglin in Cardiac Remodeling and Heart Failure

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Conclusions

Reduced expression of mEng limits TGFβ1 signaling, collagen synthesis and cardiac fibrosis.

Overexpressing sEng also limits TGFβ1 signaling, collagen synthesis and cardiac fibrosis.
Impaired Endoglin function is associated with a pro-survival phenotype in pressure overload induced heart failure characterized by uncoupling of cardiomyocyte hypertrophy and LV fibrosis.
1. Identify Endoglin as an important contributor to the biology of cardiac remodeling in heart failure.

2. Prior studies limiting total TGFb1 activity in heart failure have failed. Our studies support that targeting the subset of TGFb1 signaling mediated by Endoglin uncouples cardiac hypertrophy from fibrosis, thereby providing a potentially unique novel therapeutic approach for individuals with heart failure.
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