**ABSTRACT**

TGF-β1 induces HASM cell shortening and airway hyperresponsiveness through a Smad3-dependent signaling pathway.

Christie A. Ojiaku, Gaoyuan Cao, Wangu Zhu, Steven S. An, and Reynold A. Panettieri Jr.

1Department of Systems Pharmacology and Translational Therapeutics, University of Pennsylvania, Philadelphia, PA
2Rutgers Institute for Translational Medicine and Science, Rutgers, The State University of New Jersey, New Brunswick, NJ
3Johns Hopkins Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD

**HYPOTHESIS**

TGF-β1 signaling induces AHR by directly modifying Ca2+ dependent or Ca2+ sensitization pathways in HASM cell E-C coupling.

**REFERENCES**


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**NAMESAKE**

St. Stephen's Lab at Johns Hopkins University

**SUMMARY**

- TGF-β1 induces hPCLS bronchoconstriction and AHR
- TGF-β1 augments basal and agonist-induced HASM cell shortening and MLC phosphorylation via TβRI
- TGF-β1 induces HASM cell shortening through a ROCK-dependent, RhoA-independent pathway
- Smad3 knockdown decreases TGF-β1-induced HASM cell shortening and ROCK activation

**SIGNIFICANCE**

- TGF-β1 may induce AHR through Smad3-dependent ROCK activation.
- TGF-β1 release following repeated airway injury-repair may lead to increased bronchomotor tone and sustained airway hyperresponsiveness.
- Further elucidation of this pathway may lead to the development of novel therapeutic targets for moderate and severe persistent asthma.