This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Idiopathic pulmonary fibrosis (IPF)	Integrin α_3 (ITGA3; VLA3); β -catenin (CTNNB1); SMAD family member 2 (SMAD2)	Studies in mice and in patients suggest that interfering with the CTNNB1-SMAD2 complex could be useful for treating IPF. Lung tissue from IPF patients showed accumulation of CTNNB1-SMAD2 complexes, whereas lung tissue from control patients did not. Lung tissue from wild-type fibrotic mice showed Itga3-dependent Ctnnb1-Smad2 complex formation, whereas mice without Itga3 lacked complex formation. Mice with lung epithelial cell-specific deletion of <i>Itga3</i> did not develop fibrosis, whereas wild-type mice did. Next steps include further dissecting the pathway of the ITGA3-dependent CTNNB1-SMAD2 interaction. SciBX 2(3); doi:10.1038/scibx.2009.113 Published online Jan. 22, 2009	Unpatented; unlicensed	Kim, K. <i>et al. J. Clin. Invest.</i> ; published online Dec. 22, 2008; doi:10.1172/JCI36940 Contact: Harold A. Chapman, University of California, San Francisco, Calif. e-mail: hal.chapman@ucsf.edu