This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Pulmonary disease				
Cystic fibrosis (CF)	Ribosomal protein S6 kinase 1 (RPS6K1); CD80; major histocompatibility complex type II (MHC II); prostaglandin D2 receptor (CD294; CRTH2; GPR44); lipoxin A_4 (LXA_4); α_1-antitrypsin (α_1AT)	An *ex vivo* study suggests that targeting viable airway neutrophils could slow or prevent progression of CF. Flow cytometry analysis of sputum from CF patients showed that viable, not necrotic, neutrophils in CF airways actively released elastase that damaged pulmonary tissue. Thus, inhibiting elastase release with LXA_4, an endogenous anti-inflammatory molecule derived from arachidonic acid, could slow or prevent pulmonary damage. The study also found higher levels of a cell survival factor, phospho-RPS6K1, in the neutrophils, suggesting they were longer lived than normal neutrophils. Additionally, increased expression of other cell surface receptors (CD80, MHC II, CD294) suggests that immunomodulatory cross-talk between T cells and neutrophils plays a role in CF airway inflammation. Ongoing work will assess the therapeutic implications of the increased RPS6K1 and examine whether T cell–neutrophil cross-talk mitigates or exacerbates CF airway inflammation. Kamada Ltd. has Kamada API, a formulation of the elastase inhibitor α_1AT, in Phase II testing to treat CF.	Not patented; licensing status undisclosed	Tirouvanziam, R. *et al. Proc. Natl. Acad. Sci. USA*; published online March 3, 2008; doi:10.1073/pnas.0712386105 **Contact:** Rabindra Tirouvanziam, Stanford University School of Medicine, Stanford, Calif. e-mail: tirouvan@stanford.edu