Subacute TGFβ Exposure Drives Airway Hyperresponsiveness in CF Mice through the PI3K Pathway

Authors: Elizabeth L. Kramer[1,2], Satish K. Madala[1,2], Kristin M. Hudock[3,4], Cynthia Davidson[2], John P. Clancy[1,2]

[1]Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, OH

[2]Division of Pulmonary Medicine, Cincinnati Children's Hospital, Cincinnati, OH

[3]Division of Adult Pulmonary & Critical Care Medicine, University of Cincinnati, Cincinnati, OH

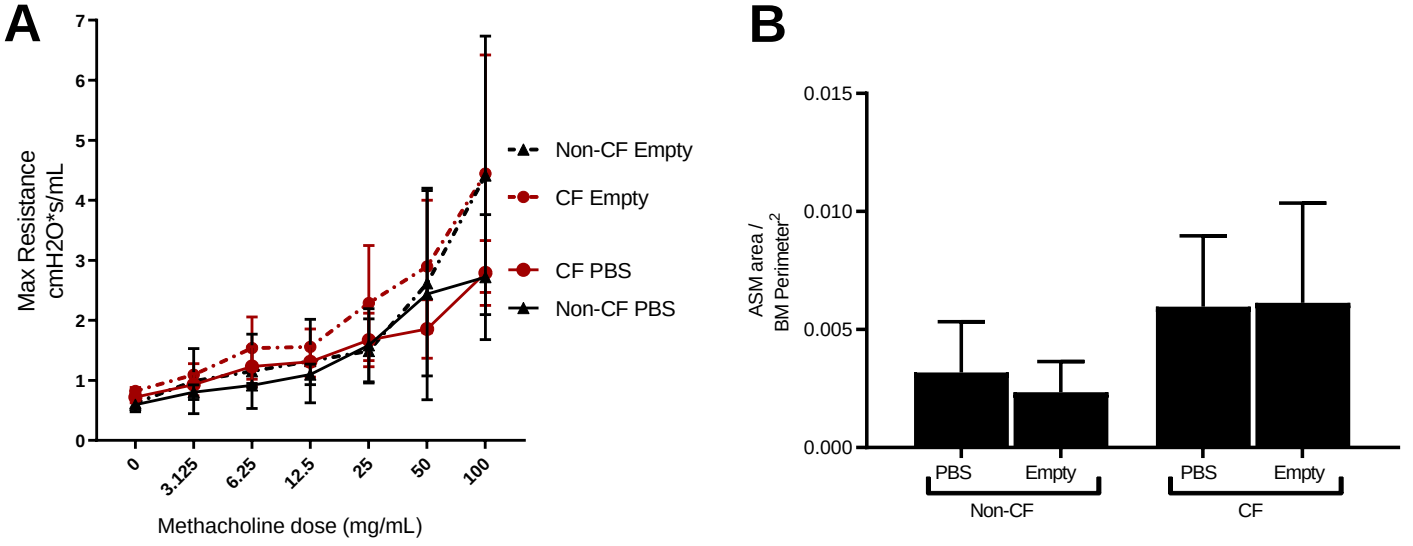
[4]Division of Pulmonary Medicine, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

Online Data Supplement

Supplemental Figure E1. A. Treatment with intratracheal Empty vector control virus at $5x10^7$ pfu did not induce airway hyperreactivity in either CF or non-CF mice compared to intratracheal PBS treatment at day 7. **B.** Treatment with Empty vector did not induce ASM area alterations in CF or non-CF mice.

Supplemental Figure E2. After pretreatment with intraperitoneal LY294002 (LY), PI3K signaling was not altered in cells recovered from bronchoalveolar lavage (BAL) from non-CF or CF mice one day after Ad-TGFβ exposure. Western blot analysis was performed on the BAL cell pellet. PI3K signaling was measured by phosphorylation of S6 and was similar in vehicle and LY294002 treated mice of both genotypes. Values are corrected to vehicle treated non-CF mice. NS indicates $p>0.05$ by two-tailed t-test.

Supplemental Figure E1



Supplemental Figure E2