through direct effects on the airway. Antigen presenting cells (APC) can process and present HDM antigens to activated T cells (Th1, Th2, Th17) that then release adaptive immune mediators and cytokines that contribute to the pathogenesis of allergic airway disease.

Supplemental Figure 1. Inhibition of EGFR, TACE, or p38 for 24 hours does not alter cell number or cell morphology in HBEC cultures. (A) HBEC were exposed to the highest dose used in the studies of Erlotinib (0.5 μM), AG1478 (0.5 μM), GM6001 (20 μM), TAPI-1 (20 μM), SB202190 (5 μM) for 24 hours, then cells were trypsinized and counted; single experiment performed in quadruplicate for each group. (B) Representative pictures of HBEC exposed to the highest dose of each inhibitor.

Supplemental Figure 2. IL-17A induced GM-CSF production by HBEC is dependent on transcriptional regulation. HBEC were stimulated with IL-17A (25 ng) with or without Actinomycin D (1 μg/mL). GM-CSF was measured in the media by ELISA from 30 minutes to 24 hours after stimulation, and statistical significance was determined by comparing samples to medium controls (* $p<0.001$) or compared to the IL-17A stimulated group (^^^ $p<0.001$); n=2 (two independent experiments, each performed in triplicate) for each group.**

Supplemental Figure 3. GM-CSF production by HBEC is not dependent on p38 signaling downstream of direct activation of EGFR signaling. HBEC were stimulated with amphiregulin (AREG; 72 nM) with or without the p38 inhibitor SB202190 (2 μM [+] or 5 μM [++]) or the MEK1/2 inhibitor ARRY162 (1μM [+]). GM-CSF was measured in the

media by ELISA 24 hours after stimulation, and statistical significance was determined by comparing samples to medium controls (* p<0.001) or compared to the Amphiregulin stimulated group (^^^ p<0.001); n=2 (two independent experiments, each performed in triplicate) for each group.**

Supplemental Figure 4. Inhibition of EGFR signaling with Erlotinib does not alter Th1, Th2, or Th17 cytokines in established allergic airway disease. Cytokine levels were measured in bronchoalveolar lavage fluid (BALF) by multiplex ELISA (A,B,D-F) or by qPCR for mRNA levels in lung homogenates (C). NS p>0.05*, p<0.05, ** p<0.01; n=6 for each group in (A-F), **which represents the total number of individual mice used in vivo.**

Supplemental Figure 5. Inhibition of EGFR signaling with Erlotinib decreases eosinophils but does not alter allergic sensitization in mice with established allergic airway disease. (A) Changes in the number of macrophages, lymphocytes, neutrophils and eosinophils were assessed after differential staining. HDM specific IgG (B) and IgE (C) were measured in serum samples from mice by ELISA. NS p>0.05, ** p<0.01, *** p<0.001; n=12-20 for each group in (A-C), **which represents the total number of individual mice used in vivo.**