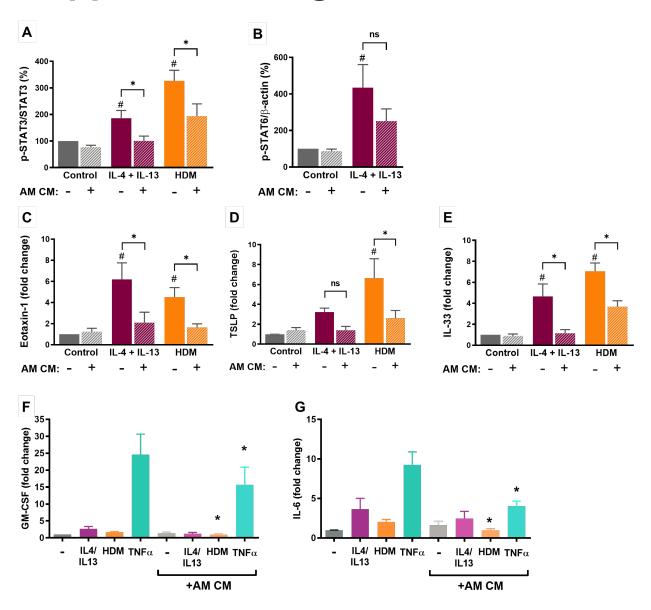
## **Supplemental Figure 2**



Supplemental Figure 2. AM-derived CM inhibits STAT activation and inflammatory gene expression in response to type 2 stimuli

(A) Activation of STAT3 (phosphorylated as percentage of total STAT3), and (B) activation of STAT6 (phosphorylated as percentage of  $\beta$ -actin) in BEAS-2B ECs pretreated with or without AM CM and thereafter stimulated with IL-4/IL-13 (10 ng/mL each) or HDM (10  $\mu$ g/mL) for 1 h.(C) Relative expression of eotaxin, (D) TSLP, (E) IL-33, (F) GM-CSF and (G) IL-6 mRNA in BEAS-2B ECs pretreated with or without AM CM and thereafter stimulated with IL-4/IL-13 (10 ng/mL each), HDM (10  $\mu$ g/mL) or TNF $\alpha$  (10ng/mL) for 6 h. All data represent fold change relative to untreated control, and are mean  $\pm$  SEM from 5-6 independent experiments. #p<0.05 as compared to not pretreated control ECs. \*p<0.05, using a one-way ANOVA followed by Sidak's multiple comparisons test.