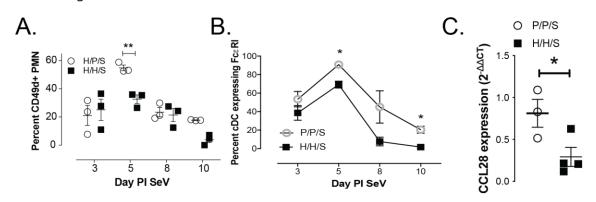
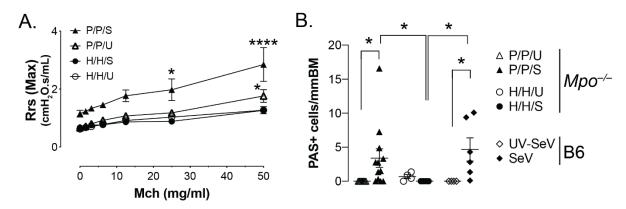
## **Supplemental material**

Supplemental Figure 1.



Supplemental Figure 1. Pre-existing atopy alters mechanistic pathway. SeV infected atopic mice (H/H/S) demonstrated a reduction in (A) CD49d<sup>+</sup> PMN in BAL and (B) high affinity IgE receptor, FcɛRI, expression on lung cDC compared to non-atopic mice (H/P/S). (C) *Ccl28* was also reduced in atopic mouse lungs at day 21 PI SeV (qRT-PCR analysis of *Ccl28* expression normalized to *Gapdh* measured as  $2^{-\Delta\Delta CT}$  of mRNA expression). n≥3 mice per group/time point; \*p<0.05, \*\*p<0.01, atopic versus non-atopic

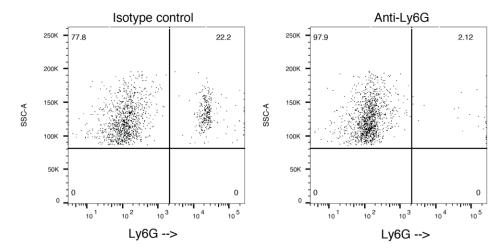
Supplemental Figure 2.



Supplemental Figure 2. Post-viral airway disease is prevented in atopic MPO deficient mice.

Mice (*Mpo*-/-) sensitized and challenged with HDM and infected with SeV (H/H/S) fail to develop (**A**) post-viral AHR (**B**) MCM, 21d PI SeV or UV-SeV (control). Note that infected non-atopic *Mpo*-/- mice (P/P/S) did develop increased AHR and MCM. For MCM, wild type C57BL6 mice (B6) are included for comparison. n=4 mice per group, \*p<0.05, \*\*\*\*p<0.0001 from baseline of each group.

## Supplemental Figure 3.



Supplemental Figure 3. Depletion of lung PMN with anti-Ly6G mAb. Cells harvested from lungs after 24h of treatment with isotype control or anti-Ly6G mAb. Representative dot plots of flow cytometry showing frequency of PMN in mouse lung 24h after isotype control (left) or anti-Ly6G mAb (right) given i.p.