Supplemental Figure 1

- □ WT Saline
- ■ WT OVA
- ● H1R−/− Saline
- ○ H1R−/− OVA

Penh vs. Methacholine [mg/mL]

Baseline Saline 1.0 3.3 10 33 100 333

**
Supplemental Figure 1: H1R−/− mice do not develop inflammatory airway hyperresponsiveness to antigen.

OVA immunized (OVA) or sham immunized (Saline) WT or H1R−/− mice were exposed to OVA by inhalation and studied for airway hyperresponsiveness. Airway hyperresponsiveness to methacholine was determined by whole body plethysmography. Data represents mean Penh ± SEM. n=9-10 mice. ** indicates p<0.01 by 2-tailed ANOVA.

Supplemental Figure 2: Dysregulated antibody isotype levels in H1R−/− mice

The levels of OVA-specific antibody levels in the serum of WT or H1R−/− mice was determined by ELISA, 31 days after i.p. immunization with OVA.

Supplemental Figure 3: IL-4 and IL-13 induce airway hyperresponsiveness in H1R−/− mice.

5 µg of recombinant mouse IL-4 or IL-13 or 5 µg of BSA was administered intranasally for 3 days. Airway hyperreactivity to methacholine in WT or H1R−/− mice was assessed by whole body plethysmography. Each group consisted of 5 individual mice. ** indicates p<0.01 by 2-tailed ANOVA.

Supplemental Figure 4: Airway hyperresponsiveness to antigen can be induced by WT T cells but not by H1R−/− T cells.

Antigen-expanded CD4+ T cells from WT or H1R−/− mice were adoptively transferred into naïve WT or H1R−/− recipients and the development of airway hyperresponsiveness to methacholine in WT or H1R−/− recipients was studied after inhaled OVA challenge. Data represents mean Penh ± SEM; n= 4-5/group.