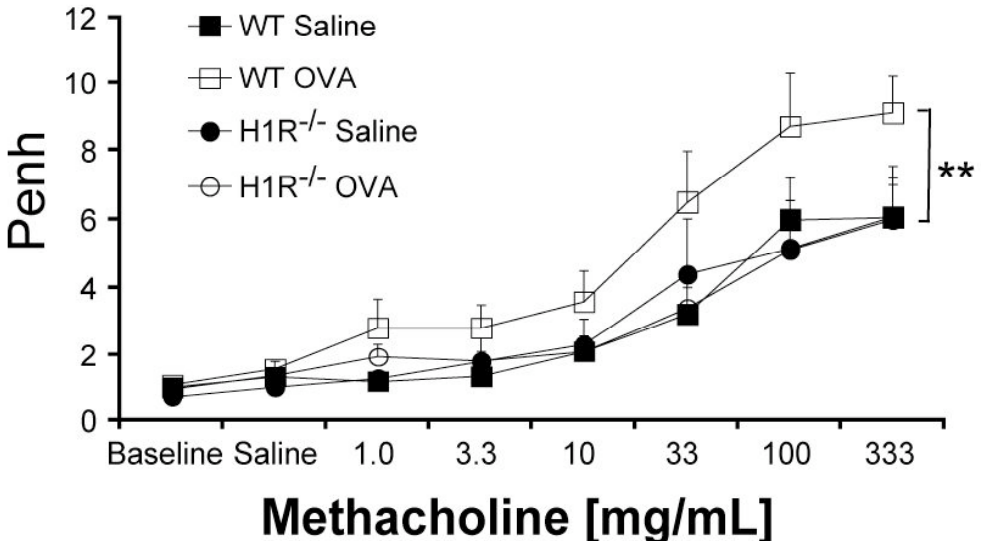
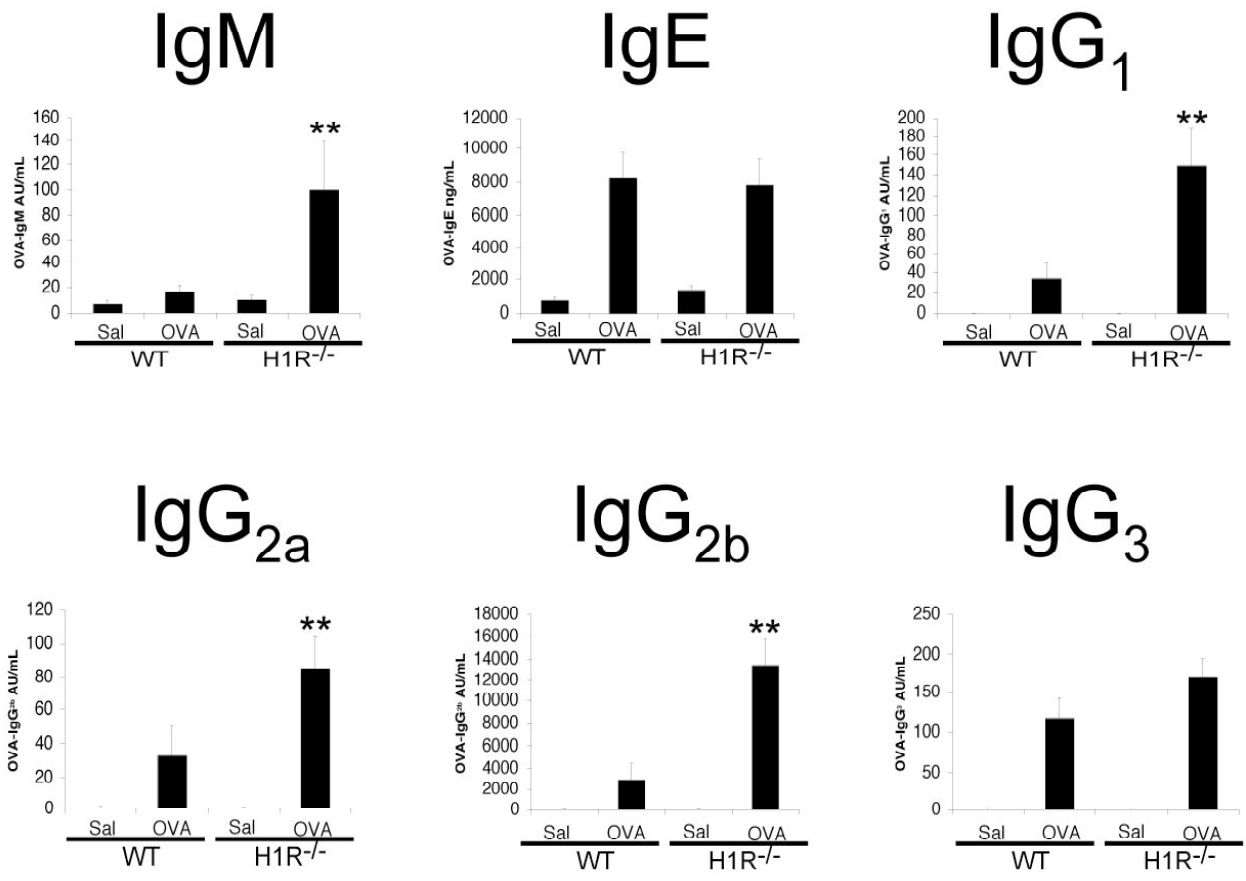
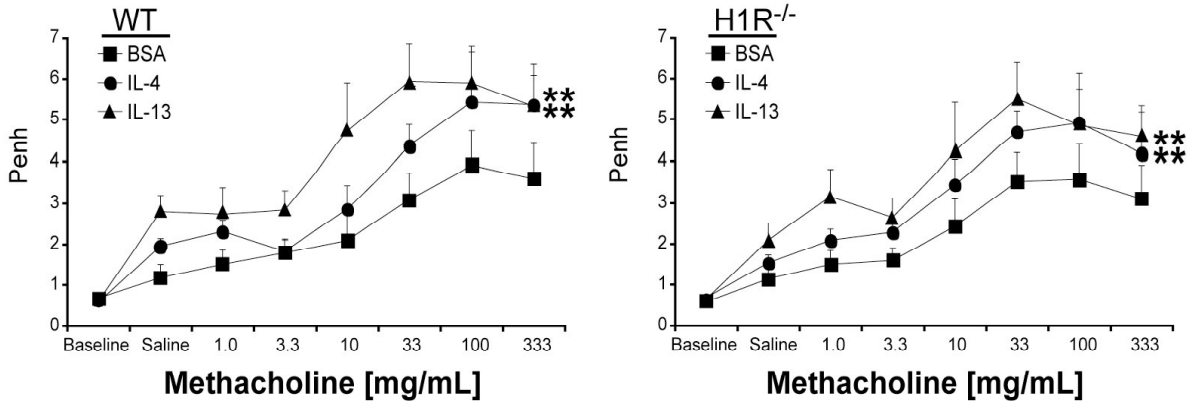
Supplemental Figure 1



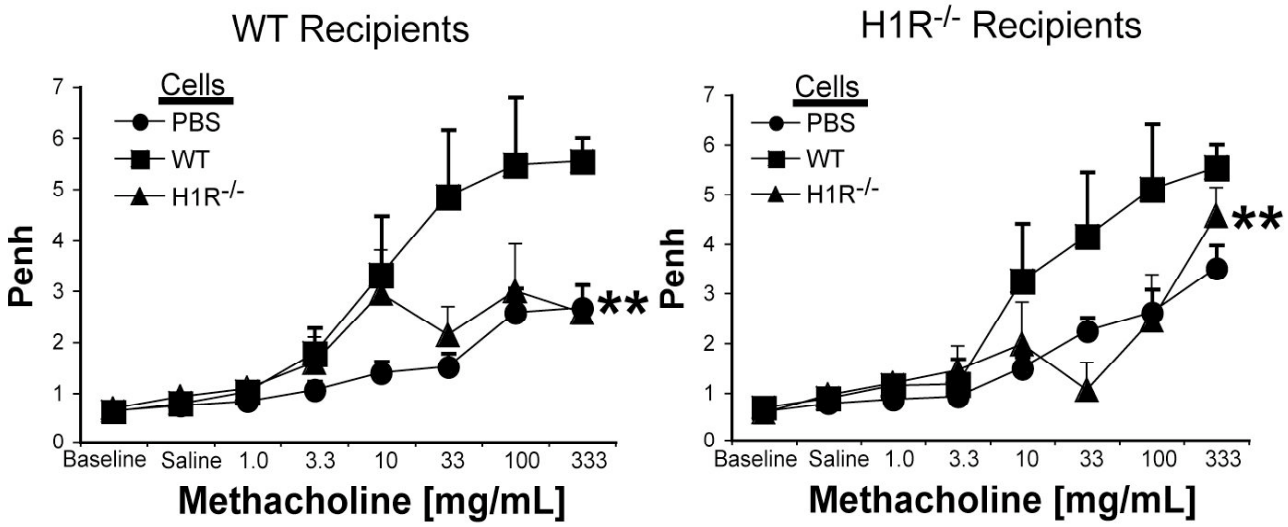
Supplemental Figure 2



Supplemental Figure 3



Supplemental Figure 4



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.

5μg of recombinant mouse IL-4 or IL-13 or 5μg 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.