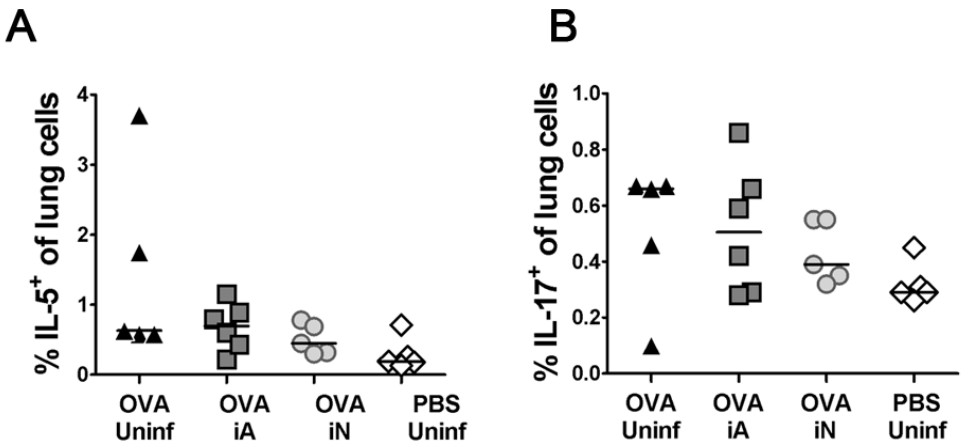
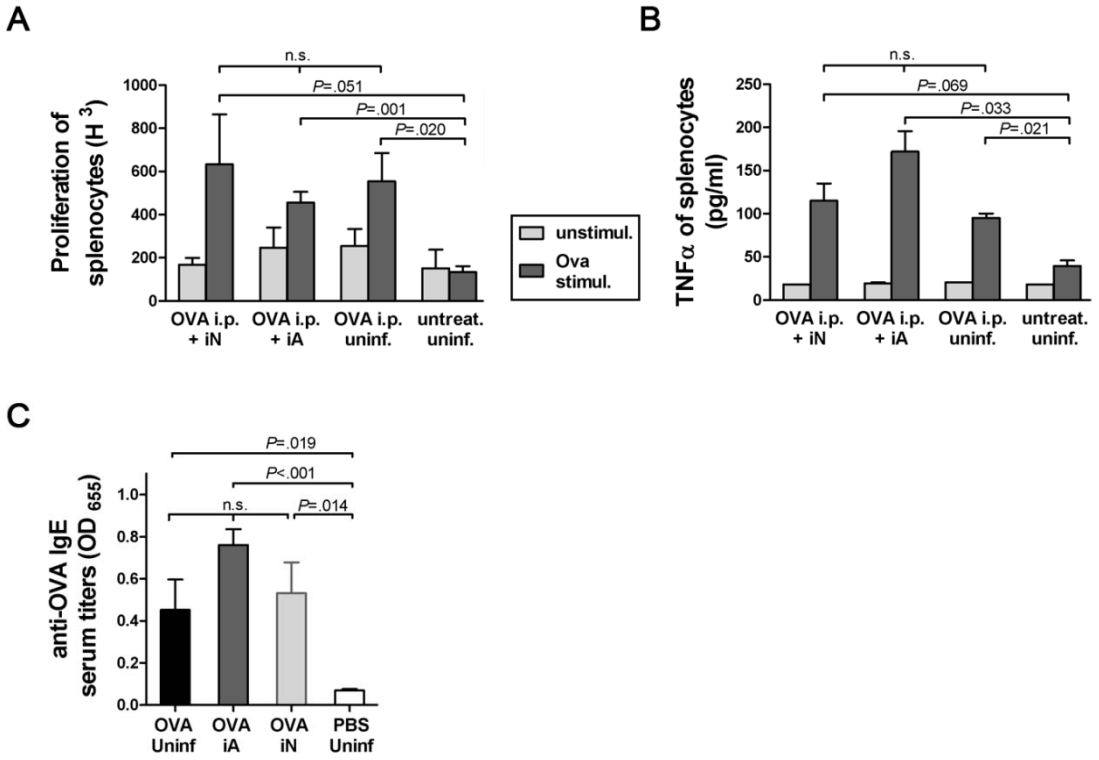
Supplemental Figure 1:



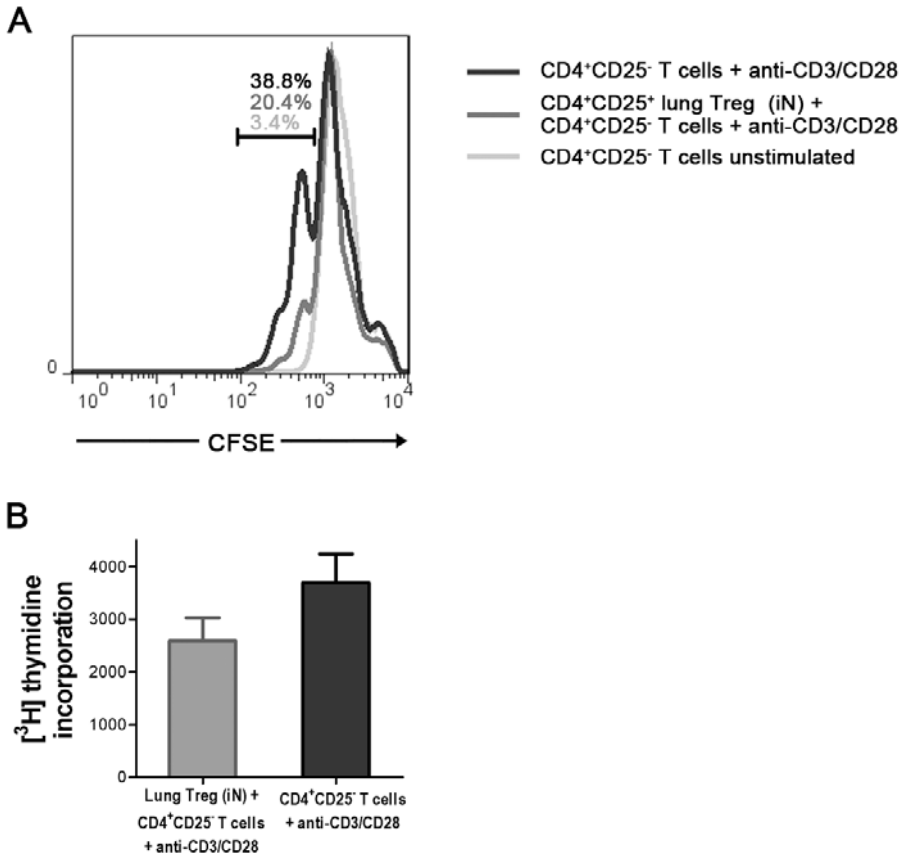
Suppl. Figure 1: Pulmonary Th2 and Th17 cell infiltration of ovalbumin-sensitized and challenged mice. Mice were orally infected as neonates (iN) or adults (iA) with *H. pylori* and sensitized with alum-adjuvanted ovalbumin 4 and 6 weeks p.i. along with an uninfected group ('OVA'). Two weeks after the second sensitization, all mice (including a mock-sensitized control group, 'PBS') were exposed to three consecutive daily doses of aerosolized ovalbumin. (**A,B**) Fraction of IL-5^+ (A) and IL-17^+ (B) cells in % of all lung cells as determined by intracellular cytokine staining of single cell preparations.

Supplemental Figure 2:



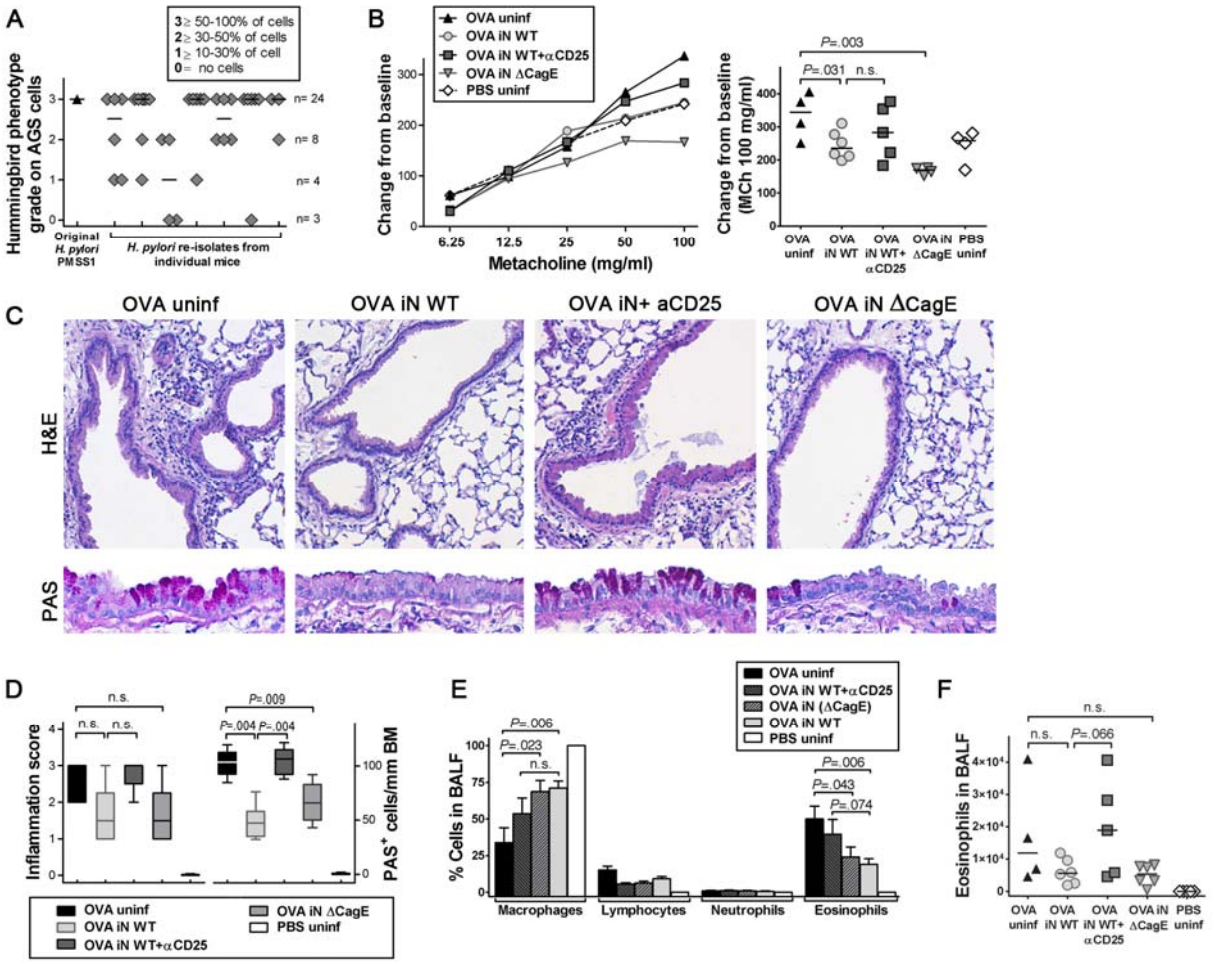
Suppl. Figure 2: Primary responses to allergen are not impaired in mice infected with *H. pylori*. Groups of 5 mice each were infected with *H. pylori* at either 6 days or 6 weeks of age (iN, iA), and sensitized with alum-adjuvanted ovalbumin 4 and 6 weeks p.i. along with an uninfected control group. An additional group remained untreated ('untreat.'). None of the mice were challenged with aerosolized ovalbumin. All mice were sacrificed 2 weeks after the last sensitization dose. (**A,B**) Splenocyte preparations of individual mice were incubated with 200 ng/ml ovalbumin for 4 days for the quantitative assessment of proliferation by [^{3}H] thymidine incorporation (A) and TNFα secretion by ELISA (B) (R&D Systems). (**C**) Serum from the same mice was assessed for ovalbumin-specific IgE by ELISA as follows: wells were coated with anti-mouse IgE (0.2 µg/well), incubated for 1h with 1:5 diluted serum, and detected with biotinylated ovalbumin (10µg/ml) followed by HRP-conjugated streptavidin. Conversion of TMB was recorded at 655 nm.

Supplemental Figure 3:



Suppl. Figure 3: Lung-infiltrating Tregs from protected mice have suppressive activity *ex vivo*. (**A,B**) $CD4^+CD25^+$ regulatory T-cells were retrieved by immunomagnetic isolation from pooled single cell suspensions of the five neonatally infected, ovalbumin-challenged mice as shown in Figure 1. Tregs were mixed at a 1:1 ratio with CFSE-labeled $CD4^+CD25^-$ effector T-cells prior to the addition of anti-CD3/anti-CD28-coated beads (Invitrogen). (**A**) The dilution of CFSE in the effector population with or without stimulation is shown in the presence or absence of Tregs. Percentages of $CFSE^{dim}$ cells are indicated. (**B**) The proliferation of identically treated co-cultures as determined by [^{3}H] thymidine incorporation.

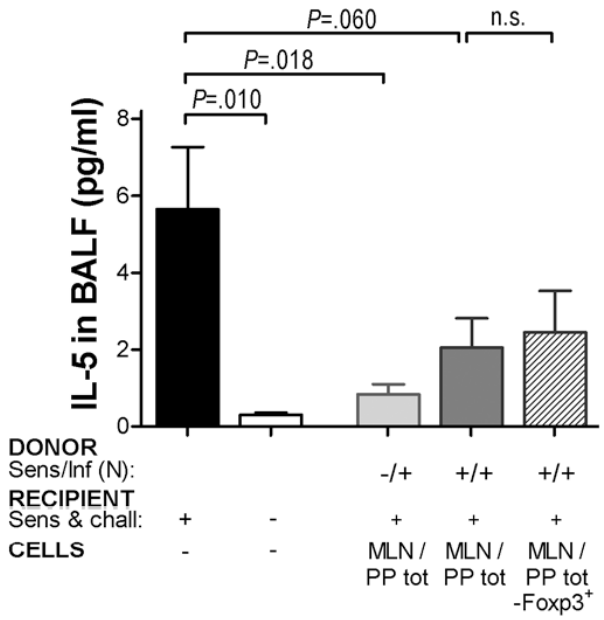
Supplemental Figure 4:



Suppl. Figure 4: Tregs are essential, whereas CagA delivery is not required for asthma protection of neonatally infected mice. (**A**) For the quantitative assessment of the CagA translocation proficiency of 39 clones re-isolated from 7 neonatally infected mice, sections of their stomachs were homogenized in Brucella broth and serial dilutions were plated on horse blood plates for obtaining single colonies as described (12). These were expanded in Brucella broth and used for 6h infections of human gastric adenocarcinoma cells (AGS, ATCC CRL 1739) at a multipiclity of infection of 200. The cellular elongation and scattering of AGS cells that represents a hallmark of CagA delivery by the Cag-PAI-encoded type IV secretion system was scored on a scale from 0-3 as indicated in the legend. Of the 39 assessed re-isolates, 24 had completely retained their ability to deliver CagA to AGS cells. CagA delivery by the original PMSS1 isolate served as a reference. (**B-F**) Mice were infected at 6 days of age with *H. pylori* PMSS1 wild type (WT) or a CagE-deficient isogenic mutant (ΔCagE). Asthma was induced by ovalbumin sensitization and challenge as described in Figure 1. One group of WT-infected mice received 3 doses of 100μg anti-CD25 antibody during ovalbumin challenge. (**B**) Airway hyper-responsiveness in response to increasing doses of inhaled metacholine and the highest dose of 100mg/ml, respectively. (**C,D**) Tissue inflammation and

goblet cell metaplasia as assessed on H&E and PAS-stained tissue sections. Representative micrographs are shown in C, and scores for all mice are shown in D. (**E,F**) Relative representation of the indicated cell types and absolute numbers of eosinophils in 1ml of BALF.

Supplemental Figure 5:



Suppl. Figure 5: Protection against ovalbumin-induced bronchoalveolar IL-5 secretion is conferred by the adoptive transfer of MLN/PP cells from infected donors. Groups of mice were sensitized with ovalbumin or remained untreated prior to intravenously receiving unsorted (total, 'tot') cell populations isolated from the MLN and PP of neonatally infected and/or ovalbumin-sensitized or Treg-depleted ('-FoxP3^{+}) donors. Treg depletion was achieved by administration of a single dose of diphtheria toxin to *foxP3*-EGFP-DTR-transgenic donors one day prior to cell isolation; the donors of Treg-proficient cell populations were non-transgenic littermates. All recipients as well as control groups (no cells) were nebulized with ovalbumin on days 2,3 and 4 post adoptive transfer and sacrificed 2 days after the last ovalbumin challenge. Bronchoalveolar IL-5 was quantified by cytometric bead array.