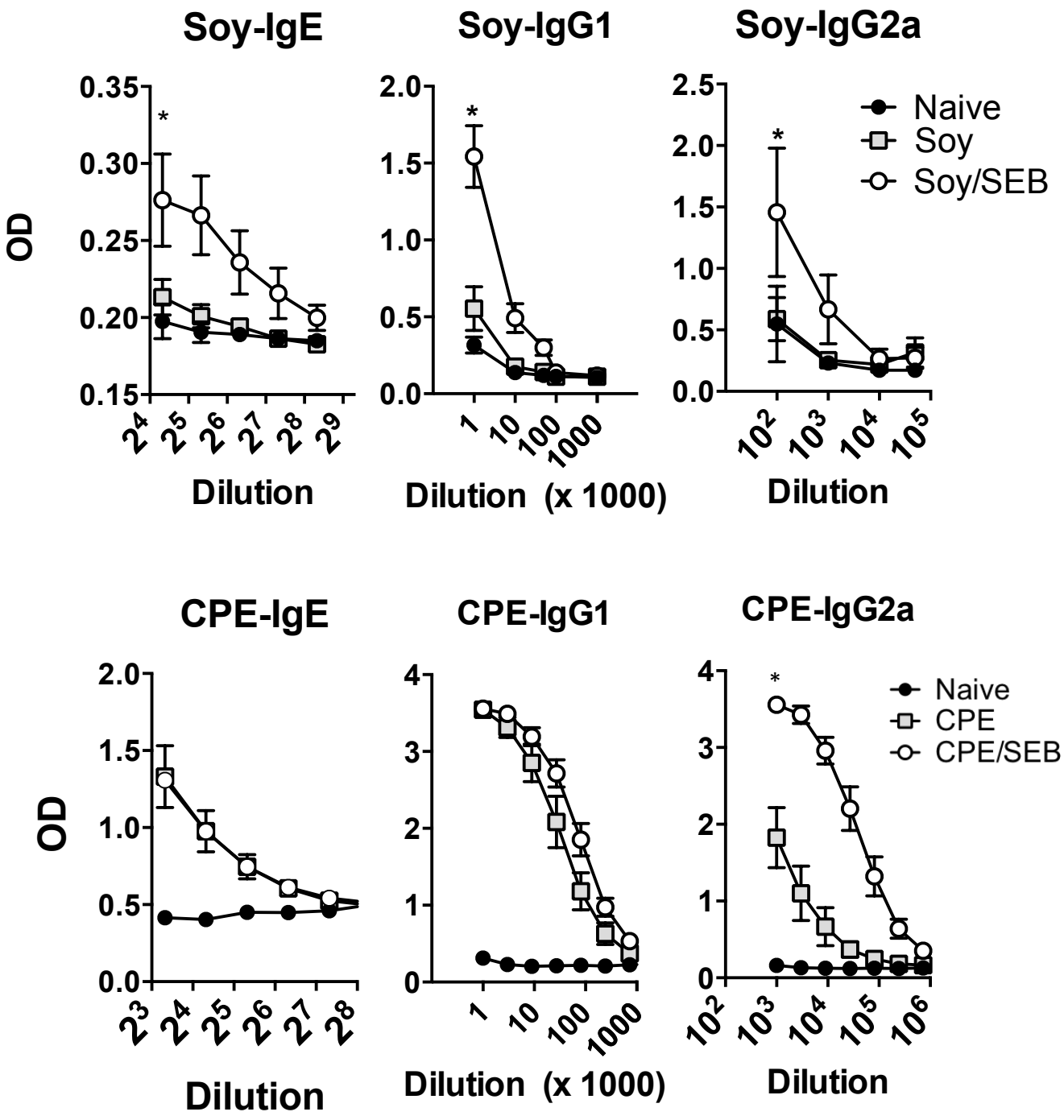
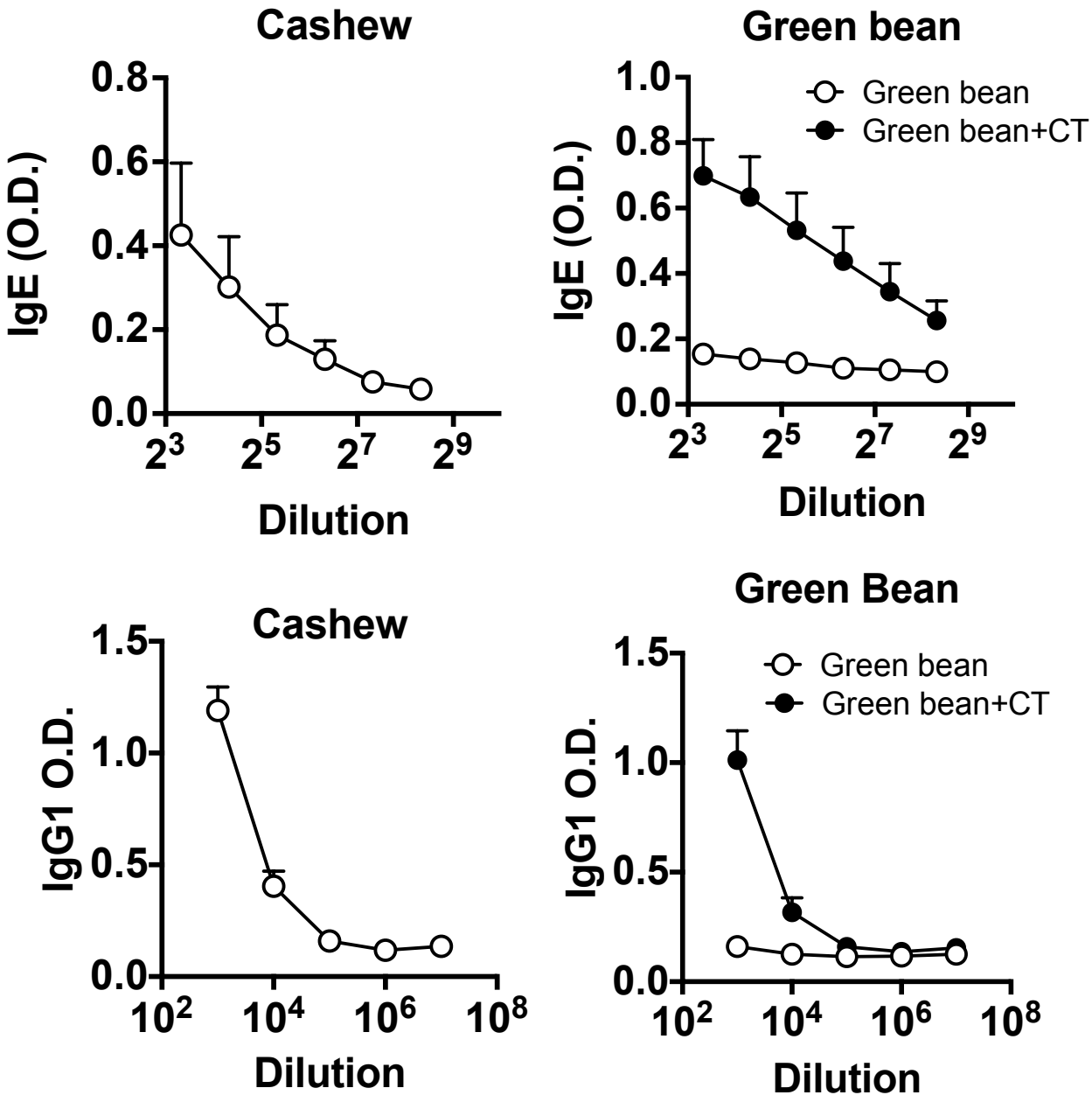
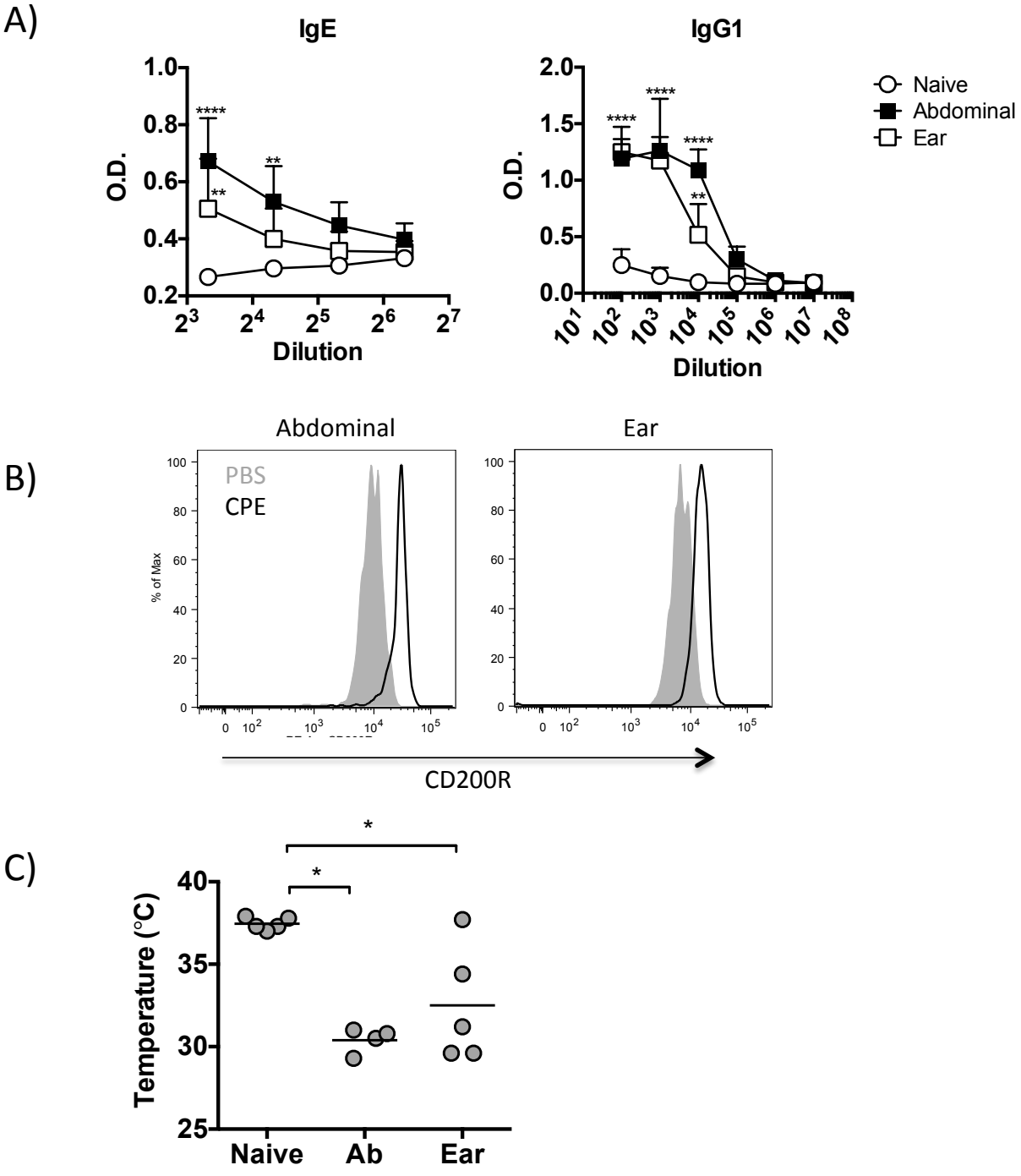


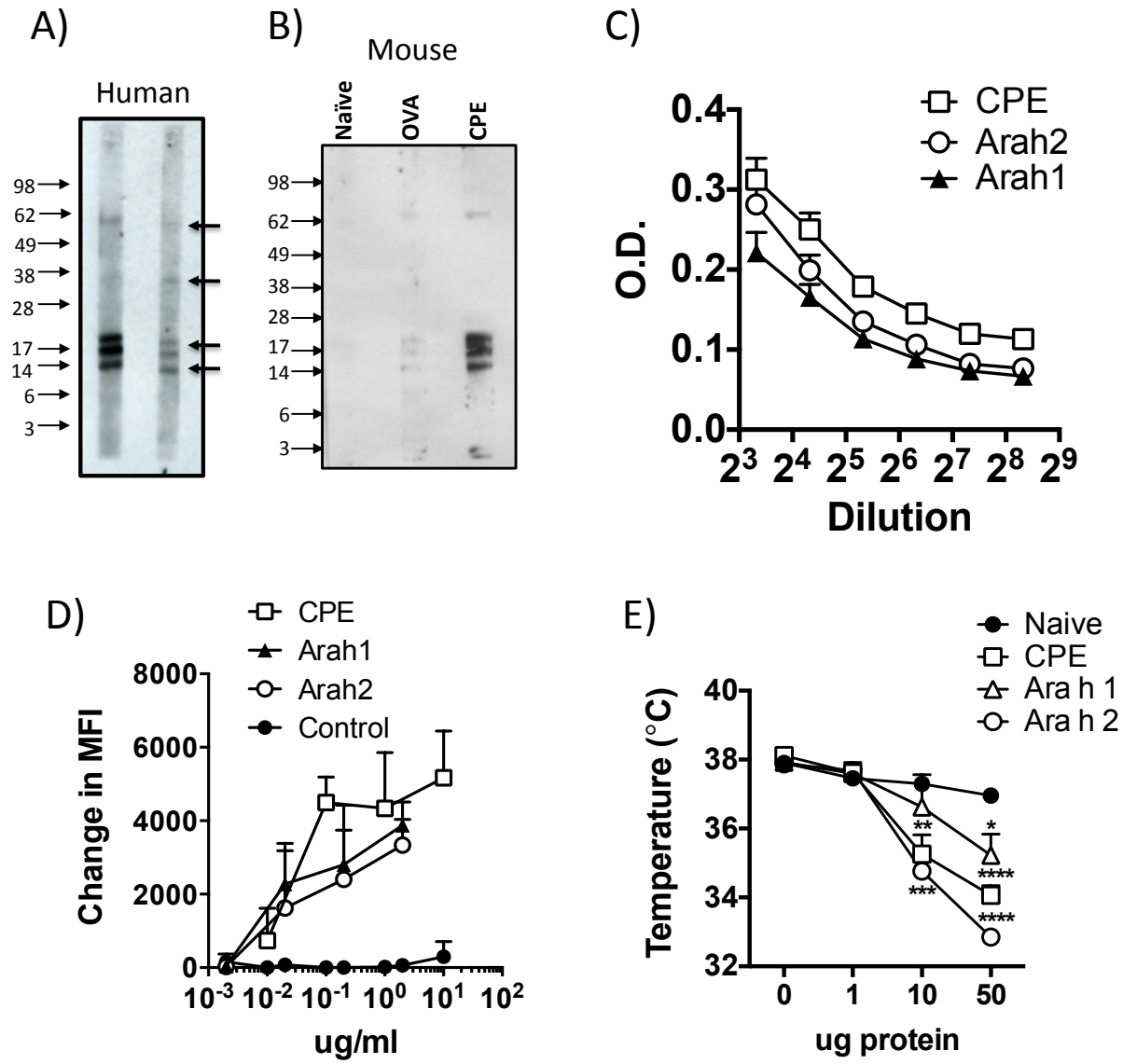
Supplementary Figure 1: Impact of SEB co-exposure on antibody responses to soy or peanut. Mice were topically exposed 1 mg of crude peanut extract (CPE) or soy extract weekly for 6 weeks in the presence or absence of 10 ug of SEB. Antigen-specific IgE, IgG1, and IgG2a were measured in serum. n = 5/group. Values for IgE and IgG1 in naïve and antigen alone groups are the same as are shown in Figure 1. *$p < 0.05$ compared to antigen alone.



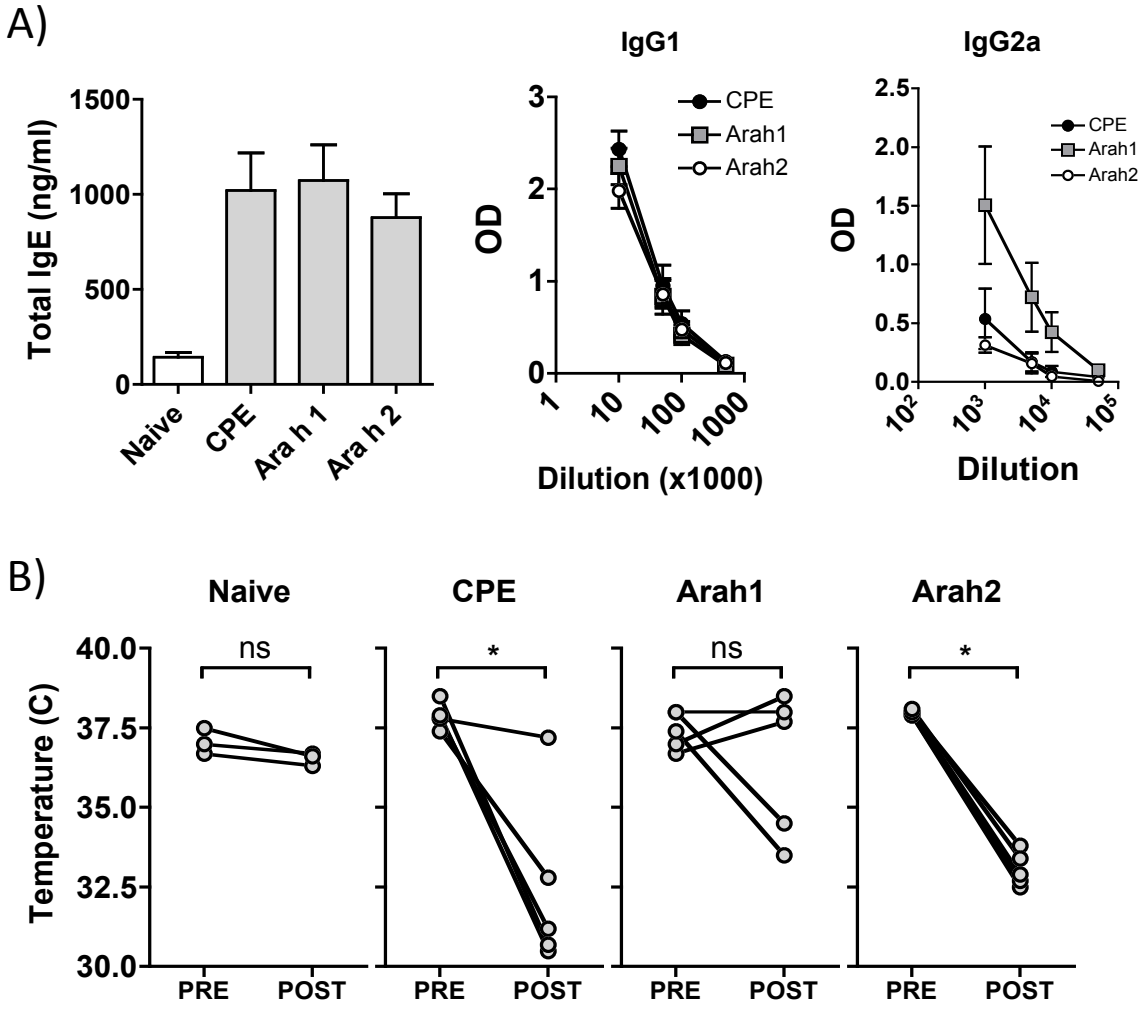
Supplementary Figure 2: In vivo allergenicity of food extracts. Mice were epicutaneously exposed to defatted cashew extract or green bean extract (in the presence or absence of the adjuvant cholera toxin, CT) weekly for 6 weeks. Serum samples were obtained and allergen-specific IgE (top) and IgG1 (bottom) were measured.



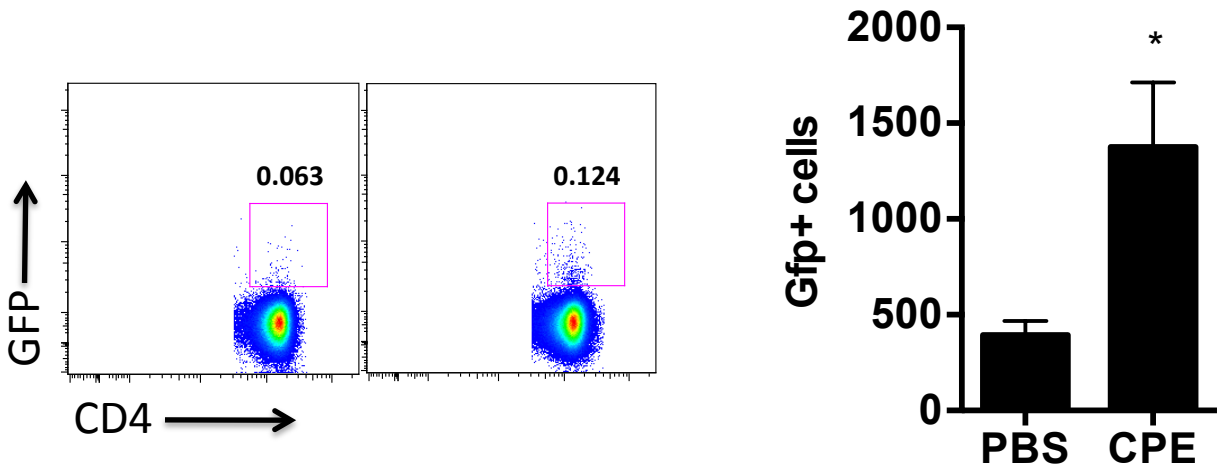
Supplementary Figure 3: Sensitization to peanut through topical exposure on the ear. Mice were exposed to CPE by painting the ear pinnae (Ear) or the abdominal region (Ab) once a week for 6 weeks. After 6 weeks, peanut-specific IgE and IgG1 were measured by ELISA (A), and peanut-induced basophil activation was measured by upregulation of CD200R (B). Anaphylaxis in response to ip challenge with 100 ug of peanut extract was measured by drop in body temperature 30 minutes after challenge (C). *p < 0.05, **p < 0.01, ****p < 0.0001 vs naïve.



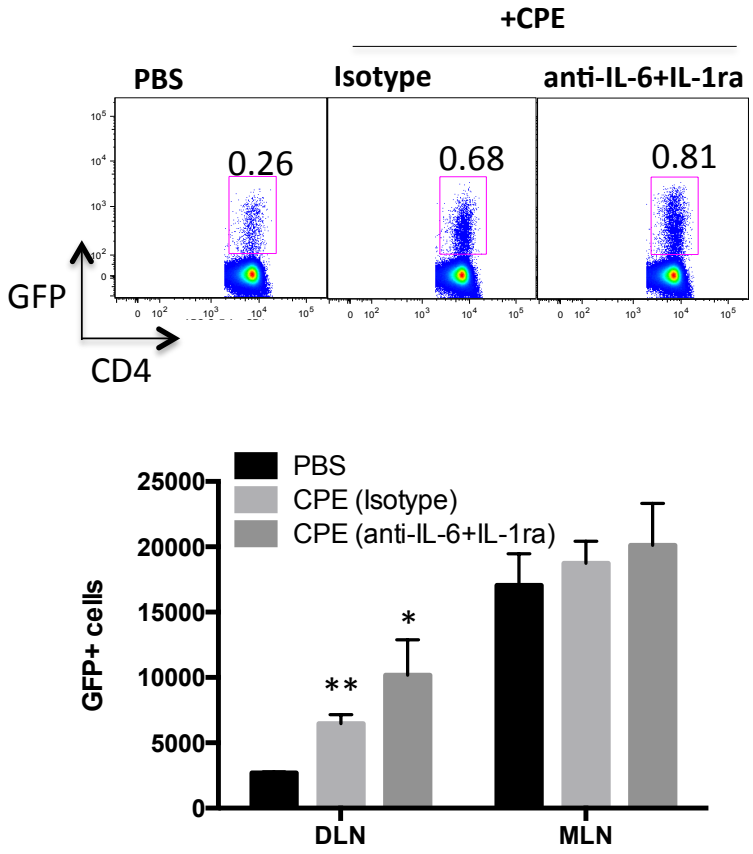
Supplementary Figure 4: Sensitization to peanut components. Crude peanut extract was run on an SDS gel, and immunoblotting was performed with serum from 2 peanut-allergic individuals (A), or with serum from mice (B) exposed to CPE, OVA as control, or from naïve mice. (C-E): Mice were epicutaneously exposed to 1 mg CPE weekly for 6 weeks, followed by challenge with CPE, Ara h 1, or Ara h 2 (5/group). (C) Allergen-specific IgE. (D) Basophil activation test. Change in MFI (media fluorescence intensity) was calculated with respect to the stimulation with media alone. Negative control was obtained by stimulating naïve sera with CPE, Ara h 1 or Ara h 2. (E) Body temperature in response to a graded challenge with increasing doses of CPE or purified allergens, given by ip injection. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, ****$p < 0.0001$ vs naïve.



Supplementary Figure 5: Sensitization using purified peanut components. A: Mice were epicutaneously exposed to 1 mg of CPE, or 0.1 mg of purified Ara h 1 or Ara h 2, once a week for 6 weeks. Mice were then challenged with their sensitizing antigen (0.1 mg of CPE or purified allergens by the intraperitoneal route). (A) Total IgE (left), and IgG1 and IgG2a against CPE, Arah1, or Arah2 in mice exposed to CPE, Arah1, or Arah2, respectively. (B) Anaphylaxis measured by drop in body temperature 30 minutes after challenge. *$p < 0.05$, ns – not significant.



Supplementary Figure 6: Induction of IL-13-expressing cells by peanut exposure. IL-13eGFP mice were epicutaneously exposed to CPE, and the draining lymph nodes were harvested 7d later. IL-13-secreting cells were assessed in total CD4+ population by flow cytometry. Representative plots are shown beside summary data (3/group) depicting number of GFP+ cells per million CD4+ T cells. *$p < 0.05$



Supplementary Figure 7: Blocking IL-6 and IL-1 does not diminish IL-4 reporter induction during CPE exposure. 4get mice were pre-treated with either isotype control or anti-IL-6+IL-1 receptor antagonist before epicutaneous exposure to peanut (CPE). A week later, DLN and MLN were harvested. Representative plots of IL-4-expressing cells in the DLN are shown above, and summary data showing number of cells per million CD4+ T cells is shown below. N=4/group. * $p < 0.05$, ** $p < 0.01$ compared to PBS.