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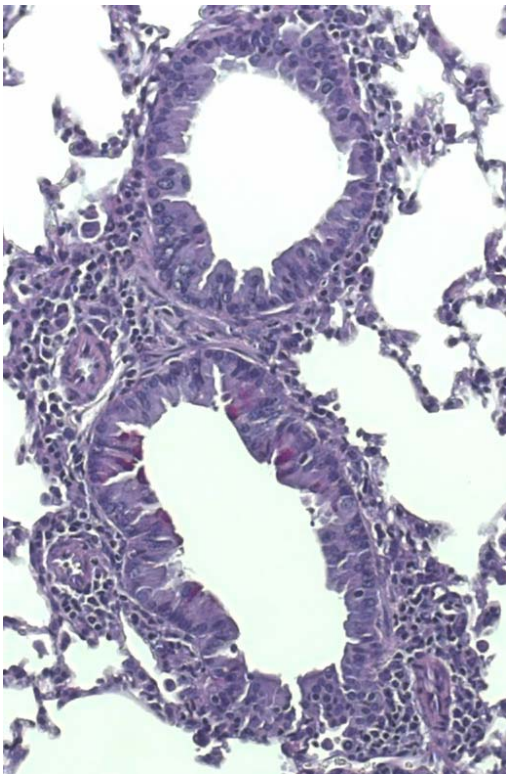

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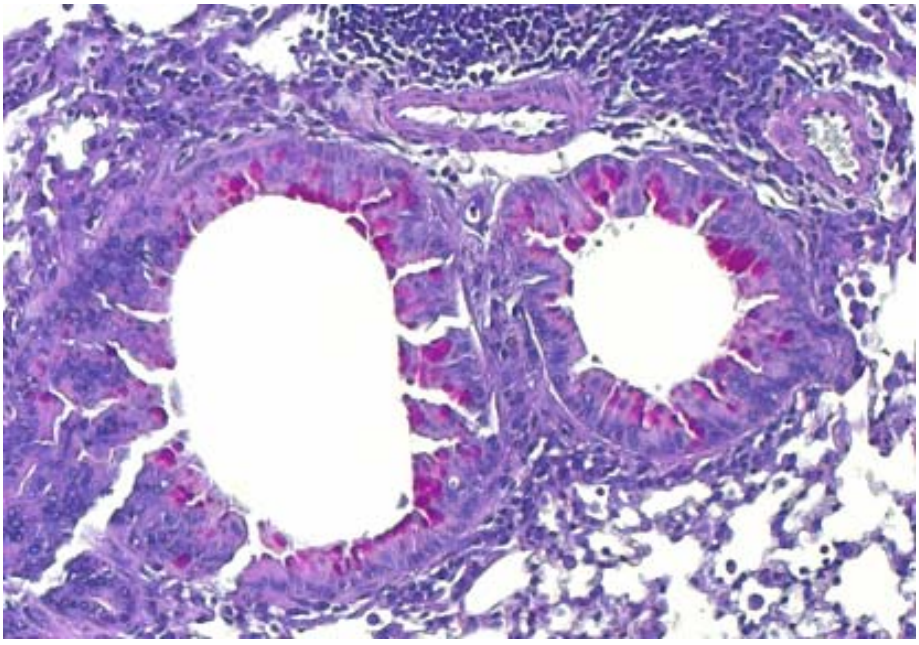
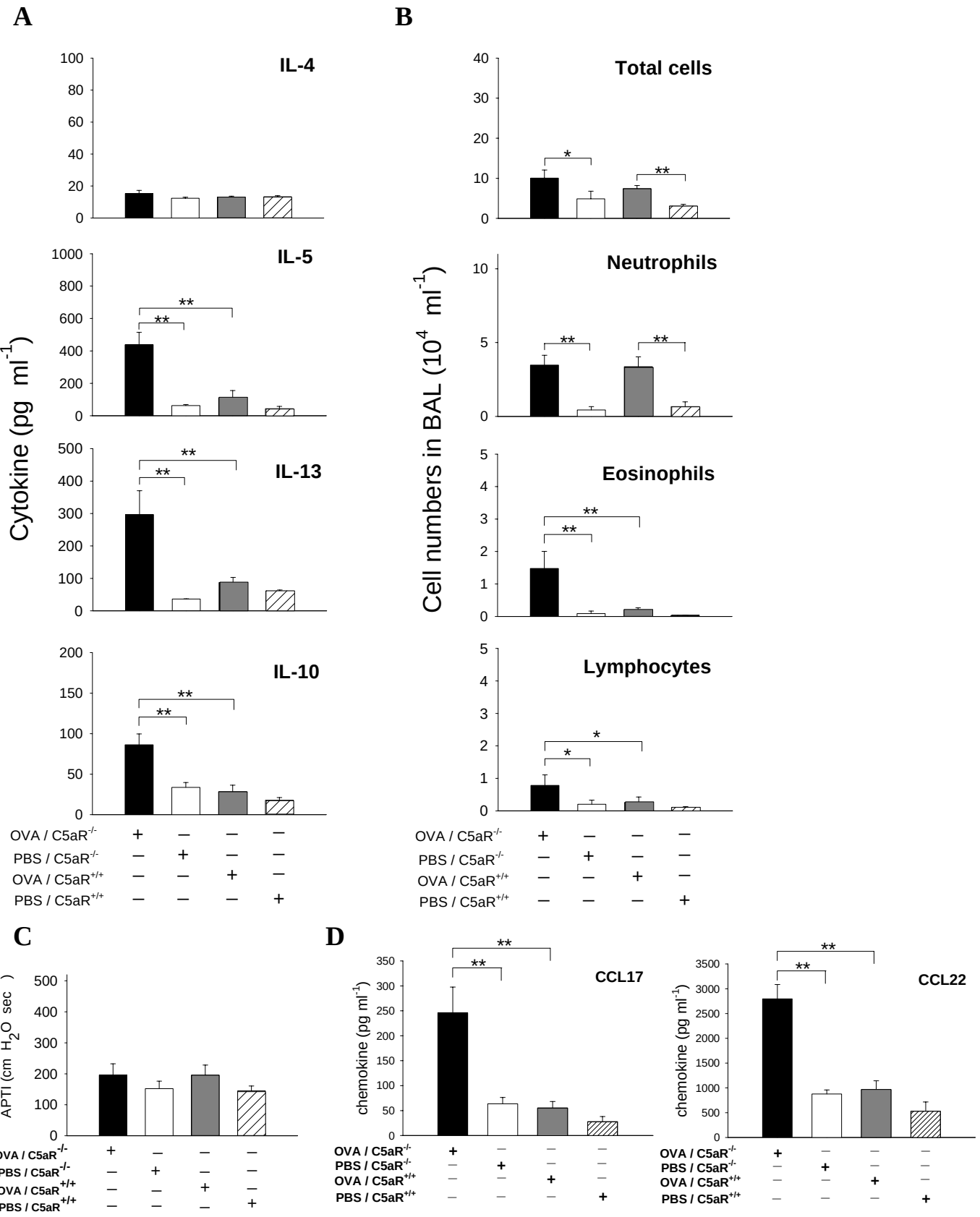
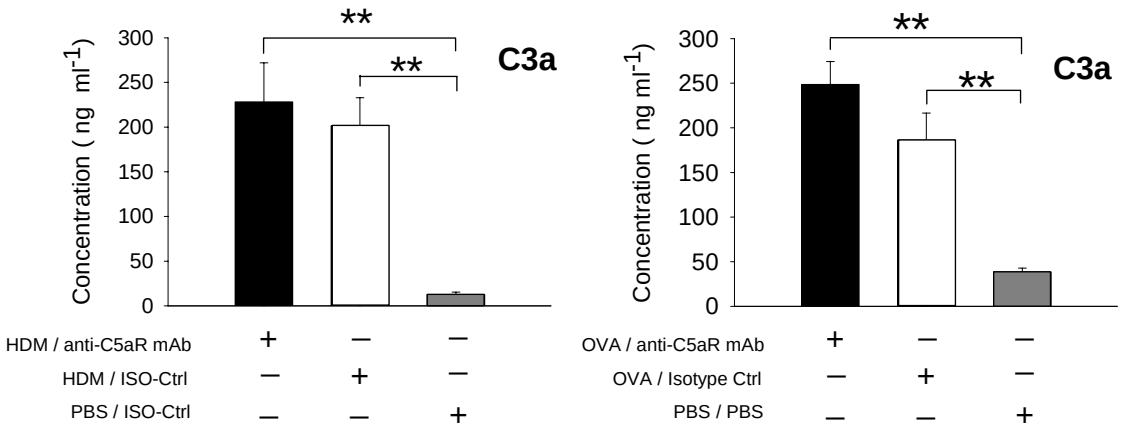


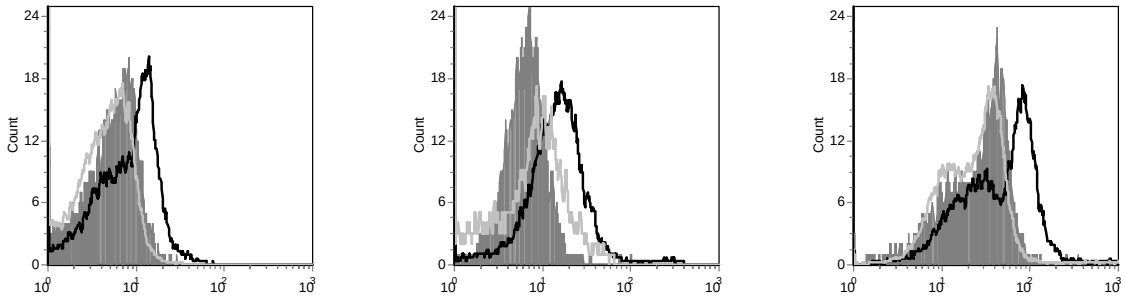



**Supplemental Figure 2 Köhl et al.**




**Supplemental Figure 3 Köhl et al.**

**A** **B** **C**




**CD40** **CD80** **CD86**

**Supplemental Figure 4 Köhl et al.**

**Supplemental Figure 1 Impact of C5aR blockade on mucus production.** (A) Enlarged sectional view of PAS-stained lung tissues of mice exposed to OVA; and (B) to OVA and the neutralizing anti-C5aR mAb. (Figure 2D) The mucus production in response to OVA (red areas) is only minor but is markedly increased in response to C5aR blockade. Original magnification, x 400.

**Supplemental Figure 2 C5aR deficiency enhances Th2 adaptive immune responses and eosinophilic airway inflammation in response to pulmonary OVA exposure.** (A) Cytokine profile of pulmonary cells harvested from $C5aR^{-/-}$ and $C5aR^{+/+}$ mice 24 h after the final in vivo OVA exposure. (B) Total and differential cell counts in BAL. (C) Airway responsiveness to i.v. Ach. (D) CC17 and CCL22 production from co-cultures of FACS-sorted $CD11c^{+}$ DCs and $CD4^{+}$ lymphocytes. (n=8-10/group); $^{**}P < 0.001$, $^{*}P < 0.05$.

**Supplemental Figure 3 Complement activation in response to repeated pulmonary allergen exposure**. Complement activation was determined by measuring the generation of C3a in BAL (A) 72h after the final pulmonary exposure to HDM or (B) 16h after the final pulmonary exposure to OVA. C3a was measured by ELISA.

**Supplemental Figure 4 Impact of OVA exposure on the surface expression of CD40, CD80 and CD86 on pulmonary-derived mDCs**. $CD11c^{+}$, $CD11b^{+}$, $Gr\text{-}1^{-}$ mDCs were FACS-sorted from lungs of naïve BALB/c mice and incubated with cell culture medium (dashed histogram), OVA ($10^{-5}$M; gray line) or LPS (100 ng/ml; black line). (A) CD40 expression; (B) CD80 expression; (C) CD86 expression.