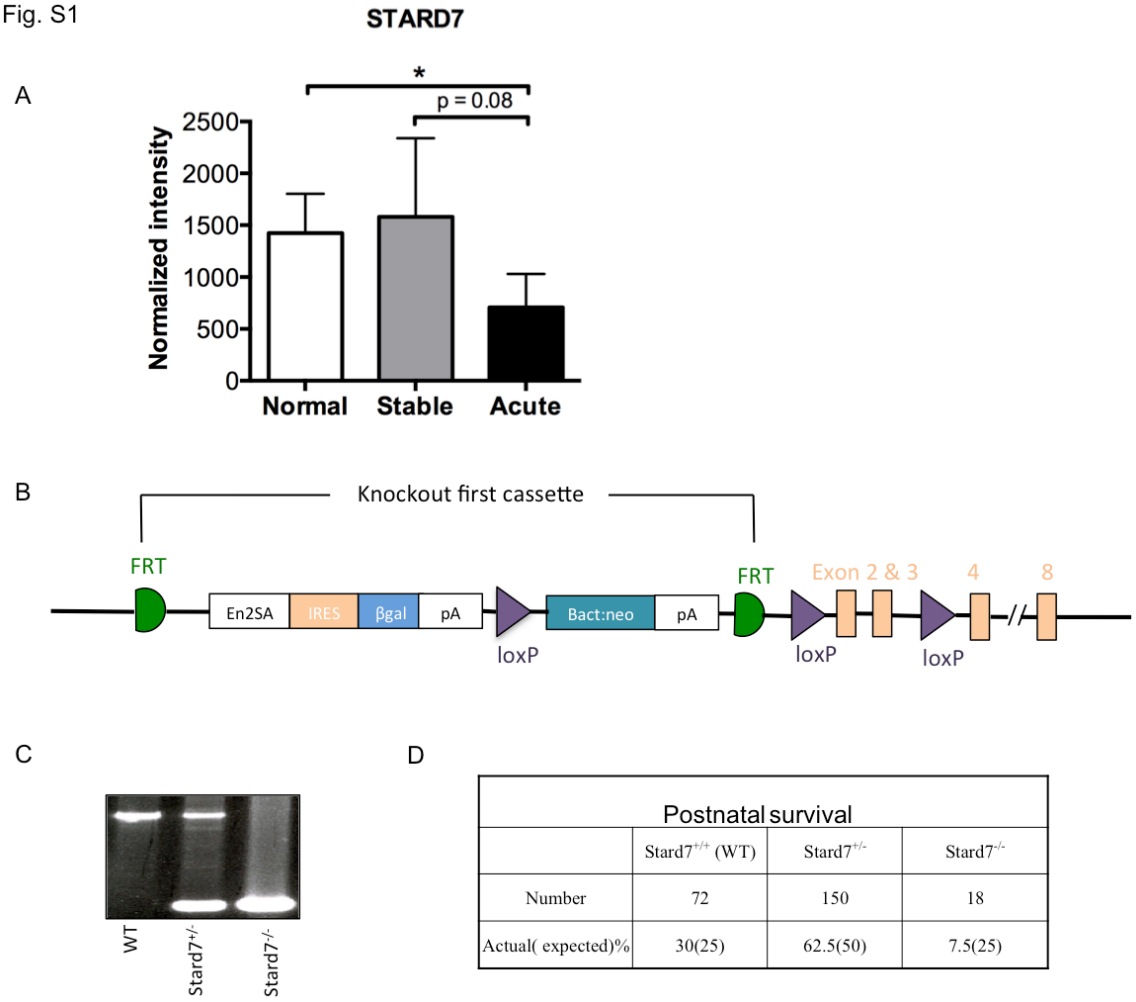
Fig. S1



Postnatal survival			
	Stard7$^{+/+}$ (WT)	Stard7$^{+/-}$	Stard7$^{-/-}$
Number	72	150	18
Actual(expected)%	30(25)	62.5(50)	7.5(25)

Fig. S1. Generation of Stard7$^{+/-}$ mice.

(A) Expression of Stard7 mRNA in nasal epithelial cells isolated from children with stable asthma or an acute asthma exacerbation (kindly provided by G.K. Hershey, M.D., Ph.D., Cincinnati Children's Hospital Medical Center; Guajardo, J. R., K. W. Schleifer, M. O. Daines, R. M. Ruddy, B. J. Aronow, M. Wills-Karp, and G. K. Hershey. 2005. Altered gene expression profiles in nasal respiratory epithelium reflect stable versus acute childhood asthma. *The Journal of allergy and clinical immunology* 115: 243-251). (B) Schematic of the targeted *Stard7* allele. A trapping cassette (SA-betagal-pA) flanked by FRT sites was targeted to intron 1 of the *Stard7* gene; exons 2 and 3 of *Stard7* are flanked

by LoxP sites for cell/tissue-specific deletion of *Stard7*. FRT, FLp-recombinase target site-directed recombination; En2SA, entrailed 2 splice acceptor site; IRES, internal ribosome entry site; β-act:neo, β-actin promoter driving neomycin gene selectable marker; pA, polyadenylation site. (C) PCR analysis of the targeted *Stard7* allele in tail DNA of 8 week-old mice. (D) Table depicting survival of 3 week-old progeny from *Stard7*$^{+/-}$ intercrosses.

Fig S2

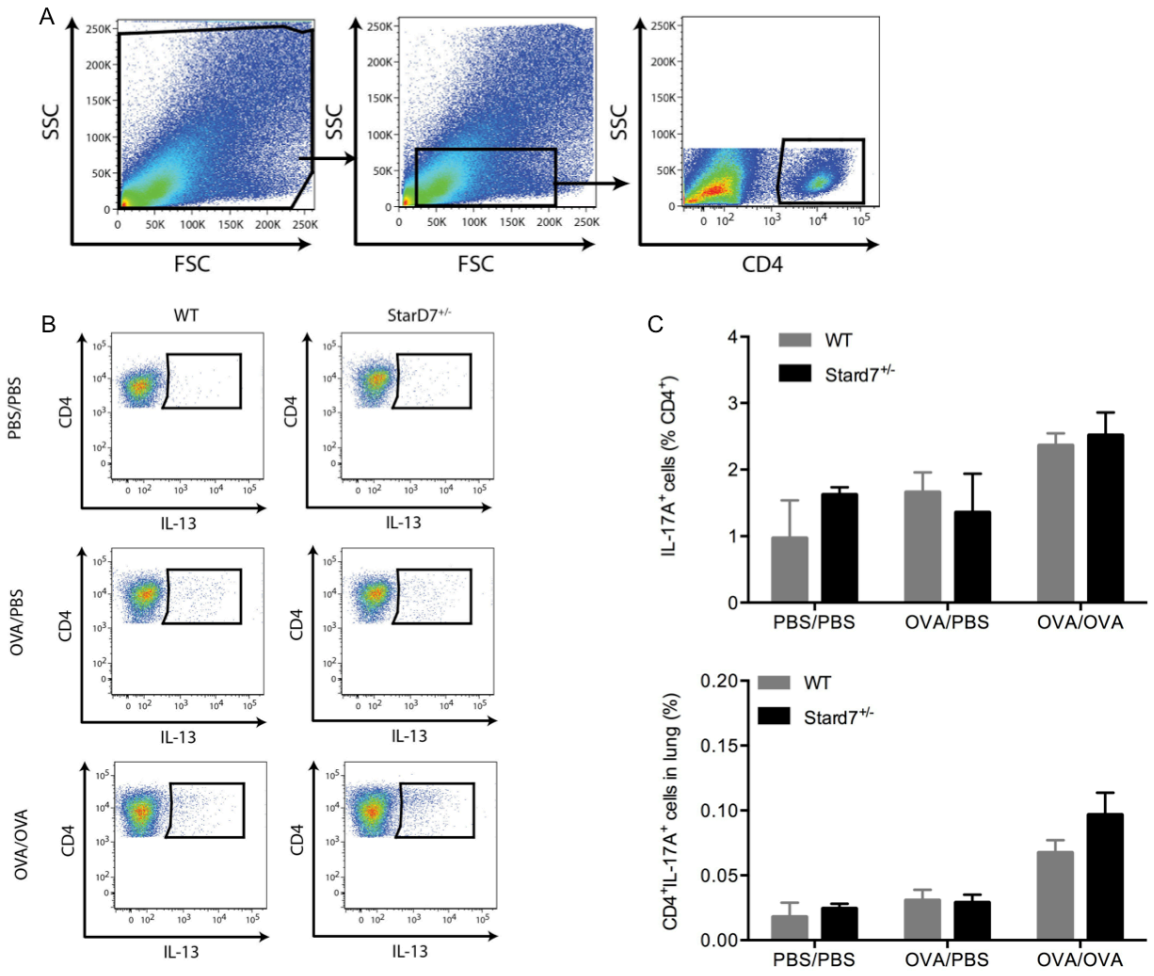


Fig. S2. Identification of IL-13-secreted T cells in the lungs of OVA-exposed WT and StarD7$^{+/-}$ mice.

Lung cells were isolated from PBS-sensitized, PBS-challenged (PBS/PBS), OVA-sensitized, PBS challenged (OVA/PBS), or OVA-sensitized, OVA-challenged (OVA/OVA) WT or StarD7$^{+/-}$ mice. Cells were stimulated overnight with PMA/Ionomycin, and cultured in the presence of brefeldin A and monensin for 4 hours. Cells were stained with mAbs to CD4 and IL-13 or IL-17A for analysis of the frequency of cytokine positive CD4 cells. (A) Gating strategy used to identify CD4+ cells. (B) Representative dot plots showing identification of IL-13+ CD4+ T cells from WT and StarD7$^{+/-}$ mice in the various treatment groups. This gating strategy was used to identify cytokine producing CD4+ T cells enumerated in Figures 3 and S2C. *n* = 4-8 mice/group.

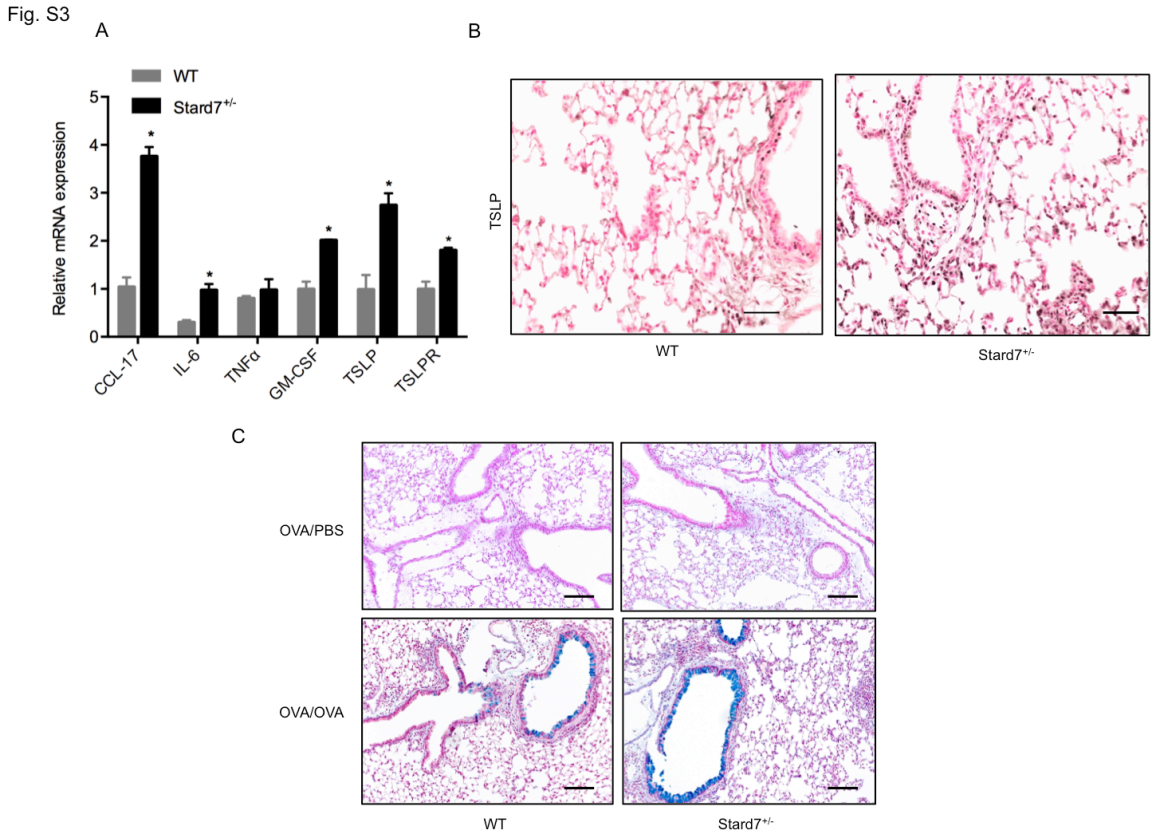


Fig. S3. Deficiency of Stard7 is associated with increased inflammatory mediators and mucous cell metaplasia following OVA sensitization and challenge.

(A) qRT-PCR of cytokine and chemokine mRNAs from lung tissue of sensitized and challenged mice. TSLP, thymic stromal lymphopoietin; TSLPR, thymic stromal lymphopoietin receptor. $n = 4$ mice/group. (B) Immunohistochemical staining of TSLP in lung tissue from OVA-sensitized and challenged mice. Scale bars = 50 μm. (C) Alcian blue staining of lung tissue following sensitization and challenge with OVA/PBS or OVA/OVA. $n = 4$ mice/group. Scale bars = 50 μm.

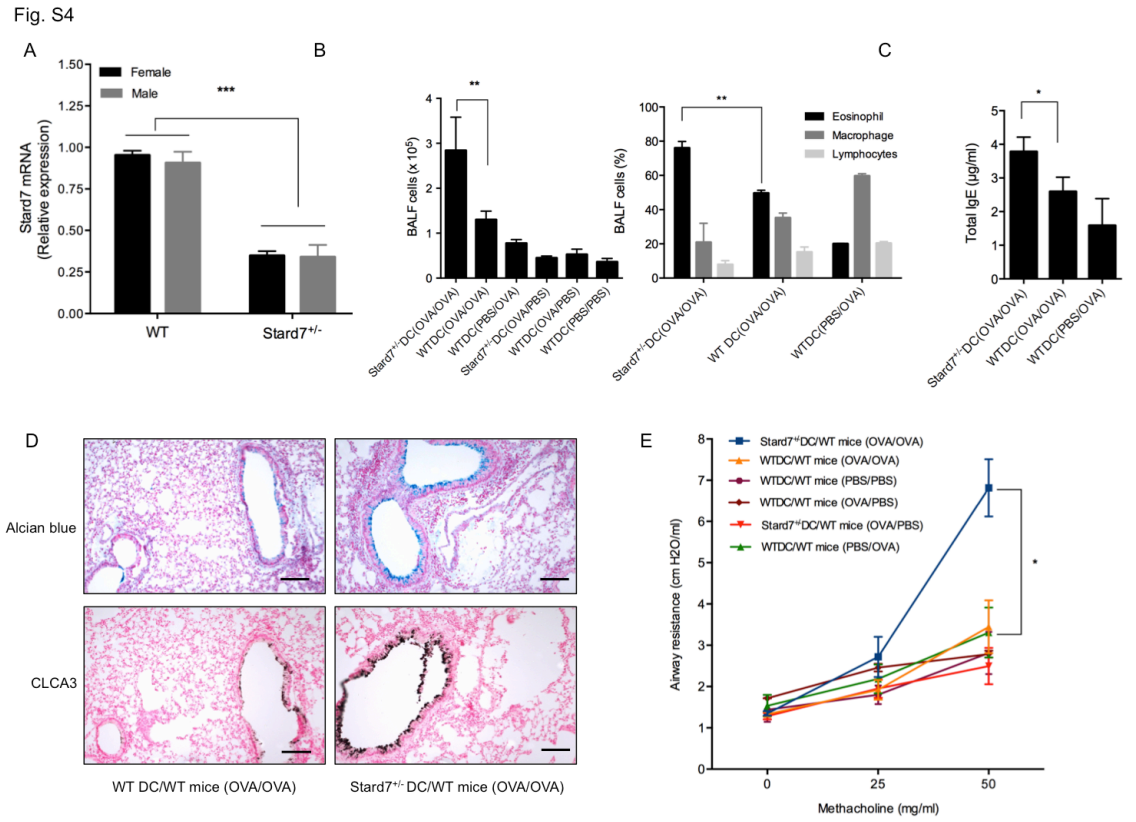


Fig. S4. Adoptive transfer of Stard7$^{+/-}$ BMDCs confers an exaggerated allergic response in WT mice.

OVA-pulsed BMDCs from WT or *Stard7*$^{+/-}$ mice were injected into the airways of WT mice followed by intratracheal OVA challenge (days 10-13) and analysis on day 14. (A) qRT-PCR of *Stard7* mRNA in BMDCs (6 days after GM-CSF differentiation) from 8

week-old donor mice. β-actin was used as an endogenous control. $n = 3$ samples/group. (B) Total (left panel) and differential (right panel) BALF cell counts in BMDC recipient mice challenged with OVA. $n = 8$ mice/group. (C) Total IgE in WT recipient mice challenged with OVA. $n = 5$ mice/group. (D) Alcian blue (upper panels) and CLCA3 immunohistochemistry (lower panels) in lung tissue of WT recipient mice challenged with OVA. Scale bars = 50 μm. (E) AHR was evaluated in BMDC recipient mice challenged with PBS or OVA. $n = 8$ mice/group; *p value, *Stard7*$^{+/-}$ DC/WT mice (OVA/OVA) vs. WT DC/WT mice (OVA/OVA).