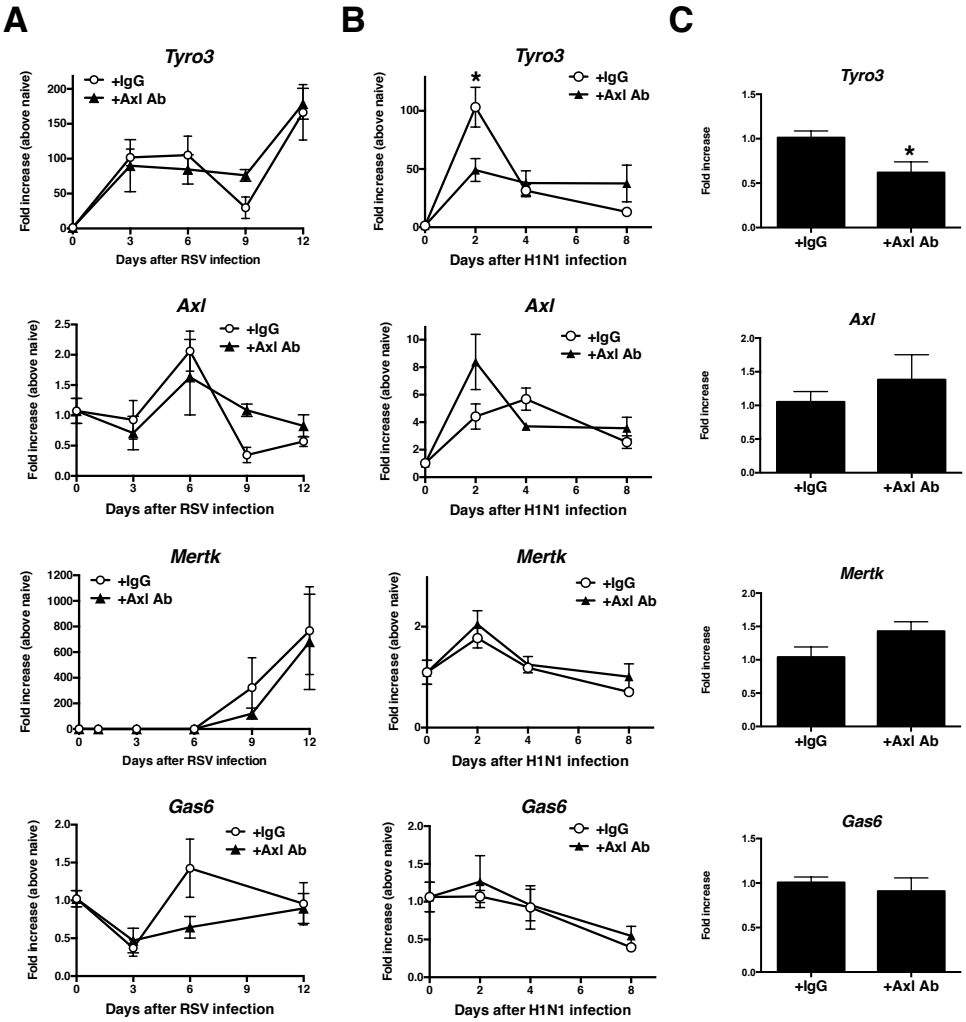
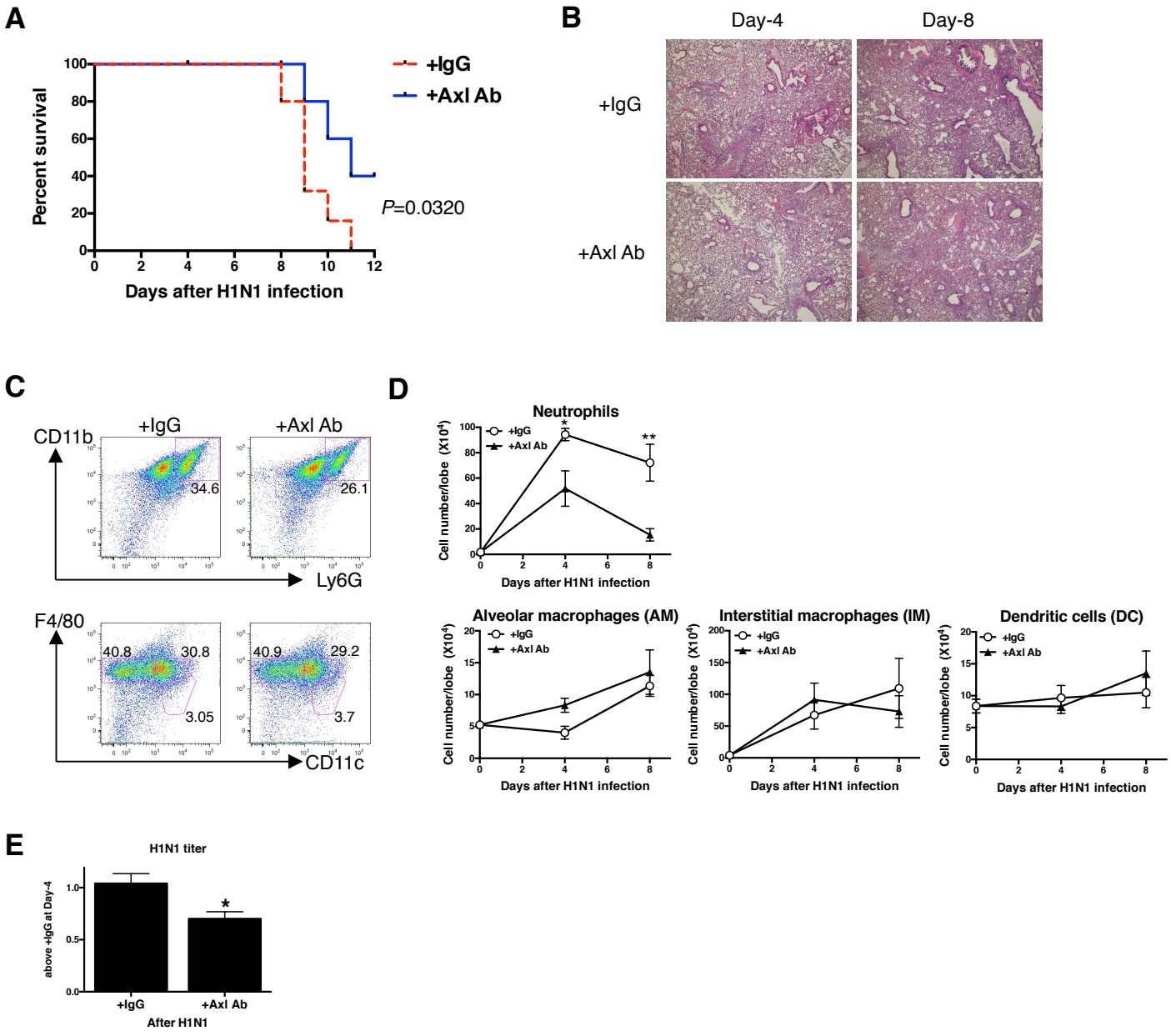
Supplemental Figure 1



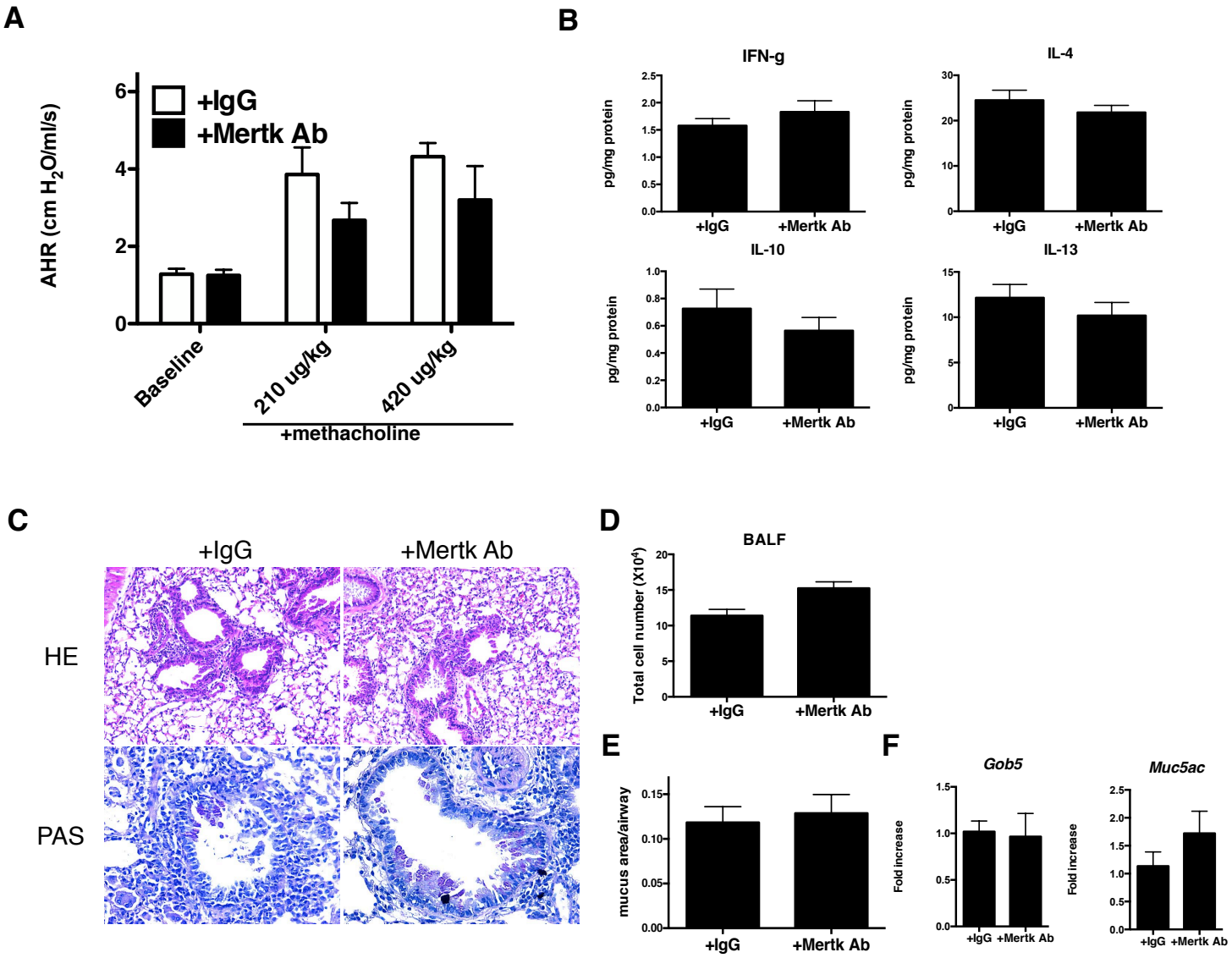
Supplemental Fig. 1. Anti-Axl mAb treatment did not alter TAM receptor expression. Quantitative PCR transcript analysis of *Tyro3*, *Axl*, *Mertk*, and *Gas6* in whole lung samples during primary RSV infection (**A**), primary influenza virus infection (**B**), and fungal asthma (**C**) are indicated.

Supplemental Figure 2



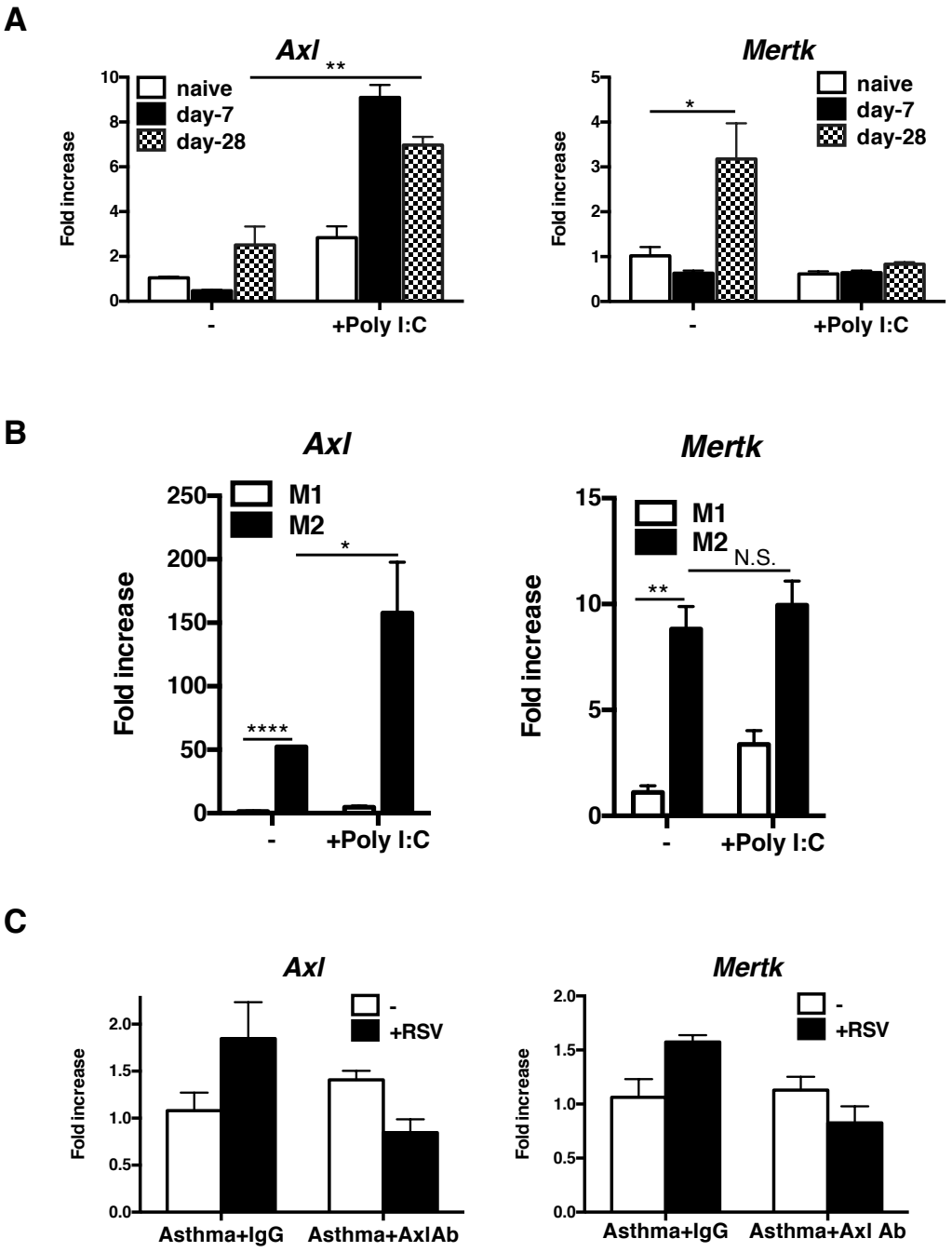
Supplemental Fig. 2. Anti-Axl mAb treatment after influenza infection significantly inhibited lethality. Naïve mice were treated with IgG1 or anti-Axl mAb during H1N1 infection at days 2, 4, 8 after intrapulmonary infection with H1N1 influenza virus. Kaplan Meier survival analysis of IgG1-treated group (red) and anti-Axl mAb-treated (blue) mice during H1N1 (1×10^4 PFU) infection **(A)**. Results are expressed as the percent survival of a starting cohort of 10 mice per group. Representative hematoxylin and eosin (H&E)-stained lung tissue sections from IgG1- (upper) and anti-Axl mAb-treated (lower) mice at days 2, 4, and 8 post-infection with influenza virus (**B**). Representative flow cytometry analysis of neutrophils (upper panels, $CD45^+CD11b^{high}Ly6G^{high}$), interstitial macrophages (lower panels, $CD45^+F4/80^+CD11C^-$), alveolar macrophages ($CD45^+F4/80^+CD11C^+$), and dendritic cells ($CD45^+F4/80^-CD11C^+$) in IgG1- and anti-Axl mAb-treated lung samples from naïve mice at day 4 after H1N1 inoculation (**C**). Quantification of neutrophils, interstitial and alveolar macrophages, and dendritic cells in IgG1- and anti-Axl mAb-treated lung after H1N1 inoculation (**D**). Lung viral titers in IgG1- and anti-Axl mAb-treated mice at day 4 after H1N1 infection (**E**). *P<0.05, **P<0.01, ***P<0.001 compared with control IgG-treated group.

Supplemental Figure 3



Supplemental Fig. 3. Anti-Mertk mAb therapy did not inhibit any airway feature in a fungal asthma model. Airway hyperresponsiveness to a dose of 210 μg/kg or 420 μg/kg of methacholine in IgG1-treated (+IgG1) or anti-Mertk mAb-treated (+Mertk mAb; 5 μg/dose i.p. x 7 doses; **A**) at day 28 after conidia. Whole lung protein levels of IFN-g, IL-4, IL-10, and IL-13 in asthmatic mice treated with IgG1 or anti-Mertk mAb at day 28 after conidia (**B**). Representative H&E- and PAS-stained lung tissue sections from both treatment groups at day 28 after conidia (**C**). Quantification of cells present in BALF from IgG1- and anti-Mertk mAb-treated asthmatic mice (**D**). Quantitative analysis of mucus area in airway from IgG1- and anti-Mertk Ab-treated groups at day 28 after conidia (**E**). Quantitative PCR analysis of *Muc5ac* and *Gob5* in the IgG1 and anti-Mertk mAb groups at day 28 after conidia (**F**). Results are expressed as the mean ± SEM for n=5/group.

Supplemental Figure 4



Supplemental Fig. 4. Synthetic double stranded RNA (Poly I:C) significantly enhanced Axl expression in macrophages from asthmatic mice. Bone marrow-derived macrophages (BMDM) from naïve mice and asthmatic mice prior to and at 7 and 28 days after conidia challenge were exposed to poly I:C (10 μg/ml for 6 h and *Axl* and *Mertk* transcript expression were determined using q-PCR (**A**). *, $P<0.05$, **, $P<0.01$. BMDM from naïve mice were stimulated with LPS/IFN-g to induce M1 macrophages (M1) and IL-4/IL-13 to induce M2 macrophages (M2) for 24 h. M1 and M2 macrophages were then exposed to poly I:C for 6 h, and *Axl* and *Mertk* transcript expression were analyzed using q-PCR (**B**). *, $P<0.05$, **, $P<0.01$ ****, $P<0.0001$ versus the naïve group. Quantitative PCR analysis of *Axl* and *Mertk* in IgG1- and ant-Axl mAb-treated asthmatic mice at day 42 after conidia with mock (-) or RSV infection (+) (**C**).