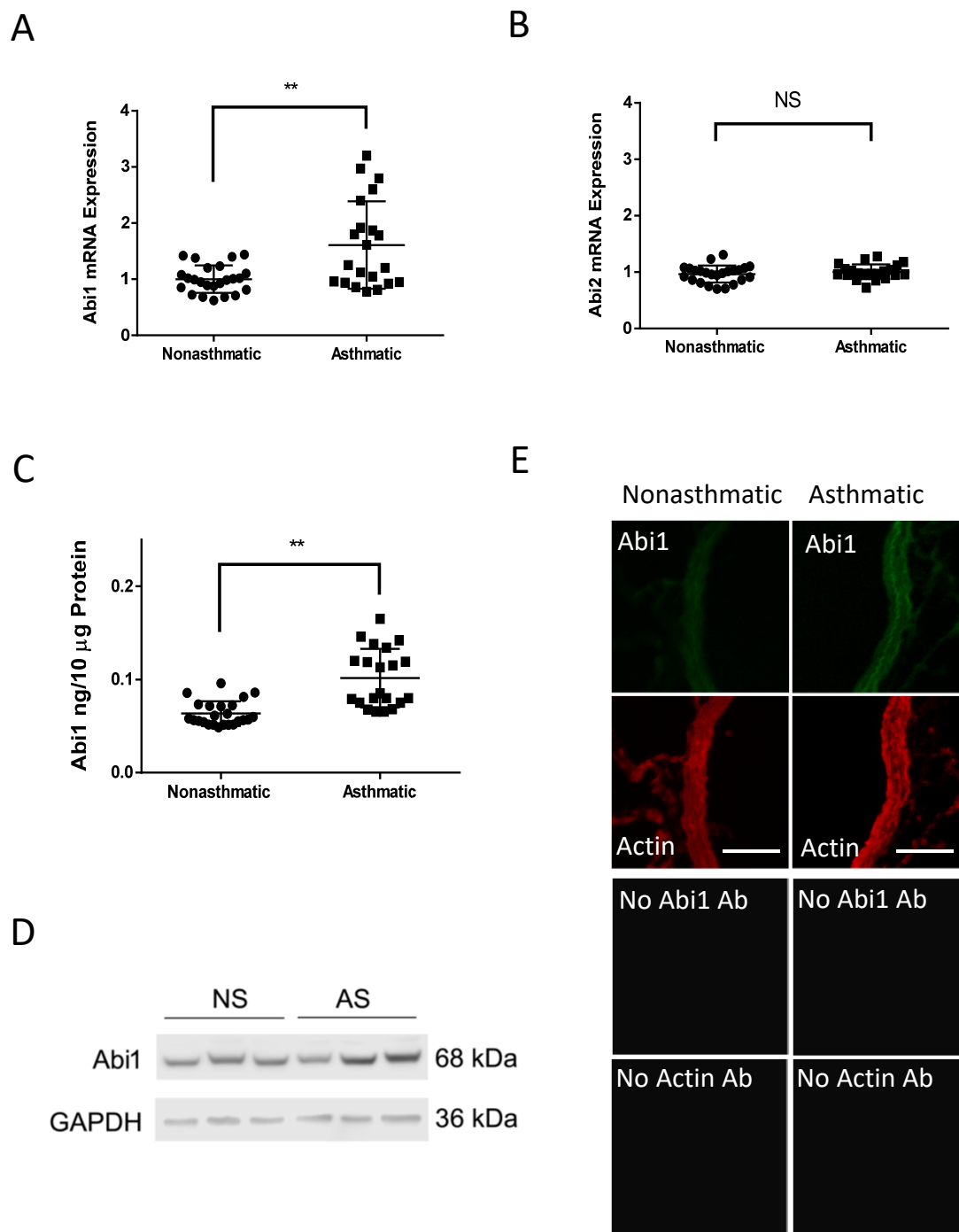


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## **Supplemental information**

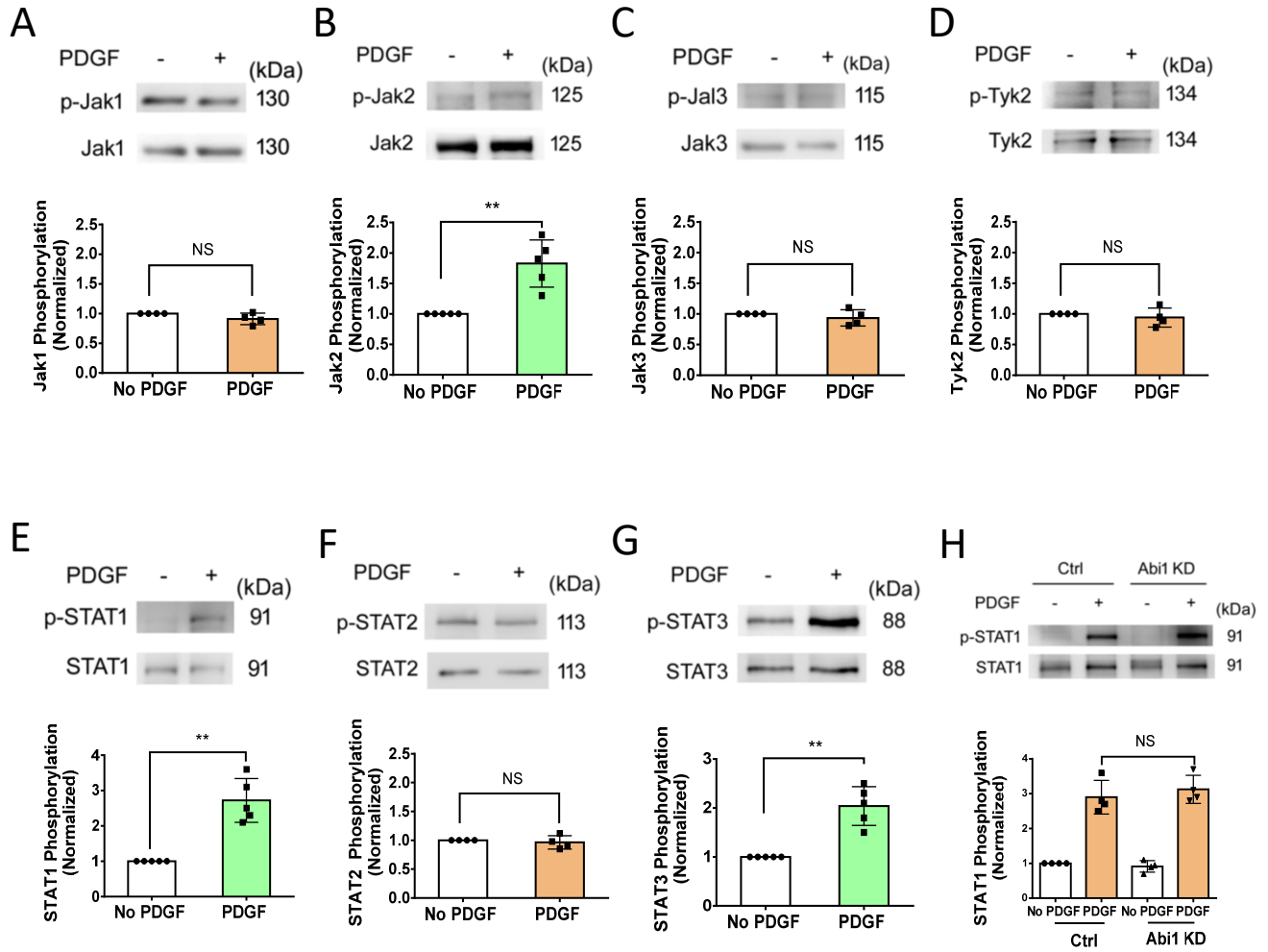
**Abi1 mediates airway smooth muscle  
cell proliferation and airway  
remodeling via Jak2/STAT3 signaling**

**Ruping Wang, Yinna Wang, Guoning Liao, Bohao Chen, Reynold A. Panettieri  
Jr., Raymond B. Penn, and Dale D. Tang**



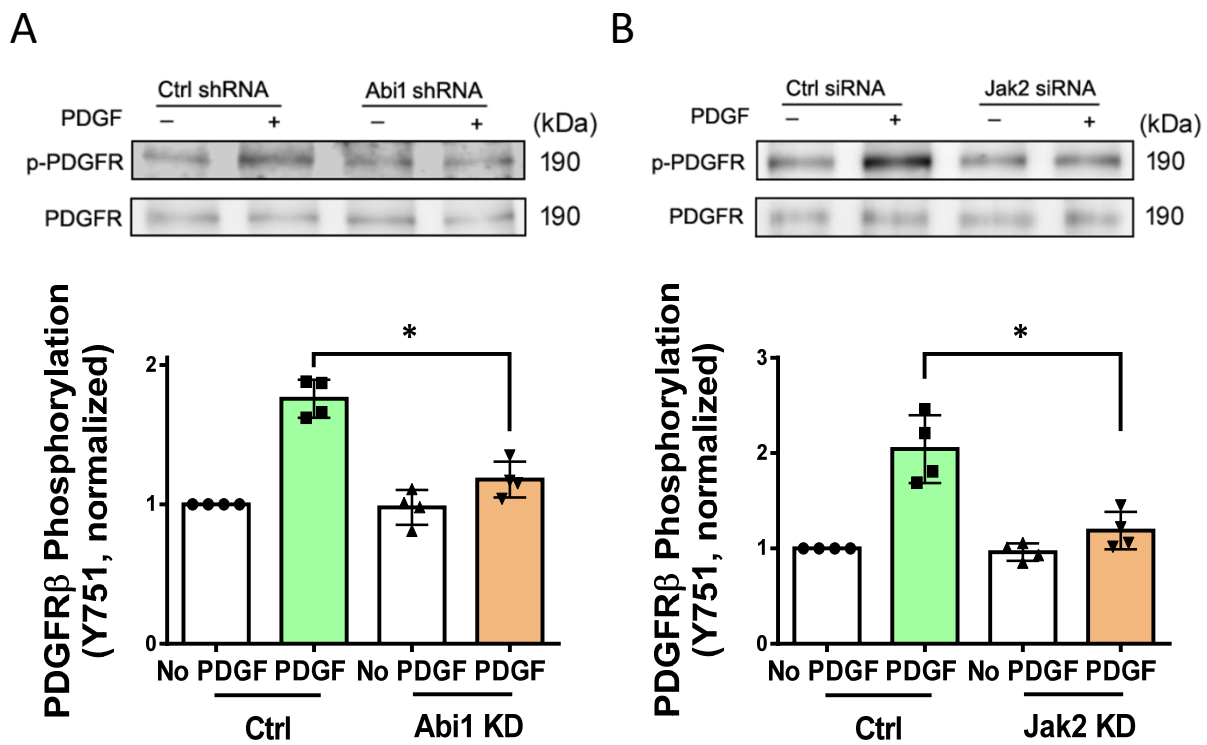
**Figure S1. Mean analysis of Abi1 mRNA, Abi2 mRNA and Abi1 protein in nonasthmatic and asthmatic human ASM cultures. Related to Figure 1.**

**A.** Abi1 mRNA. **B.** Abi2 mRNA. **C.** Abi1 protein. Data are means  $\pm$  S.E.M. *t*-test was used for data analysis. **D.** Representative IB of Abi1 expression in nonasthmatic (NA) and asthmatic (AS) cells. **E.** Abi1 is upregulated in asthmatic human small airways as evidenced by immunofluorescence microscopy. Scale bar, 120  $\mu$ m. Images of no primary antibody controls are also included.



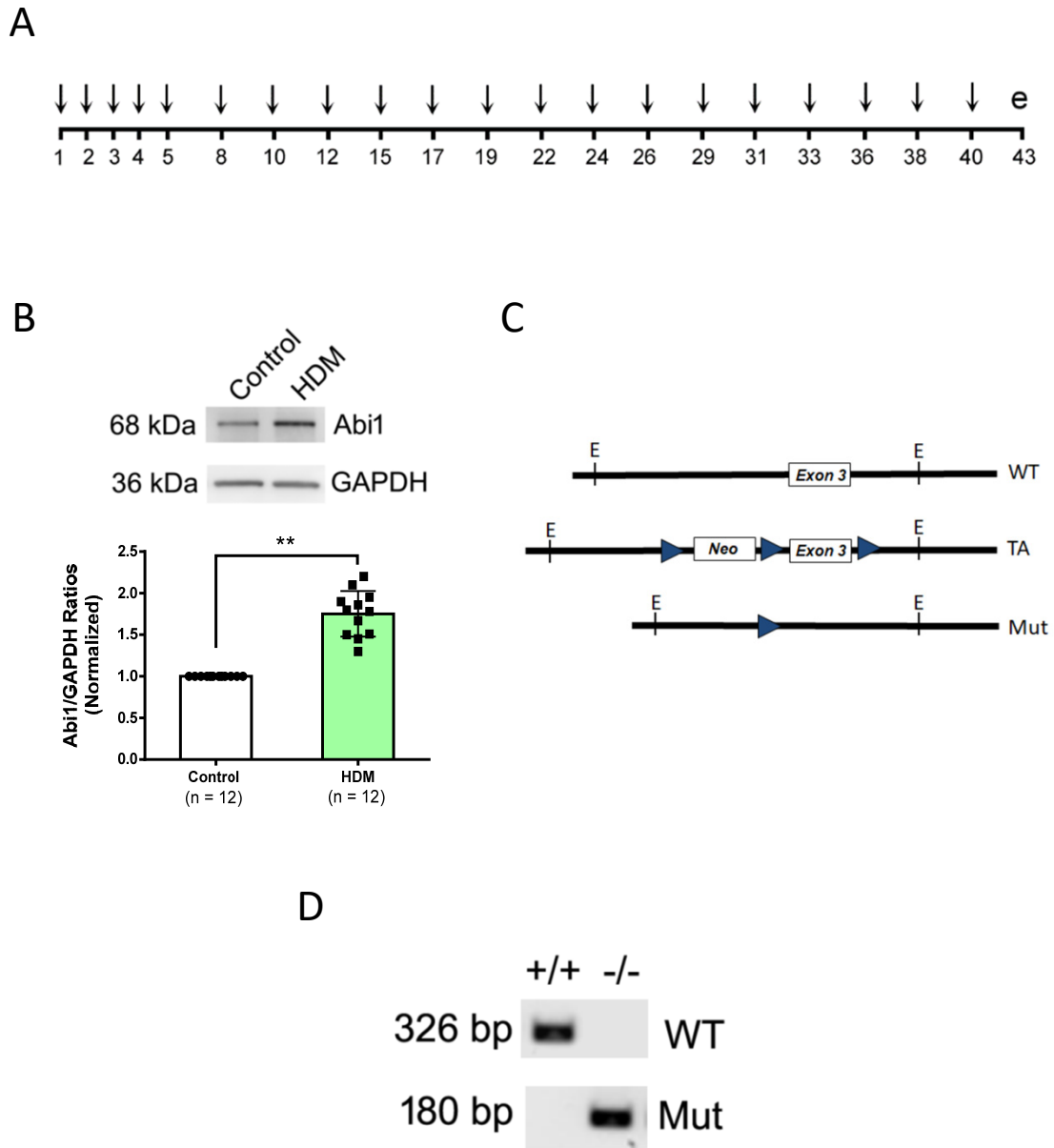
**Figure S2. Stimulation with PDGF differentially affects phosphorylation of Jaks and STATs in HASM cells. Related to Figure 3.**

**A-D.** Treatment with PDGF increases p-Jak2 (Y1007/Y1008), but not p-Jak1 (Y1034/Y1035), p-Jak3 (Y980/Y981) and p-Tyk4 (Y1054/Y1055). **E-G.** Stimulation with PDGF increases p-STAT1 (Y701) and p-STAT3 (Y705), but not p-STAT2 (Y609). **H.** Abi1 KD does not affect the phosphorylation of STAT1 (Y701). Data are means  $\pm$  S.E.M.  $n = 4-5$ . The  $t$ -test was used for statistical analysis for Figure S2, A-G. Two-way ANOVA was used for Figure S2H. NS, not significant. \*\*  $p < 0.01$ .



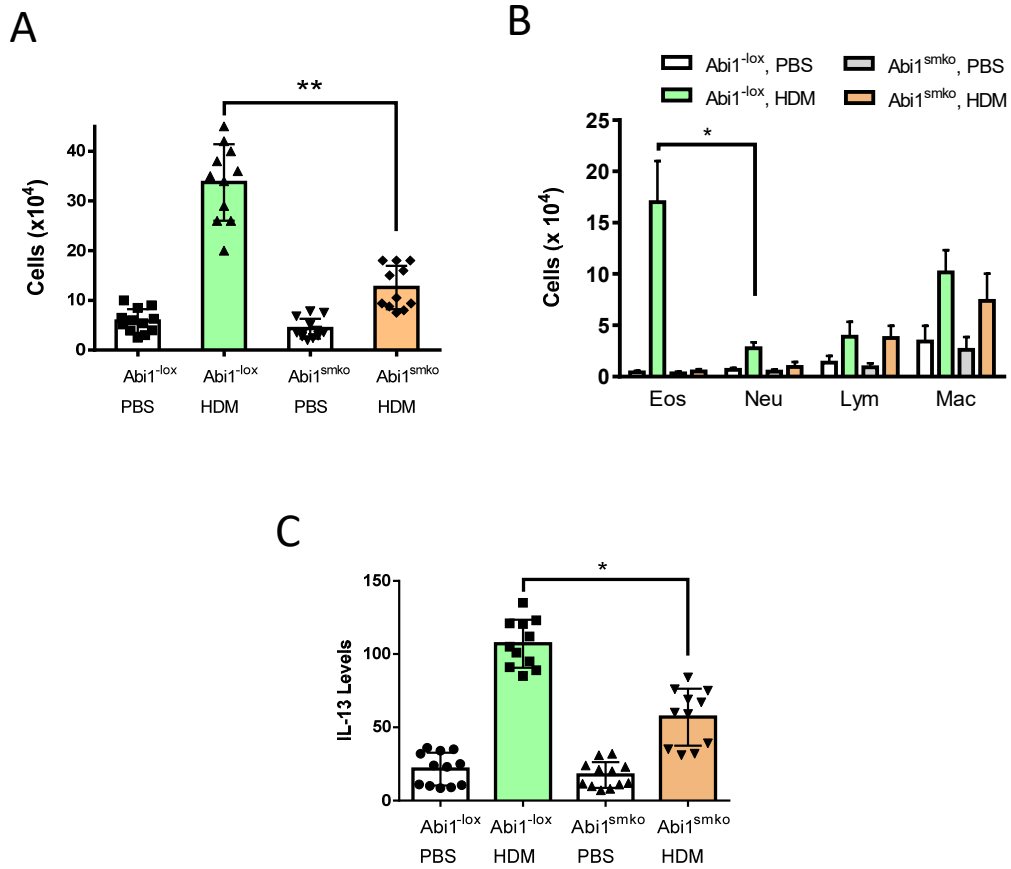
**Figure S3. Role of Abi1 and Jak2 in PDGFR phosphorylation. Related to Figure 5.**

**A.** Abi1 KD attenuates the PDGF-induced PDGFR $\beta$  phosphorylation in HASM cells. Data are means  $\pm$  S.E.M.  $n = 4$ . Two-way ANOVA was used for statistical analysis. **B.** Jak2 KD attenuates the PDGF-induced PDGFR $\beta$  phosphorylation in HASM cells. Data are means  $\pm$  S.E.M.  $n = 4$ . Two-way ANOVA was used for statistical analysis.



**Figure S4. Protocol and characterization of murine asthma model. Related to Figure 8.**

**A.** Schematic overview of study protocol of allergen exposure. Arrows; intranasal exposure; e, experiment. **B.** The expression of Abi1 in tracheal/bronchial extracts of house dust mite (HDM)-treated mice is higher than control mice (\* $P < 0.05$ ). *t*-test was used for statistical analysis. **C.** Strategy of generating Abi1-floxed mouse. Exon 3 of Abi1 is flanked by two loxP sites. Crossing of MYH11-Cre mouse with floxed mouse generates conditional KO mouse. **D.** Agarose gel of PCR products amplified from mouse tails of indicated mouse strains.



**Figure S5. Role of Abi1 in airway inflammation. Related to Figure 8.**

**A.** Conditional Abi1 knockout affects total cell counts in the lungs of animals after HDM exposure. **B.** The differential cell counts are reduced in the lungs of conditional Abi1 knockout mice treated with HDM. **C.** The IL-13 level in BALF of HDM-exposed Abi1<sup>-lox</sup> mice is increased compared to Abi1<sup>-lox</sup> treated with PBS. Abi1 conditional knockout inhibits the HDM-induced increase in IL-13 level (n = 11-12). Data are means  $\pm$  S.E.M. \*P < 0.05; \*\*P < 0.01. Two-way ANOVA was used for statistical analysis.