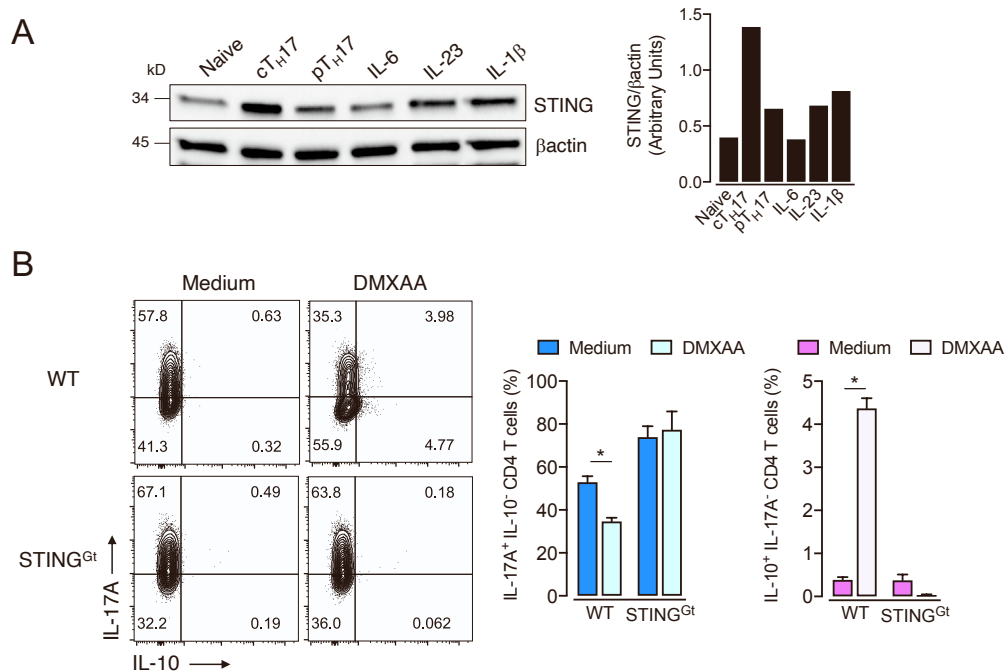


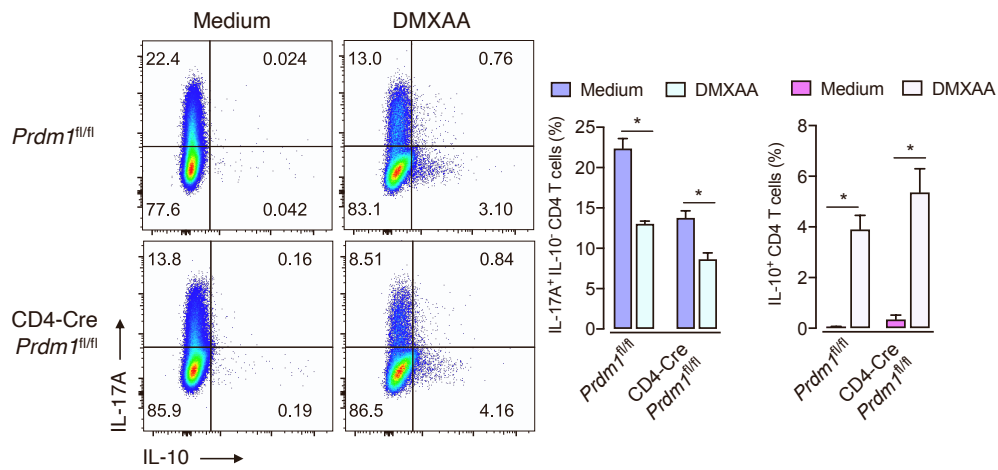
**Supplemental information**

**STING is an intrinsic checkpoint inhibitor  
that restrains the T<sub>H</sub>17 cell pathogenic program**

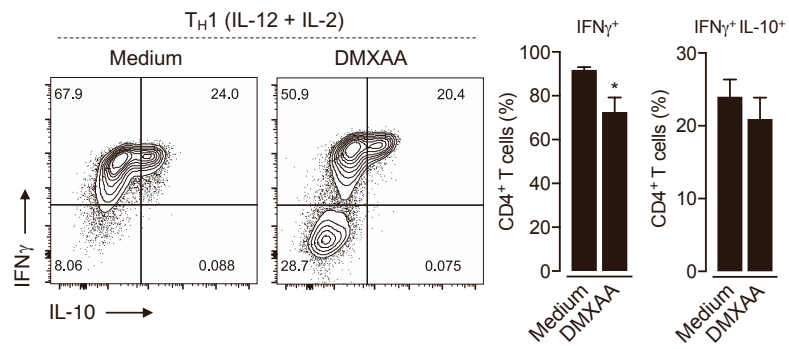
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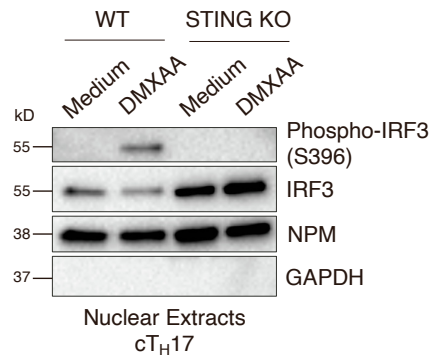
**Figure S1. STING expression is supported by the combination of IL-6 and TGFβ and a point mutation in STING phenocopies the effects observed with STING-deficient T<sub>H</sub>17 cells. Related to Figure 2.** (A) STING protein levels were evaluated in CD4 T cells cultured under c<sub>H</sub>17 or p<sub>H</sub>17-conditions or in the presence of rmIL-6, rmIL-23 and rmIL-1β alone for 72 h by immunoblot analysis. βactin was used as the loading control. (B) Naive CD4 T cells from WT or STING<sup>Gt</sup> mice were differentiated into c<sub>H</sub>17 cells in the presence of 10 μM DMXAA for 72 h, followed by flow cytometric analysis ( $n = 3$ ). Data are representative of two independent experiments and shown as mean ± SEM. \*P < 0.05 as determined by two-way ANOVA (B) followed by Tukey's post hoc test.



**Figure S2. Blimp-1 is not required for STING-induced IL-10 production in T<sub>H</sub>17 cells. Related to Figure 3.** Naive CD4 T cells from WT (*Prdm1<sup>fl/fl</sup>*) and Blimp-1-deficient (*CD4-Cre Prdm1<sup>fl/fl</sup>*) mice were cultured under cT<sub>H</sub>17-polarizing conditions in the presence or absence of 10  $\mu$ M DMXAA for 72 h, followed by flow cytometric analysis ( $n = 3-5$ ). Data are representative of two independent experiments and shown as mean  $\pm$  SEM. \* $P < 0.05$  as determined by two-way ANOVA followed by Tukey's post hoc test.



**Figure S3. STING activation does not alter IL-10 expression in T<sub>H</sub>1 cells. Related to Figure 3.** Naive CD4 T cells were cultured under T<sub>H</sub>1-skewing conditions in the presence or absence of 10  $\mu$ M DMXAA for 72 h, followed by flow cytometric analysis ( $n = 3$ ). Data are representative of two independent experiments and shown as mean  $\pm$  SEM. \* $P < 0.05$  as determined by two-tailed Student's t-test.



**Figure S4. Active IRF3 is localized in the nuclear compartment of T<sub>H</sub>17 cells in a STING-dependent manner. Related to Figure 4.** WT or STING-deficient naive CD4 T cells were differentiated into cT<sub>H</sub>17 cells for 72 h in the presence or absence of 10  $\mu$ M DMXAA and collected for nuclear fraction separation. Phospho-IRF3 and IRF3 levels were analyzed by immunoblot analysis. Nucleophosmin (NPM) was used as nuclear loading control. Data are representative of two independent experiments.