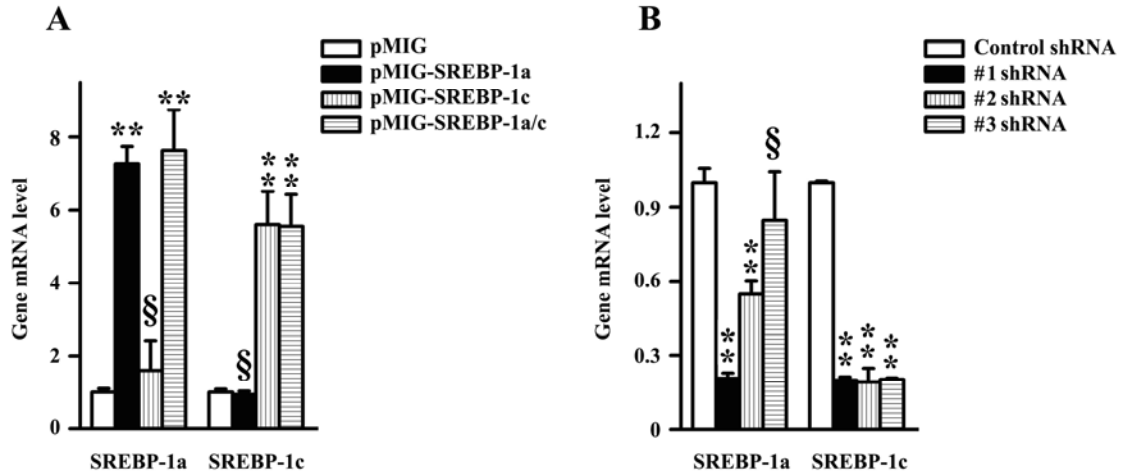


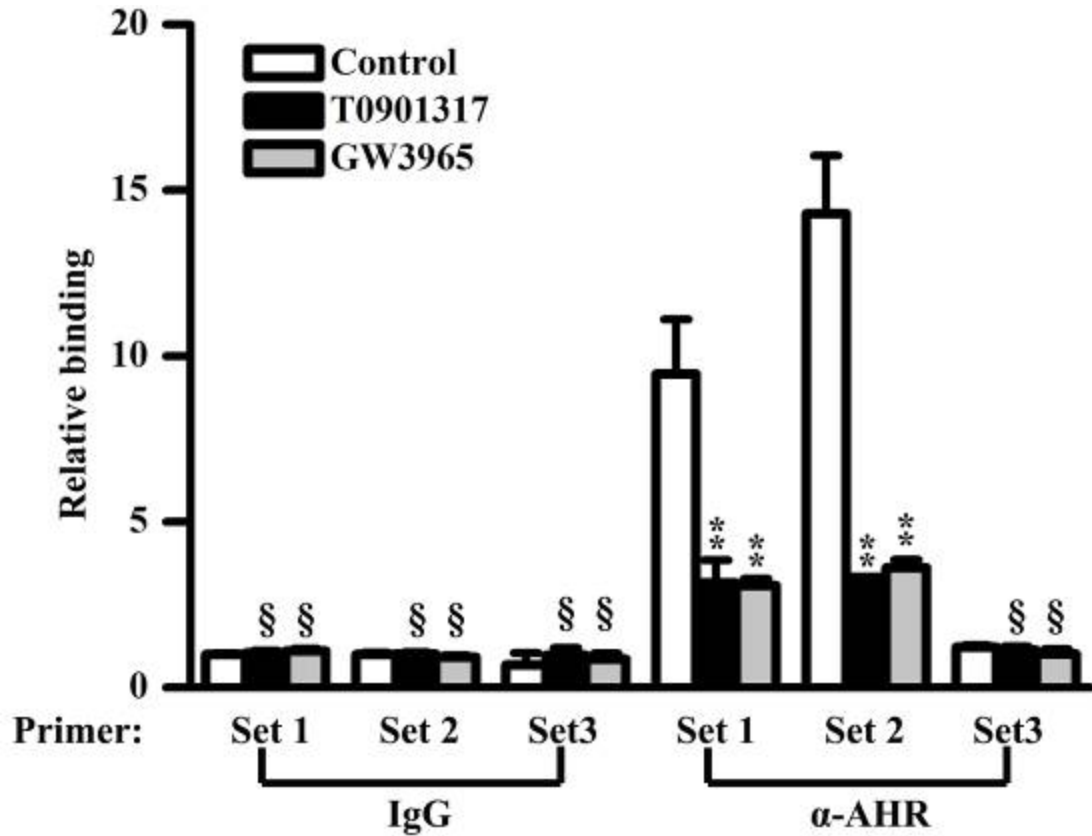
**Supplementary Figure 1. Schematic representation of SREBP-1 putative binding site, the E-box element, on IL-17 promoter and primer Set 1, Set 2 and Set 3 used in CHIP assay.**



**Supplementary Figure 2. Over-expression and knockdown of SREBP-1a and SREBP-1c in Th17 cells.**

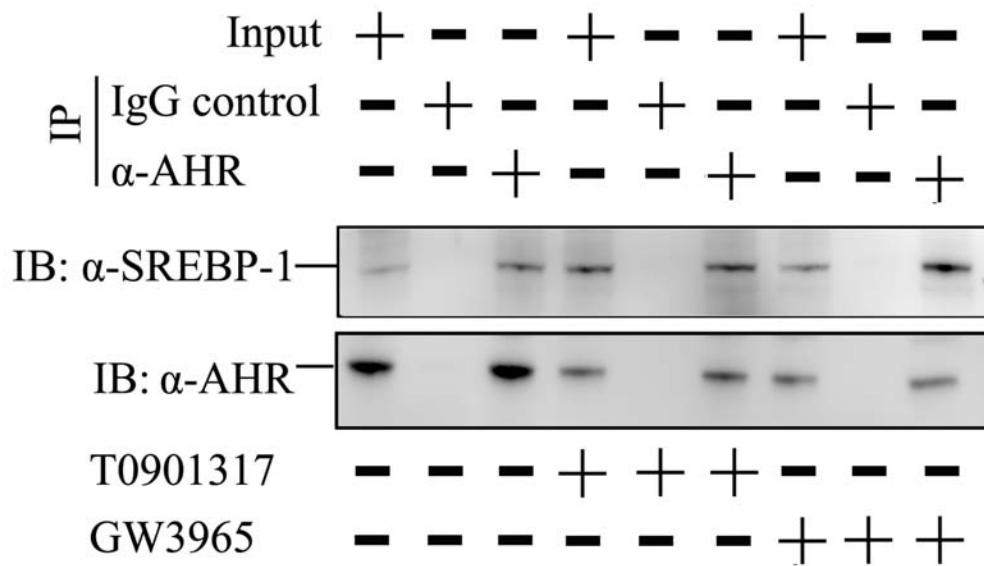
(A) Naïve CD4<sup>+</sup> T cells were cultured under Th17-inducing conditions with or without retroviral expression of SREBP-1a and SREBP-1c for 4 days before harvesting for real time PCR analysis of SREBP-1a and SREBP-1c mRNA levels.

(B) Naïve CD4<sup>+</sup> T cells were cultured under Th17-inducing conditions with or without retroviral knockdown of SREBP-1a/c for 4 days before harvesting for real time PCR analysis of SREBP-1a and SREBP-1c mRNA levels. \*\*, P<0.01; §, P>0.05.



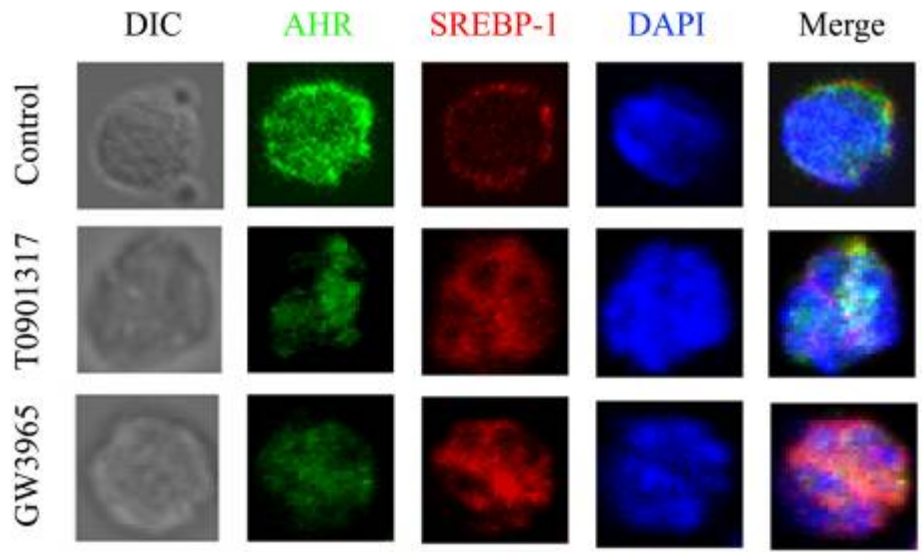
**Supplementary Figure 3. Binding of AHR onto the E-box element on IL-17 promoter.**

AHR binding to the E-box element on the IL-17 promoter in *in vitro* differentiated Th17 cells was assessed by CHIP. The primers used to detect CHIP signals are schematically represented in Supplementary Figure 1. Statistical analysis was performed between the LXR agonist-treated and untreated groups. §,  $P > 0.05$ ; \*\*,  $P < 0.01$ , compared with “control” group. This experiment has been repeated for three times with similar results.

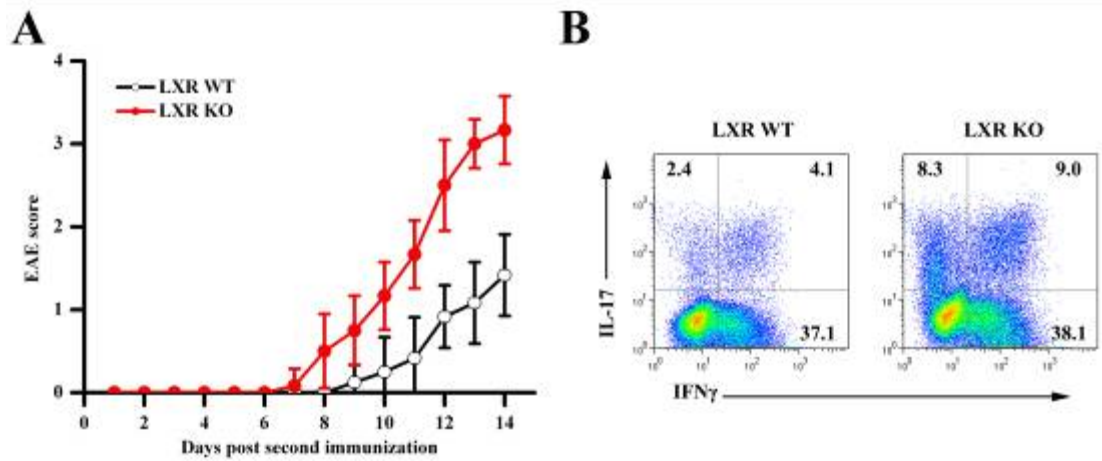


**Supplementary Figure 4. AHR physically interacted with SREBP-1.**

Naïve CD4<sup>+</sup> T cells were cultured under Th17-inducing conditions with the indicated drugs before the performing of coimmunoprecipitation. Protein complexes immunoprecipitated by anti-AHR antibody were resolved by SDS-PADGE and immunoblotted with anti-SREBP-1 or anti-AHR antibody.



Supplementary Figure 5. Enlarged Figure 7C.



**Supplementary Figure 6. LXR KO CD4<sup>+</sup> T cells conferred Rag1<sup>-/-</sup> mice stronger susceptibility to EAE development than LXR WT CD4<sup>+</sup> T cells.**

(A) Naive CD4<sup>+</sup> T were isolated from LXR WT or LXR KO mice and injected intravenously into sublethally irradiated (500 rad) Rag1<sup>-/-</sup> mice (10<sup>7</sup> cells/mouse). CFA emulsified with MOG peptide (150 $\mu$ g/mouse) was subcutaneously injected into mice on Day 0 and Day 7 while pertussis toxin (500ng/mouse) was intraperitoneally injected on Day 1 and Day 8. EAE clinical scores are expressed as means  $\pm$  s.e.m. and represent three independent experiments with similar results (6 mice per group). P=0.007, LXR WT control group vs LXR KO group.

(B) 14 days after the second immunization, infiltrates in the brain and spinal cord were isolated for intracellular staining of IL-17 and IFN $\gamma$ .