Supplementary figures

Fig.S1



Fig.S1. IL-15 decreases the frequency of IL-17A+ cells in Th17 cultures. A) Naïve T cells were left unstimulated (unstim.) or stimulated with anti-CD3 and anti-CD28 under Th17 conditions along with indicated concentrations of recombinant mouse IL-15 for 4 days. IL-17A levels were assessed in culture supernatants by ELISA.





Fig. S2. Loss of IL-15 signaling does not lead to increase in Foxp3, IFN- γ , TNF- α , IL-22 and CD25 expression. Naive WT (upper panel) or *II15r^{-/-}* CD4 cells (lower panel) stimulated under Th17 conditions in the absence of APC for 96 hours. Flow cytometric plots Foxp3, IFN- γ , IL-17F, TNF- α , IL-22 and CD25 expression.



Fig. S3. IL-15 suppresses IL-17A production independently of GMCSF. (A)WT or $ll15r^{l-}$ cells CD4 T cells were stimulated as in Fig.3C and the supernatants were collected on indicated days. CD14+ monocytes were derived from splenocytes and were stimulated using IL-4 and GMCSF for 6 days. The cells were washed and restimulated without or with zymosan and the supernatants were collected after 24 hours. GMCSF levels were determined using ELISA.(B) Th17 cells are pathogenic *in vivo*. WT Th17 or $ll15r^{l-}$ Th17 cells were adoptively transfered into CD45.1 *Rag1*^{-/-} mice (n = 14). Some mice received PBS (PBS). Percent weight change on indicated days after IBD induction is shown.

Fig.S4



Fig. S4. Loss of IL-15 signaling impairs STAT5 binding in *il-17a* **locus.** WT (grey bars) or *ll15r* ^{-/-} (white bars) naive CD4 T cells were stimulated under Th17 conditions. On day 5, the cells were restimulated with IL-15 for 20 minutes, fixed and used for ChIP followed by Taqman qPCR. P2, P3, P4 are the primer probe sets for STAT5 binding sites and NB1 and NB2 are the primer probe sets for STAT5 non-binding sites in the *il-17* locus.