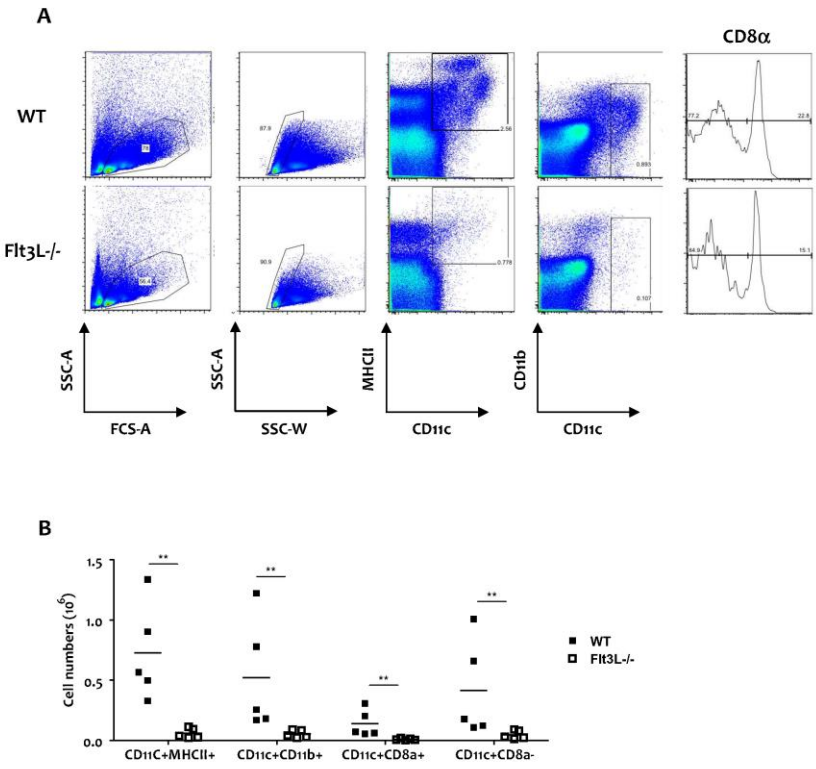
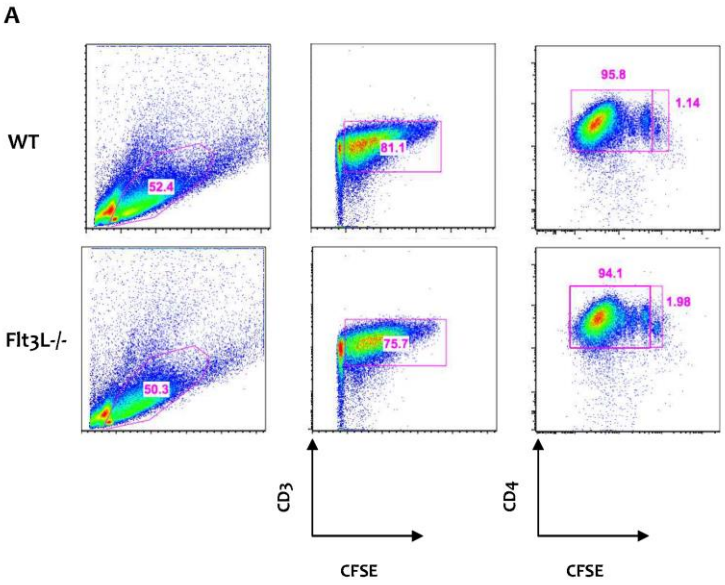
Supplementary Figures:

Supplementary Figure 1. (A) Phenotypic characterization of DC in WT and in Flt3L-/- mice in homeostatic conditions. Inguinal and axilary LNs of WT mice (n=5) and Flt3L-/- (n=5) were collected and DC subsets were identified by flow cytometry. Flt3L-/- animals show reductions in the percentage of CD11c+MHCII+ DC compared with WT animals. (B) Quantification of total DC (CD11c+MHCII+), DC myeloid-related (CD11b+CD11c+ DC,) and lymphoid-related DC (CD8α+ CD11c+ DC) in Flt3L-/- mice and WT animals. Flt3L-/- animals showed a reduction in the percentage of CD11c+MHCII+ DC (p=0.0079) compared with WT animals. The numbers of CD11b+CD11c+ DC (p=0.0079), CD8α+ CD11c+ DC (p=0.0079), CD8α– CD11c+ DC (p=0.0079) were reduced in the LN of Flt3L-/- mice compared with WT mice.



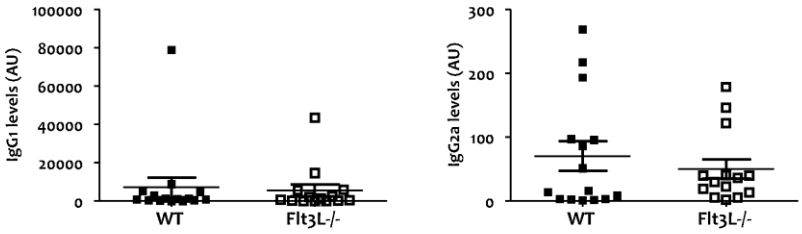
Supplementary Figure 1

Supplementary Figure 2. Capacity of WT and Flt3L-/- BMDCs to induce T cell proliferation in vitro. T cell proliferation was measured by flow cytometry as a loss of CFSE staining. The percentage of proliferating cells is shown. There were no differences in the percentage of proliferating T cells between WT and Flt3L-/- BMDCs. A representative plot is shown (n=4).



Supplementary Figure 2

Supplementary Figure 3. Serum specific collagen type II (CII) antibodies in WT and Flt3L-/- mice during the chronic of CIA as assessed by ELISA. Levels of CII-specific IgG1 and IgG2a antibodies. There were no differences in the amount of CII specific IgG1 and IgG2a antibodies at day 60.



Supplementary Figure 3