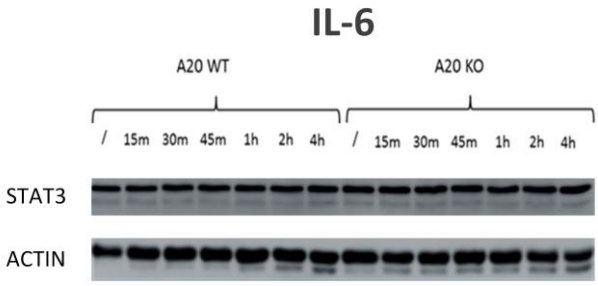
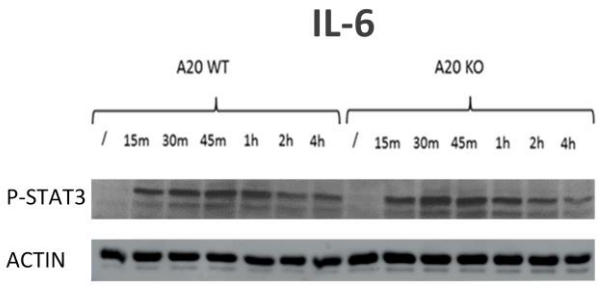
Supplementary figure 1: A20 does not negatively modulate STAT3 expression



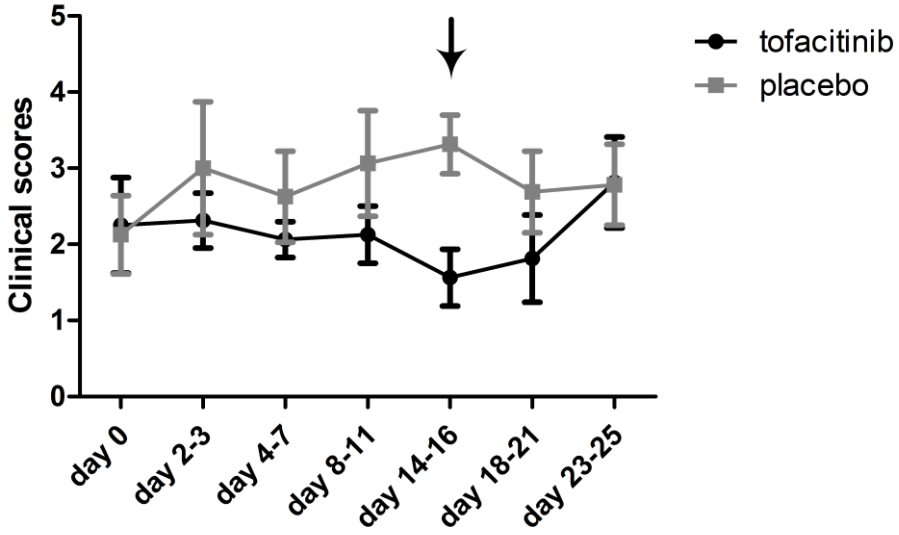
Bone marrow-derived macrophages (BMDMs) were derived from A20^{myelKO} and wild-type littermate mice and stimulated with interleukin (IL)-6 to activate the JAK-STAT pathway. Western blot analysis was performed to determine STAT3 protein levels. A20 did not have an inhibitory effect on STAT3 protein levels in BMDMs. (one out of three independent experiments is shown) (n=9 per group for the three independent experiments combined)

Supplementary figure 2: Effect of A20 on STAT3 phosphorylation status



Bone marrow-derived macrophages (BMDMs) were derived from A20^{myelKO} and wild-type littermate mice and stimulated with interleukin (IL)-6 to activate the JAK-STAT pathway. Western blot analysis was performed to determine P-STAT3 protein levels. A20 did not have an inhibitory effect on P-STAT3 protein levels in BMDMs. (one out of three independent experiments is shown) (n=9 per group for the three independent experiments combined)

Supplementary figure 3: Continued administration of tofacitinib is needed to treat enthesitis in A20^{myelKO} mice: results from a withdrawal study



A20^{myelKO} mice were treated with tofacitinib citrate (50mg/kg, twice daily) or placebo control for 16 days. The mice were clinically scored three times a week during the treatment and during 9 days after discontinuation of the treatment. Clinical scores were shown as the mean of two consecutive clinical scoring time points. During the treatment period clinical inflammation diminished in the tofacitinib citrate-treated mice, while the clinical scores of the placebo-treated mice increased over time. After discontinuing the treatment, the clinical scores of the tofacitinib citrate-treated mice increased to the same level as the clinical scores of the placebo-treated mice.