

Supplementary Figure 1: Prolonged TNF α synergizes with interferons (IFN) for induction of *CXCL9* and *CXCL11* in RA FLS. (A-B), RA FLS were cultured for 3 days in the presence or absence of TNF α (10 ng/ml), which was added on the first day of culture and was not replenished. On day 3 the cells were stimulated with IFN β (1,000 U/ml) or IFN γ (100 U/ml) for 3 hours. The expression of *CXCL9/MIG* (A) and *CXCL11/ITAC* (B) mRNA was measured by real-time quantitative reverse transcription-polymerase chain reaction (qPCR). Values are the mean \pm SEM and were normalized relative to mRNA for GAPDH. *= $p < 0.05$, **= $p < 0.01$, and ***= $p < 0.001$, by one-tailed paired Student t test. (C), *CXCL9*, *CXCL10* and *CXCL11* genes are located next to each other on the long arm of chromosome 4.

Supplementary Figure 2: Priming effect of TNF α on interferon responses of RA FLS is gene specific. RA FLS were cultured for 3 days in the presence or absence of TNF α (10 ng/ml), which was added on the first day of culture and was not replenished. On day 3 the cells were stimulated with IFN β (1,000 U/ml) or IFN γ (100 U/ml) for 3 hours. The mRNA expression of classic interferon-targets including *SOCS1* (A), *STAT1* (B), *IRF1* (C) and *IFIT2/ISG54* was measured by real-time quantitative reverse transcription-polymerase chain reaction (qPCR). Values are the mean \pm SEM and were normalized relative to mRNA for GAPDH.

Supplementary Figure 3: STAT1-amplifier function of prolonged TNF α is retained in RA FLS despite the removal of TNF α . RA FLS were cultured for 3 days in the

presence or absence of TNF α (10 ng/ml), which was added on the first day of culture and was not replenished. Then inflammatory input was removed by washing cells, adding new medium and blocking any residual TNF α with infliximab (10 μ g/ml). Following a weaning period of 24 hours, cells were stimulated with IFN β (1,000 U/ml) (**A**) or IFN γ (100 U/ml) (**B**) for 0-60 minutes. STAT1 tyrosine phosphorylation (pY) and total STAT1 protein were measured by immunoblotting. AKT was used as loading control. Results are representative of three independent experiments.