SUPPLEMENTAL MATERIAL

Noti et al., http://www.jem.org/cgi/content/full/jem.20090849/DC1

Figure S1. TNF is required for TNBS-induced colitis and GC synthesis. (A and B) Weight loss (A) and colon length (B) in control- and TNBS-treated TNF+/+ and TNF−/− mice. Means ± SD or individual values of four mice per group are shown. (C and D) CYP11B1 mRNA expression levels (C) and GC synthesis (D) in colonic tissue from control- and TNBS-treated TNF+/+ and TNF−/− mice at day 3. Means ± SD or individual values of five mice per group.

Figure S2. TNF induces expression of steroidogenic enzymes in primary intestinal crypt cells. (A–D) Crypt cells from small (A and B) and large intestine (C and D) were isolated and cultured for 4 h ex vivo in the presence of 0, 3, 10, or 30 ng/ml TNF. RNA was isolated and CYP11A1 (A and C) and CYP11B1 (B and D) expression levels were detected by quantitative RT-PCR. Means of triplicates ± SD of one typical experiment out of three are shown.
Figure S3. Therapeutic effects of TNF in DSS-induced colitis. (A–E) DSS-treated animals were injected daily up to day 8 with 1 μg mouse TNF or PBS control, and weight loss (A) was recorded daily. All animals were sacrificed at day 8 and colonic MPO levels (B) as well as colonic CYP11A1 (C) and CYP11B1 (D) mRNA expression levels were analyzed. Intestinal GC synthesis was measured after ex vivo organ culture, as described in Materials and methods (E; n = 5 mice per group). *, P < 0.05; **, P < 0.01.