Supporting Information

Normand et al. 10.1073/pnas.1100981108



Fig. S1. Generation of *NIrp6*-deficient mice. (*A*) Scheme of generation of *NIrp6*-deficient mice. (*B*) RT-PCR analysis of *NIrp6*-deficient mice in wild-type and mutant mice. (*C*) Genotyping of *NIrp6*-deficient mice by PCR on mouse tail genomic DNA. The bands corresponding to mutant and wild-type alleles are depicted.



Fig. 52. Anakinra therapy failed to rescue $Nlrp6^{-/-}$ mice from dextran sodium sulfate (DSS)-induced injury. Untreated (n = 4) and Anakinra-treated (100 mg/kg per day, n = 4) Nlrp6-deficient mice ($Nlrp6^{-/-}$) were challenged with 3% DSS for 5 d followed by a 5-d period of regular drinking water. Body weight loss (A) and clinical score (B), including stool consistency and presence of blood, was monitored daily.



Fig. S3. Intestinal tumorigenesis induces an abnormal transcriptional response in *Nlrp6*-deficient mice. Venn diagram is depicted (*A*) after transcriptional profiling of tumoral (T) and nontumoral (NT) biopsies from two wild-type and two *Nlrp6*-deficient mice (*B*). (*C*) Gene ontology analysis on up- and down-regulated genes that are differentially expressed in *Nlrp6*-deficient mice, as described in *Materials and Methods*. (*D*) Relative gene expression on tumoral (T) and nontumoral (NT) colonic specimens were determined by quantitative RT-PCR analysis. **P < 0.01, ***P < 0.001.

DN A C

S A L



Fig. S4. Enhanced tumoral expression of Ccl24 and Xbp1 independently of NLRP6. Relative gene expression on tumoral (T) and nontumoral (NT) colonic specimens (n = 8) were determined by quantitative RT-PCR analysis. *P < 0.05, **P < 0.01.

DNAS