SUPPLEMENTAL MATERIAL

Salcedo et al., http://www.jem.org/cgi/content/full/jem.20100199/DC1

Figure S1. The effect of MyD88 deficiency on susceptibility to AOM/DSS-induced colitis and CAC is maintained when they are compared with littermate WT mice. Myd88−/− mice and their corresponding heterozygote and WT littermates were subjected to AOM/DSS treatment as indicated by the scheme. During the administration of the first DSS cycle, mice were monitored for bleeding (A). At day 48 after AOM administration, mice were euthanized, colons were resected and measured (B), and polyps were counted (C). The percentage of survival during the course of the experiment is depicted (D). The data shown in A–D correspond to a representative experiment out of two performed using littermate controls. Data represent means ± SE. *, P < 0.05.
Figure S2. **Comparative effects of AOM versus DSS treatment in Myd88−/− mice.** Cohorts of 8–18 mice/group were subjected to either AOM or DSS treatment as depicted by the schemes. 6 mo after AOM administration, mice were euthanized, the colon length was measured (A), and colonic polyps were counted (B). The percentage of gain or loss of body weight was calculated as a mean per group during the course of the experiment (C). The data shown in A–C were obtained from a single experiment performed and reproduce data published by others (Rakoff-Nahoum and Medzhitov, 2007). At the end of the second DSS cycle treatment, mice were euthanized, colons were harvested and measured (D), and the number of colonic polyps were counted (E). The data pooled from three different experiments are shown in D and E. Data represent means ± SE. *, P < 0.05; **P < 0.01.
Figure S3. Unsupervised heat map of genes differentially expressed among treated WT versus treated $\text{Myd88}^{-/-}$ mice. The microarray data were deposited in the GEO database and the accession no. is GSE19793. Standard filter was applied (80% presence call, twofold ratio cutoff, P < 0.005) that allowed 1,188 genes. To obtain the microarray data, 10 mice per group were treated with AOM/DSS and six untreated controls per group were used. This experiment was performed once.
Figure S4. AOM/DSS treatment enhances the expression of genes involved in the EGFR and β-catenin pathways on Myd88−/− mice. Ingenuity pathway analysis of treated WT and treated Myd88−/− mice shows increased expression of genes involved in EGFR and β-catenin signaling. The graphic was adapted from Ingenuity System.
Figure S5. *Il18*−/− mice are highly susceptible to DSS colitis. Cohorts of 9–10 mice/group were subjected to treatment with 5% DSS in drinking water for 5 d, as depicted by the scheme. During the course of the DSS administration, mice were monitored for macroscopic bleeding (A) and diarrhea (B). Mice were euthanized, and colons were resected and measured (C). Tissue sections were analyzed and the histology scores were calculated as described in the Materials and methods (D). The percent of mucosa in the distal and middle colon exhibiting ulceration is shown (E). Photomicrographs of three representative colon sections from *Il18*-deficient mice compared with a representative control, indicating extensive ulcerated areas, are shown at 20× magnification (F). The data shown in A–F correspond to a representative experiment out of two performed. Data represent means (horizontal bars) ± SE. *, *P < 0.05; ***, *P < 0.001.

Figure S6. *Il18r*−/− mice are susceptible to AOM/DSS administration, similar to *Il18*−/− mice. Cohorts of 9–10 WT and *Il18r*−/− mice per group were injected i.v. with AOM on day 0 followed by four DSS cycles administered in drinking water. At the completion of the first DSS cycle, mice were monitored for bleeding (A). After treatment, mice were monitored for survival (P = 0.0019, WT vs. *Il18r*−/−; B). When mice reached any of the end points for euthanasia or at the completion of the experiment, colons were resected and polyps were counted (P = 0.0002, WT vs. *Il18r*−/−). Data represent means ± SE. **, *P < 0.01; ***, *P < 0.001.
Table S1. Real-time PCR primer sequences and experimental conditions

<table>
<thead>
<tr>
<th>Genes</th>
<th>Primer sequences</th>
<th>Product size</th>
<th>Tm °C</th>
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<tr>
<td>Il6</td>
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<td>Areg</td>
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<td>Mmp10</td>
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<td>Cox2</td>
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Tm, temperature of melting.