**Supplementary table and figure legends**

**Table S1. Histopathological analysis of colonic samples from sepsis-surviving and sham-operated mice.**

| | Normal mucosa (%) | Adenoma with LGD (%) | Adenoma with HGD (%) | Invasive adenocarcinoma (%) |
|---|---|---|---|---|
| Sham | 0 | 0 | 50 | 50 |
| Post-sepsis | 75 | 25 | 0 | 0 |

LGD denotes low-grade dysplasia; HGD denotes high-grade dysplasia.

**Figure S1. Cyclophosphamide-induced Treg ablation restores colonic inflammation and cancer development in post-sepsis mice.** A) Endoscopic scores of colitis at days 12, 32, and 52, evidencing colitis in Treg-depleted post-sepsis mice. D) Endoscopic number of colonic tumors and E) tumor load showing that reduction of Tregs induced by CYP increased the incidence of tumor and the tumor load. Data, means ± S.E.M. The experiments were repeated twice. Data demonstrate a representative experiment. Data, ± S.E.M. *, $P < 0.0001$

**Figure S2. Tregs mediate the inhibition of colorectal-associated cancer in post-sepsis disorder.** A) Inflammatory process induced by AOM/DSS in intestinal mucosa, producing ulcers, inflammatory cell infiltration and CAC initiation/promotion. B) Colitis/CAC impairment in the post-sepsis state, showing the role of Tregs in the inhibition of pro-tumor inflammation and, consequently, preclusion of CAC carcinogenesis.