indicated in serum-reduced media for 6h, and cell proliferation was determined by EdU incorporation for 30 minutes. Arrowheads indicate nuclear condensation.

Figure S1. Induction of CK2 by inflammatory cytokines. (A) Comparative expression of CK2α and Cdc37 pS13 in primary mouse IEC, IEC-6, SK-CO15, and SW480. Equivalent protein concentration was confirmed by Ponceau S staining prior to immunoblotting. (B) CK2α expression in IEC-6 cells after 7d treatment with the indicated cytokines (IL-10 25ng/ml; IL-22 200ng/ml; TGF-β1 50ng/ml; all others 10ng/ml). (C) CK2α expression in IEC-6 cells after 7d treatment with IL-1β. (D) CK2α and CK2α' expression in IEC-6 cells after 1-3d treatment with the indicated cytokines. (E) IEC-6 cells were treated with the indicated cytokines (10ng/ml) for 24h, and total pixel intensity of CK2α (cytosolic plus nuclear) was determined by confocal microscopy. n = 3 independent experiments with >100 cells per condition.

Figure S2. Immunolocalization of CK2α and Cdc37 pS13 in mouse and human tissues. (A) IHC of CK2α in chronic DSS colitis tissue using a polyclonal antibody. Tissue was briefly counterstained with hematoxylin. (B) IHC of PCNA and β-catenin shows correct localization to crypt base nuclei and epithelial plasma membrane, respectively. (C) IHC of Cdc37 pS13 in chronic DSS colitis samples shows enhanced nuclear staining in crypts adjacent to mucosal ulcers. (D, E) Co-immunostaining of CK2α (white) with (D) Ki67 and (E) β-catenin (red) in human tissue samples.

Figure S3. Inhibition of CK2 induces normal IEC apoptosis. (A) IEC-6 cells were treated as indicated for 8h, and analysed by immunoblot for caspase-9 cleavage and inhibition of potential off-target GSK-3β. (B) IEC-6 cells were treated as indicated for 12h. Proliferation was assessed by EdU incorporation for 30 minutes (green), and caspase-3 cleavage is shown in red. (C) IEC-6 cells were treated as indicated in the presence of IFN-γ and TNF-α (10ng/ml each) for 12h. Proliferation was determined by EdU incorporation for 30 minutes (shown in red). Active caspase-3 staining is shown in green.

Figure S4. Effect of CK2 modulation on IEC migration proliferation. (A) IEC-6 cells were scratch-wounded and migrated for 8h. Results are representative of 2 experiments, with 6 replicates per condition. (B) Representative kymographs of IEC-6 cells migrating for 1h. Arrowheads indicate the position of the leading lamellipodium. The velocity of 20 leading edge cells per condition from 2 experiments is shown on the right. (C) IEC-6 cells were scratch-wounded, treated as indicated, and migrated for 4h. Caspase-9 activation is shown in red. (D) CK2α was stably overexpressed in IEC-6 using a lentiviral SNAP-CK2α construct. Representative immunblots show CK2α expression in one experiment with three wells per condition. Membranes were reprobed for SNAP and actin (below). (D) Control and cytokine-treated (10ng/ml each for 16h) IEC-6 cells were assessed for EdU incorporation. CK2α overexpression increased baseline cell proliferation. Results show one experiment with three wells per condition.

Supplemental Table 1. Real-time PCR primers for mouse CK2.

Csnk2a1 fw	5′-TGG GTG TCT CAT TCA AAC CT-3′
Csnk2a1 rv	5′-GGA TCC TTA ATC CCC AGA GA-3′
Csnk2a2 fw	5′-GGA GGC CCT AGA TCT TCT TG-3′
Csnk2a2 rv	5′-CGC GTT AAG ACG TTT TGA TT-3′
Csnk2b fw	5′-TGA AAC TCT ACT GCC CCA AG-3′
Csnk2b rv	5′-GTC TTG ACT GGG CTC TTG AA-3′