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Supplementary data

Design and evaluation of IKK-activated GSK3β inhibitory peptide as an inflammation-responsive anti-colitic therapeutics

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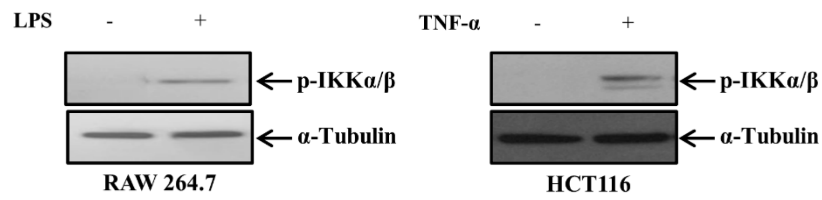

Figure S1. IKK phosphorylation by stimulation with inflammatory mediators

HCT116 and RAW 264.7 cells were stimulated with TNF-α and LPS, respectively. Cells were lysed 10 min (for TNF-α) or 1 h (for LPS) later. Western blot analysis was performed to detect p-IKK. α-Tubulin was blotted as a loading control.

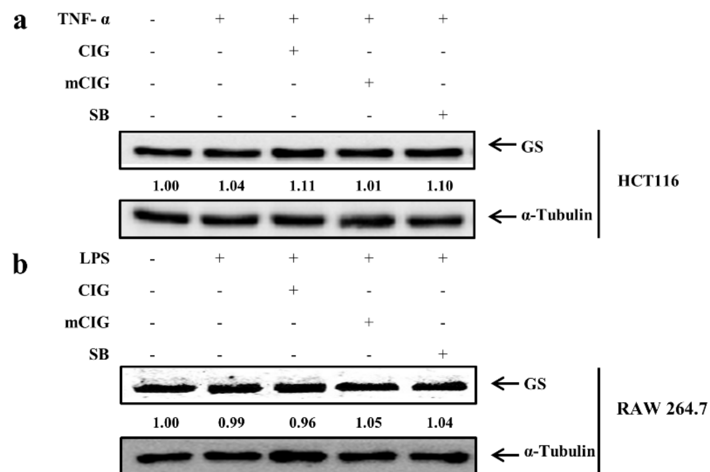

Figure S2. Levels of glycogen synthase protein after treatment with peptides and SB2106781 in cells

(A) HCT116 cells were pretreated with CTP-IAGIP (CIG, 200 μM), CTP-mIAGIP (mCIG, 200 μM) or SB216763 (SB, 10 μM) for 1 h followed by stimulation with TNF-α (10 ng/mL) for 1 h. Levels of glycogen synthase (GS) protein were monitored in whole cell lysates. (B) RAW 264.7 cells were pretreated with CTP-IAGIP (CIG, 200 μM), CTP-mIAGIP (mCIG, 200 μM), SB216763 (SB, 10 μM) for 1 h followed by stimulation with LPS for 1 h. Levels of GS protein were monitored in whole cell lysates. In (A) and (B), α-Tubulin was blotted as a loading control. Blots are representative of three separate experiments.

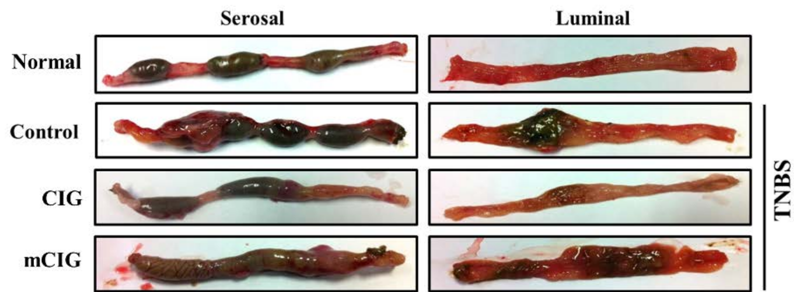

Figure S3. Photos of the luminal and serosal sides of the distal colons

CTP-IAGIP (CIG, 200 μM, 500 μL), CTP-mIAGIP (mCIG, 200 μM, 500 μL) was administered rectally to TNBS-induced colitis rats once a day for 6 days 72 h after induction of colitis. The luminal and serosal sides of the distal colons were photographed.