TableS1 Primers for qPCR

Genes	Forward	Reverse
TNFα	5'-CATCTTCTCAAAATTCGAGTGACAA-3'	5'-TGGGAGTAGACAAGGTACAACCC-3'
CCL2	5'-ACTGAAGCCAGCTCTCTCTTCCTC-3'	5'-TTCCTTCTTGGGGTCAGCACAGAC-3'
CCL5	5'-CCACTTCTTCTCTGGGTTGG-3'	5'-GTGCCCACGTCAAGGAGTAT-3'
CXCL1	5'-GGCGCCTATCGCCAATGA-3'	5'-GACTTCGGTTTGGGTGCAGT-3'
CXCL2	5'-GAAGACCCTGCCAAGGGTTG-3'	5'-AGGCAAACTTTTTGACCGCC-3'
CX3CL1	5'-GCAAGTTTGAGAAGCGGGTG-3'	5'-CTTGGGAAGTCCCCATGGTC-3'
CXCL5	5'-TGCATTCCGCTTAGCTTTCT-3'	5'-CAGAAGGAGGTCTGTCTGGA-3'
CXCL10	5'-CCAAGTGCTGCCGTCATTTT-3'	5'-CTCAACACGTGGGCAGGATA-3'
CSF1	5'-TGGCTTGGCTTGGGATGATT-3'	5'-GTCTGTCCCCATGGTTTGGT-3'
iNOS	5'-CAGGGCCACCTCTACATTTG-3'	5'-TGCCCCATAGGAAAAGACTG-3'
36B4	5'-AAGCGCGTCCTGGCATTGTCT-3'	5'-CCGCAGGGGCAGCAG TGGT-3'
G6pase	5'-CCGGTGTTTGAACGTCATCT-3'	5'-CAATGCCTGACAAGACTCCA-3'
PEPCK	5'-ATCATCTTTGGTGGCCGTAG-3'	5'-ATCTTGCCCTTGTGTTCTGC-3'
BAFFR	5'-CCAGCAAGAGTCCCTGGAAAAT-3'	5'-CTCCACTGCTGCTATTGCTCT-3'
CD40	5'-TTGTTGACAGCGGTCCATCT-3'	5'-GCGAATCTCCCTGTTCCACT-3'
LTβR	5'-CAGCTGGTGCCCCCTTATC-3'	5'-AAGACAAACTCGCCTGGGG-3'
RANK	5'-CCTTCGACTGGTTCACTGCT-3'	5'-GGACACGGGCATAGAGTCAG-3'
NIK	5'-TCTCTGGAGGAACAGGAACAA-3'	5'-GCCATTGAGAGACTGGATCTG-3'

Figure S1

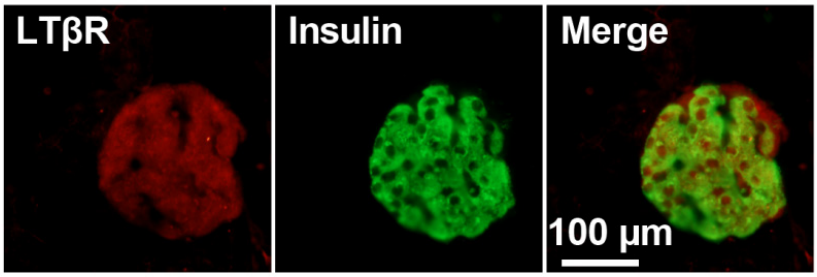

Figure S1. LTβR is present in the insulin-positive cells. Immunostaining of LTβR and insulin in the section of pancreas.

Figure S2

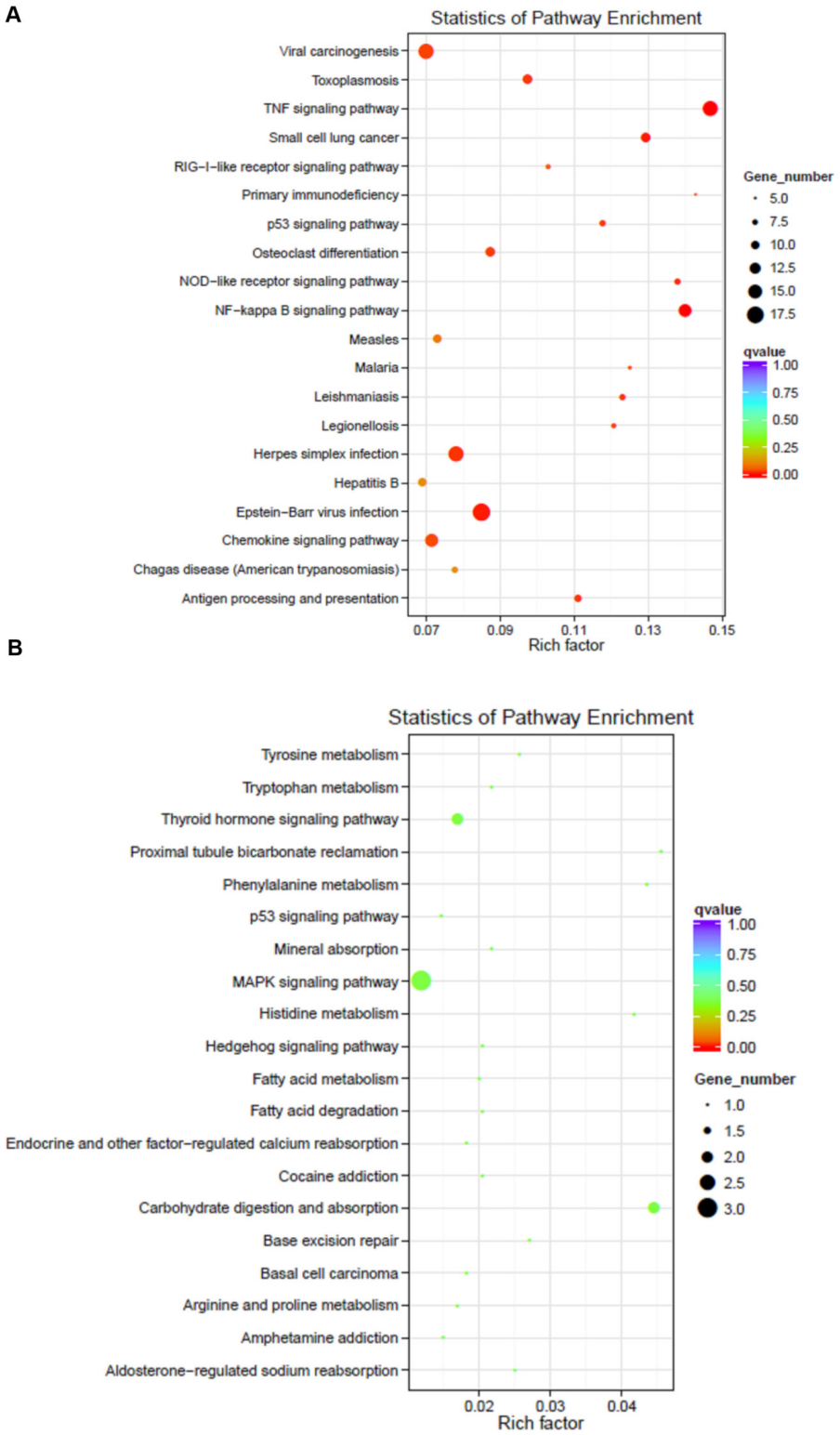

Figure S2. NIK overexpression activates immune signaling pathways in αTC1-6 cells. A-B, KEGG pathway enrichment analysis of up- and down-regulated genes in NIK-overexpressing αTC1-6 cells compared with β-Gal control.

Figure S3

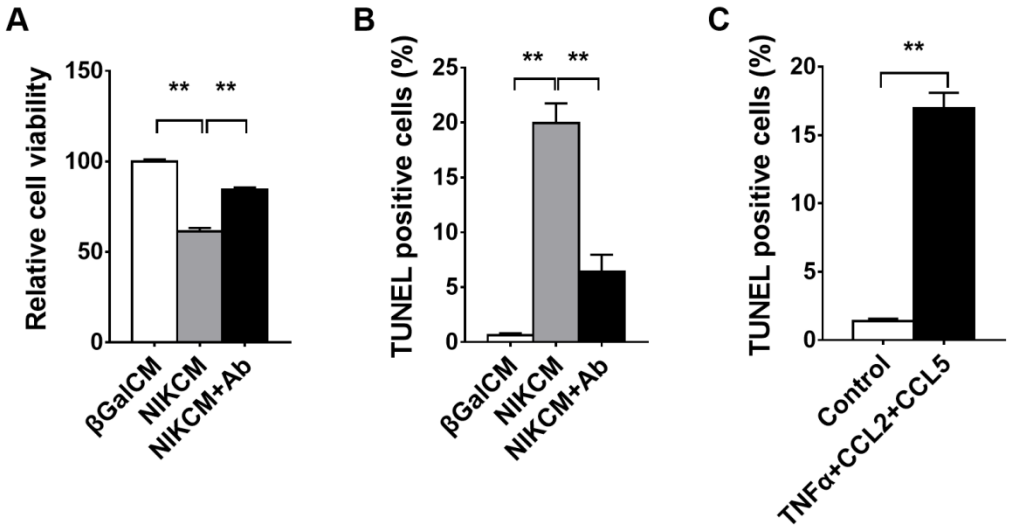

Figure S3. Conditioned media from NIK overexpressing αTC1-6 cells induces β-cell death.

Conditioned media were collected from NIK-overexpressing αTC1-6 cells (NIKCM) and control cells (βGalCM). Half of NIKCM was neutralized with antibodies against TNFα, CCL2 and CCL5, denoted as NIKCM+Ab. INS-1 832/13 cells were treated with these conditioned media for 16 h. Cell viability was measured by MTT assays (A), and cell apoptosis was measured by TUNEL staining (B). A combination of TNFα (5 ng/mL), CCL2 (100 ng/mL) and CCL5 (100 ng/mL) were used to treat INS-1 832/13 cells for 16 h, and TUNEL positive cells were measured (C) (n = 3-4/group). **, $p < 0.01$.

Figure S4

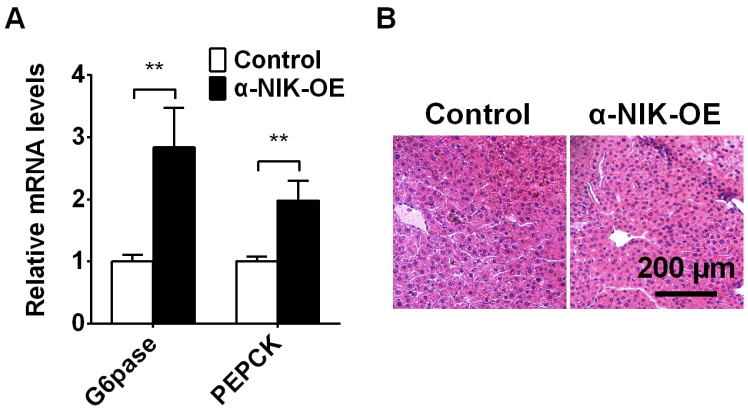

Figure S4. α-NIK-OE mice display high expression levels of *G6pase* and *PEPCK* and normal structure in the liver as compared to those in control mice. A, *G6pase* and *PEPCK* mRNA levels were measured by RT-qPCR assays (n = 10-11/group). B, H&E staining. **, $p < 0.01$.

Figure S5

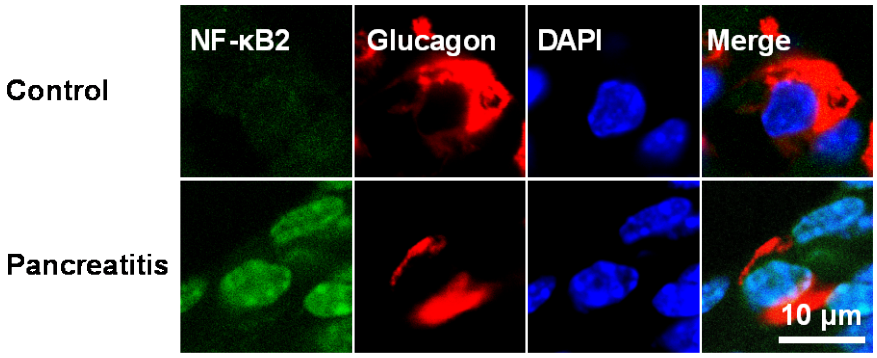

Figure S5. Nuclear NF-κB2 is increased in pancreatic islet α cells in acute pancreatitis mouse model. Pancreatic cryosections of cerulein-induced acute pancreatitis were coimmunostained with NF-κB2 and glucagon.

Figure S6.

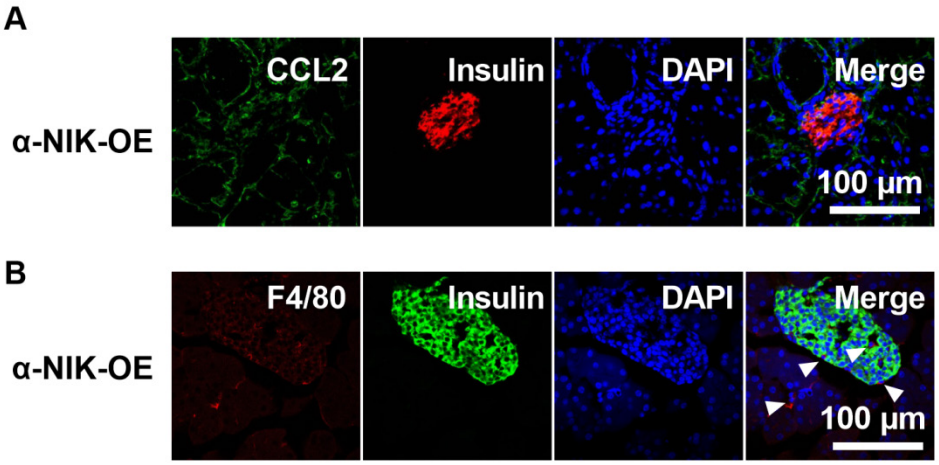

Figure S6. α-NIK-OE mice display insulitis. Pancreatic cryosections of α-NIK-OE mice were co-immunostained with CCL2 and insulin (A) or F4/80 and insulin (B).

Figure S7

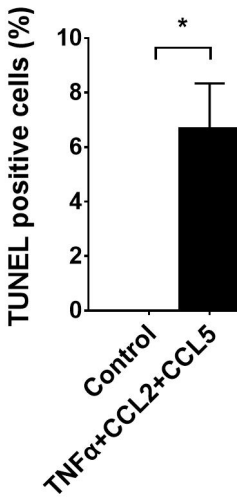

Figure S7. Cytokine and chemokine induce acinar cell death. Primary pancreatic acinar cells were isolated from C57BL/6 mice, and then treated with or without a combination of TNFα (5 ng/mL), CCL2 (100 ng/mL) and CCL5 (100 ng/mL) for 16 hours. TUNEL positive cells were measured (n = 3/group). *, $p < 0.05$.

Figure S8

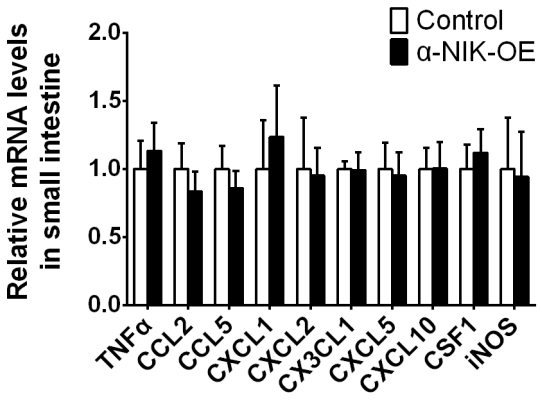

Figure S8. α-NIK-OE and control mice display similar expression of inflammatory genes in small intestine. The expression levels of inflammatory genes in small intestine were measured by RT-qPCR assay (n = 9-11/group).