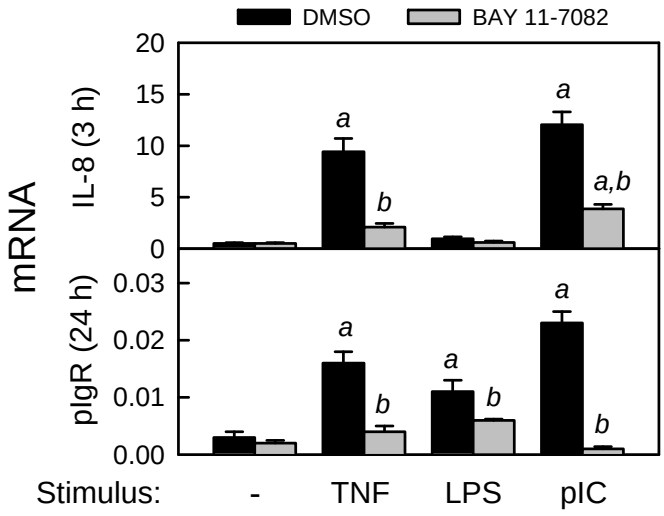
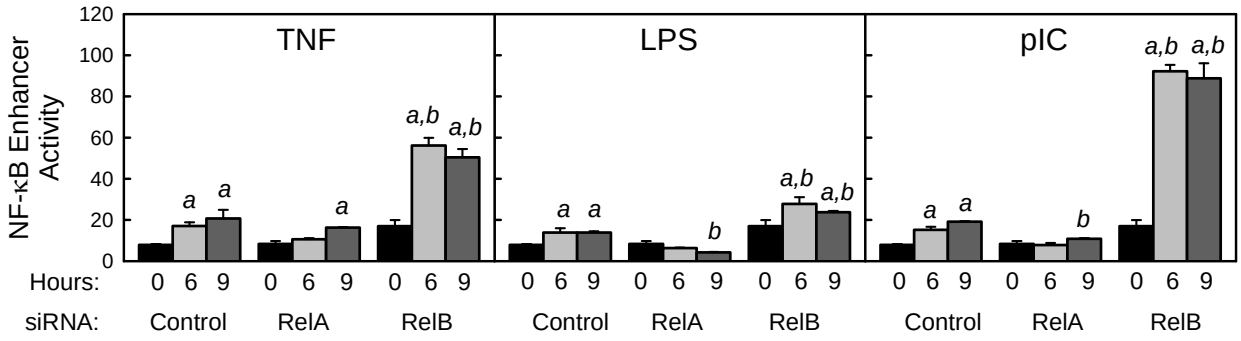


Supplementary Figure 1 Effect of NF-κB inhibition on the response of the LoVo human IEC line to LPS, pIC and TNF. LoVo cells were stimulated with TNF (10 ng/ml), LPS (1 μg/ml) or pIC (100 μg/ml) for 3 or 24 h, in the presence or absence of 10 μM BAY 11-7082, an inhibitor of IκBα phosphorylation. Cells cultured in the absence of BAY 11-7082 were treated with an equivalent volume of vehicle (DMSO). mRNA levels for IL-8 and pIgR were quantified by qRT-PCR and normalized to GAPDH mRNA. Data are expressed as mean ± SEM (n = 4): *a*, the mean for stimulated cells is significantly greater than the mean for unstimulated cells ($p < 0.05$); *b*, the mean for cells treated with BAY 11-7082 is significantly different from the mean for cells given the same stimulus in the absence of BAY 11-7082 ($p < 0.05$).



Supplementary Figure 2 Effects of RelA and RelB knockdown on NF-κB enhancer activity in the HT-29 human intestinal epithelial cell-line. HT-29 cells stably transfected with the indicated siRNA plasmids were transiently transfected with an NF-κB regulated firefly luciferase reporter plasmid (pNF-κB-TK-luc) or an enhancerless control plasmid (pTK-luc). Twenty-four h after transfection, cells were stimulated for 6 h or 9 h with TNF (10 ng/ml), LPS (1μg/ml) or pIC (100 μg/ml). Firefly luciferase activity was analyzed in cell lysates and normalized to the activity of a co-transfected Renilla luciferase plasmid. NF-κB enhancer activity was calculated by subtracting the average luciferase activity of cells treated with pTK-luc from the luciferase activity of cells treated with pNF-κB-TK-luc. Data are expressed as mean ± SEM (n = 4): *a*, the mean for stimulated cells is significantly greater than the mean for unstimulated cells expressing the same siRNA ($p < 0.05$); *b*, the mean for cells expressing RelA or RelB siRNA is significantly different from the mean for cells expressing control siRNA given the same stimulus ($p < 0.05$).