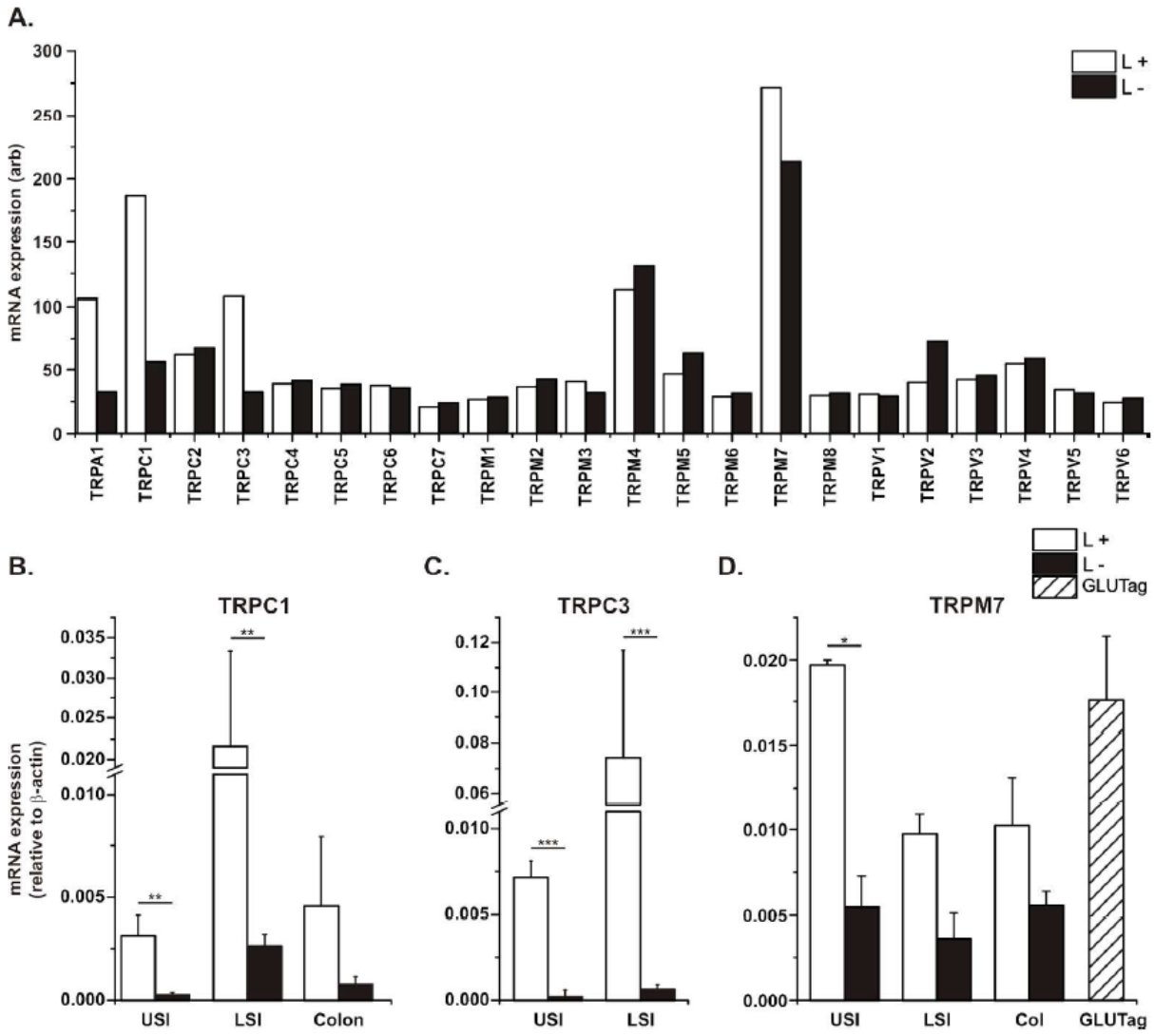
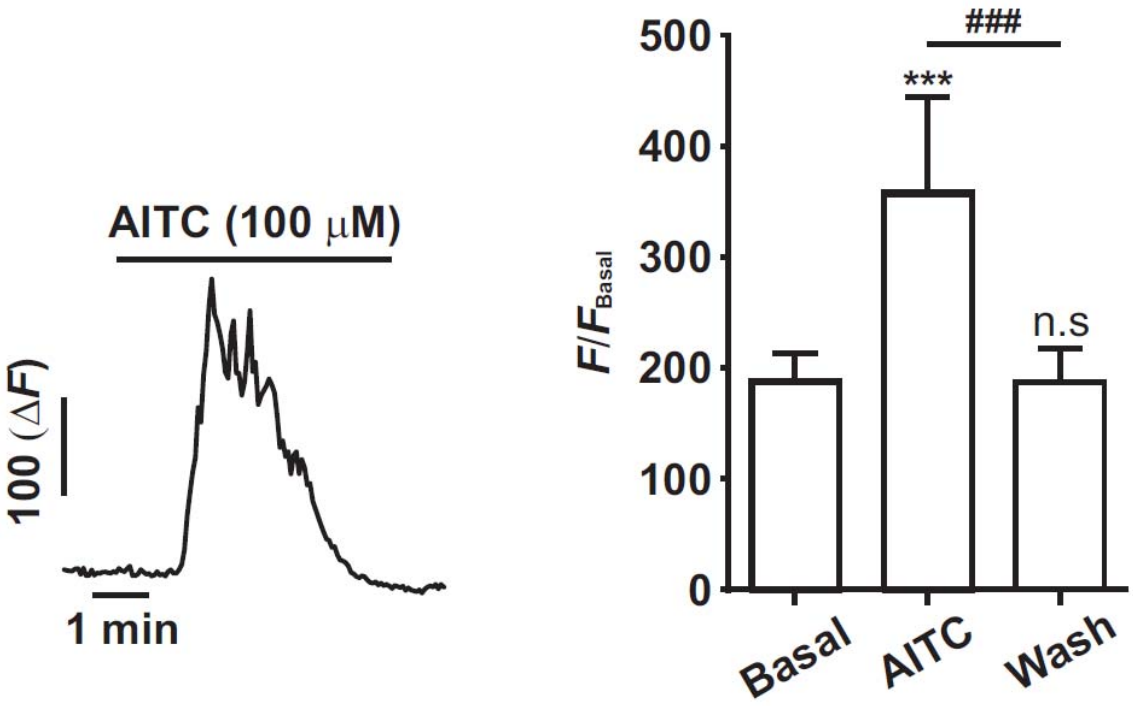
SUPPLEMENTARY DATA

Supplementary Figure 1. mRNA analysis of TRP channel expression within murine L-cells of the small and large intestine. A. Microarray screen of 22 TRP channel superfamily members. Expression of each TRP channel mRNA was assessed from murine small intestinal (L+) cells, and compared to the negative population (L−). B-D. Quantitative real-time PCR analyses for *trpc1*, *trpc3* and *trpm7* from the upper small intestine (USI), lower small intestine (LSI) and colon (Col), as well as GLUTag cells. Primary L+ cells were compared to their negative population (L−). Values were normalised to the expression of β-actin from the same cell populations (n=≥3, **p*<0.05, ***p*<0.01, ****p*<0.001, two-way unpaired Student's t-test).



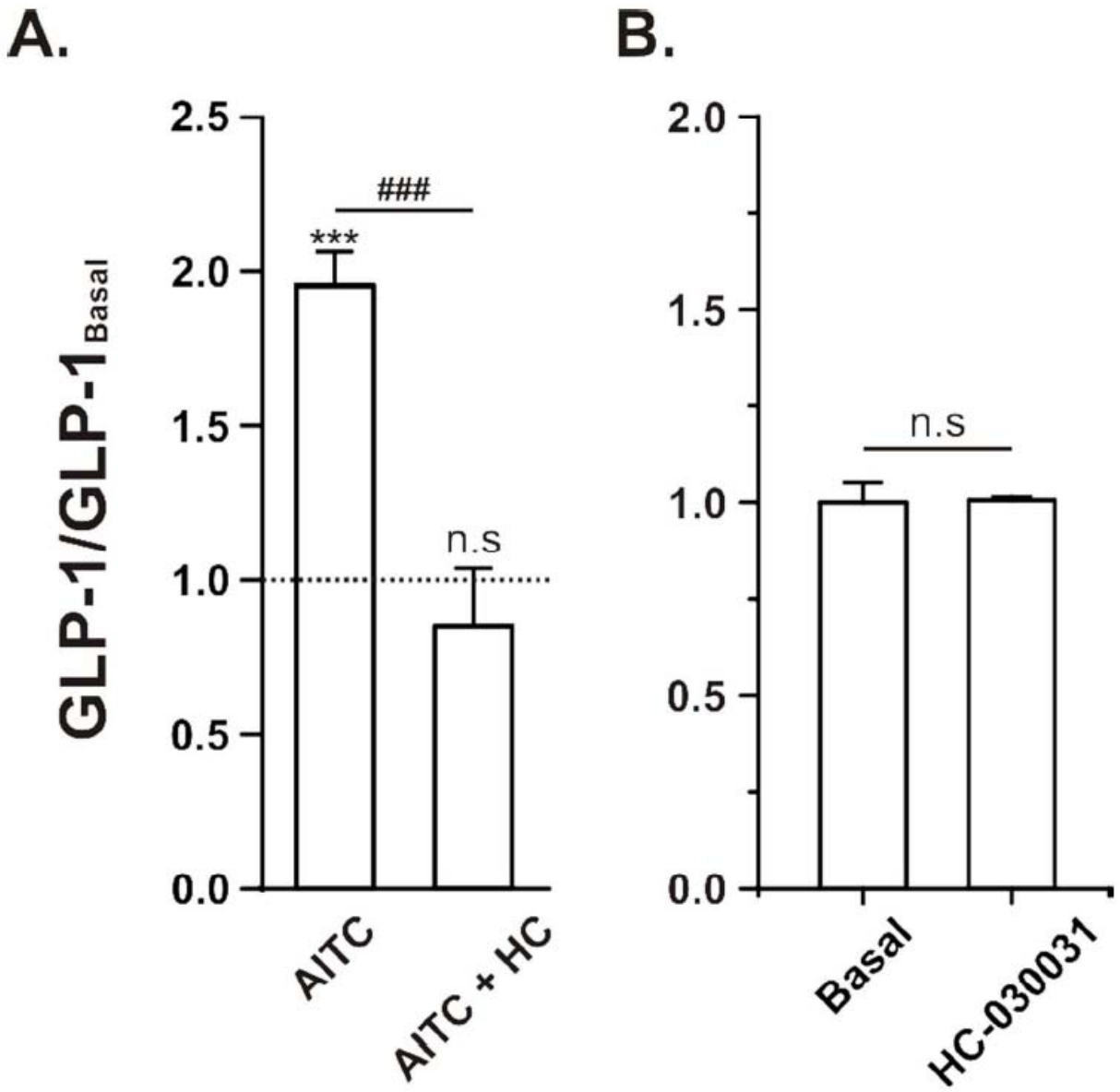
SUPPLEMENTARY DATA

Supplementary Figure 2. TRPA1 agonism causes an increase in intracellular Ca2+ levels in murine ileal L-cells. The application of AITC (100 μM) caused a significant increase in intracellular Ca^{2+} levels as measured by GCaMP3 fluorescence from ileal L-cells (n=5, ***/$^{###}p$<0.001, repeated measures ANOVA with Bonferroni post hoc test). * indicates significance level from baseline and $^{\#}$ indicates significance level between groups.



SUPPLEMENTARY DATA

Supplementary Figure 3. TRPA1 activation or inhibition is independent of glucose-stimulated GLP-1 secretion in GLUTag cells. A. The incubation of AITC (100 μM) for two hours caused an approximate two-fold increase in GLP-1 secretion from GLUTag cells, which was completely inhibited by the co-incubation of the TRPA1 inhibitor HC-030031 (50 μM) B. Incubation of TRPA1 inhibitor HC-030031 (50 μM) had no effect on basal GLP-1 secretion (10 mM glucose) from GLUTag cells. All experiments were n=≥6, ***p<0.001, two-way unpaired Student's t-test. * indicates significance level from baseline and # indicates significance level between groups. The dotted line on each graph represents the respective baseline value.



SUPPLEMENTARY DATA

Supplementary Figure 4. Effect of TRPA1 activation by carvacrol on plasma GLP-1 levels, *in vivo*. A. Intraduodenal administration of carvacrol (2 mM; 0.6 mL) or vehicle (PBS) caused no significant increase in plasma GLP-1 from blood taken from the portal vein at five minutes (n=9-10). In contrast, however, administration of glucose (20% w/v) did cause a significant increase in plasma GLP-1 (n=3, ***p<0.001, two-way unpaired Student's t-test). B. An increase in the concentration and volume of carvacrol administered (4 mM; 1.2 mL), still caused no significant increase in plasma GLP-1 concentrations (n=5; two-way unpaired Student's t-test).