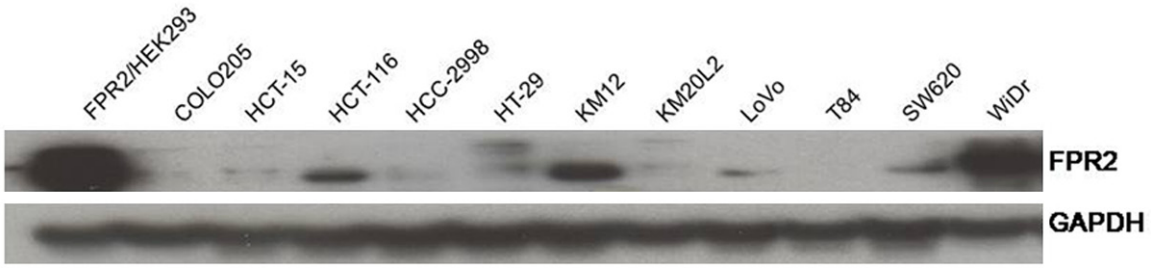
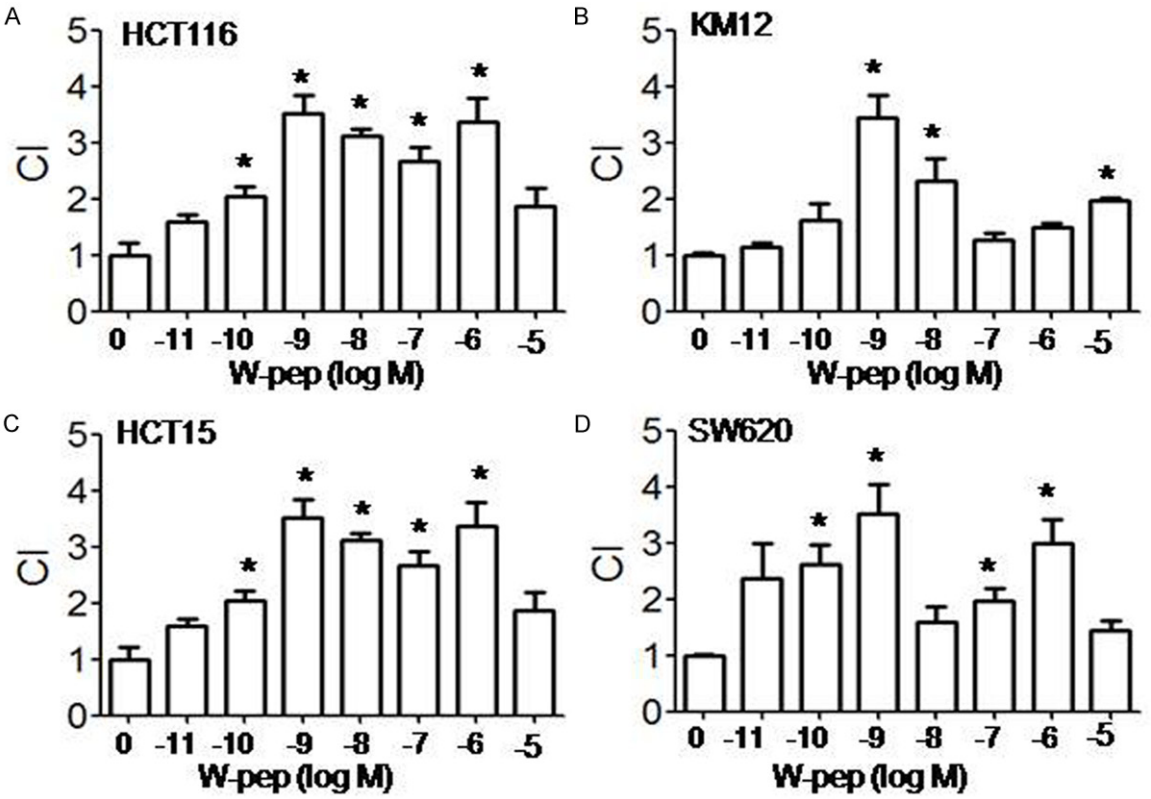
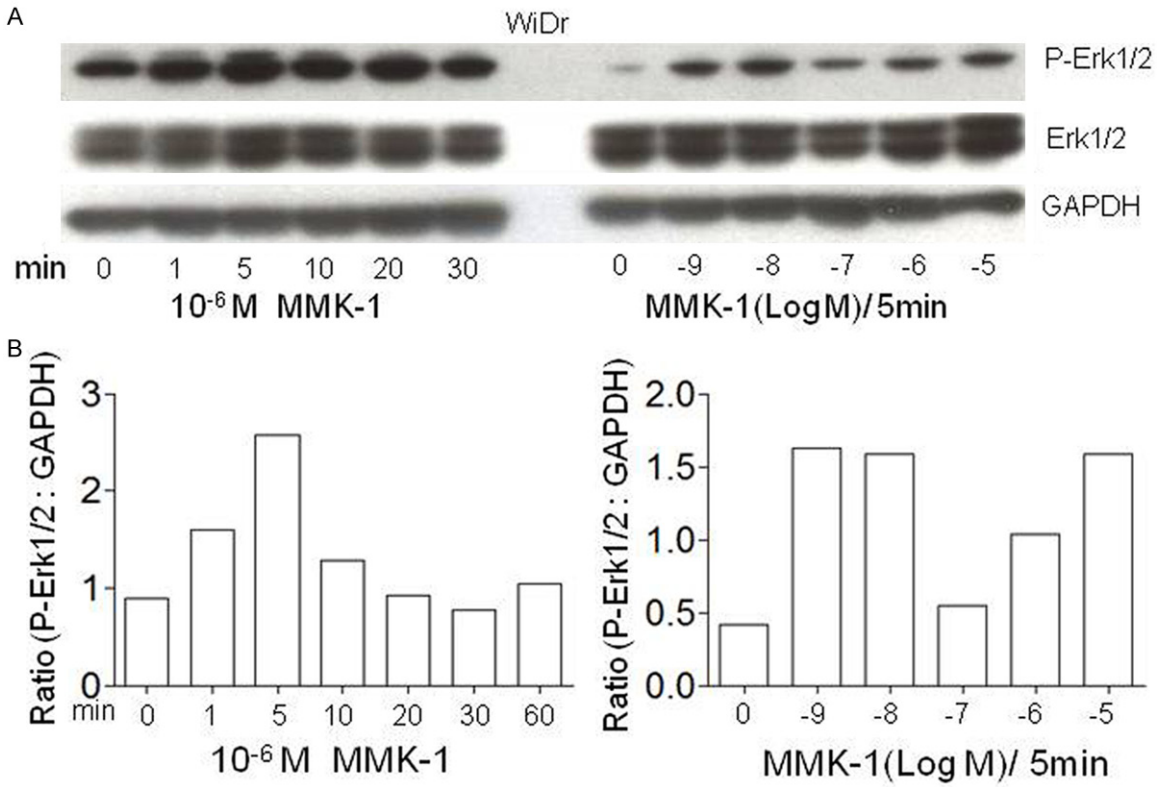


Supplementary Figure 1. FPR2 protein expression in human colon cancer cell lines. Human colon cancer cell lines grown in 60-mm dishes were harvested and lysed with 1 × SDS sample buffer. Proteins were electrophoresed and transferred onto ImmunoBlot Poly-vinylidene membranes, which were incubated with anti-human FPR2 antibodies followed by a HRP-conjugated secondary antibody. FPR2/HEK293 cells were used as positive control. COLO205, HCT-15, HCT-116, HCC-2998, HT-29, KM12, KM20L2, T84, SW620 and WiDr are human colon carcinoma cell lines.



Supplementary Figure 2. Human colon cancer cell migration in response to the FPR2 ligand W-peptide. Different concentrations of W-peptide were placed in the lower wells of the chemotaxis chamber; human colon cancer cells were placed in the upper wells, which were separated from lower wells by a 10-μm pore-size polycarbonate filter coated with collagen type I. After incubation, the cells that migrated across the filter were counted under light microscopy. The results are expressed as the mean ± SE of the chemotaxis index (CI). **P* < 0.05, significantly increased cell migration in response to FPR2 agonists compared to medium (0). A. HCT116, B. KM12, C. HCT15 and D. SW620.



Supplementary Figure 3. ERK1/2 phosphorylation in WiDr colon cancer cells induced by the FPR2 agonist MMK-1. A. ERK1/2 phosphorylation in WiDr cells induced by MMK-1. GAPDH protein was used as a loading control. B. Ratio of density for p-ERK1/2 versus GAPDH in WiDr cells after stimulation with MMK-1. Activation of ERK1/2 induced by MMK-1 at different time points (Left) with different ligand concentrations (Right).