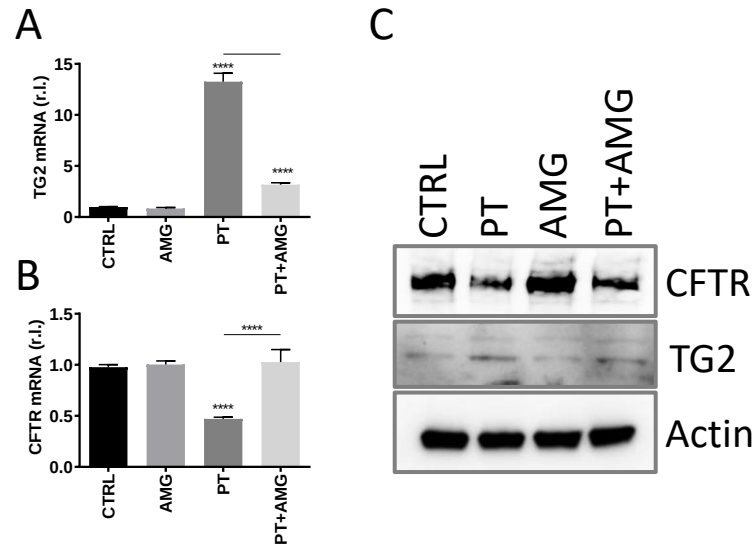
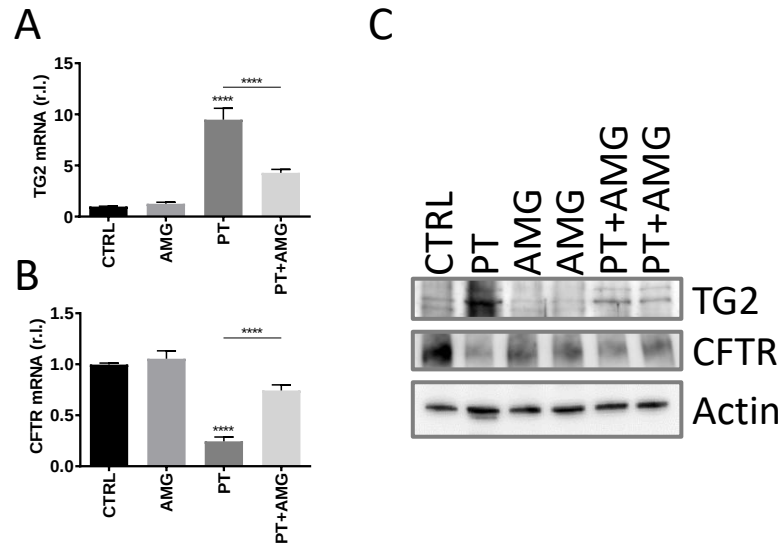




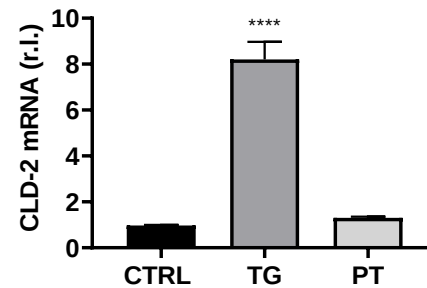
Supplementary S1. The *in vitro* PT-stimulated dysregulation of TG2 and CFTR is mediated by CXCR3. Caco-2 cells were stimulated 9h with PT (1 mg/ml) alone or in the presence of the CXCR3 inhibitor AMG 487 (AMG, 1 μ M; 8h pretreatment) and the expression of TG2 (A,C) or CFTR (B,C) was evaluated by qPCR or western blotting analysis. Data are representative of three independent experiments performed in triplicate. Histograms represent mean $\pm$ s.d.; ****p < 0.0001. Actin was used as loading control.



Supplementary S2. The *ex vivo* PT-stimulated dysregulation of TG2 and CFTR is mediated by CXCR3.

Small intestine of GF mice cultured in GEVS were stimulate 9h with PT (2.5 mg/ml) alone or in the presence of the CXCR3 inhibitor AMG 487 (AMG, 1 μ M; 8h pretreatment) and the expression of TG2 (A,C) or CFTR (B,C) was evaluated by qPCR or western blotting analysis.

Data are representative of three independent experiments performed in triplicate. Histograms represent mean $\pm$ s.d.; ****p < 0.0001. Actin was used as loading control.



Supplementary S3. CXCR3 is required to deregulate TJ proteins expression. HEK cells were stimulated 9h with PT (1 mg/ml) or TG (10 μ g/ml), and the expression of CLD-2 was evaluated by qPCR. Data are representative of three independent experiments performed in triplicate. Histograms represent mean $\pm$ s.d.; ****p < 0.0001.