Electronic Supplementary Material (ESI) for Food & Function.

Supplementary information

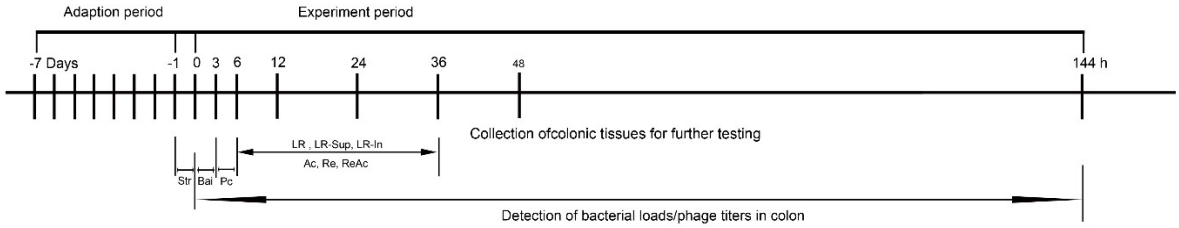

Fig. S1. Design of animal experiments. All the mice were kept normally for a week to adapt to their new surroundings. Streptomycin was orally administered to the mice to destroy their normal intestinal microbiota one day before *S. typhimurium* challenge (0.2 mL, 4×10^8 CFU/mouse). Mice in the Pc treatment group were orally administered a single dose of Pc (0.2 mL, 2×10^8 PFU/mouse) at 3 h after *S. typhimurium* infection. Mice in the PcLR, PcLR-Sup, PcLR-In, PcAc, PcRe and PcReAc groups were administered a single dose of Pc (0.2 mL, 2×10^8 PFU/mouse, at 3 h after *S. typhimurium* infection) by gavage, and then, they were gavaged with LR (0.2 mL, 4×10^6 CFU/mouse), 1:100 diluted LR-Sup (0.2 mL/mouse), 1:100 diluted inactivated LR pellets (0.2 mL, 4×10^6 CFU/mouse), Ac (300 mM), Re (1.5 mM) and ReAc (300 mM, 1.5 mM) at 6, 12, 24 and 36 h after *S. typhimurium* infection, respectively. For the controls, the mice were only gavaged with the same doses of LR, LR-Sup, LR-In, Ac, Re or ReAc at the same time points with no Pc treatment. Colonic tissues were collected from all treatment groups at different time intervals after *S. typhimurium* infection for further testing.

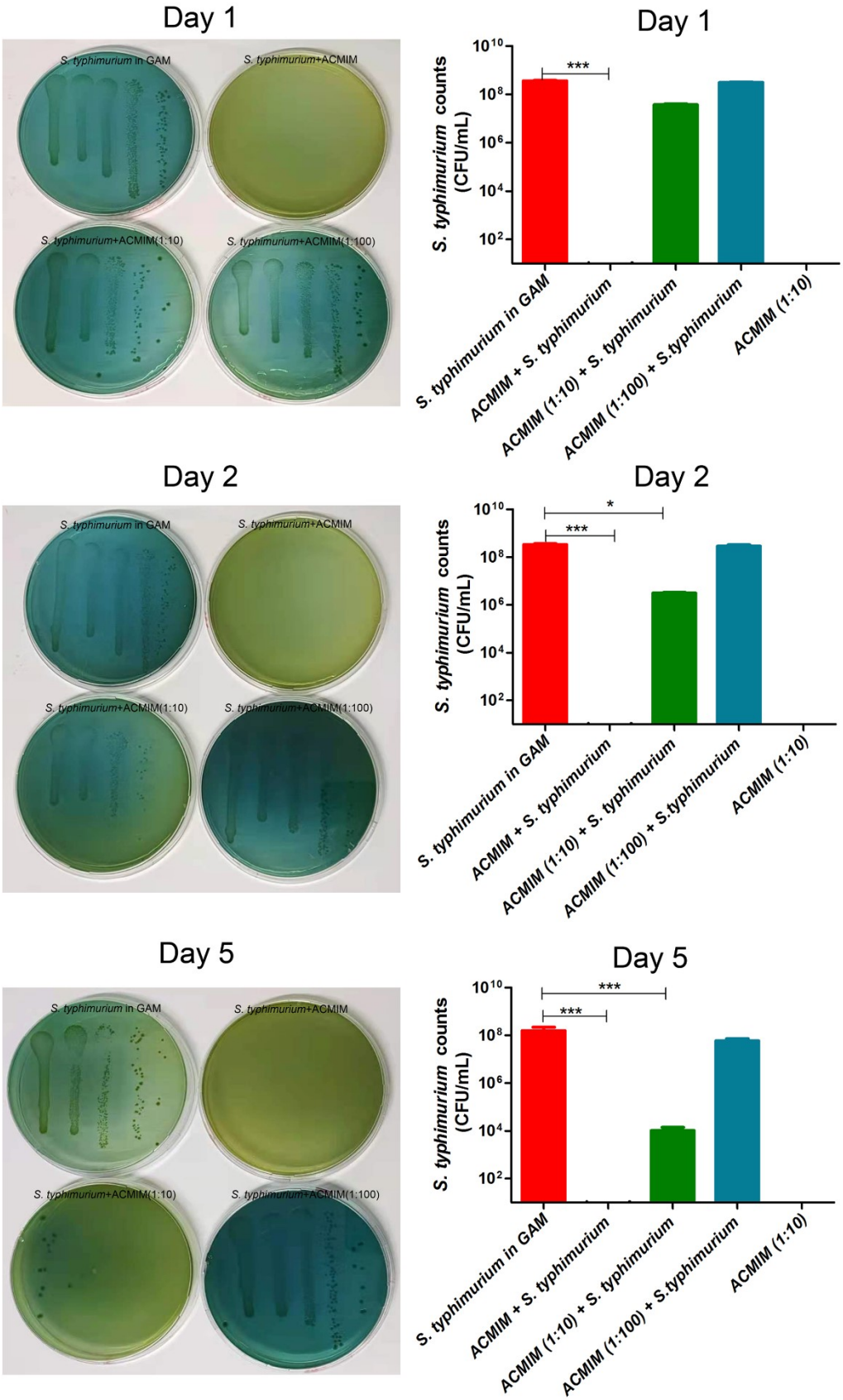

Fig. S2. The antibacterial effect of ACMIM *in vitro*. Cultures of ACMIM cocultured with *S.*

typhimurium, ACMIM (1:10) cocultured with *S. typhimurium* and ACMIM (1:100) cocultured with *S. typhimurium* were collected at the indicated days (1, 2, and 5). *Salmonella* in GAM without treatment and ACMIM (1:10) were used as the control. (Left) *Salmonella* colonies were measured by streaking 10μL of serially diluted culture onto WS agar plates. (Right) The *Salmonella* concentration was quantified on a log scale at 1, 2, and 5 days after coincubation. Data are expressed as the means ± SEM. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$. The experiment was repeated three times.

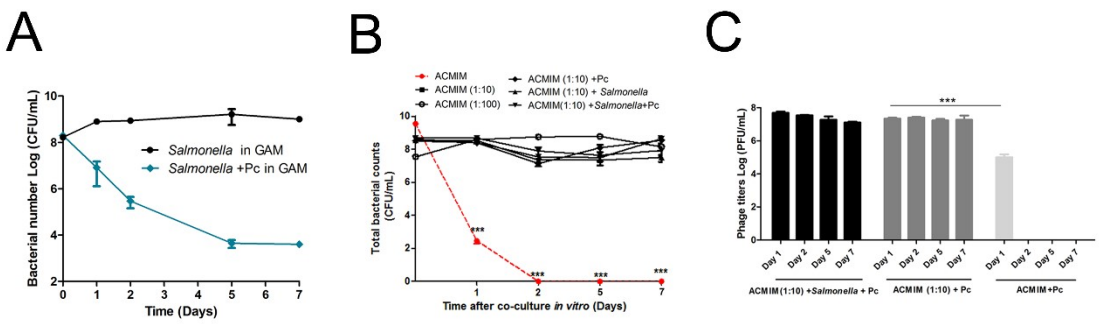

Fig. S3. Characterization of anaerobically cultured mouse intestinal microbiota (ACMIM). (A) The bactericidal effect of Pc on *Salmonella* in anaerobic condition. (B) The total bacterial counts of ACMIM at the indicated days (1, 2, 5, and 7) after coculture in GAM medium. (C) Phage titers in cultures were detected. Cultures were collected from ACMIM cocultured with Pc, ACMIM (1:10) cocultured with Pc and *S. typhimurium,* and ACMIM (1:10) cocultured with Pc at different time intervals (1, 2, 5, and 7 days after coculture). Data are expressed as the means ± SEM. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$. The experiment was repeated three times.

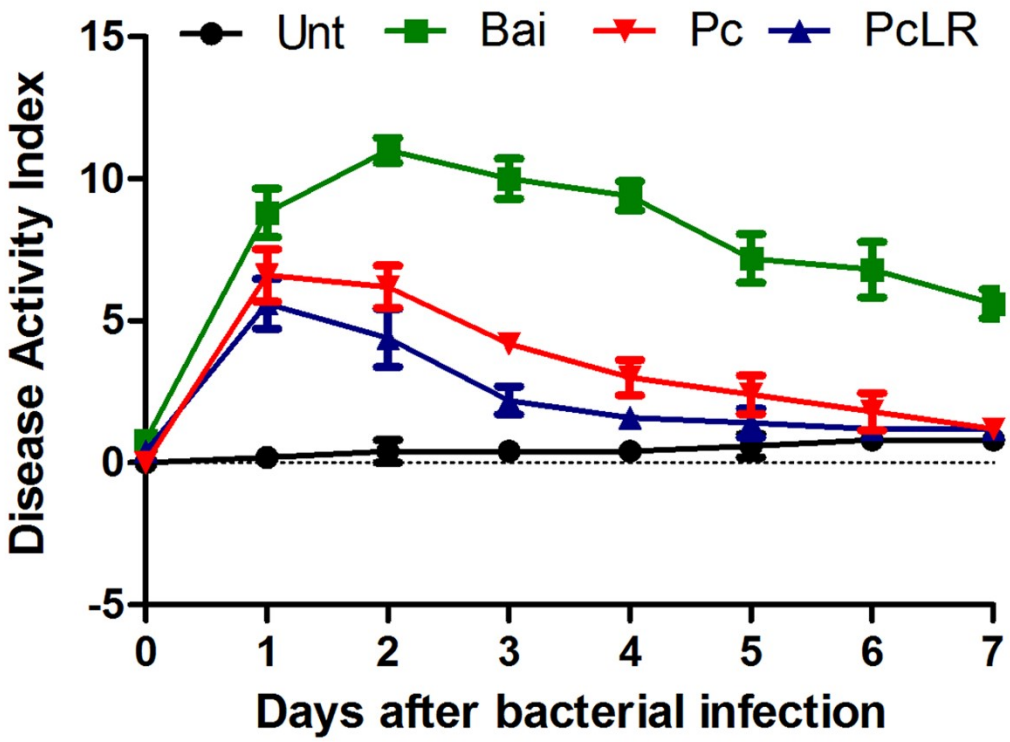

Fig. S4. Disease activity index (DAI). The DAI of different treatment groups was monitored for 7 consecutive days. i) Body weight loss (0, none; 1, 1–5%; 2, 5–10%; 3, 10–15%; and 4, > 15%). ii) Stool consistency (0, normal; 1, pasty and not adhering to the anus; 2, semi-formed adhered to the anus; 3, pasty and adhered to the anus; and 4, watery). iii) Rectal bleeding (0, no blood (–); 1, blood (±); 2, blood (+); 3, blood (++); and 4, obvious blood in stool).

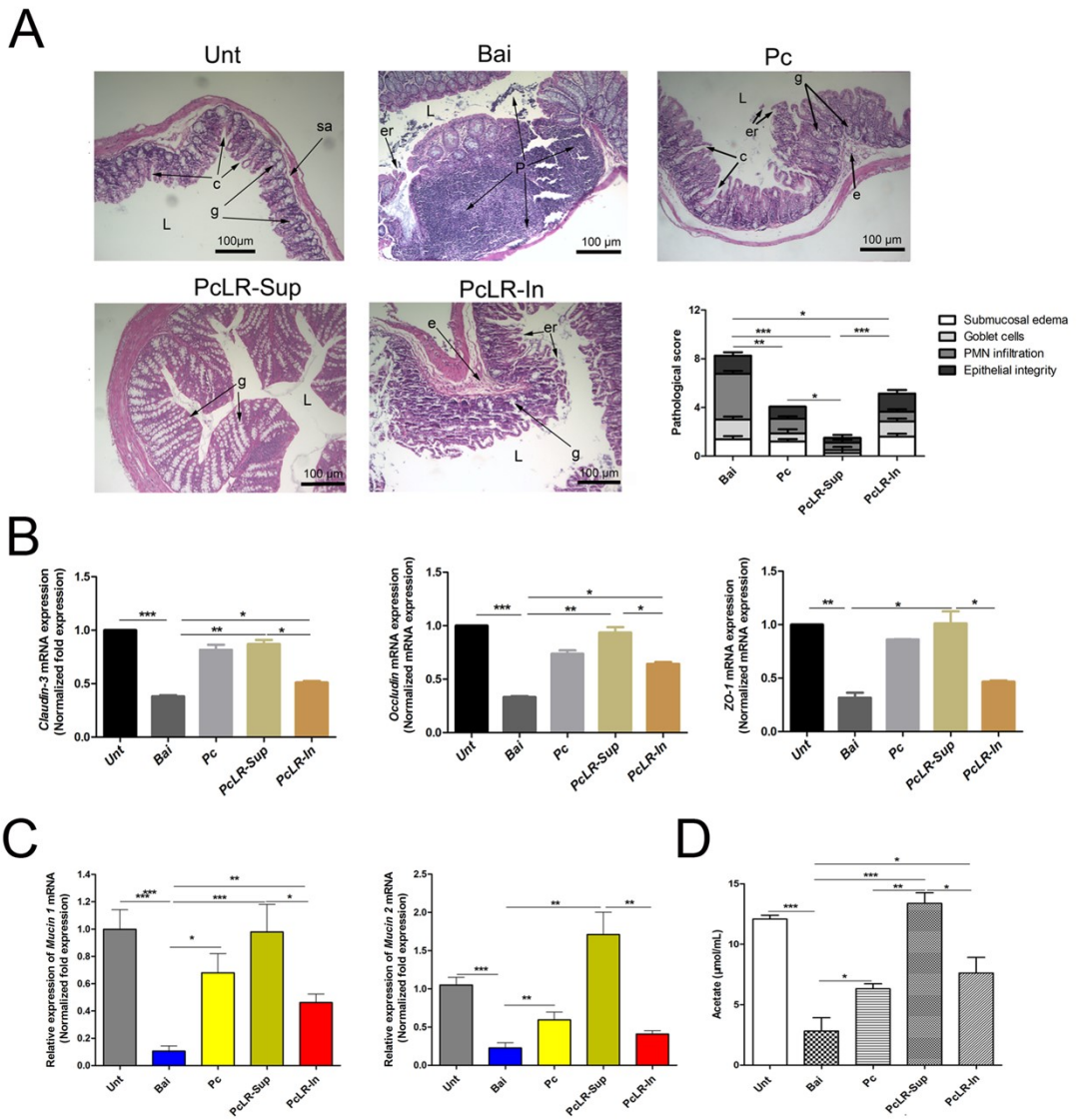

Fig. S5. Combination of Pc and LR-Sup effectively improved pathological damage and intestinal physical barrier on mouse colitis caused by *S. typhimurium*. Mice in the PcLR-Sup-treated and PcLR-In-treated groups were orally administered a single dose of Pc (0.2 mL, 2×10^8 PFU/mouse, at 3 h after 4×10^8 CFU/mouse of *S. typhimurium* infection) and then, the mice were gavaged with 1:100 diluted LR-Sup (0.2 mL/mouse) and 1:100 diluted inactivated LR pellets (0.2 mL, 4×10^6 CFU/mice) at 6, 12, 24 and 36 h after *S. typhimurium* infection, respectively. The colonic tissues were collected 48 h after infection. (A) H&E staining. The colonic tissue sections were stained with H&E (100×). L, intestinal lumen; e, edema; p, PMN; er, erosion of the epithelial

layer; c, crypt; g, goblet cell; and sa, submucosa. Magnifications are indicated by the black bars. (B) Levels of the tight junction-related genes (*Occludin*, *Claudin-3* and *ZO-1*) in colonic tissues. (C) Levels of epithelial mucus genes (*Mucin 1* and *Mucin 2*) in the colonic tissues. (D) SCFA (acetate). The fecal samples of different treatment groups were collected, and the SCFA (acetate) contents in these groups were detected. Data are the means ± SEM, n = 6. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ indicate significant differences.

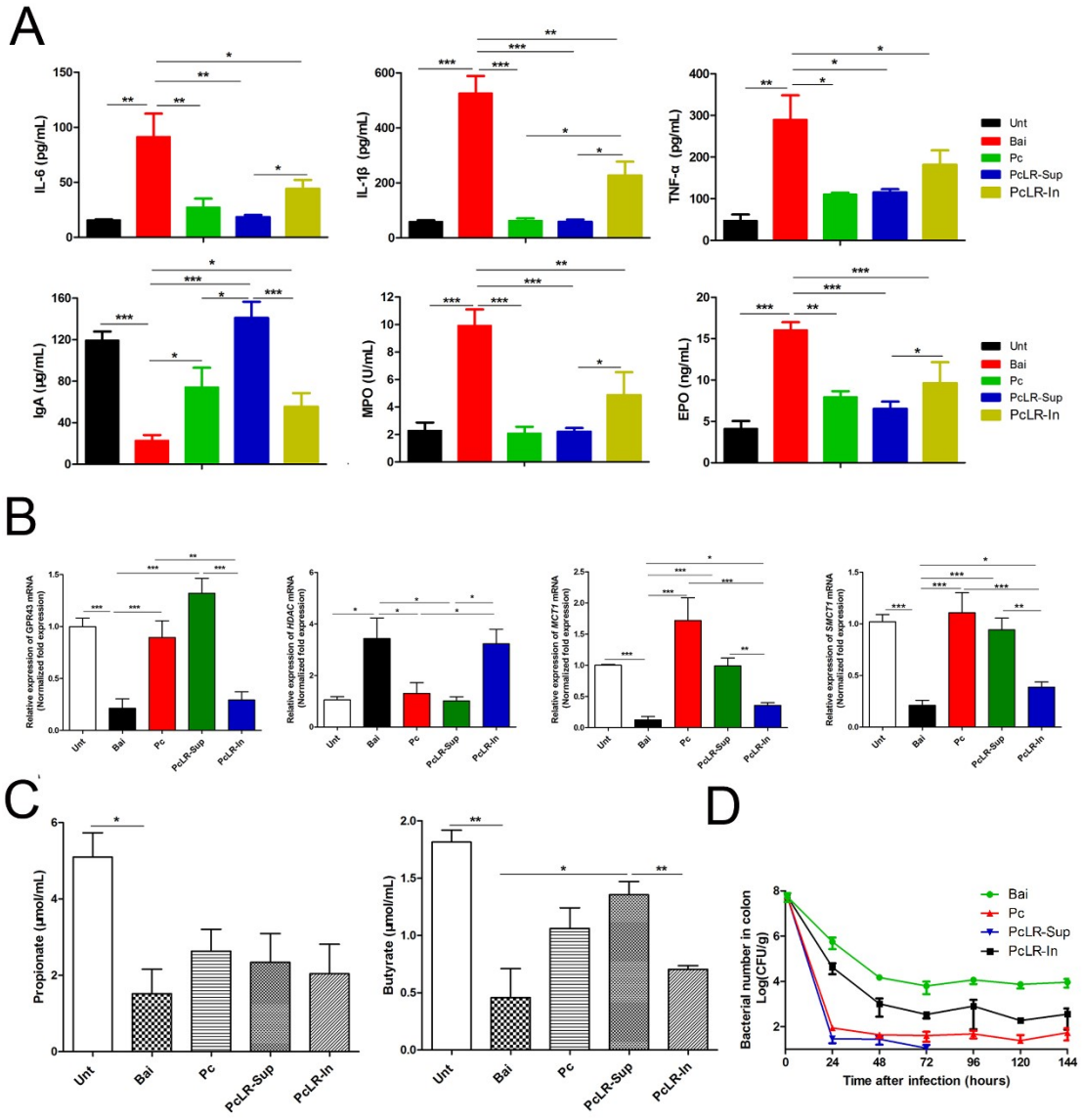

Fig. S6. Cytokine levels and SCFA metabolism after PcLR-Sup and PcLR-In treatment. Mice were orally administered 0.2 mL, 2 × 10^8 PFU/mouse single dose of Pc at 3 h after *S. typhimurium* (0.2 mL, 4 × 10^8 CFU/mouse) infection, and then, the mice were orally administered 0.2 mL/mouse of the 1:100 diluted LR-Sup and 0.2 mL of 4 × 10^6 CFU/mouse 1:100 diluted inactivated LR pellets (LR-In) at 6, 12, 24 and 36 h after *S. typhimurium* infection. Mice were sacrificed after 48 h of infection, and the colonic tissues were collected for testing. (A) The levels of inflammatory cytokines (IL-6, IL-1β and TNF-α) and MPO and EPO in the colonic tissues and blood of different treatment groups were measured at 48 h post-infection. (B) The relative copy number of SCFA

transporters *SMCT1* and *MCT1*. *β-actin* and *GAPDH* were used as reference genes. (C) SCFA contents (propionate, butyrate) in feces. (D) The bacterial loads in the colonic tissues. Data are the means ± SEM, n = 6. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ indicate significant differences.

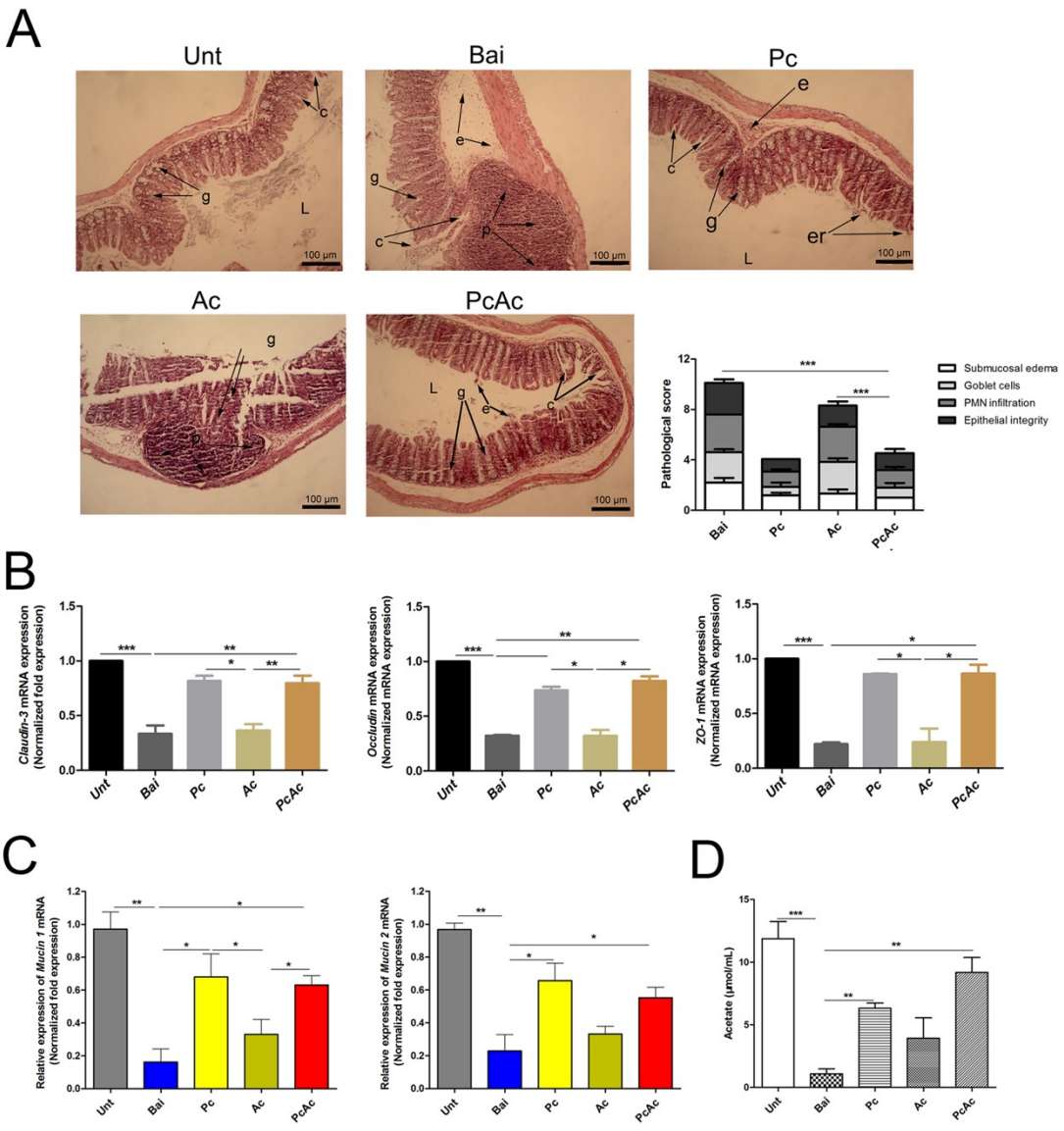

Fig. S7. The effect of PcAc on mouse colitis caused by *S. typhimurium* infection. Mice in the PcAc groups were orally gavaged with a single dose of Pc (0.2 mL, 2 × 10^8 PFU/mouse) at the initial 3 h after *S. typhimurium* (0.2 mL, 4 × 10^8 CFU/mouse) infection, and then, the mice were administered Ac (300 mM) at 6, 12, 24 and 36 h after *S. typhimurium* infection. Forty-eight hours later, the colonic tissues were collected and used for further testing. (A) H&E staining. The colonic tissue sections were stained with H&E (100×). L, intestinal lumen; e, edema; p, PMN; er, erosion of the epithelial layer; c, crypt; g, goblet cell; and sa, submucosa. Magnifications are indicated by the black bars. (B) Levels of the tight junction-related genes (*Occludin*, *Claudin-3* and *ZO-1*) in

colonic tissues. (C) Levels of the epithelial mucus genes (*Mucin 1* and *Mucin 2*) in colonic tissues. (D) The contents of SCFA (acetate) in fecal samples. Data are the means ± SEM, n = 6. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ indicate significant differences.

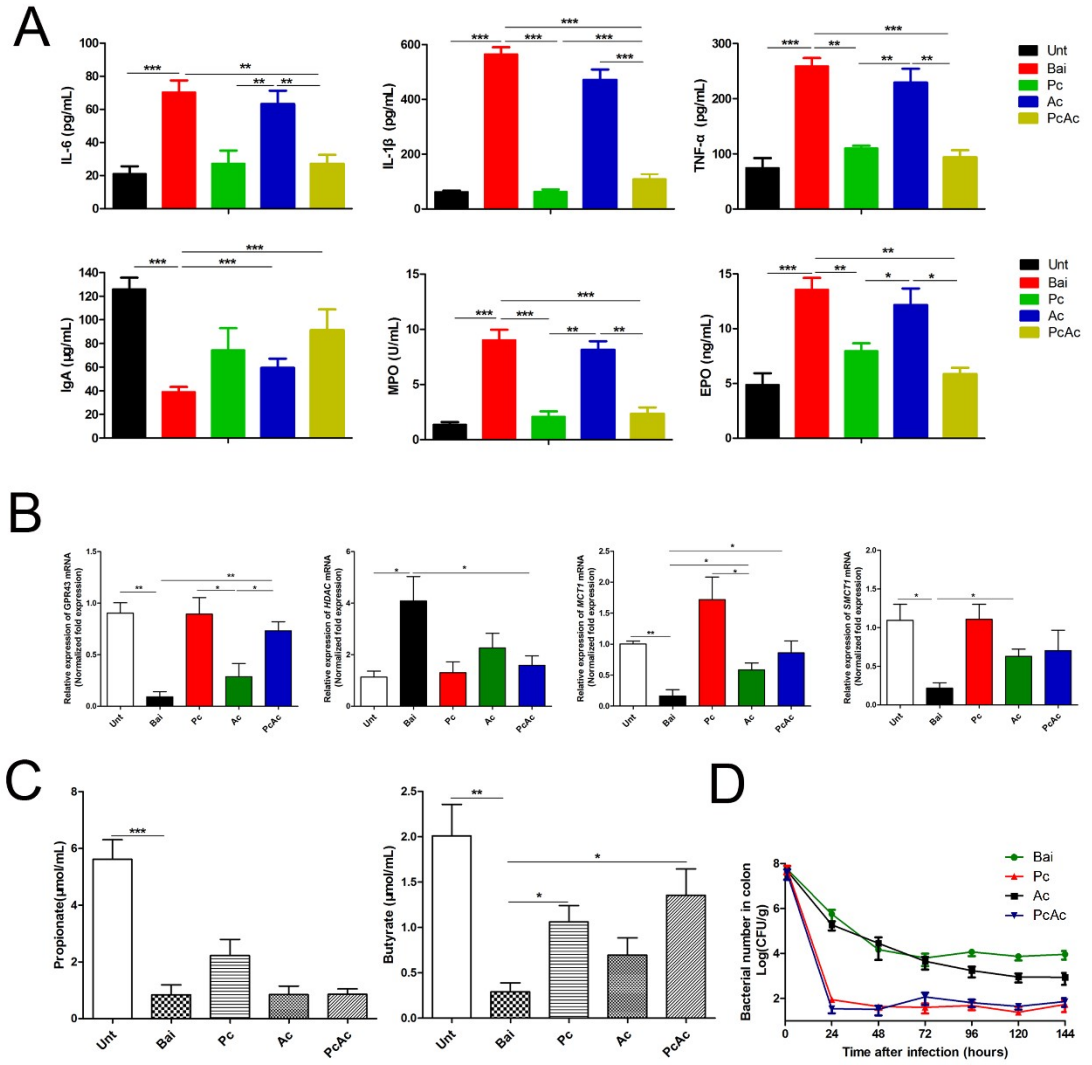

Fig. S8. Cytokine levels and SCFA metabolism after PcAc treatment. Mice were orally administered a single dose of Pc (0.2 mL, 2 × 108 PFU/mouse) at 3 h after 4 × 108 CFU/mouse *S. typhimurium* infection, and then, the mice were gavaged with Ac (300 mM) at 6, 12, 24 and 36 h after *S. typhimurium* infection. Forty-eight hours later, the colonic tissues were collected. (A) Cytokine levels (IL-6, IL-1β, and TNF-α) and levels of MPO and EPO in the colonic tissues and IgA in the blood of different treatment groups. (B) The transcription levels of SCFA transporters (*SMCT1* and *MCT1*) and receptors (*HDAC* and *GPR43*). β-actin and GAPDH were used as reference genes. (C) SCFA contents (propionate, butyrate) in feces were detected at 48 h post-infection. (D)

The bacterial loads in the colonic tissues were detected. Data are the means ± SEM, n = 6. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ indicate significant differences.

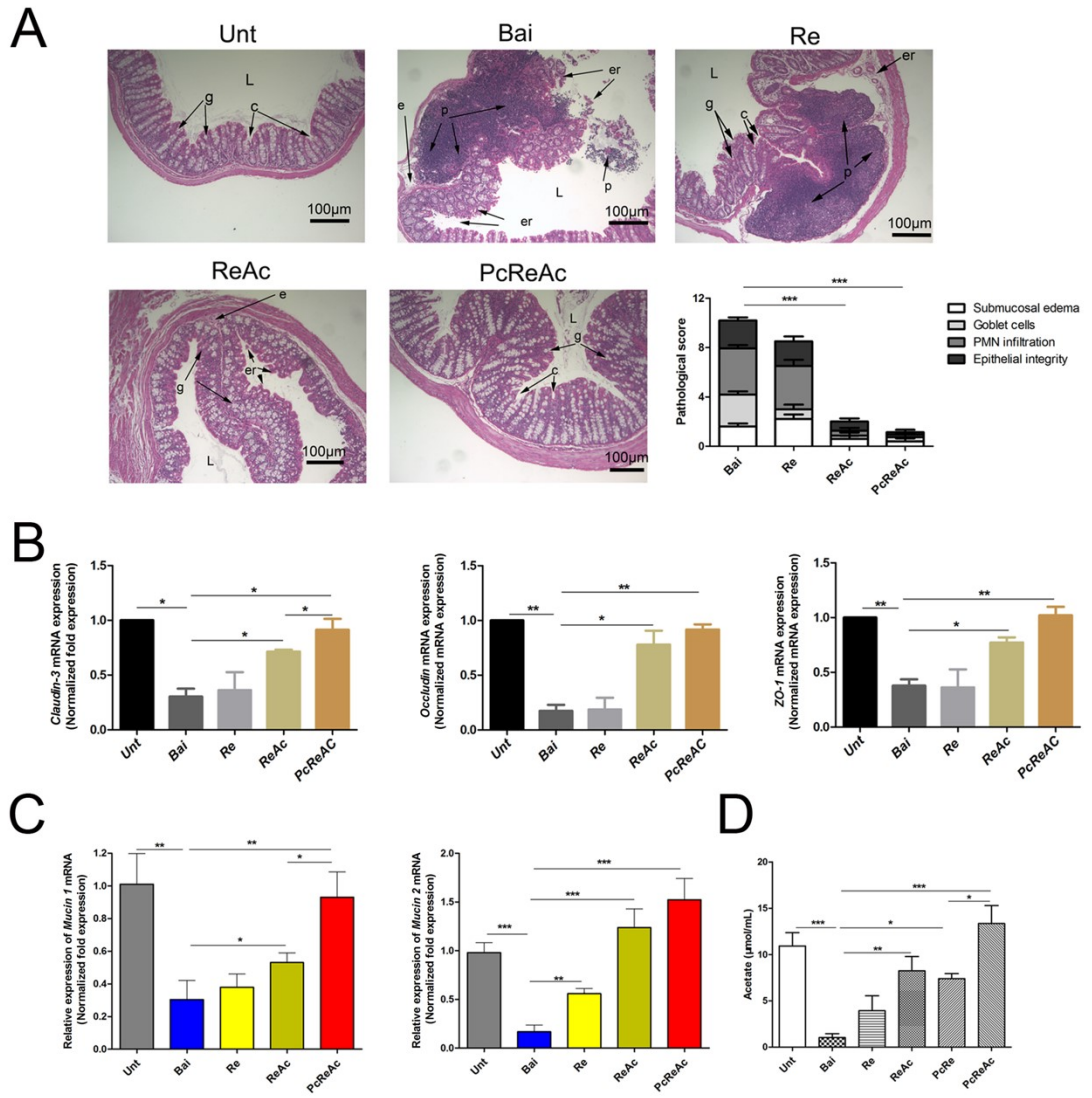

Fig. S9. Combination of Pc and ReAc (released by LR) effectively ameliorated mouse colitis caused by *S. typhimurium*. Mice were administered an oral gavage of a single dose of Pc (0.2 mL, 2×10^8 PFU/mouse, at 3 h after 4×10^8 CFU/mouse *S. typhimurium* infection) and then, the mice were gavaged with Re (1.5 mM) and ReAc (300 mM, 1.5 mM) at 6, 12, 24 and 36 h after *S. typhimurium* infection, respectively. Forty-eight hours later, their colonic tissues were collected and used for the following study. (A) H&E staining. The colonic tissue sections were stained with H&E (100×). L, intestinal lumen; e, edema; p, PMN; er, erosion of the epithelial layer; c, crypt; g, goblet cell; and sa, submucosa. Magnifications are indicated by the black bars. (B) The transcription level

of tight junction-related genes (*Occludin*, *Claudin-3*, and *ZO-1*). (C) The transcription level of epithelial mucus genes (*Mucin 1* and *Mucin 2*) in colonic tissues. (D) SCFA levels (acetate) in the feces. Data are the means ± SEM, n = 6. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ indicate significant differences.

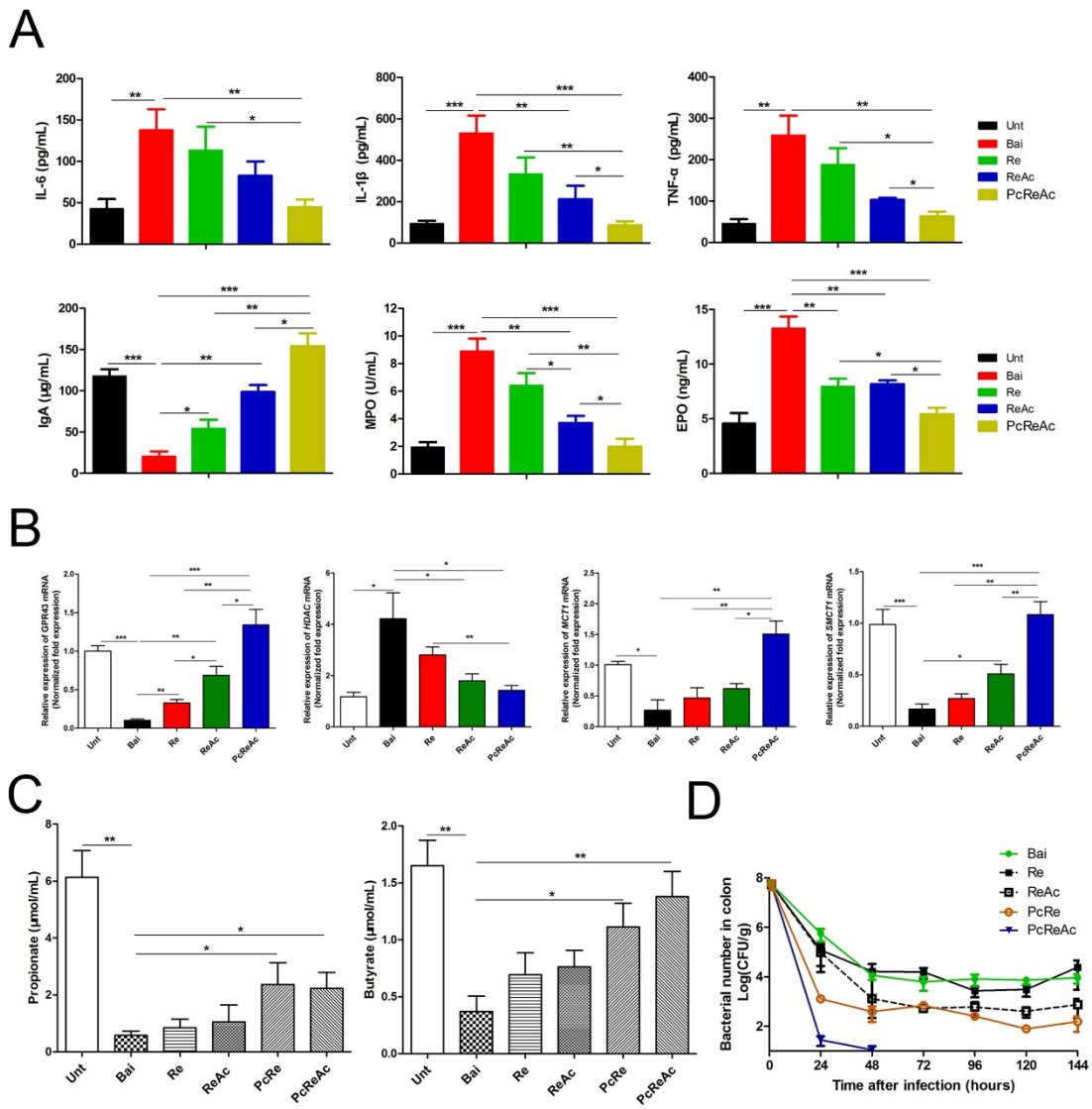

Fig. S10. Cytokine levels and SCFA metabolism after PcReAc treatment. Mice in the PcRe and PcReAc groups received a single dose of Pc (0.2 mL, 2 × 10^8 PFU/mouse, at 3 h after 4 × 10^8 CFU/mouse *S. typhimurium* infection) treatment, and then, the mice were orally administered Re (1.5 mM) and ReAc (300 mM Ac, 1.5 mM Re) at 6, 12, 24 and 36 h after *S. typhimurium* infection. The colonic tissues and faeces were collected 48 h after *S. typhimurium* infection and used for further analysis. (A) The levels of inflammatory cytokines (IL-6, IL-1β and TNF-α), MPO and EPO in the colonic tissues and IgA in blood were measured. (B) The gene expression of SCFA transporters *SMCT1* and *MCT1* and SCFA receptors *HDAC* and *GPR43* in the colonic tissues was

detected. The relative expression was calculated as the ratio of the target gene to the internal reference gene (*β-actin* and *GAPDH*). (C) SCFA contents (propionate, butyrate) in feces. (D) The bacterial loads in the colonic tissues. Data are the means ± SEM, n = 6. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ indicate significant differences.

Table S1. The primers used for the RT–PCR analysis in this study.

Targeting gene	Primer sequence (5' to 3')
β-actin	Forward: CAGCTGAGAGGGAAATCGTG
	Reverse: CTCCAGGGAGGAAGAGGATG
GAPDH	Forward: CCATGGAGAAGGCTGGGG
	Reverse: CAAAGTTGTCATGGATGACC
SMCT1	Forward: TGCCATTTCCTTATGGGTAGG
	Reverse: AGTGGAGTCCTTTCCGCATTA
MCT1	Forward: GTGCAGCA-GCCAAGGAGCCC
	Reverse: CCATGGCCAGTCCGTTGGCC
GPR43	Forward: CTGTATGGAGTGATGATCGCTGCTCTG
	Reverse: CTGCTCTTGGGTGAAGTTCTCGTAG
HDAC	Forward: CACACAGCAGACTTTCTACCAGGAC
	Reverse: GACATTGAAGCCCTCGCCACTG
Mucin 1	Forward: CCTTCAGTGCCAAGTCAATAC
	Reverse: TCCCCAGAAAATCTCCGTT
Mucin 2	Forward: ATGCCCACCTCCTCAAAGAC
	Reverse: GTAGTTTCCGTTGGAACAGTGAA