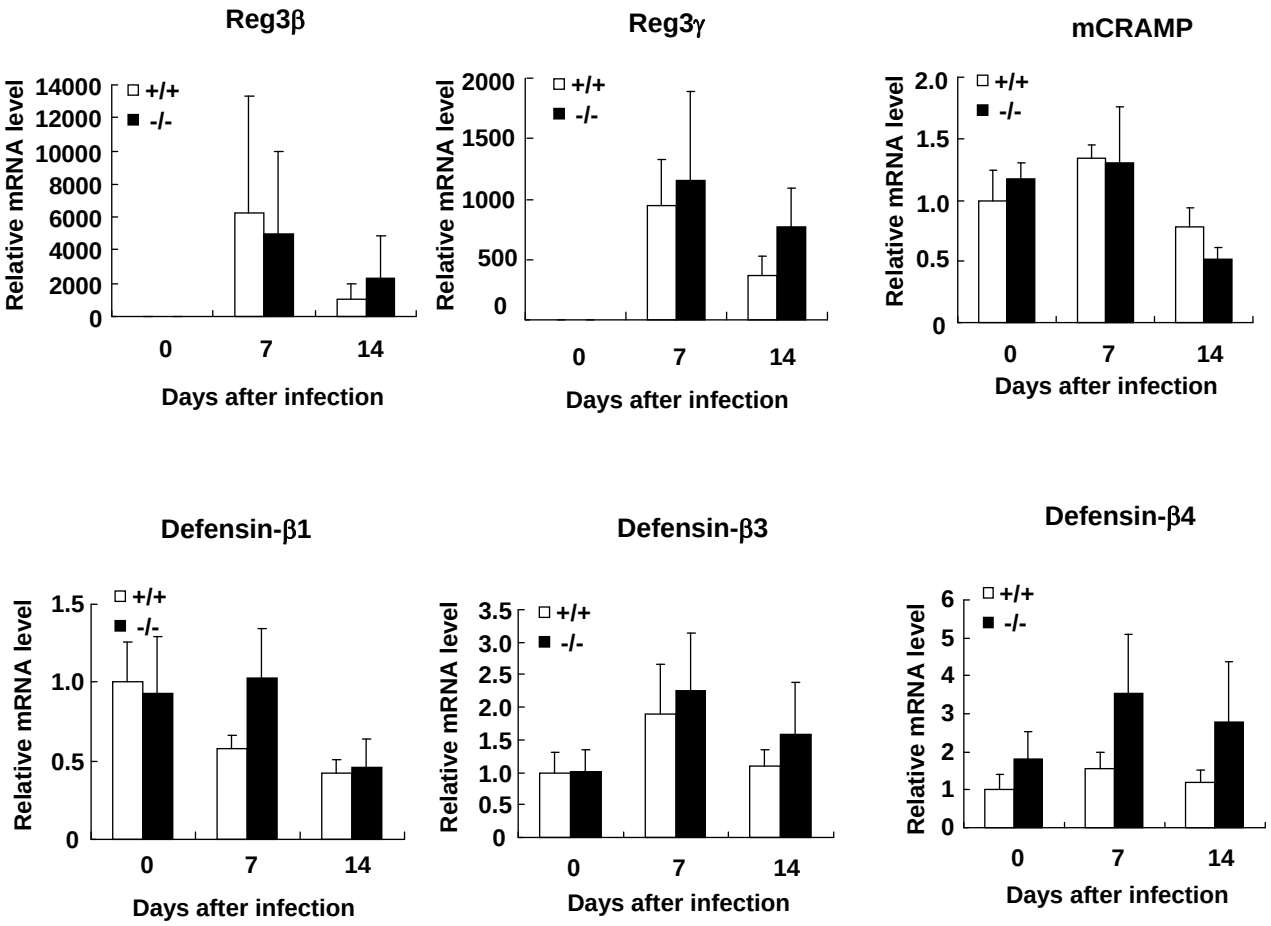

Figure S1. SRC-3 deficiency dose not impair the expression of antimicrobial genes during *C. rodentium* infection. Quantitative RT-PCR results of Reg3β, Reg3γ, mCRAMP, defensin-β1, defensin- β 3 and defensin- β 4 relative to day 0 in the distal colons of wild-type and SRC-3$^{-/-}$ mice on day 7 and day 14 after infection. Data are the means + SEM of five to ten mice per group. Results are representative of two independent experiments.

Figure S1

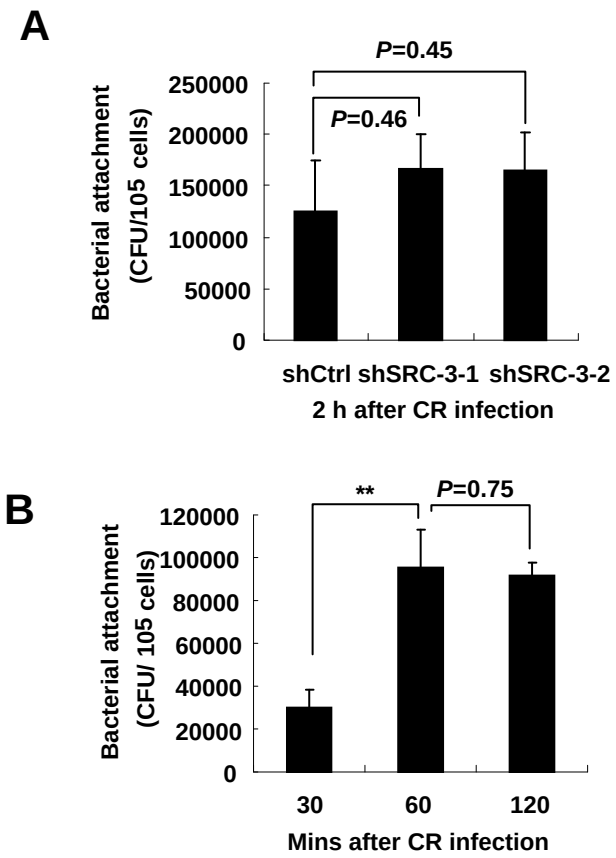

Figure S2. Adherence of *C. rodentium* to CMT93 cells A**,** shCtrl and shSRC-3 CMT-93 cells grown as adherent monolayers in 12-well plates, were infected with *C. rodentium* at MOI 200 for 2 hours. Bacterial adherence was determined by CFU assay. B,CMT-93 cells grown as adherent monolayers in 12-well plates were infected with *C. rodentium* at MOI 200 for 30 mins, 60 mins and 120 mins, respectively. Bacterial adherence was determined by CFU assay.

Figure S2

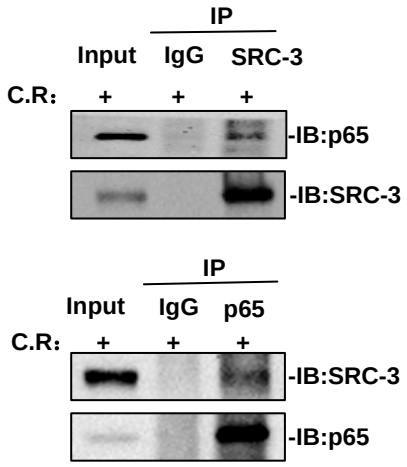

Figure S3. Co-IP analysis of the interaction of endogenous SRC-3 and p65 in CMT93 cells infected with *C. rodentium* for 2 hours. Anti-SRC-3 antibodies could immunoprecipitate p65 from cell lysates at 2 hours after *C.rodentium* infection. Reciprocally, anti-p65 antibodies could also immunoprecipitate SRC-3 from cell lysates at 2 hours after *C.rodentium* infection.

Figure S3

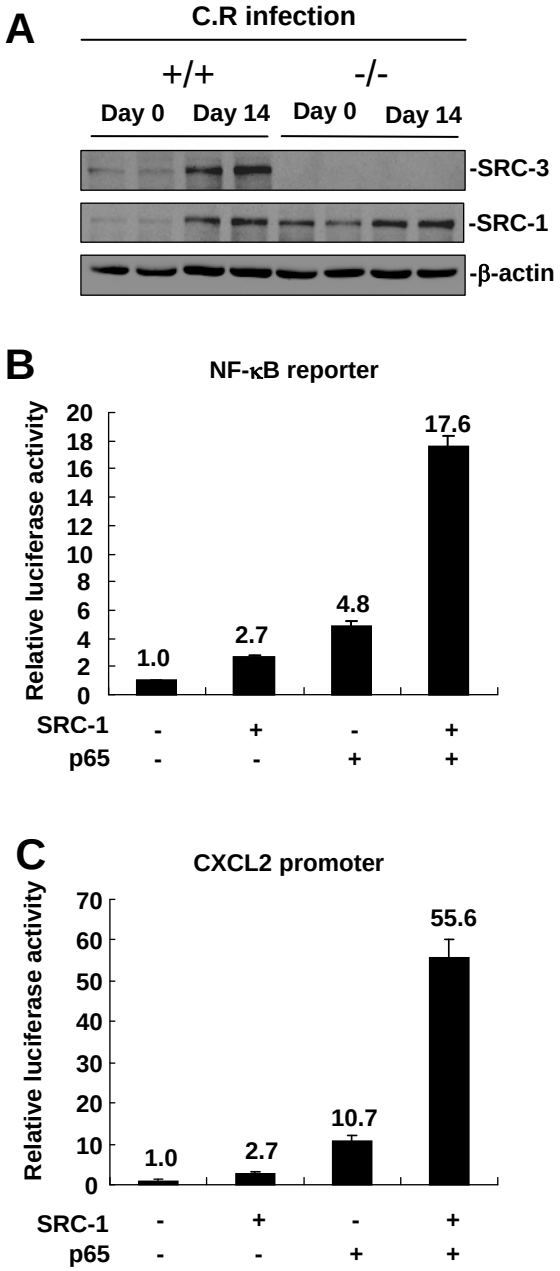

Figure S4. SRC-1 is upregulated in colonic epithelial cells in response to *C. rodentium* infection and enhances NF-κB signaling. *A,* SRC-1 was upregulated in colon in response to *C. rodentium* infection. *B,* SRC-1 cooperated with p65 to enhance the activity of NF-κB reporter. *C,* SRC-1 cooperated with p65 to enhance the activity of CXCL2 promoter reporter.

Figure S4