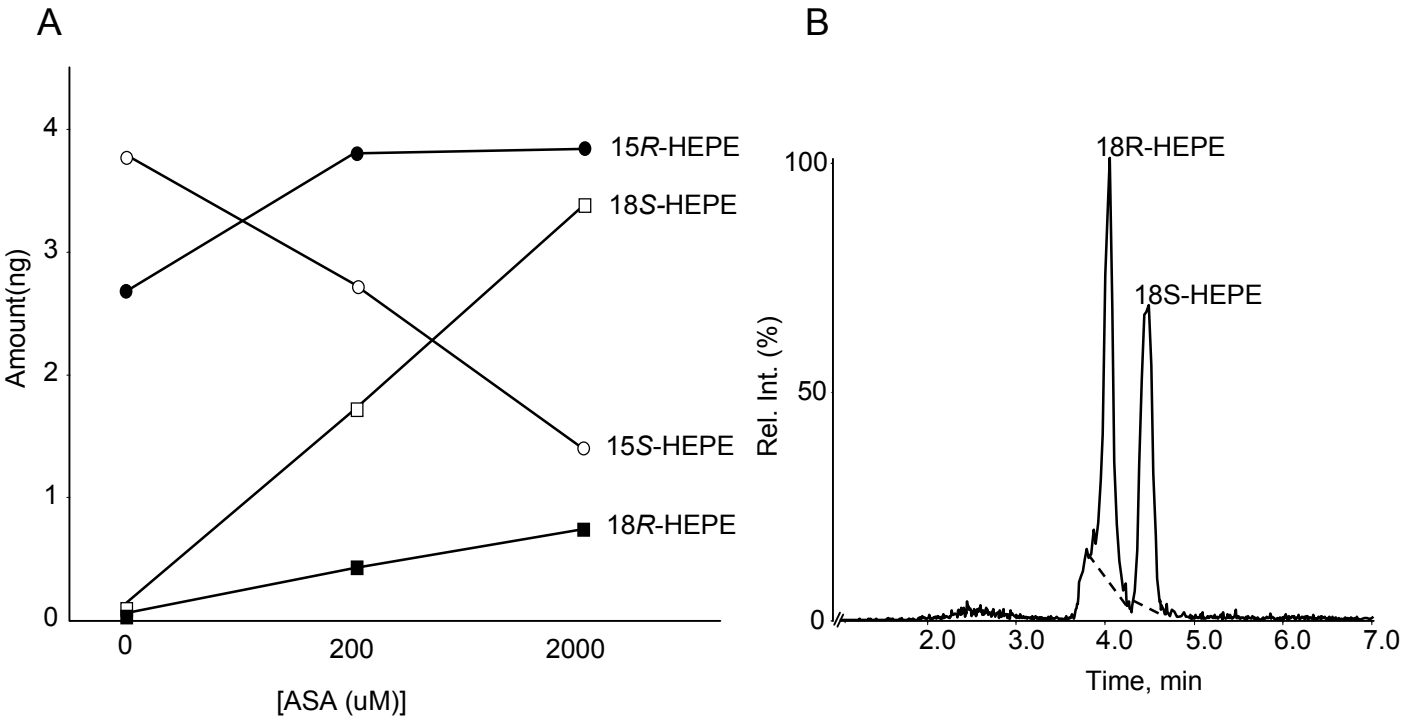

Figure S1.

Aspirin treatment increases preferential generation of 18S-HEPE. A) Human recombinant COX-2 generates both 18S-HEPE and 15R-HEPE. Human recombinant COX-2 was treated with different concentrations of aspirin and incubated for 30 min; then 15μM EPA was added and further incubated for 30 min. 15S-HEPE (open circle), 15R-HEPE (closed circle), 18S-HEPE (open square) and 18R-HEPE (closed square) were individually quantified by chiral LC-UV-MS-MS. Results are representative of three independent experiments. B) 18S-HEPE production is increased with aspirin treatment in a murine air pouch model. TNF-α (100ng) was injected into 6-day air pouch raised in mouse dorsal skin, followed by 500μg aspirin (3.5 hours post-TNF-α) and 300μg EPA (4 hours post TNF-α). Mice were sacrificed at 6 hours after initial TNF-α injection and 18R/S-HEPE were identified with chiral lipidomic analysis. Aspirin plus EPA treatment increased both 18R/S-HEPE levels when compared to EPA-only treatment (2.4ng to 14ng, ~6-fold increase) and changed the 18R/S ratio to more S-favorable (1.5:1 to 1:1).

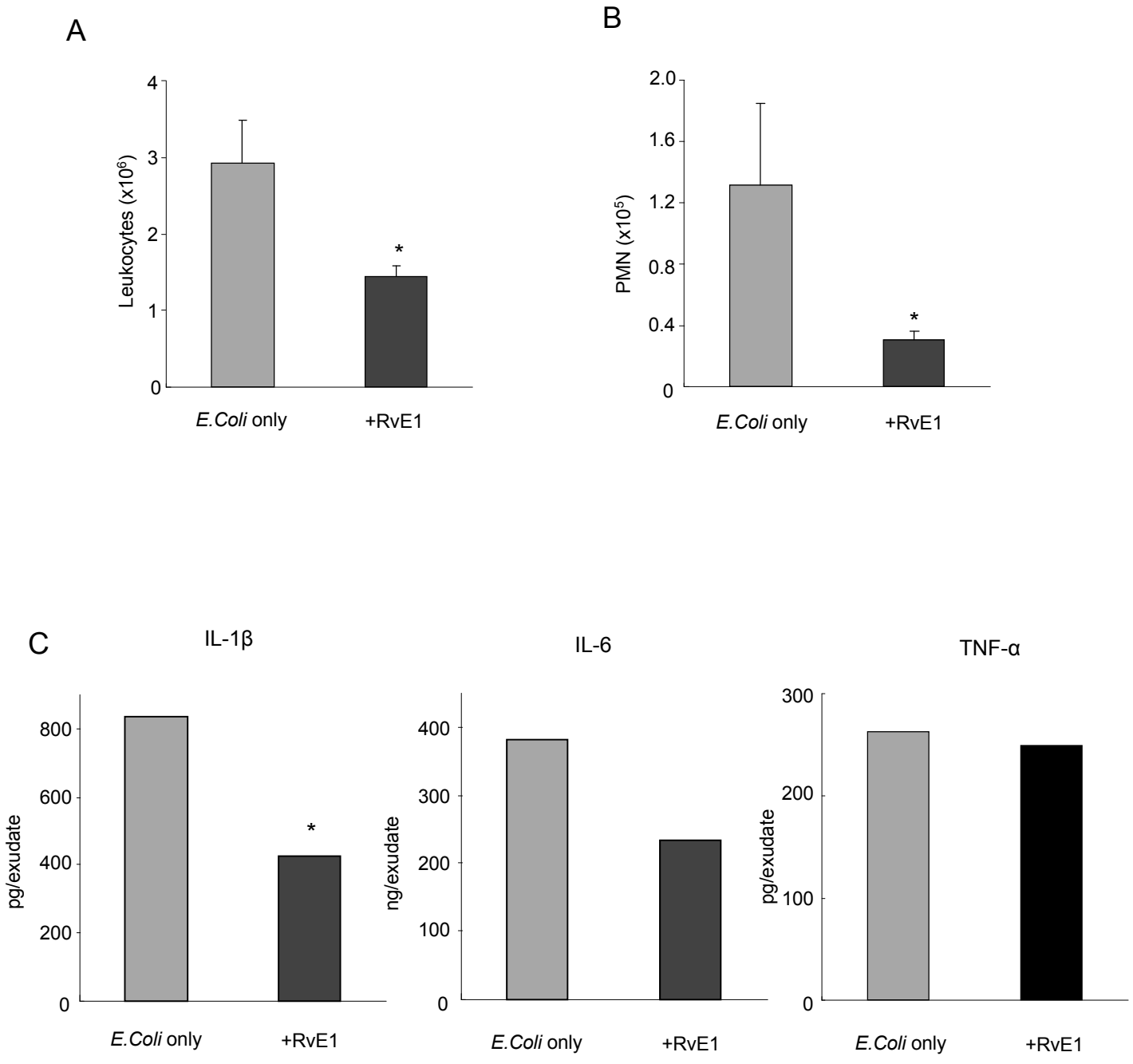

Figure S2.

RvE1 actions in E. coli peritonitis. 100ng RvE1 was administered i.v. immediately before E. coli peritoneal injection (107 CFU). Four hours later, exudates were obtained by lavage, A) total leukocyte and B) PMN numbers were enumerated, C) cytokine levels. *; $p<0.05$ compared to vehicle-treated animals.