Supporting Information

Morris et al. 10.1073/pnas.1000373107

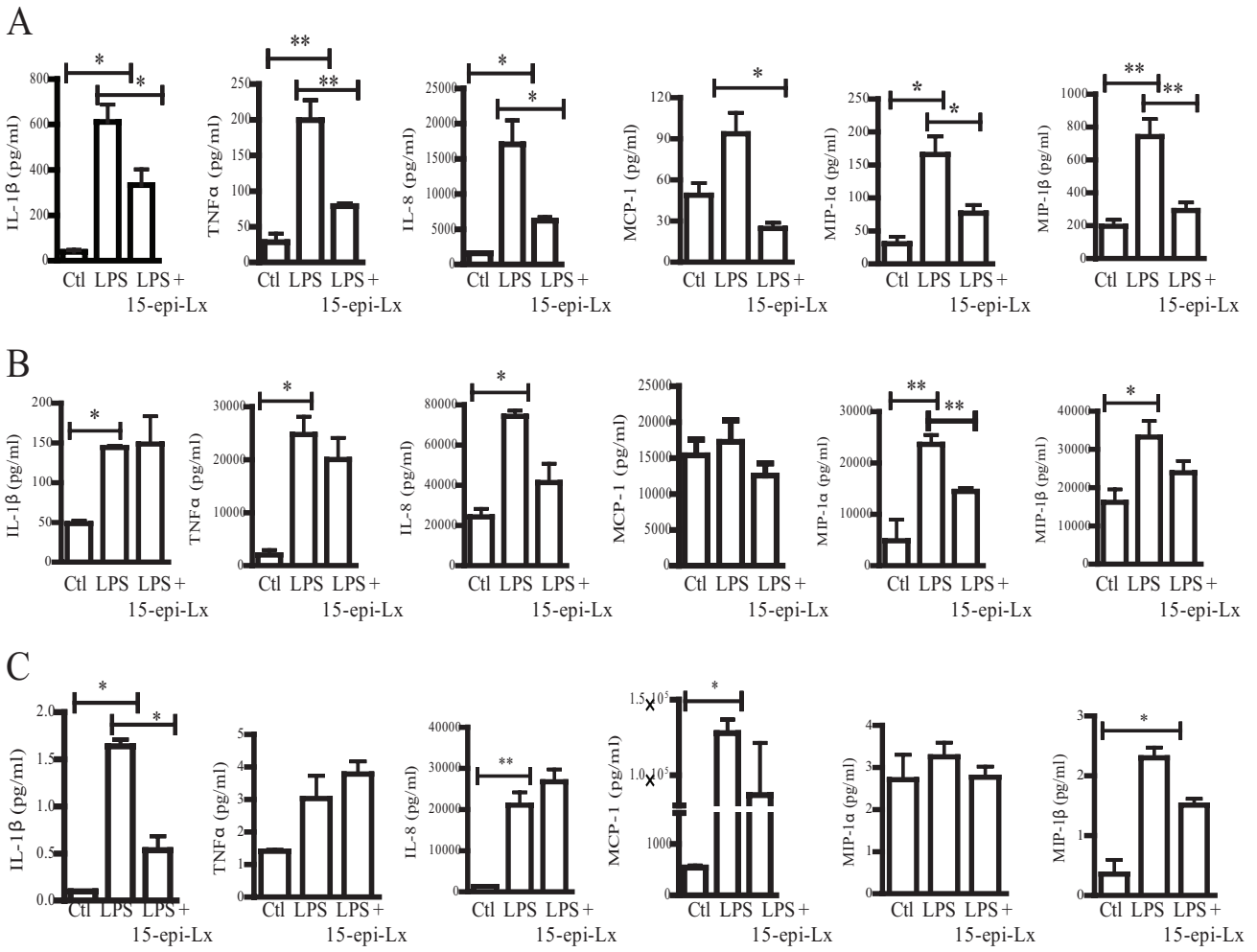

Fig. S1. Inhibitory effect of 15-epi-LxA$_4$ on inflammatory cytokine/chemokine synthesis from LPS-stimulated human monocytes (*A*), monocyte-derived macrophages (*B*), and HUVECs (*C*). Data are presented as mean ± SEM; $*P < 0.05$ and $**P < 0.01$.

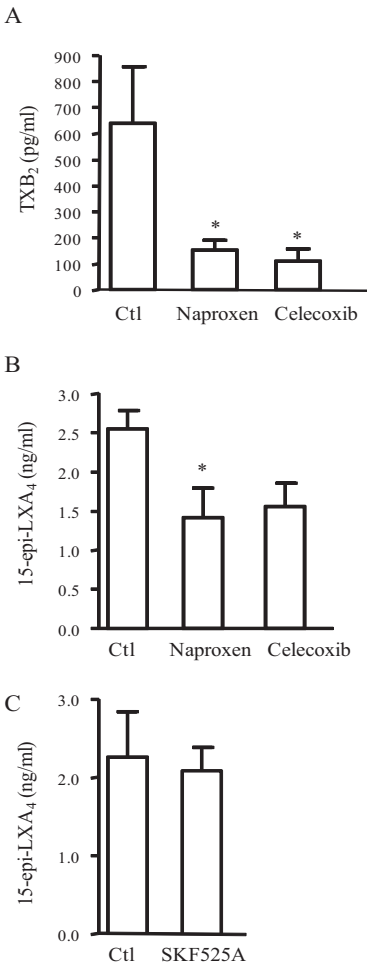

Fig. S2. Plasma levels of 15-epi-LxA$_4$ are inhibited by COX inhibition in naive animals. Naproxen and celecoxib were dosed at 10 mg/kg for 3 d, followed by plasma measurements of TxB$_2$ (*A*), the active metabolite of TxA$_2$; and 15-epi-LxA$_4$ (*B*). (*C*) No effect on plasma 15-epi-LxA4 was found with the cytochrome P450 inhibitor SKF525A (twice daily for 3 d at 30 mg/kg). Data were analyzed by ANOVA followed by Bonferroni *t* test and presented as mean ± SEM; *$P < 0.05$.

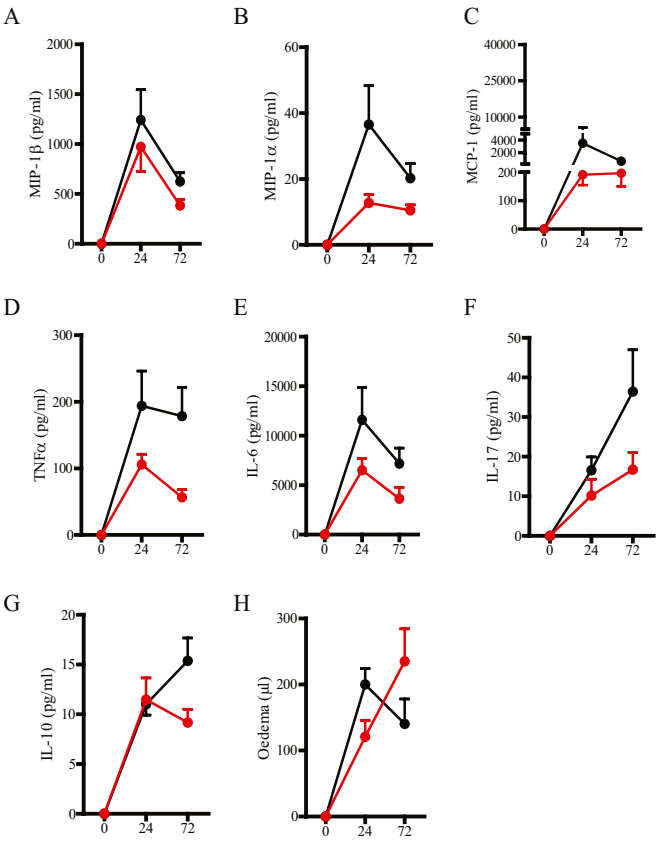

Fig. S3. Whereas ASA^{low} (solid red line) inhibited IL-1β and IL-8 in the blister fluid of volunteers possessing an early resolving phenotype (Fig. 4 *E* and *F* in the main text), it had a trend toward but no significant reduction in levels of a range of inflammatory cytokines (*A*–*G*) and blister edema (*H*). Black solid line represents controls.

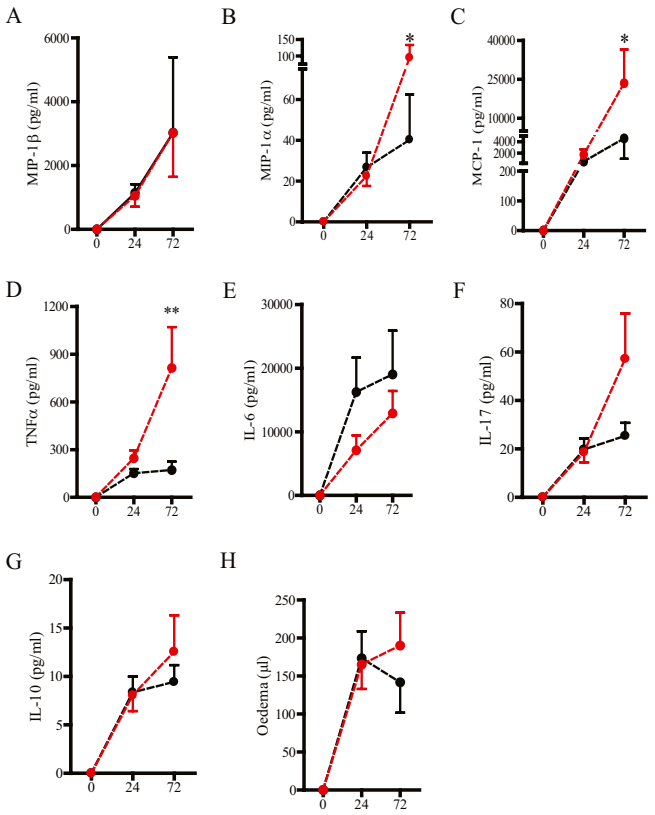

Fig. S4. In addition to having no effect on blister IL-1β and IL-8 (Fig. 5 *A–F* in the main text), ASA^{low} (dotted red line) was also without effect on a range of inflammatory cytokines (*A–G*) and edema (*H*) in volunteers with a delayed resolving phenotype. Black solid line represents controls.