Fig. S1. Inhibitory effect of 15-epi-LxA4 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-LxA₄ 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 TxB₂ (A), the active metabolite of TxA₂; and 15-epi-LxA₄ (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<sub>low</sub> (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.
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.