Supporting Information

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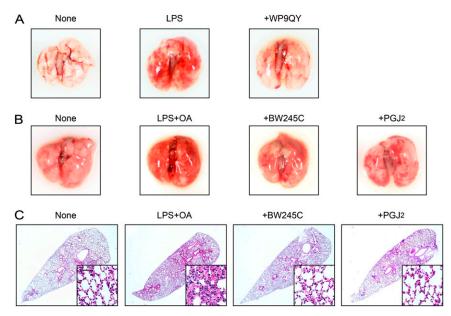


Fig. S1. The TNF- α -inhibiting peptide WP9QY (10 mg/kg every 3 h) was administered intranasally to LPS-challenged mice. The DP receptor agonist BW245C or a degraded product of prostaglandin D₂ (PGD₂) 15d-PGJ₂ was administered intranasally to LPS + oleic acid (OA)-challenged mice at 100 μ g/kg 10 min before the LPS challenge. Representative pictures of dissected lung (A and B) and H&E staining (C) are shown (Scale bar in C: 100 μ m.)

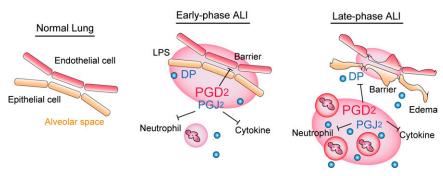


Fig. S2. PGD₂ signaling between alveolar endothelial cells/epithelial cells and neutrophils provides anti-inflammatory effects in acute lung injury.

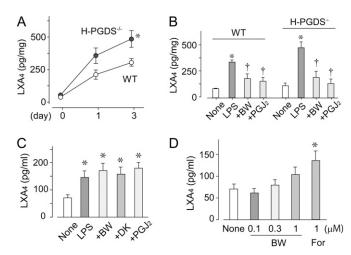


Fig. S3. Lipoxin A₄ (LXA₄) generation in inflamed mouse lungs was examined by enzyme immunoassay (Neogen). (A) LXA₄ production was greater in hematopoietic PGD synthase-deficient (H-PGDS^{-/-}) mouse lung than in inflamed WT lung at 1 d after LPS inhalation (n = 5 each). (B) Administration of the DP agonist BW245C (100 μg/kg intranasally) strongly decreased LXA₄ synthesis in inflamed WT mouse lung on day 1 (n = 5 each). (C and D) PGD₂-mediated signaling did not influence LXA₄ generation in mouse neutrophils. Treatment with LPS (50 ng/mL for 12 h) stimulated LXA₄ production from neutrophils. (C) Concomitant treatment with the DP agonist BW245C (0.3 μM), CRTH2 agonist DK-PGD₂ (1 μM), or 15d-PGJ₂ (0.3 μM) did not influence the production of LPS-induced LXA₄ (n = 4 each). (D) Treatment with BW245C (0.1–1 μM for 12 h) had no effect on LXA₄ production, whereas treatment with forskolin (1 μM for 12 h) stimulated LXA₄ production (n = 4 each).