Figure S1

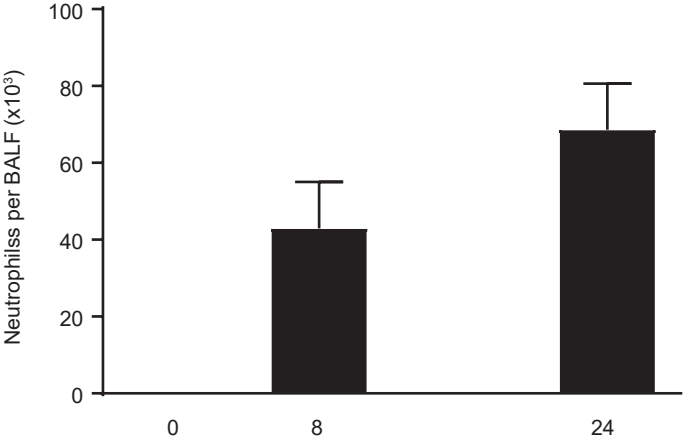

Figure S1. BAL neutrophils after ALI. BAL fluid neutrophils were enumerated by flow cytometry. Results are expressed as mean +/- s.e.m. N = 4 per group

FIGURE S2

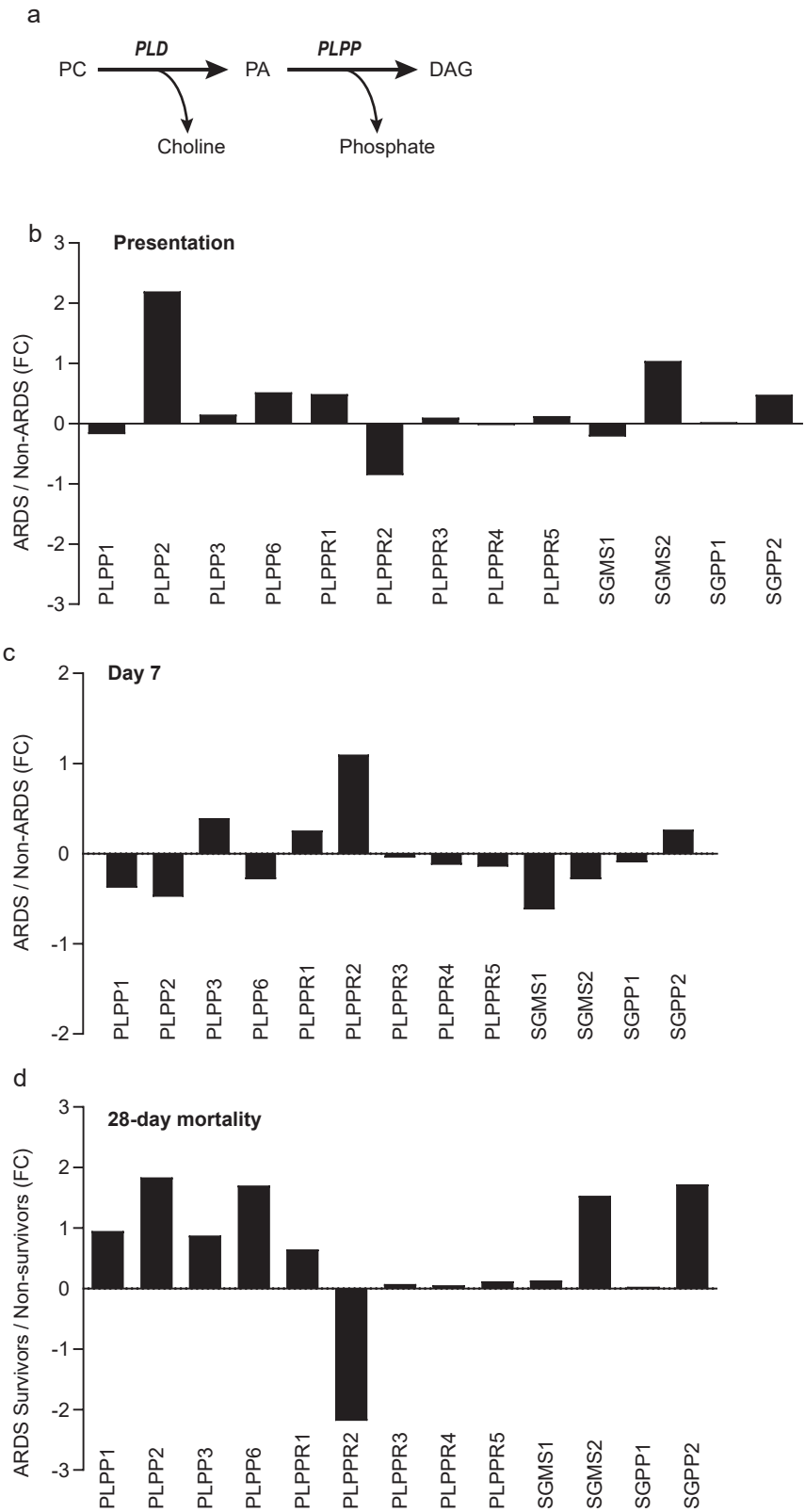

Figure S2. Phospholipid phosphatase genes are differentially regulated during ARDS. (a) Schematic representation of the phopholipase D (PLD) signaling pathway. PLD1 and PLD2 hydrolyze membrane phosphatidylcholine (PC) to phosphatidic acid (PA). The PA thus formed can be degraded by phospholipid phosphatases (PLPP1, PLPP2, or PLPP3) to produce diacylglycerol (DAG) and inorganic phosphate. Phospholipid phosphatase and phospholipid phosphatase-related gene expression fold change in ARDS / Non-ARDS (b) at presentation to the ICU, and (c) at day 7. (d) Phospholipid phosphatase and phospholipid phosphatase-related gene expression fold change at presentation in ARDS survivors / Non-survivors. All data is expressed in log2.

Figure S3

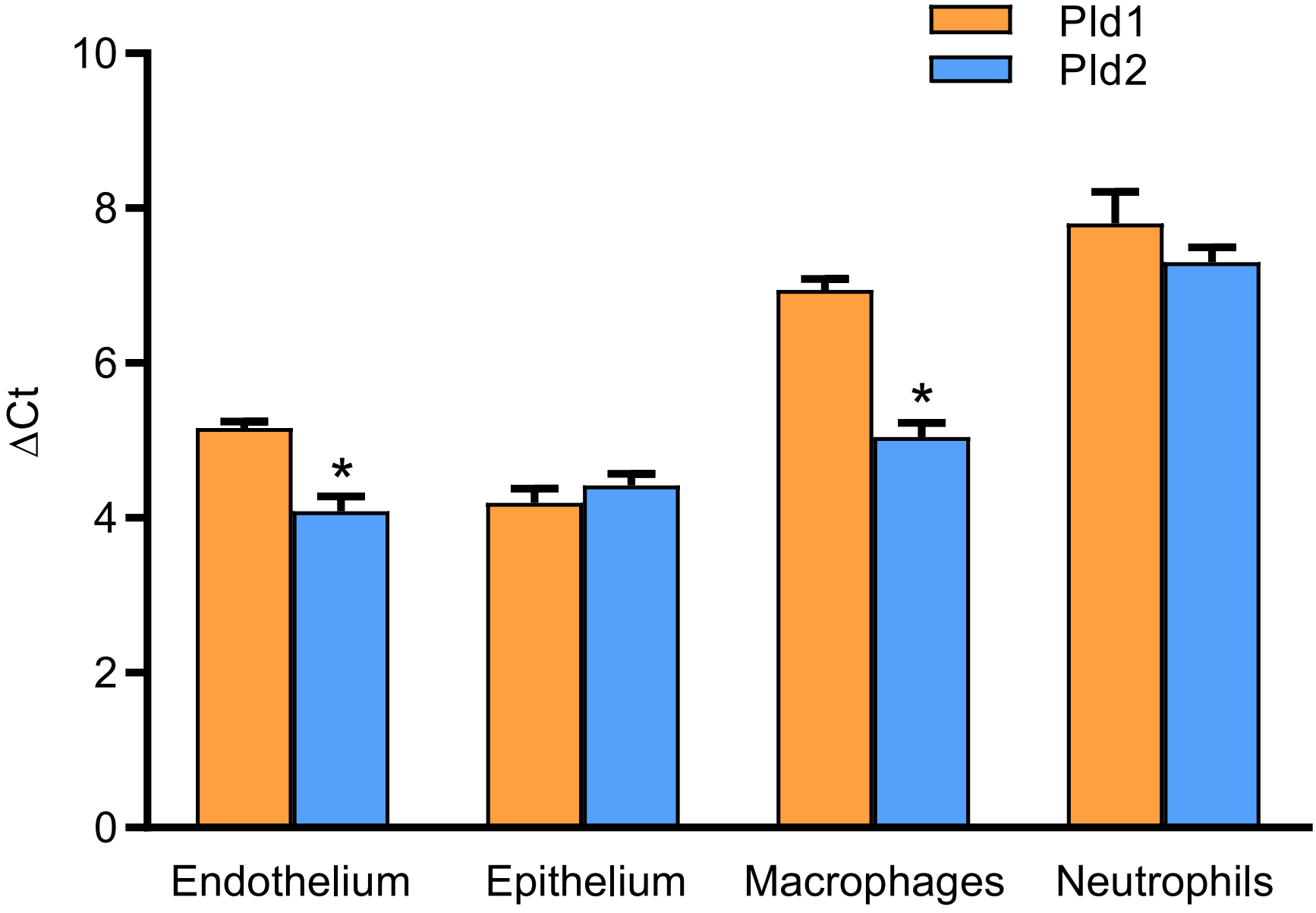

Figure S3. PLD isoforms are expressed in lung cells. *Pld1* and *Pld2* gene expression in lung endothelial cells, epithelial cells, neutrophils, and macrophages sorted by flow cytometry 24 hrs after acid-induced lung injury. Data expressed as mean ± s.e.m. N > 10, * P <, 0.01 relative to *Pld1*

Figure S4

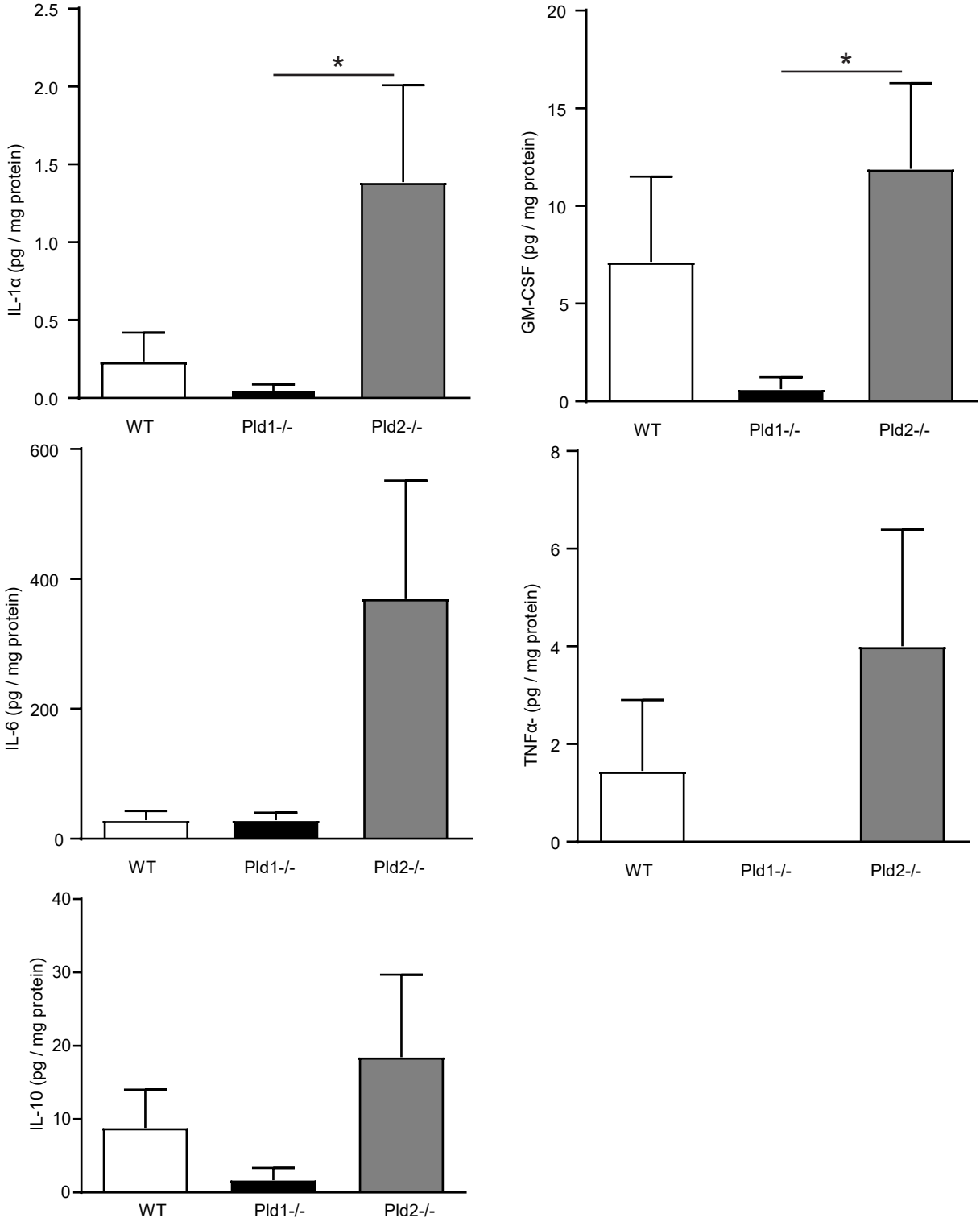

Figure S4. PLD2 deficiency selectively regulates BAL cytokine release after ALI. Levels of IL-1α, GM-SCF, IL-6, TNFα, and IL-10 24 hrs after intra-tracheal HCl determined by bead-based immunoassay. Results are expressed as mean ± s.e.m. n = > 10 per group., * $P < 0.05$

Figure S5

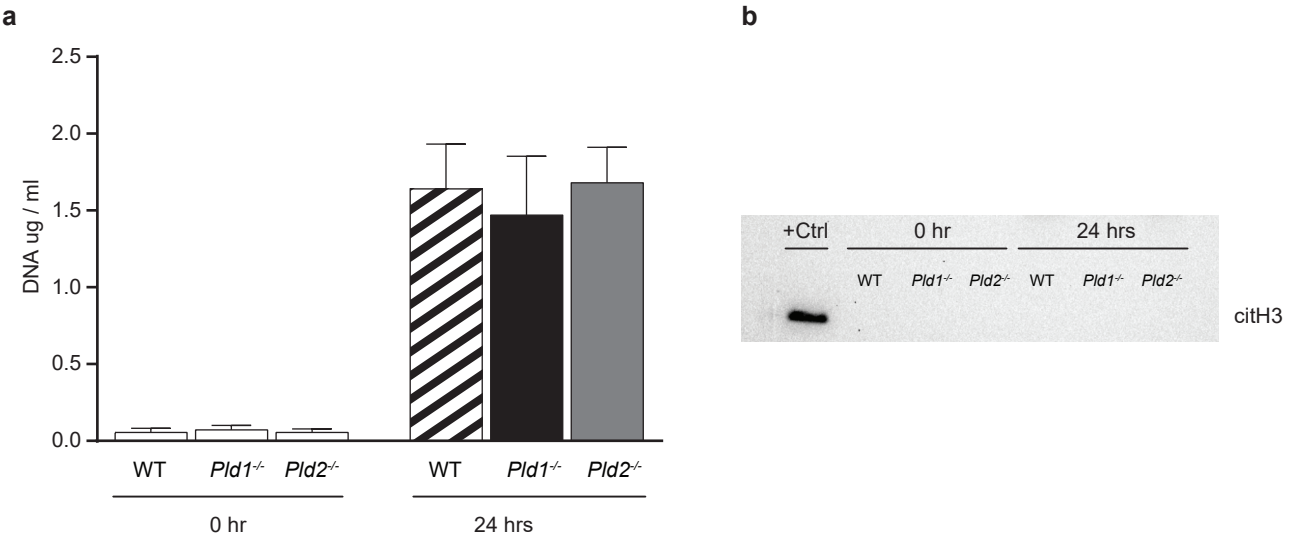

Figure S5. NETosis in acid-induced ALI. (a) DNA content in BAL fluid 24 hrs after intra-bronchial HCl. Results are expressed as mean ± s.e.m. n = > 10 per group. (b) Representative immunoblot against citrulinated histone H3 (citH3) on BAL fluid protein obtained 24 hrs after intra-bronchial HCl.