Fig. S1. POPG suppression of proinflammatory cytokine production in NHBE cells. NHBE cells from three subjects were either sham treated (CONL); or infected with RSV for 48 h, in either the absence (RSV) or presence of 200 μg/mL phospholipids (+POPG, +POPC), using a viral multiplicity of infection of 2. Cells were also treated with phospholipid alone (POPG, POPC). IL-6 and IL-8 released into media were quantified by ELISA. Values shown are means ± SE and reveal the intersubject variability as well as the variability for different treatments. Duplicate measurements were performed for each subject. *P < 0.05; **P < 0.001.

Fig. S2. POPG suppresses RSV-induced IL-6 and IL-8 production by BEAS2B cells. IL-6 (A) and IL-8 (B) production by the BEAS2B cell line were measured by ELISA after sham treatment (CONL) or infection with virus (RSV) for 48 h, in either the absence or presence of 200 μg/mL POPG or palmitoyl-oleoyl-phosphatidylcholine (+POPG or +POPC). The viral multiplicity of infection was 1. Additional control experiments included cells treated with phospholipid alone (POPG, POPC). Values shown are means ± SE for three independent experiments. *P < 0.01. Average IL-6 production upon RSV challenge was 3,568 ± 532 pg/mL. Average IL-8 production was 6,144 ± 972 pg/mL.
Fig. S3. POPG does not produce pleiotropic inhibition of metabolism or TLR5-induced signaling. (A) Beas2B cells were incubated for 48 h with 3H-leucine in either the presence or absence of 200 μg/mL of POPG as indicated. 3H-leucine incorporation into macromolecules precipitated by trichloroacetic acid was measured by liquid scintillation spectrometry. Normalized values shown are means ± SE for three independent experiments. Absolute values obtained were as follows: CONL, 11,274 ± 4,018 (cpm) for sham incubation; POPG, 12,841 ± 5351 (cpm) for phospholipid treatment. (B) Beas2B cells were treated with the TLR5 agonist flagellin (Alexis Biochemicals) at 10 ng/mL (Fla) for 48 h in either the absence or presence of phospholipids (+POPG, +POPC). Control conditions containing only phospholipid (POPG, POPC) were also included. IL-8 production by the cells was measured by ELISA and is expressed as the normalized mean ± SE for cells from three independent experiments. Absolute values obtained in the three experiments were as follows: CONL, 256 ± 104; Flagellin, 5,383 ± 745; Flagellin + POPG, 5,548 ± 873; Flagellin + POPC, 6,302 ± 1,216.7; POPG, 272 ± 111.5; and POPC, 382.9 ± 161.

Fig. S4. POPG prevents the cytopathic effects of RSV upon BEAS2B cells. BEAS2B cells were either sham treated (CONL), or challenged with virus (RSV) for 72 h, in either the absence or presence of 200 μg/mL phospholipids (+POPG, +POPC) as indicated. The viral multiplicity of infection was 1. Additional conditions exposed the cells to phospholipids alone (POPG, POPC). Bar inset in CONL panels, 50 μm.