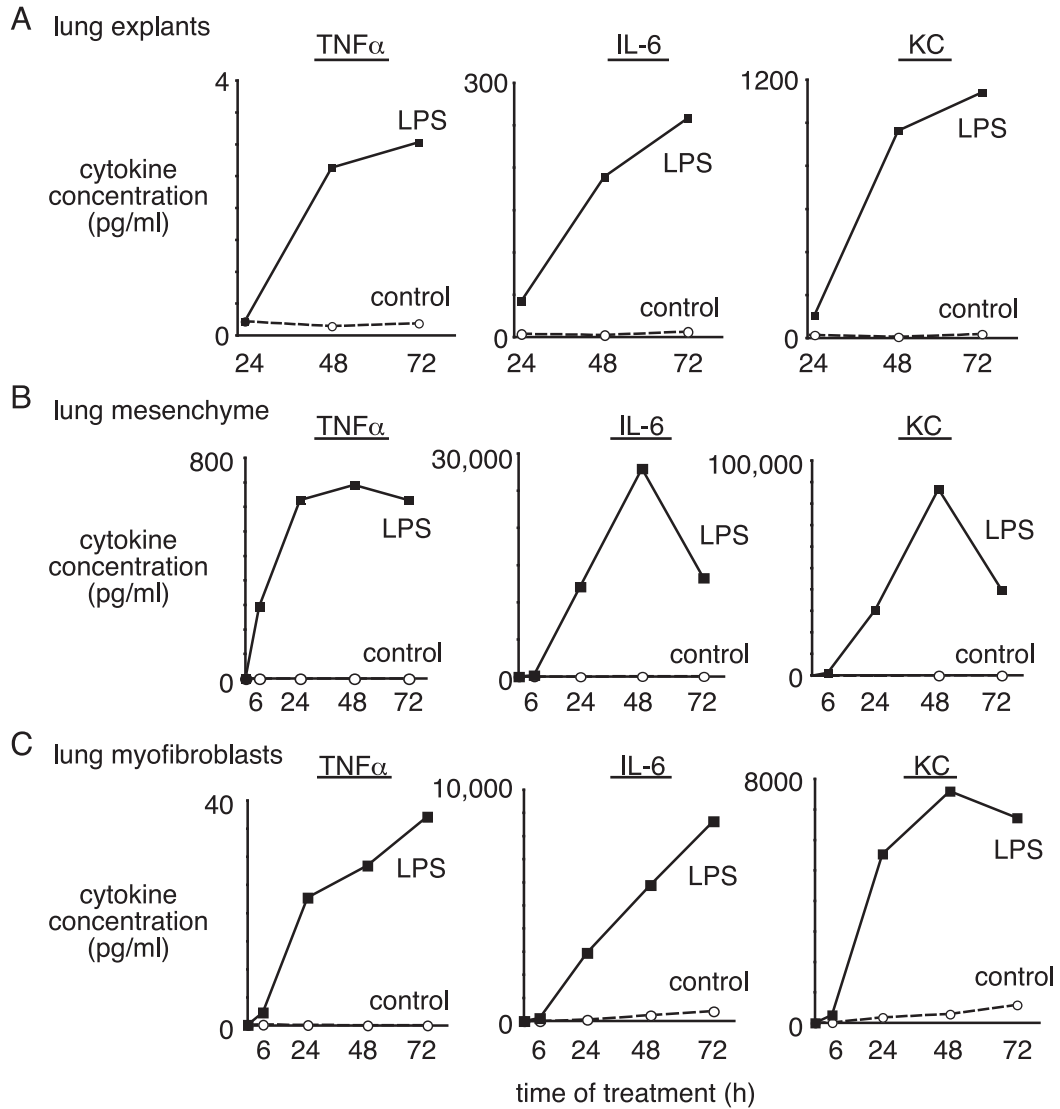


Supplemental Figure 1. Inflammatory mediators produced by fetal mouse lung explants, mesenchymal cells, and myofibroblasts. To verify that cells within the fetal mouse lung were responsive to innate immune activation, 250 ng/ml of *E. coli* LPS was added to E15 fetal mouse lung explants (A), primary cultures of fetal lung mesenchyme (B), or cultured fetal mouse lung myofibroblasts (C). Myofibroblast cultures were obtained by first isolating fetal mouse lung mesenchymal cells, and passaging over 10 times to obtain a homogeneous population of cells that contained alpha-smooth muscle actin-positive stress fibers (not shown). For all experiments, LPS was added in serum-free DMEM. At indicated time points, the media was harvested and inflammatory mediator concentrations were measured using a Luminex assay. All time points and conditions were repeated in triplicate, and mean value is shown for each data point.



Cause of preterm labor	IL-6 (pg/ml)	IL-10 (pg/ml)	IP-10 (pg/ml)	RANTES (pg/ml)
Chorioamnionitis	114.2 (+/- 138.2)	9.9 (+/- 28.3)	139.2 (+/- 726.7)	76.4 (+/- 212.8)
Pre-eclampsia	22.5 (+/- 29.8)	6.1 (+/- 4.5)	46.9 (+/- 24.3)	24.3 (+/- 11.5)
Other	14.9 (+/- 167.1)	1.8 (+/- 2.2)	22.3 (+/- 22.8)	8.8 (+/- 132.8)

Supplemental Table I. Inflammatory mediator concentrations in tracheal aspirate fluid obtained from preterm infants. Samples were obtained via endotracheal suctioning during the first 24 h of life and assayed using a SearchLight Assay platform (Pierce Endogen). Samples were grouped based on the clinical and pathological cause of preterm birth. Values are reported as median (+/- standard deviation) for 9 chorioamnionitis samples, 7 pre-eclampsia samples, and 6 other samples.