**Purpose of Study** Age-associated chronic diseases are associated with a pro-inflammatory state. It has been challenging to determine cause and effect – do age-associated pathologies increase inflammation or does inflammation induce age-associated pathologies or both? We previously showed that disease-related regenerative asynchrony in repairing lung is the cause of chronic inflammation and fibrosis. Thus, we hypothesized that the aged lung is itself asynchronously regenerating leading to a pro-inflammatory pulmonary milieu.

**Methods Used** Tracheas and intra-cardiac blood were harvested from C57BL6 mice in two age groups of both genders. Young mice were between 8 and 20 weeks of age. Aged mice were between 23 and 33 months of age. Tracheal epithelial progenitor cells were isolated and cultured for 6 days with continuous exposure to BrdU. Cellular regeneration was analyzed by flow cytometry for 7-AAD DNA staining in BrdU+ cells. Concentrations of an initial screening set of cytokines in plasma and cell culture supernatants from days 2 and 6 of culture were measured using magnetic bead-based assays.

**Summary of Results** Fewer airway epithelial progenitors underwent mitosis from the aged than the young mice (16.9±10.4% vs. 62.2±9.4% of the cultured cells at 6 days). The tracheal epithelial progenitors from aged mice were asynchronously distributed along the cell cycle (G1, S, G2/M: 44, 25, and 31%) compared to those from young mice (62, 14, and 24%). Plasma concentrations of IL-1β, IL-6, TNFα and TGFβ were not significantly different between age groups. Concentrations of TGFβ were significantly different between age groups in supernatant from day 2 (aged=112.43±16.31 pg/mL, young=171.23±13.70 pg/mL; p<0.05) but not from day 6 of culture (aged=159.60±29.83 pg/mL, young=214.15±94 pg/mL; p=NS). Concentrations of IL-1β were not significantly different between age groups in supernatant from day 2 of culture (aged=2.01±0.23 pg/mL, young=2.10±0.24 pg/mL; p=NS) but remained higher in aged compared to young progenitors on day 6 (aged: 2.17±0.31 pg/mL, young: 1.26±0.10 pg/mL; p<0.05).

**Conclusions** Our data support the concept that aging induces progenitor cell mitotic asynchrony. It is possible that this epithelial mitotic asynchrony contributes to the pro-inflammatory state associated with aging, as seen in other chronic inflammatory states.