**Background**

IL-13 transforms cultured normal human bronchial epithelial (NHBE) cells into goblet cells that secrete mucus, leukotrienes and inflammatory mediators.

We have shown that clarithromycin, but not dexamethasone, can inhibit IL-13 goblet cell transformation of NHBE cells.

**Objectives**

We hypothesized that azithromycin and dexamethasone would decrease the production of immunomodulatory mediators in goblet cells and we evaluated inflammatory mediator production by multiplex ELISA.

**Methods**

**Cell culture model**

NHBE cells were grown for 14 days at air-liquid interface (ALI) with PBS vehicle or IL-13 5 ng/mL as well as azithromycin 1 μg/mL (AZ), dexamethasone 1 μg/mL (Dex), or DMSO vehicle.

**Histological analysis**

Histology was performed using H&E and periodic acid-Schiff (PAS) stains, and immunofluorescence for MUC5AC & acetylated α-tubulin for cilia.

**Multiplex bead assay**

Multiplex bead assay of 25 inflammatory mediators was performed in the apical supernatants and basal culture medium from these cultured cells.

**Results**

Ciliated cells are weakly stained with MUC5AC and strongly stained with acetylated α-tubulin at the surface of epithelial layers, whereas goblet cells with secretory granules strongly stained with MUC5AC, but there was no acetylated α-tubulin seen.

**Discussion**

Neither AZ or Dex inhibited goblet cell hyperplasia.

Th1 cytokines & chemokines:

IFN-γ and related chemokines were inhibited by both AZ and Dex.

Th2 cytokines:

IL-4 and IL-9 were inhibited by both AZ and Dex.

IL-13 was inhibited by AZ but not by Dex.

Th17 & Neutrophil activating cytokines:

IL-17 and IL-6 were inhibited both by AZ and Dex.

Other inflammatory cytokines:

TNF-α and MIP-1α were inhibited by both AZ and Dex.

IL-1β and MCP-1 were inhibited by Dex and apical IL-7 was inhibited by AZ.

Other growth factors:

Growth factors, basolateral FGF, PDGF and VEGF, which may contribute to airway remodeling were not inhibited by AZ, however PDGF was inhibited by Dex.

**Conclusions**

Inflammasome profiling suggests that the airway goblet cell is an inflammatory effector cell capable of producing proinflammatory cytokines and chemokines.

Although both AZ and Dex showed selective anti-inflammatory effects, AZ more effectively inhibited Th2 cytokines than Dex. AZ does not appear to have an effect on mediators associated with airway remodeling.