European Respiratory Society
Annual Congress 2013

Abstract Number: 4113

Publication Number: P578

Abstract Group: 3.2. Airway Cell Biology and Immunopathology

Keyword 1: Asthma - mechanism **Keyword 2:** Experimental approaches **Keyword 3:** Anti-inflammatory

Title: Stimulation of IL-8 release in respiratory cell cultures by increasing IL1-β concentrations: Effect of budesonide

Dr. Juan 25823 Figueroa figuejuan@gmail.com MD [1], Dr. Fernanda 25824 Elias figuejuan@gmail.com [1,2], Mrs. Mariana 25825 Callello figuejuan@gmail.com [1] and Dr. Andrea 25826 Dugour figuejuan@gmail.com [1,2]. [1] CIRES, Fundación P.Cassará, Buenos Aires, Argentina, 1085 and [2] Molecular Biology, TimeDNA, Buenos Aires, Argentina, 1085 .

Body: Background: in airways of patients with severe asthma it have been described higher levels of IL-1β and IL-8. Previously we described that stimulation with IL1-β induces the release of IL-8 in human bronchial cells (Calu-3) and that this effect diminishes with budesonide (BUD). Objective: to evaluate the effect of increasing concentrations of IL1-β on the release of IL-8 by Calu 3 cells and to establish the action of BUD in this model. Materials and Methods: cells were cultured in monolayer and exposed to increasing concentrations of IL1-β (10-150 ng / ml) and H2O2 100 uM. After 4 hours of incubation inflammatory agent was removed and cells were treated with BUD (30 and 120 uM) or placebo for 24 hours. IL-8 release was then quantified. Results: with higher concentrations of IL1-β the release of IL-8 by Calu3 cells was greatest (10 ng of IL1-β induce 54 ng/ml of IL-8; 50 ng of IL1-β induce 78 ng/ml of IL-8). BUD anti-inflammatory effect was lower in cells stimulated with higher concentrations of IL1-β (IL-8 release decrease 12%) than in cells treated with lower concentrations (IL-8 release decrease 30%). Conclusions: higher IL1-β concentration increase proinflammatory response of respiratory cells and decreases the effect of BUD. The behavior of this model is consistent with clinical and biological data of patients with severe asthma.