VOLUME 46 / SUPPLEMENT 59 / SEPTEMBER 2015

# EUROPEAN RESPIRATORY *journal*

OFFICIAL SCIENTIFIC JOURNAL OF THE ERS

## Abstracts / *25th International Congress*

Amsterdam, Netherlands 26 –30 September 2015

Online ISSN: 1399-3003

ERS EUROPEAN RESPIRATORY SOCIETY
every breath counts

Citations should be made in the following way: **Authors. Title. Eur Respir J 20015; 46: Suppl. 59, abstract number**.

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## 257. Pathophysiological mechanisms at different scales: lung, airways, muscles and symptom perception

**PA2301**

**Small airways in in sedentary and endurance-trained dystrophic (mdx) mice**

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The effects of mild endurance exercise training on the small airways in mdx mice are unknown. We compared epithelial thickness and turnover, apoptosis, and stress marker expression in small airways of mdx mice and wild-type (WT) controls, at rest and during exercise training. Mdx and WT mice were randomly assigned to sedentary (mdx-S, n=17; WT-S, n=19) or trained (mdx-EX, n=14; WT-EX, n=16) groups. Low-intensity endurance training (running on a wheel) was done 5 d/wk for 6 wk at progressively increasing speed (rpm from 16 to 24) and time (15 min to 1 h). Lungs were processed for light microscopy and periodic acid Schiff (PAS) staining. Hsp60 and PCNA were quantified by immunohistochemistry. Apoptosis was assessed by TUNEL. Bronchial epithelial thickness decreased over time in WT mice irrespective of training (linear regression for time trends: WT-S: $R^2$=0.43, r= -0.65; WT-EX: $R^2$=0.68, r= -0.82, p<0.0005 for both); conversely, no significant change occurred in mdx mice. The number of PAS+ goblet cells was much lower in the bronchiolar epithelium of mdx compared to WT mice in all conditions. At 30 days, PCNA positivity was higher in EX than S animals in both groups; however, at 45 days it sharply decreased in mdx-S and -EX, but not in WT mice. The percentage of TUNEL+ cells was higher in mdx-EX than WT-EX mice at 45 days. In mdx mice, expression of Hsp60 progressively decreased (p<0.01), and was inversely related to the percentage of TUNEL+ cells ($R^2$=0.44, r=-0.66, p=0.01). In conclusion, bronchiolar epithelium in mdx mice is poor of goblet cells, and progressively deteriorates over time possibly because of loss of stress-related protective mechanism. Mild training did not cause any additional damage.

## 527. Cellular and molecular mechanisms of COPD and emphysema

**PA5049**

**Role of chronic exposure to cigarette smoke on endoglin/CD105 expression in airway epithelium**

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Dysregulation of airway epithelium function related to cigarette smoke exposure plays an important role in the pathophysiology of COPD and is associated to tissue damage and disease severity. CD105 is a component of the receptor complex of TGF-β, a pleiotropic cytokine involved in cellular proliferation, differentiation and migration. CD105 regulates the expression of different components of the extracellular matrix suggesting a role of CD105 in cellular transmigration and remodeling processes. The aim of the present study was to investigate the expression of endoglin/CD105 in airway epithelium of COPD patients and its involvement in tissue remodeling and COPD progression. We evaluated the immunoreactivity for CD105 expression in bronchial biopsies of COPD patients and healthy controls (HC). The analysis of metaplastic epithelium was performed in bronchial biopsies by Image Analysis software (Leica Quantimet). Finally, we investigated, by western blot, the expression of CD105 protein receptor in human bronchial epithelial cells (16HBE) exposed to 5% Cigarette Smoke Extract (CSE) for 12 days. We found that the CD105 immunoreactivity was significantly higher in bronchial epithelium of COPD than HC. Morphometric analysis of bioptic samples of COPD showed an increase of the CD105 immunoreactivity in the area of metaplastic than in not metaplastic epithelium. Long term exposure to CSE significantly up-regulated CD105 expression in 16HBE. Chronic inflammation due to cigarette smoke might play a critic role on the alteration of CD105 protein expression in COPD, promoting tissue remodeling, angiogenesis and dysregulation of physiological reparative mechanisms, leading to squamous metaplasia.