

# European Respiratory Society Annual Congress 2013

**Abstract Number:** 1331

**Publication Number:** P609

**Abstract Group:** 3.2. Airway Cell Biology and Immunopathology

**Keyword 1:** Biomarkers **Keyword 2:** Cell biology **Keyword 3:** COPD - mechanism

**Title:** Myofibroblast expression in airways and alveoli is affected by smoking and COPD

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**Body:** Chronic obstructive pulmonary disease (COPD) is characterized by structural changes in airways and alveoli. Our aim was to analyse the numbers of alpha-smooth muscle actin ( $\alpha$ -SMA) positive cells, as a marker of myofibroblasts, in different lung compartments in non-smokers and smokers with normal lung function or COPD.  $\alpha$ -SMA, tenascin-C (Tn-C) and EDA-fibronectin were assayed by immunohistochemistry and quantified by image analysis. There were less distal alveolar distensions containing  $\alpha$ -SMA positive cells in alveoli (Figure 1) and  $\alpha$ -SMA positive cells in bronchioles in smokers with normal lung function and COPD compared to non-smokers. Quantity of  $\alpha$ -SMA positive myofibroblasts was increased in bronchi in COPD. Tn-C expression was elevated in bronchi in COPD and smokers' lung. Myofibroblasts are localized variably in normal and diseased lung suggestive of roles in regeneration of lung and pathogenesis of COPD. Distal alveolar distensions, newly characterized histological structures, may be the source of myofibroblasts at the alveolar level.

Figure 1. The immunohistochemical characterization of distal alveolar distensions. Image 1A demonstrates alveolar walls and distal alveolar distensions with  $\alpha$ -SMA positive cells (arrows) from peripheral lung of a patient with COPD. Image 1B represents the numbers of distal alveolar distensions, which were positive for  $\alpha$ -SMA in different patient groups. \*\*\*  $p < 0.001$ .