European Respiratory Society
Annual Congress 2013

Abstract Number: 4562

Publication Number: P5159

Abstract Group: 4.3. Pulmonary Circulation and Pulmonary Vascular Disease

Keyword 1: Airway smooth muscle **Keyword 2:** Epigenetics **Keyword 3:** Cell biology

Title: Long intergenic non-coding RNAs are involved in the differentiation of smooth muscle cells

Dr. Melina M. 29632 Musri mmusri@clinic.ub.es [1,2], Dr. Victor I. 29633 Peinado vpeinado@clinic.ub.es [2], Dr. Joan Albert 29634 Barberà jbarbera@clinic.ub.es MD [2] and Prof. Dr Gunter 29635 Meister Gunter.Meister@vkl.uni-regensburg.de [1]. [1] Biochemistry Center Regensburg (BZR), Laboratory of RNA Biology, University of Regensburg, Regensburg, Germany and [2] Department of Pulmonary Medicine, Hospital Clínic-IDIBAPS, CIBER De Enfermedades Respiratorias, Barcelona, Spain .

Body: Alterations in smooth muscle cell (SMCs) plasticity play an important role in vascular remodeling associated to chronic obstructive pulmonary disease. Long intergenic non-coding RNAs (lincRNAs) have been shown to regulate a number of physiological processes by regulatory gene expression. Our objective was to deep sequence the whole transcriptome of SMCs during differentiation and to identify lincRNAs that participate in the regulation of this process. Primary human pulmonary artery SMCs (Lonza) were differentiated by allowing them to achieve cell-to-cell contact, a factor that triggers differentiation. Proliferative cells (D0) and cells at the beginning of the differentiation (D2) were analyzed. RNA-seq libraries were prepared using TruSeq RNA Sample Kit from Illumina and sequenced on a HiSeq1000. Expression of SMC markers and selected lincRNAs was measured by real time PCR. Knockdown of lincRNAs was performed by transfection of siRNA using Lipofectamine RNAiMax (Invitrogen).13 million reads were obtained after quality control. Significantly higher expression of mature markers of SMC, such as [a]-actin, growth differentiation factor 5 (GDF5) and SM-leiomodin 1 (LMOD1) at D2 confirmed the reliability of our results. We observed a number of annotated lincRNAs as well as non-annotated lincRNAs that significantly change from D0 to D2. Interestingly, knockdown of lincRNAs leads to differentiation defects suggesting an important role of them in SMC differentiation. We conclude that lincRNAs are tightly regulated during the differentiation of SMC and might play a key role during this process. Supported by grants 10/02175, and SEPAR-2009, MMM is recipient of a Sara Borrell contract from ISCiiiα.