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Title: LSC 2012 abstract – Wnt11 is identified in 3D human lung tissue model as regulator of distal airway epithelial cell differentiation

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Body: Alveolar Type II (ATII) cells repopulate the damaged alveolar surface and transdifferentiate into Alveolar Type I (ATI) cells during physiological regeneration. During pathological airway repair excessive Epithelial-Mesenchymal Transition (EMT) might occur, resulting in fibrosis. Mesenchymal signals might contribute to the differentiation and regeneration of pulmonary epithelium stimulating distal airway epithelial differentiation and preventing EMT. To identify these factors, we constructed a 3-dimensional (3D) human tissue model of primary pulmonary cells to mimic epithelial-mesenchymal interactions in the human lung. Distal airway epithelial cells differentiate into ATII-like cells as suggested by morphological changes as well as increased expression of differentiation markers AQP3, SP-A, SP-C in the 3D model. Wnt11 was identified in the model and in human lung explant cultures as one of the main regulators of ATII differerentiation. Added Wnt11 increased the expression of ATII markers, while silencing of Wnt11 resulted in elevated levels of EMT markers N-cadherin and S100A4. We conclude that the 3D lung model is applicable for studying epithelial-mesenchymal interactions in the lung. Our finding may mark Wnt11 as a potential therapeutic target in lung regenerative therapy.