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MEETING ABSTRACTS

DISRUPTION OF THE ARYL HYDROCARBON RECEPTOR (AHR) SIGNALING ALTERS FUNCTIONS AND PRODUCTION OF SURFACTANT IN A HUMAN MODEL OF ALVEOLAR TYPE II CELLS

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The aryl hydrocarbon receptor (AhR) is a well-known cellular sensor of xenobiotics and major transcription regulator of xenobiotics-metabolizing enzymes. Recent studies have indicated that AhR is also important for physiological immunological functions of barrier organs, such as skin, gut, and lung. Nevertheless, its functions in epithelial cells of barrier organs are far less explored. Alveolar epithelial type II cells (ATII), also known as type II pneumocytes, are important regulators of functions of alveolar epithelium, which contribute to its regeneration, and production of surfactant. Surfactant lipids and proteins, which cover alveolar epithelium, both reduce surface tension and provide protection to pneumocytes. Here, we studied potential role of the AhR in the production of surfactant in a human model of ATII cells, A549 cell line. We have used AhR wild type and AhR-deficient A549 cells, in order to compare their capacity to express surfactant proteins, synthesize surfactant phospholipids, and produce surfactant layer, when cultivated at air-liquid interface (ALI). We then evaluated presence of lamellar bodies, as functional markers of differentiated ATII cells. Our results show that cells lacking AhR have an altered pattern of ATII markers, as compared with wild-type cells. These results suggest that toxicants activating and/or inhibiting AhR might potentially contribute to disruption of ATII cell functions and thus alter their role in the maintenance of lung homeostasis.

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