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Vortragsankündigung
22.05.2017 16.00 Uhr H47

"In vitro studies of fibroblast-to-myofibroblast transition in bronchial asthma – attempts to limit this phenomenon"

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Bronchial asthma is a chronic inflammatory disease of the lower respiratory tract. It is related to the presence of prolonged inflammation, consequently leading to airway hyperresponsiveness and whole bronchial tree reconstruction. The process of bronchial wall reconstruction, also known as remodelling, is a complex sequence of events. Many different types of cells participate in this process. On the one hand there are structural cells, which build bronchial wall such as bronchial epithelial cells, smooth muscle cells and fibroblasts, on the other hand there are immune cells which infiltrate bronchial tissue and secrete cytokines and growth factors enhancing the process of remodelling. Bronchial wall remodelling results in irreversible changes that are referred to: increased rate of divisions and size of smooth muscle cells (hyperplasia and hypertrophy), increased deposition of extracellular matrix proteins, or phenotypic differentiation of fibroblasts into myofibroblasts. These changes lead to reduction in the airway lumen and burdensome for the patient's symptoms such as wheezing, shortening of breath, chest tightness, or recurrent cough.

In our studies we concentrate on the fibroblasts-to-myofibroblasts transition (FMT) in bronchial asthma. We use human primary cell cultures, a technique widely used to investigate the pathomechanism of diseases and the search for novel therapy targets. We are trying to find molecular mechanism responsible for much higher FMT rate observed in human bronchial fibroblasts (HBFs) isolated from patients with asthma at the presence of key profibrotic factor - TGF-β. We are also looking for possible targets for future anti-remodeling therapies in bronchial asthma. During the lecture I will present you our latest research in the searching for new effective anti-fibrotic compounds in the group of methylxanthines derivatives.