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The biological role of extracellular vesicles in COPD

Extracellular vesicles (EVs), such as exosomes and microvesicles are released by many cell types into their environment. EVs contain a subset of proteins and nucleic acids such as messenger RNA and microRNA. EVs are thought to serve as a means of cell-to-cell communication and contribute to a number of disease states as they transfer their contents. COPD is a chronic inflammatory lung disease that causes obstructed airflow from the lungs. The main pathological changes of COPD are emphysema and small airway remodeling. Cigarette smoking has been widely recognized as the main causes of COPD. The noxious effects of smoking induce airway epithelial injury. Injured lung epithelial cells act as a source of various autocrine and paracrine factors. These suggest that the reciprocal interactions between the epithelium and mesenchyme are part of the important mechanism in COPD pathogenesis. Therefore the major aim of our study is to reveal the cell-to-cell interaction via EVs in COPD pathogenesis. Within research of our group, we investigated an EV-mediated intercellular communication mechanism between primary human bronchial epithelial cells (HBECs) and lung fibroblasts (LFs) and discovered that cigarette smoke extract (CSE)-induced HBEC-derived EVs promote myofibroblast differentiation in LFs. Remarkably, we elucidated that the novel mechanism of myofibroblast differentiation in LFs is attributed to the CSE-induced HBEC-derived EV miR-210 regulating autophagy machinery. Defining these mechanisms has potential as a new therapeutic target for COPD. The results will be presented and discussed.

Biography

Tsukasa Kadota has graduated from Jikei University School of Medicine where he also completed his Residency in Pulmonary Medicine. He is a Research Associate at The Jikei University School of Medicine and a Visiting Scientist at National Cancer Center Research Institute.

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