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Extended Abstract
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COPD 2016: The biological role of extracellular vesicles in COPD_Tsukasa Kadota_The Jikei University School of Medicine, Japan

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Extracellular vesicles (EVs), like exosomes and microvesicles, are released by many types of cells into their environment. VEs contain a subset of proteins and nucleic acids such as messenger RNA and microRNA. EVs are believed to serve as a cell-to-cell communication medium and contribute to a number of disease states during the transfer of their contents. COPD is a chronic inflammatory lung disease that obstructs the air flow to the lungs. The main pathological changes in COPD are emphysema and remodeling of the small airways. Smoking has been widely recognized as the main cause of COPD. The harmful effects of smoking cause epithelial damage to the airways. The injured lung epithelial cells act as a source of various autocrine and paracrine factors. These suggest that the reciprocal interactions between the epithelium and the mesenchyme are part of the important mechanism of the pathogenesis of COPD. Therefore, the main objective of our study is to reveal the cell-to-cell interaction via EVs in the pathogenesis of COPD. As part of our group's research, we studied a mechanism of EV-mediated intercellular communication between primary human bronchial epithelial cells (HBEC) and pulmonary fibroblasts (LF) and discovered that EV derived from HBEC derived from the extract cigarette smoke (ESC) promoted the differentiation of myofibroblasts in LF. Remarkably, we have elucidated that the new mechanism of differentiation of myofibroblasts in FL is attributed to the regulatory autophagy machinery EV miR-210 derived from HBEC induced by CSE. Defining these mechanisms could become a new therapeutic target for COPD. The results will be presented and discussed.

Cell-cell communication is crucial for all multicellular organisms and can be mediated by direct cell-cell contact or by transfer of secreted molecules. Currently, new mechanisms of intercellular communication have emerged that involve the transfer of extracellular vesicles (EV). In general, electric vehicles are classified according to their approximate size, origin and freight. The three main subclasses of EV include apoptotic bodies, exosomes and microvesicles. Although the exosomes and microvesicles are structurally similar, they differ in their cellular origin, their lipid composition and their size. Exosomes are 50 to 150 nm in diameter and are generated from the endosome. Exosomes have a set of proteins conserved during evolution, including tetraspanins (CD81, CD63 and CD9), the major histocompatibility complex (MHC) of classes I and II, Alix and Tsg101. Until now, on the one hand, it is difficult to identify the origin of exosomes through the specific protein assembly of the donor cell because the exosomes are derived from the endosomal pathway. On the other hand, microvesicles are formed by the outer budding and the fission of the plasma membrane. Microvesicles, also called microparticles or ectosomes, have a particle diameter of 100 to 2,000 nm. The lipid composition of microvesicles is similar to that of the cell membrane, but lacks the asymmetric distribution of lipids generally observed across the two layers of the plasma membrane. Consequently, the membrane composition of microvesicles more closely reflects that of the donor cell than the membrane composition of exosomes. Although the origin of these vesicles has been defined, current technologies do not allow a clear distinction to be made between the different types of EV. Isolating specific vesicles with reliable quality and at high concentrations remains a major challenge in this area.

The importance of EVs lies in their ability to transfer information to other cells, thereby influencing the cellular function of the recipient. A major breakthrough in VE research has been to identify the nucleic acid content of VEs, such as mRNAs, small non-coding microRNAs (miR) and long non-coding RNAs, which are transported to receptor cells. EVs have been isolated from most body fluids, such as blood, urine, BAL fluid (BALF) and saliva, and there is growing evidence that EVs play a key role not only in the regulation of normal physiological processes but also in the pathogenesis of the disease, including the regulation of inflammation and immune responses. Based on these results, EVs are promising candidates for intercellular communication in the airway microenvironment. VE contributes to the production of various pro-inflammatory mediators by respiratory and immune cells, potentially serving as key factors in the inflammatory process of the airways and pulmonary immunity.

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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