Exosomes in lungs of patients with sarcoidosis: a contributor to immune pathogenesis or just another by-product of heightened immune activity?

Ling-Pei Ho

The concept that cells can directly communicate with, and influence the function of other cells by transfer of particulate complexes or cell surface proteins (eg, antigen-bound MHC-II, integrin, ATPase channels)\(^1\)\(^-\)\(^3\) rather than soluble factors like cytokines and chemokines has excited cell biologists for decades. Extensive efforts have been made to prove the existence of this phenomenon and understand the mechanisms by which exosomes exist and can transfer cellular material, the focus has shifted to showing that they are enriched compared to healthy controls. The study utilised multi-modal imaging modalities to show their presence and, more significantly, demonstrate that these exosomes were able to induce production of cytokines from peripheral mononuclear cells and epithelial cells. This finding forms the first step in explorations of exosomal function in lung disease. Many questions can now be raised—what is/are the parent source(s) of these exosomes? Does increased amount of exosomes contribute to amplification of the CD4 T-cell response observed in sarcoidosis? Is this a sarcoidosis-specific finding or are lungs of patient with asthma, COPD and idiopathic pulmonary fibrosis also enriched with exosomes? And do different diseases have exosomes that bear different cellular proteins? Do

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different sarcoidosis patients with different outcomes have different composition of exosomes? It is possible to envisage, for example, that membrane-bound TGFβ from dendritic cell-derived exosomes are enriched in sarcoidosis patients who show a higher propensity for pulmonary fibrosis? Gazi and colleagues provide firm evidence for the presence of functional (albeit, in vitro) exosomes in sarcoidosis and pave the way for further questions which could show that these exosomes contribute to immune pathogenesis of sarcoidosis.

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