

Macrophage - derived apoptotic bodies promote the proliferation of the recipient cells via shuttling microRNA - 221/222

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Abstract

Bacterial pneumonia is a common and serious clinical entity. Alveolar epithelial cells and alveolar macrophages are the first line of defense in the innate immunity against bacterial pathogens. Epithelial cells are known to release chemokines/cytokines that recruit and activate phagocytic cells. However, the signals sent from alveolar macrophages back to the lung epithelial cells remain largely unexplored. We found that LPS, a well - recognized stimulator derived from gram - negative (G^-) bacteria, rapidly and robustly induces the secretion of macrophage - derived extracellular vesicles (EVs). The main type of EVs found in the early stages after LPS stimulation are apoptotic bodies (ABs) and not microvesicles (MVs) or exosomes (Exos). Furthermore, LPS markedly up - regulate the levels of a repertoire of microRNAs (miRNAs) in the macrophage - derived ABs, including miR - 221 and miR - 222. Functionally, the LPS - induced, macrophage - derived ABs promote the proliferation of malignant and/or normal lung epithelial cells. We next directly transfected miR - 221 and/or miR - 222 inhibitors into the LPS - induced ABs. Deletion of miR - 221/222 in ABs significantly reduces the AB - mediated proliferation of lung epithelial cells. Mechanistically, AB - shuttling miR - 221/222 promote cell growth by modulating cyclin - dependent kinase inhibitor 1B (CDKN1B) pathways. Collectively, LPS - induced, macrophage - derived ABs promote the proliferation of their recipient epithelial cells, partially via AB - shuttling miRNAs.

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