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Compartment-specific Immunophenotyping in Chronic Obstructive Pulmonary Disease

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Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide and is pathologically marked by irreversible airway obstruction, chronic inflammation, and lung parenchyma destruction (emphysema). Its phenotypes encompass more emphysema and more airway limitation, with/without vascular changes. Increasing evidence showed specific immune infiltrates in lung compartments. However, how consistent the immune profile is between different compartments remains unknown. We hypothesize that specific immune profiles differentiate COPD phenotypes and that a specific subset of immune cells contributes to airway and/or vascular remodelling. Here we investigated compartment-specific (bronchi, pulmonary arteries (PA), and lung parenchyma) inflammatory profiles by applying multi-panel flow cytometry. In addition, we analyzed previously published COPD single-cell RNA (scRNA) datasets and performed receptor ligand analysis to infer cell-to-cell communications. The most abundant population is neutrophils in the lung parenchyma (25-30%), while T cells are in small PA (20-50%) and bronchi (30-55%). Across three compartments, macrophage and monocytes showed relatively similar proportions (20-25%), while NK cells showed the highest proportion in the lung parenchyma (10-15%), followed by small PA and bronchi (1-5%). Interaction analysis in COPD showed more communication between (1) cytotoxic T cells and antigen-presenting cells (APC) and between (2) cytotoxic T cells and endothelial cells. Receptor ligand analysis predicted MHC I genes such as B2M and HLA-E from APC and endothelial cells to be bound with T cell receptor complex genes such as CD3D and CD247. Overall, immune populations are differentially abundant in COPD lung compartments and potentially interact with structural cells. We plan to validate the interactions using functional assays and integrate current data with clinical and microbiome data from matched patients.