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Cellular metabolic basis of altered immunity in the lungs of patients with COVID-19

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Abstract

Metabolic pathways drive cellular behavior. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection causes lung tissue damage directly by targeting cells or indirectly by producing infammatory cytokines. However, whether functional alterations are related to metabolic changes in lung cells after SARS-CoV-2 infection remains unknown. Here, we analyzed the lung single-nucleus RNA-sequencing (snRNA-seq) data of several deceased COVID-19 patients and focused on changes in transcripts associated with cellular metabolism. We observed upregulated glycolysis and oxidative phosphorylation in alveolar type 2 progenitor cells, which may block alveolar epithelial diferentiation and surfactant secretion. Elevated inositol phosphate metabolism in airway progenitor cells may promote neutrophil infltration and damage the lung barrier. Further, multiple metabolic alterations in the airway goblet cells are associated with impaired muco-ciliary clearance. Increased glycolysis, oxidative phosphorylation, and inositol phosphate metabolism not only enhance macrophage activation but also contribute to SARS-CoV-2 induced lung injury. The cytotoxicity of natural killer cells and CD8+ T cells may be enhanced by glycerolipid and inositol phosphate metabolism. Glycolytic activation in fbroblasts is related to myofbroblast diferentiation and fbrogenesis. Glycolysis, oxidative phosphorylation, and glutathione metabolism may also boost the aging, apoptosis and proliferation of vascular smooth muscle cells, resulting in pulmonary arterial hypertension. In conclusion, this preliminary study revealed a possible cellular metabolic basis for the altered innate immunity, adaptive immunity, and niche cell function in the lung after SARS-CoV-2 infection. Therefore, patients with COVID-19 may beneft from therapeutic strategies targeting cellular metabolism in future.

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