Monocyte migration profiles define disease severity in acute COVID-19 and unique features of long COVID

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Immune dysfunction is a key factor in acute COVID-19 pathophysiology, with monocyte abnormalities sustained during convalescence for at least 9 months following hospital discharge and corresponding to specific long COVID symptoms https://bit.ly/3xEIY0H


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chemokine ligand 16 (CXCL16) (p<0.05), which is abundantly expressed in the lung. Monocyte CXCR6 and lung CXCL16 were heightened in patients with progressive fibrosing interstitial lung disease (p<0.001), confirming a role for the CXCR6-CXCL16 axis in ongoing lung injury. Conversely, monocytes from long COVID patients with ongoing fatigue exhibited a sustained reduction of the prostaglandin-generating enzyme cyclooxygenase 2 (p<0.01) and CXCR2 expression (p<0.05). These monocyte changes were not present in respiratory syncytial virus or influenza A convalescence.

**Conclusions** Our data define unique monocyte signatures that define subgroups of long COVID patients, indicating a key role for monocyte migration in COVID-19 pathophysiology. Targeting these pathways may provide novel therapeutic opportunities in COVID-19 patients with persistent morbidity.