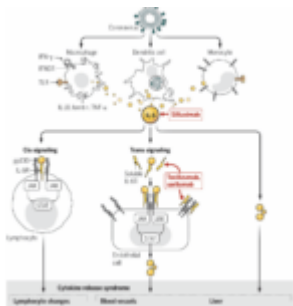
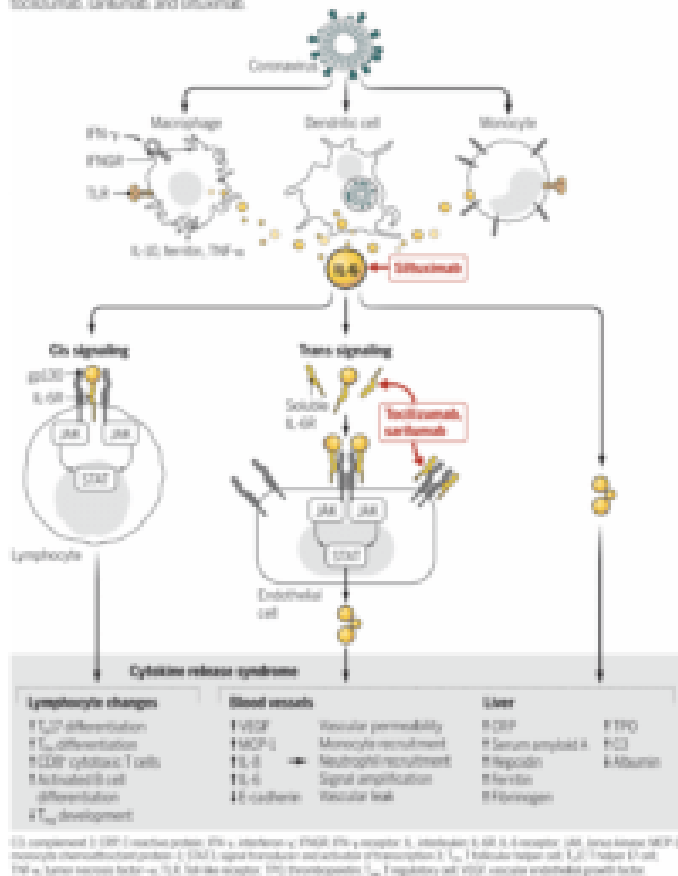


Cytokine Release Syndrome & COVID-19



Pathways leading to cytokine release syndrome

Coronavirus infection results in monocyte, macrophage and dendritic cell activation. IL-6 release then initiates an amplification cascade that results in cis signaling with T_H17 differentiation, among other lymphocyte changes, and trans signaling in many cell types, such as endothelial cells. The resulting increased systemic cytokine production contributes to the pathophysiology of severe COVID-19, including hypotension and acute respiratory distress syndrome (ARDS), which might be treated with IL-6 antagonists such as tocilizumab, canakinumab, and siltuximab.



Moore & June 2020

A recent perspective in Science asked whether there are lessons that can be learned from arthritis and cell therapy in cancer for COVID-19. Severe disease in 20% of COVID-19 cases appear to be from acute respiratory distress syndrome (ARDS),

which is often fatal. This poignant piece outlines the pathways leading to cytokine release syndrome (CRS), often seen in [hemophagocytic lymphohistiocytosis](#) and leukaemia patients receiving T cell therapy, and potential therapeutics that can suppress CRS. IL-6 release from macrophages, dendritic cells and monocytes (all activated during SARS-CoV-2 infection) is known to be central in amplifying the signalling leading to Th17 differentiation and pathology. Treatment with IL-6 antagonists may be one effective therapeutic intervention. The authors state: "The immediate goal of IL-6 antagonism is to ameliorate severe COVID-19 cases so that requirements for advanced care are minimized. The long-term goal should include a focus on the development of antivirals and vaccines that prevent or ameliorate the infection."

References: Moore & June, 2020. [Cytokine release syndrome in severe COVID-19](#). Science

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