

The role of complement in COVID-19 pathogenesis.

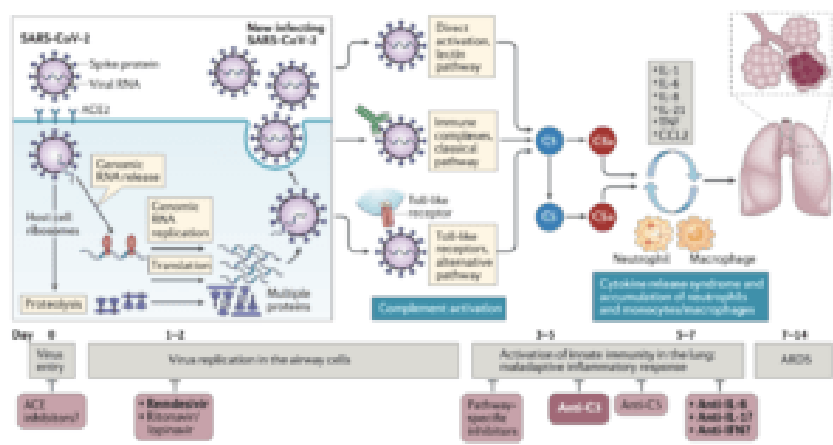
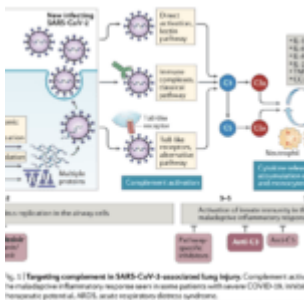


Fig. 1 | Targeting complement in SARS-CoV-2-associated lung injury. Complement activation may contribute to the maladaptive inflammatory response seen in some patients with severe COVID-19. Inhibition of C3 or C5 may have therapeutic potential. ARDS, acute respiratory distress syndrome.

Source: Risitano et al., 2020.

A recent commentary in Nature Reviews Immunology cites the role of complement in acute respiratory distress syndrome (ARDS) and that possibly targeting the inhibition of the complement cascade may be an important treatment. Complement is part of the innate immune response and is involved with the initiation pro-inflammatory responses. It has been found that activation of the complement component C3 by SARS-CoV, the closely related cousin of SARS-CoV-2, is involved in viral-associated ARDS. Inhibiting C3 with agents such as AMY-101, could have two effects: a) blocking C3a and C5a generation and the intrapulmonary C3 activation and IL-6 release from alveolar macrophages, and b) systemic inflammation affecting the microvascular beds of the kidney, brain and other vital organs – as often seen in late-stage COVID-19.

Journal Article: Risitano et al., 2020. [Complement as a target in COVID-19 ?](#) Nature

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