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Pathogenesis of COVID-19-induced ARDS: implications for an ageing population

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Review of viral ARDS pathogenesis, how it informs evolving models of COVID-19, and how hallmarks of ageing explain the age-related morbidity and mortality of severe COVID-19 <https://bit.ly/39Ca0c0>

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ABSTRACT The coronavirus disease 2019 (COVID-19) pandemic has elicited a swift response by the scientific community to elucidate the pathogenesis of severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2)-induced lung injury and develop effective therapeutics. Clinical data indicate that severe COVID-19 most commonly manifests as viral pneumonia-induced acute respiratory distress syndrome (ARDS), a clinical entity mechanistically understood best in the context of influenza A virus-induced pneumonia. Similar to influenza, advanced age has emerged as the leading host risk factor for developing severe COVID-19. In this review we connect the current understanding of the SARS-CoV-2 replication cycle and host response to the clinical presentation of COVID-19, borrowing concepts from influenza A virus-induced ARDS pathogenesis and discussing how these ideas inform our evolving understanding of COVID-19-induced ARDS. We also consider important differences between COVID-19 and influenza, mainly the protean clinical presentation and associated lymphopenia of COVID-19, the contrasting role of interferon- γ in mediating the host immune response to these viruses, and the tropism for vascular endothelial cells of SARS-CoV-2, commenting on the potential limitations of influenza as a model for COVID-19. Finally, we explore hallmarks of ageing that could explain the association between advanced age and susceptibility to severe COVID-19.