**Supplemental Table 1: Research questions raised by the SSC Research Committee:**

1) Is lasting immunity, from a clinical perspective, possible to SARS Coronavirus-2?

2) How does SARS Coronavirus-2 impair immune function?

3) Can we modulate the host response in SAR COV-2 for therapeutic benefit? (examples are Tocilizumab, plasma therapy, hydroxychloroquine but the list can be endless)

4) Should mechanically ventilated patients with ARDS and COVID-19 receive systemic corticosteroids?

5) Can we characterize macrophage activation syndrome in COVID-19 and define treatment?

6) Which are the diagnostic criteria of cytokine storm in Covid-19?

7) What is the immunobiology of COVID-19 post infective acute myocarditis?

8) Does SARS-CoV-2 viral load change the immune phenotypic response?

9) What is the optimal set of specific treatments to improve outcomes from patients with COVID19?

10) Can we develop antiviral agents for pre- and post-exposure prophylaxis​for COVID 19?

11) What characterizes the immune status in COVID-19 at different stages of the disease and should we monitor it?

12) What characterizes and explains the different phenotypes (ARDS predominant – Kawasaki like – myocarditis) in COVID-19?

13) Is there a place for modulation of renin angiotensin system (by agonist or antagonists)?

14) What are the best antihypertensive agents and vasopressor agents?

15) Which drugs are best to use for viral load modulation and when should we give them?

16) What specific cells are directly targeted by SARS-CoV-2, to what extent, and how do these cells respond to drive the complications of COVID-19? How do these cells respond to infection? Do they become dysfunctional or carry on normally while spewing out viruses?

17) Do endothelial cells play a central role in driving and/or potentiating COVID-19 disease, or in the immune response to COVID-19 disease, and could the endothelium be directly targeted to prevent COVID-19 complications (e.g.: ARDS, AKI, VTE, cardiovascular events)?

18) Can early data (e.g.: biomarkers, physiologic parameters, cognitive function, ex-vivo responses of cells to serum samples) be used to predict outcomes of COVID-19 (mild illness *vs* hospitalized but not critical *vs* critically ill), and, by extension, to guide therapies?

19) How can we perform and assess quality research during a pandemic?

20) What is the role of co-infection in COVID 19?

21) Is there a role for combination anti-viral therapies using agents with different mechanistic targets?

22) What is the role of biomarkers to de-escalate antibiotics in COVID 19?

23) What organisational changes to current ICU settings are needed in the setting of a pandemic?

24) Can heterogeneous paths of spread be exploited to arrest active transmission in a community or is heard immunity the only way to end the pandemic?

25) Can we development rapid point of care tests for screening – e.g., prior to surgical procedures, hospital admissions, etc.?

26) What is the role of asymptomatic carriage in community transmission?

27) Which are the predictors of ICU admission in Covid-19?

28) What are mechanisms of vascular dysfunction and thrombosis in COVID-19 and why are thrombotic manifestations more common in SARS CoV 2 infection compared to other infections or causes of critical illness?

29) What is the best approach to anticoagulation in patients with COVID-19?

30) What is the role of neutrophils netosis and sieving in COVID-19 microangiopathy?

31) Is there a role for activated protein C on COVID-19-induced coagulopathy?

32) Why are there age-related differences in the thrombotic and cardiovascular complication of COVID-19?

33) What is the expected recovery of chronic ICU patients from COVID-19?

34)  Do the long-term sequelae of severe COVID-19 disease differ from sequelae of sepsis/ARDS? (ie, is there anything unique to COVID-19 survivorship in terms of cardiopulmonary or other complications?

35) Does rehabilitation and physiotherapy play a role in shortening the ICU stay in patients with COVID-19?

36) Does SARS-CoV-2 induced social and physical problems influence the return to work and normal pre- Covid life for patients and families?

37) What is the impact on health outcomes unrelated to COVID19 but due to health system strain and physical distancing policies?

38) What is the incidence of dysfunction in organ systems other than the lungs? Do those who develop non-pulmonary dysfunction and survive have long-term dysfunction, like what is often following sepsis? In other words, do COVID-19 survivors get something akin to the Post Intensive Care Syndrome (PICS)?

39) What is the pathophysiologic mechanism as to why children are less severely affected than adults with acute infection?

40) What determines severity of illness (including co-morbidities) in individuals infected with COVID19? Are there immunogenetic factors associated with disease susceptibility, notably in high-risk groups?

41) Does a patient who is critically ill with COVID-19 have sepsis? What features of a “dysregulated host response” are common to both COVID-19 and sepsis? What features are different? What features are common to both COVID and “infection” (eg, pneumonia) from other viruses or from bacteria? What features are different?

42) What are good models to study COVID-19 pathophysiology, mechanisms and potential therapies?

43) Why is severe hypoxia subjectively well tolerated by patients with COVID 19?

44) What is the role of prone positioning and non-invasive ventilation in non-ventilated patients with COVID?

45) Does ECMO improve outcome in Covid-19 pneumonia?

46) Does COVID cause typical ARDS? (5, with an asterisk)

47) Should our approach to ventilator management differ from our standard approach in patients with acute hypoxic respiratory failure?
48) Should we do tracheostomy early in patients with COVID-19 respiratory failure?
49) Should the threshold for intubation differ in patients with COVID-19 respiratory failure?

50) Are there different phenotypes of respiratory failure in COVID-19 that require different strategies of ventilation?

51) In face of the lack of ICU beds, can we treat COVID 19 patients with mild to moderate ARDS with NIV outside the ICU?

52) Is the high incidence of mechanical ventilation due to a higher severity of illness in COVID-19 patients? Or is the high incidence of mechanical ventilation a function of differences in practice with early intubation in these patients?

53) Should patients above a certain age with COVID be intubated?

54) Does COVID-19 have any neurotropic effects on the brainstem and other parts of the brain?

55) What are the mechanisms responsible for encephalopathy in COVID-19?

56) In mechanically ventilated patients with ARDS, is a deep sedation strategy (e.g., target RASS -4 to -5) to achieve mechanical ventilation goals preferable to a light sedation strategy (e.g., target RASS 0 to -2) without achievement of mechanical ventilation goals?

57) Should COVID patients with shock be treated with a conservative or liberal fluid strategy for acute resuscitation?

58) Does cytokine absorbers improve organ dysfunction or survival in Covid-19?