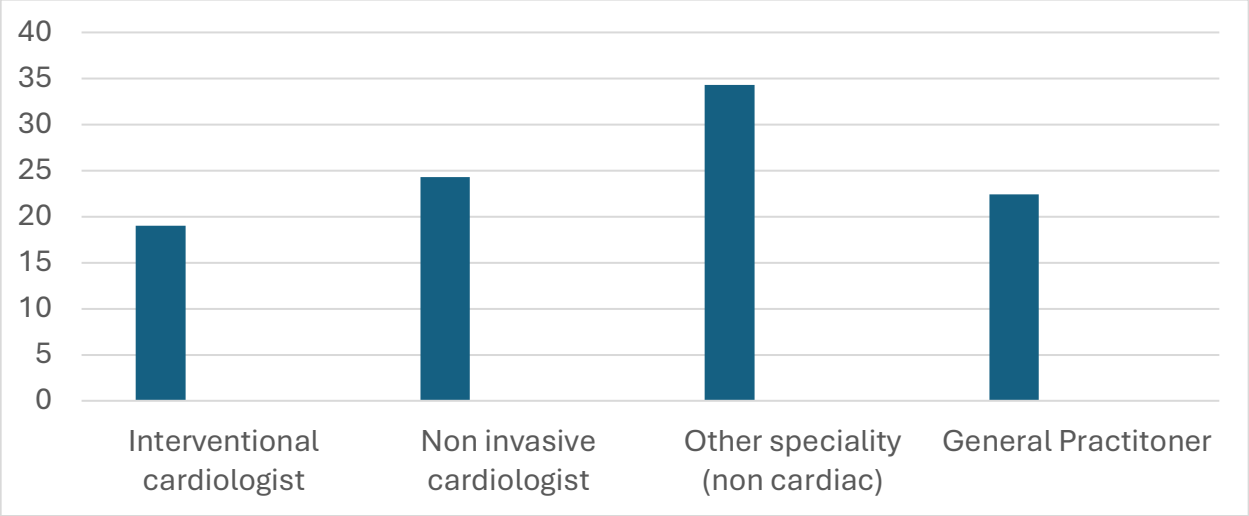
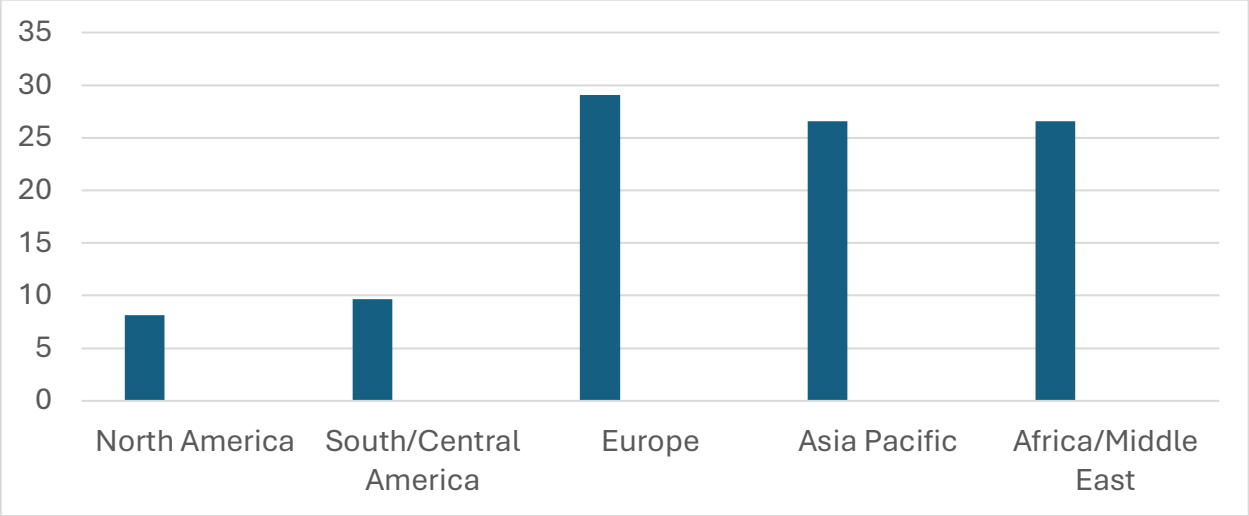
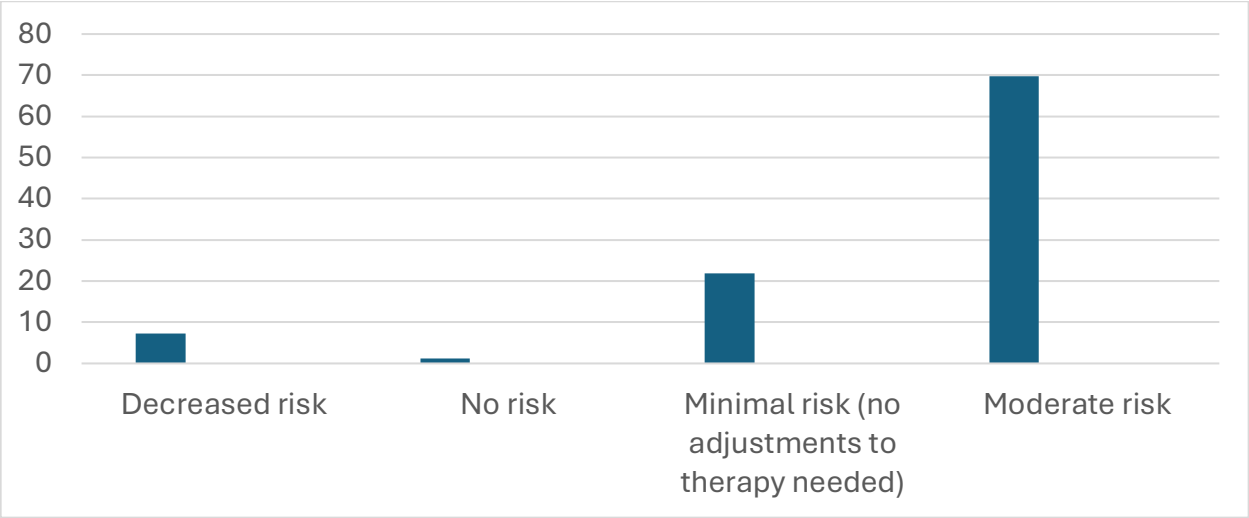
Supplementary Figure 1: "What is your specialty?"



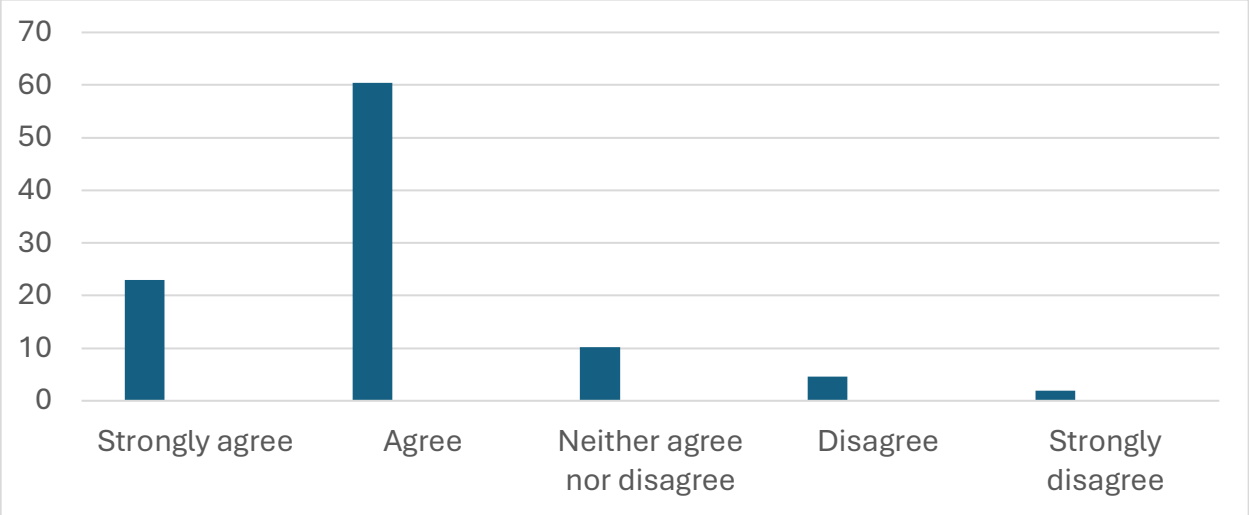
Supplementary Figure 2: "What region are you from?"



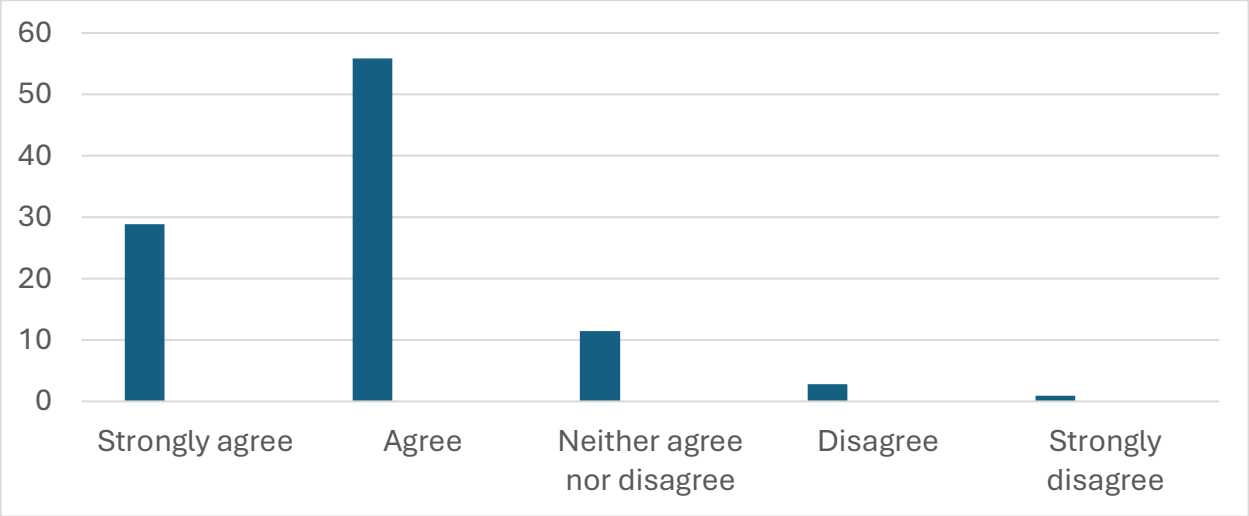
Supplementary Figure 3: "What is your perception of ischemic risk in patients with recent infective episodes?"



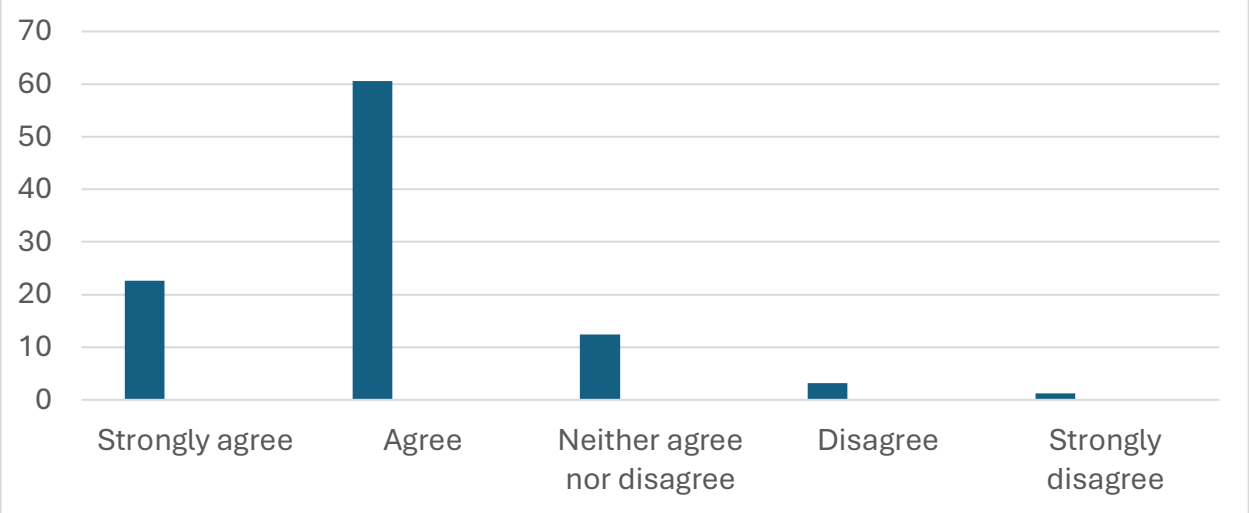
Supplementary Figure 4: "Infections including influenza, pneumonia, and acute bronchitis are associated with a transient increased thrombotic risk including myocardial infarction (MI) which appears to be proportional to time from and severity of infection."[1,2]



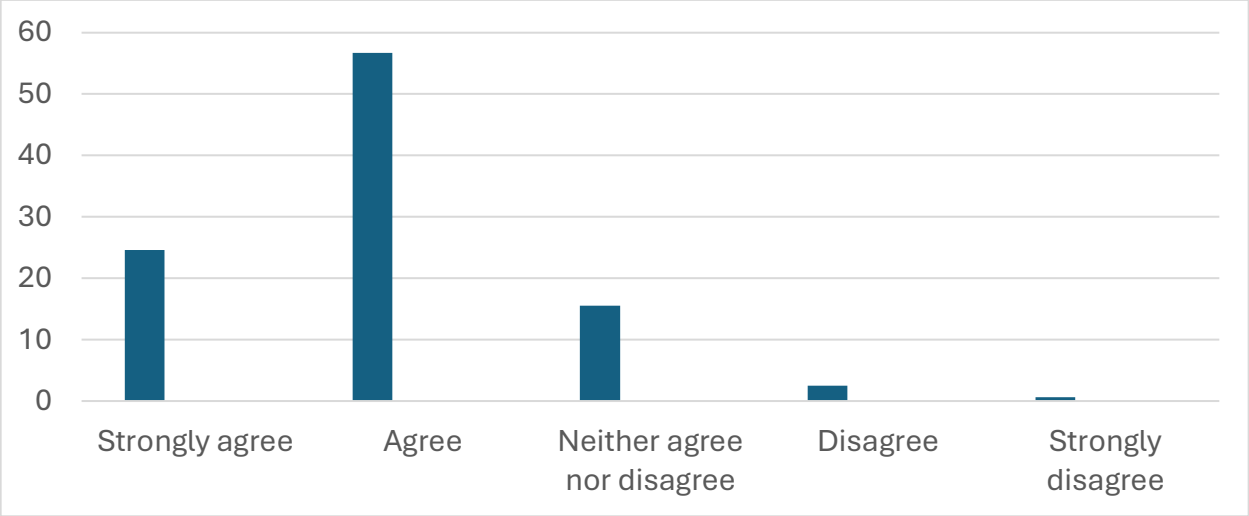
Supplementary Figure 5: "The most common thrombotic events associated with COVID-19 infection are venous thromboembolism (VTE) and thrombotic stroke."[1,3,4]



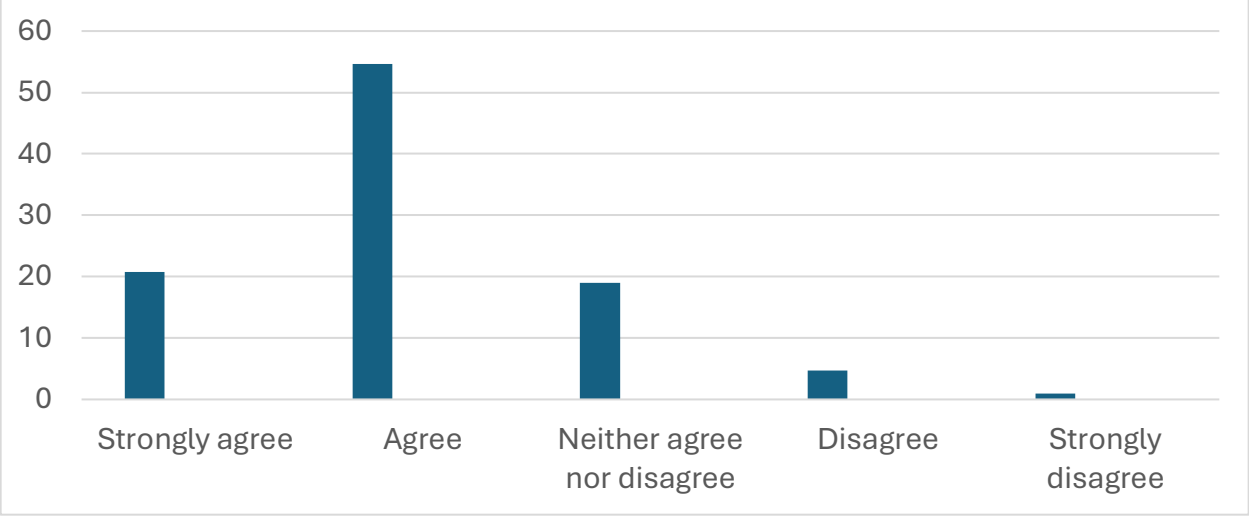
Supplementary Figure 6: "The risk of VTE is highest during the acute infection stage (0-30 days) but elevated excess risk potentially persists for up to one year 1 among patients hospitalised for COVID-19."[5,6]



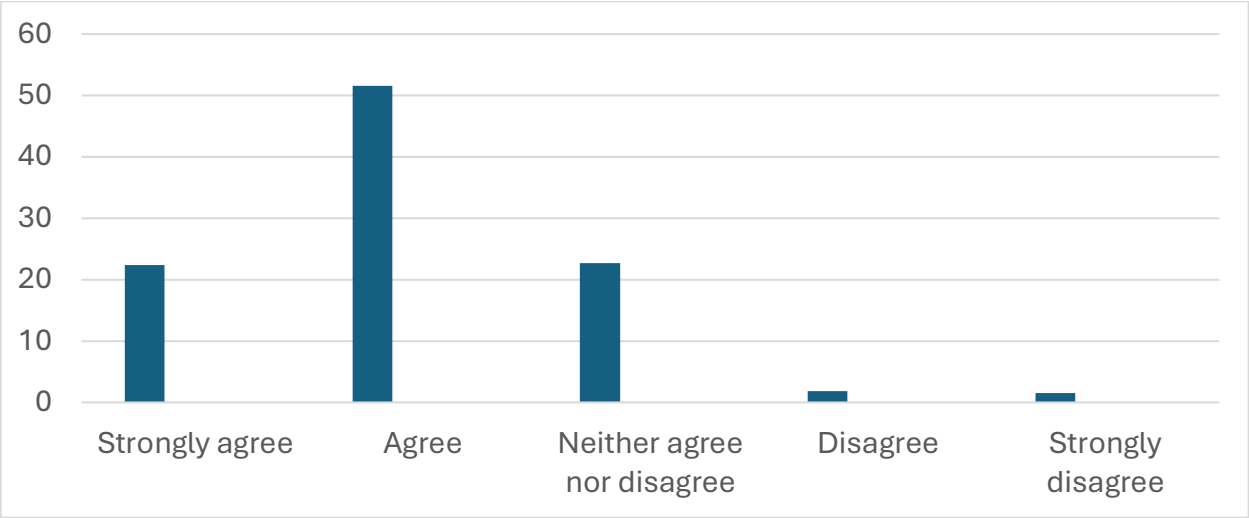
Supplementary Figure 7: "The increased risk of MI in patients with acute infections is more pronounced in patients with chest infections (viral and bacterial) and is proportional to time from infection and severity of illness."[6]



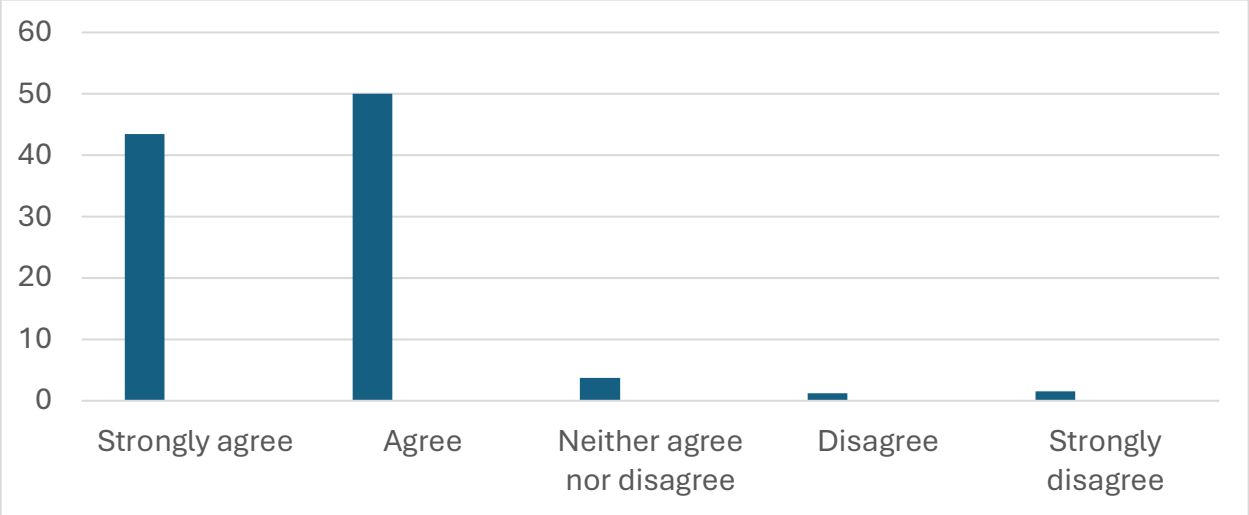
Supplementary Figure 8: "After COVID-19 recovery, adult patients have a two-fold increased risk of ischemic stroke compared to non-infected patients in the follow up period ranging between 4 and 18 months."[7]



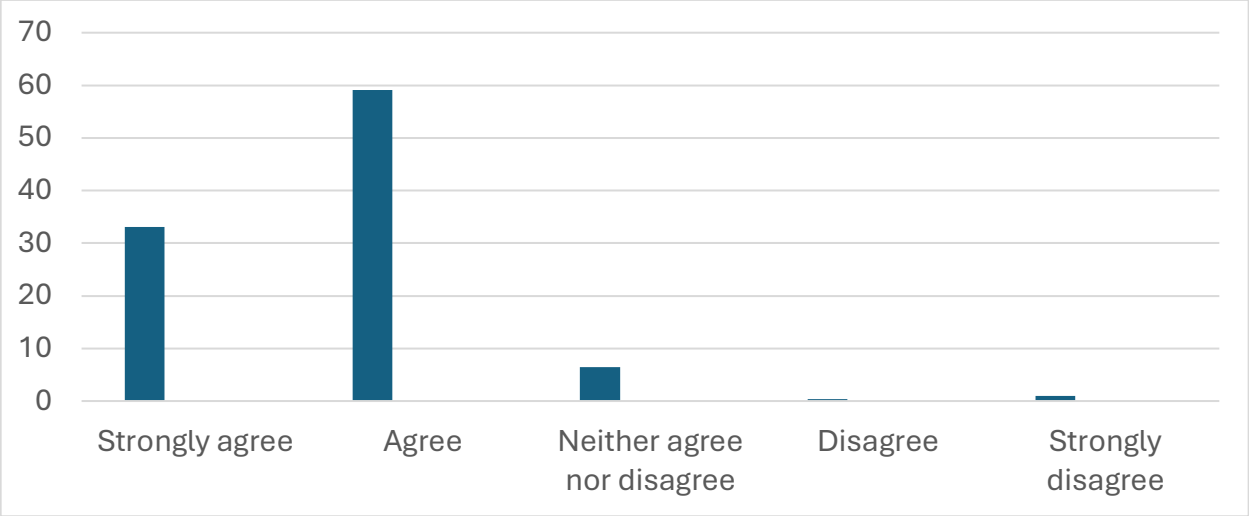
Supplementary Figure 9: "The risk for thrombotic complications of PCI with stent implantation is higher in patients infected with COVID-19 than those who are not."[8,9]



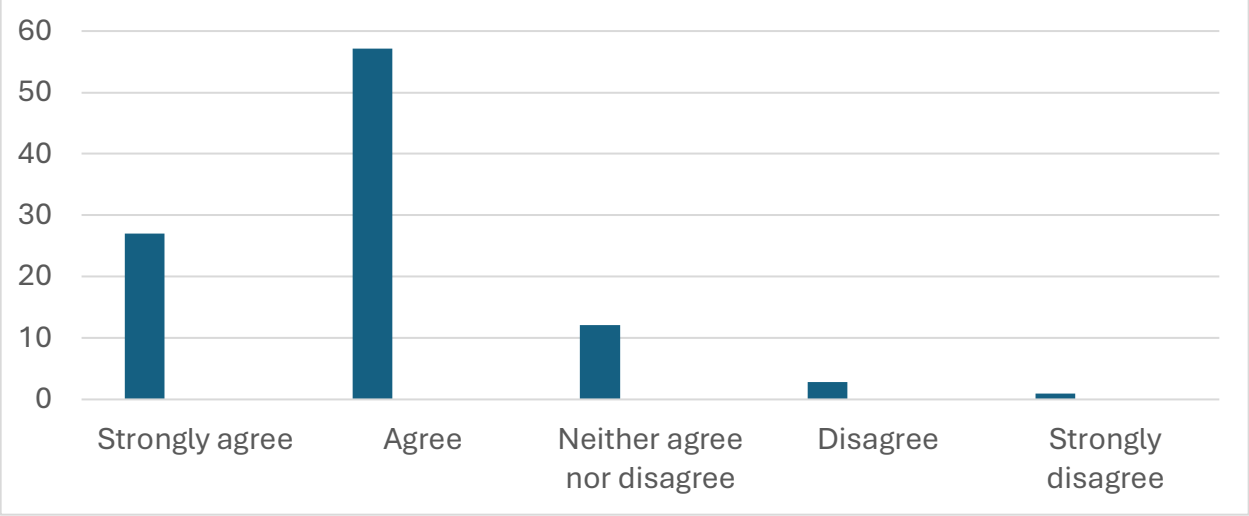
Supplementary Figure 10: "COVID-19 infection of greater severity is associated with greater risk and is related to the bidirectional relationship between the inflammatory process and thrombosis."[10–14]



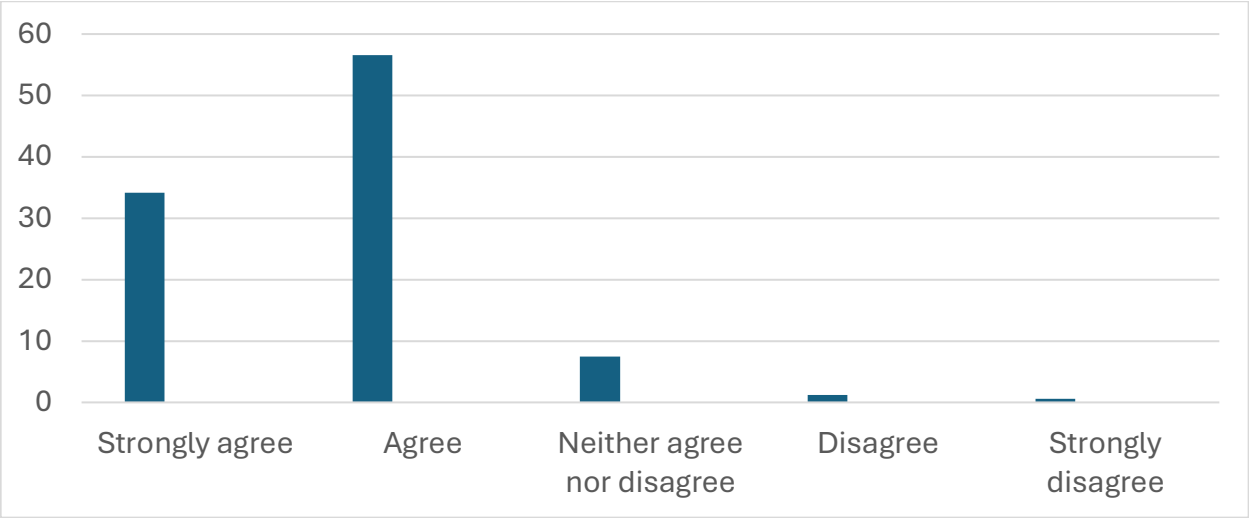
Supplementary Figure 11: "Studies emerging post pandemic that demonstrate the thrombotic risk associated with COVID-19 pose new challenges due to a multitude of factors including vaccination programs, new variants which may be less virulent and previous COVID-19 infection."[15]



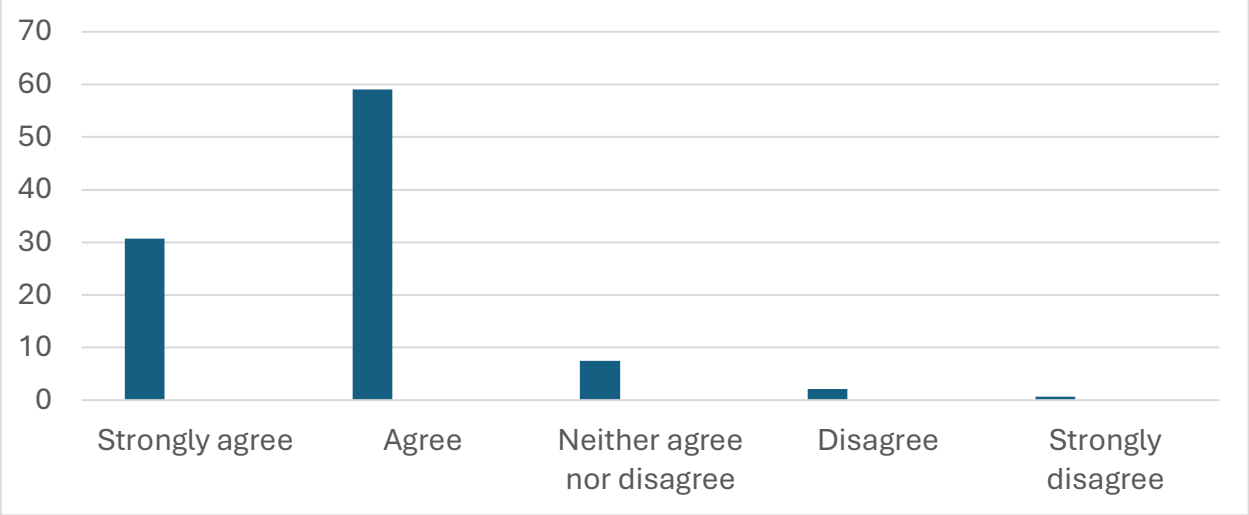
Supplementary Figure 12: "COVID-19 infection can aggravate underlying ACS and cause new-onset type 2 MI through hypercytokinaemia, direct cardiomyocyte injury, and dysregulation of the renin-angiotensin system."[16]



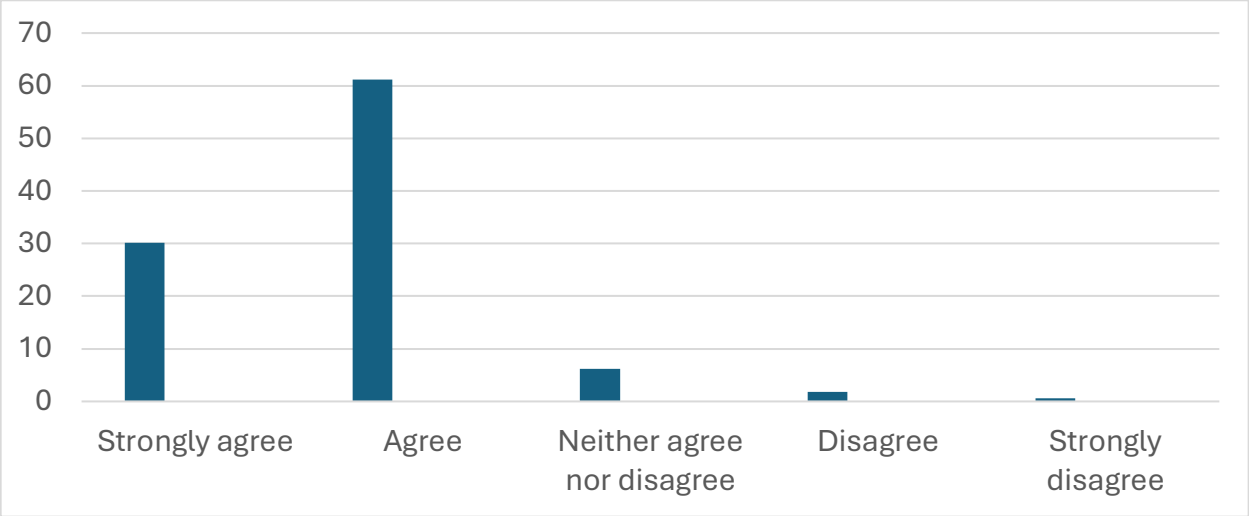
Supplementary Figure 13: "The clinical course and outcome in patients with acute MI are worse in patients co-infected with COVID-19 than those who are not."[16]



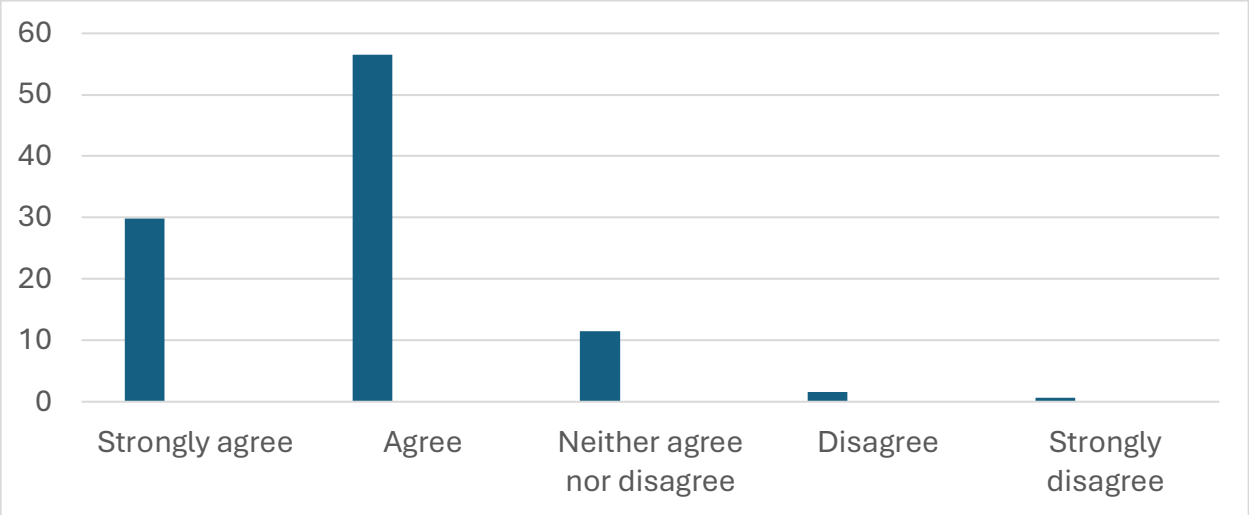
Supplementary Figure 14: "COVID-19 infection should be taken into consideration in CV risk algorithms and in particular during the acute period in patients requiring hospitalisation."



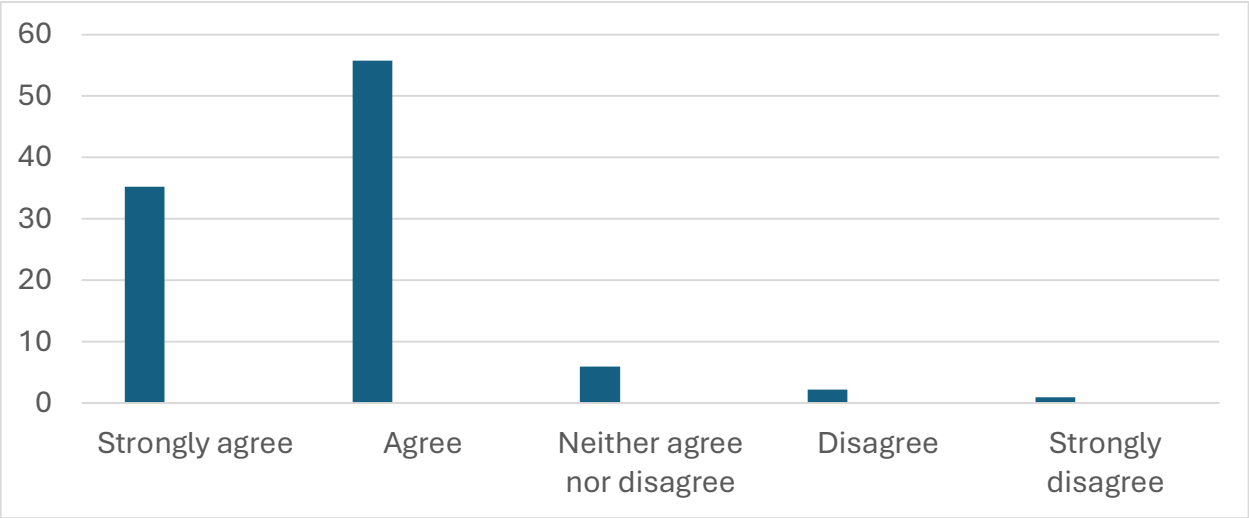
Supplementary Figure 15: "Care pathways for individuals with raised CV risk should consider the increased thrombotic risk associated with acute infection including COVID-19."



Supplementary Figure 16: "Individuals at raised CV risk with acute infective episodes including COVID-19 should undergo close monitoring during the month, and possibly during the year, following acute COVID-19 infection. Certain patient populations (e.g. those undergoing stent implantation) may benefit from close monitoring of their antiplatelet and antithrombotic therapy to carefully manage their thrombotic versus bleeding risk during this period after acute COVID-19 infection."



Supplementary Figure 17: "To understand the optimal antithrombotic regimen for prevention and management of thromboembolic events in patients with and acute infection including COVID-19, more high-quality evidence is needed to inform management strategies in these patients."[4]



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