Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

group allocation, potentially introducing bias. Lastly, it is important not to apply the findings to neonates or infusion sets used to administer chemotherapy, blood products, or lipids.

The trial provides additional learning points. The results emphasise previously reported risks inherent in use of three-way stopcocks.[12] The relatively low yield of removing central venous access devices and peripheral arterial catheters on clinical suspicion of infection is also highlighted; 9·8% and 0·14% of removed catheters, respectively, grew positive cultures. For central venous access devices, testing differential time to positivity of line and peripheral blood is an alternative diagnostic modality in stable patients.[13]

In a wider perspective, the RVSP trial should inspire clinicians to challenge other practices that require substantial resources or produce substantial waste. The non-inferiority design lends itself well to showing whether reductions in interventions are clinically safe.

I am a scientific adviser to 11 Health, a medical wearable technology company in Irvine, CA, USA, outside the area of work commented on here.

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Correlates of protection from SARS-CoV-2 infection

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Since the beginning of the COVID-19 pandemic, many scientists and public health officials assumed that infection with SARS-CoV-2 would protect from reinfection and that neutralising antibodies would correlate with protection or would be at least one of the protective immune mechanisms.[1] Early on, these assumptions were supported by non-human primate data showing protection from reinfection, a correlation between neutralising antibodies protection, and protection afforded by passive transfer of neutralising antibodies.[2–4] However, reports of rare reinfections, the notion that antibody titres might wane within weeks (which is incorrect), and the fact that human coronaviruses that cause common colds do cause reinfections have cast doubt on these initial assumptions.[5–7]

A study in the UK reported in *The Lancet* by Victoria Hall and colleagues,[8] called the SARS-CoV-2 Immunity and Reinfection Evaluation (SIREN) study, suggests that being seropositive to SARS-CoV-2 through natural infection protects robustly from asymptomatic and symptomatic reinfection. The study analysed data from 25 661 enrolled health-care workers between June 18, 2020, and Jan 11, 2021, including 8278 individuals with known previous SARS-CoV-2 infection, of whom the vast majority were antibody positive at enrolment and 17 383 individuals who were seronegative and had not previously been infected with SARS-CoV-2. 21 617 (84·2%) of 25 661 participants were women and 4010 (15·6%) were men, with a median age of 45·7 years. 87·3% of the participants were White, 6·9% were mixed race, 2·0% were Asian,

Adrian Dennis/Getty Images

1·6% were Black, 1·3% were Chinese, 0·6% were from other ethnic groups, and 0·2% preferred not to specify. Individuals were followed up with questionnaires (every 2 weeks), PCR for SARS-CoV-2 (every 2 weeks), and serology (at enrolment and every 4 weeks). 1704 infections occurred in the naive cohort, while two probable (required supportive serological data or supportive viral genomic data) and 153 possible (two positive PCR results 90 days apart or an antibody-positive individual with new positive PCR test 4 weeks after the first antibody positive test) infections occurred in the SARS-CoV-2-experienced cohort. Additionally, 864 individuals in the naive group seroconverted over the study interval but were not counted towards SARS-CoV-2 infections. The authors report previous SARS-CoV-2 infection provided a 84% risk reduction for reinfection (adjusted incidence rate ratio [aIRR] 0·159, 95% CI 0·13–0·19) and 93% risk reduction for those with symptomatic infections (aIRR 0·074, 0·06–0·10). Importantly, a variant of concern known as B.1.1.7 did circulate during the final part of the observation period, causing about 50% of all infections, but did not seem to have an effect on reinfection rates.

The findings of the authors suggest that infection and the development of an antibody response provides protection similar to or even better than currently used SARS-CoV-2 vaccines. Although antibodies induced by SARS-CoV-2 infection are more variable and often lower in titre than antibody responses induced after vaccination, this observation does make sense considering current SARS-CoV-2 vaccines induce systemic immune responses to spike proteins while natural infection also induces mucosal immune responses and immune responses against the many other open reading frames encoded by the approximately 29 900 nucleotides of SARS-CoV-2. The SIREN study adds to a growing number of studies, which demonstrate that infection does protect against reinfection, and probably in an antibody-dependent manner.[9–15]

The SIREN study does have several limitations. Different serological assay platforms were used to determine seropositivity and not all of them have the same sensitivity over time or focus on the spike of SARS-CoV-2, which is the prime target of neutralising antibody responses. Additionally, although protection against B.1.1.7 was shown, the degree to which infection with so-called garden variety SARS-CoV-2 provides protection against reinfection with antigenically distinct variants of concern such as B.1.351 and P.1 remains unclear. Furthermore, the authors did not link quantitative antibody measurements to protection. This is an important piece of the puzzle since the protective titre for SARS-CoV-2 is still unknown, although non-human primate studies suggest that it is likely to be low.[2,3] Establishment of antibody titres as a correlate of protection and defining a protective titre would be extremely important for public health considerations and for patient management. A correlate of protection and a protective threshold would also allow for the development of additional SARS-CoV-2 vaccines based on small immunogenicity-based phase 3 trials rather than large and costly field efficacy trials, which are becoming exceedingly difficult to perform. Determination of a protective titre should be a priority for future studies that investigate protection afforded by natural infection or vaccination.

I report that the Icahn School of Medicine at Mount Sinai has filed patent applications relating to SARS-CoV-2 serological assays and NDV-based SARS-CoV-2 vaccines on which I am named. I also report that I have previously published work on influenza virus vaccines with Sarah Gilbert, the lead investigator on the Oxford–AstraZeneca vaccine. I have consulted for CureVac, Merck, and Pfizer (before 2020); am currently consulting for Pfizer, Seqirus, and Avimex on SARS-CoV-2 and influenza virus vaccines; my laboratory is collaborating with Pfizer on animal models of SARS-CoV-2; my laboratory is collaborating with Norbert Pardi at the University of Pennsylvania on mRNA vaccines against SARS-CoV-2; my laboratory was previously working with GlaxoSmithKline on the development of influenza virus vaccines; and two of my mentees have recently joined Moderna.

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Guided $P2Y_{12}$ inhibitor therapy after percutaneous coronary intervention

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Antiplatelet treatment regimens for patients after percutaneous coronary intervention (PCI) have undergone major changes and substantial improvements. Extensions or a shortening of treatment as well as the use of various scores have been assessed in numerous trials to maximise anti-ischaemic efficacy and minimise bleeding.[1,2] The key limitation of these strategies is the inherent time delay in the implementation of any therapy adjustments. At the same time, several clinical trials have investigated tailored and guided antiplatelet treatment.[3] Unfortunately, the pioneering trials of tailored treatment did not meet their primary endpoints or show improvement in patients' outcomes.[4,5] Various explanations have been attributed to these disappointing results, including insufficient intensification of treatment in patients with low response to clopidogrel by limited use of potent $P2Y_{12}$ inhibitors and preferential inclusion of low-risk patients.[3] To address these issues, a series of recent trials included patients at higher risk and investigated the use of platelet function testing (phenotyping)[6] or genetic testing (genotyping)[7,8] as a strategy by including study groups with a clear distinction of testing versus no testing. Notably, these trials did not provide unequivocal results and were partly limited in sample size and sometimes underpowered to compare specific ischaemic and bleeding endpoints.

In *The Lancet*, Mattia Galli and colleagues[9] report the results of a systematic review and meta-analysis investigating the efficacy and safety of a guided antiplatelet treatment strategy by means of phenotyping or genotyping compared with standard and unguided treatment in patients undergoing PCI. Their analysis included 20 743 patients with chronic or acute coronary syndromes from 11 randomised controlled trials and three observational studies, with a mean follow-up of 11 months. Compared with standard therapy, the guided treatment approach was associated with a reduction in study-defined major adverse cardiovascular events (risk ratio [RR] 0·78, 95% CI 0·63–0·95). The subgroup of patients undergoing a guided de-escalation strategy (four of 14 studies) showed a reduction in bleeding compared with standard therapy (RR 0·81, 95% CI 0·68–0·96). Major limitations of the study include a lack of patient-level data and the inclusion of studies with cilostazol or double-dose clopidogrel treatment. Although overall results are encouraging and could provide a basis for more widespread adoption of guided antiplatelet treatment in everyday clinical practice, several hurdles need to be tackled in the future.

First, results of platelet function testing are subject to variability over time and across different available