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.

Antibody response to first BNT162b2 dose in previously SARS-CoV-2-infected individuals

Rapid vaccine-induced population immunity is a key global strategy to control COVID-19. Vaccination programmes must maximise early impact, particularly with accelerated spread of new variants.[1] Most vaccine platforms use a two-dose prime-boost approach to generate an immune response against the virus S1 spike protein, the titres of which correlate with functional virus neutralisation and increase with boosting.[2,3] To enable larger numbers of people to receive the first dose, delayed administration of the second dose has been advocated and implemented by some.[1] The impact of previous SARS-CoV-2 infection on the need for boosting is not known.

We reasoned that previous infection could be analogous to immune priming. As such, a first prime vaccine dose would effectively act as boost, so a second dose might not be needed. To test this, we undertook a nested case-control analysis of 51 participants of COVIDsortium,[4,5] an ongoing longitudinal observational study of health-care workers (HCWs) in London who underwent weekly PCR and quantitative serology testing from the day of the first UK lockdown on March 23, 2020, and for 16 weeks onwards. 24 of 51 HCWs had a previous laboratory-confirmed mild or asymptomatic SARS-CoV-2 infection, as confirmed by positive detection of antibodies against the SARS-CoV-2 nucleocapsid (Elecsys Anti-SARS-CoV-2 N ECLIA, Roche Diagnostics, Burgess Hill, UK) or the receptor binding domain of the SARS-CoV-2 S1 subunit of the spike protein (anti-S; Elecsys anti-SARS-CoV-2 spike ECLIA, Roche Diagnostics), whereas 27 HCWs remained seronegative. A median of 12·5 sampling timepoints per participant permitted the identification of peak antibody titres in seropositive individuals while avoiding false negatives.

All participants received their first dose of the BNT162b2 mRNA COVID-19 vaccine (Pfizer-BioNTech, Mainz, Germany)[2,3] and were tested 19–29 days later (median 22 days, IQR 2). Among previously uninfected, seronegative individuals, anti-S titres after one vaccine dose were comparable to peak anti-S titres in individuals with a previous natural infection who had not yet been vaccinated. Among those with a previous SARS-CoV-2 infection, vaccination increased anti-S titres more than 140-fold from peak pre-vaccine levels (figure). This increase appears to be at least one order of magnitude greater than reported after a conventional prime-boost vaccine strategy in previously uninfected individuals.[3]

These serological data suggest that for individuals receiving the BNT162b2 mRNA vaccine, a potential approach is to include serology testing at or before the time of first vaccination to prioritise use of booster doses for individuals with no previous infection. This could potentially accelerate vaccine rollout. With increasing variants (UK, South Africa, Brazil), wider coverage without compromising vaccine-induced immunity could help reduce variant emergence. Furthermore, reactogenicity after unnecessary boost risks an avoidable and unwelcome increase in vaccine hesitancy.

Whether enhanced vaccine-induced antibody responses among previously seropositive individuals will show differential longevity compared to boosted vaccines remains to be seen. In the meantime, our findings provide a rationale for serology-based vaccine dosing to maximise coverage and impact.

CM and ADO contributed equally. AS and JCM contributed equally. All authors' contributions are listed in the appendix. Funding details for this Correspondence are provided in the appendix. DMA and RJB have consulted as members of Global T cell Expert Consortium, Oxford Immunotec, UK. All other authors declare no competing interests. The corresponding author had full access to all data and had final responsibility for the decision to submit for publication.

*Charlotte Manisty, Ashley D Otter, Thomas A Treibel, Áine McKnight, Daniel M Altmann, Timothy Brooks, Mahdad Noursadeghi, *Rosemary J Boyton, Amanda Semper, James C Moon*

r.boyton@imperial.ac.uk

Institute of Cardiovascular Science (CM, TAT, JCM), Division of Infection and Immunity (MN), University College London, London, UK; Department of Cardiology, St Bartholomew's Hospital, Barts Health NHS Trust, London, UK (CM, TAT, JCM); National Infection Service, Public Health England, Porton Down, UK (ADO, TB, AS); The Blizard Institute, Queen Mary University of London School of Medicine and Dentistry, London, UK (AM); Department of Immunology and Inflammation (DMA); Department of Infectious Disease (RJB) Imperial College London, London W12 0NN, UK; Lung Division, Royal Brompton and Harefield Hospitals, London, UK (RJB)

Published **Online** February 25, 2021 https://doi.org/10.1016/S0140-6736(21)00501-8

See **Online** for appendix

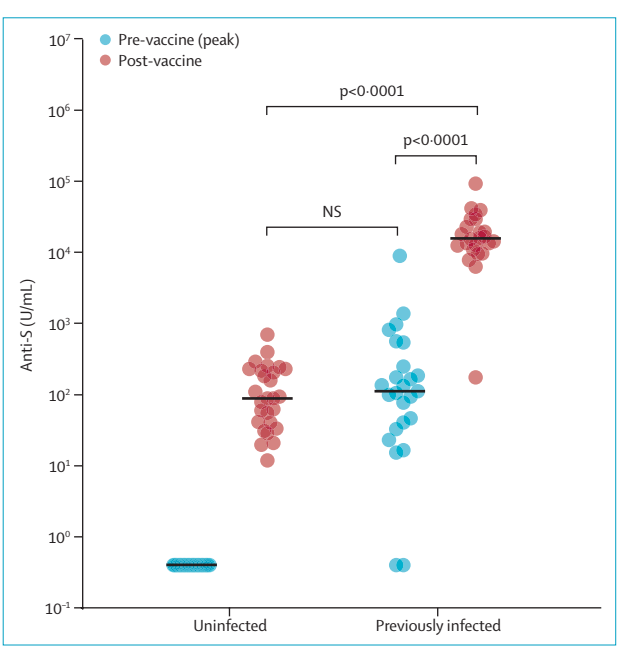

Figure: **Serological response to one dose of the BNT162b2 mRNA COVID-19 vaccine in individuals with and without laboratory-confirmed previous SARS-CoV-2 infection**

SARS-CoV-2 anti-S antibody titres in individuals with no previous infection are similar to titres in individuals who have had a mild SARS-CoV-2 infection. Anti-S titres in those with previous SARS-CoV-2 infection are more than 140-fold greater than at time of peak infection. Statistical analysis was by unpaired two-tailed *t* test. U=unit. NS=non-significant.

1 Joint Committee on Vaccination and Immunisation. Optimising the COVID-19 vaccination programme for maximum short-term impact. Jan 26, 2021. https://www.gov.uk/government/publications/prioritising-the-first-covid-19-vaccine-dose-jcvi-statement/optimising-the-covid-19-vaccination-programme-for-maximum-short-term-impact (accessed Jan 29, 2021).

2 Polack FP, Thomas SJ, Kitchin N, et al. Safety and efficacy of the BNT162b2 mRNA COVID-19 vaccine. *N Eng J Med* 2020; **383:** 2603–15.

3 Walsh EE, Frenck RW, Falsey AR, et al. Safety and immunogenicity of two RNA-based COVID-19 vaccine candidates. *N Engl J Med* 2020; **383:** 2439–50.

4 Treibel TA, Manisty C, Burton M, et al. COVID-19: PCR screening of asymptomatic health-care workers at London hospital. *Lancet* 2020; **395:** 1608–10.

5 Reynolds CJ, Swadling L, Gibbons JM, et al. Discordant neutralizing antibody and T cell responses in asymptomatic and mild SARS-CoV-2 infection. *Sci Immunol* 2020; **5:** eabf3698.

Undoing supremacy in global health will require more than decolonisation

I read with interest Seye Abimbola and Madhukar Pai's Perspective.[1] It provides an enlightening and hopeful vision of decolonised global health detangled from supremacy in its many forms. However, it left me feeling that the vast mark that colonisation has left on society, politics, and system hierarchy within low-income and middle-income countries (LMICs) has been less considered. Without paying due consideration to the challenges of supremacy and oppression within LMICs, we cannot realistically equalise global health and progress to ensure that it upholds health equity and social justice.

Globally, we observe how rich academics in high-income countries (HICs), particularly from the UK and USA, tend to get richer. For example, the ways in which global health funding and publication are dominated by prominent academics and high-income prestigious institutions mean that worthy work can be dismissed when teams are less valued. Importantly, many individuals from LMICs who are valuable in directing global health endeavours do not have the opportunities or training to prove why or how they are valuable in meaningful ways to academia. Under some circumstances, they can be actively oppressed.

There is a refusal to learn from local populations, especially those from the margins of society, and ethnic superiority exists within societal, political, and academic structures in both HICs and LMICs, which is rising amid right-wing conservatism in some settings. How do we effectively empower valuable leaders to push forward necessary global health measures when they are restricted from the outset?

Colonisation has left a pervasive mark. Its legacy in LMICs still needs to be unpicked. Creating truly equitable global health must involve diverse groups of people who view challenges through differing lenses from their backgrounds, lived experiences, and skills, and who have wider, inclusive visions that do not focus on individual career success and are not at the mercy of prescribed academic agendas in HICs.

I declare no competing interests.

Keerti Gedela
keertigedela@gmail.com

Chelsea and Westminster NHS Foundation Trust, London SW10 9NH, UK

1 Abimbola S, Pai M. Will global health survive its decolonisation? *Lancet* 2020; **396:** 1627–28.

Seye Abimbola and Madhukar Pai[1] describe eloquently how, for historical reasons, global health is operationalised as a saviourism model. To redress the balance of power between saviour and saved, they envision a utopic global health fuelled by respect and humility, and motivated by an adherence to values based on rights, equity, and justice.

Unfortunately, the disciplines that dominate global health attend to the causes of and solutions to disease endpoints on the health and wellbeing spectrum. Such disciplines have not engaged adequately with a crucial understanding of the sociostructural production of health or with the political arguments based on myriad values that fall outside of the traditional medical and health sciences. It is impossible to decolonise global health if crucial geopolitical analyses, and the impact on relationships between high-income countries (HICs) and low-income and middle-income countries (LMICs), remain chronically marginalised.

Additionally, decolonising global health extends beyond relations between LMICs and HICs; it is also about the relationships within them. Decolonisation is fundamentally about redressing inequity and power imbalance. It cannot be achieved without also addressing gender inequity, racism, and other forms of structural violence. The colonised also have to be at least as reflective about the status quo as the colonisers. This mindset goes beyond engagement and participation between HICs and LMICs, to disrupting the norms of dependency within LMICs that enable the inequities and replicate the hierarchies of neocolonialism. In real terms, LMICs must confront their own internal power relations inherent in the discourse of immutable culture, which protect cronyism, tribalism, poor governance, and patriarchy.

Ultimately, a decolonised global health can only exist within a broader geopolitical and economic environment that supports rights, equity, and justice.

We declare no competing interests.

**Pascale Allotey, Daniel D Reidpath*
pascale.allotey@unu.edu

International Institute for Global Health, United Nations University, Bandar Tun Razak, Kuala Lumpur 56000, Malaysia (PA); International Centre for Diarrhoeal Disease Research Bangladesh, Dhaka, Bangladesh (DDR)

1 Abimbola S, Pai M. Will global health survive its decolonisation? *Lancet* 2020; **396:** 1627–28.

Authors' reply

We thank Keerti Gedela as well as Pascale Allotey and Daniel Reidpath for their responses to our Perspective on decolonising global health.[1] We welcome and completely agree with the points they highlighted for additional emphasis: greater

Submissions should be made via our electronic submission system at http://ees.elsevier.com/thelancet/