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.

instrument-dependent, and of limited deployability. We agree with Larremore and colleagues[5] that test sensitivity is secondary to frequency of screening and time to result.

Finally, the excellent negative predictive value of Ag-RDTs is a mathematical computation of probability based on test sensitivity and prevalence of infection in the population. It is not misleading as long as one is clear about the purpose of testing and how the sensitivity of a test is derived.

See Online for appendix

We declare no competing interests.

*Rosanna W Peeling, Piero Olliaro, Debrah Boeras, Noah Fongwen
rosanna.peeling@lshtm.ac.uk

London school of Hygiene & Tropical Medicine, London, UK (RWP, NF); Centre for Tropical Medicine and Global Health, University of Oxford, Oxford, UK (PO); Global Health Impact Group, Atlanta, GA, USA (DB)

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SARS-CoV-2 B.1.1.7 and B.1.351 spike variants bind human ACE2 with increased affinity

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Genomic surveillance efforts have uncovered SARS-CoV-2 variants with mutations in the viral spike glycoprotein, which binds the human angiotensin-converting enzyme 2 (ACE2) receptor to facilitate viral entry.[1] Such variants represent a public health challenge during the COVID-19 pandemic because they increase viral transmission and disease severity.[2] The B.1.351 variant, first identified in South Africa, has three notable mutations in the spike receptor-binding domain (RBD)—namely, K417N, E484K, and N501Y[3]—whereas the B.1.1.7 variant, first identified in the UK, carries the N501Y mutation (appendix pp 2–4). B.1.351 is of particular concern for its potential resistance to antibodies elicited by previous SARS-CoV-2 infection and vaccination.[4]

Several mechanisms might account for increased variant transmissibility, such as increased spike protein density, greater furin cleavage accessibility, and enhanced spike protein binding affinity for the ACE2 receptor.[5] To test whether the B.1.351 and B.1.1.7 variants bind ACE2 with increased affinity, binding of purified recombinant B.1.351 and B.1.1.7 RBD was compared with binding of the Hu-1 RBD, which was originally identified in Wuhan (SCoV2) using microscale thermophoresis. The B.1.1.7 RBD bound ACE2 with 1·98-times greater affinity than the SCoV2 RBD (mean equilibrium dissociation constant [Kd] 203·7 nM [SD 57·1] *vs* 402·5 nM [112·1]; p=0·0521; appendix p 5). The B.1.351 RBD bound ACE2 with 4·62-times greater affinity than the SCoV2 RBD (mean Kd 87·6 nM [SD 25·5] *vs* 402·5 nM [112·1]; p=0·0009; appendix p 5). These data are consistent with a model in which variant spike proteins mediate increased transmissibility of the B.1.1.7 and B.1.351 variants, at least in part, by enhancing ACE2 binding affinity in line with in-silico predictions.[6]

In the initial stage of infection, virions bind lung airway epithelial cells with kinetics governed in part by spike–ACE2 binding affinities. Enhanced affinity likely mediates increased infectivity by lowering the effective concentration of virions required for cell entry and is a convergent feature in more transmissible SARS-CoV-2 variants arising in multiple geographical regions. Finally, characterisation of binding between variant spike proteins and cognate human receptor ACE2 on the basis of microscale thermophoresis represents a potential surveillance strategy for predicting enhanced transmissibility of emerging SARS-CoV-2 variants harbouring novel spike mutations.

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*Muthukumar Ramanathan†, Ian D Ferguson†, Weili Miao†, *Paul A Khavari
khavari@stanford.edu

†Contributed equally

Department of Pathology (MR) and Program in Epithelial Biology (IDF, WM, PAK), Stanford University School of Medicine, Stanford, CA 94305, USA

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What chikungunya teaches us about COVID-19

Since December, 2019, the COVID-19 pandemic has generated huge challenges, numerous breakthroughs in research and care, and innovations in public health. 1 year after its emergence, the world is discovering

that late clinical consequences of the disease are an increasing problem for patients.

For a substantial proportion of SARS-CoV-2-infected adults, the disease appears biphasic, with long-lasting clinical disorders (so-called long COVID).[1] This post-COVID status might comprise multiple mental or somatic disorders, especially among adults. The prevalence, profile, and determinants of these chronic manifestations remain to be described and understood for appropriate case management. Assessment of clinical disability, quality of life, and fitness for work is required to provide a better view of the real burden of disease and improve our response to the pandemic.

There is a cognitive bias when examining the COVID-19 burden. Considering its mode of transmission (airborne), its clinical influenza-like presentation, and the explosive shape of the epidemic curves, the main comparison for COVID-19 in human history is the influenza pandemic of 1918. Most political and public health leaders are reasoning with the image of influenza in mind. However, by contrast with COVID-19, influenza has no long-term clinical burden.

An acute viral infection with chronic sequelae is not unprecedented. Following our experience with post-chikungunya status, we were struck by the similarities between the long-lasting manifestations after COVID-19 and those after chikungunya virus infection, especially for general and mental disorders, impaired quality of life, and medico-social consequences. Post-chikungunya consequences might account for about 70% of disability-adjusted life-years following a chikungunya outbreak.[2,3] It took about 10 years to describe the post-chikungunya disorders and propose guidelines (still not evidenced-based).[4] Despite multi-continental outbreaks, there are still fewer than five well designed randomised-controlled trials, and no management strategy exists for patients who have been suffering post-chikungunya consequences for years. One reason for this neglect of the chronicity of chikungunya is a tendency to consider it a simple, short-lived infection like dengue.

A simple comparison of the global burdens of COVID-19, influenza, chikunguna, and dengue fever distinguishes roughly two biphasic infections (chikungunya and COVID-19) and two monophasic ones (influenza and dengue). In a sense, COVID-19 is much closer to chikungunya than to influenza or dengue. We support calls for urgent research on long COVID to avoid having millions of adults with long COVID left behind, with an inestimable social and economic impact. The lessons from post-chikungunya should be learnt.[5]

FS reports being the chief executive officer of RISK&VIR, on a data and safety monitoring board for Valneva, and a senior consultant on chikungunya to PAHO/WHO. HW reports shares in Sanofi. All other authors declare no competing interests.

*Fabrice Simon, Hugh Watson, Jean-Baptiste Meynard, Vincent Pommier de Santi, Jean-Nicolas Tournier
simon-f@wanadoo.fr

Hôpital d'Instruction des Armées Laveran, Service de Pathologie Infectieuse et Tropicale, Marseille, France (FS); Unité des Virus Emergents U190, INSERM-IRD-Aix Marseille Université, Marseille, France (FS); Evotec ID, Virology Department, Lyon, France (HW); Departments of Clinical Pharmacology, Hepatology and Gastroenterology, Aarhus University, Aarhus, Denmark (HW); Centre d'Épidémiologie et de Santé Publique des Armées, Marseille, France (J-BM, VPdS); Ecole du Val-de-Grâce, Paris, France (J-BM, J-NT); Institut de Recherche Biomédicale des Armées, Microbiology and Infectious Diseases Department, Brétigny-sur-Orge, France (J-NT); CNRS UMR-3569, Innovative Vaccine Laboratory, Virology Department, Institut Pasteur, Paris, France (J-NT)

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Serum neutralising activity against SARS-CoV-2 variants elicited by CoronaVac

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Emergence of multiple SARS-CoV-2 variants of concern (VOCs) harbouring mutations in the spike protein—the major target of neutralising antibodies—has raised serious concerns about the potential for reduced protective efficacy of humoral responses elicited by vaccination.[1–4] CoronaVac (Sinovac Biotech, Beijing, China) is an inactivated vaccine that has been authorised for conditional mass use against COVID-19 in China and was efficacious in preventing symptomatic and severe disease in a phase 3 trial.[5]

To evaluate the potential resistance of new variants to neutralisation elicited by CoronaVac, sera from 93 healthy health-care professionals from Nanjing Drum Tower Hospital, a tertiary hospital in Nanjing, China, who received two doses of CoronaVac were obtained before the first dose and at day 14 after two doses. We assayed their neutralisation activity against lentivirus-based SARS-CoV-2 pseudotyped viruses containing the spike protein of the Wuhan-1 reference strain (wildtype), as well as six circulating variants,[2] including D614G, B.1.1.7 (first identified in the UK), B.1.351 (first identified in South Africa), P.1 (first identified in Brazil), B.1.429 (first identified in California, USA), and B.1.526 (first identified in New York, NY, USA).

All pre-vaccine sera showed undetectable levels of neutralisation against the seven SARS-CoV-2 variants tested. After two-dose inoculation, 76 (82%; geometric mean titre [GMT] 51·3) serum samples were capable of neutralising wildtype pseudovirus. Compared with