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.

authors did additional subgroup analysis that revealed fIPV schedules with a later-in-life first dose and longer interval between two successive doses had higher immunogenicity. A limitation to the analysis is that the duration of humoral immunity and the induction of mucosal immunity is not asssesed, which may differ between fractional and full-dose IPV.

Looking forward, the importance and global demand for fractional and full-dose IPV to global polio eradication will only increase. As an immediate next step, two-dose IPV immunisation schedules are being considered in countries that currently use one IPV dose. After eradication of wild poliovirus is achieved, all countries will withdraw OPV, and give IPV-only vaccination. These schedules will likely need to include fIPV as an option and, therefore, the data summarised by Mashunye and colleagues for two and three dose IPV schedules will provide an important evidence base for policy decisions.

Several barriers still exist to wider adoption of fIPV: the programmatic challenges of intradermal administration (although eased through the development and use of intradermal devices and injectors), IPV vial sizes, and off-label use.[6,7] Given the challenges of intradermal administration, a study done in infants in Cuba tested intramuscular administration of fIPV and found non-inferior seroconversion rates for all three polio serotypes for intramuscular fIPV compared with intradermal fIPV after two doses, given at 4 and 8 months of age.[9] If the results are replicated in other settings and age groups, this could provide an alternative delivery method to increase accessibility of fIPV.

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Long COVID has exposed medicine's blind-spot

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On Feb 23, 2021, the National Institutes of Health announced a new US$1·15 billion initiative to support research and resources for so-called long COVID.[1] This is the culmination of a year that has seen more scientific attention, public commentary, and media coverage of chronic unexplained medical symptoms (either post-infectious or not) than arguably the past decade combined. Indeed, one of the most concerning stories emerging out of the COVID-19 pandemic is the quandary of long COVID. Long COVID, or post-acute sequelae of SARS-CoV-2 infection, is being seen in a growing number of patients reporting a constellation of symptoms after SARS-CoV-2 infection that are persistent, debilitating, and have yet to be fully explained by known or measurable mechanisms. These symptoms include fatigue, cognitive difficulties, mood dysregulation, headaches, insomnia, dizziness, and a variety of other neurological, neuropsychiatric, autonomic, and systemic symptoms.[2] These symptoms are being reported by patients even with mild initial infection that did not require hospitalisation or medical attention. Many physicians have reported having such symptoms post-COVID-19, which has added support to initial pleas that long-COVID symptoms exist and can be debilitating.[3] Media organisations across the world have highlighted the complexity of this topic with intrigue and concern. This attention and tone offers a relatively stark contrast to the cynicism that usually plagues chronic, unexplained symptoms and a history of patients who feel that they have been ignored by medicine.[4]

As many others have pointed out, the clusters of symptoms reported by patients post-COVID-19 are not unique or specific to long COVID. Patients with similar assortments of chronic symptoms are commonly encountered in neurology, rheumatology, infectious diseases, and other subspecialty clinics. Some patients will have similar post-infectious onsets, whereas others report other potential triggers, and, for some, there are no identifiable triggers at all. Unfortunately, for most of these symptoms, there are no validated objective biomarkers to aid in diagnosis or to quantifiably measure an abnormal structural state. Indeed, disruptions in brain and brain–body function that probably account for such symptoms cannot yet be reliably identified by conventional blood tests or brain scans. Thus, a common denominator in this field is medical consultations largely based on diagnostic exclusion, in which the absence of further answers or direction for recovery can leave patients feeling dismissed and dissatisfied.[4]

Two broad possibilities exist to explain where long COVID might fit in this complex and controversial field. First, COVID-19 could trigger post-infectious processes that generate persisting symptoms in a unique way that is distinct from previously encountered patients. Although this would traditionally defy guiding principles such as Occam's razor (favouring simple, unifying explanations), we cannot ignore SARS-CoV-2's many firsts, its use of the ACE2 receptor (similar to SARS-CoV), and the particularly aggressive interactions that have been observed with the brain, other organs, and blood vessels in some patients.[5] Second, long COVID might exemplify the category of mysterious, unexplained, chronic symptoms (either post-infectious or not), and could operate via similar mechanisms to symptoms seen in other patients. The major problem in teasing this out is that the latter, despite a long history of high numbers of patients, has remained very poorly understood and constitutes one of medicine's largest blind-spots. The collective vacuum of uncertainty has opened the floodgates to many different disease notions, diagnostic labels, and conflicting explanations from purely so-called physiological to purely so-called psychological. Starting with physiological explanations, some models present inflammatory or immune-mediated cascades that might place primary importance on a given trigger (eg, the nature of an infection and its interaction with the host). Whereas traditional psychological theories heavily weight psychological factors and the potential overlap between these constellations of symptoms and bodily manifestations of stress responses and anxiety. Alternatively, contemporary neuropsychiatry models present this polarisation as a false dichotomy and highlight the potential importance of predisposing factors, including genetic and psychosocial factors, that might result in dysfunction of brain or brain–body circuits and networks that then interact with a potential triggering event.

Every proposed explanation on this topic has gaps and they are not necessarily mutually exclusive hypotheses. There might also be substantial heterogeneity between patients, with subpopulations at different points along this spectrum. How the brain connects within itself and to the body via complex neural, neurohormonal, and neuroimmune axes is one of the final frontiers of science and medicine.[6] We still do not fully understand how these interactions occur under normal circumstances so how can we fully understand when they go wrong? Unfortunately, instead of humbly embracing the complexity of these interactions and encouraging collaboration, contrasting opinions are often ferociously defended, creating deep divisions.

For now, the most important thing is to study long COVID with no assumptions and to interrogate potential unique factors about COVID-19 that could explain why these symptoms seem to be triggered with particularly high propensity. The COVID-19 pandemic and the large (and growing) number of patients with long-term symptoms offers an unprecedented window to study these symptoms, their inter-relationships, and their puzzling pathogenesis. Attention is finally being paid to this important topic, and even if this line of research does not lead to definitive answers, we are confident that there will be valuable new insights for this field. We should not care about what ends up being right or more right, wrong or more wrong; we should care about getting closer to reliable, objective markers of these complex symptoms and easing the suffering of these oft-neglected patient populations.

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Cameroon's bold response to the COVID-19 pandemic during the first and second waves

In the early phase of the COVID-19 pandemic, Cameroon was among the top five countries in sub-Saharan Africa and the first in central Africa in terms of number of confirmed cases. From the beginning, Cameroon faced the COVID-19 pandemic with the objectives of reducing viral transmission in the community, limiting the number of deaths, and lessening the socioeconomic impact of COVID-19. The Cameroonian government has made calculated decisions, including contextualised mitigation measures, a bold testing strategy incorporating rapid diagnostic tests, treatment of patients with COVID-19 exclusively in specialised treatment centres, re-opening schools during the peak of the pandemic, and integrating mental health care into the national response.[1]

The first two cases of COVID-19 in Cameroon were confirmed on March 5, 2020. The first case was imported from France and the second was one of their close contacts in Cameroon. The number of cases increased rapidly, after first being imported from western Europe mostly before community transmission of the disease was confirmed in late April 2020.[2] In February 2020, the Ministry of Public Health drew up a preparedness and response plan for COVID-19 to quickly detect possible cases of importation of the disease and limit its spread in Cameroon. The incident-management system was therefore activated at the Public Health Emergencies Operations Centre on March 6, 2020, the day after the first cases were confirmed in Yaoundé.[2] Based on confirmed case counts and PCR positivity rates, Cameroon had a first COVID-19 peak at the end of June, 2020, and a second peak in April, 2021. In Cameroon, the mean age of people with COVID-19 is 38 years, and 53·1% are men.[3] For fatal cases, the average age at death is 58 years, and nearly 20% of these individuals have comorbidities. As of May 12, 2021, 74 946 people have had COVID-19 and 1152 have died, a mortality of 1·5%.[2]

One clear objective of the response was to detect as many cases as possible given the scarce testing resources. Despite 15 PCR-capable diagnostic laboratories being implemented in nine of ten Cameroonian geographical regions by April, 2020, it was clear that many cases were being missed.[4] Therefore, the Cameroon Ministry of Health took the bold decision to incorporate and evaluate rapid diagnostic tests for SARS-CoV-2 antigens and serology.[5] The national algorithm was validated by the Scientific Council of Public Health Emergencies in June, 2020. 629 090 people were tested by antigenic rapid tests between June 6, 2020, and Dec 31, 2020, in selected testing sites located in markets, schools, universities, administrative offices, and businesses. 43 261 (57·9%) of the 74 733 COVID-19-positive cases reported by the Ministry of Public Health were diagnosed using rapid tests.[2] In addition, the government established specialised COVID-19 care centres in regions with community transmission, thus relieving congestion in public health facilities and reducing the stigma associated with hospitals that were treating patients with COVID-19. To further limit the transmission of SARS-CoV-2 in the community, on March 18, 2020, the government implemented 19 measures, including the closure of all borders, the closure of bars and restaurants after 18:00 h, the limitation of people in public transport, the closure of schools and universities, and the compulsory wearing