Long COVID in people living with HIV

Michael J. Peluso[a] *and Annukka A.R. Antar*[b]

Purpose of review
It is now recognized that SARS-CoV-2 infection can have a long-term impact on health. This review summarizes the current state of knowledge regarding Long COVID in people living with HIV (PLWH).

Recent findings
PLWH may be at elevated risk of experiencing Long COVID. Although the mechanisms contributing to Long COVID are incompletely understood, there are several demographic and clinical factors that might make PLWH vulnerable to developing Long COVID.

Summary
PLWH should be aware that new or worsening symptoms following SARS-CoV-2 infection might represent Long COVID. HIV providers should be aware of this clinical entity and be mindful that their patients recovering from SARS-CoV-2 infection may be at higher risk.

Keywords
coronavirus disease 2019, HIV, Long coronavirus disease, postacute sequelae of severe acute respiratory syndrome coronavirus-2 infection, severe acute respiratory syndrome coronavirus-2

INTRODUCTION

In the spring of 2020, shortly after the emergence of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), individuals began to report prolonged symptoms or unexpected changes in health after coronavirus disease 2019 (COVID-19) [1]. The number, diversity, and duration of symptoms was unprecedented, and the presence of this syndrome in people who had what was considered to be non-severe acute illness (i.e. not requiring hospitalization) was unexpected. Patient anecdotes were reported widely in social and popular media, and a group of patient-researchers began conducting and publishing large internet-based surveys in the first attempts to characterize the syndrome. The medical research community followed their lead and the first peer-reviewed report of Long COVID was published in July 2020 [2]. After months of intense advocacy on the part of people experiencing this condition, the US Congress passed a more than $1 billion initiative to study Long COVID [3] and the Biden administration has also made commitments to these efforts [4]; similar programs have been developed in other settings [5,6]. Since that time, major efforts have been made to understand the epidemiology, natural history, biological mechanisms, and impact of Long COVID.

Long COVID, the term now favored by US research agencies [7], is one of several terms currently used to describe health problems that occur after COVID-19 [8]. Other names for the condition include post-COVID conditions, long-haul COVID, chronic COVID, post COVID-19 syndrome, and postacute sequelae of SARS CoV-2 infection (PASC). Ultimately, each of these serve as umbrella terms encompassing a range of conditions in the postacute period, ranging from established sequelae of severe COVID-19 (e.g. post-ICU and posthospitalization syndromes), incident medical diagnoses (e.g. pulmonary embolism, myocardial infarction, stroke, diabetes mellitus), mental health conditions (e.g. depression, anxiety), social conditions (e.g. isolation), as well as symptoms not attributable to another cause. The latter is most representative of Long COVID as described by individuals experiencing the condition.

In this review, we summarize the current state of knowledge regarding Long COVID in people living with HIV (PLWH). Although current data on the

[a]Division of HIV, Infectious Diseases, and Global Medicine, University of California, San Francisco, California and [b]Division of Infectious Diseases, The Johns Hopkins University School of Medicine, Baltimore, Maryland, USA

Correspondence to Michael J. Peluso, MD, Division of HIV, Infectious Diseases, and Global Medicine, University of California, San Francisco, San Francisco, CA 94110, USA. Tel: +1 415 476 4082 x119; e-mail: michael.peluso@ucsf.edu

Curr Opin HIV AIDS 2023, 18:126–134

DOI:10.1097/COH.0000000000000789

KEY POINTS

- Some people do not return to their baseline state of health and develop additional complications following SARS-CoV-2 infection.
- Although data are limited, several studies have reported an increased prevalence of Long COVID symptoms and other postacute sequelae of SARS-CoV-2 infection among PLWH compared with HIV-seronegative individuals.
- The risk factors for and pathophysiology of Long COVID remain incompletely understood.
- There are a number of reasons why PLWH might be vulnerable to developing Long COVID, including sociodemographic factors, medical comorbidities, and dysregulation of immunologic and/or physiologic systems that could be exacerbated by acute and postacute SARS-CoV-2 infection.
- HIV providers should assess patients for complications of SARS-CoV-2 infection when encountering them during the postacute period.

mechanisms of Long COVID are limited, we describe why PLWH might be vulnerable to developing this condition.

EPIDEMIOLOGY AND NATURAL HISTORY OF LONG COVID

Cohort studies, epidemiologic studies and systematic reviews suggest that up to 70% of individuals experience some form of Long COVID following SARS-CoV-2 infection, with smaller proportions (5–10%) experiencing severe, debilitating forms of this condition [9–13]. Prevalence estimates vary widely due to differences in case definition, which include differences in how Long COVID symptoms are ascertained and defined, particularly with regard to the inclusion of special populations (e.g. hospitalized individuals), the follow-up period (e.g. weeks or months), and the handling of symptoms that may have predated COVID-19 (e.g. exclusion of these symptoms or inadequate information to exclude them). In the United States, data from the Veterans Affairs (VA) Healthcare System has been instrumental in understanding the epidemiology of Long COVID in veterans [14–17], although a number of high-quality community-based studies have been undertaken [2,18–23]. Outside of the United States, there have been several notable efforts in the United Kingdom, Europe, Africa, and Asia [24–32]. Although the epidemiology has never been precisely understood and is potentially affected by the emergence of new variants [33], preexisting vaccine-related and infection-related immunity [16,34], and the availability of therapeutics for acute infection [17,35], it is now accepted that even if the lower range of the prevalence estimate is accurate, the absolute number of people affected is large because of the scale of the pandemic. As a result, the economic, social, and healthcare impact is likely to be substantial [36].

Many initial studies of Long COVID focused on its natural history [13,14,37–40]. The most inclusive studies of Long COVID have identified dozens of symptoms associated with this condition [41,42], although it is unlikely that all symptoms experienced by a person following COVID-19 are directly attributable to SARS-CoV-2 infection or its sequelae. For this reason, separating related from unrelated symptoms is a major challenge in Long COVID research and clinical care [38]. In addition, Long COVID is characterized by substantial within-individual variability [38], with the waxing and waning of symptoms over time in some individuals complicating measurement. Clinical trajectories are highly variable, with some individuals improving over time, others plateauing, and some occasionally worsening (although substantial worsening is felt to be rare); many cases persist for months or even years. Finally, there is increasing acceptance that Long COVID is likely to constitute multiple syndromes or symptom clusters [39,40,43,44], including cardiopulmonary, neurocognitive, gastrointestinal, and musculoskeletal phenotypes. Some of these may in addition overlap with other postinfectious syndromes such as: myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS), postural orthostatic tachycardia syndrome (POTS), mast cell activation syndrome (MCAS), and postexertional malaise syndromes (PEM). Defining these phenotypes is of increasing importance as it may be the case that different pathophysiologic mechanisms drive different phenotypes. Although they continue to be identified, there are now several well established risk factors for Long COVID [14,18,26,45]. These include female sex, older and middle age, lower socioeconomic status and access to healthcare, and the presence of certain comorbid medical conditions including diabetes and obesity. Preexisting anxiety and depressive symptoms have also been associated with the development of Long COVID in some but not all studies [46,47] and mood symptoms may co-occur with physical symptoms in some individuals experiencing this condition.

LONG COVID IN PEOPLE LIVING WITH HIV

PLWH represent a specific group of interest and may be at increased risk for both post-COVID

complications as well as Long COVID. However, the data thus far are extremely limited. The presence of some PLWH were reported in early Long COVID cohorts but they were not characterized in detail [38]. Instead, the first in-depth descriptions of Long COVID in PLWH were case series. One series of 94 PLWH in Western India who were unvaccinated at the time of COVID-19 reported that 44% of individuals had at least one Long COVID symptom 30 or more days postinfection [48]. Cough and fatigue were the most commonly reported symptoms. This cohort was composed primarily of middle-aged men (median age 51, 73% men) and most (91%) were on suppressive antiretroviral therapy (ART). In a similar case series from an HIV clinic in Padua, Italy, 75 PLWH on suppressive ART who were unvaccinated at the time of symptomatic COVID-19 were followed for a median time of 6 months [49]. The cohort was predominantly constituted of middle-aged men (>80% men), with good immune reconstitution (median CD4+ T-cell count >500 cells/μl). One or more COVID-attributable symptoms or sequelae 4 or more weeks after symptom onset were reported in 27% of individuals in this study. The most common symptoms were fatigue, dyspnea on exertion, and recurrent headache. In both of these case series, those who had Long COVID symptoms were more likely to previously have had moderate-to-severe COVID-19.

Several attempts have also been made to determine whether PLWH have an increased likelihood of Long COVID compared with HIV-seronegative people. An analysis of 530 individuals who had presented to the emergency department or were hospitalized at NY Presbyterian Hospital-Cornell University in the spring of 2020 demonstrated that PLWH were more likely to report one or more Long COVID symptoms 12 months after COVID-19, after adjusting for age, sex, race, poverty, comorbidities, and severity of acute COVID-19 [adjusted risk ratio 1.75, 95% confidence interval (CI) 1.14–2.69] [50]. However, the cohort included only 10 PLWH (<2% of the total population studied).

Two more recent studies in the University of California, San Francisco-based Long-term Impact of Infection with Novel Coronavirus (LIINC) cohort built upon these findings. In the first [51▪], 39 unvaccinated PLWH were compared with 43 unvaccinated HIV-seronegative individuals. Following adjustment for age, sex, hospitalization status, and time since infection, the authors found that PLWH had 4.01× the odds of reporting at least one COVID-attributed symptom and 2.72× the odds of reporting three or more COVID-attributed symptoms at a median of 4 months post-COVID. Certain symptoms, including fatigue, gastrointestinal symptoms, neurocognitive symptoms, and sleep problems were reported more commonly in PLWH. The authors identified lower levels of SARS-CoV-2-specific memory CD8+ T cells and higher levels of PD1+ SARS-CoV-2-specific CD4+ T cells among PLWH but did not find significant differences according to PASC status. They also found that interleukin-6 (IL-6) and IP-10 had a modest association with Long COVID overall, and that IP-10 levels in particular were associated with Long COVID symptoms in PLWH. A second study from this cohort [52▪] evaluated 54 PLWH as part of an analysis of 280 individuals with prior SARS-CoV-2 infection to assess the contribution of chronic viral infections such as Epstein–Barr virus (EBV), cytomegalovirus (CMV), and HIV. It found using adjusted models that PLWH had increased odds of neurocognitive Long COVID symptoms at 4 months, when controlling for demographic factors, serologic evidence suggesting recent EBV reactivation, and CMV serostatus. This suggested that HIV infection itself was an independent risk factor for Long COVID in this cohort.

Finally, a recent study that has not yet been peer-reviewed has attempted to ascertain the likelihood of postacute sequelae of SARS-CoV-2 using data from 69 US healthcare organizations sourced from the TriNetX federated health research network [53▪▪]. The authors evaluated nearly 29 000 individuals between January 2020 and September 2022 who had ICD10 data indicating both SARS-CoV-2 infection and HIV infection. They compared PLWH with a similarly sized cohort of HIV-negative individuals, propensity matching for age, sex, race, ethnicity, BMI, and select comorbid conditions. They found a higher odds ratio in PLWH for several new diagnoses in an undefined post-COVID period: 'mental disorders', malignancy, heart disease, diabetes, and thrombosis. They also examined Long COVID symptoms and found body aches, headaches, gastrointestinal symptoms, respiratory symptoms, fatigue, and cognitive impairment to be more likely to be reported among PLWH. This study provides valuable information from a large US database and shows that certain symptoms and new onset health conditions were more likely to occur in PLWH in a mostly unvaccinated cohort after SARS-CoV-2 infection.

Based on these analyses, it remains possible and even likely, but not definitively proven, that HIV is a risk factor for Long COVID. Whether some of the excess risk for symptoms in PLWH can be attributed to factors such as smoking or comorbid conditions more frequent in this population requires studies with PLWH who do not have COVID-19 as a control group. Below, we outline the current research on the

pathobiology of Long COVID and delineate why PLWH may be more susceptible to develop postacute symptoms and sequelae [54] (Fig. 1).

RISK FACTORS FOR AND MECHANISMS OF LONG COVID: WHY PEOPLE LIVING WITH HIV MIGHT BE VULNERABLE

There is substantial overlap between the clinical risk factors for Long COVID and the demographics and clinical comorbidities of PLWH. First, severity of acute infection has been repeatedly identified as a predictor of Long COVID. Although initial small studies suggested that PLWH did not exhibit worse outcomes during acute SARS-CoV-2 infection [55–59], larger studies have shown that HIV status is a risk factor for severe outcomes even among the virologically suppressed [60–67]. This risk is most pronounced in those with comorbidities, viremia, and/or poor immune reconstitution [63,68–72]. Second, PLWH commonly exhibit metabolic comorbidities such as diabetes and obesity, as well as cardiovascular disease [73–81], which are also risk factors for Long COVID. Furthermore, other possible contributors to Long COVID or exacerbating factors such as mental health conditions, substance use, and lower socioeconomic status are common among PLWH [82–85]. People with well controlled HIV infection can also exhibit neurocognitive impairment despite ART [86,87]; this HIV-associated neurocognitive disorder (HAND) in some ways resembles the types of deficits present in neurocognitive Long COVID [88,89]. Because of these issues, PLWH may be at increased risk for developing Long COVID.

Acute COVID-19 is a highly inflammatory syndrome [90], and SARS-CoV-2 convalescent plasma donors have higher levels of inflammation than historical donors [91]. Multiple studies have now identified immune dysregulation and inflammation, specifically pathways involving IL-6, TNF-alpha, and in some cases IL-1B, among others, in those with Long COVID in comparison to individuals with full recovery [92–101]. Whether these

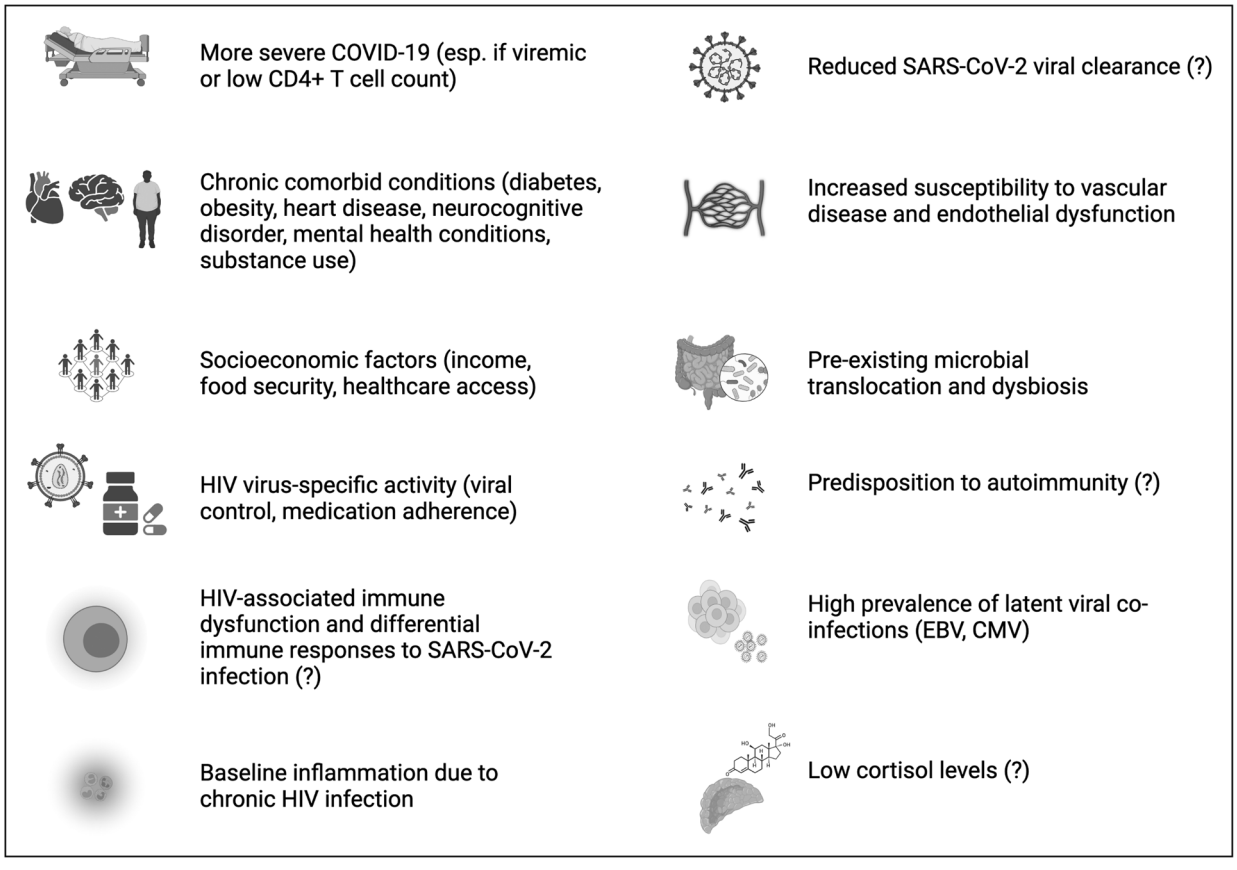

FIGURE 1. Potential contributors to Long COVID in people living with HIV. People living with HIV (PLWH) may be at elevated risk for Long COVID and other postacute sequelae following SARS-CoV-2 infection. While the risk factors for and mechanisms of such sequelae are incompletely understood, there are a variety of clinical, demographic, immunologic, and physiologic factors that could explain why PLWH might experience increased vulnerability to complications in the postacute phase of SARS-CoV-2 infection. '?' indicates potential contributors with limited strength of evidence. Created using Biorender.

perturbations drive Long COVID symptoms or are simply a result of other pathophysiologic processes as discussed below is unknown, as studies of immunomodulatory therapy in Long COVID have not yet been performed. However, it is well understood that PLWH – even those with viral suppression – have increased inflammation at baseline [102–106]. Therefore, they might be at higher risk for Long COVID following an additional SARS-CoV-2-initiated inflammatory insult. Although the cause of immune dysregulation in Long COVID is unknown, several mechanisms which may drive at least some cases of Long COVID have been proposed to contribute.

Recently, SARS-CoV-2 viral antigen persistence has been identified in a subset of individuals with Long COVID [107–111]. If persistence of viral genetic material or antigen drives Long COVID, it is possible that PLWH could exhibit reduced SARS-CoV-2 viral clearance from tissue reservoirs because of HIV-induced loss or dysfunction of tissue-specific immune cells. This could potentially be driven by dynamics of the humoral and cellular immune responses which in turn might contribute to Long COVID [94,96,98,99,112–118], although the role of the SARS-CoV-2 immune response in Long COVID and whether adaptive immune responses to SARS-CoV-2 differ by HIV status are unclear [51▪,119–123].

Other proposed immunologic mechanisms of Long COVID could exacerbate baseline levels of immune dysregulation in PLWH. PLWH are known to have higher baseline levels of endothelial dysfunction [74]. Microvascular dysfunction, platelet activation, and clotting are currently under investigation as potential drivers of Long COVID [124–128]. It is believed that these processes could drive organ dysfunction, which manifests as inflammation and symptoms. Similarly, microbial translocation because of reduced integrity of the blood–gut barrier is another plausible mechanism of Long COVID supported by data from a recent study [129], as is dysbiosis [130–134]. Disruption of the gut epithelial barrier and microbiome dysbiosis are associated with HIV [135], and so PLWH might be at increased risk of Long COVID because of gut dysfunction. PLWH may also be primed to develop autoreactive immunity [136–138], which has been demonstrated during and just after acute SARS-CoV-2 infection [96,139–143], although the data in Long COVID are mixed [96,97,140,142,144,145].

People with HIV are also known to have high rates of human herpesvirus co-infection such as EBV and human CMV [146–148]. Several recent studies identified that direct viral measurement during acute COVID-19 or indirect serologic evidence suggesting recent EBV reactivation during the postacute phase was associated with increased odds of Long COVID [52▪,96,149], and PLWH may be more susceptible to reactivation of this virus because of impairment of surveillance by CD8+ T cells [147]. Interestingly, CMV seropositivity was associated with lower odds of neurocognitive Long COVID symptoms in one Long COVID study in models controlling for HIV serostatus [52▪]. In addition, HIV-specific factors including viral activity and immune responses could contribute. For example, an early study of PLWH after COVID-19 showed an increased frequency of viral blips of unclear clinical significance in the postacute period in comparison with historical controls [150]. This suggests that there may be unique mechanisms of Long COVID in PLWH, and therefore dedicated mechanistic studies of PASC and Long COVID in this subpopulation are warranted.

Finally, two independent studies identified an association of low cortisol levels with Long COVID [96,97]. The adrenal cortex, which is the site of cortisol production, is highly vascular and is a known site of SARS-CoV-2 infection [151,152]. Many symptoms of Long COVID overlap with those of adrenal insufficiency [153], although most adrenal insufficiency among PLWH in the modern ART era is subclinical [154,155].

MANAGEMENT OF LONG COVID

Although several clinical trials are now underway, there is no accepted treatment for Long COVID, and a detailed review of Long COVID management practices is beyond the scope of this article. The US CDC provides diagnostic guidelines [156], and other management guidelines for the primary care setting are available [157]. Generally, Long COVID management requires a holistic, patient-centered approach, focused on ruling out other medically treatable conditions that might mimic symptoms of Long COVID, detection of treatable complications of COVID-19, management of unexplained symptoms, and referral as appropriate for more intensive testing. For PLWH, evaluation of medication adherence and adverse effects, confirmation of virologic suppression and stable immune status, and attention to comorbid medical and psychosocial conditions are also important. Many nascent Long COVID clinics utilize a hub-and-spoke model to deliver coordinated care through a core team that partners with many medical subspecialties (e.g. pulmonology, neurology, cardiology, and psychiatry) and support services (e.g. pharmacy, physical and occupational therapy, social work, and behavioral health) [158]; this approach has for decades been used in many HIV and primary care clinics. Finally, we also note that PLWH continue to face substantial

stigmatization [159,160], and Long COVID and other poorly understood medical conditions can also result in stigma [161,162], although the scope of the problem has not been fully explored and few studies have addressed the qualitative experiences of people with HIV also experiencing Long COVID [163]. It is particularly important that HIV providers be aware of these issues and how they might complicate care for PLWH experiencing Long COVID.

CONCLUSION

Although Long COVID is now a well established clinical entity and public health challenge, many questions remain. More research examining the epidemiology of Long COVID in PLWH, in particular whether PLWH are at higher risk for developing Long COVID and whether the clinical manifestations or natural history differ between HIV-seropositive and HIV-seronegative people are needed. In order to distinguish the burden of HIV-specific morbidity among PLWH from that caused by COVID-19, studies of the impact of COVID-19 on the long-term health of PLWH may also need to make comparisons with PLWH who did not develop COVID-19 during the study interval. In addition, ongoing efforts will be needed to determine whether the development of Long COVID in PLWH is influenced by vaccination status, acute COVID-19 treatment, and the emergence of new variants to the same degree that it might be in HIV-seronegative individuals. As the mechanisms of Long COVID in the general population are further delineated, these need to be examined in PLWH, and attention needs to be paid to contributors that might be unique or more important in driving Long COVID among PLWH. And critically, efforts must be made to ensure that PLWH experiencing Long COVID are not systematically excluded from the nascent clinical trials that will soon be implemented to determine how best to manage Long COVID symptoms. Ultimately, regardless of whether it is more common, presents differently, or has different mechanisms, Long COVID is likely to remain an important new comorbid condition among a subset of PLWH recovering from SARS-CoV-2 infection for the foreseeable future.

Acknowledgements

None.

Financial support and sponsorship

None.

Conflicts of interest

M.J.P. reports consulting fees from Gilead Sciences and AstraZeneca.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- ■ of special interest
- ■■ of outstanding interest

1. Callard F, Perego E. How and why patients made Long Covid. Soc Sci Med 2021; 268:113426.
2. Tenforde MW, Kim SS, Lindsell CJ, *et al.* Symptom duration and risk factors for delayed return to usual health among outpatients with COVID-19 in a Multistate Healthcare Systems Network - United States, March-June 2020. MMWR Morb Mortal Wkly Rep 2020; 69:993–998.
3. NIH launches new initiative to study 'Long COVID.' National Institutes of Health (NIH) 2021.
4. The White House. Memorandum on addressing the Long-Term Effects of COVID-19. The White House 2022.
5. Coronavirus: Coronavirus » Long COVID: the NHS plan for 2021/22 [date unknown].
6. Baraniuk C. Covid-19: how Europe is approaching long covid. BMJ 2022; 376:o158.
7. Office of the Assistant Secretary for Health (OASH): Health+ long COVID. HHS.gov 2022.
8. Walter J. Koroshetz, M. D., National Institute of Neurological Disorders and Stroke, Lawrence Tabak DDS, Collins F: PASC. NIH Director's Blog [date unknown].
9. Bowe B, Xie Y, Al-Aly Z. Acute and postacute sequelae associated with SARS-CoV-2 reinfection. Nat Med 2022; 28:2398–2405.
10. Groff D, Sun A, Ssentongo AE, *et al.* Short-term and long-term rates of postacute sequelae of SARS-CoV-2 infection: a systematic review. JAMA Netw Open 2021; 4:e2128568.
11. Di Gennaro F, Belati A, Tulone O, *et al.* Incidence of long COVID-19 in people with previous SARS-Cov2 infection: a systematic review and meta-analysis of 120,970 Ppatients. Intern Emerg Med 2022; 1–9; doi: 10.1007/s11739-022-03164-w. [Epub ahead of print]
12. Sudre CH, Murray B, Varsavsky T, *et al.* Attributes and predictors of long COVID. Nat Med 2021; 27:626–631.
13. Dennis A, Wamil M, Alberts J, *et al.*, COVERSCAN study investigators. Multiorgan impairment in low-risk individuals with post-COVID-19 syndrome: a prospective, community-based study. BMJ Open 2021; 11:e048391.
14. Al-Aly Z, Xie Y, Bowe B. High-dimensional characterization of postacute sequelae of COVID-19. Nature 2021; 594:259–264.
15. Xie Y, Xu E, Bowe B, *et al.* Long-term cardiovascular outcomes of COVID-19. Nat Med 2022; 28:583–590.
16. Al-Aly Z, Bowe B, Xie Y. Long COVID after breakthrough SARS-CoV-2 infection. Nat Med 2022; 28:1461–1467.
17. Xie Y, Choi T, Al-Aly Z. Nirmatrelvir and the risk of post-acute sequelae of COVID-19 [date unknown].
18. Yomogida K, Zhu S, Rubino F, *et al.* Post-acute sequelae of SARS-CoV-2 infection among adults aged ≥18 years - Long Beach, California, April 1-December 10, 2020. MMWR Morb Mortal Wkly Rep 2021; 70:1274–1277.
19. Cohen K, Ren S, Heath K, *et al.* Risk of persistent and new clinical sequelae among adults aged 65 years and older during the postacute phase of SARS-CoV-2 infection: retrospective cohort study. BMJ 2022; 376:e068414.
20. Bull-Otterson L. Post-COVID conditions among adult COVID-19 survivors aged 18-64 and ≥65 years — United States, March 2020–November 2021. MMWR Morb Mortal Wkly Rep 2022; 71:713–717.
21. Perlis RH, Santillana M, Ognyanova K, *et al.* Prevalence and correlates of Long COVID Symptoms among US adults. JAMA Netw Open 2022; 5: e2238804.
22. Wanga V, Chevinsky JR, Dimitrov LV, *et al.* Long-term symptoms among adults tested for SARS-CoV-2 - United States, January 2020-April 2021. MMWR Morb Mortal Wkly Rep 2021; 70:1235–1241.
23. Hernandez-Romieu AC, Leung S, Mbanya A, *et al.* Healthcare utilization and clinical characteristics of nonhospitalized adults in an integrated healthcare system 28-180 days after COVID-19 Diagnosis - Georgia, May 2020-March 2021. MMWR Morb Mortal Wkly Rep 2021; 70:644–650.
24. Ayoubkhani D, Bermingham C, Pouwels KB, *et al.* Trajectory of long covid symptoms after covid-19 vaccination: community based cohort study. BMJ 2022; 377:e069676.
25. Ayoubkhani D. Prevalence of ongoing symptoms following coronavirus (COVID-19) infection in the UK - Office for National Statistics. 2021.
26. Ayoubkhani D, Pawelek P, Gaughan C. Technical article: Updated estimates of the prevalence of postacute symptoms among people with coronavirus (COVID-19) in the UK - Office for National Statistics. 2021.
27. Huang C, Huang L, Wang Y, *et al.* 6-month consequences of COVID-19 in patients discharged from hospital: a cohort study. Lancet 2021; 397:220–232.
28. Roessler M, Tesch F, Batram M, *et al.* Post-COVID-19-associated morbidity in children, adolescents, and adults: A matched cohort study including more than 157,000 individuals with COVID-19 in Germany. PLoS Med 2022; 19: e1004122.

29. Taquet M, Dercon Q, Luciano S, *et al.* Incidence, co-occurrence, and evolution of long-COVID features: a 6-month retrospective cohort study of 273,618 survivors of COVID-19. PLoS Med 2021; 18:e1003773.
30. Ballering AV, van Zon SKR, olde Hartman TC, Rosmalen JGM. Persistence of somatic symptoms after COVID-19 in the Netherlands: an observational cohort study. Lancet 2022; 400:452–461.
31. Blomberg B, Mohn KG-I, Brokstad KA, *et al.* Long COVID in a prospective cohort of home-isolated patients. Nat Med 2021; 27:1607–1613.
32. Lund LC, Hallas J, Nielsen H, *et al.* Postacute effects of SARS-CoV-2 infection in individuals not requiring hospital admission: a Danish population-based cohort study. Lancet Infect Dis 2021; 21:1373–1382.
33. Morioka S, Tsuzuki S, Suzuki M, *et al.* Post COVID-19 condition of the Omicron variant of SARS-CoV-2. J Infect Chemother 2022; 28:1546–1551.
34. Azzolini E, Levi R, Sarti R, *et al.* Association Between BNT162b2 Vaccination and Long COVID After Infections Not Requiring Hospitalization in Healthcare Workers. JAMA 2022; 328:676–678.
35. Boglione L, Meli G, Poletti F, *et al.* Risk factors and incidence of long-COVID syndrome in hospitalized patients: does remdesivir have a protective effect? QJM 2022; 114:865–871.
36. Cutler DM. The costs of Long COVID. JAMA Health Forum 2022; 3: e221809–e1221809.
37. Nalbandian A, Sehgal K, Gupta A, *et al.* Postacute COVID-19P syndrome. Nat Med 2021; 27:601–615.
38. Peluso MJ, Kelly JD, Lu S, *et al.* Persistence, magnitude, and patterns of postacute symptoms and quality of life following onset of SARS-CoV-2 infection: cohort description and approaches for measurement. Open Forum Infect Dis 2022; 9:ofab640.
39. Kenny G, McCann K, O'Brien C, *et al.,* All-Ireland Infectious Diseases (AIID) Cohort Study Group. Identification of distinct Long COVID clinical phenotypes through cluster analysis of self-reported symptoms. Open Forum Infect Dis 2022; 9:ofac060.
40. Zhang H, Zang C, Xu Z, *et al.* Data-driven identification of postacute SARS-CoV-2 infection subphenotypes. Nat Med 2022; 29:226–235.
41. Lopez-Leon S, Wegman-Ostrosky T, Perelman C, *et al.* More than 50 long-term effects of COVID-19: a systematic review and meta-analysis. Sci Rep 2021; 11:16144.
42. Davis HE, Assaf GS, McCorkell L, *et al.* Characterizing long COVID in an international cohort: 7 months of symptoms and their impact. EClinicalMedicine 2021; 38:101019.
43. Deer RR, Rock MA, Vasilevsky N, *et al.* Characterizing Long COVID: deep phenotype of a complex condition. EBioMedicine 2021; 74:103722.
44. Estiri H, Strasser ZH, Brat GA, *et al.* Evolving phenotypes of nonhospitalized patients that indicate long COVID. BMC Med 2021; 19:249.
45. Subramanian A, Nirantharakumar K, Hughes S, *et al.* Symptoms and risk factors for long COVID in non-hospitalized adults. Nat Med 2022; 28:1706–1714.
46. Demko ZO, Yu T, Mullapudi SK, *et al.,* OutSMART Study Team. Post-acute sequelae of SARS-CoV-2 (PASC) impact quality of life at 6, 12 and 18 months post-infection. medRxiv [Preprint] 2022. doi: 10.1101/2022.08.08.22278543.
47. Durstenfeld MS, Peluso MJ, Peyser ND, *et al.* Factors associated with long COVID symptoms in an online cohort study. Open Forum Infect Dis 2023; 10:ofad047.
48. Pujari S, Gaikwad S, Chitalikar A, *et al.* Long-coronavirus disease among people living with HIV in western India: an observational study. Immun Inflamm Dis 2021; 9:1037–1043.
49. Mazzitelli M, Trunfio M, Sasset L, *et al.* Factors Associated with Severe COVID-19 and Post-Acute COVID-19 Syndrome in a Cohort of People Living with HIV on Antiretroviral Treatment and with Undetectable HIV RNA. Viruses 2022; 14:493.
50. Kingery JR, Safford MM, Martin P, *et al.* Health status, persistent symptoms, and effort intolerance one year after acute COVID-19 infection. J Gen Intern Med 2022; 37:1218–1225.
51. ▪ Peluso MJ, Spinelli MA, Deveau T-M, *et al.* Postacute sequelae and adaptive immune responses in people with HIV recovering from SARS-COV-2 infection. AIDS 2022; 36:F7–F16.

Detailed characterization of Long COVID in people living with HIV in comparison to HIV-seronegative individuals.

52. ▪ Peluso MJ, Deveau T-M, Munter SE, *et al.* Chronic viral coinfections differentially affect the likelihood of developing long COVID. J Clin Invest 2022; 133:e163669.

Identified HIV status as an independent risk factor for Long COVID when controlling for demographic factors, severity of initial infection, serologic evidence suggesting recent EBV reactivation, and CMV serostatus

53. ▪▪ Yendewa G, Perez JA, Patil N, McComsey GA. HIV infection is associated with higher risk of post-acute sequelae of SARS-CoV-2 (PASC) however vaccination is protective. Available at SSRN: https://ssrn.com/abstract=4276609 or http://dx.doi.org/10.2139/ssrn.4276609.

Large EMR-based study identifying higher risk of postacute sequelae following SARS-CoV-2 infection, including Long COVID symptoms, among people living with HIV.

54. Peluso MJ, Deeks SG. Early clues regarding the pathogenesis of long-COVID. Trends Immunol 2022; 43:268–270.
55. Blanco JL, Ambrosioni J, Garcia F, *et al.,* COVID-19 in HIV Investigators. COVID-19 in patients with HIV: clinical case series. Lancet HIV 2020; 7: e314–e316.
56. Härter G, Spinner CD, Roider J, *et al.* COVID-19 in people living with human immunodeficiency virus: a case series of 33 patients. Infection 2020; 48:681–686.
57. Vizcarra P, Pérez-Elías MJ, Quereda C, *et al.,* COVID-19 ID Team. Description of COVID-19 in HIV-infected individuals: a single-centre, prospective cohort. Lancet HIV 2020; 7:e554–e564.
58. Sigel K, Swartz T, Golden E, *et al.* Coronavirus 2019 and people living with human immunodeficiency virus: outcomes for hospitalized patients in New York City. Clin Infect Dis 2020; 71:2933–2938.
59. Ho H-E, Peluso MJ, Margus C, *et al.* Clinical outcomes and immunologic characteristics of coronavirus disease 2019 in people with human immunodeficiency virus. J Infect Dis 2021; 223:403–408.
60. Tesoriero JM, Swain C-AE, Pierce JL, *et al.* COVID-19 outcomes among persons living with or without diagnosed HIV infection in New York state. JAMA Netw Open 2021; 4:e2037069.
61. Mellor MM, Bast AC, Jones NR, *et al.* Risk of adverse coronavirus disease 2019 outcomes for people living with HIV. AIDS 2021; 35:F1–F10.
62. Rial-Crestelo D, Bisbal O, Font R, *et al.* Incidence and severity of SARS-CoV-2 infection in HIV-infected individuals during the first year of the pandemic. J Acquir Immune Defic Syndr 2021; 89:511–518.
63. Nomah DK, Reyes-Urueña J, Diaz Y, *et al.,* PISCIS study group. Sociodemographic, clinical, and immunological factors associated with SARS-CoV-2 diagnosis and severe COVID-19 outcomes in people living with HIV: a retrospective cohort study. Lancet HIV 2021; 8:e701–e710.
64. Yang X, Sun J, Patel RC, *et al.,* National COVID Cohort Collaborative Consortium. Associations between HIV infection and clinical spectrum of COVID-19: a population level analysis based on US National COVID Cohort Collaborative (N3C) data. Lancet HIV 2021; 8:e690–e700.
65. Bhaskaran K, Rentsch CT, MacKenna B, *et al.* HIV infection and COVID-19 death: a population-based cohort analysis of UK primary care data and linked national death registrations within the OpenSAFELY platform. Lancet HIV 2021; 8:e24–e32.
66. Geretti AM, Stockdale AJ, Kelly SH, *et al.* Outcomes of coronavirus disease 2019 (COVID-19) related hospitalization among people with human immunodeficiency virus (HIV) in the ISARIC World Health Organization (WHO) Clinical Characterization Protocol (UK): a prospective observational study. Clin Infect Dis 2021; 73:e2095–e2106.
67. Bertagnolio S, Thwin S, Silva R, *et al.* Clinical characteristics and prognostic factors in people living with HIV hospitalized with COVID-19: findings from the WHO Global Clinical Platform. In 11th International AIDS Society Conference on HIV Science. 2021. pp. 18–21.
68. Wang H, Jonas KJ. The likelihood of severe COVID-19 outcomes among PLHIV with various comorbidities: a comparative frequentist and Bayesian meta-analysis approach. J Int AIDS Soc 2021; 24:e25841.
69. Jassat W, Cohen C, Tempia S, *et al.,* DATCOV author group. Risk factors for COVID-19-related in-hospital mortality in a high HIV and tuberculosis prevalence setting in South Africa: a cohort study. Lancet HIV 2021; 8: e554–e567.
70. Madhi SA, Nel J. Epidemiology of severe COVID-19 from South Africa. Lancet HIV 2021; 8:e524–e526.
71. Hoffmann C, Casado JL, Härter G, *et al.* Immune deficiency is a risk factor for severe COVID-19 in people living with HIV. HIV Med 2021; 22:372–378.
72. Dandachi D, Geiger G, Montgomery MW, *et al.* Characteristics, comorbidities, and outcomes in a multicenter registry of patients with human immunodeficiency virus and coronavirus disease. Clin Infect Dis 2021; 73: e1964–e1972.
73. Hsue PY, Deeks SG, Hunt PW. Immunologic basis of cardiovascular disease in HIV-infected adults. J Infect Dis 2012; 205(Suppl 3):S375–S382.
74. Anand AR, Rachel G, Parthasarathy D. HIV proteins and endothelial dysfunction: implications in cardiovascular disease. Front Cardiovasc Med 2018; 5:185.
75. McLaughlin MM, Ma Y, Scherzer R, *et al.* Association of viral persistence and atherosclerosis in adults with treated HIV infection. JAMA Netw Open 2020; 3:e2018099.
76. Hsu DC, Ma YF, Hur S, *et al.* Plasma IL-6 levels are independently associated with atherosclerosis and mortality in HIV-infected individuals on suppressive antiretroviral therapy. AIDS 2016; 30:2065–2074.
77. Chow FC, Ma Y, Manion M, *et al.* Factors associated with worse cerebrovascular function in aging women with and at risk for HIV. AIDS 2021; 35:257–266.
78. Hsue PY, Waters DD. HIV infection and coronary heart disease: mechanisms and management. Nat Rev Cardiol 2019; 16:745–759.
79. Kalra S, Kalra B, Agrawal N, Unnikrishnan A. Understanding diabetes in patients with HIV/AIDS. Diabetol Metab Syndr 2011; 3:2.
80. Hernandez-Romieu AC, Garg S, Rosenberg ES, *et al.* Is diabetes prevalence higher among HIV-infected individuals compared with the general population? Evidence from MMP and NHANES 2009-2010. BMJ Open Diabetes Res Care 2017; 5:e000304.
81. Sarkar S, Brown TT. Diabetes in people with HIV. Curr Diab Rep 2021; 21:13.
82. Adams C, Zacharia S, Masters L, *et al.* Mental health problems in people living with HIV: changes in the last two decades: the London experience 1990–2014. AIDS Care 2016; 28(Suppl 1):56–59.

83. Cook JA, Burke-Miller JK, Steigman PJ, *et al.* Prevalence, comorbidity, and correlates of psychiatric and substance use disorders and associations with hiv risk behaviors in a multisite cohort of women living with HIV. AIDS Behav 2018; 22:3141–3154.

84. Lesko CR, Keil AP, Moore RD, *et al.* Measurement of current substance use in a cohort of HIV-infected persons in continuity HIV care, 2007–2015. Am J Epidemiol 2018; 187:1970–1979.

85. Ghiasvand H, Higgs P, Noroozi M, *et al.* Social and demographical determinants of quality of life in people who live with HIV/AIDS infection: evidence from a meta-analysis. Biodemography Soc Biol 2020; 65:57–72.

86. Farhadian S, Patel P, Spudich S. Neurological complications of HIV infection. Curr Infect Dis Rep 2017; 19:50.

87. Peluso MJ, Hellmuth J, Chow FC. Central nervous system effects of COVID-19 in people with HIV infection. Curr HIV/AIDS Rep 2021; 18:538–548.

88. Hellmuth J, Barnett TA, Asken BM, *et al.* Persistent COVID-19-associated neurocognitive symptoms in nonhospitalized patients. J Neurovirol 2021; 27:191–195.

89. Apple AC, Oddi A, Peluso MJ, *et al.* Risk factors and abnormal cerebrospinal fluid associate with cognitive symptoms after mild COVID-19. Ann Clin Transl Neurol 2022; 9:221–226.

90. Lucas C, Wong P, Klein J, *et al.* Longitudinal analyses reveal immunological misfiring in severe COVID-19. Nature 2020; 584:463–469.

91. Bonny TS, Patel EU, Zhu X, *et al.* Cytokine and chemokine levels in coronavirus disease 2019 convalescent plasma. Open Forum Infect Dis 2021; 8:ofaa574.

92. Peluso MJ, Lu S, Tang AF, *et al.* Markers of immune activation and inflammation in individuals with postacute sequelae of severe acute respiratory syndrome coronavirus 2 infection. J Infect Dis 2021; 224:1839–1848.

93. Peluso MJ, Sans HM, Forman CA, *et al.* Plasma markers of neurologic injury and inflammation in people with self-reported neurologic postacute sequelae of SARS-CoV-2 infection. Neurol Neuroimmunol Neuroinflamm 2022; 9:e200003.

94. Phetsouphanh C, Darley DR, Wilson DB, *et al.* Immunological dysfunction persists for 8 months following initial mild-to-moderate SARS-CoV-2 infection. Nat Immunol 2022; 23:210–216.

95. Schultheiß C, Willscher E, Paschold L, *et al.* The IL-1β, IL-6, and TNF cytokine triad is associated with postacute sequelae of COVID-19. Cell Rep Med 2022; 3:100663.

96. Su Y, Yuan D, Chen DG, *et al.* Multiple early factors anticipate post-acute COVID-19 sequelae. Cell 2022; 185:881–895.e20.

97. Klein J, Wood J, Jaycox J, *et al.* Distinguishing features of Long COVID identified through immune profiling. medRxiv 2022. doi: 10.1101/2022.08.09.22278592.

98. Vijayakumar B, Boustani K, Ogger PP, *et al.* Immuno-proteomic profiling reveals aberrant immune cell regulation in the airways of individuals with ongoing post-COVD-19 respiratory disease. Immunity 2022; 55:542–556.e5.

99. Littlefield KM, Watson RO, Schneider JM, *et al.* SARS-CoV-2-specific T cells associate with inflammation and reduced lung function in pulmonary post-acute sequalae of SARS-CoV-2. PLoS Pathog 2022; 18:e1010359.

100. Sollini M, Morbelli S, Ciccarelli M, *et al.* Long COVID hallmarks on [18F]FDG-PET/CT: a case-control study. Eur J Nucl Med Mol Imaging 2021; 48:3187–3197.

101. Durstenfeld MS, Peluso MJ, Kelly JD, *et al.* Role of antibodies, inflammatory markers, and echocardiographic findings in postacute cardiopulmonary symptoms after SARS-CoV-2 infection. JCI Insight 2022; 7:e157053.

102. Khoury G, Fromentin R, Solomon A, *et al.* Human immunodeficiency virus persistence and T-cell activation in blood, rectal, and lymph node tissue in human immunodeficiency virus-infected individuals receiving suppressive antiretroviral therapy. J Infect Dis 2017; 215:911–919.

103. Somsouk M, Estes JD, Deleage C, *et al.* Gut epithelial barrier and systemic inflammation during chronic HIV infection. AIDS 2015; 29:43–51.

104. Tenorio AR, Zheng Y, Bosch RJ, *et al.* Soluble markers of inflammation and coagulation but not T-cell activation predict non-AIDS-defining morbid events during suppressive antiretroviral treatment. J Infect Dis 2014; 210:1248–1259.

105. Serrano-Villar S, Sainz T, Lee SA, *et al.* HIV-infected individuals with low CD4/CD8 ratio despite effective antiretroviral therapy exhibit altered T cell subsets, heightened CD8+ T cell activation, and increased risk of non-AIDS morbidity and mortality. PLoS Pathog 2014; 10:e1004078.

106. Borges ÁH, O'Connor JL, Phillips AN, *et al.,* INSIGHT SMART and ESPRIT Study Groups and the SILCAAT Scientific Committee. Factors associated with plasma IL-6 levels during HIV infection. J Infect Dis 2015; 212:585–595.

107. Gaebler C, Wang Z, Lorenzi JCC, *et al.* Evolution of antibody immunity to SARS-CoV-2. Nature 2021; 591:639–644.

108. Natarajan A, Zlitni S, Brooks EF, *et al.* Gastrointestinal symptoms and fecal shedding of SARS-CoV-2 RNA suggest prolonged gastrointestinal infection. Med (N Y) 2022; 3:371–387.e9.

109. Peluso MJ, Deeks SG, Mustapic M, *et al.* SARS-CoV-2 and Mitochondrial Proteins in Neural-Derived Exosomes of COVID-19. Ann Neurol 2022; 91:772–781.

110. Swank Z, Senussi Y, Manickas-Hill Z, *et al.* Persistent circulating SARS-CoV-2 spike is associated with postacute COVID-19 sequelae. Clin Infect Dis 2022; 76:e487–e490.

111. Craddock V, Mahajan A, Spikes L, *et al.* Persistent circulation of soluble and extracellular vesicle-linked Spike protein in individuals with postacute sequelae of COVID-19. J Med Virol 2023; 95:e28568.

112. Peluso MJ, Deitchman AN, Torres L, *et al.* Long-term SARS-CoV-2-specific immune and inflammatory responses in individuals recovering from COVID-19 with and without postacute symptoms. Cell Rep 2021; 36:109518.

113. Herman JD, Atyeo C, Zur Y, *et al.* Impact of cross-coronavirus immunity in post-acute sequelae of COVID-19. medRxiv [Preprint] 2022. doi: 10.1101/2022.09.25.22280335.

114. Hu F, Chen F, Ou Z, *et al.* A compromised specific humoral immune response against the SARS-CoV-2 receptor-binding domain is related to viral persistence and periodic shedding in the gastrointestinal tract. Cell Mol Immunol 2020; 17:1119–1125.

115. Fernández-Castañeda A, Lu P, Geraghty AC, *et al.* Mild respiratory COVID can cause multi-lineage neural cell and myelin dysregulation. Cell 2022; 185:2452–2468.e16.

116. Bergamaschi L, Mescia F, Turner L, *et al.* Longitudinal analysis reveals that delayed bystander CD8+ T cell activation and early immune pathology distinguish severe COVID-19 from mild disease. Immunity 2021; 54:1257.e8–1275.e8.

117. Cervia C, Zurbuchen Y, Taeschler P, *et al.* Immunoglobulin signature predicts risk of postacute COVID-19 syndrome. Nat Commun 2022; 13:446.

118. Jia X, Cao S, Lee AS, *et al.* Antinucleocapsid antibody levels and pulmonary comorbid conditions are linked to post-COVID-19 syndrome. JCI Insight 2022; 7:7.

119. Spinelli MA, Lynch KL, Yun C, *et al.* SARS-CoV-2 seroprevalence, and IgG concentration and pseudovirus neutralising antibody titres after infection, compared by HIV status: a matched case-control observational study. Lancet HIV 2021; 8:e334–e341.

120. Verburgh ML, Boyd A, Wit FWNM, *et al.,* AGEhIV Study Group. Similar risk of SARS-CoV-2 infection and similar nucleocapsid antibody levels in people with well controlled HIV and a comparable cohort of people without HIV. J Infect Dis 2021; 225:1937–1947.

121. Martín-Vicente M, Berenguer J, Muñoz-Gómez MJ, *et al.* Similar humoral immune responses against the SARS-CoV-2 spike protein in HIV and non-HIV individuals after COVID-19. J Infect 2021; 84:418–467.

122. Snyman J, Hwa S-H, Krause R, *et al.* Similar antibody responses against SARS-CoV-2 in HIV uninfected and infected individuals on antiretroviral therapy during the first South African infection wave. Clin Infect Dis 2021; 75:e249–e256.

123. Alrubayyi A, Gea-Mallorquí E, Touizer E, *et al.* Characterization of humoral and SARS-CoV-2 specific T cell responses in people living with HIV. Nat Commun 2021; 12:5839.

124. Ahamed J, Laurence J. Long COVID endotheliopathy: hypothesized mechanisms and potential therapeutic approaches. J Clin Invest 2022; 132: e161167.

125. Pretorius E, Venter C, Laubscher GJ, *et al.* Prevalence of symptoms, comorbidities, fibrin amyloid microclots and platelet pathology in individuals with Long COVID/Post-Acute Sequelae of COVID-19 (PASC). Cardiovasc Diabetol 2022; 21:148.

126. Pretorius E, Vlok M, Venter C, *et al.* Persistent clotting protein pathology in Long COVID/Post-Acute Sequelae of COVID-19 (PASC) is accompanied by increased levels of antiplasmin. Cardiovasc Diabetol 2021; 20:172.

127. Fogarty H, Townsend L, Morrin H, *et al.,* Irish COVID-19 Vasculopathy Study (iCVS) investigators. Persistent endotheliopathy in the pathogenesis of long COVID syndrome. J Thromb Haemost 2021; 19:2546–2553.

128. Sun B, Tang N, Peluso MJ, *et al.* Characterization and biomarker analyses of post-COVID-19 complications and neurological manifestations. Cells 2021; 10:386.

129. Giron LB, Peluso MJ, Ding J, *et al.* Markers of fungal translocation are elevated during postacute sequelae of SARS-CoV-2 and induce NF-κB signaling. JCI Insight 2022; 7:e160989.

130. Yeoh YK, Zuo T, Lui GC-Y, *et al.* Gut microbiota composition reflects disease severity and dysfunctional immune responses in patients with COVID-19. Gut 2021; 70:698–706.

131. Zuo T, Zhang F, Lui GCY, *et al.* Alterations in gut microbiota of patients with COVID-19 during time of hospitalization. Gastroenterology 2020; 159:944. e8–955.e8.

132. Haran JP, Bradley E, Zeamer AL, *et al.* Inflammation-type dysbiosis of the oral microbiome associates with the duration of COVID-19 symptoms and long COVID. JCI Insight 2021; 6:e152346.

133. Liu Q, Su Q, Zhang F, *et al.* Multikingdom gut microbiota analyses define COVID-19 severity and postacute COVID-19 syndrome. Nat Commun 2022; 13:6806.

134. Liu Q, Mak JWY, Su Q, *et al.* Gut microbiota dynamics in a prospective cohort of patients with postacute COVID-19 syndrome. Gut 2022; 71:544–552.

135. Zevin AS, McKinnon L, Burgener A, Klatt NR. Microbial translocation and microbiome dysbiosis in HIV-associated immune activation. Curr Opin HIV AIDS 2016; 11:182–190.

136. Iordache L, Launay O, Bouchaud O, *et al.* Autoimmune diseases in HIV-infected patients: 52 cases and literature review. Autoimmun Rev 2014; 13:850–857.

137. Virot E, Duclos A, Adelaide L, *et al.* Autoimmune diseases and HIV infection: a cross-sectional study. Medicine (Baltimore) 2017; 96:e5769.

138. Yen Y-F, Chuang P-H, Jen I-A, *et al.* Incidence of autoimmune diseases in a nationwide HIV/AIDS patient cohort in Taiwan, 2000–2012. Ann Rheum Dis 2017; 76:661–665.
139. Song E, Bartley CM, Chow RD, *et al.* Divergent and self-reactive immune responses in the CNS of COVID-19 patients with neurological symptoms. Cell Rep Med 2021; 2:100288.
140. Seeßle J, Waterboer T, Hippchen T, *et al.* Persistent symptoms in adult patients one year after COVID-19: a prospective cohort study. Clin Infect Dis 2021; 74:1191–1198.
141. Woodruff MC, Ramonell RP, Haddad NS, *et al.* Dysregulated nave B cells and de novo autoreactivity in severe COVID-19. Nature 2022; 611:139–147.
142. Son K, Jamil R, Chowdhury A, *et al.* Circulating antinuclear autoantibodies in COVID-19 survivors predict long-COVID symptoms. Eur Respir J 2022; 61:2200970.
143. Chang SE, Feng A, Meng W, *et al.* New-onset IgG autoantibodies in hospitalized patients with COVID-19. Nat Commun 2021; 12:5417.
144. Peluso MJ, Thomas IJ, Munter SE, *et al.* Lack of antinuclear antibodies in convalescent coronavirus disease 2019 patients with persistent symptoms. Clin Infect Dis 2022; 74:2083–2084.
145. Peluso MJ, Mitchell A, Wang CY, *et al.* Low prevalence of interferon-α autoantibodies in people experiencing Long COVID symptoms. J Infect Dis 2022; 227:246–250.
146. Yan Y, Ren Y, Chen R, *et al.* Evaluation of Epstein-Barr virus salivary shedding in HIV/AIDS patients and HAART use: a retrospective cohort study. Virol Sin 2018; 33:227–233.
147. Hernández DM, Valderrama S, Gualtero S, *et al.* Loss of T-cell multifunctionality and TCR-Vβ Repertoire against Epstein-Barr virus is associated with worse prognosis and clinical parameters in HIV+ patients. Front Immunol 2018; 9:2291.
148. Zuhair M, Smit GSA, Wallis G, *et al.* Estimation of the worldwide seroprevalence of cytomegalovirus: a systematic review and meta-analysis. Rev Med Virol 2019; 29:e2034.
149. Gold JE, Okyay RA, Licht WE, Hurley DJ. Investigation of long COVID prevalence and its relationship to Epstein-Barr virus reactivation. Pathogens 2021; 10:763.
150. Peluso MJ, Bakkour S, Busch MP, *et al.* A high percentage of people with human immunodeficiency virus (HIV) on antiretroviral therapy experience detectable low-level plasma HIV-1 RNA following coronavirus disease 2019 (COVID-19). Clin Infect Dis 2021; 73:e2845–e2846.
151. Paul T, Ledderose S, Bartsch H, *et al.* Adrenal tropism of SARS-CoV-2 and adrenal findings in a postmortem case series of patients with severe fatal COVID-19. Nat Commun 2022; 13:1589.
152. Kanczkowski W, Evert K, Stadtmüller M, *et al.* COVID-19 targets human adrenal glands. Lancet Diabetes Endocrinol 2022; 10:13–16.
153. Henry M, Thomas KGF, Ross IL. Sleep, cognition and cortisol in Addison's Disease: a mechanistic relationship. Front Endocrinol 2021; 12:694046.
154. Mayo J, Collazos J, Martínez E, Ibarra S. Adrenal function in the human immunodeficiency virus-infected patient. Arch Intern Med 2002; 162:1095–1098.
155. Mifsud S, Gauci Z, Gruppetta M, *et al.* Adrenal insufficiency in HIV/AIDS: a review. Expert Rev Endocrinol Metab 2021; 16:351–362.
156. CDC. Post-COVID conditions: information for healthcare providers. Centers for Disease Control and Prevention. 2022.
157. Greenhalgh T, Sivan M, Delaney B, *et al.* Long covid—an update for primary care. BMJ 2022; 378:e072117.
158. Santhosh L, Block B, Kim SY, *et al.* Rapid design and implementation of post-COVID-19 clinics. Chest 2021; 160:671–677.
159. Geter A, Herron AR, Sutton MY. HIV-related stigma by healthcare providers in the united states: a systematic review. AIDS Patient Care STDS 2018; 32:418–424.
160. Ma PHX, Chan ZCY, Loke AY. Self-stigma reduction interventions for people living with HIV/AIDS and their families: a systematic review. AIDS Behav 2019; 23:707–741.
161. Byrne EA. Understanding Long Covid: nosology, social attitudes and stigma. Brain Behav Immun 2022; 99:17–24.
162. Macpherson K, Cooper K, Harbour J, *et al.* Experiences of living with long COVID and of accessing healthcare services: a qualitative systematic review. BMJ Open 2022; 12:e050979.
163. Santiago-Rodriguez EI, Maiorana A, Peluso MJ, *et al.* Characterizing the COVID-19 illness experience to inform the study of postacute sequelae and recovery. Int J Behav Med 2022; 29:610–623.