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## COVID-19 in patients with HIV

We read with interest the report by Blanco and colleagues<sup>1</sup> of five people living with HIV who were admitted to a Barcelona hospital with COVID-19. We believe that caution is required before drawing conclusions on the outcome of COVID-19 in this population.

Evidence is evolving that protease inhibitors developed for the treatment of HIV, both lopinavir and darunavir boosted by ritonavir or cobicistat, are not efficacious against severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) *in vivo*.<sup>2–4</sup> Therefore, antiretroviral combinations should not be changed in an attempt to treat SARS-CoV-2 infection, because neither drug combination is a first-line choice in most guidelines for HIV and changing treatment could lead to increased rates of adverse events.

Antiretroviral treatments such as non-nucleoside reverse transcriptase inhibitors and integrase inhibitors have better tolerability than boosted protease inhibitors.<sup>5</sup> Moreover, three of the five cases described by Blanco and colleagues were initiated or switched to an antiretroviral combination containing a pharmacokinetic booster, thereby introducing a substantial risk of significant drug-drug interactions.<sup>5</sup> New antiviral drugs active against COVID-19 are being developed, and interactions of such drugs with

antiretrovirals can be seen frequently. For example, remdesivir might interact with carbamazepine and other drug metabolism inducers, and no data are available on potential interactions with nucleoside analogues used in antiretroviral combinations.<sup>5</sup>

Caution is needed when interpreting the incidence of COVID-19 in people living with HIV compared with the HIV-negative population. The numbers reported by Blanco and colleagues are small and patients attended only one hospital, so the sample is subject to bias. The authors do not report on the proportion of patients with COVID-19 who were tested for HIV infection. Without universal HIV testing, it is not possible to calculate the incidence of the two viral infections occurring in the same individual simultaneously.

The statement that only 1% of people admitted with COVID-19 to one hospital in Barcelona had HIV can be misinterpreted and falsely reassuring, particularly while we still do not entirely understand which populations should be protected from COVID-19 by social interventions, such as shielding, self-isolation, and frequent testing. In the UK, large cohort studies are being done to investigate the true rate of infection, clinical characteristics, and outcomes of COVID-19 in people with HIV.

Challenges in understanding the true frequency of COVID-19 in people with HIV include the overall limited testing that has happened so far, particularly for patients not needing hospitalisation, the admission of patients in hospitals external to where the individual might access their HIV care, and the fact that people with HIV might be more vigilant at shielding and self-isolation because of the propagation of fears of higher acquisition rates and a poorer outcome of SARS-CoV-2 infection in people living with HIV.

Finally, appropriately powered and designed studies are needed to draw conclusions on the effect of COVID-19

in people with chronic diseases, including HIV infection. HIV infection is itself characterised by various clinical scenarios, ranging from viral suppression and good quality of life to HIV-associated comorbidities or virological failure with or without immunosuppression.<sup>5</sup>

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### Authors' reply

We thank Rachael Jones and colleagues for highlighting two issues prescribing caution before drawing conclusions from our case series<sup>1</sup> of COVID-19 in HIV-infected patients. The first issue is regarding treatment with boosted protease inhibitors; the second is about

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