Author Correction: High monoclonal neutralization titers reduced breakthrough HIV-1 viral loads in the Antibody Mediated Prevention trials

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Check for updates

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The originally published version of this Article contained several errors in references to previous work.

In the Introduction, references 23 and 26 were inadvertently omitted in two sentences, and have been added as follows: 'The Antibody Mediated Prevention (AMP) trials were the first human efficacy studies of infused bnAbs for HIV-1 prevention (HVTN 704/HPTN 085 and HVTN 703/HPTN 081)[23]. The efficacy of a CD4-binding site bnAb, VRC01, was tested in men, women and transgender individuals who were vulnerable to HIV-1 in the Americas, sub-Saharan Africa and Europe[24–26].'

The published Article incorrectly referenced Cale, E. M. et al. Neutralizing antibody VRC01 failed to select for HIV-1 mutations upon viral rebound. J Clin Invest 130, 3299–3304, https://doi.org/10.1172/JCI134395 (2020) in a sentence in the Discussion. The correct sentence instead cites reference 39 as follows: 'The TZM-bl target cell assay used to quantify in vitro neutralization uses cells that are highly permissive to infection[39], potentially allowing for larger reductions by VRC01.'

The published Article incorrectly referenced Gaebler, C. et al. Sequence Evaluation and Comparative Analysis of Novel Assays for Intact Proviral HIV-1 DNA. J Virol 95, https://doi.org/10.1128/JVI.01986-20 (2021) in a sentence in the Discussion. Gaebler et al. (2021) has been removed from the published Article. The correct sentence instead cites reference 64 as follows: 'Moreover, the pseudoviruses used in the assay may be more VRC01 sensitive than wild-type viruses[64].'

Due to a formatting error of the bibliography, several references were omitted in the published Article. These have been added as follows:

Reference 31 in the Introduction: 'Viral load has long been associated with HIV-1 pathogenesis and progression[31].'

Reference 36 in the Introduction: 'Previously, breakthrough infections among some PrEP users appear to admit lower viral loads[36].'

References 53–56 in the Discussion: 'Blunting or delaying HIV-1 viremia might be relevant for pathogenesis[52], reservoir creation[51], viral evolution[53], and/or onward transmission during acute infection. Furthermore, small but exciting studies[54,55] showed other bnAbs can achieve prolonged (up to 2 years) HIV-1 suppression after stopping ART potentially due to a vaccinal effect in which infused bnAbs interact with virus, e.g., forming immune complexes, to enhance immune control[56].'

References 67–69 in the Discussion: 'Another explanation may be that cell-to-cell transmission of HIV-1 effectively lowers the ability of VRC01 to find virus to neutralize[67–69].'

Reference 36 in the Discussion: 'Viral load blunting and delay was also observed in breakthrough acquisition during ART-mediated prevention trials[36].'

References 73–75 in the Discussion: 'In contrast to ART monotherapy, which rapidly and deterministically leads to viral escape[73–75], we show some evidence that bnAb monotherapy is relatively robust.'

References 56, 66 and 79 in the Discussion: 'Our viral load model does not notably consider cell-to-cell transmission, any enhancement of CD8 T cell killing via VRC01, or any active mechanism whereby VRC01 kills infected cells (such as antibody dependent cellular cytotoxicity or ADCC) and/or mediates phagocytosis[66,79]. This would be particularly important to know given the hypothesized bnAb vaccinal effect mentioned above[56] though it may be less critical here as VRC01 has demonstrated little effector functionality in prior studies[66].'

This has been corrected in the PDF and HTML versions of the Article.