Reply to Filteau

To the Editor—Filteau [1] writes in response to our recent paper [2] in which we showed that postnatally infected infants had been exposed to substantially larger amounts of cell-free human immunodeficiency virus (HIV) in breastmilk than were appropriate uninfected controls. Although Filteau does not directly comment on our study findings, she postulates that our results may confirm that exclusive breastfeeding (EBF) is not protective and that a protective association shown in other studies was confounded.

Filteau suggests that the apparent shorter EBF duration in the postnatally infected children in our case-control study was due to cessation of EBF upon infant HIV-infection diagnosis [1]. This would be in apparent contradiction of the view that EBF is protective against HIV-infection acquisition by the infant, as shown in large cohort studies [3–5]. We note that in our cohort, which had monthly follow-up visits, censoring of participants occurs close to the time of HIV acquisition, and in our case-control analysis there were no statistically significant differences in EBF duration before age at acquisition of infection between infants who got infected and those who did not. However, this analysis was not adjusted for potential confounders and it was not powered to answer the question of whether EBF cessation occurs upon HIV-infection diagnosis in the infant.

Cessation of EBF depends on multiple factors. We further compared in cases and controls the percentage of mothers who stopped EBF of their infant after the first HIV-positive test result. Fewer mothers of infected infants stopped EBF after the first HIV-positive test result than those of matched control infants (22 [61%] cases and 26 [72%] controls; $P = .32$). Among women who stopped EBF after the first HIV-positive test result, the delay between diagnosis of infant infection and cessation of EBF was shorter in cases (mean duration, 57.05 d; median duration, 59 d; interquartile range, 15–87 d) than in controls (mean duration, 66.65 d; median duration, 82 d; interquartile range, 24–101 d), although this difference was not significant ($P = .33$). We thus note that there is little or no evidence to suggest that postnatal infant HIV infection leads to cessation of EBF.

Filteau further suggests that reduced duration of EBF could be caused by poor maternal health (ie, advanced HIV infection), which is, however, also a strong driver of postnatal HIV transmission. In other words, the observed shorter duration of EBF among transmitters may only reflect that these mothers have more advanced HIV infection. To investigate this issue, we determined the correlation between the total duration of EBF over the first 28 weeks and the maternal antenatal CD4 cell count or the maternal postnatal CD4 cell count measured 28 weeks after delivery. The total duration of EBF was not correlated with maternal antenatal CD4 cell count ($\rho = 0.11$ [95% CI, −0.14 to 0.34]; $P = .39$), nor with maternal postnatal CD4 cell count ($\rho = 0.14$ [95% CI, −0.12 to 0.38]; $P = .28$). Similar results were found if only cases were analyzed ($\rho = 0.19$ [95% CI, −0.17 to 0.5]; $P = .29$; $\rho = 0.10$ [95% CI, −0.30 to 0.47]; $P = .61$). Our data, therefore, also do not support the assumption that EBF is reduced with advanced HIV infection.

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