Reply to Neogi et al

To the Editor—We appreciate the comments by Neogi et al in response to our study of human immunodeficiency virus type 1 (HIV-1) subtype and plasma viremia during early infection and their perspectives on the underlying causes for the spread of HIV-1 subtype C [1]. In our multinational population of persons with incident HIV-1 infection, we found that early viremia, as measured by mean viral load and the proportion of persons with high viral loads (>5 log_{10} copies/mL), was similar for individuals infected with HIV-1 subtype C, compared with those infected with other common African HIV-1 subtypes [2]. Prior studies, including those by Novitsky et al [3] and Neogi et al [4], found that a substantial fraction of persons with primary subtype C infection have high-level viremia, but those studies did not have contemporaneous comparison populations. Our findings suggest that the proportion of individuals with high-level viremia in early HIV-1 infection is similar for subtype C and non-C HIV-1 infections in Africa and that high-level early viremia specific to subtype C is not likely to explain the spread of subtype C in Africa.

The example from Neogi et al of additional nuclear factor–κB sites relative to other subtypes, potentially resulting in greater infectivity through increased transcriptional activation and replicative fitness [5], is a hypothesis our data could not explore. However, we recently reported the relationship between viral subtype and HIV-1 transmission among serodiscordant heterosexual couples in Africa and found that HIV-1 subtype C–infected individuals had no greater risk of transmitting virus to their uninfected partners than those with non–subtype C infection [6]. Those results suggest that transmissibility during chronic HIV-1 infection is no greater for subtype C, compared with other subtypes.

We agree with Neogi et al that host and viral genetic factors, sociobehavioral context, and other variables may contribute to the predominance of subtype C in the global HIV-1 epidemic and that a multifaceted approach will be required to control the further spread of HIV-1 in subtype C–endemic regions.

Notes

Disclaimer. The authors designed and executed the study, had full access to the raw data,
performed all analyses, wrote the manuscript, and had final responsibility for the decision to submit for publication.

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Mary S. Campbell,1 Erin M. Kahle,2,3 and Jared M. Baeten1,2,3
1Department of Medicine, 2Department of Epidemiology, and 3Department of Global Health, University of Washington, Seattle

References


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Correspondence: Mary S. Campbell, MD, University of Washington, Department of Medicine, Division of Allergy and Infectious Diseases, Box 358070, Seattle, WA 98195-8070 (msc6@u.washington.edu).