CORRESPONDENCE

Human T Cell Lymphotropic Virus Type II and Human Immunodeficiency Virus Type I Disease Progression

To the Editor—We read with interest the article by Hershow et al. [1] that suggests that human T cell lymphotropic virus (HTLV) type II does not increase the progression of human immunodeficiency virus (HIV) infection. The authors are to be lauded for addressing this issue with a multicenter seroincident cohort, a powerful methodologic approach. We agree that most of the epidemiologic studies suggesting that HTLV-I or -II accelerates the progression of HIV infection have methodologic limitations. Unfortunately, the authors misquote our study as being one of them [2]. A careful read of our article reveals that we included nothing in the results or conclusions about the effect of HTLV-I on the rate of HIV disease progression. To the contrary, we stated that the finding of more advanced HIV infection in coinfection was probably because HTLV-I is a marker of a longer duration of HIV infection. We purposely avoided the issue of coinfection and HIV disease progression because our cross-sectional data did not allow us to adequately address the issue and we had our own doubts about the validity of the hypothesis. In fact, we have data suggesting that the biologic basis for the hypothesis does not exist [3].

We believe that the authors’ article could be misconstrued to suggest that coinfection has no effect on CD4 lymphocyte counts. We found that HTLV-I coinfection in Brazil was associated with higher CD4 cell counts despite more advanced HIV infection, suggesting some degree of dissociation between CD4 cell count and HIV stage [2, 4]. This concept was recently confirmed with the report of 2 HTLV-I–coinfected patients in Baltimore with severe immunodeficiency and CD4 cell counts >3000/mm³ [5]. Although the authors acknowledge that HTLV-I and HTLV-II have different cell tropisms, they appear to treat the two viruses interchangeably when discussing this issue. Their data, which are restricted to patients with HTLV-II coinfection, cannot be considered to be applicable to patients with HTLV-I coinfection.

Lee H. Harrison and Mauro Schechter
University of Pittsburgh Graduate School of Public Health and School of Medicine, Pittsburgh, Pennsylvania; Infectious Diseases Service, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil

References


Reprints or correspondence: Dr. Lee H. Harrison, Dept. of Epidemiology, University of Pittsburgh School of Public Health, A526 Crabtree Hall, 130 DeSoto St., Pittsburgh, PA 15261.

The Journal of Infectious Diseases 1997;176:308–11 © 1997 by The University of Chicago. All rights reserved. 0022–1899/97/7601–0048$02.00

Reply

To the Editor—We wish to thank Harrison and Schechter for their thoughtful comments on our article [1]. They are correct that their paper does not assert that coinfection with human T lymphotropic virus type I (HTLV-I) hastens human immunodeficiency virus (HIV) disease progression [2]. In retrospect, we should have cited their work as having demonstrated an association between HTLV coinfection and more advanced HIV disease. A possible explanation of this finding, suggested by other investigators, has been an effect of HTLV on HIV disease progression [3]. However, such a hypothesis cannot be adequately addressed by a cross-sectional study. It was precisely for this reason that we investigated this issue in a prospectively followed cohort with well-defined dates of HIV acquisition and ample prevalence of HTLV coinfection [4]. It should be reemphasized that we studied HTLV-II, not HTLV-I, coinfection.

Harrison and Schechter are also concerned that readers may generalize our finding that HTLV-II coinfection has no apparent effect on the CD4 lymphocyte levels at which AIDS and AIDS-related mortality occur and conclude that our observations apply to both HTLV-I and HTLV-II. A careful reading of our paper should not allow such an inference. In our introduction, we discuss Harrison and Schechter’s findings regarding the dissociation between CD4 lymphocyte count and HIV disease stage in HTLV-I–coinfected persons and state that this issue has not been addressed previously in HTLV-II–coinfected persons. The importance of differentiating HTLV-I and HTLV-II infections is made in both the introduction and the discussion. In the latter, this point is made in the paragraph immediately preceding our discussion concerning the lack of effect of HTLV-II coinfection on the CD4 lymphocyte counts at which AIDS and death occur. Furthermore, our findings are contrasted with those of Harrison and Schechter.

Therefore, we hope it is clear, and would reemphasize, that our observations pertain to HTLV-II and differ from, but do not negate, Harrison and Schechter’s important findings regarding coinfection with HTLV-I.