was 18 months for the entire group. The Kaplan-Meier curves for the depressed and nondepressed groups (Figure) were compared using the log rank procedure \( P = 4.9 \). The finding from the multivariate Cox model showed a risk hazard for those with CES-D score of 16 or higher having a shorter time to death of 1.08 (95% confidence interval, 1.34 to 0.81). Analyses defining depression in other ways based on CES-D score\(^1\) or based on a visit within 6 months after AIDS showed similar results.

We conclude that there is no evidence that depression is associated with a worse survival in HIV infection at any disease stage. This further supports our previous findings\(^2,3\) and agrees with the finding by Burack et al\(^4\) that the association between depression and decline in CD4 cell count does not directly affect survival. It is important to replicate these findings using standardized psychiatric diagnosis. Depression occurs among 4% to 30% of HIV-infected persons, and it is a serious condition that causes impaired functioning and increased risk for suicide.\(^1\) Depression among HIV-infected persons should be identified and treated aggressively.

Constantine G, Lyketsos, MD, MHS
Donald R, Hoover, PhD, MPH
Marcella Guccione, MS
The Johns Hopkins University
Baltimore, MD

This study was supported by grant MH52507, from the National Institute of Mental Health, Bethesda, Md, and contracts N01-A1-25868, N01-A1-25869, N01-A1-25834, N01-A1-25874, and SM01-RR-00722 from National Institute of Allergy and Infectious Diseases, Bethesda, Md.


HIV-1 Shedding and Chlamydia Urethritis

To the Editor—Drs Schmid and Fontanarosa\(^1\) thoroughly discussed the causes and management of nongonococcal urethritis. However, they did not emphasize the link between treatment of sexually transmitted diseases and prevention of human immunodeficiency virus (HIV).\(^2\) We collected ejaculate from a 32-year-old white man with HIV-1 disease and a CD4 cell count of 0.11×10\(^9\)/L who was taking no antiretroviral therapy. The patient had chlamydial urethritis at the time the sample was obtained, with dysuria but without discharge. We performed quantitative HIV-1 culture on his seminal cell fraction, using a modification of the AIDS Clinical Trials Group quantitative cell culture protocol. We had previously performed 32 quantitative cultures for HIV-1 using seminal cells from 25 HIV-1-infected individuals; the range of excretion was <3.0 to 2.823.3 infectious units per ejaculate. Our patient excreted 6729 infectious units of HIV-1 in his ejaculate obtained at the time of chlamydial infection \( P < 0.001 \) for the comparison of subjects without symptoms of urethritis vs subjects with symptomatic urethritis. We also analyzed the seminal plasma by reverse transcriptase polymerase chain reaction, using a commercial assay (HIV-1 Amplicor Monitor, Roche Biomedical Laboratories, Research Triangle Park, NC). The patient excreted 375 000 copies of HIV-1 RNA per milliliter of seminal plasma. Four weeks after the patient completed therapy for chlamydial infection, we reexamined the semen for HIV-1. The quantitative culture could not be done. However, the copies of HIV-1 RNA in the seminal plasma at this time were reduced to 7500 per milliliter. Using an alternative amplification method that uses silica beads to separate HIV-1 RNA from potential inhibitors,\(^3\) the results from the pretreatment and posttreatment samples were 1 200 000 and 12 000 copies per milliliter, respectively. The patient received no antiretroviral therapy during this interval. These results suggest chlamydial urethritis may increase shedding of HIV-1 in semen, and treatment of chlamydial urethritis may decrease shedding of HIV-1. Moss and coworkers\(^4\) have reported that treatment of gonococcal urethritis in HIV-positive men decreases detection of HIV-1 in urethral swab specimens. These observations provide biological support to the recent observation that aggressive therapy for sexually transmitted diseases reduces transmission of HIV.\(^5\)

Joseph J. Eron, Jr, MD
Bruce Gilliam, MD
Susan Fiscus, PhD
John Dyer, MD
Myron S. Cohen, MD
University of North Carolina at Chapel Hill

This research was supported in part by grant U01-A125868-00 from the National Institutes of Health. Assay materials were provided by Roche Biomedical Laboratories, Research Triangle Park, NC, and Organon-Technika, Durham, NC.


CORRECTION

Incorrect Reference Citations in Text.—In the Letters entitled “The Relationship Between Physicians’ Malpractice Claims History and Later Claims,” published in the May 17, 1996, issue of THE JOURNAL (JAMA. 1996;275:1487-1495), the reference citations in the text of the reply by Messrs Bovbjerg and Petronis were incorrect. The first citation to reference 1 was correct; reference 2 should have been cited after the second and third sentences of the second paragraph; reference citations 3, 4, 5, and 6 should have been citations 3, 4, 5, and 6, respectively; and reference 6 should not have been cited in the sixth paragraph.