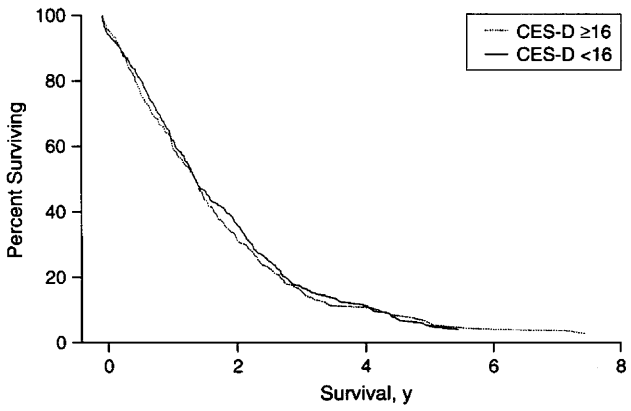

Time to death by depression status at visit within 6 months before acquired immunodeficiency syndrome. CES-D indicates Center for Epidemiologic Studies Depression Scale score.

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=.49). 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[1,2] 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.

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HIV-1 Shedding and Chlamydial 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 2523.3 infectious units per ejaculate. Our patient excreted 6729 infectious units of HIV-1 in his ejaculate obtained at the time of chlamydial infection (P<.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]

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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, 1995, issue of THE JOURNAL (*JAMA*. 1995;273:1487-1489), 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 2, 3, 4, and 5 should have been citations 3, 4, 5, and 6, respectively; and reference 6 should not have been cited in the sixth paragraph.