reported prior or current IVDU, compared with none of 35 HCV-negative patients.

A surprising finding was that a greater (highly statistically significant) number of HCV-positive patients (12 of 38) died with a CD4 cell count $>200/\text{mm}^3$ than did HCV-negative patients (1 of 35; $P < .002$; $\chi^2$ test). Similarly, a greater (highly statistically significant) number of HCV-negative patients died with a CD4 cell count $<50/\text{mm}^3$ (26 of 35) than did HCV-positive patients (15 of 38; $P < .003$). The HCV-positive patients who died with a CD4 cell count $>200/\text{mm}^3$ died of diverse causes: complications of liver disease (5); cardiomyopathy (2); systemic vasculitis (1); thrombotic thrombocytopenic purpura (1); an overdose of illicit drugs (1); metastatic undifferentiated carcinoma (1); and an unknown cause (1). Twelve HCV-positive patients, 2 of whom had a CD4 cell count $<50/\text{mm}^3$, died as a result of advanced liver disease, compared with only 1 of 35 HCV-negative patients.

Thus it would appear that an incremental mortality was linked to HCV infection, such that (at least in a mostly pre-highly active antiretroviral therapy era) a substantial minority of patients dually infected with HCV and HIV died despite relatively well-preserved CD4 cell counts. Furthermore, death due to advanced liver disease was a numerically important cause for the dually infected patients, despite their relatively young ages. It is unclear whether death was due to HCV directly or to some immunologic perturbation due to chronic HCV infection or an unidentified attribute linked to IVDU.

The absence of a demonstrable acceleration of HIV disease by chronic HCV infection may suggest that the cup is half full. It is half empty as well. HCV contributes substantially to mortality among HIV-infected patients.

**References**


**Reply**

**SIR**—We appreciate the interest of Dr. Jenny-Avital in our recent publication and would like to respond to her question about the representativeness of the cohort. During the period of our study, 676 patients were tested for antibody to hepatitis C virus (HCV); for 136 of the 676 (20.1%), iv drug abuse (IVDU) was an HIV-infection risk factor. The HIV cohort that we followed until May 1997 included 208 iv drug users among 1030 patients (20.2%). Obviously, our study population was representative of the total population. Our cohorts may differ with respect to the prevalence of both IVDU and HCV infection. Of interest, Dr. Jenny-Avital describes her cohort of patients but does not indicate the number for whom IVDU was a risk factor.

The data presented by Dr. Jenny-Avital are of interest and need to be confirmed. We analyzed all patients who died from April 1996 through September 1997 (the same period studied by Dr. Jenny-Avital), and of the 80 patients who died, 23 were HCV-negative, 11 were HCV-positive, and 46 were not tested for HCV status. Among patients who had CD4$^+$ cell counts determined during the 18 months before death, the CD4$^+$ cell counts did not differ by HCV status (for HCV-positive patients, median CD4$^+$ cell count was 6/mm$^3$, within a range of 0–519/mm$^3$; for HCV-negative patients, the median CD4$^+$ cell count was 20/mm$^3$, within a range of 2–570/mm$^3$). Data regarding cause of death are incomplete, but HCV may have contributed to the death of at least 1 patient.

The outcome for HIV-HCV–coinfected patients is obviously complex and will probably change with continued use of potent antiretroviral therapy. Continued prospective studies, including those of patients being treated for HCV infection, are needed.

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**Adverse Effects of Minocycline versus Doxycycline in the Treatment of Lyme Neuroborreliosis**

**SIR**—Both minocycline and doxycycline are completely absorbed when administered orally and seem to reach therapeutic concentrations in CSF, in contrast to the older tetracyclines [1, 2]. As Dr. Cunha points out in his letter [3], earlier studies have shown that minocycline is markedly more lipid soluble than doxycycline [4]. However, some of the reported adverse reac-
tions to minocycline therapy in humans may cause concern. Minocycline seems to cause teeth discoloration even in young adults; other described adverse effects are discoloration of the skin, nails, sclerae, and conjunctivae [1]. These side effects were reported after ≥3 weeks of minocycline therapy, and the risk of adverse effects after shorter courses has not been fully elucidated. Furthermore, vertigo, ataxia, and dizziness have been described during minocycline therapy [5, 6]. These CNS symptoms must be considered a major disadvantage for minocycline therapy, in particular for patients with neurological signs and symptoms, as in Lyme neuroborreliosis.

Tooth and bone deposition is less common with doxycycline than with other tetracyclines [1, 7]. In Sweden, doxycycline has been safely administered to children ≥8 years of age and to adult nonpregnant patients, with very few reports of these side effects. Therefore, although oral minocycline seems to be a candidate for the treatment of Lyme neuroborreliosis from the pharmacological point of view, we would still prefer oral doxycycline therapy because of the risk of adverse reactions to minocycline.

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References

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