

## **Risk Factors for Liver Disease Progression in a Cohort of US Women Coinfected With HIV and HCV**

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**Background:** There are few data on liver disease progression (LDP) among women coinfecting with HIV and HCV. We used serum markers to measure LDP among women in the longitudinal Women's Interagency HIV Study (WIHS) and described risk factors for more rapid LDP.

**Methods:** Level of fibrosis/cirrhosis, as a measure of LDP, was assessed using the SHASTA index, a combination of serum tests of hyaluronic acid (HA), aspartate aminotransferase (AST), and albumin. Subjects included 633 HCV Ab+ WIHS participants (536 HCV+/HIV+ and 97 HCV+/HIV-); about two thirds had been infected with HCV for 15 or more years. LDP was assessed at baseline entry into the study and at the most recent WIHS visit an average 9 years later. Subjects who received antiretroviral therapy (HAART) were also assessed at the 6-month visits both immediately preceding and following the onset of therapy. Thus, the SHASTA was calculated at 2 visits for HIV-negative subjects and for HIV-positive subjects not receiving HAART and at 4 visits for subjects receiving HAART. To assess LDP, annualized changes in SHASTA scores were calculated between each pair of consecutive time points with the difference being divided by the length of time in years. These changes were then regressed on potential risk factors for LDP using generalized estimating equations, adjusting for age, HAART status, baseline CD4+ levels, HCV RNA (positive vs negative), and race/ethnicity.

**Results:** We found that compared with HIV uninfected women, both pre-HAART HIV ( $P = 0.01$ ) and post-HAART HIV ( $P < 0.01$ ) liver disease progression was significantly more rapid in HIV-infected women. Women who reported current use of injection drugs at baseline also had significantly faster liver disease progression compared with never users, but pre-baseline use of injection drugs was not associated with LDP. We also found no faster progression associated with alcohol consumption at baseline; progression rates were significantly slower for subjects who reporting drinking in the past but were non-drinkers at baseline ( $P < 0.01$ ). CD4+ level at baseline were not significantly associated with LDP.

**Conclusions:** Our preliminary analysis suggests that the key factors associated with more rapid LDP are HIV status and current use of injection drugs. Baseline CD4+ counts were not associated with LDP. Future analysis will take into account changes in CD4+ counts and in alcohol and drug use over follow-up.

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