# Association between Alcohol Use and HIV Viral Load

**Evan S. Wu**[1], **David S. Metzger**[2], **Kevin G. Lynch**[1,4], and **Steven D. Douglas**[3]

[1] Division of Biostatistics, Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania

[2] Department of Psychiatry, University of Pennsylvania

[3] Division of Allergy-Immunology, Children's Hospital of Philadelphia and CHOP Research Institute

[4] Department of Psychiatry, University of Pennsylvania

## Introduction

The association between alcohol use and indicators of HIV progression through adherence has been documented (1–3); however, a direct association between alcohol use and HIV viral load and CD4 count has not been established. Immunologic studies have searched for a biological mechanism by which alcohol may directly affect the immune function of HIV positive patients. Alcohol significantly enhanced HIV R5 strain infection in monocyte-derived macrophages, treated ex vivo (4). Samet et al. conducted a seven-year longitudinal study which examined the independent association between HIV progression and alcohol use (5). Heavy alcohol consumption was not directly associated with higher HIV RNA levels among subjects receiving HAART (4).

We examined the relationship between alcohol use and viral load - independent of adherence to HAART. Focus was placed on the HAART using group, since Samet et al. observed that alcohol's effects on the liver could potentially affect the metabolism of ARV medications, which may account for the associations between alcohol and HIV disease progression (6).

## Methods

### Participants

Participants for the study were recruited from HIV treatment settings in Philadelphia between 2003 and 2005. We studied subjects with a history of alcohol and substance abuse, and as a comparison group, also enrolled a sample of non-alcohol users. Inclusion criteria required that participants had a confirmed seropositive status, and were receiving HIV treatment at the time of interview. The trial was approved by the Committee on Human Subjects of the University of Pennsylvania Institutional Review Board.

### Alcohol Use Data

All behavioral data were assessed through self-report. Participant alcohol use was collected using the Risk Assessment Battery (RAB). The RAB measure asked participants of their frequency of alcohol use in the past month with four response options: *everyday, a few times a week, a few times a month,* and *not at all.* Using this measure, we classified our sample

All correspondence to: Steven D. Douglas, M.D., Professor and Associate Chair, Department of Pediatrics, University of Pennsylvania School of Medicine, Children's Hospital of Philadelphia, Children's Hospital of Philadelphia Research Institute, 34th and Civic Center Blvd, Philadelphia, PA 19104, Tel: 215-590-3561, Fax: 215-590-3044, douglas@email.chop.edu.

into "daily drinkers", "regular drinkers", and "non-drinkers". "Daily drinkers" used alcohol *everyday*, "regular drinkers" reported drinking *a few times a week*, and "non-drinkers" reported drinking *a few times a month* or *not at all*.

### HAART Adherence

Self-reported adherence to HAART was measured using a four-point scale (*always, usually, sometimes, never*), all participants were asked the way in which they took their prescribed medication for HIV. Participants currently not on HAART were provided the choice *not on HAART.* Based on this adherence schema, we dichotomized HAART users in our sample into "adherers" (*always* taking their medication), and "non-adherers" (*usually, sometimes,* or *never taking their medicine).*

### Laboratory Data

Our primary outcomes were HIV viral load and absolute CD4 count. Levels of HIV RNA in plasma were quantified by means of RT-PCR assay (Roche Molecular Systems, Branchburg, NJ) according to the manufacturer's protocol. The minimal detectable level of HIV RNA was 50 copies/ml.

### Data Analysis

In order to investigate the adjusted association between viral load and alcohol use, we first used logistic regression to examine the relationship between detectable viral load and daily alcohol use, adjusted for adherence (for HAART users only) and age, race and gender. Linear regression was employed to study the association between CD4 count and daily alcohol use. Finally, we included "regular drinkers" in the alcohol users group, in addition to the "daily drinkers", and reran the above analyses. All statistical analyses were carried out in R 2.9.1 (Free Software Foundation, Inc, Boston, MA, USA).

## Results

### Sample Description

Of our 325 subjects, 241 (74%) were currently on HAART, and 84 (26%) were non-HAART users. Table 1 outlines the outcome measures in our study. 43.4% of HAART users in our sample had a detectable viral load, as compared to 90.5% of the non-HAART users (p<0.01). There were no significant differences in absolute CD4 count between the groups, and nearly 55% of HAART users were always adherent to HAART. With respect to the alcohol measures, 10.6% of the HAART users and 24.1% of the non-HAART subjects consumed alcohol daily in the past month; 44.2% in the HAART group and 36.1% in the non-HAART group did not drink at all in the past month (p=0.24).

### Alcohol Use and Viral Load

The relationship between detectable viral load and alcohol use was examined. Adjusting for adherence and demographic covariates, daily drinkers had nearly a four-fold increase in the odds of detectable viral load (OR=3.81, p=0.01, 95%CI = [1.42, 11.48]), as compared to the remainder of the HAART users in the sample. When we included regular drinkers in our alcohol user group, the adjusted association was no longer significant (OR=1.29, p=0.41, 95%CI = [0.70, 2.33]). For non-HAART users, daily drinking was not associated with significantly higher odds of detectable viral load (OR=2.08, p=0.62, 95%CI = [0.32, 41.20]).

### Alcohol Use and CD4

For HAART users, daily alcohol use, adjusted for adherence and demographic factors, was associated with a −41.59 cells/ml decrease in CD4 (p=0.52, 95%CI = [−169.24, 86.06]).

Among the non-HAART users, daily alcohol use was associated with a 65.6 cells/ml decrease in CD4 (p=0.31, 95%CI = [−193.01, 61.77]).

## Discussion

We investigated the direct relationship between alcohol use and HIV viral load. After controlling for adherence and demographic covariates, there remains a strong relationship between daily alcohol use and detectable viral load among participants on HAART. Daily drinkers in our sample had increased odds of detectable viral load, regular drinkers, defined as consuming a few drinks a week, did not.

### Limitations

This was a cross-sectional study; a longitudinal design may have been more appropriate to investigate the possible association between alcohol use and progression of the HIV virus over time. Givenour limited sample size (n=84) and highly skewed distribution (90.5% detectable viral load) of non-HAART participants, it was difficult to conclude whether the non-significant association for this group was due to the inherent lack of statistical power, or an underlying biological explanation.

### Implications and Suggestions for Future Research

Despite these limitations, our results suggest that there is an association among HAART users between daily alcohol use and viral load. By classifying alcohol users into "daily" and "regular", we also found that while daily consumption was significantly associated with detectable viral load, regular alcohol use was not. This finding is consistent with previous work that moderate alcohol use is associated with different impact on HIV progression and survival than heavy alcohol use (5,7). In contrast, Samet et al. (5) established a significant association between heavy alcohol use and CD4 count among non-HAART users, but not among HAART users. Heavy alcohol use in their study was not associated with higher HIV RNA levels for either medication group (5). Our study established an independent relationship for HAART users between heavy alcohol use and HIV RNA levels, but inconclusive results among participants not on HAART.

In summary, reducing alcohol use, even from daily to a few times a week, may decrease the odds of detectable viral load in patients undergoing HAART therapy. Our results provide evidence in support of Samet's hypothesis (6) that the association between alcohol use and HIV RNA levels is modified by HAART, and the possibility of drug-drug interaction between HAART and alcohol, whereby alcohol impacts the metabolism of HAART as previously suggested.

## Acknowledgments

## References

1. Samet JH, Horton NJ, Meli S, Freedberg KA, Palepu A. Alcohol consumption and antiretroviral adherence among HIV-infected persons with alcohol problems. Alcohol Clin Exp Res. 2004; 28(4): 572–577. [PubMed: 15100608]

2. Chander G, Lau B, Moore RD. Hazardous alcohol use: A risk factor for non-adherence and lack of suppression in HIV infection. J Acquir Immune Defic Syndr. 2006; 43(4):411–417. [PubMed: 17099312]

3. Miguez MJ, Shor-Posner G, Morales G, Rodriguez A, Burbano X. HIV treatment in drug abusers: Impact of alcohol use. Addict Biol. 2003; 8(1):33–37. [PubMed: 12745413]

4. Wang X, Douglas SD, Metzger DS, et al. Alcohol potentiates HIV-1 infection of human blood mononuclear phagocytes. Alcohol Clin Exp Res. 2002; 26(12):1880–1886. [PubMed: 12500113]

5. Samet JH, Cheng DM, Libman H, Nunes DP, Alperen JK, Saitz R. Alcohol consumption and HIV disease progression. J Acquir Immune Defic Syndr. 2007; 46(2):194–199. [PubMed: 17667330]

6. Samet JH, Horton NJ, Traphagen ET, Lyon SM, Freedberg KA. Alcohol consumption and HIV disease progression: Are they related? Alcohol Clin Exp Res. 2003; 27(5):862–867. [PubMed: 12766632]

7. Braithwaite RS, Conigliaro J, Roberts MS, et al. Estimating the impact of alcohol consumption on survival for HIV+ individuals. AIDS Care. 2007; 19(4):459–466. [PubMed: 17453583]

**Table 1**

Summary of Outcome Measures

| Outcome | Group | | P-value |
| --- | --- | --- | --- |
| | HAART User n=241 | Non-HAART User n=84 | |
| Log10 Viral Load, mean ± SD | 1.43 ± 1.83 | 3.58 ± 1.39 | <0.01 |
| Detectable Viral Load (%) | | | |
| Detectable | 43.4 | 90.5 | <0.01 |
| Non-Detectable | 46.6 | 9.5 | |
| CD4, mean ± SD | 442.8 ± 295.6 | 441.3 ± 239.1 | 0.964 |
| Adherence (%) | | | |
| Always take | 54.8 | N/A | |
| Usually take | 38.6 | N/A | |
| Sometimes take | 6.6 | N/A | |
| Never take | 0.0 | N/A | |
| Frequency of Alcohol Use (%) | | | 0.238 |
| Everyday | 10.7 | 24.1 | |
| A few times a week | 27.0 | 24.1 | |
| A few times a month | 18.1 | 15.7 | |
| Not at all | 44.2 | 36.1 | |
| Current DSM-IV Diagnosis (%) | | | 0.341 |
| Present | 30.4 | 44.0 | |
| Absent | 69.6 | 56.0 | |
| Past DSM-IV Diagnosis (%) | | | 0.213 |
| Dependence | 68.5 | 78.6 | |
| Abuse | 9.1 | 4.8 | |
| Absent | 22.4 | 16.7 | |
| Cocaine | | | 0.642 |
| Everyday | 5.3 | 9.7 | |
| A few times a week | 0.9 | 1.2 | |
| A few times a month | 1.8 | 3.6 | |
| Not at all | 92.0 | 85.5 | |