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Association between alcohol consumption trajectories and clinical profiles among women and men living with HIV

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Abstract

Background—Alcohol use is common among persons living with HIV (PLWH). It is unclear how alcohol consumption changes over time and if these changes are associated with clinical profiles.

Objective—We aimed to describe the association between longitudinal patterns of alcohol consumption and the clinical profiles of PLWH.

Methods—Data from the Women's Interagency HIV Study (n = 1123 women) and Multicenter AIDS Cohort Study (n = 597 men) from 2004 to 2013 were utilized. Group-based trajectory models were used to assess alcohol consumption patterns across 10 years. Generalized estimating equations were used to identify associations between clinical factors and alcohol consumption. All analyses were stratified by sex.

Results—Four trajectories of alcohol use were identified in women and men (women: abstinent 38%, low: 25%, moderate: 30%, heavy: 7%; men: abstinent 16%, low: 69%, moderate: 9%, heavy: 5%). The Framingham Risk Score (women: adjusted odds ratio [AOR] 1.07, 95% confidence interval [CI] 1.04–1.09), years on ART (women: AOR 1.02, CI 1.00–1.05; men: AOR 1.05, CI 1.01–1.09), suboptimal ART adherence (men: AOR 1.23, CI 1.07–1.42), and unsuppressed viral load (women: AOR 1.82, CI 1.56–2.13; men: AOR 1.36, CI 1.17–1.58) were associated with increased odds for moderate drinking. The Framingham Risk Score (women: AOR 1.10, CI 1.07–1.14; men: AOR 1.12, CI 1.06–1.20), sub-optimal adherence (women: AOR 1.25, CI 1.04–1.51),

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Conflict of interest

The authors declare that they have no conflict of interest.

and unsuppressed viral load (women: AOR 1.78, CI 1.42–2.24) were associated with increased odds for heavy drinking.

Conclusions—Clinicians should consider screening patients for alcohol consumption, particularly if patients have comorbid medical conditions, suboptimal antiretroviral adherence, and/or detectable viral load.

Keywords

HIV; alcohol use; longitudinal; MACS; WIHS; sex; women; men

Introduction

Alcohol use is common among persons living with HIV (PLWH) and is reported among 39–81% (1–5). Prevalence of heavy drinking has been reported in as much as 25–45% (1,6,7) of PLWH, with alcohol dependence ranging from 5.5 to 10% (4,8–10). Alcohol consumption, in general, is negatively associated with completing the steps of the HIV care continuum (11) and heavy alcohol use is associated with poor retention in HIV care and lower visit adherence, compared to those who do not drink[1]. Likewise, heavy alcohol consumption is associated with decreased antire troviral (ART) adherence (9,12–14), lower CD4 + T-cell count (9,15), and increased viral load (7). Aside from the relationship between alcohol consumption and ART adherence, alcohol abuse has been linked specifically to HIV progression through alteration of viral infectivity, inflammatory biomarkers, immune response, and tissue injury (16,17). Heavy drinking in this population is also associated with engagement in risky health behavior, such as cigarette (18,19) and substance use (20), which can lead to other chronic illnesses. Some studies have found that PLWH who use alcohol have increased chronic comorbidity (5,10,21,22), while other studies have found no such association (3,23–25). Others have found a J-curve association between alcohol consumption and risk for chronic illness. For example, Wandeler et al. (3) found that, among PLWH, low and moderate drinkers had significantly lower risk for cardiovascular disease events or death, compared to non-drinkers. While similar findings have been shown in the general population, emerging evidence suggests that PLWH may be more affected by the harmful sequela of alcohol use when compared to similar or even lower levels of use among uninfected groups (26–28). Because many of the aforementioned studies are cross-sectional, it is unclear whether moderate and heavy drinking leads to chronic illness or if alcohol use is a coping response to such illness.

The current literature is limited, with generally few studies focusing on long-term patterns of alcohol use and with cross-sectional studies differing on measurements of alcohol use. Longitudinal studies of alcohol use patterns among PLWH have focused primarily on dichotomous measures of hazardous or heavy alcohol use, which limit the variability of alcohol consumption and can result in stagnated patterns over time (29–31). These studies have also been limited by relatively short follow-up (6 months–2 years) (29,31) or have synthesized longitudinal patterns by using lifetime recall of alcohol use phases (30). To our knowledge, there is limited research on levels of alcohol use aside from hazardous/heavy use over long periods of follow-up. Cook et al. (32) conducted a group-based trajectory model (GBTM) of self-reported number of drinks consumed per week to inform emerging patterns

among HIV-positive women in the Women's Interagency HIV Study (WIHS) from 1996 to 2006. This study found five trajectories of drinking, three of which described changing drinking behavior over time. Namely, the authors found the following groups: women who were persistent heavy drinkers (3%), women who reduced from heavy to non-heavy drinking (4%), women who increased from moderate-to-heavy drinking (8%), women who were persistent non-heavy drinkers (36%), and non-drinkers (49%). These data, however, describe drinking patterns in the first half of the 20-year cohort study and may not be relevant to drinking behavior in the post highly active ART therapy era (the standard treatment to reduce HIV viral replication, consisting of a combination of drugs). Lastly, Marshall et al. (33) conducted a longitudinal analysis of patterns of the Alcohol Use Disorder Identification Test-Consumption questionnaire (AUDIT-C) (8) score among HIV-positive men who have sex with men (MSM) from 2002 to 2010 of the Veteran's Aging Cohort Study. This study found four stable trajectories (men with consistently hazardous drinking [12%; AUDIT-C score of about 6–7], potentially hazardous [35%; AUDIT-C score of about 3–4], low risk [36%; AUDIT-C score of about 1–2], and infrequent [16%; AUDIT-C score around 0]), perhaps due to the use of the somewhat prescriptive AUDIT-C score, which is used to identify alcohol use disorders (score ranging from 0 to 12) and has lower variability than that of self-reported number of drinks per week, thus limiting the detection of change in drinking. While these two studies were conducted in different populations and examined inconsistent predictors of heavy alcohol use, illicit drug use was distinctly associated with heavy consumption.

In summary, a gap exists regarding alcohol use changes over time among PLWH. Further, it is unclear if there are significant clinical factors associated with long-term moderate and heavy alcohol consumption by gender. Associated factors of alcohol consumption patterns would provide clinicians with the means to identify those with the greatest need for early intervention and alcohol abuse treatment. Given these gaps in the literature, the goals of this analysis are to (1) describe patterns of alcohol consumption among PLWH from 2004 to 2013 by gender and (2) assess the association between time-varying and -invariant clinical factors and long-term heavy and moderate alcohol consumption. By utilizing reported number of drinks per week, we hypothesized that distinct patterns will emerge that are descriptive of stable (i.e., consistent abstinent, consistent moderate, and consistent heavy) and changing alcohol use behavior (i.e., abstinent to moderate or heavy drinking; heavy to moderate or abstinence) over time. We also hypothesized that clinical factors would be identified, specifically by gender, as important predictors of long-term moderate and heavy alcohol consumption. Specifically, we hypothesized that those with poor clinical profiles (e.g., diabetes, increased BMI status and Framingham Risk Score, lower CD4 count, and higher HIV viral load) would be more likely to be heavy or moderate drinkers, compared to those who are abstinent or low drinkers. While clinical associations of longitudinal alcohol consumption were the main focus of this analysis, the Bio-Psycho-Social theoretical framework (34) was used to conceptualize other non-clinical factors needed for analytical adjustment. According to the Bio-Psycho-Social model of health, multiple biological, psychological, and social factors interact to impact indices of health and illness. Because long-term patterns of alcohol consumption are complex, with multiple biological (e.g., age, gender, clinical factor), psychological (e.g., depressed mood, other substance use), and

social (e.g., race/ethnicity, income) predisposing factors, this model may be used as a framework for understanding risk factors for alcohol consumption.

Methods

Participants

The Multicenter AIDS Cohort Study (MACS) (35–37) and WIHS (38,39) are well-established, national multicenter cohorts of MSM and of women, respectively, living with or at risk for HIV-infection. Participants from MACS were recruited from the following metropolitan areas: Baltimore, MD; Washington, DC; Chicago, IL; Pittsburgh, PA; Los Angeles, CA. Participants from WIHS were recruited from the following metropolitan areas: Brooklyn and Bronx, NY; Washington, DC; Chicago, IL; Los Angeles and San Francisco, CA. The MACS recruited MSM across three waves, in 1984–1985 (n = 4954), 1987–1991 (n = 668), and 2001–2003 (n = 1350). Women were recruited in WIHS across two waves, in 1994–1995 (n = 2625) and 2001–2002 (n = 1141). The data from these studies were collected from structured interviews, and standardized physical, psychological, and laboratory assessments. HIV status was assessed by enzyme-linked immunosorbent assay (ELISA) with Western blot for confirmation at baseline for HIV-positive participants, and semi-annually for HIV-participants. Sero-conversion was confirmed by testing HIV-participants at each semi-annual visit using the aforementioned tests. Written informed consent was obtained prior to each semi-annual assessment for both cohorts. The questionnaires are available online for MACS at www.statepi.jhsph.edu/macs/forms.html and for WIHS at https://statepiaps.jhsph.edu/wihs/index-forms.htm.

The current study utilized data from participants of the cardiovascular sub-studies (enrollment in 2004) of the MACS and WIHS, to understand the associations between clinical profiles including cardiovascular disease risk factors (i.e., Framingham Risk Score, BMI, diabetes) and alcohol consumption prior to cardiovascular disease or related events. Therefore, this study focuses on alcohol consumption patterns from 2004 to 2013. The cardiovascular sub-study enrolled a subset of HIV-positive WIHS participants (n = 1,321), aged 25–60 years and the MACS enrolled a subset of HIV-positive MSM (n = 828), over 40 years of age and under 300 lbs. Participants who seroconverted during the study and those with less than 4 alcohol consumption assessments were excluded (WIHS n = 198; MACS n = 231). The median person-years of follow-up between 2004 and 2013 were 6.2 years [interquartile range (IQR): 6.0–7.5 years] for WIHS participants and 8.5 years (IQR: 8.0–10.0 years) for MACS participants.

All MACS and WIHS participants provided written informed consent for overall study participation, and this specific analysis was approved by the Institutional Review Board at the University of Florida.

Main outcome measure

Alcohol consumption was measured via self-report by asking about the average frequency (number of days per week) and quantity (number of drinks per drinking day) of use. The average number of drinks per week was calculated by multiplying the frequency by the

quantity. In the GBTM, alcohol consumption was modeled as a continuous variable; in the GEE model, consumption was categorized as abstinence to low (< 1 drink per week), moderate (1–7[14] drinks per week for women [men]), or heavy use (> 7[14] drinks per week for women [men]) (40,41).

Independent variables

Clinical/biological—Age was assessed in years, using participants' self-reported date of birth. Use of ART was reported at each visit and weighted by the reported adherence of ART (42). Cumulative ART was calculated by adding the weighted ART use variable to reflect years of ART use by the end of the 10-year follow-up period. Optimal ART adherence was defined as taking ≥ 95% of prescribed ART doses at each semiannual visit, as this has previously been associated with sustained viral suppression (43,44). Plasma HIV RNA viral load and CD4 + T-cell count were measured, semi-annually, using standard laboratory techniques. HIV RNA viral load was subsequently categorized as undetectable (< 200 copies/mL) or detectable (≥ 200 copies/mL) (45); CD4 + T-cell count was categorized as high (≥ 500 cells/mm^3), medium (300–500 cells/mm^3), or low (< 300 cells/mm^3).

Diabetes was dichotomized as having ever been diagnosed with diabetes at any time during follow-up versus no history of diabetes. The Framingham Risk Score (46) was calculated, using the gender-based algorithms, including the following variables: age, total cholesterol, high-density lipoprotein (HDL) cholesterol, systolic blood pressure, and smoking status. Therefore, we did not adjust for these variables outside of this risk score. The Framingham Risk Score values range from negative to positive, with negative values indicating low risk and positive values indicating high risk. BMI was based on weight and height, and participants were categorized as being underweight (BMI < 18.5 kg/m^2), normal (18.5–24.9 kg/m^2), and overweight (> 24.9 kg/m^2).

Psychological—Depressive symptoms were assessed at each semi-annual visit with the Center for Epidemiologic Studies Depression Scale (CES-D) (47). Some research has found that utilizing the score of 16 or greater may inflate the rate of depression among PLWH, due to the overlapping somatic symptoms that may be present due to HIV-infection (48). Therefore, a score of 23 or greater was considered probable depression (49). Self-reported illicit drug use was dichotomous and measured at each visit by asking if participants used any of the following: crack or any form of cocaine; uppers (including crystal, methamphetamines, speed, ice); heroin or other opiates.

Social—Race/ethnicity was self-reported and categorized as non-Hispanic white, non-Hispanic black, and other races (Hispanic; Asian/Pacific Islander; Native American/Alaskan). Annual income was self-reported at each visit and categorized, based on natural cutoffs in the data, as < $10,000, $10,000–$30,000, or ≥ $30,000 a year.

Data analyses

Group-based trajectory modeling—We present two sets of longitudinal analyses to carry out our study aims. Aim 1 was to describe patterns of alcohol consumption over time. In this first longitudinal analysis, we conducted GBTM in several modeling steps. In the first

modeling step, we assessed linear patterns of 3–5 groups, as suggested by previous research (29,32,33). Models with a group(s) with less than 5% of the sample were rejected. Once the best-fitting number of groups was identified, we assessed the best-fitting change structure for each group (linear, quadratic, cubic). Goodness of fit was assessed at each step using the Akaike information criteria (AIC) and Bayesian information criteria (BIC; smaller the values, better the model), group posterior probabilities (PP ≥ 0.7 is indicative of sufficient internal reliability), and mean model entropy (≥ 0.7 is optimal; summed PP/number of groups). The PP estimate is the probability that any one group-based trajectory adequately captures the individual patterns. Therefore, an individual pattern was assigned into the group pattern with the highest probability of group membership. Models with PP and/or model entropy values < 0.7 were rejected (50). The 95% confidence intervals (CIs) of the resulting patterns were used to qualitatively assess the stability of the trajectories. Models with small CIs of trajectories were favored over wide CIs. The variance around the intercept and slope means were set to zero.

Generalized estimating equations—Aim 2 was to assess longitudinal associations between clinical factors and moderate and heavy alcohol consumption compared to abstinent/low use. In the second set of longitudinal analyses, we estimated the association of clinical factors of alcohol consumption outside of the GBTM using generalized estimating equations (GEEs). Using repeated measures of alcohol consumption and clinical factors, multivariate (GEE) were conducted, stratified by gender. Several covariance structures were tested (independent—responses are uncorrelated; exchangeable—responses are equally correlated; autoregressive—correlation between responses decrease over time) (51). We used the quasi-information criterion (QIC) to compare model fit between covariance structures and chose the model with the smallest QIC. Model fit statistics between GBTM and GEE models are available as supplemental data.

Univariate and bivariate analyses were conducted to assess frequencies and proportions of clinical factors and covariates. Clinical factors were considered significantly associated with alcohol consumption at the $p < .05$ level.

Missing data

Missing data were assessed and variables associated with missing data were identified. In MACS, 63% had less than 10% missing data. Missing greater than 10% was significantly associated with greater illicit drug use (27% vs. 19%), higher CD4 + T-cell count (41% ≥ 500 cells/mm^3 vs. 22%), less diabetes (29% vs. 38%), lower ART adherence (62% vs. 71%), cumulative years on ART (7.7 vs. 10.5), and Framingham Risk Score (10.7 vs. 11.3) compared to those with less than 10% missing. In WIHS, 64% had less than 10% missing data. Missing greater than 10% was significantly associated with greater illicit drug use (11% vs. 6%), lower diabetes (21% vs. 26%), CD4 + T-cell count (32% ≥ 500 cells/mm^3 vs. 43%), and fewer years on ART (9.3 vs. 13.6). Missingness was not associated alcohol consumption in MACS or WIHS. In order to address the potential for bias, we conducted multiple imputation, averaged across 10 imputations.

All statistical analyses were conducted using SAS 9.4 (SAS Institute, Inc., Cary, NC).

Results

Baseline characteristics by cohort are presented in Table 1

Alcohol consumption trajectories

A four-group trajectory model emerged as the best-fitting model for HIV-positive women (Figure 1; model entropy, 0.92). Alcohol consumption patterns were labeled as “abstinent” (38%; PP 0.94, IQR .93–0.99; little to zero drinks per week during most of 10 years), “low” (25%; PP 0.86, IQR .75–0.98; > 0 and < 2 drinks per week during most of 10 years), moderate (30%; PP 0.92, IQR 0.89–1.00; ≥ 2–7 drinks per week during most of 10 years), and “heavy” (7%; PP 0.97, IQR 0.99–1.0; > 7 drinks per week during most of 10 years).

A four-group trajectory model emerged as the best-fitting model for HIV-positive men (Figure 2; model entropy, 0.97): abstinent (16%; PP 0.96, IQR 0.99–1.00; little to zero drinks per week during most of 10 years), low (69%; PP 0.99, IQR .99–1.0; > 0 and < 4 drinks per week during most of 10 years), moderate (9%; PP 0.97, IQR .99–1.0; ≥ 4–14 drinks per week during most of 10 years), and heavy (5%; PP 0.99, IQR .99–1.0; >14 drinks per week during most of 10 years).

Multivariate analysis of clinical factors on alcohol consumption among women

Moderate drinking—Multivariable analyses are shown in Table 2. Controlling for race, annual income, age, probable depression, and illicit drug use, each increased unit of Framingham Risk Score was associated with 1.07 times higher odds for moderate drinking (CI 1.04–1.09, $p < .001$), compared to abstinent/low use. Each year of ART use was associated with 1.02 times higher odds for moderate use (CI 1.00–1.05, $p = .05$) and unsuppressed viral load was associated with 1.82 times higher odds for (CI 1.56–2.13, $p < .001$) for moderate drinking. Women with CD4 + T-cell count < 300 had 0.57 times lower odds for moderate drinking (CI 0.45–0.72, $p < .001$), compared to CD4 + T-cell count ≥ 500.

Heavy drinking—Controlling for race, annual income, age, probable depression, and illicit drug use, each increased unit of Framingham Risk Score was associated with 1.10 times higher odds for heavy drinking (CI 1.07–1.14, $p < .001$), compared to abstinent/low use. Suboptimal adherence was associated with 1.25 times higher odds for heavy drinking (CI 1.04–1.51, $p = .02$) and unsuppressed viral load was associated with 1.78 times higher odds for (CI 1.42–2.24, $p < .001$) for heavy drinking.

Multivariate analysis of clinical factors on alcohol consumption among men

Moderate drinking—Multivariable analyses are shown in Table 3. Controlling for race, annual income, age, probable depression, and illicit drug use, underweight BMI status and diabetes were associated with 0.54 (CI 0.45–0.64, $p < .001$) and 0.56 (CI 0.41–0.75, $p < .001$) times lower odds for moderate drinking, respectively, compared to abstinent/low use. Each year of ART use was associated with 1.05 times higher odds for moderate use (CI 1.01–1.09, $p = .02$) for moderate drinking. Suboptimal ART adherence was associated with 1.23 times increased odds (CI 1.07–1.42, $p = .004$) and unsuppressed viral load was associated with 1.36 times higher odds for (CI 1.17–1.58, $p < .001$) for moderate drinking.

Men with CD4 + T-cell count < 300 or 300–500 had 0.82 (CI 0.72–0.93, $p = .003$) and 0.46 (CI 0.38–0.55, $p < .001$) times lower odds for moderate drinking, respectively, compared to CD4 + T-cell count ≥ 500.

Heavy drinking—Controlling for race, annual income, age, probable depression, and illicit drug use, underweight BMI status and diabetes were associated with 0.59 (CI 0.38–0.91, $p = .02$) and 0.43 (CI 0.23–0.83, $p = .01$) times lower odds for heavy drinking, respectively, compared to abstinent/low use. Each increased unit of the Framingham Risk Score was associated with 1.12 times higher odds for heavy drinking (CI 1.06–1.20, $p < .001$).

Discussion

We aimed to describe alcohol consumption trajectories over time and to assess the longitudinal associations between clinical factors and moderate and heavy alcohol consumption. All patterns identified characterized steady levels of alcohol consumption, with very little change over time. These findings are consistent with existing literature finding mainly stable consumption patterns using dichotomous measures of hazardous or heavy alcohol consumption (29,33). While women tended to drink less, in general, they had higher membership in the moderate drinking and slightly higher membership in the heavy drinking patterns than men. Given the fact that it is relatively unknown whether moderate use confers health benefits or harms among PLWH, these results could suggest that women are a target for prevention/intervention strategies. This is specifically important when considering the lower threshold of number of drinks needed for intoxication (26) and given the evidence that only 30 drinks per month (i.e., moderate use) is associated with increased risk for physical injury and death in this population (27), far exceeding risk compared to the 70 drinks per month needed for similar impact among HIV-individuals. Also of significance is the lack of a decreasing trajectory from the heavy pattern across both men and women, suggesting that once heavy consumption becomes relatively common, this behavior remains overtime.

Results from the multivariate GEE models suggest that there are significant longitudinal clinical associations of moderate and heavy consumption that may help distinguish individuals for prevention and/or early intervention. The Framingham Risk Score was associated with increased odds for moderate and heavy alcohol consumption among women and for heavy drinking in men. Diabetes, however, was associated with decreased odds for moderate and heavy drinking among men, which may be due to recommendations from care providers to reduce or stop drinking due to declining health or risk for clinical illness. Conversely, this association could also be indicative of a protective effect of alcohol consumption on diabetes, described in research among the general population (52–54). Suboptimal ART adherence was associated with increased odds for moderate consumption in men and for heavy consumption in women. Furthermore, controlling for ART adherence, having an HIV RNA viral load of 200 or greater was associated with increased odds for membership in the moderate and heavy drinking in women, and moderate drinking in men. This is consistent with previous research indicating that alcohol abuse is linked to HIV progression through alteration of viral infectivity (7), inflammatory biomarkers, immune response, and tissue injury (16,17). Conversely, women and men with lower CD4 count were

less likely to be moderate drinkers, compared to the abstinent/low group, suggesting a protective effect.

The readers should consider some limitations of the current study. First, alcohol consumption quantity and frequency were assessed via self-report and is subject to recall and social desirability biases, which likely resulted in underestimated reports of alcohol consumption. Second and previously mentioned, the CES-D cutoff score of 16 or greater may inflate depression estimates among PLWH, which is why we chose a cutoff of 23 to indicate probable depression. Other methods have been proposed, such as the removal of somatic symptoms from the scale, resulting in an 11-item CES-D score. This method, however, was suggested based on the results of a factor analysis of one moderately sized sample. Also, recent studies among PLWH found that removing misfitting items resulted in little improvement in the validity of the CES-D (49,55). The CES-D is a standardized tool that has been shown to have good reliability and validity in detecting significant depressive symptoms in a variety of populations, including persons living with and without HIV. Third, there are significant demographic differences between the WIHS and MACS cohorts, making direct comparisons of stratified GBTM analyses difficult. It is possible that any differences found may be due to differences in social factors between these cohorts. Fourth, we restricted our analyses to participants with at least four alcohol consumption assessments in order to estimate stable trajectory models. Therefore, it is possible that different trajectories could have emerged had we not excluded these participants. Fifth, those with heavy drinking and comorbidities may have been more likely to drop out of the study or die. This could have affected the results relating to alcohol consumption and clinical conditions, making heavy consumption seem less common among those with diabetes or progressed HIV-infection, when, in fact, there may have been a true positive association. Lastly, GBTM is a semi-parametric and probabilistic model that estimates grouped trajectories of the most similar individual patterns. Therefore, each trajectory does not fully describe the individual-level patterns contained within them and should not be considered absolute.

In summary, the current study added to existing literature on the proportion of HIV-positive persons who consume alcohol at specific levels, particularly moderate and heavy consumption. Because alcohol consumption patterns were not limited to characterize only heavy use, we were able to describe the course of different levels of alcohol use over time. The results also reveal clinical characteristics and comorbidities that can be used to identify those at risk for moderate and heavy consumption. The US Preventive Services Task Force recommends that clinicians assess all adults aged 18 years and older for alcohol misuse, and to provide support to reduce risky alcohol consumption (6). Further, several screening and brief intervention tools have been developed specifically for clinical use in the general and specific clinical populations (56). In line with these recommendations, clinicians should consider screening all patients for alcohol consumption, particularly if patients report clinical comorbidities, suboptimal ART adherence, and if patients have unsuppressed viral load. Clinicians could also consider assessing moderate alcohol consumption, as this study found detrimental associations of moderate use on adherence and viral load, particularly among women.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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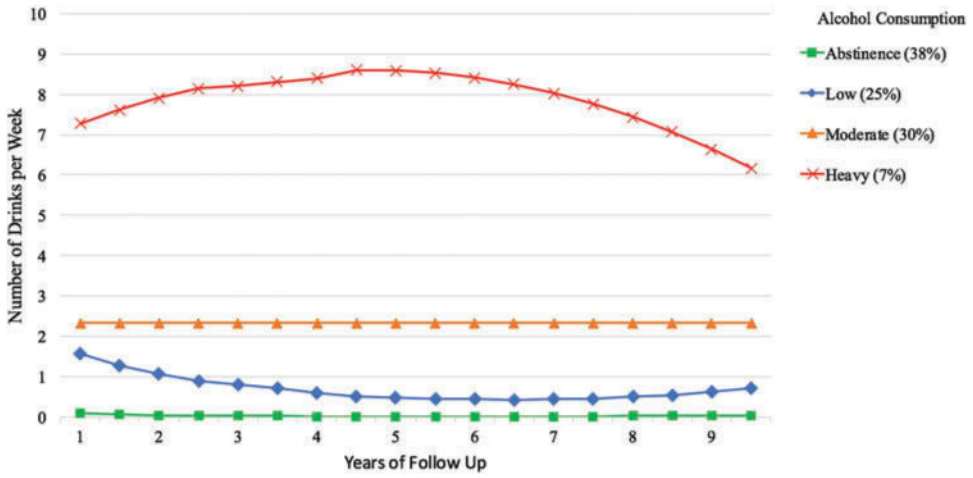

Figure 1.

Trajectories of alcohol consumption among 1,123 HIV+ women in the Women's Interagency HIV Study (WIHS).

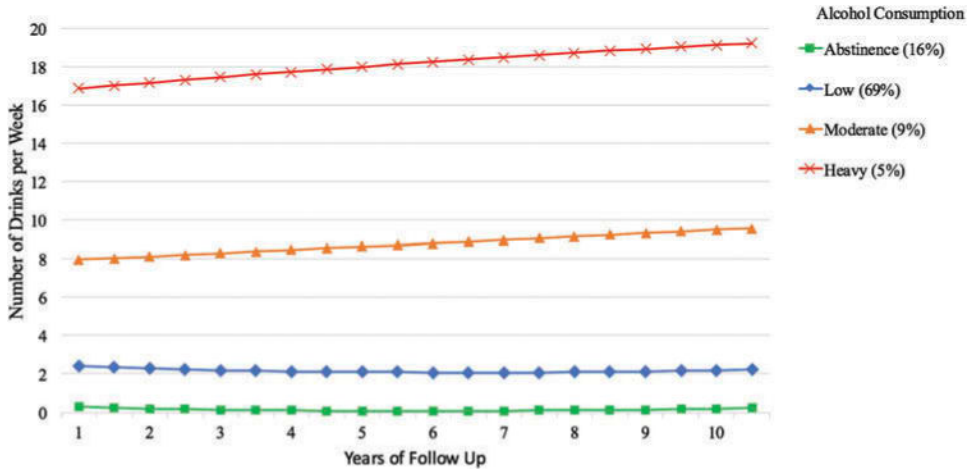

Figure 2.

Trajectories of alcohol consumption among 597 HIV+ men in the Multicenter AIDS Cohort Study (MACS).

Table 1

Baseline characteristics of persons living with HIV by cohort.

Baseline Characteristics	WIHS (*N* = 1123)	MACS (*N* = 597)
	No. (Column %)	
Race		
White	248 (22)	311 (52)
African-American/Black	676 (60)	225 (38)
Other	199 (18)	61 (10)
Age (continuous), mean (SD)	45.0 (7.6)	56.9 (7.7)
Annual Income		
< $10,000	551 (51)	150 (31)
$10,000–$30,000	339 (31)	126 (26)
≥ $30,000	189 (18)	211 (43)
Probable depression		
No	895 (80)	514 (86)
Yes	228 (20)	83 (14)
Illicit drug use		
No	990 (92)	400 (78)
Yes	81 (8)	112 (22)
Ever diagnosed with diabetes		
No	829 (74)	388 (65)
Yes	297 (26)	209 (35)
Body mass index		
< 18.5	487 (43)	133 (22)
18.5–24.9	225 (20)	228 (38)
> 25.0	411 (37)	236 (40)
HIV RNA viral load		
< 200 copies/mL	628 (57)	417 (70)
> 200 copies/mL	495 (44)	180 (30)
CD4 + T cell count		
≥ 500 cells/mm^3	443 (39)	264 (44)
300–500 cells/mm^3	323 (29)	157 (26)
< 300 cells/mm^3	357 (32)	176 (30)
HIV ART adherence		
< 95%	514 (46)	163 (32)
≥ 95%	609 (54)	353 (68)
Cumulative ART exposure, mean (SD), years	*12.1 (5.0)*	*9.4 (4.0)*
Framingham Risk Score, mean (SD)	*8.4 (6.0)*	*11.1 (3.3)*

Note. WIHS, Women's Interagency HIV Study; MACS, Multicenter AIDS Cohort Study; ART, antiretroviral therapy.

Table 2

Multivariable analysis of associated factors of the moderate alcohol use compared to the low/abstinent alcohol use among women living with HIV.

	Moderate			Heavy		
	AOR	***95% CI***	***P Value***	***AOR***	***95% CI***	***P Value***
Race (Ref = White)						
African American/Black	0.84	0.64–1.11	.23	0.97	0.64–1.46	.88
Other	0.81	0.57–1.17	.26	0.48	0.26–0.89	.02
Annual income (Ref = ≥ 30,000)						
< $10,000	0.77	0.60–0.98	.04	0.76	0.52–1.12	.17
$10,000–$30,000	0.76	0.60–0.98	.04	0.72	0.48–1.08	.11
Age, years	0.96	0.94–0.97	< .001	0.96	0.93–0.99	.01
Probable depression (Ref = No)	1.14	0.93–1.39	.19	1.46	1.13–1.90	.004
Illicit drug use (Ref = No)	2.07	1.54–2.77	< .001	4.46	3.09–6.42	< .001
BMI status (Ref = Normal)						
Underweight	0.90	0.68–1.18	.44	0.76	0.51–1.14	.19
Overweight	0.81	0.62–1.05	.11	0.80	0.53–1.21	.30
Diabetes (Ref = No)	0.90	0.71–1.15	.40	0.78	0.52–1.18	.24
Framingham risk score	1.07	1.04–1.09	< .001	1.10	1.07–1.14	< .001
Suboptimal adherence (Ref ≥ 95%)	1.12	0.97–1.30	.12	1.25	1.04–1.51	.02
Cumulative ART exposure, years	1.02	1.00–1.05	.04	0.99	0.96–1.02	.68
Unsuppressed viral load (Ref < 200)	1.82	1.56–2.13	< .001	1.78	1.42–2.24	< .001
CD4 + T-cell (Ref = ≥ 500)						
300–500	0.90	0.75–1.08	.25	0.85	0.63–1.14	.28
< 300	0.57	0.45–0.72	< .001	0.81	0.57–1.14	.23

Note. AOR, adjusted odds ratio; 95% CI, 95% confidence interval; ART, antiretroviral therapy.

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Table 3

Multivariable analysis of associated factors of the moderate alcohol use compared to the low/abstinent alcohol use among men living with HIV.

	Moderate			Heavy		
	AOR	***95% CI***	***P Value***	***AOR***	***95% CI***	***P Value***
Race (Ref = White)						
African American/Black	0.59	0.42–0.81	.001	0.75	0.40–1.42	.38
Other	0.55	0.35–0.88	.01	0.96	0.38–2.41	.94
Annual income (Ref = ≥ 30,000)						
< $10,000	0.75	0.62–0.90	.002	1.97	0.35–0.74	< .001
$10,000–$30,000	0.76	0.69–0.85	< .001	0.43	0.34–0.55	< .001
Age, years	0.98	0.96–1.00	.07	0.96	0.93–1.00	.05
Probable depression (Ref = No)	1.22	1.06–1.40	.005	1.61	1.28–2.34	< .001
Illicit drug use (Ref = No)	1.51	1.27–1.79	< .001	1.73	1.28–2.34	< .001
BMI status (Ref = Normal)						
Underweight	0.54	0.45–0.64	< .001	0.59	0.38–0.91	.02
Overweight	0.92	0.79–1.09	.35	0.97	0.72–1.30	.82
Diabetes (Ref = No)	0.56	0.41–0.75	< .001	0.43	0.23–0.83	.01
Framingham risk score	1.03	1.00–1.07	.06	1.12	1.06–1.20	< .001
Suboptimal adherence (Ref ≥ 95%)	1.23	1.07–1.42	.004	1.03	0.79–1.35	.81
Cumulative ART exposure, years	1.05	1.01–1.09	.02	0.97	0.90–1.05	.45
Unsuppressed viral load (Ref < 200)	1.36	1.17–1.58	< .001	1.24	0.89–1.73	.21
CD4 + T-cell (Ref = ≥ 500)						
300–500	0.82	0.72–0.93	.003	1.11	0.86–1.41	.45
< 300	0.46	0.38–0.55	< .001	0.79	0.55–1.15	.23

Note. AOR, adjusted odds ratio; 95% CI, 95% confidence interval; ART, antiretroviral therapy.

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