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Alcohol and Race/Ethnicity Elicit Different Changes in Lipid Profiles in HIV-Infected Individuals Receiving Highly Active Antiretroviral Therapy

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Abstract

This longitudinal study examined the impact of alcohol consumption (88 hazardous and 76 nonhazardous drinkers) and race/ethnicity on lipid profiles in individuals starting highly active antiretroviral therapy (HAART). At baseline, Whites and Hispanics had the most adverse lipid profiles, whereas Blacks had the least atherogenic. Whites and Hispanics showed higher increases in cholesterol (W = 11%; H = 6%), triglycerides (W = 40%; H = 24%), and low-density lipoprotein (10%) than Blacks (cholesterol = 4%; triglycerides = 9%; low-density lipoprotein = 4%). Hazardous alcohol consumption was correlated with increased lipids in each group. Hispanics had a clear trait risk for hypertriglyceridemia with HAART (1.9-fold) and with hazardous drinking (3.2-fold; p = .04). The highest risk for hypertriglyceridemia was found in heavy drinkers (3.75-fold; p = .05). Results underscore the importance of an alcohol/race interactive effect on HAART-associated dyslipidemia and the need for assessment and treatment of alcohol disorders.

Keywords

alcohol; antiretrovirals; cardiovascular disease; triglycerides

Dyslipidemia, consisting of hypertriglyceridemia, low high-density lipoprotein (HDL) cholesterol, and elevated low-density lipoprotein (LDL) cholesterol, is being observed with increasing frequency among persons living with HIV (PLWH) (Carr et al., 1998; Gerber et al., 2005; Gkrania-Klotsas & Klotsas 2007; Miller et al., 1998; Viraben & Aquilina, 1998). It has been reported in 40% to 80% of those on highly active antiretroviral therapy (HAART) and associated in particular with protease inhibitors (PI) or nonnucleoside reverse transcriptase inhibitors (Gkrania-Klotsas & Klotsas, 2007). Although the mechanisms underlying HAART-associated dyslipidemia have not been fully elucidated, they seem to involve hepatic overproduction of LDL, and to a lesser degree, impaired clearance (Calza, Manfredi, & Chiodo, 2004; Fauvel et al., 2001). Others argue possible interference with the

levels and activity of gene expression in lipoprotein receptors (Fauvel et al., 2001; Foulkes et al., 2006).

The high prevalence of lipoprotein disturbances observed in HIV-infected patients along with a rapid onset of metabolic abnormalities have contributed to an elevated risk of cardiovascular disease (CVD) among PLWH (Bozkurt, 2004; Friis-Moller et al., 2007). Modified diet, increased physical activity, and even moderate alcohol consumption may be recommended to patients with lipoprotein disturbances in the general population (Després et al., 2000). Such recommendations are based on published data showing that individuals who consume alcohol not only have higher serum levels of HDL cholesterol but also have lower levels of LDL cholesterol than abstainers (de Jong, de Goede, Griep, & Geleijnse, 2008). Unfortunately, alcohol effects on lipid abnormalities have not been evaluated in HIV-infected individuals. Moreover, under these conditions (HIV-infected and on HAART), it is not surprising that adding an additional stressor such as alcohol use will result in further derangement of lipid metabolism. Thus, strategies that identify individuals at increased risk of HAART-related metabolic complications are likely to facilitate more tailored selection of HAART regimens and the early use of appropriate preventive CVD therapies.

Despite a well-described relationship between race/ethnicity and lipoproteins in the general population (Albu, Murphy, Frager, Johnson, & Pi-Sunyer, 1997; Després et al., 2000; Lovejoy, de la Bretonne, Klemperer, & Tulley, 1996; Schmidt et al., 1996), ethnicity has rarely been considered in the development of metabolic complications in PLWH (Bausserman, Tashima, DiSpigno, Maceroni, & Carpenter, 2004; Foulkes et al., 2006) and has not been studied regarding alcohol use. Such differences may be of specific relevance in HAART-associated dyslipidemia, given the multiethnic distribution of PLWH and the prevalence of alcohol abuse.

Subjects and Methods

Sampling

A gender and racially diverse HIV-infected population was screened in January 2005 by the University of Miami Miller School of Medicine clinics affiliated with Jackson Memorial Hospital. HIV-infected participants, 18 to 55 years of age, were eligible for the study if they were starting a HAART regimen. Those who were not HAART-naive were eligible for the study if they were without HAART for at least 6months.The authors included 6-month interruptions because they expected a rebound of the outcome measurements within that time, namely a CD4 drop, viral load increase, and reduction in medication toxicity. The definition of HAART used for analyses was guided by published recommendations (Panel on Antiretroviral Guidelines for Adults and Adolescents, 2008).

Those who expressed willingness to participate (98%) and provided a written informed consent and a medical release were enrolled and followed for 6 months. No significant differences in sociodemographic variables existed between enrolled and nonenrolled participants. The institutional review board at University of Miami Miller School of Medicine approved the study.

Study Subgroups

After eligibility was established, participants completed a detailed alcohol drinking assessment (CAGE Assessment, Alcohol Use Disorders Identification Test [AUDIT], Alcohol Dependance Scale), specifying the number of days per week they drank and the quantity they consumed each time. The authors defined standard portions as a glass, bottle, or can of beer; a 4-ounce glass of wine; and a shot of liquor. Based on the National Institute on Alcohol Abuse and Alcoholism guidelines, participants who consumed more than 7 drinks per week for women and more than 14 drinks per week for men were considered hazardous alcohol drinkers and enrolled as cases (National Institute on Alcohol Abuse and Alcoholism, 1995). PLWH who reported drinking less than those amounts (nonhazardous alcohol drinkers) constituted the controls and were matched by stratified gender, age (± 5 years), race, and Centers for Disease Control and Prevention stage of HIV infection with hazardous alcohol users. Race/ethnicity data were self-reported, with race categorized as Black, Caucasian, American Indian, Black Caribbean, or Asian; and ethnicity as Hispanic or non-Hispanic. For this analysis, a single three-level race/ethnicity variable was defined, which represented 98% of the sample: White/non-Hispanic (White), Black/non-Hispanic (Black), or Hispanic.

Study Outcomes

Fasting blood samples were collected, processed within 6 hours of collection, and sent to a clinical laboratory. Total cholesterol (TC), HDL, LDL, and triglyceride (TG) levels were measured by routine enzymatic methods (KonePro; Konelab, Epoo, Finland). Hypercholesterolemia (>200 mg/dl) and hypertriglyceridemia (borderline high 150 to 199, high 200 to 499, and very high >500 mg/dl) were defined according to cutoff values recommended in the U.S. National Cholesterol Education Program guidelines (Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults [Expert Panel], 2001).

Covariates

At baseline and the 6-month follow-up, each participant underwent an in-depth assessment, including a detailed interview using standardized research questionnaires covering sociodemographic information, drug and tobacco use habits, HIV infection- related data (i.e., stage of HIV infection), and a complete past and present medical and medications history. Medication history included previous exposure to antiretrovirals as well as lipid lowering medications. These questionnaires have been used in the authors' previous studies (Miguez-Burbano, Lewis, Moreno, & Fishman, 2008). To determine immune status and HAART effectiveness, CD4 cell counts were determined by flow cytometry per National Institute of Allergy and Infectious Diseases laboratory protocols and HIV viral load by polymerase chain reaction (COBAS Amplicor Analyzer [Roche Molecular Diagnostics, Pleasanton, CA]).

Nutritional evaluations included 24-hour dietary intakes, anthropometrics, and albumin levels. Body weight and height were measured and used to calculate body mass index (weight in kilograms divided by height in meters squared).

Statistical Analyses

The data were analyzed with SAS version 8 and Statistical Package for the Social Sciences version 11. Following descriptive statistical analyses, mean variables were compared using Student t-test and one-way analysis of variance (ANOVA) procedures. Correlations between the main variables of interest (i.e., CD4, lipid levels, HAART, and alcohol consumption) were examined with Pearson correlation coefficients. Chi-square, Student *t*-test, and ANOVA were used to evaluate differences in lipid levels between cases and controls at baseline and after 6 months of receiving HAART. Changes in lipid profiles between the baseline and second visit evaluation were assessed using Student *t*-test for paired samples. Lipid levels were compared between nonhazardous and hazardous drinkers at baseline and 6 months. All subsequent analyses were stratified by race/ethnicity. The authors estimated the relative risk (RR) of developing hyperlipidemia according to alcohol consumption by calculating odds ratios (ORs) and the corresponding confidence intervals (CIs) through multiple conditional logistic regression analyses. Multiple logistic regression analyses were used to evaluate the effects of race/ethnicity, alcohol, and other potential risk factors on lipid levels. The initial model was constructed with age, body mass index, CD4 cell counts, gender, baseline lipids, viral loads, and being in a regimen that included a PI as predictive variables. These factors were selected because they are either recognized risk factors (Montes et al., 2005) or they were significant ($p < .05$) in the univariate analysis. Nonsignificant variables ($p \geq .05$) were removed, beginning with the least significant variables, until the final full model was determined. Regression output was reported as adjusted OR, accompanied by 95% CI. Two participants at the 6-month assessment had extremely high triglycerides that required lipid-lowering drug treatments, and they were disqualified from the analyses.

Results

Study Group Main Characteristics

Table 1 illustrates the demographic and clinical characteristics of the sample (108 men and 56 women) by alcohol group. The alcohol groups were well-matched and were comparable in sociodemographic, anthropometric, CD4 counts, dietary variables, and the use of PIs. Approximately half (51%) of the respondents identified themselves as Black, 39% as Hispanic, and 10% as White.

The study sample of hazardous drinkers reported an average use of 32 ± 22 drinks/week, along with a well-documented history of continuous alcohol consumption averaging 8 ± 7 drinks per day. A trend was observed for Whites having more years drinking (12 ± 8 years vs. Blacks with 11 ± 10 years and Hispanics with 9.4 ± 8 years; $p = .09$) and Blacks drinking more days per week (3.8 ± 2.6 days vs. Whites with 3.0 ± 2.5 days, $p = .09$ and Hispanics with 3 ± 2.7 days, $p = .08$).

Baseline Profiles

The mean serum cholesterol levels of the study group varied from 52 to 324 mg/dl (173 ± 43 mg/dl), with 5% of the group having more than 250 mg/dl. An inverse correlation was observed between weekly alcohol consumption and TC level ($r^2 = -.256$, $p = .04$). Greater

weekly intakes of alcohol were associated with significantly lower LDL values (r^2 = .208; p = .03), and alcohol consumption was linked to HDL/LDL-cholesterol ratio (r^2 = −.195, p = .04). At baseline, hazardous alcohol users tended to have lower levels of TC than nonhazardous drinkers (166.7 ± 43.1 vs. 181.4 ± 40.7 mg/dl, p = .07). Levels of LDL were also significantly lower in hazardous drinkers (81.8 ± 35.6) than in nonhazardous drinkers (98.4 ± 33.2 mg/dl, p = .02). Levels of HDL (46.9 ± 17.6 vs. 44.1 ± 12.6 mg/dl), however, were similar between the groups.

Baseline TG ranged from 43 to 373 mg/dl (179 ± 108 mg/dl) with 15% of the sample having borderline high TG levels (151–199 mg/dl), one third had TG levels > 200 mg/dl, and only one Hispanic patient had TG levels > 500 mg/dl. Mean triglyceride levels were also higher in hazardous alcohol drinkers than in the nonhazardous drinkers (199 ± 123 vs. 177.2 ± 103.3 mg/dl, p = .07).

Table 2 displays the baseline mean values of lipids by race/ethnicity, including p values from comparison tests. As illustrated in Table 2, Hispanics and Whites were at increased risk of hypertriglyceridemia (> 200 mg/dl OR = 2.4, 95% CI: 1.0–5.7, p = .02 and OR = 6, 95%CI: 1.9–20.6, p = .001, respectively) compared with Blacks. Mean TC was significantly lower in Blacks. Although at first Blacks exhibited higher mean HDL levels, additional analyses indicated that compared with Blacks, Hispanics showed a threefold increased risk (95% CI: 1.3–7.9, p = .003) of having HDL levels < 40 mg/dl, suggesting an increased CVD risk. No significant gender, drug, or tobacco effects on lipid profiles were evident.

Evaluation of Lipid Levels After 6 Months of HAART

After 6 months of HAART, a positive slope in TG levels was observed (+30.1 ± 23 mg/dl), along with small increases in TC (+4 ± 3.84 mg/dl), and LDL (+5 ± 3.5 mg/dl) levels. The incidence of hypertriglyceridemia and hypercholesterolemia was 25% and 35%, respectively. Participants were dichotomized into two groups based on whether their treatment included a PI. Regimens without PIs included nucleoside reverse transcriptase inhibitors, nonnucleoside reverse transcriptase inhibitors, and/or integrase inhibitors. For those on regimens including a PI, the authors observed a substantial elevation in mean TG levels (22 ± 27 mg/dl), small increases in HDL (8 ± 5 mg/dl) and LDL (5 ± 4 mg/dl) levels, and TC levels not differing. In those not on a PI, TG levels decreased from 208 ± 66 to 173 ± 33, although it was not statistically significant, but levels of LDL tended to increase (75 ± 7 to 89 ± 13 mg/dl, p = .09). Nonsignificant differences in lipid profiles were observed between HAART-naive and HAART-experienced groups.

The increases in lipids by alcohol and race/ethnicity groups after 6 months of HAART are presented in Table 3. After HAART initiation, mean fasting TG levels in Hispanic hazardous drinkers increased more than in Hispanic nonhazardous drinkers (42% vs. 24%). Whites had a similar pattern (38% vs. 35%), contrasting with a 6% decrease among Black hazardous alcohol users and a 9% decrease in nonhazardous users.

Similarly, TC increased in both Whites and Hispanics after HAART, but it was more prominent in the Hispanic hazardous drinker group (22.2 ± 6.5 mg/dl, p = .004). In contrast,

Blacks showed only a small and nonsignificant increase, even if they consumed hazardous amounts of alcohol.

The rise in LDL was highest in the Hispanic hazardous drinkers (8 ± 5, $p = .08$) and at least double that of any other group. A total of 40% of the Hispanic nonhazardous drinkers and 45% of the Hispanic hazardous alcohol users had HDL < 40 mg/dl, and those are considered at risk for CVD. As illustrated in Table 4, Whites were next, with Black nonhazardous users exhibiting the lowest risk. The highest elevations in mean HDL levels were seen in White hazardous drinkers and in White nonhazardous drinkers, followed by Hispanics and Blacks. It has been established that an HDL above 60 mg/dl is cardioprotective; unfortunately none of the Hispanic hazardous drinkers had this advantage.

Regression Analyses

After adjusting for age, CD4 cell count, dietary intake (total caloric intake and saturated fat intake), HAART regimen (PI vs. no PI), and being Hispanic resulted in a 1.9-fold increased ($p = .04$) risk of hypertriglyceridemia after HAART and a 1.5-fold increase among Whites ($p = .08$). Moreover, a dose-response trend was observed; being a Hispanic hazardous drinker increased the risk 3.18-fold (95% CI: .99–12.05, $p = .04$); and heavy drinkers (> 30 drinks/week) exhibited the further highest risk (RR = 3.75, $p = .05$).

Discussion

Although the effects of race/ethnicity on both alcohol consumption and plasma lipoprotein levels have been examined in several studies of the general population (Albu et al., 1997; Després et al., 2000; Lovejoy et al., 1996; Schmidt et al., 1996), the authors failed to find any study of this type or on CVD risk factors in PLWH on HAART that addressed the influence of alcohol. Such a population may be uniquely different from the general population given the high prevalence of alcohol use, its potential interactions with HAART, its presumed effects on CVD risk factors, and the multiethnic composition of PLWH from the recruitment center in Miami. Overall, the results indicate that Black patients on HAART, particularly the nonhazardous alcohol drinkers, had a less atherogenic lipid profile compared with Whites and Hispanics. In contrast, when taking HAART, alcohol drinking significantly increased CVD risk, particularly among Hispanics. These results were of more serious concern given the prevalence of cigarette smoking among PLWH, further elevating CVD risks.

After 6 months, abnormalities associated with HAART were observed and included an escalation in total cholesterol and TG levels and a decrease in HDL. These events are major risk factors for CVD (Expert Panel, 2001). The most important result was that after 6 months on HAART, Hispanics and Whites exhibited a more marked increase in lipids than Blacks. Although consistent with previous publications (Foulkes et al., 2006, Bausserman et al., 2004), this study will extend the literature, including the use of certain methodological and design advantages. Prior work in the CVD risk area with PLWH on HAART largely examined cross-sectional relationships (Foulkes et al., 2006, Bausserman et al., 2004). This study was longitudinal, which allowed for uniformity, controlled for time in treatment, and did not have the confounding effects of lipid-lowering treatments or diets, thus providing a

stronger basis for causal inference of clinical relevance. More important, as hypothesized, the authors provide the first documentation that hazardous alcohol consumption during HAART further exacerbates the development of metabolic complications among all groups, particularly in Hispanics and Whites. These results will further extend the previous study result that hazardous alcohol use significantly interfered with HAART (Míguez, Shor-Posner, Morales, Rodriguez, & Burbano, 2003).

Although race/ethnicity has been considered a surrogate for sociodemographic or dietary factors, analyses indicate no significant differences in any of these parameters across groups. The magnitude of the association between race/ethnicity (e.g., being Hispanic) and hyperlipidemia, its independence from traditional risk factors (i.e., diet or sociodemographics), and evidence of an alcohol dose-response trend suggest that these results are less likely to be because of chance. Further, a number of possible biological mechanisms by which race/alcohol can alter lipid metabolism are becoming evident. Recent studies have shown that genetic factors account for important differences in plasma lipids and alcohol metabolism across racial/ethnic groups (Fauvel et al., 2001; Foulkes et al., 2006; Freudenheim et al., 2003; Olshan, Weissler, Watson, & Bell, 2001). Moreover, further strengthening these results was the identification of an alcohol dose effect on the risk of developing hypertriglyceridemia after initiating HAART in a Hispanic patient.

In summary, these results showed that race and alcohol were independent risk factors for lipoprotein disturbances in the HIV-infected individuals in the study. The results call for caution when prescribing HAART regimens containing PI for Hispanic and White hazardous alcohol users without previous alcohol use diagnosis and counseling. Nevertheless, the study has some limitations. The size of the study allowed only moderate estimates of changes in the lipid profiles following HAART. Results were limited to a single cohort, and although the race/ethnic distribution of the sample was representative of the HIV epidemic in the United States, Black patients were overrepresented. Lipid values may differ in less advanced HIV-infected patients than those included in this study. Although these data covered only the first 6 months of therapy, previous studies have indicated that most of the changes occurred within this window (Montes et al., 2005). Therefore, larger studies with longer follow-up times are recommended to confirm these results.

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Clinical Considerations

- The high prevalence of lipoprotein disturbances observed in PLWH receiving HAART has contributed to an elevated risk of CVD.
- Although moderate alcohol consumption has been recommended to reduce lipid disturbances and CVD risk in the general population, closer examination may be needed to refine the clinical guidelines for HIV-infected individuals.
- In light of the challenge facing health care providers regarding what to recommend to HIV-infected patients about alcohol use, clarification of the role of alcohol in HIV disease and treatment is needed.
- Strategies that identify individuals at increased risk of HAART-related metabolic complications are likely to facilitate rational decision making when selecting treatment regimens and the early use of appropriate preventive CVD therapies.
- Individual treatment outcomes, reduction/management of side effects, and follow-ups may be enhanced when health care providers consider risk factors such as race/ethnicity and alcohol use when prescribing HAART.

Table 1

Baseline Sociodemographic Information by Alcohol Group

Variables	HIV-Infected Hazardous Drinkers *N* = 88	HIV-Infected Nonhazardous Drinkers *N* = 76
Age	40 ± 7.2	41 ± 6.7
Gender		
Male	62%	70%
Female	38%	30%
Race/ethnicity		
Black	64%	55%
Hispanic	29%	33%
White	7%	12%
Income		
<$10,000	89%	88%
$11,000–$20,000	9%	10%
>$20,000	2%	2%
Highest Level of Education		
<11 years	53%	56%
<12 years	47%	44%
Marital/partner status		
Single, separated, or widowed	89%	84%
Married or with a partner	11%	16%
Number of years diagnosed with HIV	8.7 ± 5.3	9.7 ± 6.5
$CD4^+$ lymphocyte percentages	14.9 ± 9.6	13.1 ± 8.1
Viral load logarithm	4.3 ± 1.5	3.98 ± 1.6
Body mass index kg/m^2	26.7 ± 6.7	27 ± 5.6

NOTE: Values are means ± SD or percentages. No significant differences in sociodemographic characteristics were reported between groups.

Table 2

Baseline Lipid Values by Race/Ethnicity

Variables	Hispanics	*p*-Value	Blacks	*p*-Value	Whites
Total cholesterol mg/dl	163.7 ± 46.9	.02	159.4 ± 35.9	.002	207.8 ± 28.8
HDL mg/dl	42.1 ± 11	.04	50.0 ± 14.1	NS	43.5 ± 18.3
LDL mg/dl	91.8 ± 40.9	NS	83.0 ± 32.4	.09	100.9 ± 29.6
Triglycerides mg/dl	216.0 ± 133	.02	150.5 ± 84.9	.002	298.0 ± 107.1

NOTE: Values are means ± SD. HDL = high-density lipoprotein, LDL = low-density lipoprotein, NS = not significant.

Table 3

Six-Month Change in Lipid Profile by Alcohol and Race/Ethnicity

	Hispanics		Whites		Blacks	
Variables	**Nonhazardous *N* = 26**	**Hazardous *N* = 30**	**Nonhazardous *N* = 8**	**Hazardous *N* = 6**	**Nonhazardous *N* = 42**	**Hazardous *N* = 52**
Total cholesterol mg/dl	9.8 ± 51	22.2 ± 6.5*	12.8 ± 6	18.2 ± 16	5.9 ± 4.5	8.3 ± 7.0
LDL mg/dl	3.8 ± 3.6	8 ± 5	1.9 ± 1.2	2.9 ± .6	3.4 ± 4.4	4.1 ± 6.1
HDL mg/dl	.4 ± 2.2	2.5 ± 2.4	7.4 ± 5.7	28 ± 4	.028 ± 1.4	1.3 ± 1.9
Triglycerides mg/dl	52.5 ± 48.7	99.5 ± 81	73.7 ± 33.7	75.5 ± 43	−10 ± 8.8	−6.6 ± 1

NOTE: HDL = high-density lipoprotein, LDL = low-density lipoprotein.

Table 4

Major Risk Factors for Cardiovascular Disease

		Hazardous Drinkers (*n* = 88)			Nonhazardous Drinkers (*n* = 76)		
	Points	**Hispanic**	**Black**	**White**	**Hispanic**	**Black**	**White**
Cigarette smoking	+1	87%	100%	94%	82%	75%	84%
Hypertension (> 140/90 mmHg)	+1	0%	1%	0%	0%	2%	0%
Low HDL cholesterol (< 40 mg/dl)	+1	45%	36%	26%	40%	28%	23%
High HDL cholesterol (> 60 mg/dl)	−1	0%	17%	19%	15%	13%	11%
Age (men > 45 years; women > 55 years)	+1	7%	14%	41%	29%	22%	28%
Diabetes	+1	1%					
LDL > 160	+1	6%	0%	0%	6%	3%	0%

NOTE: Values are percentage. HDL = high-density lipoprotein, LDL = low-density lipoprotein.