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The New Orleans Alcohol Use in HIV [NOAH] Study: Launching a translational investigation of the interaction of alcohol use with biological and socioenvironmental risk factors for multi-morbidity in people living with HIV

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Abstract

Background: Alcohol use disorders (AUDs) are highly prevalent in persons living with HIV (PLWH) and are associated with increased HIV risk behaviors, suboptimal treatment adherence, potential interaction with medication pharmacodynamics, and greater risk for disease progression. Preclinical studies show that chronic binge alcohol administration accelerates disease progression

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Authors' Contributions

All authors participated in study design, operationalization, and conduct of the study. DW drafted the manuscript with contributions from all co-authors. All co-authors have revised and approved the final manuscript.

Availability of Data and Materials

The datasets generated during the current study will be available per the *LSUHSC-NO Comprehensive Alcohol-HIV/AIDS Research Center's* data sharing policy.

Conflict of Interests

No competing interests to disclose.

and aggravates pathogenesis in the simian immunodeficiency virus (SIV)-infected rhesus macaque model despite viral suppression by antiretroviral therapy.

Methods: To translate preclinical findings in the rhesus macaque model of chronic binge alcohol administration and SIV infection and to address areas of uncertainty surrounding the biological mechanisms and socioenvironmental modifiers that contribute to the relationship between alcohol use and HIV-associated comorbidities, precocious aging, and disease progression, we designed a translational multi-project, longitudinal, cohort study, the *New Orleans Alcohol Use in HIV [NOAH] Study*. The *NOAH Study* is led by a multidisciplinary team of scientists, with a research focus on the interaction of AUD and HIV. The overarching hypothesis is that alcohol use will lead to adverse health outcomes in PLWH. In this report, we describe the study design and baseline descriptive characteristics of our cohort.

Results: Three hundred sixty-five participants completed the baseline testing. The cohort is predominantly male (69%) and African American (83.5%). The majority of participants report incomes below 200% of the federal poverty level. CD4 counts less than 200 cells/ μ L were found in 12.8% and viral loads less than 50 copies/mL were found in 73.6%. These HIV status variables did not differ based upon alcohol use.

Conclusions: The *NOAH Study* facilitates bidirectional translational investigation of alcohol's impact on PLWH. Translation of preclinical findings to PLWH permits confirmation of basic biological mechanisms in humans and also allows incorporation of sociobehavioral factors that may affect biology but are challenging to replicate in preclinical models.

Keywords

alcohol; HIV; frailty; immunosenescence; comorbidities

Introduction

Alcohol use disorders (AUDs) are increasingly common, particularly in marginalized populations (Grant BF *et al.*, 2017). Further, people living with Human Immunodeficiency Virus (PLWH) are especially vulnerable to the negative health consequences of alcohol misuse, even in the era of effective combined antiretroviral therapy (cART) (Azar MM *et al.*, 2010). At risk alcohol use is tightly linked to HIV acquisition through high risk behaviors. In addition, alcohol consumption affects every stage of the HIV Care Continuum; (Valdiserri RO, Holtgrave DR, 2018) delays in diagnosis, low engagement and retention in medical care, poor adherence to treatment, and failure to suppress circulating virus are associated with harmful alcohol use.

Effective cART has prolonged survival and shifted the demographics of PLWH. Mean ages of PLWH have steadily risen with presently more than half of PLWH in the developed world older than 50 years of age. These demographic shifts are largely driven by a marked decrease in death due to AIDS-related infections and cancers. An abundance of data suggests that HIV infection leads to precocious aging, geriatric comorbidities, and frailty (Gutierrez AD, Balasubramanyam A, 2012). Increased prevalence of cancers, chronic liver disease, osteoporosis, cardiovascular disease, diabetes, and metabolic dysregulation have been widely reported in PLWH. HIV is also associated with geriatric syndromes including

polypharmacy, physical and cognitive impairment, and frailty (Kolter DP, 2003; Guaraldi G *et al.*, 2011; Gutierrez AD, Balasubramanyam A, 2012; Piggott DA *et al.*, 2013; Williams EC *et al.*, 2016; Molina PE *et al.*, 2018).

The factors contributing to the early onset of comorbidity and aging in PLWH are incompletely understood and many mechanisms have been proposed. Social, environmental, and behavioral factors appear to be primary drivers of adverse health effects. However, preclinical and translational evidence indicate that alcohol also dysregulates biological pathways, particularly immune and metabolic systems (Gutierrez AD, Balasubramanyam A, 2012; Klatt NR *et al.*, 2013; Molina PE *et al.*, 2018). Alcohol misuse is well recognized to promote chronic inflammation. Immune system activation and a chronic pro-inflammatory state promote HIV disease progression and comorbidity pathogenesis (Molina PE *et al.*, 2018). These factors are central to biological aging and to increased risk of comorbidities in PLWH.

The impact of alcohol on viral suppression and CD4 T cell counts is predominantly linked to poor cART adherence. However, the detrimental effects of AUD on HIV-associated comorbidities may depend on direct perturbation of biological pathways. Clearly, in the context of circulating virus, HIV antigens overshadow most other immune drivers. The contribution of alcohol use to the pathogenesis of chronic, HIV-linked comorbidities becomes an increasingly important factor as the HIV epidemic shifts from an acute fulminant immunodeficiency illness toward a chronic medical condition.

Studies from our group at the Louisiana State University Health Sciences Center - New Orleans (LSUHSC-NO) National Institute on Alcohol Abuse and Alcoholism (NIAAA)-funded Comprehensive Alcohol-HIV/AIDS Research Center (CARC) have advanced the understanding of the impact of chronic binge alcohol administration on disease progression using the simian immunodeficiency virus (SIV)-infected rhesus macaque model (Molina PE *et al.*, 2008; Katz PS *et al.*, 2015; Molina PE *et al.*, 2018). Our preclinical data show that clinical outcomes are worse in alcohol-administered SIV-infected macaques despite viral suppression by cART. This raises the question whether alcohol-induced inflammation mediates comorbidity pathogenesis and precocious aging in PLWH when viral loads are suppressed.

The *New Orleans Alcohol Use in HIV [NOAH] Study*, facilitates a bidirectional translational investigation of alcohol's impact on PLWH. The *NOAH Study* aims to address areas of uncertainty surrounding the biological mechanisms and socioenvironmental modifiers that contribute to the relationship between alcohol use and HIV-associated comorbidities, precocious aging, and disease progression. This report details the study design and baseline descriptive characteristics of the *NOAH* cohort. The *NOAH* cohort data collection was designed with sufficient flexibility to support additional projects over time; however, the initial scheme focused upon three major research themes.

Alcohol & Metabolic Dysregulation in PLWH

Mechanistically, we hypothesize that chronic inflammation, accentuated by alcohol consumption and biological aging, is a significant underlying mechanism disrupting

metabolic homeostasis. Thus, the role of alcohol consumption on (a) the incidence of decreased lean body mass and muscle strength, (b) lipodystrophy (fat redistribution, altered lipid profile), and (c) prediabetes (fasting plasma glucose 100 to 125 mg/dl, and glycosylated hemoglobin 5.7% to 6.4%) will be examined in PLWH.

Metabolic alterations, particularly defects related to glucose and lipid metabolism, lead to a higher risk for insulin resistance manifested as an increased frequency of glucose intolerance or diabetes mellitus in PLWH. The mechanisms underlying dysglycemia in PLWH have remained poorly understood (Gutierrez AD, Balasubramanyam A, 2012). However, chronic subclinical inflammation is thought to contribute to adipose tissue dysregulation and metabolic derangements seen in ART-treated PLWH (Kolter DP, 2003). Our preclinical studies in SIV-infected non-human primates have shown that chronic binge alcohol administration accentuates metabolic derangements, including end-stage disease wasting that is associated with localized skeletal muscle inflammation, profound depletion of antioxidant capacity (oxidative stress), increased proteasomal activity, and decreased myoblast differentiation potential (Lecapitaine NJ *et al.*, 2011; Simon L *et al.*, 2014). We predict this translates into decreased metabolic homeostasis in the clinical setting.

Alcohol Use Disorder & Aging in HIV: The Role of the Microbiota & Inflamm-Aging

Alcohol misuse hastens aging in the general population, but its role in aging and frailty in PLWH remains unknown. Some reports have suggested a detrimental effect of substance misuse on frailty in HIV-infected patients (Piggott DA *et al.*, 2013). The term “inflamm-aging” connotes a central role for immune activation caused by chronic antigenic stress as the origin of immune cell senescence and exhaustion characteristic of aging (De Martinis M *et al.*, 2005). In this model, antigenic burden drives naïve T cells toward clonal expansion resulting in a restricted functional capacity. The intestinal tract is a major target for early HIV infection, viral expansion, and CD4+ T cell destruction. It has also become clear that loss of mucosal integrity, accompanied by microbial product translocation into the systemic circulation, is associated with HIV infection, disease progression, and mortality (Klatt NR *et al.*, 2013).

The effects of AUD on the alimentary tract microbiota in PLWH by phylogenetic analysis of ribosomal DNA sequences from oral and stool samples will be investigated. Moreover, the role of alcohol in escalating immune cell activation and senescence in PLWH and the contribution of alimentary tract microbial community structure as an intermediary in this relationship will be examined. Finally, we will examine the association between alcohol use and validated indices of aging and frailty including the deficit index and phenotypic frailty (Fried LP *et al.*, 2001; Searle SD *et al.*, 2008), along with telomere length as a biomarker of cellular aging.

Lifetime and Current Stress and Allostatic Load in PLWH

Among PLWH, higher levels of both psychosocial and physiological stress have been associated with poorer immune status, increased viral load over time, faster disease progression, and higher rates of mortality (Leserman J *et al.*, 2000; Ironson G *et al.*, 2006). Chronic psychological stress has been implicated in precocious aging and allostatic load –

the cumulative wear and tear on physiological systems and organs due to chronic stress (Geronimus A *et al.*, 2006).

The overall goal of this component is to examine the impact of both chronic psychological stress and early life adversity on behavioral and biological outcomes among men and women living with HIV. Alcohol use, allostatic load, mental health, medication adherence, retention in care, and viral suppression will be examined in relation to chronic psychological stress and early life adversity overall and how these may differ by sex in PLWH. The mediating role of alcohol use between chronic psychological stress and early life adversity and biological, mental health, and clinical outcomes will be determined using the *NOAH* cohort.

Materials and Methods

Overall Design

The *NOAH Study* aims to address areas of uncertainty surrounding the biological mechanisms and socioenvironmental modifiers that contribute to the relationship between alcohol use and HIV-associated comorbidities, precocious aging, and disease progression. Enrolled participants complete a battery of behavioral, demographic, and functional assessments at baseline and at 2.5 years with interim limited assessment visits every 10 months (Figure 1).

Study Population

A clinic-based sample of 365 HIV-infected patients under care in the Greater New Orleans, Louisiana metropolitan area, aged 18 years were enrolled in the full study after screening 655 subjects. Patients with potentially hazardous alcohol use as defined by Alcohol Use Disorders Identification Test (AUDIT) 8 were oversampled. Exclusion criteria included an acute illness within the last 6 weeks as defined by unscheduled healthcare utilization for a new or exacerbated illness, non-prophylaxis prescription of antibiotics, or pregnancy. Excluded patients were eligible for enrollment after resolution of the acute phase of identified illness.

Data Collection and Analysis

LSUHSC-NO's Human Research Protection Program and Institutional Review Board approved the study and maintains oversight of research activities. Biospecimen samples collected include a fasting blood sample, gargles, buccal swabs, vaginal swabs, and fecal samples. Anthropometric measurements, body composition estimates by air displacement plethysmography, blood pressure, oral examination, and clinical laboratory tests are also performed. Data from interviewer-administered questionnaires covering alcohol use, socioenvironmental status, psychosocial stress, medical history, physical and cognitive function, and health behaviors are supplemented by a electronic health record abstraction. *Additional details are provided in Supplemental Materials.*

A strength of the *NOAH Study* is the collection of data for a multi-dimensional characterization of alcohol exposure for the cohort (Supplemental Table S1). Alcohol exposures and consequences are categorized by drinking status, drinking patterns, quantity-

frequency of alcohol consumption, alcohol use disorder, and addiction severity, as well as by a biomarker of recent alcohol use. Other substance use (cannabis, cocaine, opioids, etc.) is assessed, as it is likely to have significant modifying effects on alcohol-related biomedical outcomes.

Sample size (N=350) was estimated to allow testing the hypotheses described under the three main research themes. Our sample size is inflated by 20% to account for dropouts and lost to follow-up.

Results

The baseline enrollment and exam/testing of the *NOAH* cohort was completed and descriptive statistics characterizing the cohort are provided in Table 1. A total of 655 participants completed the screening visit. Of these, 430 were invited to continue to full testing and a total of 365 participants (85%) attended the baseline testing. Unadjusted, descriptive statistics for the initial testing visits reveal that our cohort is predominantly non-Hispanic black, majority male, and middle age. Sex was not a consideration in our inclusion or exclusion criteria. The frequency of women in our cohort mirrors the clinic's demographics [32% female] in the low alcohol consumers but is lower in participants with potentially hazardous drinking. The majority also have annual incomes less than \$20,000 and a high school degree or less. A substantial proportion are current smokers. Compliance with the study protocol was high as illustrated in Supplemental Table S2.

Discussion

The *NOAH Study* provides data on a cohort of in-care PLWH, with and without AUD. This study provides a valuable resource for resolving emerging questions regarding the consequences of alcohol use and other health behaviors on disease process and risk of comorbidities in PLWH despite use of cART and viral suppression. While the study design establishes *a priori* hypotheses to be tested, the testing strategy is sufficiently comprehensive and intentionally flexible to allow introduction of additional projects in the future.

Many pivotal findings have been reported in cohorts of PLWH with significant immunosuppression, high levels of viremia, and/or in comparison to the seronegative population (Williams EC *et al.*, 2016; Molina PE *et al.*, 2018). The design of the *NOAH Study* offers added relevance to the current state of the HIV pandemic in which effective cART is more widely available, viral suppression can be achieved with adherence to treatment, and PLWH increasingly suffer a disproportionate burden of chronic disease and precocious aging. By focusing on a clinic-based sample, the *NOAH Study* is well positioned to investigate the translational biology, and the influence of behaviors and environment, of PLWH under care.

Challenges and learning opportunities during the initial phase of the study include assessments of alcohol exposure and AUD that rely primarily on validated but self-reported questionnaires. Early in the study we found that AUDIT scores during screening were higher than at retesting during the full baseline visit (data not shown), skewing the initial targeted distribution of alcohol exposure among recruited participants. While the reason(s) for this

are not entirely clear, social pressure and hope of secondary gain are probable confounders. This variability of self-reported consumption may reflect true variance in behaviors. We implemented a number of measures to minimize these effects including de-emphasizing the focus on alcohol and substance use, inclusion of a phosphatidylethanol (PEth), a biomarker of alcohol consumption, and frequent longitudinal reassessments.

This observational study cannot confirm mechanisms. However, several approaches partially mitigate this limitation including our longitudinal design, planned *ex vivo* mechanistic experiments, and intervention studies on subsets of the *NOAH* cohort. Furthermore, the LSUHSC CARC is operationalized as a bidirectional research program, and we are positioned to conduct mechanistic studies based on observations from the *NOAH Study* in our SIV-infected *rhesus macaque* model of alcohol consumption. Thus, the *NOAH Study* provides valuable data to test the impact of alcohol and other substance use on PLWH engaged in care. Inclusion of biological and sociobehavioral assessments offer the opportunity to test novel hypotheses highly relevant to the ongoing HIV pandemic as it enters its next stage, when the clinical importance of chronic disease outweighs overwhelming immune suppression and opportunistic infection. This knowledge will lead to clinical interventions to improve the health of PLWH.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1:
NOAH Study Design Overview

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Table 1.
Distribution of Research Participants by Alcohol Use Disorders Identification Test (AUDIT) and Demographic Parameters.

Significant differences are shown for those characteristics that differed between participants with AUDIT <8 and AUDIT ≥ 8. *1 participant was transgender. Data shown as total numbers and percentage in parenthesis.

	AUDIT		
	All	<8	8
	(n=365)	(n=218)	(n=147)
	% (n)	% (n)	% (n)
Sex			
Female	31.0 (113)	36.7 (80)	23.1 (34)
Male	69.0 (252)	63.3(138)	76.9 (113)
p-value		0.0062	
Race			
African American	83.6 (305)	82.6 (180)	85.0 (125)
White	15.6 (57)	16.1 (35)	15.0 (22)
Other	0.8 (3)	1.4 (3)	0
p-value		0.7912	
Age, years			
20–29	5.2 (19)	5.5 (12)	4.8 (7)
30–39	16.4 (60)	16.1 (35)	17.0 (25)
40–49	24.1 (88)	22.9 (50)	25.8 (38)
50–59	41.6 (152)	41.3 (90)	42.2 (62)
60+	12.6 (46)	14.3 (31)	10.2 (15)
p-value		0.5361	
Housing			
Single Family Dwelling	84.1 (307)	84.4 (184)	83.7 (123)
HIV-Specific Group Facility	10.7 (39)	11.0 (24)	10.2 (15)
Homeless/Shelter	4.4 (16)	3.2 (7)	6.1 (9)
Group Home	0.8 (3)	1.4 (3)	0
p-value		0.5219	
Income			
< \$20,000	88.7 (323)	88.1 (192)	89.1 (131)
\$20,000-\$39,999	8.5 (31)	9.2 (20)	7.5 (11)
\$40,000+	2.7 (10)	2.3 (5)	3.4 (5)
p-value		0.9180	
Education			
< High School	40.8 (149)	35.8 (78)	48.3 (71)
High School Graduate	31.2 (114)	33.5 (73)	27.9 (41)
Some College, Junior College, Community	22.2 (81)	24.8 (54)	18.4 (27)

		AUDIT	
	All	<8	8
College, Vocational/Trade School			
4-Year College Graduate	3.8 (14)	3.7 (8)	4.1 (6)
Graduate/Professional School	1.9 (7)	2.3 (5)	1.4 (2)
p-value		0.0449	
BMI (n=362)			
< 18.5	4.4 (16)	4.2 (9)	4.8 (7)
18.5–24.9	38.4 (140)	35.2 (76)	43.8 (64)
25–29.9	29.0 (106)	29.6 (64)	28.8 (42)
30–34.9	17.0 (62)	20.4 (44)	12.3 (18)
35+	10.4 (38)	10.6 (23)	10.3 (15)
p-value		0.1031	
Alcohol Measures			
30-day TLFB (grams) Median (Quartile Range)	158.2 (728)	37.1 (186.2)	770 (1811.3)
p-value		<0.0001	
PEth 50-mg/dL	162 (45.9)	60 (28.8)	102 (70.3)
p-value		<0.0001	
Smoking Status			
Non-Smoker	23.0 (84)	27.5 (60)	16.3 (24)
Current Smoker	60.8 (222)	53.2 (116)	72.1 (106)
Former Smoker	16.2 (59)	19.3 (42)	11.6 (17)
p-value		0.5991	
CD4 Count (n=357)			
< 200	13.2 (47)	12.7 (27)	13.9 (20)
200–350	13.2 (47)	14.1 (30)	11.8 (17)
> 350	73.7 (263)	73.2 (156)	74.3 (107)
p-value		0.9847	
Viral Load (n=357)			
< 50	75.4 (269)	77.5 (165)	72.2 (104)
p-value		0.2595	
< 400	87.1 (311)	86.4 (184)	88.2 (127)
400–5000	4.5 (16)	3.8 (8)	5.5 (8)
> 5000	8.4 (30)	9.9 (21)	6.3 (9)
p-value		0.3860	