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A genetic marker of risk in HIV-infected Individuals with a history of hazardous drinking

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Abstract

Impulsivity and sensation seeking have been linked to hazardous drinking, increased sexual risk behaviors, and lower treatment adherence among persons living with HIV (PLH). The *dopamine active transporter1* (*DAT1* or *SLC6A3*) gene has been linked to impulsivity and sensation seeking in several populations but has not been investigated among populations of PLH. This study used data from 201 PLH who report a recent history of heavy episodic drinking. Results indicate that *DAT1**10R vs *DAT1**9R genotype was related to higher propensity for risk taking (standardized difference score (d) = 0.30 [95% CI: 0.02;0.59]), more hazardous drinking (d = 0.35 [0.05;0.64]), and more condomless sex (rate ratio (RR) = 2.35[1.94; 2.85]), but were counter-intuitively associated with fewer sexual partners (RR = 0.65[0.43;0.91]) and possibly better treatment adherence (d = 0.32 [-0.01;0.65]). Results are consistent with the suggested associations between *DAT1* and risk-taking behavior. The counter-intuitive finding for partner selection and treatment adherence may be evidence of additional factors that place PLH at risk for engaging in hazardous drinking as well as relationship difficulties and problems with treatment adherence (e.g., depressive symptoms, avoidant coping, trauma history). Caution is required when using a single gene variant as a marker of complex behaviors and these findings need to be replicated using larger samples and additional variants.

Keywords

Hazardous Drinking; Medication Adherence; Sexual Risk Behaviors; *SLC6A3*; Dopamine Active Transporter (*DAT1*)

INTRODUCTION

Among persons living with HIV (PLH), there is consistent evidence linking hazardous drinking to lower adherence to antiretroviral therapy, higher rates of condomless sex, and greater number of sexual partners (Azar, Springer, Meyer, & Altice, 2010; Kalichman et al., 2014; Shuper, Joharchi, Irving, & Rehm, 2009; Vagenas et al., 2015), and an increased risk of HIV progression and transmission (Baliunas, Rehm, Irving, & Shuper, 2009; Shuper et al., 2010). Impulsivity and sensation seeking have been linked to hazardous drinking, increased sexual risk behaviors, and lower treatment adherence (Hendershot, Stoner, George, & Norris, 2007; Shin, Hong, & Jeon, 2012). We evaluated a genetic marker (*DAT1*) that has been associated with impulsivity, sensation seeking, and multiple risk behaviors.

The dopaminergic system is central to the reward pathway and variation in dopaminergic genes is thought to contribute to individual differences in impulsivity and reward sensation seeking (Norbury & Husain, 2015; van Gaalen, van Koten, Schoffemeer, & Vanderschuren, 2006). A critical component of the dopaminergic system is the dopamine active transporter 1 that is coded by the *SLC6A3* gene (commonly known as *DAT1*) (Ciliax et al., 1999; Vandenberg et al., 1992). Past studies have focused on the 9 and 10 repeat genotypes of the *DAT1* variable number of tandem repeat polymorphism (*DAT1**9R & *DAT1**10R), and have associated *DAT1**10R versus *DAT1**9R with greater impulsivity, sensation seeking, and risk-taking (Blum et al., 2011; Gizer & Waldman, 2012; Guo, Cai, Guo, Wang, & Harris, 2010; Mata, Hau, Papassotiropoulos, & Hertwig, 2012). There is some evidence that *DAT1**10R is more prevalent in PLH relative to uninfected individuals (Horn et al., 2013).

Few studies have examined associations with sexual risk behavior (Daw & Guo, 2011; Guo, Tong, & Cai, 2008; Guo, Tong, Xie, & Lange, 2007), finding *DAT1**10R is associated with more condomless sex among adolescent females, and the *DAT1**9R/9R associated with fewer lifetime partners among young adults. Moreover, there is inconsistency in the literature regarding all of the aforementioned associations (Koutsilieri, Riederer, Plessis, & Scheller, 2014), suggesting more work is needed to understand the role of *DAT1* in risk-taking, particularly sexual risk taking. In this brief report, we present differences between *DAT1* genotypes in sexual risk behaviors, treatment adherence, hazardous drinking, and propensity for risk taking in a sample of PLH who have a history of heavy episodic drinking.

METHODS

Participants

Participants consisted of 201 HIV-infected adults (ages 22–64) who reported at least one occasion of Heavy Episodic Drinking (HED; 5+/4+ drinks per occasion for men/women) over the past 6 months. The recruited sample was consistent with demographics of the clinic recruitment site (Table 1). There were minimal demographic differences between genotypes with largest difference only accounting for 2% of variability in years since HIV diagnosis.

Procedures

Participants were recruited by a clinic nurse during regularly scheduled appointments. The study was approved by the hospital IRB, and all participants provided written informed

consent. Data were collected using audio computer-assisted self-interview, and a computer administered behavioral task.

Genotyping—DNA was isolated from buccal cells (Kenna et al., 2009) and DAT1 genotypes were assayed via published methods (Anchordoquy, McGeary, Liu, Krauter, & Smolen, 2003). PCR product was analyzed with an ABI 3500 DNA sequencer. Alleles were scored by two investigators independently, and inconsistencies were reviewed and rerun when necessary. Consistent with previous literature (Blum et al., 2011) we coded *DAT1**9R/9R genotype as *DAT1**9R and both the *DAT1**10R/9R and *DAT1**10R/10R genotypes as *DAT1**10R. DAT1 genotypes did not differ from Hardy–Weinberg equilibrium ($\chi^2(1)=2.22$, $p=.14$).

HIV Treatment History and Medication Adherence—Participant report of HIV treatment history included history of AIDS defining illness, history of antiretroviral resistance, and most recent viral load. Medication adherence was assessed using the AIDS Clinical Trials Group (ACTG) Self-Reported Medication Adherence Measure (Chesney et al., 2000).

Risk Taking—Risk taking propensity was assessed using the Balloon Analogue Risk Task (BART), a computer administered behavioral task included in the PhenX Toolkit (Lejuez et al., 2002). Performance on this task has been associated with adolescent risk taking (Lejuez, Aklin, Zvolensky, & Pedulla, 2003) and impulsivity (Hunt, Hopko, Bare, Lejuez, & Robinson, 2005). Higher scores indicate greater risk taking propensity.

Sexual risk behaviors—Sexual risk behaviors during the past 90 days (including receptive and insertive vaginal or anal intercourse) included whether or not they were sexually active, number of sexual partners, number of condomless sex acts, number of condomless sex acts following alcohol consumption, and whether or not they used drugs, other than alcohol, prior to having sex at any time during the past 90 days. For the most recent encounter, participants indicated the type of partner (i.e., someone they just met; someone they knew well, but not a regular partner; a steady boyfriend or girlfriend), whether they drank before the sexual encounter (and if so how many drinks), and whether a condom was used.

Drinking Behavior—Participants completed the Alcohol Use Disorders Identification Test (AUDIT) (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993), a widely used brief screening tool designed for general medical settings.

RESULTS

DAT1 information was missing for 7% of the sample resulting in an analytic sample of 188 participants. Generalized linear models (GLiM) were used to test for differences between genotypes. Analyses of treatment adherence and viral load were performed using the 156 participants who reported being on antiretroviral (ARV) medication. The size of differences between genotype groups were estimated using odds ratios (OR) for dichotomous outcomes, rate ratios (RR) for behavioral counts, and standardized difference scores (standardized

using the pooled baseline standard deviation across genotypes; *d*) for continuous outcomes. Using a recommended approach to address possible bias due to population stratification (Sinha, Larkin, Elston, & Redline, 2006), race was included as dichotomous covariate (White vs. non-White) in all models. Analyses were performed using R v3.2.3. Unadjusted descriptive statistics and adjusted effect sizes with 95% confidence intervals are shown in Table 2.

DISCUSSION

One aim of this study was to replicate previous studies linking *DAT1* to risk behaviors, in a population of PLH with recent HED. Results indicate strong evidence that those with a *DAT1**10R versus *DAT1**9R genotype were more likely to report condomless sex and condomless sex after drinking, with some evidence that they were more likely to report using drugs prior to sex, hazardous drinking, and show higher propensity for risk taking on a computerized task. Some findings were unexpected: the strong evidence that *DAT1**10R genotype reported fewer partners along with some evidence of better treatment adherence and more viral suppression than the *DAT1**9R genotype.

Overall, these findings are consistent with previous literature linking the *DAT1**10R genotype to risk-taking behaviors, including multiple dimensions of sexual risk-taking. The unexpected findings for partner selection and treatment adherence may be evidence of risk factors other than impulsivity and sensation seeking that place PLH at risk for engaging in hazardous drinking as well as relationship difficulties and problems with treatment adherence. These alternatives may include depressive symptoms, avoidant coping, or trauma history which have all been implicated in hazardous drinking as well as poor adherence, sexual risk, low interpersonal trust, and relationship difficulties (Ben-Zur & Zeidner, 2009; Dale et al., 2014; Houck, Nugent, Lescano, Peters, & Brown, 2010; Kamen et al., 2013; Monson et al., 2012; Nugent, Amstadter, & Koenen, 2010; Saucedo, Wiebe, & Simoni, 2014; Whetten et al., 2013).

Limitations

Limitations include: 1) use of observational, cross-sectional data, 2) limited sample size, 3) use of self-reported viral load and adherence, 4) possibility of a third variable explanation such as another genetic variant (e.g., another variant in linkage disequilibrium with the *DAT1* VNTR), or unmeasured environmental factor, 5) possibility that population stratification of the *DAT1* gene may be distorting findings, 6) use of only one marker to index the complex phenotype of impulsivity/sensation seeking.

CONCLUSIONS

Understanding these limitations, results from this study support associations between the *DAT1**10R genotype and increased sexual risk behavior among PLH with a history of HED. Moving forward, there are several areas of future research that would help to better identify and understand the individual vulnerabilities in this population. First, more information is required to understand neurobiological mechanism that may contribute to the vulnerabilities associated with HED, sexual risk behaviors, and disease management in PLH (Litten et al.,

2015). For example, in addition to impulsivity and sensation seeking, biobehavioral influences related to PTSD, depression, and avoidant coping strategies may also be at play (Gold, Machado-Vieira, & Pavlatou, 2015; Stark et al., 2015). Second, given the complexity of behavioral genetic phenomena including likelihood for multiple markers influencing behaviors in concert, gene-gene and gene-environment interplay, and the potential impact of HIV virus on function of dopamine-rich brain tracts, more research is needed with larger samples. Finally, longitudinal event-level characterization of drinking, sexual behavior, and partner characteristics will provide more information about the full context of drinking and sexual risk behavior. Reducing risk and improving health in PLH requires moving beyond identifying patients by single risk behaviors, such as HED, to tailoring treatment recommendations according to individual vulnerabilities. As the field continues to progress, it is possible that genetic markers can inform etiological theories and may identify new intervention targets.

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Table 1

Sample Demographics.

	<i>DATI*9R</i> <i>n=82</i>	<i>DATI*10R</i> <i>n=106</i>	Effect Size [95% CI] ^a
Demographics			
Age	46.60 (9.09)	45.15 (9.05)	0.08 [−0.07;0.22]
Household Income (\$K)	68.74 (174.94)	45.47 (126.59)	0.08 [−0.08;0.23]
Education (years)	10.84 (2.83)	11.49 (3.18)	0.11 [−0.04;0.25]
Female	24% (20)	30% (32)	0.06 [−0.08;0.20]
Race			0.07 [−0.07;0.21]
White	48% (39)	44% (47)	
African American	15% (12)	17% (18)	
Mixed Heritage	20% (16)	14% (15)	
Other	6% (5)	8% (9)	
Did not report race	12% (10)	16% (17)	
Ethnicity: Hispanic	17% (14)	24% (25)	0.08 [−0.06;0.21]
Relationship Status			0.05 [−0.08;0.19]
Single	64% (51)	67% (70)	
Committed relationship	22% (18)	16% (17)	
Married	14% (11)	16% (17)	
Sexual Orientation			0.07 [−0.07;0.21]
Lesbian or gay	42% (33)	41% (40)	
Heterosexual	49% (39)	44% (43)	
Bisexual	8% (6)	14% (14)	
Other	1% (1)	1% (1)	
HIV History			
Years since diagnosis	13.90 (7.92)	11.50 (7.93)	0.15 [0.00;0.29]
Mode of Infection			0.05 [−0.09;0.19]
Condomless sex with infected partner	64% (51)	63% (65)	
Needle sharing	16% (13)	18% (19)	
Multiple possible sources	16% (13)	13% (13)	
Other	4% (3)	6% (6)	
Currently Prescribed ARV medications	86% (70)	81% (86)	0.05 [−0.09;0.19]
Ever having an AIDS defining illness	26% (21)	34% (35)	0.06 [−0.08;0.20]
Ever developing resistance to at least one medication	13% (11)	12% (13)	0.01 [−0.13;0.15]

Notes:

^aEffect sizes are presented as correlations, calculated using Cramer's V for categorical variables and from *t*-tests for continuous variables.

Table 2

Genotype Differences among Study Measures.

	<i>DATI</i> *9R <i>n</i> =82	<i>DATI</i> *10R <i>n</i> =106	Adjusted Effect Size [95% CI]	<i>p</i> -value
Global Measures of Sexual Risk				
Sexually active in past 90 days	51% (42)	49% (40)	<i>OR</i> : 1.37 [0.76;2.45]	.29
Number of Partners in past 90 days	1.88 (4.01)	1.17 (2.07)	<i>RR</i> : 0.64 [0.50;0.81]	<.01(−)*
Number of condomless Acts in past 90 days	1.71 (4.12)	4.03 (13.90)	<i>RR</i> : 2.35 [1.94;2.85]	<.01+
How many times have you had condomless sex after drinking alcohol in past 90 days?	1.94 (6.60)	3.78 (11.22)	<i>RR</i> : 1.98 [1.64;2.40]	<.01*
Used drugs before sex during the past 90 days?	26% (22)	43% (45)	<i>RR</i> : 1.92[1.02;3.62]	.04
Event-Level Measures of Sexual Risk				
Drank last sex	43% (35)	45% (48)	<i>OR</i> : 1.14 [0.62;2.08]	.68
Binged last sex	18% (15)	27% (29)	<i>OR</i> : 1.67 [0.82;3.40]	.16
Condom use last sex	55% (58)	55% (45)	<i>OR</i> : 0.78 [0.42;1.45]	.44
Partner type last sex [casual]	45% (37)	40% (42)	<i>OR</i> : 0.78 [0.42;1.44]	.42
Treatment Adherence^a				
Detectable Viral Load	58% (30)	39% (23)	<i>OR</i> : 0.46 [0.21;1.02]	.06 (−)
% missed doses during past 4 days	11.80 (26.29)	5.09 (15.37)	<i>d</i> : −0.32 [−0.65;0.01]	.06 (−)
Drinking Attitudes and Behaviors				
AUDIT: Hazardous Alcohol Use	5.40 (2.86)	6.36 (2.70)	<i>d</i> : 0.35 [0.05;0.64]	.02
AUDIT: Dependence Symptoms	2.56 (3.48)	3.03 (3.59)	<i>d</i> : 0.13 [−0.16;0.42]	.39
AUDIT: Harmful Alcohol Use	3.33 (3.29)	3.73 (3.25)	<i>d</i> : 0.13 [−0.16;0.42]	.39
Risk Performance Assessment				
BART: Adjusted Average Number of Pumps	38.60 (14.05)	42.43 (14.01)	<i>d</i> : 0.30[0.02;0.59]	.04

Notes:

^aResults for the 156 participants who reported being on ARV medication. Data are presented as % (*n*) for categorical variables and *mean (standard deviation)* for continuous variables. Abbreviations: OR=odds ratio, RR=rate ratio, *d*= standardized difference score, (−) indicates effects that were contrary to hypotheses.

* *p* < False Discovery Rate (Benjamini-Hochberg) cut-off = .01.