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Anxiety sensitivity and hazardous drinking among persons living with HIV/AIDS: An examination of the role of emotion dysregulation

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HIGHLIGHTS

- Anxiety sensitivity (AS) is associated with hazardous drinking in seronegatives.
- Little is known about anxiety sensitivity among persons living with HIV/AIDS.
- Emotion dysregulation may underlie anxiety sensitivity and hazardous drinking.
- AS was indirectly associated with outcomes via emotion dysregulation.

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ABSTRACT

Hazardous drinking is prevalent among persons living with HIV/AIDS (PLWHA). Anxiety sensitivity is a vulnerability factor that is highly associated with hazardous drinking among seronegatives, but has yet to be tested in PLWHA. Additionally, there is a need to examine potential mechanisms underlying associations of anxiety sensitivity and hazardous drinking. Emotion dysregulation is one potential construct that may explain the association between anxiety sensitivity and hazardous drinking. The current study examined emotion dysregulation as a potential explanatory variable between anxiety sensitivity and four, clinically significant alcohol-related outcomes among PLWHA: hazardous drinking, symptoms of alcohol dependence, number of days consuming alcohol within the past month, and degree of past heavy episodic drinking. The sample included 126 PLWHA ($M_{age} = 48.3$; $SD = 7.5$; 65.9% male). Results indicated significant indirect effects of anxiety sensitivity via emotion dysregulation in all models. Indirect effects (κ^2) were of medium effect size. Alternative models were run reversing the predictor with mediator and, separately, reversing the mediator with the proposed outcome(s); alternative models yielded non-significant indirect effects in all but one case. Together, the current results indicate that anxiety sensitivity is associated emotion dysregulation, which, in turn, is associated with hazardous drinking outcomes. Overall, these findings may provide initial empirical evidence that emotion dysregulation may be a clinical intervention target for hazardous drinking.

1. Introduction

Hazardous drinking, defined as a pattern of substance use that increases the risk of harmful consequences (World Health Organization [WHO], 2015), is highly common among persons living with HIV/AIDS (PLWHA; Conigliaro et al., 2006; Schneider, Chersich, Neuman, & Parry, 2012). Hazardous drinkers do not necessarily meet full diagnostic criteria for an alcohol use disorder (AUD), but their drinking volume and patterns increase their risk of health and social problems. Even using conservative standard definitions, hazardous drinking is common among PLWHA (from 37 to 68%; Conigliaro et al., 2006), which is nearly double the rate found in the general population (Dew, Elifson, & Sterk, 2007; Galvan et al., 2002). For example, hazardous drinking has been associated with severe problems, such as HIV medication non-adherence (Kleeberger et al., 2001; Samet, Horton, Traphagen, Lyon, & Freedberg, 2003), risky sexual behavior (Ehrenstein, Horton, & Samet, 2004; Stein et al., 2005), other types of substance use, (Gonzalez, Barinas, &

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O'Cleirigh, 2011), smoking (Vidrine, Marks, Arduino, & Gritz, 2012), global psychological and physical health complications (Dew et al., 1997), rapid disease progression (Conigliaro, Gordon, McGinnis, Rabeneck, & Justice, 2003), medication toxicities (Fein, Fletcher, & Di Sclafani, 1998), peripheral neuropathy (Ferrari & Levine, 2010), organ failure, and poor virologic control (Arnsten et al., 2001), and may lead to increased risk of transmission and premature death (Galvan et al., 2002).

Individual differences in psychological factors are an important consideration for better understanding hazardous drinking among PLWHA (for review, see Shuper et al., 2010). Anxiety sensitivity is one individual difference construct that may be particularly relevant to hazardous drinking among PLWHA. Anxiety sensitivity is a cognitive factor that reflects the extent to which an individual experiences physiological arousal as potentially harmful or dangerous (Kushner, Thuras, Abrams, Brekke, & Stritar, 2001; Reiss & McNally, 1985). Anxiety sensitivity is a risk factor for anxiety and depression (Naragon-Gainey, 2010) and it has consistently been related to hazardous drinking among those without HIV (seronegatives; Schmidt, Buckner, & Keough, 2007; Stewart, Peterson, & Pihl, 1995; Stewart, Samoluk, & MacDonald, 1999). Research suggests greater arousal-dampening effects of alcohol for individuals with higher anxiety sensitivity when compared with lower anxiety sensitivity (e.g., Stewart, Zvolensky, & Eifert, 2001; Zack, Poulos, Aramakis, Khamba, & MacLeod, 2007). Individuals with higher anxiety sensitivity also report greater alcohol-related problems, including increased rates of excessive alcohol consumption (Conrod, Stewart, & Pihl, 1997; Stewart et al., 1999), drinking to legal intoxication more frequently (Stewart et al., 1995, 2001), and higher rates of alcohol dependence (Lewis & Vogeltanz-Holm, 2002). Further, longitudinal studies have implicated anxiety sensitivity in the development of alcohol problems. For example, Schmidt et al. (2007) reported that individuals with high anxiety sensitivity were more likely to have developed an alcohol use disorder after 24 months than were individuals with low anxiety sensitivity. However, little is known about relationship between anxiety sensitivity and hazardous drinking among PLWHA. Anxiety sensitivity may be particularly important in PLWHA due to the common physiological arousal/distress associated with symptoms of disease progression and medication side effects (Ammassari et al., 2001).

In addition to examining the direct association of anxiety sensitivity and hazardous alcohol use among PLWHA, there is a need to explicate the processes governing such associations. Indeed, examining underlying factors may help to explicate explanatory mechanisms by which anxiety sensitivity may impact alcohol use in this population. One construct that may provide explanatory value among associations of anxiety sensitivity and hazardous drinking is emotion dysregulation (Chandley, Luebbe, Messman-Moore, & Ward, 2014). Emotion dysregulation has been defined as difficulties engaging a set of abilities wherein one can observe, understand, evaluate, and differentiate one's emotions and subsequently access strategies to regulate emotions and control behavioral responses (Gratz & Roemer, 2004; Tull & Aldao, 2015). Generally, emotion dysregulation is associated with increased alcohol consumption and dependence (Berking et al., 2011), as well as increased alcohol-related problems (Dvorak et al., 2014). Among PLWHA, those meeting criteria for hazardous drinking have greater levels of emotion dysregulation, relative to those not meeting such criteria (Garey et al., 2015).

Theoretically, individuals with greater anxiety sensitivity may respond to physiological sensations (e.g., those associated with anxiety) with less acceptance (i.e., greater emotion dysregulation), resulting in greater subjective distress (Kashdan, Zvolensky, & McLeish, 2008). As a result of such emotion dysregulation, these individuals may use alcohol as a means of regulating negative emotions. Importantly, the theoretical framework derived from other areas of research in substance use (e.g., smoking; Johnson, Farris, Schmidt, & Zvolensky, 2012) indirectly support emotion dysregulation as a factor underlying anxiety sensitivity and hazardous drinking. For example, Johnson et al. (2012) demonstrated evidence of an indirect effect from anxiety sensitivity to smoking-relevant outcomes via emotion dysregulation. Currently, no such model has been tested examining anxiety sensitivity, emotion dysregulation, and hazardous alcohol use in general or among PLWHA specifically.

Together, the current study tested the hypothesis that anxiety sensitivity would exert an indirect effect on alcohol-related criterion variables via emotion dysregulation (see Fig. 1). Specifically, anxiety sensitivity was expected to positively predict emotion dysregulation, which, in turn, would be associated with the alcohol dependent variables. In the current study, four clinically significant dependent variables identified in past work among PLWHA (e.g., Fiellin, McGinnis, Maisto, Justice, & Bryant, 2013; Surah et al., 2013) were evaluated: 1) hazardous drinking, 2) symptoms of alcohol dependence, 3) number of days consuming alcohol within the past month, and 4) past report of heavy episodic drinking. It was expected that such an effect of anxiety sensitivity via emotion dysregulation would be evident on all criterion measures over and above variance accounted for by the following covariates: gender, sexual orientation, time since HIV diagnosis, and presence of a substance use disorder. These covariates were selected as past work has shown significant associations of each with alcohol consumption (Conen et al., 2009; Marshal et al., 2008; Nolen-Hoeksema, 2004).

2. Method

2.1. Participants

Participants included 129 adults living with HIV/AIDS recruited from AIDS service organizations in Houston, Texas. Flyers were placed in local community health clinics, and doctors' offices as well as in newspaper/magazine advertisements and on webpage announcements (e.g., Craigslist.com). Further advertisement was conducted via public speaking engagements (e.g., Ryan White Foundation Houston, Houston AIDS Foundation) and word-of-mouth. Interested individuals contacted our clinic/research lab to schedule an appointment, completed confidentially (i.e., no identifying information on study materials). Participants were eligible for inclusion if they were at least 18 years old, were previously diagnosed with HIV/AIDS per self-report, and had the cognitive capacity to give written informed consent, as assessed by their ability to read the consent form and explain the study purpose to assessment personnel. Study measures were completed as part of a larger assessment battery. Participants were paid $20 in gift cards for completing a two-hour baseline assessment consisting of diagnostic interview and questionnaires.

Three participants were missing data on one or more measures of interest and were excluded from analyses yielded 126 individuals for the current study. The majority of participants (65.9%) were male and the mean age was 48.3 years ($SD = 7.5$). In terms of ethnicity, 55.1% of the sample identified as Black, 28.6% as White/Caucasian, 13.4% as Hispanic, and 4.5% identified as "mixed/other" (e.g., Native American). Regarding sexual orientation, 45.2% identified as heterosexual, 38.1% homosexual, 14.3% bisexual, and 2.4% 'other'. Although 85.7% reported completion of high school or further education, 74.3% of participants reported current unemployment and 55.6% reported earning <$10,000 annually. The average CD4 t-cell count within the sample was 567.3

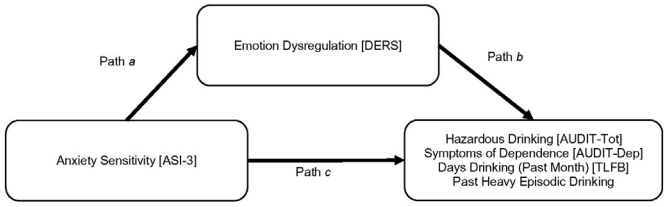

Fig. 1. Proposed model examining the indirect effect of Anxiety Sensitivity on Alcohol Use criterion variables (AUDIT-Tot, AUDIT-Dep, TLFB, and Past Heavy Episodic Drinking) via Difficulties with Emotion Regulation.

(*SD* = 264.5; range: 28–1300); CD4 counts range from 500 to 1200 in seronegative individuals with CD4 ≤200 contributing to a diagnosis of AIDS. Less than half (43.7%) self-reported diagnosis of AIDS. The majority of participants (87.3%) reported current use of anti-retroviral therapy (ART). Of those on ART, 78.2% reported having an undetectable viral load. The sample reported having a diagnosis of HIV for an average of 17.0 years (*SD* = 8.6). The most common psychological diagnoses were generalized anxiety disorder (26.2%), major depressive disorder (24.3%), and (non-alcohol) substance use disorder (SUD; 22.2%).

2.2. Measures

2.2.1. MINI international neuropsychiatric interview

(MINI; Lecrubier et al., 1997). The MINI is a brief semi-structured diagnostic interview, which was developed in order to assess for the presence of current Axis I psychological disorders (e.g., anxiety, mood, substance-use, psychosis) based on DSM-IV criteria. The MINI has been utilized in prior studies examining HIV + samples (e.g. Breuer et al., 2014) and has been deemed psychometrically sound (see Lecrubier et al., 1997). A subset of 12.5% of cases where checked for diagnostic reliability by a research staff member; no cases of disagreement were noted.

2.2.2. Anxiety sensitivity index-3

(ASI-3; Taylor et al., 2007). The ASI-3, derived in part from the original ASI, is an 18-item self-report measure of anxiety sensitivity (Reiss & McNally, 1985). Items (e.g., "When my stomach is upset, I worry that I might be seriously ill") are rated on a scale from 0 (*very little*) to 4 (*very much*) and summed to a total score. The ASI-3 maintains strong psychometric properties (Farris et al., 2015; Taylor et al., 2007). ASI-3 scores in the current sample (*M* = 26.06, *SD* = 18.54) are similar to those in past studies among PLWHA (M = 27.10, SD = 26.21; Garey et al., 2015). In the current sample, internal consistency was excellent (α = 0.96) per commonly used interpretive range (i.e., ≥0.9; George & Mallery, 2003).

2.2.3. Difficulties in emotion regulation scale

(*DERS;* Gratz & Roemer, 2004). The DERS is a 36-item multidimensional self-report measure of emotion dysregulation. Participants rate each item (e.g., "I experience my emotions as overwhelming and out of control") on a 5-point Likert scale ranging from 1 (*almost never*) to 5 (*almost always*) and summed to a total score. The DERS has demonstrated excellent psychometric properties including test-retest reliability, construct validity and predictive validity (Gratz & Roemer, 2004) and has been in samples of PLWHA (Brandt, Gonzalez, Grover, & Zvolensky, 2012). DERS scores in the current sample (*M* = 88.22, *SD* = 27.72) are similar to those in past studies among PLWHA (M = 91.41, SD = 26.21; Garey et al., 2015). Internal consistency was excellent in the current study (α = 0.95).

2.2.4. The alcohol use disorders identification test (AUDIT; Babor, Higgins-Biddle, Saunders, & Monteiro, 2001)

The AUDIT is a 10-item self-report measure that was developed by the World Health Organization to assess problematic alcohol use (Babor et al., 2001). Items (e.g., "How often do you have a drink containing alcohol?") are rated from 0 to 4 and summed to a total score. The AUDIT has strong psychometric properties, including reliability and validity (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). In addition to a total score (AUDIT-Tot), the three items targeting alcohol dependence (AUDIT-Dep; e.g., "How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?") are used to screen for problematic alcohol use. In the current study, AUDIT-Tot and AUDIT-Dep were used as criterion variables and had good internal consistencies among the current sample (α = 0.86, and 0.77, respectively), consistent with past work among PLWHA (Surah et al., 2013). Scores on the AUDIT-Tot (*M* = 4.80, *SD* = 6.28) in the current study are similar to those obtained in past work among PLWHA (median AUDIT-Tot score = 5; Surah et al., 2013), although many studies (e.g., Trillo et al., 2013; Woolf-King, Neilands, Dilworth, Carrico, & Johnson, 2014) have reported only AUDIT categories among PLWHA (e.g., abstainer, low-risk) and not average scores, highlighting a need for additional work in this area. In this sample, 20.6% met criteria for hazardous drinking on the AUDIT-Tot (total scores of 8 or greater for men/7 or greater for women; Conigrave, Hall, & Saunders, 1995).

2.2.5. Timeline follow-back (TLFB; Sobell & Sobell, 1995)

The TLFB is a self-report measure that obtains day-by-day estimates of drinking for a designated period of time. The current study used the past 30-day TLFB, which assessed the number of days during which alcohol was consumed over that period. Individuals were presented with a calendar on which they write events, which then serve as memory prompts for estimating the days during which they consumed alcohol. The TLFB has been deemed a reliable measure of alcohol consumption with excellent temporal stability (Carey, Carey, Maisto, & Henson, 2004; Sobell, Sobell, Leo, & Cancilla, 1988) and has been used as a measure of alcohol consumption among PLWHA (Fiellin et al., 2013). Number of days drinking in the past 30 days in the current sample (*M* = 4.72, *SD* = 8.63, 15.73% of days) is consistent with past work using the 30-day TLFB among PLWHA (16.60% of days; Simpson, Xie, Blum, & Tucker, 2011).

2.2.6. Past heavy episodic drinking

To approximate the degree past heavy episodic drinking, one item ("During the period in your life when you were drinking most heavily, how often did you have 6 [if you are a man]/4 [if you are a woman] drinks on one occasion?") was created. Participants provided a rating on a scale from 0 *"never"* to 4 *"daily or almost daily"*. While the cutoff of 5 or more drinks is commonly used to mark heavy episodic drinking, others (e.g., Mäkelä et al., 2001) have used the cutoff of 6 or more drinks in one occasion.

2.3. Data analytic strategy.

Analyses were conducted using the PROCESS macro for SPSS 20 (Hayes, 2012), which calculates the indirect effect of a predictor (X) on an outcome (Y) via some mediating factor (M; West & Aiken, 1997). Effect sizes (κ^2) were calculated for the indirect effects, following recommendations of Preacher and Kelly (2011). Bootstrapping with 10,000 re-samples with replacement was performed to obtain 95% confidence intervals. The association of anxiety sensitivity via emotion dysregulation was examined with four dependent variables: hazardous drinking (AUDIT-Tot), symptoms of alcohol dependence (AUDIT-Dep), number of days drinking in the past month (TLFB), and degree of past heavy episodic drinking. Covariates included gender, sexual orientation, time since diagnosis, and presence of an SUD. For each model, two planned comparison models were also evaluated. First, the predictor and mediating factor were reversed such that the effects of emotion dysregulation via anxiety sensitivity were evaluated. Next, the outcome and mediating factor were reversed to evaluate the possibility of anxiety sensitivity predicting emotion dysregulation via the alcohol-related variables. It was hypothesized that each of the comparison models would yield non-significant indirect effects, adding confidence to the focal models (Fig. 1) testing effects of anxiety sensitivity via emotion dysregulation.

3. Results

Descriptive statistics and Pearson correlations are presented in Table 1. At the bivariate level, anxiety sensitivity was strongly associated with emotion dysregulation ($r = 0.67$; $p < 0.001$) and weakly with hazardous drinking ($r = 0.19$; $p < 0.001$). Emotion dysregulation was

Table 1
Zero-order correlations among study variables.

Variable		1	2	3	4	5	6	7	8	9	10
1. Gender[a]		–	−0.56***	0.10	−0.10	−0.09	0.16	0.02	0.30**	0.10	−0.09
2. Sexual orientation[a]			–	−0.23*	0.01	0.05	−0.09	−0.03	−0.31***	−0.11	0.18*
3. Time since diagnosis[a]				–	−0.05	−0.18*	0.07	0.09	0.07	0.07	−0.21*
4. SUD[a]					–	0.13	0.13	0.15	0.09	0.07	0.12
5. ASI-3[b]						–	0.19*	0.16	0.04	0.01	0.67***
6. AUDIT-Tot[c]							–	0.82***	0.36***	0.42***	0.28**
7. AUDIT-Dep[c]								–	0.26**	0.34**	0.26**
8. TLFB[c]									–	0.14	0.13
9. HED[c]										–	0.14
10. DERS[d]											–
Descriptive statistics	Mean (n)	83	57	204.25	28	26.06	4.80	0.98	4.72	1.74	88.22
	SD (%)	65.90	45.20	103.33	22.2	18.54	6.28	2.18	8.63	1.57	27.72

Note: Gender = coded as Male = 1 and Female = 0, with descriptive statistics for number and percentage male; Sexual Orientation, coded as Heterosexual = 1 and Gay/Lesbian/Bisexual/Other = 0, with descriptive statistics for number and percentage heterosexual; Time since Diagnosis = time in months since diagnosed with HIV; SUD = Substance Use Disorder diagnosis, coded as yes = 1 and no = 0 with descriptive statistics for number and percentage with a diagnosis; ASI-3 = Anxiety Sensitivity Index-3; AUDIT-Tot = total scale score on the Alcohol Use Disorders Identification Test; AUDIT-Dep = Alcohol Use Disorders Identification Test, Dependence symptoms subscale; TLFB = Timeline Follow-Back, Number of Drinking Days (Past Month); HED = Past Heavy Episodic Drinking; DERS = Difficulties with Emotion Regulation Scale.

[a] Covariates.
[b] Predictor.
[c] Outcome Variable.
[d] Mediator.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

moderately associated with hazardous drinking ($r = 0.28$; $p < 0.001$) and symptoms of alcohol dependence ($r = 0.26$; $p < 0.001$). Data were normally distributed with skewness within acceptable range (0.2–2.0; George & Mallery, 2003). See Table 2 for model fit statistics.

In the model of hazardous drinking (AUDIT-tot), there was a significant positive indirect effect of anxiety sensitivity via emotion dysregulation (unstandardized point estimate = 0.07, $SE = 0.03$, BC 95% CI: 0.02 to 0.14; direct effect of anxiety sensitivity controlling for emotion dysregulation = 0.01, $SE = 0.04$, $t = 0.04$; $p = 0.970$). The size of the indirect effect via emotion dysregulation was medium (completely standardized point estimate = 0.20, $SE = 0.08$, BC 95% CI: 0.05 to 0.36; $\kappa^2 = 0.14$, $SE = 0.06$, BC 95% CI: 0.02 to 0.26). The first comparison model yielded non-significant indirect effect (emotion dysregulation as X, anxiety sensitivity as M, hazardous drinking as Y; unstandardized point estimate = 0.01, $SE = 0.02$, BC 95% CI: −0.04 to 0.04); however, the second comparison model yielded a significant indirect effect (anxiety sensitivity as X, hazardous drinking as M, emotion dysregulation as Y; unstandardized point estimate = 0.05, $SE = 0.04$, BC 95% CI: 0.01 to 0.15).

In predicting symptoms of dependence (AUDIT-Dep) scores, there was a significant positive indirect effect of anxiety sensitivity via emotion dysregulation (unstandardized point estimate = 0.02, $SE = 0.01$, BC 95% CI: 0.01 to 0.05; direct effect of anxiety sensitivity controlling for emotion dysregulation = −0.01, $SE = 0.01$, $t = -0.19$; $p = 0.851$). The size of the indirect effect via emotion dysregulation was medium (completely standardized point estimate = 0.19, $SE = 0.07$, BC 95% CI: 0.05 to 0.34; $\kappa^2 = 0.14$, $SE = 0.05$, BC 95% CI: 0.03 to 0.24). Neither of the comparison models were significant (comparison model 1 [emotion dysregulation as X, anxiety sensitivity as M, symptoms of dependence as Y]: unstandardized point estimate = 0.01, $SE = 0.01$, BC 95% CI: −0.02 to 0.01; comparison model 2 [anxiety sensitivity as X, symptoms of dependence as M, emotion dysregulation as Y]: unstandardized point estimate = 0.04, $SE = 0.03$, BC 95% CI: −0.01 to 0.12).

There was a significant positive indirect effect of anxiety sensitivity via emotion dysregulation in predicting number of days drinking (TLFB; unstandardized point estimate = 0.08, $SE = 0.04$, BC 95% CI: 0.01 to 0.16; direct effect of anxiety sensitivity controlling for emotion dysregulation = −0.05, $SE = 0.05$, $t = -0.96$, $p = 0.340$). The size of the indirect effect via emotion dysregulation was medium (completely standardized point estimate = 0.18, $SE = 0.08$, BC 95% CI: 0.03 to 0.34; $\kappa^2 = 0.09$, $SE = 0.06$, BC 95% CI: 0.01 to 0.21). Neither of the comparison models were significant (comparison model 1 [emotion dysregulation as X, anxiety sensitivity as M, number of days drinking as Y]: unstandardized point estimate = −0.02, $SE = 0.02$, BC 95% CI: −0.07 to 0.03; comparison model 2 [anxiety sensitivity as X, number of days drinking as M, emotion dysregulation as Y]: unstandardized point estimate = 0.01, $SE = 0.02$, BC 95% CI: −0.02 to 0.07).

For past heavy episodic drinking, there was a significant positive indirect effect of anxiety sensitivity via emotion dysregulation (unstandardized point estimate = 0.01, $SE = 0.01$, BC 95% CI: 0.01 to 0.03; direct effect of anxiety sensitivity controlling for emotion dysregulation = −0.01, $SE = 0.01$, $t = -1.16$, $p = 0.252$). The size of the indirect effect via emotion dysregulation was medium (completely standardized point estimate = 0.17, $SE = 0.09$, BC 95% CI: 0.01 to 0.35; $\kappa^2 = 0.12$, $SE = 0.06$, BC 95% CI: 0.01 to 0.24). Neither of the comparison models were significant (comparison model 1 [emotion dysregulation as X, anxiety sensitivity as M, heavy episodic drinking as Y]: unstandardized point estimate = −0.01, $SE = 0.01$, BC 95% CI: −0.02 to 0.01; comparison model 2 [anxiety sensitivity as X, heavy episodic drinking as M, emotion dysregulation as Y]: unstandardized point estimate = 0.01, $SE = 0.03$, BC 95% CI: −0.06 to 0.06).[1]

4. Discussion

The current study examined the indirect effects of anxiety sensitivity via emotion dysregulation in predicting hazardous drinking, symptoms of alcohol dependence, past-month alcohol consumption, and degree of past heavy episodic drinking among a sample of PLWHA. As hypothesized, there were significant indirect effects of anxiety sensitivity via emotion dysregulation for four all models tested. The size of these

[1] Moderated mediation models were also tested using PROCESS. Separate analyses examined each covariate (gender, sexual orientation, time since HIV diagnosis, and presence of a substance use disorder) as moderators of the indirect association of anxiety sensitivity via emotion dysregulation on each of the four outcomes. There were significant indirect effects in males and females, for heterosexual and lesbian/gay/bisexual/other individuals, for those with high, average, and low time since diagnosis, and for those with and without SUD in all four models, respectively. Full results can be obtained by contacting the corresponding author.

Table 2
Model fit statistics.

$N = 126$ Antecedent		Consequent								
		Mediator (M) DERS					Criterion AUDIT-Tot			
	Path	Coeff.	*SE*	*t*	*p*	Path	Coeff.	*SE*	*T*	*p*
ASI-3	*a*	0.98	0.10	9.70	<0.001	*c′*	0.01	0.04	0.04	0.970
DERS	–	–	–	–	–	*b*	0.07	0.03	2.60	0.011
Gender		4.56	4.65	0.98	0.329		2.34	1.37	1.71	0.090
Time since Dx		−0.01	0.02	−0.77	0.442		0.01	0.01	1.28	0.204
SO		9.77	4.50	2.17	0.032		−0.28	1.34	−0.21	0.834
SUD		2.68	4.43	0.60	0.547		1.78	1.30	1.37	0.174
constant		57.55	7.45	7.72	<0.001		−4.56	2.67	−1.71	0.090
		$R^2 = 0.48$					$R^2 = 0.14$			
		$F(5, 120) = 22.21$, $p < 0.001$					$F(6, 119) = 3.24$, $p = 0.006$			

$N = 126$ Antecedent		Consequent								
		Mediator (M) DERS					Criterion AUDIT-Dep			
	Path	Coeff.	*SE*	*t*	*p*	path	Coeff.	*SE*	*t*	*p*
ASI-3	*a*	0.98	0.10	9.70	<0.001	*c′*	−0.01	0.01	−0.19	0.851
DERS	–	–	–	–	–	*b*	0.02	0.01	2.44	0.016
Gender		4.56	4.65	0.98	0.329		0.10	0.49	0.20	0.843
Time since Dx		−0.01	0.02	−0.77	0.442		0.01	0.01	1.53	0.130
SO		9.77	4.50	2.17	0.032		−0.19	0.48	−0.40	0.689
SUD		2.68	4.43	0.60	0.547		0.64	0.46	1.39	0.166
constant		57.55	7.45	7.72	<0.001		−1.71	0.95	−1.81	0.073
		$R^2 = 0.48$					$R^2 = 0.10$			
		$F(5, 120) = 22.21$, $p < 0.001$					$F(6, 119) = 2.30$, $p = 0.039$			

$N = 126$ Antecedent		Consequent								
		Mediator (M) DERS					Criterion TLFB			
	Path	Coeff.	*SE*	*t*	*p*	Path	Coeff.	*SE*	*t*	*p*
ASI-3	*a*	0.98	0.10	9.70	<0.001	*c′*	−0.05	0.05	−0.96	0.340
DERS	–	–	–	–	–	*b*	0.08	0.04	2.25	0.026
Gender		4.56	4.65	0.98	0.329		3.38	1.84	1.84	0.069
Time since Dx		−0.01	0.02	−0.77	0.442		0.01	0.01	0.41	0.680
SO		9.77	4.50	2.17	0.032		−4.18	1.81	−2.31	0.023
SUD		2.68	4.43	0.60	0.547		2.03	1.75	1.16	0.248
constant		57.55	7.45	7.72	<0.001		−2.50	3.60	−0.70	0.488
		$R^2 = 0.48$					$R^2 = 0.17$			
		$F(5, 120) = 22.21$, $p < 0.001$					$F(6, 119) = 4.16$, $p < 0.001$			

$N = 86$ Antecedent		Consequent								
		Mediator (M) DERS					Criterion HED			
	Path	Coeff.	*SE*	*t*	*p*	Path	Coeff.	*SE*	*t*	*p*
ASI-3	*a*	0.89	0.12	7.64	<0.001	*c′*	−0.01	0.01	−1.16	0.252
DERS	–	–	–	–	–	*b*	0.02	0.01	1.80	0.076
Gender		7.66	5.27	1.45	0.150		0.12	0.42	0.30	0.769
Time since Dx		−0.01	0.02	-0.20	0.843		0.01	0.01	0.40	0.693
SO		10.66	5.26	2.03	0.046		−0.36	0.43	−0.84	0.404
SUD		0.46	5.40	0.08	0.933		0.32	0.43	0.75	0.457
constant		57.03	8.60	6.63	<0.001		0.59	0.85	0.70	0.487
		$R^2 = 0.45$					$R^2 = 0.06$			
		$F(5, 80) = 13.03$, $p < 0.001$					$F(6, 79) = 0.86$, $p = 0.530$			

effects were medium in each model $\kappa^2 = 0.09$–0.14), and evident over and above variance accounted for by the covariates (gender, sexual orientation, time since HIV diagnosis, and presence of an SUD). The lack of impact of SUD was surprising. Indeed, there were no bivariate correlations of SUD with any outcome, and SUD was not a significant covariate in any model tested. Thus, hazardous drinking appears to be present, and clinically significant, independent of SUD. Future work should follow up on the interplay of alcohol and other substance use in this population. As data were collected at one time point, competing models were run to examine potential model misspecification. For each hypothesized model (testing the indirect effect of anxiety sensitivity via emotion dysregulation), two competing models were run (i.e., eight total comparison models). The competing models yielded non-significant indirect effects in all but one case (i.e., confidence intervals for the indirect effect contained '0'), adding confidence to the hypothesized model testing the indirect association of anxiety sensitivity via emotion dysregulation. Overall, the regulation of emotions may represent an important intermediate variable linking catastrophic interpretation of physiological arousal (anxiety sensitivity) to hazardous alcohol use. Indeed, the present data suggest anxiety sensitivity may be related to emotion dysregulation, which, in turn, may affect hazardous drinking. Future longitudinal work is needed to further evaluate this model given the current cross-sectional findings.

Interestingly, the direct and total effects of anxiety sensitivity in predicting the alcohol-related outcomes were not statistically significant in any of the models, with the exception of a significant total effect for hazardous drinking (see Table 3). Additionally, there was one significant bivariate correlation (i.e., ASI-3 and AUDIT-Total), the association failed to maintain significance once covariates were entered. The lack of a consistent direct/total effect of anxiety sensitivity was unexpected

Table 3
Direct, indirect, and total unstandardized effects of anxiety sensitivity on hazardous drinking outcomes via difficulties in emotion regulation.

Criterion	Effect of IV on M	Effect of M on DV	Direct effect	Indirect effect		Total effect	
	(*a*)	(*b*)	(*c*′)	(*a* × *b*)	95% CI	(*c*)	95% CI
AUDIT-Tot	0.98	0.07	0.01	0.07	0.02–0.14	0.07	0.01–0.13
AUDIT-Dep	0.98	0.02	−0.01	0.02	0.01–0.05	0.02	−0.01–0.04
TLFB	0.98	0.08	−0.05	0.08	0.01–0.16	0.03	−0.05–0.11
HED	0.89	0.02	−0.01	0.01	0.01–0.03	0.01	−0.02–0.02

given well-documented associations of elevated anxiety sensitivity and hazardous drinking among non-HIV samples (DeMartini & Carey, 2011). One possible explanation for this set of findings is that anxiety sensitivity may be more strongly associated with drinking motives (e.g., negative affect reduction) than with drinking behavior, although this remains to be empirically tested. These data suggest the association between anxiety sensitivity and hazardous drinking outcomes (particularly symptoms of dependence, past-month drinking, and heavy episodic drinking) may be more complex among PLWHA, with the current results suggesting an indirect association via emotion dysregulation.

The findings of the present study may potentially inform interventions for hazardous drinking among PLWHA. These results suggest that targeting and improving emotion dysregulation may have benefits in terms of addressing hazardous drinking. Such an approach would be consistent with intervention work on emotion dysregulation and alcohol use disorders among non-HIV samples (e.g., Berking et al., 2011). For example, changes in the use of maladaptive emotion regulation strategies over the course of treatment have been shown to predict changes in anxiety- and alcohol-related psychopathology in a sample with comorbid anxiety and alcohol use disorders (Conklin et al., 2015). Given that newer treatments directly targeting emotion regulation are mounting growing evidence in treating a range of emotional outcomes (Gratz, Weiss, & Tull, 2015), such treatments may serve as additional options for the treatment of hazardous drinking among PLWHA.

The current study has several limitations. First, due to the cross-sectional design of the study, temporal ordering cannot be elucidated. Attempts were made to examine alternative models, which were rejected, adding confidence to the findings, but future work utilizing a longitudinal design is required to determine temporal effects and rule out the possibility of alternative explanations (e.g., alcohol use leading to emotion dysregulation, and anxiety sensitivity, in turn). Second, method variance may have influenced the current findings. All measures, while validated, were completed via self-report. Thus, future work should consider multi-method assessment approaches to index the constructs of interest. Third, although the sample was diverse in terms of ethnicity, it was limited to an older adult, primarily male group of individuals living with HIV/AIDS who volunteered to participate in a study for monetary reward. Although men comprise a large percentage of the HIV/AIDS population (Vermund, 2014), future studies would benefit from examining more heterogeneous samples of persons with HIV/AIDS. Moreover, it may be advisable to offer other types of incentives instead of those that are financial in nature to ascertain whether there is any type of sampling bias. The current study was underpowered to test more complex interactions (e.g., gender by sexual orientation by substance user) and future work using large samples should replicate and extend the current findings to probe for such interactions. Relatedly, it is possible that clusters of individuals (e.g., with specific racial, gender, and sexual orientation identities with/without certain diagnoses) may demonstrate unique associations, as is the case in any diverse and heterogeneous sample. Nevertheless, bootstrapping with 10,000 resamples of the data was used to identify significant 95% confidence bands around the indirect effects, adding confidence to the findings. Finally, the study criterion variables were limited to numerous indices of hazardous drinking. Future work could potentially benefit by further extending the present work to other alcohol-related processes, such as motives and outcome expectancies for use. It also may be advisable to explore how anxiety sensitivity and emotion dysregulation relate to a broader array of alcohol-related impairment indices, such as quality of life.

Overall, the present study serves as an initial exploration into the association between anxiety sensitivity, emotion dysregulation, and hazardous drinking among PLWHA. There was consistent empirical evidence of indirect associations of anxiety sensitivity via emotion dysregulation. Accordingly, if replicated and extended using prospective designs, treatments for hazardous drinking may benefit from consideration of emotion regulation therapeutic tactics.

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Contributors

Mr. Brandt and Dr. Zvolensky designed the parent study and data collection. Mr. Paulus and Mr. Brandt conducted literature searches and provided summaries of previous research studies for the current paper. Mr. Paulus and Mr. Jardin, conducted the statistical analysis and wrote the first draft of the manuscript. All authors edited the manuscript and provided text used in the final draft. All authors have approved the final manuscript.

Conflict of interest

This work has not been presented previously in any form. No authors have any conflicts of interests or financial disclosures to report. The study was approved by Institutional Review Board at the University of Houston. Informed written consent was obtained prior to initiating study procedures. No animals have been employed in this research.

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