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Intraindividual change in anxiety sensitivity and alcohol use severity 12-months following smoking cessation treatment

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ABSTRACT

Although past work has documented reduction in alcohol use severity among smokers following smoking cessation treatment, little is known regarding factors associated with this reduction. The current study sought to examine relations between trajectories of change in anxiety sensitivity and non-targeted alcohol use severity from baseline to one year following smoking cessation treatment. Individuals (n = 386) were adult daily smokers engaged in a smoking cessation treatment study. Measures of alcohol use severity and anxiety sensitivity were collected at baseline as well as 1-, 3-, 6-, and 12-months post-treatment. Latent growth curve modelling was used to estimate intercepts and slopes. Anxiety sensitivity (M = −0.87 95% CI [-1.19, −0.54], p < 0.001) and alcohol use severity (M = −0.22 95% CI [-0.38, −0.06], p = 0.006) each significantly reduced over time. Reductions in anxiety sensitivity were strongly associated with reductions in alcohol use over time (r = 0.63, 95% CI [0.18, 1.09], p = 0.006). Changes in anxiety sensitivity positively correlated with changes in alcohol use severity. Examinations of means suggest that anxiety sensitivity reduced earlier whereas alcohol use severity reduced later in the follow-up period. If replicated establishing temporal precedence of change, these results could implicate anxiety sensitivity reduction as one avenue towards reduced alcohol use severity, among smokers.

Although smoking rates have generally declined, a significant portion of the United States (U.S.) population initiates smoking each year (Centers for Disease Control and Prevention, 2010) and smoking remains the leading cause of preventable death (U.S. Department of Health and Human Services, 2014). Adding to the health risks associated with smoking is the use of alcohol, which has been consistently linked to smoking (Anthony & Echeagaray-Wagner, 2000; Falk, Yi, & Hiller-Sturmhöfel, 2006; Kahler et al., 2008; Palfai, Monti, Ostafin, & Hutchison, 2000). For example, the co-use of alcohol and tobacco is associated with additive health risk greater than the independent risk either alone (Hart, Smith, Gruer, & Watt, 2010; B.; Taylor & Rehm, 2006). Additionally, past work suggests approximately 85% of smokers drink alcohol and drinkers are 75% more likely to smoke relative to those who abstain (Harrison, Hinson, & McKee, 2009; Krukowski, Solomon, & Naud, 2005; Reed, Wang, Shillington, Clapp, & Lange, 2007), highlighting both the prevalence of alcohol use among smokers and the impact it can have on smoking behavior. Alcohol use is particularly important to consider during quit attempts as it is associated with greater risk for relapse following a quit attempt (Humfleet, Muñoz, Sees, Reus, & Hall, 1999). Furthermore, smokers with alcohol problems report lower tobacco quit rates (Hughes & Kalman, 2006) and die at higher rates due to smoking-related illness relative to smokers without alcohol problems (Hurt et al., 1996). Thus, the comorbidity of alcohol use and smoking represents an important public health concern (Romberger & Grant, 2004).

Importantly, alcohol use can be impacted during treatment for smoking. For example, past work has documented a lower prevalence of alcohol use disorder over time following a successful smoking quit

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attempt (Cavazos-Rehg et al., 2014). Additionally, there is some evidence for the decline of alcohol use during smoking cessation treatment. For example, among Puerto Rican daily smokers who were also at-risk drinkers, a randomized controlled trial (RCT) comparing motivational enhancement and problem solving/coping skills (MAPS) for smoking cessation to MAPS integrating smoking cessation and reduction of at-risk behaviors (MAPS +) resulted in greater reduction of at-risk drinking in the MAPS + condition among individuals quitting smoking (Correa-Fernández et al., 2017). Similarly, among military beneficiaries, a RCT comparing standard smoking cessation alone vs. in combination with weight management and alcohol reduction strategies alcohol consumption reduced by approximately one third, with no significant difference in the reduction of alcohol use based on treatment condition (Sobell et al., 2017). This available work highlights that alcohol use may be reduced over the course of smoking cessation treatment, even when not directly targeted. This is important when considering the common co-use of tobacco and alcohol, and clinical need to reduce both. Yet, relatively little is known regarding the *trajectory* of alcohol use over the course of smoking cessation treatment or correlates/predictors of the trajectory.

Although this past work shows the promise of reducing alcohol use within a smoking cessation treatment framework, such work could be aided by identifying and explicitly targeting potential mechanisms driving the use of alcohol and smoking. One potential mechanism with relevance to smoking and alcohol use is anxiety sensitivity, an individual difference construct reflecting the tendency to interpret interoceptive sensations as personally dangerous (Reiss, 1991; Reiss & McNally, 1985). Although well-validated as a risk factor for affective domains such as anxiety and depression (Naragon-Gainey, 2010; Olatunji & Wolitzky-Taylor, 2009), anxiety sensitivity has shown strong relations to substance use such as smoking (Leventhal & Zvolensky, 2015) and alcohol use (DeMartini & Carey, 2011). Specifically, individuals with elevated anxiety sensitivity report greater alcohol-related problems (Conrod, Stewart, & Pihl, 1997; Stewart, Samoluk, & MacDonald, 1999) and have higher rates of alcohol dependence (Lewis & Vogeltanz-Holm, 2002), greater likelihood of alcohol use disorder/substance use disorder (Allan, Macatee, Norr, Raines, & Schmidt, 2015) and more frequently drink to intoxication (Stewart, Peterson, & Pihl, 1995; Stewart, Zvolensky, & Eifert, 2001). Research also suggests that those with elevated anxiety sensitivity experience greater arousal-dampening effects of alcohol (e.g., Stewart et al., 2001; Zack, Poulos, Aramakis, Khamba, & MacLeod, 2007) and engage in drinking to cope with distress (Mackinnon, Kehayes, Clark, Sherry, & Stewart, 2014). Among smokers, anxiety sensitivity has also been linked to problematic alcohol use and coping motives for use (Allan, Albanese, Norr, Zvolensky, & Schmidt, 2015; Chavarria et al., 2015; Paulus et al., 2017). Additionally, there is evidence that reductions in anxiety sensitivity during smoking cessation can relate to both lower affective symptoms (Schmidt, Raines, Allan, & Zvolensky, 2016) and higher smoking abstinence rates (Zvolensky et al., 2018). Longitudinal associations have been documented between anxiety sensitivity and alcohol use disorder (Schmidt, Buckner, & Keough, 2007) and past work has shown that changes in anxiety sensitivity over the course of treatment mediate the effect of treatment type (cognitive-behavioral therapy [CBT] for substance use vs. CBT for substance use plus CBT for anxiety) on number of post-treatment drinking days among individuals with substance use disorder and a comorbid anxiety disorder (Wolitzky-Taylor et al., 2018).

Despite this body of work, it is currently unknown how *intraindividual changes* in anxiety sensitivity relate to *intraindividual changes* in alcohol related outcomes such as severity of use (i.e., levels of consumption and consequences/problems). Furthermore, little is known how such changes in anxiety sensitivity and alcohol use relate over a longer period of time (e.g., including treatment follow-up), or among smokers, specifically. In order to inform mechanisms underlying CBT, it is necessary for longitudinal research to examine intraindividual change with methods (e.g., latent growth curve modelling) that allow for measurement of *idiographic change* (Gallagher et al., 2013). Other advanced statistical methods such as cross-lagged panel models (Cole & Maxwell, 2003) and auto-regressive latent trajectory models (Bollen & Curran, 2004), although well suited for estimation of temporal precedence of change, estimate *interindividual standing over time* rather than intraindividual change (Selig & Preacher, 2009). Thus, although such lagged models can inform whether a certain variable changes prior to another, such change is not idiographic and does not inform whether within-person change in a candidate mechanism relates to within-person change in outcomes.

The current study aimed to examine the intraindividual trajectories of anxiety sensitivity and alcohol use severity from baseline to one-year follow-up after engaging in smoking cessation treatment using parallel process latent growth curve modelling. It was hypothesized that (1) anxiety sensitivity would significantly decline from baseline to one-year follow-up; (2) non-targeted alcohol use severity would significantly decline from baseline to one-year follow-up; and (3) there would be a positive association between trajectories of change in anxiety sensitivity and alcohol use severity. The association between one-year trajectories of change were expected to remain evident after accounting for theoretically-relevant covariates (treatment condition, participant sex, and baseline level of cigarette dependence). Additionally, we hypothesized that more robust changes in anxiety sensitivity would be evident earlier in the measurement period and that the greatest amount of change in alcohol use severity would occur after expected changes in anxiety sensitivity.

1. Method

1.1. Participants

The sample consisted of 386 individuals who participated in a large multi-site randomized control trial examining two smoking cessation interventions (for full protocol, see Schmidt et al., 2016). All individuals were adult daily smokers (at least 8 cigarettes per day) seeking treatment for smoking (49.9% male; $M_{age} = 38.6$ years, $SD_{age} = 13.7$). Regarding ethnicity, 83.2% of participants identified as White, 9.8% as Black non-Hispanic, 3.1% as Hispanic, 0.8% as Black Hispanic, 0.8% as Asian, and 1.8% as “mixed race/other”; 0.6% did not disclose race/ethnicity. In terms of sexual orientation, 80.6% of participants identified as heterosexual, 3.4% as gay or lesbian, 2.1% as bisexual, and 1.3% as “other”; 12.7% did not disclose sexual orientation.

1.2. Procedure

Individuals responded to study advertisements and were invited to an in-person baseline assessment to determine eligibility for the trial. Eligible individuals were 18 + years of age, regular daily cigarette users (8 + per day for 1 + years, consistent with past smoking cessation trials; Becoña et al., 2017; Langdon, Farris, Øverup, & Zvolensky, 2016), and reported a motivation to quit smoking (5 + or greater on a 10-point scale). Those with psychotic spectrum disorders, uncontrolled bipolar disorder, serious suicidal intent (i.e., immediate attention required) or undergoing other smoking cessation programs were excluded. All participants provided informed written consent and were randomized to one of two smoking cessation treatments (Schmidt et al., 2016). Both treatments were CBT-based, although one arm had specific focus on anxiety sensitivity. Each intervention consisted of four sessions. Follow-up assessments were gathered for one year. The study was approved by the Institutional Review Board at each study site. The current study is based on secondary analysis including all individuals who were randomized to one of the two interventions. Data from baseline as well as follow-ups (1-, 3-, 6-, and 12-months post-intervention) were used for the current study.

1.3. Measures

Smoking History Questionnaire (SHQ; Brown, Lejuez, Kahler, & Strong, 2002). The SHQ is a self-report measure that assesses smoking history and current use patterns (e.g., age of first cigarette, cigarettes smoked per day, years of daily smoking). The SHQ was administered at baseline.

Alcohol History Measure (AHM). The AHM is a self-report questionnaire that assesses participants' past alcohol use (e.g., days drinking per week and average drinks consumed). The AHM was administered at baseline.

Fagerström Test of Cigarette Dependence (FTCD; Fagerström, 1978; Korte, Capron, Zvolensky, & Schmidt, 2013). The FTCD is a 6-item measure of "cigarette dependence" (Fagerström, 2012; Heatherton, Kozlowski, Frecker, and Fagerström, 1991). The FTCD has shown positive associations with basic smoking variables (e.g., saliva cotinine), and has demonstrated high test-retest reliability (Heatherton et al., 1991). In the current sample, the FTCD total score internal consistency was low ($\alpha = 0.59$), which is characteristic for this measure (Korte et al., 2013). The FTCD was administered at baseline and was used as a covariate in the current study.

Anxiety Sensitivity Index-3 (ASI-3; S. Taylor et al., 2007). The ASI-3 is a reliable and valid self-report measure of anxiety sensitivity (Taylor et al., 2007). It is comprised of 18 items which are rated on a Likert scale from 0 ("*very little*") to 4 ("*very much*"). There is a total score as well as three sub-scales: (ASI-3-Phy; e.g., "It scares me when my heart beats rapidly"), cognitive (ASI-3-Cog; e.g., "It scares me when I am unable to keep my mind on a task"), and social (ASI-3-Soc; e.g., "I worry that other people will notice my anxiety") concerns (S. Taylor et al., 2007). The ASI-3 was administered at baseline as well as 1- 3- 6- and 12-month follow-up. In the current sample, internal consistency was excellent for the ASI-3 total across all time points ($\alpha = 0.91$-0.93). The current study used the three sub-scales as indicators of a latent anxiety sensitivity variable: ASI-3 physical ($\alpha = 0.85$-0.88), ASI-3 cognitive ($\alpha = 0.88$-0.93), and ASI-3 social ($\alpha = 0.83$-0.88).

The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993). The AUDIT is a reliable and valid self-report measure of alcohol consumption and problems (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001; Saunders et al., 1993). It consists of 10 questions which are rated on various scales from 0 (e.g., "*never*") to 4 (e.g., "*4 or more times a week*"). There is a total score as well as three subscales: frequency/quantity of alcohol use (AUDIT-F/Qu; three items, e.g., "How often do you have a drink containing alcohol?"), symptoms of dependence (AUDIT-Dep; three items, e.g., "How often during the last year have you failed to do what was normally expected from you because of drinking?"), and alcohol-related consequences (AUDIT-Con; four items, e.g., "Have you or someone else been injured as a result of your drinking?"). The AUDIT was administered at baseline as well as 1- 3- 6- and 12-month follow-up. In the current sample, internal consistency was good for the AUDIT total across all time points ($\alpha = 0.83$-0.88). This study used the three subscales as indicators of a latent alcohol use severity variable: AUDIT-F/Qu ($\alpha = 0.80$-0.84), AUDIT-Dep ($\alpha = 0.52$-0.64), and AUDIT-Con ($\alpha = 0.63$-0.78). Reliability estimates were consistent with past work on the AUDIT three-factor structure (Doyle, Donovan, & Kivlahan, 2007). Additionally, total scores were used to characterize the sample in terms of hazardous drinking, defined as pattern of drinking putting one at risk for alcohol-related problems (World Health Organization, 2015). Total scores of 8 (7 for women) or higher indicate hazardous drinking (Babor et al., 2001; Saunders et al., 1993).

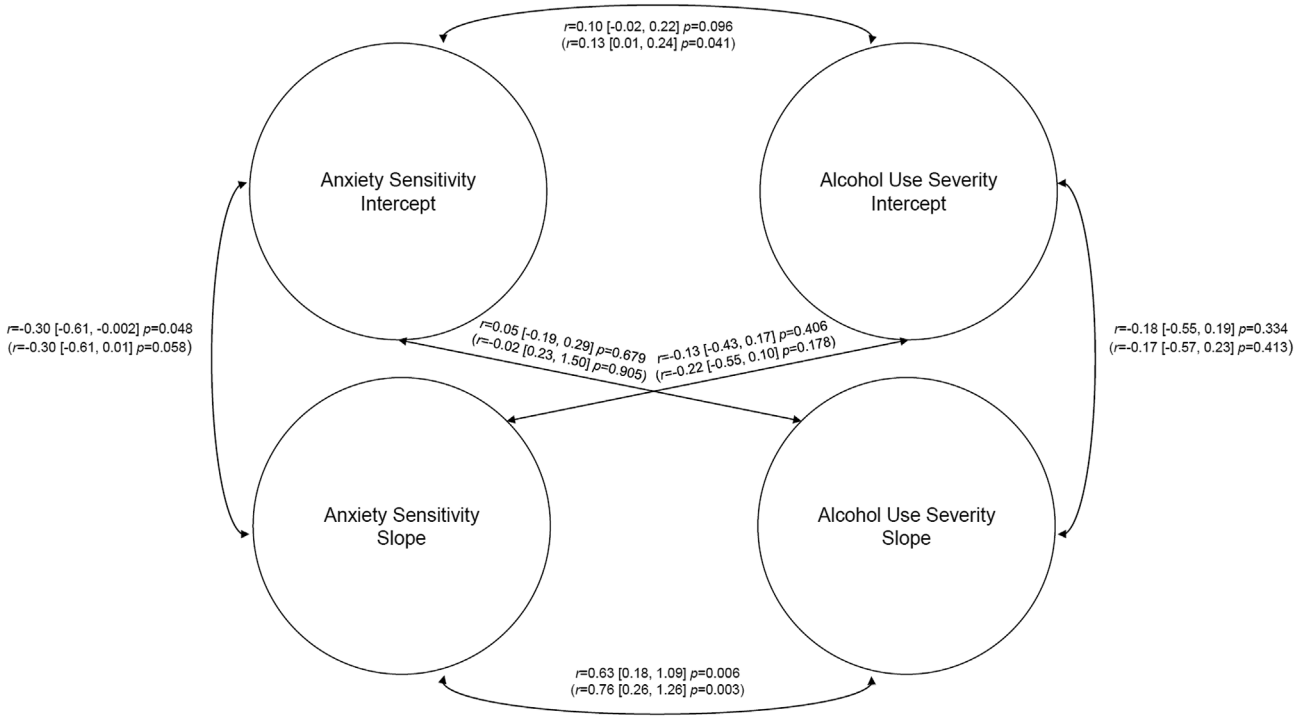
1.4. Data analytic strategy

Data analyses were conducted in Mplus version 7.4 (Muthén & Muthén, 2015) using maximum likelihood estimation with robust standard errors (i.e., MLR estimation). Maximum likelihood accounts for missing data in a manner as good as, if not better than, multiple imputation (Allison, 2003). First, longitudinal measurement invariance models for the latent constructs of anxiety sensitivity and alcohol use severity were examined using confirmatory factor analysis (CFA). In longitudinal analyses, this is an important initial step to assure that measurements are factorially invariant over time and that the constructs demonstrate a reasonable degree of stability across the measurement period; this allows for the interpretation of differences observed over time in a given measure to be attributed to the construct rather than due to changes in measurement or bias (Little, 2013). Model fit was assessed using root mean square error of approximation (RMSEA), Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and Standardized Root Mean Square Residual (SRMR). RMSEA values of 0.06 or lower, CFI and TLI values of 0.95 or greater, and SRMR values of 0.08 or less indicate good model fit (Hu & Bentler, 1999). To assess invariance, a series of three nested models adding constraints were estimated (configural invariance, weak factorial invariance, and strong factorial invariance, respectively) in accordance with procedures outlined by Little (2013). Specifically, to asses configural invariance the latent structure of each construct (anxiety sensitivity and alcohol use severity) was estimated with observed indicators (i.e. subscales of the ASI-3 and AUDIT) loading freely onto the respective construct at each time point (i.e., baseline and 1-, 3-, 6-, and 12-month). Following configural invariance, 'weak' factorial invariance was estimated by constraining the loading of each indicator to be equal over time and comparing model fit to the configural invariance model. Finally, 'strong' factorial invariance was estimated by further restricting the model to constrain intercepts to be equivalent over time in addition to factor loadings and comparing model fit to the weak factorial invariance model. Adjusted chi-square difference tests with Satorra-Bentler scaling correction were used to evaluate the change from configural to weak and weak to strong models for both anxiety sensitivity and alcohol use severity, respectively, with alpha levels corrected for the four tests (i.e., $p = 0.0125$).

To estimate intraindividual changes in anxiety sensitivity and alcohol use severity, a series of linear latent growth curve models were estimated. First, the trajectories of anxiety sensitivity and alcohol use severity from baseline to 12-month follow-up were examined in separate models.[1] Next, latent growth curve models were estimated to examine whether intraindividual change in anxiety sensitivity relates to intraindividual change in alcohol use severity.[2] A parallel process latent growth curve model was used to examine the association between change in anxiety sensitivity and change in alcohol use severity from baseline to 12-month follow-up. Intercepts and slopes were specified to correlate with one another (see Fig. 1). The parallel process model was re-run, adjusting for theoretically-relevant and statistically justified

[1] Multiple trajectories (linear, quadratic, cubic, quartic, and shape factors) were examined for each construct; linear trajectories were the only models to converge *and* demonstrate adequate model fit and were retained for all further models.

[2] Given the longitudinal design, we attempted to model whether intraindividual change in anxiety sensitivity occurred prior to intraindividual change in alcohol use severity. First, a piece-wise latent growth curve model was estimated with an intercept and two slopes (pre-treatment to 3-month follow-up; 3-month follow-up to 12-month follow-up) for each construct (anxiety sensitivity and alcohol use severity). The slopes were estimated with concurrent and cross-lagged associations to see if earlier change in anxiety sensitivity predicted later change in alcohol use severity (or vice versa). This model did not converge. Next, parallel process models estimating the slope of anxiety sensitivity from baseline to the first follow-up as a predictor of the slope of alcohol use severity over the course of the follow-ups was estimated but did not converge. Thus, the current report focuses on parallel process models, which were conducted after the previous models failed to converge. These models estimated with one slope (pre-treatment to 12-month follow-up) for each construct.



Notes: Correlations presented for parallel process models examining intercepts and slopes for anxiety sensitivity and alcohol use severity in the unadjusted (values on top) as well as covariate-adjusted (values in brackets on bottom) models. 95% confidence intervals presented in square brackets. All arrows indicate correlations.

Fig. 1. Correlations between Intercepts and Slopes in Parallel Process Models. Notes: Correlations presented for parallel process models examining intercepts and slopes for anxiety sensitivity and alcohol use severity in the unadjusted (values on top) as well as covariate-adjusted (values in brackets on bottom) models. 95% confidence intervals presented in square brackets. All arrows indicate correlations.

covariates (treatment condition, participant sex, and baseline level of cigarette dependence), which were added as predictors of intercepts and slopes. Finally, Cohen's d values were calculated to examine the effect size for changes in mean levels of anxiety sensitivity (ASI-3) and alcohol use severity (AUDIT) from one time point to the next in order to estimate at what point(s) each construct appeared to demonstrate change.

2. Results

Descriptive Statistics. On average, participants reported smoking 16.5 cigarettes per day (SD = 9.0) and had been smoking regularly for an average of 20.2 years (SD = 13.6). A majority (65.9%) of participants identified themselves as “regular” drinkers. Overall, participants reported drinking an average of 1.93 days per week (SD = 2.07) and consuming 3.53 drinks per occasion (SD = 3.04). Of those with available data, approximately one third of the sample (32.6%) surpassed the cut-off for hazardous drinking, per the AUDIT, at baseline with one quarter (25.4%) surpassing the cut-off at one-year follow-up. Regarding anxiety sensitivity, 22.1% had “high” anxiety (i.e., ASI-3 total scores of 23 or greater; Allan et al., 2014) at baseline compared to 12.6% at one year follow-up.

Measurement Invariance. The configural model for anxiety sensitivity provided a good fit to the data: [χ^2 (50) = 79.90, p = 0.005, RMSEA = 0.04 90% CI [0.02, 0.06], CFI = 0.98, TLI = 0.96, SRMR = 0.04]. The test of weak invariance resulted in a well-fitting model: [χ^2 (58) = 85.08, p = 0.011, RMSEA = 0.04 90% CI [0.02, 0.05], CFI = 0.98, TLI = 0.97, SRMR = 0.06]. The Satorra-Bentler scaled chi-square difference test between configural and weak invariance was not statistically significant: [$\Delta\chi^2$ (8) = 6.71, p = 0.568], indicating that the weak invariance model was not a worse fit than the configural model. The test of strong invariance resulted in a well-fitting model: [χ^2 (66) = 102.23, p = 0.003, RMSEA = 0.04 90% CI [0.02, 0.05], CFI = 0.98, TLI = 0.96, SRMR = 0.06]. The Satorra-Bentler scaled chi-square difference test from weak to strong invariance was not statistically significant with the adjusted alpha: [$\Delta\chi^2$ (8) = 18.21, p = 0.020], indicating that the strong invariance model was not a worse fit than the weak invariance model.

The configural model for alcohol use severity provided a good fit to the data: [χ^2 (50) = 100.62, p < 0.001, RMSEA = 0.05 90% CI [0.04, 0.07], CFI = 0.96, TLI = 0.91, SRMR = 0.06]. The test of weak invariance resulted in a well-fitting model: [χ^2 (58) = 95.96, p = 0.001, RMSEA = 0.04 90% CI [0.03, 0.06], CFI = 0.97, TLI = 0.94, SRMR = 0.06]. The adjusted chi-square difference test between configural and weak invariance was not statistically significant: [$\Delta\chi^2$ (8) = 4.90, p = 0.768]. The test of strong invariance resulted in a well-fitting model: [χ^2 (66) = 111.13, p < 0.001, RMSEA = 0.04 90% CI [0.03, 0.06], CFI = 0.96, TLI = 0.94, SRMR = 0.06]. The adjusted chi-square difference test from weak to strong invariance not statistically significant with the adjusted alpha: [$\Delta\chi^2$ (8) = 15.88, p = 0.044].

Anxiety Sensitivity Reduction. Estimating the change in anxiety sensitivity resulted in an adequate fit to the data: [χ^2 (10) = 24.52, p = 0.006, RMSEA = 0.06 90% CI [0.03, 0.09], CFI = 0.94, TLI = 0.94, SRMR = 0.08]. The slope of anxiety sensitivity demonstrated statistically significant decline (M = −0.87 95% CI [-1.19, −0.54], p < 0.001). There was statistically significant variance in intercepts (s^2 = 82.51, p < 0.001) but not slopes (s^2 = 1.19, p = 0.179), suggesting that individuals varied in terms of starting anxiety sensitivity but declined to a similar degree. The correlation between anxiety sensitivity intercept and slope was not statistically significant, although it was moderate in size (r = −0.31, 95% CI [-0.63, 0.01] p = 0.056).

Alcohol Use Severity Reduction. Estimating the change in alcohol use severity resulted in an adequate fit to the data: [χ^2 (10) = 21.66, p = 0.017, RMSEA = 0.06 90% CI [0.02, 0.09], CFI = 0.94, TLI = 0.94, SRMR = 0.06]. The slope of alcohol use severity demonstrated statistically significant decline (M = −0.22 95% CI [-0.38, −0.06], p = 0.006). There was statistically significant variance in intercepts (s^2 = 28.01, p < 0.001) but not slopes (s^2 = 0.47, p = 0.159); these data indicate varied levels of baseline alcohol use severity but similar declines across individuals. The correlation between alcohol use severity intercept and slope was not statistically significant (r = −0.31, 95% CI [-0.55, 0.28] p = 0.526), although it was moderate in size.

Parallel Process Model. The parallel process model of change in anxiety sensitivity relating to change in alcohol use severity resulted in a well-fitting model: [χ^2 (35) = 60.49, p = 0.005, RMSEA = 0.04 90% CI [0.02, 0.06], CFI = 0.96, TLI = 0.95, SRMR = 0.07]. The slope of anxiety sensitivity was statistically significantly associated with the slope of alcohol use severity and large in magnitude (r = 0.63, 95% CI [0.18, 1.09], p = 0.006; Fig. 1). The intercept of anxiety sensitivity demonstrated a moderate and statistically significantly negative correlation with slope of anxiety sensitivity (r = −0.30, 95% CI [-0.61, −0.002], p = 0.048) as well as small correlations with intercept of alcohol use severity (r = 0.10, 95% CI [-0.02, 0.22], p = 0.096) and slope of alcohol use severity (r = 0.05, 95% CI [-0.19, 0.29], p = 0.679), which were not statistically significant. The intercept of alcohol use severity was weakly associated with the slope of anxiety sensitivity (r = −0.13, 95% CI [-0.43, 0.17], p = 0.406) and slope of alcohol use severity (r = −0.18, 95% CI [-0.55, 0.19], p = 0.334), though these relations were not statistically significant.

Covariate-Adjusted Parallel Process Model. Treatment condition, sex, and FTCD were added to the model as predictors of intercept and slope parameters for both constructs. This resulted in an adequate fit to the data: [χ^2 (55) = 91.52, p = 0.002, RMSEA = 0.04 90% CI [0.03, 0.06], CFI = 0.96, TLI = 0.94, SRMR = 0.06]. The slope of anxiety sensitivity was statistically significantly associated with the slope of alcohol use severity and large in magnitude (r = 0.76, 95% CI [0.26, 1.26], p = 0.003). For all other correlations between intercepts and slopes in the covariate-adjusted model, see Fig. 1.

Treatment condition was a significant predictor of alcohol use severity intercept (β = 0.12, 95% CI [0.01, 0.22], p = 0.028) but not slope (β = 0.12, 95% CI [-0.06, 0.30], p = 0.186) or the intercept (β = 0.10, 95% CI [-0.03, 0.22], p = 0.119) or slope (β = −0.02, 95% CI [-0.24, 0.20], p = 0.860) of anxiety sensitivity. Males had statistically significantly greater alcohol use severity intercepts (β = 0.19, 95% CI [0.08, 0.30], p = 0.001) but sex did not predict intercept of anxiety sensitivity (β = −0.04, 95% CI [-0.16, 0.08], p = 0.508) or slope for alcohol use severity (β = −0.03, 95% CI [-0.22, 0.17], p = 0.804) or anxiety sensitivity (β = 0.11, 95% CI [-0.15, 0.37], p = 0.412). Baseline levels of cigarette dependence did not predict the intercept of anxiety sensitivity (β = 0.82, 95% CI [-0.03, 0.20], p = 0.158) but had a trending moderate association with slope of anxiety sensitivity (β = −0.26, 95% CI [-0.53, 0.003], p = 0.052); it was a significant predictor of intercept (β = −0.20, 95% CI [-0.31, −0.09], p < 0.001) but not slope (β = 0.19, 95% CI [-0.06, 0.44], p = 0.140) of alcohol use severity.

Effect Size. Scores of anxiety sensitivity demonstrated the largest change from baseline (M = 14.39; Table 1) to 1-month follow-up (M = 10.61; Cohen's d = −0.34, 95% CI [-0.20, −0.48]; Fig. 2), with additional small reductions thereafter. Regarding alcohol use severity, scores demonstrated some weak variation between baseline (M = 5.91) and 6-month follow-up (M = 5.95) with the largest reduction from 6-month follow-up to 12-month follow-up (M = 4.84; Cohen's d = −0.19, 95% CI [-0.05, −0.33]).

Table 1
Univariate means across time points.

		BL	1M	3M	6M	12M
ASI-3	*M*	14.39	10.61	10.71	9.73	9.30
	SD	11.92	10.15	10.62	9.05	9.03
	n	385	206	156	135	127
AUDIT	*M*	5.91	6.41	6.14	5.95	4.84
	SD	5.70	6.48	6.12	6.23	5.37
	n	342	202	147	130	122

Notes: BL = Baseline; 1M = 1 month follow-up; 3M = 3 month follow-up; 6M = 6 month follow-up; 12M = 12 month follow-up; ASI-3 = Anxiety Sensitivity Index-3 Total Score; AUDIT = Alcohol Use Disorders Identification Test Total Score. Cohen's d represents change between adjacent time points (e.g., BL to 1M). All follow-ups occurred post-treatment.

3. Discussion

The current study examined how the intraindividual trajectory of change in anxiety sensitivity relates to the intraindividual trajectory of change in alcohol use severity in a secondary analysis of a large RCT for smoking cessation. These findings support the hypothesis that changes in anxiety sensitivity correlate with changes in non-targeted alcohol use severity from baseline to one-year follow-up. The current findings replicate separate lines of past work documenting reduction of anxiety sensitivity (Schmidt et al., 2016; Zvolensky et al., 2018) and reductions in non-targeted alcohol use severity (Correa-Fernández et al., 2017; Sobell et al., 2017) among treatment-seeking smokers. Descriptively, there was approximately 10% reduction in prevalence of hazardous drinking and “high” anxiety sensitivity at one year follow-up relative to baseline. Furthermore, these current findings extend past lines of work by demonstrating that changes in anxiety sensitivity and alcohol use severity over time are positively, and strongly, related. The correlation in slopes was large in magnitude (r = .63) and remained statistically significant and large (r = 0.76) after accounting for effects of treatment condition, sex, and cigarette dependence.

Although correlational, examination of mean changes over time using Cohen's d suggest that changes in anxiety sensitivity occurred earlier (Fig. 2) whereas changes in alcohol use occurred primarily later, from 6- to 12-months post-treatment; indeed, for alcohol use severity, scores were nearly identical at baseline and 6-month follow-up. Yet, there was meaningful change in alcohol use thereafter. These data are in line with the perspective that anxiety sensitivity may be a mechanism by which alcohol use changes among smokers (i.e., that treatment impacts anxiety sensitivity and change in anxiety sensitivity results in reduction of alcohol use). However, the current results cannot definitively identify anxiety sensitivity as a mechanism per se. Yet, these findings coincide with previous work from the parent study documenting relations between change in anxiety sensitivity and smoking abstinence (Zvolensky et al., 2018).

Several other findings are noteworthy and relevant to the overall context of the observed effects for the primary analyses. Both anxiety sensitivity and alcohol use severity demonstrated longitudinal measurement invariance over the five measurement periods. Specifically, factor loadings and intercepts were consistent from baseline through one-year follow-up. Thus, reductions in scores observed over time can be attributed to change in the constructs (anxiety sensitivity and alcohol use severity) rather than changes in measurement-related factors (e.g., error, instability), adding confidence to the findings regarding trajectories of change. However, it should be noted that alcohol use severity was modeled as the outcome. Given the lack of detailed information on consumption (e.g., drinks consumed over the past week) it was not possible to separately model consumption and consequences/problems. Future work may seek to do so given evidence that they may represent unique aspects of drinking (Doyle et al., 2007) with anxiety sensitivity being more strongly related to consequences/problems than consumption among smokers (Paulus et al., 2017). Importantly, the AUDIT total score, which is commonly used, also sum items across consumption and problems/consequences to measure a unitary

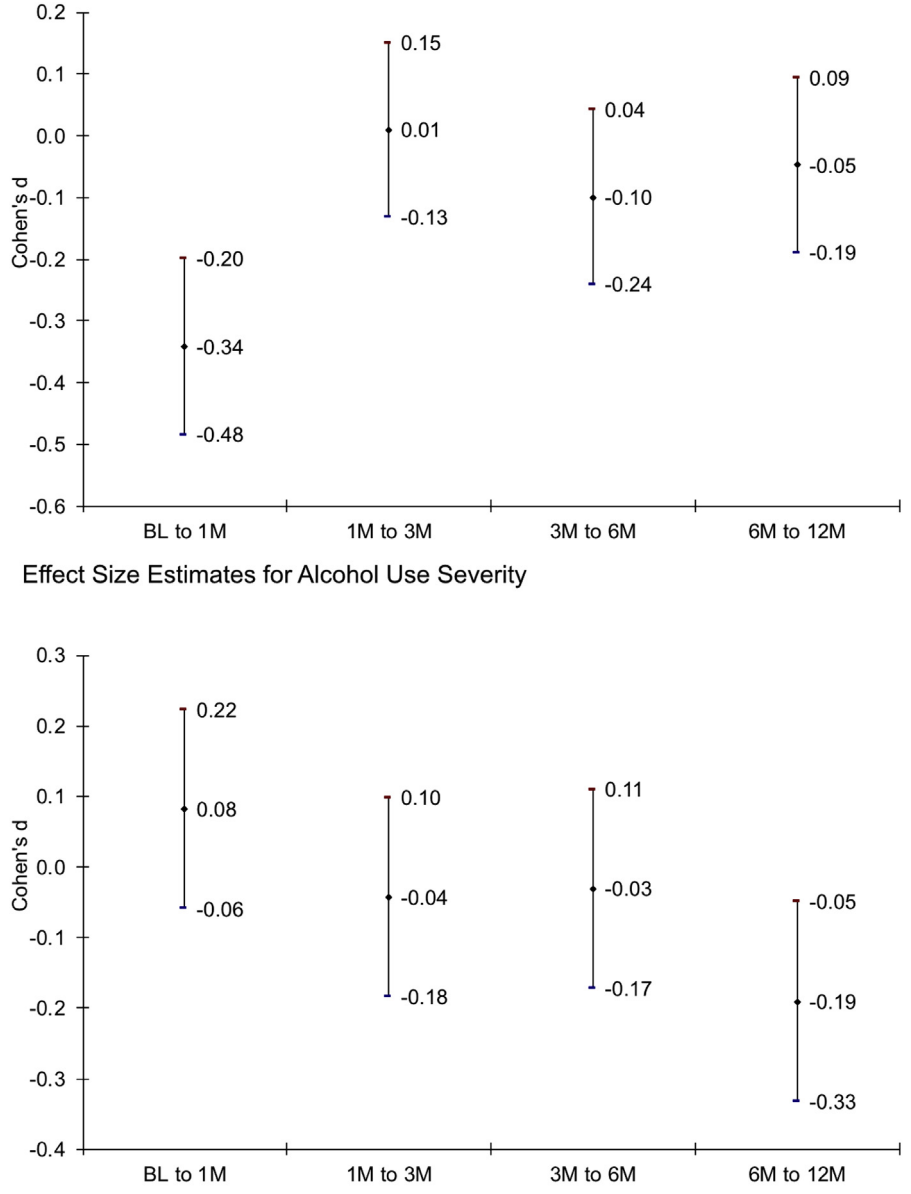


Fig. 2. Cohen's d estimates for adjacent study timepoints.
Notes: Cohen's d represents change between adjacent time points (e.g., BL to 1M). All follow-ups occurred post-treatment.

dimensional construct.

Second, greater anxiety sensitivity intercept (i.e., baseline level) was related to lower slope of anxiety sensitivity, suggesting that those with greater starting anxiety sensitivity had lower rates of change in anxiety sensitivity. Although this correlation was medium-sized, there was a wide confidence interval and in the adjusted model the association (which had the same parameter estimate) it was not statistically significant (Fig. 1). Given that both confidence bands were relatively wide, further work is needed to provide a more detailed evaluation of the associations, and to examine potential moderators. Unexpectedly, the intercept of anxiety sensitivity was not statistically significantly related to the intercept of alcohol use severity. There was a small, positive, association, but a confidence interval including 0 (−0.02 to 0.22); however, in the covariate-adjusted model the association did reach statistical significance, although with a confidence band narrowly excluding 0. Additionally, the intercept of anxiety sensitivity was not associated with slope for alcohol use severity, suggesting that starting level of anxiety sensitivity did not impact rate of change in alcohol use severity. Starting level of alcohol use severity was not significantly associated with rates of change for either variable. This finding was surprising given past work documenting that more severe alcohol use/problems are associated with poorer outcomes (e.g., depression, smoking; Humfleet et al., 1999; Sullivan, Fiellin, & O'Connor, 2005).

Third, treatment condition was not a statistically significant predictor of change in anxiety sensitivity or change in alcohol use severity. The main outcome trial from this RCT documented that treatment differences in changes in anxiety sensitivity, however these were only evident during the initial treatment phase and not the follow-up phase (Schmidt et al., 2016). Given the current study utilized data-points spanning from baseline to one-year follow-up, it is not surprising that the lack of condition effect on anxiety sensitivity was not evident, consistent with these previous findings. Nevertheless, anxiety

sensitivity did reduce in both conditions, which is not surprising given that both treatments were CBT-based which tend to reduce anxiety sensitivity even if they are not designed as 'anxiety sensitivity reduction' treatments per se (Smits, Berry, Tart, & Powers, 2008). Although changes were observed from baseline to one year follow-up suggesting that change can be maintained, future work should evaluate how to bolster and maintain these changes in treatments that directly target anxiety sensitivity, such as the anxiety sensitivity-focused treatment arm. It is possible that the current study protocol of four sessions, while enough to reduce anxiety sensitivity, may need to be extended or augmented to achieve long term gains relative to other interventions (e.g., standard CBT-based smoking cessation). Additionally, these findings suggest that although alcohol use severity significantly reduced from baseline to one year follow-up, it did not differentially reduce in the standard smoking cessation condition relative to the anxiety sensitivity focused condition, consistent with past work (e.g., Sobell et al., 2017). However, this may be a by-product of the lack of treatment condition differences in anxiety sensitivity reduction. Future work should examine whether treatments that can maintain long term anxiety sensitivity changes in targeted protocols result in larger reductions in alcohol use as well. Given the lack of treatment differences in anxiety sensitivity reduction, that question remains unanswered by the current data.

Neither sex nor baseline cigarette dependence statistically significantly related to change in anxiety sensitivity or alcohol use severity. Males did have significantly greater alcohol use severity intercepts, consistent with past work (e.g., Grant et al., 2016). Thus, men started out more severe than women, but did not differ in their rates of decline overall. Baseline cigarette dependence levels were not significantly associated with intercept for anxiety sensitivity but greater cigarette dependence was surprisingly associated lower intercept of alcohol use severity, contrary to expectation that individuals with greater cigarette dependence would experience greater drinking, given past work linking alcohol consumption and nicotine (i.e., cigarette) dependence (Dierker, Selya, Rose, Hedeker, & Mermelstein, 2016).

Despite the finding that intraindividual changes in anxiety sensitivity correlate strongly with intraindividual changes in alcohol use severity, more work is needed to better understand how such findings may be eventually translated into clinical intervention. For example, treatments such as the Unified Protocol for Emotional Disorders (Barlow et al., 2017), may be good candidates to evaluate within a substance use framework given multiple empirically supported mechanisms including anxiety sensitivity, emotion regulation, and mindfulness, which have each demonstrated strong relations with both smoking and alcohol use (DeMartini & Carey, 2011; Dvorak et al., 2014; Leventhal & Zvolensky, 2015; Paulus, Langdon, Wetter, & Zvolensky, 2018).

The current findings should be interpreted with in the context of several limitations. First, the study was based on self-report and is subject to bias in reporting. For example, it is possible that score declines over time were impacted by regression to the mean or desirability characteristics; however, it is noteworthy that alcohol use severity scores were similar at baseline and 6-month follow-up. Second, the study suffered from high rates of dropout (see Table 1), which may have biased estimates of change particularly if individuals who did not complete follow-ups did not experience reduction in anxiety sensitivity and/or alcohol use severity. Approximately 33–36% of individuals who completed baseline assessments also completed the one-year follow-up. It also is possible that dropout rate impacted convergence of models.[1,2] However, the rates of dropout in this parent trial are consistent with rates previously documented (e.g., Prochaska, Delucchi, & Hall, 2004). Third, the study focused on treatment seeking smokers and the interventions did not target alcohol use. Future work will need to examine whether such findings generalize to other samples (e.g., smokers with high anxiety sensitivity and at-risk/problematic drinking) as well as what the impact would be if alcohol use was explicitly targeted.

Fourth, as alcohol use was not the focus of treatment it was not measured throughout treatment, preventing more nuanced analysis of temporal change. We originally attempted to estimate intraindividual change in these variables with temporal precedence (e.g., using piecewise latent growth curves to examine whether earlier slope of one variable predicted later slope of another)[2]. Unfortunately, these models did not converge, possibly due to added model complexity. Therefore, the study is limited to a correlational design and temporal precedence was not present despite the rich longitudinal data. Future work may seek to examine temporal precedence of change in anxiety sensitivity and alcohol use severity with cross-lagged panel models and/or autoregressive latent change models. Such work would represent novel contributions to the field; however, these models would inform interindividual standing rather than intraindividual change. Thus, is important for additional future work to also expand upon the current findings with methodologies that allow for testing of intraindividual change in anxiety sensitivity and alcohol use severity *with* conclusions regarding temporal precedence.

Fifth, the study was unable to estimate multiple trajectories of change. Linear slopes were fit in all models and the models did not converge when estimating non-linear slopes[1]. Thus, the linear estimations documented here represent an overestimate of the true correlation. This is particularly important given effect size patterns, which suggest that anxiety sensitivity and alcohol use severity may change non-linearly; Fig. 2. Future work should seek to examine shapes of change in anxiety sensitivity and alcohol use over time. For such work, session-by-session measurement is ideal in order to capture more complex shapes of change over time (Smith, Paulus, & Norton, 2017). Sixth, the current study focused on anxiety sensitivity as one well-established correlate of alcohol use severity and other measures of interest were not available for analysis. Future work may consider multirisk models and evaluate how anxiety sensitivity and other relevant variables such as emotion dysregulation relate over time or whether emotion dysregulation mediates changes in anxiety sensitivity and alcohol use severity among smokers, extending cross-sectional work in this area (e.g., Paulus et al., 2017). Seventh, this study focused on alcohol use as an outcome. Although analyses adjusted for smoking levels at baseline, smoking behaviors were not a focus of the current study. However, previous work from this trial has documented how change in anxiety sensitivity relates to changes in smoking (Zvolensky et al., 2018). Finally, this study examined the use of alcohol among adult daily smokers seeking treatment for smoking. It is unknown how the use of other substances (e.g., cannabis, opioids) may also change over the course of smoking treatment or how the co-use of these substances with alcohol and tobacco may have impacted the current findings. Future work should examine how complex relationships between multiple substances impact one another over time and what the role of anxiety sensitivity may be in their fluctuation over time.

Overall, the results of the current study provide novel evidence of idiographic changes in anxiety sensitivity as a statistically significant and large correlate of idiographic alcohol use severity reduction following smoking cessation treatment. If replicated and extended, these findings could implicate anxiety sensitivity as a mechanism underlying treatment-related changes in non-targeted alcohol use in smoking cessation interventions. Future work is needed to examine the degree to which anxiety sensitivity reduction strategies may be beneficial in treating combined smoking and alcohol use.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://

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