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Retrospective Self-Reports of Sensitivity to the Effects of Alcohol: Trait-Like Stability and Concomitant Changes With Alcohol Involvement

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Objective: Lower sensitivity to the acute effects of alcohol is known to confer risk for the development of alcohol use disorder. Alcohol sensitivity, or level of response to alcohol's subjective effects, is heritable but also can change as a result of persistent alcohol exposure (i.e., acquired tolerance). Here, we examined how changes over time in four indices of alcohol involvement affected scores on two validated, retrospective self-report measures of alcohol response—the Self-Rating of the Effects of Alcohol (SRE) form and the Alcohol Sensitivity Questionnaire (ASQ)—in a sample of emerging adult drinkers. ***Method:*** Participants ($N = 173$; $M_{age} = 19.5$ years; 60% assigned female at birth) completed the ASQ, SRE, and measures of alcohol use and problems at two time points separated by a median of 0.77 years (range: 0.30–2.54 years). ***Results:*** Multiple linear regression showed that increases in drinking over this period accounted for increases in SRE and ASQ scores (i.e., in reported numbers of drinks needed to experience subjective effects of alcohol). Increased drinking accounted for more variance in the number of drinks needed to experience lighter drinking versus heavier drinking effects, and increases in the number of drinks consumed per occasion had a larger effect than did changes in total numbers of drinks consumed, number of binge-drinking occasions, or drinking-related problems. ***Conclusions:*** Findings suggest that both SRE and ASQ capture some stable, trait-like variability in alcohol response as well as some state-dependent, within-person variability in alcohol response acquired through increases in alcohol involvement.

Public Health Significance Statement

The findings in this report provide evidence that validated questionnaire measures of individual differences in level of response to alcohol, also known as subjective response, are sensitive to changes in young adults' drinking over time. Given that a reduction in level of response to alcohol is an indicator of possible alcohol use disorder (AUD), these findings support the use of these questionnaire measures to track changes in AUD risk over time.

Keywords: alcohol sensitivity, level of response, subjective response, test–retest reliability, acquired tolerance

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Research points to relatively low sensitivity to alcohol's acute effects (i.e., low level of response) as a potent risk factor for alcohol use disorder (AUD; Newlin & Thomson, 1990; Quinn & Fromme, 2011; Schuckit, 1994). Individuals who require more drinks to experience the acute effects of alcohol (i.e., low sensitivity/low responders) are more likely to experience alcohol-related problems, including AUD, than their higher sensitivity/higher responding peers (see Morean & Corbin, 2010; Quinn & Fromme, 2011; Schuckit, 2022, for review). Risk associated with the low-sensitivity phenotype is distinct from vulnerability attributable to other known risks, such as alcohol expectancies, externalizing behaviors, and comorbidity with other psychiatric diagnoses (Schuckit, 2022; Schuckit, Smith, Anderson, & Brown, 2004; Trim et al., 2009).

The gold standard for measuring alcohol response is alcohol administration in the laboratory (Ehlers et al., 1989; Paulus et al., 2012; Schuckit et al., 1987; Schuckit & Gold, 1988). However, this method is burdensome, costly, unethical for certain populations (e.g., underage drinkers), and unsuitable for large-scale epidemiological studies (Wood & Sher, 2000). Responding to these concerns, Schuckit and colleagues (Schuckit, Smith, & Tipp, 1997) developed the Self-Rating of the Effects of Alcohol (SRE) form, a 12-item, retrospective questionnaire asking respondents to record the number of standard drinks required to feel each of four subjective effects from drinking alcohol during each of three periods of their lives (the first five times alcohol was consumed [*SRE-5*], the most recent 3-month period during which they drank [*SRE-3*], and the heaviest drinking 3-month period in their lives [*SRE-H*]). The SRE has good test–retest reliability (Ray et al., 2011; Schuckit, Smith, & Tipp, 1997) and excellent internal consistency (Schuckit, Smith, & Tipp, 1997) and has evidenced good construct validity via strong correlations with subjective effects during laboratory alcohol challenge (e.g., Fleming et al., 2016; Schuckit et al., 2009; Schuckit, Smith, & Tipp, 1997), levels of alcohol consumption (e.g., Kalu et al., 2012; Schuckit et al., 2001, 2005, 2008, 2009; Schuckit & Smith, 2004, 2013), and alcohol-related problems (e.g., Corbin et al., 2013; Gonçalves et al., 2017; Ray et al., 2011; Schuckit et al., 2001, 2008; Schuckit & Smith, 2013; Schuckit, Smith, Goncalves, & Anthenelli, 2016).

Despite the SRE's demonstrated utility, researchers have raised some concerns about its scope (Fleming et al., 2016). The subjective experiences queried by the SRE consist mostly of effects of alcohol typically experienced when relatively large doses are consumed (feeling dizzy/slurring speech, stumbling, passing out). Thus, the SRE's ability to query effects associated with smaller doses is scant, potentially limiting the population for whom SRE scores are valid (i.e., only heavy drinkers). The one effect queried by the SRE that could result from smaller doses—feel any different—is vague and open to a variety of interpretations, thereby limiting its precision (Clark & Watson, 2016). Finally, one of the SRE's items—"feel a bit dizzy, or begin to slur your speech"—conflates two qualitatively distinct experiences, likely contributing error to item responses (Bolt & Liao, 2022).

The Alcohol Sensitivity Questionnaire (ASQ; Fleming et al., 2016; O'Neill et al., 2002) was created with these issues with the SRE in mind. The ASQ queries a wide array of effects typically experienced from both heavy (*ASQ-H*; e.g., passing out; vomiting; hangover) and light (*ASQ-L*; e.g., feeling buzzed; feeling flirtatious; becoming more talkative) alcohol exposures. Like the SRE, higher ASQ scores indicate a blunted response to the subjective effects of alcohol. ASQ scores have excellent internal consistency and have shown good construct/criterion validity via strong correlations with subjective responses to acute alcohol exposure and with SRE scores (Fleming et al., 2016), alcohol consumption levels (Bartholow et al., 2003, 2007, 2010), estimated blood alcohol concentration during real-world drinking episodes (Kohen et al., 2023), and alcohol-related consequences (Bartholow et al., 2010; Davis et al., 2021; Fleming & Bartholow, 2014; Hone et al., 2017).

SRE and ASQ scores are complex in that they are believed to reflect both an inherited "innate" sensitivity to alcohol and an aspect of tolerance acquired from drinking experience (see Fleming et al., 2016; Schuckit, 2018). This has several implications for interpretation of scores, especially changes in scores over time. In theory, the proportion of variance in SRE/ASQ scores attributable to heritable between-person constitutional differences should remain stable over time, regardless of exposure history, whereas the proportion attributable to acquired tolerance will vary over time according to changes in alcohol exposure (see Kalant, 1996). When the latter proportion is large, test–retest reliability of the SRE and ASQ will decrease. Research has shown a modest decay in the SRE's reliability over time, with 1-year test–retest correlations of .72–.82 and a 5-year κ value of .66 (Schuckit, Smith, Anderson, & Brown, 2004; Schuckit, Smith, & Kalmijn, 2004; Schuckit, Tipp, et al., 1997). However, whether this decay in stability is attributable to changes over time in level of response (e.g., due to changes in drinking) has not been examined.

More generally, no prior research has directly examined the extent to which changes over time in SRE or ASQ scores reflect concomitant changes in alcohol involvement. Work by Corbin et al. (2013; Morean & Corbin, 2008) suggests acquired tolerance to alcohol's effects, as indicated by the difference between initial/early level of response (SRE-5 scores) and recent level of response (SRE-3 scores), accounts for unique variance in recent drinking, beyond that associated with initial/early response alone. This finding suggests changes in drinking should lead to changes in subjective responses as tolerance develops. Addressing this question requires a prospective design in which changes over time in subjective response profiles can be compared against changes in alcohol involvement. This was the primary purpose of the present study.

An additional purpose of this study was to examine whether changes in alcohol use are differentially associated with changes in subjective effects typically associated with lighter versus heavier drinking. Most of the lighter drinking effects assessed by the ASQ can be considered pleasant/appetitive (e.g., feeling more flirtatious or socially at ease). The desire to experience such effects could motivate increased drinking over time, which, ironically, could induce tolerance to those effects, thereby increasing the number of drinks needed to experience them. In contrast, many of the effects associated with heavier drinking are more aversive (e.g., feeling nauseous; having a hangover). Presumably, experiencing such effects is less desirable, and therefore less likely to motivate increased drinking. Still, it remains possible that increased drinking motivated by other factors could nevertheless induce tolerance to heavier drinking effects, which could have implications for the experience of aversive consequences in other domains (e.g., interpersonal problems; role fulfillment). Thus, we compared the extent to which changes in drinking were associated with changes in numbers of drinks required to experience effects of alcohol arising from lighter versus heavier acute exposures.

Theoretically, some drinking phenotypes should more strongly influence changes in levels of response to alcohol than other

phenotypes. Increases in heavy episodic use should be especially likely to result in tolerance-related changes in subjective response. Acquisition of tolerance is believed to reflect neuroadaptations resulting from repeated cycles of intoxication and withdrawal (see Elvig et al., 2021), cycles that are more likely to result from heavy episodic exposure than from consistent low-to-moderate exposure. Thus, we compared the extent to which changes in four qualitatively distinct indices of alcohol involvement in the past 30 days—total drinks, average number of drinks consumed per occasion, number of heavy drinking days, and scores on the Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993)—were associated with changes over time in ASQ and SRE scores. Examining this question in young, nonclinical drinkers is advantageous in that such individuals are at a relatively early stage of their drinking careers when drinking is normatively increasing (Arnett, 2005; Patrick et al., 2019), and, hence, drinking-related changes in alcohol response are likely to emerge.

We anticipated that, in general, increases across time in alcohol involvement would be associated with increases in ASQ and SRE scores (i.e., reduced level of response). We also anticipated that increases in indicators of heavy drinking would be more strongly associated with changes in level of response than would increases in total volume of alcohol consumed, and that this effect would be more apparent in ASQ-L scores (sensitivity to lighter drinking/lower exposure effects) than in ASQ-H or SRE scores (sensitivity to heavier drinking/larger exposure effects). Finally, based on the idea that level of response during initial drinking experiences (i.e., SRE-5) should be unaffected by subsequent changes in drinking, we expected no appreciable change in SRE-5 scores over repeated assessments and no association between changes in drinking-related variables and changes in SRE-5 scores.

Method

Participants

Participants contributing data for this report were part of a sample of 318 healthy emerging adults (ages 18–20 at T1; 55% assigned female at birth; 87% White/Caucasian) enrolled in a large, multiwave investigation aimed at characterizing associations among measures of alcohol sensitivity, alcohol cue reactivity, and drinking-related behaviors and sequelae. Study candidates (University of Missouri undergraduates and community-dwelling age peers) completed a screening survey to determine eligibility. Candidates were sent an invitation to participate if they (a) were between the ages of 18 and 20 years old, (b) reported drinking alcohol at least once a month in the past year, with at least one binge-drinking episode (4+/5+ drinks within 2 hr for those assigned female/male at birth) in the past 6 months, (c) had normal or corrected-to-normal vision, and (d) could read and write English. Ineligibility criteria included (a) a previously unsuccessful attempt to reduce alcohol use, (b) a diagnosed neurological disease, or (c) head injury resulting in loss of consciousness for >2 min, as the parent study included electroencephalography data collection. The University of Missouri Institutional Review Board reviewed and approved all procedures used in this study. Variable selection and analyses were planned prior to data collection as part of the grant application (R01AA025451) that funded the study.

For each participant, alcohol sensitivity was measured at up to three occasions: (a) at an eligibility screening session (Time 0; T0), (b) at an initial laboratory visit (Time 1; T1), and (c) at a follow-up laboratory session (Time 2; T2) administered ∼9 months after T1. Preliminary analyses evaluated test–retest reliability of sensitivity measures for two intervals (T0–T1 and T1–T2). The median time between assessments was 45 days (range: 3–179 days) for T0–T1 and 0.77 years (range = 0.30–2.54 years) for T1–T2. As these analyses are part of an ongoing study, some participants did not have data from the follow-up T2 laboratory session. Participants were excluded from T0 to T1 analysis if the interval between these assessments was ≥6 months (n = 29). An additional participant (n = 1) was removed after identification as an extreme and consistent outlier. Sociodemographic information for the sample is reported in Table 1. Additional findings from this study have been reported elsewhere (Cofresí, Kohen, et al., 2022; Cofresí, Piasecki, & Bartholow, 2022; Cofresí, Piasecki, Hajcak, & Bartholow, 2022; Kohen et al., 2023; Waddell et al., 2023).

Measures

Alcohol Sensitivity/Level of Response

Participants completed the SRE (Schuckit, Smith, & Tipp, 1997) and the ASQ (O'Neill et al., 2002) on three occasions (eligibility screening,[1] T1, and T2). The SRE asks the respondent to indicate whether they have ever experienced each of the four effects from drinking alcohol (begin to feel different, feel a bit dizzy or begin to slur your speech, begin stumbling or walking in an uncoordinated manner, and pass out/fall asleep when you did not want to), and for each endorsed effect, to indicate the number drinks required to experience it. These four effects are queried with regard to three time periods: the first five drinking episodes in the respondent's lifetime (SRE-5), the most recent 3-month period in which they drank at least once a month (SRE-3), and the heaviest drinking 3-month period in the respondent's lifetime (SRE-H). Accordingly, higher SRE scores on any item subset—or on all items together (SRE-total)—indicate lower sensitivity/lower level of response to the subjective effects of alcohol (SRE-total α = .91–.94; SRE-5 α = .75–.84; SRE-3 α = .82–.90; SRE-H α = .87–.92).

Typically, the SRE is scored as an average of the number of drinks reported across all endorsed effects. This approach is problematic, in that item endorsement is not random across individuals. Rather, some items (e.g., passing out) will be endorsed only by heavier drinkers, whereas other items (e.g., feel different) are likely to be endorsed by both heavy and light drinkers. Prior research has documented a strong correlation between an item's mean (i.e., the average number of drinks required to experience an effect) and the item's likelihood of being endorsed (Lee et al., 2015). This, in turn, produces downwardly biased SRE scores for respondents who endorse fewer items. To avoid this problem, Lee et al. proposed an alternative scoring approach—standardized person-mean imputation—in which each item response is converted to a z-score prior to averaging responses across endorsed items for each individual. We used this alternative scoring approach for the SRE in this study. Also, because sex strongly determines the level of response to alcohol (Gandhi et al., 2004), scoring was carried out separately by sex assigned at birth. Higher

[1] Alcohol sensitivity measures were administered as part of the eligibility screener to target recruitment of a full range of alcohol sensitivity levels from individuals assigned male at birth and individuals assigned female at birth.

Table 1
Sample Characteristics

Variable	Short-term reliability sample (N = 288)	Long-term reliability subsample (N = 173)
	M (*SD*)	*M* (*SD*)
Age T0	19.31 (.72)	—
Age T1	19.47 (.73)	19.47 (.73)
Age T2	—	20.42 (.83)
	N (%)	*N* (%)
Undergraduate status	281 (98)	168 (97)
Sex assigned at birth (female)	158 (55)	103 (60)
Gender identity		
Male[a]	121 (45)	63 (43)
Female	141 (53)	82 (49)
Other	5 (2)	2 (1)
Hispanic/Latino	22 (8)	8 (5)
Race		
White/Caucasian	251 (87)	151 (88)
Black/African American	7 (2)	6 (3)
Asian	12 (4)	6 (3)
Native American Indian	2 (<1)	0
>1 endorsed	14 (5)	9 (6)

Note. T0 = Time 0; T1 = Time 1; T2 = Time 2.
[a] One participant, also present in the subsample, who was assigned female at birth, indicated that he identifies as a male.

standardized SRE scores indicate lower alcohol sensitivity/level of response relative to same-sex peers.

The 15-item ASQ (Fleming et al., 2016; O'Neill et al., 2002) has been shown to produce a two-factor structure (Fleming et al., 2016). The first factor (ASQ-L) is composed of scores on nine items querying effects of alcohol typically experienced during lighter drinking/low exposure episodes (e.g., feeling buzzed; feeling flirtatious, becoming more talkative); the second factor (ASQ-H) is composed of scores on six items querying effects typically experienced during heavier drinking/higher exposures (e.g., passing out, vomiting, hangover). All ASQ items begin by asking the respondent to indicate whether s/he has ever experienced the effect in question from drinking alcohol. If yes, the respondent is asked to indicate either the minimum number of drinks they require to experience the effect (ASQ-L) or the maximum number of drinks they can consume without experiencing the effect (ASQ-H). Higher scores on either subscale are indicative of lower sensitivity/level of response to the subjective effects of alcohol tapped by that subscale (or the entire scale; ASQ-total). Like the SRE, scores on the ASQ were derived using standardized person-mean imputation (Lee et al., 2015), separately by sex assigned at birth. Higher ASQ scores on any item subset—or on all items together (ASQ-total)—indicates lower sensitivity/lower level of response to the subjective effects of alcohol relative to same sex peers (ASQ-total α = .91–.93; ASQ-L α = .88–.89; ASQ-H α = .90–.96).

Alcohol Involvement

A computer-administered TimeLine Follow-Back (Sobell & Sobell, 1992) was used to measure drinking in the 30 days prior to the two laboratory visits. Participants indicated the days during the previous 30 on which they consumed alcohol, the quantity of alcohol consumed on each drinking day, and the length of each drinking episode. From these responses, three alcohol use indices were computed for each participant: (a) Total Drinks (TD) was calculated as a sum of the number of drinks reported for the previous 30 days; (b) Drinks per Drinking Day (DpDD) was calculated by dividing TD by the number of days on which drinking occurred; and (c) Heavy Drinking Days (HDD) was calculated as the number of days in the previous 30 on which 4 or more/5 or more drinks were reported for those assigned female/male at birth.

The 10-item AUDIT (Saunders et al., 1993) was used as an index of potential problems related to drinking during the past year. The first three items query quantity and frequency of alcohol use and the frequency of heavy drinking episodes (six or more drinks on a single occasion). The remaining items query the frequency with which specific drinking-related consequences were experienced in the past year (e.g., unable to stop drinking once started). AUDIT scores can range from 0 to 40; scores in the 8–14 range indicate potentially hazardous or harmful alcohol consumption, and a score of ≥15 indicates likely AUD.

Procedure

Participants completed the ASQ, SRE, and alcohol involvement measures at up to three time points: T0 and T1 (N = 288), or T0, T1, and T2 (n = 173). Procedures for the T1 and T2 laboratory sessions were similar. After arriving to the laboratory and providing informed consent, participants completed the self-report measures (and others not described here) and underwent a diagnostic interview (e.g., Semi Structured Assessment for the Genetics of Alcoholism, Mini International Neuropsychiatric Interview; Bucholz et al., 1994; Sheehan et al., 1998) to assess for common psychiatric disorders. They then completed a battery of alcohol cue-reactivity tasks while electroencephalography was recorded (data reported elsewhere; see Cofresí, Kohen, et al., 2022; Cofresí, Piasecki, & Bartholow, 2022). Immediately following T1, participants completed a 21-day ecological momentary assessment protocol (data reported elsewhere; see Kohen et al., 2023).

Data Analysis

All analyses were completed using R statistical software (R Core Team, 2020). Short-term semipartial reliability of ASQ/SRE scores was computed using the olsrr package (Hebbali, 2020), controlling for time between T0 and T1. Longer term semipartial reliability analyses were carried out using data from participants who completed the ASQ and SRE at both T1 and T2, controlling for time between sessions.

Two approaches were taken to characterize the nature of relationships between alcohol involvement and ASQ/SRE scores. First, as a general approach to understanding how the alcohol sensitivity and alcohol involvement variables covary at T1 and T2, canonical correlation analyses (Hotelling, 1936) were carried out separately for ASQ and SRE variables. Canonical correlation analysis aims to assign weights to a set of predictors (e.g., ASQ or SRE subscale scores) that maximize variance they account for in a set of criterion variables (e.g., alcohol involvement variables). The analysis identifies ρ, the largest correlation attainable by correlating a linear combination of the variables in Set 1 (i.e., predictor set) with

a linear combination of the variables in Set 2 (i.e., criterion set). The correlation for the first canonical pair (i.e., the first dimension) reflects ρ for the optimal linear combinations of both variable sets; additional canonical pairs (i.e., second dimension, etc.) reflect ρ for other possible, orthogonal combinations of the variable sets. The bivariate correlations between individual variables in a set and the resulting composite (i.e., loadings) and linear weights associated with this maximal correlation (i.e., coefficients) can be used to describe relations between the sets of variables in the context of their joint variance–covariance matrix.

Second, a residualized change score approach (Castro-Schilo & Grimm, 2018; Webster & Bereiter, 1963) was used to test the hypothesis that increasing alcohol involvement between T1 and T2 would result in reduced sensitivity to alcohol's effects. Regression residuals for alcohol involvement variables (TD, DpDD, HDD, and AUDIT scores) were calculated by regressing values at T2 on values at T1. Accordingly, positive residual values represent increasing alcohol involvement between T1 and T2. Next, multiple regression analyses examined whether changes in alcohol involvement (i.e., residual scores) corresponded with changes in ASQ and SRE scores. These models regressed ASQ/SRE scores at T2 on ASQ/SRE scores at T1, the alcohol involvement residuals, and covariates (sex assigned at birth and time between T1 and T2). Materials and analysis code for this study are available by emailing the corresponding author. This study was not preregistered.

Results

Sample Descriptives

Table 2 reports raw (i.e., not standardized person-mean imputation-transformed) alcohol sensitivity scores and mean levels of alcohol use reported at T1 and T2. Person-level variability in changes in alcohol sensitivity and representative alcohol involvement variables are plotted in Figure 1. Similar plots for ASQ/SRE subscales and additional alcohol involvement variables are in Supplemental Figures S1–S7.

Test–Retest Reliabilities

Table 3 reports short-term test–retest reliability (median interval = 45 days) and longer term test–retest reliability (median interval = 0.77 years) for the ASQ, SRE, and all their constituent subscales. All test–retest reliability estimates were .63 or higher, and each estimate was lower when tested over the longer versus the shorter interval.

Canonical Correlations

Table 4 gives the canonical coefficients and loadings (first canonical pairs) at T1 and T2 for the ASQ. The canonical correlation between the ASQ subscales (ASQ-L, ASQ-H) and the alcohol involvement variables on the first dimension, representing the maximum possible correlation between weighted composites of the variables, was ρ = 0.63 ($p < .0001$) at T1 and ρ = 0.57 at T2 ($p < .0001$). The canonical correlation on the second dimension, representing the next-highest correlation between an orthogonal set of weighted composites, was small at both T1 (ρ = 0.16, $p = .25$) and T2 (ρ = 0.14, $p = .33$). Additionally, ASQ-L scores were associated with smaller loadings than ASQ-H scores at both T1 (0.23 vs. 0.98, respectively) and T2 (0.34 vs. 0.97, respectively). These patterns suggest that ASQ-L scores play a smaller role than ASQ-H scores in determining the maximally predictive linear relationship between ASQ scores and the alcohol involvement variables.

Table 2
Average Raw Alcohol Sensitivity and Alcohol Use Scores Measured at Times 1 and 2

Variable	Time 1 *M* (*SD*)	Time 2 *M* (*SD*)
	Alcohol sensitivity	
ASQ-total	4.55 (1.78)	4.74 (1.89)
ASQ-L	3.24 (1.22)	3.13 (1.17)
ASQ-H	7.33 (3.34)	7.65 (3.43)
SRE-total	5.03 (2.10)	5.24 (2.04)
SRE-5	3.71 (1.76)	3.85 (1.59)
SRE-3	5.19 (2.16)	5.33 (2.26)
SRE-H	5.96 (2.62)	6.34 (2.74)
	Alcohol involvement	
TD	36.73 (30.54)	41.87 (39.80)
DpDD	5.14 (2.75)	5.02 (2.73)
HDD	3.95 (3.26)	4.53 (3.15)
AUDIT	9.95 (5.14)	9.73 (5.26)

Note. ASQ and SRE scores reported here are untransformed, representing average numbers of drinks required to experience alcohol effects. ASQ = alcohol sensitivity questionnaire; ASQ-total = Alcohol Sensitivity Questionnaire total score; ASQ-L = ASQ light-drinking subscale score; ASQ-H = ASQ heavy-drinking subscale score; SRE = Self-Rating of the Effects of Alcohol; SRE-total = Self-Rating of the Effects of Alcohol form total score; SRE-5 = SRE score representing first five lifetime drinking episodes; SRE-3 = SRE score representing the most recent 3-month period in which alcohol was consumed; SRE-H = SRE score representing the heaviest drinking 3-month period in the lifetime; TD = total number of alcoholic drinks consumed in the past 30 days; DpDD = average number of drinks consumed on days when drinking occurred in the past 30 days; HDD = number of heavy drinking days (4+/5+ drinks for those assigned female/male at birth) in the past 30 days; AUDIT = Alcohol Use Disorder Identification Test score (past year).

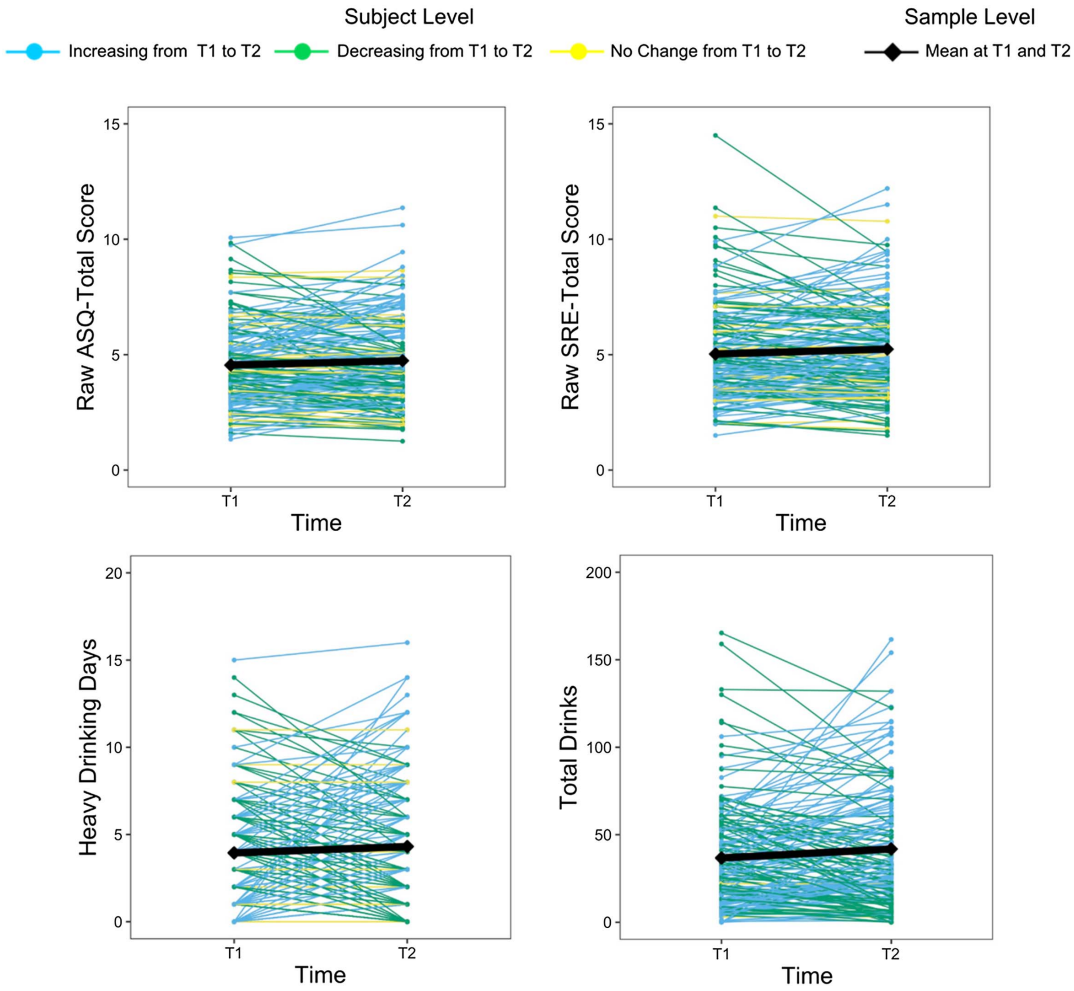
Table 5 gives the canonical coefficients and loadings (first canonical pairs) at T1 and T2 for the SRE. The canonical correlation between the SRE subscales (SRE-5, SRE-3, SRE-H) and the alcohol involvement variables on the first dimension was ρ = 0.89 ($p < .0001$) at T1 and ρ = 0.86 at T2 ($p < .0001$); the canonical correlations on the second and third dimensions were relatively small at both T1 (respectively, ρ = 0.29, $p < .01$, and ρ = 0.15, $p = .16$) and T2 (respectively, ρ = 0.26, $p = .03$, and ρ = 0.13, $p = .22$). The loadings for SRE-5 were the smallest among the three subscales at both time points, indicating that SRE-5 plays a much smaller role than SRE-3 or SRE-H in determining the maximally predictive linear relationship with the alcohol involvement variables.

Residualized Change Score Models

Summary results of models examining associations between residualized changes in alcohol involvement variables and changes in ASQ and SRE scores are presented in Table 6. Results of individual models are presented in Supplemental Tables S1 through S28. Parameter estimates for all associations between changes in alcohol involvement and changes in ASQ/SRE scores were positive, indicating that increases in drinking were associated with increases in ASQ/SRE scores (i.e., decreases in alcohol sensitivity).

Figure 1

Interindividual Variation in Changes in Alcohol Sensitivity and Alcohol Use From T1 to T2



Note. For ASQ and SRE scores, the "No Change" group represents a score difference of ±0.1 from T1 to T2. The "No Change" group for Heavy Drinking Days and Total Drinks represents a difference of exactly 0 from T1 to T2. For illustrative purposes, one outlier was removed from Heavy Drinking Days and Total Drinks plots (all analyses were robust to outlier removal). ASQ-total = Alcohol Sensitivity Questionnaire total score; SRE-total = Self-Rating of the Effects of Alcohol total score; Heavy Drinking Days = number of heavy drinking days (4+/5+ drinks for those assigned female/male at birth) in the past 30 days; TD = Total number of alcoholic drinks consumed in the past 30 days; T1 = Time 1; T2 = Time 2. See the online article for the color version of this figure.

ASQ and SRE Total Scores

Accounting for ASQ scores at T1, an increase in all alcohol involvement scores was associated with an increase in ASQ scores at T2. Change in DpDD (ΔDpDD) accounted for the most variance in changing ASQ scores (9.3%), followed by ΔTD (8.1%), ΔHDD (4.4%), and ΔAUDIT (2.5%). Similarly, an increase in any alcohol involvement score was associated with an increase in SRE scores at T2, relative to T1. As with the ASQ models, ΔDpDD accounted for the most variance in changing SRE scores (7.5%), followed by ΔTD (4.1%), ΔHDD (2.5%), and ΔAUDIT (1.4%). Overall, changes in alcohol involvement from T1 to T2 accounted for less variance in SRE scores (R^2 ranging from .014 to .075) than in ASQ scores (R^2 ranging from .025 to .093).

ASQ and SRE Subscale Scores

When ASQ subscale scores were examined separately, changes in drinking accounted for more variance in changing ASQ-L scores than in changing ASQ-H scores. Specifically, accounting for T1 scores, increases in DpDD and TD accounted for just over 8% of variance in T2 ASQ-L scores versus 5.5% and 3.6% of variance, respectively, in T2 ASQ-H scores. Similarly, increased HDD accounted for 5.2% of variance in T2 ASQ-L scores but was not associated with T2 ASQ-H scores. In contrast, whereas increases in AUDIT scores accounted for 3.7% of variance in increasing ASQ-H scores, the AUDIT was not associated with increasing ASQ-L scores.

Similarly, results for the SRE-3 and SRE-H models indicated significant positive relationships between changes in alcohol

Table 3

Shorter and Longer Term Test–Retest Reliability Estimates for the ASQ, SRE, and Their Subscale Scores

Measure	Shorter term (*Mdn*: 45 days)	Longer term (*Mdn*: 281 days)
ASQ-total	.78	.68
ASQ-L	.75	.65
ASQ-H	.73	.65
SRE	.73	.67
SRE-5	.64	.63
SRE-3	.71	.63
SRE-H	.77	.73

Note. Estimates reported here represent semipartial reliability, controlling for differences across participants in the duration between sessions. *N*s = 288–271 for shorter term retesting interval; *N*s = 173–163 for longer term retesting interval. All $p < .001$. ASQ = Alcohol Sensitivity Questionnaire; ASQ-total = Alcohol Sensitivity Questionnaire total score; ASQ-L = ASQ light-drinking subscale score; ASQ-H = ASQ heavy-drinking subscale score; SRE = Self-Rating of the Effects of Alcohol; SRE-5 = SRE score representing first five lifetime drinking episodes; SRE-3 = SRE score representing the most recent 3-month period in which alcohol was consumed; SRE-H = SRE score representing the heaviest drinking 3-month period in the lifetime.

involvement and changes in these subscale scores. Changes in alcohol involvement accounted for somewhat larger proportions of variance in changing SRE-3 scores (ΔTD: 7.4%, ΔDpDD: 2.5%, ΔHDD: 5.2%, ΔAUDIT: 4.3%) than changing SRE-H scores (ΔTD: 5.1%, ΔDpDD: 7.3%, ΔHDD: 2.6%, ΔAUDIT: 2.1%). In contrast, changes in most alcohol involvement variables from T1 to T2 were unrelated to changes in SRE-5 scores, the exception being ΔDpDD, which accounted for a modest 2.4% of variance in changing SRE-5 scores.

Table 4

Canonical Coefficients and Loadings (First Canonical Pair) for ASQ and Alcohol Involvement and Their Canonical Correlations at Times 1 and 2

Measure	Time 1 Coefficients (loadings)	Time 2 Coefficients (loadings)
ASQ variables		
ASQ-L	.0003 (.23)	.0003 (.34)
ASQ-H	1.31 (.98)	1.30 (.97)
Alcohol involvement		
DpDD	1.02 (.97)	1.03 (.98)
TD	.04 (.50)	−.08 (.20)
HDD	−.006 (.57)	.01 (.44)
AUDIT	.11 (.55)	−.02 (.38)
Canonical correlations	ρ = 0.63	ρ = 0.57

Note. Canonical coefficients are unstandardized regression weights; loadings are bivariate correlations between the individual variables and the composite within the set. Canonical correlations at both Sessions 1 and 2 are significant, $p < .0001$. ASQ = alcohol sensitivity questionnaire; ASQ-L = light-drinking subscale; ASQ-H = heavy-drinking subscale; DpDD = average number of drinks per drinking day in the past 30 days; TD = total number of alcoholic drinks consumed in the past 30 days; HDD = number of heavy drinking days (4+/5+ drinks for those assigned female/male at birth) in the past 30 days; AUDIT = Alcohol Use Disorder Identification Test total score.

Discussion

Researchers have long known that subjective response to alcohol is partly heritable (e.g., Heath et al., 1999; Kalu et al., 2012; Ray et al., 2010; Viken et al., 2003) and that consistent exposure to alcohol can dampen subjective responses through acquired tolerance (e.g., Kalant, 1996; Newman, 1941). Measures of subjective response in experienced drinkers—whether obtained during alcohol challenge or via retrospective reports—cannot readily disentangle inherited and acquired components of alcohol response. However, a prospective design affords the opportunity to estimate the extent to which changes in alcohol exposure are associated with concomitant changes in alcohol response that theoretically reflect acquired tolerance.

The present findings indicate that scores on the ASQ and SRE are reasonably stable over both shorter (*Mdn* = 45 days) and longer retesting intervals (*Mdn* = ~9 months). Similar to previous reports (Ray et al., 2011; Schuckit, Smith, & Tipp, 1997), the present data also indicate decreasing test–retest reliability for these measures as the retesting interval increases. The present study adds to the existing literature by demonstrating that a portion of the change in both ASQ and SRE scores, which contributes to their apparent decay in retest reliability, likely reflects changes in the underlying alcohol response construct resulting from increases in alcohol involvement. To our knowledge, the present study is only the second to prospectively demonstrate that sensitivity to the subjective effects of alcohol is not only a static, inherited trait but is also state like in its malleability associated with recent levels of alcohol involvement, thus corroborating a long-suspected attribute of the alcohol sensitivity construct (e.g., Corbin et al., 2013; Morean & Corbin, 2008; Trela et al., 2016). In the only other previous study of this kind, Schuckit & Smith (2004) reported divergence in SRE scores over time (between emerging adulthood and age 35) among lighter compared to heavier drinkers. However, those authors did not report the extent to which changes in drinking over time accounted for changes in alcohol sensitivity over that period.

These findings also add to the clinical utility of these measures by suggesting they can be used not only as a record of initial sensitivity but also as a within-person record of changes in subjective response to alcohol over time. As this change in response represents a change in risk profile, these measures offer important clinical information on a well-established AUD predictor, which may be useful to providers in deciding when and how to intervene in a patient's drinking or for augmenting personalized feedback interventions for high-risk drinkers (e.g., Schuckit, Smith, Clausen, et al., 2016).

Unlike the other SRE subscales (and all ASQ score variants), changes in scores on the SRE-5 subscale largely were not related to changes in alcohol involvement. Given that SRE-5 scores ostensibly represent initial levels of alcohol sensitivity prior to any opportunity for tolerance to develop (Kramer et al., 2008; Schuckit, Smith, & Tipp, 1997), this general lack of association is encouraging. However, it is notable that SRE-5 demonstrated the poorest test–retest reliability of all the scores we examined (also see Schuckit & Smith, 2013). In principle, SRE-5 should be expected to be highly stable because it is the only subscale assessing a quantity that does not change over time. In contrast, for SRE-3 (and possibly also SRE-H), participants reported about different experiences at T1 versus T2. Although our data cannot address this issue, it seems likely that SRE-5 is susceptible to the kinds of recall biases concerning early drinking experiences that have been reported in prior studies (e.g., Parra et al., 2003).

Table 5

Canonical Coefficients and Loadings (First Canonical Pair) for SRE and Alcohol Involvement and Their Canonical Correlations at Times 1 and 2

Measure	Time 1 Coefficients (loadings)	Time 2 Coefficients (loadings)
SRE subscales		
SRE-5	.00005 (.13)	.0001 (.16)
SRE-3	−.15 (.65)	.11 (.72)
SRE-H	1.28 (.996)	1.11 (.997)
Alcohol involvement		
DpDD	1.15 (.99)	1.14 (.999)
TD	.03 (.42)	.003 (.21)
HDD	−.009 (.53)	−.002 (.40)
AUDIT	.08 (.51)	.03 (.38)
Canonical correlations	ρ = 0.89	ρ = 0.86

Note. Canonical coefficients are unstandardized regression weights; loadings are bivariate correlations between the individual variables and the composite within the set. Canonical correlations at both Sessions 1 and 2 are significant, $p < .0001$. SRE = Self-Rating of the Effects of Alcohol; SRE-5 = first five lifetime drinking episodes; SRE-3 = most recent 3-month period in which drinking occurred; SRE-H = heaviest drinking 3-month period in the lifetime; DpDD = average number of drinks per drinking day in the past 30 days; TD = total number of alcoholic drinks consumed in the past 30 days; HDD = number of heavy drinking days (4+/5+ drinks for those assigned female/male at birth) in the past 30 days; AUDIT = Alcohol Use Disorder Identification Test total score.

It is also noteworthy that SRE-5 had small canonical loadings at both T1 and T2, indicating that SRE-5 plays little role in determining the SRE's optimal predictive relationship with concurrent alcohol involvement. This pattern also calls into question the clinical utility of SRE-5 scores. Overall, the SRE's utility appears to depend mainly on SRE-H scores, which had the highest loadings and largest coefficients at T1 and T2. Similarly, the ASQ's utility for understanding concurrent alcohol involvement rests largely with ASQ-H scores, which demonstrated much higher loadings and larger coefficients than did ASQ-L scores. Thus, it appears that items tapping sensitivity to heavier drinking effects, either concurrently (ASQ-H) or during the heaviest drinking period in one's lifetime (SRE-H), better determine optimal correlations between alcohol sensitivity and alcohol involvement variables than do items tapping lighter drinking effects (ASQ-L) or more recent drinking experiences (SRE-3).

The present findings largely support our primary predictions. We had hypothesized that indicators of heavier, more problematic drinking would be more strongly associated with changes in alcohol sensitivity than would a measure of total drinks consumed. This prediction was based on the idea that episodes of heavy drinking should be more likely than episodes of light or moderate drinking to contribute to the development of acquired tolerance (Elvig et al., 2021). This prediction was largely supported, in that increases in the number of drinks consumed per drinking day in the past month (DpDD) were the strongest determinant of changes over time in ASQ and SRE scores. Although DpDD is not necessarily an indicator of heavy drinking, it arguably provides a more precise index of problematic patterns of use than does a simple binge drinking threshold measure (e.g., Pearson et al., 2016). Indices of binge-drinking frequency, including our HDD measure, provide a categorical accounting of heavy drinking occasions but yield no information concerning the range of drinks consumed on such occasions. The data in Table 2 indicate that the average number of drinks consumed per drinking day in this sample was at or above the binge-drinking cutoff (e.g., Courtney & Polich, 2009), suggesting that the typical drinking day was a "heavy drinking day." Thus, DpDD has better clinical utility in populations in which heavy drinking is the norm (O'Neill et al., 2001; Pearson et al., 2016).

Table 6

Variance in T2 Alcohol Sensitivity Scores as a Function of T1 Alcohol Sensitivity Scores and Changes in Alcohol Involvement From T1 to T2

Alc. variable	ASQ-total		ASQ-L		ASQ-H			
	T1 score	Δ Alc. variable	T1 score	Δ Alc. variable	T1 score	Δ Alc. variable		
TD	.430***	.081***	.394***	.083***	.391***	.036***		
DpDD	.363***	.093***	.340***	.084***	.345***	.055***		
HDD	.422***	.043***	.379***	.052***	.399***	.010		
AUDIT	.446***	.025**	.410***	.009	.412***	.037***		
	SRE-total		SRE-3		SRE-H		SRE-5	
	T1 score	Δ Alc. variable	T1 score	Δ Alc. variable	T1 score	Δ Alc. variable	T1 score	Δ Alc. variable
TD	.410***	.041**	.360***	.074***	.463***	.051***	.384***	.003
DpDD	.383***	.075***	.370***	.025**	.420***	.073***	.386***	.024**
HDD	.431***	.025**	.382***	.052***	.480***	.026**	.389***	−.001
AUDIT	.447***	.014*	.398***	.043***	.517***	.021**	.389***	−.004

Note. Cell values are R^2 for each variable in separate models. Findings were robust to the removal of outliers. $N = 169–172$ for all models except ASQ-H ($N = 161–162$). T1 score" = T1 score for the relevant alcohol sensitivity measure; "Δ Alc. variable" = the change (residual) in the alcohol involvement variable from T1 to T2 in each model. ASQ = Alcohol Sensitivity Questionnaire; ASQ-total = Alcohol Sensitivity Questionnaire total score; ASQ-L = ASQ light-drinking subscale score; ASQH = ASQ heavy-drinking subscale score; SRE = Self-Rating of the Effects of Alcohol; SRE-total = Self-Rating of the Effects of Alcohol total score; SRE-3 = SRE score for the most recent 3-month period in which alcohol was consumed; SRE-H = SRE score for the heaviest drinking 3-month period in the lifetime; SRE-5 = SRE score for first five lifetime drinking episodes; TD = total number of alcoholic drinks consumed in the past 30 days; DpDD = average number of drinks per drinking day in the past 30 days; HDD = number of heavy drinking days (4+/5+ drinks for those assigned female/male at birth) in the past 30 days; AUDIT = Alcohol Use Disorder Identification Test total score; T1 = Time 1; T2 = Time 2.
* $p < .05$. ** $p < .01$. *** $p < .001$.

In contrast, our index of alcohol-related problems (AUDIT scores) was among the weakest correlates of changes in alcohol response across time. However, baseline (T1) AUDIT scores were uniformly larger correlates of T2 ASQ and SRE scores than were all T1 alcohol consumption measures, supporting the utility of the AUDIT for understanding variability in alcohol sensitivity.

We also predicted that effects of increasing alcohol involvement would be more apparent in changing levels of response to lighter drinking effects of alcohol compared to heavier drinking effects. Support for this prediction was mixed. The longitudinal models (Table 6) suggest that changes in alcohol involvement, especially DpDD, are somewhat stronger determinants of changes in sensitivity to lighter drinking effects (ASQ-L) compared to heavier drinking effects (ASQ-H). However, the cross-sectional canonical correlation models (Tables 4–5) provided strong evidence that scores on the ASQ-H and SRE-H subscales accounted for most of the association between the alcohol sensitivity measures and concurrent alcohol involvement.

What might account for this pattern of effects? One possibility is that sensitivity to effects generally associated with higher exposures/heavier drinking is more heritable—and therefore more resistant to change as a result of increased drinking—than is sensitivity to effects associated with lower exposures/lighter drinking. Several models (King et al., 2011, 2014; Newlin & Thomson, 1990) emphasize the importance of distinguishing sensitivity to alcohol's pleasant/stimulating effects—which tend to occur at lower acute exposures—from sensitivity to its unpleasant/sedating effects—which tend to occur at higher acute exposures (e.g., Addicott et al., 2007; Waller et al., 1986). Prior research supports a two-factor model of alcohol response in which sensitivity to alcohol's pleasant/stimulating effects and its unpleasant/sedating effects are correlated but separable phenotypes that appear to reflect distinct genotypes (see Ray et al., 2016). Thus, it is possible that sensitivity to these two broad categories of effects is differentially heritable and, thus, differentially affected by changes in alcohol involvement. To our knowledge, no research to date has tested this idea.

A related possibility is that levels of initial alcohol sensitivity determine the extent to which tolerance can be acquired. This idea has been tested extensively in rodent models, with studies generally finding that animals bred for low alcohol sensitivity are less susceptible to acquisition of tolerance than are animals bred for higher alcohol sensitivity (e.g., Khanna et al., 1985; Mayer et al., 1982, 1983; Riley & Lochry, 1977). Moreover, some evidence suggests alcohol-non-preferring rodent strains—which generally show higher alcohol sensitivity—become *more sensitive* to alcohol's sedative effects with repeated exposures, whereas alcohol-preferring rats become less sensitive (Kurtz et al., 1996). These findings suggest that initial low sensitivity might especially confer resistance to the acquisition of tolerance to alcohol's sedative effects.

To explore the possibility that initial sensitivity predicted the extent to which tolerance developed in our sample, we estimated a set of post hoc regression models in which change in the ASQ and SRE subscale scores (i.e., residuals) was predicted from an interaction between SRE-5 scores and changes in DpDD. The SRE-5 × DpDD interaction was a significant predictor of changes in ASQ-L ($t = -3.28$, $p = .001$), ASQ-total ($t = -2.54$, $p = .01$), and SRE-3 scores ($t = -2.48$, $p = .01$). In each case, the form of the interaction indicated that increases over time in DpDD led to larger changes in alcohol response for individuals with higher initial sensitivity (i.e., lower SRE-5 scores). The interaction did not emerge for ASQ-H or SRE-H scores ($t < 1$), further supporting the idea that increased drinking has a more pronounced effect on the development of tolerance to lighter drinking effects.

As noted previously, ASQ and SRE scores are believed to reflect some combination of inherited (e.g., genetic) predisposition and acquisition through drinking experience. This study provides initial support for the idea that scores on these retrospective measures can change over time in ways that are systematically related to changes in alcohol involvement. However, it would be premature to conclude that estimates of stability across time in these measures (e.g., autoregression) are representative of their heritable components, while variability in these measures associated with changes in alcohol involvement over time represents their acquired components. As there are noted genetic influences on initial alcohol sensitivity (Edwards et al., 2018), genetics could also influence the malleability of alcohol sensitivity over time, regardless of change in alcohol involvement or other factors. Conclusions regarding these matters are beyond the scope of this study, as directly addressing them would require a prospective twin study of alcohol sensitivity that incorporates repeated measures of alcohol exposure. Presumably, such a study could be designed to quantify the relative contributions of acquired tolerance, other unmeasured, nonshared environmental factors, and genetic contributions to changes in alcohol sensitivity over time (e.g., Bleidorn et al., 2014).

The present study's strengths—its longitudinal design, relatively large sample size, and measures of response to effects associated with both lighter and heavier alcohol exposures—must be considered in light of its limitations. First, our ability to estimate alcohol response and the acquisition of tolerance was limited by our use of retrospective self-report measures. Assessment of subjective responses to acute alcohol exposures in a prospective design might yield more accurate estimates of how tolerance emerges from changes in alcohol involvement. Second, the current sample was homogeneous in terms of participants' race/ethnicity and college student status. Given the evidence that subjective response to alcohol differs by race (Duranceaux et al., 2008; Pedersen & McCarthy, 2013; Wall et al., 1992), considerable caution must be used when attempting to extrapolate these findings beyond a primarily White college sample. Accordingly, it is critical for future research on alcohol sensitivity and how it changes over time to include participants who represent broader racial and sociodemographic backgrounds. Specifically, it is yet unknown whether racial groups who tend to be more highly sensitive to the subjective effects of alcohol (e.g., East Asian carriers of the ALDH2*2 allele; Goedde et al., 1992) show a similar relationship between changing alcohol involvement and alcohol sensitivity.

We used residualized scores to relate change in alcohol involvement to change in alcohol sensitivity. Residual scores represent a mixture of both change in true scores and measurement error that drive differences in observed scores over measurement occasions. The ratio of error to true score change is unknown and could vary across measures. Findings consistently supported the strong a priori hypothesis that increases in drinking would be associated with increasing tolerance to the effects of alcohol, suggesting the residuals are likely to index real changes in these constructs to some extent. However, the true effects could be underestimated if measurement error adds considerable noise to the change estimates. Finally, the analyses examined only how changes in alcohol use predict downstream levels of alcohol sensitivity.

It is also likely that both initial sensitivity and changes in sensitivity are important determinants of later alcohol use trajectories.[2] Future research should explicitly model and parse these bidirectional influences.

In conclusion, the present findings demonstrate the sensitivity of retrospective, self-report measures of alcohol response to changes in alcohol involvement during emerging adulthood. The findings further support that changes in alcohol response arising from changes in drinking are more likely to emerge for effects of alcohol typically associated with lower acute exposures. Finally, our exploratory analyses support the prediction derived from preclinical models that individuals with higher initial levels of alcohol response are more susceptible to the acquisition of tolerance to alcohol's effects. Together, these findings advance understanding of the dynamic association between alcohol use and sensitivity to alcohol's effects.

[2] Indeed, post hoc analyses predicting residualized change in drinking from sensitivity measures at T1 indicated that, with the exception of the SRE-5, lower sensitivity on all scales and subscales at T1 predicted increased ΔDpDD.

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