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RESEARCH REPORT

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Alcohol approach–avoidance task behavior and brain potentials differentially predict ecologically assessed alcohol craving and consumption in early emerging adulthood

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Abstract

Aims: The current study measured the extent to which different neurobehavioral indices of incentive-motivational salience attribution to alcohol cues predict alcohol craving and consumption in the natural environment.

Design, setting, and participants: Laboratory study at a university in Missouri, USA, followed by a smartphone-based 21-day ecological momentary assessment (EMA) protocol. Participants were emerging adults (N = 218–268 [52–56% female], age 18–20).

Measurements: Participants completed an alcohol cue approach-avoidance task while their electroencephalogram (EEG) was recorded. Behavioral measures (response time) indexed the strength of cue-activated approach vs. avoidance tendency. Cue-locked event-related potentials provided EEG-based neural measures of motivated attention (P3 amplitude) and approach-avoidance conflict (N450 amplitude). From EMA, measures of alcohol consumption dynamics (as indexed by estimated blood alcohol concentration [eBAC], g/dL) during real-world drinking episodes were obtained, as were measures of alcohol craving (7-point visual analogue scale) dynamics during and outside these episodes.

Findings: Different approach-avoidance task-derived behavioral and neural measures rank-ordered participants differently.

Participants who *approached* alcohol cues more rapidly in lab subsequently showed steeper increases in craving (ΔB ± standard error [SE] = 1.042 ± 0.499 point/hr), and eBAC (ΔB ± SE = 0.046 ± 0.017 g/dl/hr), during real-world drinking episodes. Participants who *avoided* alcohol cues more slowly in lab also showed steeper increases in eBAC (ΔB ± SE = 0.056 ± 0.017 g/dl/hr).

Participants with larger P3 during alcohol cue *approach* in lab subsequently showed steeper increases in eBAC (ΔB ± SE = 0.048 ± 0.017 g/dl/hr), as did those with smaller P3 during alcohol cue *avoidance* (ΔB ± SE = 0.025 ± 0.017 g/dl/hr).

Participants with smaller N450 during alcohol cue *approach* in lab subsequently showed steeper increases in craving during drinking episodes (ΔB ± SE = 1.465 ± 0.607 point/hr).

Tests examining lab-based neurobehavioral measures as predictors of craving dynamics during nondrinking moments, such as following incidental cue exposure, generally were inconclusive.

Conclusions: Incentive salience toward alcohol may influence alcohol seeking (including craving) and alcohol consumption through distinct behavioral risk pathways in different people.

KEYWORDS
approach bias, attention bias, appetitive motivation, craving, cue reactivity, ERP, incentive salience, neuroscience, positive affect

INTRODUCTION

The attribution of affective-motivational significance to cues associated with alcohol/drug reward, referred to as incentive salience (IS), is thought to contribute to the heterogeneity of alcohol/drug use disorders [1–4]. Nonetheless, the core IS construct was developed by psychological scientists working with non-human animal models [5, 6], so the behavioral pathways by which IS confers risk for alcohol/drug use disorder in humans remain empirically underexamined [7–9]. The present research aimed to examine the extent to which neurobehavioral manifestations of IS-related motivational and attentional processes captured in a human lab context can predict alcohol craving and consumption in natural drinking contexts.

According to incentive sensitization theory [5, 6], sensitized IS or 'wanting' attributed to reward-predictive cues can manifest in several ways, including approach, attention and craving. Focusing on alcohol reward as an example, research points to at least three behavioral pathways by which the pre-conscious mental process of IS attribution to alcohol reward-predictive cues (exteroceptive or interoceptive) may impel a person to seek and consume alcohol [7]. In the *hyper-direct* pathway, manifestations of IS attribution—cue-directed attention and approach—directly facilitate alcohol seeking or drinking. In the *direct* pathway, cue-directed attention enters a feed-forward cycle that engenders conscious alcohol use-related thoughts, imagery and feelings [10, 11], especially in consumption-associated contexts. In the *indirect* pathway, contextual barriers (e.g. 'wrong' place or time for drinking) to cue-triggered impulses generate salient internal conflict signals that almost immediately enter conscious awareness and are interpreted as an urge to imbibe [12]. Unlike the *hyper-direct* pathway, the *direct* and *indirect* pathways are theorized to result in subjective experiences of craving rather than self-administration, although in permissive real-world contexts craving may naturally progress into self-administration, which explains the strong links between momentary fluctuations in subjective craving and alcohol/drug use likelihood [13, 14]. Transient spikes in craving, resulting from the *direct* or *indirect* pathways, may also counteract naturally occurring decreases in craving over time within alcohol/drug use episodes related to satiety-like 'anti-reward' homeostatic processes [15]. Together, the *hyper-direct*, *direct* and *indirect* risk pathways represent the influence of conceptually distinct IS-related phenomena on a person's alcohol/drug seeking and taking. If truly distinct, then human laboratory measures representing these IS-related phenomena might be related to different conceptually distinct aspects of alcohol/drug use-related behavior in the natural environment, such as craving and consumption dynamics within alcohol/drug use episodes and cue- and context-related craving dynamics outside episodes, in the theoretically predicted ways described above. Alternatively, if IS-related phenomena are not truly distinct, then different human laboratory measures should be interchangeable and exhibit similar patterns of association with measures of real-world alcohol/drug use behavior.

The alcohol approach–avoidance task (AAAT) [16, 17] can provide behavioral and neural indices of these putatively distinct IS-related phenomena. In the AAAT, participants respond to images of alcoholic and non-alcoholic beverages by pushing (avoiding) or pulling (approaching) a videogame-type joystick as quickly as possible following image onset. The attribution of IS to alcohol-related cues is expected to facilitate responding (with a faster response time, RT) on alcohol approach trials and to hinder responding (with a slower RT) on alcohol avoidance trials owing to congruence and incongruence, respectively, between the activation of alcohol approach behavior and task-required behavioral responses (approach vs avoidance). Previously, we have shown that the magnitude of the N450 event-related potential (ERP), a neural index of stimulus–response conflict [18, 19], is greater on alcohol avoidance trials for persons exhibiting approach bias (in RT) and, conversely, is greater on alcohol approach trials for persons exhibiting avoidance bias [20]. In addition, the magnitude of the cue-elicited P3 (or late positive potential, LPP) ERP, a neural index of attention to a stimulus for its incentive-motivational value [21, 22], was enhanced on alcohol approach trials and dampened on alcohol avoidance trials among persons exhibiting alcohol approach bias [20], consistent with the idea that the motivational significance of alcohol-related cues is enhanced when top-down task goals and bottom-up motivations are aligned [23, 24].

Here, we examined the extent to which individual differences in the AAAT-derived behavioral RT, conflict-related N450 and attention-related P3 are differentially associated with alcohol consumption and craving dynamics in the natural environment. Ecological momentary assessment (EMA) protocols employ event- and time-based behavior sampling strategies to record alcohol/drug use (and contextual information) as it unfolds from moment to moment in the natural environment [25]. These near real-time records can be used to model the topography of alcohol/drug consumption and craving within alcohol/drug use episodes. This approach allows us to link lab-based

neurobehavioral profiles to more fine-grained aspects of real-world health-relevant behavior.

Based on the *hyper-direct* risk pathway for IS attribution to alcohol and its cues, we predicted that laboratory measures of alcohol cue-directed approach (RT) and motivated attention (P3 amplitude) would be associated with the topography of alcohol consumption within drinking episodes. Specifically, faster approach or slower avoidance (both reflecting alcohol approach bias) and heightened attention during approach trials should predict a faster ascent of estimated blood alcohol concentration (eBAC) during drinking episodes.

Based on the *direct* risk pathway, we predicted that P3 amplitude would be associated with the topography of alcohol craving within drinking episodes as well as with craving outside of drinking episodes, particularly during incidental exposure to beverage cues in consumption-associated environmental contexts. Specifically, a greater P3 amplitude during alcohol approach trials should predict spikes in craving during drinking episodes and outside of drinking episodes following incidental exposure to drinking cues in drinking-permissive contexts.

Based on the *indirect* pathway, we predicted that, as a direct measure of approach–avoidance conflict, N450 amplitude would be associated with craving outside of drinking episodes, especially reactivity to incidental cue exposure outside consumption-associated contexts. Specifically, a greater N450 amplitude during alcohol approach incongruent trials should predict spikes in craving outside of drinking episodes following incidental exposure to drinking cues outside of drinking-permissive contexts.

Predictions from the *direct* and *indirect* risk pathways were also applied to behavioral RT as a potential indirect measure of cue-related attention and conflict.

METHODS

The analyses reported herein address aims of the grant that funded data collection; however, analyses were not pre-registered on a publicly available platform, so the results should be considered tentative pending replication in an independent sample.

Participants

Participants were healthy youths (age = 19.52 ± 0.73 years, M ± SD) enrolled in a longitudinal study examining associations between laboratory-based alcohol cue reactivity, alcohol sensitivity and alcohol use in real-world contexts. A sample of 318 individuals (57% female) was available for the present report. This sample size was determined by the requirements of that longitudinal study and the practical considerations that affected data collection (e.g. COVID-19 shutdown). The primarily White (87%), non-Hispanic (93%), undergraduate student (97%) sample was recruited from Columbia, MO, USA, and represented a spectrum of alcohol involvement. Detailed information about the sample, including recruitment strategies, inclusion and exclusion criteria, and compensation are provided in prior reports [20, 26, 27]. Briefly, the eligibility criteria were: (i) age 18–20 years; (ii) monthly alcohol use across the past year and at least one binge-drinking episode in the past 6 months; (iii) no history of neurological disease or head injury; and (iv) no history of unsuccessful attempts to reduce alcohol use. The analytic sample sizes varied owing to variability in patterns of missing data across predictors and covariates. Sample characteristics did not differ among analytic subsamples (Table S1).

Procedure

All participants completed an in-person laboratory visit followed by a 21-day EMA period beginning the next day. Upon arrival, participants provided informed consent, completed a breath alcohol test, to verify sobriety, and their anthropometrics (height and body weight) were recorded. As part of a battery of lab-based tasks, participants completed the AAAT while their electroencephalogram (EEG) was recorded. At the end of the visit, a smartphone app [28] was downloaded onto the participant's phone and instructions were given for its use during the EMA protocol. Participants completed a brief EMA survey before leaving the lab to ensure that the app was working as intended and that they understood how to use it.

Materials

Alcohol approach–avoidance task (AAAT)

On each of 360 trials, a photo of an alcoholic beverage (alcohol; e.g. beer bottle), a non-alcoholic beverage (non-alcohol; e.g. juice bottle) or a non-beverage non-comestible liquid (non-beverage; e.g. motor oil), titled 3° left or right, was presented centrally (for approx. 1.5 s in duration with approx. 0.5 s between trials). Participants were instructed to push (avoid) or pull (approach) on a joystick (based on the direction of the tilt for each photo) as quickly as possible following photo onset. Pulling on the joystick caused the photos to increase in size (appearing to come toward the participant) and pushing on the joystick caused the photos to decrease in size (appearing to recede away from the participant), providing visual reinforcement of behavioral approach and avoidance action-related proprioceptive feedback [29, 30]. Stimulus–response mappings (left/right tilt : push/pull) were counterbalanced across participants. For other details (e.g. example stimuli), see our prior report [20].

Measures

Predictor: in-lab behavioral measures

Person-level median RT scores for different AAAT trial types were computed after discarding error trials (5.23% ± 3.55%, M ± SD) and correct trials with RT ≤ 200 ms (1.93% ± 3.41%) or RT ≥ 3SD from each person's mean RT (6.22% ± 2.19%). Person-level median RTs, which are robust to any remaining extreme RTs, were used for consistency with our prior report [20]; however, person-level mean RTs produce similar results. Figure S1 shows sample M ± SE values for RT scores by trial type. RT scores were similar among analytic subsamples

(Table S2). Furthermore, RT scores had excellent internal consistency [adjusted split-half (odds/evens) reliability ($r_{split\text{-}half}$) = 0.92–0.94] [31]. Raw and residualized RT bias scores, such as difference scores (e.g. alcohol avoid RT – alcohol approach RT) and double-difference scores (e.g. alcohol RT difference – non-alcohol RT difference), were also derived [32]. However, RT bias scores demonstrated poor-to-modest internal consistency ($r_{split\text{-}half}$ = −0.03–0.66) [31], and were not considered adequately reliable as measures of individual differences [33].

Predictor: in-lab neural measures

Electroencephalograms (EEGs) were recorded at 512 Hz from 32 Ag/AgCl electrodes (M2 reference, FPz ground) arranged according to the expanded 10–20 system [34]. Impedance was ≤10 kΩ. Offline, processing used eeglab [35] and erplab [36]. EEGs were re-referenced (average mastoid), resampled (256 Hz) and filtered (0.1–30 Hz). Components corresponding to blinks, eye movements and other artifacts were identified via independent component analysis (ICA) and removed (5.25 ± 2.85, M ± SD). Excessively noisy channels were discarded pre-ICA and interpolated post-ICA (1.62 ± 1.61, M ± SD). EEGs from correct-response trials were segmented into stimulus-locked epochs prior to additional artifact detection and rejection. Artifact-free epochs (45.01 ± 11.74 per type, M ± SD) were then averaged to obtain electrode × trial type ERPs. The grand average ERP (across trial types and persons) was inspected, and in consultation with the extant literature, the electrodes and time windows in which the magnitudes of the N450 and P3 components were maximal were identified. The N450 was quantified as the mean amplitude over nine frontal/central electrodes at 225–575 ms for each trial type (Figure S2). The P3 was quantified as the mean amplitude over nine parietal/occipital electrodes at 400–600 ms for each trial type (Figure S3). ERP scores were similar among analytic subsamples (Table S2) and demonstrated good-to-excellent internal consistency ($r_{split\text{-}half}$ = 0.87–0.92) [31]. The internal consistency of ERP difference and double-difference scores (raw and residualized) was poor ($r_{split\text{-}half}$ = 0.04–0.34) [31], and therefore, as with RT bias scores, no ERP bias scores were sufficiently reliable as indices of individual differences [33, 37].

Outcome: EMA alcohol consumption

Participants were instructed to initiate a report in the app upon first consuming an alcoholic beverage. Participants also received time-based survey prompts throughout the day (one at user-specified wake-up time plus four at equally spaced pseudo-random times between 8:00 AM and 11:00 PM) asking whether they had consumed alcohol in the past 2 hours. Reports of alcohol use triggered follow-up assessments at 30-minute intervals for the next 2 hours, in which participants were asked to indicate the number of standard drinks consumed since the last assessment (in increments of 0.5 drinks, up to '6 or more') aided by an infographic of standard drinks (14 g pure ethanol equivalents) contained in different alcoholic beverages varying in alcohol content by volume. Automatically generated timestamps with each submitted report were used to keep track of time. From these data, eBAC was calculated at every moment using a standard formula [38], provided in the supporting information, that takes into account the amount ingested, the timing since ingestion, biological sex, body weight (as recorded at lab visit) and elimination rate, as in our prior works [26, 27, 39, 40]. Rare instances of missing eBAC or potentially erroneous eBAC (>0.20 g/dl) values were discarded, as were the few moments determined to be on the descending limb of the BAC time course and the few moments with eBAC = 0 and ΔeBAC = 0 (relative to the previous moment), as these may be post-drinking moments. Thus, only moments on the ascending limb of the BAC curve and within the 2-hour follow-up window were analyzed as belonging to 'drinking episodes'. On average, these moments represented approximately 10% of all EMA observations, with three drinking episodes captured per person and three standard drinks consumed per episode per person. These did not differ by analytic subsample (Table S3).

Outcome: EMA alcohol craving

Participants responded to two items ('urge to drink' and 'craving a drink') using a visual analogue scale anchored at 1 (not at all) and 7 (extremely). Momentary craving was calculated as the mean across these items, based on excellent internal consistency (α = 0.99) [41]. Participants also responded to an item about exposure to beverage cues in the past 15 minutes by selecting whether alcohol was 'visible directly—bottle, glass, etc.' (coded 1) or 'no, not visible' (coded 0). These items were adapted from prior work [42, 43]. These reports were used to quantify: (i) craving across moments in which neither drinking nor beverage cues were reported (i.e. baseline or tonic craving); (ii) changes in craving level over the course of drinking episodes (as defined in Outcome: EMA Alcohol Consumption) during which beverage cues abound; and (iii) changes in craving level following incidental exposure to beverage cues outside of drinking episodes. On average, 10% of all EMA observations involved beverage cues outside of drinking episodes. This did not differ by analytic subsample (Table S3).

Covariates

Ecological momentary assessment (EMA) contextual factors were included as moment-level covariates in all analyses. Automatically generated timestamps with each submitted report were used to create a liberally defined weekend (from 6:00 PM Thursday to 6:00 PM Sunday, coded 1) versus weekday (from 6:01 PM Sunday to 5:59 PM Thursday, coded 0) variable, consistent with prior work on drinking patterns of emerging adult college students in the USA [44, 45]. Other contextual factors included: peer (i.e. friend, partner or coworker)

presence (coded 1) versus absence (coded 0), bar/pub/restaurant (coded 1) versus other location (coded 0), cannabis use (coded 1) versus non-use (coded 0) since last report, and tobacco use (coded 1) versus non-use (coded 0) since last report.

Certain participant characteristics were included as person-level covariates in all analyses unless otherwise indicated, such as: age, biological sex (female = 0, male = 1), body mass index and hazardousness of typical alcohol use, as indexed by the total score on the Alcohol Use Disorders Identification test (AUDIT) [46], which had acceptable internal consistency ($\alpha = 0.77$). For analyses of alcohol craving within drinking episodes, baseline or tonic craving, as indexed by person-level mean craving intensity outside of drinking episodes and incidental beverage cue exposures across the EMA period, was also included as a person-level covariate.

These contextual factors and personal characteristics have been associated with either alcohol craving or consumption or both in previous studies [39, 40, 47–54].

Analytic approach

Primary analyses tested the EMA outcome-predictive utility of behavioral and neural measures from AAAT alcohol trials. Separate models were tested for RT, N450 and P3 scores; however, both 'trial type' scores (e.g. alcohol approach RT, alcohol avoid RT) were included in each model to control for shared variance. Models initially included predictor interaction terms (e.g. alcohol approach RT × alcohol avoid RT), but these produced small, non-significant effects so they were dropped from consideration. Parallel analyses tested behavioral and neural measures from AAAT non-alcohol and non-beverage trials, providing insight into specificity of alcohol trial effects. Sensitivity (robustness) analyses tested alcohol trial effects accounting for non-alcohol trial effects, which is the ideal control here given the appetitive value of non-alcohol beverage cues compared with non-beverage cues [55]. To facilitate interpretation, N450 amplitudes were reverse scored. AAAT trial-derived predictors were standardized. Continuous covariates were grand mean centered. Categorical covariates were effect coded. All were modeled as simple numeric predictors.

As in prior work [26, 27], analyses involved generalized linear multi-level models (GLMMs) using the gamma distribution with a log link function. To make eBACs suitable for gamma GLMMs, 0.0001 was added to all eBACs. Unless otherwise indicated, all GLMMs incorporated random slopes and random intercepts, as appropriate, and these were allowed to correlate with one another. Separate GLMMs were fit for predicting alcohol craving and consumption (eBAC) trajectories over time (linear and quadratic trends) within drinking episodes as well as for predicting alcohol craving in non-drinking moments. Example model formulas are provided in the supporting information. EMA outcome-predictive utility tests involved evaluating specific coefficients in the GLMM, corresponding to moderation effects (e.g. alcohol approach RT × linear time), using Wald Type III tests. The threshold for statistical significance was $P < 0.050$.

Follow-up analyses involved the examination of model-estimated covariate-adjusted marginal means and simple slopes using asymptotic *Z*-tests. All follow-up analyses were conducted on the log scale; however, back-transformed (exponentiated) and 'unlinked' covariate-adjusted model-estimated values are provided, where possible, to facilitate interpretation.

Two sets of ancillary bivariate correlation analyses were also conducted. The first examined associations among AAAT-derived measures. Figure S4 shows the independence of RT and P3 or N450 measures, and only weak links between P3 and N450 measures. The second correlation analysis examined associations between AAAT-derived measures and the EMA-derived measure of baseline or tonic alcohol craving. No significant associations were detected (Table S4).

All analysis was conducted using R 4.5.1 [56] and the following packages: emmeans [57], ggplot2 [58], lme4 [59], lmertest [60], performance [61] and psych [62].

RESULTS

Table 1 presents results from GLMM-based tests of whether in-lab AAAT-derived behavioral and neural measures moderate the dynamics of alcohol consumption (specifically linear growth in eBAC) during EMA-captured drinking episodes; Table 2 presents results concerning the moderation of alcohol craving dynamics (specifically linear growth component) during drinking episodes. Table 3 presents results from GLMM-based tests of whether in-lab AAAT-derived measures moderate alcohol cue- and context-related fluctuations in alcohol craving in moments outside of drinking episodes. Only results from models involving fixed effects for which the appropriate random slopes could be specified consistently are presented below. Readers interested in moderation tests involving fixed effects for which these random slopes could not be specified consistently across models—and for which Type 1 error therefore may be inflated [63–65]—are referred to Table S5 (quadratic change in eBAC during drinking episodes), Table S6 (quadratic change in craving during drinking episodes) and Table S7 (effects of recent cannabis or tobacco use on craving outside drinking episodes) in the supporting information.

Alcohol consumption (eBAC) trajectory during alcohol use episodes

Associations with AAAT behavioral RT

The primary analysis indicated that alcohol approach and alcohol avoid RTs were associated with eBAC dynamics (Table 1). Inspection of the model-estimated eBAC time course revealed that: (i) faster compared with slower alcohol approach RTs (Figure 1a); and (ii) slower compared with faster alcohol avoid RTs (Figure 1B) predicted a faster rise in eBAC, resulting in a higher eBAC 1 hour into drinking. The parallel analyses indicated that non-alcohol and non-beverage avoid RTs were also associated with eBAC dynamics (Table 1). Simple slopes analysis confirmed a faster rise in eBAC within episodes with faster RTs on alcohol approach trials or slower RTs on

TABLE 1 Summary of GLMMs examining the linear component of change in alcohol consumption (eBAC) trajectory within alcohol use episodes captured in 21-day EMA protocol as a function of in-lab AAAT-derived behavioral and neural measures.

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Time (hours)	**1.426 ± 0.069**	**4.162 ± 1.072**	**425.743**	**<0.0001**
RT	Primary	**Alcohol approach RT (*Z*) × time (hours)**	**−0.350 ± 0.111**	**0.704 ± 1.117**	**9.951**	**0.0016**
		Alcohol avoid RT (*Z*) × time (hours)	**0.410 ± 0.110**	**1.507 ± 1.116**	**14.006**	**0.0002**
	Parallel	Non-alcohol approach RT (*Z*) × time (hours)	−0.210 ± 0.114	0.810 ± 1.121	3.396	0.0653
		Non-alcohol avoid RT (*Z*) × time (hours)	**0.251 ± 0.114**	**1.285 ± 1.120**	**4.865**	**0.0274**
		Non-beverage approach RT (*Z*) × time (hours)	−0.224 ± 0.118	0.799 ± 1.125	3.602	0.0577
		Non-beverage avoid RT (*Z*) × time (hours)	**0.279 ± 0.123**	**1.321 ± 1.131**	**5.120**	**0.0237**
	Sensitivity	Alcohol approach RT (*Z*) × time (hours)	−0.121 ± 0.174	0.886 ± 1.190	0.484	0.4868
		Alcohol avoid RT (*Z*) × time (hours)	**0.635 ± 0.188**	**1.887 ± 1.207**	**11.427**	**0.0007**
N450	Primary	Alcohol approach N450 (*Z*) × time (hours)	−0.107 ± 0.138	0.898 ± 1.147	0.606	0.4362
		Alcohol avoid N450 (*Z*) × time (hours)	0.104 ± 0.129	1.110 ± 1.138	0.650	0.4201
	Parallel	Non-alcohol approach N450 (*Z*) × time (hours)	0.097 ± 0.159	1.102 ± 1.172	0.377	0.5392
		Non-alcohol avoid N450 (*Z*) × time (hours)	−0.109 ± 0.151	0.897 ± 1.163	0.520	0.4708
		Non-beverage approach N450 (*Z*) × time (hours)	**−0.474 ± 0.151**	**0.622 ± 1.164**	**9.791**	**0.0018**
		Non-beverage avoid N450 (*Z*) × time (hours)	**0.481 ± 0.151**	**1.617 ± 1.163**	**10.152**	**0.0014**
	Sensitivity	Alcohol approach N450 (*Z*) × time (hours)	−0.114 ± 0.151	0.893 ± 1.163	0.563	0.4530
		Alcohol avoid N450 (*Z*) × time (hours)	0.100 ± 0.178	1.105 ± 1.195	0.313	0.5760
P3	Primary	**Alcohol approach P3 (*Z*) × time (hours)**	**0.446 ± 0.132**	**1.563 ± 1.141**	**11.383**	**0.0007**
		Alcohol avoid P3 (*Z*) × time (hours)	**−0.321 ± 0.134**	**0.725 ± 1.144**	**5.722**	**0.0167**
	Parallel	Non-alcohol approach P3 (*Z*) × time (hours)	0.239 ± 0.141	1.270 ± 1.151	2.874	0.0900
		Non-alcohol avoid P3 (*Z*) × time (hours)	−0.126 ± 0.138	0.881 ± 1.148	0.837	0.3602
		Non-beverage approach P3 (*Z*) × time (hours)	0.028 ± 0.145	1.028 ± 1.156	0.036	0.8488
		Non-beverage avoid P3 (*Z*) × time (hours)	0.032 ± 0.143	1.032 ± 1.154	0.050	0.8234
	Sensitivity	**Alcohol approach P3 (*Z*) × time (hours)**	**0.412 ± 0.150**	**1.509 ± 1.162**	**7.513**	**0.0061**
		Alcohol avoid P3 (*Z*) × time (hours)	**−0.319 ± 0.163**	**0.727 ± 1.177**	**3.851**	**0.0497**

Note: AAAT = alcohol approach avoidance task; eBAC = estimated blood alcohol concentration; EMA = ecological momentary assessment; GLMMs = generalized linear mixed models.

AAAT-derived measures include correct response time (RT), reverse-scored mean amplitude of the N450 component in the stimulus-locked event-related brain potential (ERP) over a frontal/central electrode cluster and mean amplitude of the P3 component in the ERP over a parietal/occipital electrode cluster for trials requiring approach- and avoidance-like behavioral responses to images of alcoholic beverages (alcohol), non-alcoholic beverages (non-alcohol) or non-beverage non-comestible liquids (non-beverage).

Primary analyses involved testing alcohol approach and avoid trial RT, N450 or P3 score moderation of the linear component of eBAC change over time within alcohol use episodes. Parallel analyses involved testing the same moderation effects for scores from non-alcohol or non-beverage trials in the AAAT. Sensitivity analyses involved testing effects of alcohol trial scores controlling for non-alcohol trial scores. GLMMs utilized correlated random slopes and intercepts and controlled for age, body mass index, biological sex, AUDIT total score, and contextual factors such as physical, social and temporal setting.

Main effect of time (hours) is taken from a model using the analytic subsample for RT score tests but without the latter lab-derived predictors in the model. This effect did not differ substantively across models with different lab-derived predictors.

Unstandardized log scale beta coefficient (*B*) and standard errors (SE), exponentiated *B* and SE and chi-square (χ^2) statistics are presented for each predictor term alongside the *P*-value for the accompanying Type 3 Wald test (which applies to both *B* ± SE and χ^2). The χ^2 statistic shows the degree to which the predictor term contributes to the GLMM model fit. The exponentiated beta coefficients are interpretable as a proportional or percentage change in the outcome as a function of unit increases in the predictor term. For example, 'primary analysis' > 'alcohol approach RT (*Z*) × time (hours)' is statistically significant, indicating that between-person differences in alcohol approach RT were associated with between-person differences in eBAC linear growth within alcohol use episodes. For this predictor term, the exponentiated *B* indicates a (1–0.704 = 0.296 × 100% =) 29.6% *decrease* in eBAC linear growth per 1 unit increase in alcohol approach RT, or alternatively a 29.6% *increase* in eBAC linear growth per 1 unit *decrease* in alcohol approach RT.

Text in bold highlights effects that were statistically significant.

TABLE 2 Summary of GLMM analyses examining the linear component of change in subjective craving trajectory within alcohol use episodes captured in a 21-day EMA protocol as a function of in-lab AAAT-derived behavioral and neural measures.

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Time (hours)	0.042 ± 0.047	1.043 ± 1.048	0.790	0.3742
RT	Primary	**Alcohol approach RT (Z) × time (hours)**	**−0.181 ± 0.086**	**0.834 ± 1.090**	**4.414**	**0.0356**
		Alcohol avoid RT (Z) × time (hours)	0.120 ± 0.084	1.128 ± 1.088	2.036	0.1536
	Parallel	Non-alcohol approach RT (Z) × time (hours)	0.084 ± 0.083	1.088 ± 1.087	1.018	0.3129
		Non-alcohol avoid RT (Z) × time (hours)	−0.112 ± 0.083	0.894 ± 1.086	1.833	0.1758
		Non-beverage approach RT (Z) × time (hours)	−0.102 ± 0.092	0.903 ± 1.096	1.230	0.2675
		Non-beverage avoid RT (Z) × time (hours)	0.068 ± 0.091	1.070 ± 1.095	0.562	0.4533
	Sensitivity	**Alcohol approach RT (Z) × time (hours)**	**−0.483 ± 0.128**	**0.617 ± 1.137**	**14.112**	**0.0002**
		Alcohol avoid RT (Z) × time (hours)	0.043 ± 0.143	1.044 ± 1.154	0.090	0.7646
N450	Primary	**Alcohol approach N450 (Z) × time (hours)**	**−0.235 ± 0.098**	**0.791 ± 1.103**	**5.758**	**0.0164**
		Alcohol avoid N450 (Z) × time (hours)	0.182 ± 0.097	1.200 ± 1.102	3.551	0.0595
	Parallel	Non-alcohol approach N450 (Z) × time (hours)	0.152 ± 0.110	1.164 ± 1.116	1.910	0.1670
		Non-alcohol avoid N450 (Z) × time (hours)	−0.165 ± 0.107	0.848 ± 1.113	2.374	0.1234
		Non-beverage approach N450 (Z) × time (hours)	0.041 ± 0.111	1.042 ± 1.117	0.138	0.7105
		Non-beverage avoid N450 (Z) × time (hours)	−0.071 ± 0.112	0.931 ± 1.119	0.399	0.5276
	Sensitivity	**Alcohol approach N450 (Z) × time (hours)**	**−0.296 ± 0.109**	**0.744 ± 1.115**	**7.442**	**0.0064**
		Alcohol avoid N450 (Z) × time (hours)	0.076 ± 0.128	1.079 ± 1.137	0.349	0.5546
P3	Primary	Alcohol approach P3 (Z) × time (hours)	0.112 ± 0.103	1.119 ± 1.108	1.195	0.2743
		Alcohol avoid P3 (Z) × time (hours)	−0.140 ± 0.102	0.870 ± 1.108	1.874	0.1710
	Parallel	Non-alcohol approach P3 (Z) × time (hours)	−0.109 ± 0.096	0.897 ± 1.101	1.268	0.2602
		Non-alcohol avoid P3 (Z) × time (hours)	0.029 ± 0.093	1.029 ± 1.098	0.096	0.7568
		Non-beverage approach P3 (Z) x time (hours)	−0.091 ± 0.108	0.913 ± 1.114	0.711	0.3992
		Non-beverage avoid P3 (Z) × time (hours)	0.033 ± 0.109	1.034 ± 1.115	0.093	0.7602
	Sensitivity	Alcohol approach P3 (Z) × time (hours)	0.198 ± 0.112	1.219 ± 1.119	3.108	0.0779
		Alcohol avoid P3 (Z) × time (hours)	0.034 ± 0.127	1.034 ± 1.135	0.071	0.7895

Note: AAAT = alcohol approach avoidance task; EMA = ecological momentary assessment; GLMMs = generalized linear mixed models; subjective craving = within-person mean of crave and urge items assessed at same occasion.

AAAT-derived measures include correct response time (RT), reverse-scored mean amplitude of the N450 component in the stimulus-locked event-related brain potential (ERP) over a frontal/central electrode cluster, and mean amplitude of the P3 component in the ERP over a parietal/occipital electrode cluster for trials requiring approach- and avoidance-like behavioral responses to images of alcoholic beverages (alcohol) or non-alcoholic beverages (non-alcohol) or non-beverage non-comestible liquids (non-beverage).

Primary analyses involved testing alcohol approach and avoid trial RT, N450 or P3 score moderation of the linear component of subjective craving change over time within alcohol use episodes. Parallel analyses involved testing the same moderation effects for scores from non-alcohol or non-beverage trials in the AAAT. Sensitivity analyses involved testing effects of alcohol trial scores controlling for non-alcohol trial scores. GLMMs utilized correlated random slopes and intercepts and controlled for age, body mass index, biological sex, AUDIT total score and contextual factors such as physical, social and temporal setting. GLMMs also controlled for between-person differences in baseline or tonic craving as indexed by person-level mean craving across all EMA moments outside of alcohol use episodes and beverage cue exposure incidents.

Main effect of time (hours) is taken from a model using the analytic subsample for RT score tests but without the latter lab-derived predictors in the model. This effect did not differ substantively across models with different lab-derived predictors.

Unstandardized log scale beta coefficient (*B*) and standard error (SE), exponentiated *B* and SE, and chi-square (χ^2) statistics are presented for each predictor term alongside the *P*-value for the accompanying Type 3 Wald test (which applies to both *B* ± SE and χ^2). The χ^2 statistic shows the degree to which the predictor term contributes to the GLMM model fit. The exponentiated beta coefficients are interpretable as a proportional or percentage change in the outcome as a function of unit increases in the predictor term. For example, 'Primary analysis' > 'alcohol approach RT (Z) × time (hours)' is statistically significant, indicating that between-person differences in alcohol approach RT were associated with between-person differences in the linear growth of craving over time within alcohol use episodes. For this predictor term, the exponentiated *B* indicates a (1–0.834 = 0.166 × 100% =) 16.6% *decrease* in craving linear growth per 1 unit increase in alcohol approach RT or, alternatively, a 16.6% *increase* in craving linear growth per 1 unit *decrease* in alcohol approach RT.

Text in bold highlights effects that were statistically significant.

TABLE 3 Summary of GLMM analyses examining alcohol cue- and context-related fluctuations in subjective craving *outside* of alcohol use episodes captured in 21-day EMA protocol as a function of in-lab AAAT-derived behavioral and neural measures.

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Alcohol cue	**0.176 ± 0.050**	**1.193 ± 1.051**	**12.573**	**0.0004**
		Alcohol physical context	−0.007 ± 0.039	0.993 ± 1.040	0.031	0.8602
		Alcohol temporal context	**0.014 ± 0.020**	**1.014 ± 1.020**	**0.479**	**0.4889**
		Alcohol social context	**0.049 ± 0.019**	**1.050 ± 1.019**	**6.932**	**0.0085**
		Alcohol cue × alcohol physical context	**0.152 ± 0.062**	**1.164 ± 1.064**	**6.068**	**0.0138**
		Alcohol cue × alcohol temporal context	**0.087 ± 0.031**	**1.091 ± 1.032**	**7.870**	**0.0050**
		Alcohol cue × alcohol social context	0.055 ± 0.035	1.057 ± 1.036	2.443	0.1181
RT	Primary	Alcohol approach RT (*Z*) × alcohol cue	−0.016 ± 0.081	0.984 ± 1.084	0.038	0.8455
		Alcohol avoid RT (*Z*) × alcohol cue	−0.008 ± 0.080	0.992 ± 1.083	0.011	0.9158
		Alcohol approach RT (*Z*) × alcohol physical context	−0.001 ± 0.075	0.999 ± 1.078	0.000	0.9946
		Alcohol avoid RT (*Z*) × alcohol physical context	0.018 ± 0.076	1.018 ± 1.079	0.055	0.8147
		Alcohol approach RT (*Z*) × alcohol cue × alcohol physical context	−0.149 ± 0.203	0.862 ± 1.225	0.539	0.4630
		Alcohol avoid RT (*Z*) × alcohol cue × alcohol physical context	0.098 ± 0.184	1.103 ± 1.202	0.283	0.5948
		Alcohol approach RT (*Z*) × alcohol temporal context	−0.026 ± 0.040	0.974 ± 1.041	0.420	0.5167
		Alcohol avoid RT (*Z*) × alcohol temporal context	0.000 ± 0.038	1.000 ± 1.039	0.000	0.9898
		Alcohol approach RT (*Z*) × alcohol cue x alcohol temporal context	0.192 ± 0.127	1.211 ± 1.135	2.293	0.1300
		Alcohol avoid RT (*Z*) × alcohol cue x alcohol temporal context	−0.143 ± 0.123	0.867 ± 1.131	1.343	0.2465
		Alcohol approach RT (*Z*) × alcohol social context	−0.038 ± 0.037	0.963 ± 1.038	1.032	0.3097
		Alcohol avoid RT (*Z*) × alcohol social context	0.054 ± 0.036	1.056 ± 1.037	2.279	0.1311
		Alcohol approach RT (*Z*) × alcohol cue x alcohol social context	−0.007 ± 0.143	0.993 ± 1.154	0.003	0.9583
		Alcohol avoid RT (*Z*) × alcohol cue × alcohol social context	0.009 ± 0.138	1.009 ± 1.148	0.004	0.9487
	Parallel	Non-alcohol approach RT (*Z*) × alcohol cue	−0.018 ± 0.084	0.982 ± 1.088	0.045	0.8318
		Non-alcohol avoid RT (*Z*) × alcohol cue	−0.013 ± 0.091	0.987 ± 1.095	0.020	0.8883
		Non-alcohol approach RT (*Z*) × alcohol physical context	0.035 ± 0.076	1.036 ± 1.078	0.217	0.6413
		Non-alcohol avoid RT (*Z*) × alcohol physical context	−0.03 ± 0.083	0.971 ± 1.086	0.131	0.7173
		Non-alcohol approach RT (*Z*) × alcohol cue × alcohol physical context	−0.167 ± 0.196	0.846 ± 1.217	0.727	0.3940
		Non-alcohol avoid RT (*Z*) × alcohol cue × alcohol physical context	0.141 ± 0.203	1.152 ± 1.225	0.487	0.4851
		Non-alcohol approach RT (*Z*) × alcohol temporal context	−0.056 ± 0.040	0.946 ± 1.041	1.969	0.1605
		Non-alcohol avoid RT (*Z*) × alcohol temporal context	0.026 ± 0.041	1.026 ± 1.042	0.388	0.5336
		Non-alcohol approach RT (*Z*) × alcohol cue × alcohol temporal context	0.014 ± 0.125	1.014 ± 1.133	0.012	0.9128
		Non-alcohol avoid RT (*Z*) × alcohol cue × alcohol temporal context	0.034 ± 0.133	1.035 ± 1.142	0.066	0.7974
		Non-alcohol approach RT (*Z*) × alcohol social context	−0.042 ± 0.038	0.959 ± 1.039	1.220	0.2693
		Non-alcohol avoid RT (*Z*) × alcohol social context	0.057 ± 0.039	1.059 ± 1.040	2.109	0.1465
		Non-alcohol approach RT (*Z*) × alcohol cue × alcohol social context	0.101 ± 0.145	1.106 ± 1.156	0.485	0.4862
		Non-alcohol avoid RT (*Z*) × alcohol cue × alcohol social context	−0.086 ± 0.157	0.917 ± 1.170	0.303	0.5822
		Non-beverage approach RT (*Z*) × alcohol cue	0.028 ± 0.085	1.028 ± 1.089	0.107	0.7433
		Non-beverage avoid RT (*Z*) × alcohol cue	−0.046 ± 0.085	0.955 ± 1.089	0.290	0.5905
		Non-beverage approach RT (*Z*) × alcohol physical context	−0.11 ± 0.070	0.896 ± 1.073	2.432	0.1188
		Non-beverage avoid RT (*Z*) × alcohol physical context	0.115 ± 0.074	1.122 ± 1.077	2.385	0.1225
		Non-beverage approach RT (*Z*) × alcohol cue × alcohol physical context	0.029 ± 0.185	1.029 ± 1.204	0.025	0.8755
		Non-beverage avoid RT (*Z*) × alcohol cue × alcohol physical context	−0.041 ± 0.195	0.960 ± 1.215	0.045	0.8323
		Non-beverage approach RT (*Z*) × alcohol temporal context	−0.021 ± 0.042	0.979 ± 1.042	0.258	0.6116
		Non-beverage avoid RT (*Z*) × alcohol temporal context	−0.018 ± 0.040	0.982 ± 1.041	0.199	0.6553

TABLE 3 (Continued)

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Non-beverage approach RT (*Z*) × alcohol cue × alcohol temporal context	0.028 ± 0.135	1.029 ± 1.145	0.043	0.8351
		Non-beverage avoid RT (*Z*) × alcohol cue × alcohol temporal context	0.017 ± 0.128	1.017 ± 1.137	0.017	0.8975
		Non-beverage approach RT (*Z*) × alcohol social context	0.003 ± 0.039	1.003 ± 1.04	0.005	0.9443
		Non-beverage avoid RT (*Z*) × alcohol social context	0.005 ± 0.038	1.005 ± 1.039	0.017	0.8977
		Non-beverage approach RT (*Z*) × alcohol cue × alcohol social context	0.080 ± 0.148	1.084 ± 1.159	0.296	0.5863
		Non-beverage avoid RT (*Z*) × alcohol cue × alcohol social context	−0.032 ± 0.145	0.968 ± 1.156	0.050	0.8239
	Sensitivity	Alcohol approach RT (*Z*) × alcohol cue	0.032 ± 0.134	1.033 ± 1.144	0.058	0.8095
		Alcohol avoid RT (*Z*) × alcohol cue	0.049 ± 0.141	1.05 ± 1.152	0.120	0.7293
		Alcohol approach RT (*Z*) × alcohol physical context	0.042 ± 0.125	1.043 ± 1.133	0.111	0.7393
		Alcohol avoid RT (*Z*) × alcohol physical context	0.145 ± 0.138	1.156 ± 1.148	1.094	0.2956
		Alcohol approach RT (*Z*) × alcohol cue × alcohol physical context	−0.138 ± 0.328	0.871 ± 1.388	0.178	0.6730
		Alcohol avoid RT (*Z*) × alcohol cue × alcohol physical context	−0.007 ± 0.380	0.993 ± 1.462	0.000	0.9844
		Alcohol approach RT (*Z*) × alcohol temporal context	0.053 ± 0.065	1.055 ± 1.067	0.670	0.4130
		Alcohol avoid RT (*Z*) × alcohol temporal context	0.022 ± 0.068	1.022 ± 1.07	0.108	0.7426
		Alcohol approach RT (*Z*) × alcohol cue × alcohol temporal context	0.256 ± 0.212	1.291 ± 1.237	1.450	0.2286
		Alcohol avoid RT (*Z*) × alcohol cue × alcohol temporal context	−0.315 ± 0.221	0.730 ± 1.247	2.042	0.1530
		Alcohol approach RT (*Z*) × alcohol social context	0.016 ± 0.063	1.016 ± 1.065	0.068	0.7946
		Alcohol avoid RT (*Z*) × alcohol social context	0.069 ± 0.063	1.071 ± 1.065	1.204	0.2726
		Alcohol approach RT (*Z*) × alcohol cue × alcohol social context	−0.241 ± 0.248	0.786 ± 1.281	0.942	0.3319
		Alcohol avoid RT (*Z*) × alcohol cue × alcohol social context	−0.033 ± 0.255	0.967 ± 1.29	0.017	0.8961
N450	Primary	Alcohol approach N450 (*Z*) × alcohol cue	0.017 ± 0.098	1.017 ± 1.103	0.029	0.8650
		Alcohol avoid N450 (*Z*) × alcohol cue	0.000 ± 0.102	1.00 ± 1.107	0.000	0.9962
		Alcohol approach N450 (*Z*) × alcohol physical context	−0.038 ± 0.087	0.963 ± 1.091	0.188	0.6649
		Alcohol avoid N450 (*Z*) × alcohol physical context	0.024 ± 0.087	1.025 ± 1.091	0.079	0.7794
		Alcohol approach N450 (*Z*) × alcohol cue × alcohol physical context	0.266 ± 0.256	1.305 ± 1.291	1.085	0.2975
		Alcohol avoid N450 (*Z*) × alcohol cue × alcohol physical context	−0.287 ± 0.251	0.750 ± 1.285	1.309	0.2525
		Alcohol approach N450 (*Z*) × alcohol temporal context	−0.087 ± 0.049	0.917 ± 1.051	3.097	0.0784
		Alcohol avoid N450 (*Z*) × alcohol temporal context	0.083 ± 0.050	1.087 ± 1.051	2.795	0.0945
		Alcohol approach N450 (*Z*) × alcohol cue × alcohol temporal context	0.010 ± 0.159	1.010 ± 1.172	0.004	0.9503
		Alcohol avoid N450 (*Z*) × alcohol cue × alcohol temporal context	−0.039 ± 0.162	0.962 ± 1.176	0.058	0.8102
		Alcohol approach N450 (*Z*) × alcohol social context	**−0.086 ± 0.043**	**0.918 ± 1.044**	**3.917**	**0.0478**
		Alcohol avoid N450 (*Z*) × alcohol social context	0.073 ± 0.044	1.075 ± 1.045	2.719	0.0992
		Alcohol approach N450 (*Z*) × alcohol cue × alcohol social context	0.115 ± 0.182	1.122 ± 1.199	0.399	0.5275
		Alcohol avoid N450 (*Z*) × alcohol cue × alcohol social context	−0.108 ± 0.185	0.898 ± 1.204	0.339	0.5606
	Parallel	Non-alcohol approach N450 (*Z*) × alcohol cue	0.010 ± 0.103	1.010 ± 1.109	0.009	0.9242
		Non-alcohol avoid N450 (*Z*) × alcohol cue	−0.005 ± 0.100	0.995 ± 1.105	0.002	0.9640
		Non-alcohol approach N450 (*Z*) × alcohol physical context	−0.037 ± 0.091	0.963 ± 1.095	0.169	0.6807
		Non-alcohol avoid N450 (*Z*) × alcohol physical context	0.032 ± 0.094	1.032 ± 1.099	0.114	0.7358
		Non-alcohol approach N450 (*Z*) × alcohol cue × alcohol physical context	−0.104 ± 0.244	0.901 ± 1.276	0.183	0.6688
		Non-alcohol avoid N450 (*Z*) × alcohol cue × alcohol physical context	0.035 ± 0.232	1.035 ± 1.261	0.022	0.8813
		Non-alcohol approach N450 (*Z*) × alcohol temporal context	0.024 ± 0.052	1.024 ± 1.054	0.215	0.6428
		Non-alcohol avoid N450 (*Z*) × alcohol temporal context	−0.038 ± 0.051	0.962 ± 1.052	0.572	0.4496

(Continues)

TABLE 3 (Continued)

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Non-alcohol approach N450 (*Z*) × alcohol cue × alcohol temporal context	−0.034 ± 0.164	0.967 ± 1.178	0.043	0.8362
		Non-alcohol avoid N450 (*Z*) × alcohol cue × alcohol temporal context	0.009 ± 0.159	1.009 ± 1.173	0.003	0.9574
		Non-alcohol approach N450 (*Z*) × alcohol social context	−0.044 ± 0.047	0.957 ± 1.048	0.898	0.3435
		Non-alcohol avoid N450 (*Z*) × alcohol social context	0.035 ± 0.045	1.036 ± 1.046	0.628	0.4282
		Non-alcohol approach N450 (*Z*) × alcohol cue × alcohol social context	−0.130 ± 0.182	0.878 ± 1.2	0.505	0.4772
		Non-alcohol avoid N450 (*Z*) × alcohol cue × alcohol social context	0.124 ± 0.173	1.132 ± 1.189	0.512	0.4741
		Non-beverage approach N450 (*Z*) × alcohol cue	**−0.212 ± 0.094**	**0.809 ± 1.099**	**5.039**	**0.0248**
		Non-beverage avoid N450 (*Z*) × alcohol cue	**0.232 ± 0.098**	**1.261 ± 1.103**	**5.610**	**0.0179**
		Non-beverage approach N450 (*Z*) × alcohol physical context	−0.077 ± 0.089	0.926 ± 1.093	0.754	0.3852
		Non-beverage avoid N450 (*Z*) × alcohol physical context	0.082 ± 0.095	1.086 ± 1.100	0.750	0.3866
		Non-beverage approach N450 (*Z*) × alcohol cue × alcohol physical context	0.017 ± 0.227	1.017 ± 1.255	0.006	0.9411
		Non-beverage avoid N450 (*Z*) × alcohol cue × alcohol physical context	−0.074 ± 0.243	0.929 ± 1.275	0.092	0.7616
		Non-beverage approach N450 (*Z*) × alcohol temporal context	0.006 ± 0.051	1.006 ± 1.052	0.014	0.9074
		Non-beverage avoid N450 (*Z*) × alcohol temporal context	−0.022 ± 0.051	0.979 ± 1.053	0.179	0.6726
		Non-beverage approach N450 (*Z*) × alcohol cue × alcohol temporal context	−0.054 ± 0.149	0.948 ± 1.161	0.128	0.7201
		Non-beverage avoid N450 (*Z*) × alcohol cue × alcohol temporal context	0.081 ± 0.156	1.084 ± 1.169	0.268	0.6047
		Non-beverage approach N450 (*Z*) × alcohol social context	−0.066 ± 0.046	0.936 ± 1.047	2.070	0.1502
		Non-beverage avoid N450 (*Z*) × alcohol social context	0.057 ± 0.046	1.059 ± 1.047	1.541	0.2145
		Non-beverage approach N450 (*Z*) × alcohol cue × alcohol social context	0.118 ± 0.171	1.125 ± 1.187	0.474	0.4909
		Non-beverage avoid N450 (*Z*) × alcohol cue × alcohol social context	−0.102 ± 0.179	0.903 ± 1.196	0.321	0.5710
	Sensitivity	Alcohol approach N450 (*Z*) × alcohol cue	0.057 ± 0.115	1.058 ± 1.122	0.241	0.6233
		Alcohol avoid N450 (*Z*) × alcohol cue	0.051 ± 0.126	1.053 ± 1.135	0.164	0.6856
		Alcohol approach N450 (*Z*) × alcohol physical context	−0.04 ± 0.098	0.961 ± 1.103	0.168	0.6816
		Alcohol avoid N450 (*Z*) × alcohol physical context	0.010 ± 0.111	1.01 ± 1.118	0.008	0.9272
		Alcohol approach N450 (*Z*) × alcohol cue × alcohol physical context	0.397 ± 0.303	1.488 ± 1.354	1.721	0.1896
		Alcohol avoid N450 (*Z*) × alcohol cue × alcohol physical context	−0.185 ± 0.291	0.831 ± 1.338	0.403	0.5254
		Alcohol approach N450 (*Z*) × alcohol temporal context	−0.048 ± 0.056	0.953 ± 1.058	0.715	0.3977
		Alcohol avoid N450 (*Z*) × alcohol temporal context	**0.133 ± 0.060**	**1.143 ± 1.062**	**4.899**	**0.0269**
		Alcohol approach N450 (*Z*) × alcohol cue × alcohol temporal context	0.011 ± 0.189	1.011 ± 1.208	0.003	0.9549
		Alcohol avoid N450 (*Z*) × alcohol cue × alcohol temporal context	−0.040 ± 0.198	0.961 ± 1.218	0.041	0.8400
		Alcohol approach N450 (*Z*) × alcohol social context	**−0.099 ± 0.050**	**0.906 ± 1.051**	**3.976**	**0.0461**
		Alcohol avoid N450 (*Z*) × alcohol social context	0.054 ± 0.055	1.055 ± 1.057	0.941	0.3321
		Alcohol approach N450 (*Z*) × alcohol cue × alcohol social context	0.128 ± 0.207	1.136 ± 1.229	0.384	0.5357
		Alcohol avoid N450 (*Z*) × alcohol cue × alcohol social context	−0.107 ± 0.231	0.899 ± 1.260	0.213	0.6445
P3	Primary	Alcohol approach P3 (*Z*) × alcohol cue	−0.021 ± 0.093	0.98 ± 1.098	0.049	0.8246
		Alcohol avoid P3 (*Z*) × alcohol cue	0.006 ± 0.095	1.006 ± 1.099	0.004	0.9474
		Alcohol approach P3 (*Z*) × alcohol physical context	−0.010 ± 0.075	0.990 ± 1.078	0.016	0.8989
		Alcohol avoid P3 (*Z*) × alcohol physical context	−0.006 ± 0.078	0.994 ± 1.081	0.007	0.9346
		Alcohol approach P3 (*Z*) × alcohol cue × alcohol physical context	−0.136 ± 0.220	0.872 ± 1.247	0.383	0.5358
		Alcohol avoid P3 (*Z*) × alcohol cue × alcohol physical context	0.327 ± 0.235	1.386 ± 1.265	1.928	0.1649

TABLE 3 (Continued)

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Alcohol approach P3 (*Z*) × alcohol temporal context	0.052 ± 0.044	1.054 ± 1.044	1.451	0.2284
		Alcohol avoid P3 (*Z*) × alcohol temporal context	−0.045 ± 0.045	0.956 ± 1.046	1.001	0.3170
		Alcohol approach P3 (*Z*) × alcohol cue × alcohol temporal context	0.027 ± 0.147	1.027 ± 1.158	0.033	0.8556
		Alcohol avoid P3 (*Z*) × alcohol cue × alcohol temporal context	−0.088 ± 0.155	0.916 ± 1.168	0.321	0.5710
		Alcohol approach P3 (*Z*) × alcohol social context	0.068 ± 0.039	1.07 ± 1.040	3.010	0.0828
		Alcohol avoid P3 (*Z*) × alcohol social context	−0.054 ± 0.040	0.948 ± 1.041	1.746	0.1863
		Alcohol approach P3 (*Z*) × alcohol cue × alcohol social context	−0.054 ± 0.165	0.948 ± 1.179	0.107	0.7440
		Alcohol avoid P3 (*Z*) × alcohol cue × alcohol social context	0.041 ± 0.170	1.042 ± 1.185	0.058	0.8092
	Parallel	Non-alcohol approach P3 (*Z*) × alcohol cue	−0.126 ± 0.083	0.881 ± 1.087	2.287	0.1304
		Non-alcohol avoid P3 (*Z*) × alcohol cue	0.108 ± 0.087	1.114 ± 1.090	1.555	0.2124
		Non-alcohol approach P3 (*Z*) × alcohol physical context	0.051 ± 0.083	1.052 ± 1.086	0.373	0.5414
		Non-alcohol avoid P3 (*Z*) × alcohol physical context	−0.07 ± 0.079	0.932 ± 1.083	0.777	0.3781
		Non-alcohol approach P3 (*Z*) × alcohol cue × alcohol physical context	−0.066 ± 0.209	0.936 ± 1.233	0.099	0.7529
		Non-alcohol avoid P3 (*Z*) × alcohol cue × alcohol physical context	0.233 ± 0.214	1.262 ± 1.239	1.181	0.2771
		Non-alcohol approach P3 (*Z*) × alcohol temporal context	0.016 ± 0.045	1.016 ± 1.046	0.120	0.7286
		Non-alcohol avoid P3 (*Z*) × alcohol temporal context	0.000 ± 0.044	1.000 ± 1.045	0.000	0.9967
		Non-alcohol approach P3 (*Z*) × alcohol cue × alcohol temporal context	0.057 ± 0.131	1.059 ± 1.140	0.190	0.6630
		Non-alcohol avoid P3 (*Z*) × alcohol cue × alcohol temporal context	−0.084 ± 0.133	0.919 ± 1.142	0.399	0.5277
		Non-alcohol approach P3 (*Z*) × alcohol social context	−0.025 ± 0.040	0.975 ± 1.041	0.405	0.5247
		Non-alcohol avoid P3 (*Z*) × alcohol social context	0.027 ± 0.039	1.028 ± 1.040	0.486	0.4856
		Non-alcohol approach P3 (*Z*) × alcohol cue × alcohol social context	0.004 ± 0.146	1.004 ± 1.158	0.001	0.9788
		Non-alcohol avoid P3 (*Z*) × alcohol cue × alcohol social context	−0.013 ± 0.155	0.987 ± 1.167	0.007	0.9314
		Non-beverage approach P3 (*Z*) × alcohol cue	0.115 ± 0.092	1.122 ± 1.097	1.560	0.2117
		Non-beverage avoid P3 (*Z*) × alcohol cue	−0.136 ± 0.094	0.873 ± 1.099	2.085	0.1488
		Non-beverage approach P3 (*Z*) × alcohol physical context	0.036 ± 0.084	1.037 ± 1.087	0.190	0.6626
		Non-beverage avoid P3 (*Z*) × alcohol physical context	−0.051 ± 0.089	0.951 ± 1.093	0.328	0.5667
		Non-beverage approach P3 (*Z*) × alcohol cue × alcohol physical context	0.164 ± 0.210	1.179 ± 1.234	0.611	0.4344
		Non-beverage avoid P3 (*Z*) × alcohol cue × alcohol physical context	−0.038 ± 0.223	0.963 ± 1.250	0.028	0.8667
		Non-beverage approach P3 (*Z*) × alcohol temporal context	0.070 ± 0.048	1.072 ± 1.049	2.111	0.1463
		Non-beverage avoid P3 (*Z*) × alcohol temporal context	−0.057 ± 0.049	1.048 ± 1.102	1.367	0.2424
		Non-beverage approach P3 (*Z*) × alcohol cue × alcohol temporal context	0.043 ± 0.149	1.044 ± 1.160	0.082	0.7743
		Non-beverage avoid P3 (*Z*) × alcohol cue × alcohol temporal context	−0.106 ± 0.160	0.899 ± 1.174	0.439	0.5078
		Non-beverage approach P3 (*Z*) × alcohol social context	−0.022 ± 0.042	0.978 ± 1.043	0.281	0.5958
		Non-beverage avoid P3 (*Z*) × alcohol social context	0.027 ± 0.043	1.028 ± 1.044	0.398	0.5284
		Non-beverage approach P3 (*Z*) × alcohol cue × alcohol social context	0.058 ± 0.168	1.06 ± 1.183	0.121	0.7285
		Non-beverage avoid P3 (*Z*) x alcohol cue x alcohol social context	−0.077 ± 0.177	0.926 ± 1.194	0.189	0.6641
	Sensitivity	Alcohol approach P3 (*Z*) × alcohol cue	0.027 ± 0.104	1.027 ± 1.110	0.068	0.7948
		Alcohol avoid P3 (*Z*) × alcohol cue	0.006 ± 0.118	1.007 ± 1.125	0.003	0.9561
		Alcohol approach P3 (*Z*) × alcohol physical context	0.001 ± 0.084	1.001 ± 1.087	0.000	0.9913
		Alcohol avoid P3 (*Z*) × alcohol physical context	0.051 ± 0.105	1.052 ± 1.111	0.232	0.6299
		Alcohol approach P3 (*Z*) × alcohol cue × alcohol physical context	−0.107 ± 0.250	0.899 ± 1.284	0.183	0.6688
		Alcohol avoid P3 (*Z*) × alcohol cue × alcohol physical context	0.287 ± 0.294	1.332 ± 1.342	0.949	0.3300
		Alcohol approach P3 (*Z*) × alcohol temporal context	0.032 ± 0.051	1.033 ± 1.052	0.410	0.5222

(Continues)

TABLE 3 (Continued)

Predictor	Analysis	Predictor term	*B* ± SE	Exponentiated *B* ± SE	χ^2	*P*
		Alcohol avoid P3 (*Z*) × alcohol temporal context	−0.071 ± 0.055	0.931 ± 1.057	1.685	0.1943
		Alcohol approach P3 (*Z*) × alcohol cue × alcohol temporal context	−0.063 ± 0.164	0.939 ± 1.178	0.148	0.7001
		Alcohol avoid P3 (*Z*) × alcohol cue × alcohol temporal context	−0.160 ± 0.187	0.852 ± 1.206	0.729	0.3933
		Alcohol approach P3 (*Z*) × alcohol social context	**0.105 ± 0.045**	**1.110 ± 1.046**	**5.507**	**0.0189**
		Alcohol avoid P3 (*Z*) × alcohol social context	−0.015 ± 0.049	0.985 ± 1.050	0.102	0.7495
		Alcohol approach P3 (*Z*) × alcohol cue × alcohol social context	−0.075 ± 0.183	0.927 ± 1.201	0.170	0.6800
		Alcohol avoid P3 (*Z*) × alcohol cue × alcohol social context	0.028 ± 0.214	1.029 ± 1.239	0.018	0.8944

Note: AAAT = alcohol approach avoidance task; alcohol cue = exposure to alcohol beverage cues within past 15 minutes; alcohol physical context = bar, pub or restaurant location versus all others; alcohol social context = presence of peers (e.g. friend, partner, coworker) versus all others; alcohol temporal context = weekend (Thu 6 PM–Sun 6 PM) versus all others; EMA = ecological momentary assessment; subjective craving = within-person mean of crave and urge items assessed at same occasion.

AAAT-derived measures include correct response time (RT), reverse-scored mean amplitude of the N450 component in the stimulus-locked event-related brain potential (ERP) over a frontal/central electrode cluster, and mean amplitude of the P3 component in the ERP over a parietal/occipital electrode cluster for trials requiring approach- and avoidance-like behavioral responses to images of alcoholic beverages (alcohol) or non-alcoholic beverages (non-alcohol) or non-beverage non-comestible liquids (non-beverage).

Primary analyses involved testing alcohol approach and avoid trial RT, N450 or P3 score moderation of alcohol cue × alcohol context exposure effects on momentary subjective craving intensity level outside of alcohol use episodes. Separate models were used for testing alcohol cue × alcohol physical context, alcohol cue × alcohol temporal context and alcohol cue × alcohol temporal context. Parallel analyses involved testing the same moderation effects for scores from non-alcohol or non-beverage trials in the AAAT. Sensitivity analyses involved testing effects of alcohol trial scores controlling for non-alcohol trial scores. GLMMs utilized correlated random slopes and intercepts and controlled for age, body mass index, biological sex and AUDIT total score, unless otherwise noted, and contextual factors other than the focal context in each analysis. For three models, certain covariates had to be removed to obtain convergence, as follows: age and body mass index for model testing moderation of alcohol cue × alcohol physical context effects by non-alcohol RT scores; age and AUDIT total scores for model testing moderation of alcohol cue × alcohol social context effects by alcohol RT controlling for non-alcohol RT scores; and age and AUDIT total scores for model testing moderation of alcohol cue × alcohol social context effects by alcohol P3 controlling for non-alcohol P3 scores.

Main effects and two-way interaction effects of alcohol cue and alcohol context are taken from a model that simultaneously tested all cue and context effects using the analytic subsample for RT score tests but without the latter lab-derived predictors in the model. Neither sets of effects differed substantively across models with or without different lab-derived predictors. Two-way interactions of lab-derived predictors with alcohol cue are taken from a model focused on these two-way interactions.

Unstandardized log scale beta coefficient (*B*) and standard error (SE), exponentiated *B* and *SE*, and chi-square (χ^2) statistics are presented for each predictor term alongside the *P*-value for the accompanying Type 3 Wald test (which applies to both *B* ± *SE* and χ^2). The χ^2 statistic shows the degree to which the predictor term contributes to the GLMM model fit. The exponentiated beta coefficients are interpretable as a proportional or percentage change in the outcome as a function of unit increases in the predictor term. For example, 'Primary analysis' > 'alcohol approach N450 (*Z*) × alcohol social context' is statistically significant, indicating that between-person differences in alcohol approach N450 were associated with between-person differences in the influence of an alcohol social context on craving for alcohol in non-drinking moments. For this predictor term, the exponentiated *B* indicates a (1–0.918 = 0.082 × 100% =) 8.2% *decrease* in craving response to an alcohol social context per 1 unit increase in alcohol approach N450 or alternatively an 8.2% *increase* in craving response to an alcohol social context per 1 unit *decrease* in alcohol approach N450.

Text in bold highlights effects that were statistically significant.

alcohol, non-alcohol or non-beverage avoid trials (Table S8). The sensitivity analysis indicated that the alcohol avoid but not the alcohol approach RT association was robust to controlling for non-alcohol RT associations (Table 1). Follow-ups confirmed that slower compared with faster alcohol avoid RTs predicted faster rises in eBAC and consequently higher eBACs 1 hour into drinking (Figure 1c; Table S9).

Associations with AAAT N450 amplitude

Neither the primary analysis nor the sensitivity analysis indicated that alcohol approach or alcohol avoid N450s were associated with eBAC dynamics, but the parallel analyses indicated that non-beverage approach and non-beverage avoid N450s were (Table 1). Simple slopes analysis confirmed that smaller non-beverage approach N450s or larger non-beverage avoid N450s were associated with a faster rise in eBAC within episodes (Table S10).

Associations with AAAT P3 amplitude

The primary analysis indicated that alcohol approach and alcohol avoid P3s were associated with eBAC dynamics (Table 1). Inspection of the model-estimated eBAC time courses revealed that larger compared with smaller alcohol approach P3s predicted a faster rise in eBAC, resulting in higher eBACs 1 hour into drinking (Figure 2a). This also appeared to be the case for smaller compared with larger alcohol avoid P3s, but differences were not statistically significant (Figure 2b).

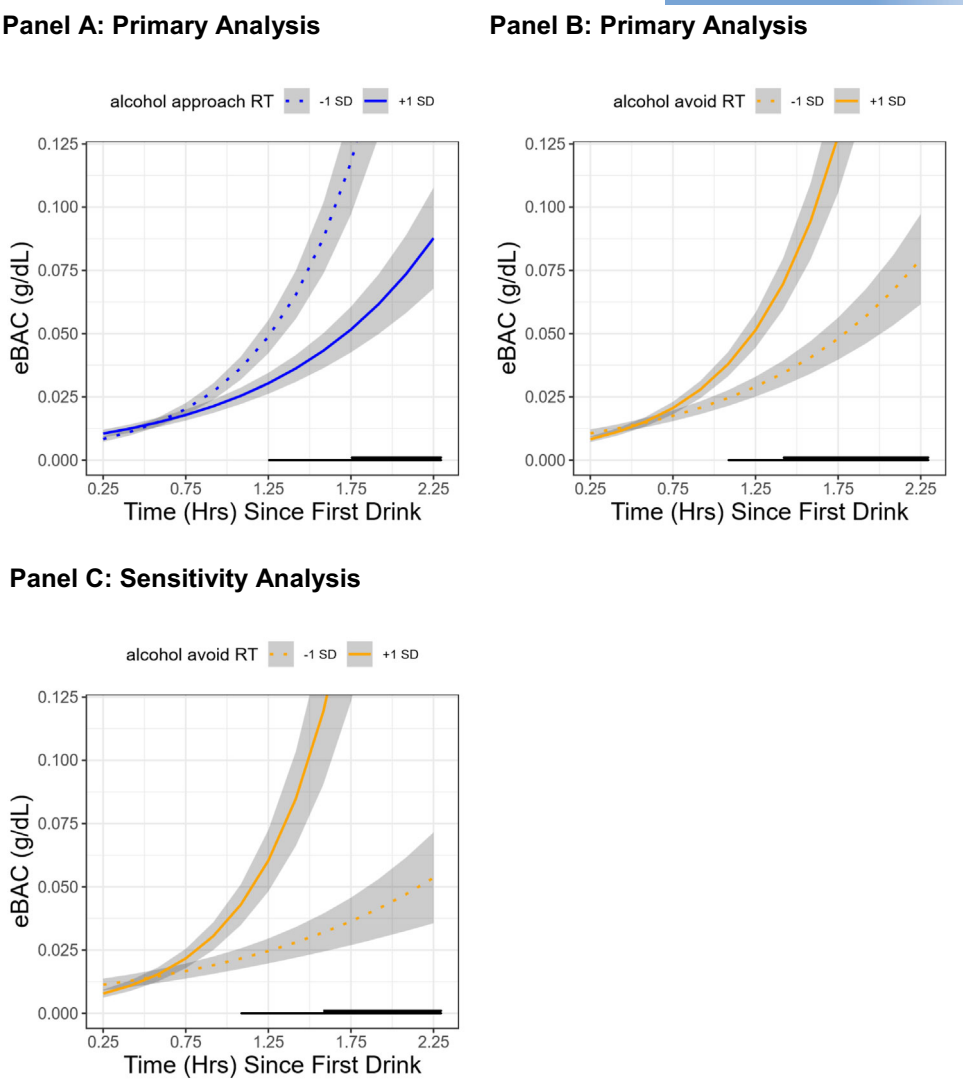

FIGURE 1 Alcohol cue approach or avoid response time (RT) scores predicting alcohol consumption (estimated blood alcohol concentration, eBAC) dynamics during alcohol use episodes. *Note*. Covariate-adjusted, model-predicted M ± SE while holding alcohol approach RT (a) or alcohol avoid RT (b, c) at *Z* = +1 SD or −1 SD are presented. Estimates in (a) and (b) are derived from the primary analysis model, which simultaneously estimates associations with alcohol approach and avoid RT. Estimates in (c) are derived from the sensitivity analysis model, which simultaneously estimates associations with alcohol approach and avoid RT as well as non-alcohol approach and avoid RT. All models were based on data from *n* = 268 participants, *n* = 800 alcohol use episodes and *n* = 1620 moments of observation. A single black solid line segment near the *x*-axis in any panel indicates areas of significant (uncorrected $P < 0.05$) difference between the two curves identified via pairwise comparisons conducted on the log scale predicted values at 13 specific timepoints spanning the *x*-axis. A second black solid line near the *x*-axis in any panel indicates areas of significant difference between the curves that survive Bonferroni correction. (a) Persons who exhibited faster response speed on approach-congruent alcohol beverage cue trials, consistent with activation of an alcohol cue approach-like behavioral tendency, are represented by negative *Z*-scores (e.g. *Z* = −1 SD) whereas persons who exhibited a slower response speed on approach-congruent alcohol beverage cue trials, consistent with activation of an alcohol cue avoidance-like behavioral tendency, are represented by positive *Z*-scores (e.g. *Z* = +1 SD). (b, c) Persons who exhibited faster response speed on avoidance-congruent alcohol beverage cue trials, consistent with activation of an alcohol cue avoidance-like behavioral tendency, are represented by negative *Z*-scores (e.g. *Z* = −1 SD), whereas persons who exhibited slower response speed on avoidance-congruent alcohol beverage cue trials, consistent with activation of an alcohol cue approach-like behavioral tendency, are represented by positive *Z*-scores (e.g. *Z* = +1 SD).

Nonetheless, simple slopes analysis confirmed a faster rise in eBAC within episodes with larger alcohol approach P3s or smaller alcohol avoid P3s (Table S11). The sensitivity analysis indicated that alcohol approach and avoid P3 associations were robust to controlling for non-alcohol P3 associations (Table 1). Follow-up on these model-estimated eBAC time courses confirmed these impressions (Figure 2c,d), but simple slopes analysis suggested that only alcohol approach P3 robustly predicted a faster rise in eBAC within episodes (Table S12).

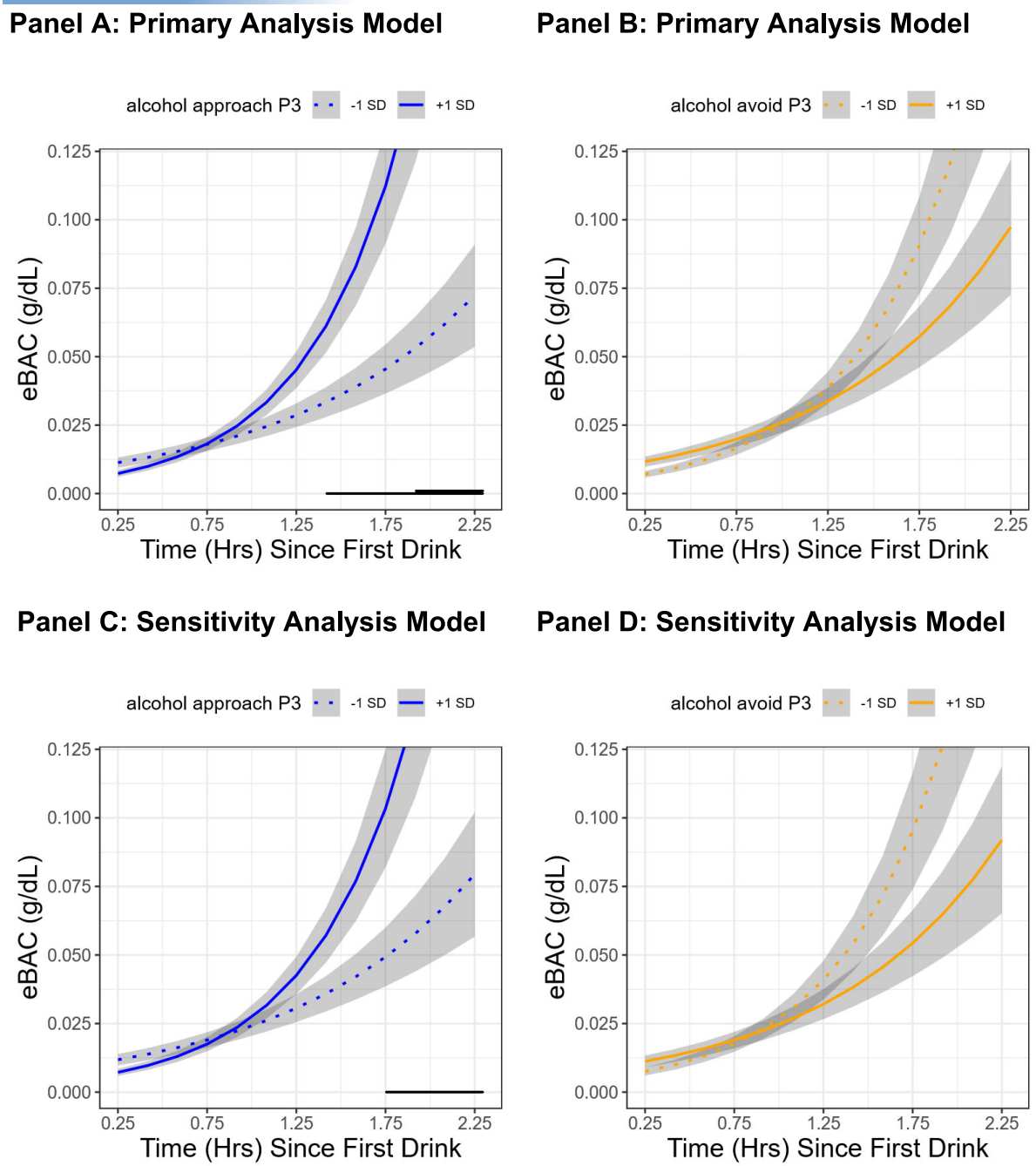

FIGURE 2 Alcohol cue approach or avoid P3 scores predicting alcohol consumption (estimated blood alcohol concentration, eBAC) dynamics during alcohol use episodes. *Note*. Covariate-adjusted, model-predicted M ± SE while holding alcohol approach P3 amplitude (a, c) or alcohol avoid P3 amplitude (b, d) at Z = +1 SD or −1 SD are presented. Estimates in (a) and (b) are derived from the primary analysis model, which simultaneously estimates associations with alcohol approach and avoid P3 amplitude. Estimates in (c) and (d) are derived from the sensitivity analysis model, which simultaneously estimates associations with alcohol approach and avoid P3 amplitude as well as non-alcohol approach and avoid P3 amplitude. All models were based on data from n = 233 participants, n = 673 alcohol use episodes and n = 1378 moments of observation. A single black solid line segment near the x-axis in any panel indicates areas of significant (uncorrected $P < 0.05$) difference between the two curves identified via pairwise comparisons conducted on the log scale predicted values at 13 specific timepoints spanning the x-axis. A second black solid line near the x-axis in any panel indicates areas of significant difference between the curves that survive Bonferroni correction. (a, c) Persons who attended less to the cue on approach-congruent alcohol beverage cue trials, consistent with dampened incentive salience, are represented by negative Z-scores (e.g. Z = −1 SD), whereas persons who attended more to the cue on approach-congruent alcohol beverage cue trials, consistent with amplified incentive salience, are represented by positive Z-scores (e.g. Z = +1 SD). (b, d) Persons who attended less to the cue on avoidance-congruent alcohol beverage cue trials, consistent with dampened incentive salience, are represented by negative Z-scores (e.g. Z = −1 SD), whereas persons who attended more to the cue on avoidance-congruent alcohol beverage cue trials, consistent with amplified incentive salience, are represented by positive Z-scores (e.g. Z = +1 SD).

Alcohol craving trajectory during alcohol use episodes

Associations with AAAT behavioral RT

The primary analysis indicated that alcohol approach RT, but not alcohol avoid RT, was associated with subjective craving dynamics (Table 2). Inspection of the model-estimated trajectories indicated different patterns of change in craving level over time within episodes: increases for persons with faster alcohol approach RTs and decreases for those with slower alcohol approach RTs (Figure 3a). Simple slopes analysis confirmed the difference in these diverging slopes (Table S13). The sensitivity analysis indicated that the alcohol approach RT association was robust to controlling for non-alcohol RT associations (Table 2). Inspection of model-estimated within-episode craving trajectories from the sensitivity analysis revealed accentuated alcohol approach RT score-related differences: craving level increased from an initial nadir for those with faster alcohol approach RTs, whereas it decreased from an initial zenith for those with slower alcohol approach RTs (Figure 3b). Simple slopes analysis confirmed the difference in these diverging slopes (Table S14).

Associations with AAAT N450 amplitude

The primary analysis indicated that alcohol approach N450, but not alcohol avoid N450, was associated with subjective craving dynamics (Table 2). Inspection of the model-estimated trajectories suggested divergent patterns of change in craving level over time within episodes: craving increased for persons with smaller alcohol approach N450s and decreased for those with larger alcohol approach N450s (Figure 4a). Simple slopes analysis confirmed the difference in these diverging slopes (Table S15). The sensitivity analysis indicated that the alcohol approach N450 association was robust to controlling for non-alcohol N450 associations (Table 2), and follow-up analyses confirmed the impressions from follow-up on the primary analysis (Figure 4b; Table S16).

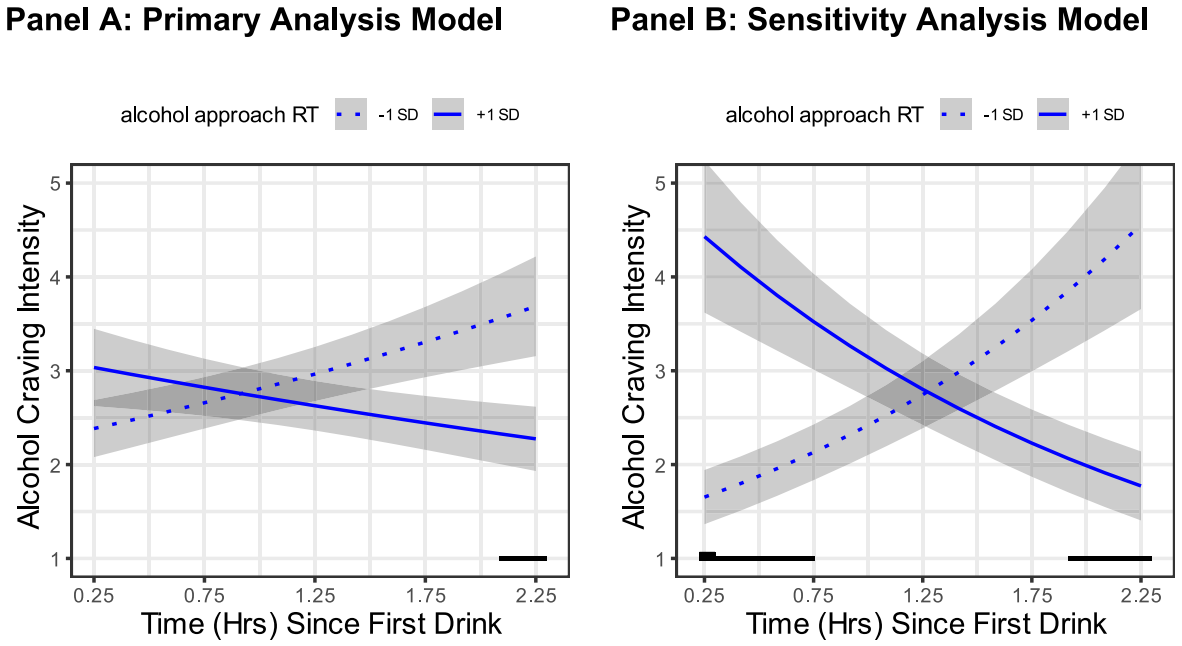

FIGURE 3 Alcohol cue approach response time (RT) scores predicting subjective craving dynamics during alcohol use episodes. *Note*. Covariate-adjusted, model-predicted M ± SE while holding alcohol approach RT at Z = +1 SD or −1 SD are presented. Estimates in (a) are derived from the primary analysis model, which simultaneously estimates associations with alcohol approach and avoid RT. Estimates in (b) are derived from the sensitivity analysis model, which simultaneously estimates associations with alcohol approach and avoid RT as well as non-alcohol approach and avoid RT. All models were based on data from n = 250 participants, n = 736 alcohol use episodes and n = 1494 moments of observation. All models included a grand mean centered covariate representing between-person differences in baseline or tonic alcohol craving level (defined as each person's mean alcohol craving intensity across all moments outside drinking episodes in which beverage cues were absent). A single black solid line segment near the x-axis in any panel indicates areas of significant (uncorrected $P < 0.05$) difference between the two curves identified via pairwise comparisons conducted on the log scale predicted values at 13 specific timepoints spanning the x-axis. A second black solid line near the x-axis in any panel indicates areas of significant difference between the curves that survive Bonferroni correction. Persons who exhibited faster response speed on approach-congruent alcohol beverage cue trials, consistent with activation of an alcohol cue approach-like behavioral tendency, are represented by negative Z-scores (e.g. Z = −1 SD), whereas persons who exhibited slower response speed on approach-congruent alcohol beverage cue trials, consistent with activation of an alcohol cue avoidance-like behavioral tendency, are represented by positive Z-scores (e.g. Z = +1 SD).

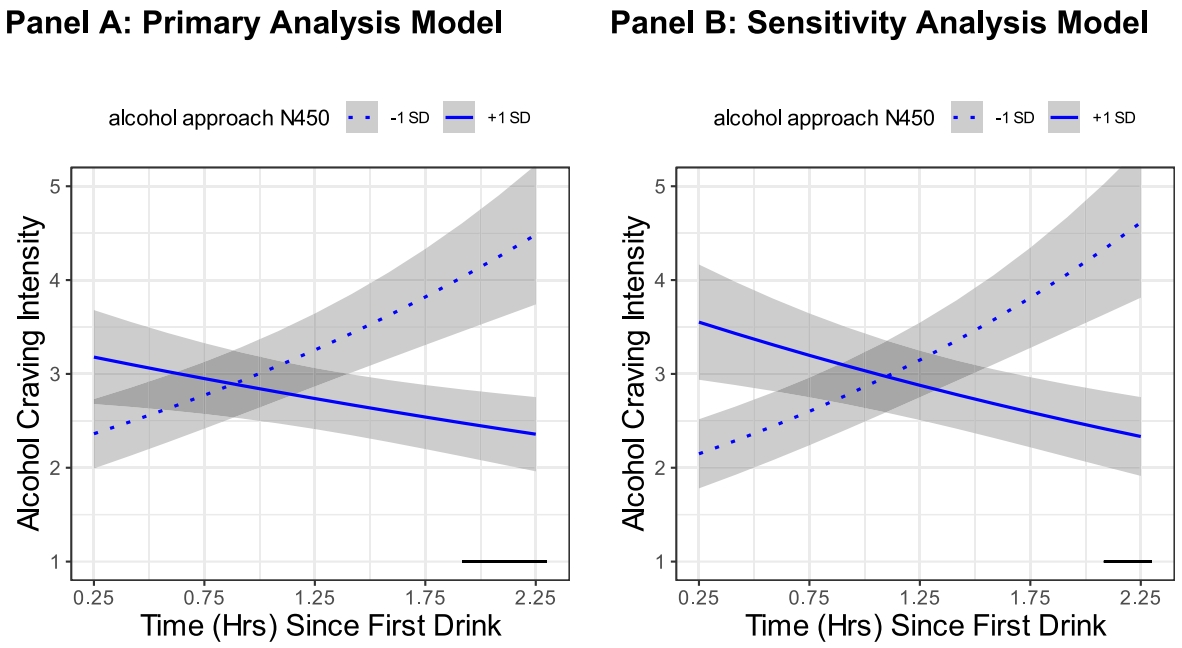

FIGURE 4 Alcohol cue approach N450 scores predicting subjective craving dynamics during alcohol use episodes. *Note.* Covariate-adjusted, model-predicted M ± SE while holding alcohol approach N450 amplitude (a, b) at $Z = +1$ SD or −1 SD are presented. Estimates in (a) are derived from the primary analysis model, which simultaneously estimates associations with alcohol approach and avoid N450. Estimates in (b) are derived from the sensitivity analysis model, which simultaneously estimates associations with alcohol approach and avoid N450 as well as non-alcohol approach and avoid N450. For ease of interpretation, N450 amplitudes were reverse-scored prior to inclusion in the models (viz., if a person's alcohol approach N450 amplitude score was −8 μV, it was reverse-scored as +8 μV for inclusion in the model). All models were based on data from $n = 218$ participants, $n = 617$ alcohol use episodes and $n = 1268$ moments of observation. All models included a grand mean centered covariate representing between-person differences in baseline or tonic alcohol craving level (defined as each person's mean alcohol craving intensity across all moments outside drinking episodes in which beverage cues were absent). A single black solid line segment near the *x*-axis in any panel indicates areas of significant (uncorrected $P < 0.05$) difference between the two curves identified via pairwise comparisons conducted on the log scale predicted values at 13 specific timepoints spanning the *x*-axis. No differences between curves survive Bonferroni correction. Persons who exhibited more internal conflict on approach-congruent alcohol beverage cue trials, consistent with activation of alcohol avoidance motivation, are represented by positive *Z*-scores (e.g. $Z = +1$ SD), whereas persons who exhibited less internal conflict on approach-congruent alcohol beverage cue trials, consistent with activation of alcohol approach motivation, are represented by negative *Z*-scores (e.g. $Z = -1$ SD).

Associations with AAAT P3 amplitude

No significant associations were detected (Table 2).

Alcohol craving outside alcohol use episodes

Associations with AAAT behavioral RT

No significant associations were detected (Table 3).

Associations with AAAT N450 amplitude

Primary analyses indicated that alcohol approach N450 was associated with the effect of alcohol-related social context on subjective craving outside of drinking episodes (Table 3). Follow-up found no significant simple slopes or slope difference (Table S17). Parallel analyses indicated that non-beverage approach and non-beverage avoid N450s were associated with the effect of incidental alcohol cue exposure outside of drinking episodes (Table 3). Follow-up found a significant negative simple slope of non-beverage approach N450 on craving following cue exposure and a significant positive simple slope of non-beverage avoid N450 on craving following cue exposure (Table S18). Sensitivity analyses indicated that the alcohol approach N450 association with the effect of alcohol-related social context was robust to controlling for non-alcohol N450 associations, and detected an alcohol avoid N450 association with the effect of alcohol-related temporal context (Table 3). Follow-up on the former confirmed impressions from the follow-up on the primary analysis (Table S19), whereas follow-up on the latter found no significant simple slopes or slope difference (Table S20).

Associations with AAAT P3 amplitude

No significant associations were detected in primary or parallel analyses, but sensitivity analyses detected an alcohol approach P3

association with the effect of alcohol-related social context on subjective craving outside of drinking episodes (Table 3). Follow-up found no significant simple slopes or slope difference (Table S21).

DISCUSSION

The present study examined the extent to which laboratory-based indices of putatively distinct facets of IS sensitization are differentially associated with alcohol craving and consumption in drinkers' natural environments. Recent theorizing on the role of IS in addiction has pointed to heterogeneity in the neurobehavioral pathways through which IS contributes to substance use [7]. We identified three constructs derived from the AAAT— approach behavior (RT), motivated attention (P3 amplitude) and approach–avoidance motivational conflict (N450 amplitude)—that map onto these neurobehavioral pathways and that should, therefore, differentially relate to specific aspects of substance use. Consistent with this idea, individual differences in these AAAT-derived constructs were found to be relatively independent and to predict different aspects of emerging adults' real-world alcohol craving and consumption. Overall, the findings support the theorized heterogeneity of IS-related neurobehavioral pathways as well as the clinical utility of laboratory analogs of these pathways in predicting aspects of real-world alcohol use [66, 67].

Tests of predictions derived from theorized IS-related risk pathways

Both predictions from the *hyper-direct* pathway were supported. First, a tendency to approach alcohol cues, reflected by either a faster RT on approach trials or a slower RT on avoid trials, was related to faster and heavier consumption within drinking episodes (Figure 1). Second, stronger motivated attention to alcohol cues, reflected in larger P3 amplitudes elicited on approach trials, robustly predict faster and heavier consumption within drinking episodes (Figure 2). As originally described by Cofresí *et al.* [7], the *hyper-direct* pathway captures the ability of attention/approach responses elicited by alcohol cues to facilitate instrumental alcohol seeking/consumption actions, namely, Pavlovian-to-instrumental transfer (PIT) effects on alcohol self-administration [68, 69]. The present findings converge with emerging evidence suggesting that between-person differences in lab-based measures of PIT effects have utility in alcohol use prognosis [70, 71].

In contrast, neither prediction from the *direct* pathway was supported. Motivated attention to alcohol cues in the lab (P3 amplitude) was not significantly associated with either craving during drinking or craving during non-drinking moments following alcohol cue exposure in alcohol-related contexts. These null results are consistent with other reports of failures to find associations between task-derived indicators of attention to drug-related cues and drug craving [27, 72] or consumption [73, 74] in the natural environment. The association between attention and craving may simply be weak [75, 76]. Alternatively, it may depend on measuring attention bias in laboratory contexts that more closely resemble real-world settings. For example, motivated attention to alcohol cues following a priming dose of alcohol—potentially in a simulated bar setting—might better capture the variability that relates to attention and craving during real-world drinking [77]. Or, if attentional bias, like craving, fluctuates with motivational states, then demonstrating their association might require measuring both constructs more closely in time [76]. In any case, given the centrality of craving to IS theory [5, 6], additional research into the *direct* pathway is warranted.

The primary prediction from the *indirect* pathway was also not supported. Motivational conflict related to incongruent alcohol cue-activated versus task-based behavioral goals (N450 amplitude) was not related to craving during non-drinking moments following alcohol cue exposure in alcohol use-incongruent contexts. This null result calls into question accounts linking alcohol craving with different forms of conflict, including approach–avoidance conflict [12, 78].

Yet, incentive-motivational conflicts abound during drinking episodes (e.g. the desire for another drink vs the goal to moderate intake), and such conflicts could also have implications for craving. Thus, associations between laboratory indices of approach–avoidance conflict and craving dynamics during drinking episodes can also support the plausibility of the *indirect* pathway. Here, RT and N450 measures from alcohol approach trials were robustly related to the topography of alcohol craving during drinking: persons exhibiting slower RTs or larger N450s on alcohol approach trials, both consistent with approach conflict, exhibited decreasing craving while drinking, whereas persons exhibiting faster RTs or smaller N450s on such trials exhibited increasing craving while drinking (Figures 3 and 4). Thus, whereas lab-based analogs of alcohol approach–avoidance conflict may have limited utility for understanding the influence of alcohol cues and contexts on craving outside drinking episodes, these measures may distinguish individuals whose craving satiates during drinking episodes from individuals whose craving sensitizes during drinking episodes. This distinction could prove important for understanding different consumption phenotypes associated with the *indirect* pathway as well as for identifying drinkers for whom craving intensity is likely to determine heaviness of use.

Implications for the role of IS in the etiology of alcohol/drug use disorders

A growing body of evidence across species suggests that only some individuals ('sign-trackers') attribute IS to reward-predictive cues [8, 79]. Hence, substantial between-person variability is expected in the relevance of IS attribution and its sensitization to the etiology of drug use disorders. Yet, as traditionally conceived, IS attribution is a dynamic, within-subject process that manifests consistently across multiple sub-mechanisms of reactivity to reward-predictive cues. The present work goes further by demonstrating between-subject variability in the expression of IS attribution on sub-mechanisms of cue reactivity. That is, different lab-based measures of IS attribution to alcohol

cues rank-ordered individuals differently and correlated with different EMA-based measures of alcohol craving and consumption. An important implication of this demonstration is that understanding the relevance of IS attribution and its sensitization to the etiology of alcohol/drug use disorders requires careful consideration of how different sub-mechanisms of alcohol/drug cue reactivity may influence different aspects of alcohol/drug use behavior in the natural environment. In other words, because the different pathways theorized to link IS sensitization to substance use manifest on distinct sub-mechanisms, advancing our understanding of IS in addictive behaviors is not a matter of whether IS attribution (as a 'monolith') is relevant to a person's substance use, but rather the extent to which specific sub-mechanisms and related risk pathways contribute to that person's substance use.

Limitations

The primary findings should be interpreted in light of the limitations of the study and some unanticipated ancillary findings. With respect to limitations, the generalizability is constrained by the sample characteristics (e.g. socio-demographics, short life histories of alcohol use, low rates of other substance use) and limitations of our EMA protocol (e.g. under-sampling of drinking episodes across study days, under-sampling of behavior within drinking episodes). Moreover, while eBACs are an improvement over simple drink counts, because eBACs capture not only how many drinks are consumed over a period of time but also what those drinks may mean in terms of expected level of intoxication and risk for related harms for a given person, similar limitations apply (e.g. inaccurate counts, uncontrolled stomach contents) [25]. Future studies may benefit from the objective measurement of alcohol consumption using portable miniaturized breath alcohol concentration (BrAC) meters [80] or transdermal sensors [81]. Furthermore, although none of the alcohol cue reactivity sub-mechanisms (approach, motivated attention, conflict) was found to be related to cue-related craving outside of drinking episodes, these null results do not rule out the existence of such associations. Statistical tests here may have been underpowered given: the expected small effects; the low levels of craving in non-drinking relative to drinking moments (Table S22); the uncontrolled frequency of alcohol cue/context exposure; the reliance on self-report of alcohol cue/context exposure; and the failure to assess person-specific cues/contexts. Model diagnostics also indicated potential multi-collinearity issues and thus reduced power for all sensitivity analysis models. On a related note, statistical tests of certain cross-level moderation effects were relegated to the supporting information and are not considered further because the relevant random slopes could not be included in the models without causing convergence issues, and the absence of these random slopes would be expected to inflate Type 1 error for the specific moderation tests [63–65]. Thus, the extent to which individual differences captured in AAAT-derived neurobehavioral measures moderate the quadratic components of change in alcohol craving and consumption dynamics during real-world drinking remains an open question, as does the possibility that these individual differences moderate the effects of incidental alcohol cue exposure while under the influence of cannabis or tobacco on craving for alcohol outside of drinking episodes. Future studies focused specifically on these questions are warranted. Additionally, it is worth emphasizing that the use of drugs other than alcohol was not a focus of the study. Studies examining the predictive utility of multiple IS domain measures with respect to real-world craving and consumption of drugs other than alcohol are needed. Finally, this study assumes that alcohol approach–avoidance measures index relatively stable between-person differences, yet recent work on food approach–avoidance measures [82, 83] supports the idea that alcohol approach–avoidance measures may fluctuate with motivational states [76], and thus there is a need for ambulatory assessment approaches that can overcome previously identified challenges to measuring approach-avoidance biases in the natural environment [74].

With respect to ancillary findings, the parallel analyses using measures derived from 'control' trials in the AAAT produced some perplexing results. Slower RTs on non-alcohol and non-beverage cue avoid trials predicted faster alcohol consumption during drinking episodes. The finding that slower RTs on avoid trials generally appeared to predict faster consumption during drinking episodes suggests a role for non-specific appetitive-motivational reactivity in shaping real-world alcohol use. Further evidence consistent with this idea comes from parallel analysis findings that smaller N450 amplitudes (less incentive conflict) during non-beverage approach trials or larger N450 amplitudes (more incentive conflict) during non-beverage avoid trials predicted faster alcohol consumption rates during drinking episodes as well as increased craving following alcohol cue exposure in non-drinking moments. The influence of non-specific appetitive-motivational reactivity could reflect the characteristics of the study participants, such as psychosocial development stage (early emerging adulthood) [84], which is marked by increased approach tendencies generally and addiction vulnerability [85], or early-stage addiction, which is characterized by appetitive motivation and positive reinforcement [2]. It will be important for future studies to disambiguate IS and its sub-mechanisms from conceptually related dispositions or traits that may also contribute to substance use, especially as present analyses controlled for only a limited number of such potential between-person third variables.

CONCLUSION

Pending validation in an independent sample, the present findings suggest that the IS of alcohol/drug may influence alcohol/drug seeking (including craving) and taking through distinct behavioral risk pathways in different people. More work is needed to determine the specificity of the proposed behavioral risk pathways as well as to examine the possibility that these pathways or their sub-mechanisms (motivated attention to cues, behavioral approach impulses and approach–avoidance motivational conflict) may cooperate or compete to shape alcohol/drug seeking and taking cycles in daily life.

AUTHOR CONTRIBUTIONS

Roberto U. Cofresí: Conceptualization (equal); data curation (lead); formal analysis (lead); investigation (supporting); software (supporting); visualization (lead); writing—original draft (lead); writing—review and editing (lead). **Sandie Keerstock:** Data curation (supporting); investigation (supporting); project administration (supporting); visualization (supporting); writing—original draft (supporting); writing—review and editing (supporting). **Casey B. Kohen:** Data curation (supporting); software (supporting); writing—review and editing (supporting). **Thomas M. Piasecki:** Conceptualization (equal); data curation (supporting); funding acquisition (equal); methodology (equal); resources (supporting); software (supporting); supervision (supporting); writing—review and editing (supporting). **Bruce D. Bartholow:** Conceptualization (equal); funding acquisition (lead); methodology (equal); resources (lead); supervision (lead); writing—review and editing (supporting).

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DECLARATION OF INTERESTS

None to declare.

DATA AVAILABILITY STATEMENT

Materials, data and analysis code are available from the corresponding author, upon reasonable request.

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