


Affective dynamics surrounding craving, non-heavy alcohol use and binge drinking in female patients with alcohol use disorder and controls: An experience sampling method study

Nicolas Leenaerts^{1,2}  | Thomas Vaessen^{2,3,4} | Stefan Sunaert^{1,5} |
Jenny Ceccarini^{1,6} | Elske Vrieze^{1,2}

¹Leuven Brain Institute, KU Leuven, Leuven, Belgium

²Mind-body Research, Biomedical Sciences Group, KU Leuven, Leuven, Belgium

³Center for Contextual Psychiatry, Biomedical Sciences Group, KU Leuven, Leuven, Belgium

⁴Center for eHealth and Wellbeing Research, Department of Psychology, Health and Technology, University of Twente, Enschede, The Netherlands

⁵Department of Radiology, University Hospitals Leuven and Department of Imaging and Pathology, KU Leuven, Leuven, Belgium

⁶Department of Nuclear Medicine and Molecular Imaging, Department of Imaging and Pathology, KU Leuven, Leuven, Belgium

Correspondence

Nicolas Leenaerts, Mind-Body Research, Leuven Brain Institute, Biomedical Sciences Group, KU Leuven, Herestraat 49, 3000 Leuven, Belgium.
Email: nicolas.leenaerts@kuleuven.be

Funding information

Special Research Fund KU Leuven C1 grant, Grant/Award Number: ECA-D4671-C14/18/096 (to E.V.); J.C. served as a PhD Scholarship for N.L.; postdoc grant from Fonds Wetenschappelijk Onderzoek (FWO, Grant/Award Numbers: 12R1619N and 1243620N (to J.C. and T.V.)).

Abstract

Background and Aims: Studies show that higher levels of positive affect (PA) and lower levels of negative affect (NA) are related to craving and alcohol consumption at a daily level in men, but little is known on these associations at a momentary level, and whether they are present in women. This study measured the dynamics of within-person NA and PA surrounding craving, non-heavy alcohol use and binge drinking in women with alcohol use disorder (AUD) and female controls without AUD.

Methods: 53 female patients with AUD and 75 female controls, all recruited in Belgium, were included in an experience sampling study where they reported on momentary NA, PA, craving and alcohol use in daily life over a period of 12 months. Assessments occurred eight times a day on Thursdays, Fridays and Saturdays in seven bursts of three weeks.

Results: Within-person NA at a previous assessment (t_{-1}) predicted craving at the current assessment (t_0) in patients with AUD in a positive linear [$\beta = 0.043$; 95% confidence interval (CI) = 0.002, 0.057; $P = 0.041$] and quadratic fashion ($\beta = 0.034$; CI = 0.011, 0.057; $P = 0.004$). Within-person PA at t_{-1} predicted craving at t_0 in patients with AUD with a positive quadratic relation ($\beta = 0.042$; CI = 0.08, 0.065; $P < 0.001$). Within-person NA at t_{-1} negatively predicted non-heavy alcohol use at t_0 in a linear fashion in controls ($\beta = -0.495$; CI = -0.677, -0.312; $P < 0.001$) and patients with AUD ($\beta = -0.276$; CI = -0.421, -0.132; $P < 0.001$). Within-person PA at t_{-1} significantly predicted non-heavy alcohol use at t_0 with a positive linear term ($\beta = 0.470$; CI = 0.329, 0.610; $P < 0.001$) in controls, but with a positive linear term ($\beta = 0.399$; CI = 0.260, 0.454; $P < 0.001$) and a positive quadratic term ($\beta = 0.203$; CI = 0.060, 0.347; $P = 0.003$) in patients with AUD. Within-person NA at t_{-1} predicted binge drinking at t_0 in patients with AUD with a significant quadratic term ($\beta = 0.236$; CI = 0.060, 0.412; $P = 0.008$), but not for controls. Within-person PA at t_{-1} predicted binge drinking at t_0 in patients with AUD with a significant quadratic term ($\beta = 0.378$; CI = 0.215, 0.542; $P < 0.001$), and this was also the case for controls ($\beta = 0.487$; CI = 0.158, 0.770; $P < 0.001$). Non-heavy alcohol use at t_0 predicted lower levels of NA at t_{+1} in both patients with AUD ($\beta = -0.161$; SE = 0.044; CI = -0.248, 0.074; $P = 0.001$) and controls ($\beta = -0.114$; CI = -0.198, -0.029; $P = 0.010$). Non-heavy alcohol use at t_0 also predicted higher levels of PA at t_{+1} in both patients

with AUD ($\beta = 0.181$; CI = 0.088, 0.274; $P < 0.001$) and controls ($\beta = 0.189$; CI = 0.101, 0.278; $P < 0.001$).

Conclusions: The momentary relation between affect and craving or alcohol use seems to be non-linear in female patients with alcohol use disorder, whereby a worse mood predicts subsequent alcohol use, though more for binge drinking than for non-heavy alcohol use.

KEYWORDS

alcohol use, alcohol use disorder, binge drinking, craving, ecological momentary assessment, experience sampling method, negative affect, positive affect

INTRODUCTION

Alcohol use disorder (AUD) is defined as a maladaptive pattern of alcohol use that leads to significant impairments or distress [1]. This pattern of alcohol use has far-reaching consequences on individuals, their surroundings and society as a whole [2–4]. Additionally, treating AUD is challenging as only 17.3% of patients receive treatment within a given year, and as up to 60% of those who receive treatment do not achieve remission [5, 6]. Crucially, literature suggests that women progress more rapidly from their first alcohol consumption to AUD than men, experience worse health outcomes and are less likely to seek treatment [7–9]. Therefore, there is an urgent need to better understand the triggers for alcohol use in female patients with AUD.

Across different theories, momentary changes in both negative affect (NA) and positive affect (PA) are thought to be important triggers of alcohol use, with craving being a mediator of these relations [10–13]. First, NA is characterized as a feeling of ‘subjective distress and unpleasurable engagement’ that includes several negative emotions such as anger, anxiety, guilt, loneliness or sadness [14]. Theories such as the tension reduction theory, the stress-response dampening model and the affective processing model of addiction propose that higher levels of NA cause patients with AUD to consume alcohol, and that a subsequent reduction in NA has a reinforcing effect on future alcohol use [10, 15, 16]. Indeed, studies have found that experimentally inducing NA can lead to more alcohol use across patients with AUD and controls (i.e. individuals without AUD), and that alcohol consumption can lower NA [17, 18]. Furthermore, patients with AUD report more often that they drink to cope with NA than controls, and relapse in patients with AUD is predicted by higher levels of NA [19, 20]. Importantly, studies show that NA could play a greater role in women, because they are more likely to drink alcohol when experiencing high levels of NA than men [21].

Second, PA reflects the extent to which somebody experiences positive emotions such as feeling alert, excited and satisfied [14]. Theories such as the motivational model of alcohol use posit that patients not only drink alcohol to cope with NA, but also drink alcohol to enhance PA, and that this behavior is influenced by factors such as social enhancement expectancies and sensation seeking [11, 12]. For example, an individual could experience lower levels of PA (i.e. feeling bored and inactive) and drink alcohol to feel more excitement [11, 12]. Indeed, patients with AUD who report lower PA also report higher

levels of craving for alcohol [22]. Furthermore, studies suggest that higher levels of PA could also lead to alcohol use by making individuals more attentive to rewards and more likely to approach them [23, 24]. Experimentally inducing PA can indeed lead to more alcohol use and drinking alcohol can increase PA [25–27]. Studies also show that women drink in response to PA at a level comparable to men, but that they could be more vulnerable to the effects of alcohol on PA [28, 29]. Importantly, it has been suggested that patients with AUD initially consume alcohol for its positive reinforcing effects (i.e. increasing PA), but that a shift occurs over time, after which alcohol is consumed for its negative reinforcing effects (i.e. decreasing NA) [30, 31]. In line with this hypothesis, individuals who experience more rewarding effects from alcohol use have a higher risk of developing AUD [32].

Third, craving can be defined as ‘an intense and conscious desire for a specific substance’, although the precise definition of craving has been the topic of discussion [13, 33]. Despite this debate, several conditioning-based, cognitive, psychobiological, and motivational models have been developed, which propose that deviations in the average levels of NA and PA can increase craving [13]. Across patients and controls, studies do report that experimentally inducing NA can lead to more craving for alcohol, and that experiencing more craving in response to NA is predictive of relapse in patients with AUD [18, 34, 35]. Compared to men, women show a stronger relation between craving and depressive symptoms and a stronger increase in craving in response to beverage cues after the induction of NA, suggesting women may be more sensitive to the effects of NA on craving [36, 37]. Additionally, there is little research on the link between PA and craving, although one study finds that experimentally inducing PA can increase craving in patients with AUD [27].

Taken together, numerous studies show that NA and PA play a significant role in alcohol use and that craving could mediate this relation. However, these studies have typically been performed in a laboratory setting or have used retrospective questionnaires, which focus on between-person differences and can suffer from limitations such as a lack of ecological validity or recall bias [38]. Therefore, there has been an increasing interest in studying the impact of within-person deviations of NA and PA on craving and alcohol use in real life using the experience sampling method (ESM), also known as ecological momentary assessment, whereby participants repeatedly report their mood, behavior and context in real-time [38]. Importantly, a meta-analysis of 69 ESM studies finds that higher levels of PA are related to

alcohol use and binge drinking in daily life, but that this is not the case for NA and alcohol use or binge drinking, which calls into question whether deviations in average NA levels actually cause individuals to drink alcohol in daily life [39].

However, these ESM studies have several limitations. First, a large number of ESM studies investigate the relation between affect and alcohol use on a daily level, meaning that they aggregate several measurements within a day. This could be an issue if the relation between NA and alcohol use exists on a smaller timescale. If so, it might be better suited to investigate whether higher or lower than average levels of NA and PA within a person at one moment predict craving and alcohol use at the next moment. Indeed, findings in the literature suggest that the relation between affect and alcohol use differs across levels. Namely, after analyzing the same dataset, one study looking at the daily level reports that higher PA is related to greater alcohol use, whereas another study looking at the momentary level found that lower PA is associated with subsequent alcohol consumption [40, 41]. However, this is not the only factor influencing the relation between affect and alcohol use, as studies investigating the role of momentary NA report conflicting results. Namely, one study finds that higher levels of anxiety predict subsequent alcohol use, whereas another study reports that lower levels of NA predict future alcohol consumption [41, 42]. Second, almost all studies have been performed in individuals without a diagnosis of AUD. This could have influenced the findings as the relation between NA and alcohol use is hypothesized to be the strongest in patients with AUD [20]. Indeed, one of the few ESM studies in patients with AUD reports that higher levels of NA do predict relapse in daily life [43]. Third, a large number of ESM studies on alcohol use primarily include men, although studies show that women are more likely to drink alcohol to regulate NA and that this tendency plays a key role in the development of AUD of women [21]. Fourth, ESM studies typically do not differentiate between various types of alcohol use. However, studies suggest that NA especially predicts binge drinking (i.e. the consumption of large amounts of alcohol within a short period of time) over non-heavy alcohol use (i.e. the consumption of a limited amount of alcohol) [44]. Additionally, studies find that individuals who binge drink are more likely to meet the criteria for AUD and that binge drinking predicts the onset of AUD [45, 46].

This study aims to overcome these limitations by exploring the role of momentary affect in the daily lives of female patients with AUD and controls without AUD and investigate the temporal relations between NA and PA on the one hand and craving, non-heavy alcohol use and binge drinking on the other hand. More specifically, it aims to investigate the following hypotheses based on the assumptions of the affective processing and motivational models of alcohol use together with the assumption that the relation between NA and alcohol use exists primarily in patients with AUD:

1. Both within-person NA and PA positively predict subsequent craving in female patients with AUD, but only within-person PA positively predicts subsequent craving in controls.
2. Both within-person NA and PA positively predict subsequent non-heavy alcohol use and binge drinking in female patients with AUD,

but only within-person PA positively predicts subsequent non-heavy alcohol use and binge drinking in controls.

3. Both non-heavy alcohol use and binge drinking predict a subsequently lower NA and higher PA in female patients with AUD and controls.

METHODS

Study sample

The participants were drawn from a larger ESM study in female patients with bulimia nervosa and/or AUD as well as controls (i.e. individuals without bulimia nervosa or AUD). This larger ESM study set out to include 70 patients with bulimia nervosa, 70 patients with AUD and 70 controls, based on recommendations for multilevel studies and previously reported drop-out rates [47, 48]. In the end, the larger ESM study included 51 patients with bulimia nervosa, 53 patients with AUD, 19 patients with both bulimia nervosa and AUD, and 76 controls. The current study only used the data of 53 patients with AUD and 75 controls, after excluding one control who drank no alcohol. The data of the patients with both bulimia nervosa and AUD were excluded, as including them could bias the results concerning the role of NA and PA in patients with AUD. Participants were recruited from September 2019 to February 2022 in Flanders, Belgium through residential and ambulatory care centers, patient groups, universities, social media and by handing out flyers on the street. The inclusion criteria were: (1) female; (2) understand Dutch; (3) body mass index (BMI) ≥ 18.5 kg/m²; and (4) age ≥ 18 years. In Belgium, the legal drinking age is 16 for beverages containing less than 1.2% of distilled alcohol and 18 years for beverages containing more alcohol [49]. Additional inclusion criteria for patients with AUD were: (5) meet the criteria for AUD of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition [1]; (6) display a binge drinking pattern according to the criteria of the National Institute of Alcohol Abuse and Alcoholism (NIAAA) (i.e. drinking 4 units of alcohol within 2 hours for women) [50]; (7) illness duration ≤ 5 years. This maximum illness duration was set as the role of affect is thought to change over the course of the disorder, with the impact of NA increasing and the impact of PA decreasing over the course of AUD [30, 31]. Participants were excluded for the following reasons: (1) major medical pathology; (2) chronic use of sedatives; (3) pregnancy; and (4) presence of psychiatric pathology for controls or major psychiatric pathology for patients with AUD (i.e. schizophrenia, autism spectrum disorder, bipolar disorder, substance use disorder other than AUD). All participants gave their written consent, and the study was approved by the ethical committee of the UZ/KU Leuven.

Study procedure

All potential participants were initially screened via telephone or email, after which they attended an in-person assessment. During this assessment, a resident of psychiatry confirmed whether an individual

was eligible to participate based on the inclusion and exclusion criteria. Afterwards, the participants had their height and weight measured with a calibrated scale and stadiometer and completed clinical interviews and questionnaires. Next, the participants were briefed on the ESM questions and practiced the use of the mobile application. All participants started with the ESM protocol on the first Thursday following the in-person assessment. An overview of the protocol can be seen in Figure 1. The protocol consisted of a repeated measurement design where seven bursts of data collection were spread out over a 12-month period. The bursts had a duration of 3 weeks and were separated by intervals of 5 weeks. During each burst, data were only collected on Thursday, Friday and Saturday to limit the protocol's impact on the participants. These days were selected to consecutively gather data on week and weekend days. In addition, these are the days with the highest alcohol consumption among young adults in Flanders [51]. This resulted in 9 days of data collection in each burst and 63 days in total. On a given day of data collection, participants received eight assessments on a signal-contingent (i.e. semi-random) basis. This meant that there were 72 assessments per burst and 504 assessments per participant. The data were initially collected with the app *MobileQ* [52]. When the app was no longer maintained in October 2020, data collection continued using *m-Path* [53]. More information on the apps can be found in eMethods 1 and eTable 1.

Measures

Baseline measures

The Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (SCID-5-S) was used to confirm the diagnosis of AUD and to screen for other psychiatric disorders [54]. AUD severity was assessed with the Alcohol Use Disorders Identification Test (AUDIT) [55]. The AUDIT had an excellent internal consistency with a standardized Cronbach's α of 0.92.

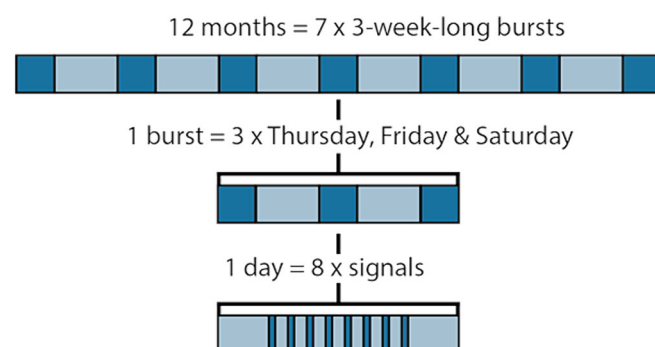


FIGURE 1 Experience sampling method protocol. The protocol consisted of seven bursts of data collection, which were spread out over a 12-month period. The bursts had a duration of 3 weeks and were separated by intervals of 5 weeks. During the bursts, data were only collected on Thursday, Friday and Saturday. On a given day of data collection, participants received eight signals, which were sent on a signal-contingent (i.e. semi-random) basis.

ESM measures

Negative and positive affect

For NA, participants needed to rate how much they agreed with feeling six emotions in the moment (afraid, distressed, guilty, insecure, lonely and sad) on a seven-point Likert scale (1: 'Totally Disagree', 7: 'Totally Agree'). For PA, participants were required to rate how much they agreed with experiencing three emotions in the moment (cheerful, relaxed and satisfied) on a seven-point Likert scale (1: 'Totally Disagree', 7: 'Totally Agree'). These scores were then averaged to get one score for NA and PA at each assessment. The questions for NA and PA were based on previous ESM studies [56–58]. However, other ESM studies measuring NA have included questions on other emotions as well [39].

Craving

Participants needed to rate their urge to drink alcohol in the moment on a five-point Likert scale (1: 'None', 5: 'Overwhelming'). This is similar to other ESM studies on craving [59].

Alcohol use

Participants were required to indicate whether they drank alcohol since the last assessment. If so, they needed to report how many units of alcohol they consumed (1, 2, 3, 4, 5, 6, >7). The participants were instructed about the definition of an alcohol unit (i.e. 250 mL beer, 100 mL wine, 35 mL liquor). For the analyses, based on the criteria of the NIAAA, non-heavy alcohol use was defined as having drunk less than 4 units of alcohol since the last assessment, whereas binge drinking was defined as having drunk at least 4 units of alcohol since the last assessment with the mean (SD) time between assessment of 103 [44] minutes. The exact phrasing of the ESM questions as well as the internal consistency and intraclass coefficients of the ESM scales is found in eMethods 2 and 3 and eTables 2 and 3.

Statistical analysis

Data preparation

The data were collected between September 2019 and January 2023. However, only the assessments answered within 90 minutes of the prompt were used in the analyses. This window was chosen to exclude assessments that were answered too late after the scheduled time. Furthermore, the different models only used a subset of all answered assessments. For craving, only the data before the first report of alcohol consumption of the day was used together with the data on days where participants drank no alcohol. For non-heavy alcohol use, only the data up to the first report of non-heavy alcohol consumption on days without binge drinking were used, in addition to data from days where participants did not drink any alcohol. For binge drinking, only the data up to the first report of a binge drinking episode were used together with the data on days where participants drank no alcohol, but excluding the days with only non-heavy alcohol

use. The reduced datasets were used to avoid an effect of cumulative alcohol use on the results. Additionally, the different models only used a subset of all participants. The analyses for craving and non-heavy alcohol use were performed in patients with AUD and controls who reported to have drunk alcohol in the year before participating in the study, whereas the analyses for binge drinking were carried out on the data from patients with AUD and controls who reported to have experienced at least one binge drinking episode in the year before starting in the study. This was done to ensure that the analyses concerning non-heavy alcohol use or binge drinking were not influenced by controls who did not drink alcohol or did not binge drink.

General

All models were fit using the lmer and glmer packages in R, version 4.1.1. The data and the scripts that support the methodological decisions and results can be found at <https://doi.org/10.48804/B1562D>.

The continuous variables in the models were standardized so that estimates can be interpreted as effect sizes. All models were fitted with maximum likelihood estimation or restricted maximum likelihood estimation and were, therefore, valid under a missing at random assumption. *P*-values below 0.05 were considered significant.

Hypothesis 1 and 2

Separate mixed-effects models were fitted to the data to investigate with either NA or PA at a previous timepoint (t_{-1}) as an independent variable, and with craving, non-heavy alcohol use or binge drinking at the current timepoint (t_0) as the outcome. This means that, unlike previous studies where observations were averaged per day, the current study investigated the relationship between lagged variables that were entered directly into the models. This makes it possible to investigate whether changes in a variable at one timepoint predict changes in another variable at the next timepoint, which is not possible when both variables exist at the same timepoint. The observations could be lagged within a day, but not across days. For craving, the use of a generalized linear mixed-effects model with a cumulative link logit was explored because of its ordinal nature. However, the models did not achieve an adequate fit of the data, which prompted a switch to a linear mixed model. For non-heavy alcohol use and binge drinking, a generalized linear mixed-effects model with a binomial distribution and a logit link was used because of their binary nature. The predictors (i.e. NA or PA) were separated into within- and between-person effects through person-mean centering and grand-mean centering, which made it possible to investigate whether higher than average levels of NA or PA at t_{-1} predicted subsequent craving, non-heavy alcohol use or binge drinking. Furthermore, all models included age, BMI, day since the start of the study and time of day as covariates. These covariates were included to control for previously reported relations between age, BMI and alcohol use and to control for changes in alcohol use over time [60, 61]. However, a forward selection

procedure with Wald tests showed that the relation between the predictors and the outcomes was non-linear, and that a model with both linear and quadratic polynomials of NA and PA, but not cubic polynomials, would best fit the data. Furthermore, to compare patients with AUD and controls, group (i.e. AUD or control) was added as a main and interaction effect with the within-person polynomials at t_{-1} . For the random effects, a maximal random effects structure (i.e. random slope and intercept for participant and a random intercept for burst) was first fit to the data and then simplified with a backward elimination procedure using likelihood-ratio tests. This was done as studies show that a maximal random effects structure can lead to overfitting and uninterpretable models, and that using a parsimonious random effects structure is preferred [62, 63]. This showed that there was no single random effects structure that would best suit all models. Therefore, the backward elimination procedure was performed for each model separately, resulting in varying random-effects structures for the individual models, with some having two levels and others having three. The different structures can be found in eTable 4. A script with the backward elimination procedure can be consulted in the data repository.

Hypothesis 3

Linear mixed models were fitted to the data to investigate whether non-heavy alcohol use and binge drinking at the current assessment (t_0) predicted a subsequently lower NA and higher PA at the following assessment (t_{+1}) in patients with AUD and controls. These models used the same data from the analyses for non-heavy alcohol use and binge drinking mentioned previously. Furthermore, all models included age, BMI, day since the start of the study and time of day as covariates. The random-effects structure was determined in the previously described backward elimination procedure and can be found in eTable 4.

Additional analyses

For the models with a significant quadratic effect, the mean and SD across all participants was calculated for the value of within-person NA or PA at which the predicted outcome was at its lowest point. To assess the robustness of the findings, we conducted sensitivity analyses incorporating additional covariates into the models. These covariates included weekday, compliance, treatment, app type, comorbidities and medication use. Furthermore, we examined the potential influence of the coronavirus disease 2019 (COVID-19) pandemic on the results by introducing a COVID-19 stringency index as a covariate, derived from the Oxford COVID-19 Government Response Tracker [64]. Furthermore, in the models examining the relationship between affect and binge drinking, it was observed that participants engaged in non-heavy alcohol use before experiencing a binge drinking episode in 36.2% (423/1168) of instances. A corresponding variable was introduced into the models as a covariate to assess its

impact. As an exploratory analysis, AUDIT-scores and baseline binge drinking frequency were added as a moderator to investigate the impact of disease severity on the results.

RESULTS

Sample characteristics

The data of 128 study participants (AUD = 53; controls without AUD = 75) were used in the analyses concerning craving and non-heavy alcohol use. One control participant who reported to not drink alcohol was excluded. Furthermore, the data of 92 study participants (AUD = 53, controls without AUD = 39) were used in the analyses on binge drinking. There were 17 (24.5%) patients with a mild AUD (2–3 positive criteria), 17 (24.5%) with a moderate AUD (4–5 positive criteria) and 27 (50.9%) with a severe AUD (≥ 6 positive criteria). The characteristics of the different study groups can be found in Table 1. There were no significant differences in age, BMI, education and ethnicity between the patients with AUD and controls. Additional information on the sample characteristics can be found in eResults 1.

Data characteristics

There were 33 (25.8%) participants (21 [39.6%] AUD; 12 [16.0%] controls) who dropped out of the study before the follow-up ended. For every participant group (AUD, controls), there was no significant difference between those who dropped out and those who did not when it came to age, BMI, illness duration and AUDIT scores. During the first burst, the median compliance per participant was 90.2% for the controls and 81.9% for the patients with AUD. This is similar to the compliance rates of previous cross-sectional ESM studies with a substance use disorder [65]. Over the course of the entire study, the controls answered 24 437 (72.2%) of their scheduled beeps, whereas the patients with AUD answered 12 338 (62.5%). This is similar to the compliance in lengthier ESM studies on substance use [65]. More information on the drop-out and compliance rates as well as the average number of data points per burst can be found in eResults 2 and eTable5.

Hypothesis testing

The main results concerning hypothesis 1 to 3 can be found in Table 2 and are visualized in Figures 2–4. The full results of all the statistical models can be found in eTables 6–8.

Hypothesis 1

(Within-person NA and PA predict subsequent craving in patients with AUD, but only within-person PA predicts subsequent craving in controls)

Within-person NA at a previous assessment (t_{-1}) predicted craving at the current assessment (t_0) in patients with AUD in a positive linear ($\beta = 0.043$; CI = 0.002–0.084; $P = 0.041$) and quadratic fashion ($\beta = 0.034$; CI = 0.011–0.057; $P = 0.004$). The mean (SD) within-person NA at the lowest predicted craving level in patients with AUD was 0.095 (0.856), deviating only slightly from the center. Together, the results suggest that when patients with AUD experienced either lower or higher NA levels than average, they were more likely to report craving afterward, but this was more pronounced when they experienced higher NA levels than usual. Within-person PA at t_{-1} predicted craving at t_0 in patients with AUD with a significant positive quadratic relation ($\beta = 0.042$; CI = 0.08–0.065; $P < 0.001$). The mean (SD) within-person PA at the lowest predicted craving level in patients with AUD was -0.279 (0.832), suggesting a small shift toward lower levels of PA. This indicates that higher than average PA levels were related to more subsequent craving, that slightly lower levels of PA were initially associated with less subsequent craving, and that much lower levels of PA were related to more subsequent craving. In contrast, within-person NA or PA at t_{-1} did not predict craving at t_0 in controls.

Hypothesis 2

(Within-person NA and PA predict subsequent non-heavy alcohol use and binge drinking in patients with AUD, but only within-person PA predicts subsequent alcohol use and binge drinking in controls)

Alcohol use

Within-person NA at t_{-1} negatively predicted non-heavy alcohol use at t_0 in a linear fashion in controls ($\beta = -0.495$; CI = -0.677 to -0.312 ; $P < 0.001$) and patients with AUD ($\beta = -0.276$; CI = -0.421 to -0.132 ; $P < 0.001$). This shows that when patients with AUD or controls reported higher NA levels than they usually experienced, they were less likely to then engage in non-heavy alcohol use. Furthermore, within-person PA at t_{-1} significantly predicted non-heavy alcohol use at t_0 in controls with a positive linear term ($\beta = 0.470$; CI = 0.329–0.610; $P < 0.001$), meaning that when they experienced higher than average levels of PA, they were more likely to then engage in non-heavy alcohol use. However, within-person PA at t_{-1} predicted non-heavy alcohol use at t_0 in patients with AUD with a positive linear term ($\beta = 0.399$; CI = 0.260–0.454; $P < 0.001$) and a positive quadratic term ($\beta = 0.203$; CI = 0.060–0.347; $P = 0.003$). The mean (SD) within-person PA at the lowest predicted probability of non-heavy alcohol use in patients with AUD was -0.527 (0.942), suggesting a shift toward lower levels of PA. This shows that higher than average PA levels were related to more subsequent non-heavy alcohol use in patients with AUD, that slightly lower levels of PA were initially associated with less subsequent non-heavy alcohol use, but much lower levels of PA were related to more subsequent non-heavy alcohol use.

Binge drinking

Within-person NA at t_{-1} predicted binge drinking at t_0 in patients with AUD with a significant quadratic term ($\beta = 0.236$; CI = 0.060–0.412;

TABLE 1 Sample characteristics.

	AUD (n = 53)		C			
			C _{Total} (n = 75)		C _{Binge drinking} (n = 39)	
	Mean (SD)	95% CI	Mean (SD)	95% CI	Mean (SD)	95% CI
Age	21.4 (3.5)	20.5–22.4	21.7 (3.1)	21.0–22.4	21.2 (2.1)	20.4–22.4
BMI	22.7 (2.0)	22.1–23.3	22.3 (2.2)	21.8–22.8	22.2 (2.0)	21.6–22.9
Illness duration AUD (y)	3.1 (1.3)	2.8–3.5	0 (0)	0–0	0 (0)	0–0
Education (y)	14.7 (1.6)	14.3–15.2	15.0 (1.6)	14.6–15.3	15.0 (1.6)	14.5–15.5
AUDIT	15.1 (5.3)	13.7–16.6	4.0 (2.5)	3.4–4.5	5.2 (2.3)	4.6–6.1
ESM measures ^a						
NA	2.3 (1.1)	2.3–2.4	2.1 (1.0)	2.1–2.1	2.0 (0.9)	2.0–2.0
PA	4.9 (1.3)	4.8–4.9	5.1 (1.2)	5.1–5.1	5.1 (1.1)	5.1–5.1
Craving	1.4 (0.8)	1.4–1.4	1.1 (0.3)	1.1–1.1	1.1 (0.3)	1.1–1.1
Alcohol use	25 (26)	12–31	14 (15)	6–15	16 (15)	5–18
Binge drinking	7 (9)	2–7	1 (3)	0–1	2 (3)	0–2
	n (%)		n (%)		n (%)	
Baseline binge drinking frequency						
Never	0 (0)	0–0	36 (48)	37–60	0 (0)	0–0
Annually	0 (0)	0–0	5 (7)	0–18	5 (13)	0–31
Semi-annually	0 (0)	0–0	10 (13)	3–25	10 (26)	13–43
Three-monthly	5 (9)	0–24	15 (20)	10–32	15 (38)	26–56
Monthly	10 (19)	8–34	5 (7)	0–18	5 (13)	0–31
Biweekly	21 (40)	28–55	3 (4)	0–16	3 (8)	0–25
Weekly	10 (19)	8–34	1 (1)	0–13	1 (3)	0–20
>Weekly	7 (13)	2–28	0 (0)	0–0	0 (0)	0–0
Therapy (general)						
Past	29 (55)	41–68	23 (31)	20–41	10 (26)	11–40
Present	5 (9)	1–17	3 (4)	0–9	1 (3)	0–8
Therapy (AUD)						
Past	1 (2)	0–6	0 (0)	0–0	0 (0)	0–0
Present	1 (2)	0–6	0 (0)	0–0	0 (0)	0–0
Ethnicity						
Caucasian	50 (94)	91–100	73 (97)	95–100	39 (100)	100–100
Latino	1 (2)	0–8	0 (0)	0–0	0 (0)	0–0
Mixed	2 (4)	0–10	1 (0)	0–4	0 (0)	0–0
Asian	0 (0%)	0–0	1 (0)	0–4	0 (0)	0–0
Psychoactive medication	6 (11%)	3–20	0 (0)	0–0	0 (0)	0–0
Comorbidities						
MDD	3 (6%)	0–17	0 (0)	0–0	0 (0)	0–0
PD	2 (4%)	0–15	0 (0)	0–0	0 (0)	0–0
SAD	1 (2%)	0–13	0 (0)	0–0	0 (0)	0–0
ADHD	3 (6%)	0–17	0 (0)	0–0	0 (0)	0–0
PTSD	2 (4%)	0–15	0 (0)	0–0	0 (0)	0–0

Abbreviations: ADHD, attention deficit hyperactivity disorder; AUD, alcohol use disorder; AUDIT, Alcohol Use Disorders Identification Test; BMI, body mass index; C, controls; C_{Binge drinking}, controls who reported at least one binge drinking episode in the past year; ESM, experience sampling method; MDD, major depressive disorder; NA, negative affect; PA, positive affect; PD, panic disorder; PTSD, post-traumatic stress disorder; SAD, social anxiety disorder.

^aThe ESM measurements refer to the mean level observed in a participant throughout the entire duration of the study.

TABLE 2 Model results.

Outcome	Variable	Group	Polynomial	β	OR	SE	95% CI	P
Craving t_0	NA t_{-1}	Controls	Linear	-0.004	-	0.017	-0.036, 0.029	0.831
			Quadratic	-0.006	-	0.008	-0.021, 0.010	0.472
		AUD	Linear	0.043	-	0.021	0.002, 0.084	0.041*
			Quadratic	0.034	-	0.012	0.011, 0.057	0.004*
		AUD vs Controls	Linear	0.047	-	0.027	-0.006, 0.099	0.083
			Quadratic	0.040	-	0.014	0.012, 0.067	0.006*
	PA t_{-1}	Controls	Linear	0.007	-	0.014	-0.021, 0.034	0.617
			Quadratic	-0.005	-	0.007	-0.019, 0.009	0.459
		AUD	Linear	0.019	-	0.019	-0.017, 0.055	0.303
			Quadratic	0.042	-	0.012	0.018, 0.065	<0.001*
		AUD vs Controls	Linear	0.012	-	0.023	-0.034, 0.058	0.606
			Quadratic	0.047	-	0.014	0.020, 0.075	0.001*
Non-heavy alcohol use t_0	NA t_{-1}	Controls	Linear	-0.495	0.610	0.093	-0.677, -0.312	<0.001*
			Quadratic	-0.024	0.976	0.097	-0.214, 0.167	0.806
		AUD	Linear	-0.276	0.759	0.074	-0.421, -0.132	<0.001*
			Quadratic	0.008	1.008	0.076	-0.141, 0.156	0.920
		AUD vs Controls	Linear	0.219	1.244	0.119	-0.014, 0.451	0.065
			Quadratic	0.061	1.064	0.112	-0.157, 0.280	0.581
	PA t_{-1}	Controls	Linear	0.470	1.599	0.072	0.329, 0.610	<0.001*
			Quadratic	0.041	1.042	0.069	-0.093, 0.176	0.547
		AUD	Linear	0.399	1.490	0.071	0.260, 0.4537	<0.001*
			Quadratic	0.203	1.226	0.073	0.060, 0.347	0.006*
		AUD vs Controls	Linear	-0.071	0.931	0.100	-0.268, 0.126	0.479
			Quadratic	0.162	1.176	0.100	-0.034, 0.358	0.106
Binge drinking t_0	NA t_{-1}	Controls	Linear	-0.028	0.973	0.253	-0.524, 0.469	0.913
			Quadratic	0.233	1.263	0.183	-0.124, 0.591	0.201
		AUD	Linear	-0.033	0.967	0.100	-0.229, 0.163	0.739
			Quadratic	0.236	1.267	0.090	0.060, 0.412	0.008*
		AUD vs Controls	Linear	-0.006	0.994	0.272	-0.539, 0.528	0.983
			Quadratic	0.003	1.003	0.203	-0.394, 0.400	0.988
	PA t_{-1}	Controls	Linear	0.286	1.331	0.199	-0.104, 0.676	0.151
			Quadratic	0.487	1.612	0.163	0.158, 0.770	0.003*
		AUD	Linear	0.017	1.460	0.088	-0.156, 0.190	0.846
			Quadratic	0.378	1.331	0.083	0.215, 0.542	<0.001*
		AUD vs Controls	Linear	-0.269	0.764	0.218	-0.696, 0.158	0.217
			Quadratic	-0.099	0.905	0.183	-0.457, 0.259	0.587
NA t_{+1}	Non-heavy alcohol use t_0	Controls	-	-0.111	-	0.042	-0.193, -0.029	0.010*
		AUD	-	-0.161	-	0.044	-0.248, -0.074	0.001*
		AUD vs Controls	-	-0.047	-	0.063	-0.170, 0.075	0.454
	Binge drinking t_0	Controls	-	-0.087	-	0.136	-0.324, 0.209	0.520
		AUD	-	-0.077	-	0.063	-0.174, 0.070	0.217
		AUD vs Controls	-	0.010	-	0.149	-0.282, 0.303	0.945
PA t_{+1}	Non-heavy alcohol use t_0	Controls	-	0.189	-	0.045	0.101, 0.278	<0.001*
		AUD	-	0.181	-	0.048	0.088, 0.274	<0.001*
		AUD vs Controls	-	-0.009	-	0.066	-0.137, 0.120	0.895
	Binge drinking t_0	Controls	-	0.068	-	0.189	-0.302, 0.439	0.720

TABLE 2 (Continued)

Outcome	Variable	Group	Polynomial	β	OR	SE	95% CI	P
		AUD	–	0.096	–	0.099	–0.098, 0.290	0.338
		AUD vs Controls	–	0.028	–	0.213	–0.390, 0.446	0.896

Table displaying the outcomes of the different mixed effects models. The ‘Outcome’ column represents the dependent variable used in the models. The ‘Variable’ column denotes the independent variable for each model. The ‘Group’ column specifies the group for which the variable estimate was calculated or the interaction effect (i.e. the difference between patients with AUD and controls without AUD). The ‘Polynomial’ column indicates the specific polynomial for which the estimate was calculated, if polynomials were used.

Abbreviations: AUD, alcohol use disorder; β , standardized estimate; NA, negative affect; OR, odds ratio; P, P-value; PA, positive affect; t_{-1} , previous assessment; t_0 , current assessment; t_{+1} , next assessment.

*Significant result.

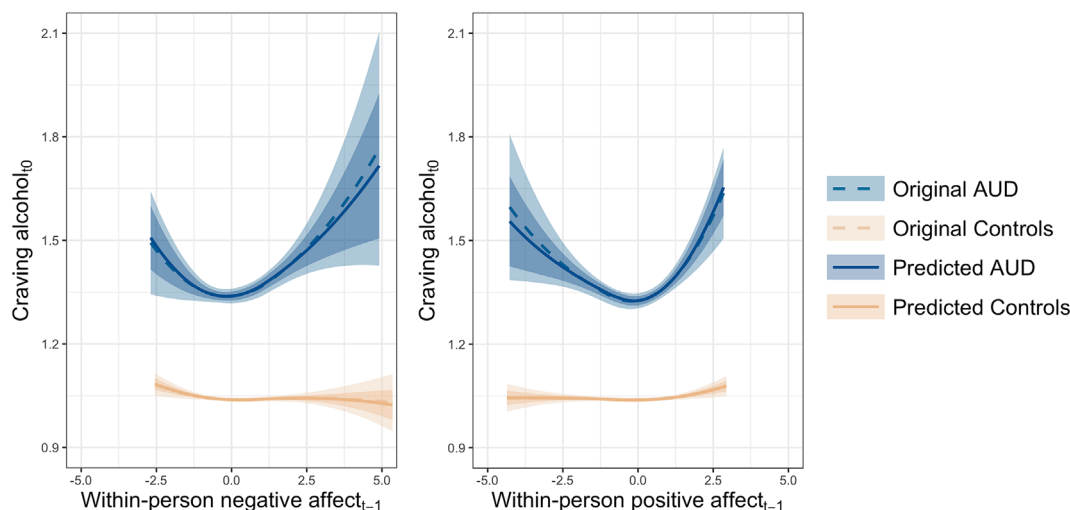


FIGURE 2 Smoothed Loess curves showing the relation between within-person negative and positive affect at the previous assessment (t_{-1}) and craving for alcohol at the current assessment (t_0) in the original data and the data predicted by the linear mixed models. Abbreviations: AUD, alcohol use disorder; t_{-1} : previous assessment; t_0 , current assessment.

$P = 0.008$), but this was not the case for controls. Stated differently, when patients with AUD experience either higher or lower levels of NA than usual, they were more likely to binge drink afterward. The mean (SD) within-person NA at the lowest predicted probability of binge drinking in patients with AUD was 0.035 (0.615), deviating only slightly from the center. Furthermore, within-person PA at t_{-1} predicted binge drinking at t_0 in patients with AUD with a significant quadratic term ($\beta = 0.378$; CI = 0.215–0.542; $P < 0.001$), and this was also the case for controls ($\beta = 0.487$; CI = 0.158–0.770; $P < 0.001$). The mean (SD) within-person PA at the lowest predicted probability of binge drinking was -0.330 (0.734) in patients with AUD and -0.598 (0.956) in controls. This means that higher than average PA levels were related to more subsequent binge drinking in patients with AUD and controls, that slightly lower levels of PA were initially associated with less subsequent binge drinking, but much lower levels of PA were related to more subsequent binge drinking.

Hypothesis 3

(Non-heavy alcohol use and binge drinking predict a subsequently lower NA and higher PA in patients with AUD and controls)

Alcohol use

Non-heavy alcohol use at t_0 predicted lower levels of NA at t_{+1} in both patients with AUD ($\beta = -0.161$; SE = 0.044; CI = -0.248 to 0.074 ; $P = 0.001$) and controls ($\beta = -0.114$; CI = -0.198 to -0.029 ; $P = 0.010$). Similarly, non-heavy alcohol use at t_0 predicted higher levels of PA at t_{+1} in both patients with AUD ($\beta = 0.181$; CI = 0.088–0.274; $P < 0.001$) and controls ($\beta = 0.189$; CI = 0.101–0.278; $P < 0.001$). This means that engaging in non-heavy alcohol use was followed by lower than average NA and higher than average PA in both patients with AUD and controls.

Binge drinking

Binge drinking at t_0 did not predict subsequent changes in NA or PA at t_{+1} in patients with AUD or controls.

Sensitivity analyses

The inclusion of week day, compliance, medication use, therapy, app type, presence of comorbidities or COVID-19 stringency did not change the significance of these results. Furthermore, when it comes

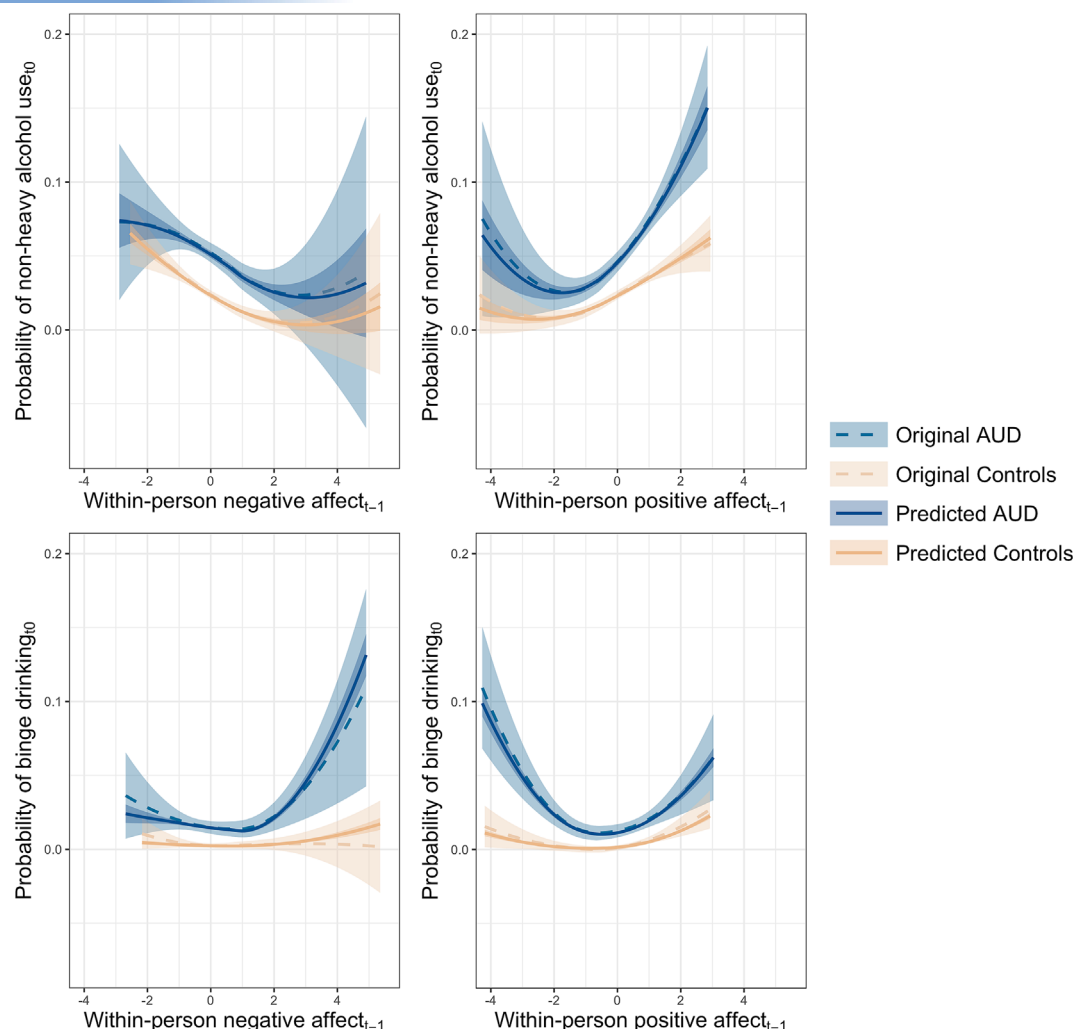


FIGURE 3 Smoothed Loess curves showing the relation between within-person negative and positive affect at the previous assessment (t_{-1}) and alcohol use or binge drinking at the current assessment (t_0) in the original data and the data predicted by the generalized linear mixed models. Abbreviations: AUD, alcohol use disorder; t_{-1} : previous assessment; t_0 , current assessment.

to the models on binge drinking, the inclusion of a variable concerning previous non-heavy alcohol use did not influence the results.

Moderation by disease severity

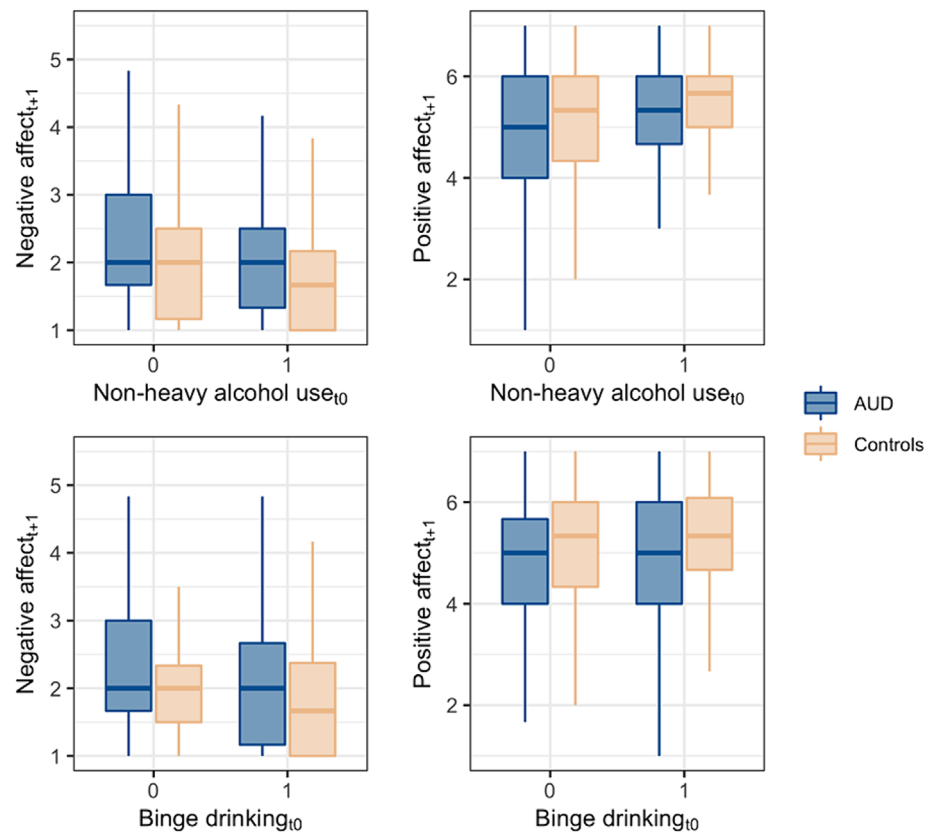
The full results concerning moderation by AUDIT scores and baseline binge drinking frequency can be found in eTables 6 and 7). For the relation between NA at t_{-1} and craving at t_0 , patients with AUD with higher AUDIT scores had a stronger linear relation ($\beta = 0.137$; CI = 0.093–0.179; $P < 0.001$), whereas a higher binge drinking frequency was related to a stronger linear ($\beta = 0.112$; CI = 0.093–0.179; $P = 0.005$) and quadratic relation ($\beta = 0.063$; CI = 0.014–0.112; $P = 0.011$). For the relation between PA at t_{-1} and craving at t_0 , patients with AUD with higher AUDIT scores had a more negative linear relation ($\beta = -0.109$; CI = -0.148 to -0.070; $P < 0.001$) and a stronger quadratic relation ($\beta = 0.040$; CI = 0.011–0.069; $P = 0.007$), whereas a higher binge drinking frequency was related to a stronger quadratic relation ($\beta = 0.090$; CI = 0.039–0.112; $P = 0.141$). These

results show that when patients with AUD with a higher disease severity experienced a deviation from their average NA or PA levels, they reported more craving afterward, and this was especially the case for higher NA and lower PA. Additionally, controls with a higher binge drinking frequency had a stronger quadratic relation between PA at t_{-1} and non-heavy alcohol use at t_0 ($\beta = 0.198$; CI = 0.023–0.372; $P = 0.026$), as well as a more negative relation between binge drinking at t_{-1} and NA use at t_0 ($\beta = -0.380$; CI = -0.688 to -0.072; $P = 0.016$). This means that controls with a higher baseline binge drinking frequency were more likely to engage in non-heavy alcohol use after experiencing higher or lower PA, and that they experienced a lower NA after binge drinking.

DISCUSSION

The purpose of this study is to compare the affective dynamics surrounding craving, non-heavy alcohol use and binge drinking between female patients with AUD and female controls without AUD.

FIGURE 4 Box plots showing the relation between alcohol use or binge drinking at the current assessment (t_0) and negative or positive affect at the next assessment (t_{+1}) in the original data. Abbreviations: AUD, alcohol use disorder; t_0 , current assessment; t_{+1} : next assessment.



Importantly, the results show that the relation between affect and craving or alcohol use is typically non-linear in women. First, both higher and lower than average levels of NA and PA predict higher subsequent craving in female patients with AUD, although slightly lower levels of PA are first associated with lower subsequent craving. In contrast, deviations from average affect do not predict subsequent craving in controls. Second, lower than average levels of NA and higher than average levels of PA predict subsequent non-heavy alcohol use in female patients with AUD and controls, although at more pronounced lower levels of PA, the probability of subsequent non-heavy alcohol use in female patients with AUD rises again. Third, both lower and higher deviations from average NA predict subsequent binge drinking in female patients with AUD, but not in controls. Furthermore, lower and higher than average levels of PA are related to subsequent binge drinking in female patients with AUD and controls, although slightly lower levels of PA are first associated with a lower probability of subsequent binge drinking in both populations. Fourth, non-heavy alcohol use, but not binge drinking, predicts subsequently lower levels of NA and higher levels of PA in both female patients with AUD and controls.

The non-linear relation between affect and craving or alcohol use

Although their number is limited, there are previous studies reporting that affect is related to craving and alcohol use in a non-linear manner

[66, 67]. One study finds that days with lower as well as higher loneliness were related to more alcohol consumption during the COVID-19 pandemic [66]. Another study reports that drinking alcohol alleviates NA in women, but that this effect is more pronounced during periods of heightened NA [67]. Such a non-linear relation is not unexpected as alcohol use is thought to be related to both NA and PA, which are negatively correlated with one another, and because both lower as well as higher levels of PA are hypothesized to lead to alcohol consumption [11, 12, 68]. Indeed, one study finds that low PA as well as high anxiety predict subsequent alcohol use [41]. However, there could be several reasons why the current study was particularly well-suited to detect a non-linear relation. The focus on the lagged relation between variables measured at a momentary level could have provided the temporal resolution necessary to detect such a relation, unlike studies that average observations at a daily level. Furthermore, the study's burst measurement design, which has resulted in a large number of observations per participant, could have facilitated the modeling of complex within-subject relations. Additionally, this study includes patients with AUD who have a short illness duration, which could imply that they are still at a stage where both NA and PA play a role [30, 31]. Regardless, the finding that NA and PA can be non-linearly related to craving and alcohol use seems to be essential for the different theories on affect and alcohol consumption to converge. Interestingly, the results indicate that slightly lower than average levels of PA were related to less craving, non-heavy alcohol use and binge drinking in female patients with AUD, whereas more pronounced lower levels of PA were associated with more craving, non-

heavy alcohol use and binge drinking. This could be related to the finding that patients with AUD display more affective urgency, whereby strong changes in NA or PA lead to rash actions [69]. Taken together, future studies should not simply assume a linear relation between affect and craving or alcohol use, but pay attention to potential non-linear effects. Furthermore, the results suggest that treatments for AUD should address the complete range of affective dynamics surrounding craving and alcohol use.

Differences between female patients with AUD and controls

The results of this study suggest that female patients with AUD and controls differ in the way that NA and PA are related to craving and alcohol use in daily life.

First, the current study finds that both lower and higher than average levels of NA and PA are associated with subsequent craving in female patients with AUD, but not in controls. Additionally, this was related to disease severity as higher AUDIT scores and a higher binge drinking frequency are associated to experiencing more craving in response to high NA and low PA in female patients with AUD. These results are in line with research showing that experimentally inducing NA or PA increases craving in patients with AUD, but contradict certain ESM studies, which report that NA predicts craving in individuals who do not misuse alcohol [18, 27, 59, 70, 71]. One possible explanation for these conflicting results is that previous ESM studies typically examine the relationship between concurrent affect and craving levels, whereas the current study focuses on lagged relations. Specifically, if the impact of affect on craving in non-problematic drinkers would be limited in size and duration, then it would be more difficult to detect this effect with a lagged analysis where the average lag was 100 minutes. Nevertheless, the difference between patients and controls is in line with most theories on craving, which posit that certain predisposing and acquired factors strengthen the relation between affect and craving in patients with AUD [13]. For example, patients are thought to have more positive expectancies of alcohol consumption compared to controls, such as the belief that drinking alcohol will help to cope with NA or enhance PA [72]. Indeed, individuals with higher drinking to cope expectancies display a stronger relation between NA and craving in daily life [59]. Future ESM studies should, therefore, further explore which predisposing and acquired factors contribute to the relation between affect and craving and how this differs between patients and controls.

Second, the current study shows that female patients with AUD could indeed be more likely to drink alcohol in daily life in response to a worse mood (i.e. low PA or high NA), and this could be more pronounced than in individuals without AUD [11, 12]. This stands in contrast to previous ESM studies reporting that there is no relation between high NA or low PA and alcohol use at a daily level [39]. However, these studies have typically been performed in individuals who do not misuse alcohol, and usually include a sample with a majority of male participants. Therefore, future ESM studies that want to

investigate the role of affect in alcohol misuse should therefore aim include a sample of patients with AUD, and balance their sample when it comes to sex.

Differences between non-heavy alcohol use or binge drinking

The findings of the current study suggest that the relation between affect and alcohol use depends on the amount of alcohol that is consumed. It appears that higher levels of NA and lower levels of PA are more strongly associated with subsequent binge drinking in daily life than with subsequent non-heavy alcohol use across female patients and controls. Indeed, studies suggest that NA increases the risk for binge drinking more so than for non-heavy alcohol use and that individuals who experience more NA are more likely to start binge drinking [44, 73]. This could be more pronounced in women, where higher levels of depression are more strongly related to the amount of alcohol consumed than in men [74]. Furthermore, it seems that non-heavy alcohol use could play a larger role in affect regulation in daily life than binge drinking. This is because non-heavy alcohol use predicts subsequently lower levels of NA and higher levels of PA in the current study, whereas this is not the case for binge drinking. Indeed, there are studies who report that a small amount of alcohol can lead to feelings of euphoria, whereas larger amounts increase feelings of depression [75]. However, there have also been studies in daily life that show that reductions in NA are higher on days with heavy drinking days than on moderate drinking days [76]. Therefore, the results suggest that more research is needed on how NA and PA are related to different types of alcohol use (i.e. non-heavy alcohol use and binge drinking) in daily life. The results also suggest that treatments for AUD should differentiate between the different ways in which alcohol is consumed, tailoring interventions on whether a patient is more likely to engage in non-heavy alcohol use or binge drinking.

Limitations

This study has several limitations. First, the sample of patients with AUD mostly consists of Caucasian non-treatment seeking female individuals with a short illness duration. Furthermore, patients with a comorbid substance use disorder were excluded although ~47.1% of female patients with AUD meet the criteria for a substance use disorder [77]. This impacts the generalizability of the results to all patients with AUD. Second, the sample of controls consists of different types of drinkers (i.e. social drinkers and heavy drinkers). However, there could be differences between these drinking patterns. Furthermore, controls were excluded if they had a psychiatric comorbidity, whereas this was not the case for patients with AUD, which could contribute to the differences between the participant groups. However, a sensitivity analysis did not confirm that the presence of psychiatric comorbidities influenced the results. Third, the larger ESM study, from which the data of the current study come, aimed to include

70 participants in every study group to ensure the analyses were well-powered. However, the current study includes fewer participants in the group of female patients with AUD, which could have impacted the power of the analyses. Fourth, the decrease in compliance over the course of the study could impact the results because of the missing data. However, the analysis techniques used in this study are valid under a missing at random assumption and a sensitivity analysis finds no impact of compliance on the results. Fifth, limiting the ESM assessments to Thursday, Friday and Saturday could have influenced the results if participants would experience a different relation between affect, craving and alcohol use on the other days of the week. However, as to our knowledge, no other studies have assessed whether individuals experience differences in this relation depending on the day of the week. Sixth, the signal-contingent assessment schedule could have influenced the measurement of alcohol use, especially at night, and could have been improved with the addition of event-contingent assessments. Seventh, binge drinking was defined in the current study as having drunk at least 4 units of alcohol since the last assessment with a mean (SD) time between assessment of 103 [44] minutes, which differs from the definition of the NIAAA that states that binge drinking for women is drinking at least 4 units of alcohol within 2 hours [50]. This needs to be kept in mind when comparing the results of the current study with those of other studies.

AUTHOR CONTRIBUTIONS

Nicolas Leenaerts: Conceptualization (lead); data curation (lead); formal analysis (lead); investigation (lead); methodology (lead); project administration (lead); software (lead); visualization (lead); writing—original draft (lead). **Thomas Vaessen:** Methodology (equal); writing—original draft (equal). **Stefan Sunaert:** Supervision (equal). **Jenny Ceccarini:** Funding acquisition (equal). **Elske Vrieze:** Conceptualization (supporting); methodology (supporting); supervision (equal); writing—original draft (equal).

ACKNOWLEDGEMENTS

The present study design and analyses were not pre-registered. However, consistent with the Transparency and Openness Promotion (TOP) guidelines, the data and scripts that support the findings of this study are available at the Research Data Repository of the KU Leuven at <https://doi.org/10.48804/B1562D>.

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

DECLARATION OF INTEREST

Concerning conflict of interest and funding, A C1 grant (ECA-D4671-C14/18/096) of the Special Research Fund KU Leuven to E.V. and J.C. served as a PhD Scholarship for N.L. J.C. and T.V. were supported by a postdoc grant from Fonds Wetenschappelijk Onderzoek (FWO; 12R1619N and 1243620N). No other grant of any kind was received. No other disclosures were reported.

DATA AVAILABILITY STATEMENT

The present study' design and analyses were not pre-registered. However, consistent with the Transparency and Openness Promotion (TOP) guidelines, the data and scripts that support the findings of this study are available at the Research Data Repository of the KU Leuven at <https://rdr.kuleuven.be/privateurl.xhtml?token=9b7f1cd5-116f-47a2-bf3d-147c9b884417>.

ORCID

Nicolas Leenaerts  <https://orcid.org/0000-0003-2421-6845>

REFERENCES

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders 5th ed.; 2013.
2. World Health Organization. Global status report on alcohol and health 2018 Geneva; 2018.
3. Anderson P, Baumberg B. Alcohol in Europe – Public Health Perspective: Report summary. [Internet]. 2009 1 [cited 2022 Dec 21];13(6):483–8. Available from: <https://doi.org/10.1080/09687630600902477>
4. Sacks JJ, Gonzales KR, Bouchery EE, Tomedi LE, Brewer RD. 2010 National and State Costs of Excessive Alcohol Consumption. *Am J Prev Med*. 2015;49(5):e73–9. Available from: <https://pubmed.ncbi.nlm.nih.gov/26477807/>
5. Mekonen T, Chan GCK, Connor J, Hall W, Hides L, Leung J. Treatment rates for alcohol use disorders: a systematic review and meta-analysis. *Addiction*. 2021;116(10):2617–34. Available from: <https://pubmed.ncbi.nlm.nih.gov/33245581/>
6. Fleury MJ, Djouini A, Huynh C, Tremblay J, Ferland F, Ménard JM, et al. Remission from substance use disorders: a systematic review and meta-analysis. *Drug Alcohol Depend*. 2016;168:293–306. <https://doi.org/10.1016/j.drugalcdep.2016.08.625>
7. Agabio R, Pisanu C, Gessa GL, Franconi F. Sex differences in alcohol use disorder. *Curr Med Chem*. 2016;24(24):2661–70. <https://doi.org/10.2174/0929867323666161202092908>
8. McCrady BS, Epstein EE, Fokas KF. Treatment interventions for women with alcohol use disorder. *Alcohol Res*. 2020;40(2):1–18. Available from: [/pmc/articles/PMC7384374/](https://pubmed.ncbi.nlm.nih.gov/35946/arcv.v40.2.08). <https://doi.org/10.35946/arcv.v40.2.08>
9. Holzhauer CG, Cucciare M, Epstein EE. Sex and gender effects in recovery from alcohol use disorder. *Alcohol Res*. 2020;40(2):1–19. Available from: [/pmc/articles/PMC7668196/](https://pubmed.ncbi.nlm.nih.gov/35946/arcv.v40.3). <https://doi.org/10.35946/arcv.v40.3>
10. Baker TB, Piper ME, McCarthy DE, Majeskie MR, Fiore MC. Addiction motivation reformulated: an affective processing model of negative reinforcement. *Psychol Rev*. 2004;111(1):33–51. <https://doi.org/10.1037/0033-295X.111.1.33>
11. Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: a motivational model of alcohol use. *J Pers Soc Psychol*. 1995;69(5):990–1005. <https://doi.org/10.1037/0022-3514.69.5.990>
12. Cox WM, Klinger E. A motivational model of alcohol use. *J Abnorm Psychol*. 1988;97(2):168–80. Available from: [/doiLanding?doiLanding?doi=10.1037%2F0021-843X.97.2.168](https://doi.org/10.1037/0022-3514.97.2.168).
13. van Lier HG, Pieterse ME, Schraagen JMC, Postel MG, Vollenbroek-Hutten MMR, de Haan HA, et al. Identifying viable theoretical frameworks with essential parameters for real-time and real world alcohol craving research: a systematic review of craving models. *Addiction Res Theory*. 2018;26(1):35–51. Available from: <https://www.tandfonline.com/doi/abs/10.1080/16066359.2017.1309525>
14. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. *J Pers*

- Soc Psychol. 1988;54(6):1063–70. <https://doi.org/10.1037/0022-3514.54.6.1063>
15. Conger JJ II. Reinforcement Theory and the Dynamics of Alcoholism. *Q J Stud Alcohol*. 1956;17(2):296–305. Available from: <https://www.jsad.com/doi/10.15288/qjsa.1956.17.296>
 16. Levenson RW, Sher KJ, Grossman LM, Newman J, Newlin DB. Alcohol and stress response dampening: pharmacological effects, expectancy, and tension reduction. *J Abnorm Psychol*. 1980;89(4):528–38. <https://doi.org/10.1037/0021-843X.89.4.528>
 17. Bresin K. A meta-analytic review of laboratory studies testing the alcohol stress response dampening hypothesis. *Psychol Addict Behav*. 2019;33(7):581–94. <https://doi.org/10.1037/adb0000516>
 18. Bresin K, Mekawi Y, Verona E. The Effect of Laboratory Manipulations of Negative Affect on Alcohol Craving and Use: A Meta-analysis. *Psychol Addict Behav*. 2018;32(6):617. Available from: <https://pmc/articles/PMC6136957/>.
 19. Witkiewitz K, Villarroel NA. Dynamic association between negative affect and alcohol lapses following alcohol treatment. *J Consult Clin Psychol*. 2009;77(4):633–44. <https://doi.org/10.1037/a0015647>
 20. Carpenter KM, Hasin DS. Drinking to cope with negative affect and DSM-IV alcohol use disorders: a test of three alternative explanations. *J Stud Alcohol*. 2015;60(5):694–704. Available from: <https://www.jsad.com/doi/10.15288/jsa.1999.60.694>
 21. Peltier MR, Verplaetse TL, Mineur YS, Petrakis IL, Cosgrove KP, Picciotto MR, et al. Sex differences in stress-related alcohol use. *Neurobiol Stress*. 2019;10:100149. <https://doi.org/10.1016/j.ynstr.2019.100149>
 22. Kathryn McHugh R, Kaufman JS, Frost KH, Fitzmaurice GM, Weiss RD. Positive Affect and Stress Reactivity in Alcohol-Dependent Outpatients. *J Stud Alcohol Drugs*. 2015;74(1):152–7. Available from: <https://www.jsad.com/doi/10.15288/jsad.2013.74.152>
 23. Tamir M, Robinson MD. The happy spotlight: positive mood and selective attention to rewarding information. *Pers Soc Psychol Bull*. 2007;33(8):1124–36. Available from: <https://pubmed.ncbi.nlm.nih.gov/17578934/>
 24. Young CB, Nusslock R. Positive mood enhances reward-related neural activity. *Soc Cogn Affect Neurosci*. 2016;11(6):934. Available from: <https://pmc/articles/PMC4884311/>.
 25. Dinc L, Cooper AJ. Positive affective states and alcohol consumption: the moderating role of trait positive urgency. *Addict Behav*. 2015;47:17–21. <https://doi.org/10.1016/j.addbeh.2015.03.014>
 26. Wilkie H, Stewart SH. Reinforcing mood effects of alcohol in coping and enhancement motivated drinkers. *Alcohol Clin Exp Res*. 2005;29(5):829–36. Available from: <https://pubmed.ncbi.nlm.nih.gov/15897728/>
 27. Mason BJ, Light JM, Escher T, Drobos DJ. Effect of positive and negative affective stimuli and beverage cues on measures of craving in non treatment-seeking alcoholics. *Psychopharmacology (Berl)*. 2008;200(1):141–50. Available from: <https://link.springer.com/article/10.1007/s00213-008-1192-x>
 28. Karpyak VM, Biernacka JM, Geske JR, Abulseoud OA, Brunner MD, Chauhan M, et al. Gender-specific effects of comorbid depression and anxiety on the propensity to drink in negative emotional states. *Addiction*. 2016;111(8):1366–75. Available from: <https://onlinelibrary.wiley.com/doi/full/10.1111/add.13386>
 29. Harder VS, Ayer LA, Rose GL, Naylor MR, Helzer JE. Alcohol, Moods and Male–Female Differences: Daily Interactive Voice Response over 6 Months. *Alcohol Alcoholism*. 2014;49(1):60–5. <https://doi.org/10.1093/alcalc/agt069>
 30. Koob GF, Le Moal M. Drug abuse: Hedonic homeostatic dysregulation. *Science (1979)*. 1997;278(5335):52–8. Available from: <https://www.science.org/doi/10.1126/science.278.5335.52>
 31. Koob GF, Volkow ND. Neurobiology of addiction: a neurocircuitry analysis. *Lancet Psychiatry*. 2016;3(8):760. Available from: <https://pmc/articles/PMC6135092/>.
 32. King AC, McNamara PJ, Hasin DS, Cao D. Alcohol challenge responses predict future alcohol use disorder symptoms: a 6-year prospective study. *Biol Psychiatry*. 2014;75(10):798–806. <https://doi.org/10.1016/j.biopsych.2013.08.001>
 33. Sayette MA, Shiffman S, Tiffany ST, Niaura RS, Martin CS, Schadel WG. The measurement of drug craving. *Addiction*. 2000;95(Suppl 2):S189. Available from: <https://pmc/articles/PMC2683662/>.
 34. Higley AE, Crane NA, Spadoni AD, Quello SB, Goodell V, Mason BJ. Craving in response to stress induction in a human laboratory paradigm predicts treatment outcome in alcohol-dependent individuals. *Psychopharmacology (Berl)*. 2011;218(1):121–9. Available from: <https://pubmed.ncbi.nlm.nih.gov/21607563/>
 35. Cooney NL, Litt MD, Morse PA, Bauer LO, et al. Alcohol cue reactivity, negative-mood reactivity, and relapse in treated alcoholic men. *J Abnorm Psychol*. 1997;106(2):243–50. Available from: <https://pubmed.ncbi.nlm.nih.gov/9131844/>
 36. Boykoff N, Schneekloth TD, Hall-Flavin D, Loukianova L, Karpyak VM, Stevens SR, et al. Gender differences in the relationship between depressive symptoms and cravings in alcoholism. *Am J Addict*. 2010;19(4):352–6. Available from: <https://onlinelibrary-wiley-com.kuleuven.e-bronnen.be/doi/full/10.1111/j.1521-0391.2010.00057.x>
 37. Rubonis AV, Colby SM, Monti PM, Rohsenow DJ, Gulliver SB, Sirota AD. Alcohol cue reactivity and mood induction in male and female alcoholics. *J Stud Alcohol*. 2015;55(4):487–94. Available from: <https://www.jsad.com/doi/10.15288/jsa.1994.55.487>
 38. Shiffman S, Stone AA, Hufford MR. Ecological momentary assessment. *Annu Rev Clin Psychol*. 2008;4:1–32. Available from: <https://pubmed.ncbi.nlm.nih.gov/18509902/>. <https://doi.org/10.1146/annurev.clinpsy.3.022806.091415>
 39. Dora J, Piccirillo M, Foster KT, Arbeau K, Armeli S, Auriacombe M, et al. The daily association between affect and alcohol use: a meta-analysis of individual participant data. 2022.
 40. Emery NN, Simons JS. The role of affect, emotion management, and attentional bias in young adult drinking: an experience sampling study. *Psychopharmacology (Berl)*. 2020;237(5):1557–75. <https://doi.org/10.1007/s00213-020-05480-5>
 41. Emery NN, Stanton K, Baumgardner S, Simons JS, Douglass MA, Prince MA. Discrete emotions and global affect: applying empirically driven approaches to experience sampling data to model state and trait affective structure and affect-alcohol use associations in a heavy drinking young-adult sample. *Behav Res Ther*. 2023;167:104356.
 42. Bresin K, Fairbairn CE. The association between negative and positive affect and alcohol use: an ambulatory study. *J Stud Alcohol Drugs*. 2019;80(6):614. Available from: <https://pmc/articles/PMC6900989/>. <https://doi.org/10.15288/jsad.2019.80.614>
 43. Waters AJ, Schoenmakers TM, Snelleman M, Szeto EH, Franken IHA, Hendriks VM, et al. Affect, motivation, temptation, and drinking among alcohol-dependent outpatients trying to maintain abstinence: an ecological momentary assessment study. *Drug Alcohol Depend*. 2020 Jan;1(206):107626. <https://doi.org/10.1016/j.drugalcdep.2019.107626>
 44. Stene-Larsen K, Torgersen L, Strandberg-Larsen K, Normann PT, Vollrath ME. Impact of maternal negative affectivity on light alcohol use and binge drinking during pregnancy. *Acta Obstet Gynecol Scand*. 2013;92(12):1388–94. Available from: <https://onlinelibrary.wiley.com/doi/full/10.1111/aogs.12259>
 45. Tavalacci MP, Berthon Q, Cerasuolo D, Dechelotte P, Ladner J, Baguet A. Does binge drinking between the age of 18 and 25 years predict alcohol dependence in adulthood? A retrospective case-control study in France. *BMJ Open*. 2019;9(5):e026375. Available

- from: <https://bmjopen.bmj.com/content/9/5/e026375>. <https://doi.org/10.1136/bmjopen-2018-026375>
46. Addolorato G, Vassallo GA, Antonelli G, Antonelli M, Tarli C, Mirijello A, et al. Binge Drinking among adolescents is related to the development of Alcohol Use Disorders: results from a Cross-Sectional Study. *Sci Rep*. 2018;8(1):1–9. Available from: <https://www.nature.com/articles/s41598-018-29311-y>
 47. Burke LE, Shiffman S, Music E, Styn MA, Kriska A, Smailagic A, et al. Ecological Momentary Assessment in Behavioral Research: Addressing Technological and Human Participant Challenges. *J Med Internet Res*. 2017;19(3):e77. Available from: <https://pubmed.ncbi.nlm.nih.gov/28298264/>
 48. Maas CJM, Hox JJ. Sufficient sample sizes for multilevel modeling. *Methodology*. 2005;1(3):86–92. <https://doi.org/10.1027/1614-2241.1.3.86>
 49. FOD Volksgezondheid. Alcohol. 2024. Available from: https://www.health.belgium.be/nl/gezondheid/zorg-voor-jezelf/alcohol-tabak/alcohol#Alcohol_enjongeren
 50. National Institute on Alcohol Abuse and Alcoholism [NIAAA]. Drinking Levels Defined. 2022. Available from: <https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking>
 51. Van Damme J, Thienpondt A, Rosiers J, Tholen R, Soye V, Sisk M, et al. In hogere sferen Volume 5: Een onderzoek naar middelengebruik bij de Vlaamse studenten [Internet]. 2022. Available from: www.vad.be
 52. Meers K, Dejonckheere E, Kalokerinos EK, Rummens K, Kuppens P. mobileQ: A free user-friendly application for collecting experience sampling data. *Behav Res Methods*. 2020;52(4):1510–5. Available from: <https://link.springer.com/article/10.3758/s13428-019-01330-1>
 53. Mestdagh M, Verdonck S, Piot M, Niemeijer K, Tuerlinckx Francis, Kuppens P, et al. m-Path: An easy-to-use and flexible platform for ecological momentary assessment and intervention in behavioral research and clinical practice. 2022; Available from: <https://psyarxiv.com/uqdfs/>
 54. American Psychiatric Association. SCID-5-S Gestructureerd klinisch interview voor DSM-5 Syndroomstoornissen. Nederlandse vertaling van Structured Clinical Interview for DSM-5® Disorders– Clinician Version (SCID-5-CV). first. 2017.
 55. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG. The Alcohol Use Disorders Identification Test Guidelines for Use in Primary Care. 2001;
 56. Lataster T, Valmaggia L, Lardinois M, van Os J, Myin-Germeys I. Increased stress reactivity: a mechanism specifically associated with the positive symptoms of psychotic disorder. *Psychol Med*. 2013; 43(7):1389–400. Available from: <https://www-cambridge-org.kuleuven.e-bronnen.be/core/journals/psychological-medicine/article/increased-stress-reactivity-a-mechanism-specifically-associated-with-the-positive-symptoms-of-psychotic-disorder/DF668A72A0D4C09399FEAE1060C1D5F5>. <https://doi.org/10.1017/S0033291712002279>
 57. Collip D, Nicolson NA, Lardinois M, Lataster T, Van Os J, Myin-Germeys I. Daily cortisol, stress reactivity and psychotic experiences in individuals at above average genetic risk for psychosis. *Psychol Med*. 2011;41(11):2305–15. Available from: <https://www-cambridge-org.kuleuven.e-bronnen.be/core/journals/psychological-medicine/article/daily-cortisol-stress-reactivity-and-psychotic-experiences-in-individuals-at-above-average-genetic-risk-for-psychosis/CBD53523B589B8BEDB09E59677F9EF98>. <https://doi.org/10.1017/S0033291711000602>
 58. Rintala A, Wampers M, Myin-Germeys I, Viechtbauer W. Momentary predictors of compliance in studies using the experience sampling method. *Psychiatry Res*. 2020;286. Available from: <https://pubmed.ncbi.nlm.nih.gov/32146247/>
 59. Waddell JT, Sher KJ, Piasecki TM. Coping motives and negative affect: an ecological study of the antecedents of alcohol craving and alcohol use. *Psychol Addict Behav*. 2021;35(5):565–76. Available from: <https://pubmed.ncbi.nlm.nih.gov/33507786/>
 60. Kleiner KD, Gold MS, Frost-Pineda K, Lenz-Brunsmann B, Perri MG, Jacobs WS. Body Mass Index and Alcohol Use. *J Addict Dis*. 2008; 23(3):105–18. Available from: https://www.tandfonline.com/doi/abs/10.1300/J069v23n03_08
 61. Bray BC, Dziak JJ, Lanza ST. Age trends in alcohol use behavior patterns among U.S. adults ages 18–65. *Drug Alcohol Depend*. 2019; 205:107689.
 62. Barr DJ, Levy R, Scheepers C, Tily HJ. Random effects structure for confirmatory hypothesis testing: keep it maximal. *J Mem Lang*. 2013; 68(3):255–78. <https://doi.org/10.1016/j.jml.2012.11.001>
 63. Bates D, Kliegl R, Vasishth S, Baayen RH. Parsimonious mixed models. *ArXiv*. 2015.
 64. Kira B, Saptarshi ML, Thayslene M, Oliveira M, Nagesh R, Phillips T, et al. BSG Working Paper Series Variation in government responses to COVID-19. 2022; Available from: www.bsg.ox.ac.uk/covidtracker
 65. Jones A, Remmerswaal D, Verveer I, Robinson E, Franken IHA, Wen CKF, et al. Compliance with ecological momentary assessment protocols in substance users: a meta-analysis. *Addiction*. 2019; 114(4):609. Available from: <https://pubmed.ncbi.nlm.nih.gov/31492133/>
 66. Bragard E, Giorgi S, Juneau P, Curtis BL. Daily diary study of loneliness, alcohol, and drug use during the COVID-19 pandemic. *Alcohol Clin Exp Res*. 2022;46(8):1539–51. Available from: <https://onlinelibrary.wiley.com/doi/full/10.1111/acer.14889>
 67. Simons JS, Emery NN, Simons RM, Wills TA, Webb MK. Effects of Alcohol, Rumination, and Gender on the Time Course of Negative Affect. *Cognit Emot*. 2017;31(7):1405. Available from: <https://pubmed.ncbi.nlm.nih.gov/29156778/>
 68. Schmukle SC, Egloff B, Burns LR. The relationship between positive and negative affect in the positive and negative affect schedule. *J Res Pers*. 2002;36(5):463–75. [https://doi.org/10.1016/S0092-6566\(02\)00007-7](https://doi.org/10.1016/S0092-6566(02)00007-7)
 69. Smith GT, Cyders MA. Integrating affect and impulsivity: the role of positive and negative urgency in substance use risk. *Drug Alcohol Depend*. 2016;163:S3–S12.
 70. Pedersen SL, Kennedy TM, Holmes J, Molina BSG. Momentary associations between stress and alcohol craving in the naturalistic environment: differential associations for black and white young adults. *Addiction*. 2022;117(5):1284–94. <https://doi.org/10.1111/add.15740>
 71. Treloar Padovano H, Janssen T, Emery NN, Carpenter RW, Miranda R. Risk-Taking Propensity, Affect, and Alcohol Craving in Adolescents' Daily Lives. *Subst Use Misuse*. 2019;54(13):2218. Available from: <https://pubmed.ncbi.nlm.nih.gov/31492133/>
 72. Marlatt GA. Cognitive factors in the relapse process. In: Marlatt GA, Gordon JR, editors. *Relapse prevention*. New York: The Guilford Press; 1985. p. 128–200.
 73. Cheng TC, Lo CC. Change in Adolescents' Alcohol-Use Patterns, From Non-Drinking to Non-Heavy Drinking or Heavy Drinking. *J Drug Issues*. 2015;45(4):447–59. Available from: <https://journals-sagepub-com.kuleuven.e-bronnen.be/doi/full/10.1177/0022042615604013>
 74. Graham K, Massak A, Demers A, Rehm J. Does the Association Between Alcohol Consumption and Depression Depend on How They Are Measured? *Alcohol Clin Exp Res*. 2007;31(1):78–88. Available from: <https://onlinelibrary.wiley.com/doi/full/10.1111/j.1530-0277.2006.00274.x>
 75. Tamerin JS, Mendelson JH. The psychodynamics of chronic inebriation: observations of alcoholics during the process of drinking in an experimental group setting. *Am J Psychiatry*. 1969;125(7):886–99. <https://doi.org/10.1176/ajp.125.7.886>

76. Russell MA, Linden-Carmichael AN, Lanza ST, Fair EV, Sher KJ, Piasecki TM. Affect relative to day-level drinking initiation: analyzing ecological momentary assessment data with multilevel spline modeling. *Psychol Addict Behav*. 2020;34(3):434–46. <https://doi.org/10.1037/adb0000550>
77. Castillo-Carniglia A, Keyes KM, Hasin DS, Cerdá M. Psychiatric comorbidities in alcohol use disorder. *Lancet Psychiatry*. 2019;6(12):1068–80. Available from: <http://www.thelancet.com/article/S2215036619302226/fulltext>. [https://doi.org/10.1016/S2215-0366\(19\)30222-6](https://doi.org/10.1016/S2215-0366(19)30222-6)

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Leenaerts N, Vaessen T, Sunaert S, Ceccarini J, Vrieze E. Affective dynamics surrounding craving, non-heavy alcohol use and binge drinking in female patients with alcohol use disorder and controls: An experience sampling method study. *Addiction*. 2025;120(1):61–76. <https://doi.org/10.1111/add.16682>