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ORIGINAL ARTICLE

Quantifying heterogeneity in mood–alcohol relationships with idiographic causal models

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Abstract

Background: Ecological momentary assessment (EMA) studies have provided conflicting evidence for the mood regulation tenet that people drink in response to positive and negative moods. The current study examined mood-to-alcohol relationships idiographically to quantify the prevalence and intensity of relationships between positive and negative moods and drinking across individuals.

Method: We used two EMA samples: 96 heavy drinking college students (sample 1) and 19 young adults completing an ecological momentary intervention (EMI) for drinking to cope (sample 2). Mood and alcohol use were measured multiple times per day for 4–6 weeks. Mood–alcohol relationships were examined using three different analytic approaches: standard multilevel modeling, group causal modeling, and idiographic causal modeling.

Results: Both multilevel modeling and group causal modeling showed that participants in both samples drank in response to positive moods only. However, idiographic causal analyses revealed that only 63% and 21% of subjects (in samples 1 and 2, respectively) drank following any positive mood. Many subjects (24% and 58%) did not drink in response to either positive or negative mood in their daily lives, and very few (5% and 16%) drank in response to negative moods throughout the EMA protocol, despite sample 2 being selected specifically because they endorse drinking to cope with negative mood.

Conclusion: Traditional group-level analyses and corresponding population-wide theories assume relative homogeneity within populations in mood–alcohol relationships, but this nomothetic approach failed to characterize accurately the relationship between mood and alcohol use in approximately half of the subjects in two samples that were demographically and clinically homogeneous. Given inconsistent findings in the mood–alcohol relationships to date, we conclude that idiographic causal analyses can provide a foundation for more accurate theories of mood and alcohol use. In addition, idiographic causal models may also help improve psychosocial treatments through direct use in clinical settings.

KEYWORDS
alcohol use, casual analysis, ecological momentary assessment, idiographic, mood

INTRODUCTION

Heavy drinking is most common in early adulthood (Kanny et al., 2018), with regular heavy drinkers reporting an average of 7 drinks per episode. Heavy drinking carries an increased risk for serious injury, chronic diseases such as cancer, high blood pressure, stroke, heart disease, and liver disease, and impairment in work and educational functioning (Centers for Disease Control and Prevention [CDC], 2019). It is estimated that excessive drinking costs the United States $249 billion annually (CDC, 2019).

Several existing theories of problematic alcohol use focus on the role of alcohol in regulating emotions (e.g., Baker et al., 2004; Cooper et al., 1995), describing positive and negative mood regulation pathways. In positive mood regulation models, drinkers are hypothesized to drink in response to positive mood, and drinking is hypothesized to increase positive mood. In negative mood regulation models, drinkers are hypothesized to drink in response to negative mood, and drinking is hypothesized to reduce negative mood. These two pathways can operate within an individual, as positive and negative emotions are related but distinct constructs (Watson & Clark, 1994).

Ecological momentary assessment (EMA) studies are well-suited to test whether alcohol use serves as a mood-regulating behavior in naturalistic environments. In fact, many EMA studies have focused on the role of mood as a proximal precursor to alcohol use. For positive mood, EMA studies have found a relationship between higher positive mood and subsequent alcohol consumption (Armeli et al., 2008; De Leon et al., 2020; Dvorak et al., 2018; Emery & Simons, 2020; Hussong et al., 2001). For negative moods, studies have found conflicting results. Some EMA studies have found that elevated negative mood precedes alcohol use (Armeli et al., 2008; Dvorak et al., 2014), however, many studies have found no association between negative mood and subsequent alcohol use (Emery & Simons, 2020; Fairlie et al., 2019; Hussong et al., 2001; O'Donnell et al., 2019; Stevenson et al., 2020), and some studies have found an inverse relationship (i.e., decreased alcohol use following negative mood; De Leon et al., 2020; Treloar et al., 2015). This pattern of results is not strongly supportive of a causal connection between negative mood to drinking. However, there is evidence that the relationship between mood and alcohol use may vary by the severity of one's problems with alcohol use, initially starting out with celebratory and social drinking which progresses into a habit of drinking to cope with negative moods as one develops AUD (Koob, 2004). That is, it is often thought that college samples drink in response to positive mood but not negative mood, and clinical samples drink in response to negative mood. Therefore, studies on mood and alcohol use must be careful to consider the clinical status of the sample when making inferences about populations.

The studies informing mood regulation theories for drinking, like most psychosocial theories, rely on the overall, group–level relationship between mood and alcohol use to support or fail to support theories. This process necessarily averages together all participants in the sample and leaves individual variation in the mood–alcohol relationship relatively unknown. For example, if some individuals are more likely to drink due to negative mood and some are less likely, averaged group models may find no relationship or mixed findings, just as the EMA literature has found for negative mood and alcohol use.

Some researchers use multilevel models to estimate random slopes by the participant, which enables them to determine if the relationship between mood and alcohol use varies significantly by individual. These studies often find that individuals do significantly differ in their mood–alcohol slopes, showing that only a subset of drinkers uses alcohol in association with negative mood (e.g., Mohr et al., 2013; Schroder & Perrine, 2007; Wardell et al., 2013). These random slopes begin to reveal the extent of heterogeneity within samples, even in those samples that are relatively homogeneous in superficial ways (e.g., public university student drinkers aged 18–24, Wardell et al., 2013). An accurate estimate of the generalizability of nomothetic mood–alcohol theories requires more precise estimates of idiographic, or individual-level, mood–alcohol heterogeneity.

To more precisely evaluate the degree to which group results accurately characterize individuals within the group, we propose using idiographic analyses (Molenaar, 2004), which can then be summarized and described at the group level as well. Constructing personal models is feasible with EMA datasets due to the large number of observations collected per person (Stevenson et al., 2021). Further, because these models are specific to one individual and based on naturalistic data, they have clear implications for use in clinical settings. For example, personal models can be used to inform assessment, deliver personalized feedback to patients, personalize treatment plans, and even assess response to treatment (e.g., Morgenstern et al., 2014; Webb et al., 2021).

However, although EMA data can be temporally ordered, EMA studies are observational (i.e., without experimental manipulation) and therefore unable to determine causal relationships between measured phenomena using traditional analytic methods. Of course, uncovering causal relationships would be the ideal result for most EMA studies. For example, to find that happiness was not just associated with drinking, but that happiness *caused* by drinking, would allow for much more precise theory-building and intervention development. And now, with the development of causal search analytic methods, which are widely used in computer science, psychological researchers can estimate causal relationships in observational data (Eberhardt, 2017; Malinsky & Danks, 2018; Spirtes et al., 2000). Causal search methods rely on logic-based algorithms to identify the most likely causal relationships among the variables in the dataset. Causal search algorithms such as those we use for this manuscript are continuously refined and verified by other investigators, using direct experimentation (e.g., simulations in datasets with known causal relationships; Malinsky & Danks, 2018; Ramsey et al., 2017).

In contrast, most past EMA studies have utilized multilevel modeling (MLM) or structural equation modeling (SEM) to analyze associations between mood and drinking. Although robust in many ways, these analysis methods are limited in their ability to make causal inferences. In MLM, associations between pre-specified variables are found when two variables covary, after accounting for the

variance in other measured variables that are included in the model, but the pattern of relationships between all other variables in the model is not taken into account. In causal search algorithms like Fast Greedy Equivalence Search (fGES), the causal structure of all measured variables is analyzed to inform other relationships in the model (Malinsky & Danks, 2018). This information is used to infer which variables in the model are causally related to each other. Further, although some EMA studies use temporal ordering to inform their models, many EMA studies include contemporaneously-measured variables in models. In this situation, causal search algorithms like fGES can infer direction by systematically analyzing how all variables in the dataset relate to one another. As a result, the fGES algorithm has a superior ability to detect causal relationships as compared to regression-based methods, such as MLM.

In this paper, we used fGES algorithms to construct personal causal models of the momentary relationship between mood and alcohol use among two samples that varied in clinical severity: 18–20-year-old, heavy drinkers who were attending college full time (sample 1), and treatment-seeking young adults who were completing an intervention for drinking to cope (sample 2). Personal causal models were examined as they related to positive and negative mood regulation theories of drinking. Results from personal causal models were also compared to group-level causal models for each sample, and more traditional multilevel models for each sample. We expected to find considerable heterogeneity in mood–alcohol relationships among participants, but given the lack of prior research examining idiographic mood–alcohol relationships, we did not have specific hypotheses.

METHOD

Participants

Sample 1

Participants were $N = 96$ college students aged 18–20 (see Table 1 for details). Participants were eligible if they had access to a smartphone with a data plan, were enrolled in a 4-year college, and reported heavy drinking, defined as either consuming 4+/5+ drinks (for women/ men) in one episode or experiencing a negative consequence due to alcohol use in the last two weeks. Exclusion criteria were: using an illicit drug other than cannabis in the past two weeks and current treatment for any substance use disorder. $N = 100$ participants completed the EMA protocol, but only $N = 96$ reported consuming alcohol during the EMA study period (current analysis sample; see Merrill et al., 2021 for more details).

Sample 2

Participants were $N = 19$ young adults (aged 18–26) who attended a partial hospital psychiatric program at a private hospital in New England and were interested in completing an ecological momentary intervention (EMI) for drinking to cope with negative mood. Participants were eligible to participate if they used alcohol at least twice per week in the last month and reported drinking to cope, moderate to severe anxiety symptoms, and high risk for depression. $N = 20$ participants completed the EMA/EMI protocol, but only $N = 19$ reported consuming alcohol during the EMA study period (current analysis sample; see Blevins et al., 2021 for more details).

TABLE 1 Characteristics of samples

	Sample 1 ($N = 96$)	Sample 2 ($N = 19$)
Participants	Heavy drinking college students	In partial hospital treatment for mental health concerns. Endorsed drinking to cope.
Age	M = 18.67 (SD = 0.66) Range 18–20	M = 21.45 (SD = 2.28) Range 18–25
Sex		
Male	46 (47.9%)	9 (40.9%)
Female	50 (52.1%)	13 (59.1%)
Intersex	0 (0%)	0 (0%)
Gender		
Man	48 (50.0%)	9 (40.9%)
Woman	44 (45.8%)	13 (59.1%)
Genderqueer	3 (3.1%)	0 (0%)
Declined	1 (1.0%)	0 (0%)
Race		
White	70 (72.9%)	20 (90.9%)
Black	7 (7.3%)	1 (4.6%)
Asian	21 (21.9%)	0 (0%)
Native Am./Alaska Native	1 (1.0%)	0 (0%)
Pacific Islander	1 (1.0%)	0 (0%)
Multiracial	13 (13.5%)	1 (4.6%)
Hispanic ethnicity	14 (14.6%)	3 (14.3%)

Procedure

Sample 1

See Table 2 for a comparison of EMA protocols by sample. Participants completed a four-week EMA protocol that included daily morning reports (prompted at 7 AM), participant-initiated assessments at the beginning of each drinking episode, hourly assessments throughout each drinking episode until 1.5 hours after

TABLE 2 EMA protocols by sample

	Sample 1	Sample 2
Length of EMA	4 weeks	6 weeks
Assessments per day	1 scheduled per day, plus participant-initiated drinking reports and follow-ups. No random prompts.	4 scheduled, no separate drinking reports
Scheduled reports	7 am daily. 5 pm reports were completed the day after each drinking episode.	Randomly delivered during four time periods: 9 AM-12 PM, 12-3 PM, 3-6 PM, 6-9 PM
When was alcohol use reported?	During morning reports or in participant-initiated drinking assessments	During any of the scheduled reports
Other details	Follow-up assessments hourly during drinking episodes	Protocol included intervention designed to reduce alcohol use. Assessments were also available on demand.

the last drink, and 5 PM assessments the day after drinking. There were no random prompts in this protocol. Morning reports were retrospective assessments of drinking the prior day, including start and stop times, and current reports of mood. The other assessments collected current reports of mood and alcohol use (see Merrill et al., 2021 for more details).

Sample 2

Participants completed a six-week ecological momentary assessment and intervention (EMA/EMI) protocol after completing 5–10 days of partial hospital treatment for anxiety, depression, or another disorder. The partial hospital treatment program did not provide any treatment for alcohol or substance use disorders, but the EMI was aimed at reducing drinking to cope. The EMA/EMI protocol involved four scheduled surveys per day, prompted at a random time between 9 AM-12 PM, 12-3 PM, 3-6 PM, and 6-9 PM. Participants could also take assessments on demand. Surveys assessed alcohol use since the last assessment and current mood and delivered personalized messages to prompt coping skills if the subject endorsed high negative mood and urges to drink. See Blevins et al. (2021) for more details on the intervention and study procedure, and Stevenson et al. (2020) for more details on the EMA protocol.

Measures

Alcohol use

Sample 1

Participants were trained in reporting standard drinks prior to beginning the EMA protocol. For current alcohol use, participants initiated 'start drink' assessments and reported the time they took their first sip of alcohol and the number of standard drinks so far. Then follow-up assessments were delivered at 1-hour increments after the start drink report to assess the total number of drinks consumed so far in the drinking episode. The difference between the current and last assessments was calculated for each drink report to extract the number of drinks consumed since the last assessment (rather than the running total for the drinking episode). To measure yesterday's alcohol use, participants were asked "Did you drink yesterday?". If participants responded 'yes' to this question, they were asked the total number of drinks consumed yesterday and the start and end times for the drinking episode.

Sample 2

Participants were trained in reporting standard drinks prior to beginning the EMA protocol. During EMA, participants were asked at each assessment if they had consumed alcohol since the last assessment. If they responded yes, they were asked how many standard drinks they had consumed.

Mood

Sample 1

Current mood was measured by asking "How [sad, irritable, stressed, relaxed, happy, energetic] do you feel right now?" Participants responded on a scale from 0 to 6, with anchors at 0 (*not at all*), 3 (*somewhat*), and 6 (*extremely*). Current mood was assessed during all surveys, including start drink assessments and drink follow-ups.

Sample 2

Current mood was measured by asking "Please rate how you feel RIGHT NOW" using a scale of 1 (least) to 10 (most) for the following emotions: sad, angry, stressed, happy, excited. Current mood was assessed during all random surveys.

Data analysis plan

Lagged variables were created for each mood and for alcohol use, encoding their value at the previous assessment (within person). Observations that were completed more than 24 hours after the last survey were dropped (3% of surveys). In sample 1, alcohol use was measured during drinking episodes and morning reports. In the event that a subject reported alcohol use in a morning report that

was not reported during the drinking episode, the morning report for alcohol use was imputed at the appropriate start and end time the day prior (33 reports, or 7.1% of drink reports).

Two types of group analyses were performed separately for each sample. Multilevel negative binomial regressions (`menbreg` command in Stata 15.1) predicted alcohol use from lagged and momentary mood. All observations of mood and alcohol use (level 1) were nested within people (level 2). Intercepts were allowed to vary randomly by person, and random slopes were tested for significant mood–alcohol relationships. Time of day was included as a level 1 covariate. For both group analyses, variables were person-centered (i.e., centered on each person's mean) prior to group analysis, meaning that mean differences between subjects were removed and results focus only on within-person variance. Although multilevel models often also add the person means to the analysis, we opted to include only within-person effects in order to facilitate comparisons to the causal models. Coefficients from the multilevel models can be interpreted as the change expected in alcohol use for each unit change in the predictor. For example, if happiness is related to alcohol use at $B = 0.40$, then each unit increase in happiness (above a person's usual mean) is associated with an increase in drinks by 0.40 (in standard drinks).

The second group analysis was a causal model using the fast greedy equivalence search (fGES; Ramsey et al., 2017) algorithm in Tetrad. This algorithm is a modified version of Greedy Equivalence Search by Ramsey and colleagues to be faster without sacrificing accuracy. This algorithm searches the dataset for all possible causal relationships and iteratively adds and subtracts these relationships from the model until it reaches maximum goodness of fit. Previous studies have shown that fGES reliably converge on an accurate causal structure by using datasets generated from a known causal structure (Chickering, 2002; Ramsey, 2015). Personal causal models were estimated separately by individual using fGES. For all causal models, prior knowledge specified that variables could not cause observations that occurred earlier in time (i.e., lagged observations). The fGES algorithm has no tolerance for missing values, so we imputed (using information from other assessments) as many values as possible prior to analyses. For sample 1, the percentage of observations dropped per person varied from 0.8% to 21.1% ($M = 5.2\%$, $SD = 4.3\%$). For sample 2, the percentage of observations dropped per person varied from 0.6% to 18.8% ($M = 5.8\%$, $SD = 4.5\%$).

Causal analyses produce directed graphs with each significant pathway shown with an r value, which varies from −1 (strongest negative relationship) to 1 (strongest positive relationship), with values closest to 0 being weakest. To determine the total effect of each mood variable (X) on the number of drinks consumed (Y), let Paths(X,Y) be the set of all directed paths from X to Y, and $r(e)$ be the r value for the edge e. The total effect (TE) of X on Y is defined as: $TE(X,Y) = \sum_{P \in Paths(X,Y)} \prod_{e \in P} r(e)$. The total effect of X on Y is the model's predicted change in the variance of Y given a single unit increase in X. Since all variables were standardized, a single unit corresponds to 1 *SD*. For example, if happiness has a single directed path to alcohol use at $r = 0.40$, this means that alcohol use increases by 0.40 *SD* for each *SD* increase in happiness.

RESULTS

Descriptive statistics

In Sample 1, participants ($N = 96$) completed an average of 71.85 surveys ($SD = 13.24$, range 40–119) over 28 days (range 28–29) for an average of 2.5 surveys per day. Participants completed 99% of the morning reports (the only consistently scheduled assessments). They reported an average of 4.93 drinking episodes per person (range 1–14) containing 2.61 drinks per drinking assessment (i.e., the number of new drinks reported per survey when drinking; $SD = 1.73$, range 0.5–12) and 4.44 drinks per episode (i.e., the total number of drinks consumed from the time one started drinking to the time they stopped; $SD = 3.06$, range 1–18).

In Sample 2, participants ($N = 19$) completed an average of 104.15 surveys ($SD = 44.69$, range 38–169) over 34 days (range 15–49), for an average of 3 surveys per day. Participants completed an average of 52.88% of scheduled assessments. They reported an average of 11.35 drinking episodes per person ($SD = 10.06$, range 1–36) containing 2.71 drinks per drinking assessment ($SD = 1.95$, range 1–11) and 3.56 drinks per episode ($SD = 2.49$, range 1–11).

Group multilevel models

In sample 1, lower stress (both current- and lagged-assessment) was related to higher number of drinks consumed with small effect sizes (current stress: $B = -0.51$, $p < 0.001$; lagged stress: $B = -0.25$, $p < 0.001$). Current happiness, current energy, and lagged energy were positively associated with the number of drinks consumed with small effect sizes (happiness: $B = 0.17$, $p = 0.003$; energy: $B = 0.54$, $p < 0.001$; lagged energy: $B = 0.19$, $p < 0.001$). Random slopes were tested for all significant moods and results uniformly indicated that there was significant variance among individuals in the relationship between each of these moods and alcohol use.

In sample 2, only lagged excitement was associated with a higher number of drinks consumed with a small effect size ($B = 0.13$, $p = 0.011$). Random slopes also indicated significant between-person variance in the relationship between lagged excitement and the number of drinks consumed.

Group causal models

See Figure 1 for the group causal model for sample 1. When including all 96 participants (a total of 6408 observations) in the causal analyses, we found that feeling energetic ($r = 0.200$, $p < 0.001$) and less stressed ($r = -0.150$, $p < 0.001$) were causally related to increased alcohol use. Higher energy at the last assessment

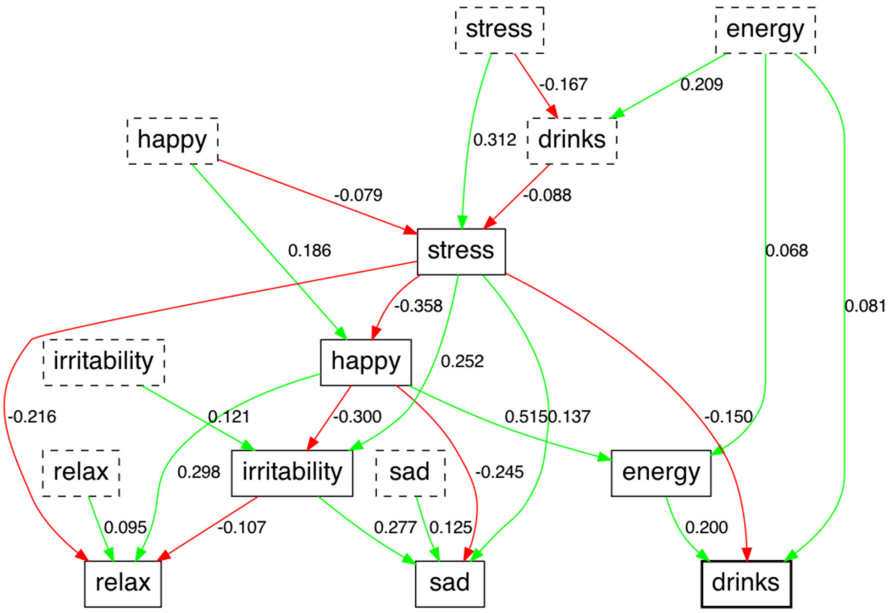

FIGURE 1 Group causal model for sample 1 (heavy drinking college students). Dashed lines indicate lagged variables (one assessment prior). Numbers indicate the standardized edge weight, or *r* of the relationship. *p* values were uniformly <0.001 for all edges due to large sample size and preference for causal discovery analyses to generate sparse models with only strong pathways. Arrows indicate the direction of causality. Red paths are negative relationships, green paths are positive.

FIGURE 2 Group causal model for sample 2 (treatment-seeking young adults who drink to cope, *N* = 19). Dashed lines indicate lagged variables (one assessment prior). Numbers indicate the standardized edge weight, or *r* of the relationship. *p* values were uniformly <0.001 for all edges due to large sample size and preference for causal discovery analyses to generate sparse models with only strong pathways. Arrows indicate the direction of causality. Red paths are negative relationships, green paths are positive.

was also causally related to an increased number of drinks at the last ($r = 0.209$, $p < 0.001$) and current assessments ($r = 0.081$, $p < 0.001$). For the smallest effect size, on average across subjects, a 1.51-unit increase in energy at the last assessment (on a scale of 0–6; 1 *SD*) led to a 0.08-drink increase (0.081 *SD*) per assessment. For the largest effect size, a 1.51-unit increase in current energy (on a 0–6 scale; 1 *SD*) led to a 0.19-drink increase (0.200 *SD*) per assessment.

See Figure 2 for the group causal model for sample 2. When including all 19 participants (a total of 1869 observations) from sample 2 in the causal analyses, we found that a higher level of excitement was causally related to alcohol use at the next assessment ($r = 0.087$, $p < 0.001$). A 2.14-unit increase in excitement (on a 1–10 scale; 1 *SD*) was related to a 0.11-drink increase (0.087 *SD*). No other moods were related to drinks consumed in the group model.

Personal causal models

In sample 1, 58 of 96 individuals' models (60.4%) contained only pathways consistent with positive mood regulation (i.e., positive mood was causally related to drinks consumed; average strongest mood's $r = 0.33$, range: 0.02, 0.62), 23 (24.0%) had no causal pathways between mood and alcohol use ($r = 0.00$), 3 individuals' models (3.1%) contained only pathways consistent with negative mood regulation (average strongest $r = 0.36$, range: 0.32, 0.40) and 2 people (2.1%) had pathways consistent with both positive and negative mood regulation (average strongest $r = 0.41$, range: 0.31, 0.52; see Figure 3). The remaining 10 individuals only had negative relationships between mood and alcohol; 9 drank *less* due to stress and irritability (average strongest $r = -0.35$, range: −0.50, −0.25) and 1 drank *less* when their energy at the previous assessment was higher ($r = -0.32$).

In raw drinks, among those with positive mood–alcohol pathways only, subjects drank on average 0.28 more drinks when their

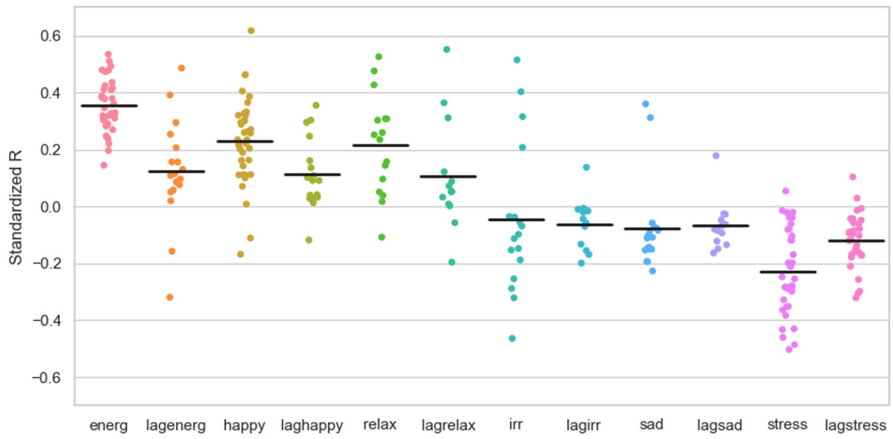

FIGURE 3 Effect sizes of mood on drinks consumed, sample 1 (heavy drinking college students). Dots represent individuals and lines represent the group average for each mood. All individuals whose models revealed a causal relationship between a mood and drinking are shown on this figure. The same individual may have had a causal relationship between multiple moods and drinking.

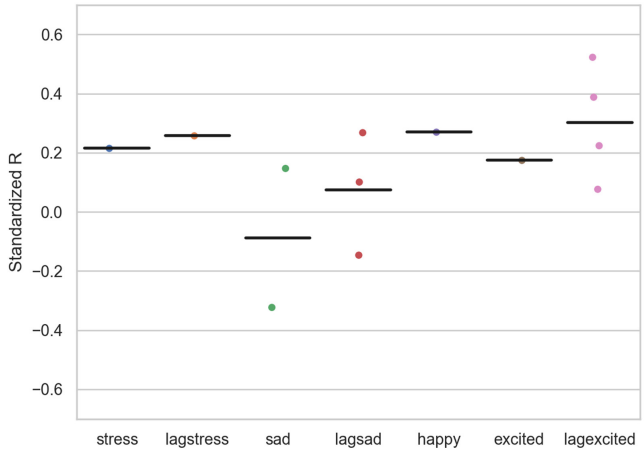

FIGURE 4 Effect sizes of mood on drinks consumed, sample 2 (treatment-seeking young adults who drink to cope). Dots represent individuals and lines represent the group average for each mood. All individuals whose models revealed a causal relationship between a mood and drinking are shown on this figure. The same individual may have had a causal relationship between multiple moods and drinking.

most strongly-related positive mood increased by 1 *SD* (range 0.02, 0.84). Among those with both positive and negative pathways, subjects drank on average 0.41 more drinks (range 0.20, 0.61) when their most relevant mood increased by 1 *SD*. Among those with only negative mood pathways, drinks increased by an average of 0.18 (range 0.05, 0.26) for each *SD* increase in a negative mood. Among those who only drank less in response to mood, drinks decreased by an average of −0.39 with each *SD* increase in the most relevant mood (range −0.58, −0.18).

In sample 2, 11 of 19 individuals (57.9%) had no causal pathways between mood and alcohol use, 4 individuals' models (21.1%) contained only pathways consistent with positive mood regulation (average strongest $r = 0.35$, range: 0.22, 0.52), and 3 individuals' models (15.8%) contained only pathways consistent with negative mood regulation (average strongest $r = 0.25$, range: 0.22, 0.27; see Figure 4). The remaining individual drank less when sad ($r = -0.32$). No individuals demonstrated both positive and negative mood regulation pathways in this sample.

In raw drinks, among those with positive mood–alcohol pathways only, subjects drank on average 0.35 more drinks when their most strongly-related positive mood increased by 1 *SD* (range 0.22, 0.52). Among those with negative mood–alcohol pathways only, subjects drank on average 0.25 more drinks when their most strongly-related negative mood increased by 1 *SD* (range 0.22, 0.27).

Case studies

To illustrate the degree of inter-individual variation and potential clinical utility, we next characterize two personal causal models from each sample. In sample 1, we highlight two cases (see Figure 5). The first personal causal model (on the left side of Figure 5) was based on 47 completed surveys and 5 drinking episodes ($M = 4.25$ drinks, range 2–7). For this person, feeling irritable was causally related to alcohol use. In raw numbers, for this individual, a 0.83-unit increase in irritability (on a scale of 0–6; 1 *SD*) was related to a 0.61-drink increase in alcohol use (0.515 *SD*). We can observe that, although irritability has the most proximal relationship with drinking, sadness is the most consequential emotion in this graph, as it leads to reduced relaxation, happiness, and energy, as well as increased irritability, stress, and drinking. Therefore, clinical interventions to decrease alcohol use for this individual would likely focus on addressing both sadness and irritability.

The second personal causal model in Figure 5 (right side) was based on 79 completed surveys and 5 drinking episodes ($M = 4.6$ drinks, range 3–5). For this person, feeling energetic increases their alcohol use. In raw numbers, for this individual, a 1.78-unit increase in energy (on a scale of 0–6; 1 *SD*) led to a 0.45-drink increase (0.535 *SD*). We can also note a drink 'cycle' with energy: alcohol use increased energy, which in turn increased alcohol use. An intervention to decrease alcohol use for this individual might focus on breaking

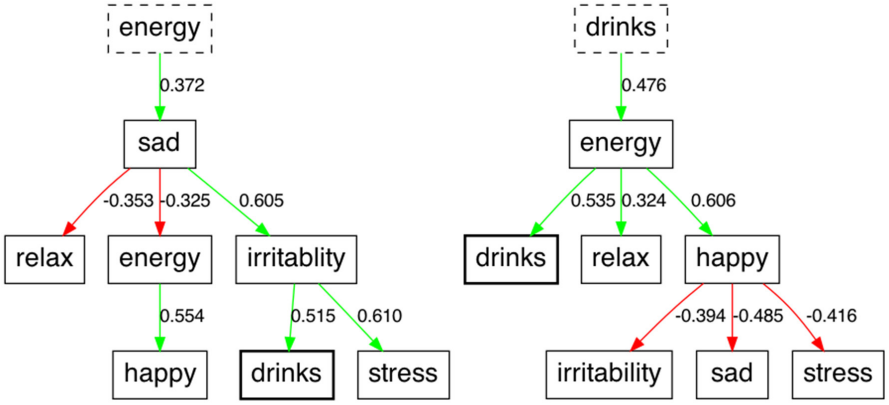

FIGURE 5 Two personal causal models from sample 1. Dashed lines indicate lagged variables (one assessment prior). Numbers indicate the standardized edge weight, or *r* of the relationship. *p* values were uniformly <0.001 for all edges due to large sample size and preference for causal discovery analyses to generate sparse models with only strong pathways. Arrows indicate the direction of causality. Red paths are negative relationships, green paths are positive.

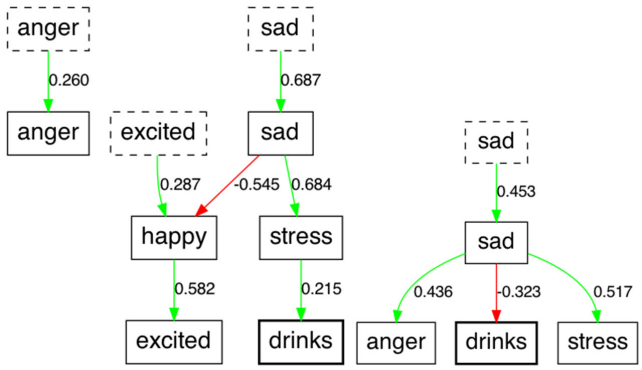

FIGURE 6 Two personal causal models from sample 2. Dashed lines indicate lagged variables (one assessment prior). Numbers indicate the standardized edge weight, or *r* of the relationship. *p* values were uniformly <0.001 for all edges due to large sample size and preference for causal discovery analyses to generate sparse models with only strong pathways. Arrows indicate the direction of causality. Red paths are negative relationships, green paths are positive.

this cycle and introducing alternative strategies to feel energetic since alcohol use is known to be effective at increasing a feeling of energy for this individual.

We will also highlight two cases from sample 2 with a causal relationship between sadness and drinking, one positive and one negative (see Figure 6). The first model (on the left side of Figure 6) was estimated based on 132 completed surveys and 12 drinking episodes ($M = 2.17$ drinks, range 1–4). In this model, sadness decreased positive emotions and increased stress, which in turn increased alcohol use. A 2.30-unit increase in stress (on a scale of 1–10; 1 *SD*) led to a 0.22 *SD* increase in alcohol use (0.10 drinks). Intervention for this person may focus on coping with sadness, which was self-sustaining from one assessment to the next, and led to drinking through stress.

The second model (on the right side of Figure 6) was estimated based on 76 completed surveys and 10 drinking episodes ($M = 1.5$ drinks, range 1–4). This individual's sadness was also self-sustaining, but for this person, sadness decreased alcohol use and increased anger and stress. A 0.77-unit increase in sadness (on a scale of 1–10; 1 *SD*) led to a 0.32 *SD* decrease in alcohol use (0.12 drinks). In this case, although increasing sadness would theoretically reduce drinking, increasing sadness is not advisable. For models like this one, it will be particularly important to utilize additional information from clinical intake assessments and explore with the patient why sadness led to less drinking (e.g., perhaps they found it was more helpful to engage in other activities when sad), and apply those strengths to reducing alcohol use.

DISCUSSION

The present study compared differences in EMA-measured mood–alcohol relationships using three analytic approaches (multilevel modeling, and group-level and idiographic causal modeling) in heavy-drinking college students (sample 1) and young adults who were completing an EMI for drinking to cope (sample 2). Despite recruiting sample 2 specifically for drinking to cope, results were similar between the two group analyses, finding only support for a positive mood regulation model of drinking. However, idiographic analyses revealed considerable heterogeneity between people in mood–alcohol relationships. In the personal causal models, many people had either no relationship between mood and alcohol use or a positive relationship between positive mood and alcohol use. A very small percentage of people drank in response to negative moods in both samples.

Both group analyses produced results consistent with a positive mood regulation model of alcohol use. However, when personal causal models were generated for the same individuals, positive relationships between positive mood and alcohol were absent for a strikingly high percentage of our more clinical sample (79% of sample 2) and a large minority of the heavy drinking college student sample (38% of sample 1). This finding is even more surprising considering that the samples studied were quite homogeneous in race, age, and

clinical severity. This raises an important question for researchers to consider about existing theories: what percentage of people do not conform to group-level results and their corresponding theories? And what is the highest percentage that is acceptable?

Although both group analyses found similar relationships between positive moods and alcohol use, the conclusions we can draw from their results differ in utility. Because mood and alcohol use were measured at the same time (except for the lagged variables), the multilevel model can only say that positive mood and alcohol use were related, but this analytic method cannot discover the direction of the relationship. In contrast, the group causal model was able to show a direction; that an energetic, excited mood very likely increases alcohol use. Both models cannot rule out the possibility of a third variable (e.g., positive mood prior to drinking events could be due to anticipation of a fun event), but the causal models can discover the direction of the relationship. In these datasets, we could have also inferred direction using multilevel modeling by restricting models to temporally ordered assessments (i.e., only modeling relationships between lagged observations and current observations) but the lag between assessments in samples 1 and 2 was on average 9 and 7 hours, frequently spanning overnight. This amount of separation between mood and alcohol use may be too much time to observe the real-time connection between these two constructs. Therefore, it may be preferable to use concurrent measurements of mood and alcohol use and employ causal modeling to discover directional relationships between variables that were measured at the same time. These methods would also have clear utility in investigating the relationship between alcohol use on mood as well.

Discovering that positive mood and alcohol use are causally related (not just associated) for specific individuals increases the likelihood that interventions focused on mood will have the ability to effect change in alcohol use. Further, the causal models have the ability to examine relationships between all variables in the model, rather than testing the relationship between predefined predictors and a single predefined outcome, as is necessary for standard multilevel regression. For these reasons (i.e., the discovery of direction between concurrent assessments, stronger causal inferences, ability to generate complex networks), we believe that causal analyses will provide a path forward from the era of finding "associations" among EMA data and enable researchers to identify the variables that are most implicated in causing their outcome of interest.

Of the mood–alcohol relationships that were found, it is important to note that some were not strong enough to be meaningful. Though there is no agreed upon limit for a meaningful effect size, changing the strongest-related mood by one standard deviation would change alcohol use by half of a drink or less for almost 90% of the sample.

Notably, there were substantial portions of both samples who had no significant mood–alcohol relationships (24% of sample 1 and 58% of sample 2). This was especially surprising for sample 2, which was selected due to trait-level endorsement of coping motives (i.e., drinking in response to negative mood), though it is also important to note that this sample was completing an intervention intended to decouple negative mood and alcohol use, potentially reducing the likelihood that we would observe these relationships, or the strength of them. In addition, this sample had just completed a partial hospitalization program to treat mood disorders among other psychiatric conditions, and many of them were taking psychiatric medications, which may have also changed the relationship between mood and alcohol use. Nonetheless, the finding that only 16% of this sample drank in response to the negative mood is consistent with existing evidence that endorsing coping motives on general (trait-level) questionnaires does not correspond to using alcohol in response to negative mood in daily life (Dora et al., under review).

The finding that a quarter to a half of people simply had no relationship between naturalistic mood and alcohol use is novel for the field of alcohol research and was made possible by idiographic analysis. This finding may provide insight the needed to resolve two challenges facing the field: (1) Prior EMA studies on the relationships between mood and alcohol use have not found consistent results, particularly for negative mood. This may be because large swaths of drinkers do not drink in response to any mood, so some samples tend toward showing a relationship and some samples tend in the opposite direction. Future EMA studies, and studies that have only previously used group-level analysis methods, may use idiographic analyses to explore the percentage of people who drink in response to negative mood, positive mood, both, or neither. Studies using burst designs (i.e., several separate periods of monitoring) may explore the within-person stability of EMA-measured drinking tendencies. In addition, future studies should attempt to determine what participant and context-specific characteristics distinguish these drinking patterns. Additionally, (2) Given the widespread use of mood regulation theories for alcohol use, many treatments for AUD have been founded on the premise that alcohol is used to regulate mood (e.g., Kadden, 1995). However, if many people do not drink in response to any mood, then treatments based on this premise will not be effective for those people. Future studies can investigate this question by examining how idiographic drinking patterns predict treatment outcomes in samples with alcohol use disorder (unlike the samples studied here). Indeed, some past research has found that CBT for AUD works best for those who report that they drink to cope (Anker et al., 2016). It is possible that other idiographic mood–alcohol patterns may be related to treatment response as well.

Overall, the two primary findings in this study–(1) many people did not drink in response to mood at all and (2) substantial heterogeneity within those who did drink in response to mood–call into question the utility of broad, group-level theories of mood regulation for alcohol use. The degree of heterogeneity is even more surprising when one considers that the two samples studied were quite demographically and clinically homogeneous. If the group result was not consistent with the individual models for more than half of the people in these relatively homogeneous groups, this raises serious concerns about the application of nomothetic mood–alcohol theories to more diverse populations. Nonetheless, idiographic analyses are limited to the variability present within one person and cannot discover if there are differences between people on average. Therefore,

we propose that idiographic analyses be used in conjunction with between-person (nomothetic) analyses to fully characterize individual heterogeneity (and group patterns in heterogeneity) while also studying group trends and interindividual variance.

Clinical implications

In terms of clinical utility, around half of the models produced in this study contained direct pathways between a mood and alcohol use that could be targeted for intervention. However, most of these pathways were positive moods leading to alcohol use, and unlike people who drink to cope with negative mood, there is no agreed-upon intervention strategy for those who drink in response to a positive mood. For these cases, it may nonetheless be informative for patients to see the naturalistic connections between their mood and drinking, and intervention techniques may be modeled after those that have been effective in young adult populations, who frequently drink in relation to positive moods, such as normative interventions, increasing protective behavioral strategies, and motivational interviewing.

The other half of the models mostly showed no relationship between mood and alcohol use, which is still clinically informative in that the clinician and patient can rule out mood regulation as the underlying mechanism for maintaining alcohol use. Particularly, if the patient self-reports using alcohol to cope or enhance their mood, demonstrating the lack of a relationship between mood and alcohol use in their EMA-informed causal model may highlight that there are many instances when the patient does not use alcohol in response to mood, which presents an opportunity to observe and further develop self-efficacy, an important predictor of treatment success (Project MATCH Research Group, 1998). Lastly, a small number of models showed that a negative mood resulted in decreased drinking. In these cases, though it may result in decreased drinking, it would not be advisable to increase negative mood. Instead, the clinician and patient can explore what the patient does to cope with their negative mood instead of drinking, using this strength and insight to successfully deploy alternative skills in situations when they may wish to reduce drinking.

Limitations

It is important to note that neither of the samples in this study was originally collected for the purpose of idiographic modeling, so future studies could include additional variables relevant to drinking in every assessment, such as social setting, alcohol cues, intentions, and urges (Beckjord & Shiffman, 2014). Though some of these variables were included in the original studies, idiographic models require a high number of observations, so these variables would need to be measured at every assessment (as were mood and alcohol use), not just daily. Ideally, variables relevant to each person's clinical presentation would also be included (e.g., Frumkin et al., 2021). This would improve the clinical utility of graphs produced and a broader understanding of momentary (i.e., proximal) antecedents to drinking. Future studies should also be attentive to the possibility that mood–alcohol relationships will differ as a function of recovery status. For example, a habitual drinker who is not attempting to quit may have no mood–alcohol relationship because they drink every day regardless of mood. In contrast, when the same drinker is attempting to reduce or stop drinking, they may only drink in response to a heightened positive or negative mood. Thus, considering one's recovery status may clarify the relationship between mood and alcohol use for some populations. In the same vein, the samples in this study were not receiving professional treatment for alcohol use disorder per se, though the second sample was completing an EMI for drinking to cope. It is possible that those with current AUD will differ in mood–alcohol relationships from those without AUD, so the nature of these relationships in AUD samples is a question for future research.

In addition, the time lag between assessments was variable for both samples. For example, when lagged drinks predicted current energy levels, it is unknown whether energy increased an hour later or 12 hours later. Further, because the models measured mood and drinking before, during, and after drinking events, we are unable to separate moods that prompt drinking initiation versus moods that maintain an ongoing drinking episode (i.e., both of these will appear as a predictor of drinking in the multilevel and causal models). We also focused almost exclusively on mood as a cause of drinking and did not use these data to examine the effects of drinking on mood, an important part of mood regulation theories for alcohol use.

Idiographic modeling generally is limited in its inability to examine variance that does not occur within one subject. For example, if a subject always reports high levels of sadness, their personal model will not be able to reveal the impact of reducing sadness. Idiographic models also require an adequate sampling of the behavior of interest within every person, which is not always possible. For example, 5 out of 120 subjects recruited for these studies did not drink at all and another 7 drank only one or two times during the EMA protocol, for a total of 10% of participants who likely did not have enough drinking episodes to produce a reliable personal model. Lastly, the causal modeling algorithm we used dropped each row (i.e., each EMA assessment) with any missing data, resulting in an average of 5–6% of rows being dropped, most of which contained some data. Causal discovery algorithms are being continually refined and modified, but at present there is a lack of algorithms that handle missing data in more sophisticated ways.

CONCLUSION

The current study found that group-level analyses, such as traditional multilevel modeling and group causal modeling, found exclusive support for a positive mood regulation model of alcohol use in two ecological momentary assessment (EMA) samples: heavy drinking college students (sample 1) and young adults who were completing

an intervention for drinking to cope with negative mood (sample 2). However, idiographic analyses revealed that only 63% and 21% of subjects (in samples 1 and 2, respectively) actually drank in response to a positive mood. Instead, large portions of both samples did not drink in response to any mood, and a very small portion of each sample drank in response to negative mood, even the sample that was recruited for drinking to cope. These results highlight the substantial heterogeneity in naturalistic mood-to-alcohol relationships even within samples that are relatively homogeneous. This heterogeneity may help to explain both the inconsistent prior findings for mood regulation theories on alcohol use and the low response rate to AUD treatments, which are often based on the tenet that people use alcohol to regulate their mood in their daily lives.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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