Neural moderators of social influence susceptibility on drinking

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Abstract

Conversations shape future behaviors. However, individuals vary in susceptibility to conversational influence and in neural responses tracking such influences. We examined whether activity in brain regions associated with social rewards and making sense of others' minds relates to a common behavior—drinking, following conversations about alcohol. We studied ten social groups of college students (N = 104 students; 4760 total observations) across two University campuses. We collected whole-brain fMRI data while participants viewed photographs of peers with whom they tended to drink at varying frequencies. Next, using ecological momentary assessment, we tracked alcohol conversations and drinking twice daily for 28 days. On average, talking about alcohol was associated with a higher probability of next-day drinking. Controlling for baseline drinking, participants who responded more strongly to peers with whom they drank more frequently—in brain regions associated with social rewards and mentalizing—showed higher susceptibility to conversational influence on drinking. Conversely, stronger neural responses to peers with whom they drank less frequently decoupled the link between alcohol conversations and next-day drinking. We conceptually replicate prior findings linking conversations and drinking in a longitudinal setting and provide new evidence that brain sensitivity to peers may exacerbate or buffer conversational susceptibility to drinking.

Key words: functional neuroimaging, health behavior, alcohol use, EMA (ecological momentary assessment), social influence, social groups

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Introduction

Humans are inherently social beings. Many of our everyday decisions, including what we eat, drink and buy, are shaped by the actions, beliefs, and emotions of other individuals[1-4]. This phenomenon—social influence—has been widely studied across many fields and under different operationalizations. One major pathway to influence involves interpersonal communication[5,6]. Online and offline conversations about health topics can affect a wide range of behaviors such as future alcohol use[7-9], dietary habits[10,11], exercise[12,] and smoking cessation[13]. However, individuals vary widely in how likely they are to change their behavior in response to health-related conversations[14.]

What kinds of psychological processes help explain susceptibility to health-related conversations? A window into the nature of these processes is offered by functional neuroimaging (fMRI), which can identify neural systems associated with affective and social cognitive processes implicated in social influence, and how their engagement varies across individuals[15]. To examine between-person differences in who is more (or less) susceptible to health-related conversations, we drew on recent paradigms that combine fMRI with ecological momentary assessment (EMA)[16]. fMRI can capture real time insights into how individuals process social cues[17] and integrate inputs into behavior change[18], while EMA can track health behaviors in real-world settings through repeated text message surveys[19]. In this study, we combined fMRI and EMA to examine how individual differences in neural responses to social cues—specifically, the faces of peers—relate to susceptibility to conversational influence, or the likelihood of drinking following alcohol conversations[9,17]. We focused on alcohol use as a

prevalent behavior that influences health and well-being, particularly among a key population of young adults[20] and in college settings[21].

Neuroimaging studies have suggested the key role of several core brain systems in processing social cues and subsequent behavior change, including the reward system[22,23] and the mentalizing system[24–26]. The reward system, including the bilateral ventral striatum, dorsal striatum (i.e., caudate and putamen), and ventromedial prefrontal cortex, is implicated in tracking rewards, for example, social rewards (e.g., approval from friends), food, or monetary rewards[27]. Individual differences in reward-related activity to social cues consistently correlate with conformity; with stronger reward-related brain activity and connectivity associated with increased susceptibility to conform across substance use[28], risky driving[29], food choices[30], and mobile app rating contexts[24,31]. A theoretical perspective underlying these findings suggests that humans value alignment with others, which fosters social connections and shared experiences[32,33].Stronger activity in reward-related brain regions may reflect the anticipation (or experience) of social rewards from interpersonal alignment and thereby reinforce future conformity promoting behaviors[34]. Thus, expecting or experiencing rewards (e.g., social approval), as reflected by stronger activity in the reward system, may also increase susceptibility to influence on future drinking.

Consistent with this perspective, college drinking is largely social[21,35] and shaped by social learning, where individuals often conform to drinking behaviors that are normative within their peer group[36]. Many students experiment with alcohol in the presence of peers and report drinking due to a desire to conform[37]. In parallel, in the brain, peer influence is often modulated by reward processing[28], with evidence that stronger reward activity to anticipated social rewards moderates the link between peer norms and risk-taking susceptibility among adolescents[38].

Consistently, we anticipate that those with stronger reward responses to drinking peers (versus non-drinking) peers may be more prone to drink after alcohol conversations than those without these reward-related brain patterns. Building on prior work[2,35], we define drinking-peers as peers from existing social groups with whom one drinks frequently and non-drinking peers as peers with whom one drinks rarely or never.

In addition to the brain's reward system, the mentalizing system is implicated in social influence and plays a central role in inferring and predicting mental states, or what peers think and expect in social settings[26,39]. Key regions involved in the mentalizing system, include the dorsomedial prefrontal cortex (dmPFC), temporoparietal junction (TPJ), posterior superior temporal sulcus (pSTS), and posterior cingulate cortex (PCC)[26,40]. Numerous studies have linked stronger activity in mentalizing-related regions, associated with understanding mental states, with increased conformity among both young adults and adolescents[24,25,31,41–44]. Stronger activity in mentalizing-related regions may render peers' viewpoints as more influential inputs to health decision-making[34]. For example, when encountering drinking influences, individuals with stronger mentalizing related activity may be more likely to consider their peers' perspectives in subsequent decisions to drink. We reasoned that, within college drinking contexts, individuals who show stronger activity within mentalizing-related brain regions, possibly indicative of sensitivity to peer mental states and expectations, may also show increased susceptibility to conversational influences on drinking. Together, previous work points to the value of considering neural responses in both the reward and mentalizing systems to better understand individual differences in conversational influence susceptibility to drink.

One key source of social influence involves peers. Peers can influence behaviors like alcohol use directly (i.e., through feedback and conversations) and indirectly (i.e., through

mental representations)[45,46]. For example, mental representations of peers may indirectly encourage drinking by making local drinking norms salient, even without explicit peer pressure, or by amplifying effects of alcohol conversations[47,48]. Specifically, mental representations of existing peers with whom one drinks often vs. rarely may evoke different values attached to drinking[49], potentially exacerbating or dampening susceptibility to conversational influences on drinking[50].

In parallel, the brain spontaneously tracks information about peer traits and behaviors during passive face processing. Knowledge and existing schemas about peers can be spontaneously retrieved during passive face processing[51], and social attributes, such as status, can be tracked by activity in neural regions associated with reward and mentalizing[17,52,53]. Such schemas allow individuals to store and activate information about others rapidly via cognitive shortcuts or heuristics[54]. These mental schemas may become more (or less) activated during alcohol conversations, thereby moderating susceptibility to drink. For instance, stronger brain activity in response to the faces of drinking (vs. non-drinking) peers may more readily evoke alcohol-related schemas and anticipated social rewards, thereby amplifying pro-drinking influences. Bridging these disparate areas of research, individual differences in brain activity to one type of social signal—a peer's face—may evoke different information about norms, peer expectations, or anticipated rewards. Individuals who more strongly call these social schemas to mind might then show greater susceptibility to conversational influence on drinking, depending on their prior drinking interactions with a given peer[2,35].

With the above considerations in mind, we examined how individual differences in reward and mentalizing-related activity to faces of drinking vs. non-drinking peers relate to conversational susceptibility—the link between talking about alcohol and next-day drinking. We

combined fMRI, with real-world social group information, and twice-a-day EMA over 28-days, to capture conversations and drinking within students' daily lives[9,19]. We focused on existing, on-campus social groups where drinking is common[55] and exacts a significant toll on the health, intellectual, and social lives of students[56]. Drawing on prior work suggesting that individuals with stronger neural sensitivity to social cues are more susceptible to social influence[24,29,31,42], we hypothesized that individuals who show stronger reward and mentalizing activity to the faces of drinking (vs. non-drinking) peers, will be more susceptible to drinking following alcohol conversations.

Results

Alcohol Conversations, Alcohol use, and Brain Activity

Throughout the 28-day EMA period, 99 out of 104 participants (95%) reported drinking at least once and 97 (93%) and reported at least one alcohol conversation. The average number of drinking occasions was 7.69 over 28 days (median = 6; SD= 6.07; range=0-28) and the average number of alcohol conversations was 9.46 (median = 8; SD=7.59; range = 0-34). Participants were highly compliant with the study protocol, responding to a median of 95% (53/56) of alcohol conversation prompts (*M* = 50.17; *SD* =7.8; range =13-56) and 96% (53.5/56) of EMA alcohol use prompts (*M* = 51.03; *SD* =7.2; range = 18-56). In total, we collected 4760 EMA data points. We observed large individual differences in neural responses to peer faces, with grand mean centered raw units of brain responses in the reward system ranging from -0.56 to 0.54 (median =-.01, SD = 0.24) and mentalizing activity ranging from -.84 to 0.80 (median =-.03; SD = 0.31). Correlations between key study variables can be found in Supplement B Table S8.

Neural Responses to Peer Faces, Alcohol Conversations and Alcohol Use

We found that talking about alcohol was associated with an increased likelihood of drinking the following day (OR =1.59, 95% CI [1.30-1.94], p <.001). We next tested whether including a neural index of activity to the faces of a drinking vs. non-drinking peers, as a moderator, would provide additional information about who is more (vs. less) susceptible to drinking following alcohol conversations (and would improve the model fit as measured via Akaike information criterion [AIC][57] and chi-square tests). Specifically, we tested whether including an interaction term between alcohol conversation and neural activity to drinking (vs. non-drinking) peers would improve model fit relative to (i) a main effects model (alcohol conversation and neural activity to peers as separate predictors) and (ii) a null model (no information about alcohol conversations and neural responses). We repeated this comparison twice to consider neural activity in the reward and the mentalizing regions separately, as presented below.

Reward and Mentalizing ROIs

The two-way interaction model (reward activity*alcohol conversation) significantly improved model fit (AIC = 3697.4), compared to a main effects model for alcohol conversation and activity in reward-related regions (AIC = 3699.6) and to a null model (AIC = 3746.9). Chi-squared tests showed that the interaction model significantly improved fit over the main effects model ($\chi^2(1)=4.16$, p = .041) as well as the null model ($\chi^2(1)=57.45$, p = <.001). We observed similar patterns when considering mentalizing-related activity. The two-way interaction model (mentalizing activity*alcohol conversation) significantly improved model fit (AIC = 3695.7) compared to a main efforts model for alcohol conversation and mentalizing activity (AIC= 3699.5) and to a null model (AIC =3746.9). The interaction model significantly improved fit

over the main effects model ($\chi^2(1)=6.05$, $p = .014$) and the null model ($\chi^2(1)=59.16$, $p = <.001$). Together, considering individual differences in neural responses to drinking (vs. non-drinking) peer faces in the reward and mentalizing systems explained additional differences in conversational susceptibility to drink.

Neural responses to peer faces moderate conversational susceptibility to drink.

We tested two hypotheses about the interaction between neural responses to peers and alcohol conversations on next-day drinking. We predicted that stronger neural responses in reward and mentalizing systems to drinking (vs. non-drinking) peers would moderate the temporal association between talking about alcohol and next-day drinking, by increasing the likelihood of next-day drinking.

Consistent with our first hypothesis, we observed a significant interaction between reward activity to drinking (vs. non-drinking) peers and alcohol conversations on next-day drinking (OR=3.05, 95% CI [1.21-7.71], p=0.018), such that participants with stronger activity in the reward system were more likely to drink following alcohol conversations. Follow-up analyses showed that talking about alcohol increased the probability of drinking among individuals whose brains showed stronger (+1 *SD*) or near average reward-related activity to the faces of drinking peers (+1 *SD* reward activity: OR=1.92, 95% CI [1.42-2.57], p<.001; near average reward activity: OR=1.46, 95% CI [1.19-1.80], p = 0.003). In other words, individuals who talked about alcohol were more likely to drink the next day by approximately 4% (95% CI 3%-5%), from 11% (95% CI [10%-13%]) to 15% (95% CI [13%-18%]), and stronger reward-related activity to drinking peers increased the risk of drinking by approximately 7% (95% CI [5%-10%]), from 10% (95% CI [8%-12%]) to 17% (95% CI [13%-22%]). By contrast, for participants whose brains showed stronger activity to non-drinking peers (-1 *SD*), talking about

alcohol did not increase or decrease the probability of next-day drinking (-1 *SD* reward activity: OR=1.12, 95% CI [0.82-1.53], p=0.47).

We observed similar patterns when considering neural responses in the mentalizing system. Consistent with our second hypothesis, we observed a significant interaction between mentalizing related-activity and alcohol conversations on next-day drinking, with stronger activity in the mentalizing system associated with increased likelihood of drinking (OR=2.68, 95% CI [1.32-5.44], p=0.006). Talking about alcohol increased the probability of drinking among individuals whose brains showed stronger (+1 *SD*) or near average mentalizing activity to the faces of drinking (vs. non-drinking) peers (+1 *SD* mentalizing activity: OR=2.00, 95% CI [1.49-2.79], p <0.001; near average mentalizing activity: OR=1.48, 95% CI [1.20-1.81], p<0.001). Stronger mentalizing activity to drinking (vs. non-drinking) peers increased the probability of drinking following alcohol conversations by 8% (95% CI [6%-11%]), from 10% (95% CI [8%-12%]) to 18% (95% CI [14%-23%]). By contrast, for people whose brains showed stronger activity to non-drinking (vs. drinking) peers, talking about alcohol was not significantly associated with next-day drinking (-1 *SD* mentalizing activity: OR=1.09, 95% CI [0.80-1.47], p=0.5929). Together, these results suggest that individuals with stronger activity within the two hypothesized brain systems—reward and mentalizing—showed an increased susceptibility to alcohol related conversational influence on drinking; or were more likely to drink following alcohol conversations.

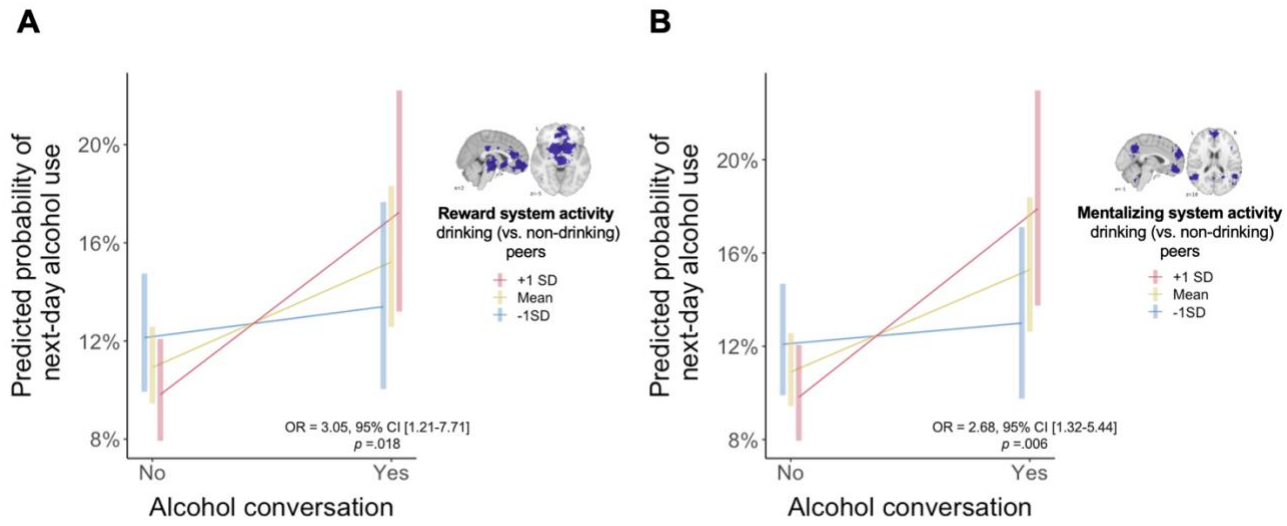

Fig 1. | Neural responses to drinking vs. non-drinking peers moderate conversational influence susceptibility on next-day drinking.

Individuals who showed stronger or near average activity in (A) reward and (B) mentalizing systems to drinking peers were significantly more likely to drink following alcohol conversations (+1 SD; pink line; mean; yellow line). Among individuals who showed stronger activity to non-drinking peers (-1SD; blue line), talking about alcohol was not significantly associated with next-day drinking.

Additional Analyses

Sensitivity analyses and robustness checks are included in the Supplement B. These analyses include tests of whether neural responses to drinking (vs. non-drinking) peers moderate associations between alcohol conversations and next-day drinking when controlling for a range of variables: age, gender, race/ethnicity, social group membership, weekend vs. weekday effects, and intervention effects as part of a parent study with the same dataset[58] , as well as when applying zero-inflated negative binomial models, which separately model the count and likelihood of alcohol use occasions. Further, we explored if the observed results were specific to brain activity tracking differences in peer drinking interactions vs. general perceived peer

closeness). Across all approaches, we observed parallel results such that stronger neural responses to drinking (vs. non-drinking) peers in the reward and mentalizing systems was associated with increased likelihood of drinking following alcohol conversations.

Discussion

Social influence plays a key part in daily life, and individuals vary widely in how they process and respond to such influences. To our knowledge, this is the first study to examine how individual differences in brain activity linked to reward and mentalizing to real peers relates to conversational influence susceptibility on alcohol use. We sampled students at two Northeastern US college campuses who were members of existing social groups (e.g. sports teams and fraternities) and who were social drinkers. Using fMRI, we observed brain activity while individuals viewed photographs of the faces of peers within their social groups. Critically, faces were chosen to show peers with whom participants engaged in social drinking at varying frequencies. Next, using EMA, we tracked alcohol conversations and drinking behavior twice daily for 28 days. Controlling for individual differences in baseline drinking, we found that individuals with stronger reward and mentalizing-related brain activity to the faces of drinking peers—with whom participants drank more frequently— showed higher susceptibility to conversational influence on drinking. Conversely, stronger neural responses to non-drinking peers—with whom participants drank less frequently—decoupled the link between alcohol conversations and next-day drinking.

Our results are consistent with a growing body of work highlighting the role of mentalizing and reward processes in tracking people's relational status with respect to others, in

general[17,52,53,59], and specifically tracking susceptibility to being socially influenced by them[24,25,42]. Our results extend existing literature in several important ways.

Prior neuroimaging studies of social influence have largely used controlled experimental conditions, manipulating participant beliefs about perceived peer preferences for appetitive stimuli (e.g., for food or art) by presenting normative ratings ostensibly made by anonymous or confederate others[25,30,60]. However, social influence in everyday life is more nuanced and complex than a controlled laboratory paradigm, and to date, there has been limited research exploring individual differences in neural responses to naturalistic peer cues in substance use contexts[61]. In this study, we found that the direction and degree of susceptibility to alcohol conversations varied as a function of one's mental representations of specific peers within their social group. While we conceptually replicated the positive relationship between alcohol conversations and future drinking in a naturalistic, longitudinal setting[9], critically, we found that the strength of this relationship varied significantly based on neural responses in brain systems involved in motivational, affective, and social cognitive processing of peer cues. As such, this study follows recent calls to bridge laboratory-based neural responses with natural real-world social contexts[33] and highlights the importance of considering individual heterogeneity[62] to better understand social influence processes.

Our findings suggest neural mechanisms that could underlie various kinds of individual differences in susceptibility to social influences on drinking. For example, stronger activity to drinking (vs. non-drinking) peers, may make anticipated rewards associated with drinking more salient by making peer alcohol norms and expectations (drawn from past social drinking experiences) more accessible in social contexts. In other words, stronger activity in the reward and mentalizing systems may suggest easier retrieval of information pertaining to a peer's

drinking habits, local drinking norms, and expectations of future rewards. In turn, greater schema activation in conversational contexts could promote future conformity.

Similarly, behavioral evidence based on social learning theory also suggests that the nature of peer drinking relationships can moderate future social influence susceptibility on drinking. For example, when peers frequently engage in drinking together, these interactions can reinforce similar behaviors and provide models for future alcohol consumption through observational learning. This often happens as young adults seek peer approval and try to avoid rejection[35]. In line with this view, a separate exploratory analysis (see Supplement B, tables S9 and S10) supports the idea that individual differences in brain activity within reward and mentalizing systems may reflect mental representations specific to drinking experiences, rather than general aspects of peer relationships like perceived closeness. Indeed, we found that susceptibility to conversational influence on drinking was specific to brain activity in response to drinking peers compared to non-drinking peers, i.e., the nature of drinking interactions. However, these associations were not found when examining differences in brain activity related to perceived peer closeness, more generally.

A key complementary finding was that stronger brain responses to faces of non-drinking peers (vs. drinking peers) provided a buffer against conversational influence on drinking. Individuals who showed increased reward and mentalizing-related activity to the faces of non-drinking peers were not influenced by conversations about alcohol, regardless of their baseline drinking levels. It is possible that stronger brain activity in response to non-drinking peers might activate schemas unrelated to drinking in social settings, potentially steering individuals away from alcohol consumption. This finding aligns with previous research among adolescents, which suggests that the nature of peer relationships can deter susceptibility to risk taking behaviors,

with perceived peer support mitigating risky behaviors[63].This insight further raises the question of how to leverage positive peer signals to counteract drinking influences, i.e., to discourage unhealthy drinking habits.

Taken together, the present results may highlight potential future intervention strategies, such as incorporating cues about non-drinking peers into perspective-taking interventions. Prior work has shown that adopting the perspectives of non-drinking peer models can reduce alcohol consumption among college students[58]. Extending this line of work, interventions that dynamically detect peer influences in real-time and deploy perspective-taking reminders among individuals who are most sensitive to social influences could help counteract these influences more effectively. Drawing on recent just-in-time paradigms, researchers could identify states of heightened peer influence vulnerability (e.g., during alcohol conversations) and trigger prompts, "at the right time and for the right person", to buffer susceptibility to conversational influence[64]. Future research may personalize such dynamic interventions based on non-drinking peer cues and evaluate their feasibility relative to stand "one-size-fits all" interventions.

The current results should be interpreted considering the strengths and limitations of the study. Here, we combined fMRI and information about peers from existing social groups within an EMA design, embedded in individuals' daily lives. This multimodal approach allowed us to capture time-sensitive links between alcohol conversations and drinking behavior in everyday environments, avoiding common biases that may arise when participants are asked to recall information about longer periods of time (e.g., alcohol consumed in the previous 30 days[65]). Also notable is the fact we employed the same study protocol across two college campuses, thereby enhancing the robustness of our findings. Although intensive assessment can raise data

compliance concerns, we found minimal non-compliance among our sample. The intensive sampling approach produced high response rates, with ~95% median response rate over 28 days.

With respect to limitations, our data cannot be used to generalize about samples beyond college students who are social drinkers (i.e., without alcohol dependence) who are part of social groups. We recruited students from pre-existing social groups on two campuses (and included groups in which 80% or more expressed interest). Although we controlled for group membership in supplemental analyses, the non-independence may have confounded effects in unmeasurable ways. Further, we did not measure who initiated the alcohol conversation and conversational involvement, which may also moderate conversational susceptibility to alcohol use[66]. Although we did measure conversational valence, i.e., how positive or negative each alcohol conversation was, our study did not indicate that valence was related to drinking behavior. In our sample, most alcohol conversations were positive, and the drinking norms on the studied college campuses favored drinking as normative. Future work may examine more precise conversational measures, possibly using natural language processing tools and passive mobile sensing[67].

Notably, in our data, individual differences in brain activity to the faces of drinking (vs. non-drinking) peers moderated the incidence of drinking episodes but did not relate to the specific amount consumed when drinking, as shown in Supplement B Tables S8 and S9. One possibility is that the neural responses to drinking vs. non-drinking peers provide important inputs when deciding whether to drink (or not) vs. when deciding how much to drink while drinking, which may be subject to additional, factors not considered here. Another possibility is in our current sample of social drinkers, we had insufficient variability in the number of drinks per occasion to detect differences. As such, future work could fruitfully explore how individual

differences in brain activity to different peers relates to health decision making across various contexts and samples.

In sum, the present study combined fMRI, with information about real social groups, and EMA to examine how individual differences in neural responses to peers relate to susceptibility to conversational influence on drinking. Individuals whose brains showed stronger responses to faces of drinking vs. non-drinking peers—in systems related reward and mentalizing—showed higher conversational susceptibility to drink. This work highlights that the ways individuals' brains gauge motivational relevance of social connections can promote drinking following alcohol conversations or provide a protective buffer, depending on their mental representations of peers. Specifically, the present data suggest that brain responses to the faces of peers with varying drinking interactions may serve as indicator of broader susceptibility to conversational influence on alcohol use among college students who are social drinkers. Future work may validate this neural index across different samples and integrate it in interventions that embed positive peer influence to promote healthy behaviors.

Methods

Participants and Procedure

We use data from the Social Health Impact of Network Effects Study (SHINE), a multimodal, multisite project designed to provide insight into mind, body, and community relationships among social groups of young adults (see Refs.[68,69]). All research, methods, and study protocols were approved by the Human Subjects Electronic Research Application (HSERA) Institutional Review Board (IRB) at the University of Pennsylvania and were acknowledged by the Human Research Protection Office of the Department of Defense. All

research, methods, and study protocols were conducted in accordance with the IRB at the University of Pennsylvania and the Human Research Protection Office of the Department of Defense.

Students in two urban universities who belonged to on-campus groups (e.g., sports teams, arts groups, Greek life, etc.) were invited to participate. We recruited groups where more than 80% of the group expressed interest to participate, with the goal of facilitating data collection from many social partners in the group. At baseline, each participant was invited to upload a photo of themselves to build a pool of stimuli for the face viewing fMRI task. We collected 588 photos of peer faces across 24 social groups. The current investigation includes participants (N = 104; M_{age}=20.56 years, SD_{age}=1.72) who completed a baseline online survey, an fMRI visit and a post-scan survey, and a 28-day EMA assessment, comprising individuals from 10 social groups (mean group size = 42.36 students; SD = 21.38, median = 32). These participants reported the following gender and racial/ethnic identities: 63 women, 40 men, 1 other; 57 white, 32 Asian, 2 Black, 5 Latino/a, 8 Other/or more than one identity. Among the participants, 32% participated as part of a sports team, 48% as part of a performing arts group, 17% as part of Greek life, and 3% as another type of group. See 'Supplement A Recruitment' for more details and Figure S1 for participant exclusion and see Ref.[68] for further details on study protocol and procedures. All participants provided written informed consent and were paid for their participation.

Measures and Tasks

fMRI face-viewing task

To measure neural responses to peer faces, we used a task adapted from Refs.[17,52,53]. In the scanner, participants viewed photographs of peers from their on-campus social group. The task stimuli were prepared from peer photographs collected during the baseline survey. Group

members were asked to face the camera, with a neutral expression, and to have no objects in the background. Photographs were inspected manually by researchers for quality and cropped and converted to grayscale with equal luminance, for standardization. See Ref.[68] for details on task development and stimuli selection.

The face-viewing task implemented a rapid event-related design across two runs. During each run, participants viewed three trial types: faces of peers who were part of their on-campus social group (*M* = 25 unique peer faces; *SD* = 3; range = 18-27), their own face, and a red dot in the center of the screen (control images), appearing one at a time. Each face appeared on the screen 6 times (3 times per run) in a randomized order. All trials were presented for one second, followed by a jittered fixation cross (*M* = 5.5s, *SD* = 2.8). To ensure engagement during the task, participants were instructed to press a button each time they saw a red dot on the screen (~10 % of total presentations), using a five-button box. See 'Supplement A Figure S2' for task visualization and instructions. The task was presented using PsychoPy (Version v3.0.0b11[70]). Following the scan, participants reported on how frequently they drank with each of their peers featured in the face-viewing task using a 9-point scale (1-9) in addition to other measures that probed dimensions of individual variation that were beyond the scope of the current report[68].

Ecological Assessment of Conversations and Drinking

Throughout the 28-day EMA period, participants received two survey prompts per day via the LifeData mobile app (www.lifedatacorp.com): in the morning (8am) and in the evening (6pm). Surveys assessed alcohol-related behaviors in addition to other measures such as craving and mood not reported here[68].

Alcohol conversations and drinking occasions *in situ*. To measure alcohol conversations, participants were asked: "Since the (EVENING/MORNING) survey, have you

talked to someone about alcohol?" Participants answered using a "No/Yes" response option. To measure alcohol use, participants were asked: "Since the EVENING/MORNING survey, have you consumed any alcohol?" Participants answered using a "No" or "Yes" response option. Both items were measured twice a day, during the morning and evening EMA. For follow-up alcohol use measures, see 'Supplement A Follow-up measures' and for analyses with these measures see Supplement B Tables S7-S11.

Baseline measures

During the initial online survey, participants reported on demographics including age, gender, and race/ethnicity, in addition to typical drinking frequency and drinking amount in the past 6 months prior to the study. The race/ethnicity variable indicated Asian, Black, Latino/a, white, and Other status. For wording and measurement of the baseline drinking measures see 'Supplement A Alcohol use baseline measures'.

fMRI Data Acquisition, Modeling, and ROI Analysis

fMRI data pre-processing, modeling, and ROI analysis are reported in detail in Supplement A. Briefly, to create a neural index of activity to peer faces with whom one drinks more vs. less frequently, we extracted mean parameter estimates from the parametrically modulated viewing of peer faces (modulated by frequency of drinking with that peer) within the (a) reward and (b) mentalizing ROIs, as defined by Neurosynth[71]. Greater values on these neural indices correspond to stronger activity to faces of peers with whom one engages in more vs. less frequent drinking-related activities (i.e., noted as drinking vs. non-drinking peers).

Data Preparation

We took several steps to prepare the data prior to modeling. We aimed to model how alcohol conversations relate to prospective alcohol use, and we slid forward the alcohol use

variable by one day (by two observations), as the questions were phrased to measure alcohol use since the previous survey, twice a day. That is, we considered whether today's alcohol conversation predicted tomorrow's alcohol use[9]. Further, to attenuate the influence of outliers in the brain data, we winsorized activity in reward regions and mentalizing regions +/- 2 standard deviations from the mean, following outlier inspection. This cutoff applied to seven participants' activity in reward-related regions and four participants' activity in mentalizing regions. As a sensitivity test, we repeated all analyses including the outliers in the brain data and observed parallel results. See 'Supplement B Tables S1 and S2' for analyses including outliers.

Analysis plan

To examine whether individual differences in neural activity in reward and mentalizing regions to drinking vs. non-drinking peers moderates the association between talking about alcohol and next-day drinking, we estimated mixed-effects models separately for each brain system. The main predictor of interest was an interaction term of (a) having a conversation about alcohol (Yes/No) and (b) a between-subject neural index (for either reward or mentalizing systems) capturing activity to drinking (vs. non-drinking) peers. The alcohol conversation variable was split into a within and between components following standards in the field[72]. Specifically, we created a person-level conversation variable by computing the overall proportion of alcohol conversations that occurred across the EMA protocol, and within-person daily variable indicating whether (1) or not (0) an alcohol conversation occurred at a given prompt. Our primary outcome was the likelihood of alcohol use, indicating whether (1) or not (0) alcohol was consumed[73]. In the manuscript, we present the most parsimonious models, controlling for baseline drinking frequency and amount in the past six months (to account for possible individual differences in overall drinking), and time in study, as on average, participants reported drinking less over the

study period. See Supplement B Tables S3 and S4 for sensitivity analyses including additional covariates: age, gender, race/ethnicity, group membership, response rates, weekend, and condition effects as part of a larger intervention study[58]. Results are robust to both the inclusion and exclusion of covariates (Supplement B tables S5 and S6). All numeric variables were grand-mean centered, and intercepts were allowed to vary randomly across people. Models did not converge when a random effect for alcohol conversations was included and, as such, a simpler model without this random effect estimate is presented.

Given we aimed to quantify the likelihood of a future drinking episode, we conducted multilevel binary logistic regression using the 'glmer' function from the lme4 package[74]. We specified the "bobyqua" algorithm to optimize model convergence. As a sensitivity test, we repeated the same analyses using more complex multi-level hurdle models using glmmTMB[75] which separately model the count in addition to binary alcohol use occasions, and we observed parallel results. See Supplement B Table 7 for results from multi-level hurdle models. All analyses were conducted in RStudio version 3.6.2[76].

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Funding Statement

Research was sponsored by the Army Research Office and was accomplished under Grant Number W911NF-18-1-0244. D.M.L. acknowledges support from the National Institute on Drug Abuse (K01 DA047417) and the Brain & Behavior Research Foundation. D.S.B. acknowledges support from the John D. and Catherine T. MacArthur Foundation, the Swartz Foundation, the Paul G. Allen Family Foundation, the Alfred P. Sloan Foundation and the NSF (IIS-1926757). D.C. and E.B.F. acknowledge support from Hopelab and the Lentic fund. K.N.O. acknowledges support from the National Institute on Alcoholism and Alcohol Abuse (R01 AA023653). M.J. acknowledges support from CSS. The views and conclusions contained in this document are those of the authors and should not be interpreted as representing the official policies, either expressed or implied, of the Army Research Office or the U.S. Government. The U.S. Government is authorized to reproduce and distribute reprints for Government purposes notwithstanding any copyright notation herein.

Citation Statement

We sought to proactively consider choosing references that reflect the diversity of the field in thought, form of contribution, gender, race, ethnicity, and other factors. First, we obtained the predicted gender of the first and last author of each reference (excluding software package citations) by using databases that store the probability of a first name being carried by a woman (https://github.com/dalejn/cleanBib). By this measure (and excluding self-citations to the first and last authors of our current paper), our non-software references contain 26% woman(first)/woman(last), 27% man/woman, 14% woman/man, and 33% man/man. This

method is limited in that names used to construct the databases may not, in every case, be indicative of gender identity and cannot account for intersex, non-binary, or transgender people.

Positionality Statement

Mindful that our identities can influence our approach to science, the authors wish to provide the reader with information about our backgrounds. With respect to gender, when the manuscript was drafted, eight authors self-identified as women, one as non-binary, and four as men. With respect to race, 12 authors self-identified as white and one as Asian. With respect to engagement with college students, when this study was conducted, two authors were doctoral students who teach and/or mentor other students, three authors were postdoctoral researchers or research scientists who teach and/or mentor students, and eight authors were professors who teach and/or mentor students.

Ethics Declaration

All research and experimental procedures were conducted in accordance with the Human Subjects Electronic Research Application (HSERA) Institutional Review Board (IRB) at the University of Pennsylvania and acknowledged by the Human Research Protection Office of the Department of Defense. All participants provided informed consent before taking part in the study and all participants were financially compensated.

CReDiT Taxonomy Statement

Conceptualization: M.J, O.S, E. B. F, K.N.O, B.D., Formal Analysis: M.J, O.S, D.C., Funding Acquisition: E.B.F, K.N.O, D.S.B, P.M., Supervision: D.C, N.C, D.L.S, K.N.O, Y.K, E.B.F.; Investigation: O.S, Y.K; Z.B., P.J.M., Methodology: M.J, B.D, C.S, O.S, K.N.O., Writing – original draft: M.J., Writing – editing and revising: M.J, O.S., C.S, B.D, E. B. F, P.J.M, D.M.L, K.N.O, F.C, Y.K, N.C, Z.B, D.C.

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Data Availability Statement

De-identified data and code to reproduce the main analyses are available on:

https://github.com/miajov/brain-responses---peer-influence.

Competing interests

The author(s) declare no competing interests.

Supplementary Information

The following document contains supplementary information for *Jovanova et al*. “Neural moderators of social influence susceptibility on drinking”. Supplement A presents information on participant enrollment and study procedures. Supplement B includes sensitivity analyses and robustness checks.

Supplement A

Information on participant recruitment is adapted from Jovanova et al[1], and information on the neuroimaging session and fMRI preprocessing is taken from our study protocol, Ref[2]. Here we provide a summary for the interested reader.

Recruitment

Recruitment materials advertised a study titled "Social Health Impact of Network Effects Study (SHINE)" to undergraduate students who were members of on-campus social groups across two urban Northeastern universities in the United States. The study was advertised through flyers, university websites, and email communication. To reach students, researchers contacted group leaders and further employed a snowball sampling approach, such that participating students could share recruitment information with their peers who were members of on-campus social clubs or sports teams. For the current report, the data collection began on March 1st, 2019 and ended on March 31st, 2020, thus including time primarily on campus and as participants transitioned home at the start of the COVID-19 pandemic.

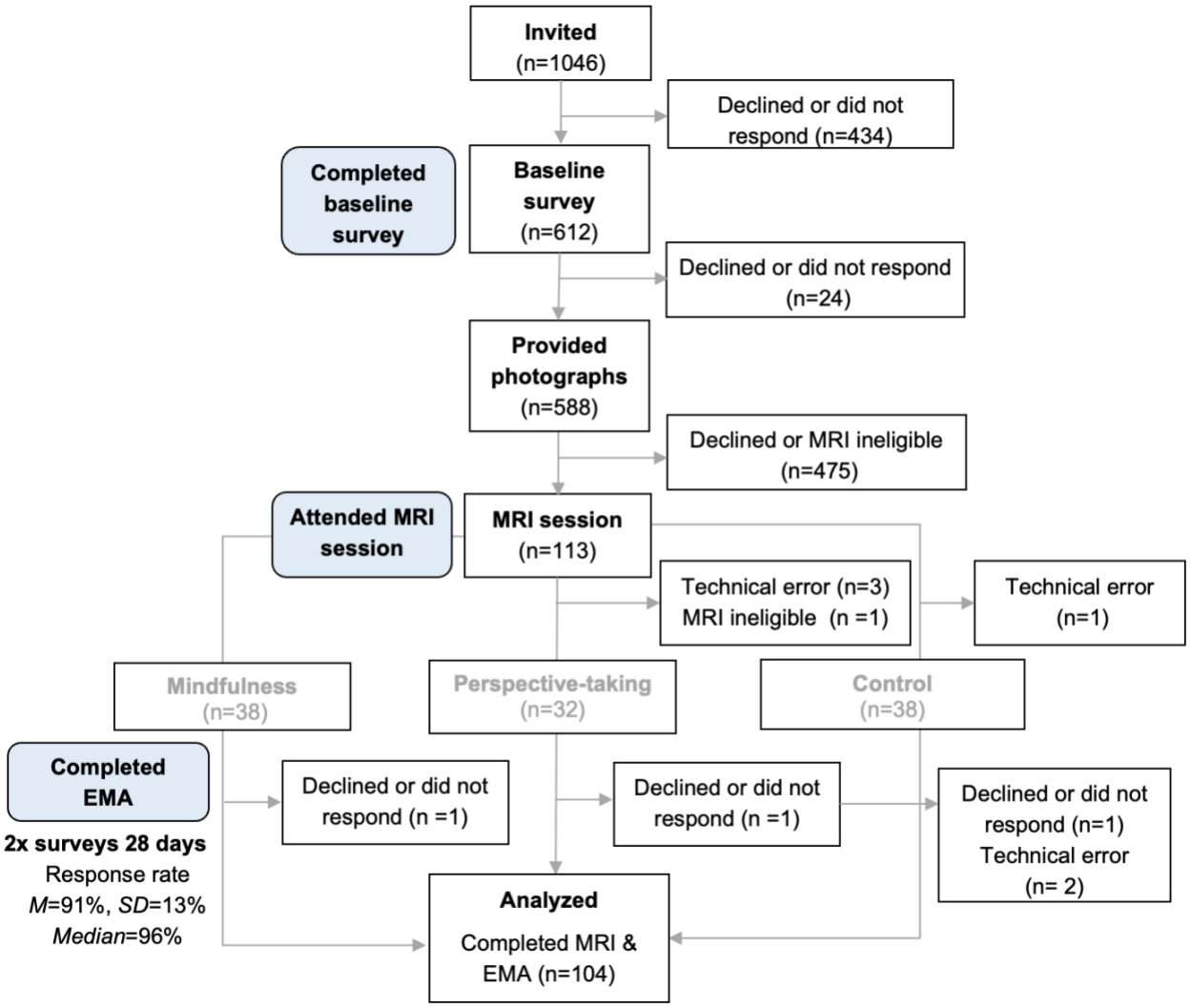

Fig S1. Participant enrollment flowchart.

Flow chart shows participant retention across three main study components: baseline survey, MRI session, and a 28-day EMA protocol. As part of a different study beyond the scope of the current report, participants were randomized into three conditions on how to respond to alcohol cues (mindfulness, perspective-taking, and control) (See Ref.[2]). See Supplement B Table S3 and S4 for analyses controlling for condition effects.

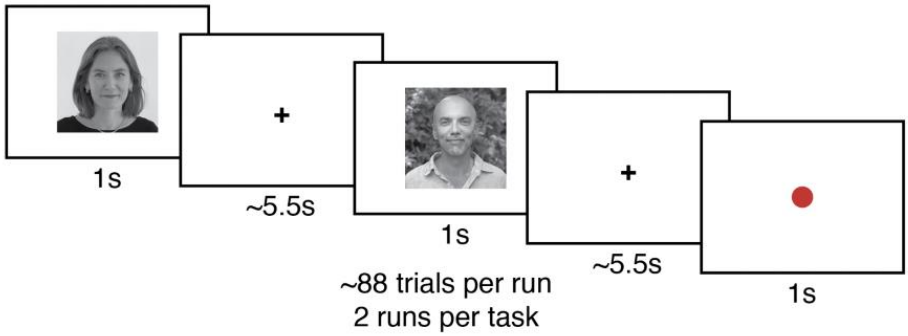

Fig S2. Passive face viewing fMRI task.

Design of the faces task. Participants viewed faces of their peers, themselves, or a control image (red dot), and pressed a button each time they saw the dot[2].

Faces task instructions

The following text presents the verbatim task instructions for the fMRI task. Non-italicized fonts are researcher instructions and *italicized* fonts are instructions included in the PsychoPy scripts. Research assistants walked participants through the instructions and asked participants to complete several practice rounds on their own prior to beginning the fMRI task session.

We're going to be doing a task which will involve looking at pictures and pressing a button every time a red "O" appears on the screen. We are going to ask you to pay attention to the screen and press a button every time you see the red "O" appear on the screen.

Please read and listen carefully, and let us know if you have any questions.
Each trial will start with a cross at the center of the screen (+). When you see this symbol, simply focus on its center.

Next, a photo will appear. You will see images of faces or a red "O". You should focus on the screen. Press any button on the keypad when you see a red "O". Do not press a button when you see a face.

You will have a few seconds to make your response. If three seconds pass and you haven't yet made a response, the computer will not record a response and will go on. To avoid this outcome, please keep your hand on the keypad and be ready to record a response.

So I will walk you through some practice now…

Face-viewing task pre-processing, modeling, and ROI analysis

fMRI data collection. Neuroimaging data were acquired on 3 Tesla Siemens Prisma scanners equipped with a 64-channel head coil. High-resolution T1-weighted structural images were collected using an MPRAGE sequence (TI=1,100ms, voxel size=0.9×0.9×1mm, 160 slices, field of view [FOV]=256, repetition time [TR]=1850ms, echo time [TE]=3.91ms, flip angle=8°). T2*-weighted functional images were also collected (voxel size=3x3x3mm, 42 slices, FOV=70, TR=1,000ms, TE=30, flip angle=62°).

fMRI preprocessing. The anatomical and functional data were preprocessed using fMRIPrep (Version 20.0.6[3], which is based on Nipype (Version 1.4.2[4];. The T1-weighted (T1w) image was corrected for intensity non-uniformity (INU) with N4BiasFieldCorrection[5], distributed with ANTs 2.2.0[6], and used as a T1w-reference throughout the workflow. The T1w-reference was then skull-stripped with a Nipype implementation of the ANTs brain extraction workflow, using OASIS30ANTs as target template. Brain tissue segmentation of cerebrospinal fluid (CSF), white-matter (WM), and gray-matter (GM) was performed on the brain-extracted T1w image using FAST (FSL 5.0.9[7]). Brain surfaces were reconstructed using recon-all (FreeSurfer 6.0.1[8]), and the brain mask estimated previously was refined with a custom variation of the method to reconcile ANTs-derived and FreeSurfer-derived segmentations of the cortical gray matter of Mindboggle[9]. Volume-based spatial normalization to one standard space (MNI152NLin2009cAsym[10]) was performed through nonlinear registration with antsRegistration (ANTs 2.2.0), using brain-extracted versions of both the T1w reference and the T1w template.

A reference volume and its skull-stripped version were generated using a custom methodology of fMRIPrep. A B0-nonuniformity map (or fieldmap) was estimated based on two echo-planar imaging (EPI) references with opposing phase-encoding directions, with 3dQwarp[11]

with AFNI 20160207. Based on the estimated susceptibility distortion, a corrected EPI reference was calculated for a more accurate co-registration with the anatomical reference. The BOLD reference was then co-registered to the T1w reference using bbregister from FreeSurfer, which implements boundary-based registration[12]. Co-registration was configured with six degrees of freedom. Head-motion parameters with respect to the BOLD reference (transformation matrices and six corresponding rotation and translation parameters) are estimated before any spatiotemporal filtering using mcflirt (FSL 5.0.9)[13]. BOLD runs were slice-time corrected using 3dTshift from AFNI 20160207[11]. The BOLD time series were resampled onto their original, native space by applying a single, composite transform to correct for head-motion and susceptibility distortions. The BOLD time series were resampled into standard space, generating a preprocessed BOLD run in MNI152NLin2009cAsym space. All resamplings were performed with a single interpolation step by composing all the pertinent transformations (i.e., head-motion transform matrices, susceptibility distortion correction when available, and co-registrations to anatomical and output spaces). Gridded (volumetric) resamplings were performed using antsApplyTransforms (ANTs), configured with Lanczos interpolation to minimize the smoothing effects of other kernels[14]. Non-gridded (surface) resamplings were performed using mri_vol2surf (FreeSurfer). Various confounds (e.g., framewise displacement, DVARS, and global signal) were also calculated for each TR and logged in a confounds file. The outputs from fMRIPrep were then manually quality checked to ensure adequate preprocessing.

Prior to first-level modeling, we generated motion regressors using an automated motion assessment tool. This tool is a predictive model that utilizes the confound files generated by fMRIPrep and classifies whether or not fMRI volumes contain motion artifacts. The classifier is applied to each participant's task run and returns a binary classification indicating the presence

or absence of motion artifacts for each volume. In addition, this tool transforms the realignment parameters into Euclidean distance for translation and rotation separately and calculates the displacement derivative of each. This yielded a total of five motion regressors for first-level modeling[2]. First, realignment parameters were transformed into Euclidean distance for translation and rotation separately, and we included the displacement derivative of each (resulting in four motion regressors). Another regressor of non-interest marked images with motion artifacts (e.g., striping) was identified via automated motion assessment[15] and visual inspection. Following the application of this threshold, no task runs were excluded from further analyses. Data were high-pass filtered at 128s, and temporal autocorrelation was modeled using FAST[16].

fMRI task modeling. Since we aimed to compute a neural index of responses to peer faces with varying drinking interactions, we extracted activity to peer faces as modulated by drinking frequency ratings, or how often each participant reported drinking with each individual peer. As such, we modeled the task using a parametric modulation design as implemented in SPM 12[17]. We constructed first level models for each participant that regressed periods of exposure to peer faces on mean centered, trial-by-trial differences in how frequently they reported drinking with each peer (range: 1-9). Models also included nuisance regressors that were modeled separately. These included periods of exposure to an individual's own face, red dot, and five motion regressors described above. Thus, to compute a brain index that captures differences in responses to peer faces with varying drinking interactions, we extracted activity to peer faces as modulated by drinking frequency ratings, or how often each participant reported drinking with each of the peers whose face they viewed in the scanner (~28 peers).

***ROI analysis*.** To define targeted regions of interest, we extracted two functionally defined maps from Neurosynth[18]. For reward-related activity, we used the search term 'reward' (922 studies; 30418 activations, p<.01, corrected). For mentalizing-related activity, we used the search term 'mentalizing' (151 studies; 6824 activations, p<.01, corrected). Next, to create a neural index of activity to peers with whom one drinks more vs. less frequently, we extracted mean parameter estimates from the parametric modulated viewing of peer faces within the (a) reward ROI and the (b) mentalizing (ROI). Greater values on these neural indices correspond to stronger activity to faces of peers with whom one engages in more (vs. less) frequent drinking activities (i.e., drinking vs. non-drinking peers). This procedure resulted in two values per participant that were used as individual difference measures of 'reward' and 'mentalizing' activity to faces of drinking (vs. non-drinking) peers, as a function of peer drinking interactions.

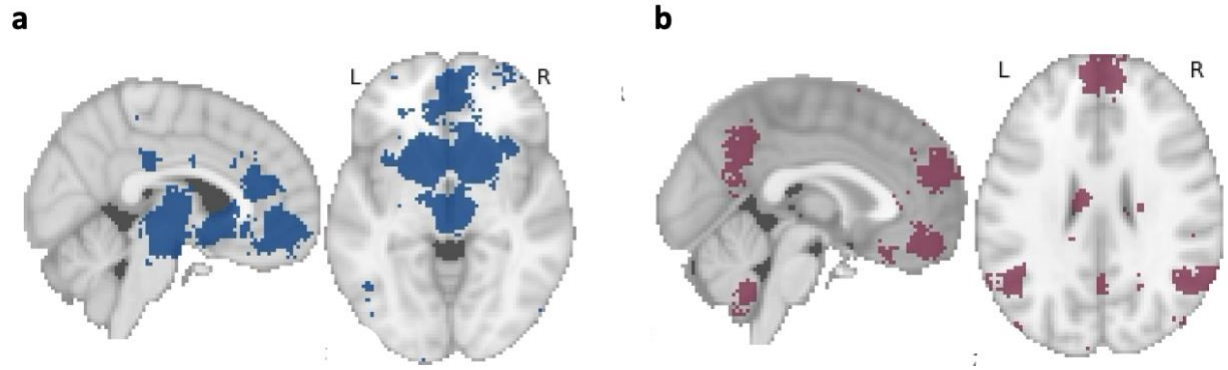

Fig S3. Region of interest (ROI) masks associated with 'reward' and 'mentalizing'.

A. Brain regions associated with "reward", as identified through Neurosynth using an association test (p<0.01, corrected). B. Brain regions associated with "mentalizing", as identified through Neurosynth using an association test (p<0.01, corrected). Figure was created using 'nilearn.plotting.plot_roi'[19] in Python (version 3.6.2).

Alcohol use baseline measures

To measure typical alcohol use prior to data collection, participants responded to two questions which measured drinking frequency and drinking amount as part of an online survey, in addition to other individual difference measures beyond the scope of the current report. Responses to these baselines drinking measures were included as covariates in the main models reported in the manuscript.

Drinking frequency. "During the last 6 months, how often did you usually have any kind of drink containing alcohol? (By a drink we mean the equivalent of a 12 oz can or glass of beer, a 5 oz glass of wine, or a drink containing 1 shot of liquor)". Response options included: "I never drank any alcohol in my life" = 1; "I did not drink alcohol in the last 6 months, but I did drink in the past" = 2; "1-2 times in the past 6 months" = 3; "3-5 times in the past 6 months" = 4; "2-3 times a month" = 5; "Once a month" = 6; "Once a week" = 7; "Twice a week" = 8; "3-4 times a week" = 9; "5-6 times a week" = 10; "Every day" = 11.

Drinking amount. "During the last 6 months, how many alcoholic drinks did you have on a typical day when you drank alcohol?" "0 drinks" = 0; "1 drink" = 1; "2 drinks = 2; "3-4 drinks" = 3.5; "5-6 drinks" = 5.5; "7-8 drinks" = 7.5; "9-11 drinks" = 10; "12 - 15 drinks" =13.5; "16-18 drinks" = 17 drinks; "19-24 drinks"= 21.5; "25 or more drinks" = 25.

Follow-up measures. Participants who responded 'yes' to having alcohol were also asked to enter the number of wine/beer/liquor beverages consumed since the last survey as part of a different study[1]. Participants who reported 'yes', to having an alcohol conversation were asked to report the valence of their most recent alcohol conversation since the previous survey (0, *negative* to 100, *positive*). In the present manuscript, we focus on the initial "No/Yes"

responses to alcohol use and alcohol conversations. Analyses with follow-up measures are included in Supplement B Tables S7-S11.

Supplement B

Brain activity effects on conversational influence susceptibility are robust to outlier inclusion. We conducted an exploratory analysis including outlier observations in brain activity in both reward and mentalizing ROIs to drinking (vs. non-drinking) peers. In both cases, we found that the interaction between reward/mentalizing activity and conversational influence on drinking remained significant. Stronger activity in reward and mentalizing-related regions increased the likelihood of drinking following alcohol conversations [reward: OR = 2.73, 95% CI [1.22=6.12], p=.014; mentalizing: OR =1.88, 95% CI [1.09-3.24], p =.023]. See Tables S1 and S2.

Table S1. Reward ROI model including outliers.

Reward ROI*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.18	0.14, 0.22	<.001***
Alcohol conversation	1.47	1.19, 1.80	<.001***
Reward activity	0.57	0.34, 1.08	.089
Baseline drinking frequency	1.34	1.19, 1.50	<.001***
Baseline drinking amount	1.08	1.00, 1.16	.040*
Proportion of alcohol conversations	11.84	4.36, 32.15	<.001***
Time in study	0.99	0.98, 0.99	<.001***
Alcohol conversation*reward activity	2.73	1.22, 6.12	.014*
	ICC	SD	
Intercept			
Participant ID	.10	.59	

Note. 4760 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

Table S2. Mentalizing ROI model including outliers.

Mentalizing activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.18	0.14, 0.22	<.001***
Alcohol conversation	1.48	1.20, 1.81	<.001***
Mentalizing activity	0.71	0.49, 1.03	.072
Baseline drinking frequency	1.34	1.19, 1.51	<.001***
Baseline drinking amount	1.07	1.00, 1.15	.052
Proportion of alcohol conversations	11.73	4.31, 31.92	<.001***
Time in study	0.99	0.98, 0.99	<.001***
Mentalizing activity*alcohol conversation	1.88	1.09, 3.24	.023*
	ICC	SD	
Intercept Participant ID	.10	.59	

Note. 4760 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

Brain activity effects on conversational influence susceptibility are robust to covariate inclusion. Parallel to the models reported in the main manuscript, we conducted an exploratory analysis with additional covariates including age, gender, race/ethnicity, social group membership, response rate, intervention effects[1], and social weekend. Social weekend is defined as Thursday-Sunday (relative to rest of the weekdays) given elevated drinking levels among college students during these days[20] .Two interventions variables were specified to include (1) between-person randomized condition assignment (mindfulness, perspective-taking, and control) and (2) within-person manipulation (active vs. inactive) weeks (for more details see Ref.[1]). Group membership is defined as belonging to one of ten on-campus social groups (i.e., sports clubs or fraternities).

Consistent with the analyses presented in the main manuscript, we predicted that stronger responses in reward and mentalizing systems to drinking (vs. non-drinking) peers would

moderate the association between talking about alcohol and next-day drinking, specifically by increasing the likelihood of drinking following alcohol conversations. We conducted two separate models for ROI activity in reward and mentalizing systems. In both cases, the interaction between reward/mentalizing activity and alcohol conversations on next-day drinking remained significant, such stronger responses in reward and mentalizing-related regions increased the likelihood of drinking following alcohol conversations [reward: OR = 2.69, 95% CI [1.07, 6.75], p=.035; mentalizing: OR =2.47, 95% CI [1.22, 4.97], p =.012]. See Tables S3 and S4.

Table S3. *Reward ROI model including covariates*

Reward activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.13	0.06, 0.29	<.001***
Alcohol conversation	1.44	1.17, 1.78	.001**
Reward activity	0.77	0.38, 1.57	.480
Gender	0.98	0.71, 1.35	.903
Age	1.19	1.08, 1.30	<.001***
Race	1.11	0.92, 1.27	.104
Social group	1.01	0.97, 1.08	.715
Social weekend (vs. weekdays)	1.23	0.92, 1.46	.016*
Proportion of alcohol conversations	16.66	9.46, 47.5	<.001***
Alcohol responses	0.97	0.65, 1.00	.028*
Time in study	0.99	0.63, 0.99	<.001***
Condition mindful (vs. control)	0.78	0.55, 1.13	.183
Condition perspective (vs. control)	0.80	0.55, 1.20	.284
Active week (vs. inactive)	1.17	1.10, 1.46	.151
Alcohol conversation*reward activity	2.69	1.07, 6.75	.035*
	ICC	SD	
Intercept Participant ID	.10	.61	

Note. 4760 observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

Table S4. *Mentalizing ROI model including covariates*

Mentalizing activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.13	0.06, 0.29	<.001***
Alcohol conversation	1.45	1.18, 1.79	<.001***
Mentalizing activity	0.82	0.48, 1.40	.457
Gender	0.98	0.71, 1.35	.890
Age	1.19	1.08, 1.30	<.001***
Race	1.12	0.98, 1.27	.096
Social group	1.01	0.95, 1.08	.732
Social weekend (vs. weekdays)	1.23	1.04, 1.45	*.018
Proportion of alcohol conversations	17.22	6.01, 49.34	<.001***
Alcohol responses	0.97	0.95, 1.00	.028*
Time in study	0.99	0.98, 0.99	<.001***
Condition mindful (vs. control)	0.77	0.53, 1.13	.180
Condition perspective (vs. control)	0.80	0.54, 1.20	.278
Active week (vs. inactive)	1.18	0.95, 1.47	.141
conversation*mentalizing activity	2.47	1.22, 4.97	.012*
	ICC	SD	
Intercept			
Participant ID	.11	.62	

Note. 4520 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

Brain activity effects on conversational influence susceptibility are robust to covariate exclusion. We also conducted exploratory analyses to inspect the robustness of our interaction effects when removing all covariates. We performed two models for reward and mentalizing ROIs separately. We observed consistent effects, such that neural responses to peers in reward and mentalizing ROIs moderated the association between alcohol conversations and next-day drinking, with stronger activity to drinking (vs. non-drinking) peers increasing the likelihood of drinking following alcohol conversations [reward: OR = 2.59, 95% CI [1.03-6.53], p=.043; mentalizing: OR = 2.35, 95% CI [1.17, 4.72], p =.017]. See Tables S5 and S6.

Table S5. *Reward ROI model without covariates*

Reward activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.12	0.10, 0.15	<.001***
Alcohol conversation	1.73	1.42, 2.12	<.001***
Reward activity	0.60	0.26, 1.39	.234
Alcohol conversation*reward activity	2.59	1.03, 6.53	.043*
	ICC	SD	
Intercept Participant ID	.18	.85	

Note. 4760 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

Table S6.

Mentalizing ROI model without covariates

Mentalizing activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.12	0.10, 0.15	<.001***
Alcohol conversation	1.74	1.42, 2.13	<.001***
Activity in mentalizing regions	0.64	0.34, 1.22	.174
Alcohol conversation*mentalizing activity	2.35	1.17, 4.72	.017*
	ICC	SD	
Intercept Participant ID	.18	.86	

Note. 4760 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$.

Brain activity effects on conversational influence susceptibility are specific to drinking occasions. As an additional test, we repeated the same analyses presented in the main manuscript using multi-level hurdle models, an alternative analytic approach which separately models the count of drinks per drinking occasion in addition to the presence (vs. absence) of alcohol use occasions. This analysis allowed us to (a) confirm that our results are robust to different analytic choices and (b) explore the extent to which the observed interaction effects are specific to likelihood of a drinking occasion (zero-inflated model) versus number of drinks per drinking occasion (conditional model).

We observed parallel results to those presented in the main manuscript, such that brain activity in reward and mentalizing ROIs, moderated the association between alcohol conversations and next-day drinking occasions, with stronger activity increasing the likelihood of drinking following alcohol conversations [reward: OR =0.32, 95% CI [0.13-0.82], p=.018; mentalizing: OR =0.36, 95% CI [0.18-0.74], p =.005]. Further, we observed no significant interaction between brain activity in reward and mentalizing ROIs and alcohol conversations on the number of drinks per occasion [reward: OR =1.88, 95% CI [0.77, 4.58], p=.166; mentalizing: OR =1.27, 95% CI [0.65-2.46], p =.485] See Table S7. Together, these results suggest that the observed interaction is specific to the likelihood of engaging in a drinking occasion versus the number of drinks consumed when drinking.

Table S7.

Neural responses to drinking (vs. non-drinking) peer faces moderate conversational influence susceptibility using zero-inflated negative binomial multi-level hurdle models

	Reward ROI*alcohol conversation effects on future drinking					
	Zero-inflated sub model			**Conditional sub model**		
Fixed Effects	OR	95%CI	*P*	OR	95%CI	*p*
Intercept	5.75	4.61, 7.12	<.001***	2.40	1.99, 2.90	<.001***
Alcohol conversation	0.69	0.56, 0.84	<.001***	1.26	1.04, 1.52	.020*
Reward activity	1.54	0.77, 3.07	.218	1.01	0.57, 1.77	.982
Baseline drinking frequency	0.74	0.66, 0.83	<.001***	1.11	1.03, 1.21	.008**
Baseline drinking amount	0.93	0.87, 1.00	.054	1.13	1.08, 1.18	<.001***
Proportion of alcohol conversations	0.08	0.93, 0.97	<.001***	1.19	0.62, 2.29	.598
Time in study	1.01	1.01, 1.02	<.001***	0.99	0.99, 1.00	.007***
Reward activity*alcohol conversation	0.32	0.13, 0.82	.018*	1.88	0.77, 4.58	.166
Random effects	Variance		SD	Variance		SD
Intercept Participant ID	.35		.59	.68		.26

	Mentalizing ROI*alcohol conversation effects on future drinking					
	Zero-inflated sub model			**Conditional sub model**		
Fixed Effects	OR	95%CI	*P*	OR	95%CI	*P*
Intercept	5.75	4.64, 7.12	<0.001***	2.39	1.97, 2.88	<.001***
Alcohol conversation	0.68	0.55, 0.84	<0.001***	1.27	1.05, 1.54	.014*
Mentalizing activity	1.41	0.84, 2.39	0.195	0.98	0.65, 1.48	.928
Baseline drinking frequency	0.74	0.66, 0.83	<0.001***	1.11	1.02, 1.20	.011*
Baseline drinking amount	0.93	0.87, 1.00	0.057	1.13	1.08, 1.19	<.001***
Proportion of alcohol conversations	0.08	0.03, 0.21	<0.001***	1.22	0.64, 2.36	.547
Time in study	1.01	1.01, 1.02	<0.001***	0.99	0.99, 1.00	.008**
Mentalizing activity*alcohol conversation	0.36	0.18, 0.74	.005**	1.27	0.65, 2.46	.485
Random effects	Variance		SD	Variance		SD
Intercept Participant ID	.35		0.59	.07		.26

Note. Number of observations: 4747; *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$. The zero inflated sub-model of the hurdle model estimates the probability of an extra zero (no alcohol use). As such, an odds ratio of less than 1 corresponds to a positive effect, i.e. more occasions of alcohol use.

Table S8.

Correlations and Descriptive Statistics of Key Variables

Variables	1	2	3	4	5
1. Activity in reward ROI	-				
2. Activity in mentalizing ROI	0.79	-			
3. Count of alcohol conversations	-0.02	-0.05	-		
4. Count of drinking occasions	-0.05	-0.09	0.53	-	
5. Number of alcohol responses	0.004	0.001	0.14	0.07	-
Mean	0.07	0.12	9.46	7.69	51.03
Standard Deviation	0.24	0.31	7.59	6.07	7.2

Note: *N*=104

Brain activity effects on conversational influence susceptibility are not driven by general peer closeness. As a control test, we examined whether our findings are specific to brain responses to faces of peers with varying alcohol related peer interactions, versus brain responses tracking peer closeness more generally. Specifically, we created a neural index of activity to peer closeness during passive face viewing (i.e., viewing faces to peers who are perceived as more vs. less close, using self-reported peer closeness ratings; range 1-9). We extracted mean parameter estimates from the parametrically modulated viewing of peer faces within the reward and the mentalizing system ROIs. Greater values on these neural indices correspond to stronger activity to faces of peers who are perceived as close vs. distant.

We then repeated the same interaction analyses as those presented in the main manuscript, however replacing the peer drinking (vs. non-drinking) neural index with a peer close (vs. distant) neural index. We found no significant interaction between brain activity to peers who are more (vs. less) close and alcohol conversations on future alcohol use in the reward system (OR =1.19, 95% CI [0.98, 1.45], p=0.073) and in the mentalizing system (OR =1.11, 95% CI [0.89, 1.39], *p*=0.341). See Tables S9 and S10 for more details. These results suggest

that the main findings presented in the manuscript are unlikely to be driven by individual differences in the brain that track peer closeness more generally.

Table S9.

Reward ROI perceived peer closeness model

Reward activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.18	0.14, 0.22	<.001***
Alcohol conversation	1.39	1.12, 1.73	.002**
Reward activity	0.98	0.84, 1.15	.783
Baseline drinking frequency	1.35	1.20, 1.52	<.001***
Baseline drinking amount	1.07	1.00, 1.15	.058
Proportion of alcohol conversations	11.69	4.26, 32.09	<.001***
Time in study	0.99	0.98, 0.99	<.001***
Alcohol conversation*reward activity	1.19	0.98, 1.45	.073
	ICC	SD	
Intercept			
Participant ID	.10	.60	

Note. 4760 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$. Reward activity is parametrically extracted to track perceived peer closeness (close vs. distant).

Table S10.

Mentalizing ROI perceived peer closeness model

Mentalizing activity*alcohol conversation effects on next-day drinking			
Fixed Effects	OR	95%CI	*P*
Intercept	0.18	0.14, 0.22	<.001***
Alcohol conversation	1.45	0.18, 1.78	<.001***
Mentalizing activity	0.95	0.78, 1.14	.566
Baseline drinking frequency	1.34	1.19, 1.51	<.001***
Baseline drinking amount	1.07	1.00, 1.15	.058
Proportion of alcohol conversations	11.80	4.31, 32.31	<.001***
Time in study	0.99	0.98, 0.99	<.001***
Mentalizing activity*alcohol conversation	1.11	0.89, 1.39	.341
	ICC	SD	
Intercept Participant ID	.10	.60	

Note. 4760 Observations. *$p \leq .05$, **$p \leq .01$, ***$p \leq .001$. Mentalizing activity is parametrically extracted to track perceived peer closeness (close vs. distant).

Brain activity effects on conversational influence susceptibility are not driven by conversation valence. We conducted additional analysis to explore if the main results presented in the main manuscript may be driven by how positively (vs. negatively) individuals talked about alcohol (a) at each time point, and (b) overall, across the 28 days.

Overall, conversations about alcohol were positive throughout the 28-day study protocol (*M* = 62.61, SD= 11.67, median = 61.6; range=32.35-91.33 from 0-100 scale). We found no significant interactions between brain activity and conversation valence on next-day drinking. Individual differences in brain responses to drinking (vs. non-drinking) peers did not significantly interact with conversation valence in the reward system, both within-person (OR = 0.99, 95% CI [0.94-1.04], *p* =0.562), and between-person (OR =1.03, 95% CI [0.97-1.109], *p* =0.352). We found similar non-significant effects for the mentalizing system both within-person

(OR =0.99, 95% CI [0.95-1.03], p=0.723) and between-person (OR =1.05, 95% CI [1.00-1.09], p=0.051). Together, these results suggest the main results reported in the manuscript, are unlikely to be driven by the degree to which individuals perceived their alcohol-related conversations to be positive or negative.

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