Reward System Activation in Response to Alcohol Advertisements Predicts College Drinking

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ABSTRACT. Objective: In this study, we assess whether activation of the brain's reward system in response to alcohol advertisements is associated with college drinking. Previous research has established a relationship between exposure to alcohol marketing and underage drinking. Within other appetitive domains, the relationship between cue exposure and behavioral enactment is known to rely on activation of the brain's reward system. However, the relationship between neural activation to alcohol advertisements and alcohol consumption has not been studied in a nondisordered population. **Method:** In this cross-sectional study, 53 college students (32 women) completed a functional magnetic resonance imaging scan while viewing alcohol, food, and control (car and technology) advertisements. Afterward, they completed a survey about their alcohol consumption (including frequency of drinking, typical number of drinks consumed, and frequency of binge drinking) over the previous month. **Results:** In 43 participants (24 women) meeting inclusion criteria, viewing alcohol advertisements elicited activation in the left orbitofrontal cortex and bilateral ventral striatum—regions of the reward system that typically activate to other appetitive rewards and relate to consumption behaviors. Moreover, the level of self-reported drinking correlated with the magnitude of activation in the left orbitofrontal cortex. **Conclusions:** Results suggest that alcohol cues are processed within the reward system in a way that may motivate drinking behavior. (*J. Stud. Alcohol Drugs, 79,* 29–38, 2018)

ALCOHOL IS THE MOST PREVALENT illicit substance used by adolescents, and its use is linked to other risky behaviors, including other drug use and risky sex (Center for Behavioral Health Statistics and Quality, 2015; Connor et al., 2013). Regardless, alcohol marketing is increasingly available to underage populations across various media outlets (Ross et al., 2014), including social media (Jernigan & Rushman, 2014), television advertisements (Tanski et al., 2015), movies (Bergamini et al., 2013), and popular songs (Primack et al., 2014). Epidemiological research has established a link between marketing receptivity and the development of risky drinking in adolescents (Henriksen et al., 2008). For example, early alcohol advertisement exposure and brand familiarity are associated with intentions to use alcohol (Pasch et al., 2007) and a greater likelihood and earlier onset of drinking and binge drinking (i.e., consuming five or more alcoholic beverages on one occasion) (Ellickson et al., 2005; McClure et al., 2013; Tanski et al., 2015).

Nevertheless, prior research has called into question the ability to estimate without bias the relationship between alcohol marketing exposure and behavior (Molloy, 2016; Nelson, 2011). One recent meta-analysis of alcohol marketing and adolescent drinking identified the heterogeneity of effects estimated across studies of alcohol marketing and emphasized evidence for publication bias; this author suggested that the true effect may be more modest (Nelson, 2011). Another econometric study raised questions about endogeneity—the idea that marketing companies target drinkers who, for a number of reasons, seek out the television shows and magazines that have more alcohol advertisements (Molloy, 2016). Endogeneity bias results from the degree to which these unmeasured third variables (e.g., drinker characteristics) create positive bias for marketing exposure among drinkers, contributing to the marketing exposure–drinking effects estimate.

Another area that needs to be addressed in making a causal argument about alcohol marketing and drinking is biological plausibility. Alcohol industry materials emphasize the notion that marketing influences the brands that legal consumers choose to drink but not how much they drink (Beer Institute, n.d.). The current study addresses whether it is biologically plausible that exposure to alcohol images acutely affects brain reward system responses that could increase the probability of a drinking event, and whether the level of reward activation is associated with the heaviness of recent drinking. Establishing biological plausibility is another way of assessing a potential causal relationship between alcohol marketing and consumption.

Neuroimaging research has demonstrated that appetitive cues, such as images of food or cigarettes, consistently activate the brain's reward system—including the orbitofrontal cortex (OFC) and ventral striatum (VS) (Beaver et al., 2006;

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David et al., 2005; Demos et al., 2012; Lopez et al., 2014; Rapuano et al., 2016; Wagner et al., 2013)—which gauges personal relevance (Northoff & Hayes, 2011) and motivational value (Berridge et al., 2009). Moreover, the magnitude of food cue-related reward activation relates to real-world behaviors and outcomes, such as eating when temptation arises (Lopez et al., 2014) and weight gain (Demos et al., 2012). Although the reward system is sensitive to multiple cues across a variety of domains, the relationship between activity to a specific cue and motivated behavior appears to be domain specific. For example, food cue-reactivity in the VS predicted a change in body mass index 6 months later, whereas activation to sexual scenes predicted participants' number of sexual partners; however, neither predicted outcomes in the other domain (Demos et al., 2012). In neuroimaging studies with heavy and disordered drinkers, alcohol cues elicit activation in the VS, suggesting that these cues carry motivational value within that population (Ihssen et al., 2011; Schacht et al., 2013).

Recent research has capitalized on the naturalistic appeal of product advertisements to demonstrate that reward activation to fast-food commercials predicts adiposity (Gearhardt et al., 2014; Rapuano et al., 2016) and food choices (Bruce et al., 2016) in an adolescent population. Likewise, alcohol advertisements—which are rich with contextual information about the drinking experience, including the surrounding social context and associated brands—should enable a similar exploration of cue-reactivity and alcohol use. Building off previous research linking reward reactivity to real-world consumption and long-term outcomes (Demos et al., 2012; Lopez et al., 2014; Rapuano et al., 2016), the present study uses functional magnetic resonance imaging (fMRI) to examine whether alcohol advertisements elicit a similar, behaviorally relevant response in the reward system that relates to real-world drinking.

This study examines alcohol cue-reactivity in college students, a population that experiences especially frequent exposure to alcohol and vulnerability to peer pressure. Heavy underage drinking has been normalized on college campuses; further, student drinking is influenced by the perceived drinking norms of peers (Neighbors et al., 2007; O'Malley & Johnston, 2002). Adolescents are particularly sensitive to peer influence of this type, demonstrating increases in risky behavior and reward sensitivity in the presence of peers (Blakemore & Robbins, 2012; Knoll et al., 2015; Logue et al., 2014; Steinberg, 2008). Environmental factors (i.e., a culture of drinking on college campuses: Neighbors et al., 2007; O'Malley & Johnston, 2002); neurobiological factors, including an imbalance in the maturation of subcortical and prefrontal brain regions (Bava & Tapert, 2010; Somerville et al., 2011); and neurochemical changes (Spear, 2000) that occur during this developmental stage contribute to a particular vulnerability toward socially endorsed rewards (e.g., drinking) during this late stage of adolescence.

Unfortunately, adolescence is also a sensitive period of brain development when alcohol and other drug use have particularly detrimental outcomes, disrupting brain development and increasing the likelihood of dependence in adulthood (Crews et al., 2007; Fuhrmann et al., 2015; Squeglia et al., 2009; Zeigler et al., 2005). For these reasons, it is crucial to explore potential links between reward system activation to alcohol cues and real-world drinking behavior in this population of late-stage adolescent college students, whose brains are likely unaffected by drinking alcohol so far but who are at risk of developing problematic drinking behaviors.

Method

Participants

Fifty-three right-handed Dartmouth undergraduates (32 women, M_{age} = 19.75, age range: 18–22 years) were recruited and completed an fMRI scanning session followed by a survey assessing their alcohol consumption over the previous month. All participants had normal or corrected-to-normal visual acuity and reported no abnormal neurological conditions. Each participant provided informed consent according to the guidelines of the Dartmouth College Committee for the Protection for Human Subjects and was compensated with cash for participating. Ten scanned participants were excluded from the final fMRI analyses because of excessive head movement during the scan (within-run movement in excess of 3 mm; n = 3); failing to respond to at least 75% of trials during any of the four functional runs, indicating compromised attention to the stimuli (n = 6); or for reporting disordered eating, which could inordinately influence the outcome measure of interest (n = 1). Of the 43 participants included in the subsequent analyses, 24 were female, and the mean age was 19.83 years (SD = 0.49, age range: 18–22).

Stimuli

Stimuli consisted of 336 advertisement images of four product types: alcohol (84), fast food (84), cars (84), and technology (84), with the brand and product clearly displayed. Car and technology advertisements were used as non-appetitive control conditions for estimating the neural response to alcohol advertisements, and food advertisements were used as an appetitive control condition in a specific subset of analyses. Alcohol brands (Bacardi, Budweiser, Coors, Corona, Heineken, Smirnoff) were selected from a list of top beer and distilled spirits brands (Siegel et al., 2011); food brands (Burger King, McDonald's, Pizza Hut, Subway, Taco Bell, Wendy's) were selected from the top 20 list of national fast-food restaurants based on the QSR Magazine Top 50 Quick-Service and Fast-Casual Chains (www.qsrmagazine.com/reports/2011-qsr-50); and equivalent car (Buick,

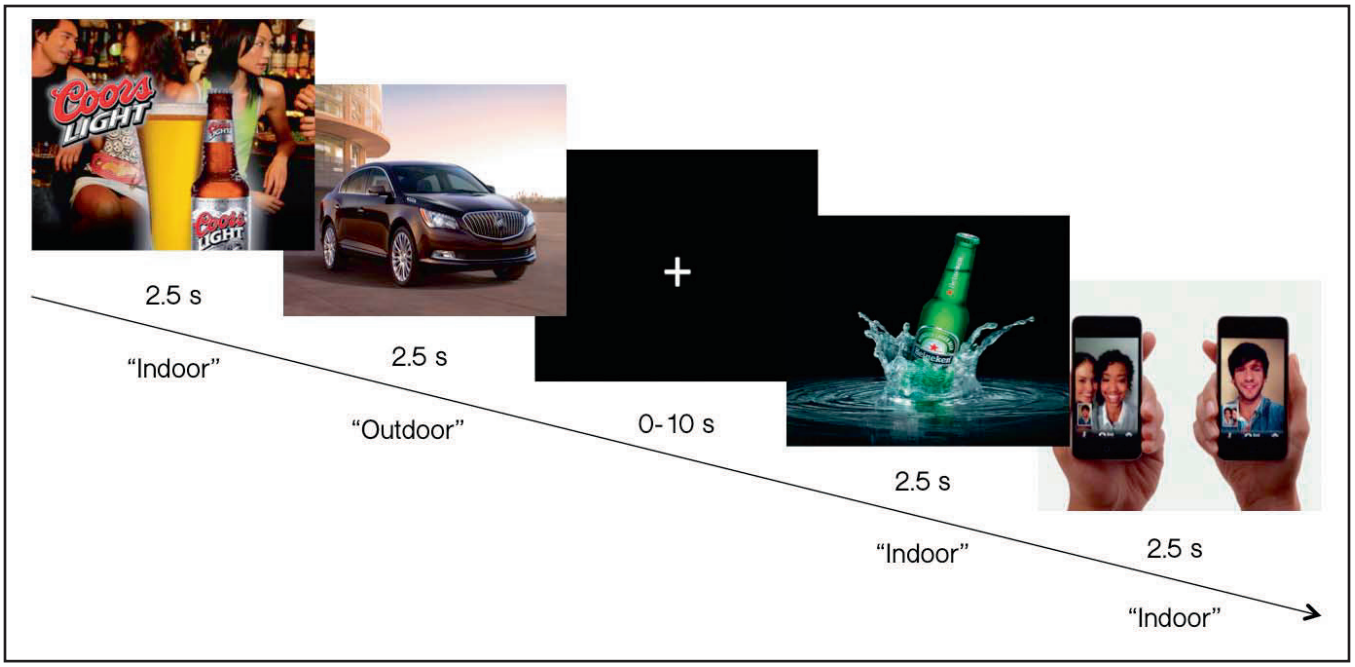

FIGURE 1. Representation of the alcohol cue-reactivity paradigm: 336 advertisements from four conditions (alcohol, food, cars, and technology) were presented for 2.5 seconds and were jittered with 30% fixation (0–10 seconds) for an estimation of baseline. Participants made indoor/outdoor judgments for each image using a button-box.

Ford, Honda, Hyundai, Jeep, Mitsubishi, Subaru, Toyota, Volkswagen) and technology (Apple, AT&T, Dell, Microsoft, Samsung, Sony, Verizon) brands were selected based on their online advertisement presence. Images were selected from an Internet search of these brands and were scaled to 800 × 600 pixels, using Adobe Photoshop (Version 12.0.4).

Procedure

Because previous reports have linked increases in cue-reactivity immediately following alcohol consumption to craving and consumption (Myrick et al., 2004; Tapert et al., 2003), we asked participants to refrain from consuming food, alcohol, and caffeine for 2 hours before the fMRI scan to ensure sobriety and a relatively equivalent state of satiety. During the fMRI scanning session, participants viewed each advertisement within a classic cue-reactivity paradigm (Figure 1; Demos et al., 2012). Each stimulus was presented for 2.5 seconds in a rapid event-related design with jittered fixation (for 30% of trials) to increase the estimability of the conditions. Data were collected over four functional runs, and the image type and brand were randomly ordered and counterbalanced across them. To ensure that participants attended to the stimuli and to conceal the true purpose of the study, we asked participants to determine whether each image was set indoors or outdoors and to make responses with a button press (left-handed response for "indoor" and right-handed response for "outdoor").

Following the scan, participants completed a survey assessing their drinking patterns over the previous month (items adapted from Tanski et al., 2015). The items included whether they had ever consumed alcohol ("Have you ever had a whole drink of alcohol, more than a sip or taste?"), their frequency of drinking ("In the past month, how often did you have a drink containing alcohol?"), typical number of drinks ("How many drinks containing alcohol do you have on a typical day when you are drinking?"), and frequency of binge drinking over the previous month ("In the past month, how often did you have five or more drinks on one occasion?"). The items measuring frequency of drinking and binge drinking were scored on a 0–4 scale (0 = *never,* 1 = *once,* 2 = *2–4 times,* 3 = *2–3 times a week,* 4 = *4 or more times a week*). The items measuring typical number of drinks consumed was scored on a 0–5 scale (0 = *nondrinker,* 1 = *1 or 2,* 2 = *3 or 4,* 3 = *5 or 6,* 4 = *7–9,* 5 = *10 or more*). The three items measuring frequency of drinking, typical number of drinks, and binge drinking were equally weighted and summed into a single scale (range: 0–13), which was created with the "scoreItems" function from the "psych" package (Revelle, 2015) in R.

Image acquisition

MRI was conducted with a Philips Achieva 3.0 Tesla scanner (Philips Medical Systems, Andover, MA) using a 32-channel SENSE (Sensitivity Encoding) head coil. Structural images were collected using a T1-weighted MP-RAGE protocol (TR = 9.9 ms; TE = 4.6 ms; flip angle = 8°; 1 × 1

× 1 mm^3 voxels). Functional images were acquired using a T2*-weighted EPI sequence (TR = 2500 ms, TE = 35 ms, flip angle = 90°, 3 × 3 × 3 mm^3 voxels). For each participant, four runs of 136 volumes (36 axial slices per whole-brain volume, 3 mm thick, 0.5 mm gap) were acquired for whole-brain coverage.

Image preprocessing

The fMRI data were analyzed using the general linear model in SPM8 (Wellcome Department of Cognitive Neurology, London, UK). For each functional run, data were preprocessed using a suite of preprocessing and analysis tools (https://github.com/ddwagner/SPM8w) to remove sources of noise and artifact and to correct for differences in slice acquisition timing. Images were realigned within and across runs to correct for head movement and were unwarped to account for remaining movement-related image distortions. Functional data were normalized into a standard stereotaxic space (3 mm isotropic voxels) based on the ICBM 152 brain template space (Montreal Neurological Institute [MNI]). Normalized images were then spatially smoothed using an 8 mm (full-width half-maximum) Gaussian kernel.

Functional magnetic resonance imaging analysis

For each participant, a general linear model incorporating task effects and covariates of no interest (a session mean, a linear trend, and six motion parameters from realignment) were convolved with a canonical hemodynamic response function and used to calculate parameter estimates for comparisons at each voxel. Individual contrast images were submitted to a group-level, random-effects analysis to create a mean whole-brain statistical image representing the conditions of interest. Monte Carlo simulations (10,000 iterations) were used to calculate the minimum cluster size (600 contiguous voxels), at a voxel-wise threshold of $p < .01$, required for whole-brain multiple comparisons correction (cluster-level, $p < .001$) using AFNI's 3dClustSim with the spatial autocorrelation function to control false positive rates (see Eklund et al., 2016; Cox et al., 2016).

To examine the involvement of putative reward system brain regions, a targeted regions-of-interest (ROI) analysis was used to extract parameter estimates from a priori regions (the left OFC and the left and right VS) identified in the group-level t-map contrasting alcohol images to control images. Importantly, each of these regions has been shown to correlate with other appetitive real-world behaviors (Demos et al., 2012; Schienle et al., 2009) and are the most commonly activated regions in meta-analyses of task-based studies of reward (Kelley et al., 2015). A 6-mm sphere was centered over peak activations in these regions—the left OFC (MNI: -33, 30, -15), left VS (MNI: -12, 3, -6), and right VS (MNI: 15, 3, -6)—identified as local maxima in the whole-brain comparison of ALCOHOL > CONTROL. In this way, ROI selection was unbiased in terms of predicting alcohol consumption, because all participants contributed equally to the selection of ROIs (Kriegeskorte et al., 2009; Vul et al., 2009). Parameter estimates were then extracted and used to predict self-reported drinking in offline statistical analysis using the R Statistical Software Package (R Core Team, 2016).

To identify additional regions in which activation to alcohol advertisements tracked with drinking, an exploratory whole-brain regression was conducted. Each participant's ALCOHOL > CONTROL contrast was entered into a regression analysis, with scores on a drinking survey entered as a covariate. Resulting statistical images were thresholded at the voxel level ($p < .01$) and cluster-corrected (327 contiguous voxels) for whole-brain multiple comparisons correction ($p < .01$).

Results

Behavioral results

The median self-reported frequency of drinking was two to four times in the previous month, with participants consuming three or four drinks on days when they were drinking. The median self-reported frequency of binge drinking (five or more drinks on one occasion) was once in the previous month (Figure 2). The three items measuring frequency of drinking, typical number of drinks, and binge drinking were correlated ($\sigma = .90$, inter-item $r = .76$) and were therefore combined into a composite scale (range: 0–13; $M = 5.53$, $SD = 2.81$) for subsequent analyses.

Brain regions responding to alcohol advertisements

Peak activations from the whole-brain contrast of ALCOHOL > CONTROL were identified in regions of the reward system, including the left and right VS (left: -12, 3, -6, $t[42] = 2.77$, $p < .01$; right: 15, 3, -6, $t[42] = 4.01$, $p < .001$) and the left OFC (-33, 30, -15; $t[42] = 5.83$, $p < .001$; Figure 3A). The left OFC peak activation was in close proximity (<6 mm) to OFC peak coordinates reported in similar studies of food reward (Rapuano et al., 2016; Van der Laan et al., 2011; Wagner et al., 2013). Additional activations were observed in the left fusiform gyrus and right inferior frontal gyrus (Table 1).

Predicting drinking behavior from LOFC and VS activation to alcohol advertisements

To identify whether activity in reward regions predicted drinking behavior, an ROI analysis (6 mm spheres) was performed on each of the three reward regions identified in

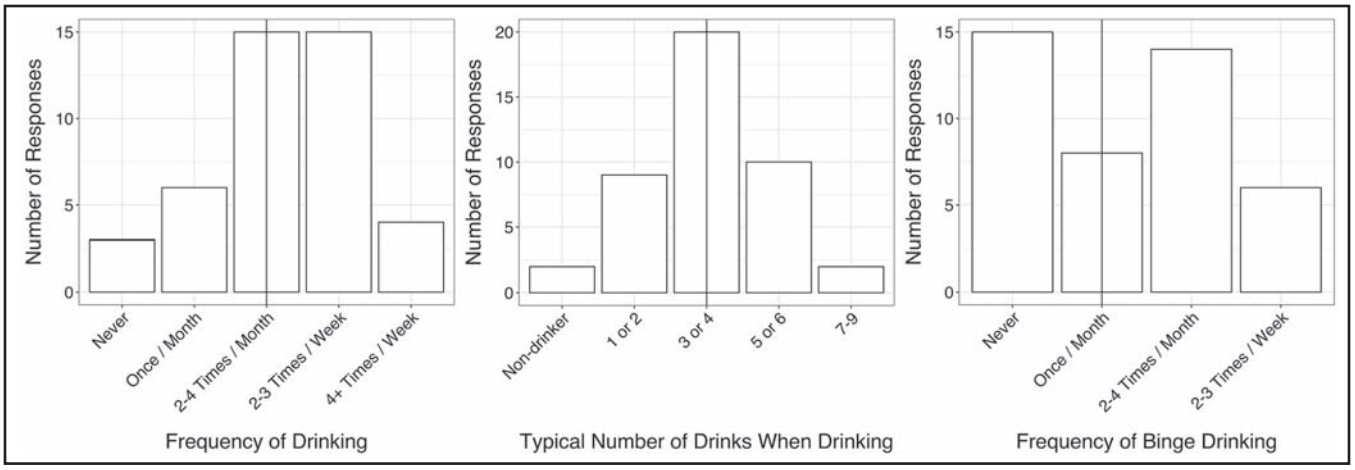

FIGURE 2. Histograms of response frequencies to each item in the drinking survey. Median responses are denoted with a vertical line (frequency of drinking: *Mdn* = 2 ["two to four times in the previous month"], interquartile range [IQR] = 1; typical number of drinks on days when drinking: *Mdn* = 2 ["three or four drinks"], IQR = 1.5; binge drinking: *Mdn* = 1 ["once in the previous month"], IQR = 2).

the ALCOHOL > CONTROL contrast and subsequently correlated with self-reported drinking scores. Whereas activity in the left OFC correlated with self-reported drinking, $r(41) = .37$, $p = .02$, bootstrapped (10,000 resamples) 95% CI [0.11, 0.57], the left and right VS did not—left VS: $r(41) = .17$, $p = .29$, bootstrapped (10,000 resamples) 95% CI [-.07, .38]; right VS: $r(41) = .28$, $p = .07$, bootstrapped (10,000 resamples) 95% CI [.05, .46] (Figure 3B).

Because activity in these regions was correlated (left OFC and left VS: $r(41) = .40$, $p < .01$; LOFC and right VS: $r(41) = .41$, $p < .01$; left VS and right VS: $r(41) = .71$, $p < .001$), a second analysis was conducted to determine whether the three reward regions collectively predicted drinking better than the LOFC alone. Specifically, the fit of a linear regression model for the LOFC predicting drinking was compared with a combined model that included the LOFC, left VS, and right VS predicting drinking. The LOFC-only model significantly predicted drinking, $F(1, 41) = 6.33$, $p = .02$, $R^2 = .13$, whereas the combined model only marginally predicted drinking, $F(3, 39) = 2.48$, $p = .08$, $R^2 = .16$. There was no significant difference in the fit of the two models, $F < 1$, $p = .55$, suggesting that VS activation in this study explained no additional variance in drinking behavior. When all three regions were included in the linear regression model, the LOFC marginally predicted drinking ($B = 2.41$, $p = .06$), whereas the left ($B = -1.16$, $p = .55$) and right VS ($B = 2.15$, $p = .28$) did not (Supplemental Figure A). (Three supplemental figures and one table appear as online-only accompaniments to the article.)

Alcohol-related activation but not food-related activation in the LOFC predicts drinking

Prior work has demonstrated domain specificity in reward brain regions such that cue-reactivity to food cues predicts weight gain, whereas cue-reactivity to erotic images predicts sexual interest and sexual behavior, but not vice versa (Demos et al., 2012). Here, we extend those findings to alcohol cues and drinking behavior. To demonstrate domain specificity in the relationship between cues and appetitive behaviors, we show that ALCOHOL > CONTROL activation predicted drinking behavior, but FOOD > CONTROL did not. Indeed, in the current study, FOOD > CONTROL activation in the LOFC did not predict drinking, $r(41) = 0.16$, $p = .30$. In addition, including both ALCOHOL > CONTROL and FOOD > CONTROL activations as predictors of drinking in the same regression model, alcohol-related OFC activation predicted drinking ($B = 3.40$, $p = .02$) but food-related OFC activation did not ($B = -0.87$, $p = .50$; coefficient plot in Supplemental Figure B). Whole-brain differences between ALCOHOL and FOOD cue-reactivity were observed in the left inferior occipital gyrus, right middle occipital cortex, right inferior frontal gyrus, left superior occipital cortex, and right postcentral gyrus (Supplemental Figure C and Supplemental Table A).

Exploratory whole-brain regression analysis with self-reported drinking

To identify additional brain regions that showed a relationship between brain activation (ALCOHOL > CONTROL) and self-reported drinking, we conducted an exploratory whole-brain regression analysis on this comparison with self-reported drinking scores. The regression analysis revealed additional positive relationships between activations and self-reported drinking (cluster-corrected to $p < .01$ [voxel-wise threshold $p < .01$], minimum cluster size = 327 contiguous voxels) in a region of the occipital cortex (15, -84, 42), left temporal lobe (-39, -12, -15), and cingulate cortex (-12, 18, 36; Table 2 and Figure 4).

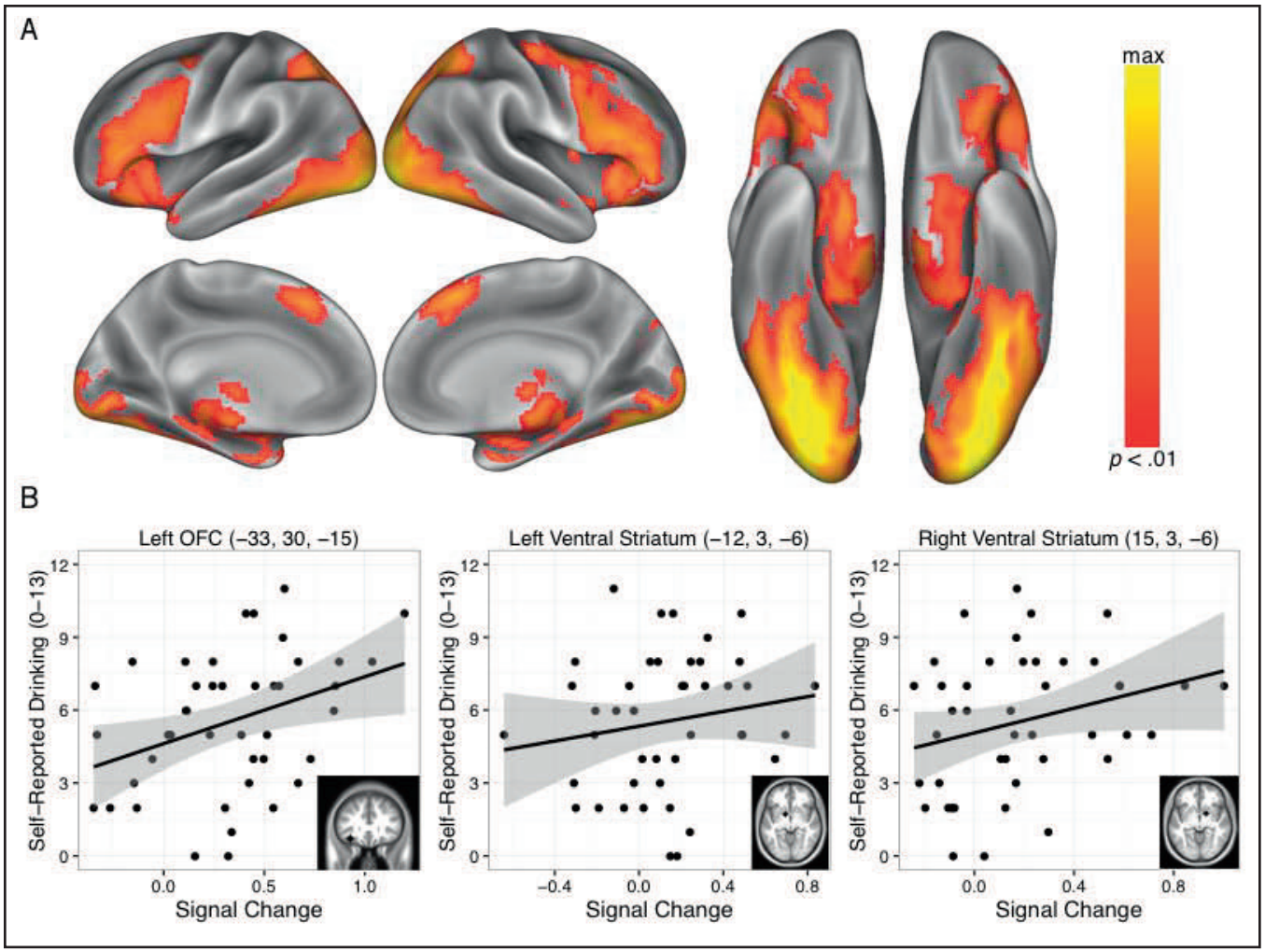

FIGURE 3. **A.** Brain regions activating to ALCOHOL > CONTROL advertisements. Whole-brain activations ($p < .01$, 600 contiguous voxels) from the ALCOHOL > CONTROL contrast are depicted on an inflated cortical surface (Marcus et al., 2011). Greater activation for ALCOHOL advertisements was observed in the left fusiform cortex, right inferior frontal gyrus, and supplementary motor area. **B.** Signal change in the left orbitofrontal cortex (OFC) region of interest defined from the whole-brain ALCOHOL > CONTROL contrast correlated with alcohol consumption (0–13), $r(41) = 0.37$, $p = .02$—the left and right ventral striatum (VS) did not: left VS: $r(41) = 0.17$, $p = .29$; right VS: $r(41) = 0.28$, $p = .07$.

TABLE 1. Regions more active (voxel-wise $p < .01$, cluster-corrected to $p < .001$) for ALCOHOL > CONTROL advertisements

	Coordinates (MNI)				
Region	X	Y	Z	Volume (mm^3)	Peak T
Left fusiform cortex[a]	-36	-60	-15	12,922	13.14
Right fusiform cortex	33	-66	-15		12.87
Left inferior occipital cortex	-27	-90	-12		12.12
Right inferior frontal gyrus[a]	51	21	24	2,207	5.89
Right precentral gyrus	48	6	27		4.65
Right inferior frontal gyrus	48	33	12		4.62

Notes: Volumes refer to entire supra-threshold clusters. Region names adapted from Automated Anatomical Labeling in SPM. MNI = Montreal Neurological Institute. X, Y, Z refer to MNI coordinates for the peak voxels in the activated cluster. Peak T value refers to the maximum t-value in the activated cluster. [a]Indicates the location of the most activated peak in the cluster. All other coordinates refer to subcluster local maxima more than 8 mm apart.

TABLE 2. Regions whose response to ALCOHOL > CONTROL advertisements covaried (voxel-wise $p < .01$, cluster-corrected to $p < .01$) with alcohol consumption

Region	Coordinates (MNI)			Volume (mm^3)	Peak T
	X	Y	Z		
Occipital cortex	15	-84	42	1,219	4.55
Left temporal lobe	-39	-12	-15	343	3.85
Cingulate cortex	-12	18	36	550	4.70

Notes: Region names adapted from Automated Anatomical Labeling in SPM. MNI = Montreal Neurological Institute. X, Y, Z refer to MNI coordinates for the peak voxel in the activated cluster. Peak T value refers to the maximum t-value in the activated cluster.

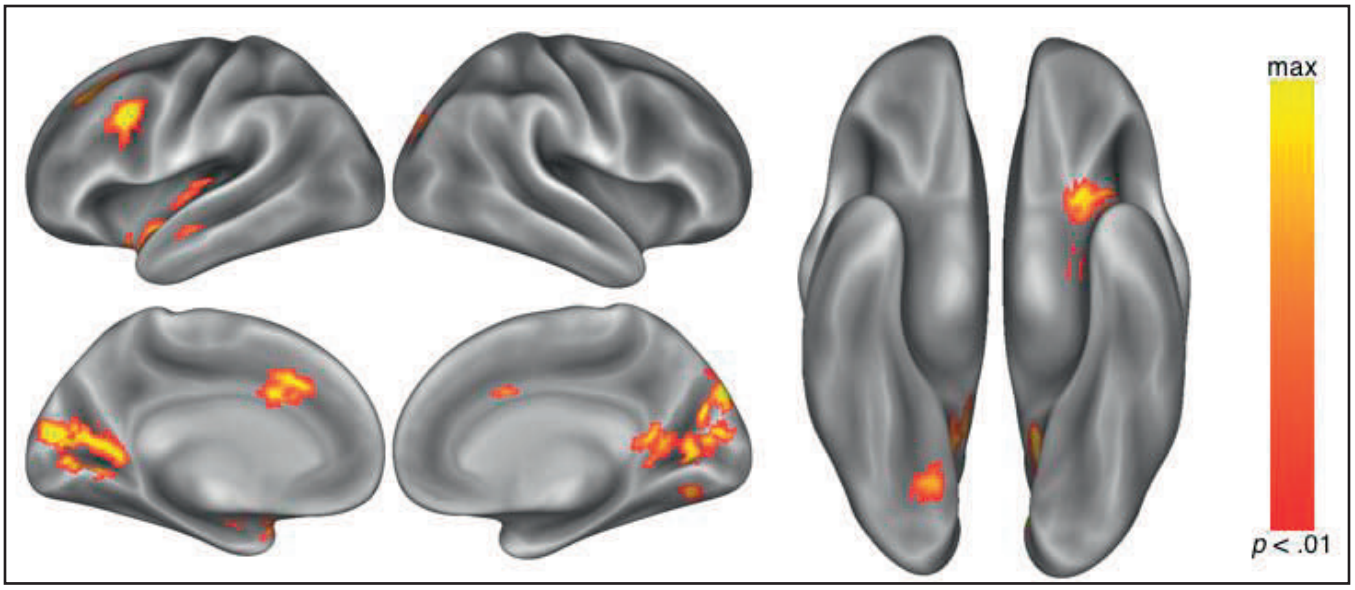

FIGURE 4. Whole-brain activations ($p < .01$, 327 contiguous voxels) from the ALCOHOL > CONTROL contrast covaried with alcohol consumption. Activations were observed in the left temporal lobe, occipital cortex, and cingulate cortex.

Discussion

The present study builds off prior epidemiological and functional neuroimaging research to confirm that alcohol advertisements are processed as rewarding stimuli within a nondisordered, college student population. Reward system activation generally reflects the motivational value of a reward: the drive to consume (Berridge et al., 2009). Our paradigm was modeled after a commonly used food cue-reactivity paradigm (e.g., Demos et al., 2012; Lawrence et al., 2012; Tang et al., 2012), which activates the reward system with images of appetizing foods. And in fact, alcohol advertisements activated brain regions that typically respond to food cues, including the left OFC (Beaver et al., 2006; Van der Laan et al., 2011) and regions of the VS (Demos et al., 2012). Whereas the VS may respond to positive and motivationally salient outcomes (Cooper & Knutson, 2008), the OFC may establish connections between cues and outcomes and attach affective value to rewards (Kringelbach, 2005; O'Doherty et al., 2001). Both brain regions facilitate approach toward valuable and personally relevant outcomes. In this study, individual differences in alcohol-related OFC activation predicted self-reported alcohol consumption.

Importantly, this relationship between activation in the OFC and drinking was specific to alcohol stimuli. These findings complement those reported by Demos and colleagues (2012), who found that food cue-reactivity in reward regions, but not alcohol cue-reactivity, predicted weight gain. Here we demonstrate the opposite—that alcohol cue-reactivity in the OFC predicts drinking but food cue-reactivity does not.

Although we did not have specific predictions regarding cingulate activation in the present study, a meta-analytic search for the peak coordinate identified in the present study using a large-scale database of fMRI studies (Yarkoni et al., 2011) revealed a strong association between cingulate activation and the term "anticipated" ($z = 6.36$). Moreover, previous studies of alcohol cue-reactivity in populations with alcohol use disorder have identified activation in the cingulate cortex in response to alcohol cues (e.g., Myrick et al., 2004). The anterior cingulate cortex is also an integral node in the cingulo-opercular network known to participate in

salience detection of relevant cues (e.g., Power & Petersen, 2013), and might play a role in anticipating and detecting salient appetitive cues in the present study. Collectively, these results suggest specificity in reward activation that is individualized and behaviorally relevant.

The findings reported here may help bridge the gap between what is known of the relationship between cue-reactivity and behavior in other appetitive domains (e.g., eating) and the literature on alcohol cue-reactivity and its relationship to alcohol dependence. Relative to nondisordered drinkers, those with alcohol dependence show increased alcohol cue-reactivity in visual, prefrontal, and limbic regions, and this activation scales with craving and consumption (Myrick et al., 2004; Schacht et al., 2013; Tapert et al., 2003). Further, alcohol cue-reactivity in these regions decreases following a period of abstinence or cue-exposure training (Brumback et al., 2015; Vollstädt-Klein et al., 2011). That we find activation in similar regions of the reward system and cingulate cortex suggests that a common neural mechanism underlying reward and motivational processes may become oversensitized during addiction. A better understanding of the neurobiological and environmental factors that promote alcohol use early in life may offer crucial insights into why some individuals are more at risk for problem drinking in adulthood.

In fact, college students are a unique population with respect to drinking behavior because the culture of drinking on college campuses permits more frequent exposure to alcohol cues (Neighbors et al., 2007; O'Malley & Johnston, 2002). Moreover, in terms of neurobiological development, most college students are in a late stage of adolescence (Crews et al., 2007). Adolescents in this age range are particularly susceptible to the context in which rewards appear, demonstrating enhanced risk-taking behavior and inhibitory control failure to rewards presented in a "hot" (e.g., socially salient) context (Blakemore & Robbins, 2012; Somerville et al., 2011; Steinberg, 2008). This proclivity has been linked to differential developmental trajectories between subcortical reward regions (maturing earlier) and prefrontal control regions (maturing later; Bava & Tapert, 2010; Somerville et al., 2010, 2011). College drinking primarily occurs in social contexts (Christiansen et al., 2002) and as a result of social motivations and peer pressure (Borsari & Carey, 2001; Kuntsche et al., 2005). Given the motivational significance of the social context for young college drinkers, future research should target this contextual effect on alcohol-related reward processing.

One factor that was not considered in the present study was the acute influence of recent alcohol consumption on brain activity. Prior research has observed drug-related increases and decreases in frontal regions up to 48 hours following consumption across a range of cognitive tasks (Goldstein & Volkow, 2011). More commonly, studies have reported increased alcohol cue-reactivity in visual, prefrontal, and limbic regions immediately following alcohol consumption in disordered drinkers (Myrick et al., 2004; Tapert et al., 2003). Although our participants were instructed not to consume any food or beverage in the 2 hours before the scan, we did not collect reports of alcohol consumption from the previous 48 hours and, therefore, cannot rule out the possibility of acute changes in measured brain activation from recent alcohol consumption.

These findings may be further limited by the level of measurement permitted by the items in our drinking questionnaire. A potentially more sensitive measure of drinking could be obtained by collecting count data for these same measures (e.g., number of times drinking in the previous month, number of drinks consumed on a typical day drinking, number of times binge drinking in the previous month). However, retrospective reports of both general drinking patterns and specific number of drinks consumed often underestimate true consumption—with underreporting increasing with higher levels of drinking (Monk et al., 2015; Stockwell et al., 2004). As new technology emerges to more directly measure drinking behavior in combination with self-reports (e.g., objective measures of blood alcohol content or smartphone/Global Positioning System indicators of participants' location near drinking establishments), we may be better positioned to more accurately characterize the relationship between reward responsivity and drinking behavior.

Conclusion

Alcohol advertisements activated regions of the reward system that commonly respond to appetitive cues, and this activation predicted self-reported drinking during the preceding month. By positioning alcohol as a reward cue in a nondisordered population, this study aimed to understand the link between predictors, motivations, and alcohol consumption that may be important for mitigating potentially risky drinking behaviors later in life. Because this relationship was established in a college student population, it raises concerns about marketing alcohol to a vulnerable, underage audience.

Conflict of Interest Statement

The authors declare no competing financial interests.

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