Aggression is Associated With Greater Subsequent Alcohol Consumption: A Shared Neural Basis in the Ventral Striatum

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Abstract

Alcohol use and abuse (e.g., binge drinking) are among the most reliable causes of aggressive behavior. Conversely, people with aggressive dispositions (e.g., intermittent explosive disorder) are at greater risk for subsequent substance abuse. Yet it remains unknown why aggression might promote subsequent alcohol use. Both aggressive acts and alcohol use are rewarding and linked to greater activity in neural reward circuitry. Through this shared instantiation of reward, aggression may then increase subsequent alcohol consumption. Supporting this mechanistic hypothesis, participants' aggressive behavior directed at someone who had recently rejected them, was associated with more subsequent beer consumption on an *ad-lib* drinking task. Using functional MRI, both aggressive behavior and beer consumption were associated with greater activity in the bilateral ventral striatum during acts of retaliatory aggression. These results imply that aggression is linked to subsequent alcohol abuse, and that a mechanism underlying this effect is likely to be the activation of the brain's reward circuitry during aggressive acts.

Keywords**:** aggression, alcohol, reward, ventral striatum, fMRI

Introduction

Alcohol and aggression share an intimate relationship. To date, this relationship has been largely portrayed as one-sided, with alcohol consumption causing aggression and not the reverse. However, recent research has shown that aggression is a significant risk factor for substance abuse (Coccaro et al., 2016; Coccaro, Fanning, & Lee, 2017). Although the mechanisms underlying alcohol's effect on aggression are well explicated (e.g., Giancola, 2004), the mechanisms linking aggression to subsequent alcohol consumption remain unknown. In what follows, we describe a brain imaging study that tested the hypothesis that aggression predicts greater alcohol consumption, which can be explained, in part, by recruitment of the brain's reward circuitry during aggression.

Alcohol-Consumption Increases Aggression

Alcohol consumption, both in the laboratory and the real-world, is reliably linked to increased aggression (Bushman & Cooper, 1990; Chermack & Giancola, 1997; Exum, 2006; Foran & O'Leary, 2008). Alcohol exerts such an aggression-increasing influence because it reduces inhibitory cognitive abilities and increases frustration-driven impulses to aggression (Bushman, 1997; Giancola, 2000, 2004; Godlaski & Giancola, 2009; Ito, Miller, & Pollock, 1996). Such alcohol-aggression links are stronger among individuals high in psychopathic traits (Birkley, Giancola, & Lance, 2013). Even simple alcohol cues are enough to elicit aggressive acts in the laboratory (Pedersen, Vasquez, Bartholow, Grosvenor, & Truong, 2014; Subra, Muller, Bègue, Bushman, & Delmas, 2010). Across all of these research findings, it is clear that alcohol consumption is a reliable and robust cause of aggressive behavior. The next section entertains the

possibility that aggression may also correlate with alcohol consumption because both activities rely on similar neurological networks related to reinforcement and reward.

Might Aggression Predict Subsequent Alcohol-Consumption?

Alcohol increases aggression, but might aggression also increase alcohol consumption? Initial evidence that this may be the case came from a longitudinal investigation of adolescents, which found that aggressive behavior predicted greater alcohol consumption and greater rates of alcohol-induced aggression in subsequent years (White, Brick, & Hansell, 1993). Subsequent work supported these findings by demonstrating that alcohol use and aggression have reciprocal relationships over time (Huang, White, Kosterman, Catalano, & Hawkins, 2001). Individuals diagnosed with intermittent explosive disorder (IED), a form of psychopathology characterized by aggressive tendencies, exhibited greater rates of substance abuse (Coccaro et al., 2016, 2017). Crucially, onset of IED preceded the onset of substance abuse by a significant amount of time, whereas substance use did not serve as a risk factor for IED. This suggests that aggressive traits and acts predispose individuals towards the abuse of substances such as alcohol.

Non-human animal models also provide support for the ability of aggression to increase subsequent alcohol consumption. Indeed, mice who had recently fought with a conspecific consumed more alcohol than controls (Hilakivi-Clarke & Lister, 1992). Thus, the alcohol-aggression link may also work in reverse. Yet how does an aggressive act predispose one towards alcohol consumptions?

A Potential Mechanism: Aggression and Neural Reward Circuitry

A growing body of research has demonstrated that aggression is not necessarily a result of negative affect (e.g., anger) but can also be motivated by positive forms of affect (e.g., hedonic reward; Chester, 2017). As evidence of aggression's rewarding nature, retaliatory (but not unprovoked) aggressive acts are associated with greater activity in the brain's reward circuitry, specifically the ventral striatum (Chester & DeWall, 2016). This mirrors findings for alcohol, which reliably link alcohol cues and consumption to greater reward sensitivity (Lyvers, Czerczyk, Follent, & Lodge, 2009) and greater activity in the ventral striatum (Boileau et al., 2003; Braus et al., 2001). This shared basis in reward may explain the reciprocal relationship between aggression and alcohol, with aggressive acts recruiting the brain's reward circuitry, which then predisposes individuals towards other reward-based acts such as alcohol consumption.

Present Research

The present research served to test the hypotheses that (A) aggression would be associated with greater subsequent alcohol consumption, (B) retaliatory aggression would be associated with greater activity in the ventral striatum, and (C) this ventral striatum activity during retaliatory aggression would be associated with more subsequent alcohol consumption. To test these hypotheses, we recruited a sample of regular alcoholic beer drinkers without histories of alcohol abuse. While undergoing functional MRI (fMRI), participants were all provoked via an experimental induction of social rejection and then they completed a behavioral aggression task. Afterwards, participants were removed from the scanner and then completed an *ad-libitum* beer drinking task and a battery of personality questionnaires that included measures of self-reported alcohol abuse and sensation-seeking. The self-report alcohol abuse measure

was included to provide an additional assessment of problematic alcohol consumption, besides the drinking task. A sensation-seeking measure was included as a covariate in subsequent analyses, given the strong links between this facet of impulsivity and aggression (Miller, Zeichner, & Wilson, 2012) and alcohol consumption (Hittner & Swickert, 2006). A brief mood measure was also included to assess aggression's impact on participants' affective levels. If the data supported the present hypotheses, this would serve as evidence for the ability of reward circuitry to predict the link between aggressive acts and alcohol use.

Methods

Participants

Participants were 24 community members and undergraduate psychology students (13 females, 11 males; *M* = 23.04, *SD* = 2.46, range: 21-30) who were compensated with images of their brain and either $50 (community members) or course credit (undergraduates). Potential participants were recruited using an online screening survey for the following inclusion criteria: between 21-35 years old, previous consumption of alcohol, clicked 'yes' when asked if they "enjoyed the taste of alcoholic beer", clicked 'yes' when asked if they "regularly drink alcoholic beer (at least once each week)", and right-hand dominance. Participants were excluded if they reported any history of claustrophobia, psychological or neurological pathology, color blindness, seizures, substance abuse or dependence, current use of psychoactive medications, pregnancy, a current body mass index of 35 or greater, or presence of metallic objects or devices inside their body (excluding certain dental devices).

Materials

Alcohol Use Disorders Identification Test (AUDIT). The 10-item AUDIT assesses the frequency with which individuals engage in various indicators of alcohol abuse (Reinert & Allen, 2007; Saunders, Aasland, Babor, De la Fuente, & Grant, 1993). Participants respond to items such as “How often do you have six drinks or more on one occasion?” along a 0 (Never) to 4 (Daily or Almost Daily) response scale.

Need Threat Scale. To assess participants’ state negative and positive affect, they completed the 30-item Need Threat Scale (Williams, 2009). This scale contains two, three-item subscales that measure negative and positive affect that were adapted to measure current levels of affect. Each item asked participants to indicate the extent to which they were currently-feeling negative (angry, bad, sad) and positive (good, happy, pleasant) affective states. Participants responded to each affective state along a 1 (Not At All) to 5 (Extremely) response scale.

UPPS-P Impulsivity Scale. The UPPS-P Impulsivity Scale is a well-validated measure of the various facets of trait impulsivity (Lynam, Smith, Whiteside, & Cyders, 2006; Whiteside & Lynam, 2001), and includes 59 statements that participants respond to along a 1 (strongly agree) to 4 (strongly disagree) Likert-style scale. The UPPSP assess five facets of impulsive behavior: negative urgency, lack of premeditation, lack of perseverance, sensation-seeking, and positive urgency.

Procedure

Participants who passed on our online screening survey were scheduled to arrive at the University of Kentucky’s Magnetic Resonance Imaging and Spectroscopy Center. Participants provided informed consent in a behavioral testing room, where they also had the study explained to them, and were given a cover story that we were exploring

how brain structure and function were involved in individual's taste preferences for different kinds of beer. Participants practiced the Taylor Aggression Paradigm that they would later complete in the MRI scanner, against a computerized avatar. Afterwards, participants were screened for metallic objects, escorted into the MRI scanner, and began MRI scanning procedures.

Cyberball task. To provoke participants towards acting aggressively, we employed the Cyberball social rejection task (as in Chester et al., 2014; Williams, Cheung, & Choi, 2000). In this task, participants played a virtual ball-tossing game with two (fictitious) partners, under the cover story that they were to mentally visualize the task as if it were occurring in real life. In the beginning of the task, participants received an equitable number of ball-tosses from their partners, an indicator of acceptance. Towards the end of the task, participants stopped receiving the ball from their partners, an indicator of rejection.

Taylor Aggression Paradigm. To measure the neural correlates of retaliatory aggression, we employed the Taylor Aggression Paradigm (as in Chester & DeWall, 2016). In this task, participants competed against an opponent (i.e., one of their rejecters from Cyberball) over the internet to see who could press a button faster. As an ostensible motivational component of the task, participants were punished if they lost the competition via an aversive noise blast. Conversely, if participants won the competition their opponent heard the noise blast and they did not. Crucially, the volume of the noise blast delivered to their opponent was set by the participant and served as the measure of aggressive behavior.

The task consisted of 14 blocks, 6 of which were classified as retaliatory aggression blocks and 8 were classified as non-retaliatory aggression blocks. Within each block, there were 6 events. Each block began with a fixation cross that modeled baseline neural activity. Then, participants set the volume of their partner's noise blast (i.e., the aggression event). A blank screen then appeared for a jittered duration, which then gave way to a competition in which participants pressed a button as fast as they could when a red square appeared on the screen. Participants then saw what volume level their opponent set for them. Finally, participants saw whether they won or lost the competition. If participants lost the competition, they heard an aversive noise blast that varied from 1 (silence) to 4 (extremely loud, though not dangerous). Whether a given block was preceded by their opponent setting a loud (3, 4) or soft (1, 2) volume level determined whether it was categorized as retaliatory (i.e., after a loud blast; 6 blocks) or non-retaliatory (i.e., after a soft blast; 8 blocks). The order of retaliatory/non-retaliatory blocks was randomized and held constant across participants. Wins and losses were split evenly with an independently-randomized order that was held constant across all participants (7 wins, 7 losses). After completing an unrelated cue reactivity task, exiting the scanner, retrieving personal items, using the restroom, walking back to the adjacent behavioral testing room, and completing a short battery of questionnaires (approximately 20-30 minutes to complete all aforementioned activities), participants then completed a short battery of questionnaires, including the Negative and Positive Affect subscales of the Need Threat Scale, which assessed participants' currently-felt levels of negative and positive affect.

***Ad-Libitum* Beer Drinking Task.** After completing the questionnaires, participants were seated in a nearby testing room, where four different refrigerated 12-ounce beers were placed in front of them in red, plastic cups. Participants were asked to evaluate the pleasantness of each beer along multiple criteria (taste, smell, color, aftertaste, mouthfeel, overall quality), using a 1 (Very Unpleasant) to 7 (Very Pleasant) response scale. Participants were led to believe that the beers were alcoholic, when in fact each of the beers was a different non-alcoholic beer. Participants were instructed to drink “a meaningful amount” of each beer and to evaluate it based on these consumption experiences. Then, participants were left alone with the beers, and a cup of water, for 15 minutes. After this allotted time, the experimenter returned and asked the participant to stop drinking. The experimenter then collected the beers and measured the weight of the remaining beer that was consumed, in ounces. This number was subtracted from the 48 fl oz. that were originally given to participants to compute a ‘beer drank’ index. Participants then completed a longer battery of personality questionnaires that included the AUDIT and UPPS-P Impulsivity Scale. Participants were then fully debriefed about the deception inherent in the study and escorted from the laboratory with thanks. Included in this debriefing were several open ended questions about whether participants believed that they were being deceived about anything in the study (e.g., whether the beer was alcoholic versus non-alcoholic). No participants expressed significant suspicion about the deceptive elements of the study.

MRI Data Acquisition and Processing

All MRI data were obtained using a 3.0-T Siemens Magnetom Trio scanner. Echo planar BOLD images were acquired with a T2*- weighted gradient across the entire

brain with a 3D shim (matrix size = 64 × 64, field of view =224 mm, echo time =28ms, repetition time = 2.5-second, slice thickness = 3.5 mm, 40 interleaved axial slices, flip angle = 90°). To allow for registration to native space, a coplanar T1-weighted MP-RAGE scan was also acquired from each participant (1 mm^3 isotropic voxel size, echo time = 2.56ms, repetition time = 1.69-second, flip angle = 12°).

The Oxford Center for Functional MRI of the Brain (FMRIB)'s Software Library (FSL version 5.0) was used to conduct all preprocessing and fMRI analyses (Smith et al., 2004; Woolrich et al., 2009). Reconstructed functional volumes underwent head motion correction to the median functional volume using FSL's MCFLIRT tool. FSL's Brain Extraction Tool was used to remove non-brain tissue from all functional and structural volumes using a fractional intensity threshold of 0.5. After a series of data quality checks, functional volumes underwent interleaved slice-timing correction, pre-whitening, spatial smoothing (using a 5-mm full-width-half-maximum Gaussian kernel), and temporal high-pass filtering (100-second cutoff). These processed brain volumes were then fed into subsequent data analyses.

MRI Data Analysis

Preprocessed fMRI data was analyzed using a two-level general linear model (GLM).

First level (within-participants). Each participant's BOLD signal was modeled at each voxel across the entire brain with a fixed-effects analysis that modeled each block's events using a canonical double-gamma hemodynamic response function with a temporal derivative. The GLM included retaliatory aggression, non-retaliatory aggression, competition, pre-competition, opponent's volume settings, and outcome

events, while leaving fixation baselines un-modeled. All six motion parameters were modeled as nuisance regressors.

A linear contrast compared retaliatory to non-retaliatory aggression (Retaliatory Aggression > Non-Retaliatory Aggression). The focus on retaliatory forms of aggression was due to previous work demonstrating that the ventral striatum tracks the degree of retaliatory aggression and not unprovoked aggression (Chester & DeWall, 2016). Resulting contrast images from these analyses were first linearly registered to native space structural volumes and then spatially normalized to a Montreal Neurological Institute (MNI) stereotaxic space template image (resampled into 2 x 2 x 2mm^3 voxels).

Second level (across-participants). Each participant's contrast volumes from the first step were then fed into a group-level, mixed-effects GLM that created group average maps for both contrasts for each voxel across the entire brain. Cluster-based thresholding (Heller, Stanley, Yekutieli, Rubin, & Benjamini, 2006; Worsley, 2001) was applied to each of the group activation maps. Family-wise error correction based on Gaussian random field theory was then applied to each voxel across the entire brain (corrected threshold: $Z > 2.3$, $p < .05$).

Parameter estimates were averaged across all voxels of a ventral striatum region-of-interest (ROI), and then were extracted, *a priori*, in units of percent BOLD signal change. The ventral striatum ROI mask was taken from the Wake Forest University Pickatlas (Maldjian, Laurienti, Kraft, & Burdette, 2003; Figure 1).

Results

Descriptive Statistics

Participants perpetrated a variable amount of aggression across the noise blast task (Table 1). Participants also consumed a wide range of beer and exhibited substantial variability on the AUDIT, the Sensation-Seeking subscale of the UPPSP, and the Negative and Positive Affect measures (Table 1). Males consumed more beer than females, $t(22) = 3.22$, $p = .004$, $d = 1.37$, and had higher AUDIT scores, $t(22) = 2.36$, $p = .028$, $d = 1.01$. However, males and females did not differ in their overall aggression, $t(22) = 1.01$, $p = .324$, $d = 0.43$, or retaliatory aggression, $t(22) = 0.55$, $p = .590$, $d = 0.23$.

Supporting the validity of the aggression task, volume settings were higher for retaliatory than non-retaliatory blocks, $t(23) = 2.63$, $p = .015$, $d = 0.23$. Further, aggressive behavior was associated with greater negative affect, $r(22) = .43$, $p = .035$, and less positive, $r(22) = -.43$, $p = .035$, affect after the aggression task. These findings fit with affective models of aggression that emphasize the momentary hedonic benefits of aggression (Chester & DeWall, 2017) that ultimately result in worsened mood (Bushman, 2002), and ultimately support the validity of this aggression task.

Neural Correlates of Retaliatory Aggression

Across the whole brain, retaliatory aggression (as compared to non-retaliatory aggression), was associated with greater activity in the left ventrolateral prefrontal cortex (VLPFC) and the right superior parietal lobe (SPL; Figure 2; Table 2).

This contrast only reflected the opportunity to retaliate and does not reflect the extent to which participants actually engaged in retaliatory aggression. As such, whole-brain regression analyses were conducted, in which participants levels of retaliatory aggressive behavior were regressed onto their whole-brain BOLD signal from the

Retaliatory > Non-Retaliatory Aggression contrast. Whole-brain analyses failed to identify any significant clusters of aggression-correlated neural activity.

However, when multiple comparisons corrections from this retaliatory aggression regression analysis were then constrained to the bilateral ventral striatum ROI, we observed a cluster of significant activation in the left ventral striatum (Figure 3; 11 voxels, peak $Z = 2.61$, $p = .030$). Supporting the idea that activity in the ventral striatum reflects the rewarding experience of appetitive outcomes, an ROI analysis of the ventral striatum exhibited a small cluster of significant activation from the Won > Lost contrast of the outcome phase of the aggression task, 4 voxels, peak $Z = 2.61$, $p = .047$; MNI coordinates for peak voxel: x = 14, y = 8, z = -8.

Linking Aggression to Alcohol Consumption Through Striatal Responses to Retaliatory Aggression

As predicted, noise blast volume settings across the aggression task were associated with greater activity in the ventral striatum during retaliatory aggression, $r(22) = .47$ [.06, .72], $p = .022$ (Figure 4A). Further, these volume settings were positively associated with greater beer consumption on the *ad-lib* drinking task, $r(22) = .42$ [.00, .70], $p = .040$ (Figure 4B). Supporting our mechanistic hypothesis, ventral striatum activity during retaliatory aggression was associated with greater beer consumption on the *ad-lib* drinking task, $r(22) = .47$ [.06, .73], $p = .022$ (Figure 4C). These aforementioned effects were not moderated by participant age or gender.

A mediation analysis (using the PROCESS macro version 2.16.2 for SPSS, model 4, 5,000 bias-corrected and accelerated bootstrapped re-samples; Hayes, 2012) failed to demonstrate a significant indirect effect of ventral striatum activity during

retaliatory aggression, 95% *CI* = -0.09, 5.02. However, when sensation-seeking was modeled as a covariate of both the mediator (retaliatory striatal activity) and the outcome (beer consumption), a significant indirect effect was observed, *B* = 1.45, *SE* = 1.16, 95% *CI* = 0.01, 5.01 (Figure 5). This model explained 38.92% of the variance in beer consumption, $F(3, 20) = 4.25$, $p = .018$.

Using the AUDIT as the dependent measure of alcohol consumption, aggression across the noise blast task was unassociated with AUDIT scores, $r(22) = .16$ [-.44, .56], $p = .470$, yet retaliatory striatal activity was associated with greater AUDIT scores, $r(22) = .46$ [.06, .73], $p = .026$. As before, a mediation analysis failed to demonstrate a significant indirect effect of ventral striatum activity during retaliatory aggression, 95% *CI* = -0.00, 0.40. Including sensation-seeking as a covariate did not result in a significant indirect effect on AUDIT scores, 95% *CI* = -0.00, 0.40.

Discussion

Aggression and alcohol use impose costs on individuals, relationships, and society. Understanding the causes of these acts is a crucial, yet complicated, task for behavioral scientists. At least one cause for aggression is alcohol consumption, which impairs inhibition and impels individuals towards harming others (Bushman & Cooper, 1990; Chermack & Giancola, 1997; Exum, 2006). But aggression may also impel people to drink alcohol. Aggressive acts can predispose individuals to alcohol and other illicit substance use (Coccaro et al., 2016, 2017; White et al., 1993). Yet what motivates aggression's link to alcohol consumption remains largely unexamined. Combining functional brain imaging techniques with a behavioral measure of alcohol consumption,

we tested the hypothesis that aggression relates to greater alcohol consumption through a shared neural basis in reward circuitry activity during aggression.

Replicating previous research (Chester & DeWall, 2016), we observed that activity in the ventral striatum during retaliatory aggressive acts was associated with greater aggression. This finding supports aggression's fundamentally rewarding nature and neural basis in the brain's reward circuitry (Chester, 2017). Building off of these findings, we observed that aggressive behavior predicted a greater amount of (non-alcoholic) beer consumed recently after the aggressive acts. This correlational evidence supports the reciprocal nature of the link between perceived alcohol consumption and aggression (Huang et al., 2001).

Suggesting that the mechanism underlying aggression's link to greater alcohol consumption is partially rooted in reward, the amount of (non-alcoholic) beer consumed in the *ad-lib* beer drinking task and self-reported alcohol abuse was associated with greater ventral striatum activation during retaliatory aggression. Mediation analyses, which included sensation-seeking as a covariate, observed a significant indirect effect whereby the link between aggression and (non-alcoholic) beer consumption was partially explained by retaliatory activity in the ventral striatum. Together, these findings provide preliminary evidence that reward circuitry activation partially accounts for the reciprocal nature of perceived alcohol consumption and aggression. Further, the use of sensation-seeking as a covariate grants some inferential confidence that our observed associations were not an artifact of a dispositional tendency to pursue rewarding activities writ large.

Despite our preliminary evidence for a striatal mechanism underlying the putative aggression-alcohol-consumption link, there are likely to be other neural mediators. Retaliatory aggressive behavior is associated with a blunted recruitment of the regulatory functions of the VLPFC (Chester & DeWall, 2016). The VLPFC subserves self-regulation more broadly (Heatherton & Wagner, 2011), and aggression's ability to blunt this neural region's functionality may facilitate subsequent alcohol consumption by impairing individuals' ability to regulate such reward-based behavior. Research that includes brain imaging techniques during (as opposed to preceding) post-aggression alcohol consumption would allow us to test this alternative mechanism empirically.

Limitations and Future Directions

This study was limited in many ways, yet the correlational nature of our findings was perhaps chief among them. It may be that another extraneous variable (e.g., poor impulse control) explains our study's observed associations. Further, it is uncertain if aggression or brain activity during aggression causes increases in alcohol consumptions, or whether these correlations do not reflect a causal relationship. Future research is needed in which aggression is experimentally manipulated and then alcohol consumption is measured, to establish aggression's causal role in substance abuse. In addition, counterbalancing the order of the alcohol and aggression tasks is necessary for future research to ensure that the observed effect of aggression on alcohol consumption is not merely due to an order effect. Our sample size was small and so future research should employ larger and more diverse samples in order to seek to replicate our findings. Another limitation comes from the reverse inference we relied upon that ventral striatum activity reflects the hedonic experience of reward, and not

some other psychological process. We did observe activity in the ventral striatum to wins (compared to losses) on the outcome phase of the aggression task, which supports the ventral striatum's association with rewarding outcomes. Further, the ventral striatum is reliably linked to reward and less reliably linked to other processes (Bartra, McGuire, & Kable, 2013; Diekhof, Kaps, & Gruber, 2012; Kühn & Gallinat, 2012). Future research should measure other indicators of reward (e.g., activation of the facial muscles involved in smiling; Cikara & Fiske, 2011). Another limitation of this project was that we did not provide a proper control beverage for the alcohol in our study. As such, it is possible that beer consumption simply reflected participants' choice to consume a 'rewarding' beverage and not alcohol. Going forward, the inclusion of a non-alcoholic, sweet-tasting beverage as a control would be an effective way to experimentally examine this possibility.

Conclusions

This paper is an initial investigation of reward's role in motivation the alcohol-aggression cycle. We observed preliminary and correlational evidence that aggression may promote consumption of beverages that people perceive to be alcoholic and that the recruitment of the brain's reward circuitry may partially explain this effect. We hope that future research replicates and extends our findings, which suggest a complex dynamic between aggression and substance use. Such a nuanced understanding of externalizing tendencies may yield practical insight that results in reduction of the violent and addictive acts that continue to harm our shared world.

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Tables

Table 1. Descriptive statistics for study variables.

Measure	*M*	*SD*	Observed Range	Possible Range	*α*
Total Aggression	2.27	0.91	1.00 - 4.00	1.00 - 4.00	.95
Retaliatory Aggression	2.40	0.97	1.00 - 4.00	1.00 - 4.00	.88
Non-Retaliatory Aggression	2.18	0.90	1.00 - 4.00	1.00 - 4.00	.91
Beer Drank (in fl oz.)	13.78	8.05	4.00 - 30.00	0.00 - 48.00	n/a
AUDIT	0.88	0.60	0.20 - 2.40	0.00 - 5.00	.83
Sensation-Seeking	2.98	0.70	1.50 - 4.00	1.00 - 4.00	.87
Post-Aggression Negative Affect	1.50	0.87	1.00 - 4.67	1.00 - 5.00	.95
Post-Aggression Positive Affect	3.97	0.93	1.00 - 5.00	1.00 - 5.00	.94

Table 2. Whole-brain fMRI main effect results from the Retaliatory Aggression > Non-Retaliatory Aggression contrast.

Brain Region	Voxels	peak *Z* (*p*)	peak MNI coordinates (x,y,z)
Ventrolateral Prefrontal Cortex	417	3.40 (.013)	-44, 50, -8
Superior Parietal Lobe	337	3.58 (.042)	58, -40, 40

Figures

Figure 1. ROI mask for the bilateral ventral striatum. Coordinate is in MNI space.

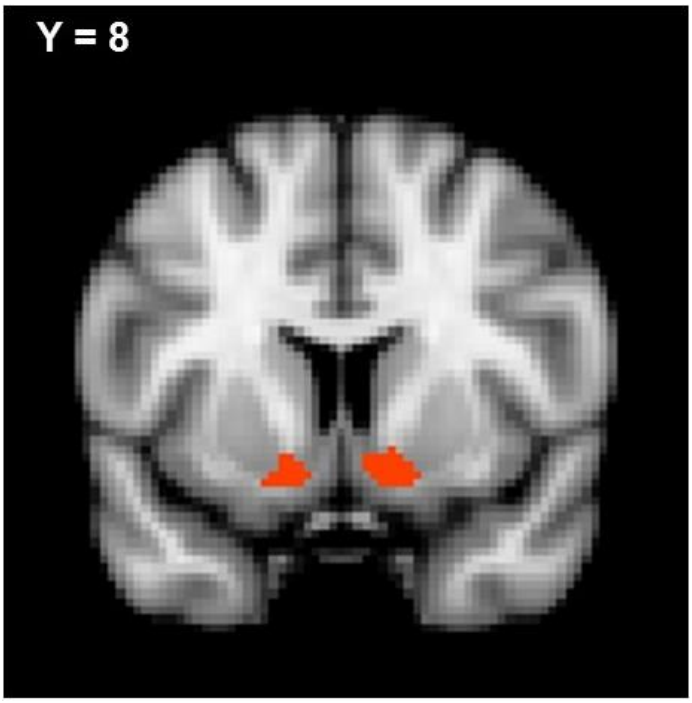

Figure 2. Greater left VLPFC activity associated with retaliatory > non-retaliatory aggression. Coordinates are in MNI space.

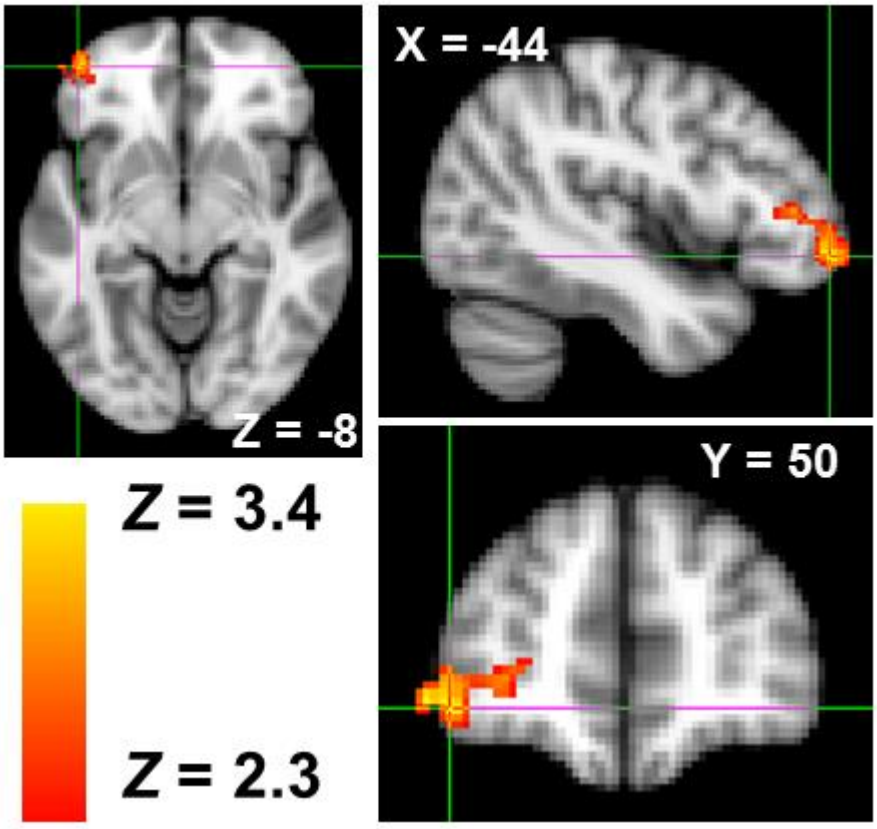

Figure 3. Activity in the left ventral striatum associated with greater retaliatory aggressive behavior.

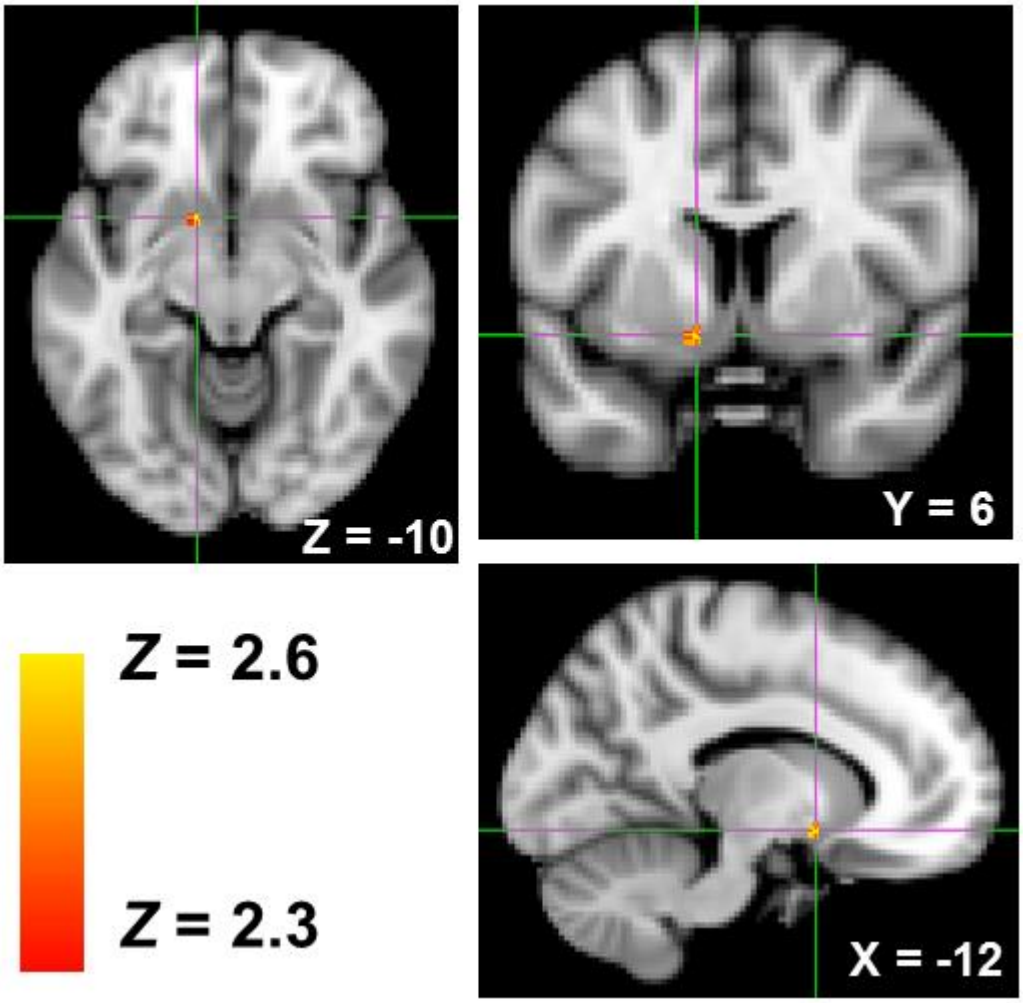

Figure 4. Scatterplots from Study 2 depicting zero-order associations between (A) aggressive behavior across the aggression task and ounces of beer consumed, (B) aggressive behavior across all the aggression task and % BOLD Signal Change estimates from the Retaliatory > Non-Retaliatory Aggression contrast of the TAP, and (C) fluid ounces of beer consumed and % BOLD Signal Change estimates from the Retaliatory > Non-Retaliatory Aggression contrast of the TAP.

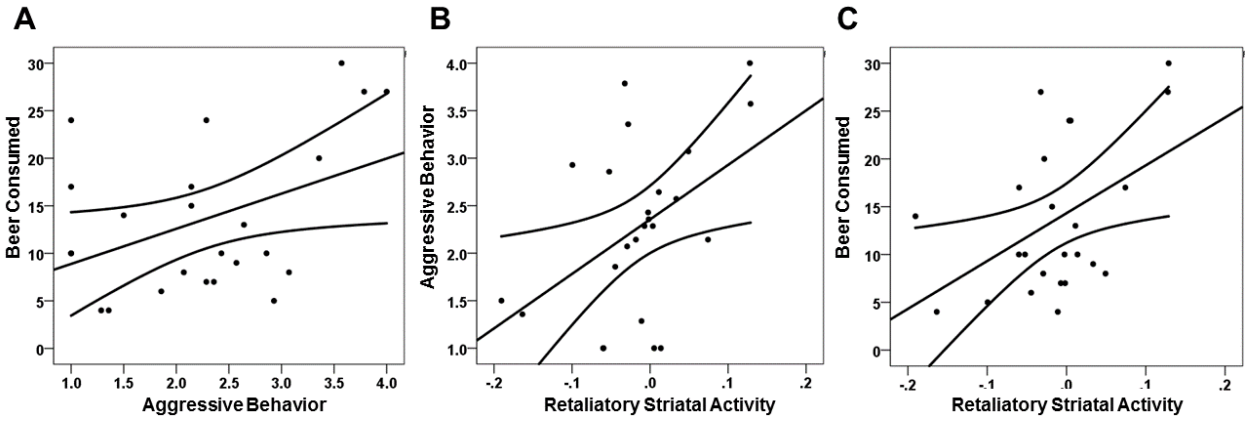

*Figure 5. The direct effect of aggressive behavior on subsequent beer consumption is mediated by greater ventral striatum (VS) recruitment during retaliatory aggression, while controlling for sensation-seeking. Values represent unstandardized regression coefficients. †p < .09, *p < .05*