The Interactive Effect of Social Pain and Executive Functioning on Aggression: An fMRI Experiment

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Abstract

Social rejection often increases aggression, but the neural mechanisms underlying this effect remain unclear. This experiment tested whether neural activity in the dorsal anterior cingulate cortex (dACC) and anterior insula in response to social rejection, predicted greater subsequent aggression. Additionally, it tested whether executive functioning moderated this relationship. Participants completed a behavioral measure of executive functioning, experienced social rejection while undergoing functional magnetic resonance imaging (fMRI), and then completed a task in which they could aggress against a person who rejected them using noise blasts . We found that dACC activation and executive functioning interacted to predict aggression. Specifically, participants with low executive functioning showed a positive association between dACC activation and aggression, whereas individuals with high executive functioning showed a negative association. Similar results were found for the left anterior insula. These findings suggest that social pain can increase or decrease aggression, depending on an individual's regulatory capability. (149 words)

Keywords: social pain, social rejection, aggression, executive functioning, fMRI, dACC

The Interactive Effect of Social Pain and Executive Functioning on Aggression: An fMRI Experiment

Why people behave aggressively continues to be an important question for scientists and laypersons alike. The world is less violent now than ever before (Pinker, 2011). But people continue to scuffle, fistfight, and brawl. One potent cause of aggression is social rejection (e.g., Gaertner et al., 2008; Leary et al., 2006; Twenge et al., 2001). Given the basic need for social acceptance (Baumeister & Leary, 1995), one might expect that people would respond to social rejection by trying very hard to gain re-inclusion from those who rejected them. However, people often do the opposite—they respond to social rejection with high levels of aggression. The present experiment focused on why this might occur.

Despite some early successes, researchers have struggled to identify the psychological and neural processes through which social rejection increases aggression, and who is most likely to respond to social rejection with aggression. To fill this gap, the present experiment integrated behavioral and functional neuroimaging methods to test the hypothesis that social rejection increases aggression through greater activation in two brain regions associated with the pain of rejection, namely the dorsal anterior cingulate cortex (dACC) and anterior insula (Eisenberger et al., 2003). Crucially, we predicted that the relationship between activation in these regions and aggression would depend on individual differences in executive functioning.

Consistent with the appraisal and decision-making component of the General Aggression Model (GAM; Anderson & Bushman, 2002; DeWall et al., 2011), we hypothesized that greater dACC and anterior insula activation would predict greater

aggression among participants relatively *low* in executive functioning because they have a propensity towards impulsive actions. Conversely, we hypothesized that greater dACC and anterior insula activation would relate to lesser aggression among participants relatively *high* in executive functioning because of their propensity toward thoughtful actions.

The General Aggression Model: Affect and Appraisal

The GAM (Anderson & Bushman, 2002) posits that provocative social encounters, such as rejection, can increase aggression through emotions such as anger. However, the GAM also states that provocation and its associated emotions do not inevitably lead to aggression. In the GAM, cognitive appraisals and decision-making strategies influence whether emotions produce impulsive, aggressive actions or thoughtful, non-aggressive ones. Such 'thoughtful' responses to provocation require the ability of individuals to control any prepotent responses to respond aggressively and, simultaneously, to consciously and effortfully reappraise the situation (Anderson & Bushman, 2002). Therefore, aggression may occur as a result of the interaction between people's affective reactions and their ability to control and reappraise them.

Social Pain and Aggression

Social pain—the aversive emotional response that accompanies social injuries such as rejection, ostracism, and exclusion (MacDonald & Leary, 2005)—is a likely candidate for the affective construct that promotes rejection-related aggression. Underscoring its importance for human survival, social pain mirrors physical pain in several ways. Social rejection is associated with activation of neural regions involved in the affective component of physical pain (Eisenberger et al., 2003; Eisenberger, 2012).

Specifically, rejection is associated with activation of the dACC and the anterior insula. But why would social pain promote aggression?

Physical pain reliably increases aggression as a defensive reaction to bodily harm (Berkowitz, 1983, 1993). This ability of physical pain to promote aggression is relevant to the rejection-aggression link because social and physical pain share neural substrates: the dACC and anterior insula (Eisenberger et al., 2003). But social pain does not imply the subjective co-occurrence of physical pain. Given this overlap between physical and social pain, we predicted that activation in the dACC and anterior insula in response to a socially painful experience—social rejection, would directly related to increased aggression.

The Role of Executive Functioning

As predicted by the GAM, the effect of social pain on aggression should be modulated by 'top-down' or 'cold' inhibitory and reappraisal processes (Anderson & Bushman, 2002). Pain is a 'hot' affect-laden psychological process (e.g., Anderson et al., 1998). The GAM posits that such affective processes promote aggression, but inhibitory and reappraisal processes can deem the expression of such affect as not useful (e.g., lashing out at your boss after he or she berates you) and limit their expression. Such aggression-related inhibitory and reappraisal processes, and their ability to regulate aggression, rely on executive functioning (Giancola, 2000). Executive functioning is a cognitive ability that regulates goal-oriented behaviors (Milner, 1995).

The relationship between social pain and aggression may depend on the amount of executive functioning individuals bring with them to aggressive situations. Indeed, individuals who perform poorly on behavioral measures of executive functioning are

more aggressive than their high executive-functioning counterparts (e.g., Hoaken et al., 1998). Individuals who are low in executive functioning are often unable to inhibit the 'hot' affective processes that promote aggression (Hoaken et al., 2003). As the pain of rejection increases, the affect-driven impulse to aggress increases (Anderson et al., 1998). As such, we predicted that for individuals low in executive functioning, greater dACC and anterior insula activation in response to social rejection would relate to *more* post-rejection aggression because they favor impulsive over thoughtful actions. More specifically, individuals low in executive functioning are unable to engage in the inhibitory and reappraisal processes that underpin thoughtful, non-aggressive action.

In contrast, individuals high in executive functioning are readily capable of engaging in the deliberative appraisal processes that inhibit 'hot' affective processes and subsequently aggression (Giancola, 2000). Regulatory functions associated with executive functioning are recruited in an increasing manner as pain and its associated affect increase (Eccleston & Crombez, 1999). As such, we predicted that greater dACC and anterior insula activation in response to social rejection would relate to *less* post-rejection aggression among individuals high in executive functioning because they favor thoughtful over impulsive actions. This is because individuals high in executive functioning are readily able to engage in the inhibitory and reappraisal processes that underpin thoughtful, non-aggressive action.

These predictions were only expected to hold under conditions in which aggression would *not* be a useful strategy for the aggressor. Specifically, when aggression is a useful option (e.g., fending off an attacker) individuals across the executive functioning spectrum are likely to aggress. As the GAM states, aggression is

sometimes an adaptive response that is thoughtfully chosen (Anderson & Bushman, 2002). In the context of purely emotional and reactive aggression, however, we predicted that individual differences in executive functioning would manifest as shifts in rejection-associated aggression.

Method

Participants

Participants were 40 healthy, right-handed undergraduate students who received course credit and $65. For safety reasons, participants were excluded from our study if they reported any history of claustrophobia, seizures, head trauma, or an injury involving a metallic object. Additionally, participants were excluded if they reported a body-mass-index greater than 30 (as individuals exceeding this cutoff might be uncomfortable in the confined MRI scanner), pregnancy or suspected pregnancy, color blindness, psychoactive medication use, or psychological/neurological pathology. One participant was excluded from analyses because of behavioral issues in the MRI environment that resulted in distorted fMRI data. Four participants did not complete the aggression measure because the MRI scan exceeded the allotted time. Analyses were therefore performed on the 35 remaining participants (17 females; M_{Age}=19.03, *SD*=1.36).

Procedure

Pre-scan measure of executive functioning. To measure executive functioning, participants completed a computerized version of the Stroop (1935) color-naming task. The Stroop color-naming task is a widely used, reliable and valid measure of executive functioning because performance on the task requires attention

maintenance, working memory and the rapid and accurate inhibition of prepotent responses (see Engle, 2002). Participants were instructed to identify the font-color of words as fast as possible, while ignoring the word's meaning. There were 40 congruent trials in which the word's meaning matched the font-color (e.g., the word RED in red font) and 40 incongruent trials in which the word's meaning did not match the font-color (e.g., the word RED in green font), ordered randomly. This 80-trial design has been effectively used in previous research to measure individual differences in executive functioning (e.g., Kiefer et al., 2005; Dinn & Harris, 2000). This task was performed several days before the MRI portion of the experiment, as the depletion of executive functioning from this task may have impacted subsequent neural activation and aggressive behavior (DeWall, Baumeister, Stillman, & Gailliot, 2007).

Scanner task. Participants were informed that they would play three rounds of a computerized ball-tossing game (Cyberball) in an MRI scanner with two same-sex partners located in nearby scanners (Williams et al., 2000). In reality, participants played with a preset computer program that was designed to produce a within-participants experience of both social acceptance and rejection. Cyberball was implemented in the MRI scanner as a block-design with three rounds (60 seconds each). Before each round, participants were presented with instructions to rest for 10 seconds. This was followed by a screen instructing them to "get ready" for the upcoming round (2 seconds). In rounds 1 and 2, participants were accepted for the entire duration of the task, receiving one-third of all ball-tosses. In round 3, participants received the ball three times, after which their partners only threw the ball to each other.

Acceptance was operationalized as occurring throughout rounds 1 and 2, as well as throughout the first part of round 3, in which participants received the ball three times. Rejection was operationalized as occurring during the second part of round 3, after participants had received the ball three times and then witnessed three more ball-tosses without receiving a toss themselves (30 second duration). After playing Cyberball and a series of anatomical MRI scans, participants were removed from the MRI scanner in order to complete the Need Threat Scale (van Beest & Williams, 2006), a 20-item questionnaire intended to assess current social distress due to social rejection.

Post-scan aggression measure. Participants then completed a behavioral measure of aggression. Participants were told they would play a computerized game against one of their partners from Cyberball. This game took the form of a competitive reaction-time task in which the winner could deliver aversive and prolonged noise to the loser through headphones. The aggression task consisted of 9 trials. Prior to each trial, participants set the volume of the noise blast their partner would receive if the participant won the round, ranging from Level 1 (60 decibels) to Level 10 (105 decibels) in 5 decibel intervals. A non-aggression option, Level 0, was also provided. Participants also controlled how long their opponent suffered by setting the duration of the noise blast, which could range from 0 seconds to 5 seconds in half-second intervals. After each trial, participants saw whether they won or lost, as well as the volume and intensity settings their partners had ostensibly set for them. Participants won five trials and lost four trials (determined randomly, despite being told that their performance was what determined the outcome of each trial). Basically, within the ethical limits of the laboratory, participants controlled a weapon that could be used to blast their opponent

with unpleasant noise. The construct validity of this task is well established (Anderson & Bushman, 1997; Bernstein et al.,1987; Giancola & Zeichner, 1995). It has been used for decades as a reliable and valid measure of laboratory aggression (Taylor, 1967).

fMRI Data Acquisition

All images were collected on a 3T Siemens Magnetom Trio scanner. Functional images were acquired with a T2-weighted gradient echo sequence with the following parameters: 2.5s repetition time, 28ms echo time, 64 x 64 matrix, 224 x 224mm field of view, 40 3.5mm axial slices acquired in interleaved order. A 3D shim was applied before functional data acquisition. These parameters allowed for whole brain coverage with 3.5mm cubic voxels. A high-resolution, T1-weighted image was also acquired from each participant so that functional data could be registered to native anatomical space and then normalized to the Montreal Neurological Institute (MNI) atlas space.

fMRI Preprocessing

All preprocessing and statistical analyses were conducted using FSL [Oxford Center for Functional Magnetic Resonance Imaging (FMRIB); Woolrich et al., 2009; Smith et al., 2004]. Functional volumes were reconstructed from k-space using a linear time interpolation algorithm to double the effective sampling rate, the first of which was removed to allow for signal equilibration. Remaining functional volumes were corrected for head movement to the median volume using MCFLIRT (Jenkinson et al., 2002), corrected for slice-timing skew using temporal sinc interpolation, pre-whitened using FILM, and smoothed with a 5-mm FWHM Gaussian kernel. To remove drifts within sessions, a high-pass filter with a cutoff period of 120s was applied. Non-brain

structures were stripped from functional and anatomical volumes using FSL's Brain Extraction Tool (Smith, 2002).

fMRI Data Analysis

FMRI analysis was performed using FSL's FMRI Expert Analysis Tool (FEAT version 5.98). A fixed-effects analysis modeled event-related responses for each run of each participant. Acceptance and Rejection blocks were modeled as events using a canonical double-gamma hemodynamic response function with a temporal derivative. Pre-block instructions were modeled as a nuisance regressor while rest blocks were left un-modeled. The contrast of interest was Rejection>Acceptance. Functional volumes and first-level contrast images from this analysis were first registered to corresponding structural volumes using 7 degrees of freedom, and then spatially normalized to an MNI stereotaxic space template image using 12 degrees of freedom with FMRIB's Linear Image Registration Tool (FLIRT; Jenkinson & Smith, 2001; Jenkinson et al., 2002). FMRIB's Local Analysis of Mixed Effects module (FLAME; Beckmann et al., 2003; Woolrich, 2004) was used to perform top-level, mixed-effects analysis, which created group average maps for contrasts of interest. *Z* (Gaussianized T/F) statistic images were thresholded using clusters determined by $Z>2.3$ and a (corrected) cluster significance threshold of $p<.005$ in an *a priori* regions-of-interest (ROI; Worsley, 2001; Heller et al., 2006). Functional data from the activated main effect cluster from our contrast- and region-of-interest were converted to units of percent signal change, averaged across each participant and extracted (as outlined by Mumford, J. http://mumford.bol.ucla.edu/perchange_guide.pdf).

Construction of Regions-of-Interest

ROIs of the dACC and anterior insula were created by Way and colleagues (2009) from the automated anatomical atlas (AAL) using MNI coordinates (Tzourio-Mazoyer et al., 2002). The dACC ROI used a rostral boundary of y=33 based on criteria established by Vogt and colleagues (2003), and a caudal boundary of y=0. The anterior insula ROIs used a caudal boundary of y=8 to correspond to the agranular insula.

Behavioral Data Analysis

Response-times from correct trials on the Stroop task were averaged for each participant, separately for congruent and incongruent trials. Response times on congruent trials were subtracted from those on incongruent trials to create an index of how much participants were able to utilize executive functioning. Higher values indicated greater interference from incongruent trials. To enhance interpretation, these values were then reverse-scored so that higher values corresponded to higher executive functioning.

In the aggression task, noise intensity and volume levels had high internal reliabilities (Cronbach α=.91 and .88, respectively) and were significantly correlated, $r(34)=.79$, $p<.001$. Thus, we standardized and summed intensity and duration levels across the nine trials to create a more reliable measure of aggression.

Results

Stroop Task Results

Stroop Task responses had an accuracy rate of 96.7 percent, across all participants. Stroop task response-times were characterized by a main effect of congruency, $t(34)=-3.71$, $p=.001$, $d=0.33$. Replicating the classic Stroop effect,

participants were faster at correctly identifying congruent word-color pairings (*M*=466.90ms, *SD*=107.95ms) than incongruent pairings (*M*=521.99ms, *SD*=149.27ms).

Imaging Results

Social rejection, compared to social acceptance, led to increased activity in the dACC and bilateral anterior insula (Figure 1; Table 1; Rejection>Acceptance contrast). This finding replicates prior social rejection research on the dACC and anterior insula as indicators of social pain (DeWall et al., 2010; Eisenberger et al., 2003; Kross et al., 2011).

Figure 1. (A) dACC and (B) bilateral anterior insula activation associated with Rejection>Acceptance. Coordinates are in MNI space.

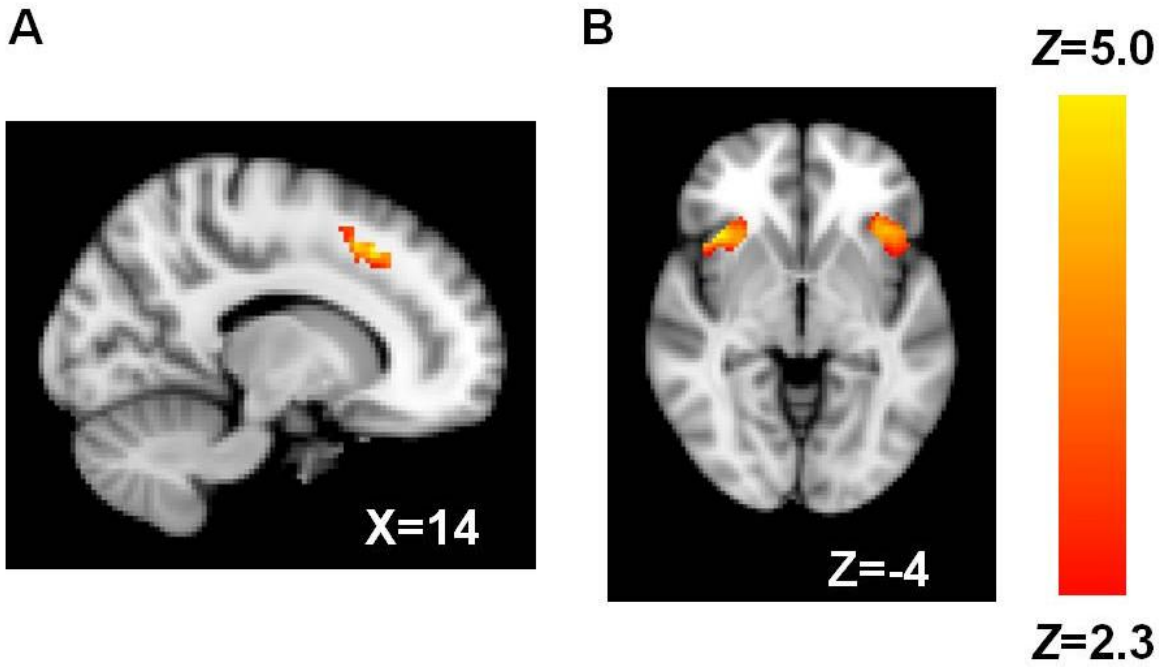


Table 1. Brain regions-of-interest associated with Rejection>Acceptance.

Brain Regions	Contiguous Voxels	Peak *Z*	Peak MNI Coordinates (x,y,z)

dACC	470	4.96	14 , 22, 44
Anterior insula	477	5.27	40, 22, 0
	542	4.75	-42, 22, -6

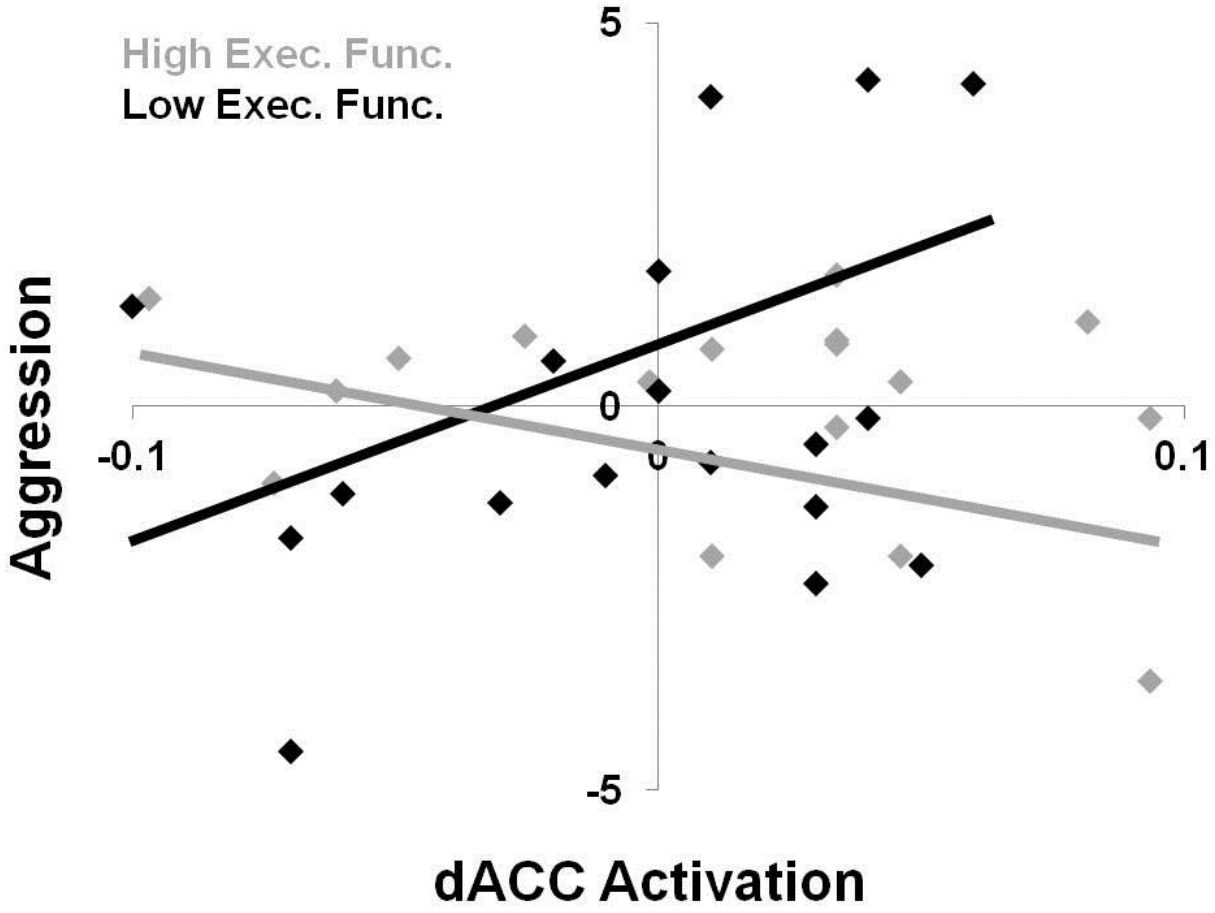
Post-rejection social distress, as measured by mean scores from the Need Threat Scale (Cronbach α=.90) taken 60 minutes after the social rejection task, was uncorrelated or marginally correlated with percent signal change units from activated clusters (obtained from rejection > acceptance contrast) in the dACC [r(34)=.14, p=.40], left insula [r(34)=.28, p=.09] and right insula [r(34)=.27, p=.10]. These null effects are similar to those obtained in previous studies that delayed giving the Need Threat Scale to participants (Zadro et al., 2006).

Participants high and low in executive functioning did not differ in activation of the social pain network in response to social rejection, as correlations between executive functioning and each of the three neural regions of the social pain network were non-significant: dACC activation, r(34)=.08, p=.65; left anterior insula activation, r(34)=-.03, p=.87; right anterior insula activation, r(34)=.09, p=.60.

Moderation Analysis

As predicted, dACC activation interacted with executive functioning to predict aggression, β=0.63, t(30)=3.82, p=.001 (Figure 2). At low levels (-1 *SD*) of executive functioning, dACC activation was positively associated with aggression, β=0.55, t(34)=2.89, p<.01. In contrast, at high levels (+1 *SD*) of executive functioning, dACC activation was negatively associated with retaliatory aggression, β=-0.47, t(34)=-2.26, p<.05.

Figure 2. Interactive effect of dACC activation and executive functioning on aggression. Aggression units are in Z-scores with higher values representing greater aggression on the Taylor Aggression Paradigm. dACC activation is in units of percent signal change from the Rejection > Acceptance contrast.



Subsequent analyses examined the effect of executive functioning on aggression at low and high levels of dACC activation. At low levels (-1 *SD*) of dACC activation, executive functioning did not correspond with aggression, β=0.31, $t(34)$=1.68, p=.11. But at high levels (+1 *SD*) of dACC activation, lower levels of executive functioning corresponded to greater aggression, β=-0.71, $t(34)$=-3.31, p<.005.

As with the dACC, anterior insula activation interacted with executive functioning to predict aggression, though only in the left hemisphere; left insula: β=0.39, t(30)=2.12, p<.05; right insula: β=0.32, t(30)=1.68, p=.10. At low levels (-1 *SD*) of executive functioning, left insula activation was marginally, positively associated with aggression, β=0.40, t(34)=2.00, p=.055, whereas at high levels (+1 *SD*) of executive functioning, left insula activation was unassociated with retaliatory aggression, β=-0.41, t(34)=-1.31, p>.2. Subsequent analyses examined the effect of executive functioning on aggression at low and high levels of left anterior insula activation. At low levels (-1 *SD*) of left anterior insula activation, executive functioning did not correspond with aggression, β=0.32, t(34)=1.21, p=.23. But at high levels (+1 *SD*) of left anterior insula activation, lower levels of executive functioning marginally corresponded to greater aggression, β=-0.49, t(34)=-2.03, p=.051.

Discussion

People often react aggressively when they are socially rejected. This experiment sought to understand why this effect occurs, and who is most likely to respond to social rejection with aggression. Utilizing fMRI, we replicated the finding that social rejection elicits activation of the dACC and anterior insula. We also offered a novel extension by showing that dACC and left anterior insula activation significantly interacted with executive functioning to predict aggression. Among individuals low in executive functioning, greater dACC and left anterior insula activation was associated with more aggression. In contrast, individuals high in executive functioning showed a negative association between dACC activation and aggression.

These findings are the first examination of the neural substrates of the rejection-aggression link. They support the GAM's predictions regarding affective processes underlying the relationship between rejection and aggression, along with how individual differences in the tendency toward impulsive and thoughtful actions influence aggression. Our results strongly suggest that social pain is a means through which rejection causes reactive aggression. Although there are other rejection-related mechanisms that facilitate the rejection-aggression link, such as hostile cognitions (DeWall et al., 2009), we have implicated pain-related neural activity as a potential contributing factor as well. This similarity provides further evidence for Pain Overlap Theory (Eisenberger & Lieberman, 2005). Building this diverse model of the neural and psychological underpinnings of the rejection-aggression link is a necessary step in understanding how to reduce aggression.

Despite the consistency of our results, the current experiment had limitations that may inform future research. First, the dACC was not significantly associated with social distress and the anterior insula was only marginally significantly associated with social distress. This was likely due to the fact that approximately 1 hour elapsed between our rejection manipulation and the post-scan assessment of social distress (due to various anatomical MRI scans and safe removal of the participant from the scanner), and self-reports of post-rejection social distress diminish after approximately 45 minutes (Zadro et al., 2006). Because of this, we cannot be certain that neural activity in these regions was indicative of underlying social pain. However, given prior research showing that these regions respond to social exclusion and tend to correlate with self-reported social distress in response to this particular task (Eisenberger, 2012), we think that the lack of

a significant relationship between self-reported social distress and neural activity is more likely to be due to the delay in the self-report assessment rather than due to a completely different psychological process taking place during the fMRI scan. Future work will be needed, however, to more carefully probe this issue. In addition, this lack of a correlation between self-reported social pain and dACC and anterior insula activation does not mean that participants were not still being affected by the rejection episode, as rejected individuals still show evidence of social pain on implicit behavioral measures well after an hour (Zadro et al., 2006).

A second limitation is that our aggression measure was direct. It is an open question as to whether our findings apply to displaced aggression, in which individuals harm innocent bystanders. Third, we relied on individual differences in executive functioning instead of experimentally manipulating them. Hence, we cannot make causal claims regarding the interactive effect of executive functioning and dACC and anterior insula activation in predicting aggression. Future research should assess whether depleting and strengthening participants' levels of executive functioning would increase and decrease the relationship between dACC and anterior insula activation and aggression, respectively.

Fourth, although dACC activity was observed in response to social rejection, activation of the dACC has also been associated with expectancy violations (e.g., Botvinick et al., 2004). Because our rejection manipulation inherently involved such a violation of participants' expectations (i.e., not receiving any ball-tosses in an ostensibly equitable ball-toss game), the dACC activation we observed during rejection may merely be due to this phenomenon and not representative of the distress associated

with social rejection. A recent study made such a possibility unlikely, as it showed that comparing the rejection condition in Cyberball to an 'over-inclusion' condition (i.e., an expectancy violation) still yields activation of the dACC (Kawamoto et al., 2012).

Fifth, participants' aggression towards their rejecter may have been affected by the lack of an expectation of future interaction with that individual. Had participants expected to interact with their partner again, individuals high in executive functioning may have aggressed in a similar fashion to their low executive functioning counterparts. Translating their social pain into aggression, individuals both high and low in executive functioning could stand to gain from retaliating against their provocateur under such conditions. Sixth, because rejection always occurred later in time than acceptance, our fMRI contrast between acceptance and rejection conditions was confounded with the inevitable changes in the MRI signal that occur over the length of a scan. To reduce the impact of this confound we utilized several widely-accepted preprocessing strategies. Specifically, our data were highpass filtered to remove low frequency shifts in the data over time, prewhitened to remove temporal autocorrelation, and a temporal derivative was included in the statistical model to account for time-based shifts in the hemodynamic response function (see Poldrack, Mumford, & Nichols, 2011). Finally, because we only measured a single outcome variable, aggression, we cannot be sure that our interactive effect of social pain and executive functioning is specific to aggression or extends to other variables.

Notwithstanding these limitations, our findings have substantial implications for theory and practice. To our knowledge, this experiment is the first to demonstrate the neural underpinnings of the rejection-aggression link. Furthermore, our findings are the

first to show that social pain-related neural activity is associated with increased aggression under certain conditions. If high levels of executive functioning can reverse the rejection-aggression link, researchers may design interventions aimed at bolstering executive functioning among socially rejected people. Although global violence appears to be on the decline, such interventions may help to reduce it further.

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