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Does the Pain of Rejection Promote the Pleasure of Revenge? A Neural Investigation of Cingulo-Striatal Contributions to Violence

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DOES THE PAIN OF REJECTION PROMOTE THE PLEASURE OF REVENGE?
A NEURAL INVESTIGATION OF CINGULO-STRIATAL CONTRIBUTIONS TO
VIOLENCE

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DISSERTATION
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A dissertation submitted in partial fulfillment of the requirements for the degree of
Doctor of Philosophy in the College of Arts and Sciences at the University of
Kentucky

By
David Skylan Chester

Lexington, Kentucky.

Director: Dr. C. Nathan DeWall, Professor of Psychology

Lexington, Kentucky

2016

ABSTRACT OF DISSERTATION

DOES THE PAIN OF REJECTION PROMOTE THE PLEASURE OF REVENGE?
A NEURAL INVESTIGATION OF CINGULO-STRIATAL CONTRIBUTIONS TO
VIOLENCE

Aggression is a dynamic and costly feature of human behavior. One reliable cause of aggression is social rejection, though the underlying mechanisms of this effect remain to be fully understood. Previous research has identified two psychological processes that are independently linked to aggressive retaliation: pain and pleasure. Given recent findings that pain magnifies the experience of pleasure, I predicted that the pain of rejection would promote the pleasure of aggression and thus, aggression itself. I also expected that this indirect effect of aggressive pleasure would only be observed among individuals with weaker self-regulatory abilities that are necessary to cope with rejection's sting. To test these hypotheses, I performed a functional neuroimaging experiment in which I acquired neural signatures of social pain and aggressive pleasure, as well as behavioral measures of aggression itself and self-regulatory abilities. Using a moderated-mediation approach, I observed that, among individuals high in self-regulatory abilities, neural signatures of social pain predicted *less* aggressive retaliation in response to social rejection. This effect was mediated by reduced activity in the brain's reward network during retaliatory aggression. Among individuals low in self-regulatory abilities, no such effects on aggression were observed. These findings suggest that social pain can buffer individuals against aggressive behavior, but only when people have the self-regulatory ability to do so. Much of human action is motivated by pain and pleasure, understanding their roles in aggression is a critical step in eliminating such violence.

KEYWORDS: Aggression, Social Rejection, Social Pain, Pleasure, fMRI

David Skylan Chester_____

April 25th, 2016_____
Date

DOES THE PAIN OF REJECTION PROMOTE THE PLEASURE OF REVENGE?
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VIOLENCE

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DEDICATION

This dissertation is dedicated to my mother, Nora L. Thompson, for giving me more support than I could have imagined, for encouraging me to chase my dreams, and for raising me to be curious.

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Thanks to my advisor, C. Nathan DeWall, for seeing something in me and guiding me along my career and shaping me as a scientist. I am grateful to my dissertation committee members, Suzanne Segerstrom, Will Gervais, Brian Gold, and Adam Craig. Your questions, suggestions, and feedback greatly improved this project and suggested avenues for future inquiry. I would like to thank the entire Department of Psychology at UK, from the chair, Robert Lorch, to the administrative staff. Our department is truly a special place and I am grateful to have been a part of it. The Social Psychology Area has been a wonderful place for me to grow as a scholar and mentor, and for that I thank Richard Smith, Sung Hee Kim, Christia Brown, Jonathan Golding, Will Gervais, and Jazmin Brown-Ianuzzi. Thanks to my friends at the magnet, Dave, Anders, and Nate, for patiently watching and helping a psychologist try and learn neuroscience, and for answering many an emergency call on a Sunday morning. I am particularly thankful for the mentorship and support of Rich Milich. I will miss our chats and your insight into what makes a good psychologist and scientist. Thanks also to Brad Bushman, who started me on the path towards trying to understand why people hurt each other. I am grateful to Jennifer Beer, Brent Hughes, Jamil Bhanji, and Gili Freedman, for teaching me about how to look at brains and for being great friends and mentors.

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Chapter One: Introduction

Nature has placed mankind under the governance of two sovereign masters, pain and pleasure.

-Jeremy Bentham

Despite millennia of cultural and technological advancement, humans are no exception. Every year, millions of people lose their lives or are injured due to human-perpetrated violence. This incalculable cost mandates that the scientific community investigates the causes of aggressive behavior and avenues through which to reduce it.

Almost a century of research on aggression has made significant strides in understanding the situations and traits that cause aggression (Anderson & Bushman, 2002). Inputs from both the situation and the person interact to influence individuals in many ways that promote and constrain aggression. The current investigation sought to expand our knowledge of a well-established cause of aggression, social rejection (Twenge, Baumeister, Tice, & Stucke, 2001). Although environmental inputs such as rejection can amplify aggression, person inputs such as self-control can restrain these violent impulses (DeWall, Finkel, & Denson, 2011). In this investigation, I tested whether the interactive effect of rejection and self-regulation on aggressive behavior can be explained, in part, by two of human life's most essential experiences: pain and pleasure.

1.1 Aggression: An Overview

Aggression is a diverse phenomenon with many manifestations and definitions and thus it is crucial to clarify what aggression is and is not. According to Bushman & Huesmann (2010), aggression is *any behavior that is intended to harm someone who does not wish to be harmed*. First and foremost, aggression is a behavior, not an emotional or cognitive state. Secondly, aggression is a social phenomenon, meaning that it can only occur between two or more individuals. Third, aggressive behavior's goals need not be realized (e.g., the clenched fist strikes home) to be considered as aggression. An attempt at the harm is all that is necessary. The final feature of aggression, harm, is what makes it so diverse, as one can harm others in many ways.

Physical aggression is the prototype in which one seeks to harm another by damaging their body. Most aggression research has focused on this form of aggression, and it is also the focus of this investigation. Aggression can also be retaliatory or unprovoked, in which the former is in response to some provocation (e.g., insult, threat) and the latter is performed with no prior provocation. Retaliatory aggression is the most frequent form of aggression and was therefore the focus of this investigation (Anderson & Bushman, 2002). Such retaliatory aggression is often characterized as reactive and 'hot' and juxtaposed against instrumental or 'cold' forms of aggression such as those performed by contracted aggressors such as soldiers. Additionally, aggression can be direct or indirect (a.k.a. displaced), in which the target of the former type of aggression is the source of provocation and in the latter type, the target is someone other than the

provocateur (Miller, Pedersen, Earleywine, & Pollock, 2003). The direct form of aggression is far more common (Anderson & Bushman, 2002) and will thus be my focus. Finally, the term 'violence' refers to extreme aggression that seeks to cause a substantial amount of harm, which is largely impossible to measure in a scientific setting. Therefore, I trained my sights on sub-violent aggression. In sum, the form of aggression adopted in this investigation was physical, retaliatory, reactive, 'hot', direct, and sub-violent. This form of aggression was chosen due to its observed frequency in the real world and logistical and ethical laboratory constraints. The now-evident complexity of aggression extends to theoretical models that seek to explain it.

1.2 The General Aggression Model

Many theories have arisen in an attempt to parsimoniously unify the vast body of findings regarding aggressive behavior. Synthesizing and extending these extant theories, a General Aggression Model (GAM) was proposed by Anderson and Bushman (2002; Figure 1) and has received immense support subsequently (e.g., DeWall, Anderson, & Bushman, 2011). The GAM outlines a temporal sequence of features that explain whether an individual will act aggressively or not. According to this process model, person and situation factors act as inputs to a given individual. These inputs then alter the individual's internal state (affect, cognition, arousal), which serves as the bottom-up motivation for aggressive acts. This motivational state then feeds into higher-order, executive functions that determine whether an individual's subsequent behavior is impulsive or thoughtful. According to the GAM, aggression can arise from either

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thoughtful (e.g., self-defense) or impulsive (e.g., drunken brawl) pathways. These

acts then feed back into the situation and person, potentially forming an

aggression-exacerbating cyclical loop.

Figure 1. The General Aggression Model (Anderson & Bushman, 2002).
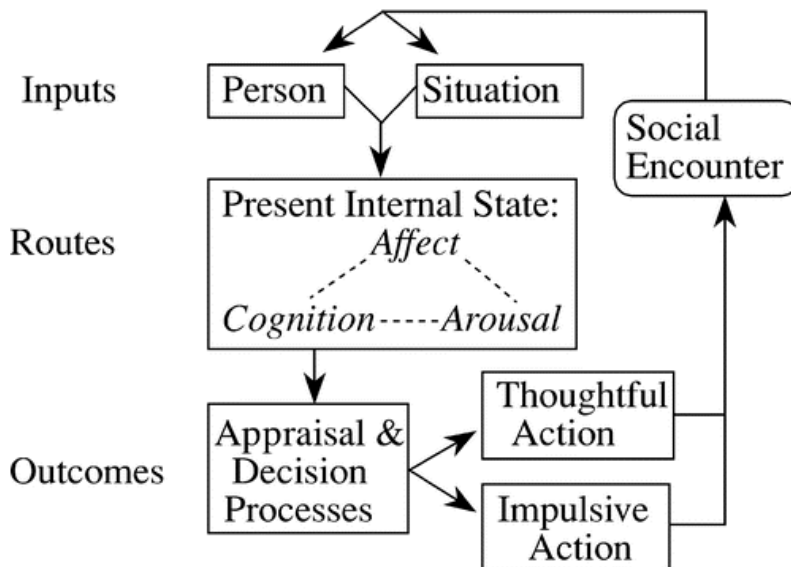
The generality of this model stems from the broad nature of the constructs

invoked. Indeed, the critical role of 'person' and 'situation' inputs into behavior

date back to Kurt Lewin's famous formulation that behavior is a function of the

person and the situation. Although the theoretical bulwark of the GAM may

appear to be imprecise given the broad nature of the constructs included therein

(e.g., personality), it is specific enough to develop targeted hypotheses such as

those tested in this investigation. From this model, I developed a set of

hypotheses to investigate one of the most reliable causes of aggression, social

rejection.

1.3 The Rejection-Aggression Link

Social connections are a deep wellspring of health, happiness, and purpose. Reflecting their importance, people across the world possess an elemental need to create and maintain these sources of acceptance (Baumeister & Leary, 1995). However, people are not always able to meet this need. Often, our attempts to connect with others are met with rejection. People respond to such rejection with a wide gamut of behaviors. Sometimes, rejected individuals lash out at the world around them. Rejection causes individuals to aggress towards both their rejecters and innocent bystanders (Buckley, Winkel, & Leary, 2004; Leary, Twenge, & Quinlivan, 2006; Twenge et al., 2001). The rejection-aggression link is highly consequential because social rejection has been identified as a common element across highly violent individuals such as school shooters (Leary, Kowalski, Smith, & Phillips, 2003).

Yet why would social rejection increase aggression? It seems odd that a person might react to social rejection with a behavior that is almost guaranteed to increase the likelihood of further ostracism. Early investigations into the underlying factors behind the rejection-aggression link identified factors that have been conventionally linked to aggression: anger (Chow, Tiedens, & Govan, 2008), hostility (DeWall, Twenge, Gitter, & Baumeister, 2009; Reijntjes et al., 2011), and the need for control (Warburton, Williams, & Cairns, 2006; Wesselmann, Butler, Williams, & Pickett, 2010). Taken together, it appears that the rejection-aggression link is largely due to a self-regulation failure in which affective and motivational states win out over more rational forces. Another proximate, psychological process that is typically linked to aggression is physical

pain (Berkowitz, 1989). The ability of physical pain to increase aggression might give us a clue as to a fourth possible mechanism of the rejection-aggression link: social pain.

1.4 Social Pain

Rejection is painful, and the aversive, emotional, and visceral experience of rejection is referred to as *social pain* (Eisenberger, 2012, 2015). The idea that rejection might be actually painful was born during Jaak Panksepp's early research on bird chicks, demonstrating that stimulating or blunting the chick's physical pain system exacerbated or diminished their distress at being separated from their mothers (Panksepp, 2011; Panksepp, Vilberg, Bean, Coy, & Kastin, 1978). It appeared that alterations in physical pain affected social functioning.

This overlap between the physical and social domains was extended to humans by a seminal functional neuroimaging experiment, which demonstrated that social rejection was associated with activity in the human brain's pain matrix (Eisenberger, Lieberman, & Williams, 2003). This finding launched a wave of research into how pain and the brain systems that subserve it may lie at the heart of far more experiences than physical injury. Yet why would a social experience activate the physical pain circuitry in the brain?

Subsequent theorizing couched the social pain results in an evolutionary framework, suggesting that social isolation in our human ancestry was so threatening (given the eusocial nature of our species) that the body co-opted the physical pain network to respond to such an event (MacDonald & Leary, 2005). According to Pain Overlap Theory (Eisenberger & Lieberman, 2004), social pain

was an adaptation that reflected our species' profoundly social nature and was

evident in other socially-dependent species. Pain is the ideal psychological entity

for this task as it is exceptionally capable of alerting us to threats. The exaptation

of the pain system to respond to rejection makes further sense given the

emphasis that natural selection makes on economy. There is no need to evolve a

new, incredibly costly brain network when one can simply take an extant system

and modify it to respond to a wider range of stimuli. Given this broad role in

responding to physical and social threats, the brain's pain matrix has become

considerably complex.

1.4.1 The brain's pain matrix. The neural literature on physical pain

reliably shows activity across a network of brain regions in response to noxious

stimuli (Eisenberger, 2012). The brain's pain network is anatomically dissociable

into two separate sub-networks: an affective network that generates the distress

of pain, and a somatosensory network that tracks the visceral and tactile

sensations associated with injury (Figure 2). The dissociability of these sub-

networks has been demonstrated by lesion studies in which surgically disabling

the affective network leads to reports that patients can feel a painful stimulus but

that it does not distress them (Foltz & White, 1962).

The affective pain matrix consists of the dorsal anterior cingulate cortex

(dACC) and anterior insula, whereas the somatic pain matrix consists of the

somatosensory cortex and the posterior insula (Eisenberger, 2012). Subcortical

structures such as the dorsomedial thalamus and periaqueductal gray have been

implicated in pain but their small size and subsequent difficulty to capture using neuroimaging techniques will exclude them from this dissertation.

Figure 2. Affective (red) and somatic (green) pain networks (Eisenberger, 2012). Reprinted with permission from Nature Publishing Group.
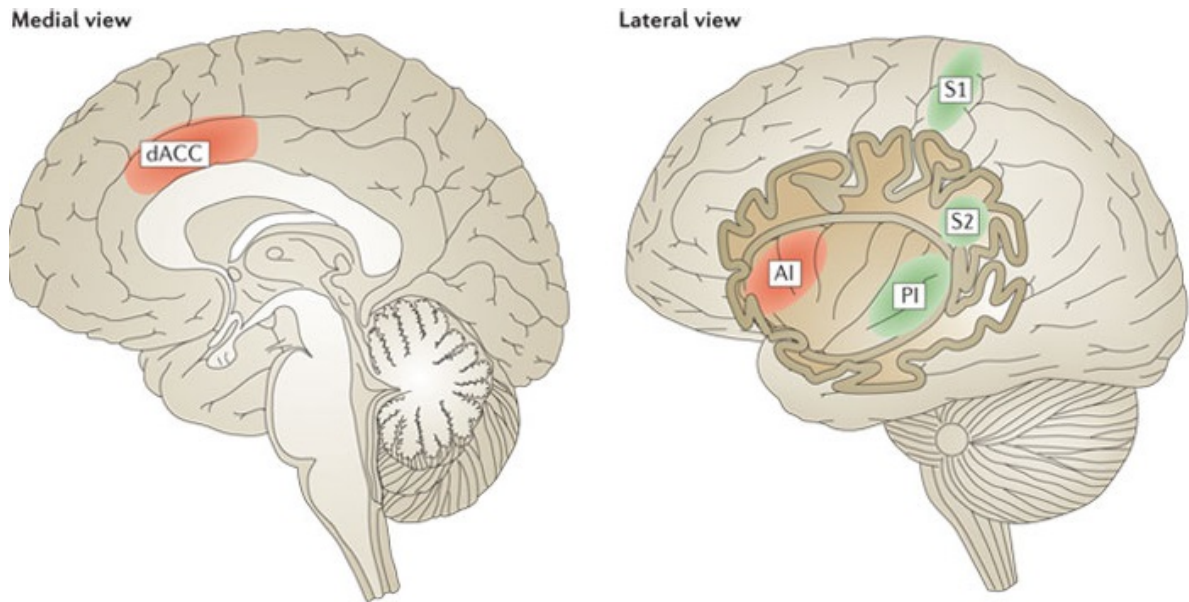
Social rejection has been associated with activity in the somatic pain matrix (Kross, Berman, Mischel, Smith, & Wager, 2011), but is far more reliably associated with activity in the affective pain matrix, particularly the dACC (Chester et al., 2014; DeWall et al., 2010; Eisenberger et al., 2003; Kawamoto et al., 2012; for meta-analytic evidence see Eisenberger, 2015; Rotge et al., 2014). Therefore it appears that social pain is characterized by the aversive, affective, distressing aspect of pain because of its neuroanatomical seats in the dACC and anterior insula. Reactivity in these regions during social rejection has substantial implications for human responses to such ostracism.

1.5 Social Pain and Aggression.

As noted earlier, physical pain is a reliable cause of aggression (Berkowitz, 1989). This phenomenon is explained by primitive reflexes ingrained in our bodies that respond to somatic harm with aggressive and escape behaviors that serve to destroy and avoid the source of a given injury, respectively (Berkowitz, 1989). According to Pain Overlap Theory (Eisenberger & Lieberman, 2004), if physical pain increases aggression, then so should social pain due to their anatomical and functional overlap. As such, I tested the hypothesis that social pain activity in the brain would be associated with greater aggression. Drawing from the General Aggression Model (Anderson & Bushman, 2002), I qualified my prediction based on the critical role that self-regulatory processes play in determining whether an aggressive impulse will actually result in aggression. I predicted that social pain would only be associated among individuals who possessed relatively weak self-regulatory abilities. Supporting this hypothesis, activity in the dACC and anterior insula during rejection has been associated with greater aggressive retaliation, though not for everyone (Chester et al., 2014). Indeed, whether activity in the brain's affective pain matrix during rejection predicts retaliatory aggression is determined by an individual's level of executive functioning.

1.5.1 The moderating role of executive functioning. According to the GAM, whether an affective state (e.g., social pain) results in aggressive behavior is contingent upon whether the present internal state can be effectively regulated by *executive functioning* (referred to as 'appraisal and decision processes'; Anderson & Bushman, 2002). Definitions of executive functioning differ greatly,

though some consensus in the self-regulation literature has been obtained, coalescing around three key processes: inhibition of prepotent responses, task-switching, and working memory (Hofmann, Schmeichel, & Baddeley, 2012). These three executive functions constitute the raw psychic materials from which effective self-regulation is achieved.

In line with this model, I provided some correlational evidence that social pain is only linked to greater aggression among individuals low in executive functioning and thus less able to regulate the pain of rejection (Chester et al., 2014). Conversely, individuals high in executive functioning exhibited a negative association between social pain and aggression, likely due to the effective recruitment of inhibitory resources. Providing converging evidence, brain stimulation techniques that artificially increased activity in brain regions that regulate social pain also reduced aggressive responses to rejection (Riva, Romero-Lauro, DeWall, Chester, & Bushman, 2015). Taken together, these findings suggest that the pain of rejection promotes aggressive behavior (though only among individuals who cannot regulate the pain). However, pain is intricately and dynamically linked with another psychological process, pleasure. Recent research suggests that this may be a mechanism underlying the rejection-aggression link as well.

1.6 Sweet Revenge: The Pleasure of Retaliatory Aggression

Conventionally, the psychological literature points to negative affect as the cause of aggression (e.g., anger, frustration, pain: Berkowitz, 1989). However, the role of positively-valenced experiences and their ability to motivate violence

are a growing trend in the aggression literature. Freudian conceptions of cathartic aggression assert that violent behavior can serve to increase positive affect and serve to increase aggressive tendencies, though this notion is likely false (Bushman, 2002; Bushman, Baumeister, & Stack, 1999). Indeed, the motivation to improve mood via aggression plays a crucial role in motivating retaliatory aggression (Bushman, Baumeister, & Phillips, 2001; Chester & DeWall, 2016). Until recently, it remained unknown whether these perceived hedonic benefits were actually realized. Recent studies have shown that retaliatory, though not displaced, aggression is rated as pleasant (Carré, Gilchrist, Morrissey, & McCormick, 2010; Ramírez, Bonniot-Cabanac, & Cabanc, 2005). Among individuals who were currently-experiencing negative affect, perpetration of retaliatory aggression appeared to temporarily repair their mood (Chester & DeWall, 2016). Thus, aggression appears to be rewarding and individuals might employ this positive affect to attempt to regulate their emotions.

Neuroimaging research has further underscored the ability of retaliatory aggression to be associated with positive affect. Using electroencephalography, retaliatory aggression has been associated with greater activity in the left hemisphere of the prefrontal cortex than the right hemisphere, a neural signature of approach and reward (Harmon-Jones & Sigelman, 2001). Using fMRI, I provided some correlational evidence that retaliatory aggression is also associated with neural activity in the nucleus accumbens (NAcc; Figure 3), a brain region critical to the experience of reward, pleasure, and positive affect (Chester & DeWall, in press[a]).

Figure 3. The nucleus accumbens (orange; Chester & DeWall, in press[a]).
Reprinted with permission from Oxford University Press.
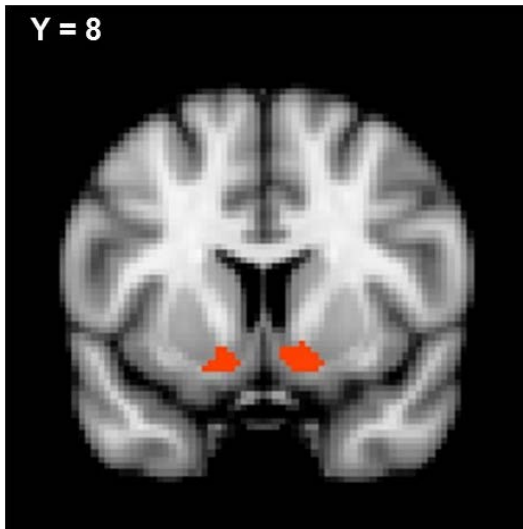
The NAcc is part of a larger dopaminergic network of brain regions that include dorsal striatal regions such as the caudate nucleus and the ventromedial prefrontal cortex (VMPFC; Berridge & Kringelbach, 2013). Despite the presence of this larger reward network, there is reason to focus on the NAcc when attempting to identify the neural substrate of positive affect and reward. For example, the caudate's link to reward and pleasure is tenuous as this region can be more accurately construed as "the neural implementation of the law of effect, responsible for [stimulus-response] learning reinforced by rewards" (Yin & Knowlton, 2006, pg. 467). Thus, the caudate is less a 'reward' region than a mechanism through which the associations between reward and various stimuli are instantiated. The VMPFC, though possessing direct anatomical connections to the NAcc, plays less of a role in the core affective experience of pleasure and serves more as an integrative hub that plays a critical role in value computations and subsequently, decision-making (e.g., do I pick the tempting cake or the

healthier apple? Damasio, Everitt, & Bishop, 1996). For these reasons, it was important to focus on the NAcc when speaking about reward and not its broader neural network.

Aside from its neurological counterparts in the dopaminergic network, the NAcc is also dynamically tied to regulatory regions of the frontoparietal regulatory network that serves as the seat of executive functioning in the brain (Heatherton & Wagner, 2011). Implicating the crucial influence of self-regulatory processes, retaliatory aggression was negatively associated with the extent to which the NAcc was regulated by an inhibitory region of the ventrolateral prefrontal cortex (VLPFC; Chester & DeWall, in press[a]). Thus, it appears that the NAcc's generation of aggressive impulses might be readily constrained by self-regulation. Aggressive retaliation is thus likely to be a pleasant experience (an experience I call *aggressive pleasure)* only when positive affect is not effectively regulated. This moderating role of self-regulation echoes the interaction between social pain and executive functioning that was summarized earlier in this manuscript. Yet how do social pain and aggressive pleasure interact to possibly explain the rejection-aggression link?

1.7 Pain Magnifies Pleasure

Pain and pleasure interact to a great extent. Due to the co-activation inherent in such opponent processes (Leknes, Brooks, Wiech, & Tracey, 2008), one experience sensitizes the individual to the other, such as when pleasure enhances subsequent pain. For example, opioid abuse leads to subsequent hyperalgesia (Silverman, 2009). Inversely, and most importantly for this project,

pain sensitizes the mind to subsequent experiences of pleasure (Bastian, Jetten, Hornsey, & Leknes, 2014). Simply removing a painful stimulus is experienced not just as an absence of pain but also as the presence of pleasure (Leknes, Lee, Berna, Andersson, & Tracey, 2011). Pain offset is further associated with dopaminergic (Leknes et al., 2013) and opioid (Sprenger et al., 2006) activity in the NAcc, which both serve to mitigate pain *and* enhance pleasure (Leknes & Tracey, 2008). Interestingly, this ability of pain to facilitate subsequent pleasure may motivate individuals to experience pain in the first place, behaviors that range from maladaptive (self-harm) to adaptive (exercise; Fields, 2007). Thus, pain magnifies subsequent pleasure. Couched within the literature of the rejection-aggression link, it is likely that social pain enhances aggressive pleasure which, in turn, motivates aggressive behavior. Further, these relationships are likely contingent on an individual's ability to regulate such experiences of pain and pleasure.

1.8 Study Overview

This investigation tested the hypothesis that social pain's effect on greater aggressive responses to rejection would be mediated by greater aggressive pleasure. To measure social pain, I imaged neural activity in the dACC and anterior insula during social rejection. To measure aggressive pleasure, I imaged neural activity in the NAcc during retaliatory. To measure aggression, I used a validated aggression task, the Taylor Aggression Paradigm.

Additionally, I tested the hypothesis that social pain's exacerbating effects on aggression and aggressive pleasure would be constrained by executive

functioning levels as measured by the well-validated Color-Naming Stroop Task. The use of neural and behavioral measures, instead of traditional self-report measures, was adopted in order to bypass many of the biases inherent in self-reflection and social desirability.

Chapter Two: Method

To test the hypotheses described above, I conducted a functional magnetic resonance imaging (fMRI) study in which brain activity was measured during instances of social rejection and subsequent retaliatory aggression. To do so, I combined two well-validated tasks that I have previously and effectively used in a neuroimaging context to both elicit social pain, aggressive pleasure, and retaliatory aggression. I also measured participants' dispositional executive functioning using a well-validated behavioral task that I have previously used to moderate the effects of social pain on aggressive behavior.

2.1 Participants

Participants consisted of 60 healthy undergraduates and general community members (38 females; Age: range = 18 – 30, M = 20.28, SD = 2.77). I recruited participants from either the University of Kentucky's Introductory Psychology Subject Pool or the general community. I compensated participants with credit towards their research requirement (students) or $50 (community members). All participants also received a compact disk that contained a selection of their brain images. At the beginning of each semester, Subject Pool students completed a short screening survey that included several measures in order to identify qualified individuals. Through the community website Craigslist, I provided interested general community members with a link to the same short screening survey. Individuals were excluded from the study if they failed to meet criteria necessary for comfort and safety in the MRI environment, as well as data

quality concerns (Table 1). I emailed all qualified respondents and asked them to

sign-up for an appointment to complete our study.

Table 1. Exclusion criteria for potential participants.

Exclusionary criterion	Reason
Body Mass Index > 30	Impairs comfort and safety in the MRI scanner.
Claustrophobia	Impairs comfort and safety in the MRI scanner.
Color blindness	Alters the efficacy of colorized stimuli.
Mental/Neurological pathology	Impairs comfort and safety in the MRI scanner and data quality.
Metallic objects in body	Impairs comfort and safety in the MRI scanner and data quality.
Non-right-hand-dominance	Impairs data quality due to hemispheric alaterality.
Prior head trauma.	Impairs data quality.
Psychoactive medication use	Impairs data quality.

2.2 Materials

    2.2.1 Rejection task (Cyberball). To induce feelings of social pain in the

functional neuroimaging environment, I employed the Cyberball social rejection

task (Chester et al., 2014; Eisenberger et al., 2003; Williams, Cheung, & Choi,

2000). In this task, participants were instructed to play a virtual ball-tossing game

with two fictitious partners. The ostensible purpose of the task was for

participants to mentally visualize the task as if it were occurring in real life, so that

we might understand the neural underpinnings of the human imagination. The task proceeded across three blocks. In the first two blocks, participants received an equal number of ball-tosses from their two partners for 60 seconds per block (Acceptance condition). However, in the third block, after 30-seconds, participants stopped receiving the ball from their partners who continuously threw it back-and-forth to one another for 50-seconds (Rejection condition). Baseline activation was captured by 10-second 'Rest' trials that preceded each of the three blocks.

2.2.2 Retaliatory aggression task (Taylor Aggression Paradigm). To measure the neural correlates of retaliatory aggression, I employed the Taylor Aggression Paradigm (Figure 4; Chester & DeWall, in press[a]; Giancola & Chermack, 1998; Krämer, Jansma, Tempelmann, & Münte, 2007; Taylor, 1967). In this classic aggression measure, 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, with each block containing 6 trials. Each block began with a fixation cross that modeled baseline neural activity. Then, participants completed an aggression trial in which they set the volume of their

partner's noise blast. A blank screen then appeared for a jittered duration, which then gave way to a competition trial 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 aggression trial was preceded by their opponent setting a loud (3, 4) or soft (1, 2) volume level determined whether the given trial was retaliatory (after a loud blast) or non-retaliatory (after a soft blast). Such retaliatory and non-retaliatory trials were split evenly (7 of each) and randomly presented with the exception of the first trial which is always non-retaliatory. Wins and losses are also randomized and split evenly (7 wins, 7 losses).

Figure 4. Temporal schematic of the Taylor Aggression Paradigm (Chester & DeWall, in press[a]). Reprinted with permission from Oxford University Press.
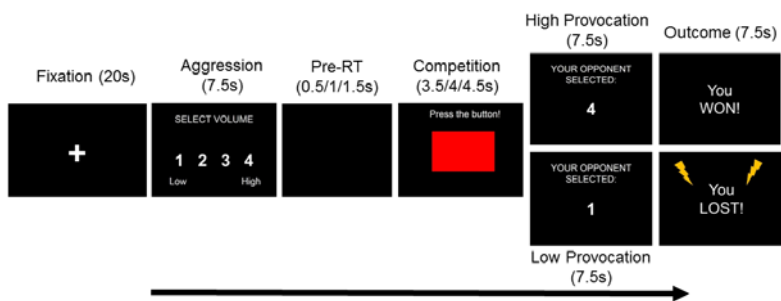
2.2.3 Self report questionnaires. In order to provide some evidentiary support for the reverse inferences that dACC activity is a neural signature of social pain and that NAcc activity is a neural signature of aggressive pleasure, I

administered four self-report measures of both social pain and aggressive

pleasure (two measures per construct).

      2.2.3.1 McGill pain questionnaire – short form. To measure

participants' experience of social pain due to social rejection, participants

completed a well-validated measure of both sensory and affective pain, the

McGill Pain Questionnaire (Melzack, 1987; see Appendix A). Previous research

on social rejection has shown that social rejection increases reports of the

affective subscale of this measure, and have effectively used it as an index of

social pain (e.g., Chen, Poon, Bernstein, & Teng, 2014; Chester, DeWall, &

Pond, in press). This measure included 15 adjectives regarding the subjective

intensity of one's current pain levels, 0 (none) to 3 (severe). Eleven items

measured sensory aspects of pain (e.g., shooting, stabbing) and four items

measured affective components (e.g., fearful, punishing-cruel). Because social

pain reflects the affective component of pain (Eisenberger, 2012), the affective

subscale of the MPQ was deemed an appropriate measure of social pain.

      2.2.3.2 Need threat scale. The 30-item Need Threat Scale served as a

manipulation check of the Cyberball paradigm's ability to elicit social pain

(Williams, 2009; see Appendix B). Aside from a two-item measure of the

subjective experience of rejection (e.g., I was excluded), the Need Threat Scale

also contained four, five-item subscales that assess the extent to which rejection

threatened the human needs for belonging (e.g., I felt like an outsider), self-

esteem (e.g., I felt liked), control (I felt I had control over the game), and

meaningful existence (I felt meaningless), and two, four-item subscales that

measures negative (e.g., angry) and positive affect (e.g., happy). Each item

asked participants to reflect back on their experience during Cyberball.

Participants responded to each item along a 5-point Likert-type scale with higher

values representing greater degrees of each subscale's latent construct.

   2.2.3.3 Aggressive pleasure scale. To assess the pleasure of

aggression, participants completed the four-item subscale of the need threat

scale that measured positive affect (Williams, 2009; see Appendix C). However,

the items had alternate instructions that asked participants to respond as to how

they felt *when they were selecting the level of noise blasts to administer to their*

partner.

   2.2.3.4 Short sadistic impulse scale. The SSIS is a brief version of the

Sadistic Attitudes and Behaviors Scale (O'Meara et al., 2011) that I used to

measure the reward derived from aggression (see Appendix D). This single-

factor, 10-item scale contained items such as 'I enjoy seeing people hurt' and

'hurting people would be exciting' that participants responded to along a 1

(disagree) to 7 (agree) Likert-type scale.

   2.2.4 Executive functioning task (Stroop). I employed one of the most

widely-used measures of executive functioning, the color-naming Stroop Task

(Engle, 2002; Stroop, 1935). In this task, participants were instructed to press a

key if a word on the screen was presented in green font and to press a different

key if the word was presented in red font. On congruent trials, the meaning of the

word matched the ink-color (e.g., the word RED displayed in red color). On

incongruent trials, the meaning of the word did not match the ink-color (e.g., the

word GREEN displayed in red ink). The task recorded participants' accuracy on each trial as well as their response time. The ability to quickly and accurately complete incongruent trials served as the measure of executive functioning, as the ability to do so reflected higher amounts of executive functioning's three major facets: inhibition of prepotent responses (not responding to the word's meaning), task-switching (flexibly shifting between congruent and incongruent trials), and working memory (maintaining task instructions in short-term memory; Hofmann et al., 2012). The task consisted of 10 congruent and 10 incongruent trials, randomly ordered.

As mentioned earlier in the manuscript, executive functioning consists of several components, including task-switching, working memory, and the inhibition of prepotent responses (Hofmann et al., 2012). It is difficult to disentangle these various components when measuring executive functioning, though doing so would allow for far more specificity in the role that self-regulatory processes play in modulating aggressive behavior. In an attempt to disentangle these components, we also employed a version of the Stroop Task in which congruent and incongruent trials were no longer randomized but segregated into two blocks of 10 congruent trials and then 10 incongruent trials. Modifying the task in this manner allowed us to eliminate the task-switching component of executive functioning and constrain inferences to working memory and inhibition.

2.2.5 Exploratory measures. Two measures were included in the battery of questionnaires to measure the extent that (A) my model predicted variance in

trait levels of aggression and (B) trait self-control functioned as a moderator in place of Stroop task performance.

      2.2.5.1 Trait aggression. To measure trait aggression, which is the dispositional tendency to exhibit anger, hostility, physical and verbal aggression, I employed the Brief Aggression Questionnaire (BAQ; Webster et al., 2014, see Appendix E). The BAQ contains twelve items that comprise four factors: anger (I have trouble controlling my temper), hostility (I sometimes feel that people are laughing at me behind my back), physical aggression (Given enough provocation, I may hit another person), and verbal aggression (My friends say that I'm somewhat argumentative). Participants responded to each item along a 1 (disagree) to 7 (agree) Likert-type scale.

      2.2.5.2 Trait self-control. To measure trait self-control, which is the dispositional tendency to restrain one's impulses to accomplish higher-order goals, I employed the Brief Self-Control Scale (BSCS; Tangney, Baumeister, & Boone, 2004; see Appendix F). This single-factor, 13-item scale contained items such as 'I wish I had more self-discipline' and 'I have a hard time breaking bad habits' that participants responded to along a 1 (not at all like me) to 5 (very much like me) Likert-type scale.

2.3 Procedure

      Participants arrived at the University of Kentucky's Magnetic Resonance Imaging and Spectroscopy Center where an undergraduate research assistant escorted them into our testing facilities. Upon obtaining informed consent, participants were given an overview of the study and then screened for safety in

the MRI environment via a questionnaire and structured interview. Participants then had a vitamin E pill attached above their right orbit for localization purposes. Participants then practiced the Cyberball and Aggression tasks outside of the scanner so that they were prepared once they encountered the tasks in the scanning environment. Participants were then led to the scanner for a final safety screening that included a metal detector test. Successfully screened participants were then led into the scanning room and introduced to the various equipment and procedures.

Once participants were secured in the scanner, I administered a high-resolution, structural scan. Once this scan was complete, participants completed the Cyberball and Aggression tasks in that order while fMRI was acquired across their entire brains. For approximately 35 of the participants, they first completed an inhibitory task and a decision-making task in the MRI scanner, prior to Cyberball (comparing whole-brain activity patterns during Cyberball revealed no significant differences between participants who had previous completed the two unrelated scanner tasks and those who did not). All participants completed a short, cue reactivity task in the MRI scanner where they viewed pleasant, neutral, and alcohol images after the Aggression task. These other three tasks were included to test other, unrelated hypotheses about impulsivity and substance abuse.

When the MRI scans were completed, participants were removed from the scanner. In a separate room, participants completed the manipulation check, the four self-report measures of social pain and aggressive pleasure, and both

versions of the Stroop Task. Finally, participants were administered a structured suspicion probe (e.g., what do you think this study was about?) and debriefed as to the actual purposes of the study and all elements of deception.

2.4 MRI Data Acquisition and Processing

All MRI data were obtained using a 3.0-T Siemens Magnetom Trio scanner at the University of Kentucky's Magnetic Resonance Imaging and Spectroscopy Center. 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 (120-second cutoff). These processed brain volumes were then fed into subsequent data analyses.

2.5 Analytic Strategy

Data analysis proceeded in several steps. First, fMRI analyses were conducted to assess patterns of brain activity during the Cyberball and Aggression tasks. Second, estimates of brain activity from clusters that showed task-related activation were extracted to create values that indicated the extent to which participants (A) activated the dACC and anterior insula during rejection (as compared to acceptance) and (B) activated the NAcc during retaliatory aggression (as compared to non-retaliatory aggression). These variables, alongside executive functioning scores from the Stroop task were mean-centered and then entered into a bootstrapped moderated mediation model to predict retaliatory aggression scores from the Taylor Aggression Paradigm. To produce the brain activity estimates, several steps were followed (described below).

2.5.1 MRI analyses. Preprocessed fMRI data from the Cyberball and Aggression tasks were separately analyzed using a two-level general linear model for each task.

2.5.1.1 First level (within-subjects). Each participant's BOLD signal was modeled at each voxel across the entire brain with a fixed-effects analysis that modeled trials as events using a canonical double-gamma hemodynamic response function with a temporal derivative. For the Cyberball task, the GLM included acceptance and rejection blocks as regressors while leaving fixation trials un-modeled. For the Aggression task, retaliatory aggression, non-retaliatory

aggression, competition, pre-competition, opponent's volume settings, and

outcome trials were included as regressors in the GLM while leaving fixation

trials un-modeled. All six motion parameters were modeled as nuisance

regressors for each GLM.

For the Cyberball task, linear contrasts compared rejection to acceptance

(Rejection > Acceptance blocks). For the Aggression Task, linear contrasts

compared retaliatory to non-retaliatory aggression (Retaliatory Aggression >

Non-Retaliatory Aggression trials). 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 2x2x2mm^3).

2.5.1.2 Second level (across subjects). 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, in percent signal change units that were averaged

across all voxels of the region-of-interest, were extracted separately from the

dACC (for the Cyberball task) and the bilateral NAcc (for the Aggression task),

yielding a social pain and aggressive pleasure value for each participant. The

dACC ROI mask was acquired from one of my previous neuroimaging studies on social pain (Chester, Pond, & DeWall, 2015), using a rostral boundary of y = 33 and a caudal boundary of y = 0 (in MNI space), with a ventral cutoff corresponding the border with the underlying corpus callosum, and a dorsal boundary corresponding to the border with the overlying cingulate sulcus (Figure 5). The NAcc ROI mask was taken from the Wake Forest University Pickatlas (Maldjian, Laurienti, Kraft, & Burdette, 2003; Figure 5).

Figure 5. Region-of-interest masks for the (A-C) dACC in blue and (D) the NAcc in orange. Coordinates are in MNI space.
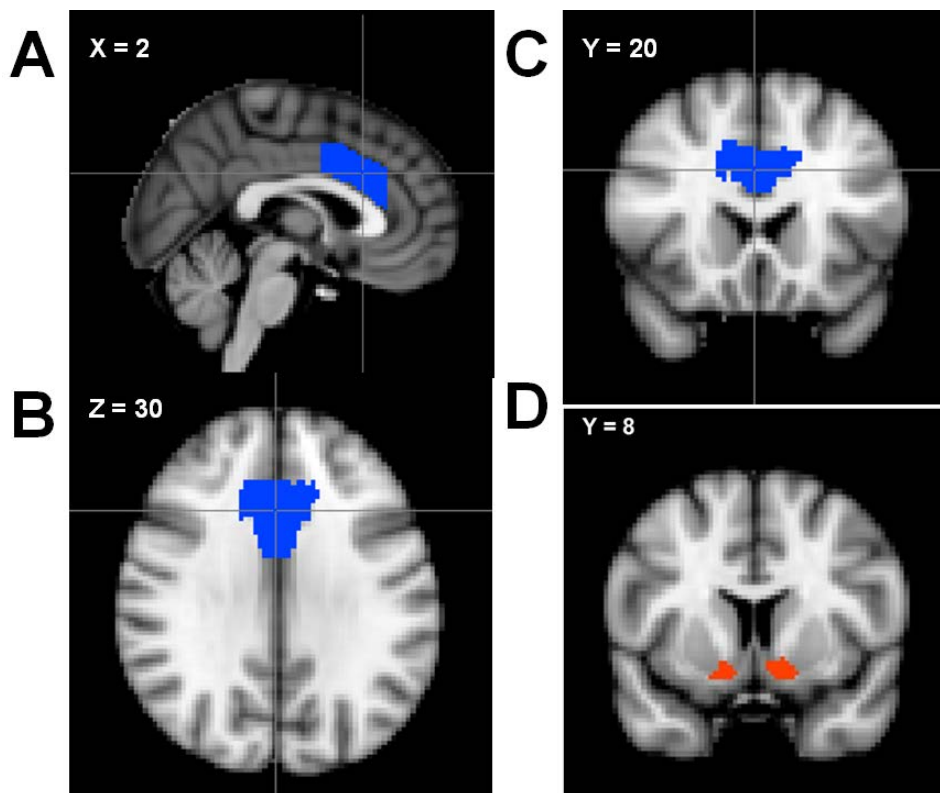
2.5.2 Moderated mediation models. Prior to entry into the moderated mediation model, values from each of the four measures (social pain, aggressive pleasure, retaliatory aggression, executive functioning) were checked for outliers

(+/- 1.5 times the inter-quartile range of the sampling distribution for each measure) and mediation paths were checked for residuals that followed a roughly normal distribution (using a Kolmogorov-Smirnov test for normality). Afterwards, values were entered in the model using PROCESS, a macro for SPSS (Hayes, 2012). The PROCESS model yielded parameter estimates for the each path of the model (e.g., *a*: dACC -> NAcc; *b*: NAcc -> retaliatory aggression; *c*: dACC -> retaliatory aggression; *c'*: dACC -> retaliatory aggression, controlling for the indirect effect) at low (-1 *SD*), mean, and high (+1 *SD*) levels of executive functioning. Using nonparametric bootstrapping (1,000 re-samples), the macro yielded 95% confidence intervals around the indirect effect of aggressive pleasure at low (-1 *SD*), mean, and high (+1 *SD*) levels of executive functioning.

     2.5.3 Reverse inference correlations. To provide additional evidence that the dACC and anterior insula activity during rejection was a signature of social pain, I correlated the affective subscale of the McGill Pain Questionnaire, and each of the seven subscales of the Need Threat Scale with dACC and anterior insula percent signal change estimates. For the NAcc-pleasure reverse inference, I correlated NAcc activity during retaliatory aggression with the Aggressive Pleasure Scale and the Short Sadistic Impulse Scale.

Chapter Three: Results

3.1 Neuroimaging

      3.1.1 Cyberball. At each voxel across the whole brain, social rejection

(compared to social acceptance) was both positively and negatively associated

with large swaths of neural activity (see Tables 2 and 3).

Table 2. Brain regions positively associated with Reject > Accept during Cyberball. Each cluster is displayed with rows for all local maxima.

Cluster	Voxels	Brain Region	Peak Z	Peak x,y,z
1	25,205	VLPFC/Anterior Insula	6.07	48,22,12
			5.98	52,22,12
			5.88	46,36,-6
			5.81	56,26,12
		Temporoparietal Junction	5.77	50,-44,20
		Middle Temporal Gyrus/ Temporal Pole	5.68	56,-10,-16
2	10,661	VLPFC	6.46	-42,20,-26
		Temporoparietal Junction	6.00	-46,-66,22
		Middle Temporal Gyrus/ Temporal Pole	5.89	-46,8,-26
		Temporoparietal Junction	5.72	-50,-66,18
			5.65	-44,-70,24
		Anterior Insula	5.61	-36,24,-6
3	9,239	Dorsal MPFC	7.52	10,52,36
			7.09	8,46,30
			7.01	6,46,26
4	1,492	Posterior Cingulate Cortex	5.49	4,-54,34
5	1,382	Thalamus/Caudate	5.34	6,-10,10
6	1,218	Occipital Cortex	5.03	-12,-94,4
7	599	Brainstem	3.53	4,-30,-30

Table 3. Brain regions negatively associated with Reject > Accept during Cyberball. Each cluster is displayed with rows for all local maxima.

Cluster	Voxels	Brain Region	Peak Z	Peak x,y,z
1	7,616	Posterior Insula	-6.54	-38,-6,16
		Precentral Gyrus	-5.69	-30,-14,64
			-5.33	-36,-16,66
		Supplemental Motor Area	-5.26	-4,-6,54
		Postcentral Gyrus	-5.14	-48,-34,56
			-4.98	-52,-28,50
2	834	DLPFC	-4.29	-40,44,8
			-3.67	-46,28,28
			-3.60	-34,32,28
			-3.58	-34,24,24
			-3.46	-40,34,34
			-3.29	-38,52,16
3	478	Superior Parietal Lobule	-3.77	30,-48,68
		Supramarginal Gyrus	-3.56	48,-36,54
		Postcentral Gyrus	-3.51	42,-28,40
		Superior Parietal Lobule	-3.46	36,-46,64
		Postcentral Gyrus	-3.09	46,-32,50
		Superior Parietal Lobule	-3.02	32,-54,64

The positively-associated regions included anticipated brain areas previously associated with social pain (bilateral anterior insula, periaqueductal gray, thalamus) and social pain regulation (ventrolateral prefrontal cortex; Figure 6).

Figure 6. Greater activity from the Reject > Accept contrast of the Cyberball task in the (A-C) left and (C-E) right anterior insula and overlying VLPFC. Green voxels represent significant clusters at corrected $p < .05$, red\yellow voxels represent significant clusters at corrected $p < .0001$. Blue voxels represent negatively-associated clusters at corrected $p < .05$.
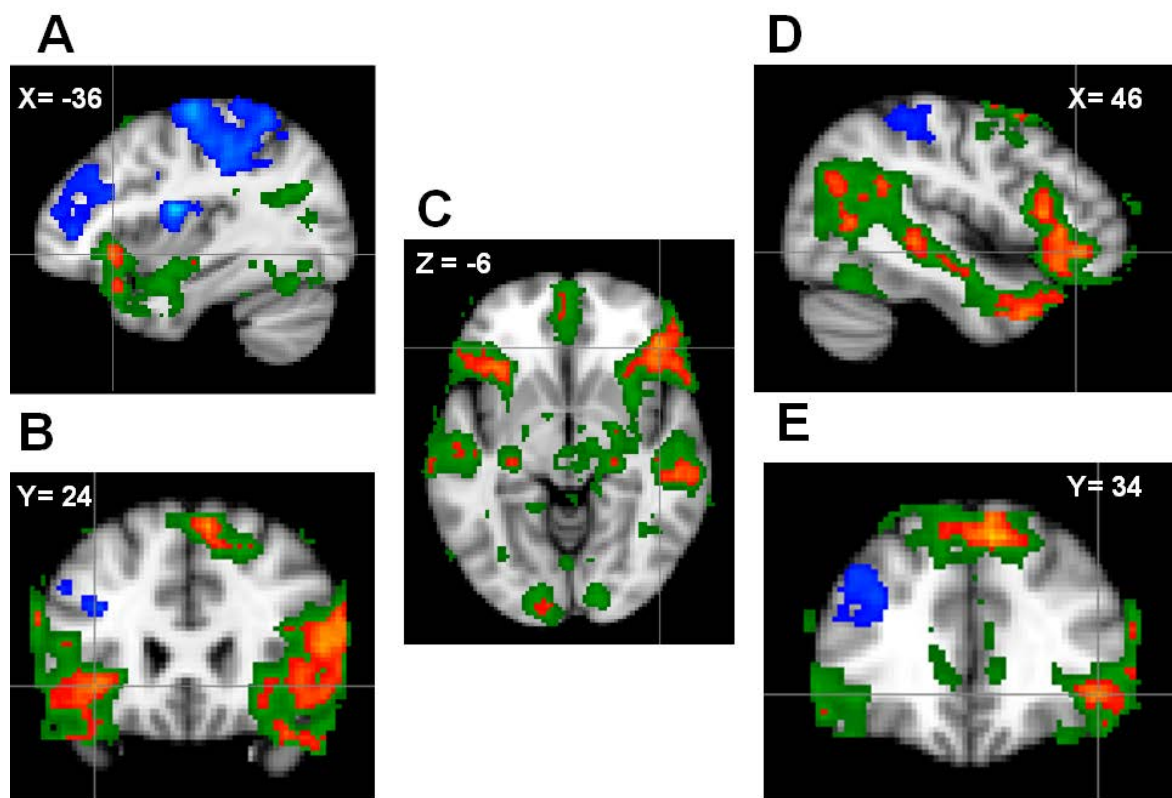
Unexpectedly, much more activity was located in what is popularly known as the mentalizing network [medial prefrontal cortex (MPFC), posterior cingulate cortex\precuneus (PCC), bilateral temporoparietal junction (TPJ), bilateral

temporal pole (TP); Figure 7]. Other positively-associated areas included the brainstem, caudate, primary visual cortex, and middle temporal gyrus. Negatively-associated areas included the dorsolateral prefrontal cortex, middle insula, precentral and postcentral gyri. The reduced activity of these motor and sensory systems was likely due to the lack of ball-toss-related button pressing that participants performed during the rejection block of the task, which was present in the acceptance condition.

Figure 7. Greater activity from the Reject > Accept contrast of the Cyberball task in the mentalizing network, which includes the (A) dorsal MPFC and PCC, (B) left TPJ and TP, and (C) right TPJ and TP.
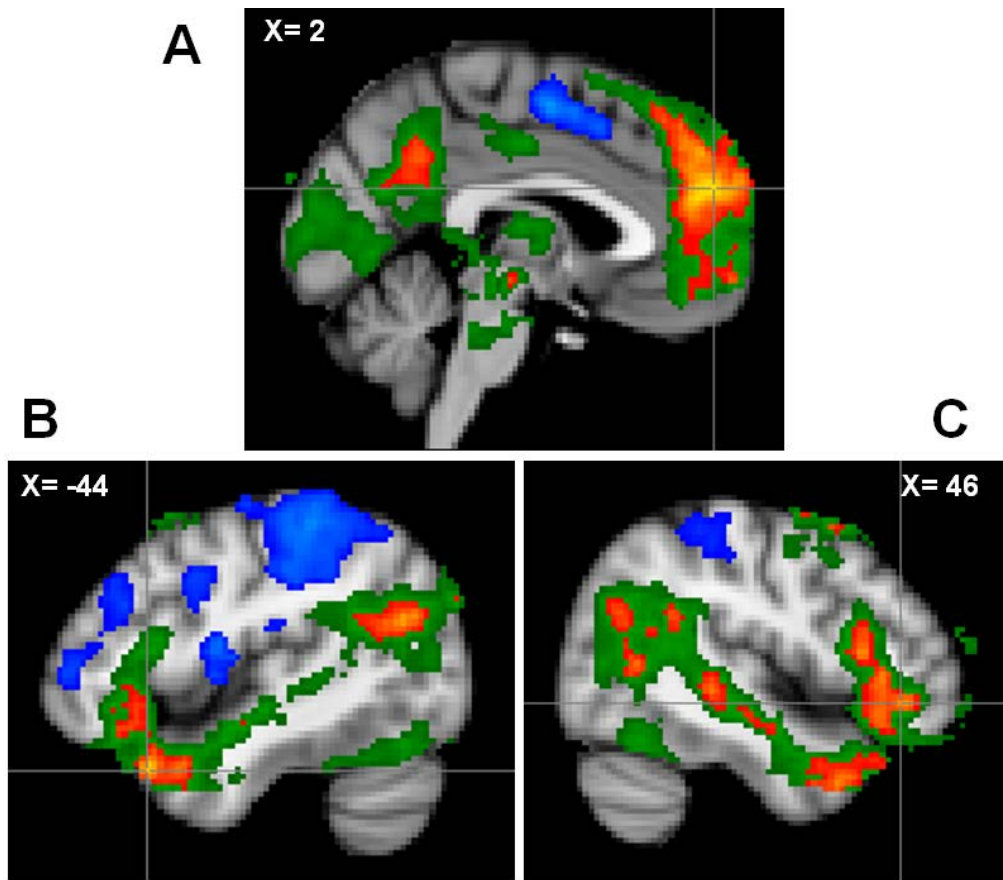
Due to the massive amount of brain activity that was associated with social rejection, I subsequently re-ran the whole brain analyses with a substantially stricter statistical threshold (family-wise error rate corrected $Z > 4.0$, $p < .0001$) to reveal the most robust clusters of statistical significance. Doing so allowed me to identify neural regions most associated with social rejection. This analysis retained large, positively-associated clusters in the pain network (bilateral anterior insula, thalamus, bilateral VLPFC), mentalizing network (MPFC, PCC, bilateral TPJ, bilateral TP), brainstem, and occipital cortex (Figures 6 and 7).

Notably absent from both of these whole-brain contrasts was rejection-related activity in the dorsal anterior cingulate cortex (dACC). To provide a more lenient test of my dACC hypothesis, I re-ran my original Reject > Accept contrast (threshold was returned to the original, family-wise error rate corrected $Z > 2.3$, $p < .05$) but I constrained these analyses to the pre-specified dACC ROI. This ROI analysis reduced the severity of my multiple comparisons correction as it reduced the number of voxels being analyzed and thus the number of statistical tests. However, this analysis revealed no significant clusters of rejection-related neural activity in the dACC. Despite the lack of above-threshold neural activity in the dACC, there existed substantial beneath-threshold brain activity in the dACC that could be entered into the proposed moderated mediation model. Brain activity estimates, in the form of percent BOLD signal change, were averaged across and extracted from all voxels in the dACC ROI. Plotting the times of the dACC, averaged across all voxels and participants, demonstrated that BOLD signal

estimates were lower during rejection blocks on average, though they seemed to peak towards the end of the rejection block (Figure 8).

Figure 8. Average timecourse of the dACC across the Cyberball Task.
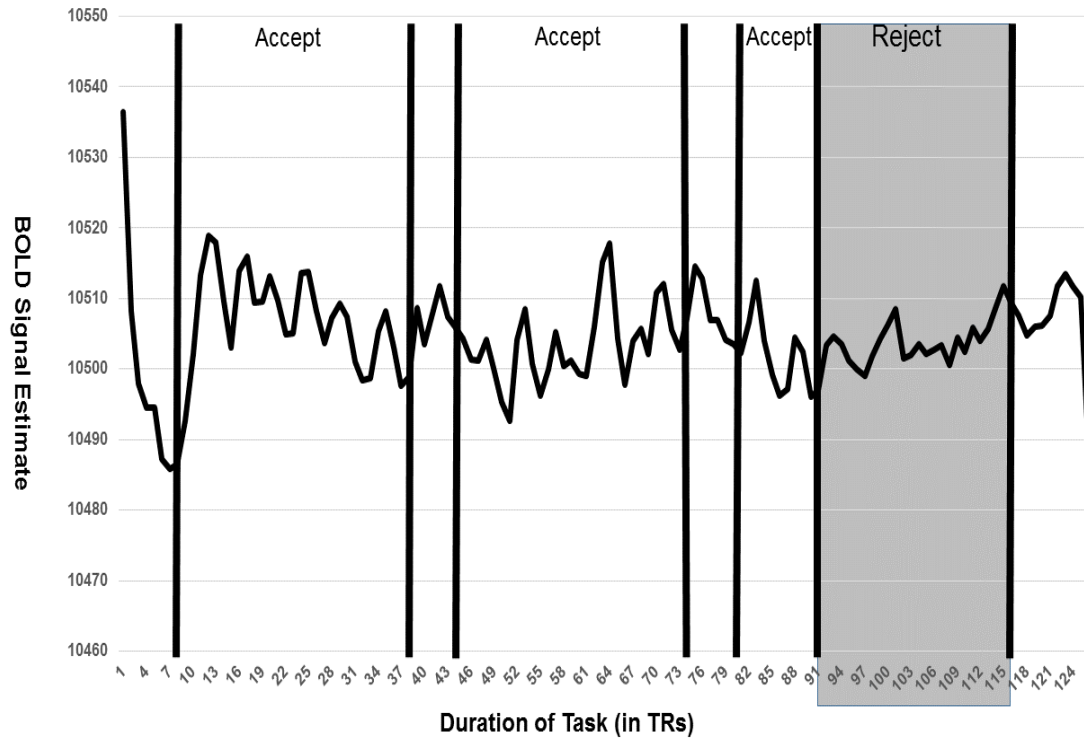
Identical ROI analyses were also performed on the anterior insula as the clusters in these regions extended contiguously into the nearby ventrolateral prefrontal cortex. Thus, an ROI approach was necessary to isolate the voxels specific to the anterior insula. Anterior insula masks were obtained from my previous research (Chester et al., 2014), using a caudal boundary of y = 8 (in MNI space) and corresponding in both the x and s planes to the agranular insula (Figure 9).

Figure 9. ROI masks of the bilateral anterior insula.
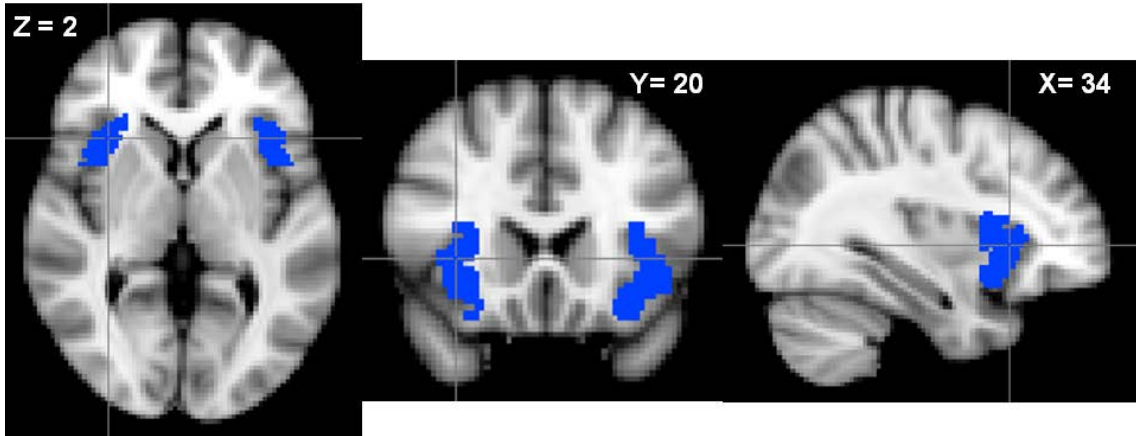
As observed in the whole-brain analysis, significant rejection-related neural activity was observed in both the left [361 voxels, peak Z = 5.61, peak MNI coordinates (x, y, z): -36, 24, -6] and right anterior insula [387 voxels, peak Z = 5.22, peak MNI coordinates (x, y, z): 32, 10, -18; Figure 10]. Activity estimates, in the form of percent BOLD signal change, were averaged across and extracted from all significant voxels in the left and right anterior insula, separately, for subsequent analyses.

Figure 10. Greater activity from the Reject > Accept contrast of the Cyberball task in the left and right anterior insula at corrected p < .05.
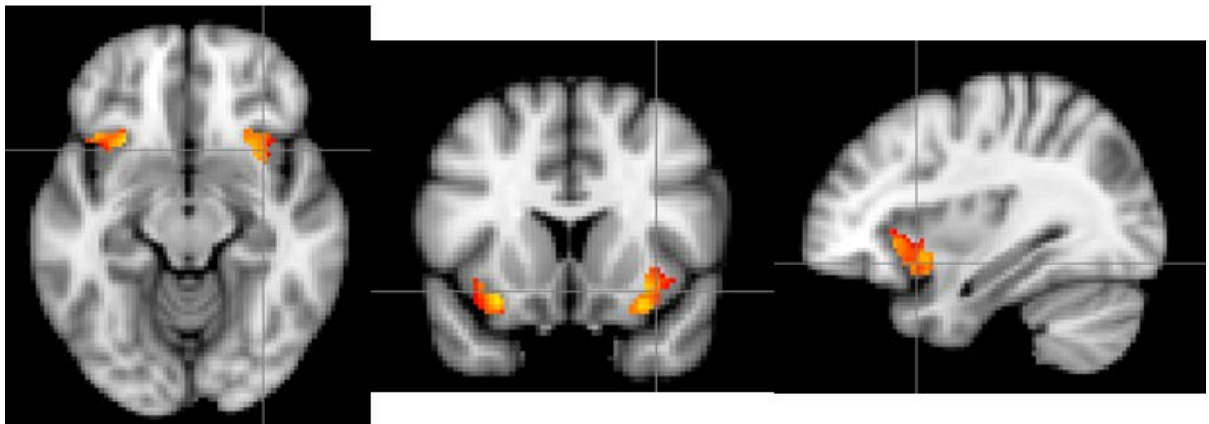
3.1.2 Taylor Aggression Paradigm. Across the entire brain, retaliatory aggression (as compared to non-retaliatory aggression) on the Taylor Aggression Paradigm was positively associated with activity in the right [952 voxels, peak Z = 3.68, peak MNI coordinates (x, y, z): 34, -14, 24] and left [688 voxels, peak Z = 4.62, peak MNI coordinates (x, y, z): -50, -20, 8] posterior insula and negatively associated with activity in the occipital lobe [2,101 voxels, peak Z = -4.92, peak MNI coordinates (x, y, z): 38, -88, -6] (Figure 11). This posterior insula finding replicates previous work from my own and other laboratories' studies on retaliatory aggression (Chester & DeWall, in press[a]; Emmerling et al., in press). Despite the lack of above-threshold neural activity in the nucleus accumbens (NAcc), there existed substantial beneath-threshold brain activity in the NAcc that could be entered into the proposed moderated mediation model. Brain activity estimates, in the form of percent BOLD signal change, were averaged across and extracted from all voxels in the bilateral NAcc ROI.

Figure 11. Bilateral posterior insula activity associated with Retaliatory Aggression > Non-Retaliatory Aggression. Blue voxels represent negatively-associated clusters in the occipital cortex.
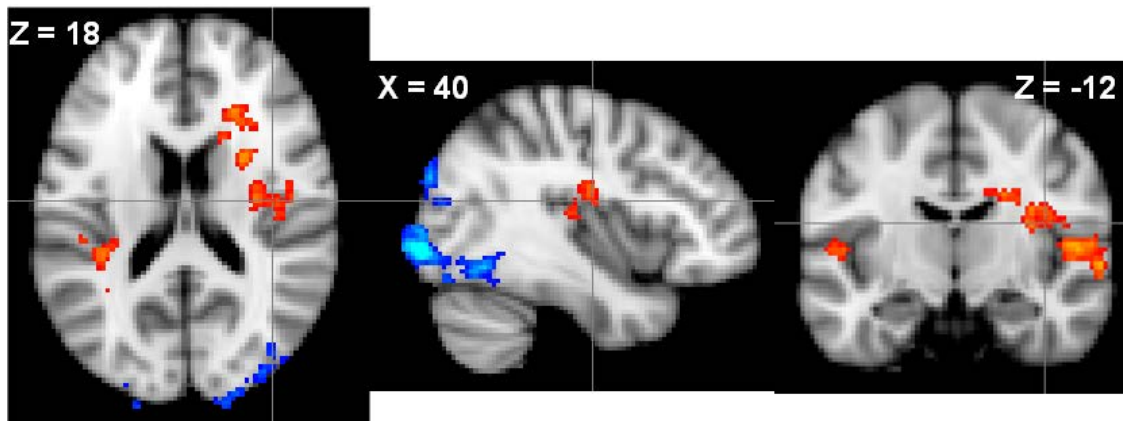
3.2 Descriptive Statistics

      3.2.1 Score computations. Several measures required computation procedures to yield values for each participant that reflected their levels of the associated latent construct. In addition to the individual dACC and left and right anterior insula estimates of social pain, I averaged together each of these percent BOLD signal change estimates (Reject > Accept contrast) to create a social pain index with higher reliability as it was constructed from three measurements instead of a single one. Retaliatory aggression was operationalized by averaging together each of the noise volume settings from retaliatory aggression trials (i.e., aggression trials that occurred after a high provocation block) of the Taylor Aggression Paradigm. The same was performed for non-retaliatory trials (i.e., aggression trials that occurred after a low provocation block).

      Finally, executive functioning was operationalized by participants' performance on the Stroop color-naming task. Due to logistical errors, randomized Stroop data were missing for two participants and blocked Stroop data were missing for 33 participants. Because of the substantial amounts of missing data for the blocked Stroop color-naming task, this measure was not analyzed. Scores from the 58 participants who completed the randomized Stroop task were retained to use in subsequent analyses. To score the randomized Stroop task, incorrect trials were first removed (congruent trial accuracy: $M = 97.93\%$, $SD = 7.44\%$, range = 50% - 100%; incongruent trial accuracy: $M = 87.41\%$, $SD = 16.92\%$, range = 30% - 100%). Then, the remaining latency

scores (the time in milliseconds it took participants to respond to the trial) were averaged across all congruent and incongruent trials separately. To isolate participants' performance on the incongruent trials and to account for general speed of responding, congruent latency scores were subtracted from incongruent latency scores. Because of this scoring system, larger values on this variable represented longer times to respond to the incongruent stimuli and thus, deficits in the executive functions necessary to correctly perform the task (e.g., inhibition of prepotent responses).

Self-report measures were scored by reversing appropriate items and then averaging together all items that corresponded to the given subscale. One participant was missing data from the Short Sadistic Impulse Scale as they spent too much time on prior tasks to finish the full battery of questionnaires.

3.2.2 Outlier detection. Box and whisker plots were used to visually inspect the distributions of the social pain index (created by averaging together the dACC and anterior insula activity estimates from the Cyberball task), NAcc activity during retaliatory aggression, retaliatory aggression, and Stroop task latencies. Boxes depicted the middle two quartiles of the distributions and whiskers extended in both directions to span 1.5 times the inter-quartile range (Figure 12). From each of these plots, three outliers were identified as extreme values by the statistical software: two from the social pain index (one of which was an outlier on the NAcc variable) and one from the randomized Stroop task. These three participants were excluded from all subsequent analyses. Combined

with the missing data, there were full, non-outlier datasets from 55 participants

that were used for all subsequent analyses.

Figure 12. Box-whisker plots demonstrating three outliers (as indicated by stars) from (A) NAcc activity during retaliatory aggression, (B) randomized Stroop latency differential, and (C) dACC\insula activity during social rejection. Y axis values represent raw data values. Numbers associated with dots represent arbitrary case numbers.
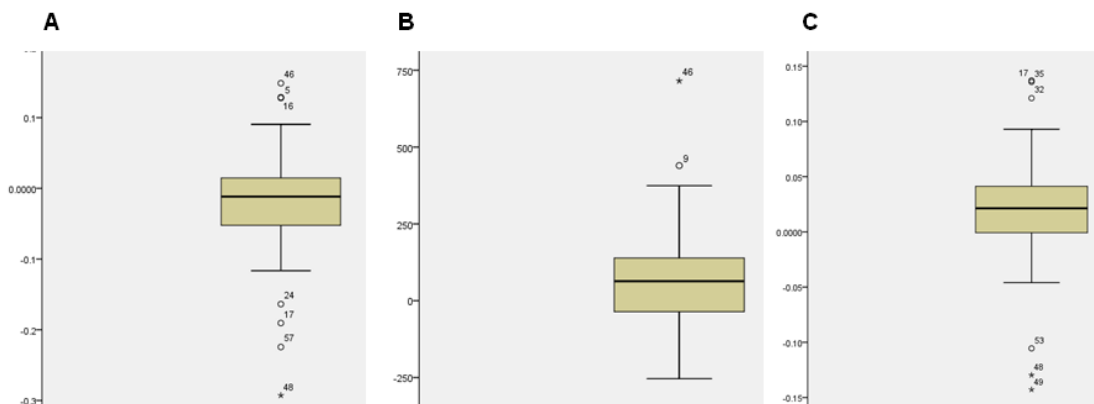
3.2.3 Behavioral effects. Replicating decades of research, a paired-samples t-test revealed that incongruent Stroop trials had longer response latencies, $M = 655.59$, $SD = 181.80$, than congruent trials, $M = 601.88$, $SD = 167.34$, $t(54) = 2.81$, $p = .007$, $d = .31$. Additionally, retaliatory aggression scores, $M = 2.55$, $SD = 0.87$, were higher than non-retaliatory aggression scores, $M = 2.37$, $SD = 0.79$, $t(56) = 3.12$, $p = .003$, $d = .22$.

3.2.4 Scale reliability. Internal consistencies were obtained for the four self-report questionnaires that were administered, and their associated subscales. Cronbach's αs were computed and indicated that for the majority of the scales, internal consistency was adequate (αs > .70). However, internal

consistency was sub-standard for the Affective subscale of the McGill Pain

Questionnaire (α = .61), the Control (α = .68) and Belonging (α = .69) subscales

of the Need Threat Scale, and the Short Sadistic Impulse Scale (α = .51). Lower

internal consistencies on these measures may have served to reduce the size of

observed correlations between these measures and other variables.

3.3 Moderated Mediation Modeling

Using a moderated mediation approach, I tested whether the effect of

rejection-related dACC activity on retaliatory aggression through NAcc activity

during such aggression was conditional upon individual's levels of executive

functioning.

3.3.1 Assumption checks. The moderated mediation approach uses

ordinary-least-squares regression from the general linear model to calculate

parameter estimates and significance tests for each path of the model. As such,

the validity of the moderated mediation model is contingent upon the

assumptions of multiple linear regression: a linear and homoscedastic

relationship between predictors and dependent measures, normality of residuals,

negligible multicollinearity of predictors, and no auto-correlation. These

assumptions were checked for each individual path of the mediation model I

tested.

3.3.1.1 Linearity, normality, and homoscedasticity. Scatterplots for

each path were visually inspected to ensure that a curvilinear, quadratic

relationship was not evident (Figure 13). To assess multivariate normality and

homoscedasticity, residuals were saved from each path of the mediation model

and a Kolmogorov-Smirnov test for normality revealed that each path met the assumptions of normality (ks < .11, ps > .07) except for the a path of the model in which rejection-related dACC activity was modeled to predict NAcc activity during retaliatory aggression, k(57) = .13, p = .015. Homoscedasticity was further assessed by examining quantile-quantile plots for deviations from a linear trend. Heteroscedasticity was observed for the relationship between NAcc activity during retaliatory aggression and retaliatory aggression scores. This issue biases variance estimates by the regression model and thus largely invalidate statistical significance estimates of the association.

Figure 13. Scatterplots depicting zero-order relationships between (A) dACC\insula activity during social rejection and retaliatory aggression scores from the Taylor Aggression Paradigm, (B) dACC\insula activity during social rejection and NAcc activity during retaliatory aggression, and (C) NAcc activity during retaliatory aggression and retaliatory aggression scores.
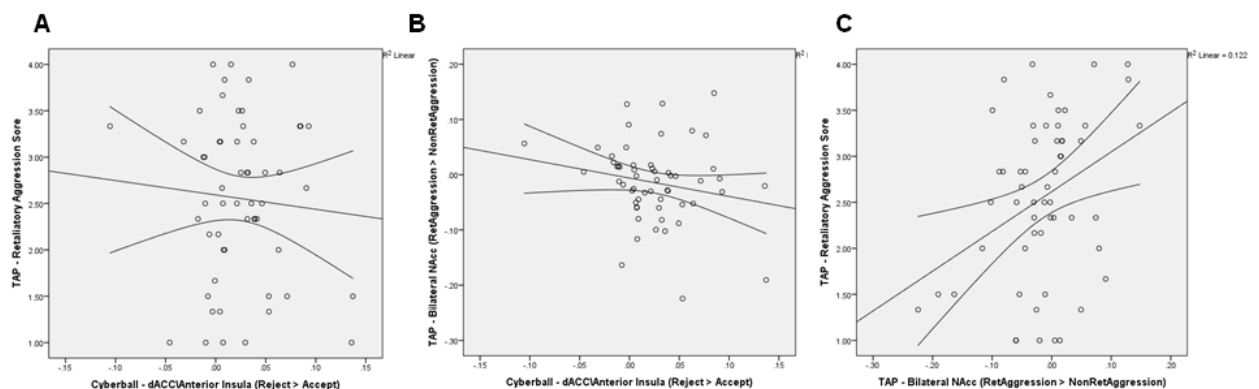
3.3.1.2 Multicollinearity of predictors. Multicollinearity was assessed with variance inflation factors that were acquired from a multiple linear regression model that included both the rejection-related dACC estimate and NAcc activity

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during retaliatory aggression as predictors. For this model, VIF = 1.02, which was well below a standard collinearity issue cutoff of 2. Further, these two predictors were unassociated with one another, $r(55) = -.13$, $p = .347$.

    3.3.1.3 Autocorrelation. Autocorrelation for each path of the mediation model was assessed using Durbin-Watson tests, which indicated that no autocorrelation was present as all computed values were substantially close to 2 (range: 1.99 – 2.33).

    The paths of the predicted mediation model appeared to meet all assumptions for ordinary least squares regression, with the exception that the residuals of the *a* path showed significant skewness. All such assumptions were met when left and right anterior insula activity during social rejection was modeled instead of dACC activity, as well as with the social pain index that was created by averaging activity estimates from all three social pain regions.

    3.3.2 Zero-order correlations. Correlating each variable in the model revealed that NAcc activity during retaliatory aggression was significantly associated with greater levels of retaliatory aggression and that rejection-related dACC activity was marginally association with *lesser* levels of retaliatory aggression (see Table 4 for all correlations). Using left and right anterior insula as a proxy for social pain instead of the dACC revealed a marginally-significant association between right anterior insula activity and *lesser* NAcc activity during retaliatory aggression. All other associations were non-significant.

Table 4. Zero–order correlations between primary study variables.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. dACC (Reject>Accept)															
2. Left Insula (Reject>Accept)	.42**														
3. Right Insula (Reject>Accept)	.53***	.73***													
4. dACC\Insula (Reject>Accept)	.74***	.87***	.91***												
5. NAcc (Ret. Agg.>Non-Ret. Agg)	-.13	-.14	-.24	-.20											
6. Retaliatory Aggression	-.23†	-.03	.01	-.09	.35**										
7. Stroop Latency	-.02	.07	.04	.04	-.08	-.02									
8. NTS – Belonging	.14	.02	.08	.09	-.17	-.02	.15								
9. NTS – Self-Esteem	.17	.22	.28*	.27*	-.05	.11	.06	.68***							
10. NTS – Meaningful Existence	.15	.10	.13	.15	-.03	.19	.01	.67***	.71***						
11. NTS – Control	.09	.08	.10	.10	-.22	-.17	.04	.48***	.45***	.54***					
12. NTS – Felt Rejection	-.01	.17	.11	.11	.06	.05	.13	.34	.33	.51***	.61***				
13. NTS – Positive Affect	-.18	-.12	-.17	-.18	-.08	-.13	.03	-.57***	-.78***	-.57***	-.33*	-.24†			
14. NTS – Negative Affect	.22	.12	.17	.20	.12	.15	.06	.59***	.66***	.69***	.23†	.38**	-.63***		
15. Aggressive Pleasure Scale	-.18	-.20	-.07	-.17	.01	-.12	.13	-.09	-.31*	-.18	.09	-.01	.32*	-.27*	
16. MPQ – Affective	.06	-.06	-.07	-.03	.10	.18	-.13	.38**	.19	.29*	-.04	.10	-.13	.23	-.24†

3.3.3 Un-moderated mediation. Prior to modeling whether my predicted indirect effect was moderated by executive functioning, I conducted unmoderated mediation analyses. In this model, rejection-related dACC activity was modeled as the predictor, NAcc activity during retaliatory aggression was modeled as the mediator, and retaliatory aggression scores were modeled as the dependent measure. Nonparametric, bias-corrected, and accelerated bootstrapping (with 1,000 resamples) was used to create a 95% confidence interval around the indirect effect. All variables were mean-centered prior to testing mediation. According to this measure, there was no significant mediation via NAcc activity, 95% C.I. = -3.48, 0.65.

No mediation was observed when dACC activity was replaced with rejection-related activity estimates from the left anterior insula, 95% C.I. = -3.57, 0.53, or the 3-region social pain index, 95% C.I. = -4.53, 0.26. However, using rejection-related activity estimates from the right anterior insula did produce evidence for significant mediation, 95% C.I. = -4.61, -0.10, R^2 = .13, $F(2,54)$ = 4.13, p = .021 (Figure 14). Despite a non-significant direct effect of anterior insula activity on retaliatory aggression, B = 1.56, $t(52)$ = 0.76, p = .453, anterior insula activity was marginally, negatively associated with NAcc activity during retaliatory aggression, B = -0.31, $t(52)$ = -1.85, p = .070, which was then positively associated with retaliatory aggression, B = 4.64, $t(52)$ = 2.88, p = .006.

Figure 14. Mediation model whereby the null effect of rejection-related right anterior insula activity on retaliatory aggression is partially accounted for by NAcc activity during retaliatory aggression. *p < .01, †p < .10
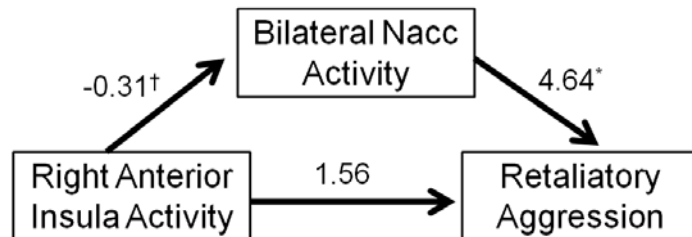
3.3.4 Moderated mediation. The initial models tested moderation of the previously discussed indirect effects by executive functioning at the *a*, *b*, and *c* paths simultaneously. Modeling the dACC as the predictor and retaliatory aggression as the dependent measure, there was no observed indirect effects of NAcc activity during retaliatory aggression at low (-1 *SD;* 95% C.I. = -2.73, 7.32), mean (95% C.I. = -3.95, 0.08), or high (+1 *SD;* 95% C.I. = -6.37, 2.78) levels of executive functioning. This lack of moderated mediation was also observed if the left or right anterior insula, or the 3-region social pain index were used as predictors instead of the dACC.

Moderated mediation can be modeled in multiple ways. I used additional analyses sought to restrict my predicted moderation to the *a* and *c* paths of the mediation model, removing the path in the model whereby executive functioning moderated the *b* path of the indirect effect. This was an empirically-supported analytic decision as the moderating effect of executive functioning had only previously been observed with social pain's effect on aggressive outcomes (Chester et al., 2014), not with aggressive pleasure or its neural substrates. When modeled in this fashion, significant moderation of the indirect effect was observed. Specifically, at high and

mean levels of executive functioning, a significant indirect effect was observed whereby the effect of dACC activity on lesser retaliatory aggression was mediated by reduced NAcc activity during such aggression (Table 5). When modeling left or right anterior insula activation or the 3-region social pain index as the predictor, this significant indirect effect was observed at high levels of executive functioning (Table 5).

Table 5. Summary of moderated mediation models. Columns a, b, and c represent unstandardized regression coefficients of each specific path of the model. C.I. represents the 95% confidence interval around the indirect effect at high (+1 *SD*), mean, and low (-1 *SD*) levels of executive functioning. *R²* represents the amount of variance in aggression accounted for by each model.

Predictor	a	b	c	95% C.I. High	95% C.I. Mean	95% C.I. Low	R^2
dACC	-0.38†	3.58*	-4.66†	-7,36, -0.08	-4.79, -0.04	-3.01, 1.20	.16
Left AI	-0.21	4.45*	-0.04	-6.19, -0.07	-4.49, 0.37	-3.70, 1.71	.11
Right AI	-0.31†	4.55*	1.99	-5.64, -0.17	-5.22, 0.16	-7.38, 0.65	.15
3-Region Index	-0.39†	4.13*	-0.43	-7.56, -0.32	-5.89, 0.20	-6.56, 0.75	.12

*$p < .05$, †$p < .10$

To test whether another measure of dispositional self-regulation would replicate these moderated mediation findings, I re-ran these models using scores from the Brief

Self-Control Scale (Tangney et al., 2004) as the moderator, in place of Stroop task performance. These models largely replicated the above models, in which social pain estimates' effect on less aggression were mediated by lesser NAcc activity during retaliatory aggression, though only among individuals high in self-regulation (Table 6).

Table 6. Summary of moderated mediation models. Columns a, b, and c represent unstandardized regression coefficients of each specific path of the model. C.I. represents the 95% confidence interval around the indirect effect at high (+1 *SD*), mean, and low (-1 *SD*) levels of trait self-control. R^2 represents the amount of variance in aggression accounted for by each model.

Predictor	*a*	*b*	*c*	95% C.I. High	95% C.I. Mean	95% C.I. Low	R^2
dACC	1.65	4.44*	-14.19	-8.71, -0.10	-3.52, 1.13	-2.33, 6.04	.23
Left AI	0.51	4.59*	-15.30	-6.33, 0.66	-3.29, 1.03	-3.57, 3.48	.24
Right AI	1.19	5.20*	-24.41	-8.69, 0.10	-4.37, -0.01	-3.61, 4.19	.27
3-Region Index	1.82	4.95*	-32.68	-9.97, -0.02	-4.49, 0.50	-3.41, 6.77	.26

*$p < .01$

3.4 Reverse Inference Correlations

3.4.1 Social pain measures. Rejection-related dACC and anterior insula activity estimates were unassociated with any of the Need Threat Scale or McGill Pain

Questionnaire subscales, $ps > .10$, with a few exceptions (Table 7). Rejection-related activity in the right anterior insula was positively associated with the Self-Esteem subscale of the Need Threat Scale, $r(55) = .28$, $p = .037$. This effect was also observed when dACC and anterior insula activations were averaged to create a neural social pain index, $r(55) = .27$, $p = .044$.

Table 7. Zero–order correlations between social pain brain activity during social rejection and self-report measures of social pain.

	dACC	Left Insula	Right Insula	dACC\Insula
NTS - Belonging	.14	.02	.08	.09
NTS - Self-Esteem	.17	.22†	.28*	.27*
NTS - Meaning	.15	.10	.13	.15
NTS - Control	.09	.08	.10	.10
NTS - Felt Rejection	-.01	.17	.11	.11
NTS - Pos. Affect	.19	-.12	-.17	-.18
NTS – Neg. Affect	.22	.12	.17	.20
MPQ - Affective	.06	-.06	-.07	-.03

†$p < .10$, *$p < .05$

3.4.2 Aggressive pleasure measures. NAcc activity during retaliatory aggression was unassociated with scores on the Aggressive Pleasure Scale, $r(55) = .27$, $p = .955$, or the Short Sadistic Impulse Scale, $r(54) = .05$, $p = .739$.

3.5 Exploratory Analyses

3.5.1 Mentalizing network. An unexpected, though coherent set of findings from my neuroimaging analyses was the robust association between social rejection and

activity in the mentalizing network, including the MPFC, PCC, TPJ, and TP. To better

understand the role that these regions played in relation to my hypotheses, I examined

the MPFC's and PCC's relation to retaliatory aggression, NAcc activity during retaliatory

aggression, and our self-report measures, as these two regions of the mentalizing

network are considered the 'hubs' of it. I extracted percent signal change values from

the activated clusters in the MPFC and PCC (voxels determined by the stricter $p <$

.0001 threshold). One outlier was observed for the MPFC, which was already

considered an outlier based on its place in the sampling distribution of other measures.

Bivariate correlations were performed (Table 8). It appeared that mentalizing activity

during social rejection was unassociated with aggression or NAcc activity. Instead, the

MPFC activity during social rejection was associated with reports of distress (e.g.,

positively with self-esteem threats and negative affect, and negatively with positive

affect). Paradoxically, both the MPFC and PCC were negatively associated with reports

of social pain.

Table 8. Zero-order correlations between rejection-related activity in mentalizing regions (medial prefontal cortex, posterior cingulate cortex) and the right ventrolateral prefrontal cortex and primary study variables.

	MPFC	PCC	VLPFC
dACC (Reject > Accept)	.50***	.29*	.45***
Left Insula (Reject > Accept)	.51***	.38**	.58***
Right Insula (Reject > Accept)	.66***	.49***	.67***
dACC\Insula (Reject > Accept)	.67***	.47***	.01
NAcc (Ret. Agg. > Non-Ret. Agg)	.10	.02	-.09
Retaliatory Aggression	.05	-.03	.04
Stroop Latency	.18	.13	.10
NTS – Belonging	.06	-.01	.32*
NTS – Self-Esteem	.37**	.16	.15
NTS – Meaningful Existence	.22†	.18	.12
NTS – Control	.04	.20	-.01
NTS – Felt Rejection	.06	.17	-.36**
NTS – Positive Affect	-.31*	-.17	.17
NTS – Negative Affect	.33*	.23†	-.14
Aggressive Pleasure Scale	-.13	.08	-.13
MPQ – Affective	-.22†	-.32*	.68***

$^†p < .10$, $^*p < .05$, $^{**}p < .01$, $^{***}p < .001$

3.5.2 VLPFC. The VLPFC also exhibited substantial activity during social rejection, though this was expected based on previous research implicating the VLPFC

as a regulatory region of social pain (Chester & DeWall, 2014; Eisenberger et al., 2003). In an exploratory fashion, I sought to test associations between this brain region and the aggression, NAcc, and self-report measures of social pain. Similarly to the MPFC, the VLPFC was associated with reports of distress (e.g., positively with self-esteem threats and negatively with positive affect; Table 8).

3.5.3 Trait aggression. I measured aggression in this study in both situational (via the Taylor aggression paradigm) and dispositional (via a trait aggression questionnaire) modalities. To test whether the observed effects from the study were specific to behavior aggression situated in provocation, or if they would generalize to a general tendency to act aggressively, I re-ran the moderated mediation models that produced significant effects but with trait aggression as the outcome measure (as quantified by the Brief Aggression Questionnaire; Webster et al., 2014). None of the models exhibited a significant indirect effect at any level of executive functioning. In an exploratory manner, Brief Aggression Questionnaire scores were correlated with neural activity estimates from Cyberball and the Taylor Aggression Paradigm, as well as aggression scores themselves (Table 9). BAQ scores were positively associated with retaliatory, $r(54) = .31$, $p = .019$, and non-retaliatory, $r(54) = .32$, $p = .017$, aggression from the Taylor Aggression Paradigm and marginally, positively associated with NAcc activity during retaliatory aggression, $r(54) = .23$, $p = .086$.

Table 9. Associations between Brief Aggression Questionnaire scores and aggression-related study variables.

	dACC (Reject>Accept)	Left Insula (Reject>Accept)	Right Insula (Reject>Accept)	NAcc (Ret. Agg>Non-Ret. Agg)	Ret. Agg
BAQ	-.19	-.02	-.04	**.23†**	**.31***

†$p < .10$, *$p < .05$

3.5.4 Gender differences. Using independent-samples t-tests, we explored whether males and females differed on any of the primary study variables. The only significant differences that emerged were in self-reports of sadism and trait aggression (see Table 10).

Table 10. Significant gender differences observed in primary study variables.

	Males *M*(*SD*)	Females *M*(*SD*)	T statistic
Short Sadistic Impulse Scale	1.66(0.47)	1.32(0.38)	2.93*
Brief Aggression Questionnaire	3.94(0.87)	2.87(0.81)	4.50**

*$p < .01$, **$p < .001$

Chapter Four: Discussion

Sigmund Freud said that "civilization began the first time an angry person cast a word instead of a rock" (p. 342, Freud & Freud, 1960). Indeed, mankind's trend away from using physical aggression to solve our problems is perhaps one of the largest contributors to our species' collective success. The psychological and neural sciences have been handed the mantle of violence reduction. In the work presented in this dissertation, I sought to better understand, and thus hopefully reduce, aggression, by exploring its underlying mechanisms. Towards this end, I investigated a common cause of aggressive behavior, social rejection (Twenge et al., 2001), and the role that pain and pleasure might play in the link between rejection and aggression. I used a multidisciplinary approach, combining social psychological methods and theories with neuroscientific models to look under the hood of the rejection-aggression link.

4.1 Summary of Hypotheses

I tested a series of hypotheses that cohered around a single, moderated-mediation framework. This model largely sought to test a single question: does the pain of social rejection promote the pleasure of revenge? To provide some preliminary evidence regarding this question, I tested several novel hypotheses. First, that rejection-related activity in the brain's pain circuitry would predict activation of the brain's pleasure circuitry during a subsequent opportunity to aggressively retaliate against one's rejecter. Second, that this increased neural signature of aggressive pleasure would predict increased levels of retaliatory aggression. Third, that this sequence of effects would only occur for individuals who possessed relatively impaired levels of self-regulation. To test these hypotheses, I performed a functional neuroimaging experiment

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that quantified the dynamic interactions between pain and pleasure circuitry in the brain during experiences of social rejection and retaliatory aggression, and individuals' dispositional self-regulatory abilities.

4.2 Summary of Hypothesis Tests

To provide a foundation on which to build evidence for my novel hypotheses, I first sought to replicate the association between social rejection (as operationalized by the Cyberball task) and activity in the brain's affective pain matrix, the dACC and the anterior insula (Eisenberger et al., 2003). The results were mixed. I observed robust clusters of activity in the bilateral anterior insula, yet no activation was observed in the dACC, even when an ROI approach was adopted. It is uncertain why the Cyberball task failed to elicit a response in the dACC, as this identical task has been previously used on similar types of participants (i.e., psychology undergraduates) to reliably produce clusters of activity in the dACC (Chester & DeWall, in press[b]; Chester et al., in press; Chester et al., 2014, 2015).

To test my first hypothesis, activity in the social pain matrix was correlated with NAcc activity estimates during retaliatory aggression. Contrary to my predictions, the associations were negative in sign (though only marginally significant). It appeared that the more that social rejection recruited the pain matrix, the less that individuals went on to recruit reward circuitry during retaliatory aggression. This finding stands in conflict with opponent process models of pain and pleasure, which postulate that pain sensitizes people to pleasure and vice-versa (Leknes et al., 2008). Instead, it appears that social pain and aggressive pleasure function more in a hydraulic fashion in which

activity in one system inhibits activity in the other. This may be a unique case where social and physical modalities of pain diverge and one does not resemble the other.

To test my second hypothesis, I sought to replicate the association between NAcc activity during retaliatory aggression and greater retaliatory aggression (Chester & DeWall, in press[a]). This effect was successfully replicated. Expanding upon this finding, I observed that the effect of social pain (as indexed by right anterior insula activity during social rejection) on subsequently reduced aggression was mediated by a blunted NAcc response to retaliatory aggression. This indirect effect suggests that not only do social pain and aggressive pleasure operate in a hydraulic fashion, but that this dynamism has implications for the rejection aggression link. Social pain appears to inhibit aggressive pleasure and thus, aggression itself. By reducing the rewarding and reinforcing nature of aggression, social pain may represent an avenue through which aggression can be reduced.

Using moderated mediation modeling to test my third hypothesis, I replicated the critical role of executive functioning (and trait self-control) in determining whether social pain yields aggressive or non-aggressive dividends (as in Chester et al., 2014). Across the models that I performed, neural indices of social pain were associated with lesser aggression (via reduced NAcc activity during aggression), but only among individuals that were relatively high in executive functioning and trait self-control. This replicates and extends my previous research showing that individuals high in executive functioning show a negative association between neural indices of social pain and retaliatory aggression in response to social rejection (Chester et al., 2014). Crucially, this moderated mediation model was only significant when I constrained my moderation to

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the *a* and *c* paths of the mediation model, leaving the *b* path unmoderated. Thus, it appears that executive functioning and trait self-control largely achieves their aggression-reducing effects by operating upon the effect that social pain has upon aggression outcomes. This mechanistic extension of past literature may be a crucial step in translating such findings into treatments designed to reduce aggression.

Critically, I failed to replicate my previous finding that, among individuals lower in executive functioning, social pain was associated with greater aggression (Chester et al., 2014). Further, I did not provide support for my prediction that, among individuals low in executive functioning, that social pain would be associated with greater aggressive pleasure. Indeed, it appears that the opposite is likely to be the case, that social pain blunts aggressive pleasure, which then reduces aggression.

4.3 Theoretical Implications

The findings of my dissertation have implications beyond my specific hypotheses and suggest support, lack-of-support, and revisions for certain theoretical models.

4.3.1 Social pain and aggression-reduction. The findings of the present research and previous studies (Chester et al., 2014) suggest that social pain might have a pacifying effect on rejected individuals. Yet how might social pain reduce aggression? I argue that the pain of rejection, much like physical pain, (A) interrupts psychological processing, (B) mobilizes and directs self-regulatory resources (e.g., executive functioning) to the sources of the social injury, and (C) motivates social healing.

A leading theory of physical pain functioning is the interruptive model (Eccleston & Crombez, 1999), which contends that nociceptive afferent inputs interrupt ongoing psychological processes (chiefly, attention) by having privileged access to these

executive functions. According to the interruptive model, not only does pain interrupt psychological processes, it adaptively changes them to escape the source of the injury. In line with pain overlap theory (Eisenberger & Lieberman, 2004), this interruptive ability should hold across modalities and apply to social pain. Therefore, social pain should interrupt psychological processes during and after an instance of perceived social rejection, and then mobilize and alter affective, cognitive, and motivational resources to effectively escape the harm being done by the social injury. This may explain why an individual's dispositional levels of executive functioning are so critical in determining whether social pain results in aggression, because pain can only mobilize resources to the extent that they are available. Because aggression is an approach-related behavior (e.g., Harmon-Jones & Sigelman, 2001), the avoidant escapism that pain promotes may be one reason that social pain can reduce aggression.

Recent research has shown that social pain can reliably predict prosocial outcomes (Chester et al., in press). Across several studies, both self-report and neural indices of social pain were associated with greater motivation and behavioral attempts to reconnect with one's social rejecters. This fits with a larger literature that shows how one of physical pain's chief functions is to motivate heal and other reparative behaviors (Bolles & Fanselow, 1980). Drawing from pain overlap theory (Eisenberger & Lieberman, 2004), it is likely that healing is an abstraction that can be either physical (e.g., bandaging a wound) or social (e.g., affiliating with one's rejecter) in nature. Indeed, other forms of non-physical pain such as empathic pain (i.e., the shared distress of another's injury) predict prosocial behaviors (e.g., Masten, Morelli, &

Eisenberger, 2011). This prosocial, healing motivation that social pain engenders may then result in the inhibition of aggression and the promotion of affiliation.

4.3.2 Opponent processes. An unresolved question regarding this dissertation is: how can social pain blunt aggressive pleasure when pain and pleasure usually magnify one another? Opponent process models, writ large, argue that a chronic psychological state of deviation from the body's desired state (e.g., hunger) will elicit motivation to regain homeostasis (Solomon & Corbit, 1974). The opponent process model has been a productive approach to understanding human experiences of pain, as chronic pain tends to motivate individuals to experience pain relief and can exacerbate feelings of pleasure (Leknes & Tracey, 2008). However, motivation states must play a key role in these models of pain. According to the Motivation-Decision Model of Pain (Leknes & Tracey, 2008), one's motivational state can readily modulate nociceptive activity in the nervous system. For example, wounded soldiers can ignore grave injuries when they are motivated by a threat to their comrades. Rejected individuals' motivation to regain social connections might alter their experience of social pain and transmute the experience into a behavioral tendency towards affiliation and away from aggression. Both of these behavioral tendencies would serve to promote re-inclusion and a return to social homeostasis (i.e., belonging; Baumeister & Leary, 1995).

Because of the integration of pain and pleasure through their shared instantiation in the brain (Leknes & Tracey, 2008), this critical role of motivational states is just as likely to apply to pleasure as it is to pain. As such, one's motivational state should readily gate one's experience of pleasure. If social pain places individuals in a motivational state to heal their social wounds via social reconnection, than it follows that

this volitional state should constrain the experience of pleasure. Yet how does social pain exert its potential influence on aggressive pleasure?

It is well-documented that social rejection recruits the affective pain matrix (e.g., dACC, anterior insula) and regions in the lateral prefrontal cortex task with pain regulation (e.g., VLPFC; Eisenberger, 2012). The VLPFC plays a broad role in self-regulation, primarily through the inhibition of affective states and behaviors (Aron, Robbins, & Poldrack, 2004). Using functional neuroimaging, the VLPFC has been repeatedly tied to the effective inhibition of pleasure and reward activity arising in the nucleus accumbens (Chester & DeWall, 2014; Wagner, Altman, Boswell, Kelley, & Heatherton, 2013). Due to the co-activation of the pain matrix and the VLPFC during rejection, it is possible that the VLPFC goes on to inhibit the NAcc during retaliatory aggression. Future analysis of the data collected in this dissertation will allow for a test of this hypothesis.

4.3.3 Meta-theories of aggression. The findings of this dissertation cohere with existing meta-theories of aggressive behavior. Meta-theories are so-called because they synthesize across theories and tend to be largely unfalsifiable. The General Aggression Model describes a process by which situational and person factors interact to create aggressive impulses in one's current state, which then interacts with self-regulatory processes to determine whether an aggressive impulse will result in an aggressive act (Anderson & Bushman, 2002). Couched in this model, I observed that social rejection (situation factor) interacted with dispositional levels of executive functioning (person factor) to alter participants' internal state (social pain and aggressive pleasure), which then fed into self-regulatory processes that reduced aggression. These

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findings support the utility and validity of the General Aggression Model by demonstrating how a complex model can be simply sorted into pre-defined components to refine theories and construct further hypotheses.

The I[3] meta-theory of aggression (Finkel, 2014) is a complementary process-based model, akin to the General Aggression Model. It proposes that certain behaviors such as aggression arise due to an interaction between three factors: an instigating trigger (i.e., an input that creates an aggressive impulse), an impelling force (i.e., a factor that determines the strength of the aggressive impulse), and an inhibiting force (i.e., a factor that restrains the aggressive impulse). In the context of this dissertation, I examined the interaction between social rejection (i.e., an instigating trigger), the resulting social pain (i.e., an impelling force), and executive functioning (i.e., an inhibiting force). Despite the correlational evidence that social pain is an inhibiting force rather than an impelling one, the significant moderated mediation models tested in this dissertation highlight the importance of looking at the dynamic interactions between these three types of components.

4.3.4 Aggression and positive affect. A more recent theoretical advancement in the aggression literature is the emphasis on positive affect as a cause of aggressive behavior (e.g., Chester & DeWall, in press[a]). Supporting this burgeoning approach to aggression, I replicated the effect of NAcc activity during retaliatory aggression with greater aggressive behavior in this dissertation project. This NAcc activity also positively correlated with trait aggression in my exploratory analyses. Taken together, these findings suggest that aggression is rewarding and that this reinforcing feedback from aggression may play a significant role in why individuals tend to develop stable levels of

aggressive acts. Much like an addictive behavior such as substance abuse, the

momentary, dopaminergic, positive affect that participants experience in response to

aggression may create a feedback loop that exaggerates the use of this behavior to

obtain positive affect.

Beyond the simple desire to experience positive affect, individuals may

strategically use the positive affect that is associated with retaliatory aggression. Recent

findings from my laboratory suggest that individuals use the hedonic reward of

aggression to combat the negative affect that arises from certain experiences such as

social rejection (Chester & DeWall, 2016). Although social pain indices were negatively

associated with such NAcc activity in this study, negative affect arises from a distributed

array of neural networks, and individuals may have still experienced anger, sadness, or

other forms of aversive emotions that they sought to combat with aggression's pleasant

qualities.

4.3.5 Social pain controversy. The fields of social neuroscience and social

psychology have not reached agreement on the existence of social pain or its potential

biological substrates. A chief criticism of pain overlap theory is that the presence of pain

matrix activity during social rejection does not actually represent the experience of pain

(Zaki, Wager, Singer, Keysers, & Gazzola, 2016). These critiques arise from the fact

that the dACC, the most reliable neural correlate of social pain, is also involved in a host

of other psychological processes such as expectancy violation (Somerville, Heatherton,

& Kelley, 2006), and salience (Iannetti, Salomons, Moayedi, Mouraux, & Davis, 2013).

Other results have demonstrated that social pain and physical pain, despite co-

activating the dACC, have distinct patterns of activity within this brain region that allow it

to be distinguished from each other (Woo et al., 2014). Aside from the limitations inherent in many of these studies, such as the confounding of rejection with retrospection (as in Woo et al., 2014), there are good reasons to retain the reverse inference that dACC activity during rejection subserves social pain.

These challenges to the social pain literature are exciting and have spurred substantial theoretical development. For example, the involvement of the dACC in processes beyond pain has been elegantly integrated by the conceptualization of the dACC as a neural 'alarm system' that exhibits painful distress when regulatory goals are not met (Eisenberger & Lieberman, 2004). Further, meta-analytic evidence has shown that the dACC is reliably associated with social rejection (Rotge et al., 2014) and most reliably associated with pain and less so with other processes such as salience or conflict-monitoring (Eisenberger, 2015; Lieberman & Eisenberger, 2015).

Further criticisms have been levied against the claim that the dACC is selective for pain (as argued by Lieberman & Eisenberger, 2015). Much of the evidence for the dACC's selectivity for pain takes the form of meta-analyses of search terms and their associations with BOLD activations across thousands of studies. Scholars, including the founder of one of the more popular meta-analytic fMRI databases, have argued that flawed statistical methods were used to assert this meta-analytic link between dACC activation and pain (Wager et al., 2016). Further, the authors of this critique argue that the dACC's reliable association with tasks that do not include a painful component (e.g., motor encoding) are inconsistent with the proposal that the dACC is selective for pain. In a response, Lieberman, Burns, Torre, and Eisenberger (2016) refute these claims and provide additional evidence that the dACC is selective for painful stimuli and

experiences. The concept of social pain is alive and well, as is its association with the dACC. Sadly, in this dissertation, I was unable to provide evidence for the dACC-social pain link. Though, it should be noted that failures to replicate are expected, even among 'real' effects.

  4.3.6 Extension to other causes of aggression. Social rejection is far from the only cause of aggressive behavior. Insults, hot weather, violent media, physiological arousal, alcohol, and many other inputs cause aggression (Anderson & Bushman, 2002). Because many of these experiences are painful and recruit affective pain structures in the brain such as the dACC (e.g., heat; Büchel et al., 2002), it may be that the interactions between pain during situational inputs that cause aggression and its dynamic interactions with the pleasure systems that show greater metabolic activity during aggression might be a fruitful area of inquiry for future research.Rejected individuals are not the only aggressive persons. Indeed, many people act aggressively for reasons tied to their social inclusion. For example, individuals with strong identity ties to their ingroup show an exaggerated tendency to aggress, even violently, on their behalf (Swann, Gómez, Dovidio, Hart, & Jetten, 2010). Future research is needed to understand the underlying mechanisms that explain why social acceptance can motivate aggression.

4.4 Practical Implications

  Beyond theory, the findings of my dissertation suggest some routes for the practical exercise of creating and modifying interventions that seek to reduce aggression. Firstly, my dissertation echoes decades of research by implicating the critical role of self-regulation in preventing aggressive acts (Denson, 2015; Denson,

DeWall, & Finkel, 2012; DeWall et al., 2011). Both dispositional self-regulatory abilities and those that are increased through self-control training are potent buffers of instigating and impelling forces ability to result in aggression. Interventions that increase executive functions and other substrates of self-control are likely to curtail aggressive reactions to rejection by modifying the effect that rejection's sting has on subsequent behavior.

Secondly, these findings reify the notion that aggression can be viewed in an addiction framework. By replicating the link between NAcc activity and aggression, my dissertation joins the chorus of other findings that speak to a brief moment of positive affect associated with aggressive retaliation. This momentary spike in positive affect mirrors other types of addictive behaviors such as gambling and risky sexual behavior. This addiction framework might prove informative to aggression interventions that may also benefit from approaches that are successful at reducing addictive behaviors. Such effective treatments are cognitive-behavioral therapies that train individuals to re-frame their impulses and to structure their life in a way to avoid them, mindfulness techniques that help manage the strength and severity of impulses, and social support networks. These three avenues are likely to be fruitful ways to approach aggression-reduction by managing aggressive impulses. Further, pharmacological tools that blunt the pleasure of substances , such as the opioid antagonist Naltrexone, might be an effective avenue to attempt to reduce the pleasure of aggression and therefore, aggression itself.

A third avenue through which these findings might inform intervention is to suggest benefits that may be inherent in harnessingsocial pain. Across this dissertation and my other research (Chester et al., in press[a]; Chester et al., 2014), social pain

appears robustly able to promote affiliation and reduce aggression in response to provocations like rejection. Interventions may seek to encourage at-risk individuals (e.g., narcissists; Chester & DeWall, in press[b]) to better regulate their experience of pain such that it can be used to increase prosocial outcomes. Teaching individuals to psychologically accept and embrace (and potentially communicate) the painful experience of rejection may help them effectively manage their daily provocations and slights. Further testing is needed to determine whether such an approach is possibly misguided.

4.5 The Role of the Mentalizing Network in Social Rejection

Alongside several of the expected patterns of brain activity I observed during social rejection, I observed activity in the stereotypical mentalizing network: MPFC, PCC, TPJ, and TP. Together, these four regions cooperate to allow individuals to perspective-take with others and understand the target's experiences (Frith & Frith, 2006). This 'cognitive empathy' is distinct from 'affective empathy', because the latter recruits the affective pain matrix in response to others' misfortunes (Singer et al., 2004). Given the social significance of exclusion, there are clear reasons to expect to observe the mentalizing network in this context. Activity the central hubs of this network (i.e., the MPFC and PCC) were unassociated with aggression or NAcc activity during such aggression. As such, it is uncertain whether activity in these brain regions represented an affiliative mindset or a more aggressive one. However, MPFC and PCC activity during social rejection were associated with self-reports of negative affect and self-esteem threat during social rejection. Therefore, individuals who experienced rejection as a more aversive and threatening experience also showed greater recruitment of the

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mentalizing network. This might represent a dispositional, other-attunement in which individuals who tend to recruit the mentalizing network are more negatively affected by social rejection. Future research should investigate the role of the mentalizing network during social rejection.

4.6 Limitations and Future Directions

The current research provided mixed support for my hypotheses. Several limitations of this study may have influenced the results of this dissertation and suggest avenues for future research. First among these is the fact that each of the independent variables in the study were measured and not experimentally manipulated (e.g., brain activity, executive functioning). Thus, all inferences from the moderated mediation model cannot be causal and were purely correlational. Such correlational research is often appropriate when it is an initial foray into an area of empirical ambiguity. However, future research can leverage experimental manipulations of social pain and aggressive pleasure to buttress the claims of this dissertation through methods such as drug administration, brain stimulation, and behavioral interventions such as mindfulness training.

Second, my sample size of 60 participants, while large for a neuroimaging study, prevented an adequate level of statistical power for my tests of moderated mediation. Therefore, I may have detected a spurious effect due to the higher Type 1 error rates inherent in underpowered designs. Further, I may have been unable to observe my predicted indirect effects among individuals low in executive functioning because my dissertation was not well powered enough to detect them, a Type II error.

Third, I relied on reverse inference to claim that dACC and anterior insula activity during rejection was a signature of 'social pain' and that NAcc activity during aggression was a signature of 'aggressive pleasure'. Reverse inference, the practice of inferring a given psychological process from a pattern of brain activity, is a potential issue as a pattern of neural activity can represent a host of psychological processes (Poldrack, 2006). It should be noted that almost all research suffers from issues of reverse inference (e.g., inferring the presence of extraversion from a high score on an extraversion questionnaire). As covered previously, a wealth of evidence exists to support the claim that dACC and anterior insula activity is indicative of pain and not other processes such as conflict monitoring (Eisenberger, 2012, 2015; Lieberman & Eisenberger, 2015) and that NAcc activity is symbolic of reward and not another mental state such as learning (Bartra, McGuire, & Kable, 2013). To further assist these reverse inferences, I included self-report measures of social pain and aggressive pleasure. However, these self-report measures did not significantly correlate with their corresponding patters of neural activity, for the most part. Therefore, the reverse inferences I have made must be qualified by this lack of supporting evidence.

4.7 Conclusions

Does the pain of rejection promote the pleasure of revenge? The findings of my dissertation do not provide evidence for such a possibility. Instead it appears that the story is a little more complicated. In fact, I observed evidence that the opposite is true, that the pain of rejection blunts the pleasure of revenge. This effect appears to arise from individuals who possess the capability to effectively regulate their feelings and behaviors. These results painted a far more hopeful vision of the underlying mechanics

of the rejection-aggression link than I had expected. My dissertation suggests that by harnessing self-regulatory forces and the pain of rejection, people might be able to reduce aggressive responses to social rejection. This possibility underscores the need for psychological and neuroscientific investigations of societal ills such as violence, that we may better understand them and one day, be rid of them.

Appendix A

McGill Pain Questionnaire – Short Form

The word below describes a type of pain. Please rate the intensity with which you were experiencing this type of DURING THE CYBERBALL GAME.

0 – Severe 1 – Mild 2 – Moderate 3 – Severe

1. Throbbing
2. Shooting
3. Stabbing
4. Sharp
5. Cramping
6. Gnawing
7. Hot-Burning
8. Aching
9. Heavy
10. Tender
11. Splitting
12. Tiring-Exhausting
13. Sickening
14. Fearful
15. Punishing-Cruel

Appendix B

Need Threat Scale

For each question, please choose the number that best represent the feelings you were experiencing DURING THE CYBERBALL GAME, from 1 (Not At All) to 5 (Extremely).

1. I felt "disconnected"
2. I felt rejected
3. I felt like an outsider
4. I felt I belonged to the group
5. I felt the other players interacted with me a lot
6. I felt good about myself
7. My self-esteem was high
8. I felt liked
9. I felt insecure
10. I felt satisfied
11. I felt invisible
12. I felt meaningless
13. I felt non-existent
14. I felt important
15. I felt useful
16. I felt powerful
17. I felt I had control over the course of the game
18. I felt I had the ability to significantly alter events
19. I felt I was unable to influence the actions of others
20. I felt the other players decided everything
21. I was ignored
22. I was excluded
23. Good
24. Bad
25. Friendly
26. Unfriendly
27. Angry
28. Pleasant
29. Happy
30. Sad
31. What percentage of throws did you receive? (type a number between 0 and 100)

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Appendix C

Aggressive Pleasure Scale

When you think back to the noise blasts that you gave to your partner, how did they make you feel? Please pick a number from 1 (Not At All) to 5 (Extremely).

1. Good
2. Friendly
3. Pleasant
4. Happy

Appendix D

Short Sadistic Impulse Scale

Please use the scale below to indicate the extent to which you agree or disagree with each of the following statements. Please pick a number from 1 (Strongly Disagree) to 7 (Strongly Agree).

1. I enjoy seeing people hurt.
2. I would enjoy hurting someone physically, sexually, or emotionally.
3. Hurting people would be exciting.
4. I have hurt people for my own enjoyment.
5. People would enjoy hurting others if they gave it a go.
6. I have fantasies which involve hurting people.
7. I have hurt people because I could.
8. I wouldn't intentionally hurt anyone.
9. I have humiliated others to keep them in line.
10. Sometimes I get so angry I want to hurt people.

Appendix E

Brief Aggression Questionnaire

Please use the scale below to indicate the extent to which you agree or disagree with each of the following statements. Please pick a number from 1 (Strongly Disagree) to 7 (Strongly Agree).

1. Given enough provocation, I may hit another person.
2. If I have to resort to violence to protect my rights, I will.
3. There are people who pushed me so far that we came to blows.
4. I tell my friends openly when I disagree with them.
5. When people annoy me, I may tell them what I think of them.
6. My friends say that I'm somewhat argumentative.
7. I am an even-tempered person.
8. Sometimes I fly off the handle for no good reason.
9. I have trouble controlling my temper.
10. Other people always seem to get the breaks.
11. I sometimes feel that people are laughing at me behind my back.
12. When people are especially nice, I wonder what they want.

Appendix F

Brief Self-Control Scale

Please use the scale below to indicate the extent to which you agree or disagree with each of the following statements. Please pick a number from 1 (Not at all like me) to 5 (Very much like me).

1. I refuse things that are bad for me.
2. I am lazy.
3. I say inappropriate things.
4. I do certain things that are bad for me, if they are fun.
5. I have trouble concentrating.
6. I often act without thinking through all the alternatives.
7. I am good at resisting temptation.
8. People would say that I have iron self-discipline.
9. Pleasure and fun sometimes keep me from getting work done.
10. I have a hard time breaking bad habits.
11. I am able to work effectively toward long-term goals.
12. Sometimes I can't stop myself from doing something, even if I know it is wrong.
13. I wish I had more self-discipline.

References

Anderson, C. A., & Bushman, B. J. (2002). Human Aggression. *Annual Review of Psychology, 53*(1), 27–51.

Bartra, O., McGuire, J. T., & Kable, J. W. (2013). The valuation system: A coordinate-based meta-analysis of BOLD fMRI experiments examining neural correlates of subjective value. *NeuroImage, 76*, 412–427.

Bastian, B., Jetten, J., Hornsey, M. J., & Leknes, S. (2014). The Positive Consequences of Pain A Biopsychosocial Approach. *Personality and Social Psychology Review, 18*(3), 256–279.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin, 117*(3), 497–529.

Berkowitz, L. (1989). Affective aggression: The role of stress, pain, and negative affect. In R. G. Geen & E. Donnerstein (Eds.), *Human aggression: Theories, research, and implications for social policy* (pp. 49–72). San Diego: Academic Press.

Berridge, K. C., & Kringelbach, M. L. (2013). Neuroscience of affect: brain mechanisms of pleasure and displeasure. *Current Opinion in Neurobiology, 23*(3), 294–303.

Bolles, R. C., & Fanselow, M. S. (1980). A perceptual-defensive-recuperative model of fear and pain. *Behavioral and Brain Sciences, 3*(02), 291–301.

Büchel, C., Bornhövd, K., Quante, M., Glauche, V., Bromm, B., & Weiller, C. (2002). Dissociable Neural Responses Related to Pain Intensity, Stimulus Intensity, and Stimulus Awareness within the Anterior Cingulate Cortex: A Parametric Single-Trial Laser Functional Magnetic Resonance Imaging Study. *The Journal of Neuroscience, 22*(3), 970–976.

Buckley, K. E., Winkel, R. E., & Leary, M. R. (2004). Reactions to acceptance and rejection: Effects of level and sequence of relational evaluation. *Journal of Experimental Social Psychology, 40*(1), 14–28.

Bushman, B. J. (2002). Does Venting Anger Feed or Extinguish the Flame? Catharsis, Rumination, Distraction, Anger, and Aggressive Responding. *Personality and Social Psychology Bulletin, 28*(6), 724–731.

Bushman, B. J., Baumeister, R. F., & Phillips, C. M. (2001). Do people aggress to improve their mood? Catharsis beliefs, affect regulation opportunity, and aggressive responding. *Journal of Personality and Social Psychology, 81*(1), 17–32.

Bushman, B. J., Baumeister, R. F., & Stack, A. D. (1999). Catharsis, aggression, and persuasive influence: Self-fulfilling or self-defeating prophecies? *Journal of Personality and Social Psychology, 76*(3), 367–376.

Bushman, B. J., & Huesmann, L. R. (2010). Aggression. In S. T. Fiske, D. T. Gilbert, & G. Lindzey (Eds.), *Handbook of social psychology (Vol. 2).* New York, NY: John Wiley & Sons, Inc.

Carré, J. M., Gilchrist, J. D., Morrissey, M. D., & McCormick, C. M. (2010). Motivational and situational factors and the relationship between testosterone dynamics and human aggression during competition. *Biological Psychology, 84*(2), 346–353.

Chen, Z., Poon, K.-T., Bernstein, M. J., & Teng, F. (2014). Rejecting another pains the self: The impact of perceived future rejection. *Journal of Experimental Social Psychology*, *50*, 225–233.

Chester, D. S. & DeWall, C. N. (in press)[a]. The pleasure of revenge: Retaliatory aggression arises from a neural imbalance toward reward. *Social Cognitive and Affective Neuroscience*.

Chester, D. S. & DeWall, C. N. (in press)[b]. Sound the alarm: The effect of narcissism on retaliation is moderated by dACC reactivity to rejection. *Journal of Personality*.

Chester, D. S. & DeWall, C. N. (2016). Combating the Sting of Exclusion with the Pleasure of Revenge: A New Look at How Emotion Shapes Aggressive Responses to Rejection. *Manuscript in preparation*.

Chester, D. S., DeWall, C. N., & Pond, R. S. (in press). The push of social pain: Does rejection's sting motivate subsequent social reconnection? *Cognitive, Affective, and Behavioral Neuroscience.*

Chester, D. S., Pond, R. S., & DeWall, C. N. (2015). Alexithymia is associated with blunted anterior cingulate response to social rejection: Implications for daily rejection. *Social Cognitive and Affective Neuroscience, 10*(4), 517-522.

Chester, D. S., Eisenberger, N. I., Pond, R. S., Richman, S. B., Bushman, B. J., & DeWall, C. N. (2014). The interactive effect of social pain and executive functioning on aggression: An fMRI experiment. *Social Cognitive and Affective Neuroscience, 9*(5), 699-704.

Chow, R. M., Tiedens, L. Z., & Govan, C. L. (2008). Excluded emotions: The role of anger in antisocial responses to ostracism. *Journal of Experimental Social Psychology*, *44*(3), 896–903.

Damasio, A. R., Everitt, B. J., & Bishop, D. (1996). The Somatic Marker Hypothesis and the Possible Functions of the Prefrontal Cortex [and Discussion]. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *351*(1346), 1413–1420.

Denson, T. F. (2015). Four promising psychological interventions for reducing reactive aggression. *Current Opinion in Behavioral Sciences*, *3*, 136–141.

DeWall, C. N., Anderson, C. A., & Bushman, B. J. (2011). The general aggression model: Theoretical extensions to violence. *Psychology of Violence*, *1*(3), 245–258.

Denson, T. F., DeWall, C. N., & Finkel, E. J. (2012). Self-Control and Aggression. *Current Directions in Psychological Science*, *21*(1), 20–25.

DeWall, C. N., Finkel, E. J., & Denson, T. F. (2011). Self-Control Inhibits Aggression. *Social and Personality Psychology Compass*, *5*(7), 458–472.

DeWall, C. N., Twenge, J. M., Gitter, S. A., & Baumeister, R. F. (2009). It's the thought that counts: The role of hostile cognition in shaping aggressive responses to social exclusion. *Journal of Personality and Social Psychology*, *96*(1), 45–59.

Eccleston, C., & Crombez, G. (1999). Pain demands attention: A cognitive–affective model of the interruptive function of pain. *Psychological Bulletin*, *125*(3), 356–366.

Eisenberger, N. I. (2012). The pain of social disconnection: examining the shared neural underpinnings of physical and social pain. *Nature Reviews Neuroscience, 13*(6), 421-434.

Eisenberger, N. I. (2015). Social pain and the brain: Controversies, questions, and where to go from here. *Annual Review of Psychology*, *66*(1), 601–629.

Eisenberger, N. I., & Lieberman, M. D. (2004). Why rejection hurts: a common neural alarm system for physical and social pain. *Trends in Cognitive Sciences*, *8*(7), 294–300.

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science, 302*(5643), 290-292.

Emmerling, F., Schuhmann, T., Lobbestael, J., Arntz, A., Brugman, S., & Sack, A. T. (in press). The role of the insular cortex in retaliation. PLoS ONE.

Engle, R. W. (2002). Working Memory Capacity as Executive Attention. *Current Directions in Psychological Science*, *11*(1), 19–23.

Fields, H. L. (2007). Understanding How Opioids Contribute to Reward and Analgesia. *Regional Anesthesia and Pain Medicine*, *32*(3), 242–246.

Finkel, E. J. (in press). The I³ Model: Metatheory, theory, and evidence. In J. M. Olson, & M. P. Zanna (Eds.), *Advances in experimental social psychology*, *49*. San Diego: Academic Press.

Foltz, E. L., & White, L. E. (1962). Pain "relief" by frontal cingulumotomy. *Journal of Neurosurgery*, *19*, 89–100.

Freud, S., & Freud, E. L. (1960). *Letters of Sigmund Freud*. Courier Corporation.

Frith, C. D., & Frith, U. (2006). The Neural Basis of Mentalizing. *Neuron*, *50*(4), 531–534.

Giancola, P. R., & Chermack, S. T. (1998). Construct validity of laboratory aggression paradigms: A response to tedeschi and quigley (1996). *Aggression and Violent Behavior*, *3*(3), 237–253.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology*, *80*(5), 797–803.

Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling. Retrieved from http://www.afhayes.com/public/process2012.pdf

Heatherton, T. F., & Wagner, D. D. (2011). Cognitive neuroscience of self-regulation failure. *Trends in Cognitive Sciences*, *15*(3), 132–139.

Heller, R., Stanley, D., Yekutieli, D., Rubin, N., & Benjamini, Y. (2006). Cluster-based analysis of fMRI data. *NeuroImage*, *33*(2), 599–608.

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences*, *16*(3), 174–180.

Iannetti, G. D., Salomons, T. V., Moayedi, M., Mouraux, A., & Davis, K. D. (2013). Beyond metaphor: contrasting mechanisms of social and physical pain. *Trends in Cognitive Sciences*, *17*(8), 371–378.

Kawamoto, T., Onoda, K., Nakashima, K., Nittono, H., Yamaguchi, S., & Ura, M. (2012). Is dorsal anterior cingulate cortex activation in response to social exclusion due

to expectancy violation? An fMRI study. *Frontiers in Evolutionary Neuroscience*, *4,* 1-10.

Krämer, U. M., Jansma, H., Tempelmann, C., & Münte, T. F. (2007). Tit-for-tat: The neural basis of reactive aggression. *NeuroImage*, *38*(1), 203–211.

Kross, E., Berman, M. G., Mischel, W., Smith, E. E., & Wager, T. D. (2011). Social rejection shares somatosensory representations with physical pain. *Proceedings of the National Academy of Sciences, 108*(15), 6270-6275.

Leary, M. R., Kowalski, R. M., Smith, L., & Phillips, S. (2003). Teasing, rejection, and violence: Case studies of the school shootings. *Aggressive Behavior*, *29*(3), 202–214.

Leary, M. R., Twenge, J. M., & Quinlivan, E. (2006). Interpersonal Rejection as a Determinant of Anger and Aggression. *Personality and Social Psychology Review*, *10*(2), 111–132.

Leknes, S., Berna, C., Lee, M. C., Snyder, G. D., Biele, G., & Tracey, I. (2013). The importance of context: when relative relief renders pain pleasant. *PAIN*, *154*(3), 402–410.

Leknes, S., Brooks, J. C. W., Wiech, K., & Tracey, I. (2008). Pain relief as an opponent process: a psychophysical investigation. *European Journal of Neuroscience*, *28*(4), 794–801.

Leknes, S., Lee, M., Berna, C., Andersson, J., & Tracey, I. (2011). Relief as a Reward: Hedonic and Neural Responses to Safety from Pain. *PLoS ONE, 6*(4), e17870.

Leknes, S., & Tracey, I. (2008). A common neurobiology for pain and pleasure. *Nature Reviews Neuroscience*, *9*(4), 314–320.

Lieberman, M. D., Burns, S. M., Torre, J. B., & Eisenberger, N. I. (2016). Reply to Wager et al.: Pain and the dACC: The importance of hit rate-adjusted effects and posterior probabilities with fair priors. *Proceedings of the National Academy of Sciences*, 201603186.

Lieberman, M. D., & Eisenberger, N. I. (2015). The dorsal anterior cingulate cortex is selective for pain: Results from large-scale reverse inference. *Proceedings of the National Academy of Sciences*, 201515083.

MacDonald, G., & Leary, M. R. (2005). Why Does Social Exclusion Hurt? The Relationship Between Social and Physical Pain. *Psychological Bulletin, 131*(2), 202–223.

Maldjian, J. A., Laurienti, P. J., Kraft, R. A., & Burdette, J. H. (2003). An automated method for neuroanatomic and cytoarchitectonic atlas-based interrogation of fMRI data sets. *NeuroImage*, *19*(3), 1233–1239.

Masten, C. L., Morelli, S. A., & Eisenberger, N. I. (2011). An fMRI investigation of empathy for "social pain" and subsequent prosocial behavior. *NeuroImage*, *55*(1), 381–388.

Melzack, R. (1987). The short-form McGill pain questionnaire. *Pain*, *30*(2), 191–197.

Miller, N., Pedersen, W. C., Earleywine, M., & Pollock, V. E. (2003). A Theoretical Model of Triggered Displaced Aggression. *Personality and Social Psychology Review*, *7*(1), 75–97.

O'Meara, A., Davies, J., & Hammond, S. (2011). The psychometric properties and utility of the Short Sadistic Impulse Scale (SSIS). *Psychological Assessment, 23*(2), 523–531.

Panksepp, J. (2011). The basic emotional circuits of mammalian brains: do animals have affective lives?. *Neuroscience & Biobehavioral Reviews, 35*(9), 1791-1804.

Panksepp, J., Vilberg, T., Bean, N. J., Coy, D. H., & Kastin, A. J. (1978). Reduction of distress vocalization in chicks by opiate-like peptides. *Brain Research Bulletin, 3*(6), 663-667.

Poldrack, R. A. (2006). Can cognitive processes be inferred from neuroimaging data? *Trends in Cognitive Sciences, 10*(2), 59–63.

Ramírez, J. M., Bonniot-Cabanac, M.-C., & Cabanac, M. (2005). Can Aggression Provide Pleasure? *European Psychologist, 10*(2), 136–145.

Reijntjes, A., Thomaes, S., Kamphuis, J. H., Bushman, B. J., Castro, B. O. de, & Telch, M. J. (2011). Explaining the Paradoxical Rejection-Aggression Link: The Mediating Effects of Hostile Intent Attributions, Anger, and Decreases in State Self-Esteem on Peer Rejection-Induced Aggression in Youth. *Personality and Social Psychology Bulletin, 37*(7), 955–963.

Riva, P., Lauro, L. J. R., DeWall, C. N., Chester, D. S., & Bushman, B. J. (2014). Reducing aggressive responses to social exclusion using transcranial direct current stimulation (tDCS). *Social Cognitive and Affective Neuroscience, 53*.

Rotge, J. Y., Lemogne, C., Hinfray, S., Huguet, P., Grynszpan, O., Tartour, E., … Fossati, P. (2015). A meta-analysis of the anterior cingulate contribution to social pain. *Social Cognitive and Affective Neuroscience, 10*(1), 19–27.

Silverman, S. M. (2009). Opioid induced hyperalgesia: clinical implications for the pain practitioner. *Pain Physician, 12*(3), 679–684.

Singer, T., Seymour, B., O'Doherty, J., Kaube, H., Dolan, R. J., & Frith, C. D. (2004). Empathy for Pain Involves the Affective but not Sensory Components of Pain. *Science, 303*(5661), 1157–1162.

Smith, S. M., Jenkinson, M., Woolrich, M. W., Beckmann, C. F., Behrens, T. E. J., Johansen-Berg, H., … Matthews, P. M. (2004). Advances in functional and structural MR image analysis and implementation as FSL. *NeuroImage, 23*(1), S208–S219.

Solomon, R. L., & Corbit, J. D. (1974). An opponent-process theory of motivation: I. Temporal dynamics of affect. *Psychological Review, 81*(2), 119–145.

Somerville, L. H., Heatherton, T. F., & Kelley, W. M. (2006). Anterior cingulate cortex responds differentially to expectancy violation and social rejection. *Nature Neuroscience, 9*(8), 1007–1008.

Sprenger, T., Valet, M., Boecker, H., Henriksen, G., Spilker, M. E., Willoch, F., … Tölle, T. R. (2006). Opioidergic activation in the medial pain system after heat pain. *PAIN, 122*(1–2), 63–67.

Stroop, R. J. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*(6), 643–662.

Swann, W. B., Gómez, Á., Dovidio, J. F., Hart, S., & Jetten, J. (2010). Dying and Killing for One's Group Identity Fusion Moderates Responses to Intergroup Versions of the Trolley Problem. *Psychological Science, 21*(8), 1176–1183.

Tangney, J. P., Baumeister, R. F., & Boone, A. L. (2004). High Self-Control Predicts Good Adjustment, Less Pathology, Better Grades, and Interpersonal Success. *Journal of Personality, 72*(2), 271–324.

Taylor, S. (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. *Journal of Personality, 35*, 297-310.

Twenge, J. M., Baumeister, R. F., Tice, D. M., & Stucke, T. S. (2001). If you can't join them, beat them: Effects of social exclusion on aggressive behavior. *Journal of Personality and Social Psychology, 81*(6), 1058–1069.

Wager, T. D., Atlas, L. Y., Botvinick, M. M., Chang, L. J., Coghill, R. C., Davis, K. D., … Yarkoni, T. (2016). Pain in the ACC? *Proceedings of the National Academy of Sciences*, 201600282.

Wagner, D. D., Altman, M., Boswell, R. G., Kelley, W. M., & Heatherton, T. F. (2013). Self-Regulatory Depletion Enhances Neural Responses to Rewards and Impairs Top-Down Control. *Psychological Science, 24*(11), 2262–2271.

Warburton, W. A., Williams, K. D., & Cairns, D. R. (2006). When ostracism leads to aggression: The moderating effects of control deprivation. *Journal of Experimental Social Psychology, 42*(2), 213–220.

Webster, G. D., DeWall, C. N., Pond, R. S., Deckman, T., Jonason, P. K., Le, B. M., … Bator, R. J. (2014). The brief aggression questionnaire: psychometric and behavioral evidence for an efficient measure of trait aggression. *Aggressive Behavior, 40*(2), 120–139.

Wesselmann, E. D., Butler, F. A., Williams, K. D., & Pickett, C. L. (2010). Adding injury to insult: unexpected rejection leads to more aggressive responses. *Aggressive Behavior, 36*(4), 232–237.

Williams, K. D. (2009). Ostracism: A temporal need-threat model. In Mark P. Zanna (Ed.), *Advances in Experimental Social Psychology* (Vol. 41, pp. 275–314). Academic Press.

Williams, K. D., Cheung, C. T, K., & Choi, W. (2000). Cyberostracism: Effects of being ignored over the Internet. *Journal of Personality and Social Psychology, 79*(5), 748–762.

Woo, C.-W., Koban, L., Kross, E., Lindquist, M. A., Banich, M. T., Ruzic, L., … Wager, T. D. (2014). Separate neural representations for physical pain and social rejection. *Nature Communications, 5*, 5380.

Woolrich, M. W., Jbabdi, S., Patenaude, B., Chappell, M., Makni, S., Behrens, T., … Smith, S. M. (2009). Bayesian analysis of neuroimaging data in FSL. *NeuroImage, 45*(1), S173–S186.

Worsley, K. J. (2001). Statistical analysis of activation images. *Functional MRI: an introduction to methods, 14*, 251-270.

Yin, H. H., & Knowlton, B. J. (2006). The role of the basal ganglia in habit formation. *Nature Reviews Neuroscience, 7*(6), 464–476.

Zaki, J., Wager, T. D., Singer, T., Keysers, C., & Gazzola, V. (2016). The Anatomy of Suffering: Understanding the Relationship between Nociceptive and Empathic Pain. *Trends in Cognitive Sciences, 20*(4), 249–259.

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Educational Institutions and Degrees Already Awarded
    Warren Wilson College, B.A., 2008
    University of Kentucky, M.S., 2013

Professional Positions Held
    Lab Manager, University of Michigan, 2008 - 2010
    Lab Manager, University of Texas at Austin, 2010 - 2011
    Lab Instructor, University of Kentucky, 2011 - 2012
    Graduate Research Assistant, University of Kentucky, 2012 - 2015

Scholastic and Professional Honors
    Dissertation Year Fellow, University of Kentucky, 2015
    Dissertation Research Award, Heritage Foundation for Personality and Social
    Psychology, 2015
    Samuel Gunto Award, University of Kentucky, 2013, 2014
    Outstanding Research Award, Society for Personality and Social Psychology,
    2012
    Honorable Mention, National Science Foundation Graduate Research Fellowship
    Program, 2012

Professional Publications
    Pearson, C. M., Chester, D. S., Powell, D. K., Wonderlich, S. A., & Smith, G. T.
        (in press). Investigating the reinforcing value of binge anticipation.
        International Journal of Eating Disorders.
    Chester, D. S. & Riva, P. (in press). Brain mechanisms to regulate negative
        reactions to social exclusion. In P. Riva & J. Eck (Eds.), *Social exclusion:
        psychological approaches to understanding and reducing its impact*.
        Cham, Switzerland: Springer International Publishing.
    Chester, D. S., DeWall, C. N., & Pond, R. S. (in press). The push of social pain:
        Does rejection's sting motivate subsequent social reconnection?
        Cognitive, Affective, and Behavioral Neuroscience.
    Chester, D. S., DeWall, C. N., Derefinko, K. J., Estus, S., Lynam, D. R., Peters,
        J. R., & Jiang, Y. (in press). Looking for reward in all the wrong places:
        Dopamine receptor polymorphisms correlate with aggression through
        increased sensation-seeking. *Social Neuroscience.*
    Chester, D. S. & DeWall, C. N. (in press). The pleasure of revenge: Retaliatory
        aggression arises from a neural imbalance toward reward. *Social
        Cognitive and Affective Neuroscience.*

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Chester, D. S., Lynam, D. R., Powell, D. K., & DeWall, C. N. (in press). Narcissism is associated with weakened frontostriatal connectivity: A DTI study. *Social Cognitive and Affective Neuroscience.*

Chester, D. S. & DeWall, C. N. (in press). Sound the alarm: The effect of narcissism on retaliation is moderated by dACC reactivity to rejection. *Journal of Personality.*

DeWall, C. N., Baumeister, R. F., Chester, D. S., & Bushman, B. J. (in press). How often does currently felt emotion predict social behavior and judgment? A meta-analytic test of two competing theories. *Emotion Review.*

DeWall, C. N. & Chester, D. S. (in press). Taming the impulsive beast: Understanding the link between self-regulation and aggression. In E. Hirt (Ed.), *Self-regulation and ego control.* San Diego, CA: Elsevier.

Chester, D. S., Lynam, D. R., Milich, R., & DeWall, C. N. (in press). Craving versus control: Negative urgency and neural correlates of alcohol reactivity. *Drug and Alcohol Dependence.*

Chester, D. S. & DeWall, C. N. (in press). Adaptations to avoid ostracism. In T. Shackelford & V. Weekes-Shackelford (Eds.), *Encyclopedia of evolutionary psychological science.* New York, NY: Springer.

Chester, D. S., Lynam, D. R., Milich, R., Powell, D. K., Andersen, A.H., & DeWall, C. N. (2016). How do negative emotions impair self-control? A neural model of negative urgency. *NeuroImage, 132*, 43-50.

Chester, D. S., Merwin, L. M., & DeWall, C. N. (2015). Maladaptive perfectionism's link to aggression and self-harm: Emotion regulation as a mechanism. *Aggressive Behavior, 41*(5), 443-454.

Chester, D. S., Pond, R. S., & DeWall, C. N. (2015). Alexithymia is associated with blunted anterior cingulate response to social rejection: Implications for daily rejection. *Social Cognitive and Affective Neuroscience, 10*(4), 517-522.

Riva, P., Romero-Lauro, L. J., DeWall, C. N., Chester, D. S., & Bushman, B. J. (2015). Reducing aggressive responses to social exclusion using transcranial direct current stimulation (tDCS). *Social Cognitive and Affective Neuroscience, 10*(3), 352-356.

Chester, D. S., DeWall, C. N., Derefinko, K. J., Estus, S., Peters, J. R., Lynam, D. R., & Jiang, Y. (2015). Monoamine oxidase A (MAOA) genotype predicts greater aggression through impulsive reactivity to negative affect. *Behavioural Brain Research, 283*, 97-101.

DeWall, C. N., Chester, D. S., & White, D. S. (2015). Can acetaminophen reduce the pain of decision-making? *Journal of Experimental Social Psychology, 56,* 117-120.

Chester, D. S. & DeWall, C. N. (2014). Prefrontal recruitment during social rejection predicts greater subsequent self-regulatory imbalance and impairment: Neural and longitudinal evidence. *NeuroImage, 101*, 485-493.

Chester, D. S., Eisenberger, N. I., Pond, R. S., Richman, S. B., Bushman, B. J., & DeWall, C. N. (2014). The interactive effect of social pain and executive

functioning on aggression: An fMRI experiment. *Social Cognitive and Affective Neuroscience, 9*(5)*,* 699-704*.*

DeWall, C. N. & Chester, D. S. (2014). The neurobiology of aggression: Looking underneath the hood. In M. DeLisi & M.G. Vaughn (Eds.), *Routledge handbook of biosocial criminology* (pp. 251 - 262). New York, NY: Routledge.

Pond, R. S., Richman, S. B., Chester, D. S., & DeWall, C. N. (2014). Social pain and the brain: How insights from neuroimaging advance the study of social rejection. In T.D. Papageorgiou, G. Christopolous, & S. Smirnakis (Eds.), *Advanced brain neuroimaging topics in health and disease: methods and applications.* New York, NY: InTech.

Chester, D. S., Powell, C. A. J., Smith, R. H., Joseph, J. E., Kedia, G., Combs, D. J. Y., & DeWall, C. N. (2013). Justice for the average Joe: The role of envy and the mentalizing network in the deservingness of others' misfortunes. *Social Neuroscience, 8*(6), 640-649.

Beer, J. S., Chester, D. S., & Hughes, B. L. (2013). Social threat and cognitive load magnify self-enhancement and attenuate self-deprecation. *Journal of Experimental Social Psychology, 49*(4), 706-711.

Chester, D. S., & DeWall, C. N. (2013). Psychopathology and susceptibility to violence. In M.S. Eastin (Ed.), *Encyclopedia of media violence* (pp. 296-299)*.* Thousand Oaks, CA: Sage.

Chester, D. S., & DeWall, C. N. (2013). Trait aggression. In M.S. Eastin (Ed.), *Encyclopedia of media violence* (pp. 352-356)*.* Thousand Oaks, CA: Sage.

Chester, D. S., Pond, R. S., & DeWall, C. N. (2013). The evolution of argument: A commentary on Mercier. In P.H. Crowley & T.R. Zentall (Eds.), *Comparative decision making* (pp. 316-318). New York, NY: Oxford University Press.

Chester, D. S., Pond, R. S., Richman, S. B., & DeWall, C. N. (2012). The optimal calibration hypothesis: How life history modulates the brain's social pain network. *Frontiers in Evolutionary Neuroscience, 4*, 10.

Chester, D. S., & Burleson, K. A. P. (2009). Xbox as therapy? An experimental investigation into persuasion, catharsis, and violent video games. *Undergraduate Research Journal for the Human Sciences, 8*.

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