BIOLOGICAL BASES OF SOCIAL AGGRESSION: SEX, GENES, PUBERTY, AND NEURAL FUNCTIONING

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ABSTRACT

Social aggression (e.g., gossiping, ostracism, and threatening to end a friendship) is a form of antisocial behavior that puts both the victim and perpetrator at increased risk for mental illness and socio-emotional suffering. Social aggression is perpetrated across the lifespan, but our understanding of its developmental origins remain limited, in part because the biological factors involved remain understudied. Furthermore, sex differences in its perpetration, which has remained a primary area of investigation since the field's inception, remain inconclusive, in part due to key moderating factors not being considered.

Therefore, the purpose of this dissertation was to identify and integrate distinct biological risk factors (e.g., sex, genes, pubertal development, and neurological processes) to explore whether their interactions can help resolve extant inconsistencies in the literature regarding the development and presentation of social aggression. We capitalized on a unique longitudinal twin study of emotional and behavioral development that consists of both childhood and adolescent waves of data collection.

First, we took a behavioral genetic approach to examining the roles of sex, genes, and puberty on the etiology of social aggression. One of the most striking findings from this investigation was that although univariate results were consistent with prior research suggesting that there were no sex differences in the etiology of social aggression, a two-moderator GxE model indicated that there were indeed sex differences in the etiology of social aggression once we adjusted for the effects of puberty. Not only were genetic influences on social aggression were stronger for boys than for girls and non-shared environmental influences were stronger for girls than for boys once we adjusted for puberty, but they were also jointly moderated by sex and

puberty together such that genetic influences more than doubled from pre-puberty to puberty in girls but not boys.

Second, we took a neuroscience approach to examining the roles of sex and pubertal development on the association between social aggression and neural functioning. Specifically, we examined the association between social aggression and amygdala reactivity during a socio-emotional face processing task. Although we anticipated finding that social aggression was associated with increased amygdala reactivity, we observed that social aggression was not associated with amygdala reactivity during this task, even after controlling for sex, age, puberty, and their interactions. Although we did not observe associations between neural activation and social aggression, neuroscience studies remain a promising area of future investigation.

Taken together, the analyses conducted in this dissertation emphasize the importance of taking a developmental psychopathology approach to studying social aggression. The defining features of developmental psychopathology include investigating psychopathology developmentally, across all relevant levels of analysis, and consideration of the interactions between puberty and sex did indeed led to new insights into the etiology of social aggression. Future research should continue to employ longitudinal and person-centered approaches to investigate social and biological risk factors and processes implicated in social aggression across time.

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CHAPTER 1: INTRODUCTION

INTRODUCTION

Defining social aggression

Social aggression is a form of antisocial behavior (Burt et al., 2012) in which social relationships and social status are used to damage reputations and inflict emotional harm on others, and includes behaviors such as gossiping, ostracism, and threatening to end a friendship. Numerous theoretical models and traditions have guided the study of these behaviors over the years. As a consequence, different terms have been used by various researchers to refer to behaviors conceptually similar to social aggression, including relational aggression (e.g., Crick & Grotpeter, 1995) and indirect aggression (e.g., Feshbach, 1969; Björkqvist et al., 1992). Social aggression was initially narrowly defined as the manipulation of group acceptance through alienation, ostracism, or character defamation (Cairns et al., 1989). Unlike indirect aggression (i.e., verbally or physically aggressive behaviors in which it is difficult to identify the aggressor), which was thought to capture both dyadic and group-level behaviors, social aggression was originally limited to group-level transgressions. Because of this, it was theorized that social aggression was not perpetrated by younger children because peer networks and social status do not become salient to most children until late childhood or early adolescence (Cairns et al., 1989). The definition of social aggression was later expanded to include dyadic relationship manipulation and more subtle nonverbal aggressive behaviors that were believed to be typical of girls' peer interactions, including eye rolling, negative facial expressions, and tossing of hair (Galen & Underwood, 1997).

More recently, Crick and Grotpeter (1995) coined the term relational aggression, which was defined as the purposeful manipulation and damage of peer relationships, such as harming

friendships and feelings of exclusion. Unlike indirect aggression, the goal of these behaviors is to use interpersonal relationships to harm others rather than to avoid detection or retribution. Like indirect and social aggression, relational aggression was also conceptualized as, and observed to be, more common among girls than boys. Specifically, girls were hypothesized to utilize relational over physical aggression because it is consistent with the focus on interpersonal relationships and social functioning that particularly characterizes female peer groups relative to male peer groups.

Critically, however, research has shown that the behaviors assessed by these three terms overlap substantially (Archer & Coyne, 2005). Therefore, except when referencing a specific study or theory, the current investigation uses the term social aggression because it encompasses both overt and covert behaviors, verbal and nonverbal behaviors, and involves both dyadic and group-level transgressions (Cairns et al., 1989; Galen & Underwood, 1997).

Sex differences in social aggression

Non-physical or social aggression was conceptualized relatively recently, with the earliest studies published in 1961 (versus 1892 for physical aggression; Spencer, 1892). As alluded to above, these early foundational studies (Buss, 1961) were rooted in exploring sex differences in aggressive behaviors that were not physical in nature. One of the first studies of this group of behaviors, for example, found that girls displayed significantly higher levels of indirect aggression (e.g., gossiping; Buss, 1961), than did boys (Feshbach, 1969). This approach represented a real advance in the literature at the time, since it had previously been assumed that, because girls engage in very little physical aggression, they did not engage in any aggression at all (i.e., the "benign childhood" hypothesis; Crick & Zahn-Waxler, 2003).

Although interesting in their own right, these findings were also notable given the sharp contrast with the robust empirical and meta-analytic evidence documenting a clear male preponderance in physical aggression. Males engage in notably higher rates of physical aggression than do females, with a 2:1 to 10:1 male-female ratio beginning in the toddler years and continuing throughout the lifespan (e.g., Hyde, 1984; Monuteaux et al., 2004; van Lier et al., 2009). Moreover, sex differences in physical aggression persist across age, informant, and cultures (e.g., Archer, 2004; Card et al., 2008; Lansford et al., 2012). As a consequence, theories incorporating evolutionary traits, biological characteristics, and social learning factors have been proposed to explain sex differences in physical aggression (e.g., Côté, 2007).

Early theory in regards to social aggression was strongly influenced by the different patterns of sex differences observed for social and physical aggression. Crick and Grotpeter (1995), for instance, argued that sex differences in physical aggression were due to researchers' failure to assess aggressive behaviors that are salient for females (i.e., those that used interpersonal relationships to harm others). They further proposed that different types of aggressive behaviors correspond to the different social goals of the perpetrator, and that these social goals differed across sex, driving the sex differences in types of aggression. Indeed, there is evidence that relationally aggressive children experience significant emotional distress in response to relationally provocative scenarios, which contributes to their aggressive responses in these social situations. These interpersonal provocations may be particularly distressing for girls due to the value that girls often place on establishing close relationships in childhood (Maccoby, 1990; Crick & Dodge, 1994; Crick, Bigbee, & Howes, 1996; Leadbeater et al., 1999; Rudolph et al., 2000; Crick, Grotpeter, & Bigbee, 2002; French, Jansen, & Pidada, 2002; Crick & Nelson, 2002).

Crick and Gropteter (1995) similarly hypothesized that girls preferentially utilized relational over physical aggression because it is consistent with the focus on the interpersonal relationships and social functioning that particularly characterizes female peer groups relative to male peer groups (Crick & Grotpeter, 1995). Subsequent work argued that norms against aggression are especially salient for girls, who are socialized against physical aggression by both adults and peers to a greater extent than are boys. It was suggested that these socialization experiences reduce girls' use of physical aggression and instead promote the use of more socially acceptable and covert forms of aggression (Keenan & Shaw, 1997; Crick et al., 2007). Girls' smaller physical size and lower physical strength was also thought to limit their capacity to use physical aggression effectively and, instead, make them more reliant on indirect strategies (Björkqvist, 1994). Finally, girls' earlier cognitive maturation was hypothesized to both buffer against engaging in physical aggression and promote the perpetration of more sophisticated and covert aggressive behaviors (Björkqvist et al., 1992; Keenan & Shaw, 1997; Silverthorn & Frick, 1999).

Despite these reasonable theoretical arguments and the early empirical findings supporting sex differences in social aggression, more recent studies have only inconsistently supported the presence of these sex differences (e.g., Österman et al., 1998; Vaillancourt, 2005). Indeed, some studies actually suggest that boys engage in higher levels of social aggression (e.g., Moroschan, Hurd, & Nicoladis, 2009; Artz, Kassis, & Moldenhauer, 2013), while others find no evidence of sex differences (e.g., Hart et al., 1998; Delveaux & Daniels, 2000). Meta-analytic work attempting to resolve these inconsistencies has concluded that while social aggression is statistically more common in females than in males, the magnitude of this difference is small (Cohen's d = .06 to .18; Archer, 2004; Card et al., 2008; Scheithaur, Haag, Mahlke, & Ittel,

2008). Of note, however, the presence of sex differences was highly variable across studies, with effect sizes ranging from d = -1.04 to d = 1.17 (Scheithauer et al., 2008). The sheer size of this effect size distribution was larger than expected by chance alone.

One possible explanation for these potentially meaningful inconsistencies across studies is that specific socially aggressive behaviors (e.g., gossiping, rudeness, ostracism) do vary consistently across sex, but are inconsistently represented on the various scales used across studies. Slawinski (2016) thus estimated item-level sex differences in socially aggressive behaviors and the degree to which these observed differences reflected true differences in the latent trait as opposed to measurement non-invariance. While we observed consistent, if small, item-level sex differences across samples, follow-up analyses revealed that they were largely a function of measurement non-invariance. These findings suggest that sex differences observed in prior work may also be overestimated due psychometric bias.

Other possible explanations for observed effect size differences across studies include sampling differences and/or the presence of key moderators. Unfortunately, meta-analytic work examining potential moderators of sex differences in social aggression has been inconclusive. While Card et al. (2008) did not find any evidence of moderation by age, Scheithauer et al. (2008) found that higher age was associated with larger sex differences across childhood and adolescence (i.e., 18 years and younger), but Archer (2004) observed that higher female social aggression was limited to later childhood and adolescence (i.e., 11 to 17 years only; not early childhood or young adulthood). In short, the presence or absence of sex differences in social aggression is as yet inconclusive and may be confounded by developmental differences (e.g., Côté, 2007) or other underlying moderator effects (e.g., Keenan, 2007).

Developmental changes and social aggression

Building on the final point, evidence suggests that relational aggression emerges earlier than previously theorized, as observational studies have documented these behaviors in preschoolers as young as 30 months old (Crick et al., 2006). At the other end of the lifespan, relational aggression has also been reported in the elderly residing in assisted living facilities as old as 100 years (Trompetter, Scholte, & Westerhof, 2011). Even so, there are important differences in the use of social aggression across the lifespan as a result of cognitive, social, and biological development. For example, social aggression has been theoretically and empirically associated with heightened social-cognitive and verbal abilities, suggesting that advances in these skills across development may facilitate socially aggressive behavior. Additionally, significant social changes in peer relationships across childhood and adolescence may also promote social aggression. Because social aggression targets relationships as the vehicle of harm, these behaviors may be most effective in the context of established social networks (Archer & Coyne, 2005) and close, intimate friendships (Murray-Close, Ostrov, & Crick, 2007), both of which become more common and salient during adolescence (Berndt, 1996).

Consistent with the latter observations, an emerging body of work indicates that social aggression becomes increasingly common as youth approach adolescence (Murray-Close et al., 2007; Kistner et al., 2010; Kawabata et al., 2012). For example, Murray-Close et al. (2007) observed that the frequency of relational aggression increased linearly over the course of one year among fourth grade girls. Importantly, this increase in relational aggression was further associated with time-dependent increases in friendship intimacy. Similarly, Karriker-Jaffe et al. (2008) reported that social aggression exhibited a curvilinear change across adolescence, with a peak around 14 years of age. Finally, Cleverley et al. (2012) found that while a subset of youth

maintained fairly high levels of indirect aggression across adolescence, most exhibited decreases. Taken together, this emerging literature suggests that social aggression increases in frequency from early childhood to early adolescence, peaks in early- to mid-adolescence, and then declines in frequency as individuals approach adulthood, but there may be significant individual differences in this trajectory.

One potential explanation for social aggression peaking in early- to mid-adolescence is due to cognitive and biological changes related to puberty. Puberty is accompanied by a host of physical changes, including increases in reproductive hormones such as testosterone. It has been theorized that these hormonal changes facilitate social aggression in the context of intrasex competition for resources (e.g., access to sexual partners). According to this theory, social aggression is a strategy used to denigrate sexual rivals (Kistner et al., 2010; Pellegrini & Long, 2003; White, Gallup, & Gallup, 2010). Consistent with this, relational aggression has been associated with dating popularity, especially among girls (Pellegrini & Long, 2003), and indirect aggression has been linked to earlier age of first intercourse (White et al., 2010).

However, very few empirical studies have directly examined associations between pubertal development, reproductive hormones, and social aggression. In the only study assessing the association between social aggression and reproductive hormones, Sánchez-Martín et al. (2011) found that testosterone was positively associated with indirect aggression in 9-year-old boys and girls. Interestingly, there is some evidence suggesting that precocious puberty may be a particular risk factor for social aggression. Indeed, Susman et al. (2007) found that early puberty was associated with higher relational aggression in girls and Hemphill et al. (2010) concluded that pubertal stage was positively associated with social aggression among younger, but not older, adolescents.

Biological processes (genetic and neural) associated with social aggression

Surprisingly, only two studies have taken a neuroscience approach to investigating social aggression. In the only fMRI study related to social aggression, Baird et al. (2010) found that, in adolescent girls, experiences of relational victimization were related to decreased activity in brain regions associated with executive functioning (i.e., decreased recruitment of bilateral dorsolateral prefrontal, anterior, and posterior cingulate cortices) when viewing peer faces with varying affective expressions (Baird, Silver, & Veague, 2010). These results suggest that girls who are unable to recruit specific frontal networks to improve cognitive and executive control may be more sensitive to relational aggression victimization. Similarly, Godleski et al. (2010) found associations between event related potentials and relational aggression in response to relationally provocative situations (i.e., increased P300 amplitude at Fz), which suggests that frontal brain function may play an important role in the perpetration of relational aggression.

However, neither the neural activation patterns nor connectivity associated with the perpetration of social or aggression has ever been explored using fMRI methods. This is a rather shocking gap in the literature given the hypothesized importance of social information processing and emotional reactivity to social situations and stressors among socially aggressive individuals (Crick, Grotpeter, & Bigbee, 2002). Furthermore, it has been implied that the increase in social aggression in late childhood and adolescence is, in part, due to improvements in cognitive and social abilities that are a result of neural maturation processes during these developmental periods (Baird, Silver, & Veague, 2010).

Until recently, theory and research have also been largely silent on the role of genetic transmission and genetic risk for social aggression. Indeed, there have been no molecular genetic studies of social aggression. However, genetically-informed twin designs can also be

used to estimate genetic influences on social aggression, with the added advantage that they are able to simultaneously estimate environmental influences as well. To date, however, only a handful of studies have examined its genetic-environmental etiology, with somewhat inconsistent results.

The first of these assessed social aggression via teacher and peer ratings in a small sample of six year-old twins and found that 20% of the variability in social aggression was due to additive genetic influences (Brendgen et al., 2005). However, a follow-up study of these same twins only a year later suggested that 43% of the variance in social aggression was due to genetic factors. Although such findings could suggest a change in the heritability of social aggression from ages 6 to 7 years old, this is unlikely, particularly given the very small sample sizes. Indeed, although heritability estimates for other antisocial behaviors do change throughout development (Burt & Neiderhiser, 2009), they rarely do so in such dramatic fashion over the course of single year.

Following up on these results, Tackett et al. (2009) made use of a multivariate psychometric model to test etiological influences on the variance common to both mother- and twin-reported child relational aggression in a large sample of 6-18 year-old twins. In contrast to Brendgen et al.'s (2005, 2008) studies, Tackett et al. (2009) found evidence for substantial genetic contributions to the latent relational aggression factor (i.e., 63% of the variance). Similarly, Slawinski, Klump, and Burt (2018) examined a large, multi-informant sample of twins in middle childhood (i.e., the Twin Study of Behavioral and Emotional Development – Child (TBED-C)), and again found that social aggression was largely additive genetic in origin. The importance of the latter findings was bolstered by our use of the nuclear twin family model, which adds the twins' biological parents' phenotypic information to the classical twin model.

This addition was important because the nuclear twin family model uniquely allows for the estimation of both additive and dominant genetic influences while also decomposing shared environmental influences into sibling-level and family-level environmental influences (only the latter of which relates to genotype-environment correlation (rGE) confounds found in shared environmental variance). Using this model, we found that social aggression was both additive genetic (*A*=0.15-0.77) and sibling environmental (*S*=0.42-0.72) in origin. Such findings offer strong additional support for the role of genetic and legitimate (not rGE) environmental influences on social aggression.

It is also worth highlighting a related study (Slawinski et al., 2019), which examined the sources of etiologic overlap between physical and social aggression in the TBED-C. We found that the covariance between social and physical aggression was explained by overlapping genetic factors and common environmental conditions. Specifically, 50-57% of the genetic factors, 74-100% of the shared environmental factors, and 28-40% of the unique environmental factors influencing physical aggression also influenced social aggression. What's more, these unique and shared etiological factors did not differ across sex. These findings argue against the common assumption that social aggression is the 'female version' of male physical aggression, and instead suggest that social aggression may be best conceptualized as a form of antisocial behavior that shares developmental pathways with other manifestations of externalizing pathology.

Although these studies provide important information regarding the etiology of social aggression, much remains unknown. Most notably, none of the aforementioned studies examined their data through a developmental lens (save Brengden et al., 2008 who were unfortunately not sufficiently powered to conduct longitudinal analyses). It is thus unclear whether or how

additional risk factors (e.g., pubertal status, age, or sex) may interact with genetic risk in the development of social aggression.

Overarching aims

There is surging interest in understanding the sex-specific and developmental origins of social aggression. Indeed, countless studies have examined the role of socialization processes and psychosocial risk factors, such as peer relationships (e.g. popularity, victimization, quality of dyadic relationships), parenting (e.g. psychological control, coercive parenting), and media exposure on the etiology of social aggression. In comparison, the biological underpinnings of social aggression have rarely been investigated and, therefore, are not well understood. These are critical omissions from the literature because, as suggested in this review, the etiology of social aggression consists of processes ranging from genetic risk to socialization experiences. Further, these risk factors likely interact across levels of analysis, which makes the investigation of biological factors a priority in developing multi-level models of the etiology of social aggression.

Therefore, the overarching aim of this dissertation is to investigate the roles of sex, genes, pubertal development, and neurological processes in the perpetration of social aggression. To do so, we will capitalize on a unique longitudinal twin study of emotional and behavioral development from the Michigan State University Twin Registry. This study consists of two waves in which data is available for analyses. Wave 1 (Twin Study of Behavioral and Emotional Development in Children; TBED-C) consists of two independent sub-samples of twins in middle childhood. The first sample consists of a population-based epidemiologic sample of 528 families (1,056 twins and their parents). The second, 'at-risk' sample consists of 502 families (1,004 twins and their parents), for whom inclusion criteria also specified that they reside in modestly-to-severely disadvantaged neighborhoods. Wave 2 (Michigan Twin Neurogenetics Study;

MTwiNS) currently consists of a follow-up sample of 275 at-risk TBED-C twin pairs, now in adolescence. Thus, this dissertation project presents us with an opportunity to examine, from a developmental, transdiagnostic, and multi-disciplinary perspective, which biological factors or combination of factors are associated with the perpetration of social aggression during different developmental periods.

To achieve this aim, multiple parallel analyses must occur in both waves of the study. First, the development of social aggression will be examined from a phenotypic and genotypic perspective. More specifically, we will investigate the overall associations between sex, pubertal stage, and age in both waves of the study separately. As discussed previously, potentially meaningful inconsistencies across studies of social aggression (e.g., sex differences) may be in part due to developmental differences in these relationships and pathways. We specifically anticipate observing a positive association between social aggression and age and pubertal stage, respectively, such that both older and more developed participants will be more socially aggressive than younger and less developed participants. Further, moderation models that integrate these factors will be examined to explore whether their interactions resolve extant inconsistencies in the literature regarding the development of social aggression. For example, it may be that the most socially aggressive participants are those who are both older and more developed. However, it may be that the most aggressive participants in middle childhood are those who are younger, but more developed. In that way, it may be that precocious puberty in particular is a risk factor for social aggression.

Next, this dissertation will take a behavioral genetic approach to examining the etiology of social aggression. Previous research has demonstrated that variance in social aggression is partially due to genetic factors. However, it is unknown how developmental risk factors, such as

puberty, might interact with genetic risk for social aggression. It may be that the proportion of genetic risk for social aggression decreases and environmental risk increases as individuals undergo puberty given the rapidly changing social environment that occurs during this developmental stage. Alternatively, the proportion of genetic risk for social aggression may increase following pubertal onset if the increases in reproductive hormones associated with pubertal development "active" genes associated with social aggression. Additionally, it is possible that both puberty and social aggression are influenced by overlapping genetic factors, which may explain their association. These questions have not yet been investigated for social aggression.

Finally, this will be the first investigation of neural correlates associated with the perpetration of social aggression. As stated previously, neither the neural activation patterns nor connectivity associated with the perpetration of social aggression has ever been explored using fMRI methods. This is a rather shocking gap in the literature given the hypothesized importance of emotional reactivity to social situations and stressors among socially aggressive individuals (Crick, Grotpeter, & Bigbee, 2002). The current investigation will not only begin to assess these correlates, but will also examine whether sex, pubertal status, or age moderate the relationship between social aggression and neural activation.

Together, these analyses will integrate these distinct biological risk factors to explore whether their interactions resolve extant inconsistencies in the literature regarding the etiology and presentation of social aggression.

CHAPTER 2: BEHAVIORAL GENETIC ANALYSES

INTRODUCTION

Social aggression is a form of antisocial behavior (Burt et al., 2012) in which social relationships and social status are used to damage reputations and inflict emotional harm on others, and includes behaviors such as gossiping, ostracism, and threatening to end a friendship. Although socially aggressive behaviors are legal and relatively typical during particular developmental periods, they have been associated with pathological outcomes in both victims and aggressors. Victims of social aggression often experience as much emotional distress as victims of physical aggression, including emotional and social difficulties such as peer rejection, loneliness, and internalizing problems (Crick et al., 2002). The perpetrators of social aggression also exhibit a number of maladaptive outcomes, including poor quality friendships marked by conflict and instability, depression, externalizing behavior, and borderline personality disorder features (e.g., Keenan, Coyne, & Lahey, 2008; Spieker et al., 2012; Crick, Murray-Close, & Woods, 2005). Not surprisingly then, there is substantial interest in uncovering the etiology of social aggression.

Extant research regarding the origins of social aggression primarily focuses on socialization processes and psychosocial risk factors, especially peer relationships (e.g. popularity, victimization, quality of dyadic relationships; Crick & Grotpeter, 1995, Sijtsema et al., 2010) and parenting factors (e.g. psychological control, coercive parenting; Kawabata et al., 2011; Kuppens et al., 2013). Other work has focused on marital conflict, sibling relationships, media exposure, and cultural values (e.g., Forbes et al., 2009; Gentile et al., 2011; Karriker-Jaffe et al., 2013). In comparison, the biological underpinnings of social aggression have rarely been investigated and, therefore, are not well understood. Furthermore, even fewer of these studies

have examined both biological and environmental risk factors simultaneously. These are critical omissions from the literature because the development of social aggression is likely due to not only psychosocial and socialization processes, but also biological factors such as genetic risk and hormonal influences.

One risk factor for social aggression that has received a considerable amount of empirical attention is biological sex. Indeed, much of the early research regarding social aggression was rooted in exploring sex differences in these behaviors. This focus was predicated on the hypothesis that, due to their smaller physical size and reduced strength, more rapid cognitive maturation, and focus on interpersonal relationships in female peer groups (Björkqvist, 1994; Crick & Grotpeter, 1995; Keenan & Shaw, 1997; Silverthorn & Frick, 1999), social aggression served as a "female version" of male physical aggression. Despite these theoretical claims and the early empirical findings supporting sex differences in social aggression, meta-analytic work has concluded that while social aggression is statistically significantly more common in females than in males, the magnitude of this difference is generally small (Cohen's d = .06 to .18; Archer, 2004; Card et al., 2008; Scheithaur et al., 2008), albeit with considerable variability across studies (effect sizes ranging from d = -1.04 to 1.17; Scheithauer et al., 2008).

One possible reason for this variability is the presence of key developmental moderators. Unfortunately, meta-analytic work examining potential moderators of sex differences in social aggression has been inconclusive. While Card et al. (2008) did not find any evidence of moderation across age, Scheithauer et al. (2008) found that higher age was associated with larger sex differences across childhood and adolescence (i.e., 18 years and younger), but Archer (2004) observed that higher female social aggression was limited to later childhood and adolescence only (i.e., 11 to 17 years; not early childhood or young adulthood). In short, the presence or

absence of sex differences in social aggression is as yet inconclusive and may be confounded by developmental differences (e.g., Côté, 2007).

Consistent with this hypothesis, there is evidence for age differences in the perpetration of social aggression, such that social aggression increases in frequency from early childhood to early adolescence, peaks in early- to mid-adolescence (i.e., 14 years old), and then declines in frequency as individuals approach adulthood (Murray-Close et al., 2007; Karriker-Jaffe et al., 2008; Kistner et al., 2010; Kawabata et al., 2012; Cleverley et al., 2012). What's more, one potential explanation for social aggression peaking during adolescence is due to biological changes related to puberty. Puberty is accompanied by a host of physical changes, including increases in reproductive hormones such as testosterone. These hormonal changes may facilitate social aggression in the context of intrasex competition for resources (e.g., access to sexual partners). According to this theory, social aggression is a strategy used to denigrate sexual rivals (Kistner et al., 2010; Pellegrini & Long, 2003; White, Gallup, & Gallup, 2010). Consistent with this, relational aggression has been associated with dating popularity, especially among girls (Pellegrini & Long, 2003), and indirect aggression has been linked to earlier age of first intercourse (White et al., 2010).

However, very few empirical studies have directly examined associations between pubertal development and social aggression. In the only study assessing the association between social aggression and reproductive hormones, Sánchez-Martín et al. (2011) found that testosterone was positively associated with indirect aggression in 9-year-old males and females. Similarly, there is some evidence suggesting that precocious puberty may be a particular risk factor for social aggression. Indeed, Susman et al. (2007) found that early pubertal onset was associated with higher relational aggression in girls and Hemphill et al. (2010) concluded that

pubertal stage was positively associated with social aggression among younger, but not older, adolescents.

Individual genetic risk for social aggression

Until recently, theory and research had been largely silent on the role of genetic transmission and genetic risk for social aggression. Indeed, there have been no molecular genetic studies of social aggression. However, genetically-informed twin designs can also be used to estimate genetic influences on social aggression, with the added advantage that they are able to simultaneously estimate environmental influences as well. Even so, only a few studies have examined social aggression's genetic-environmental etiology, with somewhat inconsistent results.

The first of these assessed social aggression via teacher and peer ratings in a small sample of six year-old twins and found that 20% of the variability in social aggression was due to genetic influences (Brendgen et al., 2005). However, a follow-up study of these same twins only a year later suggested that 43% of the variance in social aggression was due to genetic factors. Although such findings could suggest a change in the heritability of social aggression from ages 6 to 7 years old, this is unlikely, particularly given the very small sample sizes. Although heritability estimates for other antisocial behaviors do change throughout development (Burt & Neiderhiser, 2009), they rarely do so in such dramatic fashion over the course of single year. Following up on these results, Tackett et al. (2009) found evidence for substantial genetic contributions (i.e., 63% of the variance) to mother- and twin-reported child relational aggression in a large sample of 6 to18 year-old twins. Most recently, Slawinski, Klump, and Burt (2018, 2019) have also found that social aggression is genetic in origin (i.e., 15-77% of the variance) using a large, multi-informant sample of twins in middle childhood, further highlighting the

potentially important role of genetic risk in the etiology of social aggression. Although these studies provide important information regarding the developmental origins of social aggression, their heritability estimates range dramatically. Further, these few studies span early childhood, middle childhood, adolescence, and emerging adulthood, which is less than optimal should genetic risk differ across development. Indeed, developmental factors (e.g., pubertal status or age) may even interact with genetic risk in the etiology of social aggression.

Current Study

Taken together, there is an emerging body of evidence supporting the role of biological and developmental influences on the etiology of social aggression, including sex and genetic risk. However, there is significant variability amongst these findings, which suggests that they may be moderated by additional developmental factors. A likely candidate for one of these moderators is puberty, which is associated with important changes in cognitive, social, and biological development. Unfortunately, many of the studies that purport to investigate the relationship between social aggression and puberty do not actually assess pubertal development, but instead use age as a proxy for puberty. Age is an imprecise estimate of pubertal development due to the substantial individual variability in pubertal timing. For example, the age at which children enter Tanner Stage 2 (i.e., the beginning of pubertal development) ranges from 7 to 13 years-old (Grumbach & Styne, 2003). Similarly, age at menarche (i.e., first menstrual cycle), which is one of the most commonly used indices of pubertal timing in girls, ranges from 8 to 15 years-old (Chumlea et al., 2003).

There is thus a clear need for a detailed investigation of the roles of sex, genes, age, and pubertal development on the perpetration of social aggression. The current study aims just to do this by examining these phenotypic and etiological relationships using a unique longitudinal twin

study of emotional and behavioral development. I will first clarify associations between social aggression and age, sex, and pubertal development. I will then explore sex, age, and pubertal differences in the etiology of social aggression.

METHODS

Participants

Participants consisted of twin families who participated in a longitudinal study of brain and behavior development within the Michigan State University Twin Registry (MSUTR; Klump & Burt, 2006; Burt & Klump, 2013). Children gave informed assent, while parents gave informed consent for themselves and their children.

Wave 1 (Twin Study of Behavioral and Emotional Development in Children; TBED-C) consists of two independent sub-samples of twins in middle childhood. The first sample consists of a population-based epidemiologic sample of 528 families (1,056 twins and their parents). The second, 'at-risk' sample consists of 502 families (1,004 twins and their parents) in the same general recruitment radius, for whom inclusion criteria also specified that they reside in modestly-to-severely disadvantaged neighborhoods. Recruitment procedures have been described previously (Burt & Klump, 2013; Burt et al., 2016). TBED-C twins were 48.7% female and ranged in age from 6 to 11 years-old (mean age (SD) = 8.02 years (1.49)). Twins' racial and ethnic background was provided by their parents (81.7% non-Hispanic White, 9.5% African American/Black, 1.1% Native American, 0.8% Asian, 0.7% Hispanic, 0.3% Pacific Islander, and 5.9% multiracial or other ethnic groups). Twin zygosity was determined via parent report using a standard 5-item questionnaire that assesses within-pair physical similarity and is over 95% accurate (Peeters et al., 1998). Twin pairs were 41.4% monozygotic (n = 426 pairs

(202 female pairs)), 40.4% same-sex dizygotic, (n = 416 pairs (207 female pairs)), and 18.3% opposite-sex dizygotic (n = 188 pairs).

Wave 2 (Michigan Twin Neurogenetics Study; MTwiNS) consists of a follow-up sample of 275 TBED-C twin pairs, now in adolescence. MTwiNS twins recruited thus far were 43.6% female and ranged in age from 7 to 18 years-old (mean age (SD) = 13.99 (2.37)). Twins' racial and ethnic background was provided by their parents (77.5% non-Hispanic White, 14.5% African American/Black, 2.2% Hispanic, 1.1% Asian, 0.7% Native American, and 4.0% multiracial or other ethnic groups). Twin pairs were 39.6% monozygotic (n = 109 pairs (44 female pairs)), 40.0% same-sex dizygotic (n =110 pairs (48 female pairs)), and 20.4% opposite-sex dizygotic (n = 56 pairs).

Measures

Social aggression was assessed via self-, parent-, and teacher-report using the Subtypes of Antisocial Behavior Questionnaire (STAB; Burt & Donnellan, 2009). The STAB is a 32-item measure assessing three major dimensions of antisocial behavior, one of which is social aggression. The Social Aggression Scale (SA) includes 11 behaviors (e.g., gossips, gives others the silent treatment, and excludes others from group activities). Participants report on the frequency with which they commit each behavior using a scale that ranges from 1 (never) to 5 (nearly all the time). Prior work has confirmed the factor structure of the STAB in multiple samples and provided consistent support for its criterion-related validity (Burt & Donnellan, 2009, 2010). Namely, the STAB scales (1) converge with other measures of antisocial behavior and criminal convictions, (2) show expected patterns of mean differences across treatment groups of adjudicated adults, and (3) correlate as expected with measures of personality (Burt & Donnellan, 2009). Similarly, a study using experience sampling methodology (i.e., participants

reported on specific momentary behaviors six times a day while living in their natural environments), found that high scores on the STAB SA scale was uniquely associated with momentary reports of socially aggressive behaviors (Burt & Donnellan, 2010).

In middle childhood, maternal-reported STAB data were available for 96.0% of the twins ($\alpha = .85$) and teacher-reported STAB data were available for 80.9% of the twins ($\alpha = .91$). In adolescence, maternal-reported STAB data were available for 99.1% of the twins ($\alpha = .87$), teacher-reported STAB data were available for 50.0% of the twins ($\alpha = .94$), and self-report STAB data were available for 98.0% of the twins ($\alpha = .83$). To adjust for positive skew, teacher reports in middle childhood and all informant reports in adolescence were log-transformed and standardized by sex prior to analysis to better approximate normality.

Pubertal development at waves 1 and 2 was assessed via maternal-report using the Pubertal Development Scale (PDS; Peterson et al., 1988). The PDS is a 5-item measure assessing major indices of pubertal growth by sex. Parents report on growth spurt (i.e., height), body hair (e.g., underarm and/or pubic hair), and skin changes (e.g., pimples) for all children. For boys, they also report on voice changes (i.e., deepening) and facial hair growth. For girls, they additionally report on breast development and onset of menarche. Participants respond to each item except menarche using a scale that ranges from 1 (not yet started) to 4 (seems complete). Menarche is coded dichotomously (i.e., yes or no).

Pubertal stages were calculated using the scoring algorithms described in Peterson et al. (1988). For boys, body hair growth, voice change, and facial hair growth item responses are summed and this sum score is used to categorize participants into one of the five standardized pubertal development stages (Tanner, 1962), which range from pre-pubertal (Stage 1) to post-pubertal (Stage 5). For girls, body hair growth and breast development item responses are

summed and this sum score and menarche are used to categorize participants into one of the same pubertal stages described above. Importantly, menarche is necessary for girls to be categorized as late pubertal (Stage 4) or post-pubertal (Stage 5).

Prior work has confirmed the reliability and validity of the PDS (Carskadon & Acebo, 1993) and provided consistent support for its criterion-related validity. Namely, PDS categorization converges with other indices of pubertal status, including similar self-report questionnaires, picture-based interviews, physical exams, levels of basal hormones responsible for advancing pubertal development (i.e., testosterone and dehydroepiandrosterone), and onset of romantic and sexual activities (e.g., Bond et al., 2006; Chan et al., 2010; Shirtcliff, Dahl, & Pollak, 2009; Skoog et al., 2013). In middle childhood, PDS data were available for 96.4% of female twins (n = 968) and 95.1% of male twins (n = 1004; 95.7% of all twins, n = 1972). In adolescence, PDS data were available for 97.5% of female twins (n = 234) and 97.7% of male twins (n = 303; 97.6% of all twins, n = 537).

Quantitative Genetic Analytic Strategy

First, univariate twin models were used to estimate the proportion of genetic and environmental influences, respectively, on the variance within social aggression and pubertal stage, separately by informant and developmental stage. These models decompose phenotypic variance into additive genetic (A), shared environment (C), and non-shared environmental (E) components. Additive genetic variance (A) is the effect of individual genes summed over loci and acts to increase familial correlations relative to the proportion of genes shared (e.g., MZ twins share 100% of their segregating genes and DZ twins share on average 50%). Shared environmental variance (C) is the effect of environmental influences common to family members that act to make them similar to each other regardless of the proportion of genes shared (e.g.,

these environmental influences effect MZ and DZ twins equally). Non-shared environmental variance (E) is the effect of unique environmental influences that serve to differentiate family members regardless of the proportion of genes shared. Measurement error is also captured by E. For these univariate analyses, we tested both a sex differences model and a no-sex differences model. In the former, genetic and environmental parameter estimates are allowed to freely vary across sex. In the latter, genetic and environmental parameter estimates are constrained to be equal across sex. The relative fits of these two models were then compared to reveal whether there are sex differences in the etiology of each phenotype.

Next, bivariate correlated factor models were then used investigate the extent to which overlapping etiological factors underlie the observed phenotypic correlation between social aggression and pubertal stage. This model decomposes shared sources of covariance into genetic (R_A), shared environmental (R_C), and non-shared environmental (R_E) correlations. These correlations reveal the extent to which genetic and environmental factors associated with one phenotype (e.g., social aggression) overlap with the genetic and environmental factors associated with the other phenotype (e.g., pubertal stage). Because these analyses decompose the association between the two variables into its genetic and environmental components, bivariate analyses were only conducted in those cases in which a significant association between pubertal stage and social aggression was observed.

Finally, GxE models were used to examine whether sex and pubertal stage independently and/or jointly moderate the etiology of social aggression. Like the univariate model described above, GxE models decompose the variance in a given phenotype (e.g., social aggression) into A, C, and E components. However, they additionally allow each of the ACE estimates to vary as a function of a single moderator variable (controlling for the other) or jointly by two moderator

variables. Prior to GxE model-fitting, pubertal stage was dichotomized (i.e., pre-pubertal (Stage 1) and pubertal (Stages 2-5)) to facilitate interpretation of these analyses.

Importantly, given the theoretical and phenotypic correlation between age and puberty, all univariate, bivariate, and GxE analyses were re-run controlling for age. The results of these analyses were comparable to those described below and, therefore, will not be explained separately.

Mx, a structural-equation modeling program (Neale et al. 2003), was used to perform the model-fitting analyses. Because of missing data, we made use of Full-Information Maximum-Likelihood (FIML) raw data techniques, which produce less biased and more efficient and consistent estimates than pairwise or listwise deletion in the face of missing data. When fitting models to raw data, variances, covariances, and means are first freely estimated to get a baseline index of fit (minus twice the log-likelihood; −2lnL). Model fit was evaluated using −2lnL and four information theoretic indices that balance overall fit with model parsimony: the Akaike's Information Criterion (AIC; Akaike, 1987), the Bayesian Information Criteria (BIC; Raftery, 1995), the sample-size adjusted Bayesian Information Criterion (SABIC; Sclove, 1987), and the Deviance Information Criterion (DIC; Spiegelhalter et al., 2002). The lowest AIC, BIC, SABIC, and DIC among a series of nested models is considered best. As fit indices do not always agree, we reasoned that the best fitting model should yield lower or more negative values for at least three of the five fit indices.

RESULTS

Descriptive Statistics and Correlations

Descriptive statistics and correlations are presented in Table 1, and a complete breakdown of frequency of pubertal stages in middle childhood and adolescence are presented

separately by sex in Figures 1 and 2. There were significant differences in informant reports of social aggression. Twins were rated as more socially aggressive by their mothers than their teachers in both childhood ($t(1346) = 21.22$, $p < 0.000$) and adolescence ($t(193) = 4.16$, $p < 0.000$). Twins also rated themselves as more socially aggressive than did their teachers in adolescence ($t(195) = 3.18$, $p = 0.002$). However, there were no significant differences between maternal-report and twin self-report of social aggression in adolescence ($t(319) = -0.99$, $p = 0.324$). This suggests that mothers and their children report comparable levels of child social aggression, and their reports indicate higher levels of child social aggression than do teacher reports.

As seen in Table 1, there were no sex differences in social aggression in middle childhood or adolescence according to any informant. By contrast, age was positively associated with maternal reports of social aggression in middle childhood and self-reports of social aggression in adolescence. Age was also positively correlated with participant sex in adolescence, such that female participants tended to be older than male participants.

Pubertal stage was positively associated with maternal reports of social aggression in girls in middle childhood and self-reports of social aggression in both boys and girls in adolescence. However, pubertal stage was negatively associated with teacher reports of social aggression in boys in adolescence. Pubertal stage was positively associated with age and sex in both middle childhood and adolescence, such that older participants were more developed than younger participants and female participants were more developed than male participants. As seen in Figures 1 and 2, the majority of participants are in the pre-pubertal stage in middle childhood, but more girls than boys are in the later stages of development. In adolescence, the

majority of participants are in the middle to later stages of development, but more girls than boys are in the final two stages.

Moderation Analyses

Moderated linear regression was used to test whether social aggression varied phenotypically as a function of sex, age, puberty, or their interactions. Analyses were conducted via multi-level modeling, in which the child was the lower-level unit and the family was the upper-level unit, to control for the non-independence of twins within families. Results are presented separately for each informant (Table 2).

In middle childhood, we observed a positive association between maternal-reported social aggression and pubertal stage, such that more advanced pubertal status predicted increased social aggression. This association between maternal-reported social aggression and pubertal status was not moderated by sex, age, puberty, or their interactions. Teacher-reported social aggression did not vary with sex, age, puberty, or their interactions.

In adolescence, maternal-reported social aggression was no longer associated with pubertal stage. An association emerged between teacher-reported social aggression and sex, such that boys were rated as more socially aggressive than girls, but this relationship was moderated by age (Figure 3) and pubertal stage (Figure 4). According to teachers, there were no age differences in social aggression for boys, but younger girls were more socially aggressive than older girls. Similarly, teachers reported large pubertal differences in social aggression for girls, but small differences for boys.

Twin Correlations

Intraclass correlations within twin pairs, computed separately by degree of genetic relatedness, offer a preliminary indication of genetic and environmental influences on social

aggression and pubertal status, respectively. MZ intraclass correlations that are double those of DZ intraclass correlations are indicative of genetic effects, whereas MZ correlations that are less than double but still greater than DZ correlations suggest the importance of genetic and shared environmental effects. Intraclass and cross-twin, cross-trait correlations for social aggression and pubertal stage in middle childhood and adolescence are presented separately by sex in Tables 3 and 4.

In middle childhood, MZ intraclass correlations for pubertal status and both informant reports of social aggression were generally larger than their corresponding DZ correlations, suggesting that genetic effects are important for their respective etiologies. However, these MZ correlations were not double those of DZ correlations, suggesting that environmental effects are also important for the etiologies of pubertal status and social aggression, respectively, in middle childhood. Additionally, there were no statistically significant differences in MZ and DZ correlations for teacher reports of social aggression in girls, which also supports the importance of shared environmental effects.

In adolescence, MZ intraclass correlations for pubertal status in both sexes and maternal reports of social aggression in girls were again larger than, but not twice as large as, their corresponding DZ correlations, which suggests that both genetic and environmental factors are important for their respective etiologies. However, there were no statistically significant differences in MZ and DZ intraclass correlations of maternal reports of social aggression in boys, teacher reports of social aggression in both sexes, or self-reports of social aggression in both sexes. This suggests that environmental effects are important for the etiology of teacher- and self-reported social aggression in adolescence.

Additionally, cross-twin, cross-trait correlations (e.g., social aggression of Twin 1 and pubertal status of Twin 2) offer a preliminary indication of genetic and environmental influences on the covariation between these traits (see Table 4). These analyses were restricted to those reports of social aggression that were correlated with pubertal status (i.e., it does not make sense to decompose a correlation near zero into its genetic and environmental variance components). For maternal reports of social aggression in middle childhood, there were significant positive cross-twin, cross-trait correlations for same-sex female twins only, suggesting that there may be sex differences in the etiological covariation between social aggression and pubertal stage. However, these cross-twin, cross-trait correlations in girls did not differ by zygosity, indicating that shared environmental effects may drive the covariation between puberty and maternal reports of social aggression in middle childhood girls.

For self-reports of social aggression in adolescence, there were significant positive cross-twin, cross-trait correlations for same-sex DZ male twins and opposite sex twins. However, the MZ and DZ cross-twin, cross-trait correlations for self-reports of social aggression in males were not significantly different from one another, again suggesting that the etiology of the covariation between puberty and social aggression may be largely shared environmental in origin. The overall pattern of cross-twin, cross-trait correlations supports the hypothesis that there may be sex differences in etiological covariation between social aggression and puberty, but further suggests that there may also be developmental variation in these sex differences.

Quantitative Genetic Analyses

Univariate model fitting results and parameter estimates for social aggression and pubertal stage are presented in Tables 5 and 6. For social aggression, the best-fitting model across all informants in both middle childhood and adolescence was the ACE no sex differences

model, indicating that additive genetic, shared environmental, and non-shared environmental influences significantly contribute to the etiology of social aggression, and more importantly, that these magnitudes do not differ across sex. However, examination of the parameter estimates revealed that the magnitude of these influences on social aggression may change across development and informant. In middle childhood, there were clear contributions of additive genetic (27-36%) shared environmental (23-49%), and non-shared environmental (24-41%) influences on the etiology of social aggression. In adolescence, parameter estimates were less consistent across informants. Maternal and self-reports indicated that shared (36-61%) and non-shared (30-64%) environmental influences accounted for nearly all of the variance in social aggression. However, teacher reports suggested that variation in social aggression was largely due to additive genetic (43%) and non-shared environmental influences (26%).

For pubertal stage, the best-fitting model in both middle childhood and adolescence was the ACE sex differences model, indicating that additive genetic, shared environmental, and non-shared environmental influences significantly contribute to the etiology of puberty, and more importantly, that these magnitudes differ between males and females. In middle childhood, additive genetic influences were larger (52 vs. 35%) and non-shared environmental influences were smaller (11 vs. 21%) for girls compared to boys, but shared environmental influences were equivalent (36 vs. 37%). In adolescence, additive genetic influences were smaller (18 vs. 37%) and shared environmental influences were larger (76 vs. 57%) for girls compared to boys, but non-shared environmental influences were comparable (7 vs. 3%). These findings are consistent with prior research demonstrating that the variability in pubertal development is largely explained by genetic factors, likely via gene-hormone interplay (Eaves et al. 2004; Mustanski et al. 2004; van den Berg et al. 2006). The low heritability of pubertal stage in adolescent girls

likely reflects the fact that nearly all girls (91%) have reached sexual maturity by mid- to late-adolescence. Sex differences in genetic effects during these two developmental periods may also be a consequence of sex differences in the timing of these genetic-hormonal processes. Additionally, environmental factors also contribute significantly to pubertal development, particularly those associated with socioeconomic status, such as nutrition and household stress levels (Parent et al., 2003; Da Silva et al., 2004; Webster et al. 2014).

Bivariate model fitting results and genetic and environmental correlations between maternal reports of social aggression and pubertal stage in girls in middle childhood are presented in Table 7. The best-fitting bivariate model was the ACE model, but parameter estimates indicate that although there was a moderate correlation between shared environmental factors ($R_C = 0.33$), the respective overlap between additive genetic and non-shared environmental influences on puberty and social aggression, respectively, were not significant. Put another way, 11% of the shared environmental factors influencing social aggression and pubertal status are the same in girls in middle childhood. Bivariate models were not tested for maternal reports of boys' social aggression or for teacher reports of social aggression in middle childhood given the absence of phenotypic associations with pubertal stage. Additionally, bivariate models were not tested in adolescence given sample size constraints (Verhulst, 2017).

GxE ICCs, model fitting results, and parameter estimates for sex, pubertal stage, and social aggression in middle childhood are presented in Tables 8, 9, and 10. These path and moderator estimates were then used to calculate and plot the unstandardized A, C, and E variance components at each level of the moderator(s) in Figures 5 and 6. Like before, GxE models were not tested in adolescence given sample size constraints (Verhulst, 2017).

The best-fitting joint GxE model for maternal reports of social aggression was the full moderation model, indicating that sex and pubertal stage jointly moderated the etiology of social aggression. When examining the moderating effect of sex alone controlling for the effects of pubertal status (Figure 5), genetic influences on social aggression were stronger for boys than for girls (A = 0.32 for boys vs. 0.06 for girls) and non-shared environmental influences were stronger for girls than for boys (C = 0.45 for boys vs. 0.58 for girls). In contrast, pubertal stage by itself did not moderate the etiology of social aggression. However, genetic influences on social aggression were jointly moderated by sex and pubertal stage (Figure 6).

For boys, genetic influences on social aggression increased minimally from pre-puberty to puberty. For pre-pubertal boys, 33% of the variance in social aggression was explained by genetic factors (A = 0.32), 47% was explained by shared environmental factors (C = 0.45), and 20% was explained by non-shared environmental factors (E = 0.19). For pubertal boys, 40% of the variance in social aggression was explained by genetic factors (A = 0.60), 54% was explained by shared environmental factors (C = 0.80), and 7% was explained by non-shared environmental factors (E = 0.10).

For girls, however, genetics influences more than doubled from pre-puberty to puberty, although they remained small. For pre-pubertal girls, 7% of the variance in social aggression was explained by genetic factors (A = 0.06), 65% was explained by shared environmental factors (C = 0.58), and 28% was explained by non-shared environmental factors (E = 0.25). For pubertal girls, 16% of the variance in social aggression was explained by genetic factors (A = 0.21), 73% was explained by shared environmental factors (C = 0.97), and 11% was explained by non-shared environmental factors (E = 0.14).

The best-fitting joint GxE model for teacher reports of social aggression was inconclusive because no model tested yielded lower or more negative values for at least three of the five fit indices.

DISCUSSION

The overarching aim of this study was to investigate the extent to which interactions between biological sex, age, and pubertal status predicted differences in the prevalence and etiology of social aggression in middle childhood and adolescence. Notably, this was the first twin study to examine pubertal moderation of the etiology of social aggression. Results suggested that not only was puberty associated with the perpetration of social aggression, at least when assessed via maternal report, but it also moderated its prevalence and etiology differently across sex. Critically, these findings were robust to the effects of age on social aggression. However, they did not persist to other informant-reports.

Specifically, pubertal stage was positively associated with maternal reported social aggression, but this effect was stronger for girls than for boys. Furthermore, quantitative genetic analyses revealed that additive genetic influences significantly contributed to the etiology of maternal-reported social aggression, but that the magnitude of these influences was moderated by pubertal stage in girls, such that there was a significant, though small, increase in genetic effects on social aggression from pre-puberty to puberty. While additive genetic influences also significantly contributed to the etiology of social aggression in boys, they were not moderated by pubertal stage. Instead, pubertal stage moderated the magnitude of shared environmental effects on social aggression in boys, such that there was a significant increase in shared environmental effects on social aggression from pre-puberty to puberty. Therefore, association between pubertal

stage and social aggression appears may be driven by different developmental mechanisms for boys and girls.

One possible mechanism is that pubertal increases in reproductive hormones may influence increases in social aggression (Archer, 2006; Kistner et al., 2010). Our findings suggest that this may occur via the genomic effects of hormones and their role in altering gene expression (i.e., "activating" genes associated with social aggression), especially in girls. Given that ovarian hormones drive pubertal development in girls, increasing genetic effects across puberty could indicate an effect of ovarian hormones on genetic risk for social aggression. A second, related possibility is that pubertal changes in brain development influence increases in social aggression. Early theories regarding social aggression suggested girls engage in more sophisticated and covert aggressive behaviors than boys due to their earlier cognitive maturation (Björkqvist et al., 1992; Keenan & Shaw, 1997; Silverthorn & Frick, 1999), and it may be that these improvements in cognition are a result of neural maturation processes that occur during late childhood and adolescence. Indeed, neuroimaging research has shown that neural development continues throughout childhood and adolescence, with grey matter development in the frontal cortex peaking at approximately 12 years of age and white matter volume increasing (i.e., myelination) in the frontal cortex between 9 and 14 years (Blakemore, 2012). However, despite both these early theories regarding the development of social aggression and modern advances in neuroimaging research, the neural processes implicated in social aggression have not yet been investigated directly.

It has also been suggested that significant social changes in peer relationships promote social aggression. Our findings suggest that this may occur via peer processes that change or intensify during puberty (e.g., close interpersonal relationships) rather than those that are

common across childhood and adolescence (e.g., deviant peer affiliation). For example, the cultivation of close, intimate friendships and the establishment of social networks become more common during adolescence (Berndt, 1996; Archer & Coyne, 2005; Murray-Close, Ostrov, & Crick, 2007). However, these peer relationships and dynamics that become more pronounced during puberty may not be more potent for girls than boys as had been suggested (e.g., Crick & Grotpeter, 1995).

Limitations

A number of limitations to the current study should be considered. First, our findings of etiologic moderation were specific to maternal reports of social aggression in middle childhood and did not extend to teacher reports of social aggression during the same developmental period. Similarly, we were unable to investigate these relationships in the adolescent sample due to sample size constrains, such that a minimum of 1000 twin pairs are recommended to detect moderation with reasonable power (Verhulst, 2017). Because of this, we were unable to fully investigate the relationships between puberty and self-reports of social aggression. Self-reports and peer-ratings are especially useful since social aggression is characterized by both overt behaviors (that adults are likely to witness) and covert behaviors (of which only the child and his or her peers may be aware). Indeed, behavioral manifestations of social aggression may become more covert and sophisticated as individuals mature (Crick et al., 2007). Although there are concerns regarding the reliability and validity of peer- and self-reports in young children, peer ratings are more frequently used with adolescents and have been suggested to be more valid than teacher, parent, or self-ratings of social aggression during later developmental periods (Archer & Coyne, 2005). Likewise, we were limited by the variability in pubertal development in middle childhood. Because the majority of these participants were pre-pubertal, pubertal stage was

dichotomized in order to have sufficient power for the moderation analyses. This prevented us from investigating both quadratic and more nuanced linear effects across puberty.

Regardless, the current study is a critical step forward in advancing our understanding of the roles of sex and puberty in the prevalence and etiology of social aggression. Not only was puberty associated with maternal reports of social aggression, but it also moderated its prevalence and etiology differently across sex. Such findings build on emerging literature that suggests puberty is likely a key flash point in the development and etiology of social aggression. Future studies should expand on these findings by investigating more nuanced measures of puberty, such as pubertal timing (e.g., precocious puberty) and pubertal tempo, across diverse samples. It remains imperative that future research continues to employ longitudinal and person-centered approaches to explore social and biological developmental processes implicated in its development.

TABLES

Table 1: Descriptive statistics and correlations by sex

	1.	2.	3.	4.	5.	Males	Females
Middle Childhood						M (SD)	M (SD)
1. Age	-	.208**	.119**	-.043	--	7.98 (1.47)	8.06 (1.51)
2. Pubertal stage	.546**	-	.035	.003	--	--	--
3. Social Aggression – Maternal Report	.114**	.142**	-	.224**	--	18.05 (4.93)	17.80 (4.81)
4. Social Aggression – Teacher Report	-.016	.008	.144**	-	--	14.44 (4.65)	14.32 (5.38)
Sex	.026	.339**	-.026	-.012	--	--	--
Adolescence							
1. Age	-	.785**	.081	-.066	.243**	13.53 (2.56)	14.58 (1.94)
2. Pubertal stage	.705**	-	.101	-.160*	.232**	--	--
3. Social Aggression – Maternal Report	.090	.055	-	.219**	.161**	16.51 (4.78)	16.97 (4.86)
4. Social Aggression – Teacher Report	-.037	-.115	.179	-	-.030	15.47 (6.48)	14.63 (4.96)
5. Social Aggression – Self Report	.155*	.141*	.343**	-.021	-	16.31 (5.03)	16.45 (4.68)
Sex	.220**	.491**	.047	-.071	.014	--	--

Note: Male correlations are above the diagonals and female correlations are below. The STAB Social Aggression Scale ask informants to report on the frequency with which the child commits each behavior, ranging from 1 (never) to 5 (nearly all the time). Correlations with participant sex are indicated in gray shading. The Social Aggression Scale contains 11 behaviors, so overall scale scores could range from 11 to 55. * $p < .05$; ** $p < .01$

Table 2: Predicting social aggression as a function of child sex, age, pubertal stage, and their interactions

		Middle Childhood				Adolescence			
Informant	Predictor variable	*B*	*SE*	*t*	*p*	*B*	*SE*	*t*	*p*
Mother	Sex	.374	.201	1.864	.062	.755	.697	1.083	.279
	Age	.105	.119	.885	.376	-.097	.235	-.412	.680
	Pubertal stage	.595	.201	2.963	.003**	.694	.697	.996	.320
	Sex x Age	-.052	.119	-.438	.661	.077	.235	.328	.743
	Sex x Pubertal stage	-.015	.201	-.073	.942	-.401	.697	-.576	.565
Teacher	Sex	-.027	.261	-.102	.918	2.647	1.076	2.461	.014*
	Age	-.130	.152	-.856	.392	-.645	.360	-1.792	.074
	Pubertal stage	.149	.261	.569	.569	1.466	1.076	1.363	.174
	Sex x Age	.035	.152	.229	.819	.818	.360	2.272	.024*
	Sex x Pubertal stage	-.107	.261	-.410	.682	-2.865	1.076	-2.663	.008**
Self-report	Sex	--	--	--	--	1.207	.804	1.501	.134
	Age	--	--	--	--	.159	.274	.580	.562
	Pubertal stage	--	--	--	--	.749	.804	.932	.352
	Sex x Age	--	--	--	--	.289	.274	1.054	.293
	Sex x Pubertal stage	--	--	--	--	-.847	.804	-1.054	.293

Note: Age was grand-mean centered prior to analysis. Pubertal stage (i.e., pubertal = -1 or pre-pubertal = 1) and child sex (i.e., -1 = female or 1 = male) were effect coded prior to analysis. * $p < .05$; ** $p < .01$

Table 3: Intraclass correlations for social aggression and pubertal stage for each sex-zygosity cohort

		Middle Childhood		Adolescence		
		Maternal Report	Teacher Report	Maternal Report	Teacher Report	Self Report
Males	Social Aggression	.792**/.623**	.621**/.445**	.637**/.720**	.669**/.553**	.355**/.415**
	Pubertal Stage	.728**/.629**	--	.966**/.770**	--	--
Females	Social Aggression	.707**/.696**	.532**/.545**	.787**/.604**	.728**/.694**	.225*/.314**
	Pubertal Stage	.884**/.633*	--	.925**/.864**	--	--
Opposite Sex	Social Aggression	.572**	.252**	.602**	.390*	.449**
	Pubertal Stage	.289**	--	.522**	--	--

Note: ICCs are presented separately across zygosity (rMZ/rDZ). * $p < 0.05$, ** $p < 0.01$

Table 4: Cross-twin, cross-trait correlations for social aggression and pubertal stage

	Maternal Report (Middle Childhood)	Self Report (Adolescence)
Males	.074/.041	.185/.286**
Females	.101*/.141*	-.072/.306
Opposite Sex	.075	.330**

Note: Correlations are presented separately across zygosity (rMZ/rDZ). *$p < 0.05$; **$p < 0.01$

Table 5: Univariate quantitative genetic model fitting results for social aggression and pubertal status

			-2LnL	*df*	AIC	BIC	SABIC	DIC
Social Aggression – Maternal Report								
Middle Childhood	ACE	Sex Differences	4032.341	1625	782.341	-3426.130	-855.938	-1942.855
	ACE	**No Sex Differences**	**4038.353**	**1628**	**782.353**	**-3442.189**	**-858.234**	**-1947.157**
Adolescence	ACE	Sex Differences	1085.69	425	235.690	-601.360	72.036	-219.811
	ACE	**No Sex Differences**	**1088.416**	**428**	**232.416**	**-608.074**	**70.075**	**-214.768**
Social Aggression – Teacher Report								
Middle Childhood	ACE	Sex Differences	3015.997	1123	769.997	-2113.940	-331.235	-1081.972
	ACE	**No Sex Differences**	**3019.393**	**1126**	**767.393**	**-2121.918**	**-334.450**	**-1087.193**
Adolescence	ACE	Sex Differences	558.115	219	120.115	-260.477	85.944	-59.229
	ACE	**No Sex Differences**	**558.744**	**222**	**114.744**	**-267.553**	**83.614**	**-63.549**
Social Aggression – Self Report								
Adolescence	ACE	Sex Differences	1196.642	421	354.642	-533.175	133.865	-146.302
	ACE	**No Sex Differences**	**1197.591**	**424**	**349.591**	**-540.764**	**131.030**	**-151.134**

Note: Additive genetic, shared environmental, and non-shared environmental influences are represented with A, C, and E, respectively. The best fitting model for each phenotype (as indicated by the lowest -2LnL, AIC, BIC, SABIC, and DIC values for at least 3 of the 5 fit indices) is highlighted in bold font.

Table 5 (cont'd)

			-2LnL	*df*	AIC	BIC	SABIC	DIC
Pubertal Status								
Middle Childhood	**ACE**	**Sex Differences**	**3847.917**	**1616**	**615.917**	**-3495.186**	**-929.292**	**-2010.182**
	ACE	No Sex Differences	3870.993	1619	632.993	-3493.709	-923.051	-2005.947
Adolescence	**ACE**	**Sex Differences**	**847.750**	**423**	**1.750**	**-713.973**	**-43.755**	**-325.262**
	ACE	No Sex Differences	856.680	426	4.680	-717.578	-42.607	-326.111

Table 6: Best-fitting univariate model parameter estimates for social aggression and pubertal status

			A	C	E
Social Aggression – Maternal Report					
Middle Childhood	ACE	No Sex Differences	0.2737* [0.1685, 0.3842]	0.4946* [0.3854, 0.6045]	0.2354* [0.2057, 0.2710]
Adolescence	ACE	No Sex Differences	0.0861 [0.0000, 0.3044]	0.6108* [0.4007, 0.8172]	0.3003* [0.2332, 0.3821]
Social Aggression – Teacher Report					
Middle Childhood	ACE	No Sex Differences	0.3633* [0.1495, 0.5800]	0.2306* [0.0498, 0.4049]	0.4066* [0.3429, 0.4863]
Adolescence	ACE	No Sex Differences	0.4310* [0.0472, 0.8638]	0.3167 [0.000, 0.6590]	0.2576* [0.1754, 0.4006]
Social Aggression – Self Report					
Adolescence	ACE	No Sex Differences	0.0000 [0.0000, 0.2667]	0.3608* [0.1425, 0.4990]	0.6356* [0.5252, 0.7568]
Pubertal Status					
Middle Childhood	ACE	Males	0.3526* [0.1974, 0.5270]	0.3674* [0.2025, 0.5285]	0.2086* [0.1726, 0.2554]
	ACE	Females	0.5238* [0.3889, 0.6914]	0.3584* [0.1817, 0.5315]	0.1065* [0.0879, 0.1307]
Adolescence	ACE	Males	0.3675* [0.2387, 0.5721]	0.5702* [0.3120, 0,8635]	0.0341* [0.0246, 0.0490]
	ACE	Females	0.1782* [0.0471, 0.3599]	0.7633* [0.4906, 1.1201]	0.0729* [0.0491, 0.1150]

Note: Additive genetic, shared environmental, and non-shared environmental influences are represented with A, C, and E, respectively. 95% confidence intervals are presented below the point estimate in brackets. *$p < 0.05$.

Table 7: Bivariate quantitative genetic model fitting results and parameter estimates for the covariation of maternal reports of social aggression and pubertal stage in girls in middle childhood

	-2LnL	*df*	AIC	BIC	SABIC	DIC	r_G	r_C	r_E
ACE	**3804.62**	**1576**	**652.62**	**-2820.93**	**-320.54**	**-1372.68**	-0.146 [-1.00, 1.00]	0.329* [0.081, 0.622]	0.045 [-0.096, 0.183]
AE	3863.10	1579	705.10	-2800.68	-295.54	-1349.678			
CE	3878.55	1579	720.55	-2792.96	-287.81	-1341.96			

Note: Additive genetic, shared environmental, and non-shared environmental influences are represented with A, C, and E, respectively. The best fitting model (as indicated by the lowest -2LnL, AIC, BIC, SABIC, and DIC values for at least 3 of the 5 fit indices) is highlighted in bold font.

Table 8: Twin intraclass correlations of social aggression across levels of pubertal status (dichotomized) in middle childhood

	Maternal Report					Teacher Report				
	MZ Males	MZ Females	DZ Males	DZ Females	OS DZ	MZ Males	MZ Females	DZ Males	DZ Females	OS DZ
Pre-pubertal	.814**	.734**	.633**	.748**	.688**	.634**	.397**	.450**	.620**	.258*
Pubertal	.948**	.782***	.813**	.771**	.511	.674	.700**	.823	.283	.664

Note: These correlations are computed with only twin pairs who are concordant for pubertal status. However, both concordant and discordant twin pairs are included in the GxE model fitting analyses. * $p < .05$; ** $p < .01$

Table 9: GxE quantitative genetic model fitting results for social aggression, sex, and pubertal status (dichotomized) in middle childhood

	-2LnL	df	AIC	BIC	SABIC	DIC
Maternal Report						
Full moderation model	**4796.620**	**1927**	**942.620**	**-4231.930**	**-1171.851**	**-2461.135**
Sex moderation only	4824.924	1933	958.924	-4238.422	-1168.815	-2462.114
Pubertal moderation only	4825.336	1933	959.336	-4238.216	-1168.610	-2461.908
Sex and pubertal moderation only	4813.197	1930	953.197	-4233.963	-1169.121	-2460.412
No moderation	4837.419	1936	965.419	-4242.497	-1168.126	-2463.432
Teacher Report						
Full moderation model	**3601.216**	1321	959.216	-2567.532	-470.199	-1353.615
Sex moderation only	3607.276	1327	**953.276**	-2584.342	**-477.482**	-1364.911
Pubertal moderation only	3616.319	1327	962.319	-2579.821	-472.961	-1360.390
Sex and pubertal moderation only	3606.386	1324	958.386	-2574.867	-472.770	-1358.193
No moderation	3618.582	1330	958.582	**-2588.609**	-476.986	**-1366.421**

Note: Note: Additive genetic, shared environmental, and non-shared environmental influences are represented with A, C, and E, respectively. The best fitting model for each sex (as indicated by the lowest -2LnL, AIC, BIC, SABIC, and DIC values for at least 3 of the 5 fit indices) is highlighted in bold font.

Table 10: Unstandardized path and moderator estimates for the best-fitting GxE model of maternal reports of social aggression and pubertal status (dichotomized) in middle childhood

Model	Paths			Linear Moderation						Joint Moderation		
	a	c	e	a_{sex}	c_{sex}	e_{sex}	$a_{puberty}$	$c_{puberty}$	$e_{puberty}$	a	c	e
Full moderation	**0.5663*** [0.4221, 0.6730]	**0.6727*** [0.5622, 0.7730]	**0.4368*** [0.3971, 0.4831]	**-0.3163*** [-0.5685, -0.0978]	0.08 71 [-0.04 78, 0.21 48]	**0.0658*** [0.00 14, 0.12 98]	0.20 54 [-0.15 78, 0.52 43]	0.22 43 [-0.14 61, 0.55 39]	-0.12 48 [-0.23 49, 0.06 24]	**-0.8831*** [-1.28 11, -0.46 10]	-0.17 03 [-0.54 94, 0.24 92]	0.11 25 [-0.09 42, 0.26 80]

Note: Significant parameter estimates are highlighted in bold font. 95% confidence intervals are presented below the point estimate in brackets. *$p < 0.05$.

FIGURES

Figure 1: Distribution of pubertal stages in middle childhood

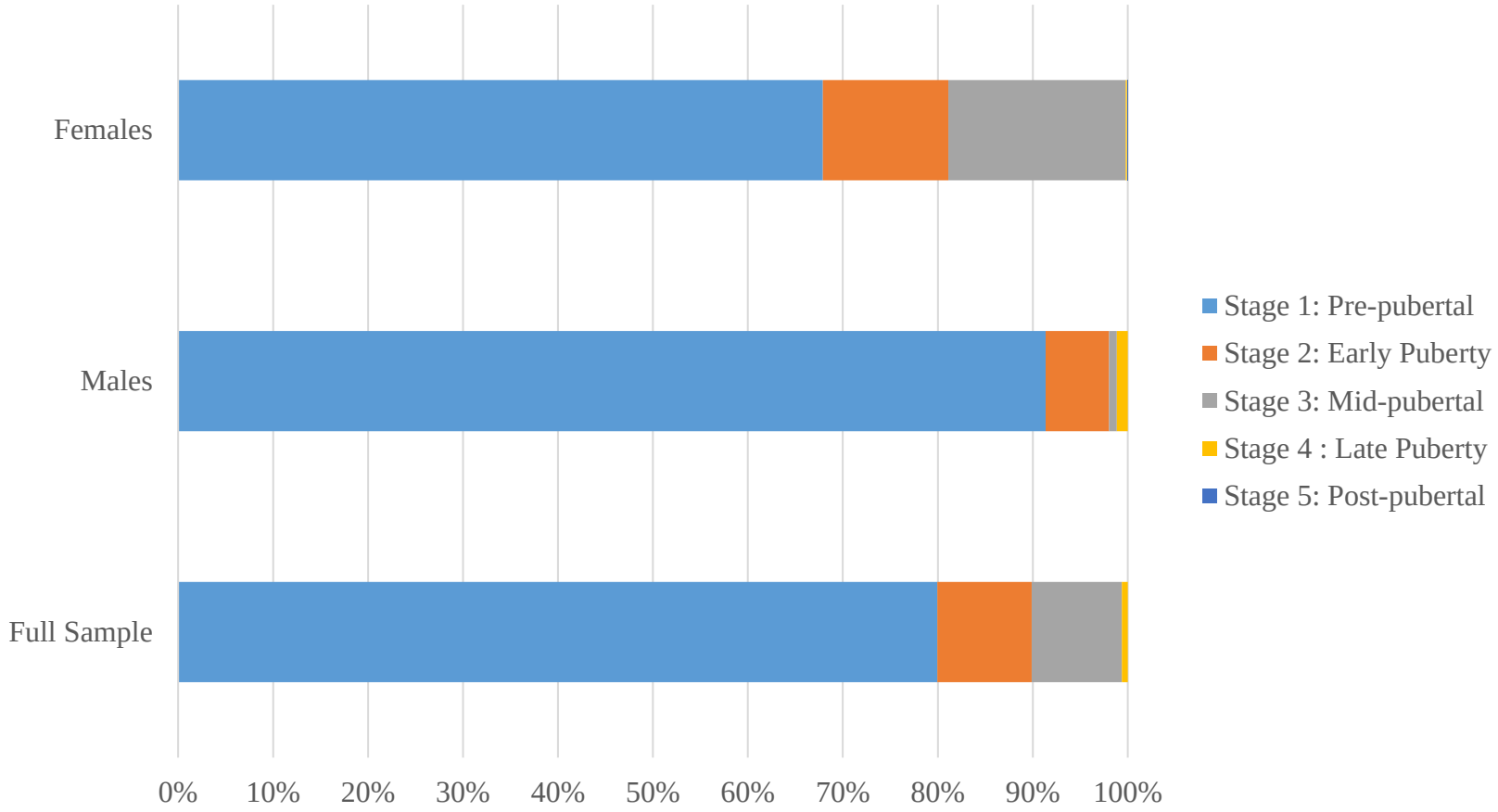


Figure 2: Distribution of pubertal stages in adolescence

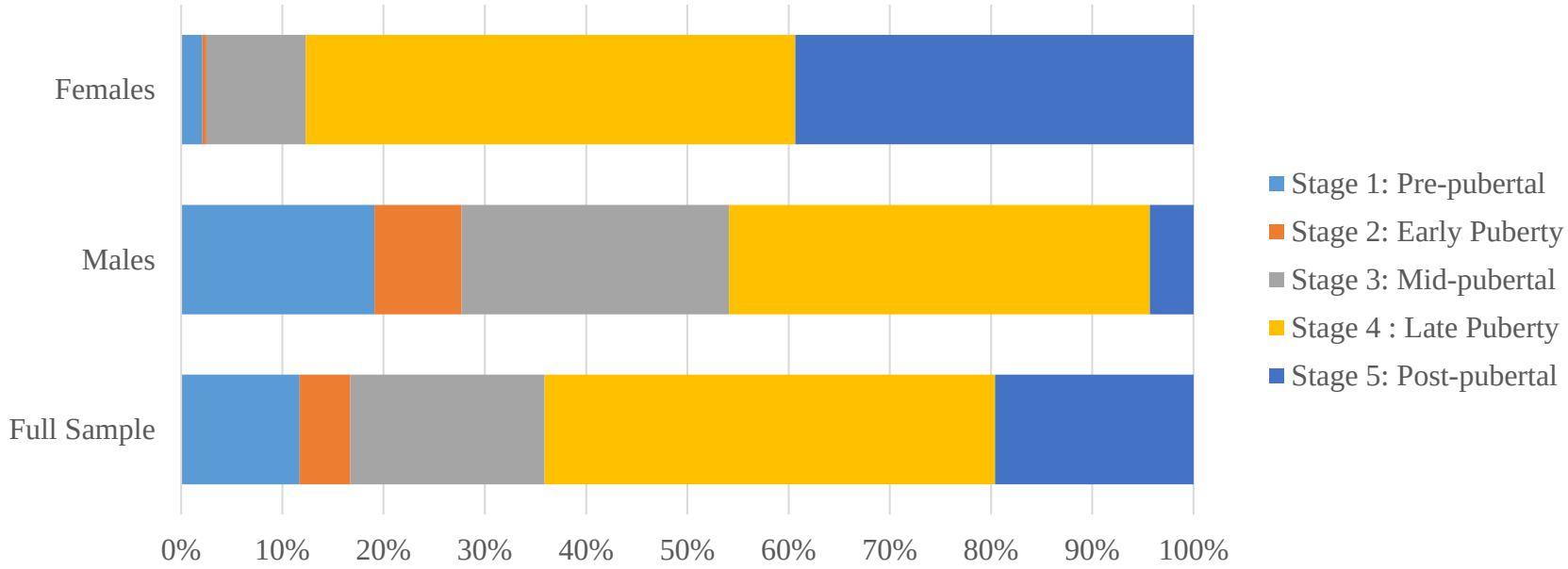


Figure 3: Predicting teacher reports of social aggression in adolescence as a function of child age, sex, and their interaction

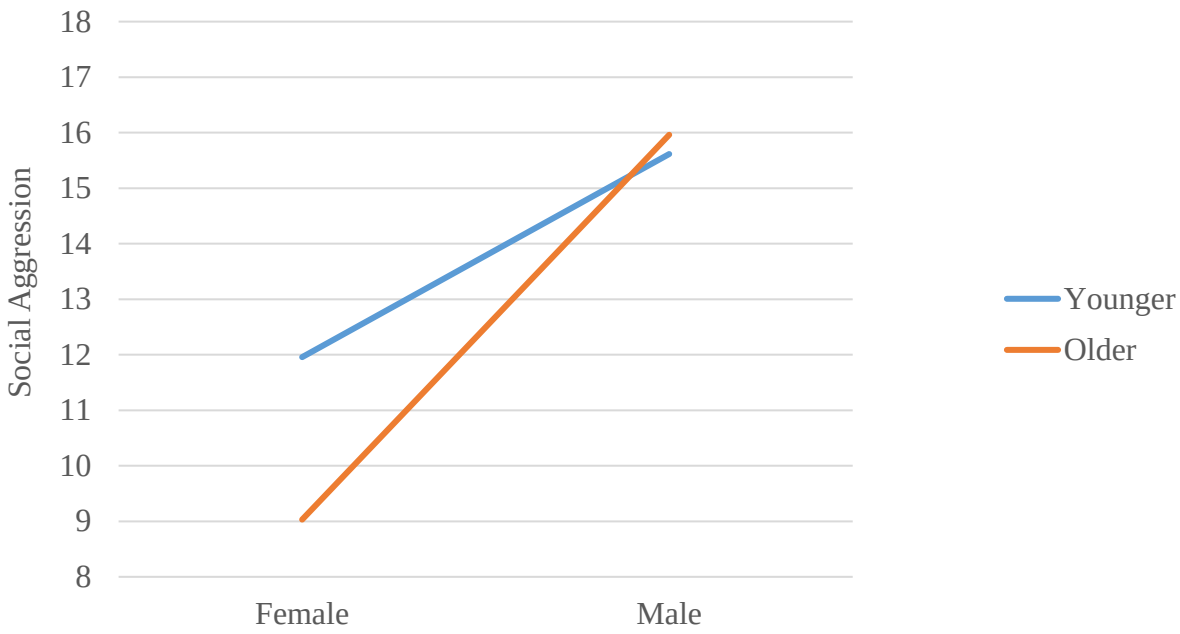


Figure 4: Predicting teacher reports of social aggression in adolescence as a function of child sex, pubertal stage, and their interaction

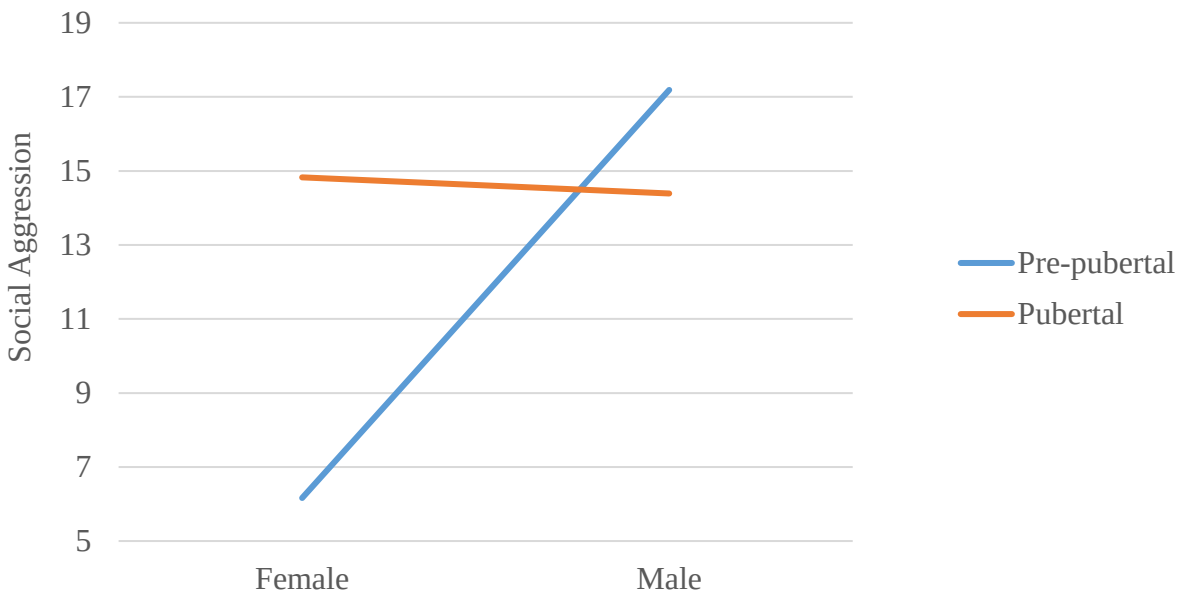


Figure 5: Moderation of the etiology of maternal reports of social aggression by sex in middle childhood

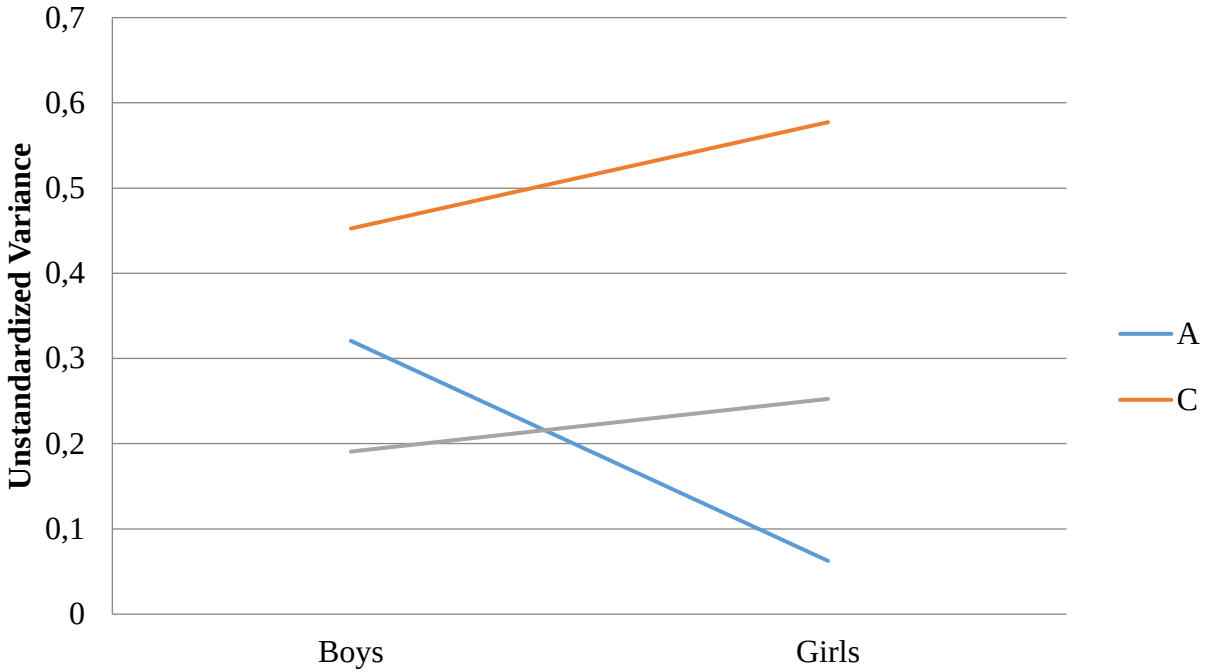
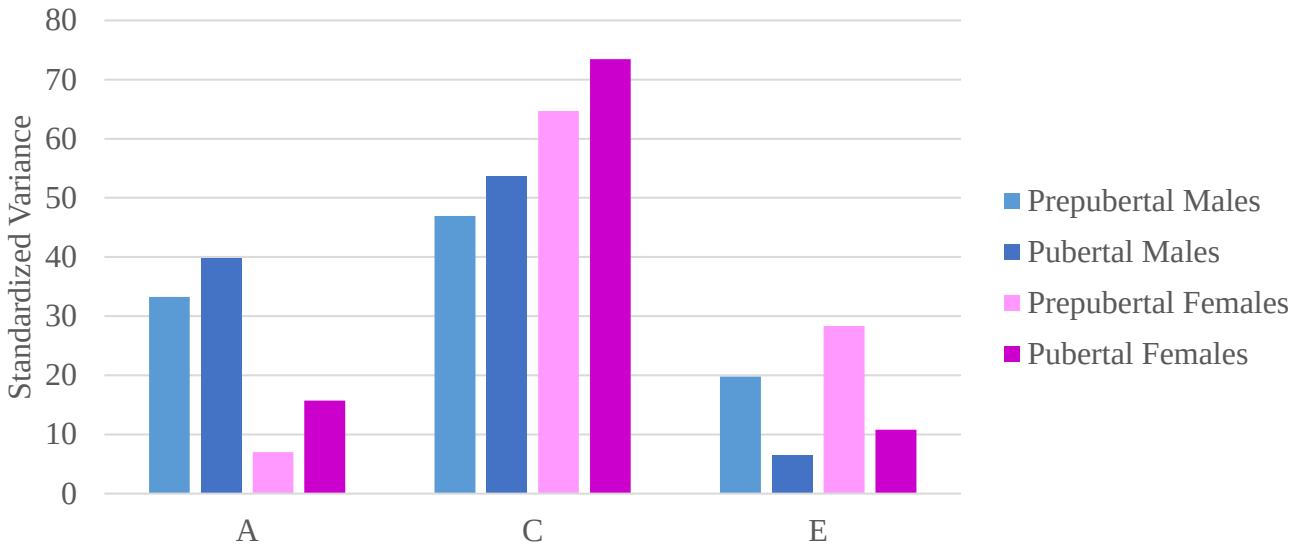


Figure 6: The etiology of maternal reports of social aggression by sex and pubertal stage



Note: A, C, and E represent genetic, shared, and non-shared environmental influences, respectively, on social aggression.

CHAPTER 3: NEUROIMAGING ANALYSES

INTRODUCTION

Social aggression is a form of antisocial behavior (Burt et al., 2012) in which social relationships and social status are used to damage reputations and inflict emotional harm on others, and includes behaviors such as gossiping, ostracism, and threatening to end a friendship. Although socially aggressive behaviors are legal and relatively typical during particular developmental periods, they have been associated with pathological outcomes in both victims and aggressors. Victims of social aggression often experience as much emotional distress as victims of physical aggression, including emotional and social difficulties such as peer rejection, loneliness, and internalizing problems (Crick et al., 2002). The perpetrators of social aggression also exhibit a number of maladaptive outcomes, including poor quality friendships marked by conflict and instability, depression, externalizing behavior, and borderline personality disorder features. Not surprisingly then, there is surging interest in understanding the developmental origins of social aggression. Although social aggression is most common during adolescence, there is evidence that it is perpetrated across the lifespan. Indeed, relational aggression has been observed in preschoolers as young as 30 months old (Crick et al., 2006) and in the elderly residing in assisted living facilities as old as 100 years (Trompetter, Scholte, & Westerhof, 2011). Furthermore, there are important mechanisms theoretically involved in the development of social aggression, including biobehavioral, cognitive, emotional, and social processes.

Frick and Morris (2004) identified a promising potential pathway to aggression, such that dysregulated negative emotions, especially anger and frustration, may serve as risk factors for aggressive behavior. From this perspective, uncontrolled angry reactions may increase the likelihood that an individual will react to real or perceived provocation aggressively. Further,

Frick and Morris (2004) suggested that the inability to regulate exaggerated negative emotional responses may also interfere with the development of social-cognitive and social skills necessary to inhibit physically aggressive behaviors (Frick & Morris, 2004).

Negative emotionality and poor regulation of negative affect have been implicated in the development of social aggression across the lifespan. In a person-centered, longitudinal study, anger was associated with increases in relational aggression over the course of four months in preschoolers (Ostrov et al., 2013). In older children and adolescents, social and relational aggression have been linked to increased hostility, angry responses to provocation, and the inability to tolerate anger and frustration (Little et al., 2003; Musher-Eizenman et al., 2004; Marsee & Frick, 2007). Anger and hostility have also been associated with relational aggression against romantic partners in adulthood (Murray-Close et al., 2010). Importantly, social and relational aggression have been uniquely associated with the regulation of anger and frustration rather than general emotion regulation deficits (Ostrov et al., 2013). These findings are consistent with developmental psychopathological theory which suggests that the ability to manage negative emotional reactions is a key developmental task that improves throughout childhood, and a failure to develop these skills is broadly associated with maladaptive behavioral and emotional patterns (Izard et al., 2007).

The amygdala is the neural structure that has received the most empirical attention in regards to emotion generation (Sabatinelli et al., 2011; Mechias et al., 2010; Costafreda et al., 2008; Murphy et al., 2003; Wager et al., 2008; McHugh et al., 2014; McNally et al., 2011; Furlong et al., 2010; Spoormaker et al., 2011; Cauda et al., 2012; Menon, 2011; Beissner et al., 2013). It is a subcortical structure in the limbic system that it is theorized to regulate the detection and appraisal of affective stimuli broadly, but there is evidence that it is more

responsive to aversive stimuli (e.g., angry or fearful facial expressions) than positive stimuli (e.g., rewards; Cunningham et al., 2012; Cunningham, Van Bavel, & Johnsen, 2008; Neta & Whalen, 2011; Vuilleumier & Purtois, 2007; Whalen et al., 2004). In fMRI studies investigating neural activation patterns associated with emotion generation, there is robust evidence that physical aggression is associated with exaggerated amygdala reactivity to emotionally salient stimuli (e.g., Herpertz et al., 2001; Donegan et al., 2003; Koenigsberg et al., 2007; Minzenberg et al., 2007; Buades-Rotger et al., 2016). For example, Coccaro and colleagues (2007) reported that impulsive physical aggression was associated with increased amygdala activation in response to angry faces in patients with intermittent explosive disorder. Additionally, Kramer et al. (2011) found that individuals who behaved aggressively on a Taylor Aggression Paradigm task exhibited greater amygdala activation on the task. Critically, however, researchers have yet to examine neural activation patterns (e.g., amygdala reactivity) associated with the perpetration of social aggression, despite the central role ascribed to emotional processes in its perpetration.

Furthermore, the amygdala is not a fully uniform structure, and its nuclei can be subdivided into two broad regions, the basolateral (BL) and centromedial (CM), based on distinct cellular architecture and differing patterns of neural connectivity (Davis & Whalen, 2001; LeDoux, 2007), and these two regions may function or respond differently to stimuli (Mosher, Zimmerman, & Gothard, 2010). Specifically, the nuclei in the BL region have more connections to sensory and neocortical areas, particularly the temporal and parietal lobes, and therefore it is hypothesized that their primary function is to evaluate the emotional significance or context of sensory inputs and social cues, such as facial expressions (McDonald, 1998; Stefanacci & Amaral, 2002; LeDoux, 2007; Mosher, Zimmerman, & Gothard, 2010). Oppositely, the nuclei in the CM region have more connections to subcortical structures, such as

the diencephalon and brainstem, and therefore it is hypothesized that their primary function is to focus attention on significant stimuli and initiate stimuli-appropriate autonomic responses in general (McDonald, 1998; Stefanacci & Amaral, 2002; LeDoux, 2007; Mosher, Zimmerman, & Gothard, 2010).

What's more, the differential function and reactivity of these two distinct subregions of the amygdala could mean that they may each play a unique role in the perpetration of aggressive behavior, and, furthermore, given its association with the emotional salience and social relevance of stimuli, the reactivity and connectivity of the BL region may be particularly important for our understanding of social aggression specifically. Indeed, evidence from non-human mammals suggest that the reactivity and co-reactivity of specific subregions of the amygdala may be differentially associated with distinct types of aggression, such that medial regions have been implicated in rivalry aggression, central regions in predatory aggression, and both regions in abnormally violent attacks, such maternal aggression (Haller, 2018). Although studies have not yet investigated how the BL and CM regions may be differentially associated with social aggression in humans, there is emerging evidence suggesting that the differential activation and dysfunction of amygdala subregions is associated with individual psychopathic personality traits, trait anger, and delinquency (Carré, Fisher, Manuck, & Hariri, 2012; Moul, Zimmerman, & Gothard, 2012; Carré, Hyde, Neumann, et al., 2013; Hyde, Shaw, Murray, Gard, et al., 2016).

Although research has not yet been conducted on the association between social aggression and amygdala reactivity, and emerging body of work has linked social aggression and sympathetic nervous system (SNS) activation. Further, prior research suggests that the amygdala triggers sympathetic nervous system activation via projections from its central nucleus to the lateral hypothalamus which prompt strong sympathetic activation in terms of tachycardia, blood

pressure elevation, pupil dilation, and increases in skin conductance (Weymar & Schwabe, 2016). Exaggerated SNS reactivity is hypothesized to reflect dysregulated emotional reactions to stress (e.g., anger), thus increasing risk for aggressive responses (Murray-Close et al., 2013). Indeed, research has provided some evidence that exaggerated stress reactivity is related to social aggression. For example, Murray-Close and Crick (2007) found that heightened systolic blood pressure was associated with social aggression, especially in in girls. Likewise, heightened SNS reactivity to stress has been associated with social aggression against romantic partners in a study of female college students (Murray-Close, 2011). Therefore, the exaggerated SNS reactivity to stress seen in socially aggressive individuals may reflect dysregulated emotional responses that could be assessed via amygdala reactivity in these same individuals.

In short, there is a clear need for an investigation of neural activations patterns associated with the perpetration of social aggression. The current study aims just to do this by examining the association between social aggression and amygdala reactivity during a socio-emotional face processing task. Critically, amygdala reactivity will be examined for the total structure and BL and CM subregions separately to investigate whether differential activation is associated with social aggression. Furthermore, moderation models that integrate sex and pubertal development will be examined in order to explore whether these additional risk factors influence the relationship between social aggression and amygdala reactivity. Finally, we will examine the incremental validity of social aggression in predicting amygdala reactivity over physical aggression, as there is fMRI evidence for greater amygdala reactivity to angry faces in physically aggressive individuals.

SPECIFIC AIMS

This study aims to examine the association between social aggression and amygdala reactivity when viewing faces with varying affective expressions for the very first time. We will also evaluate sex and pubertal status as moderators of our findings, and compare these results to those for physical aggression, about which far more is known. The following aims guide this study:

SA1: Determine the association between social aggression and amygdala reactivity**.**

Given the evidence that the perpetration of socially aggressive behaviors may reflect dysregulated emotional responses to stressful stimuli, we predict that (1) social aggression will be positively associated with increased total amygdala reactivity to emotional (e.g., angry and/or fearful) faces as compared to shapes. However, given emerging evidence regarding differential functioning of amygdala subregions in antisocial behavior (Buades-Rotger, Engelke, & Krämer, 2019; Hyde, Shaw, Murray, Gard, et al., 2016), we hypothesize that (2) social aggression will also be positively associated with increased BL amygdala reactivity to emotional faces as compared to shapes, but (3) negatively or not associated with increased CM amygdala reactivity to emotional faces as compared to shapes.

Specifically, BL amygdala reactivity is theorized to contribute to the evaluation of the emotional significance of social cues, including facial expressions (McDonald, 1998; Stefanacci & Amaral, 2002; LeDoux, 2007; Mosher, Zimmerman, & Gothard, 2010), and empirical findings from rodent studies implicate a corresponding amygdala subnucleus in the perpetration of social rivalry aggression (Haller, 2018). Therefore, it is possible that increased reactivity of the BL region of the amygdala may be particularly relevant for social aggression due its relational nature. In contrast, CM amygdala reactivity is theorized to contribute to focusing attention and

initiating stimuli-appropriate autonomic responses (McDonald, 1998; Stefanacci & Amaral, 2002; LeDoux, 2007; Mosher, Zimmerman, & Gothard, 2010) and, although current empirical evidence is mixed, evidence from a similar young, at-risk sample suggests that CM reactivity might be negatively associated with antisocial behavior (Hyde et al., 2016).

SA2: Explore potential sex and pubertal differences in the association between social aggression and amygdala reactivity.

Building on prior behavioral research highlighting negligible-to-small sex differences in social aggression, we hypothesize that the above associations between social aggression and amygdala reactivity will be largely constant across sex. However, pubertal stage has been positively associated with increased amygdala reactivity in prior research (e.g., Pagliaccio et al., 2015), so we will also explore whether pubertal stage and sex jointly moderate the associations between social aggression and amygdala reactivity.

SA3: Explore the incremental validity of social aggression in predicting amygdala reactivity over physical aggression.

Prior fMRI studies have reported greater amygdala reactivity to angry faces in physically aggressive individuals. Empirical research also suggests that social and physical aggression are often highly intercorrelated, such that prior meta-analytic work has shown that aggressive individuals typically perpetrate both forms of aggression (r = 0.76; Card et al., 2008). Therefore, if social aggression is also positively associated with amygdala reactivity to emotional (e.g., angry and/or fearful) faces, it will be important to determine whether social aggression has incremental validity in predicting unique variance in amygdala reactivity above and beyond physical aggression.

METHODS

Participants

Participants consisted of twin families who participated in the Michigan Twin Neurogenetics Study (MTwiNS), which is a longitudinal study of brain and behavior development within the Michigan State University Twin Registry (MSUTR; Klump & Burt, 2006; Burt & Klump, 2013). Children gave informed assent, while parents gave informed consent for themselves and their children (N=398). Twins were 43.6% female and ranged in age from 7 to 18 years-old (mean age (SD) = 13.99 (2.37)). Twins' racial and ethnic background was provided by their parents (77.5% non-Hispanic White, 14.5% African American/Black, 2.2% Hispanic, 1.1% Asian, 0.7% Native American, and 4.0% multiracial or other ethnic groups). Twin zygosity was determined via parent report using a standard 5-item questionnaire that assesses within-pair physical similarity and is over 95% accurate (Peeters et al., 1998). Monozygotic twins constituted 39.6% of the pairs (n = 109 pairs (44 female pairs)), same-sex dizygotic twins constituted 40.0% (n =110 pairs (48 female pairs)), and opposite-sex dizygotic twins constituted 20.4% (n = 56 pairs).

Study Procedure.

Youth and their primary caregivers (95% biological mothers) participated in a day-long protocol that included questionnaires, parent-child interaction tasks, collection of biological samples, an MRI mock scanning session, and an MRI scan. Twin pairs were randomized to determine which twin participated in the protocol activities first (except for the questionnaires, which were completed throughout the day and split by twin so the caregiver would not confuse which youth was being rated). Parents and youth consented to participate in the study (minors

provided informed assent), and the study protocol was approved by the Institutional Review Board at the University of Michigan.

Measures

Social and physical aggression was assessed via self-, parent-, and teacher-report using the Subtypes of Antisocial Behavior Questionnaire (STAB; Burt & Donnellan, 2009). The STAB is a 32-item measure assessing three major dimensions of antisocial behavior, two of which are social and physical aggression. The Social Aggression Scale (SA) includes 11 behaviors (e.g., gossips, gives others the silent treatment, excludes others from group activities) and the Physical Aggression Scale (PA) includes 10 behaviors (e.g., hits others, gets into physical fights, angers easily). For each, participants report on the frequency with which they commit each behavior using a scale that ranges from 1 (never) to 5 (nearly all the time). Prior work has confirmed the factor structure of the STAB in multiple samples (Burt & Donnellan, 2009, 2010), and provided consistent support for its criterion-related validity. Namely, the STAB scales (1) converge with other measures of antisocial behavior and criminal convictions, (2) show expected patterns of mean differences across treatment groups of adjudicated adults, and (3) correlate as expected with measures of personality (Burt & Donnellan, 2009). Similarly, a study using experience sampling methodology (i.e., participants reported on specific momentary behaviors six times a day while living in their natural environments), found that high scores on the STAB PA and SA scales were uniquely associated with momentary reports of physically and socially aggressive behaviors, respectively (Burt & Donnellan, 2010).

For the SA scale, maternal reports were available for 99.1% of the twins ($\alpha = .87$), teacher reports were available for 50.0% of the twins ($\alpha = .94$), and self-reports were available for 98.0% of the twins ($\alpha = .83$). For the PA scale, maternal reports were available for 98.9% of

the twins (α = .86), teacher-reports were available for 49.3% of the twins (α = .94), and self-reports were available for 96.5% of the twins (α = .84).

Pubertal development was assessed via maternal-report using the Pubertal Development Scale (PDS; Peterson et al., 1988). The PDS is a 5-item measure assessing major indices of pubertal growth by sex. Parents report on growth spurt (i.e., height), body hair (e.g., underarm and/or pubic hair), and skin changes (e.g., pimples) for all children. For boys, they also report on voice changes (i.e., deepening) and facial hair growth. For girls, they additionally report on breast development and onset of menarche. Participants respond to each item except menarche using a scale that ranges from 1 (not yet started) to 4 (seems complete). Menarche is coded dichotomously (i.e., yes or no).

Pubertal stages were calculated using the scoring algorithms described in Crockett (1988). For boys, body hair growth, voice change, and facial hair growth item responses are summed and this sum score is used to categorize participants into one of the five standardized pubertal development stages (Tanner, 1962), which range from pre-pubertal (Stage 1) to post-pubertal (Stage 5). For girls, body hair growth and breast development item responses are summed and this sum score and menarche are used to categorize participants into one of the same pubertal stages described above. Importantly, menarche is necessary for girls to be categorized as late pubertal (Stage 4) or post-pubertal (Stage 5).

Prior work has confirmed the reliability and validity of the PDS (Carskadon & Acebo, 1993) and provided consistent support for its criterion-related validity. Namely, PDS categorization converges with other indices of pubertal status, including similar self-report questionnaires, picture-based interviews, physical exams, levels of basal hormones responsible for advancing pubertal development (i.e., testosterone and dehydroepiandrosteron), and onset of

romantic and sexual activities (e.g., Bond et al., 2006; Chan et al., 2010; Shirtcliff, Dahl, & Pollak, 2009; Skoog et al., 2013).

PDS data were available for 97.5% of female twins (n = 234) and 97.7% of male twins (n = 303; 97.6% of all twins, n = 537).

fMRI Face Processing task

The Emotional Faces Task is a widely-used paradigm designed to robustly engage the amygdala and ventromedial PFC during socio-emotional processing (Carre et al., 2013; Hyde et al., 2014). This task consists of four blocks of a face-processing task interleaved with five blocks of a sensorimotor control task. Participant performance (accuracy and reaction time) was monitored during all scans. During the face-processing blocks, participants saw a trio of faces arranged in a triangular formation and chose which of the two faces presented in the bottom row was identical to the target face in the top row. Each face processing block was balanced for sex (i.e., male or female) and race (i.e., White or Black). Stimuli were made up of the NimStim Face Stimulus Set, which is a well-validated set of 646 facial expression stimuli used in emotion recognition studies (Tottenham et al., 2009). Each of the four face processing blocks consisted of a different emotional facial expression (i.e., anger, fear, happy, neutral), and participants were randomly assigned to one of four different orders of block presentation. During the sensorimotor control blocks, participants saw a trio of simple geometric shapes (i.e., circles, squares, or triangles) arranged in a triangular formation and chose which of the two shapes presented in the bottom row was identical to the target shape in the top row. In the face processing blocks, each of the 18 face trios was presented for 2 seconds with a variable inter-stimulus interval of 2 to 6 seconds (*M* = 4s) for a total block length of 98 seconds. A variable ISI was used to minimize expectancy effects and resulting habituation, as well as to maximize amygdala reactivity

throughout the paradigm. In the sensorimotor control blocks, each of the 12 shape trios was presented for 2 seconds followed by a fixation cross for 0.5 seconds, for a total block length of 30 seconds. An additional 4 seconds of crosshair presentation followed each block. Total task time was 578 seconds.

Imaging Data Acquisition

As described in Tomlinson et al. (2020), Suarez et al. (2022), and Michael et al. (2023), prior to the actual MRI scan, each adolescent participated in a "mock scan" during which they experienced what the scanner looks and sounds like and practiced lying motionless with the assistance of a motion compliance system. Head movement was minimized through (a) instructions to the participant and (b) padding and pillows placed around the head. Each participant was scanned with one of two research-dedicated GE Discovery MR750 3T scanners. To take advantage of improvements in MRI data acquisition and harmonize our protocol with the Adolescent Brain Development Cognitive Development Study (Casey et al., 2018), we altered our acquisition protocol after the first 140 families. For the first 140 families, one run of 298 volumes was collected for each participant. Blood oxygenation level–dependent (BOLD) functional images were acquired with a reverse spiral sequence (TR/TE=2000/30 milliseconds, flip angle = 90°, FOV = 22cm), which covered 43 interleaved oblique slices of 3-mm thickness. High-resolution T1-weighted SPGR images (156, 1mm-thick slices) were aligned with the AC-PC plane, and later used during normalization of the functional images. For the remaining 100 families, one run of 730 volumes was collected for each participant. BOLD functional images were acquired with a gradient-echo sequence with multiband acquisition (TR/TE=800/30 milliseconds, flip angle = 52°, FOV = 21.6cm), which covered 742 interleaved axial slices of 2.4-mm thickness. High-resolution T1-weighted SPGR images (208, 1mm-thick slices) were

aligned with the AC-PC plane, and later used during normalization of the functional images. For both acquisition sequences, BOLD functional images encompassed the entire cerebrum and most of the cerebellum to maximum coverage of limbic structures.

Imaging Data Processing

As described in Tomlinson et al. (2020), Suarez et al. (2022), and Michael et al. (2023), preprocessing for both acquisition sequences was identical, unless otherwise specified. Functional data were preprocessed using Statistical Parametric Mapping version 12 (SPM12; Wellcome Trust Centre, London, United Kingdom). Raw k-space data from reverse-spiral sequence acquisition were de-spiked before reconstruction to image space. For gradient-echo sequence data with multiband acquisition only, task-specific field maps are constructed from volumes of both anterior-to-posterior and posterior-to-anterior phase encoding; field maps are applied after image construction to reduce spatial distortions and minimize movement artifacts. Slice timing correction was performed using the 23rd slide as the reference slice (reverse-spiral data) or the 2nd slice of each 10-slice band (gradient-echo data with multiband acquisition). Data from both acquisition sequences were then spatially realigned to the 10th slice of the volume. These spatially realigned data were coregistered to the high-resolution T1-weighted image, and segmented and spatially normalized into standard stereotactic space (MNI template). Finally, functional data were smoothed to minimize noise and residual difference in gyral anatomy with a Gaussian filter set at 6mm FWHM. After preprocessing, the Artifact detection Tools (ART) software package (http://www.nitrc.org/projects/artifact_detect/) was used to detect global mean intensity and translation or rotational motion outliers (> 4.5 SD from the mean global brain activation, >2mm movement or 2^{o} translation in any direction); for each participant, nuisance covariates were created and included in the individual-level model for all volumes exhibiting one

of these criteria. Additionally, because of the relatively extensive signal loss typically observed in the amygdala, single-subject BOLD fMRI data were only included in subsequent analyses if there was a minimum of 90% signal coverage in the amygdala bilaterally, defined using the Automated Anatomical Labeling (AAL) atlas in the WFU PickAtlas Tool, version 1.04 (Maldjian, Laurienti, Kraft, & Burdette, 2003).

Missing data

Of the 550 youth in the current sample, usable fMRI data was available for 398 (72%) participants. See Table 11 for a summary of available fMRI data for analyses. Independent samples t-tests revealed that youth with valid imaging data did not differ (all *p*s > .10) from youth without valid imaging data on youth characteristics (i.e., chronological age, pubertal status, sex, or race/ethnicity) or primary caregiver characteristics (i.e., education or annual income).

fMRI analysis

As described in Suarez et al. (2022), the general linear model of SPM12 was used to estimate condition-specific (e.g., fearful faces > shapes) blood oxygen level–dependent (BOLD) activation for each individual scan. Individual contrast images were then used in second-level random effects models to determine mean expression-specific reactivity using one-sample t-tests. As our goal was to examine amygdala reactivity to specific contrasts, the following were estimated and mean cluster values were extracted from SPM12: (1) fearful facial expressions > shapes, (2) angry facial expressions > shapes, and (3) fearful and angry faces > shapes to measure neural reactivity to interpersonal distress and threat (Whalen et al., 2001), which have been implicated in antisocial behavior (Hyde et al., 2013). We also examined (4) neutral faces > shapes because recent studies show similar amygdala responses to unmasked neutral faces and

other expressions of threat (Marusak, Carre, & Thomason, 2013; Maursak et al., 2016), suggesting that ambiguity may also be interpreted as threatening (Pollak et al., 2000).

Consistent with prior research examining the role of amygdala reactivity during an equivalent face processing task (Hyde, Shaw, Murray, Gard, et al., 2016; Suarez, 2022), amygdala subregions were defined using maximum probability maps of cytoarchitectonic boundaries developed by Amunts et al (2005) and implemented through the SPM Anatomy Toolbox (Eickhoff et al., 2005). Contrast-specific BOLD parameter estimates were extracted from these subregions, and those that survived the family wise error correction of $p < .05$ were examined (Table 12). Notably, we also attempted to examine contrasts between fearful/angry facial expressions and neutral facial expressions (e.g., fearful faces > neutral faces), but we did not observe main effects for these contrasts at any correction threshold. Similarly, we did not observe main effects for the neutral faces > shapes contrast in the bilateral centromedial region of the amygdala at any correction threshold.

Statistical analyses

Hierarchical linear regression was used to test whether amygdala reactivity varied as a function of social aggression, sex, pubertal stage, physical aggression, or their interactions. Analyses were conducted via multi-level modeling, in which the twins were nested within their family unit, to control for the non-independence of the observations. Given the novel and exploratory nature of this investigation, each step of each hierarchical regression is reported so that all nested regression models can be examined. Results are presented separately for each informant due to significant differences in informant reports of social aggression. We focus on estimates that survived Bonferroni correction for multiple comparisons (p = .05 / 72 regressions = adjusted p value of .0069).

RESULTS

Descriptive Statistics and Correlations

Descriptive statistics and correlations are presented in Table 1. There were significant differences in informant reports of social aggression, such that both twins ($t(195) = 3.18$, $p = 0.002$)) and their mothers ($t(193) = 4.16$, $p < 0.000$) rated the twins as more socially aggressive than did the twins' teachers. However, there were no significant differences between maternal-report and twin self-report of social aggression ($t(319) = -0.99$, $p = 0.324$). This suggests that mothers and their children report comparable levels of social aggression, and their reports indicate higher levels of twin social aggression than do teacher reports.

There were no sex differences in the perpetration of social aggression according to any informant. Age was positively associated with maternal reports of social aggression and participant sex, such that female participants tended to be older than male participants. Pubertal stage was positively associated with maternal reports of social aggression in girls and self-reports of social aggression in both boys and girls, but negatively associated with teacher reports of social aggression in boys. Pubertal stage was also positively associated with age and sex, such that older participants were more developed than younger participants and female participants were more developed than male participants. A complete breakdown of frequency of pubertal stages is presented separately by sex in Figure 2. The majority of participants are in the middle to later stages of pubertal development, but more girls are in the final two stage than males.

Social aggression, sex, pubertal stage, and amygdala reactivity

Zero-order correlations (Tables 13-15) indicated that social aggression was largely unrelated to amygdala reactivity to angry, fearful, or neutral facial expressions. However, self-reports of social aggression were associated with decreased right basolateral amygdala reactivity

to neutral faces (r = -.181). Participant sex and pubertal stage were also not associated with amygdala reactivity.

Hierarchical linear regression models (Tables 16-18) similarly revealed that social aggression was largely unrelated with amygdala reactivity to angry, fearful, or neutral facial expressions. However, there was a significant interaction between maternal-reports of social aggression and sex on right bilateral total amygdala reactivity to angry faces (Table 16), such that there was an inverse relationship between sex and social aggression (Figure 7), but this observation did not survive Bonferroni correction (p = .03).

Social aggression, physical aggression, and amygdala reactivity

Zero-order correlations (Table 13-15) indicated that physical aggression was also largely unrelated to amygdala reactivity to angry, fearful, or neutral facial expressions. However, self-reports of physical aggression were associated with decreased right basolateral amygdala reactivity to neutral faces (r = -.148).

Hierarchical linear regression models (Tables 19-21) similarly found that physical aggression was not consistently related to amygdala reactivity to angry, fearful, or neutral facial expressions. Self-reported physical aggression was predictive of increased right basolateral amygdala reactivity to fearful faces (Table 20), but this observation did not survive Bonferroni correction (p = .019). Additionally, there was a significant interaction between self-reported social and physical aggression on right basolateral amygdala reactivity to fearful faces (Table 20), such that the effect of physical aggression on amygdala reactivity is strongest in those who are low in social aggression (Figure 8), but this observation also did not survive Bonferroni correction (p = .028).

DISCUSSION

The overarching aim of this study was to investigate the associations between social aggression and amygdala reactivity when viewing emotional faces. A secondary aim was to further investigate whether activation in amygdala subregions is differentially associated with social aggression. We also evaluated sex and pubertal status as moderators of our findings. Results suggested that social aggression, sex, and pubertal stage were not associated with either total nor subregion amygdala reactivity during this socio-emotional face processing task. Notably, this was just the first study to examine neural activation patterns that may be associated with social aggression. However, these findings were not consistent with prior work that did observe associations between pubertal stage and physical aggression and amygdala reactivity to emotional faces (e.g., Pagliaccio et al., 2015; Coccaro et al., 2007; Carré et al., 2012)

These null results were surprising given the hypothesized importance of social information processing and emotional reactivity to social situations and stressors among socially aggressive individuals (Crick, Grotpeter, & Bigbee, 2002). As previously mentioned, an emerging body of work has linked social aggression and sympathetic nervous system (SNS) activation. Heightened systolic blood pressure has associated with social aggression, especially in in girls (Murray-Close and Crick, 2007). Likewise, heightened SNS reactivity to stress has been associated with social aggression against romantic partners in a study of female college students (Murray-Close, 2011). Exaggerated SNS reactivity is hypothesized to reflect dysregulated emotional reactions to stress (e.g., anger), thus increasing risk for aggressive responses (Murray-Close, 2013). However, these investigations did not specifically examine SNS reactivity to an in vivo socio-emotional processing task. As discussed below, alternative

socio-emotional tasks may be better suited for capturing emotional reactivity and/or emotional dysregulation in socially aggressive individuals.

Furthermore, it remains possible that other neural processes associated with pubertal development may predict social aggression. Most notably, neural connectivity may be a particularly promising avenue for increasing our understanding of the neural correlates of social aggression. While no study to date has examined associations between social aggression and functional or structural connectivity, there is increasing evidence that puberty plays an important role in the structural and functional brain development seen in adolescence, including changes in functional connectivity.

Resting-state functional connectivity studies have found that that while functional connectivity between spatially distant, functionally-related brain regions increases between childhood and adulthood, connectivity between more spatially proximal regions decreases (Qin et al., 2015; Vogel et al., 2010; Dosenbach et al., 2010, Fair et al., 2008, Fair et al., 2009). Furthermore, developmentally-informed studies investigating functional connectivity during social cognition tasks suggest that there are age-related increases in functional connectivity in face processing networks and related social brain regions between childhood and adulthood. For example, Shaw et al., (2011) observed adolescent age-related increases in task-dependent functional connectivity between action observation and social brain regions (e.g., between the TPJ, left OFC, IFG, and insula) during observation of angry versus neutral hand gestures in a longitudinal fMRI study. Similarly, Klapwijk et al., (2013) observed increased functional connectivity in the mentalizing network (e.g., between the DMPFC, right pSTS, and right TPJ) during social relative to basic emotion processing as a function of estradiol levels and pubertal stage in adolescent girls. Structural connectivity research indicates that white matter volume

increases in the frontal cortex between 9 and 14 years (Blakemore, 2012) and that many white-matter tracts only reach mature levels of integrity after puberty (Asato et al., 2010).

These potentially puberty-induced changes in connectivity may be especially relevant for the perpetration of social aggression. Early theories regarding social aggression suggested girls engage in more sophisticated and covert aggressive behaviors than boys due to their earlier cognitive maturation (Björkqvist et al., 1992; Keenan & Shaw, 1997; Silverthorn & Frick, 1999). More recent researchers have fine-tuned this cognitive maturation hypothesis, stating that increases in social aggression in late childhood and adolescence is, in part, due to improvements in cognition and social abilities that are a result of neural maturation processes during these developmental periods (Baird, Silver, & Veague, 2010).

Because social aggression is associated with advanced social-cognitive skills (e.g., theory of mind), it may also be associated with greater connectivity between the bilateral temporoparietal junction (TPJ), medial prefrontal cortex (mPFC), and right posterior superior temporal sulcus (pSTS). However, as previously discussed, social aggression is also associated with affective deficits (e.g., emotional reactivity and dysregulation), so it may be associated with decreased connectivity between the amygdala and the dorsolateral (dlPFC) and ventrolateral (vlPFC) regions of the prefrontal cortex. Further, a core assumption of the developmental psychopathology approach is that etiology is due not only to specific vulnerabilities and risk factors, but also to their interactions (Beauchaine & Gatzke-Kopp, 2012; Beauchaine & Gatzke-Kopp, 2013; Rutter et al., 2006). Therefore, while heightened social-cognitive abilities, such as theory of mind, are likely important for the perpetration of social aggression, these advanced skills are more likely to be predictive of social aggression in individuals who exhibit impairments in other domains or in those who do not master salient developmental tasks, such as

emotion regulation. As such, future research investigating neural correlates of social aggression should explore both functional and structural connectivity of a variety of neural networks implicated in social aggression.

Limitations

One possible major limitation of the current study is the paradigm used to elicit amygdala reactivity. By definition, social aggression exploits social relationships and social status in order to damage reputations and inflict emotional harm on others. Socially aggressive behaviors are typically dyadic or group-level transgressions, such gossiping, ostracism, and threatening to end a friendship. As such, the emotional face processing task used here may not elicit the same reactions that a socially aggressive individual would have in a dynamic social context. Indeed, the studies that observed pubertal differences in functional connectivity employed tasks that may be better suited for social aggression, including reading a story or watching a video in which a person commits a social transgression (Shaw et al., 2011; Klapwijk et al., 2013). To test this, future research should utilize tasks that feature social dynamics and transgressions. One task that may be especially effective in eliciting an emotional reaction in socially aggressive individuals is the O-Cam, which consists of a simulated Web conference during which participants are either ostracized or included by two pre-taped confederates (Godwin et al., 2014; Goodacre & Zadro, 2010).

Despite this limitation, the current study is a critical step forward in advancing our understanding of the roles of neural mechanisms associated with social aggression in adolescence. Our investigation included a large sample size of diverse youth and our null findings persisted across multiple informants. Future studies should continue to explore the associations between social aggression, puberty, sex, and neural activation and connectivity in

order to increase our understanding of biological developmental processes implicated in the etiology of social aggression.

TABLES

Table 11: Summary of available fMRI data for analyses

	Spiral Sequence		Echo-planar Sequence with Multiband Acquisition	
	Number Lost	Participants with data	Number Lost	Participants with data
Original sample		238		270
- Refused scan	16		18	
- Medical restriction*	25		12	
- Data collection/scanner error	2		1	
- Partial data	0		3	
Total lost	43			34
Sample with imaging data		195		236
- Whole brain coverage loss by visual inspection	2		1	
- Low amygdala coverage (< 90%)	0		6	
- Low task performance (< 70%)	11		11	
- Ghosting on functional scans	1		1	
- Exceeded movement thresholds	0		0	
Total lost	14			19
Sample with usable imaging data		181		217

Note: *includes having a permanent retainer or braces, non-MRI safe implanted medical devices, having BBs/pellets or other non-removable metal inside of body, recent surgery, exceeds table weight limit, impaired vision not correctable with MRI-safe glasses. Note that an additional 21 families (42 twins) received an earlier version of the task that was not comparable to the current version; these participants were excluded from all analyses.

Table 12: Main effects of the fMRI implicit emotion processing task, not controlling for scanner sequence

Contrast	**Bilateral Amygdala (x,y,z), *t* extent threshold, *k* cluster size**
Fear > shapes	Left*±: (-30,2,-22) t = 5.48, *k* = 139 Right*±: (20,-4,-16) t = 6.97, *k* = 139
Fear < shapes	no suprathreshold clusters
Anger > shapes	left*±: (-26,0,-18) *t* = 8.12, *k* = 168 Right*±: (22,-2,-16) *t* = 7.83, *k* = 190
Fear + anger > shapes	Left*±: (-28,-4,-20) *t* = 9.03, *k* = 165 Right*±: (20,-4,-16) *t* = 10.39, *k* = 204
Fear + anger < shapes	no suprathreshold clusters
Neutral > shapes	Left*±: (-20,0,-18) *t* = 4.79, *k* = 64 Right*±: (20,-2,-16) *t* = 5.14, *k* = 94
Neutral < shapes	no suprathreshold clusters

Note. *N* = 398. All effects were corrected for multiple comparisons at FWE $p < .05$ within a bilateral amygdala mask created using maximum probability maps developed by Amunts et al (2005) and implemented through the SPM Anatomy Toolbox (Eickhoff et al., 2005).
* indicates that the amygdala ROI results were also observed at whole brain correction FWE
± indicates that results were also observed at 3dClustSim voxelwise $p<.001$ to achieve a whole-brain correction of $p< .05$

Table 13: Zero-order correlations between social aggression, physical aggression, sex, pubertal stage, and bilateral total amygdala reactivity to facial expressions vs. shapes

	Fear		Anger		Anger + Fear		Neutral	
	Left	Right	Left	Right	Left	Right	Left	Right
Social Aggression								
Self Report	-0.016	-0.062	-0.022	0.012	-0.025	-0.034	-0.015	-0.068
Maternal Report	0.070	0.000	-0.020	0.010	0.044	0.004	0.018	-0.002
Teacher Report	0.023	0.073	-0.105	0.002	-0.062	0.058	-0.067	-0.135
Physical Aggression								
Self Report	0.034	0.052	-0.061	-0.009	-0.009	0.041	-0.063	-0.092
Maternal Report	0.038	0.041	-0.014	-0.017	0.022	0.015	-0.026	-0.016
Teacher Report	0.038	0.070	-0.090	-0.009	-0.042	0.055	0.006	-0.061
Pubertal Stage	0.058	-0.014	-0.073	-0.013	0.001	-0.015	-0.029	-0.011
Age	0.028	-0.050	-0.039	-0.036	-0.003	-0.056	0.004	0.017
Sex	-0.032	-0.050	-0.045	0.025	-0.053	-0.013	-0.064	-0.082

Table 14: Zero-order correlations between social aggression, physical aggression, sex, pubertal stage, and bilateral basolateral amygdala reactivity to facial expressions vs. shapes

	Fear		Anger		Anger + Fear		Neutral	
	Left	Right	Left	Right	Left	Right	Left	Right
Social Aggression								
Self Report	-0.018	-0.055	-0.009	0.031	-0.029	-0.010	-0.016	-0.035
Maternal Report	0.070	0.019	-0.025	0.010	0.049	0.009	0.008	0.014
Teacher Report	0.045	0.057	-0.124	-0.075	-0.049	0.004	-0.044	-.181*
Physical Aggression								
Self Report	0.028	0.056	-0.020	-0.017	0.004	0.039	-0.064	-0.096
Maternal Report	0.037	0.062	-0.011	-0.028	0.031	0.015	-0.030	-0.016
Teacher Report	0.069	0.082	-0.103	-0.087	-0.020	0.018	0.012	-.148*
Pubertal Stage	0.048	-0.013	-0.090	0.013	-0.011	0.012	-0.025	0.009
Age	0.004	-0.048	-0.083	-0.033	-0.050	-0.040	0.005	0.025
Sex	-0.022	-0.045	-0.014	0.092	-0.023	0.053	-0.054	-0.043

* $p < .05$

Table 15: Zero-order correlations between social aggression, physical aggression, sex, pubertal stage, and bilateral centromedial amygdala reactivity to facial expressions vs. shapes

	Fear		Anger		Anger + Fear	
	Left	Right	Left	Right	Left	Right
Social Aggression						
Self Report	0.039	0.035	0.000	-0.030	0.026	0.023
Maternal Report	0.050	0.012	0.047	0.003	0.059	0.004
Teacher Report	0.047	0.062	0.030	0.098	0.067	0.105
Physical Aggression						
Self Report	0.038	0.049	0.020	-0.031	0.043	0.027
Maternal Report	0.024	0.035	0.027	-0.023	0.038	0.001
Teacher Report	0.057	0.050	0.021	0.060	0.072	0.073
Pubertal Stage	-0.028	-0.033	-0.025	-0.010	-0.051	-0.029
Age	-0.045	-0.057	-0.049	-0.031	-0.070	-0.038
Sex	0.017	-0.068	0.034	0.074	0.035	-0.007

* $p < .05$

Table 16: Predicting bilateral total amygdala reactivity to facial expressions vs. shapes as a function of social aggression, sex, pubertal stage, and their interactions

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Self-Report	Step 1	Social Aggression	-.003	-.009	-.003	.000	-.003	-.004	-.002	-.011
	Step 2	Social Aggression	-.005	-.010	-.002	.000	-.003	-.004	-.001	-.012
		Sex	.059	.046	.030	.000	.043	.017	.052	.083
		Pubertal Stage	.078	.030	-.015	.013	.030	.018	-.001	.055
	Step 3	Social Aggression	.004	-.002	-.005	.000	.000	.000	-.011	-.017
		Sex	-.001	.013	.044	.001	.019	.002	.068	.100
		Pubertal Stage	.011	-.009	.000	.012	.004	.000	.028	.078
		Social Aggression x Sex	.012	-.004	.006	.006	.008	.001	.004	.001
		Social Aggression x Pubertal stage	-.014	-.014	.006	-.001	-.005	-.007	.019	.010
		Sex x Pubertal stage	.090	.053	-.024	-.004	.034	.022	-.026	-.027
	Step 4	Social Aggression	.003	-.003	-.003	-.001	.000	-.001	-.013	-.019
		Sex	.001	.015	.037	.003	.017	.003	.072	.106
		Pubertal Stage	.011	-.008	-.004	.013	.003	.000	.030	.082
		Social Aggression x Sex	.012	-.003	.003	.007	.007	.002	.005	.004
		Social Aggression x Pubertal stage	-.014	-.014	.004	.000	-.005	-.007	.020	.012
		Sex x Pubertal stage	.089	.052	-.019	-.006	.036	.022	-.029	-.032
		Social Aggression x Sex x Pubertal stage	-.001	-.001	.005	-.001	.002	.000	-.003	-.005

Note: Regression values are unstandardized B coefficients.; * $p < .05$

Table 16 (cont'd)

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Maternal-Report	Step 1	Social Aggression	.013	.001	-.003	.002	.005	.001	.002	-.002
	Step 2	Social Aggression	.013	.002	-.002	.002	.005	.001	.002	-.002
		Sex	.059	.044	.031	.005	.043	.018	.051	.074
		Pubertal Stage	.068	.019	-.012	.016	.027	.015	-.002	.048
	Step 3	Social Aggression	.009	.001	-.004	-.004	.003	-.002	-.005	-.002
		Sex	-.005	.015	.037	-.004	.014	.000	.059	.091
		Pubertal Stage	.016	-.006	-.006	.011	.004	.001	.011	.062
		Social Aggression x Sex	.005	.002	.004	.015*	.004	.009	.004	.002
		Social Aggression x Pubertal stage	.007	.002	.005	.012	.006	.008	.016	.000
		Sex x Pubertal stage	.100	.046	-.011	.010	.045	.026	-.016	-.027
	Step 4	Social Aggression	.015	.006	-.005	-.003	.005	.002	-.008	-.006
		Sex	-.004	.015	.037	-.004	.015	.001	.059	.091
		Pubertal Stage	.018	-.005	-.006	.011	.005	.002	.010	.061
		Social Aggression x Sex	-.001	-.003	.004	.013	.001	.005	.007	.005
		Social Aggression x Pubertal stage	.002	-.003	.005	.011	.004	.004	.019	.003
		Sex x Pubertal stage	.099	.046	-.011	.010	.044	.026	-.015	-.026
		Social Aggression x Sex x Pubertal stage	.008	.006	.000	.002	.003	.005	-.003	-.005

Table 16 (cont'd)

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Teacher Report	Step 1	Social Aggression	0.003	0.009	-0.011	.000	-0.004	-0.004	0.004	-0.020
	Step 2	Social Aggression	0.002	0.009	-0.012	0.001	-0.006	-0.006	0.005	-0.021
		Sex	-0.006	-0.003	-0.010	-0.056	-0.006	-0.006	-0.040	0.058
		Pubertal Stage	-0.052	-0.037	-0.069	0.003	-0.062	-0.062	-0.018	-0.009
	Step 3	Social Aggression	0.002	0.005	-0.014	0.000	-0.007	-0.007	0.002	-0.008
		Sex	-0.022	0.027	0.027	-0.053	0.004	0.004	-0.025	-0.038
		Pubertal Stage	-0.074	-0.020	-0.034	0.013	-0.055	-0.055	-0.006	-0.090
		Social Aggression x Sex	-0.002	0.009	0.005	0.004	0.002	0.002	0.006	-0.034
		Social Aggression x Pubertal stage	0.014	0.022	-0.004	-0.008	0.004	0.004	0.007	-0.035
		Sex x Pubertal stage	0.044	-0.016	-0.065	-0.011	-0.010	-0.010	-0.012	0.090
	Step 4	Social Aggression	0.011	0.024	-0.021	-0.013	-0.004	-0.004	0.002	0.019
		Sex	-0.044	-0.026	0.044	-0.018	-0.003	-0.003	-0.024	-0.111
		Pubertal Stage	-0.097	-0.073	-0.017	0.048	-0.062	-0.062	-0.005	-0.163
		Social Aggression x Sex	-0.010	-0.011	0.011	0.017	-0.001	-0.001	0.006	-0.061
		Social Aggression x Pubertal stage	0.006	0.003	0.002	0.005	0.002	0.002	0.008	-0.061
		Sex x Pubertal stage	0.067	0.037	-0.082	-0.046	-0.003	-0.003	-0.013	0.164
		Social Aggression x Sex x Pubertal stage	0.009	0.020	-0.007	-0.014	0.003	0.003	-0.001	0.028

Table 17: Predicting bilateral basolateral amygdala reactivity to facial expressions vs. shapes as a function of social aggression, sex, pubertal stage, and their interactions

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Self-Report	Step 1	Social Aggression	-.003	-.008	-.002	.003	-.003	-.001	-.002	-.007
	Step 2	Social Aggression	-.005	-.008	.000	.002	-.003	-.002	-.002	-.008
		Sex	.040	.038	-.005	-.044	.014	-.020	.045	.063
		Pubertal Stage	.053	.018	-.045	.000	.001	.005	-.001	.070
	Step 3	Social Aggression	.001	-.002	-.001	.004	-.001	.002	-.013	-.017
		Sex	-.012	.003	.014	-.052	-.003	-.040	.064	.061
		Pubertal Stage	-.004	-.021	-.028	-.010	-.019	-.017	.032	.080
		Social Aggression x Sex	.015	.000	.005	.001	.011	.000	.003	.010
		Social Aggression x Pubertal stage	-.009	-.011	.003	-.004	-.003	-.006	.020	.017
		Sex x Pubertal stage	.077	.055	-.032	.012	.023	.031	-.029	.001
	Step 4	Social Aggression	.003	.000	.001	.004	.001	.003	-.014	-.023
		Sex	-.016	-.001	.007	-.052	-.009	-.044	.066	.076
		Pubertal Stage	-.007	-.024	-.032	-.010	-.023	-.019	.033	.089
		Social Aggression x Sex	.013	-.002	.001	.001	.008	-.002	.004	.018
		Social Aggression x Pubertal stage	-.011	-.012	.000	-.004	-.005	-.007	.021	.022
		Sex x Pubertal stage	.081	.059	-.026	.012	.028	.034	-.032	-.012
		Social Aggression x Sex x Pubertal stage	.003	.003	.005	.000	.004	.003	-.002	-.012

Note: Regression values are unstandardized B coefficients.; * $p < .05$

Table 17 (cont'd)

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Maternal-Report	Step 1	Social Aggression	.013	.004	-.003	.001	.005	.001	.000	.001
	Step 2	Social Aggression	.013	.004	-.002	.001	.006	.001	.001	.001
		Sex	.040	.035	-.002	-.038	.015	-.018	.043	.053
		Pubertal Stage	.046	.010	-.040	.006	-.001	.005	-.001	.066
	Step 3	Social Aggression	.009	.004	-.004	-.003	.002	-.001	-.007	-.002
		Sex	-.019	.002	.010	-.052	-.009	-.040	.054	.046
		Pubertal Stage	-.002	-.019	-.028	-.005	-.020	-.013	.013	.061
		Social Aggression x Sex	.007	-.001	.001	.011	.004	.002	.006	.006
		Social Aggression x Pubertal stage	.008	.000	.004	.008	.007	-.040	.017	.007
		Sex x Pubertal stage	.091	.053	-.021	.020	.037	-.013	-.020	.010
	Step 4	Social Aggression	.019	.009	-.003	-.008	.009	.004	-.007	-.008
		Sex	-.018	.003	.010	-.052	-.009	.001	.054	.045
		Pubertal Stage	.000	-.018	-.028	-.006	-.018	.033	.013	.060
		Social Aggression x Sex	-.003	-.006	.000	.016	-.002	.003	.006	.011
		Social Aggression x Pubertal stage	-.001	-.005	.003	.012	.001	.002	.017	.012
		Sex x Pubertal stage	.090	.053	-.021	.020	.036	-.040	-.020	.010
		Social Aggression x Sex x Pubertal stage	.013	.006	.001	-.007	.008	-.013	.000	-.007

Table 17 (cont'd)

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Teacher-Report	Step 1	Social Aggression	0.004	0.006	-0.012	-0.007	-0.003	0.000	-0.005	-0.030
	Step 2	Social Aggression	0.004	0.006	-0.013	-0.006	-0.004	0.001	-0.006	-0.030
		Sex	-0.028	-0.029	0.005	-0.051	-0.013	-0.067	0.028	0.001
		Pubertal Stage	-0.062	-0.052	-0.073	0.016	-0.070	-0.022	-0.045	-0.023
	Step 3	Social Aggression	0.004	0.006	-0.016	-0.008	-0.005	0.000	0.001	-0.014
		Sex	-0.050	-0.030	0.022	-0.044	-0.020	-0.067	0.014	-0.124
		Pubertal Stage	-0.090	-0.062	-0.050	0.034	-0.075	-0.022	-0.062	-0.122
		Social Aggression x Sex	-0.002	-0.004	0.010	0.010	0.005	0.004	-0.027	-0.043
		Social Aggression x Pubertal stage	0.014	0.011	-0.005	-0.009	0.005	0.004	-0.022	-0.053
		Sex x Pubertal stage	0.054	0.011	-0.026	-0.016	0.022	0.007	-0.027	0.109
	Step 4	Social Aggression	-0.008	0.011	-0.028	-0.015	-0.017	0.006	0.022	-0.017
		Sex	-0.018	-0.043	0.055	-0.026	0.010	-0.083	-0.042	-0.116
		Pubertal Stage	-0.058	-0.076	-0.016	0.053	-0.044	-0.038	-0.118	-0.114
		Social Aggression x Sex	0.009	-0.009	0.022	0.016	0.016	-0.002	-0.047	-0.040
		Social Aggression x Pubertal stage	0.026	0.006	0.008	-0.002	0.016	-0.002	-0.043	-0.050
		Sex x Pubertal stage	0.022	0.025	-0.060	-0.034	-0.009	0.024	0.030	0.102
		Social Aggression x Sex x Pubertal stage	-0.012	0.005	-0.013	-0.007	-0.012	0.006	0.022	-0.003

Table 18: Predicting bilateral centromedial amygdala reactivity to facial expressions vs. shapes as a function of social aggression, sex, pubertal stage, and their interactions

			Fear		Anger		Anger + Fear	
			Left	Right	Left	Right	Left	Right
Self-Report	Step 1	Social Aggression	.005	.006	-.001	-.004	.002	.002
	Step 2	Social Aggression	.005	.006	-.002	-.005	.002	.002
		Sex	-.018	.079	-.004	-.035	-.015	.012
		Pubertal Stage	-.017	.035	.019	.005	-.007	.014
	Step 3	Social Aggression	.005	.000	-.005	-.005	.000	.001
		Sex	-.028	.102	.003	-.015	-.016	.040
		Pubertal Stage	-.026	.061	.030	.020	-.005	.038
		Social Aggression x Sex	.003	.012	-.001	.002	-.001	.003
		Social Aggression x Pubertal stage	.000	.012	.006	-.001	.003	.002
		Sex x Pubertal stage	.015	-.040	-.010	-.032	.002	-.046
	Step 4	Social Aggression	.005	.008	-.004	-.002	.119	.007
		Sex	-.028	.080	.001	-.022	.000	.024
		Pubertal Stage	-.026	.048	.029	.016	-.016	.028
		Social Aggression x Sex	.003	.001	-.002	-.001	-.004	-.005
		Social Aggression x Pubertal stage	.000	.005	.005	-.003	.000	-.004
		Sex x Pubertal stage	.015	-.022	-.009	-.027	.003	-.032
		Social Aggression x Sex x Pubertal stage	.000	.016	.001	.005	.002	.012

Note: Regression values are unstandardized B coefficients.; * $p < .05$

Table 18 (cont'd)

			Fear		Anger		Anger + Fear	
			Left	Right	Left	Right	Left	Right
Maternal-Report	Step 1	Social Aggression	.007	.002	.005	.001	.006	.001
	Step 2	Social Aggression	.007	.003	.005	.000	.005	.001
		Sex	-.017	.083	-.001	-.038	-.013	.013
		Pubertal Stage	-.013	.036	.017	-.001	-.006	.014
	Step 3	Social Aggression	.009	-.002	.007	.001	.007	-.001
		Sex	-.026	.098	.005	-.020	-.014	.038
		Pubertal Stage	-.024	.051	.021	.015	-.008	.036
		Social Aggression x Sex	.003	.013	.000	-.001	.002	.006
		Social Aggression x Pubertal stage	-.006	.012	-.004	.000	-.004	.004
		Sex x Pubertal stage	.015	-.027	-.009	-.029	.001	-.042
	Step 4	Social Aggression	.016	.006	.012	.002	.013	.006
		Sex	-.026	.098	.005	-.020	-.014	.039
		Pubertal Stage	-.022	.053	.022	.015	-.007	.037
		Social Aggression x Sex	-.003	.005	-.005	-.002	-.003	-.001
		Social Aggression x Pubertal stage	-.012	.004	-.008	-.001	-.009	-.003
		Sex x Pubertal stage	.014	-.028	-.009	-.029	.000	-.043
		Social Aggression x Sex x Pubertal stage	.008	.011	.007	.002	.007	.009

Table 18 (cont'd)

			Fear		Anger		Anger + Fear	
			Left	Right	Left	Right	Left	Right
Teacher-Report	Step 1	Social Aggression	0.005	0.009	.002	0.010	0.004	0.009
	Step 2	Social Aggression	0.004	0.009	.078	0.011	0.004	0.010
		Sex	-0.024	0.009	.003	-0.083	-0.023	-0.047
		Pubertal Stage	-0.073	0.010	-.007	0.013	-0.043	0.013
	Step 3	Social Aggression	0.006	0.005	.004	0.011	0.004	0.006
		Sex	-0.051	0.069	.054	-0.063	-0.022	0.007
		Pubertal Stage	-0.100	0.058	.001	0.032	-0.044	0.058
		Social Aggression x Sex	-0.006	0.005	.017	0.000	-0.001	0.005
		Social Aggression x Pubertal stage	0.001	0.006	.025	-0.005	0.002	0.002
		Sex x Pubertal stage	0.041	-0.090	.002	-0.040	0.000	-0.086
	Step 4	Social Aggression	0.004	-0.014	.000	-0.029	0.011	-0.038
		Sex	-0.046	0.121	-.040	0.043	-0.041	0.125
		Pubertal Stage	-0.095	0.110	.081	0.139	-0.063	0.177
		Social Aggression x Sex	-0.005	0.024	.011	0.039	-0.008	0.049
		Social Aggression x Pubertal stage	0.003	0.025	-.010	0.034	-0.005	0.045
		Sex x Pubertal stage	0.036	-0.143	-.002	-0.148	0.019	-0.025
		Social Aggression x Sex x Pubertal stage	-0.002	-0.020	-.008	-0.041	0.007	-0.046

Table 19: Predicting bilateral total amygdala reactivity to facial expressions vs. shapes as a function of social aggression, physical aggression, and their interaction

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Self-Report	Step 1	Social Aggression	-.011	-.022*	.000	.000	-.005	-.010	.005	-.004
		Physical Aggression	.012	.019*	-.005	.002	.003	.011	-.012	-.010
	Step 2	Social Aggression	-.007	-.017	.001	.001	-.003	-.007	.004	-.007
		Physical Aggression	.015	.022*	-.005	.003	.005	.012	-.012	-.012
		Social x Physical Aggression	-.002	-.003*	.000	-.001	-.001	-.002	.001	.002
Maternal-Report	Step 1	Social Aggression	0.017	-.010	-.003	.005	.007	-.002	.014	.004
		Physical Aggression	-0.006	.014	.001	-.005	-.002	.003	-.015	-.006
	Step 2	Social Aggression	0.021	-.007	.002	.007	.011	.000	.012	.009
		Physical Aggression	-0.003	.016	.005	-.003	.001	.006	-0.16	-.003
		Social x Physical Aggression	-0.001	-.001	-.001	-.001	-.001	-.001	.000	-.001
Teacher-Report	Step 1	Social Aggression	-.004	.007	-.013	.001	-.008	.002	-.027	-.041
		Physical Aggression	.011	.003	.003	.000	.006	.004	.032	.033
	Step 2	Social Aggression	-.004	.007	-.012	.001	-.008	.002	-.027	-.040
		Physical Aggression	.012	.005	.010	.006	.009	.008	.031	.035
		Social x Physical Aggression	.000	.000	-.001	-.001	-.001	-.001	.000	.000

Note: Regression values are unstandardized B coefficients.; * $p < .05$

Table 20: Predicting bilateral basolateral amygdala reactivity to facial expressions vs. shapes as a function of social aggression, physical aggression, and their interaction

			Fear		Anger		Anger + Fear		Neutral	
			Left	Right	Left	Right	Left	Right	Left	Right
Self-Report	Step 1	Social Aggression	-.011	-.021	-.001	.005	-.007	-.006	.005	.005
		Physical Aggression	.012	.019*	.000	-.002	.005	.008	-.012	-.019
	Step 2	Social Aggression	-.007	-.016	.000	.005	-.004	-.004	.003	.003
		Physical Aggression	.014	.022**	.001	-.002	.007	.010	-.013	-.020
		Social x Physical Aggression	-.002	-.003*	-.001	.000	-.001	-.001	.001	.002
Maternal-Report	Step 1	Social Aggression	.017	-.008	-.005	.006	.006	-.001	.011	.012
		Physical Aggression	-.007	.015	.003	-.007	-.001	.003	-0.14	-0.12
	Step 2	Social Aggression	.023	-.004	-.001	.009	.010	.003	.011	.019
		Physical Aggression	-.002	.019	.007	-.004	.003	.006	-0.14	-.006
		Social x Physical Aggression	-.002	-.001	-.001	-.001	-.001	-.001	.000	-.002
Teacher-Report	Step 1	Social Aggression	-.005	-.003	.014	-.004	-.008	-.005	-.020	-.026
		Physical Aggression	.015	.016	.005	-.005	.009	.009	.025	-.009
	Step 2	Social Aggression	-.005	-.003	-.014	-.004	-.008	-.005	-.020	-.026
		Physical Aggression	.018	.017	.011	-.001	.013	.013	.026	-.006
		Social x Physical Aggression	.000	.000	-.001	-.001	-.001	-.001	.000	.000

Note: Regression values are unstandardized B coefficients.; * $p < .05$

Table 21: Predicting bilateral centromedial amygdala reactivity to facial expressions vs. shapes as a function of social aggression, physical aggression, and their interaction

			Fear		Anger		Anger + Fear	
			Left	Right	Left	Right	Left	Right
Self-Report	Step 1	Social Aggression	.003	.003	-.003	-.004	-.001	.001
		Physical Aggression	.004	.005	.005	.000	.005	.002
	Step 2	Social Aggression	.003	.005	-.005	-.006	-.002	-.001
		Physical Aggression	.004	.006	.004	-.001	.004	.001
		Social x Physical Aggression	.000	-.001	.001	.001	.001	.001
Maternal-Report	Step 1	Social Aggression	.009	-.006	.005	.005	.005	.001
		Physical Aggression	-.004	.011	.000	-.006	.000	.000
	Step 2	Social Aggression	.012	-.004	.006	.005	.007	.001
		Physical Aggression	-.001	.013	.001	-.006	.001	.000
		Social x Physical Aggression	-.001	-.001	.000	.000	-.001	.000
Teacher-Report	Step 1	Social Aggression	.003	.011	-.001	.016	.001	.013
		Physical Aggression	.004	-.004	.005	-.010	.006	-.007
	Step 2	Social Aggression	.003	.012	-.001	.016	.001	.013
		Physical Aggression	.005	.000	.007	-.008	.008	-.004
		Social x Physical Aggression	.000	-.001	.000	.000	.000	.000

Note: Regression values are unstandardized B coefficients.; * $p < .05$

FIGURES

Figure 7: Predicting right total amygdala reactivity to angry faces as a function of sex, pubertal stage, and their interaction

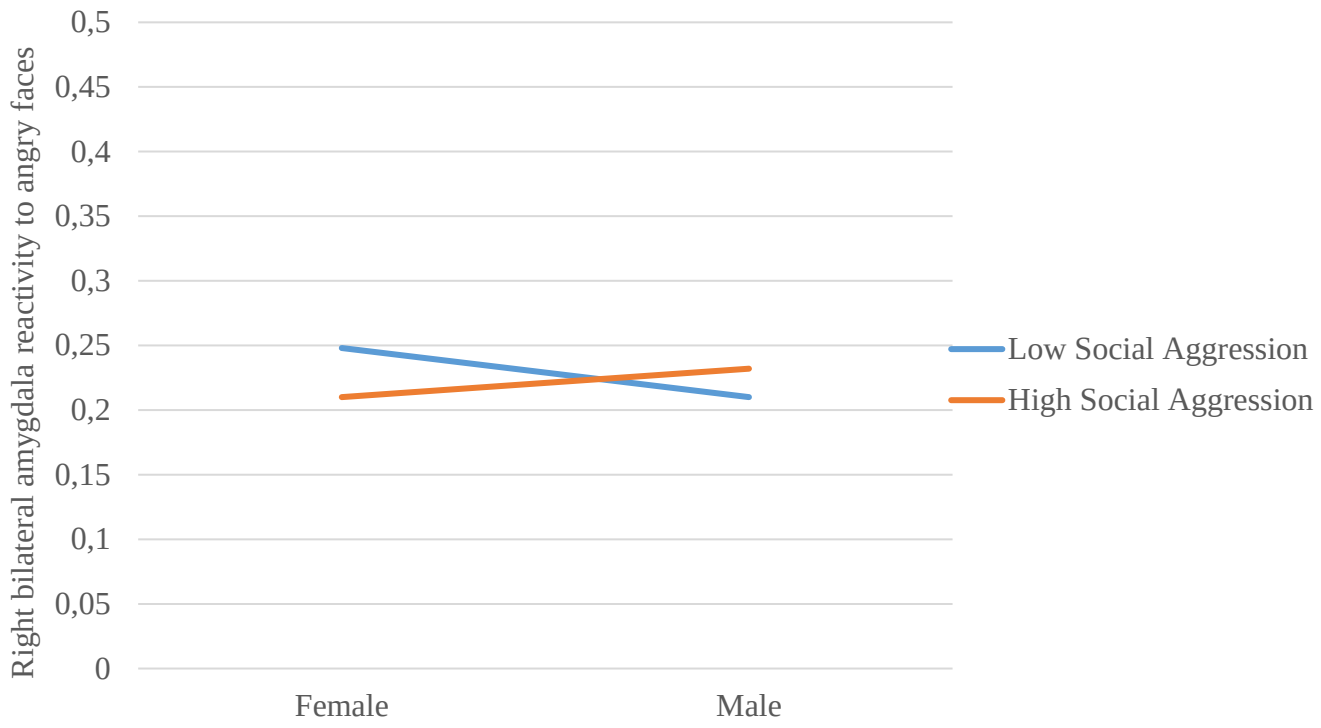
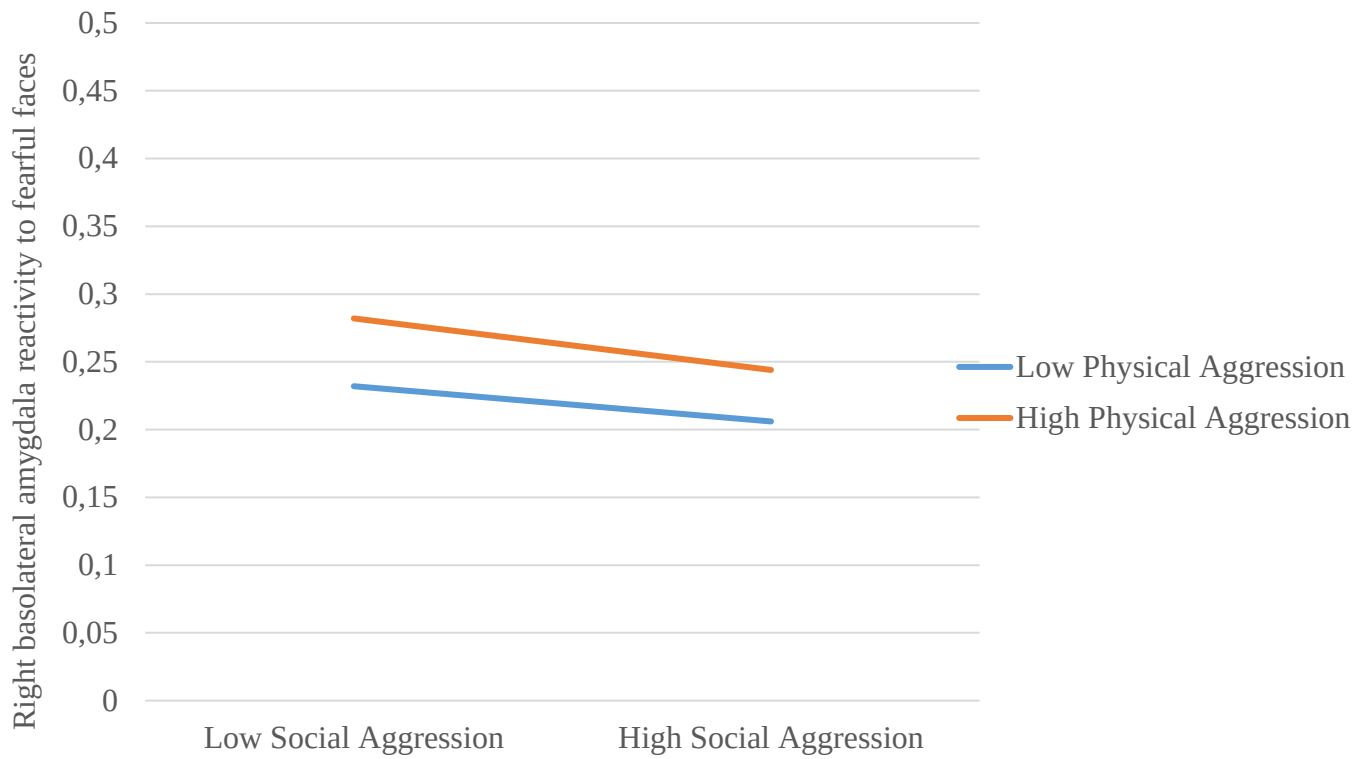


Figure 8: Predicting right basolateral amygdala reactivity to fearful faces as a function of social aggression, physical aggression, and their interaction



CHAPTER 4: CONCLUSIONS AND IMPLICATIONS

CONCLUSIONS AND IMPLICATIONS

Discussion and Future Directions

The overarching aim of this dissertation was to deepen our understanding the sex-specific and developmental origins of social aggression by investigating the roles of sex, genes, pubertal development, and neural processes in its perpetration. While countless studies have examined the role of socialization processes and psychosocial risk factors, the biological underpinnings of social aggression have rarely been investigated and, therefore, are not well understood. These are critical omissions from the literature as there is now ample evidence that etiology of social aggression includes genetic risk. Further, these genetic risk factors seem likely to vary across development and perhaps across socialization experiences, which makes the inclusion of biological factors a priority when developing multi-level models of the origins of social aggression.

We first took a behavioral genetic approach to examining the etiology of social aggression. Prior research indicated that variance in social aggression is partially due to genetic factors and suggested that there are no sex differences in its etiology. However, no behavior genetic study had yet taken a developmental approach and it was largely unknown how developmental risk factors, such as puberty, might shape genetic and environmental risk for social aggression. Therefore, successive quantitative genetic models were used to gain an increasingly nuanced understanding of the relationships between sex and puberty in the etiology of social aggression.

One of the most striking findings from this investigation was that although univariate results were consistent with prior research suggesting that there were no sex differences in the

etiology of social aggression, a two-moderator GxE model indicated that there were indeed sex differences in the etiology of social aggression (maternal reports only) once we adjusted for the effects of puberty. Specifically, we found that genetic influences on social aggression were stronger for boys than for girls and non-shared environmental influences were stronger for girls than for boys.

The question of sex differences in social aggression has remained at the forefront of empirical investigation for over fifty years, largely due to inconsistent evidence both supporting and refuting phenotypic differences. Meta-analytic work has attempted to resolve these inconsistencies, and these studies consistently conclude that while social aggression is statistically more common in females than in males, the magnitude of this difference is small to very small (Cohen's d = .06 to .18; Archer, 2004; Card et al., 2008; Scheithaur, Haag, Mahlke, & Ittel, 2014). However, the most recent meta-analysis emphasized that the presence of sex differences is highly variable across studies, with effect sizes ranging from d = -1.04 to 1.17, and concluded that the sheer size of this effect size distribution was larger than expected by chance alone (Scheithauer et al., 2014).

The results of this dissertation support the explanation that inconsistent evidence for sex differences in social aggression is in part due to confounding by key developmental moderators, such as puberty. Moreover, the joint moderation GxE model not only indicated that there are sex differences in the etiology of maternal-reports of social aggression once we adjusted for pubertal development, but also that genetic influences on social aggression are jointly moderated by sex and puberty together such that genetic influences more than doubled from pre-puberty to puberty in girls but not boys. It should be noted that although there were not significant changes in the

magnitude of genetic influences on social aggression from pre-puberty to puberty in boys, genetic influences were important for the etiology of social aggression in males as well.

One possibility for the observed increase in the proportion of genetic risk for maternal reported social aggression following pubertal onset is that increases in female reproductive hormones associated with pubertal development may activate genes associated with social aggression in girls. Unfortunately, very few empirical studies have directly examined associations between pubertal development, reproductive hormones, and social aggression. In the only study assessing the association between social aggression and reproductive hormones, Sánchez-Martín et al. (2011) found that testosterone was positively associated with physical, verbal, and indirect aggression in 9-year-old males and females. However, the effects of other reproductive hormones, such as estrogen or progesterone, have yet to be investigated for social aggression. This remains a significant gap in our understanding of the development of social aggression, since changes in reproductive hormones have been implicated in the development of other disorders, such as eating disorders and depression, in women (Schiller et al., 2015; Baker, Girdler, & Bulik, 2012; Harden et al., 2014; Bromberger et al., 2010; Schmidt, 2005).

A second possibility, one that we were able to partially test in Chapter 3, is that pubertal changes in brain development facilitate increases in social aggression. Early theories regarding social aggression suggested girls engage in more sophisticated and covert aggressive behaviors than boys due to their earlier cognitive maturation (Björkqvist et al., 1992; Keenan & Shaw, 1997; Silverthorn & Frick, 1999). Therefore, it may be that these improvements in cognition and subsequent increases in social aggression are a result of neural maturation processes that occur during late childhood and adolescence. We thus examined the association between social aggression and neural activation patterns (e.g., amygdala reactivity) during a socio-emotional

face processing task as well. Given the evidence that the perpetration of socially aggressive behaviors may reflect dysregulated emotional responses to stressful stimuli, we hypothesized that social aggression will be positively associated with increased amygdala reactivity to emotional faces. However, we observed that social aggression was not associated with amygdala reactivity during this task, even after controlling for sex, age, puberty, and their interactions.

Although these null results were unexpected, it remains possible that other neural changes associated with pubertal development predict social aggression. Most notably, neural connectivity may be a particularly promising avenue for increasing our understanding the development of social aggression. Neuroimaging research indicates that neural development continues throughout childhood and adolescence, with white matter volume increasing in the frontal cortex between 9 and 14 years (Blakemore, 2012). Further, there is some longitudinal evidence to suggest that there may be sex and pubertal differences in white matter development. Many white-matter tracts only reach mature levels of integrity after puberty (Asato et al., 2010), and there are sex differences in the onset of puberty such that puberty typically begins earlier in girls than boys (7–13 years old vs. 9–13 years old; Grumbach & Styne, 2003). As such, adolescent males on average reach peak white matter volumes later than females (Ladouceur et al., 2012). Moreover, an emerging body of functional connectivity research has found that not only does resting-state functional connectivity between spatially distant, functionally-related brain regions increases between childhood and adulthood (Vogel et al., 2010), but also task-dependent functional connectivity also increases during adolescence (Klapwijk et al., 2013). However, few studies have examined sex differences in task-dependent functional connectivity, and those that have mixed findings (Shaw et al., 2011; Klapwijk et al., 2013).

Limitations

The various research designs were both a major strength and a limitation of this project. This dissertation capitalized on a unique longitudinal twin study of emotional and behavioral development from the Michigan State University Twin Registry. Wave 1 (Twin Study of Behavioral and Emotional Development in Children; TBED-C) consists of two independent sub-samples of twins in middle childhood. The first sample consists of a population-based epidemiologic sample of 528 twin pairs. The second, 'at-risk' sample consists of 502 twin pairs, for whom inclusion criteria also specified that they reside in modestly-to-severely disadvantaged neighborhoods. Wave 2 (Michigan Twin Neurogenetics Study; MTwiNS) currently consists of a follow-up sample of 275 at-risk TBED-C twin pairs, now in adolescence. This unique design allowed us to examine, from a developmental, transdiagnostic, and multi-disciplinary perspective, which biological factors or combination of factors were associated with the perpetration of social aggression during different developmental periods.

However, there were several limitations that should be considered. Due to differences in study design and sample size across the two waves of the study, not all analyses could be conducted in both developmental periods. Namely, the neuroimaging analyses were limited to adolescence only because neuroimaging was not conducted in middle childhood. Similarly, the behavior genetic analyses were largely limited to middle childhood because a minimum of 1,000 twin pairs are necessary to conduct quantitative genetic moderation analyses, such as those using the joint moderation GxE model. While the middle childhood sample is adequately powered to conduct this work, there was less variation in pubertal development in middle childhood versus adolescence. Although this is to be expected, it limited our ability to conduct a nuanced investigation of pubertal development. Specifically, puberty was dichotomized into pre-pubertal

and pubertal periods for the quantitative genetic moderation analyses, which prevented us from examining etiological changes as they may occur throughout pubertal development.

Additionally, the behavioral genetic findings were specific to maternal reports of social aggression in middle childhood and did not extend to teacher reports of social aggression during the same developmental period. Indeed, teacher reports suggested that there was little association between sex, puberty, and social aggression, and moderation analyses examining teacher reports were inconclusive. One possible explanation for the differences observed between maternal and teacher informants is attribution bias, such that mothers and teachers are exposed to the child's socially aggressive behavior in different settings and, therefore, develop different attributions regarding the same child (De Los Reyes & Kazdin, 2005). Teachers typically observe children in structured classroom settings, but are less informed regarding their behavior outside of school. In comparison, mothers typically observe their children in less structured settings, but are less informed regarding their behavior during the school day. Therefore, it may be that children are differentially perpetrating social aggression across settings, and, if so, these results apply to social aggression observed outside of scholastic contexts only.

Alternately, the current findings may be a function of differential validity of maternal and teacher reports of social aggression, such that one informant may be providing more valid information regarding the child's social aggression than the other (Burt, Slawinski, & Klump, 2018). In addition to observing children in different settings, mothers and teachers also typically differ in the closeness of their relationships with them. Because children are typically closer with their mothers than their teachers, they may confide in their mothers regarding problematic peer interactions more than they do with their teachers. This is may be particularly relevant for the covert nature of some socially aggressive behaviors. Indeed, paired sample t-test indicated that

both child self-reports and maternal reports indicated the presence of higher rates of social aggression in adolescence than did teacher reports, but maternal and child self-reports did not differ significantly from one another. Maternal self-reports were also higher than teacher reports in childhood. Moreover, these mean differences appeared to be especially pronounced for girls.

Furthermore, although twin self-reports of social aggression were collected during adolescence, they were not collected in middle childhood. Because of this, we were unable to fully investigate the relationships between puberty and self-reports of social aggression. Self-reports and peer-ratings are especially useful since social aggression is characterized by both overt behaviors (that adults are likely to witness) and covert behaviors (of which only the child and his or her peers may be aware). Indeed, behavioral manifestations of social aggression may become more covert and sophisticated as individuals mature (Crick et al., 2007). Although there are concerns regarding the reliability and validity of peer- and self-reports in young children, peer ratings are more frequently used with adolescents and have been suggested to be more valid than teacher, parent, or self-ratings of social aggression during later developmental periods (Archer & Coyne, 2005).

Conclusion

Social aggression is a form of antisocial behavior that puts both the victim and perpetrator at increased risk for mental illness and socio-emotional suffering. Social aggression is perpetrated across the lifespan, but our understanding of its developmental origins remain limited, in part because the biological factors involved remain understudied. Furthermore, sex differences in its perpetration, which has remained a primary area of investigation since the field's inception, remain inconclusive, in part due to key moderating factors not being considered. Therefore, the purpose of this dissertation was to integrate distinct biological risk

factors (e.g., sex, genes, pubertal development, and neurological processes) to explore whether their interactions can help resolve extant inconsistencies in the literature regarding the development and presentation of social aggression.

Consideration of the interactions between puberty and sex did indeed led to new insights into the etiology of social aggression. Most notably, we observed that both sex and pubertal development jointly moderated the genetic influences on the etiology of maternal-reported social aggression, such that genetic influences increased from pre-puberty to puberty in girls, but not boys. Although we did not observe associations between neural activation and social aggression, neuroscience studies remain a promising area of future investigation.

Taken together, the analyses conducted in this dissertation emphasize the importance of taking a developmental psychopathology approach to studying social aggression. The defining features of developmental psychopathology include investigating psychopathology developmentally, across all relevant levels of analysis (Cicchetti & Dawson, 2002; Cicchetti, 2008). While a significant volume of research is conducted on social processes (e.g., peer dynamics) implicated in social aggression, there is a clear need for more evaluations of individual-level processes (e.g., genetic, neural, hormonal, temperamental) in order to refine multi-level models of the origins of social aggression. What's more, future research must continue to employ longitudinal and person-centered approaches to investigate social and biological risk factors and processes implicated in social aggression across time.

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