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Physiological Correlates of Aggression in Adolescent Females

Ashley Dibble
Virginia Commonwealth University

College of Humanities and Sciences
Virginia Commonwealth University

This is to certify that the thesis prepared by Ashley Dibble entitled "Physiological Correlates of Aggression Adolescent Females" has been approved by her committee as satisfactory completion of the thesis requirement for the degree of Master of Science.

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Wendy Kliewer, Ph.D., Director of Thesis
Department of Psychology

_____

Dace Svikis, Ph.D., Committee Member
Department of Psychology

_____

Catherine Bagwell, Ph.D., Committee Member
Department of Psychology, University of Richmond

_____

Wendy L. Kliewer, Ph.D., Director of Graduate Studies

_____

Fred M. Hawkridge, Ph.D., Interim Dean, College of Humanities and Sciences

_____

F. Douglas Boudinot, Ph.D., Dean, School of Graduate Studies

_____

Date

PHYSIOLOGICAL CORRELATES OF AGGRESSION IN ADOLESCENT FEMALES

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

by

ASHLEY ENGELS DIBBLE
M.S. Criminal Justice, Virginia Commonwealth University, 2005
B.A. Psychology, University of Virginia, 2001

Director: Wendy Kliewer, Ph.D.
Professor, Department of Psychology

Virginia Commonwealth University
Richmond, Virginia
May, 2009

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ABSTRACT

PHYSIOLOGICAL CORRELATES OF AGGRESSION IN ADOLESCENT FEMALES

By Ashley Dibble, M.S.

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Major Director: Wendy L. Kliewer, Ph.D., Professor, Department of Psychology

Recently, with the development of new technology, researchers have focused on physiological predictors of aggressive behavior, specifically cortisol and alpha amylase. Gordis, Granger, Susman, and Trickett (2006) found the interaction between cortisol and alpha-amylase significantly predicted parent reports of aggression indicating that low levels of physiological reactivity was associated with higher levels of problem behavior. While this research has provided valuable information about aggressive behavior, a major limitation is the majority of research focuses on males, or has not examined gender differences explicitly.

This study expanded on work by Gordis et al. (2006) and other researchers on the HPA axis and sympathetic nervous system responses and aggression by using a larger sample, focusing on female adolescents, examining both physical and relational aggression, and utilizing parent and adolescent reports of aggressive behavior. Based on prior literature, I expected that lower levels of salivary cortisol taken at the beginning of the interview and the beginning of the stress task would be associated with higher levels of physical and relational aggression in girls. I also hypothesized that

lower levels of cortisol and α-amylase reactivity will be associated with higher levels of physical and relational aggression. Finally, I hypothesized that lower levels of cortisol reactivity coupled with higher levels of α-amylase reactivity will be associated with lower levels of aggressive behavior.

Participants in the current study live in moderate- to high-violence areas in Richmond, VA. Participants were 146 adolescent females who were enrolled in a larger longitudinal study on coping with exposure to violence. Most of the adolescents were African-American (91.1%) with a mean age of 13.9 years old (range from 11-17). The changes in physiological responses were monitored during the interview process which included the administration of the Social Competence Interview (SCI). Aggression was measured using the Child Behavior Checklist and Problem Behavior Frequency Scales.

In the analyses, I controlled for pubertal status, medication usage, race, and time of day which are all factors that can influence the level of cortisol and alpha-amylase. Results indicated that higher levels of basal cortisol were associated with higher levels of aggressive behavior. In contrast to previous research and prediction, results indicated that symmetry in α-amylase and cortisol predicted lower levels of self-reported physical aggression in girls. Asymmetry in the two systems was associated with higher levels of self-reported physical aggression.

These results contribute to the mixed results on female physiological responses and aggression. It also provides support for symmetry in cortisol and α-amylase as a predictor of lower levels of aggressive behavior. Studying a child's physiological

reactions to stress can give insight into behavior regulation, help identify adolescents for prevention/intervention, and serve as markers of treatment progress. These data suggest that physiological associations with aggression may not be the same for males and females, or for youth living in extremely stressful circumstances. Further research is needed to replicate these finding, and specifically to compare these patterns of associations across gender.

Statement of the Problem

Aggressive behavior in adolescents is a concern because of the short- and long-term implications it can have for the aggressor, the victim, and society. Aggression is associated with a wide range of outcomes such as social adjustment problems, criminal behavior, and substance use. Research on aggression has focused on the different forms of aggression, outcomes of aggression, and causes of aggression. However, this research often has been gender specific with the majority of work focusing on adolescent males. The focus on males may be because of the stereotype that boys are more aggressive than girls, but in recent years aggressive behavior in adolescent females has been increasing at a faster rate than adolescent males (Crick, 1997). Another limitation is research on aggression has focused largely on physical aggression ignoring other forms, such as relational aggression which is more salient to females. Further, while previous research has provided a better understanding of aggressive behavior and allowed for the development of prevention and intervention programs that target risk factors that could lead to aggressive behavior, it has done little to improve the understanding of aggression in females. Researchers have begun to focus on physiological correlates of aggression. With the development of new technology, research on the physiological states that co-occur with or predict aggression became easier to study. This research has included the stress hormone, cortisol, and the enzyme, α-amylase. Although charting new territory, this physiological research on aggression also primarily has focused on adolescent males, or has not analyzed results by gender. With the recent increase in female aggression and variability across genders in a variety

of areas, it is important to expand the research of physiological correlates of aggression to the adolescent female population.

Understanding physiological correlates of aggression could help with prevention and intervention efforts by helping to identify individuals who would benefit the most from certain prevention and intervention programs. By utilizing physiological information, individuals prone to aggressive behavior could be placed into prevention and intervention programs that teach skills to limit aggression and regulate behavior.

Review of the Literature

In the following review, I will discuss several forms of aggression and research findings associated with them, as well as introduce some of the recent literature associated with physiological correlates of aggression.

Aggression

Most people have a general idea about what qualifies as aggression, but specific definitions of aggression vary widely. In fact, aggression has been defined in the literature in over 200 ways (Underwood, 2003). Aggression can refer to the expression of destructive and violent tendencies (Plutchik & van Praag, 1997). Most often, aggression brings the latter to mind. A frequently used definition of aggression is that it is behavior that is intended to inflict harm or injury (Eagly & Steffan, 1986). This definition is broad enough that it can encompass various forms of aggression, including physical and nonphysical forms. Aggression can include the infliction of emotional as well as physical harm.

Forms of aggression typically fall into one of two categories- direct and indirect. Direct aggression is verbal and physical behavior that is aimed at individuals with the intent to harm (Björkqvist, Lagerspetz, & Kaukiainen, 1991; Little, Henrich, Jones, & Hawley, 2003). Verbal attacks (mean names, insults) and humiliation of others are forms of aggression that are not physical but have obvious intent to cause psychological harm (Crick, 1997; Crick et al., 1999). Indirect aggression involves inflicting pain in such a manner that the perpetrator gives the impression that there has been no intention to hurt (Björkqvist et al., 1991). Indirect aggression is more subtle compared to the "in

your face" aspects of direct aggression (Little et al., 2003, p. 122; Underwood, 2003). It can be a more covert form of aggression that often allows the aggressor to go undetected.

Both indirect and direct aggression can be either instrumental or reactive. Instrumental aggression occurs for self-serving outcomes and is a deliberate behavior, whereas reactive aggression occurs in response to provocation (Little et al., 2003). Although both genders engage in both forms of aggression, direct and indirect, boys are more often associated with direct forms of aggression and girls are more frequently associated with indirect forms of aggression.

Physical Aggression

Two frequently researched kinds of aggression are physical and relational. Physical aggression is a direct form of aggression. Physical aggression involves the intent to harm using physical force such as hitting, punching, or kicking (Ostrov, 2006). Researchers have found that boys engage in physically aggressive acts more frequently than girls (Björkqvist et al., 1991; Ostrov, 2005; Zalecki & Hinshaw, 2004). However, some researchers believe that girls may be engaging in physically aggressive acts as much as boys but are better at hiding it from observing adults and do not admit it as readily during interviews (Loeber & Hay, 1997). Regardless of who is engaging in the behavior more frequently, physical aggression leads to negative outcomes for both genders. Boys and girls who engage in aggressive behavior are prone to many psychological and social problems. Some studies show that the prevalence of physical aggression is high early in life and then rapidly decreases throughout adolescence

(Loeber & Hay, 1997). However, the consequences of engaging in physically aggressive behavior can impact the individual as an adolescent and into adulthood.

Children who engage in physically aggressive behaviors are at greater risk for criminal behavior, alcohol and drug abuse, depression, spouse abuse, and neglectful and abusive parenting (Tremblay et al., 2004). In fact, physical aggression has been described as the "single strongest and most robust risk-factor" for antisocial behaviors (Werner & Crick, 2004, p. 495). A six-site cross-national study found that physical aggression in childhood is linked to both violent and non-violent behavior in adulthood (Broidy et al., 2003). This study included both genders, but physical aggression was a robust predictor of future problems only in males. These researchers found that although aggressive behavior developed similarly in males and females, it was much more difficult to predict violent and non-violent behavior in females than males.

The strong association between physical aggression and criminal behavior makes research on identifying aggressive adolescents and developing interventions for these adolescents all the more important. This is also true for the less often studied adolescent female population. Even though research on physical aggression has yielded inconsistent results for males and females, many researchers are not pursuing why these differences may exist and continue to ignore the female population. In contrast, females have received a great deal of attention when relational aggression, versus physical aggression, is considered.

Relational Aggression

Relational aggression is defined as acts that are intended to damage another individual's friendships or social status (Little et al., 2003). Relational aggression usually involves social manipulation such as spreading rumors, gossiping, or ignoring the individual (Crick, 1997; Crick, Ostrov, & Werner, 2006; Henington, Hughes, Cavell, & Thompson, 1998; Ostrov, 2005; Sullivan, Farrell, & Kliewer, 2006; Underwood, 2003). Unlike physical aggression, relational aggression can be both direct and indirect. These behaviors may also be nonverbal (Underwood, 2003). In research, an overlap is often seen between the usage of the term relational aggression and social aggression. Underwood (2003) proposes a model where the term social aggression is used to describe both direct, or relational, forms of aggression, and more indirect forms of aggression. Whether direct or indirect, these behaviors share the same goal, which is to cause social harm (Underwood, 2003). Research on relational aggression leads some researchers to believe that girls are just as aggressive as boys, but they use different acts to express their aggression (Underwood, 2003).

Although some studies have found that girls engage in relational aggression more frequently than boys, others have indicated this may be age dependent. As young children, boys are more likely than girls to engage in relational aggression, but between the ages of 8 to 11, the situation reverses (Henington et al., 1997). As children enter adolescence, there is a greater desire for intimacy in relationships which may be the reason for the increase in relational aggression (Zimmer-Gembeck, Geiger, & Crick, 2005). Also, relational aggression involves a certain level of maturity because

relationally aggressive acts require verbal and social skills (Bjorkqvist, Lagerspetz, & Kaukiainen, 1992). Higher levels of social intelligence are positively related to the use of relational aggression (Kaukiainen et al., 1999).

As with physical aggression, relational aggression is associated with peer rejection, internalizing problems, and externalizing behaviors (Crick et al., 2006). However, a relationship between popularity and aggressive behavior has also been found. Research in this area has focused on the notion of perceived and sociometric popularity. Sociometric popularity is how well-liked an individual is by peers, while perceived popularity is the social reputation of an individual (Cillessen & Mayeux, 2004). Sociometrically popular individuals are not necessarily members of the "in" crowd and individuals with perceived popularity are not always well liked (Cillessen & Mayeux, 2004). It is possible for a girl who is identified as popular to frequently engage in relationally aggressive acts because relational aggression is a way of establishing dominance in a peer group (Zimmer-Gembeck et al., 2005). However, her dominance in the peer group does not mean that she is well-liked. In a longitudinal study of children in grades 5 through 9, Cillessen and Mayeux (2004) examined this relationship between aggressive behavior and popularity. Each year, participants were provided with a list of peers in the grade and asked to identify peers who fit the description provided in various sociometric questions (Cillessen & Mayeux, 2004). The sociometric items measured status, physical aggression, and relational aggression. Researchers found that an increase relational aggression resulted in a decrease in sociometric popularity, but resulted in increases in perceived popularity (Cillessen &

Mayeux, 2004). These increases were seen as the participants got older and were stronger in girls. The changes over time may indicate that the aggressive behavior is being reinforced with some social benefits (Cillessen & Mayeux, 2004). In another study, of 3rd, 5th, 7th, and 9th grade students, researchers examined the friendships of relationally aggressive youth who were either disliked or perceived popular (Rose, Swenson, & Carlson, 2004). Results indicated that youth who were perceived popular had less friendship conflict than those who were disliked. One theory is that these youths are not engaging in relational aggression towards their friends, but rather working with their friends to aggress towards others. These studies show that the negative impact of relational aggression may depend on the status of the individual among peers.

Consequences of Aggressive Behavior

Adolescents engaging in any form of aggressive behavior experience similar social and psychological adjustment issues, but certain factors can influence the severity of these problems. The type of aggression used and the gender of the child may influence social and psychological problems the child faces. Engaging in aggression that is non-normative for the child's gender can lead to more severe social and psychological problems than engaging in aggression that is more normative for one's gender (Crick, 1997). This may be because relational aggression has been reported to be more accepted by girls and physical aggression is more accepted by boys. Physical aggression has been associated with higher levels of peer rejection in females than males (Underwood, 2003). Another study confirmed this finding and also found that

boys who engaged in relational aggression were at higher risk for social and psychological problems, including depression, anxiety, low self-esteem, and low well-being (Crick, 1997). Overall, girls are more likely than boys to suffer from peer rejection and social adjustment problems (Henington et al., 1997). Although low self-esteem is often cited as consequence of aggression, researchers also have found that aggressive individuals have over-inflated egos. Aggressive individuals have higher levels of self-esteem and show aggressive responses to situations they perceive as threats to their ego (Loeber & Hay, 1997).

When physical and relational aggression were compared, children, ages 9 to 12 years old, who engaged in physical aggression displayed more externalizing behaviors than nonaggressive peers, while those who engaged in relational aggression exhibited more externalizing and internalizing behaviors than nonaggressive peers (Crick, 1997). In a longitudinal study of 3rd and 4th graders, Crick et al. (2006) found that youth who engaged in both physical and relational aggression had more severe problems than those youth that engaged in one form of aggression or the other.

Aggression is a characteristic that can be identified at an early age (e.g., by age 2 in many children) and is stable over the course of childhood and adolescence. Relational and physical aggression are associated with lying, deception, and delinquent behavior (Ostrov, 2006). Frequently, aggression early in life is a significant predictor of later criminal behavior (Stattin & Magnusson, 1989). Some researchers have studied aggression, antisocial behavior, and conduct disorder because of their common comorbidity (Tremblay, 2000). Individuals who were rated highly aggressive in

adolescence, were more likely at age 26 to have a criminal record, to engage in more serious crimes, to engage in confrontative and destructive offenses, and were less likely to have a specialized crime pattern than individuals with low or normal aggression (Stattin & Magnusson, 1989). Antisocial girls become women who have up to a 40 times higher rate of criminal behavior than other women (Pajer, Gardner, Rubin, Perel, & Neal, 2001). As adults, aggressive women face a high risk of early death, complex psychiatric problems, higher rates of substance abuse and poor physical health. They also pass along the antisocial behavior to their children (Pajer et al., 2001). In reviewing the literature, aggression and antisocial behavior are used interchangeably at times even though the terms are different. This seems indicative of the strong association often seen between these two constructs.

Development of Aggression

Typically, it is not one factor that contributes to the development of aggressive behavior, but rather a combination of factors. These risk factors can be biological or environmental. It is important to identify accurate, economical predictors of aggression that can foretell such behavior from childhood to adolescence and from adolescence to adulthood (Broidy et al., 2003). Predicting risk is best when based on multiple risk domains in the child's life and the interaction of those domains (Loeber & Hay, 1997). Garbarino (1999) likens the accumulation of risk factors to juggling:

> Give me one tennis ball, and I can toss it up and down with ease. Give me two, and I can still manage easily. Add a third, and it takes special skill to juggle them. Make it four, and I will drop them all. So it is with threats to development.

(p. 76)

The accumulation of risk factors impose heavy burdens on development and will likely lead to substantial costs to the individual later in life (Shonkoff & Phillips, 2001).

The majority of theories take a transactional approach to development that recognizes the interaction between genetic and environmental risks in influencing aggressive behavior. Aggression is a behavior that virtually everyone expresses at some point, but some people more frequently engage in aggressive behavior and have a more aggressive disposition (Blackburn, 1998). This disposition increases the likelihood that the individual will react to a situation in a hostile or destructive manner. Essentially, someone may be predisposed to aggressive behavior genetically but it is the individual's environment that either puts the individual more at risk for engaging in such behavior or provides factors that protect the individual from such behavior. Parenting techniques, parental characteristics, neurological deficits, and child temperament are all associated with aggressive behavior (Barnow, Lucht, & Freyberger, 2005).

A child with a difficult temperament, who is not easy to soothe, may elicit negative responses from the parent (Loeber & Hay, 1997). This could lead to increased frustration on the part of child and eventually aggressive behavior, which in turn gets more negative responses from the parent. This reciprocal pattern can continue and significantly deteriorate the relationship between parent and child. Other factors that lead to aggressive behavior include low social intelligence, low levels of empathy, and the inadequate development of normative beliefs and problem solving strategies (Kaukianinen et al., 1999; Loeber & Hay, 1997; Werner & Crick, 2004).

Parental characteristics associated with aggressive behavior in youth include low levels of education, antisocial behavior, and family dysfunction (Tremblay et al., 2004). The process of learning to regulate emotions begins in early childhood. During development, a child must learn when and where certain emotions are appropriate (Shonkoff & Philips, 2000). Infant girls are better able to regulate their own emotions, while boys more often look to their mothers for cues (Loeber & Hay, 1997). If a mother does not regulate her emotions appropriately, the child may model her inappropriate responses. Failing to learn appropriate emotional responses and how to control behaviors can lead to impulsive and aggressive behavior. Research by Gottman et al. (1996) established the term meta-emotion to describe the awareness the parent has of his/her own emotions and those of the child. Emotion coaching is a meta-emotion philosophy where the parent assists his/her child with the emotions of anger and sadness. Parents who engage in emotion coaching are aware of low intensity emotions in themselves and their child, utilize negative emotions experienced by the child as a teaching opportunity, validate their child's emotion, help the child label the emotion, and brainstorm with the child ways to solve the problem while helping to set behavioral limits. A longitudinal study examined the relationship between meta-emotion philosophy, child emotion regulation abilities, and child outcomes. Researchers hypothesized that physiological characteristics influence emotion regulation in children. Families were assessed when the child was age 4 to 5 years old and again when the child was 7 to 8 years old. During the first assessment, data were gathered on the parent-child interaction, parent's meta-emotion philosophy, child's intelligence, and

child's physiological functioning while watching emotion-inducing films. Physiological functioning was assessed by collecting data on the cardiac interbeat interval, the pulse transmission time to finger, finger pulse amplitude, skin conductance level, and general somatic activity. For the second assessment, teachers rated child outcomes using the Child Behavior Checklist and a measure of peer aggression, the children completed the Peabody Individual Achievement Test- Revised, and mothers completed a form on the child's health, a measure about temperament, and a questionnaire about the child's emotion regulation abilities. Results indicated that meta-emotion philosophy, parenting, and the child's regulatory physiology and behavior are related to child outcomes. Specifically, emotion coaching was significantly related to child's physiology.

An insecure attachment between mother and child may predict future aggressive behavior, especially in boys (Loeber & Hay, 1997). In contrast, secure attachments can buffer children against the development of behavior problems (Shonkoff & Phillips, 2001). Disciplinary techniques can also influence aggressive behavior. Coercive interactions, physical or punitive punishment, and physical abuse as forms of discipline may lead to aggressive behavior, or may be the action the parent takes to stop aggressive behavior (Loeber & Hay, 1997). This is another example of the reciprocal relationship between the child and the environment. The parent may use physical punishment to discipline the child for an aggressive act which reinforces the aggressive behavior.

Neighborhood influences can impact the development of aggressive behavior, especially if a child lives in an area where they are frequently exposed to traumatic events (Loeber & Hay, 1997). Some of the factors that influence the development of aggression can also be outcomes of engaging in aggressive behavior, such as poor social relationships. Whereas positive peer experiences help individuals learn appropriate skills such as negotiating conflict, negative peer relationships can influence the development of aggressive behavior (Zimmer-Gembeck et al., 2005). The association between peer relationships and aggressive behavior has been described as bidirectional (Zimmer-Gembeck et al., 2005). Certain behaviors predispose adolescents to peer rejection and the peer rejection often exacerbates those behaviors. Peer rejection does not mean that adolescents engaging in relational aggression will not have any friends. It is more likely that they will associate with other aggressive or deviant peers who also engage in relationally aggressive behaviors (Werner & Crick, 2004).

The level of aggression expressed by an individual tends to change over time. Some children may use aggression to express themselves prior to developing verbal skills (Bjorkqvist, Osterman, & Kaukiainen, 1992). From early to middle childhood, aggressive behavior tends to decrease as interpersonal skills increase, with only a small cohort of children failing to regulate their aggressive behavior (Loeber & Hay, 1997). During adolescence and into early adulthood, aggressive behavior is very different from behavior displayed by young children. It may increase in impact, it may be instrumented by a peer group placing pressure on another child to do something, and it may be across genders (Loeber & Hay, 1997).

There are theories that classify individuals based on changes in their aggressive behavior between early childhood and adulthood. Loeber and Hay (1997) believe that prevalence rates of aggressive behavior indicate different groups of individuals that need to be distinguished. These groups include: (1) youth who stop aggressive behavior, (2) youth whose aggression is stable and continue the behavior without interruption or occasionally at the same level, (3) youth who escalate in their aggression and its severity, (4) youth who experience the onset of aggression during adolescence. Moffitt (2003) distinguished individuals who engaged in aggressive and antisocial behavior as life-course persistent or adolescent limited. The life-course persistent individuals are those individuals who participate in antisocial behavior during every stage of their life (Moffitt, 2003). In contrast, adolescent limited individuals engage in antisocial behavior from their teens into their mid-20s. In defining the two categories, Moffitt takes into account a variety of factors that are genetic, phenotypic, and environmental. Life-course persistent individuals have more genetic factors that are influencing their participation in antisocial behavior than the adolescent limited group. Adolescent limited individuals participate in antisocial behavior because they are trying to assert the independence they feel ready for but society does not recognize. Moffitt is careful to explain that not all adolescents fall into one of these two categories and that some individuals do not participate in antisocial behavior at all. Patterson, DeBaryshe, and Ramsey (1989) also present a theory placing antisocial individuals into early and late-starters. The early-starter may have received training and reinforcement for antisocial behavior, experienced social rejection because of the non-normative

behaviors, and been unsuccessful at academics. Late-starters do not begin committing offenses until middle to late adolescence and likely have not had training, faced peer rejection, or failed academically. Patterson et al. (1989) believe that the decrease in participation in antisocial behavior is largely due to the late-starters ceasing the behaviors.

Physiological Correlates of Aggression

An enormous body of research has focused on the correlates, causes, and consequences of aggressive behavior. However, predictors such as behavioral problems are only moderately predictive of later psychopathology (Bauer et al., 2002). Researchers are now looking at physiological correlates of aggressive behavior in order to understand how physiological responses may indicate adjustment problems and if they are better predictors of these problems. Traditionally, physiological researchers monitored heart rate, vagal tone, and skin conductance, or collected plasma samples. In the study by Gottman et al. (1996), which was not focused on aggression specifically, researchers examined the physiological basis for regulating emotion by assessing the vagal tone. The vagal nerve is the major nerve of the parasympathetic nervous system and it travels throughout the body. A child's baseline vagal tone and ability to suppress the vagal tone is associated with the child's ability to regulate emotions, greater ability to focus attention, and greater ability to self-soothe and explore novel stimuli (Gottman et al., 1996). Poor emotion regulation is often associated with aggressive behavior.

Gottman et al. (1996) found that the child's ability to suppress vagal tone at age 5 predicted good emotion regulation skill at age 8.

Of particular interest to aggression researchers are the stress hormone, cortisol, and the enzyme, α-amylase. Both cortisol and α-amylase are released by the body when it is responding to stress. Cortisol is secreted following the activation of the hypothalamic-pituitary-adrenal axis. The HPA axis influences activity of the immune system and organizes behavioral responses to threat (Dettling, Gunnar, & Donzella, 1999). Healthy adaptation depends upon the body's ability to increase production of cortisol in stressful situations and reduce production when the stressor is removed (Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001). Alpha-amylase is measured to assess the response of the sympathetic nervous system to stress. The sympathetic nervous system is responsible for the "fight or flight" reaction in the body (Gordis et al., 2006). It increases heart rate, blood flow to muscles, and blood glucose. Salivary α-amylase (sAA) increases in the saliva during parasympathetic activity and is produced by the salivary glands (Gordis et al., 2008; Granger et al., 2007). Although it is not representative of α-amylase throughout the body, increases in sAA have been found in the body following physically and psychologically stressful situations (Kivlighan & Granger, 2006; Granger et al., 2007). Until recently, much of the research on physiological correlates of aggression has been restricted because of invasive procedures to collect data and the difficulty in implementing the practices in a real world setting (Granger et al., 1998). Fortunately, saliva samples, which are relatively non-invasive, can be used to assess cortisol and sAA as markers of stress response.

Assaying for both cortisol and sAA may give a better picture of the physiological responses associated with aggressive behavior than just using one of the two (Bauer, Quas, & Boyce, 2002).

Genetic and Environmental Influences on Cortisol

Similar to the development of aggressive behavior, genetic and environmental factors impact cortisol levels. The hereditability of cortisol variation has been explored in twin studies. Researchers have studied cortisol levels throughout the day. A study of 20 monozygotic and 20 dizygotic male twin pairs found genetic influences on variation in morning cortisol levels (Meikle, Stringham, Woodward, & Bishop, 1988). Similarly, in a study of 52 monozygotic and 52 dizygotic twin pairs, researchers found that the stability of cortisol awakening levels indicated there is a genetic influence (Wüst, Federenko, Hellhammer, & Kirschbaum, 2000). However, a genetic influence was not found in daytime cortisol profiles. Linkowski et al. (1993) studied 11 monozygotic and 10 dizygotic twin pairs at a sleep laboratory for four nights. Using a catheter, cortisol was sampled every 15-minutes for 25 hours, which allowed research to see the 24-hour cortisol profile of each individual. Results indicated that genetics influenced the circadian rhythmicity, but environment controlled the mean level of cortisol secretion. Additionally, the timing of the lowest level in the daily cortisol cycle remains relatively stable and is uninfluenced by changes in meal schedule or shifts in the light-dark cycle. The timing of the daily peak is environmentally influenced and may shift based on life events.

Kirschbaum, Wüst, Faig, and Hellhammer (1992) studied 13 monozygotic and 11 dizygotic male and female twin pairs to determine the heritability of cortisol in response to stimulation. Baseline cortisol and reactive cortisol levels were measured surrounding three tasks: 1) an injection of hCRH, 2) a physical task that involved bicycling until exhausted, and 3) a public speaking and serial subtraction task. Results indicated that baseline levels of cortisol and the response to hCRH were influenced by genetic factors. However, genetic factors only mildly influenced the response to the psychological (i.e. public speaking) task and had no influence on the response to the physical task (Kirschbaum et al., 1992).

Early environmental experiences can also influence HPA responsivity (Bartels, Van den Berg, Sluyter, Boomsma, & Geus, 2003; Levine, 1994; Young, Aggen, Prescott, & Kendler, 2000). Early prenatal and developmental stress can permanently alter the HPA axis (Bartels et al., 2003). Rats have frequently been used to study the impact of early adverse experiences. The system of an infant develops in stages. Cortisol in the infant's first stage of life is relatively low and difficult to influence, which may be adaptive (Levine, 1994). In rats, this period lasts from the fourth day after birth until day 14. During this time, the physiological processes are largely influenced by the mother and separation from the mother results in decreased heart rate, decreased growth hormone production, and changes to the HPA axis. Rats that were separated from their mother during this period and then exposed to novel stimuli had higher basal levels and higher stress-induced levels of cortisol (Levine, 1994). This indicated that maternal factors my impact the regulation of the infant HPA axis.

In humans, the early loss of a parent and poor quality family relationships had long-term impacts on cardiovascular and cortisol responses (Luecken, 1998). Sixty-one college students were divided into loss and no-loss groups. Saliva samples and blood pressure readings were collected surrounding two tasks; a speech task and a video stressor. During the speech task, the participant had 30-seconds to prepare a 3-minute speech on one of three controversial topics. The video stressor was a 7-minute movie clip depicting two boys experiencing the death of their mother. Individuals in the loss group showed an increase in cortisol levels during the speech task, but individuals in the no-loss group showed a decrease (Luecken, 1998). For the movie task, individuals with poorer quality family relationships showed an increase in cortisol across samples, but individuals with more positive family relationships showed a decrease. The researchers concluded that early attachment experiences can have a permanent impact on cardiovascular and neurohormonal output (Luecken, 1998).

Cortisol and Aggressive Behavior

Aggressive behavior has been linked to low levels of stress reactivity in adolescents (Gordis et al., 2006; Moss et al., 1995; van Goozen et al., 1998). Some researchers have studied adolescents to see if low levels of cortisol predicted later aggressive behavior. Shoal et al. (2003) found that the relationship between low cortisol levels and aggressive behavior persisted over time. As part of a larger study, Shoal et al. (2003) studied 314 boys at age 10 to 12 and again at age 15 to 17. Boys participated in an event-related potential task with saliva samples being collected before and after the task. The Multidimensional Personality Questionnaire was used to

measure personality and the Youth Self Report version of the Child Behavior Checklist measured aggression. Preadolescent cortisol levels for boys aged 10 to 12 were related to aggressive behavior in middle adolescence at age 15 to 17. This study expanded on previous research by showing not only the link between cortisol and aggression but that this link extended over a five-year period.

A prior cross-sectional study by Moss et al. (1995) supported this finding. Moss et al. studied salivary cortisol responses in two groups of prepubertal boys, those with fathers who had a substance use disorder or antisocial behavior and those with fathers who did not. The youth participated in a 26-hour research protocol that included psychological and psychiatric testing, data collection on peer and family relationships, and stress arousal activities. Parents, mothers and fathers, completed information about their own substance use. Mothers also were interviewed about the psychiatric status of their child, and along with the child's teacher, completed the Child Behavior Checklist. Boys at higher risk for substance use disorder had lower cortisol responsivity when faced with an anticipated stressor than boys who were at average risk for substance abuse disorder. The reduced responsivity may be an adaptation to chronic stress or alternatively, based on research, it represents diminished brain arousal. Further, cortisol hyporesponsivity in the higher risk boys was associated with the magnitude of their aggressive behavior (Moss et al., 1995).

van Goozen et al. (1998) also examined cortisol levels in their study of 8-11 year old boys with oppositional-defiant disorder or conduct disorder. The participants were chosen from patients at an inpatient clinic and special schools for young boys with

aggressive and antisocial behavior. The primary caregiver and teachers completed the Child Behavior Checklist. The boys in the study completed a video task that involved a period of non-stress, a period of stress that included competition with a videotaped opponent and provocation from the opponent, and another period of non-stress. Cortisol levels, as well as blood pressure and heart rate, were monitored during non-stress, stress, and post-stress situations. (1998) also examined cortisol levels in their study of 8-11 year old boys with oppositional-defiant disorder or conduct disorder. Results indicated that boys with low anxiousness and high levels of externalizing behaviors had lower levels of cortisol during stress. Reduced basal cortisol levels were linked to the level of severity of conduct disorder. This study also found lower levels of cortisol at baseline and during nonstress for those boys who were rated as high by their teachers in externalizing behavior. Individuals with low cortisol levels have reportedly less peer contact, less preoccupation with school, and more hostility towards teachers (Bauer et al., 2002).

In contrast to the above studies, Klimes-Dougan et al. (2001) found no relationship between lower basal cortisol levels and externalizing behaviors. In their study of 195 adolescents (both male and female) and their parents, researchers collected saliva samples before and after two stress inducing tasks, the Conflict Discussion Paradigm (CDP) and the Social Performance Paradigm (SPP). The CDP elicited conflict between the mother and youth. The SPP requires the youth to initiate conversation with a researcher described as shy and then give a speech on themselves and what their school is like. Salivary samples were collected on four baseline

occasions that spanned a 24 hour time period. Parents and adolescents completed the Child Behavior Checklist and were administered the Diagnostic Schedule for Children, Version IV. Researchers found that factors such as age, gender, and time of day are linked to cortisol reactivity but did not find that underarousal was associated with externalizing behavior.

Another finding contrary to other research is from a study of children in day care. Researchers studied 36 preschool age children and 34 school age children by collecting saliva samples mid-morning and mid-afternoon on two days at the school and two days at the home. Parents and teachers of the children completed modified versions of the Child Behavior Questionnaire. The cortisol levels of the children in the preschool classes increased throughout the day, whereas the level of increase for school age children was less. Controlling for age, the researchers found that cortisol levels in aggressive children actually increased throughout the day (Dettling et al., 1999). Researchers believed the increase was due to the poor regulatory skills of the young children and the stress it caused them.

Some researchers believe that low resting cortisol is related to personality traits and not necessarily aggression (Shoal et al., 2003). Individuals who engage in aggressive behaviors can be characterized by certain personality traits. Shoal et al. (2003) found that the relationship between cortisol and aggressive behavior was largely accounted for by self-control. People with high levels of resting cortisol are more likely to be cautious and sensitive to punishment, while people with lower resting cortisol rates may have reduced self-control, low harm-avoidance, and irritability (Shoal

et al., 2003). Young boys on the extremes of behavior, behavior dysregulation compared to behavior inhibition, have cortisol levels on opposite ends of the spectrum (van Goozen et al., 1998). Therefore, boys who engaged in problem behaviors had low cortisol levels and boys who were shy and inhibited had high cortisol levels.

α-amylase and Aggression

Although the research is growing, studies using α-amylase are even fewer than studies involving cortisol and aggression. Some research has indicated that individual differences in salivary α-amylase are associated with problem behavior (Kivlighan & Granger, 2006). However, most studies that look at the autonomic nervous system have used heart rate and skin conductance to measure arousal. One of the strongest, most replicated findings with regards to heart rate is that antisocial children and adolescents have lower resting heart rates (Scarpa & Raine, 1997). However, these studies did not differentiate aggressive and non-aggressive antisocial behavior. Studies of skin conductance found some evidence of underarousal in antisocial individuals (Scarpa & Raine, 1997).

Recently there has been an appeal for research focusing on the relationship between the HPA axis and autonomic SNS and on how they each individually relate to aggressive behavior. A recent study by Gordis et al. (2006) used a multiple system integrative approach to studying aggressive behavior. It is believed that interactions between the two systems could have an impact on behavior. Gordis et al. (2006) examined the asymmetry between physiological stress and aggressive behavior in 67 maltreated youth. A modified version of the Trier Social Stress Task was used to

induce a stress response in the youth. A total of six saliva samples were collected before and after the stress task. Parents and the youth were also asked to complete the Reactive-Proactive Aggression questionnaire that measured the frequency that the youth engaged in certain retaliatory and unprovoked aggressive behaviors. Researchers found the interaction between cortisol and alpha-amylase significantly predicted parent reports of aggression. Furthermore, asymmetry in the two systems was associated with lower rates of aggressive behavior, whereas symmetry in the direction of low activity was related to higher rates of aggression. Individuals with low levels on both systems may have extremely uninhibited behaviors which leads to this increase in aggression. This study also had important implications for future research using these two markers of physiological stress response. Although researchers included boys and girls in this study, data was not analyzed by gender. Also, researchers did not look at subtypes of aggression.

Aside from the Gordis et al. study, there has been limited research on the interactions between the SNS and HPA axis. One study utilized basal levels of cortisol, sAA, and skin conductance. El-Sheikh and colleagues (2008) examined the interaction between the two systems and its relationship to internalizing and externalizing behaviors. Similar to the work of Gordis et al. (2006), researchers found symmetry indicated higher levels of problem behaviors, especially for participants with high activity for both systems (El-Sheikh et al., 2008). However, when cortisol and sAA were examined individually, no relationship to internalizing or externalizing behaviors was found. Stroud et al. (2006) found that the direction of the asymmetry predicted the

outcome. Low cortisol and high sAA was associated with more positive behaviors (e.g. activities, social, school) as measured by the CBCL, but high cortisol and low sAA was associated with more externalizing behaviors (e.g. social problems, thought problems, aggression, delinquency) (Stroud et al., 2006). The above studies provide support for the importance of a multisystem approach to physiological studies.

Cortisol and α-amylase have been associated with aggression, but results are equivocal. Additionally, much of the research that has been conducted has not distinguished between various types of aggression. The mixed results of research in this area may be the result of some issues associated with studying cortisol and α-amylase.

Confounds in Physiological Research

As with studies of aggressive behavior, many of the studies on cortisol have involved boys. However, researchers have determined that there are gender differences associated with cortisol. In adults, a series of four studies examined gender differences in basal cortisol and cortisol reactivity surrounding psychological and physiological stressors (Kirschbaum, Wüst, & Hellhammer, 1992). Researchers concluded that gender differences in response to these situations do exist, which could influence cortisol secretion. Females show higher levels of cortisol at midday and in late afternoon than males (Klimes-Dougan et al., 2001). Low cortisol levels are not always associated with externalizing behaviors in females (Shirtcliff, Granger, Booth, & Johnson, 2005). This may be due to biological differences in how genders deal with stress or the fact that researchers have overlooked females in previous research. One study on adolescent girls in their final stages of puberty who met the criteria for conduct

disorder found an association between conduct disorder and low cortisol levels (Pajer et al., 2001). In a study of both boys and girls, the association between low cortisol levels and externalizing behaviors was only found in boys (Shirtcliff et al., 2005).

The time of day can impact cortisol levels. For most people, cortisol levels peak during the final few hours of sleep in the morning and decrease throughout the day (Dettling et al., 1999; Pajer et al., 2001; Klimes-Dougan et al., 2001). Susman et al. (2007) examined the relationship between a.m. and p.m. cortisol ratios and aggressive behavior problems. In their study, 111 boys and girls ages 8 to 13 were assessed to determine the relationship between morningness and eveningness, morning to afternoon cortisol ratios, pubertal timing, and antisocial behavior. Morningness and eveningness describes an individual's sleep wake patterns, preference for when to engage in activities, and level of alertness during the morning (Susman et al., 2007). Eveningness is associated with behavior problems, such as poorer adjustment and antisocial behavior, in adolescence. The morning-to-afternoon cortisol ratio was important to researchers because they believed the morning-to-afternoon cortisol ratio were a better indicator of the relationship between cortisol and antisocial behavior than obtaining cortisol levels at any one point during the day. Researchers found that eveningness was associated with rule-breaking behavior, total antisocial behavior, and conduct disorder symptoms in boys, but not for girls. However, eveningness was significantly associated with relational aggression in girls. The morning-to-afternoon cortisol ratio was not related to aggressive behavior or rule breaking in any of the sample (Susman et al., 2007).

Despite the changes that occur in cortisol levels, studies have frequently involved single measurements of cortisol levels (Moss et al., 1995; van Goozen et al., 1998). By collecting a basal measurement, as well as follow up measurements, surrounding an anticipated event, a researcher can monitor changes associated with the event. It has been determined that cortisol peaks at 10 minutes following a stressor, but sAA peaks immediately following the stressor (Gordis et al., 2006). This means when collecting saliva samples, multiple samples will be necessary to get accurate measures of peak levels. A restricted range of variability in cortisol levels may be a better indicator of aggression than a low concentration of cortisol captured at one point in time (McBurnett, Lahey, Rathouz, & Loeber, 2000).

Another concern when conducting research on adolescents is the impact of puberty. Changes during puberty influence hormones. The morning increase in cortisol levels is lower in adolescents than in adults, which indicates there might be a maturational component to the morning rise (Susman et al., 2007). Some research has shown that there may also be a maturational component to sAA. Pubertal status and age were found to have a positive relationship with sAA, but this result has been replicated only in boys (El-Sheikh et al., 2005; Susman et al., 2006).

Research on the physiological correlates of aggression, in particular research that focuses on aggressive behavior in females is sparse. The increase in aggressive behavior in females makes research in this area important to prevention and intervention development. This paper will focus on the physiological correlates of aggressive behavior in females. This study expanded on work by Gordis et al. (2006) and other

researchers on the HPA axis and sympathetic nervous system responses and aggression by using a larger sample, focusing on female adolescents, examining both physical and relational aggression, studying a predominantly urban population, and utilizing parent and adolescent reports of aggressive behavior. In particular, I examined salivary cortisol and sAA as markers of physiological response. Based on prior literature, I expected that lower levels of salivary cortisol taken at the beginning of the interview and the beginning of the stress task would be associated with higher levels of physical and relational aggression in girls. I also hypothesized that lower levels of cortisol and α-amylase reactivity would be associated with higher levels of physical and relational aggression. Finally, I hypothesized that lower levels of cortisol reactivity coupled with higher levels of sAA reactivity would be associated with lower levels of both physical and relational aggression.

Methods

Participants

This paper is based on a longitudinal study that examines exposure to violence and coping in at-risk youth. The parent study, Project COPE, collected information on stressors, coping, substance use, problem behavior, parenting/caregiver practices, and psychological adjustment. At Wave 1, 358 families (a 5th or 8th grade student and a female caregiver) completed interviews, and 88% of those families were retained in Wave 2 (N=319). A 5th or 8th grade student and a maternal caregiver participated from each family and 86% of Wave 2 families were retained in Wave 3 (N = 274). Each wave of data was collected approximately one year apart. Only caregivers and female students were used in the current study. Female youth who had 5 saliva samples and completed assays for cortisol and sAA were used in the sample. There were 146 female adolescents used for analysis in the current study, and most (91.1%) self-identified as African-American. The mean was 13.9 years old (age range from= 11-18). The majority of female caregivers who participated in the study were the biological mother (83.6%) and 91.8% of these caregivers self-identified as African- American. Caregiver education was diverse: 26.7% did not complete high school, 24.7% had a high school diploma or a GED, 24% had some college, but no degree, 9.6% had a vocational degree, and 15.1% had an associate's degree or beyond. The majority of participants reported household earnings between $201-400 per week and 54.6% of households had incomes below the poverty line.

Procedure

Participants were recruited from areas that had moderate to extreme amounts of violence, based on police crime statistics. Flyers were placed in these communities as well as at local community centers, Boys and Girls Clubs, YMCAs, churches, and with tenant organizations. To be eligible, families must have had a 5th or 8th grade student and be able to complete the protocol in English. Just under two-thirds (62%) of the eligible households that were contacted gave consent to enroll in the study. Data was collected on a yearly basis using face-to-face parent and child interviews. Most interviews were conducted in the home, though at the families' request some interviews were conducted elsewhere. Interviewers completed approximately 20 hours of training on interviewing techniques and on the specific protocol, including conducting and being evaluated on practice interviews prior to conducting interviews. Before beginning the interview, parents and youth were reminded about confidentiality for the project and their right to withdraw at any time. Both parent and child were required to complete consent or assent form to participate (Appendix A). Families were compensated $50 in Wal-Mart gift cards at each wave of the study. Participants were included if they completed assays on five samples of saliva at either Wave 2 or Wave 3. This is due to the fact that initial saliva collection was based on cortisol reactivity and not adequate to capture the reactivity in α-amylase. Additional saliva collection samples were added to Wave 2 in October 2006 and included in the procedure for Wave 3. Data for the current study are based on the unique families who participated in the study beginning in

October, 2006 and for whom adequate saliva samples were obtained. Thus, some data from Waves 2 and Waves 3 was used, but no families' data was used more than once.

The study protocol and saliva collection procedures were approved by Virginia Commonwealth University's Institutional Review Board. The interview consisted of multiple instruments, a parent questionnaire, a child questionnaire, and a child booklet that was completed without the assistance of the interviewer if the child's reading level allowed. The parent interview contained the Child Behavior Checklist. The Problem Behavior Frequency scales and Social Competence Interview (SCI; Ewart & Kolodner, 1991) were part of the child protocol. The SCI was the only portion of the interview that was audiotaped.

Measures

Physiological Measures

The physiological data was collected using salivettes. Saliva samples were taken at the beginning of the child interview, as well as before, during, and after the SCI. The SCI measures physiological changes that occur when the participant is asked to relive a stressful life situation (Ewart & Kolodner, 1991). The SCI is designed to promote physiological arousal and has been repeatedly correlated with changes in blood pressure and heart rate (Chen, Matthews, Salomon, & Ewart, 2002; Ewart & Kolodner, 1991). These physiological changes are different for each individual. Unlike other studies that use performance based tasks as a stressor, the SCI elicits details about social and environmental stressors in the participant's life. The SCI has two phases, a hot phase and a cold phase. During the hot phase, the interviewer asks the child to re-

experience the stressful event and asks questions about the participant's thoughts and feelings during the event. The cool phase follows with the interviewer asking the participant to describe how the situation would have ideally ended and what could be done to achieve that outcome. Thus, the specific stressor discussed differs for each individual.

Saliva samples were used to collect the physiological data with samples being taken prior to starting the SCI, at the end of the hot phase, 10 minutes after the end of the hot phase, 20 minutes after the end of the hot phase of the SCI, and then again 20 minutes later, for a total of 5 samples. An additional sample was collected at the start of the interview. Adolescents were asked by the interviewer to place a cotton swab in their mouth and chew for about one minute. The adolescents were informed not to eat or drink anything with caffeine after the first sample was taken, and they were allowed to consume only water between samples #2 and #6. The child spit the swab into the salivette tube and the samples were frozen at a -70 degrees Centigrade or below until the samples were taken to the laboratory for analysis. The procedure for saliva collection is in Appendix B. The saliva samples were assayed at the General Clinical Research Center at Virginia Commonwealth University for the stress hormone cortisol and the enzyme α-amylase.

Aggressive Behavior Outcomes

Aggression was measured using the aggression subscale of the Child Behavior Checklist (CBCL) and Problem Behavior Frequency Scales. The Child Behavior Checklist contains a series of 113 items that help assess a child's behavioral and

emotional problems over the past three months; it is completed by the parent (Appendix C) (Achenbach & Ruffle, 2000). The CBCL is widely used and has excellent reliability and validity (Achenbach, 1991). Syndromes on the CBCL are classified into one of six areas; anxious/depressed, withdrawn, sleep problems, somatic problems, aggressive behavior, and destructive behavior. Respondents on the CBCL rate each item on a three-point scale; not true, somewhat or sometimes true, very true or very often (Hudziak, Copeland, Stanger, & Wadsworth, 2004). The aggression subscale of the CBCL contains 20 items, including "is mean to others," "destroys own things," and "is disobedient at school." The internal consistency of the Aggressive syndrome scale in a sample of urban youth was .91 (Kliewer et al., 2004). The test-retest reliability for the externalizing subscales ranges from .64 to .69 (Achenbach, 1991). The CBCL is widely used and convergence has been demonstrated between the DSM-IV disorders and the CBCL syndromes (Hudziak et al., 2004). Higher scores indicate more aggressive behavior. Internal consistency (Cronbach's alpha) for the current study was .92.

The Problem Behavior Frequency Scales (PBFS; Farrell et al., 2000) is a self-report measure that assesses problem behaviors including aggression, victimization, drug use, and delinquency. Aggression subscales include measures of physical, non-physical, and relational aggression. Respondents are asked how frequently they engaged in problem behaviors over the past 30 days (Appendix D) (Sullivan, Farrell, & Kliewer, 2006). Responses were rated on a six-point scale: never, 1-2 times, 3-5 times, 6-9 times, 10-19 times, and 20 times or more. The physical aggression subscale included seven items such as "threatened to hit or physically harm another kid" and "hit

or slapped another kid." These items were based on the Centers for Disease Control's Youth Risk Survey (Sullivan et al., 2006). The non-physical aggression subscale consisted of five items including "teased someone to make them angry," "put someone down to their face," and "gave mean looks to another student." The relational aggression subscale items were based on a measure of relational aggression developed by Crick and Grotpeter. This scale was comprised of six items that included direct and indirect forms of relational aggression such as "spread a false rumor about someone" and "told another kid you wouldn't like them unless they did what you wanted them to do." The reliability was strong for the physical aggression scale (.86) and the relational aggression scale (.76) when the scale was utilized in an urban sample of adolescents (Sullivan et al., 2006). Internal consistency (Cronbach's alpha) in the current study was .78 for physical aggression and .65 for relational aggression.

Control Variables

Pubertal Status. Pubertal status was measured using the Pubertal Development scale developed by Peterson, Crockett, Richards, and Boxer (1988) (Appendix E.). This scale is a non-verbal assessment of pubertal status that requires the adolescent to answer questions pertaining to the degree of his or her own pubertal status (Peterson et al., 1988). Regardless of gender, all adolescents are asked to answer items on growth spurt, pubic hair, and skin change. Boys have additional questions about facial hair and girls have additional questions about menarche and breast development. The four item response scale provides responses that allow the adolescent to tell where they are in pubertal development; has not yet begun, has barely started, is definitely underway, and

growth or development is complete. The reliability of the items ranges from .68 to .83 (Peterson et al., 1988). Developmental differences in cortisol and sAA have been described in the literature. Differences between adolescents and adults in the morning rise of cortisol imply that there is a maturational component to cortisol levels (Susman et al., 2006). With α-amylase, older participants (13-17 years) have been found to show greater response to interpersonal stressors compared to younger participants (7-12 years) (Granger, Kivlighan, El-Sheikh, Gordis, & Stroud, 2007). Studies have found higher basal cortisol levels in older adolescents (Stroud et al., in press).

Medication status. Previous studies have shown that medication can impact salivary cortisol and α-amylase. Medications such as steroid based anti-inflammatories, oral contraceptives, and diuretics cause individual differences in cortisol (Hibel, Granger, Kivlighan, & Blair, 2006). Antipsychotics and hypotensives have also been associated with atypically flat cortisol levels throughout the day (Hibel, Granger, Cicchetti, & Rogosch, 2007). There have been similar findings for sAA. Prescription medications that control high blood pressure and have beta-blocking properties or consumables that stimulate the SNS, such as caffeine, can increase salivary α-amylase (Granger et al., 2007). Conversely, nicotine is negatively associated with sAA activity (Granger et al., 2007). To control for medication, medication was coded use or no use. A sizable percentage of the sample (43.8%) reported being on medication. Two questions in the interview asked if the participant has ever smoked cigarettes and how frequently the participant smoked in the past month. Tobacco was coded as use in the

past month or no use to control for nicotine. Just under 10% (9.7%) of the sample reported smoking in the past month.

Race. Although research is limited, several studies have examined race and cortisol levels. These studies have found flatter diurnal cortisol rhythms in African-Americans and Hispanics compared to Caucasians (DeSantis et al., 2007). Results of this study also indicated that cortisol levels at bedtime and waking are higher for African-Americans. Race was controlled for by comparing African American adolescents to adolescents in other racial groups. Most (93.3%) of the sample was African American.

Time of day. Time of day was controlled due to the variations in cortisol levels throughout the day described in the literature. Interviews in this study were designed to meet the schedules of the families and therefore, interviews took place throughout the day which could influence levels of cortisol and sAA.

Results

Preliminary Data Analyses

Cortisol and sAA data was examined for outliers. The data of three participants were eliminated from further data analysis because cortisol or sAA values were greater than 3 standard deviations from the mean._Additionally, five participants with missing data were removed from analyses, which made the total sample size 138. Descriptives were calculated for the aggression measures; CBCL aggressive behavior, M= 9.92 SD = 8.08, PBFS physical aggression, M = 3.19 SD = 4.28, PBFS relational aggression, M = 2.01 SD = 3.37. I ran a t-test to examine differences on my control and outcome variables between data collected at Wave 2 and data collected at Wave 3. There were no significant differences on any variable, *p*s < .05. I also examined correlations between all variables used in analysis. Table 1 presents correlations among the outcome, predictor, and control variables. Notably, cortisol and sAA were uncorrelated. This result is similar to Gordis et al.'s (2008) finding showing no correspondence between cortisol and sAA in their maltreated sample.

I reviewed the SCI for each participant prior to beginning analysis. Participants were excluded from analyses if the SCI (Ewart & Kolodner, 1991) was incomplete, if a stressful event was not recalled, or if the participant was not engaged in the process based on the interviewer's impression. The distribution of cortisol and sAA at each of the five time points can be seen in Figure 1, with further information on descriptives available in Table 2. The increase in sAA at the final timepoint is of some concern and may be associated with sensitive questions being asked towards the end of the

interview. Therefore, analyses were run without the final saliva sample which was collected 40 minutes after the hot phase.

Table 1

Intercorrelations among all covariates, predictors, and outcome variables

	1	2	3	4	5	6	7	8
1. Aggression- parent report	---							
2. Physical aggression- adolescent report	.353**	---						
3. Relational aggression- adolescent report	.092	.559**	---					
4. Cortisol AUC_G	.166*	.134	.055	---				
5. AA AUC_G	-.041	.042	-.030	-.044	---			
6. Cortisol AUC_I	.160	.142	.063	.992**	-.030	---		
7. AA AUC_I	-.042	.031	-.020	-.036	.994**	-.022	---	
8. Average cortisol	.135	.007	-.020	.834**	-.025	.839**	-.020	---
9. Average AA	-.048	.049	-.032	-.008	.973**	.005	.964**	.014
10 Cortisol reactivity	-.099	.061	.060	-.485**	.079	-.430**	.081	-.687**
11. AA reactivity	-.060	-.072	-.020	-.003	.213**	-.017	.184*	-.056
12. Time of day	-.046	.009	.048	-.475**	.097	-.458**	.086	-.398**
13. Race	-.045	-.026	-.078	.023	.047	.044	.052	.042
14. Pubertal status	.099	.118	.027	-.004	.036	.010	.050	-.061
15. Medication status	.050	-.085	-.049	.112	.036	.107	.035	.156
16. Tobacco use	.208*	.116	-.086	.059	.061	.065	.055	.008

Note: AA = α-amylase, AUC_G = Area under the curve ground, AUC_I = Area under the curve increase.
*$p < .05$.

	9	10	11	12	13	14	15
1. Aggression- parent report							
2. Physical aggression- adolescent report							
3. Relational aggression- adolescent report							
4. Cortisol AUC_G							
5. AA AUC_G							
6. Cortisol AUC_I							
7. AA AUC_I							
8. Average cortisol							
9. Average AA	---						
10 Cortisol reactivity	.052	---					
11. AA reactivity	.213**	.016	---				
12. Time of day	.079	.228**	.033	---			
13. Race	.067	.038	-.097	.018	---		
14. Pubertal status	.010	.194*	-.040	.110	-.039	---	
15. Medication status	.009	-.148	.029	-.173*	-.140	.076	---
16. Tobacco use	.082	.042	.223**	-.027	.022	.118	-.071

Note: AA = α-amylase, AUC_G = Area under the curve ground, AUC_I = Area under the curve increase.
*$p < .05$

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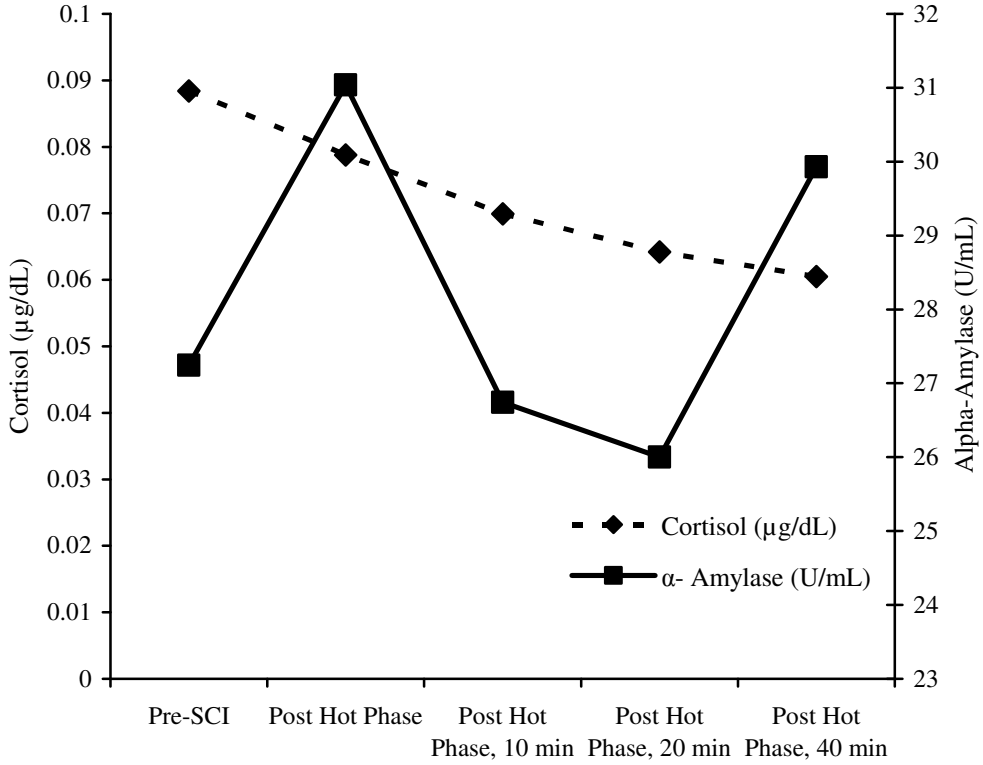

Figure 1 Cortisol and sAA means surrounding the SCI.

Previous research studies with more than three cortisol and α-amylase collection time points have used different analysis techniques to examine data. Three different methods were used in the current study; 1) Area Under the Curve, 2) Reactivity and Recovery Phase, 3) Averaging Samples. Each of these methods will be described below prior to presenting the results.

Gordis et al. (2008) used area under the curve (AUC) to reflect the total output for cortisol and α-amylase. Pruessner et al. (2003) presented two formulas to measure

hormonal output: Area Under the Curve Ground (AUC_G) and Area Under the Curve Increase (AUC_I). The AUC_G was used based on previous research on cortisol and α-amylase with multiple data collection time points. The AUC_I was utilized because it is a significant measure of how an individual responds to stress (Pruessner et al., 2003).

Table 2

Change in time for cortisol and sAA

	M	SD	Range
Cortisol (µg/dL)			
Pre –SCI	.088	.110	.00 – .87
Post hot phase	.079	.104	.00 - .85
Post hot phase, 10 min	.070	.075	.00 - .58
Post hot phase, 20 min	.064	.063	.00 - .50
Post hot phase, 40 min	.061	.087	.00 - .88
α- Amylase (U/mL)			
Pre –SCI	27.246	22.215	.66 – 130.08
Post hot phase	31.039	29.844	.98 – 220.19
Post hot phase, 10 min	26.741	24.084	1.31 – 159.24
Post hot phase, 20 min	26.007	20.712	.98 – 99.90
Post hot phase, 40 min	29.931	25.514	.98 – 161.86

For this method, I began by calculating AUC based on the four data collection time points. For the data, there is no standard time for the first interval, so the average time was calculated and substituted for the first interval. For the AUC analyses, seven participants had AUC scores that were outliers (> 3 SDs) and were eliminated from analyses. Due to the skew of the cortisol and α-amylase scores, transformations were performed based on previous research and statistical recommendations (Gordis et al., 2006; Tabachnick & Fiddell, 2001). The cortisol (AUC_G skew = 2.46, S.E. = .20; AUC_I skew = 2.06, S.E. = .20) were transformed using the natural log (AUC_G skew = .34, S.E.

= .20; AUC_I skew = .56, S.E. = .20). sAA scores (AUC_G skew = 1.72, S.E. = .20; AUC_I skew = 1.69, S.E. = .20) were transformed using a square root transformation (AUC_G skew = .58 , S.E. = .20; AUC_I skew = .60, S.E. = .20).

The second method utilized was to calculate the amount of change between the baseline value and the phase where the measure peaks and the difference. Susman's (2008) work suggests that there is a different process involved with initial reactivity compared to recovery. The reactivity phase for cortisol was calculated based on the value before the SCI and 10 minutes after the hot phase. For sAA, the difference was calculated between the measurement before the SCI and immediately following the hot phase. The recovery phase for cortisol was calculated using the saliva sample 20 minutes after the SCI and 40 minutes after the SCI. Four outliers were removed for being greater than three standard deviations from the mean. The recovery phase for sAA was calculated using samples at 10 minutes post-SCI and 20 minutes post-SCI. Five outliers had to be removed for values greater than three standard deviations from the mean. The saliva samples selected were based on previous literature about the reactivity and recovery of cortisol and sAA.

The final analytic strategy utilized was to calculate an average across all of the time points. El-Shiekh et al. (2008) indicated that when there is stability of cortisol and sAA surrounding a task, the scores can be averaged to represent a basal level. For the current study, the values across the SCI task remained stable for cortisol, $r = .73$, $p < .01$, and sAA, $r = .77$, $p < .01$. The average values were calculated using the first three saliva samples collected around the SCI. This focused on reactivity and eliminated the

final two timepoints, which are recovery values. Six participants had averages greater than 3 SDs from the mean and were eliminated from analyses. Again, the cortisol and sAA data was skewed and had to be transformed. The cortisol (skew = 1.01, S.E. = .21) were transformed using the natural log (skew = .87, S.E. = .21). sAA scores (skew = 1.22, S.E. = .21) were transformed using a square root transformation (skew = .34 , S.E. = .21). The averages were centered prior to regression analysis.

Regression Analyses

The main effect of basal cortisol and cortisol at the start of the stress task on aggression was examined using hierarchical linear regression (hypotheses 1 and 2). Regression analyses controlled for time of day, pubertal status, gender, race, medication use, and tobacco use. Two participants were identified as outliers based on Cook's D distance measure (Cook & Weisberg, 1982) and removed from analysis. A significant main effect was found for cortisol at the start of the interview and parent report of aggressive behavior. A significant main effect was also found for cortisol at the start of the SCI and parent report of aggressive behavior. For both main effects, higher levels of basal cortisol and cortisol at the start of the SCI were associated with higher levels of parent-reported aggressive behavior. No significant main effect was found for adolescent reported physical or relational aggression.

The final two hypotheses were based on the interaction between cortisol and sAA. Hierarchical multiple regression was used to examine the interaction between the HPA axis and sympathetic nervous system as measured by salivary cortisol and sAA.

The regression analyses controlled for pubertal status, medication use, tobacco use, race, and time of day.

The analyses using the first method described above were run using the AUC values. Two to six participants were identified as outliers based on Cook's D distance measure (Cook & Weisberg, 1982) and removed from analysis. As seen in Table 3, a significant interaction was not found for parent-reported aggressive behavior, adolescent-reported physical aggression, or adolescent-reported relational aggression. However, significant main effects were found for cortisol when predicting adolescent-reported physical aggression, and parent-reported aggressive behavior. Similar to the data for hypotheses 1 and 2, higher levels of cortisol were associated with more aggressive behavior. There was not a significant main effect of cortisol for adolescent-reported relational aggression.

Table 3

AUC regression equations predicting parent-reported and adolescent-reported aggression from cortisol, sAA, Cortisol X sAA interactions and controls

	β	T	F	ΔR^2
Aggression (Parent report)				
Step 1: Covariates			1.63	.06
Step 2: Main Effects			2.07*	.05
Cortisol AUC_G	.23	2.44*		
sAA AUC_G	-.02	-.27		
Step 3: Cortisol AUC_G X sAA AUC_G	-.08	-.90	1.91	.01
Step 1: Covariates			1.63	.06
Step 2: Main Effects			2.01	.04
Cortisol AUC_I	.23	2.36*		
sAA AUC_I	-.03	-.29		
Step 3: Cortisol AUC_I X sAA AUC_I	-.07	-.77	1.83	.00
Physical aggression (Adolescent report)				
Step 1: Covariates			1.40	.05
Step 2: Main Effects			1.70	.04
Cortisol AUC_G	.21	2.16*		
sAA AUC_G	-.02	-.26		
Step 3: Cortisol AUC_G X sAA AUC_G	.02	.19	1.48	.00
Step 1: Covariates			1.40	.05
Step 2: Main Effects			1.79	.04
Cortisol AUC_I	.22	2.27*		
sAA AUC_I	-.03	-.38		
Step 3: Cortisol AUC_I X sAA AUC_I	.02	.26	1.56	.00

	β	T	F	ΔR^2
Relational aggression (Adolescent report)				
Step 1: Covariates			.40	.02
Step 2: Main Effects			.62	.02
Cortisol AUC_G	.15	1.45		
sAA AUC_G	-.04	-.46		
Step 3: Cortisol AUC_G X sAA AUC_G	.03	.36	.55	.00
Step 1: Covariates			.40	.02
Step 2: Main Effects			.68	.02
Cortisol AUC_I	.15	1.52		
sAA AUC_I	-.07	-.71		
Step 3: Cortisol AUC_I X sAA AUC_I	.02	.24	.60	.00

Note. Equations control for pubertal status, time of day, race (African American vs other), medication use, and tobacco use. * $p < .05$

Analyses were also run by breaking the samples into a reactivity phase and a recovery phase. Hierarchical regression was used to determine if the amount of change between the start SCI and the physiological variable's peak predicted aggressive behavior. The results for the reactivity phase can be seen in Table 4. Two to five participants were identified as outliers based on Cook's D distance measure (Cook & Weisberg, 1982) and removed from analysis. A significant interaction was not found in the reactivity phase for parent-reported aggressive behavior, adolescent-reported physical aggression, or adolescent-reported relational aggression. No significant main effects were found for cortisol. Hierarchical regression was also used to determine the association between aggressive behavior and the amount of change during the recovery period and these results are presented in Table 5. One to two participants were identified as outliers based on Cook's D distance measure (Cook & Weisberg, 1982) and removed from analysis. Although the overall model was not significant, a significant interaction was found for adolescent-reported physical aggression. In that model, when both cortisol and sAA were low or both were high, aggressive behavior was low. When cortisol was high and sAA was low, the adolescents reported higher levels of physical aggression. The graph of the interaction can be seen in Figure 2. A significant interaction was not found for parent-reported aggressive behavior or adolescent-reported relational aggression. No significant main effects were found for cortisol.

Table 4

Regression equations predicting parent-reported and adolescent-reported aggression from the reactivity phase of cortisol, sAA, Cortisol X sAA interactions and controls

	β	*T*	*F*	*Δ R²*
Aggression (Parent report)				
Step 1: Covariates			1.42	.05
Step 2: Main Effects			1.57	.03
Cortisol reactivity phase	-.17	-1.89		
sAA reactivity phase	-.05	-.60		
Step 3: Cortisol reactivity X sAA reactivity	-.16	-1.65	1.74	.02
Physical aggression (Adolescent report)				
Step 1: Covariates			1.27	.05
Step 2: Main Effects			1.05	.01
Cortisol reactivity phase	.01	.16		
sAA reactivity phase	-.09	-.98		
Step 3: Cortisol reactivity X sAA reactivity	-.02	-.18	.92	.00
Relational aggression (Adolescent report)				
Step 1: Covariates			1.21	.05
Step 2: Main Effects			.97	.01
Cortisol reactivity phase	.08	.84		
sAA reactivity phase	-.01	-.16		
Step 3: Cortisol reactivity X sAA reactivity	.02	.25	.85	.00

Note. Equations control for pubertal status, time of day, race (African American vs other), medication use, and tobacco use.
* $p < .05$

The interaction was also examined utilizing the average value of three of the saliva samples. As with other analyses, hierarchical regression controlling for the same variables was used. Two to seven participants were identified as outliers based on Cook's D distance measure (Cook & Weisberg, 1982) and removed from analysis. As reported in Table 6, a significant interaction was not found for parent-reported

aggressive behavior, adolescent-reported physical aggression, or adolescent-reported relational aggression. Significant main effects were found for average cortisol on adolescent-reported physical aggression and parent-reported aggressive behavior. As with prior analyses, higher levels of cortisol were associated with higher levels of aggressive behavior.

Table 5

Regression equations predicting parent-reported and adolescent-reported aggression from the recovery phase of cortisol, sAA, Cortisol X sAA interactions and controls

	β	*T*	*F*	ΔR^2
Aggression (Parent report)				
Step 1: Covariates			.75	.03
Step 2: Main Effects			.57	.00
Cortisol recovery phase	-.04	-.48		
sAA recovery phase	-.02	-.25		
Step 3: Cortisol recovery X sAA recovery	-.14	-1.46	.77	.02
Physical aggression (Adolescent report)				
Step 1: Covariates			1.38	.05
Step 2: Main Effects			.99	.00
Cortisol recovery phase	.03	.47		
sAA recovery phase	-.01	-.13		
Step 3: Cortisol recovery X sAA recovery	-.21	-2.24*	1.52	.04
Relational aggression (Adolescent report)				
Step 1: Covariates			1.40	.05
Step 2: Main Effects			.99	.00
Cortisol recovery phase	-.02	-.17		
sAA recovery phase	-.01	-.14		
Step 3: Cortisol recovery X sAA recovery	-.05	-.49	.89	.00

Note. Equations control for pubertal status, time of day, race (African American vs other), medication use, and tobacco use.
* $p < .05$

Table 6

Regression equations predicting parent-reported and adolescent-reported aggression from the average of cortisol, sAA, Cortisol X sAA interactions and controls

	β	*T*	*F*	*Δ R²*
Aggression (Parent report)				
Step 1: Covariates			.99	.04
Step 2: Main Effects			1.97	.06
Cortisol average	.28	2.29*		
sAA average	-.01	-.07		
Step 3: Cortisol average X sAA average	-.09	-.98	1.85	.08
Physical aggression (Adolescent report)				
Step 1: Covariates			1.40	.05
Step 2: Main Effects			1.64	.03
Cortisol average phase	.20	2.07*		
sAA average phase	-.01	-.12		
Step 3: Cortisol average X sAA average	-.01	-.12	1.42	.00
Relational aggression (Adolescent report)				
Step 1: Covariates			.40	.02
Step 2: Main Effects			.48	.01
Cortisol average phase	.11	1.09		
sAA average phase	-.04	-.40		
Step 3: Cortisol average X sAA average	.04	.46	.44	.00

Note. Equations control for pubertal status, time of day, race (African American vs other), medication use, and tobacco use.
*$p<.05$

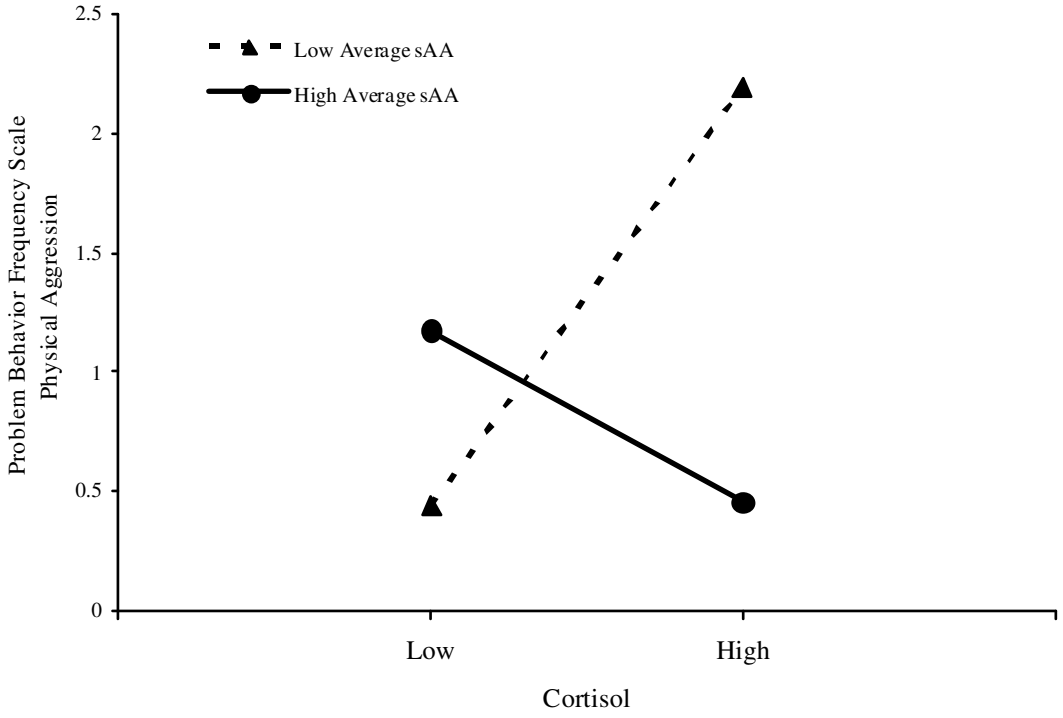

Figure 2 Relationship between recovery phase cortisol and sAA and adolescent-reported physical aggression.

Discussion

This study examined the relationship between cortisol and sAA in predicting aggressive behavior in adolescent females living in low-resources areas of a mid-sized southern city. The first two hypotheses were not supported, and in fact, findings opposite to what was expected were obtained. I found that *higher* levels of cortisol were associated with higher levels of parent-reported aggressive behavior and adolescent-reported physical aggression in this sample of girls. This main effect was found consistently across the various analytic techniques. I should note that cortisol levels overall in our sample were low, and only a quarter (27%) of the sample showed increases in cortisol in response to the task. Thus, the data should be interpreted with this in mind. Although previous research on females has been somewhat mixed, research on cortisol and aggressive behaviors has indicated that *lower reactivity* is associated with higher levels of aggressive behavior (Gordis et al., 2006; Moss et al., 1995; Shoal et al., 2003; van Goozen et al., 1998). This result has consistently been found in boys. With girls, these results have been more mixed. El-Shiekh and colleagues (2008) note that the positive association between externalizing behaviors and cortisol levels has been found more frequently in community samples, like the one in this study, than clinic samples. It is possible that with girls there is something different happening. This is especially true for girls in the age range included in the current study. It has been established that cortisol levels are influenced by puberty, but puberty interferes with hormones in ways that researchers do not yet fully understand. Further

research is needed to determine whether the current findings are consistent with other samples of girls.

I also hypothesized that the interaction between cortisol and sAA reactivity would influence levels of aggression. Specifically, I thought that lower levels of cortisol and α-amylase reactivity would be associated with higher levels of physical and relational aggression and that lower levels of cortisol reactivity coupled with higher levels of sAA reactivity would be associated with lower levels of both physical and relational aggression.

A significant interaction between salivary cortisol and sAA was found for the recovery phase. When the cortisol reactivity was high and sAA was high or when both were high, adolescent females engaged in less physically aggressive behavior. When cortisol reactivity was low and sAA was high, adolescent females engaged in more aggressive behavior. This was also true when cortisol was high and sAA was low. For the current population, *symmetry* in the systems was associated with lower levels of self-report physical aggression and *asymmetry* was associated with higher levels of aggressive behavior. This finding on asymmetry is supported by the work presented by Stroud et al. (2006) where adolescents with high cortisol and low sAA had higher levels of externalizing behaviors. However, the finding is contrary to the work of Gordis et al. (2006) who found that asymmetry was associated with lower levels of parent-reported aggressive behavior. Gordis et al. (2006) also found that symmetry in the direction of low activity in both systems was associated with more aggression. It is also is contrary to the "additive" hypothesis posited by Bauer et al. (2002). The "additive" hypothesis

states that moderate arousal or asymmetrical arousal will be associated with fewer behavior problems (Bauer et al., 2002; El-Shiekh et al., 2008). In the current study, the asymmetry was associated with higher levels of physically aggressive behavior but not relationally aggressive behavior. Given the significant association of physical and relational aggression in this sample, this finding was surprising. Despite their association, physical and relational aggression have different profiles. Both forms of aggression involve the intent to harm, but harm is achieved through different means. Physical aggression is characterized by behaviors that physically injure another individual such as kicking and punching (Underwood, 2003). Relational aggression is a more subtle form of aggression that typically involves manipulation of relationships (Underwood, 2003). Differences in the affective and cognitive processes underlying these two forms of aggression could account for the differences in physiological correlates of these types of aggression in our data.

Some of the reasons for the different findings for this study relative to other research reports are that the population is quite disparate from populations in other studies that have focused on physiological correlates of adjustment. The population in the current study consisted of adolescent females living in an urban, high-risk environment. The adolescents had many stressors in their lives with the majority of them having 3 or more risk factors for negative outcomes. The average participant reported witnessing 11 violent events in the past year. These events included muggings, shootings, knifings, drug deals, and home break-ins. The environment of the participants could make them physiologically less sensitive to these stressful situations,

including aggressive behavior, resulting in the lowered cortisol reactivity. Unlike previous studies, I used both parent and adolescent report on aggression, as well as subtypes of aggression. Additionally, the SCI (Ewart & Kolodner, 1991) is a task that focuses on social and environmental stressors, not performance-based stress like tasks frequently used in physiological research. As noted by several researchers (Dickerson & Kemeny, 2004; Stroud et al., in press), the type of cognitive and affective processing evoked by the task affects productivity of cortisol and sAA. Distinct intraindividual differences have been found between cortisol and sAA reactivity to a challenge (Granger et al., 2007). It is possible that the SCI did not influence change in the HPA axis in the same way it influenced the SNS. This is likely why overall increases in sAA but not cortisol to the task were observed.

This study contributes to the literature on cortisol and sAA by further examining the relationship between cortisol and aggressive behaviors in females. Previous research has been mixed on this relationship, and the results of this study support studies that found an association between higher reactivity and higher levels of aggression. The current study also explored the relationship between the two systems, which is a fairly new area of research. Unlike previous studies, symmetry in the systems was associated with lower levels of aggressive behavior. This indicates more research is needed on this interaction. Additionally, although no relationship was found, this study examined various forms of aggression as reported by different individuals.

This study had several limitations. The sensitive nature of some questions in the interview protocol that preceded the SCI may have resulted in cortisol and sAA being affected prior to the start of the stress task. However, as the authors of the SCI have argued, having the SCI at the start of the interview would not be as effective because the interviewer needs to build a rapport with the participant in order for the participant to fully disclose during the task. Another issue with the SCI has already been discussed and that is that the SCI may not be the best task to select when examining cortisol reactivity. A third limitation is that although puberty was controlled for, the participant's pubertal phase was not examined. Where an individual is in the pubertal process can affect cortisol levels and the diurnal cycle. However, a one-way ANOVA was used to examine if where a participant was in their menstrual cycle influenced cortisol. The results indicated that stage in menstrual cycle did not make a difference in cortisol. A final limitation is that the current study only looked at externalizing behaviors. It is possible that physiological patterns may differ in youth with both internalizing and externalizing symptoms.

Future studies should further examine the relationship between cortisol and sAA in females, but also compare patterns of interaction between cortisol and sAA across gender. More information is needed on the cause of the interaction and whether there are specific factors that are influencing the symmetry or asymmetry. Although the multisystem approach should be a primary focus, the mixed results on cortisol reactivity and aggression females warrant further exploration. Conclusions as to directions of reactivity, interactions, and the association between cortisol and sAA and aggression are

key to intervention and prevention programs. Some research has indicated that changes in the diurnal production of cortisol can provide information on the effectiveness of programs (Dozier et al., 2006). However, if it is uncertain what those patterns and associations are, it is impossible to monitor.

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Appendix A. Consent and Assent Forms.

Virginia Commonwealth University
Project COPE (#3768)
Parent Consent for Participation

Dear Parent,

This letter is to ask permission for you and your child to take part in a research study designed to learn more about what things best help students cope with stress. This study is being conducted by Virginia Commonwealth University. The funding is provided by the National Institutes of Health in Washington, D.C. A total of 400 families – half with children in the fifth grade and half with children in the eighth grade – are being asked to participate. You are being asked to participate because you live in the greater Richmond area and have a child in the 5th or 8th grade. You may have received a flyer from one of the community agencies or churches that serve the greater Richmond area.

What am I being asked to do?

If you agree to allow your family to participate, this is what will happen:

We will ask you and your child to complete four interviews over the next three years. The interviews with you and your child will be conducted separately to insure everyone's privacy. The first three interviews will be in your home, or if you prefer, at Virginia Commonwealth University. The last interview will be over the phone. The home interviews will take about 2 hours each; the phone interview will last about 30 minutes.

The interviews include a number of topics, such as

* things adolescents and families might find stressful, like personal or neighborhood violence (such as
seeing others harmed or killed), major life events such as moving, and everyday problems;
* how youth and families cope with stress, including things you and your child do that may work well and
things that don't work as well;
* the resources and strengths you have to cope with stress, including how your family relates to each other
and how you view your neighborhood;
* your child's behavior, including use of alcohol or drugs;

* ways you help your child cope with stress, and the reasons you use specific strategies to help your child;
* your child's physical reactions to stress. We will ask your child to give us 4 samples of saliva (spit) during
the interview. We will look in the saliva for the hormones which are made by the body during stress.
- your child's behavior, including use of alcohol or drugs

The National Institutes of Health, who is sponsoring the project, is very interested in why some youth turn to alcohol and drugs to cope with stress while other youth do not. We are trying to understand if there are ways that adolescents react to stress and cope with stress that make it easier or harder to turn to alcohol and drugs as a way to cope.

What are the potential risks and benefits of taking part in this research?

Some of the questions may make you or your child feel uncomfortable. You and your child can choose not to answer any question for any reason and can stop the interview at any time. If your child should become upset, a member of our staff will be glad to continue to talk to your child and address their concerns for as long as they would like. In addition, we can also provide a referral for your child if needed. Although we will assist in providing any referral that is needed, Virginia Commonwealth University or your health insurance may not provide compensation for these services. A potential benefit of this study is that by answering these questions, you and your child may help us learn how to help youth and families cope better with stress.

What will my family receive for participating?

We want to thank families who complete the interviews for the time and energy it took. So, at the end of the first interview in your home, you will receive a $45 gift certificate to Wal-Mart and your child will receive a $5 gift card. In some cases, your child will already have received this gift card for returning the consent form. After the second and third interviews in your home, you will receive $50 in gift certificates to Wal-Mart. When you complete the phone interview, you will receive a $30 gift certificate to Wal-Mart. Families who complete all 4 interviews will be entered into a drawing for $300, $200, and $100 prizes. Families in the study who stay in touch with us each month will be entered into a monthly drawing for a $25 gift certificate. One $25 gift certificate will be given away each month of the project.

If your child has given you this consent form to review, he or she will receive a $5 gift certificate if you review and return this consent form even if you decide that you do not want your family to participate.

What about privacy and confidentiality?

All of the information that you and your child provide will be kept private. Nothing that either of you tell us will be shared with anyone. But, if your child tells us that someone is hurting her or him, or that he or she might hurt himself/herself or someone else, the law says that we have to let people in authority know so they can protect your child. Even if this should happen, we would attempt to talk with you and tell you exactly what our concerns are regarding your child's safety. You will not see your child's information and your child will not see your information. All information you and your child provide will be coded with an identification number (ID number). Your name or your child's name and your ID number will not be kept together with any of the information you and your child provide. We tape record 10 -15 minutes of the interview with your child to help us keep track of the answers better. The tapes are kept in a locked cabinet at the VCU project office. Once we have written down the answers, names are

changed on the forms, and the tapes are erased. VCU or the sponsor of this project may review research records and the consent form signed by you.

When results of the research are published or discussed, no information will be included that will reveal your child's or your identity.

To help us protect your privacy, we have obtained a Certificate of Confidentiality from the National Institutes of Health. With this Certificate, the researchers cannot be forced to disclose information that may identify you, even by a court subpoena, in any federal, state, or local civil, criminal, administrative, legislative, or other proceedings. The researchers will use the Certificate to resist any demands for information that would identify you, except as explained below.

The Certificate cannot be used to resist a demand for information from personnel of the United States Government that is used for auditing or evaluation of Federally funded projects or for information that must be disclosed in order to meet the requirements of the federal Food and Drug Administration (FDA).

You should understand that a Certificate of Confidentiality does not prevent you or a member of your family from voluntarily releasing information about yourself or your involvement in this research.

The Certificate of Confidentiality does not prevent the researchers from disclosing voluntarily, without your consent, information that would identify you as a participant in the research project if your child tells us that that someone is hurting her or him, or that he or she might hurt himself/herself or someone else.

Voluntary participation and withdrawal

You and your child can choose whether to be in this study or not. Your participation is voluntary. If you volunteer to be in the study, you or your child may withdraw at any time with no consequences of any kind. You and your child may also refuse to answer any question and still remain in the study.

Who should I contact if I have questions?

If you have a question at any time, call Dr. Wendy Kliewer or the study staff at Virginia Commonwealth University at (804) 828-8793.

You may also feel free to contact the Office for Research Subjects Protection at the address and phone number below:

Virginia Commonwealth University
Bio-Tech Park, Building One
800 East Leigh Street, Suite 114
P.O. Box 980568
Richmond, VA 23219-0568

Telephone: (804) 828-0868

Consent

Signing your name below shows that you agree to be in the study. If there is any part of the form that is unclear to you, be sure to ask questions about it. Do not sign the form until you get answers to all of your questions.

I have read this consent form and understand the information about the study. All my questions about the study and my participation in it have been answered.

Federal law requires both parents to sign this consent form, unless the other parent is deceased, unknown, incompetent, not available, or does not have legal custody.

Please sign and print names below

_____
Printed name of student

_____ _____
Parent 1/ Signature of parent/legal guardian Date

_____
Printed name of Parent 1

_____ _____
Witness signature Date

Please check this box if there is no other parent/legal guardian in the home ☐

_____ _____
Parent 2/ Signature of parent/legal guardian Date

_____
Printed name of Parent 2

_____ _____
Witness signature Date

_____ _____
Signature of researcher verifying parental signature requirements (if needed) Date

_____ _____

Principal Investigator Signature Date

Virginia Commonwealth University
Project COPE (#3768)
Student Assent for Participation

We are asking you to be in a research study to help us learn more about what things best help students cope with stress. Stress can include things like experiencing or witnessing violence in the community, or dealing with everyday hassles in life, like having enough time to get everything done, or problems at school or in your neighborhood. This study is being done by Virginia Commonwealth University (VCU). About 400 students from the greater Richmond area and their mothers are being asked to participate.

Here is what we will do if you decide to participate:

- We will ask you and your mother to complete four interviews over the next three years. The first three interviews will be at your home, or at VCU if your family prefers. The interviews will be done separately to insure your privacy. Each in-person interview will take about two hours. The fourth interview will be a 30 minute phone interview.
- For the interviews we do in your home, we will ask you questions, and write your answers in a private booklet. During the interview we will ask you about things that you have done and things that have happened to you. These include questions about violence (such as seeing people being harmed or killed), your thoughts, feelings, and behavior, and drug and alcohol use. We will also ask questions about your family, friends, school, and neighborhood.
- We will ask you to talk about something that is stressful for you. We will tape record this part of the interview, because we won't be writing down what we say. Later, project staff will listen to the tape and type up what was said. Only your family number will be on the tape, not your name.
- We will also ask you to give us 6 samples of your saliva (spit). You will chew on a cotton swab for about 1 minute then spit the swab into a tube. This tells us how much of a stress hormone called *cortisol* and Alpha Amylase your body makes.

All of the information that you provide will be kept private. We won't share anything that you tell us with your parents, teachers, or anyone else. The only time we will share information about you is if you tell us that you are in danger or may harm others.

We want to thank families who do the interviews for the time and energy it took. So, at the end of the first interview in your home, your family will receive a $45 gift certificate to Wal-Mart. You will receive a $5 gift card at that time if you have not already received one. After the second and third interviews in your home, your family will receive $50 gift certificates to Wal-Mart. After you finish the phone interview, your

family will receive a $30 gift certificate. Also, at the end of the study, names of families who finish all four interviews will be put in a drawing for $300, $200, and $100 prizes. Families who stay in touch with us each month will be entered into a monthly drawing for a $25 gift certificate. One $25 gift certificate will be given away each month of the project.

It is possible that some of the interview questions may make you feel uncomfortable. You can choose not to answer any question for any reason and you can stop the interview at any time.

Although we cannot promise that you and your family will benefit from being in the study, by being in the study, you may teach us how to help other students cope better with stress.

To help us protect your privacy, we have asked the government for a Certificate of Confidentiality. Because we have this Certificate, we cannot be forced to tell others information about you that may identify you, even if a court subpoena is used. Of course, having this Certificate does not mean that you or your parent cannot share information about yourselves and your involvement in this research study. As noted above, having the Certificate does not prevent us from telling others if you are in danger or may harm others.

Being in this study is totally up to you and your parents. Nothing will happen if you or your parents decide you don't want to be in the study. If you decide to be in the study you can drop out at any time for any reason.

You can ask questions about the study now or later. If you have a question at any time, you can call Dr. Wendy Kliewer or the study staff at Virginia Commonwealth University at (804) 828-8793. They will be very happy to talk to you.

If you have been given this form and consent form to show to your parents, please return this form and the form for your parents to let us know whether you do or do not agree to be in this study. If your parent reviews and returns this consent form you will receive a $5 gift certificate even if your parent decides that they do not want your family to participate. We are giving you two copies of this form. One is for you to keep and the other is for you to return.

Signing your name below shows that you agree to be in the study. If there is any part of the form that is unclear to you, be sure to ask questions about it. Do not sign the form until you get answers to all of your questions. Remember, being in this study is up to you and your parents.

I agree to be in the study

_____ _____
Signature of student Date

_____
Printed name of student

_

_____

_____

Signature of person conducting assent discussion Date

_____ _____
Principal Investigator signature Date

Appendix B. Saliva Collection Procedure.

Assessment of cortisol in Project COPE

The youth in Project COPE will provide six samples of saliva, which will be assayed (tested) for cortisol. (The saliva will not be tested for other substances.) You will be given 6 salivettes (tubes) for the saliva collection, as well as a zip-lock bag to put the samples in, and a lunch bag with an ice pack to store the samples.

When will the samples be taken?

We will take 6 samples of saliva from youth during the interview process:

1) SAMPLE 1 - The first baseline sample – at the start of the interview
2) SAMPLE 2 - The second baseline sample – immediately before we conduct the Social Competence Interview (SCI)(e.g., before the tape recorder is turned on)
3) SAMPLE 3 - The first post-task sample – taken right after the end of the HOT phase of the SCI
4) SAMPLE 4 - The second post-task sample - taken 10 min after the first post-task sample/Sample 3
5) SAMPLE 5 – The third post-task sample – taken 10 minutes after Sample 4
6) SAMPLE 6 – Taken 20 minutes after Sample 5

How are the samples taken?

Youth chew on the cotton swab (which comes with the salivette) for about 1 minute. They need to get the cotton really wet. Sometimes it helps to have the child pretend to chew before you give them the cotton. The child then spits the cotton into the salivette tube. You (the interviewer) seal the tube and write the following on the label: ID#, time, and SAMPLE #. Once the sample has been collected, put the tube into the ziplock freezer bag. The ziplock bag will be labeled with the ID# and date (month/day/year). The zip-lock bag will be placed into the lunch bag with the icepack to keep the samples cold. IT IS ESSENTIAL THAT THE SAMPLES BE KEPT COLD.

Eating and drinking affect cortisol

The youth should not be consuming anything that has caffeine during the interview. After sample #1 is taken, the youth may drink water and eat a snack as long as the snack does not have caffeine. Nothing but water should be consumed between Sample #2 (right before the SCI) and Sample #6 (45-50 min).

Storage of cortisol samples

Saliva samples are kept in a freezer at -70 degrees Centigrade or colder until they are taken to our General Clinical Research Center (GCRC) laboratory at MCV for analysis. We have a freezer in the Project COPE research office at the Center for the Promotion of Positive Youth Development. It is critical that samples be kept cold (or if

kept overnight, frozen) until delivered to the Project COPE office at the Center.

Appendix C. Child Behavior Checklist- Aggression Subscale.

Now I am going to read you a list of items that describe children. For each item, think about whether this describes *(child)* **within the past 3 months**. Choose number 2 if the item is very true or often true of *(child)*, choose number 1 if the item is somewhat or sometimes true of *(child)*, and choose 0 if this is not true of *(child)* as far as you know. You can just tell me the number if you want.

	[0]	[1]	[2]
In the past three months. . .	Not True (as far as you know)	Somewhat or Sometimes True	Very True or Often True
1. Argues a lot.	0	1	2
2. Bragging, boasting.	0	1	2
3. Cruelty, bullying, or meanness to others.	0	1	2
4. Demands a lot of attention.	0	1	2
5. Destroys his/her own things.	0	1	2
6. Destroys things belonging to his/her family or others.	0	1	2
7. Disobedient at home.	0	1	2
8. Disobedient at school.	0	1	2
9. Easily jealous.	0	1	2
10. Gets in many fights.	0	1	2
11. Physically attacks people.	0	1	2
12. Screams a lot.	0	1	2
13. Showing off or clowning.	0	1	2
14. Stubborn, sullen, or irritable.	0	1	2

15. Sudden changes in mood or feelings.	0	1	2
16. Talks too much.	0	1	2
17. Teases a lot.	0	1	2
18. Temper tantrums or hot temper.	0	1	2
19. Threatens people.	0	1	2
20. Unusually loud.	0	1	2

Appendix D. Problem Behavior Frequency Scale.

We are interested in how often students your age do different kinds of things. Think about how often **YOU** have done the following things **IN THE LAST 30 DAYS**. Circle the number choice for your answer to each question. Remember, your answers are private and will not be shared with anyone.

Physical Aggression

1. Thrown something at someone to hurt them
2. Been in a fight in which someone was hit
3. Threatened to hurt a teacher
4. Shoved or pushed another kid
5. Threatened someone with a weapon (gun, knife, club, etc.)
6. Hit or slapped another kid
7. Threatened to hit or physically harm another kid

Relational Aggression

1. Not let another student be in your group anymore because you were mad at them
2. Told another kid you wouldn't like them unless they did what you wanted them to do
3. Tried to keep others from liking another kid by saying mean things about him/her
4. Spread a false rumor about someone
5. Left another kid out on purpose when it was time to do an activity
6. Said things about another student to make other students laugh

Response Options:
0= Never
1= 1-2 times
2= 3-5 times
3= 6-9 times
4= 10-19 times
5= 20 or more times

Appendix E. Pubertal Development Scale.

The next questions are about some of the physical changes your body may or may not be going through. Please be honest in your responses.

Answer the next questions **ONLY IF YOU ARE A GIRL**.

	[1]	[2]	[3]	[4]
Girls:	**Has Not Yet Started**	**Has Barely Started**	**Is Definitely Underway**	**Growth or Development Is Complete**
6. Have you developed body hair under your arms or down below?	**1**	**2**	**3**	**4**
7. Have your breasts started to develop?	**1**	**2**	**3**	**4**
8. Has your skin become oily, greasy, pimply, etc.?	**1**	**2**	**3**	**4**
9. Have you grown much taller very fast?	**1**	**2**	**3**	**4**

10. Have you started to menstruate (started your period)? [1] Yes / [2] No [circle one]

10a. IF YES, have you had at least 3 periods in a row? [1] Yes / [2] No [circle one]

10b. IF you have regular monthly cycles, where are you currently on your monthly cycle?

1. I'm on my period now
2. I'm in the week after my period
3. I am mid-way through my cycle
4. I am in the week before my period

Vita

Ashley Engels Dibble was born on October 16, 1979, in Lynchburg, Virginia. She received her Bachelor of Arts degree in Psychology from the University of Virginia in 2001, and her Master of Science in Criminal Justice from Virginia Commonwealth University in 2005. She has worked on several research projects covering topics such as probation and parole, violent crimes, aggressive behavior, violence exposure in youth, and substance abuse.