

**Social Information Processing, Cortisol Secretion, and Aggression in Adolescents**

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(ABSTRACT)

While both social information processing and cortisol secretion in childhood aggression have generated a great deal of interest and research in the past few decades, these social-cognitive and physiological components of aggressive behavior have not been examined in the context of an integrative model. This lack of an integrative framework may underlie some of the inconsistencies that have plagued the literature in this area to date, especially with respect to hypothalamic-pituitary-adrenal (HPA) axis functioning in aggressive children. This investigation tested a mediational model of the relationship between social-information processing, cortisol secretion, and reactive and proactive aggression. Specifically, it was hypothesized that social-information processing variables would mediate the proposed relationship between reactive and proactive aggression and cortisol secretion. One hundred and twenty-six children between the ages of 13 and 18 were administered the Child Behavior Rating Form (CBR), the Home Interview with Child (HIC), the Response Decision and Social Goals Instrument (RDSGI), the Antisocial Processes Screening Device (APSD), the Buss-Durkee Hostility Inventory (BDHI), the Children's Depression Inventory (CDI), and the Revised Children's Manifest Anxiety Scale (RCMAS). Each child also contributed two samples of saliva for cortisol assay, and each child's teacher completed a teacher-version of the APSD and the CBR. Regression analyses revealed no significant associations between proactive or reactive aggression and cortisol secretion, or between any of the social-information processing variables and cortisol secretion. Predicted associations between proactive and reactive aggression and social-information processing variables were found. Overall, therefore, the mediational model

was not supported. However, cortisol secretion was found to be associated with both anxiety and depression, and exploratory analyses revealed significant associations between cortisol secretion and Psychopathy as measured by the APSD. Taken together, the findings suggest that while the specific relationship proposed here among social-cognitive, psychophysiological, and behavioral variables was not found, an integrative model examining each of these components may be useful in further investigations of the complex phenomenon of childhood aggression.

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## Introduction

The study of childhood aggression has become increasingly refined over the past two decades, and substantial research has examined both the cognitive and psychophysiological variables associated with childhood aggressive behavior. With respect to information-processing, Dodge and his colleagues have conducted extensive studies that have identified several areas where aggressive children demonstrate clear deficiencies or distortions (Crick & Dodge, 1994; Dodge, 1980; Dodge, Laird, Lochman, & Zelli, 2002; Dodge, Murphy, & Buchsbaum, 1984; Dodge & Newman, 1981; Dodge & Somberg, 1987; Dodge & Tomlin, 1987; Fontaine, Burks, & Dodge, 2002; Nasby, Hayden, & DePaulo, 1980; Quiggle, Garber, Panak, & Dodge, 1992; Schwartz et al., 1997; Steinberg & Dodge, 1983). At the same time, investigators examining the relationship between psychophysiological functioning and aggression have found that aggressive children do, indeed, seem to exhibit unique patterns of psychophysiological functioning (McBurnett, Lahey, Rathouz, & Loeber, 2000; McBurnett, Pfiffner, Capasso, Lahey, & Loeber, 1997; Pajer, Gardner, Rubin, Perel, & Neal, 2001; Scarpa, 1997).

Unfortunately, the literature on psychophysiological functioning in aggressive children is fraught with inconsistencies. Though evidence strongly suggests a link between hypothalamic-pituitary-adrenal (HPA) axis functioning and aggressive behavior in children, the exact nature of that link has been elusive. For example, some aggressive children have been found to exhibit high levels of cortisol secretion or reactivity (McBurnett et al., 1991; Scarpa, 1997), while others have demonstrated lower than average cortisol secretion (McBurnett, Lahey, Capasso, & Loeber, 1996; Pajer et al., 2001). The purpose of this investigation, therefore, was to attempt to resolve some of these discrepancies in the literature by proposing a model that takes into account social-information processing in examining HPA axis functioning aggressive children.

To introduce the model, it is of primary importance to distinguish between two types of childhood aggression, proactive and reactive, each of which appears to be associated with unique forms of social-information processing deficits. Once this distinction is made, it is possible to suggest that the particular processing patterns associated with each form of aggression may lead to either perceptions of threat or feelings of control over the environment. Since substantial research has suggested that feelings of threat and uncontrollability can be powerful activators of the HPA axis (Stansbury & Gunnar, 1994), it may be hypothesized that particular ways of processing social cues may result in not only aggressive behavior, but may also be associated with particular patterns of HPA axis functioning in aggressive children.

## Literature Review

### Proactive and Reactive Aggression

Based initially on ethological studies of aggression in animals, theorists have begun to acknowledge a distinction between two forms of human aggression, reactive aggression and proactive aggression. In his seminal piece, “The structure and function of reactive and proactive aggression,” Dodge (1991) outlined the theoretical bases for the constructs of childhood proactive and reactive aggression. He noted that two explanations for aggressive behavior have been fundamental to the literature. The first, often associated with J. Dollard and his colleagues at Yale University (Dollard, Doob, Miller, Mowrer, & Sears, 1939), suggests that aggression stems from frustration, and that in aggressing individuals are attempting to defend themselves, or to harm the agents of frustration. Berkowitz summarized this position by writing that “a barrier to expected goal attainment generates an instigation to emotional [reactive] aggression – an inclination to hurt someone primarily for the sake of inflicting injury” (Berkowitz, 1993, p. 32). On the other hand, social learning theorists like Bandura (1973) posit that aggression is the result

of learning and social reinforcement, and that people aggress because they have observed or experienced that such behavior leads to positive consequences.

Dodge maintains that both of these explanations are correct, but that each leads to a unique form of aggression. Thus, reactive aggression occurs within the context of anger, is volatile and impulsive, and is intended as defensive or as a retaliation for perceived harm. This form of aggression is sometimes characterized as “hot-blooded.” Proactive aggression, however, is used as a means of obtaining something of value, such as an object or status. This type of aggression is usually more premeditated, and is executed in a cooler, less volatile fashion. Proactive aggression thus has a more appetitive function, and may be identified as “cold-blooded” (Dodge, 1991).

Based on this distinction, Dodge had developed predictions as to the developmental, social, and cognitive variables associated with each form of aggression. He suggests that reactive-aggressive children are likely to have histories of physical or sexual abuse and to be rejected by peers, and that they will have a tendency to attribute hostile intentions to others. Children who engage primarily in proactive aggression, on the other hand, are likely to come from environments characterized by lack of structure or guidance, and are likely to have adult role models who have demonstrated that aggression is an appropriate and effective method for achieving goals. While proactive-aggressive children may be feared by peers and thus not universally liked by them, they may also possess leadership and social skills that earn them status (Dodge, 1991).

Empirical research has found that proactive and reactive aggressive behaviors are often highly correlated in children. However, several investigators have found the distinction to be reliable, and to correlate with a variety of social and environmental variables. For example,

using confirmatory factor analysis of Dodge and Coie's (1987) teacher rating scale of aggression, Poulin and Boivin (2000) found that a two-factor model of proactive and reactive aggression better fit the data obtained from a sample of 149 4<sup>th</sup> through 6<sup>th</sup> grade boys than did a one-factor model. Further, each factor contributed uniquely to predictions of peer reports of children's aggression. In a sample of 193 third through sixth grade boys, these researchers also found that when they statistically controlled for proactive aggression, reactive aggression correlated positively with negative peer status and peer victimization, and negatively with leadership. When reactive aggression was statistically controlled, proactive aggression correlated positively with peer status and leadership, and negatively with peer victimization.

With respect to developmental history, a history of physical abuse and low peer approval has been found to distinguish reactive-aggressive children from both proactive-aggressive children and non-aggressive children in a community sample. Neither of these factors, however, distinguished between proactive-aggressive and non-aggressive children (Dodge, Lochman, Harnish, Bates, & Pettit, 1997).

Researchers have also found that reactive aggression is associated with increased problems with attention and impulsivity, whereas proactive aggression is not (Dodge et al., 1997). Further, children who tend to exhibit hostile aggression (i.e., reactive aggression) are more likely than control children to meet diagnostic criteria for ADHD, whereas children exhibiting instrumental aggression (i.e., proactive aggression) are not (Atkins & Stoff, 1993). Finally, in both community (Francisco, Van Voorhees, Bowser, Travers, & Scarpa, 2002) and psychiatrically-impaired samples (Dodge et al., 1997) of children, reactive aggression has been associated with higher rates of psychological disturbances such as depression, anxiety, somatization, and personality disorders than proactive aggression.

The distinction between these two types of aggression may have predictive value as well. Vitaro, Gendreau, Tremblay, and Oligny (1998) found that at age 12, proactive, but not reactive, aggressive behaviors predicted delinquency and disruptive behavior in mid-adolescence. Finally, in one of the few studies of proactive and reactive aggression in adult murderers, Raine and colleagues (1998) found differences in glucose metabolism using positron emission tomography between offenders exhibiting extremes of these two types of aggression. Specifically, affective (reactive) murderers exhibited lower left and right prefrontal functioning, and higher subcortical (amygdala, midbrain, hippocampus, and thalamus) functioning, than did comparison subjects. Predatory (proactive) murderers, on the other hand, exhibited increased subcortical functioning, but relatively normal prefrontal functioning.

While the above research is relevant in that it provides empirical support for distinguishing between proactive and reactive aggression, more central to this investigation are the findings that suggest that these two forms of aggression can be distinguished by specific social-information processing deficits as well. Crick and Dodge (1994) have proposed a model of social-information processing in children that follows discretely identifiable stages of processing, but allows for information to be processed in parallel through feedback loops at every stage. Though it is not accurate to view the stages as progressing in a fixed, linear sequence, a great deal of heuristic value is provided by the ability to examine each component of the process in depth.

The first stage of Crick and Dodge's (1994) social-information processing model involves Encoding of Cues, both internal and external. At this stage, biological limitations and personal history can affect which cues are attended to for encoding, and a predisposition or context of high emotional arousal can create physiological cues that must be interpreted. The

second stage is Interpretation of Cues, and includes causal attributions, intent attributions, and self- and other-evaluations. High emotional arousal or distress may create a tendency towards a more hostile interpretation of the cues, suggesting that children with a history of depression or anxiety could be more likely to demonstrate disturbances at this stage. The third stage is Clarification of Goals. As defined by Crick and Dodge (1994), “goals are focused arousal states that function as orientations toward producing (or wanting to produce) particular outcomes” (p. 76). Therefore, the clarification of goals is largely focused on “arousal regulation,” and at this stage the child’s processing will involve both pre-existing orientations as well as a response to the immediate social context. Stage four is Response Access and Construction, and involves the child’s ability to retrieve potential responses from long-term memory. Stage 5 is Response Decision, and involves evaluation of responses accessed from long-term memory, outcome expectations, and evaluations of personal efficacy for enacting these responses. Finally, Stage 6 is Behavioral Enactment.

Research has found that aggressive children in general exhibit deficiencies in each of the first five steps (Dodge, 1980; Dodge, Laird, Lochman, & Zelli, 2002; Dodge, Murphy, & Buchsbaum, 1984; Dodge & Newman, 1981; Dodge & Somberg, 1987; Dodge & Tomlin, 1987; Fontaine, Burks, & Dodge, 2002; Nasby, Hayden, & DePaulo, 1980; Quiggle, Garber, Panak, & Dodge, 1992; Schwartz et al., 1998), but that children who tend to demonstrate reactive versus proactive aggression exhibit problems at different points in the process (Crick & Dodge, 1996; Dodge & Coie, 1987; Dodge et al., 1997). Specifically, reactive aggression has been associated with an increased tendency to demonstrate errors in encoding relevant social cues (Stage 1) (Dodge et al., 1997) and to attribute hostile intentions to peers in ambiguous situations (Stage 2) (Crick & Dodge, 1996). Conversely, proactive aggression has been associated with an increased

likelihood of valuing instrumental over social goals in interpersonal interactions (Stage 3), of evaluating verbal and physical aggression positively, and of expecting more positive interpersonal consequences from aggressive behavior (both reflecting Stage 5). Increased likelihood of accessing or identifying aggressive responses to social problems (Stage 4) has not yet been associated specifically with reactive or proactive aggression, but has been linked with increased aggressive behavior in general (Crick & Dodge, 1996).

It may also be possible to draw parallels between proactive aggression and the construct of psychopathy as it applies to children. Children who score high on the Psychopathy Screening Device (Frick, Bodin, & Barry, 2000) demonstrate increased callousness and decreased empathy, increased narcissism, and increased antisocial behavior. The lack of emotionality described in high psychopathic children may be similar to the “cold-blooded” nature of proactive aggression, which is more instrumental than emotional. Fisher and Blair (1998) have hypothesized that these children may have an impairment in their ability to respond to distress cues from others that would normally serve to inhibit aggressive behavior. Blair has presented evidence to suggest that there may be dysfunctions within the amygdala that contribute to “muting” of psychopathic children’s responsiveness to distress cues in others (Blair, 2001). If Blair’s hypotheses are true, children scoring high on measures of psychopathy would be expected to evaluate aggression more positively and to expect more positive interpersonal consequences from aggressive behavior than normal children. Further, the lack of empathy that may stem from an inability to interpret distress in others could lead to a decreased interest in feelings of connectedness, therefore increasing the value of instrumental over social goals.

In sum, it appears that there may be some utility to the distinction between proactive and reactive aggression in examining aggressive behavior patterns of children, and that, further, there

may be identifiable neurophysiological correlates of this distinction in addition to the developmental, social, and information-processing differences (Scarpa & Raine, 1997; Scarpa & Raine, 2000). While it is possible that other physiological differences could be identified through a variety of measures, including heart rate, heart rate variability, electrodermal activity, and brain imaging, the model presented here focused specifically on the final product of the hypothalamic-pituitary-adrenal (HPA) axis, cortisol, one of the major stress response systems of the body. Cortisol has been studied in antisocial and aggressive behavior in children, with mixed results. However, it is possible that the discrepant findings regarding cortisol and aggression to date are due to the lack of distinction between these two forms of aggression, and a lack of consideration of the information-processing differences between them.

#### Emotional and Behavioral Correlates of Cortisol Secretion

Cortisol is the final product of the hypothalamic-pituitary-adrenal (HPA axis). This steroid hormone serves to terminate the stress response through feedback at various levels of the system, and its catabolic action allows for the release of energy needed by the body to respond to stressors (Sapolsky, 1998). Upon initial activation of the system, cortisol has been found to stimulate cortical arousal, increase energy, and improve the ability to concentrate. The delayed effects seem to involve a reversal of these actions, such that exposure to cortisol over the span of a few days contributes to decreased energy, a decreased ability to concentrate, and depressed mood. Researchers have suggested that these dual effects of cortisol may provide for an optimal response to the stressor at the time of exposure, and then may allow for the withdrawal required to recover from the acute effects (Stansbury & Gunnar, 1994).

Though the HPA axis can be activated by physical stressors such as surgery, it can be activated by emotional stressors as well. The most potent of these emotional stressors appear to

be those involving novelty, negative emotional valence, and feelings of uncontrollability (Stansbury & Gunnar, 1994). For example, neonates exposed to the noxious stimuli of repeated heel sticks showed an increase in cortisol secretion over time, whereas those exposed to the more benign stimuli of repeated physical exams showed the opposite pattern (Gunnar, Hertzgaard, Larson, & Rigatuso, 1992). Kagan has found that behaviorally inhibited children tend to exhibit greater cortisol secretion than bold children (Kagan, Reznick, & Snidman, 1987). Relevantly, however, the combination of behavioral inhibition and a lack of a feeling of security may actually be required for the HPA stress response to take place. In a study of 18-month-old toddlers and their mothers, Nachmais and colleagues found that only behaviorally inhibited toddlers in insecure attachment relationships exhibited elevations in cortisol in response to a series of novel events. Neither behaviorally inhibited toddlers in secure attachment relationships nor behaviorally uninhibited toddlers displayed such cortisol reactivity. These researchers suggested that a secure attachment relationship may reflect a history of sensitive caregiving, thus providing the child with the resources to cope with novel environmental stimuli. Therefore, the vulnerability of an inhibited temperament may only be a liability if the child is not provided with the caregiver support that allows him/her to feel safe in the face of potentially threatening stimuli (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996).

Just as the combination of novelty, uncertainty, and negative emotionality appears to precipitate increases in cortisol secretion, pleasant or soothing experiences, even if they are novel, have been found to reduce cortisol levels. For example, when 6- to 13-month-old infants participated in a mother-baby swim class, their cortisol levels were found to lower to below baseline (Hertzgaard, Gunnar, Larson, Brodersen, & Lehman, 1992). Taken together, these findings seem to suggest that pleasant experiences, when paired with the perception of control

over the environment, may serve to reduce cortisol secretion, whereas perceptions of threat, uncontrollability, and uncertainty may increase cortisol secretion.

#### Cortisol secretion and aggression.

Several studies have examined the relationship between cortisol secretion and antisocial behavior in children. Unfortunately, many of the studies have operationalized the definition of antisocial behavior differently, producing results that are somewhat difficult to interpret. For example, Kruesi and colleagues examined 24-hour urinary free cortisol output in 19 males with ADHD, conduct disorder, and/or oppositional defiant disorder and found no relationship between cortisol and behavior (Kruesi et al., 1989). Scerbo and Kolko (1994) pointed out that hormone levels may be related more to specific behaviors or symptoms than to diagnoses. Therefore, these researchers examined staff, parent, and teacher ratings of aggression, inattention/overactivity, and internalizing behavior in a sample of 40 children at a treatment program for children with disruptive behavior disorders. They found that cortisol levels correlated positively with parent-rated internalizing behavior and depression, and negatively with staff-rated inattention/overactivity and oppositional behavior. However, they did not find significant correlations between cortisol and aggression.

Other research has found relationships between aggression and cortisol, however. Pajer and colleagues (2001) found that cortisol levels were significantly lower in a sample of 47 adolescent girls with conduct disorder than in the comparison sample of 37 normal control girls, and that those girls with conduct disorder without comorbid diagnoses had the lowest cortisol levels of all (Pajer et al., 2001). Van Goozen et al (2000) found that children with either Oppositional Defiant or Conduct Disorder showed a decline in cortisol levels after a provocation task, whereas normal controls showed an increase in cortisol in the same situation.

This research suggests that there is some association between cortisol and antisocial behavior, but that the relationship may be more complex than could be detected by earlier studies. Two possible moderators of the association have been recently examined: the presence of anxiety, and the age of onset of aggressive behavior. With respect to anxiety, two studies have found that anxiety moderates the relationship between conduct disturbance and cortisol levels in boys. McBurnett et al (1991) found no difference in cortisol levels between 8 to 13 year old boys with anxiety disorders and no conduct disorder. However, these researchers did find that, among boys with conduct disorder, those with anxiety had significantly higher levels of cortisol. Interestingly, conduct disordered boys with anxiety had significantly fewer contacts with the police and lower peer ratings of aggression than did conduct disordered boys without anxiety. Similarly, van Goozen et al (1998) found that 8 to 11 year old boys with Oppositional-Defiant Disorder had lower cortisol levels overall than normal controls, but that anxiety played an important role in individual differences in cortisol secretion in response to stress. Specifically, those boys who were both high in externalizing behavior and high in anxiety exhibited the greatest cortisol response to stress, while boys who were high in externalizing behavior and low in anxiety exhibited the lowest cortisol response to stress.

With respect to age of onset, two studies by McBurnett and colleagues suggest that antisocial behavior characterized by early onset and overt aggression may be reflected in lower cortisol levels. In the first study, McBurnett, Lahey, Capasso, & Loeber (1996) found that cortisol levels in 7 to 12 year old boys were significantly negatively related to aggressive conduct disorder symptoms (i.e., initiating physical fights, using weapons, forcing sex onto someone), but not to covert conduct disorder symptoms (i.e., setting fires, destroying property, breaking into homes). Additionally, the pattern of results suggested that the children who began

to act aggressively early, and who maintained this pattern throughout their childhood years, were likely to have the lowest cortisol levels. In a later study, these researchers sampled cortisol on years two and four of a four-year annual psychodiagnostic evaluation, and found that aggressive conduct disorder symptoms were again negatively correlated with cortisol levels. Though the two cortisol samples were only correlated 0.20, indicating significant within-subject variability over time, it was observed that those boys who exhibited cortisol levels below the median at both time points had significantly higher levels of aggressive and covert conduct disorder symptoms, peer aggression nominations, and oppositional-defiant symptoms. These researchers replicated their earlier findings that early emergence of aggression was directly associated with lower cortisol levels (McBurnett, et al., 2000).

Though the specific dysregulation of the HPA axis leading to reduced cortisol in aggressive children is unclear, recent research has suggested that there is, indeed, a link. Interestingly, a link has also been found between the presence of antisocial personality disorder with a history of conduct disorder in fathers and low cortisol levels in their sons (Vanyukov et al., 1993). While low cortisol levels in one study were associated only with aggression in clinic-referred boys, parent-child relations was associated with both aggressive and covert conduct disorder symptomatology. These findings led researchers to tentatively conclude that low biological arousal and deviant parenting may both uniquely contribute to persistent aggression (McBurnett et al 1997). Scarpa (1997) examined the relationship between biological predisposition and non-optimal rearing environments from a similar perspective. In her study of 7 to 15 year old children, she found that children who were both physically abused and exhibited high levels of internalizing behavior were most likely to behave aggressively. She also found that those children who showed increased cortisol secretion in response to a provocation task had

higher rates of aggression than those who did not show such a response, and that these “cortisol reactors” were less likely than the “cortisol nonreactors” to show improvements over the course of a 7-week treatment program. Scarpa suggested that those children who were able to regulate their emotions most appropriately, as reflected by lack of internalizing behavior and lack of cortisol reaction to provocation, may be less vulnerable to the effects of their adverse family environments and more able to benefit from treatment. Thus, it may be that both excessive cortisol reactivity, as observed in children with internalizing disorders, and deficient cortisol secretion, as found in children with early-onset aggression without comorbid internalizing disorders, may both be vulnerabilities to aggression when combined with deviant parenting practices. Again, discrepancies in the literature may be clarified by distinguishing the types of aggression exhibited.

### Synthesis and Hypotheses

Despite definitional problems and inconsistent findings, the literature does provide a foundation for tentative conclusions and testable hypotheses. First, there is evidence for the existence of two distinct forms of aggression, proactive and reactive, each of which has unique and identifiable developmental, social, and information processing correlates. Second, the research on aggression and cortisol secretion suggests that there is a relationship between these two variables, but that the relationship may be moderated by other factors such as age of onset of aggression or the presence of comorbid psychopathology such as anxiety. The findings of McBurnett and colleagues (1991) and van Goozen and colleagues (1998), for example, suggest that anxiety may moderate the relationship between cortisol and aggression, such that children with conduct disorder and high levels of anxiety exhibit the highest levels of cortisol secretion. These findings are unique in the literature on the relationship between cortisol and aggression,

where it has most typically been found that aggressive children exhibit low levels of cortisol secretion.

Another explanation of these findings, however, can be posited when Dodge's theory is used as a framework for interpreting the childhood aggression and cortisol literature, and when the nature of cortisol responsivity is taken into account. That is, childhood reactive aggression, characterized by a hostile attribution bias and a defensive interpersonal stance, is likely to be associated with high levels of cortisol secretion in response to chronic perceptions of threat in the environment. That highly reactive-aggressive children would also be most likely to exhibit anxiety is supported by the findings that reactive children do tend to have higher rates of comorbid emotional disorders, including anxiety and depression (Francisco et al., 2002). Conversely, childhood proactive aggression is not likely to be associated with exaggerated feelings of threat, and should therefore not be associated with elevated cortisol secretion. In fact, since proactive aggression has been associated with positive outcome expectations for the use of aggression, this form of aggression may actually be associated with greater feelings of social control, and therefore with lower levels of cortisol secretion. Such feelings of control may also be augmented by the preference for instrumental over interpersonal gain, since it is more likely that aggressive behavior will lead to instrumental, rather than interpersonal, goals.

Based on the above research, the following hypotheses were developed:

1) a) Proactive aggression will be related to disruptions to stages 3, 4, and 5 of social information processing. That is, proactive aggression will be related to a preference for achieving instrumental over interpersonal goals in aggressive situations (stage 3), to an increased likelihood of constructing aggressive strategies to address social problems (stage 4), and to increased self-efficacy for aggressive behavior (stage 5). b) Proactive aggression will be

associated with decreased levels of cortisol secretion. Disruptions to stages 3, 4, and 5 of social information processing will be associated with lower levels of cortisol secretion.

2) a) Reactive aggression will be related to a greater tendency to demonstrate hostile attribution bias, reflecting a disturbance in stage 2 of social information processing. b) Reactive aggression will be associated with increased levels of cortisol secretion. Disruptions to stage 2 of social information processing will be associated with increased levels of cortisol secretion.

3) These information processing biases will explain the cortisol differences in reactive and proactive aggression.

### Method

Reactive and proactive aggression are both measured on a continuum, and they have been found to be highly correlated, though distinct, constructs. Unfortunately, much of the research to date has utilized statistical techniques such as ANOVA that require an artificial split of the data, with children categorized as high or low proactive-aggressive children, or high or low reactive-aggressive children. While this may be convenient for creating categories consistent with the widely used medical model upon which the DSM is based, it artificially dichotomizes constructs for which there is little evidence for such discrete categories. Because both proactive and reactive aggression can occur in the same child, assigning the child to one group based on a cutoff score disregards valuable information about the behavior of the child overall. Thus, for example, a therapist deciding upon an intervention targeting the child's aggressive behavior may treat a child labeled as "proactive" with a treatment based on social learning theory, whereas the child may also show reactive characteristics that would benefit from an intervention targeting hostile attribution biases as well. Based on this line of thinking, regression analyses of

continuous measures were chosen to analyze the data for all primary and post-hoc analyses.

Partial correlations were used for all exploratory analyses.

A preliminary power analysis (Buchner, Erdfelder, & Faul, 1997) indicated that in order to detect a medium effect size for the regression analysis at  $\alpha = .05$  with a power level of 0.8, a sample of 80 participants was necessary. Twenty-five pilot (8 boys, 17 girls, ages 14 to 18, mean age = 16.6, SD = 1.0) and one-hundred and one experimental participants (45 girls, 56 boys; ages 13-16, mean age = 14.1, SD = 0.71) were recruited from students in grades 8 through 12. All participants were students in a local public school or in a local school for behaviorally disturbed students. A letter and a consent form were mailed to the parents or legal guardian of each of the children requesting permission for the child to participate in this project. Once the consents from the parents were returned, consents from the teachers were obtained. Teachers were asked to complete the Teacher Rating Form (Brown, Atkins, Osborne, & Milnamow, 1996) and the Psychopathy Screening Device (Frick, Bodin, & Barry, 2000) for each of the participating students.

Assent (if under the age of 18) and consent (if 18 or older) was obtained from the students. For their participation the students were entered into a raffle where they were eligible to win a first prize of \$50, a second prize of \$25, or a third prize of \$15.

### Measures

Child Behavior Rating Form (CBR) (Brown et al., 1996). This measure is a 28-item scale designed to provide continuous measures of proactive and reactive aggression in schoolchildren as reported by teachers. Teachers rate proactive aggression (i.e., has hurt others to win a game or contest) and reactive aggression (gets mad when corrected) on a scale of 1 to 3, where 1 = never, 2 = sometimes, and 3 = very often. Proactive scores could fall along a range from 10 to 30, and

reactive scores could fall along a range of 6 to 18. Though proactive and reactive aggression have been found to be highly correlated (i.e.,  $r = .67$ ; Brown et al., 1996), each factor has been found to have discriminant validity as well (Poulin and Boivin, 2000). In a sample of predominately white lower middle class boys, each factor was found to have high internal consistency ( $\alpha = .94$  for Proactive Aggression and  $.92$  for Reactive Aggression) (Brown et al., 1996).

In this sample, the distributions for both proactive and reactive aggression on the teacher report form of the CBR demonstrated a very strong right skew. For proactive aggression, the range of scores was from 10 to 25, with a mean of 11.1 and a standard deviation of 2.36. Sixty-six percent of the proactive aggression scores as rated by the teachers fell at the minimum, with a score of 10. For reactive aggression the range of scores was from 6 to 16, with a mean of 6.9 and a standard deviation of 1.85. Sixty-eight percent of the reactive aggression scores fell at the minimum, with a score of 6. A principal component factor analysis with a promax rotation was conducted using the proactive and reactive criterion items of teacher report form of the CBR, forcing a two-factor solution. It was found that 48.3% of the variance was explained by the first factor, and 7.6% of the variance was explained by the second factor. Four items predicted to be reactive and four items predicted to be proactive loaded on the first factor, while four items predicted to be proactive and no items predicted to be reactive loaded on the second factor. Therefore, it appears that the CBR-teacher form did not perform well in distinguishing reactive from proactive aggression in this sample. Proactive and reactive aggression were found to be highly correlated on the CBR-teacher form,  $r(123) = 0.836$ ,  $p < .01$ . The reliability for the proactive factor was  $\alpha = 0.88$ , and the reliability for the reactive factor was  $\alpha = 0.87$ .

While the teacher report form is the version of the CBR to have been validated thus far in the literature, a child version has also been developed. As an alternative measure of proactive and reactive aggression, this child version was administered to each participant. Both the reactive and proactive factors for the CBR-child form fell along a roughly Gaussian distribution. For proactive aggression, the scores fell along a range from 10 to 23, with a mean of 14.1 and a standard deviation of 2.95. Only 10% of the scores fell at the minimum score of 10. Similarly, for reactive aggression the scores fell along a range from 6 to 15, with a mean of 9.3 and a standard deviation of 2.14, and with 7.4% of the scores at the minimum score of 6. It appears that the child version of the CBR was capable of a finer distinction of both reactive and proactive aggression along the entire range of scores in this sample than was the teacher version. As with the teacher version, a principal component factor analysis with a promax rotation was conducted using the proactive and reactive criterion items of the child report form of the CBR, forcing a two-factor solution. The first factor of the child form explained 26.1% of the variance, and the second factor explained 9.1% of the variance. Six of the items predicted to reflect proactive aggression and one of the items predicted to reflect reactive aggression loaded on the first factor. Three of the items which loaded on a reactive factor in a previous factor analysis (Brown et al., 1996) loaded on the second factor. None of the items predicted to reflect proactive aggression loaded on the second factor. On the CBR-child form proactive and reactive aggression were significantly positively correlated,  $r(117) = 0.572$ ,  $p < .01$ . The reliability for the proactive factor was  $\alpha = 0.76$ , and the reliability for the reactive factor was  $\alpha = 0.63$ . It appears that for this sample the child form of the CBR was more effective at making theoretically relevant distinctions between reactive and proactive aggression than was the teacher form; therefore the CBR-child form as developed by Brown et al., 1996, was used as the primary measure of

reactive and proactive aggression in all analyses. See Table 1 for descriptive statistics of proactive and reactive aggression.

Demographics Questionnaire. This measure consisted of demographic information such as the participant's age, gender, race, approximate height and weight, and select medical information (medications, recent ingestion of licit or illicit drugs, the possibility of pregnancy, use of birth control, use of tobacco, and presence or absence of illness), as well as whether the participant had consumed any food or caffeine in the hour prior to cortisol sampling. This information was necessary to account for possible spurious influences on cortisol.

Home Interview With Child. This instrument is a measure adapted from an earlier instrument developed by Dodge (1980) for use in assessing intent attributions in proactive and reactive aggressive children (Dodge, Laird, Lochman, & Zelli, 2002). Though it was originally designed for use with younger children, minor adaptations in language were made to make it appropriate for adolescents. The measures consisted of eight story vignettes, each describing a situation where the intent of the agent is ambiguous. The stories were designed to present four problematic group entry situations (i.e., not being allowed to join a basketball game) and 4 ambiguous peer provocation situations (i.e., being cut in front of in a line for lunch).

To index hostile attribution bias, a measure of Stage 2 of Crick and Dodge's (1994) social-information processing model, each participant was asked to respond as to why he/she thought the agent engaged in the behavior described in each vignette. The participant's response was recorded verbatim by the interviewer, and was immediately scored as "accidental," i.e., belief that the agent acted without hostile intent (score = 0), or "hostile," i.e., belief that the agent acted with hostile intent (score = 1), using the criteria outlined in the Scoring Manual for the Child Interview (Brown & Lemerise, 1990). If the interviewer was unable to determine whether

the response was accidental or hostile from the initial answer, follow-up questions were asked to elicit enough information for a score to be assigned. The responses were summed across all eight situations, creating a Hostile Attribution Bias (HAB) score with a range of 0 to 8. The HAB scores for this sample fell along a Gaussian distribution, with a reliability of  $\alpha = 0.60$ .

After responding to the question regarding the intent of the provocateur, the respondent was then asked what his/her likely response would be to the situation. Responses were recorded verbatim, and then immediately coded as irrelevant or unable to provide a further response (“I don’t know”), passive (“I wouldn’t do anything”), competent, (“I would ask her why she did that,” or, “I would ask her to help me clean off my shirt”), authority-punish (“I would ask the coach to suspend her from the game”), and aggressive (“I would swear at her,” or “I would smack her”). The total number of aggressive and authority-punish responses provided were summed across all eight situations, creating a Aggressive Problem Solving (APS) score with a range of 0 to 8 (Dodge et al., 2002). The APS scores for this sample fell along a range of 0 to 5, with a significant right skew (71.4% of the scores were 0). The reliability coefficient for this measure was  $\alpha = 0.65$ .

Response Decision and Social Goal Instrument (Things That Happen to Me). This instrument was adapted from a scale developed by Crick and Dodge (1996) to assess Outcome Expectations (Stage 5) and Social Goals (Stage 3) for aggression. Again, though it was originally designed for use with younger children, the appropriate adaptations in language were made to make it appropriate for an adolescent population. Four situations were presented to each participant, two of which reflected peer provocation and two of which reflected peer group entry situations. To obtain the Outcome Expectation score (OE), the participant were asked to evaluate whether two aggressive and two assertive strategies for each of the situations would be

effective in obtaining instrumental (i.e., getting to play basketball) and social (i.e., making a positive impression on peers) goals. Each item score was obtained by subtracting the number of positive evaluations of assertive responses from the number of positive evaluations of aggressive responses. The total Outcome Expectation score was calculated by summing the four item scores. The total scores could therefore range from -8 to +8, with higher scores signifying more positive outcome expectations for aggression. For this sample the range of scores was -7 to +2, with a Gaussian distribution along this range. In previous samples, Cronbach's alpha for the outcome expectation scale has been found to be .65 for both the conflict and peer group entry situations (Crick & Dodge, 1996).

The participant's Social Goals (SG) were assessed by asking which he/she would prefer to achieve in the four vignettes: the positive relational outcome, or the positive instrumental outcome (Crick & Dodge, 1996; Dodge et al., 2002). Participants' responses to the four situations were summed, with total scores ranging from 0 (greater relational goals) to 4 (greater instrumental goals). The range of scores for this sample was 0 to 4, and the distribution demonstrated a right skew, with 40.5% of the participants obtaining a SG score of 0. The reliability of this scale for this sample was  $\alpha = 0.68$ .

Finally, the ease with which the participants believed they could enact an aggressive response, or Response Efficacy, was determined by asking for a rating on a scale of 1 (very hard) to 4 (very easy) for each of the aggressive response options in the four vignettes (Brown & Lemerise, 1990). The Response Efficacy (RE) score, another component of Stage 5 of the social-information processing model, was summed across the vignettes. The possible range of scores was 4 to 16. The scores fell along a Gaussian distribution across the entire range of possibilities. The reliability of this scale for this sample was  $\alpha = 0.79$ . See Table 2 for a

summary description of each of the social information processing (SIP) variables, and Table 3 for descriptive statistics of the SIP variables.

Cortisol samples. Salivary cortisol samples were collected from each participant. Cortisol secretion follows a diurnal rhythm, which peaks at the time of awakening in the morning and levels out between 4 and 12 hours after awakening (Stansbury & Gunnar, 1994; Salivadiagnostics website, 2002). Thus, to keep timing consistent all testing was conducted between 9:00 am and 11:00 am. Since food intake can also affect salivary cortisol concentrations, participants were asked not to eat for one hour prior to cortisol sample collection.

Because cortisol is known to be secreted in response to situations of novelty, uncontrollability, and negative emotional valence, each experimental participant was asked to submit a “novelty” saliva sample by soaking a cotton swab with saliva for approximately one to two minutes. This initial sample was taken to control for the effects of the novelty of the procedure on the secretion of cortisol. After a five minute rest period during which the students wrote their names on index cards for entry into the raffle, a second “test” saliva sample was collected using the same procedure. Both samples were immediately placed in a cooler until they could be returned to the lab at Virginia Tech, where they were frozen until they were assayed.

The 25 pilot participants contributed only one saliva sample. However, the first (novelty) and second (test) samples of the participants for which both were collected were found to be highly correlated,  $r(96) = .86, p < .01$ , and a paired-sample t-test revealed no significant difference between the first and second samples ( $t(95) = -1.597, p = .114$ ; sample 1 mean = 1.617, SD = .189; sample 2 mean = 1.603, SD = .180). Therefore, the first (novelty) samples contributed from both the pilot and experimental participants were used for all further analyses.

Cortisol samples were assayed by the principle investigator at the endocrinology laboratory of the Dairy Science Department at Virginia Tech, under the guidance and supervision of Frank Gwazdauskas, Ph.D. Cortisol saliva levels were determined from a 90- $\mu$ L sample, which was assayed in duplicate using the DiaSorin GammaCoat radioimmunoassay for cortisol (DiaSorin Inc., Stillwater, MN). Participant duplicates exceeding a 15% CV were re-assayed. The intra-assay and inter-assay coefficient of variation (CV) means were 5.97% and 8.26%, respectively. These CV are within the ranges typically reported in the literature (Goenjian et al., 1996; Goodyer et al., 1991 & 2000; Kaufman et al., 1997; Stone et al., 2001; Young & Nolen-Hoeksema, 2001). See Table 4 for descriptive statistics of the cortisol variables.

#### Secondary/Alternative Measures of Central Constructs

##### Antisocial Processes Screening Device (APSD) (Frick, Bodin, & Barry, 2000).

Based on research into the application of the construct of psychopathy to children, Frick and colleagues have identified a subset of antisocial children who appear to demonstrate a particularly severe form of aggressive behavior. Much like their adult counterparts, children with high levels of psychopathic traits tend to engage in aggressive behavior that is more premeditated and instrumental than do their aggressive, non-psychopathic peers (Frick, Cornell, Barry, Bodin, & Dane, 2003). More specifically, antisocial children that are high on the callous-unemotional (CU) factor of psychopathy have been demonstrated to show increased thrill-seeking behavior, to be less reactive to emotional words and to threatening stimuli, to show less empathic concern towards others, to be more reward-focused and less sensitive to cues of punishment in reward-primed situation, and to expect more instrumental gain from their aggressive behavior than other antisocial youth (Frick, 2000). Thus, it was expected that in this

sample psychopathy generally, and CU traits specifically, would be significantly positively correlated with proactive but not with reactive aggression.

The APSD, a measure developed to assess for the presence of psychopathic traits in children, was used to provide an alternative measure to the CBR for instrumentally aggressive behaviors. The APSD is a modified form of a measure widely used to assess psychopathic characteristics in adults, the PCL-R (Hare, 1991). This measure has been found to have three factors: Callous-Unemotional traits, Impulsivity, and Narcissism, which have demonstrated correlations of factor loadings of items across two independent samples of 81, .68, and .90, respectively (Frick, Bodin, & Barry, 2000).

The APSD is a 20-item rating scale, and both teacher-report and student-report versions were administered. The reliability coefficients of the student-report and teacher-report versions of the scale for the current sample were  $\alpha = 0.65$ , and  $\alpha = 0.74$ , respectively. The teacher and child scores were significantly correlated,  $r(116) = 0.242$ ,  $p < .01$ . See Table 5 for descriptive statistics for the variables derived from the APSD.

Irritability Subscale of the Buss-Durkee Hostility Inventory (BDHI) (Buss & Durkee, 1957). This instrument was used to provide an alternative measure of hostile or reactive aggression. The 12-item Irritability scale is a subscale of the 66-item, true/false, self-report Buss-Durkee Hostility Inventory (BDHI), which was designed for use with individuals age 14 to adult. The Irritability scale was designed to tap the tendency to react with negative affect at minimal provocation (Buss & Durkee, 1957). Research suggests that this scale has good convergent validity, and also that high-scorers on this scale are less likely to engage in socially constructive behavior in role-play situations than low-scorers (Biaggio, Supplee, & Curtis,

1981). The alpha coefficient for this sample was  $\alpha = 0.67$ . See Table 6 for descriptive statistics for the Irritability scale of the BDHI.

### Supplementary Measures

Previous research has demonstrated the reliability and validity of the distinction between reactive and proactive aggression along developmental, social, and cognitive variables (Atkins & Stoff, 1993; Dodge et al., 1997; Vitaro et al., 1998), and recently some evidence has suggested that these two forms of aggression differ in terms of the presence of comorbid psychopathology as well (Dodge et al., 1997; Francisco et al., 2002). Supplementary measures of anxiety and depression were included with the battery of questionnaires administered to participants to determine if these findings with regard to comorbid psychopathology and cortisol secretion could be replicated on this sample of adolescents.

Children's Depression Inventory (CDI) (Kovacs, 1985). This measure is a 27-item self-report inventory for use with children ages 7 to 17, in which the child is asked to choose one of three sentences best describing his/her state. The measures assess affective, cognitive, and behavioral symptoms of depression. Several studies of internal consistency have reported alpha coefficients between .85 and .87, and one month test-retest reliability coefficients between .65 and .85 (Silverman & Rabian, 1999). The alpha coefficient for this sample was 0.88.

Revised Children's Manifest Anxiety Scale (RCMAS) (Reynolds & Richmond, 1985). This measure is a 37-item self-report measure for use with school-age (ages 5 to 19) children. The measure yields three factor scale scores: Physiological Anxiety, Worry/Oversensitivity, and Social Concerns/Concentration, as well as a Lie scale score. Internal consistency studies have yielded alpha coefficients around .82, and a 9 month test-retest reliability coefficient of .68 has been found for the total scale score (Silverman & Rabian, 1999). The alpha coefficient for this

sample was 0.86. See Table 7 for descriptive statistics for the anxiety and depression variables in this sample.

### Procedures

Consent was obtained from teachers and parents prior to beginning data collection. On the days of data collection, the investigators arrived at the school between 8:00 am and 10:00 am. The study was briefly explained to the participants by the principle investigator, and written assent was obtained. Between 9:00 am and 11:00 am each participant was asked to contribute a saliva sample by soaking a cotton swab in his/her mouth and depositing the swab into a sterile tube marked with a participant number followed by the letter "A". This first sample was taken to diminish the "novelty" effect of donating saliva. The participant was then asked to rest for 5 minutes, while writing his/her name and contact information on an index card for entry into a raffle. Finally, each participant contributed a second saliva sample, depositing it in a sterile tube marked only with his/her participant number.

Numbered folders corresponding to participant numbers and containing the following self-report questionnaires were distributed: the CBR-Student Version, the APSD-Student Version, the BDHI, the CDI, and the RCMAS. After collection of the second saliva sample, participants were provided with juice and donuts, and were asked to begin completing the questionnaires. The principle investigator remained in the room during this time to monitor the questionnaire completion and to answer any questions.

As the participants were completing the questionnaires, trained undergraduate research assistants selected students individually to undergo the interview portion of the investigation. Each participant was taken by the assistant to a separate classroom and was administered the Demographics Questionnaire, the Intent Attribution Instrument, and the Response Decision and

Social Goals Instrument. After completion of the interview, the participants finished the questionnaires if they had not already done so.

After signing the informed consent, teachers were asked to complete the CBR-Teacher Version and the APSD-Teacher Version. The teacher questionnaires were marked with each participant's name and participant number. After completion of the questionnaires, the teachers were instructed to tear each participant's name from the page so that only the participant number could be associated with the responses. Each teacher was compensated \$25 for participating in the project.

After the data collection was complete, all item responses to the questionnaires were entered into a spreadsheet by trained undergraduate research assistants. Each data point entered was subsequently verified by a second research assistant. Errors in data entry were identified and corrected either by the principle investigator or by a research assistant. The interview portion of the data was scored by hand by a trained research assistant, according to the protocol used by Dodge and colleagues (Brown & Lemerise, 1990). The hand-scored protocols were then re-scored by the principle investigator. Discrepancies between the research assistants' and the principle investigator's scorings of participant responses were identified. The principle investigator discussed these discrepancies with Jennifer Lansford, a graduate research assistant of Kenneth Dodge who has worked extensively with the interview measure. Each response was re-scored as necessary. The final scored responses were entered into the database by the principle investigator.

## Results

The three hypotheses outlined above describe a mediating model of the relationship among the variables tested. This model suggests that proactive and reactive aggression, social-

information processing, and cortisol secretion are related such that the social-information processing variables will account for the relationship between proactive and reactive aggression and cortisol secretion (see Figure 1). To test this model, the techniques for testing mediating models described by Baron and Kenny (1986) were used. According to these authors,

A variable functions as a mediator when it meets the following conditions: (a) variations in levels of the independent variable significantly account for variations in the presumed mediator (i.e., Path *a*), (b), variations in the mediator significantly account for variations in the dependent variable (i.e., Path *b*), and (c) when Paths *a* and *b* are controlled, a previously significant relation between the independent and dependent variables is no longer significant, with the strongest demonstration of mediation occurring when Path *c* is zero (p. 1176).

Hypothesis one addressed the relationship between proactive aggression and social-information processing stages 3, 4, and 5, between proactive aggression and cortisol secretion, and between social information processing stages 3, 4, and 5 and cortisol secretion. This hypothesis was designed to test conditions a and b for the mediating model involving proactive aggression. Similarly, hypothesis two addressed the relationship between reactive aggression and social-information processing stage 2, between reactive aggression and cortisol secretion, and between social information processing stage 2 and cortisol secretion. Thus, hypothesis two tested conditions a and b for the mediating model involving reactive aggression. Finally, hypothesis three addressed the mediating effects of social-information processing on the relationship between reactive and proactive aggression and cortisol secretion, and thus tested condition c for both the reactive and proactive aggressive models (see Figures 2 and 3). The results of each of the three hypotheses is presented separately below.

Because age and gender were found to be significantly associated with many of the variables of interest, both were controlled in all of following analyses. See Table 8 for association of age and gender with proactive and reactive aggression, social information processing, and cortisol secretion.

### Hypothesis I

Hypothesis one predicted that proactive aggression would be related to a greater anticipation of interpersonal and instrumental gain from aggressive behavior, and to a preference for achieving instrumental over interpersonal goals in interpersonal situations. Further, hypothesis one predicted that both proactive aggression and these social information processing biases would be negatively correlated with cortisol secretion (see Table 2).

The proactive score from the child-report version of the Child Behavior Rating form was used to measure proactive aggression in these analyses. To measure anticipation of interpersonal and instrumental gain from aggressive behavior, the Outcome Expectation (OE) and the Response Efficacy (RE) scores from the Response Decision and Social Goal Instrument, and the Aggressive Problem Solving Score (APS) from the Home Interview With Child, were used. As measured by Response Decision and Social Goal Instrument, The OE score reflected the degree to which the respondent expected aggressive strategies to be effective in obtaining instrumental and interpersonal goals, and the RE score reflected the ease with which the respondent believed he/she could enact the aggressive response to the social situations presented. The APS score as measured by the Home Interview with Child reflected the degree to which the respondent spontaneously offered an aggressive response to a social problem. To measure preference for instrumental versus social goals in interpersonal situations, the Social Goals (SG) score from the Response Decision and Social Goal Instrument was used, with an increased SG score reflecting

an increased preference for instrumental over interpersonal goals in the situations presented (see Table 2).

To address the relationship between proactive aggression and the social information processing variables, reactive aggression, age, and gender were entered into the first block, and proactive aggression was entered into to second block of a separate hierarchical regression equation for each social information processing variable. Aggressive Problem Solving (APS) was found to be significantly predicted by proactive aggression ( $\beta = .264$ ,  $R^2\Delta = .045$ ,  $p < .05$ ). Response Efficacy (RE) was found to be significantly predicted by proactive aggression ( $\beta = .314$ ,  $R^2\Delta = .063$ ,  $p < .01$ ). Neither Social Goals (SG) ( $\beta = 0.153$ ,  $R^2\Delta = .015$ ,  $p = .176$ ) nor Outcome Expectation (OE) ( $\beta = -.116$ ,  $R^2\Delta = .009$ ,  $p = .318$ ) were found to be predicted by proactive aggression. As expected, Hostile Attribution Bias (HAB) was also not found to be predicted by proactive aggression ( $\beta = .094$ ,  $R^2\Delta = .006$ ,  $p = .415$ ) (see Table 9).

As has been commonly done in the literature (Goodyer et al., 2000; Kaufman, 1991; Pajer et al., 2001), a log 10 transformation was performed on the cortisol data to normalize the distribution. In examining the association of cortisol secretion with the potential confounding variables, it was found that pregnancy/use of birth control was significantly correlated with cortisol secretion ( $r(71) = .253$ ,  $p < .05$ ) in the sample of girls. Therefore, this variable was entered with age, gender, and reactive aggression in the first step of the hierarchical regression model. Proactive aggression was not found to be associated with cortisol secretion ( $\beta = .121$ ,  $R^2\Delta = .009$ ,  $p = .267$ ). Further, none of the social information processing variables were associated with cortisol secretion (see Tables 10 and 11).

## Hypothesis II

Hypothesis two predicted that reactive aggression would be related to an increased tendency to exhibit a hostile attribution bias. Hypothesis two also predicted that reactive aggression and hostile attribution bias would each be positively correlated with cortisol secretion (see Table 2).

The reactive score from the child-report version of the Child Behavior Rating form was used to measure reactive aggression in these analyses. To measure hostile attribution bias, the Hostile Attribution Bias (HAB) score from the Home Interview With Child instrument was used, with higher HAB scores reflecting increased hostile attribution bias.

To address the relationship between reactive aggression and hostile attribution bias, the Hostile Attribution Bias score (HAB) was modeled as a function of reactive aggression using a hierarchical regression equation controlling for proactive aggression, age, and gender. Reactive aggression was not found to be significantly related to any of the social information processing variables, including hostile attribution bias (HAB) ( $\beta = .102$ ,  $R^2\Delta = .007$ ,  $p = 0.384$ ) (see Table 12).

To address the relationship between reactive aggression and cortisol secretion, cortisol secretion was modeled as a function of reactive aggression controlling for proactive aggression, age, gender, and birth control/pregnancy using a hierarchical regression equation. No relationship between reactive aggression and cortisol secretion was found ( $\beta = -.001$ ,  $R^2\Delta < .001$ ,  $p = .995$ ). Again, no relationship between any of the social information processing variables and cortisol was found (see Tables 10 and 11).

### Hypothesis III

Hypothesis three was designed to assess a mediating model of the relationship between aggression and cortisol secretion, with social-information variables as putative mediators. Because no relationship was found between either proactive or reactive aggression and cortisol, the mediating model could not be tested.

### Post-hoc analyses

Post-hoc analyses: tests of associations with Hostile Attribution Bias (HAB) with peer entry items removed.

Though a relationship between Hostile Attribution Bias (HAB) and reactive aggression has been demonstrated in several studies (Crick & Dodge, 1996; Dodge & Coie, 1987; Dodge, Price, Bachorowski, & Newman, 1990), such an association was not replicated here. To explore why the data reported here are inconsistent with what has been found by previous researchers, the responses to the interview questions designed to tap HAB were examined. It was found that some of the responses provided by the adolescents were ambiguous, and that this was particularly the case for questions regarding peer group entry situations. Unfortunately, this ambiguity was not clarified during data collection, and therefore it was often difficult to identify responses as clearly reflecting an accidental or hostile intent attribution for these questions. For example, in one peer entry vignette the participants were asked why peers refused a request to sit at the lunch table. A common answer was, “they don’t know me.” After consultation with Jennifer Lansford, a graduate student of Kenneth Dodge, it was decided that this response could reasonably be interpreted as reflecting a hostile attribution bias, primarily because such an attribution could lead to more general expectations of peer rejection in group entry situations. All responses of “they don’t know me” were therefore coded as hostile.

However, an argument could be made for the decision to regard this response as reflecting non-hostile attribution as well (i.e., a participant might think, “since the people don’t know me and they are shy, this is unique to this situation and does not necessarily mean I will be rejected in other such situations”). As this measure was originally designed for younger children with less sophisticated attributional styles, the inconsistent findings in this data set may stem from the inability of the peer entry vignette and questions to adequately measure the HAB construct in adolescents, as well as from a lack of clarification on the part of the interviewers during data collection.

In an effort to clarify the results, a test of whether the association between HAB and reactive aggression would be found without these questionable items included in the scale was run. All of the peer group entry questions were removed, and analyses for the hypotheses involving HAB were run using the scores from the remaining four items only.

In hierarchical regression modeling the revised HAB variable as a function of reactive aggression controlling for proactive aggression, age, and gender, no significant relationship was found ( $\beta = .125$ ,  $R^2\Delta = .010$ ,  $p = 0.283$ ). Similarly, no significant relationship was found when HAB was modeled as a function of proactive aggression controlling for reactive aggression, age and gender ( $\beta = .094$ ,  $R^2\Delta = .006$ ,  $p = 0.412$ ).

Post-hoc analyses: Analysis of girls and boys separately.

Much of the research examining proactive and reactive aggression in children has been conducted on male samples, with some notable exceptions (i.e., Connor, Steingard, Anderson, & Melloni, 2003; Crick & Dodge, 1996). While combining gender for analyses in this data set increased the sample size and thus the power to detect important relationships, it also introduced the risk of obscuring associations that may be unique to one gender, and may explain the lack of

replication of previously reported findings in this data set. To determine if gender-specific associations were present in this sample, hierarchical regressions were run separately for both males and females, controlling for age. As with the previous analyses, all regressions involving proactive aggression controlled for reactive aggression, and vice versa.

For girls, proactive aggression was found to be significantly related only to Outcome Expectation ( $\beta = -.316$ ,  $R^2\Delta = .059$ ,  $p < .05$ ), though this association was opposite of the predicted direction (See Table 13). Reactive aggression was found to be significantly associated with Social Goals ( $\beta = .318$ ,  $R^2\Delta = .062$ ,  $p < .05$ ), again counter to hypotheses (see Table 14).

In boys, proactive aggression was significantly associated with Social Goals ( $\beta = .388$ ,  $R^2\Delta = .104$ ,  $p < .05$ ), Aggressive Problem Solving ( $\beta = .334$ ,  $R^2\Delta = .077$ ,  $p < .05$ ), and Response Efficacy ( $\beta = .368$ ,  $R^2\Delta = .093$ ,  $p < .05$ ) (see Table 13). Reactive aggression was not associated with any of the social information processing variables in boys (see Table 14). Thus, the hypotheses regarding reactive and proactive aggression and social information processing were largely supported for boys, but not for girls.

When regression analyses were run separately for each gender, cortisol secretion was not predicted by proactive or reactive aggression in either boys or girls. Similarly, regression analyses run separately for each gender did not reveal a significant association of cortisol secretion with any of the social information processing variables.

Post-hoc analyses: Comparison of extreme groups of proactive and reactive aggression.

Finally, in this sample regression analyses were chosen for data analysis to avoid the necessity of artificial cut-points in continuous data, and because there is insufficient evidence to suggest the presence of qualitatively distinct groups of proactive- and reactive-aggressive children. However, much of the research in the area of proactive and reactive aggression has

used extreme groups for comparison and analysis. To explore the possibility of significant differences among the extreme groups of reactive and proactive aggression, the 33.3<sup>rd</sup> and 66.7<sup>th</sup> percentile of reactive and of proactive aggression scorers on the child-report form of the CBR were used as high and low cut-points for extreme group analyses. Independent sample t-tests were used as high and low cut-points for extreme group analyses. Independent sample t-tests were conducted to compare the top and bottom scoring groups on proactive aggression along cortisol secretion and social information processing. Similarly, independent sample t-tests were conducted to compare the top and bottom scoring groups on reactive aggression along each of these variables.

With regard to cortisol secretion, no significant differences were found between high- and low-scorers on either reactive ( $t(98) = -1.304, p = .20$ ) or proactive aggression ( $t(101) = -1.640, p = .10$ ) (See Tables 15 and 16).

With regard to social information processing, the hypotheses would have predicted high- and low-scorers on proactive aggression to differ on later stages in the information processing sequence, including Aggressive Problem Solving (APS), Outcome Expectation (OE), Social Goals (SG), and Response Efficacy (RE). The earlier stage (HAB) would not necessarily be expected to demonstrate a difference on these variables reflective of the later stages of processing. On the other hand, high- and low-scorers on reactive aggression would be expected to differ on Hostile Attribution Bias (HAB), but not necessarily on the later stages.

With respect to proactive aggression, the results of the t-tests were similar to the results of the regression analyses. Just as Aggressive Problem Solving (APS) and Response Efficacy (RE) were associated with proactive aggression in the regression analyses, in the t-test analyses high- and low-scorers on proactive aggression differed significantly on APS ( $t(102) = -3.979, p < .01$ ) and RE ( $t(102) = -2.614, p = .01$ ). In addition, high- and low-scorers on proactive

aggression also differed significantly on Social Goals (SG) ( $t(102) = -2.090, p < .05$ ). However, similar in pattern to findings of the regression analyses, Outcome Expectation (OE) was not found to differ among extreme scorers on proactive aggression ( $t(102) = .429, p = .70$ ). As expected, HAB did not differ between high- and low-scorers on proactive aggression ( $t(101) = -.860, p = .39$ ), even with the questionable items removed from the HAB measure.

For reactive aggression, a significant difference was found in the predicted direction between high- and low-scorers on HAB ( $t(98) = -2.368, p < .05$ ). However, extreme scorers on reactive aggression also differed significantly on Response Efficacy (RE) ( $t(99) = -.1.964, p = .05$ ). Thus, while the hypotheses regarding reactive and proactive aggression and social information processing were partially supported by the extreme group analyses, there was also one unexpected finding of a significant difference on Response Efficacy between high and low scorers on reactive aggression.

### Exploratory analyses

Three sets of exploratory analyses were planned. The first set of analyses tested whether the secondary measures of aggression, psychopathy, and hostility could be empirically demonstrated to reflect similar constructs to proactive and reactive aggression. The second set of analyses explored the relationships between these putative secondary measures, the social information processing variables, and cortisol secretion. Finally, the third set of analyses was conducted in an effort to replicate previously observed relationships among proactive and reactive aggression and comorbid psychopathology, and to examine the relationship among anxiety, depression, and cortisol secretion in this sample.

Exploratory analyses: Proactive/reactive aggression, psychopathy, and hostility.

*Proactive and Reactive Aggression and Psychopathy.*

As an alternative measure of proactive aggression, the Antisocial Process Screening Device (ASPD; Frick, Bodin, & Barry, 2000), a measure of psychopathic traits in children, was administered to participants and teachers.

Partial correlations of proactive aggression controlling for reactive aggression revealed that child-rated proactive aggression was significantly correlated with child-rated psychopathy ( $r(111) = 0.445, p < 0.01$ ), and that teacher-rated proactive aggression was significantly correlated with teacher-rated psychopathy ( $r(119) = 0.335, p < .01$ ). Child-rated proactive aggression was not significantly associated with child-rated CU traits ( $r(111) = 0.023, p = 0.83$ ), and teacher-rated proactive aggression was not associated with teacher-rated CU traits ( $r(119) = .072, p = .43$ ). Similar results were found when partial correlations were run to examine the relationship of reactive aggression controlling for proactive aggression and these two variables. Specifically, child-rated reactive aggression was significantly correlated with child-rated psychopathy ( $r(111) = 0.290, p < 0.01$ ), and teacher rated reactive aggression was significantly correlated with teacher-rated psychopathy ( $r(119) = 0.392, p < 0.01$ ). Child-rated reactive aggression was not significantly associated with child-rated CU traits ( $r(111) = 0.020, p = 0.83$ ), and teacher-rated reactive aggression was not associated with teacher-rated CU traits ( $r(119) = 0.137, p = .13$ ). Finally, Impulsivity and Narcissism were significantly related to both reactive and proactive aggression on both child- and teacher-reports. Overall, these results do not seem to support a unique relationship between proactive aggression and psychopathy, nor between proactive aggression and the callous-unemotional cluster of psychopathic traits (see Tables 17a

and 17b). However, they do suggest that CU traits may tap a dimension of behavior distinct from both proactive and reactive aggression.

*Proactive and Reactive Aggression and Hostility.*

To provide a secondary measure of reactive aggression, the Irritability subscale of the Buss-Durkee Hostility Inventory (BDHI) was used. Partial correlations of child-rated reactive and proactive aggression (each controlling for the other) and the Irritability scale were run, with the expectation that reactive aggression, but not proactive aggression, would correlate positively with Irritability. This unique association was not found, in that both reactive aggression ( $r(113) = 0.289, p < .01$ ) and proactive aggression ( $r(113) = 0.235, p < .05$ ) were significantly positively correlated with the Irritability scale. Thus, it does not appear that the Irritability scale provided an adequate secondary measure of reactive aggression in this sample.

*Exploratory analyses: psychopathy, hostility, social-information processing, and cortisol secretion.*

To examine the relationship between psychopathy and social-information processing, correlations were run among Psychopathy and each of the social-information processing variables, as well as between Callous-Unemotional (CU) traits and each of the social-information processing variables. Because of the association of Psychopathy and Callous-Unemotional (CU) traits with decreased emotional reactivity, with a more reward-dominant style, with decreased sensitivity to punishment, and with greater expectation of social and instrumental gain from aggressive behavior (Frick, 2000), it was expected that Psychopathy in general, and CU traits specifically, would be associated with disruptions to the later steps in Dodge's model of social-information processing (Crick & Dodge, 1994). That is, it was hypothesized that both psychopathy and CU traits would be associated with aggressive biases in goal selection, response

construction, and behavioral enactment as reflected in the measures of Social Goals (SG), Aggressive Problem Solving (APS), Outcome Expectations (OE), and Response Efficacy (RE).

Disruptions to the earlier stages Dodge's model involved with encoding and interpretation of social cues have been associated with hostile attribution bias and emotional reactivity (Dodge & Coie, 1987). Because it has been reported that antisocial children without psychopathic traits are likely to exhibit high emotionality during aggression, whereas psychopathic children are not (Cornell et al., 1996), it was hypothesized that hostile attribution bias (HAB) would not be significantly associated with Psychopathy or with CU traits.

These hypotheses were largely supported by the data (See Table 18). Both teacher- and child-rated psychopathy and CU traits correlated significantly with Response Efficacy (RE). Child- and teacher-rated psychopathy, and child-rated CU traits correlated significantly with Aggressive Problem Solving (APS). Child-rated psychopathy and child-rated CU traits both correlated significantly with Social Goals (SG). Outcome Expectations (OE) did not correlate significantly with any of the psychopathy or CU trait measures. In sum, 3 of the 4 measures of social-information processing that had been predicted to correlate with psychopathy and CU traits did so on the child-rated measures, and 2 of the 4 also correlated with the teacher-rated measures. Thus, it appears in this sample that aggressive biases in goal selection, response construction, and behavioral enactment were associated with psychopathic and CU traits.

As predicted, Hostile Attribution Bias (HAB), an earlier step in the social-information processing model, was not correlated with Psychopathy or CU traits on either the teacher or child report forms. Thus, the hostile attribution bias, reflective of a distortion in the earlier steps of the social-information processing model, does not appear to be associated with psychopathic or CU traits in children.

With respect to cortisol secretion, partial correlations (controlling for age, gender, and pregnancy/use of birth control) were run between cortisol secretion and Psychopathy, as well as between cortisol secretion and CU Traits. It was predicted that Psychopathy and CU traits would be negatively associated with cortisol secretion, with the strongest negative association between CU traits and cortisol secretion. These hypotheses were partially supported for the teacher ratings only. Specifically, teacher-rated CU traits was significantly negatively associated with cortisol secretion ( $r(118) = -.210, p = .02$ ), but teacher-rated Psychopathy was not ( $r(117) = -.133, p = .15$ ). Child-rated CU traits were not significantly associated with cortisol secretion ( $r(116) = .080, p = .39$ ), while child-rated Psychopathy was significantly positively associated with cortisol secretion ( $r(114) = .187, p = .05$ ) (See Table 19).

Finally, based on the initial hypothesis of an association between reactive aggression and Irritability, it was predicted that cortisol secretion would be positively associated with Irritability. A partial correlation analysis was run between cortisol secretion and Irritability, again controlling for age, gender, and pregnancy/use of birth control. Irritability was not significantly associated with cortisol secretion ( $r(119) = .014, p = .88$ ). Correlation analyses of Irritability with each of the social information processing variables revealed that Irritability was associated with Social Goals ( $r(125) = .186, p < .05$ ), Aggressive Problem Solving ( $r(125) = .213, p < .05$ ), and Response Efficacy ( $r(125) = .222, p < .05$ ) (see Table 20).

#### Exploratory analyses: proactive and reactive aggression and psychopathology.

Regression analyses were run to examine the relationship between reactive and proactive aggression and anxiety and depression, with the hypothesis that reactive aggression would predict anxiety and depression, but that proactive aggression would not (Dodge et al., 1997; Francisco et al., 2002). This hypothesis was supported for both anxiety and depression.

With respect to anxiety, in the first regression analysis anxiety was entered as the criterion variable, proactive aggression was entered as the predictor in the first block, and reactive aggression was entered as the predictor in the second block. Reactive aggression significantly contributed to the model, after the effects of proactive aggression were taken in to account ( $\beta = 0.32$ ,  $R^2\Delta = .07$ ,  $p < .01$ ). When anxiety was entered as the criterion variable, reactive aggression was entered as the predictor variable in the first block, and proactive aggression was entered as the predictor in the second block, proactive aggression failed to contribute significantly to the model after the effects of reactive aggression were taken into account ( $\beta = .083$ ,  $R^2\Delta = .005$ ,  $p = .44$ ).

With respect to depression, in the first regression analysis depression was entered as the criterion variable, proactive aggression was entered as the predictor in the first block, and reactive aggression was entered as the predictor variable in the second block. Reactive aggression contributed significantly to the model, after the effects of proactive aggression were taken into account ( $\beta = .255$ ,  $R^2\Delta = .044$ ,  $p < .05$ ). When depression was entered as the criterion variable, reactive aggression was entered as the predictor variable in the first block, and proactive aggression was entered as the predictor variable in the second block, proactive aggression failed to contribute significantly to the model after the effects of reactive aggression were taken into account ( $\beta = .095$ ,  $R^2\Delta = .006$ ,  $p = .38$ ).

Finally, regression analyses were run to examine the relationship between anxiety, depression, and cortisol secretion, with the hypothesis that both anxiety and depression would predict cortisol secretion (Dodge et al., 1997; Francisco et al., 2002). Age, gender, and pregnancy/use of birth control were controlled by entry into the first step of the hierarchical regression analysis. Both hypotheses were supported. Specifically, anxiety predicted cortisol

secretion ( $\beta = 0.204$ ,  $R^2\Delta = .039$ ,  $p = .02$ ), and depression also predicted cortisol secretion ( $\beta = 0.190$ ,  $R^2\Delta = .033$ ,  $p = .03$ ).

## Discussion

### Review of proposed model and primary hypotheses

This study was designed to examine the relationship between reactive and proactive aggression, social information processing, and cortisol secretion in adolescents. Crick and Dodge's model of social information processing was used as a foundation for a model integrating Dodge's research on proactive and reactive aggression with current research on cortisol secretion in aggressive children.

In general, it was hypothesized that the inconsistencies in the literature on cortisol secretion in aggressive children could be explained if aggression were more clearly defined. The distinction between proactive and reactive aggression in humans has been supported by significant research, and there is some evidence that parallel psychobiological distinctions may be made in animals as well. Therefore, the distinction between proactive and reactive aggression was used as a starting point for exploring differences in cortisol secretion in aggression in children. To explain why behavioral differences in aggression would be reflected in biological differences in cortisol secretion, a social information processing model of aggressive behavior was introduced. Specifically, it was hypothesized that proactive and reactive aggression would be associated with unique styles of social information processing, and that these styles of information processing, in turn, would explain the differences in cortisol secretion that would be observed in proactive and reactive aggression. Thus, a mediational model of the relationship among reactive and proactive aggression, social information processing, and cortisol secretion

was proposed, such that social information processing would mediate the relationship between aggression and cortisol secretion.

### Review and explanation of primary findings

In general, a mediational model of the relationships among proactive and reactive aggression was not supported by the data because the proposed relationships between proactive and reactive aggression and cortisol secretion were not found, and because no relationships were found between cortisol secretion and any of the social information processing variables.

However, some of the previously reported associations between proactive and reactive aggression and social information processing in children were replicated in this sample of adolescents, lending support to the utility of a distinction between the constructs of proactive and reactive aggression. Further, significant differences in anxiety and depression were also found between proactive and reactive aggression in this sample. Finally, consistent with the literature, an association of anxiety and depression with cortisol secretion was observed, and exploratory analyses suggested that a negative association may exist between cortisol secretion and Callous-Unemotional Traits of Psychopathy. The significance of each of these findings will be discussed separately below.

### Association of aggression and social information processing.

#### Social information processing and proactive aggression.

The theoretical association of proactive aggression with disruptions to the social information processing stages of goal selection, response development, and response implementation was largely supported in this sample of adolescents, replicating findings in previous reports (Dodge & Coie, 1987; Dodge et al., 1997). The lack of an association of reactive aggression with these later stages of social information processing was also replicated.

Specifically, disruptions to stages 4 (response development) and 5 (response implementation) of social information processing (SIP) were significantly positively associated with proactive aggression but not with reactive aggression. In post-hoc t-tests comparing extreme high- and low-scorers on proactive aggression, however, participants with high levels of proactive aggression were found to exhibit a significantly higher preference for instrumental over social goals in interpersonal situations, reflective of a disruption to stage 3 of SIP as well.

The only measure of social information processing that was expected to correlate with proactive aggression and did not was Outcome Expectation (OE), a secondary measure of the response development stage. There are two possible explanations for this finding. First, it is possible that the way in which this variable was measured in adolescents was not adequate to capture the construct it was intended to measure. Second, it may be that OE is not as relevant a component of response development as is Aggressive Problem Solving (APS).

Overall, then, it appears that proactive aggression, but not reactive aggression, was associated in this adolescent sample with a tendency to value instrumental over social goals and to develop aggressive responses as options for achieving goals in social situations, and with an enhanced sense of self-efficacy for enacting aggressive responses. These findings are consistent with those reported by Dodge and Coie (1987) and by Crick and Dodge (1996). They also add support to the validity of the constructs of reactive and proactive aggression by providing replications of the original findings using a separate measure of proactive and reactive aggression.

The data reported here support the idea that proactive aggressive behaviors are developed and maintained through the expectation of rewards, rather than by frustration or emotional dysregulation. Because it is more likely for an aggressive strategy to result in instrumental gain

than in positive evaluation by the victim, the valuing of instrumental over social goals would increase the overall likelihood of aggression in a proactive-aggressive child. Over time, as the child is reinforced for aggressive behavior by obtaining the valued instrumental goal, the incidence of aggressive behavior would be expected to increase. On the other hand, reactive aggression is theorized to be associated with intense affect and emotion dysregulation, and may be enacted in a relatively out-of-control manner. Therefore, the consideration of instrumental goals would not be expected to play as strong of a part in the expression of reactive aggressive behavior as they would in proactive aggressive behavior.

With regard to response selection, the cooler consideration and instrumental goal orientation of proactive aggressive behavior would be consistent with a conscious selection of aggression as a means of controlling a social situation. Thus, as the child learns that an aggressive response is likely to result in the acquisition of the instrumental goal, the likelihood that such an aggressive response will be selected in the future is increased. And finally, each time the child successfully enacts the aggressive response and obtains the desired goal, the child's self-efficacy for enacting aggressive responses is enhanced.

#### Social information processing and reactive aggression.

The theoretical association of reactive aggression with a tendency to attribute hostile motivations to peers in ambiguous social situations was not supported in this sample by the regression analyses, even when the data were re-analyzed with the ambiguous per group entry questions removed.

However, post-hoc t-tests comparing extreme scorers on reactive aggression on Hostile Attribution Bias did produce significant differences in the expected direction. Though these findings from the post-hoc analyses must be interpreted with caution, they do suggest several

possibilities as to why an association between reactive aggression and hostile attribution bias was not replicated in this sample. First, previous consistent findings of hostile attribution biases in reactive aggression have been based on studies where video-taped vignettes were used (i.e., Crick & Dodge, 1996). It is possible that lack of visual stimuli in this study made it more difficult for the respondent to place him or herself into the scenario. Because reactive aggression is theoretically based on an affective, unplanned response to a social situation, this lack of realism in the stimuli may account for the ability to uncover the predicted association only in the extreme groups.

Another possibility is that because most of this data was collected from a sample of relatively well-adjusted adolescents at a high school summer camp, it is likely that reactive aggressive responses to social situations were supplanted by more socially appropriate and mature responses in the majority of these adolescents. Thus, only the most extreme members of this sample would be likely to demonstrate dysfunctional processes significant enough to be detected by the crude measure of the early stage of social-information processing used here. Finally, previous research has demonstrated a link between hostile attribution bias and a history of early harsh discipline (Weiss, Dodge, Bates, & Pettit, 1992). It is likely that the incidence of harsh discipline was lower in this sample than in the previously reported sample of boys in a juvenile correctional facility (Dodge et al., 1997). In sum, a sort of floor effect for hostile attribution bias in this sample may have decreased the likelihood of an association.

For the most part, a lack of association was found between reactive aggression and the later stages of social information processing, which is consistent with both the theoretical distinction between reactive and proactive aggression and with previous findings. The only exception to this was the finding of increased Response Efficacy in high scorers on reactive

aggression. Though this unexpected finding is difficult to explain, it is possible to suggest that a reactive child with a limited repertoire of social problem solving alternatives who has successfully used aggression in the past may be more likely to positively evaluate his/her likelihood of future success in implementing aggressive strategies.

Though the hypothesized social-information processing distinctions between reactive and proactive aggression were largely supported for the combined-gender group analyses, a very different picture emerged on the post-hoc analyses of girls and boys separately. Specifically, while the hypothesis of an association of deficits in social-information processing stages 3, 4, and 5 with proactive aggression was found in boys, such an association was not found in girls. In fact, in girls an association of Outcome Expectation (stage 5) with proactive aggression was found in the opposite of the predicted direction, and deficits in Social Goals (stage 3) was found to be associated with reactive aggression.

These findings are important for several reasons. First, Crick & Rose (2000) have suggested that, for girls, aggression is enacted more by attacks against social relationships than by physical attacks. They have termed this more feminine form aggression “relational aggression,” and have presented arguments clearly distinguishing it from physical aggression. The unexpected nature of the findings reported here may suggest that the instrument being used to measure aggression in this study was not adequate to capture female aggression.

An important theoretical possibility is that the association of a preference for instrumental over social goals in females is more unusual in adolescent girls than in adolescent boys, given the intensity of relational involvement that typically characterizes feminine identity in adolescence (Gilligan, Lyons, & Hanmer, 1990). Such a lack of valuing of social goals in females may thus be seen as more likely to occur when more severe disruptions to normal

socialization processes take place, and rejection of the social orientation expected of girls at this age might be reasonably associated with the angry, emotionally aggressive interpersonal style of reactive aggression. Thus, the valuing of instrumental over social goals in girls may not reflect the cold preference for material gain or power associated with an instrumental orientation in boys, but may be instead more associated with a rejection of the social orientation expected of girls.

In sum, these findings of unique social-information processing patterns in reactive and proactive aggression in girls are provocative and potentially important. However, it will be crucial to determine whether these findings can be replicated on another sample of girls.

#### Reactive and proactive aggression and cortisol secretion.

Previous research has suggested that there is some relationship between cortisol secretion and aggression in children, but inconsistencies in the findings to date make it difficult to draw firm conclusions as to the nature of this association. It was proposed here that these inconsistencies in the literature might be resolved by specifically defining the type of the aggression studied, and by developing theoretically-driven hypotheses as to why specific forms of aggression may be related to specific patterns of cortisol secretion. Thus, it was hypothesized that proactive aggression would be related to decreased cortisol secretion based on both empirical evidence that some forms of aggression are associated with decreased cortisol secretion (i.e., McBurnett et al., 2000; Pajer et al., 2001), and on the understanding that decreased cortisol secretion may reflect an increased sense of control over the environment and social situations associated with proactive aggression. Similarly, it was hypothesized that reactive aggression would be related to increased cortisol secretion, based both on evidence that aggression coupled with anxiety is associated with increased cortisol secretion (i.e., McBurnett et

al., 1991), and on the understanding that increased cortisol secretion may reflect an increased sense of uncontrollability and negative emotional valence (Stansbury & Gunnar, 1994).

Unfortunately, cortisol secretion as measured in this sample was associated with neither reactive nor proactive aggression, and it was also not found to be associated with any of the social information processing variables proposed to mediate the aggression-cortisol relationship. Thus, the inconsistencies of the previous literature are replicated in this study, at least for this adolescent sample.

There are several possible explanations as to why the predicted associations were not found in this sample. First, cortisol secretion may be reflective more of a state of being, or of a reaction to a particular situation, than of a trait-like predisposition to behavior. Thus, the hypothetical vignettes presented to the respondents in this study may not have elicited the same emotional and physiological response than would have been produced by an actual event. Similarly, retrospective ratings of general behavior patterns as reflected in the Child Behavior Rating Scales may not capture the dynamic changes in emotion and behavior in different situations that could drive changes in cortisol secretion.

This possibility is supported by findings of researchers who have examined cortisol reactivity to specific provocation situations, as opposed to baseline cortisol secretion. Van Goozen et al (1998) found that boys who were high in externalizing behavior and high in anxiety exhibited the greatest cortisol response to stress, while boys who were high in externalizing behavior and low in anxiety exhibited the lowest cortisol response to stress. Similarly, Scarpa (1997) found higher rates of aggression in children who demonstrated increased cortisol secretion in response to a stressor task than in those who demonstrated decreased cortisol secretion in response to the same task. Van Goozen et al (2000) found a decline in cortisol

secretion in response to a stressor task in children with Oppositional Defiant or Conduct Disorder, as opposed to the increase in cortisol secretion to the same stimuli in normal children. Thus, cortisol secretion may be associated with a specific pattern of social information processing and aggressive behavior in the context of a particular social stressor, rather than with a general predisposition toward a pattern of social information processing or an aggressive style.

A second possibility is that while there may be an association baseline cortisol secretion with a particular form of aggression (i.e., McBurnett et al., 1991; Pajer et al., 2001), that age of onset of aggression may be a critical variable in this tonic “setting” of the HPA axis. For example, McBurnett et al (1996) found that children who began to act aggressively early, and who maintained this aggressive behavior throughout childhood, were most likely to demonstrate low tonic levels of cortisol secretion. In the sample of adolescents investigated here it was not possible to distinguish those whose aggression began early from those whose aggression followed an adolescent-onset pattern. Thus, the lack of association between cortisol secretion and either form of aggression here is not surprising in light of McBurnett’s association of low cortisol secretion specifically with early onset aggressive behavior. To address this issue, the association of cortisol secretion with proactive and reactive aggression in much younger children will have to be explored, and longitudinal investigations examining the development of cortisol secretion patterns in aggression over time will need to be conducted.

#### Review of Exploratory Findings: Proactive and Reactive Aggression and Secondary Measures

Several sets of exploratory analyses were run on this data set. First, the constructs of reactive and proactive aggression were explored through the administration and analysis of measures of constructs that were thought to be theoretically similar to these main variables of interest. Specifically, it was argued that Psychopathy, and particularly the Callous-Unemotional

(CU) traits of Psychopathy, should be similar to proactive aggression because of the instrumental, unemotional characteristics with which both are associated. Therefore, the teacher- and child-report forms of the Antisocial Processes Screening Device (Frick, Bodin, & Barry, 2000) were administered as putative secondary measures of proactive aggression. It was also suggested that the Irritability subscale of the Buss-Durkee Hostility Inventory, designed to measure “readiness to explode with negative affect to the slightest provocation” (Buss & Durkee, 1957, p. 343), would be logical secondary measure of reactive aggression.

To test the empirical validity of these hypothesized associations, analyses were run to determine if Psychopathy generally, and CU traits specifically, were associated uniquely with proactive aggression. Likewise, analyses were run to determine if Irritability was associated uniquely with reactive aggression.

The results of the partial correlation analyses examining the relationship among Psychopathy and reactive and proactive aggression were surprising, given the expectation that CU traits would uniquely distinguish proactive from reactive aggression. In fact, Psychopathy correlated significantly and positively with both reactive and proactive aggression, while CU traits did not. A further exploration of the data revealed that of all of the factors of Psychopathy, only CU traits failed to correlated significantly with proactive or reactive aggression. That is, Psychopathy generally, and the factors of Impulsivity and Narcissism specifically, all correlated positively and significantly with both proactive and reactive aggression, but CU traits did not.

These results must be interpreted cautiously, as no a-priori hypothesis as to how the factors of Impulsivity and Narcissism would be related to proactive or reactive aggression were formulated. However, the data are certainly intriguing, in that they suggest that the CU traits of

psychopathy may reflect unique characteristics of behavior that are not tapped by either proactive or reactive aggression.

Blair's (1995) research into the existence of a dysfunctional Violence Inhibition Mechanism (VIM) in Psychopathy may shed some light on what these characteristics may be. Blair has proposed the VIM as a "cognitive mechanism which, when activated by non-verbal communications of distress (i.e., sad facial expression, the sight and sound of tears), initiates a withdrawal response; a schema will be activated predisposing the individual to withdraw from the attack" (Blair, 1995, p. 3). According to Blair, such a mechanism may be subserved by neural circuits involving the orbito-frontal cortex and the amygdala. Dysfunction in these areas would tend to disrupt an individual's ability to respond to distress cues in others, thus giving the person the appearance of being relatively callous, unemotional, and unconcerned about the welfare of others (Fisher & Blair, 1998). Dysfunction to these same brain regions could also disrupt behavioral functioning associated with response to punishment, suggesting why Impulsivity is associated with but distinguishable from Callous-Unemotional traits.

The findings of an association of both Psychopathy and CU traits with the later stages of SIP (stages 3, 4, and 5), as well as the lack of association of these variables with Stage 2 of SIP, are consistent with Blair's formulation as well. Specifically, if an individual were to lack a mechanism which allowed him or her to experience punishment in association with others' distress, the individual would be more open to responding to the positive consequences of aggressive behavior. Such a lack of empathic responding would also set the stage for diminished interest in interpersonal goals, and thus a relative focus of attention on instrumental goals in social conflict situations. Relevantly, these social information processing characteristics of

Psychopathy and CU traits were found to be more similar to proactive aggression than reactive aggression in this sample.

Given that CU traits seemed to reflect a unique characteristic of Psychopathy that was not captured by either reactive or proactive aggression, the unique, negative association between CU traits and cortisol secretion is particularly intriguing. The amygdala has been associated with activation of the HPA axis in the context of fear, and it has been found that lesioning of the amygdala in rats impairs the HPA axis response to stress (Graham, Heim, Goodman, Miller, & Nemeroff, 1999; Steckler & Holsboer, 1999). Thus, it is tempting to speculate that the deficiencies in amygdala functioning postulated by Blair in association with VIM deficits may also be associated with the diminished cortisol secretion found to be associated with CU traits in this sample. However, the fact that the overall child-report ratings of Psychopathy are positively associated with cortisol secretion raises interesting questions as to the different aspects of behavior that child- and teacher-reports of Psychopathy are measuring. Finally, in interpreting these findings with respect to cortisol secretion and Psychopathy, it is critical to keep in mind the exploratory nature of these analyses, and the extreme caution with which any of these results must be interpreted.

The Irritability scale of the Buss-Durkee Hostility Inventory was not found to distinguish between reactive and proactive aggression, and was, in fact, found to correlate significantly with both. Though it is unclear why this scale did not distinguish between these two constructs, it is possible that the types of negative affect characteristic of reactive aggression are specifically those associated with withdrawal, such as anxiety and depression, but not necessarily of approach, such as anger (see Harmon-Jones, 2003). While anxiety and depression are theoretically associated uniquely with reactive aggression, anger may be associated with both

proactive and reactive aggression. Since the Irritability scale may reflect aggression associated with anger more than with anxiety or depression, (i.e., “I often feel like a powder keg ready to explode”), it is possible that this scale was not actually appropriate to distinguish between reactive and proactive aggression.

#### Proactive and reactive aggression, comorbid psychopathology, and cortisol secretion.

As mentioned above, previous research has suggested that comorbid psychopathology, especially anxiety and depression, are associated with reactive but not with proactive aggression (Francisco et al., 2002; Dodge et al., 1997). Partial correlations of proactive and reactive aggression, each controlling for the other, were run with scores from the Children’s Depression Inventory (CDI) and the Revised Children’s Manifest Anxiety Scale (RCMAS), to determine if these findings could be replicated in this sample.

The results of these analyses did replicate previous research, in that both anxiety and depression were associated with reactive but not proactive aggression. These findings lend further support to the idea that reactive aggression is created and maintained by negative affect, perhaps associated with negative cognitive schema or negative attribution biases in social situations, while proactive aggression is likely to stem from factors distinct from such emotional pathology.

While the hypothesis that reactive aggression would be associated with increased cortisol secretion was not supported, the positive association of depression and anxiety found in previous research was replicated in this sample. This replication of findings consistently reported in the literature suggests that the measure of cortisol secretion used in this study was satisfactory.

Anxiety and depression were found to be associated with both cortisol secretion and with reactive aggression, but reactive aggression was not found to be associated with cortisol

secretion. This lack of association between reactive aggression and cortisol secretion is difficult to explain. However, it is possible that emotional dysregulation is only part of a combination of factors that contributes to reactive aggressive behavior. Other factors, such as environmental modeling or reinforcement of reactive aggressive behavior, may not be associated with cortisol secretion, and therefore the physiological contribution to reactive aggression associated with the emotional variables is obscured. In support of this explanation, Scarpa (1997) observed that the combination of a history of physical abuse and internalizing behavior was most predictive of aggressive behavior in a sample of 7 to 15 year old children.

#### Limitations and Directions for Further Research

While the association between reactive and proactive aggression and some social information processing disturbances found in previous research was replicated here, the hypotheses that cortisol secretion could be associated both with specific forms of aggressive behavior, and with specific information processing biases, were not supported. Several limitations in this investigation may have obscured such relationships, if they do indeed exist. First, cortisol secretion was measured here at a tonic, baseline level, while the social information processing and behavioral variables of interest may be more reflective of dynamic and situation-driven processes. Thus, measuring cortisol reactivity in the face of specific stressors, and examining the association of these with real-time behavioral observations of aggression and social-information processing may have been a more appropriate approach to testing these relationships.

Second, the measures used in this investigation to examine social information processing in adolescents may not have been emotionally salient enough to elicit the hostile attribution biases associated with reactive aggression. Real-time responses to salient stressors, such as the

“accidental overhearing” of a peer engaging in an ambiguously-motivated antagonistic activity (i.e., Dodge, 1980), may be a more powerful approach to accessing these early, emotionally-driven stages of social information processing.

A final limitation of this study is that this sample was comprised of relatively high-functioning adolescent participants. Most were participating in a summer school program to earn extra high school credits in health and physical education. Thus, it can be expected that most had developed sufficiently effective, non-aggressive social problem solving skills to allow them to succeed in school. Such an assumption was borne out by informal observations of the students’ interactions during the administration of the research.

Perhaps the most surprising but provocative finding of this investigation was the unique nature of Callous-Unemotional traits of Psychopathy, in that these traits were associated with neither reactive nor proactive aggression. Blair’s theory regarding a Violence Inhibition Mechanism (VIM) is intriguing in this context. Though most of Blair’s previous work has been done examining the construct of Psychopathy as a whole in relation to the VIM, the exploratory findings presented here suggest that the Callous-Unemotional traits of Psychopathy may be worthy of focused study as well. However, the results reported here based on the Psychopathy measure the Antisocial Processes Screening Device (APSD) must be interpreted with caution, as the teacher and parent versions have been validated only for children ages 6 to 13. While the scores in this sample fell largely along a Gaussian distribution and demonstrated acceptable reliability ( $\alpha = .65$  for the child-report version and  $\alpha = .74$  for the teacher-report version), more work will have to be done to determine the validity of this measure on this age group.

It is important to note when considering the speculations regarding gender differences in proactive and reactive aggression, and when considering the suggestions for alternative

delineations of aggression based on Psychopathy, that the findings which stimulated these ideas are based on exploratory analyses. Not only were no a-priori hypotheses developed before these tests were run, the sheer number of post-hoc and exploratory analyses that were conducted significantly elevate the danger of experiment-wise error. In fact, many of these findings must be qualified by the understanding that they may no longer remain significant if statistical corrections such as the Bonferroni technique were employed.

In considering the practical and theoretical importance of the findings reported here, it is worthwhile to address the concerns regarding the continued relevance of the reactive/proactive distinction in the study of aggression as highlighted by Bushman and Anderson (2001) in their article “Is it time to pull the plug on the hostile versus instrumental aggression dichotomy?” These authors suggest that while a dichotomization of aggression into hostile/instrumental (or, as formulated here, reactive/proactive) may continue to be appropriate for nonhuman aggression, it does not accommodate the complexity of human aggression. They suggest that a model based on knowledge structures would be more able to capture the complex motives and subtle information processing components of aggression.

It is true that proactive and reactive aggression were found to be highly correlated in this sample, as they have been found to be in previously reported studies. Even when significant differences were observed, the effect sizes were often quite small. Thus, these data may not support a reification of such distinct categories of aggression in the relatively normally functioning sample studied here. And as Bushman and Anderson point out, it would certainly be simplistic to deny that aggression is created and maintained by a complex array of factors, all of which need to be considered in a comprehensive understanding of aggressive behavior.

However, the authors themselves note that “placing such variables under the scientific microscope has been and will continue to be a key strategy in furthering the field’s understanding of human aggression” (Bushman & Anderson, 2001, p. 278). In examining the psychophysiology of aggression, it may continue to be necessary to study more extreme examples of particular types of aggressive behavior to zero in on the physiological components involved. Such an examination may require a somewhat artificial dichotomization of a more complex continuum of behavior, and extremely reactive-aggressive or proactive-aggressive individuals may need to be studied to uncover potentially relevant neuro-physiological correlates of such behavior. Once such “pure” examples are more clearly understood, more subtle and complex variations of these can be explored. Similarly, it is certainly worthwhile to simultaneously examine the more subtle and complex contributors to aggression using a more fine-grained analysis of knowledge structures as suggested by Bushman and Anderson (2001).

The theoretical position of this investigation was that physiological and information processing components of aggression may be integrally linked. The participants studied here may not have exhibited extreme enough forms of aggression for physiological distinctions to be relevant or detectable, and therefore it may have been premature to introduce the additional subtlety of an information processing mediator. Perhaps in such a well-adjusted sample the use of a more subtle, knowledge structure approach to examining aggression may have been more successful. In fact, much of the social-information processing data reported in the literature was supported here, in a “normal” sample. This does not mean that extreme or more “pure” groups of proactive or reactive aggressive individuals do not exist, or that attempting to study the physiological correlates of such aggressive behavior is not a worthwhile pursuit. It simply means that the sample was not appropriate to test such a dichotomous formulation. Different

tools may be necessary to examine aggression at different points along a continuum. Though Bushman and Anderson (2001) may have identified an effective way to examine aggression at a more subtle social cognitive level, this does not necessarily mean that the dichotomous models derived from animal research are no longer useful in the extremes.

It is also worthwhile to consider that while cortisol is a commonly measured index of the functioning of the hypothalamic-pituitary-adrenal axis because it is easily measured in saliva, a more sensitive measure of the functioning of this stress system may be corticotrophin releasing factor (CRF). However, CRF is more difficult to measure, and is often only examined in humans indirectly through the use of the CRF stimulation test (i.e., Holsboer, von Bardeleben, Heuser, & Steiger, 1988).

In sum, the research presented here does demonstrate that there are many ways to delineate aggression, including examining personality traits associated with aggression such as impulsivity or callousness, as well as exploring behavioral distinctions such as reactive versus proactive. Efforts to identify empirically valid distinctions between specific types of aggressive behavior can still contribute to our efforts to understand the social and biological contributions to aggression, and to identify potential solutions to this pervasive and ageless problem.

## References

- Atkins, M.S., & Stoff, D. M. (1993). Instrumental and hostile aggression in childhood disruptive behavior disorders. Journal of Abnormal Child Psychology, 21(2), 165-178.
- Bandura, A. (1973). *Aggression: A Social Learning Analysis*. Englewood Cliffs, NJ: Prentice Hall.
- Baron, R.M., & Kenny, D.A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. Journal of Personality and Social Psychology, 51(6), 1173-1182.
- Berkowitz, L. (1993). Aggression: Its Causes, Consequences, and Control. Philadelphia: Temple University Press.
- Biaggio, M.K., Supplee, K., & Curtis, N. (1981). Reliability and validity of four anger scales. Journal of Personality Assessment, 45, 639-648.
- Blair, R.J.R. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. Journal of Neurology, Neurosurgery, and Psychiatry, 71, 727-731.
- Blair, R.J.R. (1995). A cognitive developmental approach to morality: Investigating the psychopath. Cognition, 57, 1-29.
- Brown, K., Atkins, M.S., Osborne, M.L, & Milnamow, M. (1996). A revised teacher rating scale for reactive and proactive aggression. Journal of Abnormal Child Psychology, 24(4), 473-480.
- Buchner, A., Erdfelder, E., & Faul, F. (1997). How to Use G\*Power [WWW document]. URL [http://www.psych.uni-duesseldorf.de/aap/projects/gpower/how\\_to\\_use\\_gpower.html](http://www.psych.uni-duesseldorf.de/aap/projects/gpower/how_to_use_gpower.html).
- Bushman, B.J., & Anderson, C.A. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy? Psychological Review, 108(1), 273-279.

- Buss, A.H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. Journal of Consulting Psychology, 21, 343-349.
- Buss, K.A., Malmstadt Schumacher, J.R., Dolski, I., Kalin, N.H., Hill Goldsmith, H., Davidson, R. J. (2003). Right frontal brain activity, cortisol, and withdrawal behavior in 6-month-old infants. Behavioral Neuroscience, 117(1), 11-20.
- Connor, D.F., Steingard, R.J., Anderson, J.J., & Melloni, R.H. (2003). Gender differences in reactive and proactive aggression. Child Psychiatry and Human Development, 33(4), 279-294.
- Cornell, D.G., Warren, J., Hawk, G., Stafford, E., Oram, G., & Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. Journal of Consulting and Clinical Psychology, 30, 1-17.
- Crick, N.R., & Dodge, K.A. (1996). Social information-processing mechanisms in reactive and proactive aggression. Child Development, 67, 993-1002.
- Crick, N.R., & Dodge, K.A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. Psychological Bulletin, 115(1), 74-101.
- Crick, N.R., & Rose, A. J. (2000). Toward a gender-balanced approach to the study of social-emotional development: A look at relational aggression. In P. A. Miller and E. Kofsky Scholnick, Ellis, (Eds.), Toward a Feminist Developmental Psychology, 153-168.
- Dodge, K.A. (1980). Social cognition and children's aggressive behavior. Child Development, 51, 162-170.

- Dodge, K.A. (1991). The structure and function of reactive and proactive aggression. In Pepler, D.J., Rubin, K.H., et al (Eds.), The Development and Treatment of Childhood Aggression. Hillsdale, NJ: Lawrence Erlbaum Associates, (pp.201-218).
- Dodge, K.A. (1980). Social cognition and children's aggressive behavior. Child Development, 51, 162-170.
- Dodge, K.A., & Coie, J.D. (1987). Social-information processing factors in reactive and proactive aggression in children's peer groups. Journal of Personality and Social Psychology, 53, 1146-1158.
- Dodge, K.A., Laird, R., Lochman, J.E., & Zelli, A. (2002). Multidimensional latent-construct analysis of children's social information processing patterns: Correlations with aggressive behavior problems. Psychological Assessment, 14(1), 60-73.
- Dodge, K.A., Lochman, J.E., Harnish, J.D., Bates, J.E., Pettit, G.S. (1997). Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. Journal of Abnormal Psychology, 106(1), 37-51.
- Dodge, K.A., Murphy, R.R., & Buchsbaum, K. (1984). The assessment of intention-cue detection skills in children: Implications for developmental psychopathology. Child Development, 55, 163-173.
- Dodge, K.A., & Newman, J.P. (1981). Biased decision-making processes in aggressive boys. Journal of Abnormal Psychology, 90(4), 375-379.
- Dodge, K.A., Price, J.M., Bachorowski, J., & Newman, J.P. (1990). Hostile attribution biases in severely aggressive adolescents. Journal of Abnormal Psychology, 99(4), 385-392.
- Dodge, K.A., & Somberg, D.R. (1987). Hostile attributional biases among aggressive boys are exacerbated under conditions of threat to self. Child Development, 58, 213-224.

- Dodge, K.A., & Tomlin, A.M. (1987). Utilization of self-schemas as a mechanism of interpretational bias in aggressive children. *Social Cognition*, 5(3), 280-300.
- Dollard, J., Doob, L.W., Miller, N.E., Mowrer, O.H., & Sears, R.R. (1939). Frustration and Aggression. New Haven, Conn.: Yale University Press.
- Fisher, L., & Blair, R.J.R. (1998). Cognitive impairment and its relationship to psychopathic tendencies in children with emotional and behavioral difficulties. Journal of Abnormal Child Psychology, 26(6), 511-519.
- Fontaine, R.G., Burks, V.S., & Dodge, K.A. (2002). Response decision processes and externalizing behavior problems in adolescents. Development and Psychopathology, 14, 107-122.
- Francisco, J., Van Voorhees, E., Bowser, F., Travers, J., & Scarpa, A. (2002, June). The relationship of anxiety and depression with reactive and proactive aggression. Poster presented at the 14<sup>th</sup> Annual Convention of the American Psychological Society, New Orleans, LA.
- Frick, P.J. (October, 2000). Using the construct of psychopathy to understand antisocial and violent youth. Paper presented at Festschrift honoring the work of Robert D. Hare at the University of British Columbia, Vancouver, Canada.
- Frick, P.J., Bodin, S.D., & Barry, C.T. (2000). Psychopathic traits and conduct problems in community and clinic-referred samples of children: Further development of the psychopathy screening device. Psychological Assessment, 12(4), 382-393.
- Frick, P.J., Cornell, A.H., Barry, C.T., Bodin, S.D., & Dane, H.E. (2003). Callous-unemotional traits and conduct problems in the prediction of conduct problem severity, aggression, and self-report of delinquency. Journal of Abnormal Child Psychology, 31(4), 457-470.

- Gilligan, C., Lyons, N.P., and Hanmer, T.J. (1990). Making Connections: The Relational World of Adolescent Girls at Emma Willard School. Cambridge, MA: Harvard University Press.
- Goodyer, I.M., Herbert, J., Tamplin, A., & Altham, P.M.E. (2000). Recent life events, cortisol, dehydroepiandrosterone and the onset of major depression in high-risk adolescents. British Journal of Psychiatry, 177, 499-504.
- Graham, Y.P., Heim, C., Goodman, S.H., Miller, A.H., & Nemeroff, C.B. (1999). The effects of neonatal stress on brain development: Implications for psychopathology. Development and Psychopathology, 11, 545-565.
- Gunnar, M., Hertzgaard, L., Larson, M., & Rigatuso, J. (1992). Cortisol and behavioral responses to repeated stressors in the human newborn. Developmental Psychobiology, 24, 487-505.
- Harmon-Jones, E. (2003). Clarifying the emotive functions of asymmetrical frontal cortical activity. Psychophysiology, 40(6), 838-848.
- Hertzgaard, L., Gunnar, M., Larson, M., Brodersen, L., & Lehman, H. (1992). First time experiences in infancy: When they appear to be pleasant, do they activate the adrenocortical stress response? Developmental Psychobiology, 25, 319-333.
- Hare, R.D. (1991). Manual for the Hare Psychopathy Checklist-revised. Toronto, Ontario, Canada: Multi-Health Systems.
- Holsboer, F., von Bardeleben, U., Heuser, I., Steiger, A. (1988). Human corticotropin-releasing hormone challenge tests in depression. In A.F. Schatzberg & C.B. Nemeroff (Eds.), The Hypothalamic-Pituitary-Adrenal Axis: Physiology, Pathophysiology, and Psychiatric Implications (pp. 79-100). New York: Raven Press, Ltd.

- Kagan, J., Reznick, J.S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. Child Development, 58, 1459-1473.
- Kaufman, J. (1991). Depressive disorder in maltreated children. Journal of the American Academy of Child and Adolescent Psychiatry, 30:2, 257-265.
- Kovacs, M. (1985). The children's depression inventory (CDI). Psychopharmacology Bulletin, 21, 995-998.
- Kruesi, M.J.P., Schmidt, M.E., Donnelly, M., Hibbs, E.D., & Hamburger, S.D. (1989). Urinary free cortisol output and disruptive behavior in children. Journal of the American Academy of Child and Adolescent Psychiatry, 28(3), 441-443.
- McBurnett, K., Lahey, B. B., Capasso, L., & Loeber, R. (1996). Aggressive symptoms and salivary cortisol in clinic-referred boys with conduct disorder. In Craig F. Ferris, & Thomas Grisso (Eds.), Understanding Aggressive Behavior in Children, Annals of the New York Academy of Sciences, 794, 169-178.
- McBurnett, K., Lahey, B.B., Frick, P.J., Risch, C., Loeber, R., Hart, E.L., Christ, M.A.G., Hanson, K.S. (1991). Anxiety, inhibition, and conduct disorder in children: II. Relation to salivary cortisol. Journal of the American Academy of Child and Adolescent Psychiatry, 30(2), 192-196.
- McBurnett, K., Lahey, B.B., Rathouz, P.J., & Loeber, R. (2000). Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. Archives of General Psychiatry, 57, 38-43.
- McBurnett, K., Pfiffner, L.J., Capaso, L., Lahey, B.B., & Loeber, R. (1997). Children's aggression and DSM-III-R symptoms predicted by parent psychopathology, parenting practices, cortisol, and SES. In A. Raine, P.A. Brennan, D.P. Farrington, & S.A.

- Mednick (Eds.), Biosocial Bases of Violence, NATO ASI Series, Series A: Life Sciences, 292 (pp. 345-348). New York: Plenum Press.
- Nachmias, M., Gunnar, M., Mangelsdorf, S., Parritz, R.H., & Buss, K. (1996). Behavioral inhibition and stress reactivity: The moderating role of attachment security. Child Development, 67, 508-522.
- Nasby, W., Hayden, B., & DePaulo, B.M. (1980). Attributional bias among aggressive boys to interpret unambiguous social stimuli as displays of hostility. Journal of Abnormal Psychology, 89(3), 459-468.
- Pajer, K., Gardner, W., Rubin, R.T., Perel, J., & Neal, S. (2001). Decreased cortisol levels in adolescent girls with conduct disorder. Archives of General Psychiatry, 58, 297-302.
- Poulin, F., & Boivin, M. (2000). Reactive and proactive aggression: Evidence of a two-factor model. Psychological assessment, 12(2), 115-122.
- Quiggle, N.L., Garber, J., Panka, W.F., & Dodge, K.A. (1992). Social information processing in aggressive and depressed children. Child Development, 63, 1305-1320.
- Raine, A., Meloy, J.R., Bihrl, S., Stoddard, J., LaCasse, L., & Buchsbaum, M.S. (1998). Reduced prefrontal and increased subcortical brain functioning assessed using positron emission tomography in predatory and affective murderers. Behavioral Sciences and the Law, 16, 319-332.
- Reynolds, C.R., & Richmond, B.O. (1985). Revised Children's Manifest Anxiety Scale: Manual. Los Angeles: Western Psychological Services.
- Salivadiagnostics. Retrieved June 20, 2002 from <http://www.ibl-hamburg.com/cortisol.html>.
- Sapolsky, R.M. (1998). Why Zebras Don't Get Ulcers: An Updated Guide to Stress, Stress-Related Diseases, and Coping. New York: W.H. Freeman and Company.

- Scarpa, A. (1997). Aggression in physically abused children: The interactive role of emotion regulation. In A. Raine, P.A. Brennan, D.P. Farrington, & S.A. Mednick (Eds.), Biosocial Bases of Violence, NATO ASI Series, Series A: Life Sciences, 292 (pp. 341-343). New York: Plenum Press.
- Scarpa, A., & Raine, A. (2000). Violence associated with anger and impulsivity. In J. Borod (Ed.), The Neuropsychology of Emotion. New York: Oxford University Press (pp.320-339).
- Scarpa, A., & Raine, A. (1997). Psychophysiology of anger and violent behavior. The Psychiatric Clinics of North America, 20(2), 375-394.
- Scerbo, A.S., & Kolko, D.J. (1994). Salivary testosterone and cortisol in disruptive children: Relationship to aggressive, hyperactive, and internalizing behaviors. Journal of the American Academy of Child and Adolescent Psychiatry, 33(8), 1174-1184.
- Schwartz, D., Dodge, K.A., Coie, J.D., Hubbard, J.A., Cillessen, A.H.N., & Bateman, H. (1998). Journal of Abnormal Child Psychology, 26(6), 431-440.
- Silverman, W. K., & Rabian, B. (1999). Rating scales for anxiety and mood disorders. In D. Shaffer, C.P. Lucas, & J.E. Richters (Eds.), Diagnostic Assessment in Child and Adolescent Psychopathology. New York, NY: The Guilford Press, (pp. 127-166).
- Stansbury, K., & Gunnar, M. (1994). Adrenocortical activity and emotion regulation. In N.A. Fox (Ed.), The Development of Emotion Regulation: Biological and Behavioral Considerations, Monographs of the Society for Research in Child Development, 59(2-3), 108-134.
- Steckler, T., & Holsboer, F. (1999). Corticotropin-releasing hormone receptor subtypes and emotion. Biological Psychiatry, (46), 1480-1508.

- Steinberg, M.S., & Dodge, K.A. (1983). Attributional bias in aggressive adolescent boys and girls. Journal of Social and Clinical Psychology, *1*(4), 312-321.
- van Goozen, S.H.M., Matthys, W., Cohen, K.T., Buitelaar, J.K., & van Engeland, H. (2000). Hypothalamic-pituitary-adrenal axis and autonomic nervous system activity in disruptive children and matched controls. Journal of the American Academy of Child and Adolescent Psychiatry, *39*(11), 1438-1445.
- van Goozen, S.H.M., Matthys, W., Cohen-Kettenis, P.T., Gispens-de Wied, C., Wiegant, V.M., van Engeland, H. (1998). Salivary cortisol and cardiovascular activity during stress in oppositional-defiant disorder boys and normal controls. Biological Psychiatry, *(43)*, 531-539.
- Vanyukov, M.M., Moss, H.B., Plail, J.A., Blackson, T., Mezzich, A.C., & Tarter, R.E. (1993). Antisocial symptoms in preadolescent boys and in their parents: Association with cortisol. Psychiatry Research, *46*, 9-17.
- Vitaro, F., Gendreau, P.L., Tremblay, R.E., & Oligny, P. (1998). Reactive and proactive aggression differentially predict later conduct problems. Journal of Child Psychiatry, *39*(3), 377-385.
- Weiss, B., Dodge, K.A., Bates, J.E., & Pettit, G.S. (1992). Some consequences of early harsh discipline: Child aggression and a maladaptive social information processing style. Child Development, *63*, 1321-1335.

Table 1.

Descriptive statistics: Proactive and reactive aggression

	N	Mean	Standard Deviation	Range	Coeff. $\alpha$
Proactive Aggression	120	14.13	2.95	10 to 23	0.76
Reactive Aggression	121	9.34	2.14	6 to 15	0.63

Table 2.

Social information processing variables, with hypothesized associations to proactive and reactive aggression and cortisol secretion

Name of Variable	Description	Expected Association
Hostile Attribution Bias (HAB) (stage 2)	Likelihood that respondent will interpret an ambiguous social provocation as reflecting hostile intent	Reactive aggression: + Cortisol secretion: +
Social Goals (SG) (stage 3)	Degree to which respondent values instrumental over social goals	Proactive aggression: + Cortisol secretion: -
Aggressive Problem Solving (APS) (stage 4)	Likelihood of respondent identifying aggressive responses to social problems	Proactive aggression: + Cortisol secretion: -
Outcome Expectation (stage 5)	Degree to which respondent believes aggressive strategy would be effective in obtaining goal	Proactive aggression: + Cortisol secretion: -
Response Efficacy (Stage 5)	Ease with which respondent rates own ability to enact an aggressive response	Proactive aggression: + Cortisol secretion: -

Table 3.

Descriptive statistics: Social information processing variables

	N	Mean	Standard Deviation	Range	Coeff. $\alpha$
Hostile Attribution Bias (HAB)	125	3.38	2.21	0 to 8	0.60
Social goals (SG)	126	1.21	1.31	0 to 4	0.68
Aggressive Problem Solving (APS)	126	.56	1.08	0 to 5	0.65
Outcome Expectation (OE)	126	-2.69	2.12	-7 to 2	
Response Efficacy (RE)	126	8.87	2.99	0 to 16	0.79

Table 4.

Descriptive statistics: Cortisol (log10 transformation)

	N	Mean	Standard Deviation	Range
Sample 1	125	1.617	.189	1.145 to 2.322
Sample 2	98	1.603	.180	1.185 to 2.238

Table 5.

Descriptive statistics: psychopathy variables

	N	Mean	Standard Deviation	Range	Coeff. $\alpha$
Child-rated Psychopathy	120	11.33	4.30	1 to 25	0.65
Impulsivity Factor	122	4.47	1.91	0 to 9	
Callous-Unemotional Factor	122	3.36	2.01	0 to 10	
Narcissistic Factor	120	3.52	2.10	0 to 9	
Teacher-rated Psychopathy	123	6.45	4.87	0 to 23	0.74
Impulsivity Factor	124	2.18	1.66	0 to 9	
Callous-Unemotional Factor	124	3.23	2.45	0 to 8	
Narcissistic Factor	123	1.07	2.21	0 to 11	

Table 6.

Descriptive statistics: Irritability scale of the Buss-Durkee Hostility Inventory

	N	Mean	Standard Deviation	Range	Coeff. $\alpha$
Hostility Scale (BDHI)	125	5.04	2.52	0 to 11	0.67

Table 7.

Descriptive statistics: Anxiety and depression

	N	Mean	Standard Deviation	Range	Coeff. $\alpha$
Anxiety (RCMAS)	118	9.34	5.93	0 to 28	0.86
Depression (CDI)	124	8.36	6.82	0 to 32	0.88

Table 8.

Associations of age and gender with proactive and reactive aggression, social information processing variables, and cortisol secretion.

	Reactive Aggression	Proactive Aggression	Cortisol (log 10 transform)	HAB	SG	APS	OE	RE
Age	.11 (ns)	.13 (ns)	.39**	.14(ns)	.03(ns)	.09(ns)	.13(ns)	.13 (ns)
Gender	.18*	-.04 (ns)	-.10 (ns)	.01(ns)	-.14(ns)	-.30**	.06(ns)	-.17(ns)

\*  $p < .05$

\*\*  $p < .01$

Table 9.

Proactive aggression and social information processing

	Degrees of Freedom	Standardized $\beta$	R <sup>2</sup> $\Delta$	p-value of R <sup>2</sup> $\Delta$
Hostile Attribution Bias (HAB)	112	.094	.006	.415
Social goals (SG)	113	.153	.015	.176
Aggressive Problem Solving (APS)	113	.264	.045	.014
Outcome Expectation (OE)	113	-.116	.009	.318
Response Efficacy (RE)	113	.314	.063	.004

Table 10.

Cortisol secretion (log10 transformation) and reactive/proactive aggression

	Degrees of Freedom	Standardized $\beta$	$R^2\Delta$	p-value of $R^2\Delta$
Proactive Aggression	111	.121	.009	.267
Reactive Aggression	111	-.001	<.001	.995

Table 11.

Cortisol secretion (log10 transformation) and social information processing

	Degrees of Freedom	Standardized $\beta$	R <sup>2</sup> $\Delta$	p-value of R <sup>2</sup> $\Delta$
Hostile Attribution Bias (HAB)	119	-.006	<.001	.942
Social goals (SG)	120	-.014	<.001	.870
Aggressive Problem Solving (APS)	120	.083	.006	.345
Outcome Expectation (OE)	120	.044	.002	.600
Response Efficacy (RE)	120	.074	.005	.388

Table 12.

Reactive aggression and social information processing

	Degrees of Freedom	Standardized $\beta$	$R^2\Delta$	p-value of $R^2\Delta$
Hostile Attribution Bias (HAB)	112	.102	.007	.384
Social goals (SG)	113	.019	.007	.342
Aggressive Problem Solving (APS)	113	.099	.006	.353
Outcome Expectation (OE)	113	.103	.007	.381
Response Efficacy (RE)	113	.076	.004	.487

Table 13.

Proactive aggression and social information processing

		Degrees of Freedom	Standardized $\beta$	R <sup>2</sup> $\Delta$	p-value of R <sup>2</sup> $\Delta$
HAB	Girls	65	.006	.003	.663
	Boys	44	.058	.002	.746
SG	Girls	65	-.081	.004	.597
	Boys	45	.388	.104	.025
APS	Girls	65	.249	0.37	.094
	Boys	45	.334	.077	.046
OE	Girls	65	-.316	.059	.045
	Boys	45	.212	.031	.221
RE	Girls	65	.228	0.31	.116
	Boys	45	.368	.093	.031

Table 14.

Reactive aggression and social information processing: Boys and girls separately

		Degrees of Freedom	Standardized $\beta$	$R^2\Delta$	p-value of $R^2\Delta$
HAB	Girls	65	.257	.040	.090
	Boys	44	-.022	.<.001	.903
SG	Girls	65	.318	.062	.038
	Boys	44	-.074	.004	.665
APS	Girls	65	.085	.004	.559
	Boys	45	.142	.013	.396
OE	Girls	65	.091	.005	.551
	Boys	45	.120	.010	.493
RE	Girls	65	.167	.017	.239
	Boys	45	.031	.001	.853

Table 15.

Extreme group comparisons: Proactive aggression

	df	t-value	Mean Low Grp	Mean High Grp	p-value
Cortisol secretion	101	-1.640	1.585	1.647	.10
SIP: Hostile Attribution Bias	101	-.860	1.02	1.22	.39
Social Goals	102	-2.090	.95	1.50	.04
Aggressive Prob. Solving	102	-3.979	.22	1.07	<.01
Outcome Expectation	102	.429	-2.67	-2.85	.70
Response Efficacy	102	-2.612	8.14	9.70	.01

Table 16.

Extreme group comparisons: Reactive aggression

	df	t-value	Mean Low Grp	Mean High Grp	p-value
Cortisol secretion	98	-1.304	1.594	1.639	.20
SIP: Hostile Attribution Bias	98	-2.368	.84	1.38	.02
Social Goals	99	-.542	1.22	1.36	.59
Aggressive Prob. Solving	99	-7.64	.48	.65	.45
Outcome Expectation	99	-.0510	-2.76	-2.55	.61
Response Efficacy	99	-1.964	8.09	9.22	.05

Table 17a.

Proactive Aggression and Psychopathy (child-child, teacher-teacher ratings only)

	Psychopathy	CU-traits	Impulsivity	Narcissism
Teacher-rated	0.335 **	0.072 (ns)	0.252 **	0.489 **
Child-rated	0.445 **	0.114 (ns)	0.336 **	0.478 **

\* p &lt; .05

\*\* p &lt; .01

Table 17b.

Reactive Aggression and Psychopathy (child-child, teacher-teacher ratings only)

	Psychopathy	CU-traits	Impulsivity	Narcissism
Teacher-rated	0.392 **	0.137 (ns)	0.217 *	0.555 **
Child-rated	0.290 **	0.020 (ns)	0.279 **	0.304 **

\* p &lt; .05

\*\* p &lt; .01

Table 18.

Psychopathy and Social-Information Processing

	Hostile Attribution Bias	Social Goals	Aggressive Problem Solving	Outcome Expectation	Response Efficacy
Psychopathy teacher-rated	0.067 (ns)	0.131 (ns)	0.296 **	0.144 (ns)	0.348 **
Psychopathy child-rated	0.077 (ns)	0.359 **	0.389**	0.094 (ns)	0.458 **
CU traits teacher-rated	-0.140 (ns)	0.018 (ns)	0.126 (ns)	0.097 (ns)	0.286 **
CU traits child- rated	0.040 (ns)	0.343 **	0.311 **	0.122 (ns)	0.385 **

\* p &lt; .05

\*\* p &lt; .01

Table 19.

Psychopathy and Cortisol Secretion (controlling for age, gender, and pregnancy/use of birth control)

	Psychopathy	CU-traits	Impulsivity	Narcissism
Teacher-rated	-.133	-.210*	-.060	-.043
Child-rated	.187*	.080	.154	.133

\*  $p < .05$

\*\*  $p < .01$

Table 20.

Irritability Scale of BDHI and Social Information Processing

	Hostile Attribution Bias	Social Goals	Aggressive Problem Solving	Outcome Expectation	Response Efficacy
Irritability Scale (BDHI)	.053 (ns)	.186*	.213*	-.047 (ns)	.222 *

\*  $p < .05$ \*\*  $p < .01$

Figure 1. General model.

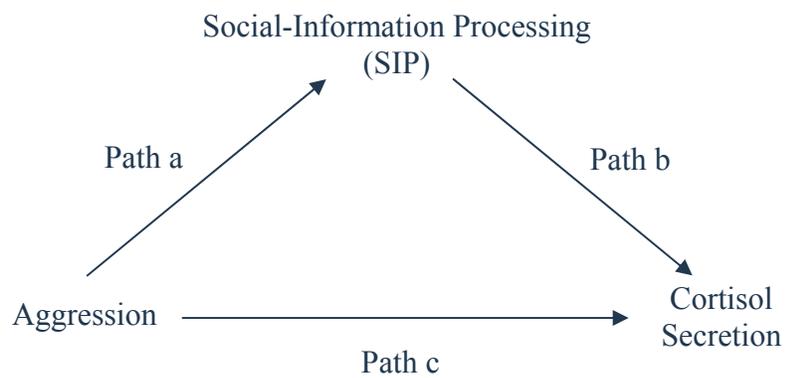


Figure 2. Model a: Proactive aggression

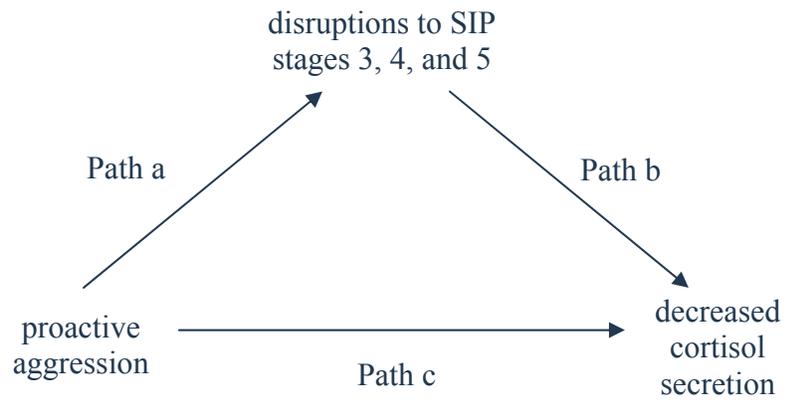
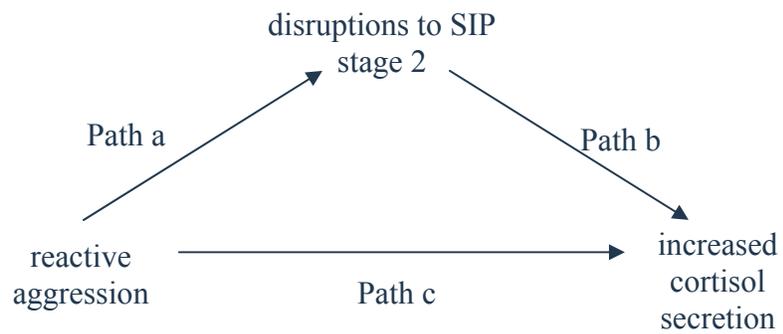


Figure 3. Model b: Reactive aggression



Curriculum Vitae  
 Elizabeth Van Voorhees  
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## **Education**

- 2000 - present **Virginia Polytechnic Institute and State University**, Blacksburg, VA
- Coursework requirements completed and dissertation defended for Ph.D. in Clinical Psychology; expected graduation date of May 2004
  - Dissertation topic: Social Information Processing, Cortisol Secretion, and Aggression in Adolescents
  - Funding for dissertation project provided by grants from the Graduate Student Assembly of Virginia Tech and the Child Clinical Research Fund of the Department of Psychology
- 1996 - 1999 **Loma Linda University**, Loma Linda, CA
- Master of Arts, Experimental Psychology
  - Thesis topic: The Relationship Between Posttraumatic Stress Disorder and Interpersonal/Self-Disturbance in Battered Women
  - Funding for thesis project provided by a grant from the Department of Psychology
- 1988 - 1992 **Brown University**, Providence, RI  
 Degree Earned: Bachelor of Arts, Religious Studies
- 1990 - 1991 **University of Wisconsin**, Madison, WI
- South Asian Studies Program
  - College junior year in Nepal studying Tibetan culture and language

## **Employment Experience**

- 08/2003 - 07/2004 **Pre-doctoral Psychology Intern**  
 Veterans Affairs Medical Center, Salem, VA
- Assist with program development of new Residential Treatment Program associated with SARRTP
  - Conduct inpatient groups and assessments with patients in the substance abuse treatment program
  - Conduct outpatient individual and group psychotherapy and clinical assessments with veterans, including those who have experienced military sexual trauma
  - Act as behavioral medicine liaison to primary and acute medical services, cardiology, extended care rehabilitation, and oncology
  - Conduct neuropsychological assessments in the Memory Disorders Clinic
  - Engage in research examining effectiveness of a follow-up aftercare program to the inpatient Substance Abuse Recovery and Rehabilitation Treatment Program (SARRTP)
  - Engage in research and case presentations to Psychology staff
- 05/2000 - present **Assessment Specialist**  
 Respond, Lewis-Gale Medical Center, Salem, VA

Assess individuals in crisis at Lewis Gale, Pulaski Regional, and Montgomery Regional Hospitals for admission to acute psychiatric care facility

06/2001 - 07/2003 **Psychometrist**

Appalachian Counseling Center, Roanoke, VA  
Administered, scored, and assisted in interpretation of neuropsychological tests

**Graduate Teaching Assistant**

Department of Psychology – Virginia Tech, Blacksburg, VA  
Taught Introductory Psychology, Psychology of Learning, Developmental Psychology

11/1998 - 05/2000 **Therapist and Behavior Management Specialist**

Action Youth Care, Williamson, WV

- Supervised behavior management plans for case managers in three West Virginia counties
- Developed and conducted training seminars for case managers on behavior modification techniques
- Provided in-home therapy to low-income families with at-risk youth

11/1996 - 01/1998 **Counselor**

Helicon Youth Center, Riverside, CA  
Supervised and counseled female and male juveniles at a minimum security treatment center for adolescent offenders

1995 - 1996 **Psychiatric Treatment Specialist**

North Florida Evaluation and Treatment Center, Gainesville, FL  
Supervised and provided milieu therapy for men adjudicated Not Guilty by Reason of Insanity or Incompetent to Proceed at a maximum security forensic treatment center

**Research Experience**

2002 - present

**Co-Principle Investigator**

*Program Assessment of the St. Francis of Assisi Prison PUP Program*

- Design, implement, and analyze data in a longitudinal investigation of the effects of a service dog training program on inmates at a Level II state correctional facility in Bland, Virginia
- Project funded by a grant from the Human-Animal Bond Foundation, Virginia-Maryland Regional College of Veterinary Medicine

**Co-Principle Investigator**

*Animal-Assisted Occupational Therapy with Autistic Children*

- Designing investigation of the effectiveness of animal-assisted occupational therapy with autistic children at Lewis-Gale Medical Center
- Analyzing results from pilot study, and preparing manuscript for publication

2002 - 2003

**Laboratory Coordinator**

Emotion Regulation Laboratory  
Department of Psychology, Virginia Tech, Blacksburg, VA

- Managed research laboratory of both graduate and undergraduate research assistants
- Engaged in research design, conducted data collection and analysis, and prepared conference presentations and manuscripts for a study examining psychophysiological and emotional correlates of proactive and reactive aggression in children

2000 - 2002     **Research Assistant**

Laboratory for the Study of Aggression and Psychological Trauma  
Department of Psychology, Virginia Tech, Blacksburg, VA

Conducted data collection and analysis, adapted questionnaires, and prepared conference presentations and manuscript for a study examining the effects of exposure to community violence in a low-risk young adult population

**Military Service**

March, 2004     Honorable Discharge, United States Army Reserve

1999 - 2004     E-4, Inactive Ready Reserve, United States Army

1996 - 1999     Heavy Wheeled Vehicle Mechanic  
United States Army Reserve, Irvine, CA & Kenova, WV

1996             Distinguished Honor Graduate, Class 63S, U.S. Military Heavy Wheeled Vehicle  
Mechanic Training School, Ft. Jackson, SC

**Selected Papers and Presentations**

Publications

*Peer Reviewed Journals:*

Van Voorhees, E., & Scarpa, A. (in press). The effects of child maltreatment on the hypothalamic-pituitary-adrenal axis. Trauma, Violence, and Abuse: A Review Journal.

Scarpa, A., Fikretoglu, D., Bowser, F., Hurley, J., Pappert, C., Romero, N., & Van Voorhees, E. (2002). Community violence exposure in university students: A replication and extension. Journal of Interpersonal Violence, 17(3), 253-272.

*Other Publications:*

Van Voorhees, E. (Summer, 2003). Dogs in jail: The animal-human bond at the Bland Correctional Facility. The Newsletter of the American Association of Human-Animal Bond Veterinarians, 9, p. 6.

Van Voorhees, E., & Scarpa, A. (2002). Psychophysiological variables in childhood proactive and reactive aggression. Psychophysiology, 39 (Supplement 1), p. S82.

Van Voorhees, E. (July/August, 2001). Individual Differences in Memory and Suggestibility: Factors Influencing the Accuracy of Remembering. American Psychological Society Observer, 14 (6).

Presentations:

Fournier, A. K., Van Voorhees, E., & Budowle, R. E. (May 2004). When the Going Gets “Ruff”: Pitfalls of Applied Research on the Human-Animal Bond. Paper to be presented at the 30th annual meeting of the Association for Behavior Analysis, Boston, MA.

Van Voorhees, E., Fournier, A.K., & Suthers-McCabe, H.M. (April 2004). The Prison PUP Service Dog Training Program at the Bland Correctional Unit: Inmate Trainer Outcomes. Paper presented at the 2004 meeting of the Eastern Psychological Association, Washington, D.C.

Suthers-McCabe, H.M., Van Voorhees, E., & Fornier, A. (August 2003). Program assessment of the St. Francis of Assisi prison PUP program. Paper presented at the Annual Convention of International Society of Anthrozoology at Kent State University in Canton, OH, and at the Annual Convention of the American Association of Veterinary Medicine in Denver, CO.

Shumate, H., Van Voorhees, E., & Scarpa, A. (May, 2003). Skin conductance, executive cognitive functioning, and reactive aggression in children. Poster presented at the 15<sup>th</sup> Annual Conference of the American Psychological Society, Atlanta, GA.

Van Voorhees, E., Kovacs, S., Phillips, L., Jarvis, E., & Scarpa, A. (May, 2003). Heart rate and child abuse potential in childhood reactive and proactive aggression. Poster presented at the 15<sup>th</sup> Annual Conference of the American Psychological Society, Atlanta, GA.

Van Voorhees, E., & Scarpa, A. (October, 2002). Psychophysiological variables in childhood proactive and reactive aggression. Poster presented at the 42<sup>nd</sup> Annual Meeting of the Society for Psychophysiological Research, Washington, D.C.

Van Voorhees, E., Shumate, H., Johnson, J., Travers, J., & Scarpa, A. (June, 2002). Physiological and family factors influencing reactive and proactive aggression in children. Paper presented at the 14<sup>th</sup> Annual Conference of the American Psychological Society, New Orleans, Louisiana.

Francisco, J., Van Voorhees, E., Bowser, F., Travers, J., & Scarpa, A. (June, 2002). The relationships of anxiety and depression to reactive and proactive aggression. Poster presented at the 14<sup>th</sup> Annual Conference of the American Psychological Society, New Orleans, Louisiana.

Fikretoglu, D., Hurley, J., Van Voorhees, E., & Scarpa, A. (December, 2001). Community violence exposure: Psychological and physiological outcomes. Poster presented at the 17<sup>th</sup> Annual Meeting of the International Society for Traumatic Stress Studies, New Orleans, Louisiana.

Van Voorhees, E., & Scarpa, A. (June, 2001). Exposure to trauma and coping in university students. Poster presented at 13<sup>th</sup> Annual Convention of the American Psychological Society in Toronto, Ontario, Canada.

Van Voorhees, E., & Scarpa, A. (June, 2001). Aggression and exposure to interpersonal abuse: A preliminary investigation. Poster presented at 13<sup>th</sup> Annual Convention of the American Psychological Society in Toronto, Ontario, Canada.

**Community Activities and Additional Training**

Founder and Executive Director of PetSafe of the New River Valley, a non-profit affiliated agency providing temporary safe shelter for the companion animals of women and children living in domestic violence shelters (2003-present).

Big Sister through Big Brothers/Big Sisters of the New River Valley (2000-present).

Mediation Training (October, 2003), Conflict Resolution Center, Roanoke, Virginia.

**Professional Membership**

Student member/affiliate:

American Psychological Association

American Psychological Society

Association for Behavior Analysis

American Society of Clinical Hypnosis

Society for Psychophysiological Research

References furnished upon request.