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The Association between Trauma Exposure, Maladjustment, and Aggression in Detained Boys

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The Association between Trauma Exposure, Maladjustment, and Aggression in Detained Boys

A Thesis

Submitted to the Graduate Faculty of the University of New Orleans in partial fulfillment of the requirements for the degree of Master of Science in Psychology

by

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Abstract

Previous research has demonstrated an association between violence exposure and aggression; however, research exploring the association between violence exposure and the forms and functions of aggression is scarce. The aim of this study was to explore the associations between trauma exposure with both reactive and proactive functions of aggression by examining two potential mediators (e.g., psychopathic traits and emotional dysregulation). Participants included 132 male juvenile offenders mandated to treatment in a residential facility ($M = 16.78$ years old; $SD = 1.25$). Results indicate emotional dysregulation partially mediated the association between trauma exposure and reactive aggression while controlling for proactive aggression. However, no evidence was found to support the hypothesis that psychopathic traits mediated the association between trauma exposure and proactive aggression. Results suggest trauma exposure is important in the development of reactive aggression. Thus, treatment approaches for aggressive youth should address issues of trauma exposure.

Reactive Aggression, Proactive Aggression, Trauma Exposure, Detained Youth, Juvenile Delinquency, Emotional Dysregulation, Psychopathic Traits
**Introduction**

Research on human aggression has evolved into an increasingly complex and detailed field of study over the past several decades. It has not been exempt from many of the same major philosophical debates facing the field of psychology in general such as the nature-nurture debate and the increasing interest in developmental psychology. The first comprehensive theory regarding the development of aggression was the Frustration-Aggression model (Dollard, Doob, Miller, Mowrer, & Sears, 1939). Like many theories of psychopathology at this time, it was rooted in the historical perspective that aggression was inherent and innate in human beings. Dollard and his colleagues (1939) borrowed from the popular psychoanalytic philosophy of Sigmund Freud, positing that frustration occurs when pleasure seeking and/or pain avoidance are thwarted whereby aggression is the direct result of human frustration. However, research began to emerge challenging the basic premise of the theory that frustration inevitably precedes aggression (Buss, 1963; Cohen, 1955; Pastore, 1952).

This newly emerging research emphasized the importance of environmental influences on the development of aggression rather than the inherent features intrinsic to all human beings. Again these theories were not exempt from the popular philosophy of the time, in this case social learning theory (Bandura, 1973). It was hypothesized that aggression was learned and maintained vicariously through a series of constantly re-occurring environmental experiences such as cues, responses, rewards, and punishments. According to this model, aggression is learned as a result of reinforcement and unlearned as a result of punishment; again, research emerged that did not fully support this
hypothesis. Eron and colleagues (1971) found evidence that the children who had been punished the most for their aggressive actions were actually the most aggressive.

Researchers began to incorporate evidence from both the frustration-aggression and social learning models leading to the development of social cognitive models. The Social Information Processing model (Crick & Dodge, 1994; Dodge, 1986) recognizes critical socialization periods for the formation and use of cognitive scripts supporting acts of aggression. This model suggests that all social situations are filtered through a sophisticated set of social cognitions governed by biologically limited capabilities and past experiences. Children receive information about social situations and decide upon behavioral responses through a series of time-related parallel processes. The model starts with encoding and interpretation of external and internal cues (Steps 1 and 2), followed by goal formation and clarification (Step 3), and ending with response construction and behavioral enactment (Steps 4, 5, and 6).

It seems as if the environments most favorable for developing aggressive cognitive scripts are those in which aggression is easily observed and typically reinforced as well as those in which the child is the victim of aggression (Eron, 1994). These environments share many of the same features as those environments with a high risk of trauma exposure such as homes with negligent or coercive parenting, parental psychopathology, substance abuse, community violence, crime, and other experiences of poverty (Greenwald, 2002). Social information processing theory would theorize that those individuals living in environments with a high-risk of trauma exposure may be operating in extended periods of post-traumatic survival mode characterized by states of heightened awareness or alertness leading one to misinterpret minor and/or neutral stimuli as threatening, thereby
increasing the likelihood of reacting aggressively (Greenwald, 2002). This in turn typically elicits an aggressive response from the environment which reinforces the belief that the environment is dangerous and threatening (Dodge, 2006).

On the surface defining aggression may appear to be an easy task as most people can list a number of actions that they consider to be aggressive; however, aggression researchers have often struggled to definitively and adequately define their construct of interest (Tremblay, 2000 for a review). Aggression has been conceptualized and defined in very broad terms as any action intended to hurt or harm (Berkowitz, 1993; Coie & Dodge, 1998). However, recent literature indicates that aggression is best understood and measured as a multifaceted construct consisting of several subtypes based on the forms and functions of the aggressive action (Little, Jones, Henrich, & Hawley, 2003; Marsee et al., 2011; Ostrov & Crick, 2007). The forms of aggression refer to the method by which the harm is inflicted and are generally classified as either overt or relational (see Archer, 2004; Card, Stucky, Sawalani, & Little, 2008 for a review). The functions of aggression refer to the reason or purpose for which the harm is inflicted and are generally classified as either reactive or proactive (see Card & Little, 2006 for a review).

The forms of aggression have gone by many different names in the literature, with overt aggression sometimes called direct aggression, physical aggression, and even verbal aggression. However, many researchers use the term overt to refer to this form of aggression because it includes both physical and verbal acts that are intended to harm, damage, or threaten the physical well-being of a victim such as hitting, kicking, pushing, insulting or threatening bodily harm (Little et al., 2003). Overt aggression is a more direct
and “in your face” form of aggression (Little et al., 2003) making it easier to view and thus easier to study.

The relational form of aggression likewise has gone by many different names in the literature such as indirect aggression, social aggression, and covert aggression (e.g., Card et al., 2008). While the terminology varies, generally speaking these terms refer to acts intended to harm or threaten to harm an individual by damaging their social relationships, and may take the form gossiping, rumor spreading, social exclusion, or ostracism. Due to the covert nature of some acts of relational aggression, it is often difficult to directly observe, measure, and study (Crick & Grotpeter, 1995). Additionally, relational aggression often goes unpunished despite the fact that children and adolescents report that it is just as damaging as overt aggression (Crick, Bigbee, & Howes, 1996). Relational aggression is often thought of colloquially as the female form of aggression; however, meta-analyses have yielded only negligible gender differences (Archer, 2004; Card et al., 2008). These studies suggest that boys tend to prefer overt forms of aggression, but do still show similar levels of relational aggression as girls.

The reactive function of aggression stems from the frustration-aggression model (Dollard et al, 1939) and generally occurs as an angry response to provocation, threat, or goal blocking. In contrast, the proactive function of aggression stems from social learning theory (Bandura, 1973) and generally occurs as an unprovoked, premeditated action with a self-serving purpose such gain or dominance. Proactive aggression is often used to achieve desired goals and it is often learned and reinforced through this successful goal achievement process.
Despite a rich history of research demonstrating the differential nature of psychosocial adjustment as well as factor analytic studies indicating the statistical distinctiveness of the forms and functions of aggression, a cursory review of any aggression literature reveals a high intercorrelation among the subtypes of aggression (Crick, 1996; Crick & Grotpeter, 1995; Little et al., 2003; Marsee et al., 2011). Recent research suggests that this high intercorrelation is partly a function of the method of assessment, where observational methods yield lower intercorrelations than teacher, parent, and/or self-report measures; however, observational methods are innately more difficult to implement as they are more costly and time consuming (Card et al., 2008; Card & Little, 2006). To this end, much attention has been paid in recent literature to improve upon self-report measures of aggression (Little et al., 2003; Marsee et al., 2011; Ostrov & Houston, 2008).

Generally the forms and functions of aggression have been measured and studied in isolation, where the focus was either the forms of aggression alone or the functions alone. Attempts to integrate both the forms and functions together in a single self-report measure have recently been conducted and validated (Little et al., 2003; Marsee et al., 2011; Ostrov & Houston, 2008). Particularly, Marsee and colleagues (2011) used confirmatory factor analysis to validate a four factor model incorporating forms and functions together for both boys and girls in three different juvenile populations (a detained sample, a high school community sample, and a residential treatment sample). Results indicate assessing all four domains simultaneously paints a clearer picture of exactly how and why youth use aggression. Additionally, all four distinct forms and functions of aggression were differentially associated with variables of psychosocial adjustment, further illustrating the importance of measuring forms and functions together.
Numerous studies have shown that the forms and functions are differentially correlated with variables of psychosocial adjustment (Marsee & Frick, 2010). Reactive aggression has repeatedly been shown to be associated with internalizing symptoms, peer rejection, victimization, and emotional and behavioral dysregulation such as impulsivity and other ADHD symptoms (see Card & Little, 2006 for a review). Further, reactive aggression has been shown to be uniquely associated with low frustration tolerance and hostile attribution bias, leading individuals high in reactive aggression to misinterpret social cues as hostile and impulsively respond to the provocation with aggression (Crick & Dodge, 1996; Dodge, Coie, & Lynam, 2006; Munoz et al., 2008; Phillips & Lochman, 2003). This pattern of impulsive and combative behavior appears to draw more attention from law enforcement as both reactive subtypes (i.e., reactive relational and reactive overt) have been shown to be associated with higher rates of self-reported arrest history after controlling for both proactive subtypes (Marsee et al., 2011).

Exposure to traumatic events is one key factor that is often studied in association with emotional and behavioral dysregulation and aggression. Exposure to potentially traumatic stressors is a relatively normative shared experience for both children and adolescents; however, the rate of trauma exposure appears to be alarmingly high for incarcerated youth (Arroyo, 2001; Ford, Hartman, Hawke, & Chapman, 2008). Furthermore, research indicates that detained youth are at risk for exposure to complex trauma, a category of traumatic stressors in which the victim suffers not only a traumatic shock but also a disruption in self-regulatory abilities and/or attachment bonds (see Ford, Chapman, Connor, & Cruise, 2012 for a review). An overwhelming majority of incarcerated youth (90%) report exposure to at least one potentially traumatic event in their lifetime,
ranging from physical, sexual, and mental abuse to various forms of violence exposure (Abram et al., 2004; Ford, Hartman, Hawke, & Chapman, 2008). Ford and colleagues (2010) used hierarchical cluster analysis to detect two complex trauma subgroups in a large sample of detained youth. Results indicated that 20% of the sample comprised a group of youth with a combination of exposure to sexual or physical abuse and family violence while another 15% of the sample comprised a group of youth who had been exposed to emotional abuse and family violence. The authors suggest this combined prevalence of 35% of the sample reporting complex trauma was well above the average rate of complex trauma exposure among the general population of children and adolescents (10-13%; Finkelhor, Ormrod, & Turner, 2009; Ford, Elhai, Connor, & Frueh, 2010).

Trauma exposure has been linked to a host of negative internalizing and externalizing consequences for youth, including anxiety, depression, PTSD, and other behavioral problems such as aggression (Fantuzzo & Mohr, 1999; Kliewer, Lepore, Oskin, & Johnson, 1998; Singer, Anglin, Song, & Lunghofer, 1995). In a recent review of the literature on community violence exposure and mental health symptoms, McDonald and Richmond (2008) found strong associations between aggression and PTSD symptoms related to community violence exposure. Additionally, trauma exposure has been shown to be associated with a number of cognitive malfunctions implicated in social information processing such as diminished arousal reactions, episodic maladaptive hyperarousal, impaired information processing, impulse control, aggressive schemas, and delinquent behavior (Ford et al., 2012).
Trauma-exposed youth exhibit higher rates of reactive aggression as well as symptoms indicative of reactive aggression such as impulsivity and emotional dysregulation in the form of an inability to suppress anger (Connor, Doerfler, Volungis, Steingard, & Melloni, 2003; Shields & Cicchetti, 1998). Marsee (2008) found an association between trauma exposure and reactive aggression even after controlling for proactive aggression in a sample of trauma-exposed youth affected by Hurricane Katrina. These results suggest differential risk factors and potentially differential developmental pathways for reactive and proactive aggression subtypes.

Both reactive and proactive aggression have been shown to be associated with delinquency, criminality, and general antisocial behavior; however, proactive aggression is associated with more severe forms of antisocial behavior (see Frick & Dickens, 2006 for a review). In contrast to reactive aggression, proactive subtypes are associated with lower rates of victimization and reduced emotional responsiveness to negative stimuli (Card & Little, 2006; Frick et al., 2003; Hubbard et al., 2002). The most differentiating feature of proactive aggression is its association with callous and unemotional (CU) traits even after controlling for reactive subtypes (Crapanzano, Frick, & Terranova, 2010; Marsee & Frick, 2010; Marsee et al., 2011; Ostrov & Houston, 2008).

Callous and unemotional traits are viewed as a key feature of psychopathy, a psychological construct with a characteristic constellation of interpersonal, affective, and behavioral/antisocial features (Cleckley, 1976; Hare, 1998). Psychopathy is generally thought to be a stable personality disorder with presenting factors early in childhood (Frick, Kimonis, Dandreaux, and Farrell, 2003). There is evidence that children and adolescents high in psychopathic features such as CU traits also demonstrate
characteristically low emotional reactivity and fearfulness as well as a lack of response to punishment (Kimonis, Frick, Fazekas, & Loney, 2006; Loney, Frick, Clements, Ellis, & Kerlin, 2003; Vitale, Newman, Bates, Goodnight, Dodge, & Petit, 2005). Additionally, this unique subgroup has also exhibited a preference for thrill seeking behavior and dangerous activities (Frick, Lillienfeld, Ellis, Loney, & Silverthorn, 1999).

With regard to the forms of aggression, overt aggression is by far the most well-studied and it is generally associated with severe psychosocial outcomes and delinquency (Coie, Dodge, & Kupersmidt, 1990; Prinstein, Boergers, & Vernberg, 2001). More specifically, it is strongly and uniquely associated with externalizing problems, low prosocial behavior, and low peer acceptance (see Card et al., 2008 for a review). In contrast, relational forms of aggression are strongly and uniquely associated with internalizing problems and prosocial behaviors (see Card et al., 2008 for a review). Research suggests compared to overt aggression, relational aggression may not warrant immediate attention from authority figures and thus highly relationally aggressive children are rarely directed to treatment or intervention programs (Crapanzano et al., 2010).

Taken together, research indicates that the reasons why youth aggress against others (i.e., the functions of aggression) may have unique emotional and behavioral correlates. Reactive aggressive subtypes show stronger associations with emotional and behavioral dysregulation while proactive aggressive subtypes show stronger associations with blunted emotional responsivity and psychopathic traits. Furthermore, the ways that youth choose to use aggression (i.e., the forms of aggression) have shown key differential associations with psychosocial adjustment with overt aggression showing strong
associations with externalizing behaviors and peer rejection and relational aggression showing stronger associations with internalizing symptoms.

Any adequate developmental theory of aggression must address the differential correlates of psychosocial adjustment between the forms and functions of aggression. Results suggest that reactive and proactive aggression are associated with differential correlates of adjustment (i.e., that reactive aggression is associated with emotional dysregulation while proactive aggression is associated with higher levels of psychopathic traits and positive outcome expectations). Youth who have been exposed to trauma use both proactive and reactive functions of aggression. It may be that different types of violence exposure and/or differences in severity of violence exposure may lead some individuals to respond more reactively to provocation while in others it may lead to an emotional numbing or callousness, which may be more associated with proactive aggression. Proactive aggression is often implemented by individuals with a goal in mind; it may be the case that repeated exposure to violence increases the likelihood that an individual will view aggression as an appropriate means of accomplishing goals. Moreover, due to the exponentially high rate of trauma exposure among incarcerated youth it is vitally important that we understand the developmental pathways of both proactive and reactive aggression and trauma exposure together. A clearer picture of how and why youth use aggression leads to better treatment and intervention opportunities.

With this in mind, the purpose of this study is to explore the associations between trauma exposure and both reactive and proactive aggression by examining potential mediators (e.g., psychopathic traits and emotional dysregulation). Additionally, much of the research to date concerning the associations between psychopathic traits and proactive
aggression has been performed with self-reported measures of CU traits. The current study will further investigate this association with a measure of psychopathy utilizing a semi-structured interview format. Therefore, this study proposes the following hypotheses:

1. Psychopathic traits will mediate the association between trauma exposure and proactive aggression.
   a. These associations are expected to remain significant after controlling for reactive aggression.

2. Emotional dysregulation will mediate the association between trauma exposure and reactive aggression.
   a. These associations are expected to remain significant after controlling for proactive aggression.
Method

Participants

Participants were selected from a group of male juvenile offenders mandated by the courts to participate in a treatment program at an Alabama Department of Youth Services residential facility. Parental consent was not required because all students at the facility are under state custody. Table 1 displays demographic information for the sample. The final sample consisted of 132 male juvenile offenders (54.5% African American, 40.9% Caucasian, 4.6% other). Participants ranged in age from 12 to 19 years old ($M = 16.78; SD = 1.25$). The mean grade level was seventh grade. Participants had a mean number of arrests of 6.73 ($SD = 6.01$) and a range of committing offenses (12% violation of probation or aftercare, 28% sexually based charges, 35% property charges, and 7% drug charges).

Procedures

All juvenile offenders were given a full psychological evaluation upon admission to and prior to release from the facility as a requirement of the treatment program. Graduate students in a clinical psychology doctoral program completed evaluations. Evaluations included intellectual and achievement measures, clinical diagnostic interviews, and a series of self-report measures intended to assess personality dimensions pertinent for treatment recommendations and dormitory placement at the facility. While completion of the psychological evaluation is a court mandated treatment requirement, the students are allowed to choose if they would like their information to be used for research purposes. Both the Auburn University and University of Alabama Institutional Review Boards have approved the evaluation process, data collection and data storage.
Measures

Trauma exposure. The Screen for Adolescent Violence Exposure (SAVE; Hastings & Kelley, 1997) was used to measure trauma exposure. The SAVE is a 32-item self-report measure of violence exposure in three different settings (i.e., home, school, and neighborhood). Each setting can also be subdivided according to three factors: traumatic violence, indirect violence, and interpersonal aggression. Items are scored on a 5-point Likert-type scale from 0 (never experienced) to 4 (very frequently experiences). All subscales were combined to produce an overall violence exposure rating in this study. The SAVE has demonstrated good internal consistency with alphas of .96 in adolescent samples (Self-Brown, LeBlanc, & Kelley, 2004). Cronbach’s alpha for the current sample (.97) is in line with previous research.

Aggression. The Peer Conflict Scale was used to measure participant’s self-report of aggression (PCS; Marsee et al., 2011). The PCS is a 40-item measure operationalizing the forms and functions of aggression via four domains: reactive overt, proactive overt, reactive relational, and proactive relational. Ten distinctive items load onto each of the four domains: reactive overt (e.g., “When someone hurts me, I end up getting into a fight”), proactive overt (e.g., “I start fights to get what I want”), reactive relational (e.g., “If others make me mad, I tell their secrets”), and proactive relational (e.g., “I gossip about others to become popular”). Items are measured on a 4-point Likert-type scale, from 0 (not at all true) to 3 (definitely true). Since our research question is more concerned with the functions instead of the forms, the proactive overt and proactive relational scales were combined to create a total proactive aggression scale and the reactive overt and reactive relational scales were combined to create a total reactive aggression scale for this study.
The coefficient alphas from a combined sample of adolescents from school, residential, and detained settings range from .79 to .89 in previous studies (Marsee et al., 2011). The coefficient alphas for the current study are good with a Cronbach’s alpha of .96 on the total aggression scale for all 40 items. Alphas for the total proactive aggression scale (20 items) and the total reactive aggression scale (20 items) are .96 and .90 respectively. Previous research has also demonstrated good convergent validity of the four aggression domains with internalizing and externalizing symptoms (Stimmel, Cruise, Ford, & Weiss, 2013; Munoz, Frick, Kimonis, & Aucoin, 2008; Marsee & Frick, 2007).

*Emotional dysregulation.* The Borderline Tendency Scale from the Millon Adolescent Clinical Inventory (MACI; Millon, 1993) was used to measure emotional dysregulation. The MACI is a 160-item, 31-scale self-report inventory used to assess personality styles, significant problems or concerns, and clinical symptoms in adolescents, including emotional dysregulation, sexual discomfort, substance abuse proneness, suicidal tendency, and eating dysfunctions. Items are rated using a true/false format. Both raw and standardized base rate scores were calculated using computer software from NCS Assessments, the publisher of the MACI. The Borderline Tendency Scale measures personality features marked by disturbances in perception of self, relationship to others, and regulation of affect such as intense mood fluctuations and tumultuous personal relationships. Criterion validity has been supported in studies indicating high correlation with base rate scores of the Borderline Tendency Scale and Social Skills subscale of the POSIT (0.63) and Ineffectiveness (0.60), Interoceptive Awareness (0.55), and Impulse Regulation (0.62) subscales of the EDI-2 (McCann, 1999). Elevated BR scores on this scale typically represent adolescents who are experiencing significant emotional turmoil and
instability indicated by shifting periods of anxiety, anger, depression, happiness, and irritability (McCann, 1999). Alphas at .92 for the entire MACI scale in the current sample are good.

*Psychopathic traits.* Adolescent psychopathic traits were measured using the Hare Psychopathy Checklist: Youth Version (PCL: YV; Forth, Kosson, & Hare, 2003). The PCL:YV is a downward extension of the most widely used measure of psychopathy in adults, the Hare Psychopathy Checklist - Revised (PCL-R; Hare, 1991, 2003). The PCL:YV measures the same constellation of features as the PCL-R (e.g., interpersonal, affective, and behavioral/antisocial features) and maintains the same expert-rater and multiple source format. The standard assessment procedure involves a review of collateral information (such as police reports, school records, court documents, and/or previous psychological assessments) and the administration of a semi-structured interview. The 20-item clinical rating scale assesses psychopathic traits in 12- to 18-year-old male and female adolescents.

Items are rated on a 3-point ordinal scale (0, 1, or 2) based on the assessment of the adolescent’s functioning and how well that assessment matches the behaviors and personality traits representing the item of concern. Rating items requires strict standardization and training as well as the use of considerable clinical judgment. According to the technical manual (Forth, Kosson, & Hare, 2003), raters must possess an advanced degree in the social, medical, or behavioral sciences and have the appropriate professional credentials (e.g., licensure to legally conduct psychological assessment according to state regulations or supervision by a licensed professional) as well as experience working with adolescents. They must also be familiar with the most current literature on psychopathy in both adults and adolescents and adequate training in the
standard administration procedures of the PCL:YV. Graduate students conducting the interviews were working under supervision of a licensed clinical psychologist in the State of Alabama. Additionally, graduate students underwent 2 weeks of training in the PCL:YV during which they were required to observe an experienced rater conduct the interview and score the items for one week. This was followed by a week of supervision in which the graduate student conducted interviews and rated items under the supervision of the experienced rater. Weekly meetings with the treatment team were held in which students were given feedback on their item ratings from the treatment team leader. Studies have shown the interrater reliability for the measure to be excellent with intraclass correlation coefficients ranging from .90 to .96 (Forth, Kosson, & Hare, 2003). PCL:YV total scores have shown to be correlated with elevations in substance abuse, ADHD, narcissism, mania, and conduct problems (Forth, Kosson, & Hare, 2003).

**Full Scale IQ.** Intelligence was assessed using the Wechsler Abbreviated Scale of Intelligence (WASI; Weschsler, 1999), an individually administered intelligence test for ages 6 to 90. WASI scores are nationally standardized and yield three traditional Verbal, Performance, and Full Scale IQ scores that are linked to the Wechsler Intelligence Scale for Children-Fourth Edition (WISC-IV) and the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III). The WASI consists of four subtests: Vocabulary, Similarities, Block Design, and Matrix Reasoning. The Verbal IQ score is a measure of crystallized abilities of verbal reasoning and concept formation and is derived from the Vocabulary and Similarities subtests. The Performance IQ score is derived from the Matrix Reasoning and Block Design subtests and is a measure of visual processing and abstract reasoning skills.
Results

Initial correlational analyses were conducted to test if the main study variables met the requirements for meditational analysis. Table 2 displays correlations, means, and standard deviations of the main study variables. All main study variables (i.e., reactive aggression, proactive aggression, total violence exposure, total psychopathic traits, and emotional dysregulation) showed significant correlations with one another indicating that assumptions were met for meditational analysis. Additionally, correlations between the nine subtypes of violence exposure (e.g., traumatic/indirect/interpersonal school, home, and neighborhood violence exposure) and the subtypes of aggression were conducted to examine differential associations. All subscales were significantly and positively associated with proactive and reactive aggression with Pearson correlations ranging from .20 to .55, demonstrating there was no differential association among the subscales of the SAVE and proactive and reactive aggression in this sample. Age in years approached a significant correlation with emotional dysregulation ($r = -.16, p = .06$). Race (coded 0=Caucasian, 1=non-Caucasian) was positively correlated with violence exposure ($r = .30, p < .001$) and psychopathic traits ($r = .33, p < .001$). Full scale IQ scores on the WASI (Weschsler, 1999) were significantly correlated with violence exposure ($r = -.19, p < .05$) and psychopathic traits ($r = -.20, p < .05$). Age in years, race, and full scale IQ were added as covariates based on these significant correlations.

Previous research has demonstrated a considerable amount of multicollinearity between the forms and functions of aggression (Marsee et al. 2011). Partial correlation analyses were conducted to explore the overlap shared between reactive and proactive aggression. Table 2 displays the results of the partial correlation analyses between reactive
and proactive aggression and the main study variables. Results indicate that when controlling for proactive aggression, the associations between the main study variables and reactive aggression remain significantly correlated ($r$s ranging from .24 to .38). However, the main study variables are no longer associated with proactive aggression after controlling for reactive aggression (see Table 2). Data from these preliminary analyses appear to indicate the association between trauma exposure and aggression is driven primarily by the associations with reactive aggression.

A series of regression analyses were conducted to examine whether psychopathic traits mediated the association between trauma exposure and proactive aggression. Based on a model for meditational analyses developed by Baron and Kenny (1986), four conditions must be met for a variable to be considered a mediator. The first condition is that the predictor, trauma exposure, be associated with the outcome variable, proactive aggression. The second condition is that the predictor, trauma exposure, be associated with the mediator variable, psychopathic traits. The third condition is that the mediator variable, psychopathic traits, be associated with the outcome variable, proactive aggression. Condition four determines mediation status if the predictor, trauma exposure, is no longer associated with the outcome variable, proactive aggression, after controlling for the mediator variable, psychopathic traits. Figure 1 demonstrates that the first three conditions were met as indicated by the significant beta weights. However, the fourth condition was not satisfied because trauma exposure remained a significant predictor of proactive aggression after controlling for psychopathic traits ($\beta = .430, p < .001$). Due to the relative reduction in the standardized coefficient (from .483 to .430) the Sobel test was conducted to test for partial mediation (Holmbeck, 2002). The Sobel test was not
significant ($z = 1.38, p = .17$) indicating that the mediator did not carry a significant portion of the effect. Given these non-significant results, hypothesis 1A (i.e., that the association between trauma exposure and proactive aggression are expected to remain significant after controlling for reactive aggression) was not tested.

A similar series of regression analyses were conducted to examine whether emotional dysregulation mediated the association between trauma exposure and reactive aggression. Figure 2 demonstrates that the first three conditions were met as indicated by the significant beta weights. The fourth condition was not satisfied because trauma exposure was still a significant predictor of reactive aggression while controlling for emotional dysregulation ($\beta = .507, p < .001$). Again, a Sobel test was conducted based on the relatively large reduction in the standardized coefficients (from .614 to .507). The Sobel test was significant for this model ($z = 3.03, p < .01$) indicating that emotional dysregulation partially mediated the association between trauma exposure and reactive aggression by carrying a significant portion of the effect. A final regression analysis was conducted to test the hypothesis that emotional dysregulation would mediate the association between trauma exposure and reactive aggression after controlling for proactive aggression. Total reactive aggression was entered as the dependent variable and trauma exposure, emotional dysregulation, and total proactive aggression were entered as the independent variables. Age, race, and IQ were included as covariates. Results indicate that emotional dysregulation partially mediated the association between trauma exposure and reactive aggression even after controlling for total proactive aggression ($\beta = .243, p < .001$).
Additionally, due to the overlap between reactive and proactive aggression a series of regression analyses were conducted to further investigate the associations between the main study variables and the functions of aggression. Table 3 represents a summary of the hierarchical linear regression analysis used to investigate the associations between the main study variables and proactive aggression while controlling for reactive aggression. In the first analysis, age, race, IQ, trauma exposure, psychopathic traits, and emotional regulation accounted for a significant amount of variance, $R^2 = .29$ (i.e., 29% of the variance in proactive aggression). Results indicate that age was negatively associated with proactive aggression ($\beta = -.17$, $p < .05$) and trauma exposure was positively associated with proactive aggression ($\beta = .38$, $p < .001$). A second analysis was conducted to evaluate if these associations remained after controlling for reactive aggression. As seen in Table 3, the associations between trauma exposure and proactive are no longer significant after controlling for reactive aggression ($\beta = .01$, $p = .86$). Results indicate that trauma exposure is no longer associated with proactive aggression after controlling for reactive aggression. There was no evidence for a differential association between trauma exposure and the functions of aggression. Rather, it appears that the association between trauma exposure and aggression is driven primarily by the associations with reactive aggression.

Table 4 reports a summary of the hierarchical linear regression analysis used to investigate the associations between the main study variables and reactive aggression while controlling for proactive aggression. In the first analysis, age, race, IQ, trauma exposure, psychopathic traits, and emotional regulation accounted for a significant amount of variance, $R^2 = .46$ (i.e., 46% of the variance in reactive aggression). Results indicate that age was negatively associated with proactive aggression ($\beta = -.15$, $p < .05$). Additionally,
trauma exposure and emotional regulation was positively associated with proactive aggression ($\beta = .46, p < .001$ and $\beta = .28, p < .001$ respectively). A second analysis was conducted to evaluate if these associations remained after controlling for proactive aggression. As seen in Table 3, the associations between trauma exposure and emotional regulation remain after controlling for proactive aggression ($\beta = .22, p < .001$ and $\beta = .19, p < .01$ respectively).
Discussion

Youth in detention centers are at a higher risk of developing internalizing and externalizing problems associated with violence exposure. While there is a wealth of research exploring the association between trauma exposure and aggression, there is little to no research exploring this association with the forms and functions of aggression. Therefore, the purpose of this study was to explore two differential pathways for trauma exposure with both reactive and proactive functions of aggression by examining two potential mediators. Contrary to expectations that the associations between trauma exposure and reactive and proactive aggression would demonstrate differential mediators, results indicate that trauma exposure and reactive aggression was partially mediated by emotional dysregulation while the associations between trauma exposure and proactive were not mediated by psychopathic traits. While the data does not suggest differential pathways to reactive and proactive aggression, it does suggest that trauma exposure explains some of the variance in the rates of aggression in detained youth. These boys very well may be detained because of the trauma exposure they have experienced in their lives. This trauma then leads to emotional dysregulation and then to reactive aggression. If we recognize the important role that trauma exposure plays in the development of reactive aggression, then we realize that treating the trauma is a key link in treating the emotional dysregulation and thus reducing the aggressive symptoms that often lead to incarceration for many of these boys.

Previous research has demonstrated a link between proactive aggression and psychopathic traits (Crpanzano, Frick, & Terranova, 2010; Frick et al., 2003; Marsee & Frick, 2010; Marsee et al., 2011) as well as a link between psychopathic traits and
emotional numbing (Loney et al., 2003). Similar emotional deficits have been demonstrated in youth exposed to violence and various other types of trauma (Farrell & Bruce, 1997). In contrast to this previous research, results from the current study indicated that the association between trauma exposure and proactive aggression was not mediated by psychopathic traits. Previous research investigating the association between trauma exposure and proactive aggression has used a self-report measure for CU-traits (Frick, 2004). In this study, we investigated if a semi-structured interview format measuring psychopathic traits would predict similar results. While there is conceptual support that these two measurements would be compatible, it may be the case that the PCL:YV captures some of the key features of the construct of CU-traits while not fully capturing the entire construct in the same way that the self-report measure for CU-traits used in past research. Schraft, Kosson, and McBride (2013) reported somewhat contrasting results in a study investigating exposure to violence and psychopathic tendencies when using the PCL:YV rather than the self-report measure of CU-traits. The authors suggest that while the results appear to be contradictory, results still indicated a significant relationship between violence exposure and affective components of psychopathy. Results from the current study likewise point to associations between violence exposure and psychopathic traits ($\beta = .406, p < .001$), but rather failed to demonstrate that this relationship was a significant mediator to proactive aggression. Regardless, results still imply that environmental influences such as violence and trauma exposure are not solely limited to externalizing psychopathology and may contribute to affective deficits as well.
Additionally, some researchers suggest that there may be differential developmental pathways to psychopathic traits making the construct of psychopathy a bit more complex than originally thought. For example, research in adults suggests a unique distinction between primary and secondary psychopathy (Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). While both subtypes would appear callous and still score high on psychopathic measures, the secondary subtype may or may not display specific affective deficits. Kimonis and colleagues (2008) suggest that this secondary subtype may result as an adaptive emotional response to harsh environments. Furthermore, it has also been demonstrated that the link between emotional deficits and CU-traits in children has been stronger for Caucasian individuals and less strong for African American individuals (Kimonis, Frick, Fazekas, et al., 2006). They propose that these differences in psychopathic traits may be explained by social and cultural differences related to living in highly threatening environments experienced by many African American individuals. This may explain the insignificant results in the current study with a 55% African American sample.

It is also possible that the current sample of detained youth may not be fully representative of youth who have been exposed to violence and trauma, at least not at the levels necessary to constitute emotional numbing. While the mean level of violence exposure in the current sample ($M = 84.08; SD = 55.85$) appears to be a somewhat heightened level of violence exposure as seen in previous studies (e.g., Allwood, Bell, & Horan, 2011; Allwood & Bell, 2008; Hastings & Kelley, 1997), the racial differences discussed above may play a more significant role in the results. As suggested by Kimonis and colleagues (2008), the unique environments experienced by many African Americans...
in urban areas may demand more emotional reactivity rather than emotional numbing. It may very well be an adaptive function of the environment to be more reactive.

Consistent with previous research (e.g., Dodge, Coie, & Lynam, 2006; Marsee, 2008; Shields & Cicchetti, 1998), results of the second mediation analysis indicated that the association between trauma exposure and reactive aggression was partially mediated by emotional dysregulation even after controlling for proactive aggression. These findings suggest that youth who have experienced trauma in the form of violence exposure exhibit emotional dysregulation leading to increased levels of reactive aggression. This theoretical model of reactive aggression is in line with the rich history of research supporting the social information processing theory (Crick & Dodge, 1994; Dodge, 1986) in which high rates of trauma exposure are characterized by states of heightened awareness and emotional dysregulation leading to the development of aggressive cognitive scripts (Eron, 1994; Greenwald, 2002). These aggressive cognitive scripts could be responsible for the misinterpretation of social cues increasing the likelihood of reacting aggressively in benign or neutral situations.

Several important limitations should be noted. First, this study was cross-sectional in nature. While the results continue to lend support for differential associations between the forms and functions of aggression, understanding the true nature of the developmental pathways to reactive and proactive aggression would require the use research methods capable of inferring causation, such as an experimental randomized clinical trial that is longitudinal in nature. This would allow for analyses that could speak to the directionality of effects as well as causation. Second, it is also possible that use of all self-report measures in the reactive aggression model may have led to artificially inflated correlations due to
shared method variance and measurement bias. Finally, the sample was an all male
detained adolescent population and thus no conclusions about gender differences can be
drawn. Results of this study should not be generalized to girls or boys of different ages.
Furthermore, it is unclear if the results could be replicable in a community setting and
further research is needed to determine the generalizability of the results to other settings.

These findings have important intervention implications. Given the wealth of
research supporting differential emotional and behavioral adjustment problems associated
with the forms and functions of aggression, every little step made toward better
understanding the differential correlates of the forms and functions of aggression can help
both researchers and clinicians better understand how to appropriately measure, screen,
and treat aggression. A better understanding of the emotional pathways associated with
reactive and proactive aggression can help fuel a better understanding of the etiology of
aggression as well as the treatment of aggression. Better treatment of the differential
forms and functions of aggression before, during, and after juveniles get involved in the
justice system will help reduce the negative consequences experienced by justice-involved
youth.
References


### Demographic Characteristics of the Sample ($n = 132$)

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<tr>
<th>Category</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean Age in Years (SD)</strong></td>
<td>16.78 (1.25)</td>
</tr>
<tr>
<td><strong>Ethnicity %</strong></td>
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</tr>
<tr>
<td>White</td>
<td>40.9</td>
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<tr>
<td>African American</td>
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<tr>
<td>Hispanic</td>
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<td>Biracial</td>
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<tr>
<td>Other</td>
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<tr>
<td><strong>Mean Number of Arrests (SD)</strong></td>
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<td><strong>Mean Grade Level (SD)</strong></td>
<td>7.02 (4.82)</td>
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<td>Property Charges</td>
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<td>Drug Charges</td>
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Table 2

Correlations, Means, and Standard Deviations of the Main Study Variables

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<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>M</th>
<th>SD</th>
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<td></td>
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<td>16.78</td>
<td>1.25</td>
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<td></td>
<td></td>
<td>4.00</td>
<td>8.55</td>
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<td>6. Trauma Exposure</td>
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<td>.30***</td>
<td>-.19*</td>
<td>.57*** (.38***)</td>
<td>.45*** (.000)</td>
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<td>7. PCL Total</td>
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<td>-.20*</td>
<td>.37*** (.24**)</td>
<td>.29** (.003)</td>
<td>.48***</td>
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<td>18.45</td>
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<td>8. Emotional Dysregulation</td>
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<td>.48*** (.38***)</td>
<td>.33*** (-.10)</td>
<td>.29**</td>
<td>.18*</td>
<td>35.41</td>
<td>21.87</td>
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Note. Race was coded 0 = Caucasian, 1 = Non-Caucasian.

* = p < .05, ** = p < .01, *** = p < .001.
**Figure 1**

*Model for Trauma Exposure and Proactive Aggression*

1. \( = .483, p < .001 \)
2. \( = .416, p < .001 \)
3. \( = .309, p = .001 \)
4. \( = .430, p < .001 \)

**Controlling for Psychopathic Traits**

Sobel test results: \( (z = 1.38, p = .17) \)
Figure 2

*Model for Trauma Exposure and Reactive Aggression*

1. \( r = .614, p < .001 \)

2. \( = .381, p < .001 \)

3. \( = .478, p < .001 \)

4. \( = .507, p < .001 \)
   - Controlling for Emotional Dysregulation

Sobel test results: \( z = 3.03, p < .01 \)
Table 3.

*Summary of Regression Analyses Predicting Proactive Aggression*

<table>
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<tr>
<th>Variable</th>
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<th>Model 2</th>
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<td>Trauma Exposure</td>
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<td>Psychopathic Traits</td>
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<td>1.22</td>
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<tr>
<td>Emotional Dysregulation</td>
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<td>1.88</td>
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<tr>
<td>Reactive Aggression</td>
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<tr>
<td>$R^2$ value for Model</td>
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*Note. *$p < .05$, **$p < .01$, ***$p < .001$*
## Table 4.

*Summary of Regression Analyses Predicting Reactive Aggression*

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>t</th>
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<th>β</th>
<th>t</th>
<th>p</th>
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<td>Age</td>
<td>-.15</td>
<td>-2.19</td>
<td>.03*</td>
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<td>Race</td>
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<td>Full Scale IQ</td>
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<td>Trauma Exposure</td>
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<td>.62</td>
<td>11.05</td>
<td>.00***</td>
<td>.52</td>
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</table>

| R² value for Model    | .46 | .73 |

*Note. *p < .05, **p < .01, ***p < .001.*
Complete this form using Adobe Acrobat Writer (versions 5.0 and greater). Hand written forms will not be accepted.

1. Protocol Number: 00-187 MR0008


4. PROJECT TITLE: Accountability Based Juvenile Treatment and Assessment

5. Barry Burkhart  Professor  Psychology  334-844-6476  burkhhbr@auburn.edu

   PRINCIPAL INVESTIGATOR

   PI SIGNATURE

   FACULTY ADVISOR

   SIGNATURE

   DEPT

   MAILING ADDRESS

   AU E-MAIL

   ALTERNATE E-MAIL

   Department of Psychology, Auburn University

   Name of Current Department Head: Dan Svyantek

   AU E-MAIL: svyandj@auburn.edu

6. Current External Funding Agency: Alabama Department of Youth Services

7. List any contractors, sub-contractors, or other entities or IRBs associated with this project:
   Margaret Kelley is PI on an associated project assessing the impact of family therapy with our subjects. She is a sub-contractor through my contract with DYS. Her protocol number is 04-206MR0501
   An additional associated project is the ongoing evaluation of staff training, Protocol number 11-283-AR 1109.

8. Briefly list (numbered or bulleted) the activities that occurred over the past year, particularly those that involved participants.

This is an ongoing project in which pretreatment, post treatment, and follow-up psychological test data and re-arrest rates are collected as the foundation for a comprehensive evaluation of the Accountability Based Sex Offender Program (ABSOP). Beginning in 2012, all adolescent delinquent offenders, not just adolescents with sex offense charges, were included in the standard intake evaluation. Through the end of this reporting period, we have collected data on 1222 participants on the pretreatment protocol. Total number of post treatment evaluations conducted is 778. Beginning this year, we will add approximately 100-250 new participants to the study. The evaluation is a standard part of the intake for all juvenile offenders admitted to the facility.

9. Explain why you are requesting additional time to complete this research project.

This is a multi-year, longitudinal project now in its thirteenth year and is to be continued indefinitely. The evaluation is part of the contract between the Department of Psychology and the Alabama Department of Youth Services.
10. Do you plan to make any changes in your protocol if the renewal request is approved? 
   (e.g., research design, methodology, participant characteristics, authorized number of participants, etc.)

   □ NO  ☑ YES (If "yes", please complete and attach the "REQUEST for PROTOCOL MODIFICATION" form. The IRB will review both requests at the same time.)

11. PARTICIPANT INFORMATION
   a. How many individuals have actually participated in this research? 1222
      If retrospective, how many files or records were accessed?

   b. Were there any adverse events, unexpected difficulties or unexpected benefits with the approved procedures?
      □ NO  ☑ YES (If YES, please explain)

   d. How many participants have withdrawn from the study? 0
      None or Not Applicable.
      NOTE: If any participants withdrew from the study, please explain.

   e. How many new participants do you plan to recruit during the renewal period? 200
      None / NA

   f. During the renewal period, will you re-contact any individual that has already participated in your research project?
      □ NO  ☑ YES  If "YES", please explain reasons for re-contacting participants.
      (If "YES" and the procedure to re-contact has not been previously approved, please complete and attach a "REQUEST for PROTOCOL MODIFICATION" form. The IRB will review both requests at the same time.)

   All juveniles complete a posttreatment evaluation as outlined in original IRB approved protocol.
12. PROTECTION OF DATA

a. Is the data being collected, stored and protected as previously approved by the IRB?

☐ NO (if "NO", explain) ☑ YES

b. Are there any changes in the "key research personnel" that have access to participants or data?

Attach CITI proof of completion for all new key personnel.

☐ NO ☑ YES (If "YES", identify each individual and explain the reason(s) for each change.)

New Graduate research assistants- Additional new graduate research assistants will be Jamie Gauthier, Ian Cero, Kevin Feiszli and Sarah Lyle. 
Melissa Cypraski, Matthew Roth, Danny Lee, Kelly Zuromski, Mark Silvestri, Rebecca Fix, Lisa Simmons, Lacey Kantra and Ashley Norwood will continue as graduate research assistants. 
Patrick Cook, Amber Ritter, Allison Croysdale, Hugo Moralis, Jan Newman, Karlene Brown-Cunningham, Kelli Thompson, and Chastity Farr will continue to be involved in data collection or analyses in 2013-2014.

c. What is the latest date (month and year) you now expect all identifiable data to be destroyed?

(Identifiable data includes videotapes, photographs, code lists, etc.)

DATE: 11/13/2020 ☐ Not Applicable - no identifiable data has been or will be collected.

11. Attach a copy of all "stamped" IRB-approved documents used during the previous year.

(Information letters, Informed Consents, Parental Permissions, etc.).

12. If you plan to recruit participants, or collect human subject data during the renewal period, attach a new copy of the consent document or information letter you will use during the extension.

(Be sure to review the OHSR website for current consent document guidelines and updated contact information:
http://www.auburn.edu/research/irb/hsrsample.htm)

PLEASE NOTE: If you do not plan to collect additional data and/or you do not have access to any identifiable data (including code lists, etc.) you may be able to file a "FINAL REPORT" for this project.
Contact the Office of Human Subjects Research for more information.

When complete, submit hard copy with signatures to the Office of Human Subjects Research, 307 Samford Hall, Auburn University, AL 36849

3 of 3
Guardian Permissioin/Minor Consent for Accountability Based Juvenile Treatment and Assessment

You are invited to take part in a study about how treatment for juvenile offenders works. You were selected as a possible participant because you are a student in residence in the Division of Youth Services and will be completing the assessment phase in the next few days. As part of this process, you will be required to fill out questionnaires and complete tests twice, at the beginning of your placement and at the end of your placement, during your time at Mt. Meigs. These include completing an interview conducted by one of the psychology staff, filling out several questionnaires about your attitudes, beliefs, and behaviors, and taking several standardized psychological tests. Also, your teachers at the Mt. Meigs Wallace School will be asked to complete brief rating forms about how you work in school. The total time to complete this study is from four to six hours. We would like your permission to use this information in a research project. We hope that this research will help us improve the treatment for juvenile offenders.

Although you are required to complete the assessment as part of your placement at DYS, you are not required to allow your data to be used in this research project. If you do, you will be helping us learn how to help juveniles with problems which cause them to get in trouble with the law. If you participate your name will be separated from the data when we do the analysis. All information that you provide will be kept strictly confidential. Other than your case manager and treatment staff, only the researchers will see the information you provide in this assessment.

Remember that you do not have to participate if you do not want to help with this project. Your decision whether or not to participate will not harm your future relations with Mt. Meigs, the Alabama Department of Youth Services, or Auburn University. We plan to present the information we gather as one or more scientific publications and/or presentations.

If you have any questions, please ask them now. If you have questions later, Barry Burkhart, Ph.D. (334-844-6476) or your case manager will be happy to answer them. You will be provided a copy of this form to keep. For more information regarding your rights as a research participant you may contact the Auburn University Office of Human Subjects Research or the Institutional Review Board by phone (334) 844-5966 or email at IRBadmin@auburn.edu.

HAVING READ THE INFORMATION ABOVE, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH PROJECT. YOUR SIGNATURE INDICATES THAT YOU AGREE TO PARTICIPATE. CHECK YES IF AGREE

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226 Thach Hall, Auburn, AL 36849-5214; Telephone: 334-844-4412; Fax: 334-844-4447

www.auburn.edu
VITA

The author was born in Montgomery, Alabama. She obtained her Bachelor's degree in human development and family studies from Auburn University in 2005. She then obtained her Master's of Art in theological studies from Fuller Theological Seminary in 2009. She joined the University of New Orleans psychology graduate program to pursue a PhD in applied developmental psychology and became a member of the Youth Social and Emotional Development Lab under the direction of Professor Monica A. Marsee in 2012.