EXPLORING OXYTOCIN AND CALLOUS-UNEMOTIONAL TRAITS AS MEDIATORS OF THE RELATIONSHIP BETWEEN MALTREATMENT AND AGGRESSION

by

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Recent advances in our understanding of the consequences of childhood maltreatment have offered new insights into the biological and psychological mechanisms that underlie the increased risk for aggression among abused and neglected children; however, the majority of this research has examined reactive (i.e., impulsive) aggression. The processes by which maltreatment increases the risk of proactive aggression are less understood. The present study tested a serial mediation model to explore the potential intermediary roles of oxytocin (OXT) and callous-unemotional (CU) traits in the maltreatment-proactive aggression relationship. Two at-risk samples of young adults (54 probationers/parolees and 47 undergraduate students with histories of adverse childhood experiences) provided saliva samples and completed self-report measures of maltreatment histories, CU traits, and aggression. Separate serial mediation tests of the indirect effects of OXT and CU traits were conducted for each form of maltreatment.

As hypothesized, low salivary OXT predicted elevated CU traits and proactive aggression, but not reactive aggression. Analyses supported the primary hypothesis that low OXT and elevated CU traits sequentially mediated the relationship between total maltreatment and proactive aggression. Although emotional abuse and neglect were expected to exert the strongest effects on OXT, serial mediation was only supported for the model of emotional abuse.

Specifically, severity of emotional abuse predicted lower levels of salivary OXT, which in turn predicted elevated CU traits, and ultimately more proactive aggression.

This study illustrates the role of reduced peripheral levels of OXT in CU traits and proactive aggression that develop in the context of childhood maltreatment. Findings support neurodevelopmental and biosocial theories of psychopathy, which postulate that socio-emotional adversity in early childhood hinders the development of biological systems that are responsible for prosocial emotions. Although the hypothesized serial mediation model was statistically significant, the cross-sectional design precludes our ability to establish causal order. Furthermore, the indirect pathway from maltreatment to proactive aggression through OXT and CU traits only explained a small portion of the total variance in proactive aggression, and should be interpreted as one of several mechanisms that lead to proactive aggression.

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CHAPTER I: INTRODUCTION

There is strong evidence of a link between childhood maltreatment and a multitude of negative psychosocial outcomes, including emotional dysregulation, criminal offending, and aggression (Norman et al., 2012; Richeya, Browna, Fitea, & Bortolato, 2016; Schimmenti, Di Carlo, Passanisi, & Caretti, 2015). Much of the research on the maltreatment-aggression relationship has not differentiated between reactive (i.e., impulsive) and proactive (i.e., goal-directed) forms of aggression (e.g., Bandura, 1973; Lee & Hoaken, 2007); however, empirical findings from the few studies that have examined these subtypes separately suggest that they are preceded by different childhood experiences and are driven by different biological, emotional, and social-cognitive processes (Hoeve et al., 2015; Hubbard, McAuliffe, Morrow, & Romano, 2010; Shields & Cicchetti, 1998). Proactive aggression, which is considered a behavioral marker of psychopathy, was historically thought to be genetically driven, though growing evidence suggests that adverse psychosocial experiences in childhood play an important role in the development of proactive aggression, albeit indirectly through biological mechanisms (Braun & Bock, 2011; Shonkoff, Boyce, & McEwen, 2009).

Proactively aggressive individuals exhibit elevated callous-unemotional (CU) traits, such as reduced empathy and guilt, and do not respond to interventions that are otherwise effective for reactive aggression (Cornell et al., 1996; Fite, Wimsatt, Elkins, & Grassetti, 2012). Accordingly, there is a need to identify the underlying mechanisms that may yield appropriate targets for treatment. One plausible biological mechanism involves the neuropeptide oxytocin (OXT). OXT's role in facilitating empathy and prosocial behavior (Heim et al., 2008; Pierrehumbert et al., 2010) provides indirect evidence that reduced levels of OXT may underlie the characteristic lack of empathy in CU traits and proactive aggression. Furthermore, there is evidence to suggest

that childhood maltreatment, particularly emotional maltreatment, leads to long-term alterations in the oxytocinergic system (e.g., Cecil et al., 2014).

The present study was intended to add to the existing literature on the biological and affective processes underlying proactive aggression in the context of childhood maltreatment, by exploring OXT levels in saliva and CU traits in two at-risk samples of young adults. A serial mediation model was tested where maltreatment exerts its effect on proactive aggression indirectly through reduced concentrations of OXT, which increases the risk for CU traits, and ultimately proactive aggression. A secondary aim of this study was to identify whether these pathways to proactive aggression (i.e., through OXT and CU traits) were stronger for emotional forms of maltreatment than for physical maltreatment.

The following sections of this paper review the literature on childhood maltreatment and aggression. This discussion is then followed by a description of potential mechanisms linking the two, including CU traits and OXT.

Childhood Maltreatment

Childhood maltreatment has emerged as a major public health concern due to its pervasive and enduring adverse effects on children's physical, emotional, and behavioral development (Margolin & Gordis, 2000). According to the World Health Organization (WHO, 2006), maltreatment is defined as any physical, sexual, emotional, or negligent mistreatment by a caregiver that is harmful to a child's health, development, or dignity. Maltreatment is often characterized into one of the following types: physical abuse, emotional (i.e., psychological) abuse, sexual abuse, physical neglect, and emotional neglect, which are elaborated upon in Table 1.

Table 1. Definitions and types of child maltreatment.

Type of Maltreatment	Description
Physical abuse	The intentional use of physical force against a child that results in harm to the child's physical health or development. This includes hitting, kicking, shaking, biting, strangling, scalding, burning, poisoning, and suffocating. Much physical violence against children is inflicted for the purpose of punishing.
Emotional abuse	Involves patterns of behavior by a caregiver that result in impairment of emotional development and the child's sense of self-worth. Acts in this category may have a high probability of damaging the child's mental health and/or moral or social development. Abuse of this type includes the following: patterns of belittling, blaming, terrorizing, threatening, frightening, exploiting, corrupting, discriminating against, and other non-physical forms of hostile treatment.
Sexual abuse	The involvement of a child in sexual activity that he or she does not fully comprehend, is unable to give informed consent to, is not developmentally prepared, or else that violates the laws or social taboos of society. Children can be sexually abused by both adults and other children who are—by virtue of their age or development—in a position of responsibility, trust, or power over the victim.
Physical neglect	Includes isolated incidents as well as a pattern of failure over time on the part of a caregiver to provide for the physical development and well-being of the child—where the parent is in a position to do so—in one or more of the following areas: health, nutrition, shelter, and safe living conditions.
Emotional neglect	Failure to provide basic emotional nurturance, adequate (e.g., rejecting, isolating or ignoring a child) emotional or cognitive stimulation, and/or opportunities for experiential learning. This may include rejecting, isolating, or ignoring a child, or being unresponsiveness or insensitive to the child's basic psychological needs. Caregivers may be unable to respond to the child's emotional needs, with no provision of an adequate alternative.

Adapted from Butchart et al. (2006), DHHS (2003), and Norman et al. (2010)

The estimated prevalence rate of child maltreatment in the U.S. is between 25% and 41% (Finkelhor, Turner, Shattuck, & Hamby, 2013; Hussey, Chang, & Kotch, 2006). In addition to the immediate consequences of maltreatment (i.e., direct physical injury), there is growing recognition of the long-term consequences to a child's physical, neurologic, cognitive, and emotional health (Butchart et al., 2006). Although much of the literature has focused on the adverse effects of physical and sexual abuse, more recent studies have shown that emotional forms of maltreatment (i.e., emotional abuse or neglect) may have equally detrimental effects on children's psychological development (Cicchetti & Nurcombe, 1991; Iwaniec, 1995). Emotional maltreatment is often more difficult to recognize, but is surprisingly common; the worldwide prevalence rate is estimated to be 27% (Stoltenborgh et al., 2012).

Childhood Maltreatment and Aggression

Maltreatment is a strong risk factor for aggression and other antisocial outcomes (Kotch et al., 2008), although these outcomes vary significantly across individuals. Widom (1989) conducted a groundbreaking study showing that child maltreatment increased the risk for violent and chronic offending in youth and adults by comparing arrests for adults who were abused or neglected as children with matched controls with no history of maltreatment. Research has continued to support this "cycle of violence" hypothesis (Widom, 2014). For instance, having a history of maltreatment significantly increases the risk of arrests and violent acts in childhood (Stouthamer-Loeber, Loeber, Homish, & Wei, 2001), and increases the risk of violent crime in adulthood by 30% (Widom, 2014). Furthermore, a systematic review and meta-analysis of 124 prospective and retrospective studies revealed strong support for a causal relationship between maltreatment and similar outcomes, including childhood conduct disorder, drug use, and risky sexual behavior (Norman et al., 2006).

Despite the strong correlation between maltreatment and aggression as a whole, aggression is a multifaceted construct, and individuals with aggression problems make up a very heterogeneous group. Aggressive behaviors may be distinguished into two clinically meaningful subtypes (i.e., reactive and proactive aggression) based on their function, intensity of emotions associated with the act, and the level of planning involved. These two subtypes appear to have distinct behavioral, biological, emotional, and neurocognitive correlates, and also respond differently to treatment.

Reactive aggression. Reactive aggression refers to an emotionally charged form of aggression that occurs in response to provocation or frustration (e.g., after a heated argument). It is generally defensive in nature, and is also referred to as "impulsive" or "hot blooded"

aggression (Frick & White, 2008; Skeem, Polaschek, Patrick, & Lillienfeld, 2011). Reactive aggression is the most common form of aggression across community and clinical samples, and is associated with impulse control problems, emotional dysregulation, and hostile attribution biases. The role of emotional dysregulation was supported by a meta-analysis by Card and Little (2006), which revealed a consistent and positive association between internalizing problems (e.g., anxiety and depression) and reactive aggression, but not proactive aggression.

Maltreatment and reactive aggression. Strong correlations have been reported between maltreatment and reactive aggression among diverse samples, including community youth (Richey, Brown, Fite, & Bortolato, 2016; Shields & Cicchetti, 1998) and violent offenders (Kolla et al., 2013). Across studies, physical abuse tends to be most consistently associated with this type of aggression. For example, a longitudinal study by Dodge and colleagues (1997) found that physical abuse in kindergarten predicted reactive aggression in the 3rd grade, but not proactive aggression.

Several theories have been proposed to explain how physical abuse may increase the risk for reactive aggression. According to social learning theory, aggression may be modeled by caregivers or learned as a strategy for coping in a hostile environment (Bandura, 1973). It has also been argued that reactive aggression may develop as a result of high stress environments and inconsistent or harsh parenting, which make it difficult to predict the behavior of caregivers. As a result, children may develop hostile attribution biases that lead them to misinterpret neutral social cues as threatening or hostile, and subsequently respond with aggression (Richey, Brown, Fite, & Bortolato, 2016).

The notion that reactive aggression may develop from an overactive threat detection system has been supported by neuroimaging studies. Multiple independent studies have

demonstrated that individuals with high levels of reactive aggression and impulsive antisocial behavior display hyper-reactivity of the amygdala in response to stress or emotional stimuli (Aghajani et al., 2016; Cohn et al., 2013; Lozier et al., 2014; Sebastian et al., 2012; Viding et al., 2012). The amygdala is a structure in the limbic system with a primary role in emotional regulation and processing, particularly for negative emotions such as fear and anger. In addition, individuals with high levels of reactive aggression have higher than average concentrations of grey matter in the amygdala (e.g., Cohn et al., 2016).

Moreover, neurocognitive accounts of reactive aggression suggest that it may be related to deficits in impulse control and associated aspects of executive functioning (e.g., planning, response inhibition). These functions, along with emotional and behavioral regulation, are governed by the prefrontal cortex. Moffitt (1990; 1993) found that individuals with high levels of reactive aggression scored lower on neuropsychological tests that are sensitive to frontal systems dysfunction. In a sample of murderers, 65% exhibited impaired executive functioning in a study by Blake, Pincus, and Buckner (1995). When murderers were analyzed separately based on the type of murder committed (i.e., reactive/impulsive versus planned/premeditated), reactive murderers showed reduced activity in the anterior prefrontal cortex during a sustained attention test, while those who committed planned or premeditated murders did not (Raine et al., 1994).

Maltreatment and other adverse early experiences can result in frontal systems dysfunction either directly (e.g., traumatic brain injuries sustained from physical abuse) or indirectly through exposure to neurotoxins or teratogens, such as alcohol, nicotine, and lead exposure (e.g., Raine et al., 2002). In fact, the neurotoxic properties of lead appear to selectively target the prefrontal cortex (Wright, Boisvert, & Vaske, 2009). Taken together, these findings

suggest that maltreatment may influence reactive aggression indirectly through frontal systems dysfunction, which results in difficulty regulating or inhibiting emotions (Frick et al., 2003).

Proactive aggression. In contrast to the impulsive and emotional features of reactive aggression, proactive aggression is characterized by purposeful and calculated acts that are used to achieve a desired goal (e.g., revenge, intimidation, to obtain material goods). This type of aggression is also referred to as "cold-blooded" or "predatory" because it is not preceded by strong emotions and generally occurs with little emotional or physiologic arousal (Glenn & Raine, 2009). Given the relative infrequency of proactive aggression compared to reactive aggression, its presence is considered a behavioral marker of CU traits and/or psychopathy (Kolla et al., 2013).

Maltreatment and proactive aggression. Proactive aggression was not empirically studied in the context of childhood maltreatment until recently. This is in part due to the historical notion that psychopathy (and by association, proactive aggression) was driven largely by genetic or biological predisposition. Nonetheless, emerging evidence contradicts this notion and instead points toward a more complex etiology involving a combination of environmental and biological factors.

In a sample of 439 detained adolescent males in the juvenile justice system, Vahl and colleagues (2016) found that severity of proactive aggression increased with the number of types of maltreatment experienced. Multiple studies have also demonstrated surprisingly high prevalence rates of maltreatment in chronically violent offenders–up to 77% in one Italian sample (Craparo, Schimmenti, & Caretti, 2013). Additionally, Hoeve and colleagues (2015) examined the inter-relationships between maltreatment, mental health problems, and both types of aggression in a large sample of 767 adolescent boys at juvenile delinquent facilities. They

found a direct relationship between maltreatment and proactive aggression, which persisted after controlling for a number of mental health problems. Conversely, the association between maltreatment and reactive aggression was fully mediated by internalizing problems, including depression and anxiety.

These findings suggest that while maltreatment increases the risk for both types of aggression, the specific etiological pathways to proactive and reactive aggression are distinct. Several theories have been proposed to explain how childhood maltreatment increases the risk of proactive aggression, which are described in subsequent sections of this paper.

Callous-Unemotional Traits

In contrast to reactive aggression, high levels of proactive aggression are unlikely to occur without the presence of elevated CU traits, which are characterized by a callous lack of empathy and remorse (Frick, 2006). In other words, CU traits are thought to involve reduced capacity for prosocial emotions (i.e., emotions that facilitate social cooperation) such as empathy, guilt, and remorse. These emotions involve feelings of discomfort and visceral reactions at the thought of wrongdoing (Damasio, 1994). The presence of CU traits in childhood is a particularly strong predictor of future juvenile arrests among community youth, recidivism, and persistent criminal offending into adulthood, more so than any other psychological or environmental predictor (Hare & Neumann, 2008; McMahon et al., 2010). CU traits distinguish psychopathic offenders from non-psychopathic offenders, and seem to characterize a particularly severe and violent subgroup of antisocial youth and adults (Frick, 2006; Loper, Hoffschmidt, & Ash, 2001; Porter, Birt, & Boer, 2001). For example, Caputo, Frick, and Brodsky (1999) found that the presence of CU traits differentiated between violent sex offenders and other types of offenders, while other features of psychopathy (e.g., narcissism, impulsivity) did not.

The importance of this distinction is reflected in the inclusion of a "with limited prosocial emotions" specifier for the diagnosis of conduct disorder in the DSM-5 (APA, 2013). This identifies youth with conduct disorder who have CU traits as evidenced by at least two of the following characteristics: lack of remorse or guilt, callous lack of empathy, lack of concern about performance (e.g., at school or work), and shallow, deficient, or superficial affect (e.g., when the displayed emotions contradict one's actions, or are displayed to manipulate others). These criteria were developed from item analyses using the Inventory of Callous-Unemotional Traits (ICU; Frick & Moffit, 2010).

Individuals with elevated CU traits are thought of as being incapable of forming genuine affectionate bonds (Blair, 2006; Cleckley, 1976; Patrick, Durbin, & Moser, 2012), and this lack of close relationships or attachments is often apparent in early childhood (see Frick, 2006; Marsee & Frick, 2006 for reviews). An abundance of research has demonstrated the diminished emotional responsiveness to the distress of others among adults and youth with elevated CU traits (e.g., Buss & Plomin, 1984; Sterzer et al., 2004; Watson & Clark, 1984, 1992), which is in direct contrast to that of individuals with high levels of reactive or impulsive antisocial behavior, but without CU traits. CU traits are also associated with reduced amygdala activity in response to emotional stimuli or moral decision making paradigms (Glenn, Raine, & Schug, 2009; Jones et al., 2009). This reduced autonomic response suggests indifference to others' distress and may reflect an impaired ability to empathize with the fear and sadness of others (Finger et al., 2008).

Additional studies have indicated that individuals with CU traits experience reduced intensity of a broader range of emotions, beyond those that are prosocial. They also appear to experience reduced negative and "self-conscious" emotions, such as embarrassment and shame, which involve higher cognitive processes of self-reflection and awareness of other people's

reactions to us (Cleckley, 1941; Hicks & Patrick, 2006; Tangney & Dearing, 2002). Consistent with the lack of association between proactive aggression and internalizing disorders observed by Hoeve and colleagues (2015), individuals with CU traits have been described as fearless and immune to stress. In fact, the presence of CU traits is a protective factor for depression, anxiety, and suicide completion (Cleckley, 1941; Patrick, Fowles, Krueger, 2009).

Psychophysiological studies have also demonstrated reduced autonomic responses (e.g., startle reflexes, heart rate, and skin conductance reactivity) to aversive and fear-provoking stimuli (Blair, 2006; Levenston, Patrick, Bradley, & Lang, 2000; Patrick, 1994; Patrick, Bradley, & Lang, 1993). Furthermore, individuals with CU traits demonstrate an "insensitivity to punishment" on aversive learning or fear conditioning paradigms (van Goozen et al., 2004). While most individuals attempt to escape from noxious stimuli relatively quickly in aversive learning paradigms, those with CU traits elect to endure a much greater magnitude and duration of punishment in order to access a reward than those with low levels of these traits (Maharaj, 2014). This abnormally low sensitivity to punishment cues has also been demonstrated outside of laboratory tests. For instance, youth with CU traits are less responsive to positive punishment-based discipline strategies than other youth with conduct problems (Blair, 2006; Blair, Colledge, & Mitchell, 2001; Patrick, Fowles, & Krueger, 2009). It is possible that this apparent fearlessness and insensitivity to punishment may also predispose individuals with CU traits to risky behavior and aggression (Hicks & Patrick, 2006; Tellegen & Waller, 1992).

Callous-unemotional traits as affective features of psychopathy. In the first thorough conceptualization of psychopathy, The Mask of Sanity, Cleckley (1941) asserted that the core feature of psychopathy was a deficiency in the intensity and/or range of emotional experience, and that this general poverty of "moral" emotions was responsible for psychopaths' patterns of

manipulative and antisocial behavior (1941; 1988). These emotional features are analogous to what are now referred to as CU traits; however, the term “psychopathy” tends to be reserved for adults in forensic contexts, while “CU traits” is regarded as a more appropriate label for youth and non-forensic populations with these features.

Current conceptualizations of psychopathy are based on research using Hare’s Psychopathy Checklist-Revised (PCL-R; Hare, 1991), which is considered the “gold standard” for diagnosing psychopathy. According to this manual, the affective features of psychopathy include 1) a lack of remorse or guilt, 2) lack of empathy, and 3) shallow emotions. Hare’s (1993) description of these features is worth quoting at length:

> While at times they appear cold and unemotional, they are prone to dramatic, shallow, and short-lived displays of feeling. Careful observers are left with the impression that they are play acting and that little is going on below the surface. Sometimes they claim to experience strong emotions but are unable to describe the subtleties of various affective states. For example, they equate love with sexual arousal, sadness with frustration, and anger with irritability. … A psychopath in our research said that he did not really understand what others meant by “fear.” However, “When I rob a bank," he said, "I notice that the teller shakes. One barfed all over the money. She must have been pretty messed up inside, but I don't know why. If someone pointed a gun at me I guess I'd be afraid, but I wouldn't throw up." When asked if he ever felt his heart pound or his stomach churn, he replied, "Of course! I'm not a robot. I really get pumped up when I have sex or when I get into a fight." (Hare, 1993, p. 52-53)

Although there is overlap, psychopathy is distinct from the diagnosis of antisocial personality disorder (ASDP) as defined by the DSM-5 (APA, 2013). ASPD criteria reflect a disregard for and violation of others' rights since at least age 15 as evidenced by at least one of the following: failure to obey laws and norms by engaging in behavior that warrants criminal arrest; lying, deception, and manipulation for profit or self-amusement; impulsive behavior, irritability, and aggression; blatant disregard for the safety of self and others; a pattern of irresponsibility; and lack of remorse for actions. These criteria are very broad and weakly represent CU traits, which are central to the construct of psychopathy. While 50% to 80% of prison inmates meet DSM criteria for ASPD (Ogloff, 2006), only 15-20% of inmates and 1-3% of the general population meet criteria for a diagnosis of psychopathy based on the PCL-R (Hare, Hare, & Harpur, 1991). While only a subgroup of antisocial individuals has elevated CU traits, it should also be noted that not all individuals with CU traits engage in antisocial behavior. As such, CU traits can exist outside of criminal populations (see Cleckley, 1941 on the "successful psychopath").

Only within the past one to two decades have empirical studies begun to focus on adverse childhood experiences and maltreatment as precursors to CU traits and proactive aggression– which are together referred to as "psychopathic traits" in this paper. Evidence from this growing literature base suggests that the precursors and developmental pathways to these outcomes are distinct from those leading to reactive aggression and other non-psychopathic forms of antisocial behavior. In order to develop more appropriate assessment and treatment strategies, additional research is needed to elucidate the underlying mechanisms.

Linking Maltreatment to Psychopathic Traits

As stated earlier, although many researchers have historically posited that the CU traits

and related features of psychopathy are primarily driven by genetic vulnerabilities rather than environmental factors, recent research has suggested otherwise. For instance, a large study by Weiler and Wisdom (1996) with a sample of 1,141 adolescent and young adult offenders concluded that those who had a history of childhood maltreatment scored higher on measures of psychopathy. This finding has been replicated across many different studies (e.g., Borja & Ostrosky, 2013; Carlson, Oshri, & Kwon, 2015; Craparo, Schimmenti, & Caretti, 2013; Hoeve et al., 2015; Schimmenti et al., 2014).

Some researchers have suggested that severe maltreatment leads to deficits in prosocial emotions and moral development, which in turn increase the risk for CU traits (Porter, 1996). Hoeve and colleagues (2015) expanded this notion by asserting that maltreated children may become emotionally "numb" as a coping mechanism to avoid the emotional pain of abuse, which in turn results in interpersonal callousness or emotional detachment, and ultimately increases the likelihood of engaging in proactive aggression (Kimonis, Fanti, Isoma, & Donoghue, 2013; Porter, 1996). This hypothesis was supported by recent research on juvenile justice youth showing that the association between childhood trauma and CU traits was mediated by emotional numbing (Kerig, Bennett, Thompson, & Becker, 2012).

Differential effects of emotional maltreatment. Given the likelihood that the processes by which maltreatment leads to proactive aggression are distinct from those that lead to reactive aggression, it is possible that specific characteristics of maltreatment (e.g., specific forms, chronicity, age of onset) differentially affect these outcomes. Among the few studies that have examined the differential effects of specific forms of maltreatment on CU traits and proactive aggression, emotional maltreatment often emerges as the strongest predictor, though this varies across samples.

Several researchers have argued that emotional maltreatment reduces the child's empathy and ability to "mentalize" the emotions of others, which are both reduced in individuals with CU traits (Gergley & Watson, 1996; Schimmenti et al., 2014). In a sample of 78 white male inmates convicted of violent crimes in Italy, Schimmenti and colleagues (2014) found that emotional abuse uniquely predicted both CU traits and total psychopathy scores, while physical and sexual abuse only predicted the impulsive lifestyle and antisocial features of psychopathy. In an overlapping sample of 139 violent offenders, Schimmenti and colleagues (2015) found that participants with the highest psychopathic traits had experienced severe degrees of multiple types of child maltreatment concurrently. Specifically, "relational" trauma (e.g., psychological abuse, rejection, neglect, at least 1 year in residential care) were frequently endorsed in this sample, and were found to be risk factors for disorganized attachment, which is characterized by a lack of clear attachment behavior.

Similarly, in a sample of 193 prisoners, Borja and Ostrosky (2013) found that psychopathic inmates had been exposed to more severe childhood emotional abuse than purely antisocial inmates (i.e., without CU traits). Furthermore, maltreatment was reported by 88% of violent male offenders who scored high in psychopathic traits, and emotional neglect was endorsed most frequently (i.e., 68%) in this subgroup (Craparo, Schimmenti, & Caretti, 2013). In a study by Hoeve and colleagues (2015) with juvenile delinquents, proactive aggression was most strongly associated with emotional abuse (r = .45) and physical abuse (r = .41), followed by a small but significant correlation with emotional neglect (r = .14). Sexual abuse was not significantly correlated with proactive aggression.

Long-term deficits in affective and interpersonal functioning among children with histories of emotional neglect have been reported as early as the 1940's. Bowlby (1944) coined

the term “affectionless” children (Follan & Minnis, 2010) to describe the features (e.g., inability to feel remorse, indifference to others’ feelings, delinquency) that he observed among children who had been separated from their mothers and placed in institutions at a young age. He posited that limited opportunities to form close attachments to a caregiver in early childhood resulted in difficulties forming attachments in the future.

This early research on attachment led to the development of diagnostic criteria for reactive attachment disorder (e.g., emotionally withdrawn and inhibited phenotype) and disinhibited social engagement disorder (e.g., indiscriminately social/disinhibited phenotype), which are thought to result from emotional neglect or severely disrupted attachment with a primary caregiver (APA, 2013). Children in the foster care system, who often experience multiple placement changes and lack stable attachments with caregivers, are particularly at risk for both disorders (Zeanah, Smyke, & Dumitrescu, 2002). Bowlby’s theories were supported by the English and Romanian Adoptees longitudinal study of institutionalized youth, which revealed that attachment problems at age 4 years were associated with interpersonal insensitivity and callous lack of concern for others at the age of 15 (Sonuga-Barke, Schlotz, & Kreppner, 2010).

Operant conditioning and behavioral analytic theories have also been applied to the manipulative or indiscriminately friendly behaviors that are characteristic of these youth that lack stable attachments with caregivers. Such behaviors are likely adaptive in environments where adult attention is inconsistent or infrequent. Furthermore, manipulative and charming behaviors continue to be reinforced with attention from adults, as new caregivers and strangers may consider their behavior “cute and endearing” (Chisholm, 1998; Golden, 2007).

All of these findings support the hypothesis that emotional maltreatment disrupts affective development. Other studies, however, have yielded inconclusive and at times

contradictory findings regarding the differential effects of specific types of maltreatment. For instance, Kolla and colleagues (2013) showed that childhood physical abuse, but not emotional abuse, predicted psychopathy scores on the PCL-R in a small sample of 25 violent offenders. Additionally, Carlson, Oshri, and Kwon (2015) found that physical, sexual, and emotional abuse equally predicted CU traits, though all correlations were small in magnitude. Some of the inconsistent findings may be a factor of the different samples that were used in each study. The majority of studies only included forensic samples that were limited to participants with low SES. It is possible that the observed relations may differ in community samples or among individuals with high SES. Furthermore, many studies sampled only extreme populations (e.g., white male violent offenders, murderers) that are not representative of the full spectra of outcomes of maltreatment, so the degree to which these findings would generalize to more typical populations is unclear.

Neurobiological processes underlying the outcomes of maltreatment. As the brain develops in an experience-dependent manner throughout childhood and beyond, it is impossible to fully understand how maltreatment influences aggression without acknowledging its impact on neurobiological, cognitive, and emotional development. There is considerable evidence to suggest that maltreatment can fundamentally and chronically alter development of these systems. The remaining sections of this literature review elaborate on the potential biological processes that underlie the development of psychopathic traits.

Emotional, social, and cognitive development are heavily influenced by experiences during "sensitive periods" of neurodevelopment (Sowell et al., 1999), where neural circuitry is particularly sensitive to environmental stimuli. With regard to emotional development, certain socio-emotional experiences (e.g., attachment with a primary caregiver) are necessary to guide

neural differentiation and pruning in regions of the brain that are responsible for emotionality, including connections between the limbic system and prefrontal cortex (Braun & Bock, 2011). These processes occur in an experience-dependent or "use it or lose it" manner, such that the synaptic pathways that are frequently activated will strengthen, while synapses that are rarely activated will be pruned. In this fashion, one's social experiences in childhood guide synaptic organization to form a child's "emotional template" (Braun & Bock, 2011).

The majority of brain development occurs within the first 5 years of life, though synaptic "pruning" away of relatively inactive neurons continues throughout adolescence. The most intense period of synaptic pruning occurs between the ages of 7 and 16 (Pihl & Benkelfat, 2005); therefore, social experiences during this period would be expected to play a major role in emotional development (Pihl & Benkelfat, 2005). Based on evidence from animal models of maternal deprivation, it is likely that chronic emotional neglect results in excessive neural pruning in prefronto-limbic pathways, leaving these circuits that are responsible for processing and experiencing prosocial emotions underdeveloped (Sonuga-Barke, Schlotz, & Kreppner, 2010). Within the limbic system, the amygdala is particularly sensitive to psychosocial adversity within the first three years of life (Sanchez, Hearn, Do, Rilling, & Herndon, 1998; Teicher et al., 2003). This may explain the finding of reduced amygdala volume among maltreated youth (Whittle et al., 2013), and also support supports Blair's (2007) theory that amygdala hypoactivity underlies the high levels of proactive aggression among individuals with psychopathy and CU traits (e.g., Jones et al., 2009).

These findings highlight the potential for early childhood adversity to hinder the development of prosocial emotions via changes in the central nervous system; however, changes in the neuroendocrine system may also underlie the emotional and behavioral consequences of

maltreatment. It is widely acknowledged that childhood adversity dramatically alters the hypothalamic-pituitary-adrenal (HPA) axis, which is responsible for regulating the body's response to stress through the release of stress hormones (Gabbard 2005; Tyrka et al., 2009). More recently, the effects of childhood adversity have been found to extend to other neuroendocrine systems, including the hypothalamic–neurohypophyseal system, which regulates secretion of the neuropeptide OXT (Heim et al., 2008; Pierrehumbert et al., 2010). The following sections review the oxytocinergic system, its role in social behaviors, and evidence that suggests the presence of abnormalities of this system in CU traits and proactive aggression.

Oxytocin

Although the functions of OXT were once thought to be limited to the female reproductive system (see Yang, Wang, Han, & Wang, 2013, for a review of the non-social functions of OXT), it is now known to play a crucial role in social bonding. In humans, OXT has gained recognition as the "social neuropeptide" because of its role in a number of complex social emotions and behaviors, including attachments, empathy, and emotion recognition (Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011). In fact, reduced OXT levels are implicated in a number of psychiatric disorders that are characterized by social dysfunction, including autism spectrum disorders, schizophrenia, and trait aggression (Beitchman et al., 2012; Lee, Ferris, van de Kar, & Coccaro, 2009).

OXT is released both centrally and peripherally. Although much of the knowledge on the social roles of OXT is based on non-human animal studies measuring central levels of OXT in cerebrospinal fluid, human studies often rely on peripheral measurements (e.g., in saliva, plasma, or urine). In the brain, OXT is primarily synthesized in magnocellular neurons in the paraventricular and supraoptic nuclei of the hypothalamus. These oxytocinergic neurons are

transported along axonal projections to the posterior pituitary gland, where they are then released into the bloodstream for peripheral circulation (Ross et al., 2009; Yang, Wang, Han, & Wang, 2013). There are also smaller parvocellular neurons in the hypothalamus that project OXT directly to the cerebral cortex and other areas of the limbic system, including the amygdala and other hypothalamic regions. Formerly, parvocellular neurons were thought to be the only neurons responsible for releasing OXT in the brain–which cast doubt on the relevance of using peripheral OXT as a proxy for central OXT release–though it is now recognized that magnocellular neurons contribute to central release as well (Ludwig & Leng, 2006; Quirin, Kuhl, & Dusing, 2011).

Prosocial roles of oxytocin. OXT is essential for the development of attachments between mother and child, as well as other types of social bonds, including romantic attachments, close friendships, and even bonds between humans and pets (Nagasawa, Kikusui, Onaka, & Ohta, 2009). Its role in facilitating maternal caregiving behaviors was first demonstrated in a 1979 study by Pedersen and Prange, who found that injections of OXT caused virgin female rats to seek out and care for abandoned rat pups (Marlin, Mitre, D'Amour, Chao, & Froemke, 2015). Studies with socially monogamous animals, such as prairie voles, have yielded similar effects on the formation of social bonding and preference for physical contact with their partners (e.g., Bales et al., 2013; Young & Wang, 2004). In humans, the prosocial effects of intranasally-administered OXT have been examined in psychiatric disorders involving social dysfunction, including autism spectrum disorder, social anxiety, and schizophrenia (Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005; Mikolajczak, Pinon, Lane, de Timary, & Luminet, 2010). In individuals with autism spectrum disorder, intranasal OXT increases emotion recognition, eye contact, trust, willingness to interact socially, eye gaze, empathy, “theory-of-

mind," and amygdala activity in response to social stimuli (Andari et al., 2010; Domes et al., 2013; Guastella, Mitchell, & Dadds, 2008; Macdonald & Macdonald, 2010). Similar effects (e.g., increases in generosity, trust, emotional empathy, and emotional responsiveness toward children) have been observed in healthy individuals as well (Domes et al., 2010; Naber, van Ijzendoorn, Deschamps, van Engeland, & Bakermans-Kranenburg, 2010). For instance, one noteworthy study revealed that emotional empathy levels in men who received intranasal OXT increased to the levels found in untreated women (Hurlemann et al., 2010).

Although there are many promising findings regarding the therapeutic potential of OXT for social functioning deficits, closer inspection of the data reveals a more complex role in interpersonal emotions and prosociality (Domes, Heinrichs, Michel, Berger, & Herpetz, 2007; Guastella, Mitchell, & Dadds, 2008). A systematic review by Bartz, Zaki, Bolger, and Ochsner (2011) found that 20% of published studies actually reported negative (i.e., antisocial) effects, and that contextual and inter-individual differences appear to influence whether or not OXT has a positive social effect. For instance, among individuals with borderline personality disorder, who generally have more insecure attachments, OXT appears to decrease trust and cooperative behaviors (Bartz et al., 2011). Correspondingly, while peripheral OXT levels in humans are significantly higher during the early stages of romantic attachment, high OXT is also correlated with new partners' anxiety about their relationship with the partner (Schneiderman, Zagoory-Sharon, Leckman, & Feldman, 2012). Rodent studies have also yielded similar effects under certain conditions. OXT increases maternal aggression among female rats when in the presence of an intruder, though this behavior is ultimately to protect offspring (Bosch, Meddle, Beiderbeck, Douglas, & Neumann, 2005). One hypothesized mechanism of OXT's effects on behavior, which could account for these divergent findings, is that OXT increases the salience of

social stimuli (Shamay-Tsoory & Abu-Akel, 2016). By increasing one's attention to social information, OXT may facilitate a stronger experience of interpersonal emotions regardless of valence (positive or negative).

Oxytocin, CU traits, and proactive aggression. Few studies have examined the relationship of OXT and CU traits. The majority of evidence supporting the role of reduced OXT in psychopathic traits is indirect and based on the beneficial effects of OXT on other clinical populations with deficits in social cognition. Despite a number of important differences between autism and CU traits, both are associated with reduced empathy, reduced attention to the eyes of emotional faces (Gillespie, Rotshtein, Wells, Beech, & Mitchell, 2015), and other neurobiological evidence of reduced salience of social-emotional stimuli. Levy and colleagues (2015) published one of the first studies examining salivary OXT, CU traits, and conduct problems. In a sample of 67 male adolescents in a residential treatment facility, the authors found that participants with low OXT and severe conduct problems were significantly more likely to have elevated CU traits. This provides support for the role of salivary OXT in the subgroup of antisocial individuals with elevated CU traits.

Additionally, Fetissov and colleagues (2006) found increased levels of an antibody reactive to OXT in aggressive male participants compared with non-aggressive controls, suggesting that low OXT levels may account for persistent instrumental aggression. In support of these findings, Lee, Ferris, Van de Kar, and Coccaro (2009) found that central OXT levels were negatively correlated with a history of lifelong aggressive behavior. More recent studies have identified single nucleotide polymorphisms (SNPs) in the OXT receptor gene that are associated with CU traits, aggression, and psychopathy (Beitchman et al., 2012; Dadds et al., 2014). One study by Malik, Zai, Abu, Nowrouzi, and Beitchman (2012), which examined in a

sample of 160 highly aggressive children with 160 matched adult controls with no history of aggressive behavior. Results demonstrated a statistically significant positive correlation between two SNPs and persistent aggression and antisocial behavior, although no significant differences were found with respect to CU traits. However, CU analyses were conducted in children who were selected for highly aggressive behavior because of the established correlation with CU traits, so there was limited variability among the degree of CU traits in this sample and this may have contributed to the lack of statistically significant findings in this area.

Maltreatment and oxytocin. One mechanism through which maltreatment may impede the development of prosocial emotions is by altering the release of OXT. One of the first published studies concerning the effects of early social experience on OXT was conducted by Fries and colleagues (2005). These authors compared OXT and vasopressin levels in a group of 18 previously orphaned children with a group of 21 typically-reared children. In a counterbalanced order, urine samples were collected 1) at baseline, 2) after the children engaged in physical contact with their mother, and 3) after physical contact with an unfamiliar adult. Interestingly, basal OXT levels among groups of children did not differ; instead, the between-group differences were observed in response to their interactions with the adults. Specifically, while family-reared children exhibited a greater increase in OXT after contact with their mothers than after contact with the unfamiliar adult, whereas OXT levels for the orphaned children did not differ in the presence of their mothers versus strangers. However, there were several limitations of this study, including relatively high variability among the orphaned children's OXT responses. While all of the orphaned children experienced severe neglect, any concurrent experience of other types of maltreatment was unknown. The authors also noted that this study occurred after an average of 3 years of rearing in relatively stable family environments, so many

of the children could have since developed satisfactory interpersonal relationships. Furthermore, no emotional or behavioral outcomes were reported in this study, so it is unknown how these additional variables would relate to OXT.

One way that maltreatment could increase the risk of CU traits is through epigenetic mechanisms. For instance, cumulative exposure to environmental stressors can promote methylation, which leads to long-term changes in gene expression. Methylation of the promoter regions of OXT receptor genes reduces gene expression by approximately 70%. One study found a significant association between CU traits and OXT receptor methylation in adolescents, but not for younger children (Dadds et al., 2014), which suggests that only adolescents with CU traits had reduced OXT receptor expression. These findings demonstrate the potential for early psychosocial adversity to influence CU traits by promoting methylation of the OXT receptor gene.

In one of the only published studies examining the relationships between specific forms of maltreatment and OXT, Heim and colleagues (2009) found that early exposure to abuse led to OXT reductions that were proportional to the frequency, severity, and duration of abuse. The authors examined OXT concentrations in cerebrospinal fluid in 22 adult women after early-life trauma. The participants were categorized into either having no or mild childhood maltreatment or having moderate to severe exposure to multiple forms of maltreatment (i.e., emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect). They found that exposure to maltreatment was significantly and negatively associated with OXT concentrations. Furthermore, emotional abuse had the strongest effect on OXT. There were also inverse associations between OXT and the number of types of maltreatment the women were exposed to as children, and the severity and duration of the maltreatment (Heim et al., 2009).

It is important to note that while multiple studies have demonstrated significant associations between maltreatment and OXT, the direction of these effects are not all consistent. Some of these inconsistencies may reflect contextual or inter-individual factors, such as the inclusion of only women versus both sexes. For instance, among women, OXT levels increase following stress, which is thought to occur as a result of OXT's suppressing effects on the HPA axis (Seltzer, Zeigler, Connolly, Prososki, & Pollack, 2013). Another notable study that only included female participants found that salivary OXT levels were higher among women with histories of emotional maltreatment, and that high OXT levels were associated with more positive ratings of happy infant faces (Bhandari et al., 2014).

Existing studies of peripheral OXT are also limited by small samples and methodological heterogeneity, which contribute to much of the variability in findings. Unextracted samples yield considerably higher and more variable values, presumably due to plasma proteins interfering with antibody binding, although this is less problematic in saliva samples, where there are fewer of these proteins (Carter et al., 2007; Leng & Sabatier, 2016). Furthermore, OXT concentrations differ across biological fluids, so OXT levels in saliva, plasma, and urine are not directly comparable.

Purpose of the Present Study, Hypotheses, and Analyses

The studies reviewed above reveal a strong biological basis for CU traits, which may involve abnormalities in the oxytocinergic system. Evidence of the impact of maltreatment on neuroendocrine systems, in combination with emerging support for reduced levels of OXT in individuals with CU traits and other social-emotional deficits, supports the hypothesis that reduced levels of OXT may mediate the relationship between maltreatment and proactive aggression. Identification of the underlying mechanism is essential to the development of

effective interventions for this population. If reduced levels of OXT do contribute to the development of CU traits and associated antisocial behaviors, then it is possible that the social-emotional deficits that characterize CU traits may be responsive to interventions targeted at the OXT system.

This study investigated several hypotheses regarding the inter-relationships among maltreatment, OXT, CU traits, and proactive aggression, which formed the basis for the primary serial mediation hypothesis.

Hypothesis 1. First, it was hypothesized that salivary OXT would be inversely associated with CU traits and proactive aggression, while any association with reactive aggression would be weaker because this type of aggression is associated with emotional dysregulation and poor impulse control, rather than reduced empathy. Moreover, multiple studies have linked OXT with defensive aggression under certain circumstances (e.g., maternal, protective, and relational aggression; Campbell, 2007).

Analysis of Hypothesis 1. Correlational analyses were completed to identify the strength of associations for salivary OXT with CU traits (Inventory of Callous-Unemotional Traits), proactive aggression, and reactive aggression (respective subscales of the Reactive-Proactive Aggression Questionnaire).

Hypothesis 2. Another aim of this study was to explore the association between OXT and maltreatment history. Hypothesis 2 stated that total childhood maltreatment would be inversely related to OXT. Based on the affective deficits and neurobiological differences that are frequently reported among emotionally maltreated youth, it was anticipated that this association would be stronger for emotional forms of maltreatment than for physical maltreatment.

Analysis of Hypothesis 2. Bivariate correlational analyses were performed for OXT with

the total score on the Childhood Trauma Questionnaire (CTQ) and separately for each subscale score (i.e., Physical Abuse, Physical Neglect, Emotional Abuse, Emotional Neglect, and Sexual Abuse).

Hypothesis 3. Following from these initial hypotheses, the primary aim of this study was to evaluate the fit of a conceptual model of the relationship between maltreatment and proactive aggression, where OXT and CU traits operate in serial to serve as biological and emotional mediators, respectively. Specifically, it was hypothesized that childhood maltreatment (X) would be associated with lower levels of salivary OXT (M_1) and in turn, greater CU traits (M_2), and thus higher levels of proactive aggression.

Analysis of Hypothesis 3. All mediation models were evaluated using the PROCESS macro for SPSS developed by Hayes and Preacher (2014), which uses an ordinary-least-squares path analysis to estimate unstandardized coefficients for the direct and indirect effects. Variables were included as covariates in mediation analyses if their effect was significant on outcome variables included in the model. To test the statistical significance of all effects, bootstrapping was used taking 10,000 samples from the data set to construct 95% bias-corrected confidence intervals. This procedure is recommended because it does not make any assumptions about the normality of the sampling distribution (Preacher & Hayes, 2008; Shrout & Bolger, 2002) and it reduces the probability of making Type I errors relative to other methods, including Baron and Kenny's (1986) causal steps approach and Sobel's (1982) product of coefficients test. Furthermore, bootstrapping is preferred when there is the potential for suppression (i.e., when the indirect effect $a \times b$ has the opposite sign of the direct effect c') or when the effect sizes are small.

To test Hypothesis 3, a serial mediation model (PROCESS Model 6) was used with

salivary OXT and CU traits entered as the two mediators. As depicted in Figure 1, this model yields three indirect effects that link maltreatment and proactive aggression: one through OXT only (a_1b_1), one through CU traits only (a_2b_2), and one through both OXT and CU traits in serial ($a_1a_3b_2$). Serial mediation would be supported if the final specific indirect effect ($a_1a_3b_2$) is statistically significant (i.e., if 95% confidence intervals do not include 0).

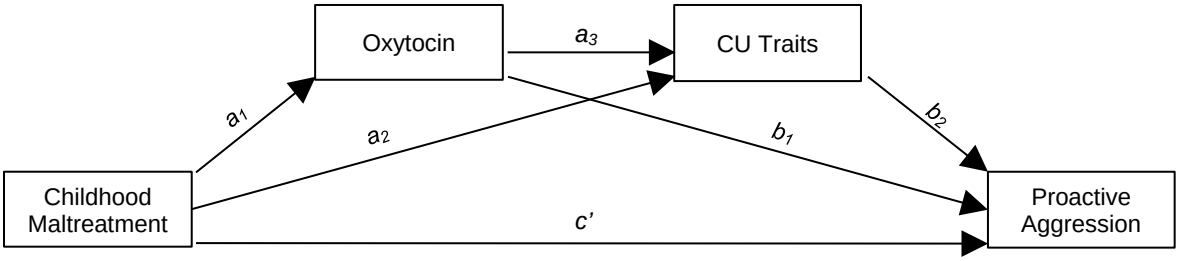

Figure 1. Primary serial mediation model in Hypothesis 3.

Hypothesis 4. It was hypothesized that the mediation effects of OXT and CU traits would be stronger for emotional forms of maltreatment, relative to physical forms of maltreatment.

Analysis of Hypothesis 4. The same analysis using PROCESS Model 6 was run separately for each type of maltreatment by entering each subscale score on the CTQ as the predictor variable, with other types entered into the model as covariates. To compare the magnitude of indirect effects across models, analyses were re-run after standardizing all continuous variables and the same covariates were included in each analysis included. The point estimates and confidence intervals of each model's final specific indirect effect were then compared to determine whether mediation was stronger for emotional abuse and emotional neglect than for physical abuse and physical neglect.

CHAPTER II: METHODS

Participants

The intended sample size was a minimum of 100, based on recommendations by MacKinnon, Lockwood, and Williams (2004) for studies using mediation analyses with bootstrapped confidence intervals. Although bootstrapping methods can be used with smaller sample sizes (Efron & Tibshirani, 1993; Preacher & Hayes, 2004), results of a simulation study by Koopman, Hower, Hollenbeck, and Sin (2015) showed that 100 cases are required to detect a moderate effect size with 80% power.

A total of 101 participants (ages 18-29) completed the study. Participants consisted of two subgroups of young adults that were at risk for aggression and antisocial behavior. Forty-seven participants comprised the group of "non-offenders." This subsample was recruited from East Carolina University's (ECU) introductory psychology courses, where they received course credit for participating in research activities. Students were invited to register for the study if they endorsed a history of adverse childhood experiences (e.g., childhood maltreatment, multiple changes in caregivers). Once registered for the study, no additional eligibility criteria were imposed regarding maltreatment histories in order to prevent deceptive responding (i.e., endorsing greater maltreatment in order to earn course credit) and to allow more variability in the data.

An additional 54 participants ("offenders") were recruited from two local probation/parole offices in North Carolina. Participants in this subsample had all been convicted of at least one criminal offense for which they were currently serving probation or parole. Although childhood maltreatment was frequently endorsed in this subsample, it was not a prerequisite for participation in the study. Exclusionary criteria included having less than a 9th

grade education, having a diagnosed developmental/intellectual disability or psychotic disorder, or not speaking English.

Characteristics of the sample in total and by offender status are provided in Table 2. Non-offenders (M_{age} = 19.65; *SD* = 1.10) were significantly younger than offenders (M_{age} = 24.10; *SD* = 3.50), $t(61.49) = -8.73$, $p < .001$, 95% CI [-5.47, -3.43]. Twenty-four participants (7 offenders, 17 non-offenders) reported having at least one psychiatric diagnosis (range 0 – 4), seven of whom endorsed diagnoses characterized by antisocial or aggressive behavior (i.e., conduct disorder, ASPD, and intermittent explosive disorder).

Table 2. Participant characteristics

Characteristic	Non-offender *n* (% of subsample)	Offender *n* (% of subsample)	Total *n* (% of total *N*)
Sex			
Female	28 (60%)	11 (20%)	39 (39%)
Race/Ethnicity			
White	30 (64%)	20 (37%)	50 (50%)
Black	16 (34%)	31 (57%)	47 (47%)
Hispanic	0 (0%)	1 (2%)	1 (1%)
Mixed	1 (2%)	2 (4%)	3 (3%)
Highest Education Level			
Some High School	0 (0%)	11 (22%)	11 (12%)
GED	0 (0%)	10 (20%)	10 (10%)
High School Diploma	0 (0%)	16 (33%)	16 (17%)
Postsecondary Education	47 (100%)	12 (25%)	59 (62%)
Relationship Status			
In a Relationship or Married	17 (37%)	30 (56%)	47 (47%)
Number of Changes in Caregivers			
0	32 (70%)	37 (69%)	69 (69%)
1-2	8 (17%)	11 (20%)	19 (19%)
3-21	6 (13%)	6 (12%)	12 (12%)
Medical Conditions			
Asthma/Allergies	22 (44%)	11 (20%)	33 (33%)
Head Injuries[1]	15 (33%)	10 (19%)	25 (25%)
CVD/HTN	3 (6%)	0 (0%)	3 (3%)
Kidney Disease	0 (0%)	1 (2%)	1 (1%)
Seizure Disorder	0 (0%)	2 (4%)	2 (2%)
Psychiatric Diagnoses			
Depression	6 (13%)	6 (12%)	12 (12%)
Anxiety	7 (15%)	5 (10%)	12 (12%)
ADHD	7 (15%)	4 (8%)	11 (11%)
Bipolar Disorder	0 (0%)	4 (8%)	4 (4%)
Conduct Disorder	1 (2%)	2 (4%)	3 (3%)
Antisocial Personality Disorder	1 (2%)	2 (4%)	3 (3%)

Borderline Personality Disorder	1 (2%)	1 (2%)	2 (2%)
PTSD	0 (0%)	2 (4%)	2 (2%)
Intermittent Explosive Disorder	0 (0%)	1 (2%)	1 (1%)
Current Medications			
CNS Stimulant	8 (17%)	1 (2%)	9 (9%)
SSRI	3 (6%)	2 (4%)	5 (5%)
Antihistamine	2 (4%)	1 (2%)	3 (3%)
Anti-Epileptic Drug	0 (0%)	1 (2%)	1 (1%)
Narcotic Analgesic[2]	0 (0%)	1 (2%)	1 (1%)
Menstruation Data for Female Participants			
Oral Contraceptive Use	10 (33%)	1 (11%)	11 (28%)
Day 1-14 of Menstrual Cycle[3]	19 (63%)	6 (67%)	25 (64%)

Note. CVD/HTN = cardiovascular disease/hypertension; SSRI = selective serotonin reuptake inhibitor
[1] All head injuries were mild and occurred at least one year prior to the time of this study.
[2] One participant was prescribed methadone for maintenance treatment of opioid dependency.
[3] Days 1-14 correspond with the follicular/ovulatory phases of the menstrual cycle, where peripheral OXT levels may be highest (Salonia et al., 2005).

Primary Study Measures

All study materials and questionnaires (described in the following sections) were at or below the 6th grade reading level, based on Microsoft Office Word readability software that derives the Flesch-Kincaid Grade Level.

Childhood Trauma Questionnaire (CTQ). Participants completed the CTQ (Bernstein & Fink, 1997), a 28-item retrospective self-report rating scale that measures five types of maltreatment: physical abuse, sexual abuse, emotional abuse, physical neglect, emotional neglect. Each item is rated on a 5-point scale assessing frequency of occurrence before the age of 18. Subscale scores range from 5 to 25 and a total maltreatment score can be derived by summing the five scaled scores (ranging from 25 to 125). The manual provides clinical cutoff scores for the presence of significant (i.e., at least "low" levels of) maltreatment for each subscale (i.e., >7 for physical abuse, >7 for physical neglect, >7 for sexual abuse, >9 for emotional abuse, and >14 for emotional neglect. The Flesch-Kincaid reading level of the CTQ is 4.8 (Flesch, 1948).

This scale is one of the most well-validated self-report measures of childhood

maltreatment, demonstrating strong internal consistency and criterion validity with therapists' ratings of maltreatment (see Baker & Maiorino, 2010 for review). Additionally, scores derived from the CTQ correlate with long-term structural and functional alterations in the limbic system of the brain (e.g., Dannlowski et al., 2012; Grant et al., 2015; Swartz et al., 2015; Teicher et al., 2014).

Inventory of Callous-Unemotional Traits (ICU). Participants' total score on the self-report version of the ICU (Frick, 2004) was used as the primary measure of CU traits in this study. The ICU contains 24 items that are rated on a 4-point rating scale from 0 (not at all true) to 3 (definitely true), with higher scores indicating greater degree of CU traits. Factor analyses (Fanti et al., 2009; Kimonis et al., 2008) have generally supported a three-bifactor model with three subscales: Callousness (9 items; $\alpha = .80$; "I do not care who I hurt to get what I want"), Unemotional (5 items; $\alpha = .62$; "I do not show my emotions to others"), and Uncaring (8 items; $\alpha = .83$; "I do not like to put the time into doing things well"). The ICU has demonstrated adequate internal consistency with incarcerated adolescents ($\alpha = .87$; Kimonis, Fanti, Isoma, & Donoghue, 2013) and adults ($\alpha = .80$; Byrd, Kahn, & Pardini, 2013; Kimonis et al., 2013). The construct and convergent validity of the ICU has been supported across undergraduate, community, and incarcerated samples based on correlations with self-report and official records of criminal activity (Byrd, Kahn, & Pardini, 2013; Fanti et al., 2009; Kimonis et al., 2008; Kimonis et al., 2013). The Flesch-Kincaid reading level for the ICU is 5.6. The internal consistency for the current study, estimated by Cronbach's alpha, was .90.

Reactive-Proactive Aggression Questionnaire (RPQ). The RPQ (Raine et al., 2006) includes 12 items measuring proactive aggression (i.e., goal-oriented, predatory aggression thought to occur with minimal autonomic arousal) and 11 items measuring reactive aggression

(i.e., emotionally provoked or impulsive aggression associated with disinhibition) rated on a 3-point scale. According to Raine and colleagues (2006), the Cronbach's alpha coefficient for the total score is .90, and is .86 and .84 for proactive and reactive aggression, respectively. This measure has also demonstrated adequate criterion, convergent, and discriminant validity (Raine et al., 2006). Participants' proactive aggression subscale score was included as a primary outcome variable in this study. The Flesch-Kincaid reading level for the RPQ is 3.5. In the current study, the internal consistency for the total scale was $\alpha = .91$, $\alpha = .86$ for the Reactive subscale, and $\alpha = .87$ for the Proactive subscale.

Salivary oxytocin (OXT). Saliva samples were taken using a passive drool collection method, where participants were instructed to pool saliva in their mouths without swallowing for approximately 1 minute before expectorating into a 2-mL cryovial using a straw-like collection material by Assay Designs. Because of the short half-life of OXT in human saliva (i.e., 4-10 minutes), participants were only allowed three minutes to provide at least 1-mL of saliva (Leng & Sabatier, 2016). This was the minimum volume required to run the samples in duplicate (n = 2 per sample). The time of day that samples were collected and the amount of time needed to provide a sample were recorded to be added as covariates. Saliva samples were immediately put on ice and transported in a cooler to the Behavioral Neuroscience Lab at ECU. Samples were stored at -79°C until they were shipped overnight on dry ice to the University of North Carolina's Stress and Health Research Lab in the Department of Psychiatry.

Salivary samples were first extracted then assayed in duplicate using a competitive OXT Enzyme Immunoassay (EIA) kit by Enzo Life Science (cat. # 900-153), using the protocol that was developed and validated for salivary OXT as previously described (Grewen, Davenport, & Light, 2010; Holt-Lunstad et al., 2008). The extraction step concentrated the sample 3.2 times to

reduce matrix interference and to ensure that OXT concentrations were above the lower limit for sensitivity (2.0 pg/mL). Of note, this procedure yields lower values than those provided with the same kit before it was updated in 2011 (e.g., Carter et al., 2007). Extraction efficiency was 93%, as determined by spiking with a known amount of hormone and extracting this amount with the other samples. OXT levels were quantified using the Enzo Life Science OT EIA as described by Grewen, Davenport, and Light (2010), where "the endogenous OXT competes for the OXT antibody binding sites with added OXT linked to alkaline phosphatase. After the overnight incubation at 4°C, the excess reagents were washed away and the bound OT phosphatase was incubated with substrate." This enzyme reaction generates a yellow color and is stopped after one hour. The optical density was read on a Sunrise plate reader (Tecan, Research Triangle Park, NC) at 405 nm, where the intensity of the color is inversely proportional to the amount of OXT. OXT concentration in picograms per milliliter (pg/ml) was calculated using an immunoassay software package supplied by the plate reader manufacturer, which plotted the optical density of each sample against a standard curve.

All samples were above the lower limit of sensitivity. The intra- and inter-assay variations for this assay, which were practically validated using control samples with known OXT concentration on each plate, were 4.8% and 8%, respectively. According to the manual, cross-reactivity for similar neuropeptides found in mammalian sera is less than 0.001.

Control and Ancillary Variables

Participants completed the following measures, which were used as ancillary or control variables in the current study. Variables that could potentially confound the results (based on prior research or theory) and that had statistically significant effects on any outcome variable were retained in the final mediation models.

Demographics. As shown in Appendix F, participants completed a demographics questionnaire (Flesch-Kincaid = 6.1) where they provided information on their care history as a child (e.g., the number of changes in caregiver, types of placements outside the home), relationship status, medical/psychiatric history, and the names of any medications or substances they had taken on the day of data collection. Female participants were asked about current oral contraceptive use and phase of menstrual cycle (i.e., number of days since last menstrual period), in light of potential menstrual cycle-related fluctuations in OXT (e.g., Salonia et al., 2005).

Recent stressful life events. Participants completed an inventory of recent life stressors (Appendix G; Flesch-Kincaid = 5.9) by endorsing any event that occurred over the past 12 months. Total number of recent stressors was included as a covariate in order to control any potential effects of recent stress on OXT (Seltzer, Zeigler, Connolly, Prososki, & Pollack, 2013). This questionnaire was adapted from the Holmes and Rahe Stress Scale (1967). Desirable events or items having positive valence (e.g., vacation) were removed.

Response validity. Response validity was assessed using two embedded measures. Cases with extreme scores on either validity scale were reviewed individually and evaluated to determine response consistency across questionnaires in order to determine whether cases would need to be removed.

CTQ Minimization Scale. Minimization or denial of childhood maltreatment was evaluated using the 3-item *Minimization Scale* of the CTQ (MacDonald et al., 2016). Higher scores on these items convey an exaggerated or "naively positive" depiction of one's childhood, such as "my family was the best in the world." Minimization scores are dichotomized so that scores of 5 on any item are coded as 1, and scores lower than 5 are coded as 0, yielding a maximum score of 3.

Behavioral Emotional and Executive Functioning Instrument (BEEF) Social Desirability Scale. Deceptive responding was also evaluated using four items from the *Social Desirability Scale* of the BEEF, a self-report 4-point Likert-type rating scale that was developed by the primary author in an unpublished manuscript to capture the affective, dysexecutive, and behavioral features of psychopathy. The embedded validity scale includes four items designed to detect virtuous responding (e.g., "I have never talked badly about another person") and overly exaggerated or deviant responding (e.g., "every time I get mad, I can hear my brain pounding like thunder").

Initial reliability, content validation, and exploratory factor analyses of the BEEF were completed in a sample of 504 undergraduate students. Analyses yielded six internally consistent factors that reflect clinically relevant aspects of psychopathy. Items assessing the affective features of psychopathy (i.e., CU traits) were distributed across three factors: Lack of Prosocial Emotions, Antagonism, and Punishment Insensitivity (e.g., fearlessness, disregard for consequences). Executive Dysfunction (e.g., disinhibition, impulsivity, proneness to boredom) items loaded onto one factor as hypothesized. Antisocial Behavior items loaded onto two factors: "Victimless" Risky Behaviors (e.g., substance abuse, skipping class) and Antisocial Behavior (e.g., criminal acts, aggression). Readability analysis yielded a Flesch-Kincaid grade level of 4.0. This measure is provided in Appendix H.

Criminal offense history. Conviction and sentence information for offenders was gathered from the NC Department of Public Safety (NCDPS) Offender Public Information website (http://webapps6.doc.state.nc.us/opi/offendersearch.do?method=list). Data were collected on participants' total number of offenses, crime classification, felony/misdemeanor status, and number of previous incarcerations. To synthesize the types of crime committed in

this sample, offenses were coded into one of four categories: aggressive crimes, property crimes, drug/alcohol crimes, and other consensual crimes. Aggressive crimes were defined as those involving using or threatening to use force with the intent to harm (e.g., assault, child abuse, use or possession of firearms). "Robbery with a firearm" was included in this category because it involves the use of force (i.e., a dangerous weapon) and thus meets criteria for felony assault, as was "indecent liberty with a child," which constitutes sexual abuse of a minor and is therefore considered a crime of violence according to N.C. General Statute § 14–202.1(a). Property crimes were defined as crimes relating to theft or destruction of someone else's property without the actual or threatened use of physical force. Drug and alcohol-related crimes comprised their own category. Consensual crimes included all other crimes that do not include unwilling victims (e.g., disorderly conduct, resisting an officer).

In the offender subsample, the median number of criminal offenses was 2.5 (M = 4.54, SD = 4.02, range = 1-14). Twenty-two of the 54 offenders (41%) had been incarcerated at least once (range = 0-5). As shown in Figure 2, assault was the most common crime committed in the sample, and 61% of participants had been charged with at least one aggressive crime. Out of the four categories of crime, however, property crimes were committed with the highest frequency, accounting for 32% of all crimes in the sample.

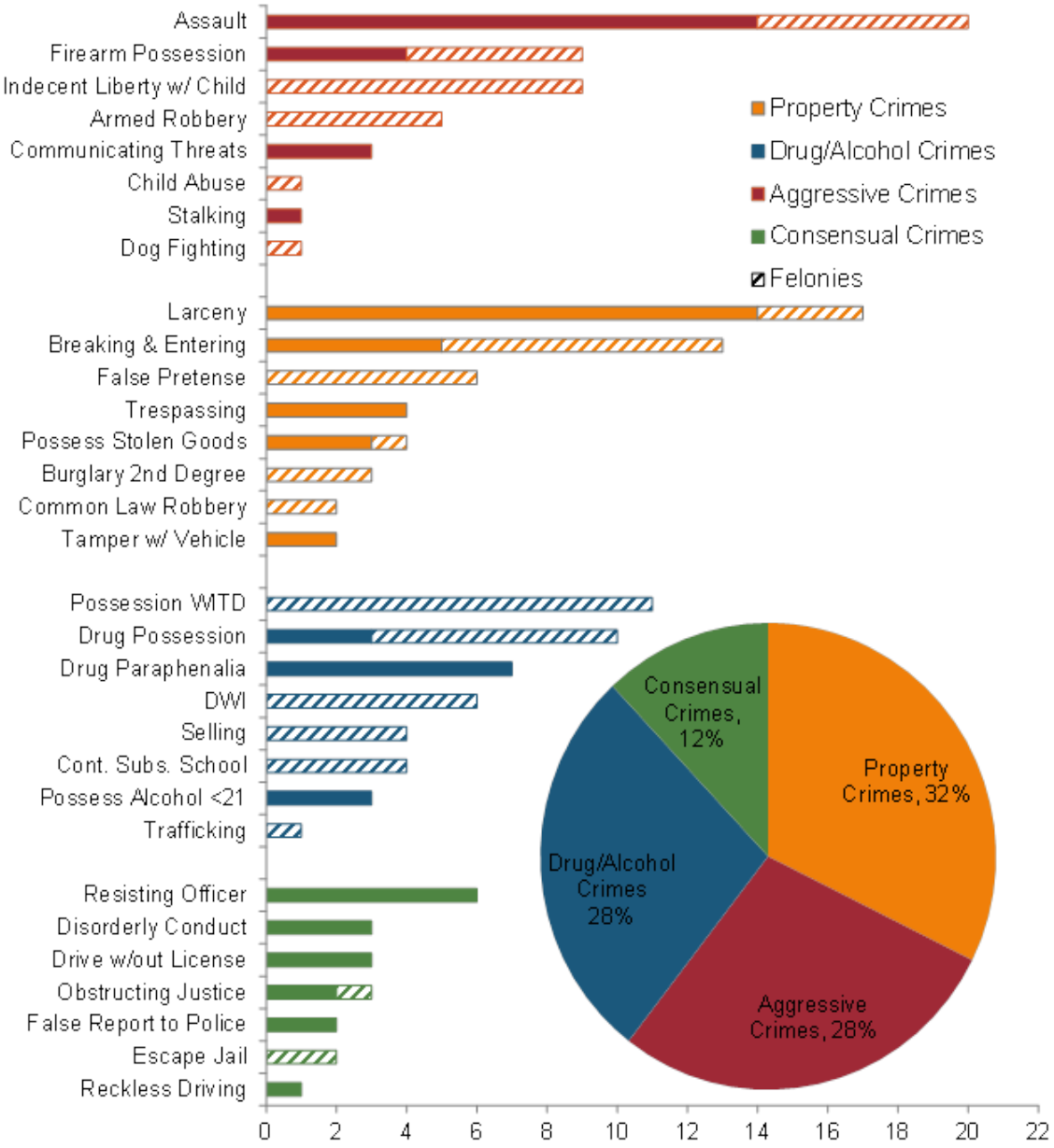

Figure 2. Types and classifications of crimes committed. The pie chart depicts the relative proportions of each category of crime. Bar charts show the total number of charges for each type of criminal offense by category. Solid bars represent misdemeanors and striped bars represent felonies.

Misdemeanor Assaults include simple assault and assault on a female. Felony Assault is defined as an unlawful attack upon another person for the purpose of inflicting severe or aggravated bodily injury, with either the intent to kill and/or resulting in serious injury. Drug charges (i.e., selling, trafficking) in this sample involved Schedule I, II, III, and IV drugs.

Procedures

All study procedures were approved by the University and Medical Institutional Review Board (UMCIRB) and NCDPS prior to any contact with potential participants (Appendices A and B). The PI was present for all data collection sessions, along with one to two trained graduate research assistants.

Data collection occurred at the locations from which participants were recruited. Participants from the offender subsample were recruited at their probation/parole office as they arrived for their scheduled office visit. Probation officers assisted in the identification of individuals from their caseloads who met the inclusionary criteria. An officer then informed them of their eligibility to participate in a research study and, if interested, led them to a conference room where the PI provided an overview of the study. Probationers were informed that their decision whether or not to participate would not affect their probation/parole terms in any way. It was emphasized that NCDPS employees would not have access to their responses. Only one probationer declined to participate. Participants from the non-offender subsample were scheduled in advance and completed the study in a private classroom in the Rawl building at ECU. Although not required, they were encouraged to hydrate beforehand and to avoid stimulant medications or other substances that could interfere with saliva production or consistency on the day of data collection.

Participants completed the study individually or in small groups (2-4 participants). All study procedures took approximately 30 minutes to complete. Data collection sessions were all held between 12:30pm and 5:30pm to account for diurnal variations in OXT (see Amico, Levin, & Cameron, 1989; White-Traut et al., 2009). The demographic questionnaire was completed first, and the remaining self-report measures were administered in a counterbalanced order across

groups of participants. As participants completed each measure, researchers immediately collected and reviewed their responses to ensure that all items were completed. Saliva samples were collected last to ensure that participants had not eaten for at least 30 minutes or consumed any liquids for at least 10 minutes before expectorating.

CHAPTER III: RESULTS

Data Screening and Preliminary Analyses

Four participants were unable to produce sufficient saliva samples within the allotted time period and were subsequently excluded from OXT analyses. One participant who endorsed breastfeeding and/or being pregnant was removed from analyses. One additional participant, who scored the maximum on the BEEF Social Desirability scale, was removed from analyses. Further investigation of this case revealed that this participant (from the offender subsample) had study materials read aloud to him due to reading difficulties, and there was evidence of an inconsistent response pattern across multiple measures. Inspection of the validity scales revealed that 21% of participants endorsed extreme scores on at least one on the CTQ Minimization Scale and 13% endorsed extreme scores on at least one validity item on the BEEF, though these scores were not correlated with any outcome variable. In total, 95 cases were included in the mediation analyses.

All analyses were conducted using SPSS statistical software (IBM SPSS Statistics Version 24.0). Raw data and scatterplots were inspected for missing data, normality, and linearity of relationships among all study variables. Research assistants detected the majority of missing data at the time of data collection. This prevented all but four instances of missing data. Because these observations appeared arbitrary and unpredictable, missing data were replaced with the median value of the respective subscale for that participant. Descriptive statistics and correlations for primary study variables are presented in Tables 3 and 4, respectively.

Sample characteristics and ancillary analyses. Data on types of maltreatment and placement changes participants experienced in childhood is presented in more detail in Figure 3. In this sample, 78% endorsed at least “low” levels of at least one type of maltreatment, as

defined by the CTQ manual, and 61% met criteria for multiple types of maltreatment. All types of maltreatment except sexual abuse were significantly inter-correlated. Overall, maltreatment and care histories were similar between groups, though non-offenders reported significantly greater levels of emotional abuse, $t(99) = 2.89$, $p = .005$, 95% CI [0.95, 5.12], and more recent stressful events ($M = 5.32$, $SD = 2.38$) than offenders ($M = 3.72$, $SD = 2.64$), $t(99) = 3.19$, $p = .002$, 95% CI [0.60, 2.60]. Similarly, non-offenders reported greater levels of reactive aggression than offenders, $t(99) = 2.04$, $p = .044$, 95% CI [0.05, 3.73].

Exploratory independent samples *t*-tests were run with sex as the grouping variable to examine group differences between men and women on primary study variables. Women reported higher levels of emotional abuse ($M = 13.85$; $SD = 4.87$) than men ($M = 9.55$; $SD = 4.87$), $t(99) = -4.16$, $p < .001$, 95% CI [-6.35, -2.25]. Additionally, men tended to have lower levels of OXT ($M = 16.40$; $SD = 8.19$) than women ($M = 19.42$; $SD = 8.29$), though this difference fell short of statistical significance, $t(94) = -1.75$, $p = .084$, 95% CI [-6.44, 0.41].

Table 3. Descriptive statistics for primary study variables by offender status

Variable	Non-offenders *M* (*SD*)	Offenders *M* (*SD*)	Total (*N* = 100) *M* (*SD*)	Skewness	*SE*	Kurtosis	*SE*
CTQ - Total	50.83 (15.70)	45.39 (16.79)	47.92 (16.44)	0.19	0.24	-1.24	0.48
CTQ - EA	12.83 (5.70)	9.80 (4.85)	11.21 (5.45)**	0.37	0.24	-1.22	0.48
CTQ – PA	8.64 (3.73)	9.15 (4.21)	8.91 (3.98)	0.77[a]	0.24[a]	-.51[a]	0.48[a]
CTQ – SA	Mdn = 5 (5.27)	Mdn = 5 (3.94)	Mdn = 5 (4.61)	2.37	0.24	4.84	0.48
CTQ - EN	12.45 (5.00)	11.67 (5.41)	12.03 (5.21)	0.16	0.24	-1.11	0.48
CTQ - PN	9.17 (3.99)	8.22 (4.29)	8.56 (4.16)	0.83	0.24	-0.52	0.48
OXT (*n* = 95)	21.85 (9.40)	14.10 (5.24)	17.57 (8.31)**	0.68	0.25	-0.19	0.49
ICU-Total	22.77 (8.36)	22.56 (9.22)	22.00 (8.83)	0.64	0.24	0.52	0.48
RPQ-Proactive	2.13 (2.17)	2.74 (2.45)	2.46 (2.34)	0.92	0.24	0.13	0.48
RPQ-Reactive	11.00 (5.11)	9.11 (4.20)	9.99 (4.72)*	0.24	0.24	-0.85	0.48
RPQ-Total	13.36 (6.61)	11.67 (6.18)	12.46 (6.40)	0.40	0.24	-0.53	0.48

Note. Medians are reported for skewed variables.
*t-test significant at the $p < .05$ level, ** $p < .01$
CTQ Total = Child Trauma Questionnaire Total Score; SA = Sexual Abuse; EA = Emotional Abuse; PA = Physical Abuse; EN = Emotional Neglect; PN = Physical Neglect; OXT = oxytocin (pg/mL); ICU-Total = Inventory of Callous-Unemotional Traits-Total Score; RPQ-Proactive = Reactive Proactive Aggression Questionnaire-Proactive Aggression Subscale; RPQ-Total = Reactive Proactive Aggression Questionnaire-Total Score
[a] Values reflect square root transformed scores.

Table 4. Zero-order correlations for primary study variables

	1	2	3	4	5	6	7	8	9	10
1. CTQ-Total	--									
2. CTQ-EA	.831**	--								
3. CTQ-PA	.635**	.383**	--							
4. CTQ-PN	.810**	.629**	.438**	--						
5. CTQ-EN	.854**	.696**	.487**	.711**	--					
6. CTQ-SA[a]	.323**	.133	.118	.138	.116	--				
7. OXT (*n* = 95)	-.130	-.151	-.006	-.217*	-.223*	.112	--			
8. ICU-Total	.367**	.309**	-.188	.396**	.524**	-.029	-.397**	--		
9. RPQ-Total	.398**	.408**	.258**	.307**	.447**	-.041	-.196	.506**	--	
10. RPQ-Proactive	.325**	.319**	.213*	.327**	.441**	-.116	-.457**	.521**	.737**	--
11. RPQ-Reactive	.356**	.340**	.213*	.248*	.379**	.019	-.119	.478**	.908**	.500**

Note. *p<.05, **p<.01. N = 100 unless reported otherwise. To correct for positive skew, correlations were calculated using square root transformed scores for physical abuse, and winsorization procedures for sexual abuse. CTQ-Total = Child Trauma Questionnaire-Total Score; SA = Sexual Abuse; EA = Emotional Abuse; PA = Physical Abuse; EN = Emotional Neglect; PN = Physical Neglect; SA = Sexual Abuse; OXT = oxytocin (pg/mL); ICU-Total = Inventory of Callous-Unemotional Traits-Total Score; RPQ-Proactive = Reactive Proactive Aggression Questionnaire-Proactive Aggression Subscale; RPQ-Total = Reactive Proactive Aggression Questionnaire-Total Score
[a] Kendall's tau (τ_b) correlation coefficients are reported for correlations with sexual abuse.

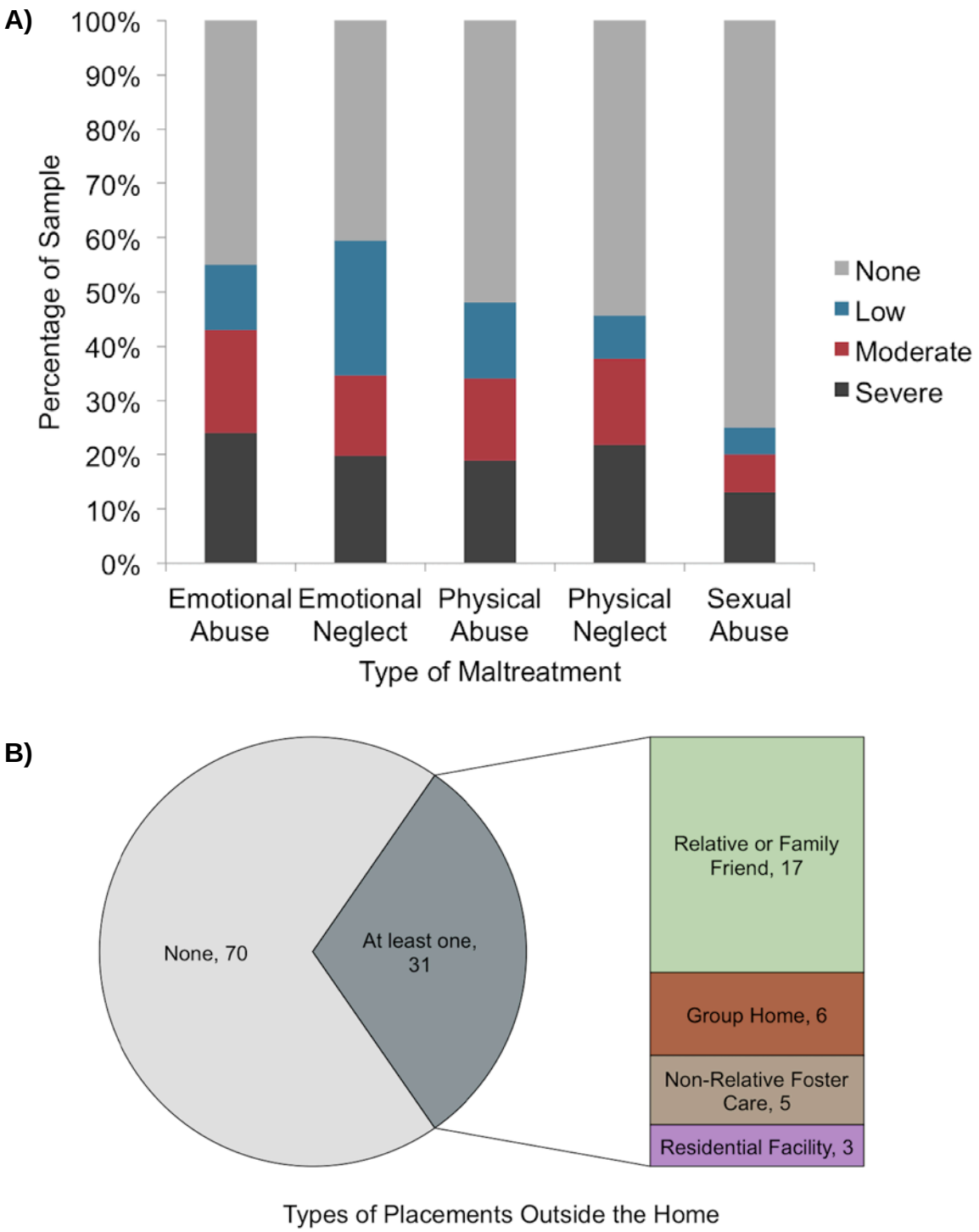

Figure 3. Care history of participants. A) Stacked columns depicting the percentage of participants who experienced low, moderate, and severe degrees of each type of maltreatment (Bernstein & Fink, 1998). B) Types of out-of-home placements or caregiver changes during childhood that were endorsed by participants.

Bivariate Hypotheses

Hypothesis 1. Hypothesis 1 predicted that salivary OXT would be inversely associated with CU traits and proactive aggression, but not reactive aggression. As predicted, OXT had medium-to-large size relations with self-reported CU traits and proactive aggression. Participants with lower levels of OXT reported significantly higher levels of CU traits, r = -.397, p = < .001, and proactive aggression, r = -.457, p < .001. Additionally, the association between OXT and reactive aggression was small and nonsignificant, r = -.119, p = .247, as expected.

Exploratory analyses were run to investigate the role of low OXT among other indicators of antisocial behavior. An independent samples *t*-test revealed that salivary OXT was significantly lower for offenders than non-offenders, $t(67) = 4.83$, $p < .001$. Because of great heterogeneity of variance between groups for OXT ($F = 10.09$, $p = .002$), degrees of freedom were adjusted downward from 93 to 64. Among the offender subsample, an exploratory analysis was also run to investigate whether number of aggressive offenses was inversely correlated with OXT, though this association was nonsignificant, r = -.110, p = .441. A square root transformation was used to correct for positive skewness for number of aggressive offenses.

Hypothesis 2. It was also hypothesized that childhood maltreatment would be inversely related to OXT. As shown in Table 4, the zero-order correlation between total childhood maltreatment and OXT fell short of statistical significance, r = -.130, p = .143. However, OXT was significantly negatively correlated with physical neglect, r = -.217, p = .034, and emotional neglect, r = -.223, p = .029. The relations between OXT and all other forms of maltreatment were nonsignificant.

Mediation Models

All mediation models were tested with and without outliers, with no substantial

differences in results. To reduce the likelihood of epiphenomenal or spurious associations that could account for the observed effects, mediation models were also tested with and without covariates. Potential covariates were selected based on their clinical relevance and plausible effects on an outcome variable. The number of covariates was reduced using backward elimination, such that covariates were only retained in the model if their effects were statistically significant.

Hypothesis 3. The primary hypothesis stated that OXT and CU traits would serially mediate the relationship between total maltreatment and proactive aggression. All unstandardized path coefficients for the PROCESS model are shown in Figure 4 and Table 5. These values are reported in text unless specified otherwise. Sex, offender status, and recent stressors were retained as covariates in this mediation model because of their significant unique effects on a mediator or outcome variable. Accounting for both mediators (unstandardized total indirect effect = 0.026, 95% BC CI [0.011, 0.046]) reduced the direct effect of childhood maltreatment on self-reported proactive aggression to a nonsignificant level (c' = 0.026, p = .051, 95% BC CI [-0.001, 0.052), which supports full mediation. As shown in Table 6, the predicted serial mediation model in Hypothesis 3 was supported because the bootstrapped confidence interval for the specific indirect effect through both mediators excluded zero ($a_1a_3b_2$ = 0.003, 95% BC CI [0.001, 0.009]). Specifically, participants who reported greater levels of childhood maltreatment generally had lower levels of salivary OXT, which in turn was associated with higher CU traits, and ultimately more proactive aggression. The total model explained 48% of variance in proactive aggression. To aid in the interpretation of relative strength of each path, standardized coefficients are also provided in Table 5.

Pairwise comparisons of each specific indirect effect in the model are provided in Table

6. The remaining specific indirect effects were also statistically significant. When comparing the strength of each mediator, contrasts show that the indirect effects of OXT and CU traits are similar in magnitude; however, when comparing the strength of the serial mediation model to the two individual mediation paths for OXT and CU traits, both single mediation models were observed to have stronger mediation effects than the serial mediation model in Hypothesis 3.

Table 5. Unstandardized and standardized regression coefficients, standard errors, and model summary data for the serial mediation model in Hypothesis 3.

		Consequent													
		M_1 (OXT)					M_2 (CU Traits)					Y (Proactive Aggression)			
Antecedent		β	Unstd.	*SE*	*p*		β	Unstd.	*SE*	*p*		β	Unstd.	*SE*	*p*
X (Total Maltreatment)	a_1	-0.23	-0.12	0.05	.021	a_2	0.35	0.19	0.05	.000	c'	0.18	0.03	0.01	.051
M_1 (OXT)		--	--	--	--	a_3	-0.37	-0.40	0.11	.000	b_1	-0.38	-0.10	0.03	.001
M_2 (CU Traits)		--	--	--	--		--	--	--	--	b_2	0.24	0.06	0.03	.012
C_1 (Recent Stressors)	f_1	0.04	0.14	0.30	.655	g_1	0.17	0.59	0.32	.063	b_1	0.31	0.27	0.08	.000
C_2 (Sex)	f_2	0.06	1.0	1.76	.533	g_2	-0.24	-4.87	1.83	.017	b_2	-0.17	-0.81	0.44	.068
C_3 (Offender Status)	f_3	-0.46	-7.69	1.70	.000	g_3	-0.08	-1.40	1.95	.462	b_3	0.02	0.11	0.46	.813
Constant	i_{M1}	--	25.22	3.49	.000	i_{M2}	--	24.09	4.53	.000	i_Y	--	1.39	1.22	.257
			R^2 = .262					R^2 = .340					R^2 = .477		
			$F(4, 91) = 8.08$, $p < .001$					$F(5, 90) = 9.28$, $p < .001$					$F(6, 89) = 13.52$, $p < .001$		

Note. β = standardized coefficients; Unstd. = unstandardized coefficients.
Standard errors and *p* values are provided for unstandardized regression coefficients. Standardized (β) coefficients are also provided as a unit-free index to determine relative strength of each path.

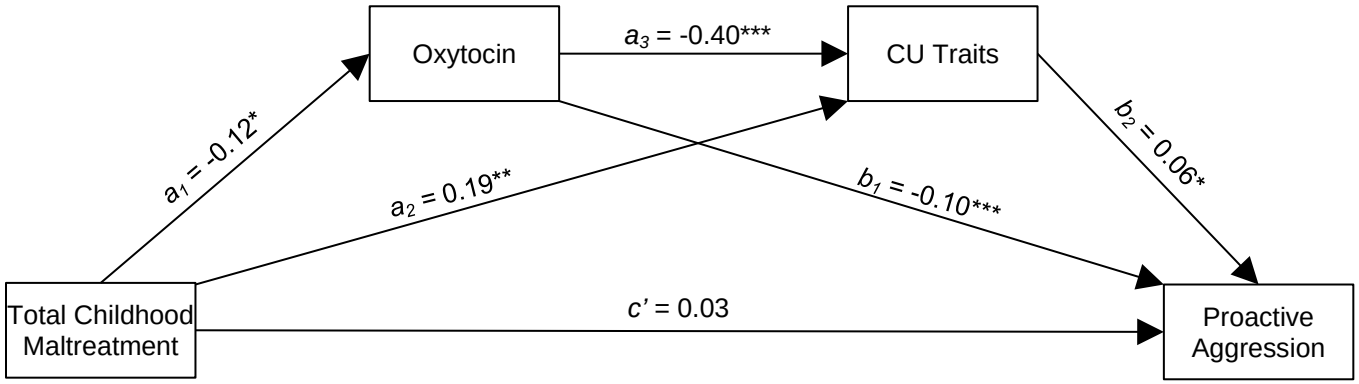

Figure 4. Serial mediation model in Hypothesis 3. Unstandardized regression coefficients are used to label the paths in this diagram.
*$p < .05$, **$p < .01$, ***$p < .001$

Table 6. Comparison of the indirect effects of OXT and CU traits in the relationship between total maltreatment and proactive aggression.

	Product of Coefficients				
Effects	β	Unstd.	*SE*	BootLLC	BootULCI
Total Indirect Effects	.184	.026	.009	.012	.046
(a_1b_1) Mal → OXT → Pro. Aggression	.078	.011	.005	.003	.026
($a_1a_3b_2$) Mal → OXT → CU → Pro. Aggression	.021	.003	.002	.001	.009
(a_2b_2) Mal → CU → Pro. Aggression	.086	.012	.005	.004	.026
Contrasts					
Model 1 (a_1b_1) minus Model 2 ($a_1a_3b_2$)	.057	.008	.005	.001	.022
Model 1 (a_1b_1) minus Model 3 (a_2b_2)	-.008	-.001	.005	-.016	.015
Model 2 ($a_1a_3b_2$) minus Model 3 (a_2b_2)	-.065	-.009	.005	-.022	-.002

Notes. $N = 95$, $k = 10{,}000$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.
β = standardized coefficients; Unstd. = unstandardized coefficients; BootLLCI = bootstrapping lower limit confidence interval; BootULCI = bootstrapping upper limit confidence interval.
Standard errors and CIs reflect unstandardized values. For contrasts, CIs that do not include zero indicate that the indirect effects of the variables are significantly different from each other.

Hypothesis 4. Hypothesis 4 stated that the indirect effects of OXT and CU traits would be stronger in serial mediation models of the effects of emotional abuse and emotional neglect, relative to physical abuse and physical neglect. Unstandardized path coefficients for each specific form of maltreatment are shown in the Table 7, and standardized coefficients are provided in Tables 8 and 9. All models included the same covariates to assist with comparison of the size of paths within and across models. Covariates were selected based on their unique effect in at least one model, and included the following: the other four specific forms of maltreatment, number of changes in caregiver, recent stressors, number of psychiatric disorders, offender status, sex, relationship status, and phase of menstrual cycle for female participants. The total model, including all covariates, accounted for 50.1% of the variance in proactive aggression, $F(13, 82) = 6.322$, $p < .001$.

Emotional abuse was the only specific form of maltreatment that influenced proactive aggression indirectly through OXT and CU traits in serial to a statistically significant degree. Serial mediation was supported because the confidence interval for the final specific indirect

effect included zero ($a_1a_3b_2$ = 0.010, 95% BC CI [0.001, 0.038]). When comparing the strength of each specific indirect effect, the path through OXT in isolation was significantly stronger than the individual path through CU traits, based on the 95% CIs [0.006, 0.123] for this pairwise comparison excluding zero.

In contrast, the indirect path from emotional neglect to proactive aggression through OXT and CU traits in serial was not statistically significant ($a_1a_3b_2$ = 0.006, 95% BC CI [0.000, 0.029]), so there was insufficient support for serial mediation. Both specific indirect effects through OXT and CU traits in isolation also fell short of statistical significance. Further examination of the individual paths in Tables 7 and 9 reveal that emotional neglect uniquely predicted CU traits, but not OXT.

The standardized indirect effects in Table 8 allow better comparison of the magnitude of indirect effects across each model. Based on standardized point estimates of the specific indirect effect through both mediators, Hypothesis 4 was only partially supported. Emotional abuse yielded the only statistically significant serial mediation effect (β = .024), though point estimates of this indirect effect were larger in the models with physical abuse (β = -.013) and sexual abuse (β = -.011) than they were for emotional neglect (β = .004), which had the smallest indirect effect.

Individual paths from each form of maltreatment were explored in more detail by examining standardized regression weights (βs) to index the strength of each predictor on OXT, CU traits, and proactive aggression (Table 9). Given the shared variance among each specific form of maltreatment (e.g., large correlations between emotional neglect and physical neglect, r = .711, p < .001), semi-partial correlation coefficients (sr) were also calculated to estimate the unique effect size of each predictor, as these values are less influenced by multicollinearity.

Accordingly, Table 9 compares the unique contributions of each predictor, mediator, and covariate to all subsequent outcomes in the model.

Table 7. Unstandardized path coefficients and standard errors from serial mediation models for each type of maltreatment

Path	Emotional Abuse Model	Emotional Neglect Model	Physical Abuse Model	Physical Neglect Model	Sexual Abuse Model
Direct effect (*c'*)	0.069 (0.061)	0.062 (0.058)	0.014 (0.056)	-0.031 (0.067)	-0.055 (0.051)
95% CI	-0.053, 0.191	-0.054, 0.178	-0.098, 0.126	-0.165, 0.103	-0.158, 0.047
a_1	-0.619 (0.213)	-0.374 (0.207)	0.499 (0.202)	-0.221 (0.241)	0.421 (0.180)
95% CI	-1.042, -0.195	-0.784, 0.037	0.015, 0.900	-0.699, 0.258	0.063, 0.780
a_2	-0.136 (0.239)	0.914 (0.217)	-0.102 (0.228)	0.047 (0.271)	-0.289 (0.205)
95% CI	-0.610, 0.339	0.484, 1.345	-0.555, 0.352	-0.491, 0.586	-0.695, 0.118
a_3	-0.277 (0.117)	-0.299 (0.110)	-0.286 (0.118)	-0.308 (0.121)	-0.286 (0.118)
95% CI	-0.509, -0.045	-0.519, -0.080	-0.520, -0.051	-0.548, -0.067	-0.521, -0.052
b_1	-0.076 (0.031)	-0.082 (0.028)	-0.085 (0.030)	-0.080 (0.031)	-0.084 (0.030)
95% CI	-0.137, -0.015	-0.138, -0.026	-0.145, -0.025	-0.141, -0.018	-0.144, -0.024
b_2	0.058 (0.028)	0.051 (0.026)	0.050 (0.027)	0.055 (0.027)	0.054 (0.027)
95% CI	0.002, 0.113	-0.001, 0.104	-0.003, 0.103	0.002, 0.109	0.001, 0.107
Indirect effects					
a_1b_1	0.047 (0.027)	0.031 (0.029)	-0.042 (0.025)	0.018 (0.023)	-0.035 (0.021)
95% CI	0.007, 0.114	-0.04, 0.108	-0.103, -0.007	-0.020, 0.075	-0.093, -0.005
$a_1a_3b_2$	0.010 (0.008)	0.006 (0.006)	-0.007 (0.006)	0.004 (0.006)	-0.006 (0.006)
95% CI	0.001, 0.038	0.000, 0.029	-0.028, 0.000	-0.003, 0.024	-0.027, 0.000
a_2b_2	-0.008 (0.017)	0.047 (0.028)	-0.005 (0.016)	0.003 (0.019)	-0.016 (0.017)
95% CI	-0.053, 0.017	-0.001, 0.113	-0.053, 0.017	-0.030, 0.051	-0.067, 0.004
Total indirect effect	0.046 (0.030)	0.083 (0.047)	-0.054 (0.033)	0.024 (0.035)	-0.057 (0.031)
95% CI	-0.001, 0.135	-0.011, 0.197	-0.130, -0.004	-0.035, 0.109	-0.139, -0.008

Note. Standard errors are in parentheses.

Table 8. Standardized coefficients for indirect paths from each type of maltreatment to proactive aggression

Path	Emotional Abuse Model	Emotional Neglect Model	Physical Abuse Model	Physical Neglect Model	Sexual Abuse Model
Specific Indirect Effects					
a_1b_1	.116	.019	-.063	.037	-.054
95% CI	.017, .266	-.080, .189	-.165, -.005	-.032, .154	-.153, -.002
$a_1a_3b_2$	.024	.004	-.013	.008	-.011
95% CI	.001, .089	-.014, .051	-.054, .000	-.003, .051	-.048, .000
a_2b_2	-.008	.101	-.005	.016	-.013
95% CI	-.102, .056	-.007, .249	-.089, .042	-.031, .126	-.104, .019
Direct Effect (*c'*)	.137	.166	.040	-.056	-.083
95% CI	-.154, .428	-.127, .458	-.159, .238	-.307, .195	-.272, .106

Note. Covariates include all other forms of maltreatment, # of changes in caregiver, recent stressors, # of psychiatric diagnoses, offender status, sex, relationship status, and phase of menstrual cycle for female participants.

Table 9. Comparison of the magnitude of all individual paths included in models of specific forms of maltreatment using standardized coefficients and semi-partial correlations

		OXT			CU Traits			Proactive Aggression	
		β	*sr*		β	*sr*		β	*sr*
Predictor Variables									
Emotional Abuse	a_1	-.423**	-.237**	a_2	-.038	-.020	*c'*	.137	.073
Emotional Neglect	a_1	-.070	-.040	a_2	.506***	.289***	*c'*	.166	.088
Physical Abuse	a_1	.231*	.187*	a_2	.080	-.019	*c'*	.040	.031
Physical Neglect	a_1	-.136	-.085	a_2	-.064	.049	*c'*	-.056	-.035
Sexual Abuse	a_1	.197	.166*	a_2	-.077	-.053	*c'*	-.083	-.068
Mediators									
OXT		--	--	a_3	-.283*	-.211*	b_1	-.274*	-.197*
CU Traits		--	--		--	--	b_2	.200	.149
Covariates									
Recent Stressors		.102	.089		.135	.117		.285**	.243**
Offender Status		-.487***	-.404***		-.037	-.027		.036	.026
Sex		.097	.069		-.107	-.076		-.135	-.095
Menstrual Cycle		-.036	-.032		-.045	-.040		-.047	-.042
# Psych Diagnoses		.244**	.226**		-.150	-.133		-.145	-.127
# Caregiver Changes		.013	.011		-.143	-.117		.030	.024
Relationship Status		-.188	-.160		-.041	-.034		.097	.081

Note. β = standardized partial coefficient; *sr* = semi-partial correlations.
Each column represents one linear regression model where all predictor variables, including covariates, are listed in the rows. *sr* represents the correlation between the outcome variable and unique aspects of the predictor variable, and may be interpreted using Cohen's (1992) heuristics for small (r = .10), medium (r = .30), and large (r = .50) correlations. Discrepancies in the values of β and *sr* reflect the degree of multicollinearity between predictors, which is reflected through inflated estimates of β.

Alternative Mediation Models Predicting Proactive Aggression

To further examine the specificity of the serial mediation model of the effects of emotional abuse on proactive aggression through OXT and CU traits as serial mediators in that

order, alternative mediation analyses were tested where the order of the mediators were reversed (i.e., emotional abuse → CU traits → OXT → proactive aggression) and where OXT was entered as the predictor variable (i.e., OXT → CU traits → emotional abuse → proactive aggression). All covariates from previous models were included in these models.

Reversing the order of the two mediators significantly modified the strength of the indirect effects, to the extent that the specific indirect effect through CU traits and OXT in serial was no longer statistically significant ($a_1a_3b_2$ = 0.001, *SE* = 0.005, 95% BC CI [-0.008, 0.042]). The only specific indirect effect that was statistically significant in this model was the one through OXT in isolation (a_2b_2 = 0.048, *SE* = 0.026, 95% BC CI [0.008, 0.112]).

Furthermore, when OXT was entered as the predictor variable, and CU traits and emotional abuse were entered as serial mediators in that order, the specific indirect effect through both CU traits and emotional abuse fell short of statistical significance ($a_1a_3b_2$ = -0.000, *SE* = 0.015, 95% BC CI [-.001, .006]), so serial mediation was not supported. Additionally, each individual specific indirect path through both mediators in isolation were also nonsignificant (a_1b_1 = -0.015, *SE* = 0.010, 95% BC CI [-0.041, 0.001], a_2b_2 = -0.008, *SE* = 0.010, 95% BC CI [-0.032, 0.010]), while the direct path from OXT to proactive aggression was statistically significant (*c'* = -0.079, *SE* = 0.031, 95% BC CI [-0.141, -0.017]).

CHAPTER IV: DISCUSSION

Summary of Results and Relevant Implications

This study was an exploratory investigation of the potential mediating roles of OXT and CU traits in the relationship between childhood maltreatment and proactive aggression in two samples (i.e., probationers/parolees and university students) who were at risk for problems with proactive aggression. The secondary aim was to determine whether the indirect effects of OXT and CU traits on proactive aggression were stronger for certain forms of maltreatment relative to others. Specifically, it was hypothesized that these effects would be strongest for emotional abuse and neglect.

Salivary OXT and psychopathic traits. As expected, individuals with elevated CU traits and higher levels of proactive aggression tended to have lower concentrations of OXT in saliva. These findings are consistent with the results of other empirical studies, and highlight the contribution of reduced peripheral levels of OXT in the development of psychopathic traits. The strong biological basis for these characteristics is well established, though the role of peripheral levels of OXT in these outcomes is only beginning to be understood.

Maltreatment and salivary OXT. Zero-order correlations revealed that OXT was significantly negatively associated with physical and emotional neglect, while correlations with total maltreatment and all three types of abuse fell short of statistical significance. However, in the primary serial mediation model, the regression path from total maltreatment to OXT was statistically significant when controlling for other variables. When examining what other variables affected the strength of this relationship, it was revealed that offender status had a medium to large sized effect on OXT (sr = .487; d = 1.02), and actually predicted the greatest amount of variance in OXT compared with all other predictors in the model. Additionally,

number of reported psychiatric diagnoses was uniquely associated with OXT (*sr* = .226).

The existing literature on the association between maltreatment and OXT has also yielded complex, and at times, inconsistent results (e.g., Bhandari et al., 2014). Inter-individual differences (e.g., biological sex) and specific patterns of maltreatment (e.g. subtype, severity, onset, and chronicity) represent other potential sources of heterogeneity. For example, there is strong evidence that early-onset emotional maltreatment (i.e., from infancy to toddlerhood) predicts conduct problems and aggression, while early-onset physical neglect predicts emotional problems and withdrawn behavior in childhood and adolescence (Keiley, Howe, Dodge, Bates, & Pettit, 2001; Manly, Kim, Rogosch, & Cicchetti, 2001). Additionally, two studies by Kaplow and colleagues (Kaplow, Dodge, Amaya-Jackson, & Saxe, 2005; Kaplow & Widom, 2007) found that maltreatment that occurred before the age of 5 years was a much stronger predictor of psychiatric diagnoses in adulthood than was maltreatment at later stages. Thus, future studies should obtain more detailed information on maltreatment to determine the periods of development it occurred, which subtype(s), and at what severity or frequency.

Serial mediation model for total maltreatment. The findings of the current study provide support for the hypothesized serial mediation model where childhood maltreatment exerts its influence on proactive aggression indirectly through OXT and CU traits in serial. The total model accounted for 48% of the variance in proactive aggression. Furthermore, these results are consistent with full mediation, to the extent that maltreatment no longer exerted a statistically significant effect on proactive aggression when the effects of OXT and CU traits were accounted for. When comparing the serial mediation model with single mediation models through OXT and CU traits separately, both individual mediation paths were stronger than the combined path, and the effects of OXT and CU traits were similar in magnitude.

Differential effects of specific forms of maltreatment. Separate mediation models were examined for each form of maltreatment as the predictor variable, OXT and CU traits as serial mediators, and proactive aggression as the outcome variable, in order to investigate whether they each influence proactive aggression through the same mechanisms. The shared variance between each type of maltreatment likely confounded the comparison of each model separately; however, results supported serial mediation in the model with emotional abuse. Specifically, the association between emotional abuse and proactive aggression was fully explained by low levels of OXT and elevated CU traits, which operated in serial.

It is notable that different forms of maltreatment differentially affected OXT. Although the unique associations between emotional neglect and physical neglect with OXT fell short of statistical significance, the directions of these correlations were negative, which is consistent with that of emotional abuse and OXT. In contrast, however, physical abuse and sexual abuse were associated with significantly higher levels of salivary OXT after controlling for all other variables.

These findings provide further evidence that emotional forms of maltreatment, as well as neglect, affect OXT and emotional development in a distinct manner from physical abuse, and these types of maltreatment yield two different pathways to aggression, accordingly. Consistent with prior theory (Lee & Hoaken, 2007), physical abuse may increase the risk of reactive aggression by resulting in problems with emotional regulation, while emotional abuse and associated experiences of social-emotional deprivation may predispose individuals to CU traits and proactive aggression as a result of experience-dependent synaptic reorganization and alteration of the oxytocinergic system.

Emotional maltreatment, including neglect, is more common than physical and sexual

forms of maltreatment (USDHHS, 2004) and some evidence suggests it may actually have more detrimental effects on development (Hildyard & Wolfe, 2003). In relation to CU traits and psychopathy, emotional neglect has been particularly understudied despite its associations with affective deficits (e.g., shallow or blunted affect, lack of concern for others, strong attachments) that are similar to those in psychopathy (Sonuga-Barke, Schlotz, & Kreppner, 2010). Studies that have examined this relationship support the notion that emotional deprivation precipitates psychopathic traits. For example, Farrington and colleagues found that childhood physical neglect predicted psychopathy scores on the PCL-R through middle age (Farrington, Ullrich, & Salekin, 2010). Additionally, Krischer and Sevecke (2008) found that psychopathy was associated with emotional–but not physical–neglect in a sample of incarcerated adolescents using the same measure of maltreatment used in the present study.

Strengths, Limitations, and Directions for Future Research

This study was intended as an exploratory investigation of one theoretical model of the processes by which maltreatment increases the risk of proactive aggression. Although the indirect effects of maltreatment–particularly in the form of emotional abuse–on proactive aggression through OXT and CU traits in serial were statistically significant, this path should be interpreted as only one of many possible mechanisms leading to proactive aggression. Existing evidence strongly suggests the presence of multiple pathways from childhood maltreatment to delinquent outcomes in adulthood, with insecure attachment, poor parental mental health, poverty, and community violence all recognized as precursors to aggression (e.g., Jaffee, Caspi, Moffitt, & Taylor, 2004).

Although mediation analyses using cross-sectional data cannot convincingly demonstrate mediation or causality, this type of analysis provided an opportunity to compare the hypothesized

serial mediation model to alternative pathways to proactive aggression, by specifying a different temporal order. There was no evidence of mediation when the model was respecified so that OXT preceded emotional abuse, which provides greater support for the proposed direction of the mediation paths. Furthermore, serial mediation was also not supported when only the temporal order of the mediators was reversed (i.e., emotional abuse → CU traits → OXT → proactive aggression). Accordingly, the directionality of the pathways modeled in this study were supported. Although experimental studies of the deleterious impact of maltreatment in humans cannot be conducted for obvious ethical reasons, prospective studies using objective measures of maltreatment have provided more robust evidence that exposure to maltreatment precedes the onset of mental health problems (e.g., Horwitz, Widom, McLaughlin, & White, 2001). Early evidence also indicates that cumulative exposure to maltreatment in childhood reduces expression of the OXT receptor gene through epigenetic mechanisms, and that these changes are not present in early childhood before the onset of maltreatment (Dadds et al., 2014).

This study addressed several limitations to the existing literature. First, this is the only study to date to evaluate the indirect effects of OXT and CU traits within the maltreatment-proactive aggression relationship. Another strength of this study was the inclusion of two samples of young adults, who are more representative of the typical populations of individuals with histories of maltreatment than many of the extreme samples (e.g., murderers) in prior published studies examining similar relationships (e.g., Raine et al., 2006). The participants in this study had diverse histories of maltreatment, placement changes, and criminal backgrounds, and thus are more likely to represent the variability in outcomes of maltreatment. The inclusion of an offender sample also permitted a higher base rate of the variables of interest, given that the prevalence of psychopathy is 16% among adult male offenders as compared to 1% among non-

institutionalized adult men (Hare, 1996).

The current findings should be interpreted in light of several methodological limitations. Like the majority of other published studies on the outcomes of maltreatment, this study relied on retrospective self-report measures of maltreatment, which are subject to recall bias. Future research should incorporate longitudinal designs to provide stronger evidence for the causal direction of the effects observed in the current study. However, evidence suggests scores on the CTQ are stable over time, even in the context of reduced psychopathology after therapy (Paivio, 2001). Underreporting of aggression and CU traits is also a reasonable concern where the participant is the sole informant, particularly in criminal samples, who may be more motivated to present themselves in a more positive light. This concern may be warranted in the current study in light of 1) the small and nonsignificant association between criminal records of aggressive offenses and self-reported aggression among the offenders (r = .21, p = .137), and 2) the finding that the undergraduate sample reported slightly higher levels of reactive aggression than the offenders did. Nonetheless, participants' responses on validity scales were not suggestive of obvious overly virtuous responding. A meta-analysis by Ray and colleagues (2012) found that the influence of socially desirable responding on self-report measures of psychopathy is small, and the self-report measures that were used to measure CU traits and aggression have strong reliability (Cima et al., 2013; Falkenbach et al., 2003; Raine et al., 2006; Vahl et al., 2014). Additionally, self-report measures of CU traits also have the advantage of assessing internal motivations (e.g., using charm to manipulate others) and emotions (e.g., guilt) that are best understood by the individual (Colins, Bijttebier, Broekaert and Andershed, 2014; Colins, Grisso, Mulder and Vermeiren, 2015; Raine et al., 2006).

The measurement of OXT represents an additional factor that limits the generalizability

of these findings. OXT concentrations across the majority of published studies are not directly comparable due to measurement in different biological fluids (e.g., saliva, plasma) and different extraction and assay procedures that yield different results. Thus, there are no normative data for salivary OXT to date. Furthermore, measurements of OXT in biological fluids outside the central nervous system, which have relatively low concentrations of OXT, are subject to multiple sources of error, including cross-reactivity of other molecules (Leng & Sabatier, 2016). Nonetheless, the extraction and assay procedures used in the present study are currently the recommended standard for peripheral OXT, and recent studies have supported the reliability of these values (e.g., Feldman, Gordon, & Zagoory-Sharon, 2010; White-Traut et al., 2009; Weisman, Zagoory-Sharon, & Feldman, 2012).

In the current study, offender status exerted a statistically significant effect on all models. It is unclear what underlying construct this actually reflects, as the two subsamples differed across several factors, including sex, age, education, and likely SES, though this was not measured in the current study. Additionally, sex significantly influenced OXT in the model of total maltreatment. Future studies should evaluate whether the indirect effects of OXT and CU traits are conditional on certain contextual and inter-individual factors, including biological sex, SES, and parental psychopathic traits. Additionally, it would be of clinical value for future research to evaluate the impact of interventions or quality of placement changes after maltreatment to evaluate whether these factors protect against maltreatment-related changes in OXT and psychopathic traits.

Clinical Implications and Conclusions

These findings illustrate the cascading effects of childhood maltreatment and underscore the importance of prevention and early intervention strategies to attenuate the negative outcomes.

With respect to prevention, the Triple P (Positive Parenting Program) has been effective in reducing perpetration of maltreatment and out-of-home placements (Prinz, Sanders, Shapiro, Whitaker, & Lutzker, 2009). For instance, if aggression is manifested via different pathways in emotionally abused versus physically abused children, or for children with low versus high OXT and CU traits, optimal interventions should specifically target these pathways. In addition, differences in neurocognitive and/or emotional processes may warrant different approaches to intervention (Fishbein, Hyde, Coe, & Paschall, 2004). Low OXT's unique association with CU traits and proactive aggression, but not reactive aggression, adds to the existing evidence that the underlying mechanisms for these types of aggression are different, and their treatments should be different, accordingly.

Clinical studies demonstrating the prosocial effects of OXT in individuals with autism spectrum disorder (e.g., Kimura, Tanizawa, Mori, Brownstein, & Okayama, 1992) have attracted attention on the potential therapeutic benefits of OXT for individuals with CU traits and psychopathy (e.g., Rice & Derish, 2015), though no clinical studies have been published to date. Findings of this study suggest that the therapeutic use of OXT is worthy of further investigation in individuals with psychopathic traits. OXT could potentially increase the effectiveness of psychotherapeutic interventions aimed at promoting empathy and reducing aggression by increasing the salience of social-emotional stimuli. There is a great need for more effective treatments for this population, given the lack of effectiveness of current interventions, and the increased risk of recidivism among individuals with CU traits.

In conclusion, this study underscores the significance of childhood maltreatment, particularly emotional abuse, as well as reduced concentrations of OXT in the development of both CU traits and proactive aggression. Our findings suggest that these mechanisms are distinct

from those that underlie reactive aggression. Although this design cannot demonstrate any causal relationships, this study illustrates one potential model for the development of psychopathic traits in the context of childhood maltreatment, and our findings support the hypothesis that early psychosocial adversities may become “biologically embedded” via chronic reductions in OXT, which ultimately increase the risk of CU traits and proactive aggression.

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APPENDIX A: IRB APPROVAL LETTER

EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board Office
4N-70 Brody Medical Sciences Building· Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office 252-744-2914 · Fax 252-744-2284 · www.ecu.edu/irb

Notification of Continuing Review Approval: Expedited

From: Social/Behavioral IRB
To: Emma-Catherine Scott
CC:
Jeannie Golden
Emma-Catherine Scott
Date: 9/12/2016
Re: CR00004873
UMCIRB 14-001140
Biological, Emotional, and Neurocognitive Effects of Early Adverse Experiences

The continuing review of your expedited study was approved. Approval of the study and any consent form(s) is for the period of 9/11/2016 to 9/10/2017. This research study is eligible for review under expedited category #8C.

Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminat an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The Investigator must adhere to all reporting requirements for this study.

Approved consent documents with the IRB approval date stamped on the document should be used to consent participants (consent documents with the IRB approval date stamp are found under the Documents tab in the stud workspace).

The approval includes the following items:

Document	Description
Application for Research with Probationers(0.03)	Study Protocol or Grant Application
BEEF(0.03)	Surveys and Questionnaires
Caregiver demographics & maltreatment survey.docx(0.03)	Surveys and Questionnaires
Demographic survey.docx(0.02)	Surveys and Questionnaires
Dissertation Proposal revised 9-26-15.docx(0.04)	Study Protocol or Grant Application
Informed Consent for Probationers.doc(0.05)	Consent Forms
Informed Consent Young Adult.doc(0.08)	Consent Forms
Inventory of CU Traits - Self report (page 1)(0.04)	Surveys and Questionnaires
Inventory of CU Traits - Self report (page 2)(0.01)	Surveys and Questionnaires
letter of offender willingness to participate.docx(0.01)	Additional Items

APPENDIX B: RESEARCH CERTIFICATION LETTER FROM THE NCDPS

EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board Office
4N-70 Brody Medical Sciences Building · Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office 252-744-2914 · Fax 252-744-2284 · www.ecu.edu/irb

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Approved consent documents with the IRB approval date stamped on the document should be used to consent participants (consent documents with the IRB approval date stamp are found under the Documents tab in the study workspace).

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Informed Consent for Probationers.doc(0.05)	Consent Forms
Informed Consent Young Adult.doc(0.08)	Consent Forms
Inventory of CU Traits - Self report (page 1)(0.04)	Surveys and Questionnaires
Inventory of CU Traits - Self report (page 2)(0.01)	Surveys and Questionnaires
letter of offender willingness to participate.docx(0.01)	Additional Items

APPENDIX C: FINAL AUTHORIZATION FROM THE NCDPS

EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board Office
4N-70 Brody Medical Sciences Building · Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office 252-744-2914 · Fax 252-744-2284 · www.ecu.edu/irb

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Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The Investigator must adhere to all reporting requirements for this study.

Approved consent documents with the IRB approval date stamped on the document should be used to consent participants (consent documents with the IRB approval date stamp are found under the Documents tab in the study workspace).

The approval includes the following items:

Document	Description
Application for Research with Probationers(0.03)	Study Protocol or Grant Application
BEEF(0.03)	Surveys and Questionnaires
Caregiver demographics & maltreatment survey.docx(0.03)	Surveys and Questionnaires
Demographic survey.docx(0.02)	Surveys and Questionnaires
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Inventory of CU Traits - Self report (page 2)(0.01)	Surveys and Questionnaires
letter of offender willingness to participate.docx(0.01)	Additional Items

APPENDIX D: CONSENT FORM FOR UNDERGRADUATE STUDENTS

East Carolina University

Informed Consent to Participate in Research

Information to consider before taking part in research that has no more than minimal risk.

Title of Study: Biological, Emotional, and Neurocognitive Effects of Early Adverse Experiences
Principal Investigator: Emmi Scott, M.A.
Institution/Department or Division: East Carolina University, Department of Psychology
Address: 109A Rawl Building, E. 5th Street, Greenville, NC, 27858
Telephone #: 252-328-5812

Study Sponsor/Funding Source: Pitt Property Management

Researchers at East Carolina University (ECU) study problems in society, health problems, environmental problems, behavior problems and the human condition. Our goal is to try to find ways to improve the lives of you and others. To do this, we need the help of volunteers who are willing to take part in research.

We invite you to participate in this research study at ECU. Before you can decide whether or not to volunteer for this study, you must understand the purpose, how it may affect you, any risks to you, and what is expected of you. This process is called informed consent. Once you understand the study, and if you agree to participate, you will be asked to sign this Informed Consent document.

Why is this research being done?
The purpose of this research is to identify the effects of early experiences such as abuse or problems in the parent-child relationship. We will look at how biological factors, such as heart rate and hormones (like oxytocin) influence emotional problems and aggression among youth. Oxytocin is a hormone that helps people form close relationships and bond with others. By doing this research, we hope to better understand why negative childhood events (like abuse) can affect some people more than others. By collecting oxytocin in saliva (spit), we hope to understand the biological reason that some people have more emotional problems and are more likely to offend (break the law) than others. We also hope that results from this study will eventually help to identify people in need of interventions. It is completely your decision whether or not to take part in this research study.

Why am I being invited to take part in this research?
You are being invited to take part in this research because you are between the ages of 16 and 29, and are currently in foster or adoptive care, or have experienced some type of early trauma, maltreatment, or disruption in the parent-child relationship. If you volunteer to take part in this research, you will be one of about 100 people to do so.

Are there reasons I should not take part in this research?
I understand I should not volunteer for this study if I am younger than age 16 or older than age 29, or have been diagnosed with an intellectual or developmental disability.

What other choices do I have if I do not take part in this research?
You can choose not to participate or to withdraw from this study at any time. There will be no negative consequences if you decide to do so.

*Consent Version # or Date:*_____

_____
Participant's Initials

Where is the research going to take place and how long will it last?
The research procedures will be conducted at your local adoption or foster care agency, Department of Social Services (DSS), your therapist's office, or at ECU (Rawl building) in Greenville, NC. You will only need to come to this site one time for the study. All visits will take place in the afternoons (between 12:30 and 5:30pm) and last about 1 hour.

What will I be asked to do?
First, you will complete a few questionnaires. They ask questions about your health, care history, and social and emotional well-being. Some questions ask if you have experienced any stressful events over the past year (e.g., death of a family member, abuse).

Next, we will measure your resting heart rate (number of beats per minute) using a device that clips onto your fingertip. You will also complete a quick decision-making game on a hand-held device, and spit into a small test tube. This sample will be used to gather information on oxytocin levels.

Several things can affect saliva (spit) samples, so please let the researcher know if you have done any of the following **before this visit**:

- Had dental work performed within the last 24 hours,
- Took a stimulant medication (like Adderall, Concerta, Ritalin for ADD/ADHD) or other medication that can cause dry mouth, or
- Ate a major meal or food/drinks with high caffeine or high sugar within the last 45 minutes.

What possible harms or discomforts might I experience if I take part in the research?
If you have experienced any traumatic events, some questions in this study may make you feel uncomfortable or distressed by reminding you of those events. If you become upset by any of the questions, a licensed and experienced therapist will be available to talk to you. We can also give you a referral sheet with trauma-related services that are available in the community.

What are the possible benefits I may experience from taking part in this research?
We do not know if you will get any benefits by taking part in this study. There may be no personal benefit from your participation, but the information gained by doing this research may help others in the future. By helping us learn more about the effects of trauma and maltreatment, future treatments may be developed for emotional problems that result from maltreatment. Some people who have participated in similar research reported feeling good about themselves for volunteering for research that could potentially help others.

Will I be paid for taking part in this research?
We will not be able to pay you for the time you volunteer while being in this study. However, we may provide refreshments (e.g., snacks, bottled water) to all potential participants at the study location. If you are completing this study as part of your research participation requirement for an undergraduate psychology course, you will receive research credit.

What will it cost me to take part in this research?
It will not cost you any money to be part of the research.

*Consent Version # or Date:*_____

_____
Participant's Initials

Title of Study: Biological, Emotional, and Neurocognitive Effects of Early Adverse Experiences

Who will know that I took part in this research and learn personal information about me?
To do this research, ECU and the people and organizations listed below may know that you took part in this research and may see information about you that is normally kept private. With your permission, these people may use your private information to do this research:

- Any agency of the federal, state, or local government that regulates human research. This includes the Department of Health and Human Services (DHHS), the North Carolina Department of Health, and the Office for Human Research Protections.
- The University & Medical Center Institutional Review Board (UMCIRB) and its staff, who have responsibility for overseeing your welfare during this research, and other ECU staff who oversee this research.

How will you keep the information you collect about me secure? How long will you keep it?
In order to make sure your responses are kept private, we will not use your name on any study records. Instead, a unique study number will be assigned to you and only this number will be used on study documents. The key to the code will be in a password-protected database. At the end of this project, this key will be destroyed. No individual information will be shared with peers, parents, or teachers. All coded study documents will be kept in a locked file cabinet in the principal investigator's office. Data collected for this study will be kept for 5 years after the end of this study.

What if I decide I do not want to continue in this research?
If you decide you no longer want to be in this research after it has already started, you may stop at any time. You will not be penalized or criticized for stopping. You will not lose any benefits that you should normally receive. You are still invited to have snacks.

Who should I contact if I have questions?
The people conducting this study will be available to answer any questions concerning this research, now or in the future. You may contact Emmi Scott, the Principal Investigator, at 252-328-5812 or at scotte07@students.ecu.edu, or Dr. Jeannie Golden, the faculty supervisor, at 252-328-6026 or at goldenj@ecu.edu.
If you have questions about your rights as someone taking part in research, you may call the Office of Research Integrity & Compliance (ORIC) at phone number 252-744-2914 (days, 8:00 am-5:00 pm). If you would like to report a complaint or concern about this research study, you may call the Director of the ORIC, at 252-744-1971.

I have decided I want to take part in this research. What should I do now?
The person obtaining informed consent will ask you to read the following and if you agree, you should sign this form:

- I have read (or had read to me) all of the above information.
- I have had an opportunity to ask questions about things in this research I did not understand and have received satisfactory answers.
- I know that I can stop taking part in this study at any time.
- By signing this informed consent form, I am not giving up any of my rights.
- I have been given a copy of this consent document, and it is mine to keep.

Participant's Name (PRINT)	Signature	Date

APPENDIX E: CONSENT FORM FOR PROBATIONERS/PAROLEES

East Carolina University

Informed Consent to Participate in Research

Information to consider before taking part in research that has no more than minimal risk.

Title of Study: Biological, Emotional, and Neurocognitive Effects of Early Adverse Experiences
Principal Investigator: Emmi Scott, M.A.
Institution/Department or Division: East Carolina University, Department of Psychology
Address: 109A Rawl Building, E. 5th Street, Greenville, NC, 27858
Telephone #: 252-328-5812

Study Sponsor/Funding Source: Pitt Property Management

We invite you to participate in this research study at East Carolina University (ECU). Once you understand the study, and if you agree to participate, you will be asked to sign this Informed Consent document.

Why is this research being done?
The purpose of this research is to understand how people's biology and environment as a child can make some people more aggressive and more likely to break the law than others. We think that negative childhood events can change people's hormones - particularly one called "oxytocin" - , and these changes in our biology control how aggressive we are. Oxytocin is often called the "love hormone" because it helps people form close relationships and bond with others. When we experience bad or stressful things as children, our bodies start to either make more or less oxytocin. It may be harder for people with less oxytocin to bond with others, and they may be more aggressive and feel less guilty about it than people with high oxytocin. By collecting oxytocin in saliva (spit) from young adults on probation, we hope to understand the biological reasons for these behaviors. It may also help us understand how negative events in childhood (like abuse) continue to influence emotions and behavior into adulthood.

Why am I being invited to take part in this research?
You are being invited to take part in this research because you are between the ages of 18 and 29, and are currently on probation. If you volunteer to take part in this research, you will be one of about 100 other people (50 on probation, and 50 not on probation) to do so.

Are there reasons I should not take part in this research?
You should not volunteer for this study if you are younger than age 18 or older than age 29, or have been diagnosed with an intellectual or developmental disability.

What other choices do I have if I do not take part in this research?
You can choose not to participate or to withdraw from this study at any time. There will be no negative consequences if you decide to do so and your terms of supervision/probation will not be affected.

Where is the research going to take place and how long will it last?
The research procedures will be conducted at your probation office or another public location that will be set up ahead of time. This study will take about 30 minutes to complete.

What will I be asked to do?

1. You will complete a few questionnaires asking about your health, history of any abuse or neglect, and your personality and behavior. Some questions ask if you have experienced any stressful events (e.g., death in the family, abuse).
2. We will measure your pulse rate (number of beats per minute) from your fingertip.
3. You will complete a quick decision-making game.
4. Finally, you will spit into a small test tube. This will be used to measure your oxytocin.

Several things can affect saliva (spit) samples, so please let the researcher know if you have done any of the following before this visit:

- Had dental work performed within the last 24 hours,
- Took a stimulant medication (like Adderall, Concerta, Ritalin for ADD/ADHD) or other medication that can cause dry mouth, or
- Ate a major meal or food/drinks with high caffeine or high sugar within the last 45 minutes.

What possible harms or discomforts might I experience if I take part in the research?
If you have experienced any traumatic events, some questions in this study may make you feel uncomfortable or distressed by reminding you of those events. If you become upset by any of the questions, a licensed and experienced therapist will be available to talk to you. We can also give you a referral sheet with trauma-related services that are available in the community.

What are the possible benefits I may experience from taking part in this research?
We do not know if you will get any benefits by taking part in this study. There may be no personal benefit from your participation, but your participation may help us develop better treatments for others in the future. Some people who have participated in similar research reported feeling good about themselves for volunteering for research that could potentially help others.

Will I be paid for taking part in this research?
We will not be able to pay you for volunteering for this study.

What will it cost me to take part in this research?
It will not cost you any money to be part of the research.

Who will know that I took part in this research and learn personal information about me?
To do this research, ECU and the people and organizations listed below may know that you took part in this research and may see information about you that is normally kept private. With your permission, these people may use your private information to do this research:

- Any agency of the federal, state, or local government that regulates human research. This includes the Department of Health and Human Services (DHHS), the North Carolina Department of Health, and the Office for Human Research Protections.
- The University & Medical Center Institutional Review Board (UMCIRB) and its staff, who have responsibility for overseeing your welfare during this research, and other ECU staff who oversee this research.

The Department of Public Safety staff are not conducting this research project. They will not get a copy of your name or of your answers. The Department may receive a copy of the overall results at the end of the study but will not be able to identify you personally from the copy they receive.
If you indicate plans to harm yourself, to harm someone else, or to escape or abscond supervision that information is not confidential and will immediately be reported to DPS staff.

How will you keep the information you collect about me secure? How long will you keep it?
In order to make sure your responses are kept private, we will not use your name on any study records. Instead, a random number will be used. No individual information will be shared. All study documents will be kept in a locked file cabinet in the researcher's office for 5 years.

What if I decide I do not want to continue in this research?
If you decide you no longer want to be in this research after it has already started, you may stop at any time. You will not be penalized or criticized for stopping. Your release date, terms of supervision, medical care, or your general living conditions will not be affected by whether you chose to be in the study or if you chose to stop participating at any point. You may refuse to answer questions or stop taking part in the study at any time.

Who should I contact if I have questions?
The people conducting this study will be available to answer any questions concerning this research, now or in the future. You may contact Emmi Scott, the Principal Investigator, at 252-328-5812 or at scotte07@students.ecu.edu, or Dr. Jeannie Golden, the faculty supervisor, at 252-328-6026 or at goldenj@ecu.edu. If you have questions about your rights as someone taking part in research, you may call the Office of Research Integrity & Compliance (ORIC) at phone number 252-744-2914 (days, 8:00 am-5:00 pm). If you would like to report a complaint or concern about this research study, you may call the Director of the ORIC, at 252-744-1971.

I have decided I want to take part in this research. What should I do now?
The person obtaining informed consent will ask you to read the following and if you agree, you should sign this form:

- I have read (or had read to me) all of the above information.
- I have had an opportunity to ask questions about things in this research I did not understand and have received satisfactory answers.
- I know that I can stop taking part in this study at any time.
- By signing this informed consent form, I am not giving up any of my rights.
- I have been given a copy of this consent document, and it is mine to keep.

_____

Participant's Name (PRINT) **Signature** **Date**

.....

(Completed by researcher)
Person Obtaining Informed Consent: I have conducted the initial informed consent process. I have orally reviewed the contents of the consent document with the person who has signed above, and answered all of the person's questions about the research.

_____

Person Obtaining Consent (PRINT) **Signature** **Date**

APPENDIX F: DEMOGRAPHIC QUESTIONNAIRE

Please answer the following questions about you honestly.

Age: _____

Gender:

- ☐ Male
- ☐ Female
- ☐ Transgender
- ☐ Other

1. Do you have any medical conditions? Check all of the following that apply:

- ☐ Asthma
- ☐ Allergies
- ☐ Arthritis
- ☐ Birth Defects
- ☐ Cancer
- ☐ Chronic Fatigue Syndrome
- ☐ Deafness
- ☐ Diabetes
- ☐ Gastrointestinal Disease
- ☐ Heart Disease/Problems
- ☐ High Blood Pressure
- ☐ High Cholesterol
- ☐ Kidney Problems
- ☐ Seizures
- ☐ Thyroid Problems
- ☐ T/B Cystic Fibrosis
- ☐ None
- ☐ Other: _____

2. Have you ever sustained a brain injury or concussion?

- ☐ Yes
  - o If yes, circle whether it was:
    - *Mild* (i.e., concussion; loss of consciousness was less than 30 minutes)
    - *Moderate* (loss of consciousness that lasted between 1 hour and 24 hours)
    - *Severe* (loss of consciousness or coma for over 24 hours)
  - o If yes, how many? _____ At what age(s)? _____
- ☐ No

3. Have you been diagnosed with any of the following mental health disorders <u>in the past</u>?

- ☐ Depression
- ☐ Anxiety
- ☐ Reactive attachment disorder
- ☐ Adjustment disorder
- ☐ Oppositional defiant disorder
- ☐ Conduct disorder
- ☐ Attention deficit hyperactivity disorder (ADHD)
- ☐ Antisocial personality disorder
- ☐ Other: _____ _____

Do you <u>currently</u> have any mental health diagnoses? If yes, specify all:

_____

_____

4. Who do you <u>currently</u> live with? (Check all that apply)

- ☐ Biological mother
- ☐ Biological father
- ☐ Other relative
- ☐ Foster parent
- ☐ Grandparent
- ☐ Aunt, uncle, or other family member
- ☐ Adoptive parents (not family member)
- ☐ Other (roommate, boy/girlfriend)
- ☐ Live by yourself

4a. If you do NOT live with your biological *mother*, please check all that are true:

- ☐ I have never met her
- ☐ My mother is no longer living
- ☐ I have seen her once or twice
- ☐ I used to see her a lot, but I have not seen her in over a year
- ☐ I see her once a year
- ☐ I see her once a month
- ☐ I see her twice a month
- ☐ I see her on weekends

4b. If you do NOT live with your biological *father*, please check all that are true:

- ☐ I have never met him
- ☐ My father is no longer living
- ☐ I have seen him once or twice
- ☐ I used to see him a lot, but I have not seen him in over a year
- ☐ I see him once a year
- ☐ I see him once a month
- ☐ I see him twice a month
- ☐ I see him on weekends

5. **Changes in caregivers**: Have you ever had to move into a home with a different caregiver?
   - ☐ No.
   - ☐ Yes. If yes, how many times? _____
     - If yes, were you in foster care? YesNo
       - If yes, specify number of foster care placements: _____
       - From age _____ to _____
     - Did you live in a group home? YesNo
       - From age _____ to _____
     - Orphanage? Yes No
       - From age _____ to _____
     - Children's home? YesNo
       - From age _____ to _____
     - Institution? YesNo
       - From age _____ to _____
     - Other (specify whether family member, other adoptive parent, residential treatment facility, etc.):_____

       _____

- **Last change in placement/caregiver to whom**:_____
  - Your age at time of last change: _____

6. Have you taken any medications (prescription or over-the counter) or illegal drugs today?

- ☐ Yes (please specify):_____
  _____
- ☐ No

7. What is your current relationship status?

- ☐ Single
- ☐ In a relationship
- ☐ Married
- ☐ Divorced
- ☐ Widowed
- ☐ Other: _____

***** ***The section below is for WOMEN only*** *****

10. How long ago was your last menstrual period? (Check <u>one</u> option that applies)

- ☐ I have never had a period
- ☐ I am having my period today (How many days ago did it start? _____)
- ☐ Between 5 and 8 days ago
- ☐ Between 9 and 12 days ago
- ☐ Between 13 and 15 days ago
- ☐ Between 16 and 18 days ago
- ☐ Between 19 and 22 days ago
- ☐ Between 23 and 28 days ago
- ☐ Between 1 month and 3 months ago (How many weeks ago: _____)
- ☐ Over 3 months ago (How many months ago? _____)

11. Do you take birth control? (either a pill, patch, shot, implant, or NuvaRing)

- ☐ Yes
- ☐ No

12. Are you pregnant or breastfeeding?

- ☐ Yes
- ☐ No

APPENDIX G: RECENT STRESSFUL LIFE EVENTS QUESTIONNAIRE

Recent Stressful Life Event Scale

In the past 12 months, which of the following major life events have taken place in your life? Place a check mark each item that you have experienced this year.

1. __ Death of parent
2. __ Parents separated
3. __ Parents divorced
4. __ Parent had jail time
5. __ Death of close family member (not parents)
6. __ A parent got married
7. __ You went to jail
8. __ You got suspended
9. __ You got fired from work
10. __ Change in family member's health
11. __ Pregnancy (or of girlfriend/wife)
12. __ Addition to family (new baby)
13. __ Death of close friend
14. __ Got married
15. __ Started or finished school
16. __ Change in living conditions (new home, etc.)
17. __ Changes in personal habits (quit smoking, etc.)
18. __ Changed to a new school (or started college)
19. __ Change in sleeping habits (a lot more or a lot less)
20. __ Break up with spouse or steady boyfriend/girlfriend
21. __ Serious physical illness or injury requiring hospital treatment
22. __ Problems with alcohol or drugs
23. __ Someone physically forced sex with you against your wishes
24. __ Family or caregiver repeatedly ridiculed you, put you down, etc.
25. __ Partner/spouse repeatedly ridiculed you, put you down, etc.
26. __ Family/caregiver kicked, beat, slapped, or physically harmed you
27. __ Partner/spouse kicked, beat, slapped, or physically harmed you
28. __ You were threatened with a weapon (gun, knife, etc.)

APPENDIX H: BEHAVIORAL EMOTIONAL AND EXECUTIVE FUNCTIONING INSTRUMENT

Remember: 1 – Very Untrue, 2 – Slightly Untrue, 3 – Slightly True, 4 – Very True

Behavioral Emotional and Executive Functioning Instrument

Participant ID:_____

Instructions: The following items contain sentences that people may use to describe how they feel or act. Read each sentence carefully and answer each item. There are no right or wrong answers. Just answer *honestly*. Circle the choice that best describes you using the following scale:

Circle 1 if the item is **not true at all**.
Circle 2 if the item is **slightly untrue**.
Circle 3 if the item is **slightly true**.
Circle 4 if the item is **very true**.

Please answer every item!

Mark: 1 = Very Untrue 2 = Slightly Untrue 3 = Slightly True 4 = Very True				
1. I don't regret anything I've done in the past.	1	2	3	4
2. I hate it when I know someone is upset with me.	1	2	3	4
3. I believe that most people are only nice to others to get what they want.	1	2	3	4
4. If people are stupid enough to get conned (scammed), it is their own fault.	1	2	3	4
5. Sometimes I enjoy teasing people just to get a reaction.	1	2	3	4
6. I feel very deep emotional connections with others.	1	2	3	4
7. If I get mad at someone, I will make sure they get what they deserve.	1	2	3	4
8. I agree with the saying, "what others don't know won't hurt them."	1	2	3	4
9. I believe that when people cry, it's usually just to get attention.	1	2	3	4
10. I usually try my best in school and work.	1	2	3	4
11. I get in trouble for not thinking before I act.	1	2	3	4
12. Sometimes I get mad when I don't get my way.	1	2	3	4
13. I believe that deep down, most people only care about themselves.	1	2	3	4
14. I can stay calm in situations where others can't.	1	2	3	4
15. I am a spur-of-the-moment type of person.	1	2	3	4
16. Nothing scares me.	1	2	3	4
17. I worry that things I say may hurt people's feelings.	1	2	3	4
18. I am very self-conscious.	1	2	3	4
19. I feel sad when I see someone cry.	1	2	3	4
20. Other people's feelings are not my problem.	1	2	3	4
21. I don't care if something I do hurts others along the way.	1	2	3	4
22. I'm not scared of anybody.	1	2	3	4
23. I don't care if I get punished for doing something that I want to do.	1	2	3	4
24. I tend to do things without thinking.	1	2	3	4
25. I have never talked badly about someone else.	1	2	3	4
26. Every time I get mad, I hear my brain pounding like thunder.	1	2	3	4
27. Other people seem to be more emotional than I am.	1	2	3	4
28. I could never cheat someone because I would feel too guilty.	1	2	3	4
29. I get embarrassed easily.	1	2	3	4

Remember: 1 – Very Untrue, 2 – Slightly Untrue, 3 – Slightly True, 4 – Very True

30. It is hard for me to wait patiently for things I want.	1	2	3	4
31. I never get mad.	1	2	3	4
32. I tend to get bored and lose interest in things easily.	1	2	3	4
33. I believe that I could beat a lie detector.	1	2	3	4
34. I feel guilty easily.	1	2	3	4
35. I am good at getting my way.	1	2	3	4
36. I have never lied directly to someone's face before.	1	2	3	4

Circle the answer choice that reflects how often you participated in each activity ***OVER THE PAST 12 MONTHS.***

Circle N if the behavior **never** occurs. Circle R if the behavior **rarely** occurs.
Circle S if the behavior **sometimes** occurs. Circle O if the behavior **often** occurs.

N = Never	**R = Rarely**	**S = Sometimes**	**O = Often**			
37. How frequently did you skip class?			N	R	S	O
38. How frequently did you use drugs illegally?			N	R	S	O
39. How frequently did you hurt an animal on purpose?			N	R	S	O
40. How frequently did you steal something from a store?			N	R	S	O
41. How frequently did you have unprotected sex?			N	R	S	O
42. How frequently did you sell drugs?			N	R	S	O
43. How frequently were you arrested?			N	R	S	O
44. How frequently did you drink alcohol?			N	R	S	O
45. How frequently were you suspended from school (or work)?			N	R	S	O
46. How frequently did you steal something from a person?			N	R	S	O
47. How frequently did you start a rumor about someone you know?			N	R	S	O