Probing associations between structural brain connectivity, childhood maltreatment, and later antisocial behavior

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Childhood maltreatment, a form of early life stress, impacts an extraordinary number of children per year and has profound implications for mental health and other psychosocial outcomes. Abuse experienced as a child is well-linked to antisocial behavior such as aggression and misconduct and to alterations in neural structures and pathways. Many of the neural structures implicated in childhood maltreatment are crucial components of emotional regulatory brain networks, connected through pathways including the uncinate fasciculus, the cingulum bundle, and the fornix. Poorer emotion regulation has been pointed to as an underlying factor contributing to antisocial behavior; therefore, we seek to explore if alterations in neural emotion-regulatory pathways significantly account for a portion of the established link between childhood maltreatment and adult antisocial behavior. We explored these related questions in a subsample of the Pittsburgh Youth Study dataset – a unique, multi-decade longitudinal study of youth and family processes. Related to childhood maltreatment and violence in adulthood, we did not find significant associations between childhood maltreatment and violent adult criminal versatility within our sample. Connected to potential neural mediators, we also did not find any significant associations between childhood maltreatment and white matter integrity in the aforementioned tracts involved in emotion regulation. Further, we found no associations between white matter integrity in these tracts and violent criminal versatility. Potential explanations for this pattern of null findings, as well as implications for this field of research, are discussed. Further work is needed to continue to understand the associations between childhood abuse, the brain, and violent

antisocial behavior in order to elucidate the downstream associations of early life stress on later behavior.

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1.0 Overview

Early life stress (ELS) such as physical abuse, child neglect, or violence exposure is sadly quite common and may impact psychological well-being and mental health (McLaughlin et al., 2012; Evans et al., 2013). Many forms of ELS – and of particular concern, childhood maltreatment – are linked to adult antisocial behavior, a set of heterogeneous actions including violating societal rules and others' rights (Wilson et al., 2009; Hyde et al., 2013). The associations between maltreatment and antisociality are well-studied and well-replicated; however, little is known about the underlying processes through which childhood maltreatment is related to antisocial behavior. In particular, understanding the neural consequences of early life stress and discovering neural predictors of violence can contribute to our understanding of the development of antisocial behavior by illuminating the pathways through which such behavior may manifest. Indeed, identifying key behavioral and biological pathways related to early stress and later antisociality is a step towards understanding how life experiences may come to affect a range of behaviors and underlying susceptibility to adverse outcomes. Probing these associations more deeply may have crucial basic science, clinical, and public-policy implications due to the high societal impact of aggression, violence, and crime (Scott et al., 2001; Colman et al., 2009; Rivenbark et al., 2018).

A growing body of neuroimaging studies has linked ELS to differences in neural circuitry related to emotion processing and regulation. Interestingly, work focused on the neurobiological correlates of antisocial behavior, independent of adversity, has suggested alterations in similar, emotion-related brain networks. Limited work has connected these constructs and attempted to understand the relations between ELS, neurobiology, and antisocial behavior. Here, we propose a study focused on white matter tracts critical for emotion processing and regulation, including the uncinate fasciculus, the cingulum bundle, and the fornix, in understanding connections between

ELS and adult antisocial behavior. Focusing on these white matter pathways may be particularly important as they connect brain areas critical for emotional functioning, including portions of the prefrontal cortex, the hippocampus, and the amygdala. ELS may be associated with alterations in structural connectivity and give rise to challenges in regulating emotions throughout development, potentially increasing antisocial behavior during adolescence and adulthood.

1.1 Early Life Stress and Antisocial Behavior

Childhood maltreatment is a particularly pernicious and prevalent form of ELS that includes neglect, emotional abuse, physical abuse, and sexual abuse (Jaffee, 2017). Childhood maltreatment is widespread, with at least 1 in 7 children experiencing abuse or neglect a year (Fortson et al., 2016). The prevalence of these experiences is particularly alarming given that child abuse is associated with the onset of psychopathology and other adverse outcomes throughout development (McLaughlin et al., 2012). Antisocial behavior is one such outcome of particular interest due to the high cost of violent and non-violent antisocial behavior (Ludwig, 2006, 2010). The construct of antisocial behavior describes a heterogeneous set of behaviors that disregard and infringe on others' rights and includes physical and sexual aggression, the violation of societal rules, and the destruction of property (Hyde et al., 2013).

Adult individuals who exhibit persistent and deleterious behaviors in extreme forms such as aggression and cruelty towards people, deceitfulness or theft, and potentially a lack of remorse, guilt, empathy, and affect may be given a diagnosis of Antisocial Personality Disorder (American Psychiatric Association, 2013). A related construct, psychopathy, denotes a particularly severe and chronic course of antisocial actions (Hare, 1991, 2003). This condition is marked by interpersonal

and affective dimensions (e.g., narcissism), as well as impulsive and antisocial facets (e.g., heightened emotional distress, deviancy) (Cunningham & Reidy, 1998; Hicks & Patrick, 2006).

A thorough body of work has established a strong connection between childhood maltreatment and antisocial, criminal, and aggressive behavior, finding that childhood maltreatment increases overall risk for antisocial and aggressive behaviors (Pollock et al., 1990). Victims of childhood sexual abuse are more likely than non-abused counterparts to be both victims and offenders of criminal acts, including both violent and sexual offenses (Mallett & Schall, 2019). Abuse and neglect experienced in childhood have been found to increase the likelihood of adult criminality by 29-38%, underscoring a clear connection between these ELS and antisocial criminal behavior (Widom & Maxfield, 2001). Meta-analyses further reinforce this linkage, though with varying effect sizes. For example, a meta-analysis on studies of maltreatment and adolescent antisocial behavior compiled by Wilson and colleagues (2009) found that overall effect sizes varied depending on the methodology; prospective studies had a modest effect size (Cohen's d = .31) while cross-sectional studies had a more robust effect size (Cohen's d = .88). Other recent meta-analyses have found that individuals maltreated as children were nearly twice as likely to engage in antisocial behavior as adults (mean-weighted odds ratio = 1.96) (Braga et al., 2018) and that maltreatment was associated with higher rates of both general juvenile antisocial behaviors (r = 0.11) and aggressive juvenile antisocial behaviors (r = 0.11) (Braga et al., 2017).

While these associations are consistently found across studies, this body of literature is not without limitation. The majority of the studies reviewed are cross-sectional in design and rely on self-report; thus, there may be recall and other biases associated with retrospective reports of childhood maltreatment (Widom & Shepard, 1996; Widom & Morris, 1997). As a result, there is a relative scarcity of prospective studies of maltreatment and antisocial behavior. Longitudinal

prospective research designs are needed to more precisely establish a causal association between childhood maltreatment and subsequent antisocial behavior. Despite differences in methodological choices, a majority of the literature converges on the conclusion that there is a strong connection between childhood maltreatment and antisociality.

1.2 Etiological Connections between Childhood Maltreatment and Antisociality

Several theories have been advanced to explain the associations between childhood maltreatment and later antisocial behavior and to explore predictive factors within these relations. A majority of these theories have focused on psychosocial and sociocultural models of early life and behavior and often build or expand on one another. For example, Shaw and Bell's (1993) bridging model integrates previous perspectives on developmental psychopathology and criminology to examine child, family, and contextual factors on the development of antisocial behavior in children. Other prominent models include social learning theory and the integrated cognitive antisocial potential (ICAP) theory. Social learning theory postulates that exposure to abuse in childhood might teach children to model aggressive and violent behaviors later in life (Bandura, 1973), while the ICAP integrates numerous risk factors for antisocial behavior such as impulsivity, inadequate parental supervision, child abuse, and more, to consider an individual's overall antisocial potential (Farrington, 2005).

Across a preponderance of these theories, emotion regulation challenges are implicitly and explicitly a central mechanism or a critical cascading factor connecting childhood maltreatment and antisocial behavior. Emotion regulation is broadly understood as an individual's conscious or unconscious ability to cope with or manage their emotions (Gross, 1999; Röll et al., 2012).

Emotion regulation skills are cultivated throughout development and are thus susceptible to the impact of childhood maltreatment. Studies have found that childhood maltreatment may lead to a number of problems in basic processes underlying emotion regulation such as emotion expression, recognition, understanding, and communication (Maughan & Cicchetti, 2002). Work has consistently implicated deficits in emotion regulation circuitry as a central element of severe antisocial behavior (Garofalo et al., 2018; Roberton et al., 2014) and as a critical risk factor for reactive aggression and impulsivity (Röll et al., 2012). While the majority of research linking these constructs relies on concurrent measures of emotion regulation and antisociality, few prospective studies have made similar associations (e.g., Trentacosta & Shaw, 2009).

Maladaptive emotion regulation strategies may contribute to aggressive and delinquent antisocial behavior in several ways. Prominent theories of self-regulation (Baumeister et al., 1994; Baumeister & Heatherton, 1996) suggest that some individuals may experience diminished cognitive control when faced with negative emotions such as anger. The failure to appropriately respond to these negative emotions may lead to increased physiological arousal, heightened negative affect, lowered behavioral inhibition, and compromised decision-making faculties (Roberton et al., 2014). For example, in research focusing on a group of incarcerated violent offenders, challenges in emotion regulation were the most robust predictor of physical aggression (Garofalo & Velotti, 2017). Such dysfunctional emotion regulation strategies have consequential effects on behavior and outcomes; in another retrospective study, violent offenders with maladaptive emotion regulation strategies were found to have more extensive histories of aggression than offenders with more adaptive strategies (Roberton et al., 2014).

A few studies have directly linked poor emotion regulation to both childhood maltreatment and antisocial behavior. For example, maladaptive emotion regulation strategies have been found

to partially account for the influence of childhood maltreatment on aggressive and disruptive behavior (Teisl & Cicchetti, 2008; Shields & Cicchetti, 1998). As discussed later, our work proposes to further characterize this relation by probing structural connections in brain circuitry critical for emotion regulation. Evidence characterizing the neurobiological correlates of ELS, as well as describing typical neural phenotypes associated with antisocial behavior, reveals a pattern of implicated brain areas critical for emotion regulation – namely, the prefrontal cortex, the hippocampus, and the amygdala.

1.3 Neural Phenotypes Associated with Childhood Maltreatment and Antisocial Behavior

Many research groups have been interested in exploring the developmental impact of stress on neural structures and circuits involved in information processing and emotion regulation. This body of work converges on evidence that childhood maltreatment and other forms of ELS significantly impact neurobiology, often compromising the typical trajectories. Neuroimaging studies highlight protracted post-natal neural development, providing evidence that gray matter, white matter, and other aspects of the brain continue developing well into adolescence. As such, neurobiological development may be highly susceptible to the impact of traumatic experiences during this period (Carlson, 2013; Giedd & Rapport, 2010; Cabrera et al., 2020).

The experience of child abuse is associated with deficits in many systems essential to social information processing and emotion regulation (Hanson et al., 2017), due in large part to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. A large body of literature has investigated if chronic stress and associated HPA axis dysregulation impacts developmentally sensitive brain-regions and has highlighted differences in brain volume and gray matter in portions

of the prefrontal cortex, the hippocampus, and the amygdala (Mallett & Schall, 2019; Hart & Rubia, 2012; Hanson et al., 2015a). Similarly, prominent theories of antisociality similarly implicate dysfunction in these same circuits as a potential etiology of antisocial behaviors such as impulsivity and aggression (Shields & Cicchetti, 1998; Cappadocia et al., 2009; Waller et al., 2017). Reviews of the neural associations of antisocial behavior report that psychopathy is typically associated with abnormalities in the prefrontal cortex, the hippocampus, and the amygdala (Koenigs et al., 2011; Anderson & Kiehl, 2012).

Examining past neurobiological studies, a large number of studies have focused on the prefrontal cortex, a brain region that plays a central role in stress responsivity, emotion regulation, reappraising negative emotions, and modulating the activity of the amygdala (Davidson 2002; Hiser & Koenigs, 2018; Cabrera et al., 2020). A plethora of evidence demonstrates prefrontal cortex dysregulation and reduced volumes in response to childhood maltreatment (De Bellis et al., 2002; Hanson et al., 2010; Pechtel & Pizzagalli, 2011; Cabrera et al., 2020). With respect to antisocial behavior, reduced prefrontal structure and function are widely considered neural phenotypes of individuals with antisocial behavior (Yang & Raine, 2009). These findings indicate potentially compromised top-down functionality in regulating emotions among maltreated children and individuals engaging in severe antisocial behavior.

A second central structure for emotional processing and regulation is the hippocampus, which plays a vital part in learning, consolidating memories, and responding to stress (Hanson et al., 2015a). Beyond the vulnerability associated with such roles, the hippocampus's strong connection to the amygdala further increases the area's potential susceptibility to stressors (Moriceau et al., 2004). To this end, smaller hippocampal volumes have been found in children exposed to maltreatment such as physical abuse (Hanson et al., 2015a). Research findings on

antisocial behavior reveal a similar pattern; studies of criminal offenders have found both smaller hippocampal volumes (Emer et al., 2012) and abnormal hippocampal morphology within these populations (Boccardi et al., 2010). These results again highlight abnormal structure within the limbic system among maltreated children and adults who engage in antisocial behavior.

Finally, the amygdala is a central information-processing hub that processes emotions, assigns emotional valence to stimuli, and detects threats (Adolphs, 2010; Bufkin & Luttrell, 2005). The amygdala is directly affected by the HPA axis, the responsivity of which is modulated by chronic stress exposure (Mallett & Schall, 2019). Childhood maltreatment consistently has been associated with amygdala dysfunction and structural abnormalities. For example, McCrory and colleagues (2011, 2013) have found heightened amygdala activation in response to emotional stimuli among maltreated children. Structurally, amygdala abnormalities are commonly found among maltreated populations, with evidence pointing to both smaller amygdala volumes (Hanson et al., 2015a) and larger amygdala volumes in individuals who experienced maltreatment as children (Mehta et al., 2009; Tottenham et al., 2010).

With respect to antisociality, meta-analytic reviews report that antisocial behavior typically associates with overall amygdala volume reductions; for example, amygdala volume reductions have been found in groups of psychopathic individuals and incarcerated individuals (Boccardi et al., 2011; Ermer et al., 2012). However, in regard to amygdala function, diverging patterns of amygdala activity have been found among antisocial populations, with reports of severe antisocial behavior being associated with both *reduced* amygdala activity (Hyde et al., 2014) and *greater* amygdala reactivity in response to emotional stimuli (Blair et al., 2010; Hyde et al., 2014). These inconsistencies are likely due in part to varying study designs, conceptual foci, and potentially unreliable methodologies (i.e., functional neuroimaging). While outside the scope of our work,

these diverging findings illustrate two key points: first, the atypicality of emotional brain circuitry with respect to antisocial behavior, and second, that research might have ignored other neurobiological influences in the investigation of these constructs thus far. However, examined collectively, amygdala dysregulation is strongly implicated in antisociality, potentially by contributing to hypervigilance to threat signals or poor inhibition of aggression (Ishikawa & Raine, 2003).

1.4 The Associations between Childhood Maltreatment, Antisocial Behavior, and White Matter Connectivity in Emotion-Regulatory Circuits

Surveying the critical roles that the prefrontal cortex, hippocampus, and amygdala play in emotion, how neural signals (and information) "move" between these hubs may be particularly important for understanding the impact of childhood maltreatment and potential links to antisocial behavior. Connective tracts, also referred to as *white matter tracts*, interlink brain regions and are critical for information processing and transfer. The examination of these tracts enables the delineation of the connectivity between brain areas and can provide valuable insight into how complex processes, such as emotion regulation, occur in the brain. Diffusion Weighted Imaging (DWI) methods, such as fractional anisotropy (FA) and quantitative anisotropy (QA), allow researchers to measure the "integrity" of white matter tracts by tracing how water molecules are conducted throughout axon bundles. These axon bundles serve as insulation and reduces biological "signal loss" as information moves quickly throughout the brain. DWI has strong reliability and consistency over time, especially in contrast to functional neuroimaging, the test-retest reliability of which has come under question in recent years (Elliott et al., 2020). As such, structural imaging

of white matter tracts may provide a better platform to understand neural mechanisms underlying complex relations between antisocial behavior and other constructs. Several white matter tracts of interest are integral components of emotion regulation circuitry. These tracts include the uncinate fasciculus, the cingulum bundle, and the fornix. The typical developmental trajectory for these white matter tracts is characterized by an increase in volume throughout childhood and adolescence; such changes are crucial for cognitive, behavioral, and emotional development (Cabrera et al., 2020).

The uncinate fasciculus, an association pathway which reciprocally connects the prefrontal cortex to the amygdala, is one of the most commonly implicated tracts in these investigations (Waller et al., 2017). Specifically, this white mater tract connects the amygdala to the ventromedial prefrontal cortex (vmPFC), a subregion of the prefrontal cortex key in regulating the emotionally reactive amygdala (Davidson et al., 2000). Related to our constructs of interest, evidence shows that childhood maltreatment impacts this key limbic connection; for example, lower white matter integrity in the uncinate fasciculus has been associated with higher childhood maltreatment scores (Hanson et al., 2015b). Furthermore, post-mortem studies have linked childhood abuse to lower numbers of myelin-producing cells in the vmPFC, suggesting lower white matter integrity in this connective tissue and potentially less efficient transfer of information from this area to the amygdala (Tanti et al., 2018). Related to antisociality, numerous studies have found dysfunctional prefrontal connectivity and reduced uncinate fasciculus integrity among individuals with severe antisocial behavior and psychopathy (Motzkin et al., 2011; Wolf et al., 2015). For example, studies have reported that adults high on antisocial behavior and psychopathy measures had lower FA values, i.e., lower tract integrity, in either the right uncinate fasciculus (Craig et al., 2009; Sundram et al., 2012) or bilaterally (Hoppenbrouwers et al., 2013). Taken together, these findings

underscore that delineating the connective integrity between these two critical hubs could be crucial to understanding the dysfunctional emotion regulation circuitry potentially underlying relations between childhood maltreatment and antisocial behavior.

The second tract of interest, the cingulum bundle, is an association pathway that connects frontal and temporal lobes including areas of the prefrontal cortex, the amygdala, and the hippocampus. This pathway has been thoroughly explored due to its role in cognitive control and the integration of multiple areas across the limbic system (Bubb et al., 2018). The cingulum bundle has been repeatedly associated with socioemotional deprivation in early childhood (Eluvathingal et al., 2006), maltreatment (Huang et al., 2012), and childhood abuse (Ugwu et al., 2015). Both antisocial behavior and psychopathy were related to lower FA values in the cingulum bundle in two recent studies (Hoppenbrouwers et al., 2013; Sethi et al., 2014). These findings underscore the atypicality of cingulum bundle integrity in childhood maltreatment and antisocial behavior, pointing to potential dysfunction in integrating emotional responses in these individuals.

The final pathway, the fornix, is a projection pathway that connects areas of the hippocampus to subcortical regions (Waller et al., 2017). The fornix has been less explored than the tracts mentioned above, but there is preliminary evidence to suggest that lower white matter diffusivity in this tract is associated with childhood maltreatment (Choi et al., 2009). While no studies to date have found an association between the structure of the fornix and antisocial behavior or psychopathy in adults, Breeden and colleagues (2015) reported lower FA values in youth with high antisocial behavior. Taken together, evidence exists that the fornix is vulnerable to the effects of childhood maltreatment and may be yet another corticolimbic tract implicated in antisocial behavior. Future studies should continue to explore the roles of each of these limbic and prefrontal pathways, particularly the pathways that have been understudied such as the fornix.

1.5 The Current Work

Motivated by these different but related bodies of work, the present study seeks to understand further the significance of these emotion regulation pathways in the relation between maltreatment and later antisocial behavior. This association will be examined using behavioral and neuroimaging data from a subset of the Pittsburgh Youth Study, a 31-year longitudinal study that followed young males from the Pittsburgh area. Based on the work above, we propose that these three white matter tracts – the uncinate fasciculus, the cingulum bundle, and the fornix – may be neurobiological pathways through which exposure to childhood maltreatment leads to antisocial behavior (Figure 1). We specifically hypothesize that childhood maltreatment will lead to lower white matter integrity and heightened antisocial behavior in adulthood. We further predict that the association between maltreatment and antisociality will be accounted for, in part, by variations in white matter integrity in these tracts of interest.

2.0 Methods

2.1 Participants

We conducted a secondary analysis of behavioral and neuroimaging data from a subset of adults (N = 205) who participated in the Pittsburgh Youth Study (PYS). The PYS is a longitudinal study that followed young males from the Pittsburgh area starting in the 1987-1988 academic year. Participants were either in first or seventh grade at their initial interview, and follow-up phases

were conducted every year until the present day. Data was gathered from participant self-report measures and was also collected from other sources such as the Allegheny County Office of Children, Youth, and Families (CYF).

2.2 Procedure

The Pittsburgh Youth Study is an ongoing longitudinal study that has aimed to document explanatory factors in childhood and adolescence that give rise to maladaptive behaviors, including aggressive and delinquent behaviors. The PYS participants were males recruited from either first, third, or seventh-grade classes in the Pittsburgh Public School system during the 1987-1988 academic year. The final sample of participants was selected based on a screening assessment combining parent, teacher, and self-report on externalizing problems. Boys who scored in the upper 30% of the screening measure (n = ~250) were invited to participant in PYS, as well as an equal number of boys from the remaining distribution of the externalizing screener (n = ~250). The final PYS sample consisted of 1,517 boys, with 503 from the first-grade cohort, 508 from the third-grade cohort, and 506 from the seventh-grade cohort. PYS involved the collection of a variety of rich measures on risk and protective factors (e.g., maltreatment, externalizing symptoms) from multiple informants (e.g., teachers, parents, official records) and were collected semiannually and then annually. Across these assessments, the PYS has maintained a high retention rate (mean = 91%). For a richer description of the project, please see the work of Loeber and colleagues (1998).

This analysis pertained to a subsample of the first-grade cohort and seventh-grade cohort that underwent a brain scan during a follow-up session, and subsequent details focus on these cohorts. Table 1 depicts the assessment ages for individuals in the first grade and seventh-grade

cohorts. A subgroup of 205 PYS participants was recruited as part of the 2011-2012 assessment for a study on neural markers of violence. This sub-group contained three different groups recruited for varying histories of violence. The first group were recruited for a history of no violence (Violence Group 0, n = 64), the second group recruited for a history of 1-3 years of violence (Violence Group 1, n = 64) and a third group recruited for a history of 4+ years of violence (Violence Group 2, n = 43) (Meier et al., 2019). Participants were excluded from participation if they had a history of neurological disease, were currently using psychotropic medications, had a lifetime diagnosis of Axis I disorders (besides Substance Use Disorder), had irremovable ferromagnetic metal in the body, and had an estimated IQ score below 70.

A total sample of 171 men completed neuroimaging scans. Thirty-four participants were excluded for failure to complete the scans, excessive movement, or otherwise unusable neuroimaging scans. More than half of this sub-sample (65%) was Black, while Black participants made up 56% and 55% of the overall 1st-grade and 7th-grade cohorts, respectively. Importantly, participants in this sub-study did not differ significantly from the larger PYS sample on high-risk status, family socioeconomic status, the number of biological parents in the home, and parent- and teacher-reported internalizing and externalizing problems (assessed using the Child Behavior Checklist and the Teacher's Report Form). The average participant age at the neuroimaging scan was 32.60 years old (SD = 3.60, Range = 26.45-40.82).

2.3 Measures

2.3.1 Childhood Maltreatment

In this analysis, childhood maltreatment was operationalized by official CYF abuse reports filed before the participant was 18 years old. Complete CYF data on each participant include referrals for physical abuse, sexual abuse, emotional maltreatment, educational maltreatment, failure to provide for the youth, and a number for total CYF referrals. Data also included referrals filed on behalf of a sibling living in the same household, and whether a referral was substantiated or unsubstantiated by a follow-up investigation. Table 2 displays the breakdown of CYF referrals reported on behalf of either a) participants or b) siblings of participants. The table also indicates whether the referral was a) substantiated, or b) unsubstantiated. Given that the overall number of participants with substantiated CYF referrals is small (n = 30) compared to the overall sample (N = 171), we decided to consider referrals filed on behalf of participants and siblings, as well as both substantiated and unsubstantiated referrals (n = 51), in order to maximize our likelihood of detecting an effect. We divided the overall referral data (i.e., participant/sibling, unsubstantiated/substantiated) into three subgroups indicating low, moderate, and severe abuse. A CYF score of 0 indicated zero referrals, a score of 1 indicated 1-2 referrals and a score of 2 indicated 3+ referrals. We entered CYF maltreatment score as a factor in our analyses. Table 3 displays the number of participants within each CYF category (also shown in Figure 2).

2.3.2 Antisocial Behavior

Antisocial behavior was operationalized by a variation of the Psychopathy Checklist-Revised (PCL-R) criminal versatility item (Hare, 1991, 2003). The PCL-R is a semi-structured clinical interview with an additional review of collateral information such as official criminal records. The measure contains 20 items scored on a 3-point scale (0, 1, 2), with 40 being the highest score possible; higher scores indicate more psychopathic features (Blais & Ritchie, 2016). The PCL-R is a valid measure of psychopathy that reliably predicts violence cross-culturally (Hare et al., 2000) and has excellent interrater reliability (Blais & Ritchie, 2016). Here, we used the PCL-R's criminal versatility item as our primary outcome of interest (PCL-R Item 20). The criminal versatility item measures an individual's adult criminal activity using information from the structured PCL-R interview and official files such as criminal records. Specific offenses that individuals commit as adults are classified into distinct categories (e.g., theft, assault, drug offenses); the final criminal versatility score is based on the number of categories that an individual's criminal history spans. This choice was motivated by empirical evidence suggesting that criminal versatility is generally associated with severe antisocial behavior. For example, criminal versatility has been established as a risk factor for recidivistic offending (Pflueger et al., 2015), more violent offending (Vittaco et al., 2007), and greater psychopathic traits in adolescents (Campbell et al., 2004). Additionally, in a similarly design project to PYS, offenders who engaged in criminal behaviors more frequently were also the most versatile and violent (Farrington, 2003).

Based on the organization of two main national crime databases in the United States – the FBI Uniform Crime Report (UCR) (FBI, 2019) and the Bureau of Justice Statistics' National Crime Victimization Survey (NCVS) (Morgan & Truman, 2020) – and the available data within our dataset, we included the following crimes in a Violent Criminal Versatility (VCV) index: robbery,

assault, murder, and kidnapping. The PCL-R Violent Criminal Versatility score was compiled using the Self Report and Official Record item for each of the crimes (as opposed to the Official-Record-only report). The PCL-R uses a particular scoring scheme to categorize criminal versatility into a score from 0-2. This scoring suggests that 0 = <3 type of offenses, 1 = 4-5 types of offenses, 2 = 6+ types of offenses. Given we considered 5 violent crimes, we adapted the scoring scheme to accommodate the fewer number of crimes. A score of 0 = 0 offenses, 1 = 1-2 types of offenses, and 2 = 3+ types of offenses. Table 4 displays the Violent Criminal Versatility scores based on the PCL-R scoring adaptation, and Figure 3 represents the distribution of this scored variable within our dataset. In our supplemental materials, we considered alternative metrics of antisociality, including continuous violent criminal versatility variable, a continuous overall criminal versatility variable, a continuous total PCL-R score, a continuous PCL-R Factor 2 score, a continuous total number of crimes variable, and an Antisocial Personality Disorder severity score.

2.3.3 Covariates

Given that childhood maltreatment is correlated with several other risk variables, we considered the potentially confounding effects of various factors in sensitivity models and in our analyses. These choices were based on the data available and motivated by past PYS publications. Our analysis considered the following demographic covariates in all statistical models: peer delinquency (measured by the Self-Reported Delinquency Scale and the Substance Use Scale at age 15; Elliott et al., 1985) socioeconomic status (measured by the Hollingshead two-factor index at Phase A for both samples, mean age for 1st grade cohort = 6.7, mean age for 7th grade cohort = 13.1), welfare status (measured by a binary variable indicating whether or not the family was on welfare at Phase A), income (annual household income at Phase A), age at MRI scan, and race

(dichotomized as 0 for White/other participants and 1 for Black participants). We also used the juvenile delinquency item from the PCL-R as a covariate to account for a history of youth antisocial behavior (PCL-R Item 18). Similar to the scoring of the criminal versatility item, this item has a score of 0, 1, or 2 with 0 representing no history of arrests for antisocial behavior, 1 representing a history of minor offenses (e.g., possession of drugs), and 2 representing a history of serious offenses (e.g., murder). Finally, we created a cumulative risk score by dichotomizing the following continuous risk factors at the upper 25th percentile and taking the sum across variables: socioeconomic status, welfare status, income, peer delinquency, and juvenile delinquency (for this item, a score of 0 will be coded as 0, and a score of 1 or 2 will be coded as 1). This cumulative risk variable was included as a covariate in our analyses. Due to the longitudinal nature of this data set, there was incomplete data for a few of the demographic variables; most notably, the welfare (n = 16) and income (n = 28) data. Missing data was imputed using multivariate techniques.

2.3.4 Neuroimaging Data

MRI images were collected on a 3.0 Tesla Siemens MAGNETOM Trio MRI scanner at the University of Pittsburgh's Magnetic Resonance Research Center. A high-resolution anatomical image was acquired using an axial 3D MPRAGE sequence, parallel to the AC-PC line (TE/TI/TR = 3.29ms/900ms/2200ms, 1mm^3 voxel, matrix size =256x192). Diffusion-weighted images were acquired using single-shot, spin-echo planar imaging (EPI). The diffusion scan had a total of 61 diffusion sampling directions, with a b-value of 1000 s/mm^2. Anterior to posterior phase encoding was used, and planes were parallel to the AC-PC line (TR=8400ms, TE=90ms, FOV=256×256, sixty-four 2 mm slices, , matrix size=128x128).

2.3.5 White Matter Structure

Diffusion data were reconstructed using DSI-Studio; specifically, Q-space diffeomorphic reconstruction in MNI space (Yeh et al., 2011). A sampling length ratio of 1.25 was used, and the final resolution was 2 mm^3. To assay our specific white matter tracts of interest, mean quantitative anisotropy (QA) for each tract was extracted using a population-based average template. This atlas based on the Human Connectome Project HCP-842 was constructed using a total of 842 subjects' diffusion MRI data (900-subject release). This template data included diffusion images acquired using a multi-shell diffusion scheme (i.e., b-values = 1000, 2000, and 3000 s/mm^2). We extracted QA values for the uncinate fasciculus, the cingulum bundle, and the fornix. Of note, QA values range from 0 to 1, with higher values indicating less water diffusivity (and higher integrity) and lower values indicating more water diffusivity (and lower integrity) (Budde et al., 2009). Values were averaged the left and right side of the white matter tracts for an overall index of structural integrity across each pathway. We additionally ran analyses on a major visual association pathway, the Vertical Occipital Fasciculus (VOF), as a comparison tract. We hypothesized that there would be no relation between the VOF and either childhood maltreatment or antisocial behavior.

2.4 Analytic Plan

We constructed three path models testing associations between maltreatment, brain connectivity, and antisociality. The first path in this model used a regression analysis to model the relation between CYF reports and PCL-R violent criminal versatility scores using age, race, and cumulative risk exposure as covariates. Next, we conducted a second set of four regression

analyses in order to test the association between CYF reports and the three white matter bundles of interest, i.e., the uncinate fasciculus, the cingulum bundle, and the fornix, plus our control pathway (the vertical occipital fasciculus). These four regressions included age, race, and cumulative risk exposure as covariates. The third set of regression models tested for associations between the neural pathways of interest and PCL-R criminal versatility scores. This set of regressions included age, race, and cumulative risk exposure as covariates. All results reported in this document will use standardized beta coefficients. Since we planned to run 9 regression models, we planned to correct for multiple comparisons by using the Benjamini-Hochberg Procedure to reduce the false discovery rate. If pathway *a* and pathway *b* were found to be significant, we planned to use a bootstrapping method to test for an indirect effect of neural pathways on the association between CYF reports and criminal offense records. We intended to interpret the statistical significance of indirect effects using bootstrapped 95% confidence intervals based on 1000 samples with replacement. We planned to consider our effects significant if the confidence interval did not contain zero (Preacher & Hayes, 2008).

2.5 Sensitivity Analyses

In addition to our primary statistical models, we performed supplemental sensitivity analyses. These models were intended to demonstrate the robustness (or lack thereof) of our findings and evaluate the extent to which our results may have been affected by model parameters, and initially, unmodeled factors. Based on conceptual and empirical reviews suggesting the importance of family-level (e.g., income, parental education) and neighborhood factors (e.g., presence of antisocial peers) in externalizing and conduct problems (Ingoldsby & Shaw, 2002;

Shaw & Shelleby, 2014), these sensitivity analyses included a range of variables pertinent to a participant's development and bioecology.

We first intended to investigate if differences in our primary outcome of interest, PCL-R violent criminal versatility scores, could be explained by variances in violence history within our subsample. Individuals within this subsample of the PYS were recruited for a) a history of no violence, b) a history of 1-3 years of violence, and c) a history of 4+ years of violence, based on annual data collected from when the participants were 11-25 years old. For the purposes of sensitivity analyses, the two groups recruited for a history of violence were combined (men with 1-3 years of violence, n = 64, men with 4+ years of violence, n = 43). This resulted in a subgroup of 107 men recruited for a history of violence, with which we compared the group of men without any history of violence (n = 64). We also ran sensitivity analyses on our cumulative risk variable (see Covariates). We compared associations between violent criminal versatility on childhood maltreatment, race, and age ("base model") to an additional model containing the latter variables plus the cumulative risk variable ("risk model"). For all sensitivity analyses, we considered regression estimates trending in the same direction and magnitude, confidence intervals, p-values, and tests of significance between correlations to rule out strong biases.

3.0 Results

3.1 Descriptive Statistics

Descriptive statistics of the key variables used in our analysis are noted in Table 5. Table 2 and Table 3 in the "Descriptive Tables" section of the Supplement display the same variables broken down by violence group and by maltreatment group, respectively. Table 6 in this document shows a correlation matrix between the key variables in this analysis.

3.2 Childhood Maltreatment and Antisocial Behavior (*c* path)

A multiple linear regression was conducted to determine the association between childhood maltreatment and adult antisocial behavior. An unadjusted or base model that considered a simple set of covariates (age and race) found an association between moderate childhood maltreatment and violent criminal versatility, $\beta = 0.235$, $p < 0.01$. Severe childhood maltreatment was not significantly associated with violent criminal versatility, $\beta = 0.124$, $p = 0.092$. Based on findings from sensitivity analyses (please see *Results: Sensitivity Analyses*) we next ran a more complex model using a cumulative risk variable as an additional covariate (as shown in Table 7). There was no significant association between violent criminal versatility and either moderate or severe childhood maltreatment (moderate $\beta = 0.116$, $p = 0.116$; severe $\beta = 0.0479$, $p = 0.4994$). There was a significant effect of cumulative risk score, $\beta = 0.3594$, $p < 0.01$, Race, $\beta = 0.1419$, $p = 0.0452$, and age at scan, $\beta = 0.1458$, $p = 0.034$ on violent criminal versatility. As such, the results

of this analysis do not provide support for our hypothesis that childhood maltreatment in this sample is associated with heightened violent criminal versatility in adulthood upon considering relevant bioecological factors in the model.

3.3 Childhood Maltreatment and White Matter Tracts (*a* path)

Multiple regression analyses were conducted predicting the association between childhood maltreatment group (none, moderate, and severe) and the average of left and Right QA values for each brain tract, while adjusting for noted covariates. There was no significant effect of moderate childhood maltreatment on average uncinate fasciculus QA, $\beta = 0.1165$, $p = 0.1581$. There was, however, a significant effect of severe childhood maltreatment on average uncinate fasciculus QA, $\beta = 0.1877$, $p = 0.0192$. This result indicated that individuals who had experienced severe childhood maltreatment had higher levels of average QA, or *increased* structural integrity, within the uncinate fasciculus tract (see Figure 4). This association ran contrary to our hypotheses; namely, we predicted increased violent criminal versatility would be associated with *decreased* structural integrity with these tracts. None of the other variables were significant in our model exploring childhood maltreatment and uncinate fasciculus QA.

There was no significant effect of either moderate maltreatment on average fornix QA, $\beta = 0.0091$, $p = 0.911$, or severe maltreatment, $\beta = 0.0176$, $p = 0.824$. None of the other variables were significant in our model exploring childhood maltreatment and fornix QA (all p's > 0.1). There was no significant effect of either moderate maltreatment, $\beta = 0.0432$, $p = 0.606$, or severe maltreatment on average cingulum bundle QA, $\beta = 0.0316$, $p = 0.696$. None of the other variables were significant in our model exploring childhood maltreatment and cingulum bundle QA (all p's

> 0.3). Lastly, we investigated associations between childhood maltreatment and average QA in the Vertical Occipital Fasciculus, a white matter tract selected as a control tract. There was no significant effect of either moderate maltreatment, $\beta = 0.0412$, $p = 0.623$, or severe maltreatment, $\beta = 0.0825$, $p = 0.309$, on average Vertical Occipital Fasciculus QA.

Overall, we did not find support for our hypotheses that childhood maltreatment was associated with differences in any of the white matter tracts critical for emotion regulation processes. More specifically, neither moderate maltreatment nor severe maltreatment predicted QA values in the uncinate fasciculus, the cingulum bundle, and the fornix.

3.4 White Matter Tracts and Violent Criminal Versatility (*b* path)

Multiple regression analyses were conducted predicting the association between the average of left and right QA values for each neural tract and the violent criminal versatility score while adjusting for noted covariates. The average uncinate fasciculus QA value did not significantly the predict violent criminal versatility score, $\beta = -0.0347$, $p = 0.612$. Next, the average fornix QA value did not significantly the predict violent criminal versatility score, $\beta = -0.0288$, $p = 0.6799$. The average cingulum bundle QA value also did not significantly the predict violent criminal versatility score, $\beta = -0.0395$, $p = 0.5647$. In terms of the control tract, as expected, the average Vertical Occipital Fasciculus QA value did not significantly the predict violent criminal versatility score, $\beta = -0.0398$, $p = 0.5605$. These results do not support our hypotheses that lower white matter integrity in three tracts critical for emotion regulation would be associated with antisocial behavior. We found no evidence that there was any association between violent criminal versatility and QA values in the uncinate fasciculus, the cingulum bundle, and the fornix.

3.5 Indirect Effects Analysis

Due to the fact that we did not find a significant association between childhood maltreatment and the three white matter tracts (path *a*) or between the white matter tracts and violent criminal versatility (path *b*), we were not able to test for an indirect effect of white matter tracts in the association between childhood maltreatment and antisocial behavior.

3.6 Cumulative Risk, Race, Age & Violent Criminal Versatility

While included as a covariate, cumulative risk was related to many of our outcomes of interest. The cumulative risk variable significantly predicted violent criminal versatility across all the statistical tests we ran with VCV as the outcome (i.e., in models predicting VCV with childhood maltreatment as the main independent variable of interest and with white matter tracts as the main independent variable of interest). Additionally, the associations between a) race and b) scan age with violent criminal versatility were either statistically significant or trending towards significant in each of the models predicting violent criminal versatility. Please refer to the Supplement for details on these associations.

3.7 Sensitivity Analyses

We first completed sensitivity analyses related to violent criminal versatility and violence history, constructing separate regressions for each of our two groups (N=64, with no history of

violence; N=107, with a history of violence) considering the association between Maltreatment Group and Violent Criminal Versatility score. Regression estimates were variable across the different subgroup analyses, but with large standard errors (without a history of violence: Maltreatment Group 1 β =-0.0169, SE=0.3852; Maltreatment Group 2 β =-0.0979, SE=0.2798; with a history of violence, Maltreatment Group 1 β =0.1743, SE=0.1567; Maltreatment Group 2 β =0.0357, SE=0.1416, See Figure 5). Comparison of correlations across the two models, however, suggested no difference between the coefficients for Maltreatment Group 1 across the two models (z = 1.12, p=0.23) or Maltreatment Group 2 (z = 0.83, p = 0.41).

We also constructed regression models examining the association between criminal versatility on childhood maltreatment, with and without a cumulative risk variable. For the base model without the cumulative risk variable, the standardized estimate for Maltreatment Group 1 was β =0.235 (SE=0.0928) and for Maltreatment Group 2, β =0.12414 (SE=0.134). Upon including a cumulative risk variable, these estimates dropped – Maltreatment Group 1 β =0.12145 (SE= 0.157) and Maltreatment Group 2 β =0.0583 (SE=0.131). Comparison of correlations across the two models, however, suggested no difference between the coefficients for Maltreatment Group 1 (z = 1.08, p=0.28) and for Maltreatment Group 2 (z = 0.61, p =0.51) across the two models. Figure 6 displays the coefficients for these two models. However, and of note, p-values for individual coefficients for associations between maltreatment and violent criminal versatility did change across our models. For example, Maltreatment Group 1, or moderate maltreatment, was associated with violent criminal versatility in the base model (β = 0.2352, p = 0.001), but not in the more complex model including cumulative risk (β = 0. 12145, p = 0.110). We therefore decided to retain this more complex model, as the inclusion of this

cumulative risk variable influenced the strength of the association between childhood maltreatment and violent criminal versatility.

4.0 Discussion

The present study investigated whether childhood maltreatment was related to violent antisocial behavior, whether childhood maltreatment was associated with lower structural integrity in white matter tracts critical for emotion regulation, and whether variations in white matter integrity of these tracts would be related to violent antisocial behavior. Our analyses did not provide support for our hypotheses that a) childhood maltreatment would be related to increased violent criminal versatility, b) childhood maltreatment would be associated with lower structural integrity in white matter tracts critical for emotion regulation, and c) lower structural integrity in these tracts would be associated with violent criminal versatility. Overall, in contrast to our predictions, our study does not provide support for these hypotheses.

4.1 Childhood Maltreatment and Antisocial Behavior

The association between adverse early life experiences, including childhood maltreatment, and antisocial behavior is well-documented and replicated (Wilson et al., 2009; Braga et al., 2018). Notably, we did not replicate this in our study; this could be for a variety of different reasons including a) the operationalization of childhood maltreatment and b) the conceptualization of violent antisocial behavior.

Past research focused on childhood maltreatment has used of a variety of different definitions in order to measure this complex early life stressor. For example, studies focused on childhood maltreatment have considered samples who suffered institutionalization (Tottenham, 2012), harsh punishment at the hands of caregivers (Afifi et al., 2013), or sexual trauma (Tyler, 2002). In addition to the different experiences of maltreatment, there is variability in whether reports are prospective (often through official Child Protective Services [CPS] records) or retrospective (through self-reports) in nature. A recent meta-analysis has found poor agreement between prospective and retrospective reports, further complicating the picture and finding that each type of report has unique advantages and disadvantages (Baldwin et al., 2019). Asking individuals for their own reports of abuse may have the benefit of identifying more true cases of abuse (Baldwin et al., 2019). On the other hand, individuals may underreport abuse due to memory biases, including because they have forgotten abuse that occurred when they were much younger (Travaglia et al., 2016), because distress can interfere with the process of consolidating memories (Roozendaal et al., 2009), or because it was suggested or told to them that their experience was not abuse (Brewin et al., 2017), among many other reasons.

We leveraged official CPS referrals in this study, which do not suffer from challenges common in retrospective reports (e.g., recall biases). Examining official referrals also has the benefit of allowing researchers to examine abuse referrals of siblings living in the same household, information which presents an additional window into life in the household that may have been overlooked. However, using official reports to measure childhood maltreatment may miss instances of this early life stressor due to failures in reporting or the occurrence of more subtle forms of abuse (e.g., neglect, emotional maltreatment). As such, official reports generally document more severe cases of abuse (Christian, 2015; Baldwin, 2019). An added complexity to

operationalizing and measuring childhood abuse is the nuanced ways in which a host of other variables, such as severity, chronicity, and age at abuse may impact the experience of abuse (Manly et al., 2001; Cowell et al., 2015). Moving forward, it will be important to consider all of these factors in understanding links between maltreatment and violent antisocial behavior to determine if certain aspects of abuse (e.g., severity, chronicity) are more likely to be associated with antisocial behavior.

Antisocial behavior is another construct that is notorious for its complexity and multi-faceted quality. Demonstrating this heterogeneity is the fact that a DSM-5 diagnosis of Antisocial Personality Disorder requires individuals to meet three or more out of seven different criteria, giving rise to many different combinations of symptoms and behaviors (American Psychiatric Association, 2013). The symptom profiles for individuals scoring high marks on the PCL-R, another common way to distinguish severe antisocial behavior, can also be similarly heterogenous (Brinkley et al., 2004). In this study, we hypothesized that several white matter connections key for emotion regulation would have an association with antisocial behavior; we anticipated that irregular or dysfunctional emotion regulatory pathways would be associated with *violent* and *versatile* antisocial behavior specifically. As such, we chose to consider violent criminal versatility as our main dependent variable of interest. First, a greater number of adverse childhood experiences have been found to be associated with more versatile and persistent criminal behavior (Levenson & Socia, 2015). Criminal versatility further seemed an appropriate choice as an outcome variable given that a sizable body of research has consistently found faulty emotion regulation to be a major risk factor for severe antisocial behavior (Garofalo et al., 2018; Roberton et al., 2014), reactive aggression, and impulsivity (Röll et al., 2012). Work has also found that frequent and violent criminal offending also tends to be more versatile (Farrington, 2003) pointing

to a possible connection between aggression based in faulty emotion-regulation and versatile criminal offending.

Postulating that deficits in emotion-regulation contribute to antisocial and criminal behavior is consistent with *general propensity* theories of criminal behavior; that is, the belief that underlying traits and vulnerabilities (such as emotion dysregulation) contribute to antisocial tendencies and criminality (Gottfredson & Hirschi, 1990; Baumesiter & Heatherton, 1996). Criminal versatility is an expected association within this framework, given that underlying problems with emotion regulation (and subsequently, antisocial behavior) are unlikely to be confined to a specific situation (DeLisi et al., 2019). Alternative conceptualizations of criminality include more of an emphasis on contextual factors driving criminal behavior, such as general strain theory (Agnew, 1992) or social information processing (Crick & Dodge, 1994); under these theories, more specialized offending and less criminal versatility might be hypothesized depending on the context. The choice of criminal versatility as an outcome variable was consistent in theory with a) our interest in neural connections underlying emotion regulation (i.e., a general propensity perspective) and b) with overall evidence suggesting that criminals tend to exhibit versatility over specialization (DeLisi et al., 2019).

In our sample, the criminal versatility variable was derived from the PCL-R Criminal Versatility Item, which scores individuals with a 0, 1, or 2 depending on the number of crime categories that their criminal history covers. Importantly, this criminal versatility item considers *all* crimes (including non-violent offenses, such as drug-related offenses), and is one item that contributes to an overall PCL-R score. We decided to only consider crimes deemed by the FBI and UCR as "violent" in order to better approximate our construct of interest, and to analyze this "violent criminal versatility" as a stand-alone measure. In supplemental analyses of the

associations between childhood maltreatment and different operationalizations of violent antisociality (see Figure 7) these alternative outcome variables were generally significantly associated with one or both levels of maltreatment (i.e., moderate or severe). While the violent criminal versatility score was a novel conceptualization of violent antisocial behavior and thus adds unique dimensions to this work, our supplemental analyses suggest that other operationalizations in this sample may have better served our research questions.

Additional work is needed to more richly conceptualize and measure violent antisocial behavior in relation to the hypothesized emotion dysregulation underpinning. There are complex and overlapping associations between different dimensions of aggression, antisocial behavior, and criminal versatility that may be underlying this discrepancy. Broadly, literature suggests that a) different subtypes of antisocial behavior (e.g., proactive and reactive aggression) may have opposite underlying patterns of emotion dysregulation, and b) it is unclear whether certain features of antisocial behavior (i.e., instrumental aggression and psychopathic traits) may be more related to criminal versatility than others (i.e., reactive aggression).

4.1.1 Proactive and Reactive Aggression

One common way that researchers have understood aggression has been to distinguish between proactive (or instrumental) and reactive aggression. Proactive aggression, or an "emotionally cool form of aggression used to achieve instrumental goals" (Lozier et al., 2014), is associated with low autonomic nervous system arousal (Raine et al., 2006) while reactive aggression tends to reflect emotional dysregulation in response to provocation (Frick, 2012). In a sample of incarcerated adolescent females, reactive aggression and not proactive aggression was associated with low emotion regulation and anger to perceived provocations; meanwhile, proactive

aggression was associated with callous-unemotional traits (Marsee & Frick, 2007). Further complicating the picture, however, is the fact that these types of aggression can co-occur and are correlated at $r = .70$ (Vitaro & Brendgen, 2005).

Taken together, this evidence suggests that while both reactive and proactive aggression may have underlying emotion regulation deficits, reactive aggression is associated more with *heightened* emotion reactivity and proactive aggression is associated with *blunted* reactivity. With respect to criminal versatility, a group of offenders who had committed at least one act of proactive aggression demonstrated significantly more psychopathic traits, criminal versatility, and violent antisocial behavior compared to offenders who solely committed reactive aggression (Cornell et al., 1996). This finding suggests that criminal versatility may be more strongly associated to proactive aggression and less so to reactive aggression.

4.1.2 Callous-Unemotional Traits

A related distinction that has been used to understand antisocial behavior is the presence, or lack thereof, of psychopathic traits (in adults) or callous-unemotional (CU) traits (in children and adolescents). Individuals with such traits are described as having a "lack of guilt, a lack of empathy, and shallow affect" (Frick, 2016) and are at risk for particularly severe and chronic violent offending throughout the lifespan compared to antisocial individuals without these traits (Frick & White, 2008). Importantly, children with antisocial behavior *and* CU traits tend to be emotionally *under* reactive to threats; their AB tends to be more premeditated or proactive, and they may have an impoverished experience of fear and guilt (Viding et al., 2012b). This pattern is consistent for adults, too, with studies finding associations between psychopathic traits and proactive aggression (Cornell et al., 1996). Individuals engaging in antisocial behavior without

CU, on the other hand, tend to be emotionally overactive to threats (Viding et al., 2012a), a distinction that maps more clearly onto reactive aggression.

Research on individuals with psychopathic or CU traits have found associations not only with proactive aggression, but also with earlier onset, frequency, and versatility of criminal offending (Forth & Book, 2010, p. 263). This suggests that criminal versatility may be in some cases a characteristic of offenders with psychopathic traits and proactive aggression and less related to reactive aggression. It is important to reiterate, however, the extremely high correlation between these two types of aggression, which may further obfuscate any associations between emotion regulation, aggression, violence, and criminal versatility.

4.1.3 PLC-R Factor 1 and Factor 2

A third distinction among antisocial behavior lies in the two factors of the PCL-R. The PCL-R Factor 1 represents the interpersonal and affective dimensions of psychopathy (e.g., narcissism, superficial charm) and Factor 2 represents the impulsive and antisocial dimensions (e.g., heightened emotional distress, deviancy) (Cunningham & Reidy, 1998; Hicks & Patrick, 2006). Empirical evidence suggests that PCL-R Factor 2 is positively correlated with amygdala hyperreactivity and with negative emotionality (NEM), or the tendency to experience negative emotional states such as anxiety and anger. Conversely, Factor 1 is related to amygdala hyporeactivity and is inversely associated with NEM (Hicks & Patrick, 2006; Hyde et al., 2014). Factor 2 scores have also been found to be predictive of reactive aggression (Skeem & Mulvey, 2001) whereas Factor 1 is more predictive of instrumental aggression (Woodworth & Porter, 2002). Work has consistently implicated faulty emotion regulation circuitry as predictive of reactive aggression (Röll et al., 2012), including as a link in the association between childhood

maltreatment and reactive aggression (Shields & Cicchetti, 1998). Research exploring subtypes of psychopathy has found that criminal versatility is characteristic of offenders high on both F1-type traits and F2-type traits, further highlighting the muddled picture depicting antisocial behavior and criminal versatility (Swogger & Kosson, 2012).

Overall, findings from literature examining proactive/reactive aggression, antisocial behavior with/without CU traits, and Factor 1/Factor 2 of the PCL-R illuminate that while emotion dysregulation appears to be a central component to all of these constructs, these associations may be quite different (as evidenced by, for example, a pattern of over-reactivity for antisocial behavior *without* CU traits and under-reactivity for antisocial behavior *with* CU traits). This suggests that there are likely patterns of emotion dysregulation unique to subtypes of antisocial behavior that were not modeled in our study and could therefore explain the lack of significant associations between brain regions underlying emotion regulation and violent criminal versatility. Further evidence for this is provided by our supplemental analyses considering the association between a self-regulation questionnaire, the Abbreviated Dysregulation Inventory (ADI), and violent criminal versatility in this sample (please see *Supplement: Abbreviated Dysregulation Inventory*). The ADI provides an overall dysregulation score, as well as an affective sub-score, neither of which were significantly associated with violent criminal versatility.

Literature also suggests that these subtypes of antisocial behavior may also have differential associations with childhood maltreatment. For example, one study found that among violent offenders, childhood physical abuse was strongly associated with reactive aggression, but not proactive reaction (Kolla et al., 2013). However, other work has found strong associations between childhood maltreatment and both proactive and reactive aggression (Hoeve et al., 2015). This brief review exposes many gaps in the literature that represent exciting and novel prospects

for future studies. In sum, work continuing to explore the associations between subtypes of antisocial behavior, emotion regulation, and maltreatment could provide critical and much needed contributions to the field.

4.2 White Matter Tract Integrity

We selected three tracts with empirically demonstrated roles in emotion regulation and investigated their association with both childhood maltreatment and violent criminal versatility. Again, we hypothesized that these tracts would be associated with both these constructs based on past literature. We did not observe any such associations in our analyses. While this may be due to how we operationalized and measured childhood maltreatment and violent criminal versatility as discussed above, it is also worth considering the way we chose to measure the brain.

We chose to investigate white matter tracts motivated by the fact that these tracts connect brain areas central to emotion regulation, such as the prefrontal cortex, hippocampus, and amygdala. Instead of examining the structure or function of any one area in particular, examining the connective tracts may provide insight into how information is "communicated" between these areas; furthermore, DWI has stronger reliability and consistency over time compared to other neuroimaging methods. In this way, the study of white matter tracts combines both structural and functional elements, as it measures connectivity between areas while preserving the reliability of structural neuroimaging. An additional reason we examined white matter tracts is that the literature exploring white matter connectivity and antisocial behavior is still at a fledgling stage, with reports of inconsistent findings and calls for continued research in this space (Waller et al., 2017).

There are many different ways to measure white matter tract integrity. The most common is Fractional Anisotropy (FA), but several other metrics may be derived including the one we used in this study, Quantitative Anisotropy (QA). Unlike FA, QA is calculated in a way such that it allows the researcher to distinguish between crossing white matter tract fibers, potentially providing more accurate insight into the composition of a given tract (Yeh et al., 2018). While QA has the benefit over FA of increased sensitivity to crossing fibers, traditional measures of both QA and FA coarsely estimate white matter tract integrity. A tract's density or integrity may change across the length of the fibers; therefore, extracting one QA value to represent an entire tract may neglect valuable variability in the tract. DSI studio, the software used to extract these values, does allow for a more nuanced calculation of QA that generates QA values for different subsections of a given tract. Future research should consider exploring this alternative extraction to gain a deeper understanding of the associations between the brain, childhood maltreatment, and antisocial behavior.

Furthermore, the algorithm we used in DSI Studio generates one QA value for each side of the brain – for example, there are two separate values for the uncinate fasciculus tract, one for the left hemisphere and one for the right hemisphere. In order to avoid running an extreme number of statistical tests (and thereby increasing our chances of a spurious finding), we averaged the left and right QA values for each tract into one mean QA value. This process may have further masked important differences between two halves of the same tract. For example, decreased white matter integrity in the *right* uncinate fasciculus specifically has been associated with increased antisocial behavior across various studies (Wolf et al., 2015; Sobhani et al., 2015).

4.3 Other Considerations

An impressive and growing body of research has delineated the various bioecological factors that may contribute to maladaptive outcomes such as antisocial behavior (Ingoldsby & Shaw, 2002; Evans et al., 2013; Baskin-Sommers, 2016). These factors exist at multiple levels of the individual (e.g., macrosystem: neighborhood violence; microsystem: peer deviance and childhood maltreatment in the home). Motivated by these facts and past research, we derived a cumulative risk variable that measured several known correlates of adult antisocial behavior. Throughout our analyses, this cumulative risk variable was consistently a significant predictor of violent criminal versatility. This effect fits in with the literature suggesting that composite adversity scores explain much of the variance in negative outcomes (Evans et al., 2013). However, the predominant challenge in interpreting and understanding cumulative risk is that, while explanatory, these models are atheoretical and lack a mechanism. Put another way, it is still poorly understood *why* multiple risk exposures are more likely to result in worse outcomes. Additionally, while cumulative risk variables have excellent explanatory power at the population level, research suggest these scores fail to predict outcomes at the individual level (Baldwin et al., 2021). Research in this space should attempt to balance specificity and sensitivity, with an eye toward the collinearity of different adversities (e.g., community violence versus interpersonal violence) and also potential mechanisms to understand links between maltreatment, adversity, and antisociality.

4.4 Future Directions and Concluding Remarks

While the study characteristics of the PYS dataset contributed to many of the decisions made in this analysis (particularly with respect to the operationalization of key constructs), the plethora of data available also holds promise for future analyses. While we used CYF reports here as a measure of maltreatment, more nuanced maltreatment data exist for a portion of our sample. Future analyses could make use of this additional information to further capture the complex construct of childhood maltreatment. With respect to the measure of antisocial behavior, a subset of our individuals also participated in an additional follow-up a year after the scan, during which data on antisocial behavior was collected. Specifically, researchers asked about study participants about delinquency, arrests, and aggression. Connecting these differential measures of our constructs of interest could aid in elucidating links between maltreatment and antisociality.

The results of these analyses did not confirm our hypotheses that childhood maltreatment, emotion regulatory neural pathways, and violent antisocial behavior would be all associated. However, sustained progress exploring these ideas is still warranted and will contribute to our understanding of the impact of childhood abuse in underpinning antisocial behavior. This knowledge can lend further legitimacy to the claim that child abuse has deleterious effects on individuals through establishing that early life stress in part predicts antisocial behavior through altering key emotion regulatory pathways. Establishing that deficient emotion regulation circuitry following childhood abuse may be driving some aspects of maladaptive behaviors would allow clinical interventions to target specific symptoms, to focus on strengthening these pathways, and to be applied before maladaptive behavior has fully manifested.

Appendix A Tables

Table 1

PYS Assessments for 1st-grade and 7th-grade Cohorts

1st-grade Cohort																		
Age	6.5	7	7.5	8	8.5	9	9.5	10	11	12	13	14	15	16	17	18	19	25	28
7th-grade Cohort																			
Age	12.5	13	13.5	14	14.5	15	16	17	18	19	20	21	22	23	24	25	35		

Table 2

Substantiated and Unsubstantiated CYF Referrals for Study Participants and Siblings

	Substantiated	Unsubstantiated	Total
Participant	30	11	41
Sibling	8	2	10
Total	38	13	51

Table 3

CYF Scores for each participant

CYF Score	Number of Participants
0	121
1	21
2	29

Table 4

Violent Criminal Versatility scored mirroring PCL-R Criminal Versatility Score.

Violent Criminal Versatility Score	Number of Participants
0	46
1	90
2	35

Table 5

Descriptive summary of study variables

	Overall (N=171)
Race	
0 (White/other)	55 (32.2%)
1 (Black)	116 (67.8%)
Age	
Mean (SD)	32.655 (3.627)
Range	26.449 - 40.823
Socioeconomic Status	

	Overall (N=171)
Missing Data	3
Mean (SD)	34.363 (13.534)
Range	6.000 - 66.000
Welfare Status	
Missing Data	16
0 (not on welfare)	78 (50.3%)
1 (on welfare)	77 (49.7%)
Income (in USD)	
Missing Data	28
Mean (SD)	$15650.543 (12481.733)
Range	$3480.0 - $60000.0
Peer Delinquency	
Missing Data	4
Mean (SD)	6.006 (6.936)
Range	0.000 - 34.000
Juvenile Delinquency	
0 (no offense Hx)	21 (12.3%)
1 (Hx minor offenses)	59 (34.5%)
2 (Hx major offenses)	91 (53.2%)
Cumulative Risk	
Missing Data	32
Mean (SD)	2.432 (1.104)
Range	0.000 - 5.000
Maltreatment (Mltx)	
0 (No Mtlx)	121 (70.8%)
1 (Moderate Mtlx)	21 (12.3%)
2 (Severe Mtlx)	29 (17.0%)
Viol Crim. Vers. (VCV)	
0 (No VCV)	46 (26.9%)
1 (Moderate VCV)	90 (52.6%)
2 (Severe VCV)	35 (20.5%)

Note. M and *SD* represent mean and standard deviation, respectively. Socioeconomic status, welfare status, and income were measured by parent report at Phase A (mean age of participants in 1st grade cohort = 6.7, mean age for 7th grade cohort = 13.1). The descriptive statistics here correspond to the original, non-imputed data.

Table 6

Means, standard deviations, and correlations with confidence intervals

Variable	*M*	*SD*	1	2	3	4	5	6	7
1. Mltx	0.46	0.77							
2. VCV	1.37	1.27	.18* [.03, .32]						
3. Age	32.65	3.63	.05 [-.10, .20]	.13 [-.02, .28]					
4. SES	34.36	13.53	-.25** [-.39, -.10]	-.21** [-.35, -.06]	.09 [-.06, .24]				
5. Peer Delinquency	6.01	6.94	.09 [-.06, .24]	.27** [.12, .40]	.06 [-.09, .21]	-.05 [-.20, .11]			
6. Welfare Status	0.50	0.50	.17* [.02, .32]	.26** [.11, .40]	-.16 [-.31, .00]	-.48** [-.60, -.35]	.05 [-.11, .20]		
7. Cumulative Risk	1.67	0.93	.27** [.11, .41]	.45** [.32, .57]	.05 [-.11, .21]	-.00 [-.16, .15]	.58** [.46, .67]	.49** [.36, .60]	
8. Race	0.68	0.47	-0.01 [-.16, .14]	.22** [.08, .36]	-.03 [-.18, .12]	0.15* [-.30, -.00]	-.06 [-.21, .09]	.27** [.11, .41]	.20* [.04,]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$. Mltx = Maltreatment, VCV = Violent Criminal Versatility, SES = Socioeconomic Status. Maltreatment was coded as continuous for the purposes of including the variable in the correlation table, although it was treated as a factor in our analyses. This table was produced using *non-imputed* data.

Table 7

Output for analysis regressing Violent Criminal Versatility on Childhood Maltreatment and covariates

	Estimate	Std. Error	t-value	p
Intercept	0.00	0.09217	8.135	0.000
Maltreatment Grp 1	0.1160	0.15319	1.582	0.1156
Maltreatment Grp 2	0.0479	0.12945	0.677	0.4994
Age at Scan	0.1458	0.04681	2.141	0.0338
Race (Black)	0.1419	0.10324	2.018	0.0452
Cumulative Risk	0.3594	0.05191	4.759	0.000

Note. The estimates represent standardized regression beta coefficients.

Appendix B Figures

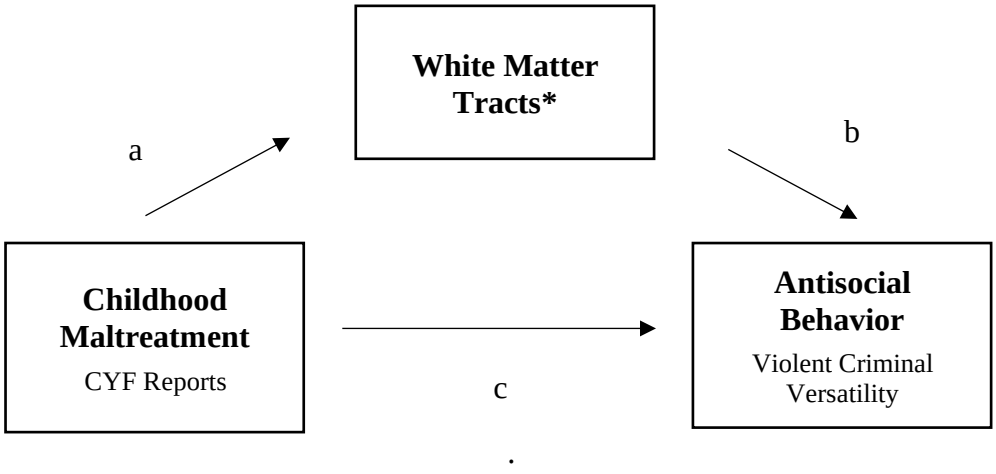

Figure 1

A representation of the model testing the indirect effect of white matter tracts in the relation between childhood maltreatment and antisocial behavior

Note. **White matter tracts*** include the Uncinate Fasciculus, the Cingulum Bundle, the Fornix, and Vertical Occipital Fasciculus pathways

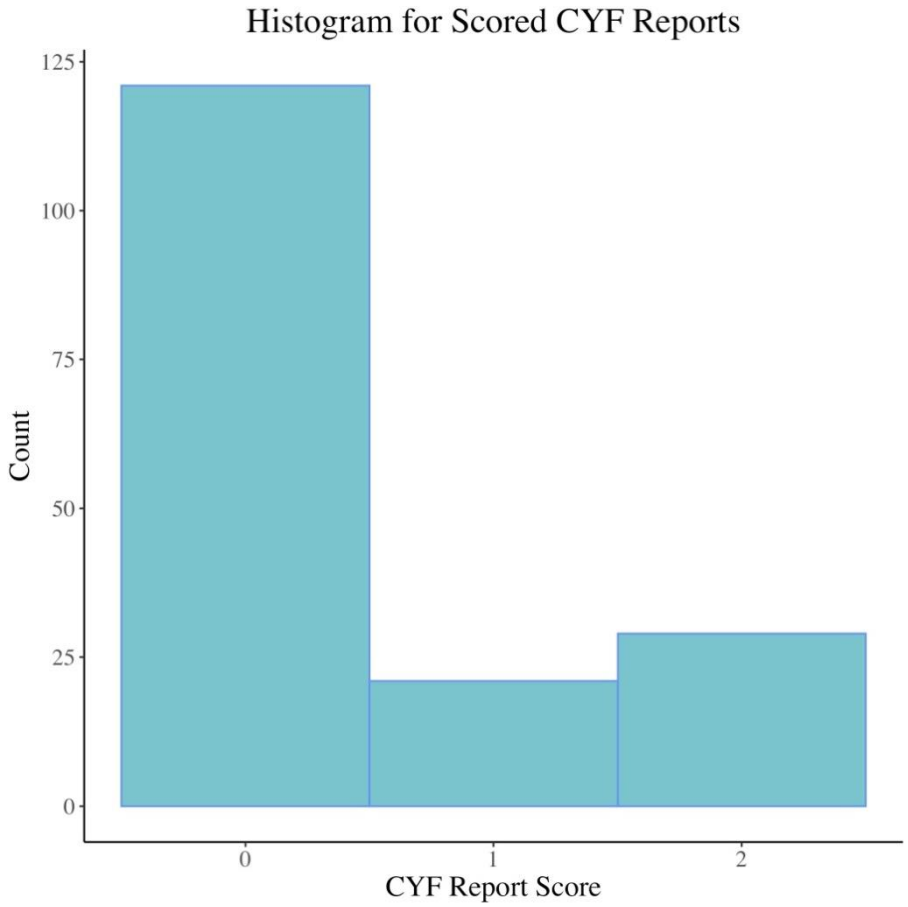

Figure 2

Histogram for Scored CYF Reports

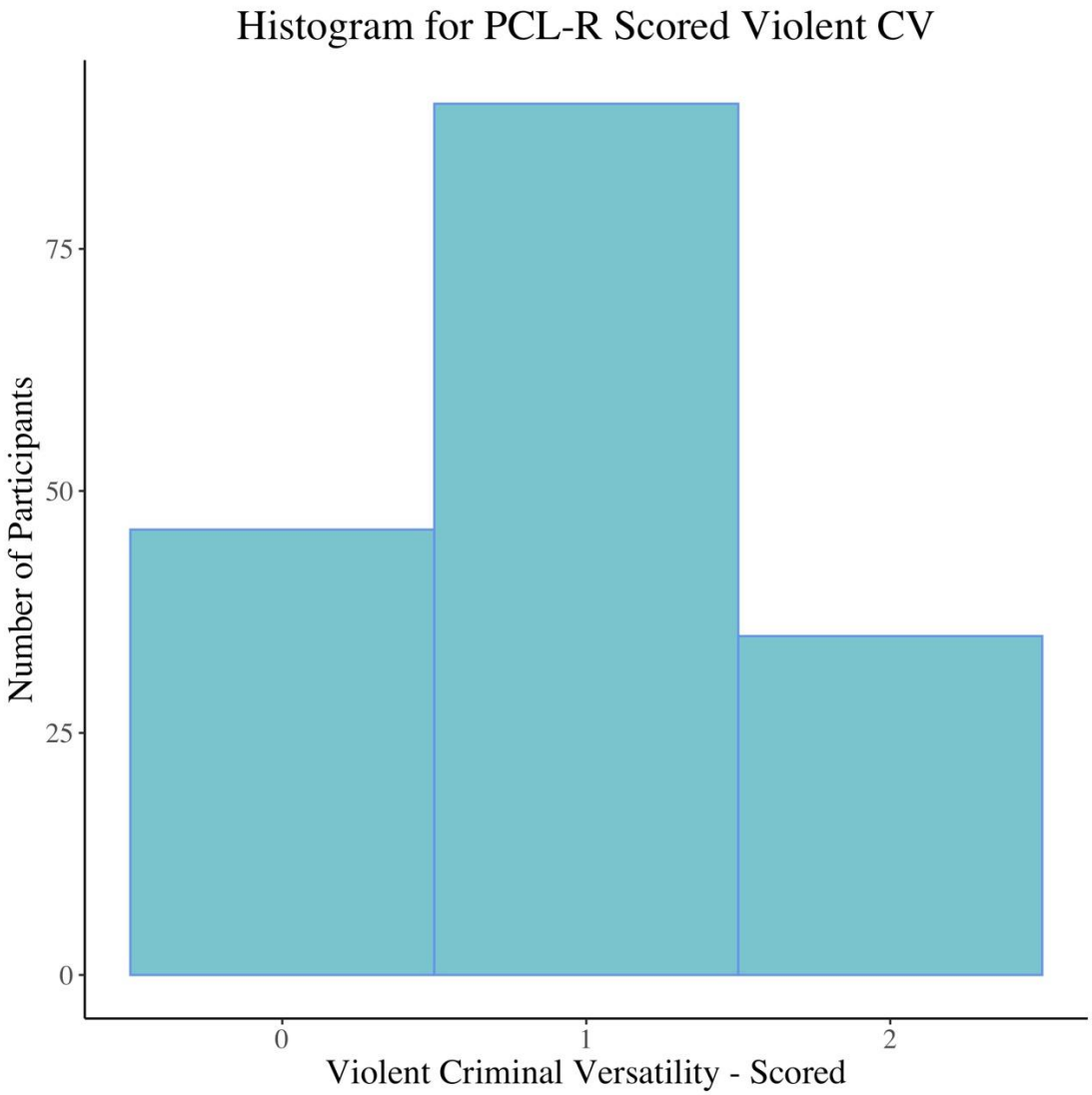

Figure 3

Histogram for Scored Violent Criminal Versatility Variable

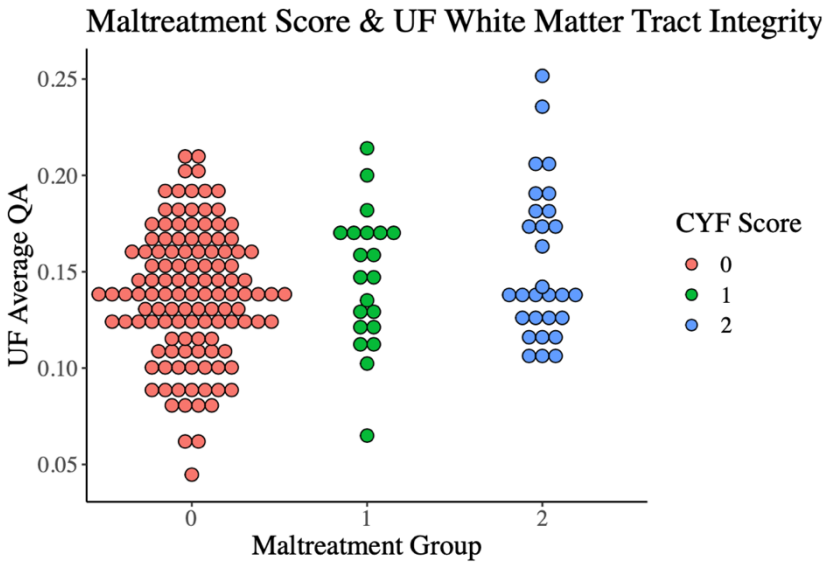

Figure 4

Uncinate Fasciculus Average Quantitative Anisotropy by Maltreatment Group

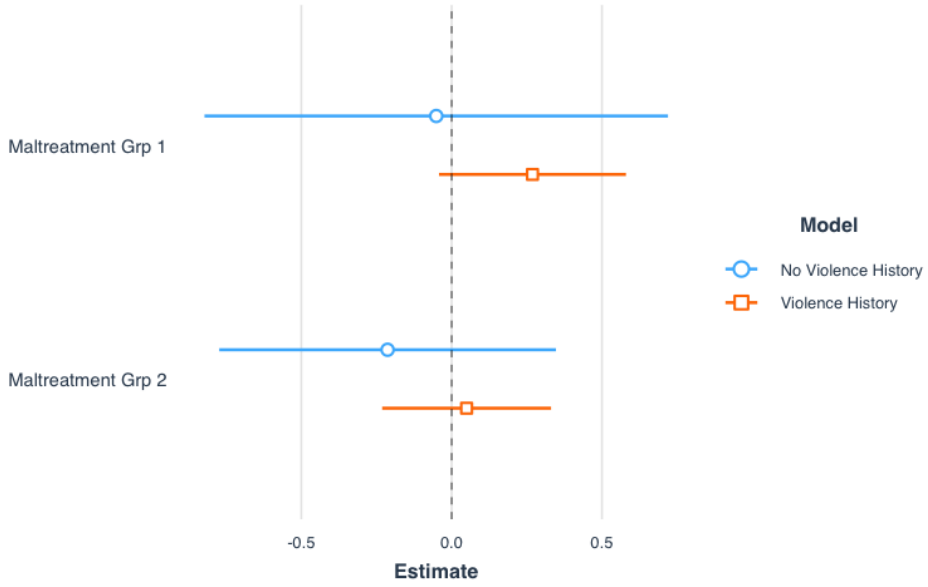

Figure 5

Standardized coefficient estimates comparing two models predicting Violent Criminal Versatility Score from Maltreatment Group

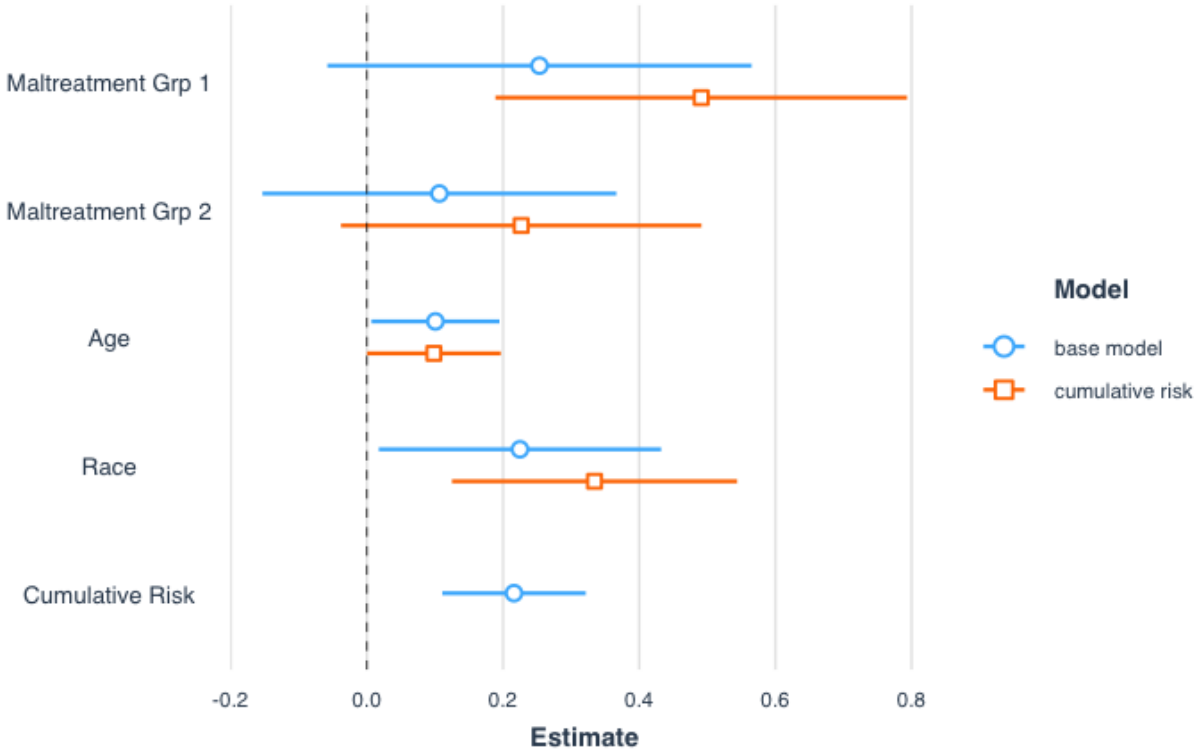

Figure 6

Standardized coefficient estimates comparing a base model and a cumulative risk model predicting Violent Criminal Versatility score from Maltreatment Group

Antisocial Behavior Operationalization	**Moderate Maltreatment**	**Severe Maltreatment**
Continuous VCV Variable	*b* = .43, t(165) = 1.47, p = 0.14	*b* = .18, t(165) = 0.76, p = 0.45
Continuous Overall CV Variable	*b* = 1.46, t(165) = 2.01, p = 0.05	*b* = .75, t(165) = 1.24, p = 0.22
Continuous Total # of Crimes	*b* = 1.46, t(165) = 2.01, p = 0.05	*b* = .75, t(165) = 1.24, p = 0.22
Continuous Total PCL-R Score	*b* = 4.10, t(165) = 1.98, p = 0.05	*b* = 4.45, t(165) = 2.57, p = 0.01
Continuous PCL-R Factor 2 Score	*b* = 2.85, t(165) = 2.42, p = 0.02	*b* = 2.24, t(165) = 2.28, p = 0.02
Antisocial Personality Disorder Severity Score	*b* = 1.54, t(165) = 1.86, p = 0.06	*b* = 1.32, t(165) = 1.91, p = 0.06

Figure 7

Estimates regressing alternative operationalizations of antisocial behavior on childhood maltreatment and covariates

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