Developmental Risk Factors and Neurocognitive Functioning of Antisocial Behavior and Callous-Unemotional Traits

by

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Dedication

To my family (O’Doyle rules) and to 16-year-old Hailey- we did it!

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Abstract

Youth antisocial behavior (AB; lying, aggression, rule-breaking) is a major public health concern due to its high prevalence and harmful consequences. Recent research has suggested that youth with AB are a heterogeneous group, which may undermine intervention success. For instance, elevated callous-unemotional (CU) traits are associated with more severe and persistent AB. Moreover, CU traits are a downward extension of psychopathic traits, and are a risk factor for adult psychopathy. Previous work suggests that the combination of AB and elevated CU traits may be etiologically distinct from AB alone. However, previous research has often been limited by the use of case-control designs in highly specialized populations, at severe levels of both AB and CU traits (e.g., adjudicated, clinical or criminal). This dissertation is comprised of three studies that examine developmental trajectories and neurocognitive deficits of AB versus CU traits, in community populations, across varying levels of AB and CU traits. To better understand developmental precursors of CU traits, Study 1 examines associations among parental psychopathic traits, parenting practices, and offspring CU traits in adolescence, using a genetically-informed design. Parental interpersonal-affective psychopathic traits were associated with adolescent CU traits and parenting (increased conflict, reduced involvement). Moreover, increased conflict and reduced involvement partially explained associations between parental interpersonal-affective traits and adolescent CU traits. Finally, using a twin difference design, we confirmed that adolescent CU traits were significantly impacted by non-shared environmental parenting influences (increased conflict, reduced involvement). Study 2 identifies neurocognitive deficits associated with the combination of AB and CU traits, in contrast to AB alone. Neither

AB, nor CU traits alone, were associated with cognitive functioning when accounting for demographic factors. However, AB and CU traits interacted to predict reaction time variability. At low levels of CU traits, AB was associated with higher reaction time variability (traditionally thought to reflect worse sustained attention). At high levels of CU traits, antisocial behavior was associated lower reaction time variability (thought to reflect better sustained attention). Finally, Study 3 examines patterns of neural network connectivity underlying psychopathic traits in young adulthood using a person-specific approach and determines whether specific features of psychopathy are characterized by distinct network features. There was significant heterogeneity in neural networks of participants, which were characterized by person-specific connections and no common connections across the sample. Psychopathic traits, particularly affective traits, were associated with connection density between the default mode network and central executive network, such that greater density was associated with elevated psychopathic traits. The general discussion chapter of this dissertation highlights the implications of this research for intervention approaches, empirical considerations, and future directions.

Chapter 1 General Introduction

Youth antisocial behavior (AB), including lying, aggression, and rule-breaking, is highly prevalent, extremely costly to perpetrators, victims, families, and society, and, unfortunately, historically difficult to treat (McCart et al., 2006; Rivenbark et al., 2018). Research suggests that AB can be parsed into etiologically distinct subtypes (Frick et al., 2014b), which may impact intervention success. For example, elevated callous-unemotional (CU) traits characterize a more stable and severe trajectory of AB (Frick et al., 2014b). CU traits are now included in the DSM-5 as the 'limited prosocial emotions' specifier for CD and ODD (American Psychiatric Association, 2013). Research suggests that elevated levels of both AB and CU traits may be characterized by distinct etiological mechanisms and neurocognitive deficits, even compared to AB and CU traits in isolation (Frick et al., 2014a). Thus, the aim of the current dissertation is to specify developmental trajectories and neurocognitive deficits that are unique to AB versus CU traits, which could then inform targeted interventions.

Antisocial Behavior and Psychopathic Traits

AB is broadly defined as actions or attitudes that violate the personal or property rights of others and/or societal norms (American Psychiatric Association, 2013). AB includes clinical diagnoses (e.g., Conduct Disorder and Oppositional Defiant Disorder in childhood, Antisocial Personality Disorder in adulthood) and harmful behaviors more broadly, such as conduct problems, substance use, and delinquency (American Psychiatric Association, 2013). AB is a significant public health concern due to its serious long-term effects on both victims and

perpetrators, and substantial financial cost, with the cost of crime estimated at $1.7 trillion in the United States (Anderson, 2012; Krug et al., 2002). To address this public health crisis, researchers have thus sought to identify risk factors for AB to inform prevention and treatment. One of the strongest dispositional predictors of AB is psychopathy (e.g., Aharoni & Kiehl, 2013; Dolan & Doyle, 2000; Neumann & Hare, 2008). Psychopathy is a personality construct in adulthood defined by a constellation of harmful traits and behaviors, including superficial charm, dishonesty, callousness, irresponsibility, and poor impulse control (Cleckley, 1941). Psychopathic traits have been shown to predict violence across community, college, and criminal samples (Reidy et al., 2011), and are associated with more severe violence and increased rates of recidivism (Reidy et al., 2015). Psychopathy alone has been estimated to account for approximately $460 billion of the annual cost of crime (Kiehl & Hoffman, 2011). Unfortunately, individuals with psychopathic traits are notoriously difficult to treat (Reidy et al., 2013). Although personality is considered to be relatively stable in adulthood, personality appears to be more malleable in childhood (Roberts & DelVecchio, 2000). Thus, researchers have sought to identify developmental risk factors, including dispositional precursors, of both adult psychopathic traits and AB that may serve as effective targets for early intervention.

Callous-Unemotional Traits

Accumulating evidence suggests that the presence of callous-unemotional (CU) traits in childhood is a dispositional risk factor for more severe and persistent forms of AB (Frick et al., 2014a). CU traits (including remorselessness, lack of empathy, shallow emotion) are considered to be a downward extension of interpersonal and affective components of psychopathy (Salekin, 2017), and there is some evidence that CU traits are a risk factor for adult psychopathy (Lynam et al., 2007). Youth with both AB and CU traits perpetrate more severe aggression and

premeditated aggression (Frick et al., 2003; Lawing et al., 2010; Marsee & Frick, 2007). Additionally, CU traits are associated with an earlier onset of severe AB (Dandreaux & Frick, 2009; Frick et al., 2014b). Importantly, beyond severity of AB, CU traits have also been associated with distinct biological, cognitive, emotional, and social characteristics from broader AB, consistent with research on adults with psychopathic traits (Frick et al., 2014b). Understanding unique etiological mechanisms contributing to AB versus CU traits is therefore critical to developing appropriate interventions.

Theoretical Approach

This dissertation uses multiple frameworks to understand developmental precursors and neurocognitive functioning associated with AB versus CU traits. First, within a development psychopathology framework, psychopathology is thought to emerge via interactions between the child and context, which change across development (Frick et al., 2014a). That is, AB and CU traits most likely emerge as a result of the combination of individual factors, such as genetic phenotype, and environmental factors, such as parenting practices (Frick & Viding, 2009; Waller et al., 2015). Moreover, the developmental psychopathology framework highlights the importance of studying development in both typically developing youth and youth with psychopathology, including the use of dimensional analyses to understand developmental processes across a range of functioning over the lifespan (Cicchetti & Rogosch, 2002). Study 1 examines the role of both inherited risk (via parent personality) and environmental factors (parenting) on the emergence of CU traits, using a dimensional measure of CU traits in a community sample of adolescent twins, and thus also benefits from a genetically-informed design. Study 2 also examines associations between individual-level factors, specifically neurocognitive abilities and AB versus CU traits, dimensionally in a community sample.

A second framework utilized in this dissertation is Belsky's determinants of parenting, which states that parenting is influenced by: 1) parent characteristics, such as personality, 2) child characteristics, such as temperament, and 3) contextual sources of stress and support (i.e., other factors in the family's social context that can impact parenting), such as marital quality, parental occupation, and broader social networks (Belsky, 1984). Within this model, parent characteristics are thought to be the most important determinant of parenting. Study 1 heavily draws on this framework by examining associations among parent psychopathic traits, parenting practice, and offspring CU traits, using a genetically-informed design.

A final framework that guides this dissertation is the Research Domain Criteria (RDoC), a dimensional approach recently developed by the National Institute of Mental Health. The RDoC framework posits that psychopathology represents a disruption (of varying degrees) in normative functioning of biological symptoms (Cuthbert & Insel, 2013). Psychopathology is therefore characterized using biological and cognitive data (i.e., "biocognitive"; e.g., genetic, neural, cognitive, and affective systems) (Cuthbert, 2014). Thus, objective markers, rather than subjective accounts or clinician judgement, are used to diagnose psychological disorders, as is done in the broader field of medicine (Cuthbert & Insel, 2013). Brazil and colleagues recently proposed a similar framework for AB and psychopathic traits, suggesting that biocognitive data could be used to identify subgroups characterized by distinct deficits, which would entail distinct intervention approaches (Brazil et al., 2018). All three studies draw from this framework by attempting to identify biocognitive markers (including genetic risk, cognitive functioning, and neural circuitry) associated with AB, CU traits, and psychopathic traits.

The goal of my dissertation is to better understand the etiology of AB, CU traits, and psychopathy from these multiple perspectives. The aim of the first study is to understand how

CU traits develop – that is, what contexts promote them (e.g., parenting and parental personality). The aim of the second study is to identify individual-level cognitive factors that are associated with CU traits versus AB. The aim of the third study is to examine patterns of brain connectivity that underlie psychopathic traits in young adulthood.

The Development of CU Traits and AB: The Importance of Parenting and Parents

A large body of work has examined developmental precursors of both CU traits and AB (Frick et al., 2014a; Viding & McCrory, 2018). Identifying *modifiable* risk factors of CU traits and AB in particular will be more directly applicable to the development of effective intervention strategies. Importantly, although AB and CU traits have been found to be moderately heritable (Gard et al., 2019; Viding et al., 2005), CU traits and AB are also strongly influenced by environmental factors, such as parenting practices (Frick et al., 2018; Viding & McCrory, 2018). In fact, adoption studies have found that parenting practices appear to *counter* inherited risk for AB and CU traits (Hyde et al., 2016; Waller et al., 2016). Thus, researchers have focused on determining which parenting practices may be most influential on AB or CU traits.

Warm and harsh parenting. A substantial developmental literature has demonstrated the importance of parenting on the development of AB (Loeber & Hay, 1997; Patterson, 2002), and parenting also appears to play a significant role in the growth and stability of CU traits (Waller et al., 2013; Waller & Hyde, 2017). Recent research has demonstrated that parenting consists of discrete dimensions (Darling & Steinberg, 1993; Grusec, 2011; Maccoby, 1994; Smetana, 2017), including warmth (or responsiveness, support) and harshness (or demandingness, control). Negative parenting practices, including inconsistent and harsh parenting practices, have been associated with both AB and CU traits (Frick et al., 2018; Waller et al., 2013). Some research suggests that negative parenting is more strongly associated

specifically with AB in the absence of CU traits (Frick et al., 2014b). Reduced positive parenting practices, including fewer warm and responsive parenting behaviors, on the other hand, may be more strongly associated with elevated CU traits, (Frick et al., 2018; Waller et al., 2013).

Of note, numerous studies have utilized composite measures of negative versus positive parenting, collapsing across different types of parenting behaviors within these categories (Frick et al., 2018). However, recent research suggests that *specific* parenting practices may be more critical to the emergence of AB versus CU traits. For example, in a parenting intervention study (e.g., Fast Track), Pasalich and colleagues (2016) identified distinct pathways for parental discipline versus warmth. Specifically, the intervention was associated with reduced harsh discipline, which predicted lower levels of conduct disorder symptoms, and with increased parental warmth, which predicted lower levels of CU traits (Pasalich et al., 2016). Further, a genetically-informed study of CU traits in childhood found low parental warmth, rather than parental harshness, to be a unique environmental predictor of CU traits, when accounting for their overlap (Waller, Hyde, et al., 2018). Parenting therefore certainly appears to play a key role in the development of both AB and CU traits, but further research is necessary to delineate what parenting practices are most impactful on AB versus CU traits specifically.

Moreover, whereas several studies have examined the impacts of parenting on AB and CU traits, fewer studies have examined whether individual factors within parents may impact parenting practices. Developmental theory suggests that parent personality traits can predict parenting practices (Belsky, 1984). Indeed, a previous meta-analysis found that parenting practices were strongly predicted by parent personality traits, as measured by the Big Five personality factors (Prinzie et al., 2009). Thus, parent personality traits may meaningfully predict parenting practices that could influence the development of AB or CU traits.

Parent psychopathic traits and parenting practices. One personality construct that may impact parenting practices is psychopathy. Individuals with elevated levels of psychopathic traits have been found to demonstrate a callous and antagonistic interpersonal style (Mooney et al., 2019; Skeem et al., 2011). As such, individuals with elevated levels of psychopathic traits may have similarly antagonistic interactions with their children. In this way, parental psychopathic traits may predict child AB or CU traits via parenting practices. Preliminary research suggests that parental psychopathic traits are indeed associated with parenting practices that may exacerbate levels of CU traits (Beaver et al., 2014; Cox et al., 2018). These findings suggest that parents high in psychopathic traits may be more likely to use specific parenting practices that have been found to predict CU traits and AB (i.e., more harsh parenting, low parental warmth; Waller et al., 2013). Moreover, given similarities between CU traits and adult psychopathy, children with CU traits specifically may have "inherited" these features from parents with high levels of psychopathic traits. That is, there may also be direct associations between parental psychopathic traits and child CU traits. Indeed, an abundance of family, twin, and adoption studies have supported the familial resemblance of AB (Labella & Masten, 2018).

In contrast to the substantial literature on the intergenerational transmission of AB, only two studies have directly examined the association between parent psychopathy, parenting practices, and child CU traits (Diaz et al., 2018; Loney et al., 2007). Importantly preliminary results do suggest that parents with psychopathic or CU traits may be more likely to have children with CU traits (Diaz et al., 2018; Loney et al., 2007). However, when researchers also examined parenting, parenting practices appeared to at least partially explain associations between parental psychopathy and child CU traits (Diaz et al., 2018; Loney et al., 2007). Further, when accounting for the mediating role of parenting dysfunction, one study found that the direct

association between maternal psychopathy and child CU traits was no longer significant (Loney et al., 2007). Notably, these studies differed in sample type (i.e., clinical and community), and were focused on different age periods (i.e., early versus late childhood) (Diaz et al., 2018; Loney et al., 2007). Thus, additional research is needed in adolescent samples, a developmental period when youth begin to spend more time outside of the home and rates of AB begin to increase (Moffitt, 2018), particularly because the role of genetic effects versus environmental influences appear to differ across development (Dick et al., 2016; Ferguson, 2010; Moore et al., 2019). Further, neither of these studies utilized genetically informed designs and were therefore unable to separate out potential non-heritable transmission. Taken together, parenting clearly plays a role in the emergence of both CU traits and AB, and individual differences in parents (including their personality) may also contribute to differential developmental pathways of CU traits and AB either directly or via parenting practices. However, further examination is needed in larger adolescent community samples that can parse apart direct versus indirect pathways, using genetically informed designs that allow researchers to determine the extent to which non-shared environmental factors influence the emergence of CU traits.

Neurocognitive Functioning within CU Traits and AB

Similar to the developmental literature, research suggests that youth AB and CU traits are associated with differing behavioral phenotypes, potentially reflecting unique deficits in neurocognitive functioning (Blair et al., 2018; Hyde et al., 2013; Waller, Dotterer, et al., 2018). AB has been historically associated with emotional dysregulation and reactive aggression. As such, researchers have theorized that AB is marked by deficits in executive functioning (Patrick et al., 2012). Indeed, several meta-analyses have supported associations between AB and poor

executive functioning (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011), as well as lower intelligence (Ogilvie et al., 2011; Sánchez de Ribera et al., 2019).

In contrast, CU traits are often associated with diminished emotional responding and proactive aggression (Frick & White, 2008; Hyde et al., 2013). Thus, CU traits may instead be associated with higher intelligence (reflected by planning involved in premeditated aggression) and/or better executive functioning (reflected by better emotional regulation). Importantly, few studies have empirically tested the extent to which CU traits demonstrate distinct associations with intelligence and executive functioning compared to AB. Within the existing literature, studies have not typically found significant associations between CU traits and intelligence, particularly when accounting for AB (e.g., Allen et al., 2013; Fanti et al., 2016; Loney et al., 2006; Loney et al., 1998; Pardini, 2011). However, Rydell and colleagues (2019) found that CU traits were associated with *higher* IQ when controlling for both AB and ADHD symptoms in an adolescent community sample.

The literature on CU traits and executive functioning has been similarly mixed. Whereas some studies have found no significant associations with CU traits when accounting for AB (Bohlin et al., 2012; Fanti et al., 2016; Graziano et al., 2019; Hadjicharalambous & Fanti, 2018), other research suggests that CU traits are associated with *better* executive functioning (Rydell & Brocki, 2019). Importantly, the studies varied greatly in their measurement of executive functioning, including types of tasks used and the specific functions of executive functioning measured. For instance, AB has often been associated with response inhibition, which refers to the ability to suppress a prepotent response (Munakata et al., 2011) and selective attention, which is the ability to attend to necessary information in the environment while ignoring less relevant information (Brodeur et al., 1997). Previous studies did not find associations between CU traits

and response inhibition or selective attention. However, executive functioning also involves sustained attention, defined as the ability to maintain focus over time (Hofmann et al., 2012). One study did find that CU traits were associated *better* sustained attention (Rydell & Brocki, 2019). Thus, further research is needed to clarify the extent to which AB versus CU traits may be characterized by deficits in specific components of executive function.

Moreover, although studies have often looked at direct effects of CU traits and AB on cognitive abilities, other research suggests that interactive effects between CU traits and AB may better explain cognitive deficits. While some studies have found that impairments emerge only at elevated levels of both AB and CU traits (Hadjicharalambous & Fanti, 2018; Platje et al., 2018; Wall et al., 2016), other research suggests that elevated AB and CU traits are associated with *better* executive functioning (Baskin-Sommers et al., 2015; Fanti et al., 2016; Graziano et al., 2019). Thus, the combination of elevated CU traits and AB may be differentially associated with specific functions of executive functioning, compared to either elevated AB or CU traits alone.

Neural Circuitry within AB versus CU Traits

Resting-state networks. Importantly, the cognitive deficits observed within AB and CU traits are each associated with unique systems within the brain (Blair et al., 2018; Hyde et al., 2013; Waller, Dotterer, et al., 2018). To address inconsistencies in the behavioral literature, researchers have also utilized brain imaging techniques to determine whether AB and CU traits are also related to differences in the functioning or organization of neural circuitry. Functional magnetic resonance imaging (fMRI) is a prominent technique used to study brain function. fMRI assesses activation, or changes in blood flow associated with activity, and functional connectivity, or changes in the contribution of activation in one area to activation in another across different conditions (Friston et al., 1995). Functional connectivity is thought to reflect the

level of functional communication between regions. Cognitive abilities, including executive functioning, rely on the functioning of, and communication between, multiple regions within the brain (i.e., neural networks; Menon, 2011). Thus, examining functional connectivity of neural networks may be particularly informative to understanding unique neurocognitive profiles associated with AB versus CU traits.

One way to assess network functional connectivity is by measuring the correlation of spontaneous activation patterns of brain regions (Greicius et al., 2003; van den Heuvel & Pol, 2010); that is, measuring network connectivity "at rest". In these "task-free" experiments, participants are typically instructed to relax with their eyes closed or looking at a fixation cross (van den Heuvel & Pol, 2010). In this way, resting-state functional connectivity can thus be thought of as one's "baseline" or "intrinsic" patterns of connectivity. Importantly, resting-state functional connectivity has been found to predict behavioral performance on tasks (e.g., Sala-Llonch et al., 2012; Tian et al., 2012; Zou et al., 2013). Moreover, differences in the connectivity of resting-state networks have been linked to various psychiatric disorders (Greicius, 2008; Kaiser et al., 2015; Menon, 2011; Sylvester et al., 2012).

In addition to functional connectivity, which primarily examines the strength of connections, more recent work has also begun to examine the extent to which the *organization* of functional brain networks predicts cognitive abilities (van den Heuvel & Pol, 2010). "Topology" can be conceptualized as the way a brain network is arranged and describes the characteristics of relations between brain regions within a network (e.g., number, length, and direction, as well as strength, of connections) (Bullmore & Sporns, 2009; Stam & Reijneveld, 2007). Organizational properties of a network directly influence the efficiency of the network, including its ability to communicate and integrate information (Bullmore & Sporns, 2009; De Vico Fallani et al., 2014;

Latora & Marchiori, 2001). Thus, differences in network topology likely impact cognitive abilities. Indeed, efficient organization of neural networks has been associated with higher intelligence (Song et al., 2008; van den Heuvel et al., 2009). Taken together, differences in the functional connectivity and organization of resting-state networks may contribute to observed behavioral deficits within AB versus CU traits.

Implicated neural networks in AB and CU traits. Altered connectivity of three resting-state networks in particular have been posited to underlie psychopathology broadly, including AB and CU traits: (1) the default mode network (DMN), including the ventromedial prefrontal cortex (vmPFC) and posterior cingulate cortex (PCC), (2) the salience (or cingulo-opercular) network (SN), including the anterior insula and anterior cingulate cortex (ACC), and (3) the central executive (or frontoparietal) network (CEN), including the dorsolateral prefrontal cortex (dlPFC) and posterior parietal cortex (PPC) (Menon, 2011). The DMN is a task-negative network because it is typically de-activated during cognitive tasks and is activated in resting-state, as well as during tasks that require self-referential thinking and perspective taking (Buckner et al., 2008; Greicius et al., 2003). In contrast, the CEN and SN are task-positive networks. The CEN is involved in working memory and decision-making during goal-directed behavior, and brain regions in this network are strongly co-activated during cognitively challenging tasks (Menon & Uddin, 2010). The SN adjusts arousal and attention based on external cues and internal states and enables switching between other networks (Sridharan et al., 2008), thus modulating the activity of both the CEN and DMN (Goulden et al., 2014; Menon & Uddin, 2010).

Recent theories have suggested that differences in connectivity the SN and the DMN impede the processing of complex sensory information in a way that may lead to severe AB

(Hamilton, Hiatt Racer, & Newman, 2015; Menon, 2011). That is, network functioning may impact affective and cognitive abilities, including executive functioning, crucial to the development of prosocial behavior (Blair, 2017). For example, the impaired integration theory hypothesizes that psychopathy is characterized by intact functioning in the CEN, but decreased functioning in the SN and DMN, as well as less coordination and flexible switching between networks (Hamilton et al., 2015). Recent studies have begun to link AB, psychopathic traits, and CU traits to impaired resting-state connectivity within the SN and DMN in youth and adults (e.g., Aghajani et al., 2017; Broulidakis et al., 2016; Motzkin, Newman, Kiehl, & Koenigs, 2011; Philippi et al., 2015; Pu et al., 2017; Thijssen & Kiehl, 2017). Additionally, preliminary findings suggest unique associations between network topology and AB and psychopathic traits (Lindner et al., 2018; Lu et al., 2017; Tillem et al., 2018; Yang et al., 2012), such as differences in the functional organization of the DMN (Lindner et al., 2018). However, it is still unclear whether different features *within* psychopathy (i.e., interpersonal, affective, impulsive, or antisocial features) are characterized by unique patterns of network topology. Moreover, previous research has been primarily conducted in offender or adjudicated populations, and thus further research is needed to examine network topology in community samples to determine whether differential network organization is present across varying levels of psychopathic traits.

Specific Aims of this Dissertation

The purpose of this dissertation is to better understand heterogeneity within individuals with AB and CU/psychopathic traits via several perspectives, which all emphasize dimensional approaches and examining multiple levels of risk. Specifically, the first study focuses on unique developmental precursors (including genetic versus environmental risk) of CU traits in adolescence. The aim of the second study is to examine associations between cognitive abilities

and the combination of AB and CU traits versus AB alone. Finally, the third study utilizes neuroimaging to determine whether psychopathic traits are also associated with differences in neural circuitry in young adulthood, which may underlie cognitive functioning.

Notably, there are several ongoing issues in the current literature on unique developmental and neurocognitive correlates of AB versus CU traits that have yet to be addressed. First, previous studies have often been limited by lack of specificity in observed associations. That is, several of the previously reviewed findings have been observed in youth with both elevated AB and CU traits, whereas other studies did not account for their overlap (i.e., did not include measures of both). Thus, it is often unclear if previous results are reflective of AB or CU traits uniquely.

Second, previous findings may be more attributable to *severity* of CU traits or AB. Importantly, accumulating evidence now suggests that both AB and CU traits are dimensional in nature (Kreuger et al., 2007). Thus, it is unclear whether previously found impairments are present across the spectrum of AB and CU traits or are only apparent in clinical-threshold presentations. For example, much of the existing evidence has been found within clinical or adjudicated samples (e.g., Jones et al., 2009) that are inherently characterized by severe levels of AB and CU traits, and which likely display the most functional impairment.

Moreover, a majority of work has focused on examining elevated CU traits in the context of elevated AB, often using case-control designs to compare antisocial youth with and without CU traits (Blair, 2013; Blair et al., 2014; Frick et al., 2014a). Children with elevated CU traits often also demonstrate more severe AB than children without CU traits (Frick et al., 2003). Thus, previous findings may be more attributable to *severity* of AB specifically, rather than CU traits uniquely. Indeed, larger studies of relatively healthy community samples have not been able to

replicate previous associations, with results at times contradicting traditional theories of AB and CU traits (e.g., Dotterer et al., 2017; Dotterer et al., 2019). In addition, previous research has often been limited to single gender, Caucasian samples, further limiting the generalizability of results (e.g., Jones et al., 2009; Sebastian et al., 2014; Viding et al., 2012).

As such, it is critical to investigate these questions in more diverse community samples (i.e., mixed gender, mixed race) to clarify whether previously identified developmental factors and neurocognitive deficits are characteristic of AB and CU traits dimensionally, in less specialized populations. Each study detailed in the chapters that follow seeks to better understand specificity in associations and utilize analytic strategies to parse apart unique effects of AB versus CU or psychopathic traits using dimensional measures in large community samples. The dissertation also benefits from a developmental perspective by examining associations within various age groups (i.e., childhood/early adolescence and young adulthood).

Study 1: Associations Between Parental Psychopathic Traits, Parenting, and Adolescent Callous-Unemotional Traits

Aim 1. Examine whether parental (maternal or paternal) psychopathic traits are associated with levels of adolescent CU traits.

Aim 2. Examine whether parental psychopathic traits are related to parenting practices.

Aim 3. Test whether parenting explains some of the association between psychopathic traits and adolescent CU traits.

Aim 4. Utilize a monozygotic (identical) twin difference design to confirm whether associations between parenting and adolescent CU traits are due, at least in part, to non-shared environmental influences.

Study 2: Neurocognitive Abilities Associated with Antisocial Behavior with and without Callous-Unemotional Traits in a Community Sample

Aim 1. Examine associations between cognitive functioning and dimensional measures of AB and CU traits, controlling for their overlap.

Aim 2. Determine whether there are any significant interactive effects of AB x CU traits associated with cognitive functioning.

Study 3: Connections that Characterize Cunning: Affective Traits are Associated with Personalized Patterns of Resting-State Network Connectivity

Aim 1. Generate individual-specific connectivity maps for each participant consisting of connections both across and within the DMN, SN, and CEN using a state-of-the-art data-driven directed connectivity network approach (group iterative multiple model estimation; GIMME).

Aim 2. Examine whether psychopathic traits are uniquely associated with specific network features (i.e., network density; node centrality) across the sample.

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Chapter 2: Associations Between Parental Psychopathic Traits, Parenting, And Adolescent Callous-Unemotional Traits

Introduction

Callous-unemotional (CU) traits, including callousness, lack of empathy, and shallow affect, are thought to distinguish a distinct subgroup of youth with serious antisocial behavior (AB) (Frick et al., 2014; Waller et al., 2019). CU traits are associated with more stable and severe conduct problems, as well as more severe antisocial outcomes in adulthood (Frick et al., 2014). As such, understanding the development of CU traits is critical to identifying targets for intervention to prevent serious AB, particularly during childhood when AB may be more malleable (Reid et al., 2004).

Psychopathy and Callous-Unemotional Traits

CU traits have been conceptualized as a downward extension of some of the interpersonal and affective components of adult psychopathic traits, including grandiosity and lack of remorse (Salekin, 2017), and are a developmental risk factor for later psychopathy (Lynam et al., 2007). Given the conceptual links between CU traits and psychopathy, as well as the moderate heritability of CU traits and psychopathy (Moore et al., 2019), children with CU traits may be more likely to have parents with elevated psychopathic traits. That is, we might expect there to be direct associations (via heritable or familial factors) between parental psychopathy and CU traits in offspring.

Somewhat surprisingly, only two studies have examined whether this hypothesis is true. In a community sample, Loney and colleagues (2007) found that *mother* psychopathic

interpersonal-affective traits were associated with child CU traits (in children age 7 to 14, mean age 10 years, n=83), but did not include fathers. In a clinical sample, Diaz and colleagues (2018) found that mother psychopathic traits (both interpersonal-affective traits and impulsive-antisocial traits) and father interpersonal-affective traits were associated with offspring CU traits in early childhood (in children age 3 to 15, mean age 8 years; n=306). Notably, the role of genetic effects versus environmental influences appear to differ across development (Dick, Adkins, Sally, & Kuo, 2016; Ferguson, 2010; Moore, Blair, Hettema, & Roberson-Nay, 2019). Thus, it is still unclear whether parental psychopathy is also associated with offspring CU traits during adolescence in particular, a developmental period when youth begin to spend more time outside of the home and rates of AB increase (Moffitt, 2018). Additionally, previous associations between parental psychopathy and child CU traits appeared to differ depending on informant. For instance, Diaz and colleagues (2018) found that self-reported mother interpersonal-affective traits were associated with father-reported CU traits, but not mother-reported CU traits. Loney and colleagues (2007) used a combined mother and teacher reported CU traits scale, and thus is it unclear the extent to which associations generalized across different informants. Taken together, associations between parental psychopathy and offspring CU traits require further replication in different developmental stages and across multiple informants.

In addition, developmental theory suggests that parent's personality traits can influence parenting practices (Belsky, 1984). Indeed, a previous meta-analysis found that parenting practices were broadly predicted by parent personality traits, as measured by the Big Five personality factors (Prinzie et al., 2009). Psychopathic traits are characterized by a callous and antagonistic interpersonal style (Hare & Neumann, 2008; Mooney et al., 2019). As such, individuals high in psychopathic traits may have similarly antagonistic interactions with their

children. Indeed, preliminary research suggests that psychopathic traits are related to more negative parenting (Beaver et al., 2014), less positive parenting (Schwartz et al., 2017), poor supervision (Schwartz et al., 2017) and higher levels of authoritarian parenting (i.e., display low warmth and high levels of control), as well as permissive parenting (i.e., have little concern for rules or structure, and place their own needs and desires before those of the child) (Cox et al., 2018). It is thus possible that parental psychopathic traits also predict child CU traits indirectly via parenting practices, in addition to (or in place of) any direct (heritable) effects from parental psychopathy to offspring CU traits.

Consistent with this possibility, Loney and colleagues (2007) found that a broad measure of negative parenting ("parenting dysfunction") mediated the association between mother interpersonal-affective traits and CU traits in children, and that the direct association between mother psychopathy and child CU traits was no longer significant (Loney et al., 2007). In their clinical sample at an earlier developmental period, Diaz and colleagues (2018) found that specific parenting practices (i.e., negative parenting versus parental warmth) were associated with child CU traits above and beyond levels of parental psychopathy. However, no other studies have examined whether parenting practices explain the association between parental psychopathy and offspring CU traits. As such, further research is needed to examine associations among parental psychopathic traits, distinct parenting dimensions, and offspring CU traits in at-risk adolescent community samples, where there is a range of CU traits, and with a sample size large enough to separate indirect and direct effects.

Importantly, the few studies examining parent psychopathy, child CU traits, and parenting practices did not utilize genetically informed study designs and thus were unable to control for the effects of common genes within families. As a result, previously observed

associations between parental traits, parenting, and offspring psychopathic traits may reflect gene-environment correlations (rGEs). That is, biological parents may provide both direct genetic risk (i.e., psychopathy) and environmental risk (i.e., negative parenting, low parental warmth) (passive rGE; Knafo & Jaffee, 2013). Alternatively, children at genetic risk for callous-unemotional traits that display disruptive behaviors may evoke specific parenting reactions (evocative rGE; Hawes et al., 2011; Klahr & Burt, 2014; Moore et al., 2019). One method to confirm the presence of environmental (i.e., non-genetic) transmission is examining monozygotic (MZ) twin differences. By examining differences in exposure and outcomes for twins who share 100% of their DNA, researchers can determine the extent to which nonshared environmental factors influence the emergence of CU traits. Indeed, a recent paper in the current sample, at an earlier developmental period (in children age 6 to 11) found that twin differences in parenting practices (combined mother and father report) were related to twin differences in child CU traits. That is, the twin who experienced higher levels of harsh parenting and less parental warmth also had higher levels of CU traits (Waller et al., 2018). However, as this study focused on childhood, it is unclear whether there are similar environmental influences of parenting on *adolescent* CU traits, as opposed to CU traits in early childhood.

Finally, developmental research has often focused specifically on the impact of mothering on child behaviors and traits. However, research suggests that there are gender differences in the expression of psychopathic traits (Efferson & Glenn, 2018). As such, the association between psychopathic traits and parenting may also differ between mothers and fathers. Moreover, previous research suggests that there are unique associations between father versus mother psychopathic traits and behavioral phenotypes in child CU traits (Dadds et al., 2014). For example, in their clinical sample of young children, Diaz and colleagues (2018) found

differential associations among psychopathic traits, parenting, and child CU traits between mothers and fathers; however, the pattern of results differed when looking within versus across informant. However, no other studies have compared associations between mother versus father psychopathic traits, parenting, and child CU traits. Further, some research suggests etiological mechanisms of CU traits may also differ for boys versus girls (Essau, Sasagawa, & Frick, 2006; Fontaine, Rijsdijk, McCrory, & Viding, 2010). To this point, both Diaz and colleagues (2018) and another study in adult offspring (Auty, Farrington, & Coid, 2015) found that offspring gender significantly moderated associations among father psychopathy, parenting, and CU traits. Thus, further research is needed to examine these associations in adolescence, within a community sample including varying levels of CU traits, including multiple informants.

Current Study

In the current study we sought to expand on the existing literature on associations among parental psychopathic traits, parenting, and offspring CU traits in a community sample of adolescent twins that included data from both mothers and fathers. First, we examined whether parental psychopathic traits were associated with levels of adolescent CU traits. Based on findings in younger children (Diaz et al., 2018; Loney et al., 2007), we hypothesized that both mother and father psychopathic traits would be directly associated with higher levels of CU traits. Second, we examined whether parental psychopathic traits were related to parenting practices. Because we focused on adolescents (versus early childhood in other parenting-CU traits studies), we examined measures of harshness (parental conflict) and involvement, as involvement captures a developmentally appropriate expression of warmth and engagement during late childhood and adolescence. We hypothesized that parents with higher levels of psychopathic traits would demonstrate harsher parenting and lower involvement. Third, we

tested whether parenting explained some of the variance in the association between psychopathic traits and child CU traits. We hypothesized that there would be an indirect effect such that parental psychopathic traits would be associated with adolescent CU traits via higher levels of conflict and lower levels of involvement. Fourth, we utilized a monozygotic twin difference design to confirm whether associations between parenting and adolescent CU traits are due, at least in part, to non-shared environmental influences. We hypothesized that the twin that experienced more conflict and less involved parenting would show higher level of CU traits. In a set of exploratory analyses, we examined whether twin gender moderated any of these associations. Finally, to examine potential reporter and shared method effects, for all aims we first looked at associations within informant and then examined whether associations were significant when looking across different informants.

Methods

Participants

Participants in this study included 550 twins from 275 families living in south-central Michigan that are part of the ongoing Michigan Twin Neurogenetics Study (MTwiNS). Twins were originally recruited at age 6 – 10 for the Twin Study of Behavioral and Emotional Development in Children (TBED-C) within the Michigan State University Twin Registry (see Burt & Klump, 2019). Twins were recruited into one of two cohorts. The population-based cohort was sampled from birth records to represent all families with twins living within 120 miles of Michigan State University. The second, at-risk cohort was recruited from the same area, but only included families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (i.e., the mean for the state of Michigan at the onset of recruitment) (see Burt & Klump, 2019). The MTwiNS follow-up study was recruited from the latter subsample, as

well as those in the first sample that would have qualified for the second sample (i.e., they lived in neighborhood with above mean levels of poverty), and thus represents families with twins living in neighborhoods with above average levels of family poverty. The average reported combined annual family income within MTwiNS was between $60,000 and $69,999, ranging from less than $4,999 to greater than $90,000. 12% of MTwiNS families reported an annual income below the 2017 federal poverty line of $24,600 per year and 59% reported annual income below the living wage for a family of 4 in Michigan (http://livingwage.mit.edu/states/26), consistent with a relatively low-income sample. Due to missing data (i.e., missing data on mother psychopathic traits, a main predictor variable), most analyses within the current study included 502 twins (56.4% male). Parent-reported race of the 502 children included was as follows: 75.7% White/Caucasian, 15.0% Black/African American, 1.2% Hispanic, 1.2% Pacific Islander, .8% Asian, 0.8% Native American, and 5.3% Other. Participants were primarily adolescence though the sample ranged in age from 7 to 18 years (Mean age = 13.84 years; SD 2.70; only 7.6% of the sample was 10 or younger). The included 502 participants did not significantly differ from the original 550 participants in family annual income ($t(537)=1.30$, $p=.193$), mother's education ($t(546)=.13$, $p=.90$), gender ($x^2(1)=.09$, $p=.76$), or race ($x^2(1)=1.24$, $p=.27$), but did significantly differ in age ($t(546)=-6.591$, $p<.001$). Included participants were younger.

Additionally, father self-reported psychopathic traits were only available for 205 out of the 275 families (409 participants); thus, analyses in which father psychopathic traits were the main predictor were limited to 409 participants. The 409 participants with father data (self-reported psychopathic traits) did not significantly differ from the original 550 participants in: mother's education ($t(546)=.96$, $p=.34$), gender ($x^2(1)=.02$, $p=.90$), or age ($t(546)=-1.90$, $p=.06$), but did significantly differ in family annual income ($t(537)=5.65$, $p<.001$) and race ($x^2(1)=26.01$,

p<.001). Included participants with father-reported data had higher family annual income and were more likely to be White. Race and annual family income were included as covariates in all analyses. The study protocol was approved by the University of Michigan Institutional Review Board.

Measures

Parent psychopathic traits. Parent psychopathic traits were assessed using the 29-item Self-Report Psychopathy Short-Form (Paulhus et al., 2015), a self-report measure of psychopathy derived from and shown to correlate highly with the Psychopathy Checklist-Revised (PCL-R; Hare, 1999) The items can be grouped into two dimensions of psychopathy to mirror the PCL-R: an interpersonal-affective factor (e.g., "I have pretended to be someone else in order to get something"; "I never feel guilty over hurting others") and an impulsive-antisocial factor (e.g., "I've often done dangerous things just for the thrill of it"; "I have tried to hit someone with a vehicle"). We calculated separate summed scores of each factor for mothers and fathers. Descriptives are provided in Table 1. Of note, parental psychopathy was a family-level variable (i.e., the variable is the same for twins in the same family).

CU traits. CU traits were assessed using parent and child report on the 24 item Inventory of Callous-Unemotional Traits, which includes callousness (e.g., "unconcerned about feelings of others"), uncaring (e.g., "always tries best"), and unemotionality (e.g., "hides feelings") (ICU; Essau et al., 2006; Kimonis et al., 2008). Consist with prior studies (Waller et al., 2015), we calculated a total 22-item summed score, excluding items 10 and 23. We calculated separate scores for father-, mother-, and child-reported total adolescent CU traits. Descriptives are provided in Table 1.

Parenting. Perceptions of parenting were assessed using parent and child report on the 42-item Parent Environment Questionnaire (PEQ; Elkins et al., 1997). Consistent with previous research (Sypher et al., 2019), we used the 12-item conflict scale (harsh parenting) and 12-item involvement scale (warm/involved parenting). Higher scores on the conflict scale (e.g., "My parent sometimes hits me in anger") indicate greater levels of harsh, conflictual parenting. Higher scores on the involvement scale (e.g., "My parent comforts me when I am discouraged or have had a disappointment") indicate more involved, warm, and supportive parenting. We calculated separate sum scales for self-reported (mothers and fathers) and child-reported conflict and involvement of maternal parenting. Notably, child-report of paternal parenting was not collected in the current study. Descriptives are provided in Table 1.

Analytic Plan

All analyses were conducted in MPlus version 8.3 (Muthén & Muthén, 2020). To account for the nesting of siblings in the model, all analyses were carried out using the Type=COMPLEX command. For all aims we examined mother versus father psychopathic traits separately. For our primary aims, we examined a series of analyses comparing "within" informant models (i.e., same reporter for all variables) to "across" informant models (i.e., different reporters of variables). By comparing results within versus across informant, we were able to determine whether any significant associations were influenced by shared informant variance with parent self-reports of his or her own personality. In all analyses, we controlled for parent-reported adolescent gender (0= Male, 1=Female), age, and annual family income. We additionally included parent-reported adolescent race, a socially constructed category, as a covariate to control for differences in exposure to systemic racism and the various unequal exposures to stress, trauma, and opportunity

for people of color in the United States (0=Non-White; 1=White as White is the largest group) (Jones, 2001).

To address our first aim, we examined parental psychopathic traits as predictors of adolescent CU traits (Figure 2.1A-B). To address our second aim, we examined parental psychopathic traits as predictors of parenting practices (parental involvement and conflict) (Figure 2.1C-D). To address our third aim, we used path modeling to determine whether there were indirect effects between either of the psychopathy factors and adolescent CU traits via 1) parental conflict 2) parental involvement (four indirect paths total in each model; Figure 2.2). We only tested for indirect effects when we had found a significant association between 1) psychopathic factor and adolescent CU traits and 2) parenting dimension and adolescent CU traits. Parameters were estimated using ML and 95% confidence intervals (CI) for indirect effects that were obtained using bias corrected bootstrapping (iterations = 5000) (Falk, 2018). For all results, we highlight associations that met a strict conservative threshold to account for our six primary models (i.e., three primary aims, separate models for mothers and fathers) that were tested (i.e., Bonferroni-correction $0.05/6 = p < .008$).

To determine whether associations between parenting and adolescent CU traits were at least partially due to non-shared environmental influences (i.e., not inadvertently showing 3 variables all influenced by the same genes and thus are all associated), we examined whether MZ twin differences in experiences of parenting were related to twin differences in CU traits (e.g., whether twin with higher exposure to parental conflict or less exposure to parental involvement had higher levels of CU traits). We created MZ twin difference scores for CU traits, parental conflict, and parental involvement by subtracting Twin 2's score from Twin 1's score (see Supplemental Table 2.1 for descriptives). We then examined zero-order correlations between

parenting difference scores and adolescent CU traits difference scores (both across and within informant; Supplemental Figure 2.1A). In a set of exploratory analyses, we also examined regressions that included difference scores for both dimensions of parenting as predictors of adolescent CU traits difference scores to determine whether associations were specific to parental involvement versus conflict, consistent with previous work from this sample (Waller et al., 2018) (Supplemental Figure 2.1B).

Finally, to examine whether twin gender moderated any associations, we ran multi-group models for each primary aim of interest in which parameters were fixed and freed with fit compared across models using the Satorra-Bentler scaled x^2 difference test (Satorra, 2000). Additionally, all analyses were repeated in the subsample of participants older than 10 years old. The patterns of findings remained the same and thus we present the results using the full sample.

Results

Zero-order correlations between adolescent CU traits and parental psychopathy as well as parenting dimensions are presented in Table 2.1. As expected, within informant, higher levels of both factors of psychopathy were associated with higher levels of adolescent CU traits; however, these associations were not present across informant. Additionally, lower levels of parental involvement, and higher levels of harsh parenting were associated with adolescent CU traits. These parenting associations were significant both within and across informant (except for father-reported parenting and child-reported CU traits) (see Supplemental Table 2.1).

Are Parental Psychopathic Traits Related to Adolescent CU Traits and Parenting Practices?

Consistent with predictions, parental interpersonal-affective traits, but not impulsive-antisocial traits, were related to higher adolescent CU traits (Table 2.2). This was true for both

mothers and fathers, and one association survived correction for multiple comparisons within informant (mother interpersonal-affective traits). There were no significant associations across informants, however.

Somewhat consistent with predictions, parental interpersonal-affective traits were associated with reduced involvement and increased conflict. However, impulsive-antisocial traits were not associated with parenting when accounting for the overlap among the factors for both mothers and fathers (Table 2.2). Most associations (one exception) survived correction for multiple comparisons but were only present within informant.

Does Parenting Explain the Association Between Parental Psychopathic Traits and Adolescent CU Traits?

Given that no associations *across informant* met requirements to test indirect effects (i.e., significant associations between psychopathic trait and adolescent CU traits, and parenting and adolescent CU traits), we only examined indirect effects within informant. Mother interpersonal-affective traits were associated with adolescent CU traits indirectly via harsh and (less) involved parenting (Table 2.3; Table 2.4). That is, mothers higher in interpersonal-affective traits were higher in conflict and lower in involvement, which in turn predicted higher adolescent CU traits. The direct pathway from mother interpersonal-affective traits to adolescent CU traits was also significant. All paths in the model survived correction for multiple comparisons.

Similarly, father interpersonal-affective traits were associated with adolescent CU traits indirectly via both parenting constructs (Table 2.3; Table 2.4). That is, fathers higher in interpersonal-affective traits were higher in conflict and lower in involvement, which in turn predicted higher adolescent CU traits. The direct pathway from father interpersonal-affective traits to adolescent CU traits was not significant. All significant paths in the model survived

correction for multiple comparisons except for one (path from father interpersonal-affective traits to father involvement).

Do Non-Shared Environmental Influences Explain Associations Between Parenting and Adolescent CU Traits?

Within informant, MZ twin differences in harsh parenting were significantly associated with twin differences in adolescent CU traits for all reporters (Table 2.5). Twin differences in parental involvement were significantly associated with twin differences in adolescent CU traits for mother and child report, but not father report. Across informant, there were no significant associations between differences in parental involvement and twin differences in adolescent CU traits (Table 2.5). Differences in self-reported harsh parenting (for both mothers and fathers) were significantly associated with twin differences in adolescent CU traits as reported by the other parent. There were no significant associations across informant when using child-reported mother conflict. Three of these associations survived correction for multiple comparisons (twin differences in mom-reported harsh parenting with twin differences in mom-reported adolescent CU traits; twin differences in mom-reported harsh parenting with twin differences in dad-reported adolescent CU traits; twin differences in dad-reported harsh parenting with twin differences in dad-reported adolescent CU traits).

Are Associations Between Parenting and Adolescent CU Traits Explained by Non-Shared Environmental Influences, Accounting for the Overlap Between Parenting Dimensions?

In a set of exploratory analyses, we also examined whether there were significant associations between MZ twin differences in parenting practices and twin differences in adolescent CU traits when controlling for the overlap between parenting dimensions (Supplemental Table 2.3). Within informant, when accounting for the overlap of parenting

dimensions (i.e., whether there were unique effects of involvement versus conflict on CU traits), there were significant associations between differences in parental conflict (mother and father report) and differences in MZ twin CU traits (Supplemental Table 2.3). Specifically, based on mother report, when accounting for overlap, differences in mother conflict were positively associated with differences in MZ twin CU traits, whereas the association with mother involvement was not significant. Similarly, there was a significant positive association between differences in father conflict and differences in MZ twin CU traits, whereas the association with involvement was not significant. There were no significant associations between differences in child-reported mother parenting practices and differences in child-reported MZ twin CU traits.

Across informant, there was one significant negative association between differences in mother conflict (mother report) and differences in MZ twin CU traits (father report) (Supplemental Table 2.3). Specifically, based on mother report of parenting, when accounting for their overlap, differences in mother conflict were positively associated with differences in father reported MZ twin CU traits, whereas the association with involvement was not significant. There were no other significant associations between differences in parenting practices and differences in MZ twin CU traits across informant when accounting for the overlap between parenting dimensions.

Does Twin Gender Moderate Associations?

In a set of exploratory analyses, we also examined whether associations differed across boys and girls. First, regarding parental psychopathic traits and adolescent CU traits, two associations significantly differed across boys and girls within informant (paternal interpersonal-affective traits: Satorra-Bentler Scaled $x^2(1) = 8.84$; p=.004; significant correcting for multiple comparisons; paternal impulsive-antisocial: Satorra-Bentler Scaled $x^2(1) = 4.62$; p=.03; not

significant correcting for multiple comparisons;). Father interpersonal-affective traits were only related to higher adolescent CU traits in boys (B= .35; p<.001), but not in girls (B= -.01; p=.96). The association between father impulsive-antisocial traits and adolescent CU traits was not significant in either boys or girls, but they differed in directionality (B= .10, p=.31; B= -.14, p=.18, respectively). Second, regarding parental psychopathic traits and parenting practices, there were no associations that differed across boys and girls.

Third, in our models including psychopathic traits, parenting, and adolescent CU traits, there were only significant differences in pathways by twin gender for fathers: father interpersonal-affective traits to involvement (Satorra-Bentler Scaled $x^2(1) = 4.52$; p=.03), father interpersonal-affective traits to adolescent CU traits (Satorra-Bentler Scaled $x^2(1) = 4.25$; p=.04), and father impulsive-antisocial traits to involvement (Satorra-Bentler Scaled $x^2(1) = 3.97$; p=.05). Specifically, in boys, father interpersonal-affective traits were associated with reduced involvement (B= -.40; p=.001), whereas this was not significant in girls (B= -.06; p=.60). The association between interpersonal-affective traits and adolescent CU traits was not significant in either boys or girls, but they differed in directionality (B= .13; p=.16; B= -.10; p=.33, respectively). This was also the case for the association between impulsive-antisocial traits and parental involvement (boys: B= -.01; p=.83; girls; B= .19; p=.07). None of these associations survived correction for multiple comparisons.

Fourth, within informant, twin gender did not moderate associations between twin differences in parenting practices and twin differences in adolescent CU traits (Supplemental Table 2.4*).* Across informant, gender moderated the association between twin differences in father involvement and twin differences in mother-reported adolescent CU traits. However, although the associations differed in directionality, the association was not significant in either

boys or girls (Supplemental Table 2.4). Additionally, gender moderated the association between twin differences in adolescent-reported mother involvement and twin differences in father-reported adolescent CU traits. Within boys, this association was negative and significant, whereas within girls this association was positive but not significant (Supplemental Table 2.4).

Finally, within informant, twin gender did not moderate associations between twin differences in parenting practices and twin differences in adolescent CU traits, when accounting for the overlap of parenting dimensions. Across informant, there was only one significant interaction, when accounting for the overlap of parenting dimensions. Gender moderated the association between twin differences in child-reported mother involvement and twin differences in father-reported adolescent CU traits. Within boys, this association was negative and significant, whereas within girls this association was positive but not significant (Supplemental Table 2.4).

Discussion

In a community sample of twins recruited from neighborhoods with above average levels of poverty, we found that parental psychopathic traits were associated with adolescent CU traits directly and indirectly via parenting practices. Both mother and father interpersonal-affective traits were associated with higher levels of adolescent CU traits, as well as reduced parental involvement and increased harsh parenting. Additionally, we found that both mother and father interpersonal-affective traits were associated with adolescent CU traits via reduced parental involvement with child and increased harsh parenting. The direct effect from mother interpersonal-affective traits to adolescent CU traits remained significant when accounting for these indirect pathways. Moreover, by examining MZ differences, we confirmed that the association between parenting and CU traits was at least partially environmental in origin and not

simply the result of gene-environment correlation. Taken together, parental interpersonal-affective traits may be transmitted to offspring indirectly via non-shared environmental experiences of parenting. However, many of the findings did not replicate when examining cross-informant models and were only present within single informant models, highlighting a role for shared informant variance as well. Finally, in our exploratory analyses, we found that most associations were similar across child gender, beyond two exceptions (one of which did not survive correction for multiple comparisons); however, these results suggest that further research may be warranted to clarify the impact of child gender on pathways of transmission.

Parental Interpersonal-Affective Traits Are Associated with Adolescent CU Traits

As hypothesized, both mother and father interpersonal-affective features were associated with higher adolescent CU traits when looking within informant. However, parental impulsive-antisocial traits were not associated with adolescent CU traits. The specificity of this association is not surprising, but important to establish, given that adolescent CU traits (e.g., lack of remorse, shallow affect) overlap more directly with the interpersonal-affective features of adult psychopathy, rather than impulsive-antisocial features (Salekin, 2017). Of note, the pattern of findings was similar for both mothers and fathers, highlighting that associations between parental psychopathy and adolescent CU traits did not differ according to parent gender. Moreover, our findings are generally consistent with previous work linking parental interpersonal-affective traits with adolescent CU traits (Diaz et al., 2018; Loney et al., 2007).

Parental Interpersonal-Affective Traits Are Associated with Parenting Practices

As hypothesized, both mother and father interpersonal-affective features were associated with reduced parental involvement with their children and harsher parenting when looking within informant. In contrast, impulsive-antisocial traits were not associated with parenting practices.

This was somewhat surprising given that parent antisocial behavior, which overlaps with the impulsive-antisocial traits of psychopathy, has been associated with harsher parenting (Blazei et al., 2006). However, the interpersonal-affective traits of psychopathy capture interpersonal style and social interactions more so than the impulsive-antisocial traits (Cooke & Michie, 2001), which may explain the specificity of this association, particularly in a community sample with less severe levels of AB. Overall, expanding on previous studies (Beaver et al., 2014; Cox et al., 2018; Loney et al., 2007; Schwartz et al., 2017), our findings suggest that parent psychopathic traits may be critical in shaping parenting practices for mothers and fathers.

Associations Between Parental Interpersonal-Affective Traits and Adolescent Callous-Unemotional Traits Are Partially Explained by Parenting Practices

As hypothesized, there was a significant indirect pathway from parental psychopathic traits and adolescent CU traits via parenting (within informant). Specifically, consistent with previous studies (Diaz et al., 2018; Loney et al., 2007), there were significant indirect effects between both fathers' and mothers' interpersonal-affective traits and adolescent CU traits via reduced involvement and increased harsh parenting. Thus, one mode of transmission of parental psychopathic interpersonal-affective traits to adolescent CU traits may be via parenting, including both harsh (i.e., conflict) and warm (i.e., involvement) dimensions of parenting. Moreover, this indirect pathway was significant for both mothers and fathers, demonstrating further similarities in the mechanisms underlying the transmission of both mother and father psychopathic traits to adolescent CU traits. The consistency of findings across mothers and fathers is notable since we had a fairly large sample of fathers, which is rare in developmental studies (Cabrera et al., 2018).

However, mother (but not father) interpersonal-affective traits were also still significantly directly associated with adolescent CU traits when including indirect pathways via parenting. Our findings therefore suggest additional modes of transmission of parental psychopathy for mothers compared to fathers. The remaining direct effect from mother psychopathic interpersonal-affective traits may reflect genetic transmission that does not overlap with parenting or could capture other environmental processes such as neighborhood effects (Burt, 2009; Raine, 2002). Notably, previous studies have not found significant direct associations between mother psychopathic traits and adolescent CU traits when accounting for parenting (Diaz et al., 2018; Loney et al., 2007), though the current study is much larger, with greater power to identify both direct and indirect effects. Thus, further research is needed to better understand the sources of genetic and environmental transmission from parental psychopathic traits to child CU traits, and how these pathways may differ between mothers and fathers.

Non-shared Environmental Influences Contribute to Differences in Monozygotic Twin Callous-Unemotional Traits

Consistent with our hypotheses and a previous study using the same sample at an earlier developmental stage (childhood; Waller et al., 2018), differences in parenting between MZ twins were associated with differences in CU traits between those twins (at least when looking within informant). Thus, our results emphasize that parenting continues to influence CU traits at least in part via environmental mechanisms into adolescence. However, associations with parental involvement difference scores were less robust, such that the association when using father report was only at trend-level. This finding was somewhat in contrast to Waller et al. (2018), in which parental involvement was significantly associated with CU traits using a combination of both mother and father report. Thus, it could be that the impact of parental involvement is greater

earlier in life, whereas harsh parenting is a strong risk factor for CU traits across childhood and adolescence. Overall, these results provide further evidence that parenting practices are critical environmental influences on the emergence of CU traits, as has been demonstrated in previous genetically informed studies (Hyde et al., 2016; Viding et al., 2009; Waller et al., 2018; Waller et al., 2016). Although both CU traits (Moore et al., 2019) and parenting practices (Klahr & Burt, 2014) are both somewhat heritable, taken together, our results suggest a nonshared environmental pathway from parenting to adolescent CU traits, which is not attributable to passive or evocative rGE. These results therefore highlight both the treatment potential and challenges to preventing CU traits. That parenting is strongly associated with the emergence of CU traits highlights parenting as a malleable target for intervention (a focus of multiple empirically supported treatments for broad AB). At the same time, that parents' own psychopathic traits are associated with CU traits and parenting, suggests that for children with CU traits, some parents may have personality traits that may be challenging for treatment providers (Viding & Pingault, 2016).

Informant Effects

Similar to Diaz et al. (2018), we did not find associations between parental psychopathic traits and adolescent-reported CU traits, nor did we find associations between mother psychopathic traits and adolescent-reported parenting practices. We did find some significant cross-informant associations in our MZ differences analyses, but these were confined to cross-parent report and did not extend to adolescent report. In fact, generally, we found little when using combinations of adolescent report and either parent report across aims. These findings raise the concern that our study and others like ours, may be over-estimating the true association between parental psychopathy, parenting, and CU traits because these associations may be due,

at least in part, to shared informant variance. On the other hand, these informant discrepancies could reflect important contextual variation in children's behavior and/or differences in informants' perspectives of the behaviors (De Los Reyes, 2011). Nevertheless, the overall pattern of findings was generally similar when comparing within-informant and cross-informant reports, arguing against informant effects as the primary explanation for our results. Moreover, previous studies utilizing observational measures of parenting have similarly identified parenting practices as a casual factor in the development of CU traits (e.g., Hyde et al., 2016).

Strengths and Limitations

The current study had several strengths, including the inclusion of multiple informants (mother, father, child), the examination of fathering and mothering, the examination of both parental harshness and involvement in a lower-income sample that is at greater risk for AB given the association between neighborhood poverty and AB (Ingoldsby & Shaw, 2002). Moreover, this is the first time that associations between parental psychopathy and adolescent CU traits have been examined in twins and thus the study benefitted from a genetically informed design. Despite these strengths, there are limitations worth noting. First, we examined associations in a community sample (albeit one with higher risk). Thus, our results may not be generalizable to clinical or adjudicated samples with potentially more severe and clinical levels of CU traits. Second, though it is important to examine father effects (Cabrera et al., 2018), not every family had fathers who participated in the study, which may have impacted the power of analyses involving father reports. Children additionally did not report on their fathers' parenting practices, and thus we were unable to examine associations between father psychopathic traits and parenting with youth reports. Third, parental psychopathy was assessed using a self-report measure. Historically, the validity of self-report measures of psychopathy has been questioned

given that deceitfulness and manipulation are core features of the construct, although more recent research has not been able to find associations between psychopathic traits and response style (Ray et al., 2013). Nevertheless, future research incorporating multiple informants would be beneficial in further evaluating the impact of reporter bias on associations (South et al., 2011). Fourth, we utilized a cross-sectional design, and thus we were unable to determine whether earlier bidirectional associations between adolescent CU traits and parenting practices throughout childhood may have influenced our findings in childhood and early adolescence (Hawes et al., 2011; Trentacosta et al., 2019; Waller et al., 2014; Waller & Hyde, 2017).

Conclusions

The current study found that both mother and father interpersonal-affective features were associated with parenting practices and adolescent CU traits in a community sample of twins. Moreover, the association between parental interpersonal-affective features and adolescent CU traits was at least partially explained by parenting practices for both mothers and fathers. We found that associations identified within informants were not robust across different informants (particularly child report). Additionally, by using a genetically informed design, we demonstrated that the associations between parenting and CU traits were, at least partially, environmental in origin. Our results provide further evidence that 1) CU traits are not entirely attributable to genetic risk and 2) that parenting significantly impacts child outcomes via environmental mechanisms, while also demonstrating that parent personality can influence parenting practices. Thus, considering both parent personality and parenting practices are likely critical to designing effective intervention strategies targeting CU traits (Viding & Pingault, 2016).

Table 2.1: Descriptives and zero-order correlations between parental psychopathy, adolescent callous-unemotional traits, and dimensions of parenting

	Descriptives				Zero-Order Correlations		
	n	M(SD)	Range	α	Adolescent CU Traits (Mom Report)	Adolescent CU Traits (Dad Report)	Adolescent CU Traits (Child Report)
Mother Interpersonal-Affective Traits	504	18.80(5.31)	13-39	.80	.27***	.09^{+}	.07
Mother Impulsive-Antisocial Traits	502	17.25(3.65)	14-29	.67	.12*	.01	.07
Father Interpersonal-Affective Traits	409	22.67(7.02)	14-42	.83	.09	.22***	.03
Father Impulsive-Antisocial Traits	414	19.78(5.43)	13-42	.76	.07	.18**	.05
Mother Involvement (Mom Report)	475	43.26(4.12)	27-48	.80	-.54***	-.25***	-.22***
Mother Conflict (Mom Report)	475	20.19(5.90)	11-43	.88	.51***	.30***	.17***
Mother Involvement (Child Report)	469	40.35(6.15)	15-48	.89	-.31***	-.30***	-.47***
Mother Conflict (Child Report)	468	20.75(6.72)	11-47	.87	.26***	.22***	.30***
Father Involvement (Dad Report)	333	40.64(5.46)	16-48	.88	-.18**	-.52***	-.08
Father Conflict (Dad Report)	335	20.11(5.82)	12-41	.89	.21***	.54***	.08
Adolescent CU Traits (Child Report)	523	17.97(6.82)	1-45	.77	.39***	.36***	
Adolescent CU Traits (Dad Report)	390	17.51(8.35)	0-52	.87	.50***		
Adolescent CU Traits (Mom Report)	543	16.43(8.55)	0-45	.87			

Note. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***. CU = callous-unemotional. M = mean. SD = standard deviation.

Table 2.2: Associations among parental psychopathic traits, parenting, and adolescent callous-unemotional traits

Associations Between Parental Psychopathic Traits and Adolescent CU Traits									
	Adolescent CU Traits (Mom Report)			Adolescent CU Traits (Dad Report)			Adolescent CU Traits (Child Report)		
	B	SE	*p*	B	SE	*p*	B	SE	*p*
Mothers									
Mother Interpersonal-Affective Traits	.31[1]	.06	<.001	.14	.09	.14	.04	.06	.50
Mother Impulsive-Antisocial Traits	-.06	.06	.27	-.06	.09	.46	.02	.06	.77
Fathers									
Father Interpersonal-Affective Traits	.10	.08	.25	.22	.09	.01	-.00	.09	.99
Father Impulsive-Antisocial Traits	-.03	.08	.70	.00	.09	.93	-.01	.09	.91

Associations Between Parental Psychopathic Traits and Dimensions of Parenting				
	Mother Involvement (Mom Report)	Mother Conflict (Mom Report)	Mother Involvement (Child Report)	Mother Conflict (Child Report)

	B	SE	*p*	B	SE	*p*	B	SE	*p*	B	SE	*p*
Mothers												
Mother Interpersonal-Affective Traits	-.23[1]	.07	.001	.22[1]	.08	.003	-.06	.06	.32	.12	.07	.11
Mother Impulsive-Antisocial Traits	-.03	.07	.68	.06	.08	.40	-.03	.07	.72	-.04	.07	.55
Fathers												

	Father Involvement (Dad Report)			Father Conflict (Dad Report)		
	B	SE	*p*	B	SE	*p*
Father Interpersonal-Affective Traits	-.24	.10	.02	.31[1]	.09	.001
Father Impulsive-Antisocial Traits	.07	.11	.52	.01	.10	.90

Note. [1] = survived for multiple comparisons (.05/6 = $p < .008$). CU = callous-unemotional. All models included parent-reported adolescent gender, race, age, and annual family income. Models also included both factors as predictors to account for their overlap.

Table 2.3: Path coefficients and direct effects for models of parental psychopathic traits, parenting, and adolescent callous-unemotional traits

Mothers									
Within Informant Model									
	Mother Involvement (Mom Report)			Mother Conflict (Mom Report)			Adolescent CU Traits (Mom Report)		
	B	SE	*p*	B	SE	*p*	B	SE	*p*
Mother Interpersonal-Affective Traits	-.23[1]	.07	.001	.22[1]	.07	.003	.17[1]	.06	.003
Mother Impulsive-Antisocial Traits	-.02	.06	.74	.06	.07	.46	-.10	.05	.05
Mother Involvement (Mom Report)							-.34[1]	.06	.00
Mother Conflict (Mom Report)							.32[1]	.05	.00
Fathers									
Within Informant Model									
	Father Involvement (Dad Report)			Father Conflict (Dad Report)			Adolescent CU Traits (Dad Report)		
	B	SE	*p*	B	SE	*p*	B	SE	*p*
Father Interpersonal-Affective Traits	-.25	.10	.02	.31[1]	.09	.001	.04	.09	.67
Father Impulsive-Antisocial Traits	.09	.10	.41	-.004	.10	.97	.03	.08	.70

Father Involvement (Dad Report)	-.34[1]	.08	<.001
Father Conflict (Dad Report)	.38[1]	.07	<.001

Note. [1] = survived for multiple comparisons (.05/6 = $p < .008$). CU = callous-unemotional. All models included parent-reported adolescent gender, race, age, and annual family income.

Table 2.4: Indirect effects for models of parental psychopathic traits, parenting, and adolescent callous-unemotional traits

Mothers				
Within Informant: Mother-reported Mother Involvement, Mother Conflict, and Adolescent CU Traits				
	Estimate			Bootstrap 95% CI
	B	SE	*p*	
Total Interpersonal-Affective Traits	.32[1]	.07	<.001	.19, .45
Interpersonal-Affective→ Involvement → CU Traits	.08[1]	.03	.003	.03, .14
Interpersonal-Affective → Conflict → CU Traits	.07	.03	.009	.03, .13
Total Impulsive-Antisocial Traits	-.07	.06	.26	-.19, .05
Impulsive-Antisocial → Involvement → CU Traits	.01	.02	.33	-.03, .06
Impulsive-Antisocial → Conflict → CU Traits	.02	.02	.47	-.03, .07
Fathers				
Within Informant: Father-reported Father Involvement, Father Conflict, and Adolescent CU Traits				
	Estimate			Bootstrap 95% CI
	B	SE	*p*	
Total Interpersonal-Affective Traits	.24	.09	.009	.05, .41
Interpersonal-Affective→ Involvement → CU Traits	.09	.04	.04	.02, .22
Interpersonal-Affective → Conflict → CU Traits	.12[1]	.04	.006	.01, .18

Total Impulsive-Antisocial Traits	.000	.09	1.00	-.17, .19
Impulsive-Antisocial → Involvement → CU Traits	-.03	.04	.42	-.11, .04
Impulsive-Antisocial → Conflict → CU Traits	-.002	.04	.97	-.08, .07

Note. [1] = survived for multiple comparisons (.05/6 = $p < .008$). CU = callous-unemotional. All models included parent-reported adolescent gender, race, age, and annual family income.

Table 2.5: Zero-order correlations between MZ twin difference scores of adolescent callous-unemotional traits and dimensions of parenting

	Adolescent CU Traits (Mom Report)	Adolescent CU Traits (Dad Report)	Adolescent CU Traits (Child Report)
Mother Involvement (Mom Report)	-.25*	-.22^{+}	-.07
Mother Conflict (Mom Report)	.31**[1]	.39**[1]	.15
Mother Involvement (Child Report)	-.15	-.20	-.27*
Mother Conflict (Child Report)	.12	.18	.27*
Father Involvement (Dad Report)	-.13	-.23^{+}	-.19
Father Conflict (Dad Report)	.26*	.52***[1]	.11

Note. MZ = monozygotic. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***. [1] = survived for multiple comparisons (.05/6 = $p < .008$). CU = callous-unemotional. MZ = monozygotic. There were 109 monozygotic twin pairs (218 twins) out of 275 total twin pairs.

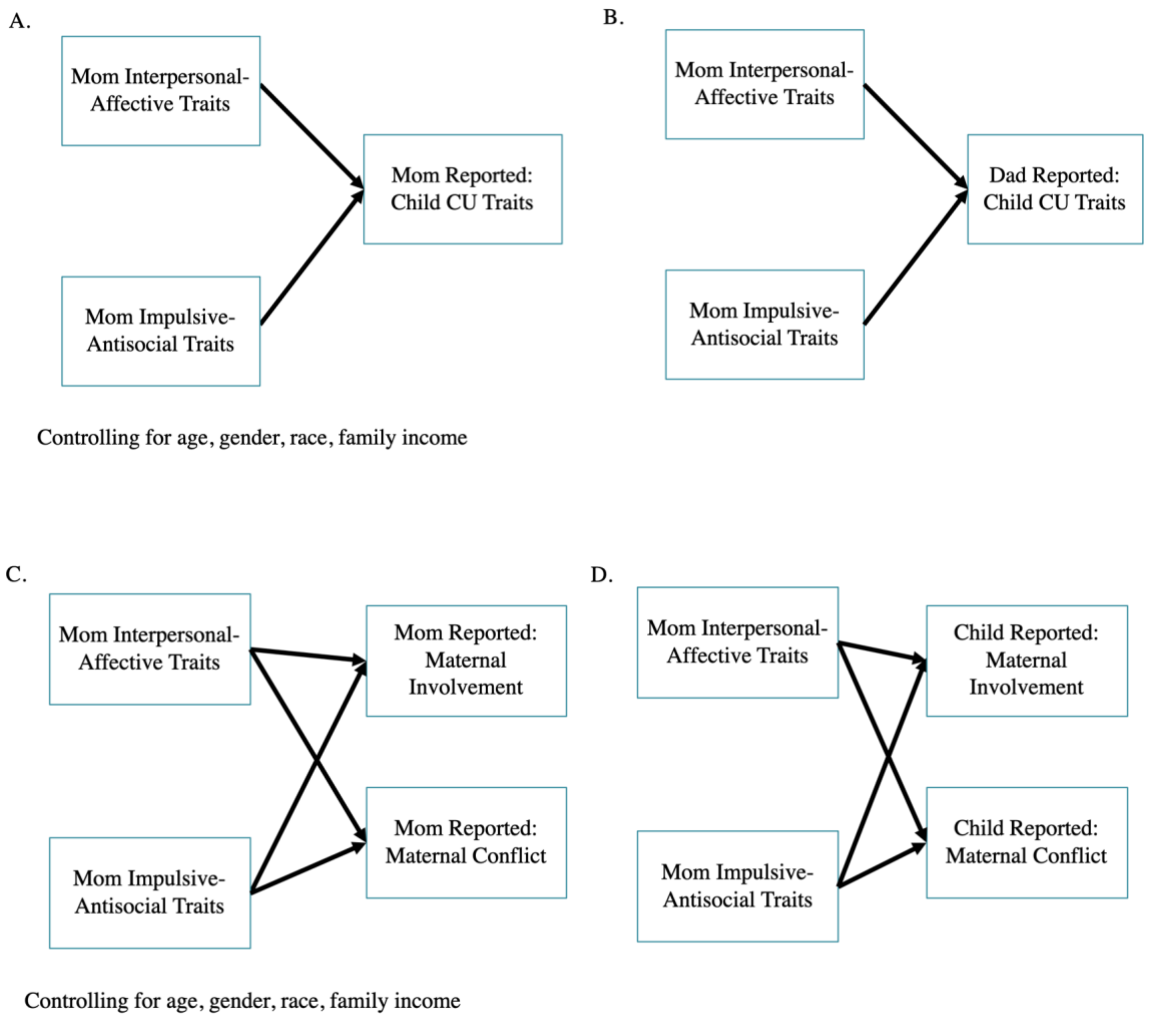

Figure 2.1: Example regression models of associations among parental psychopathic traits, parenting, and adolescent callous-unemotional traits.

All models include child gender, race, age, and annual family income. 1A. Parental interpersonal-affective traits and parental impulsive-antisocial traits are predictors of adolescent callous-unemotional traits. A "within-informant" model, such that the same

informant reports on each construct within the model (i.e., mother reports on both her own psychopathic traits and the child's callous-unemotional traits). 1B. Parental interpersonal-affective traits and parental impulsive-antisocial traits are predictors of adolescent callous-unemotional traits. An "across-informant" model, such that there are unique reporters for different constructs within the model (i.e., mother reports her own psychopathic traits, dad reports on child's callous-unemotional traits). 1C. Parental interpersonal-affective traits and parental impulsive-antisocial traits are predictors of parental involvement (warm/involved parenting) and parental conflict (harsh parenting). A "within-informant" model, such that the same informant reports on each construct within the model (i.e., mother reports on both her own psychopathic traits and her own parenting). 1D. Parental interpersonal-affective traits and parental impulsive-antisocial traits are predictors of parental involvement and parental conflict. An "across-informant" model, such that there are unique reporters for different constructs within the model (i.e., mother reports her own psychopathic traits, child reports on mother parenting).

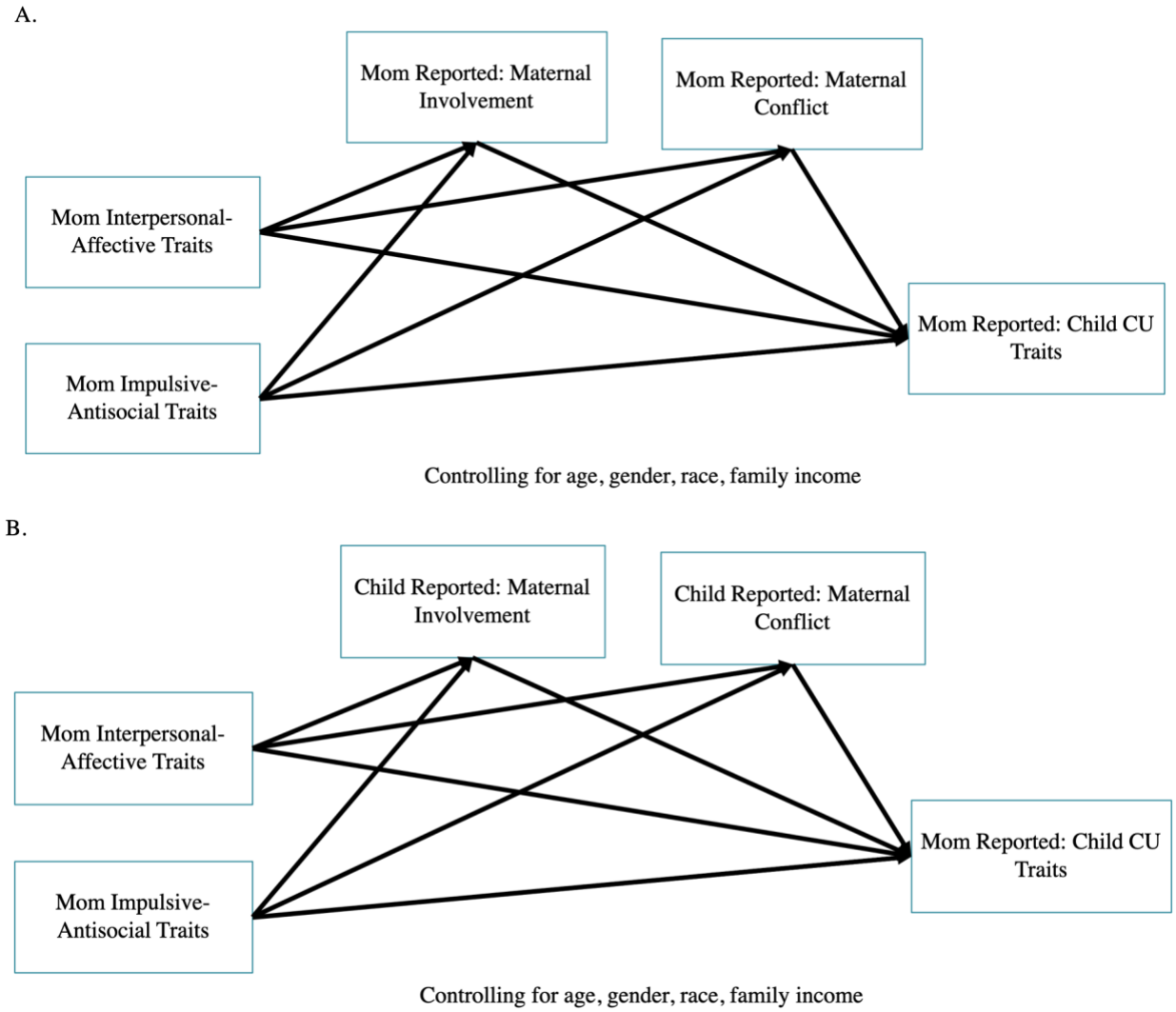

Figure 2.2: Example mediation models of associations among parental psychopathic traits, parenting, and adolescent callous-unemotional traits.

In all models, parental interpersonal-affective traits and parental impulsive-antisocial traits are predictors of child callous-unemotional traits. We also modeled indirect pathways from parental interpersonal-affective traits and parental impulsive-antisocial traits to child callous-unemotional traits via parental involvement (warm/involved parenting) and parental conflict (harsh parenting). All models include child gender, race, age, and annual family income. Figure 2.1A demonstrates a "within-informant" model, such that the same informant reports on each construct within the model (i.e., mother reports on her own psychopathic traits, her own parenting, and child callous-unemotional traits). Figure 2.1B demonstrates an "across-informant" model, such that there are unique reporters for different constructs within the model (i.e., mother reports her own psychopathic traits and adolescent callous-unemotional traits, but child reports on mother parenting).

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Supplemental Table 2.1: Zero-order correlations between parental psychopathy and dimensions of parenting

	Mother Interpersonal-Affective	Mother Impulsive-Antisocial	Father Interpersonal-Affective	Father Impulsive-Antisocial	Mother Involvement (Mom Report)	Mother Conflict (Mom Report)	Mother Involvement (Child Report)	Mother Conflict (Child Report)	Father Involvement (Dad Report)
Mother Impulsive-Antisocial Traits	.61***								
Father Interpersonal-Affective Traits	.15**	-.01							
Father Impulsive-Antisocial Traits	.06	.03	.74***						
Mother Involvement (Mom Report)	-.28***	-.18***	-.03	-.04					
Mother Conflict (Mom Report)	.28***	.21***	.13*	.03	-.51***				
Mother Involvement (Child Report)	-.12*	-.08^{+}	-.06	-.05	.33***	-.33***			

Mother Conflict (Child Report)	.10*	.04	.07	.01	-.25***	.47***	-.64***		
Father Involvement (Dad Report)	-.08	-.02	-.19**	-.11*	.19***	-.17**	.18**	-.15**	
Father Conflict (Dad Report)	.06	.06	.32***	.24***	-.14*	.28***	-.19**	.21***	-.36***

Note. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***.

Supplemental Table 2.2: Descriptive statistics and intra-class correlations for MZ twins only

	n	Twin 1	Twin 2	Total	
		M (SD)	*M (SD)*	*r*	95% CI
Adolescent CU Traits (Mom Report)	108	16.20 (8.52)	15.02 (8.06)	.47***	.30-.60
Adolescent CU Traits (Dad Report)	77	16.95 (8.12)	16.45 (7.81)	.70***	.56.79
Adolescent CU Traits (Child Report)	102	18.51 (7.08)	17.85 (6.83)	.46***	.29-.60
Mother Involvement (Mom Report)	93	43.75 (3.99)	43.56 (3.93)	.72***	.60-.80
Mother Conflict (Mom Report)	93	19.91 (6.31)	19.44 (5.98)	.73***	.62-.81
Mother Involvement (Child Report)	91	40.15 (6.59)	39.45 (6.98)	.67***	.54-.77
Mother Conflict (Child Report)	90	21.79 (7.76)	21.03 (7.26)	.69***	.56-.78
Father Involvement (Dad Report)	66	40.94 (4.94)	41.17 (5.21)	.86***	.77-.91
Father Conflict (Dad Report)	67	20.01 (5.38)	18.90 (5.45)	.68***	.52-.79

Note. *p<.05, **p<.01, ***p<.001. CU = callous-unemotional. MZ = monozygotic. Computed to establish associations within monozygotic (mz) twin pairs (n= 109 families out of 275 total sample), including 95% confidence intervals. Smaller *ns* represent missing data.

Supplemental Table 2.3: Associations between MZ difference scores of adolescent callous-unemotional traits and dimensions of parenting

	Difference in Adolescent CU Traits (Mom Report)				Difference in Adolescent CU Traits (Dad Report)				Difference in Adolescent CU Traits (Child Report)			
	B	SE	β	*p*	B	SE	β	*p*	B	SE	β	*p*
Mothers												
Difference in Mother Involvement (Mom Report)	-.41	.33	-.14	.22	-.17	.36	-.06	.64	-.01	.30	-.01	.96
Difference in Mother Conflict (Mom Report)	.49	.22	.25	.03	.53	.19	.36	.007	.23	.19	.15	.23
Difference in Mother Involvement (Child Report)	-.25	.23	-.15	.27	-.18	.21	-.14	.40	-.23	.18	-.16	.22
Difference in Mother Conflict (Child Report)	.03	.21	.02	.89	.11	.19	.09	.57	.23	.17	.18	.16
Fathers												
Difference in Father Involvement (Dad Report)	.03	.44	.01	.95	.10	.30	.04	.75	-.37	.32	-.17	.25
Difference in Father Conflict (Dad Report)	.53	.28	.27	.06^{+}	.81	.19	.53	<.001	.06	.21	.04	.78

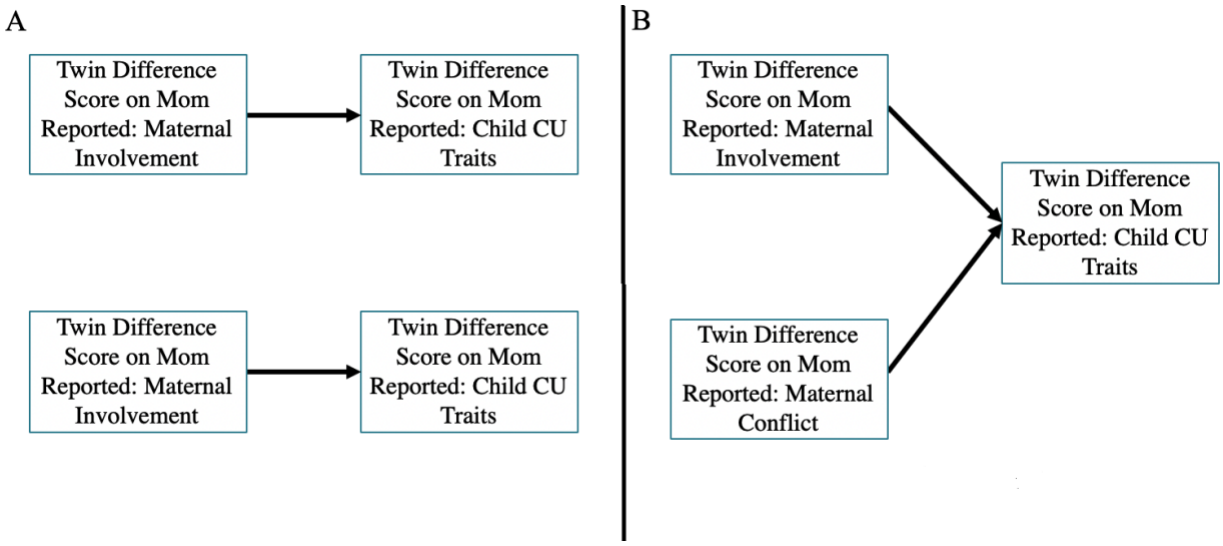
Note. CU = callous-unemotional. Separate regression models were run to examine associations among parenting difference scores for each informant (including both conflict and involvement) predicting child CU traits difference scores for each informant (9 models total). Models included both dimensions of parenting to control for their overlap. There were 109 monozygotic twin pairs (218 twins) out of 275 total twin pairs.

Supplemental Table 2.4: Zero-order correlations between MZ twin difference scores of adolescent callous-unemotional traits and parenting split by gender

Boys			
	Adolescent CU Traits (Mom Report)	Adolescent CU Traits (Dad Report)	Adolescent CU Traits (Child Report)
Mother Involvement (Mom Report)	-.33*	-.22	-.06
Mother Conflict (Mom Report)	.43**	.43*	.15
Mother Involvement (Child Report)	-.27^{+}	-.52**	-.24
Mother Conflict (Child Report)	.11	.28	.31*
Father Involvement (Dad Report)	.23	-.11	-.03
Father Conflict (Dad Report)	.08	.60***	.05
Girls			
	Adolescent CU Traits	Adolescent CU Traits	Adolescent CU Traits

	(Mom Report)	(Dad Report)	(Child Report)
Mother Involvement (Mom Report)	-.24	-.30^{+}	-.11
Mother Conflict (Mom Report)	.24	.38*	.17
Mother Involvement (Child Report)	-.09	.02	-.35*
Mother Conflict (Child Report)	.13	.07	.20
Father Involvement (Dad Report)	-.32^{+}	-.29^{+}	-.30^{+}
Father Conflict (Dad Report)	.41*	.45**	.16

Note. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***. MZ = monozygotic. CU = callous-unemotional. There were 109 monozygotic twin pairs (44 female; 65 male) out of 275 total twin pairs.



Supplemental Figure 2.1: Example models of associations between monozygotic difference scores in parenting and adolescent callous-unemotional traits.
Figure 2.1A demonstrates zero-order correlations between monozygotic differences in one dimension of parenting (i.e., involvement or conflict) and adolescent callous-unemotional traits. Figure 2.1B demonstrates partial correlations between monozygotic differences in both dimensions of parenting and adolescent callous-unemotional traits, controlling for twin gender.

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Chapter 3: Neurocognitive Abilities Associated with Antisocial Behavior With And Without Callous-Unemotional Traits In A Community Sample

Introduction

Antisocial behavior (AB), including rule-breaking and aggression, is highly prevalent and extremely harmful to perpetrators, victims, and their families (Eddy et al., 2002). Moreover, youth AB is associated with significantly increased levels of criminal justice, health, and social welfare service use in adulthood, leading to large societal costs (Rivenbark et al., 2018). Importantly, research suggests that the group of youth engaging in these behaviors is heterogenous, with many transiently engaging in AB, but others involved in a more chronic course of AB (Moffitt, 2018). One way to identify adolescents at risk for more chronic and severe AB is the presence of callous-unemotional (CU) traits (Frick et al., 2014). CU traits are defined by low empathy, lack of remorse, and shallow interpersonal affect (Frick & White, 2008; Waller et al., 2019). These traits have been conceptualized as a "downward extension" of the callousness component of adult psychopathy (Salekin, 2017), and are a developmental risk factor for psychopathy (Lynam et al., 2007; Waller & Hyde, 2017). CU traits were recently added to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) as a subtyping specifier ("with limited prosocial behavior") to the diagnosis of Conduct Disorder. Importantly, research suggests that AB with CU traits may be characterized by unique cognitive deficits compared to AB without CU traits, which could imply distinct targets for intervention (Baskin-Sommers, Curtin, et al., 2015; Frick et al., 2014).

Cognitive Impairments Within Antisocial Behavior

Research suggests that AB is marked by significant impairments in cognitive abilities that are thought to underlie the impulsive and aggressive nature of AB. Meta-analyses have found that AB is associated with lower intelligence (Ogilvie et al., 2011; Sánchez de Ribera et al., 2019). Similarly, meta-analyses have broadly linked AB to poor executive function, a conceptual category which includes several highly overlapping, but putatively distinct components including response inhibition (i.e., the ability to suppress a prepotent response) and selective attention (i.e., the ability to attend to necessary information in the environment, while ignoring less relevant information) (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). More recently, AB has also been linked to deficits in sustained attention (i.e., the ability to maintain focus over time), with preliminary research studies suggesting that AB is associated with higher reaction time variability (i.e., average standard deviation of reaction time across trials task) within inhibitory tasks (e.g., Rydell & Brocki, 2019; Scholtens et al., 2012). Deficits in executive functioning (including response inhibition and attention) are thought to underlie the impulsivity, emotional dysregulation, and inattention to punishment cues that are characteristic of AB (Patrick et al., 2012).

Interactive Effects of AB and CU Traits

AB without CU traits is typically associated with lower IQ and poorer EF (Cruz et al., 2020), which may lead to observed deficits in emotion regulation and reactive aggression. In contrast, AB with CU traits has been associated with diminished emotional responding to others and increased proactive aggression (Frick et al., 2014). This type of proactive behavior involves impulse control, strategic planning, and goal-directed behavior (Frick et al., 2014; Frick & White, 2008). However, most studies have not found significant direct associations between CU traits and executive functioning when accounting for AB and intelligence, including those in

forensic (Pardini, 2011), clinic-referred (Jezior et al., 2016; Loney et al., 1998), and community samples (Allen et al., 2013; Fanti et al., 2016; Fontaine et al., 2008; Loney et al., 2006). Similarly, CU traits have not been consistently associated with executive functioning when accounting for AB in clinic-referred (Graziano et al., 2019) and community samples (Bohlin et al., 2012; Fanti & Kimonis, 2017; Fanti et al., 2016; Hadjicharalambous & Fanti, 2018; Platje et al., 2018; Rydell & Brocki, 2019; Wall et al., 2016; Waller, Hyde, et al., 2015). These null findings suggest that CU traits specifically (i.e., without AB) may not be characterized by cognitive deficits, but that CU traits may only be associated with cognitive functioning due to their overlap with AB, or only in their interaction with AB (i.e., only at high levels of AB, does CU traits predict these outcomes). Importantly, previous research indicates that AB *with* CU traits may identify youth with a distinct cognitive profile from AB *without* CU traits (Frick et al., 2014). Several studies have found AB+CU traits to be differentially associated with components of executive functioning, compared to AB alone (Allen et al., 2013; Baskin-Sommers, Waller, et al., 2015; Fanti et al., 2016; Graziano et al., 2019; Hadjicharalambous & Fanti, 2018; Platje et al., 2018; Wall et al., 2016). However, the directionality of findings has been mixed in clinic-referred (Graziano et al., 2019), forensic (Baskin-Sommers, Waller, et al., 2015), and community samples (Allen et al., 2013; Fanti et al., 2016; Hadjicharalambous & Fanti, 2018; Platje et al., 2018; Wall et al., 2016).

The inconsistent pattern of findings is likely driven by several remaining gaps in the literature. First, the existing research on CU traits has been limited by the use of executive functioning composite scores, which combine indices from several different tasks of executive functioning, as well as questionnaire scales (Bohlin et al., 2012; Fanti & Kimonis, 2017; Hadjicharalambous & Fanti, 2018). CU traits may be more strongly associated with performance

on specific executive functioning tasks, or even specific metrics within a task (i.e., response inhibition versus reaction time variability within a Go/No-Go task), which would not be evident using a composite score (Fanti et al., 2016; Rydell & Brocki, 2019). Second, there is accumulating evidence that AB and CU traits are dimensional in nature (Blonigen et al., 2006; Krueger et al., 2007). However, many of the previous studies have utilized case-control designs, comparing relatively small groups that only model the extreme ends of the distribution (i.e., very low versus high). Moreover, a meta-analysis found that the effect sizes of associations between AB and executive functioning were largest in forensic samples and when using non-antisocial comparison groups (Ogilvie et al., 2011). Thus, further research is needed to clarify whether associations exist across the naturally occurring dimensions of these behaviors/traits in community samples, particularly among youth in the community who are exposed to more risk for AB (e.g., those living in disadvantaged neighborhoods). Finally, previous studies have not consistently controlled for symptoms of other forms of psychopathology that may also explain cognitive deficits, including attention deficit/hyperactivity disorder (ADHD), anxiety, and depression (e.g., Iorfino et al., 2016; Seidman, 2006; Vilgis et al., 2015). This omission is surprising given the high comorbidity between AB and these other forms of psychopathology which are also linked to deficits in executive functioning. Thus, it is unclear whether cognitive deficits are uniquely associated with AB or CU traits versus general psychopathology, particularly in community samples.

Current Study

The goal of the present study was to investigate relations between neurocognitive abilities (including IQ and various executive function metrics) and AB and CU traits dimensionally in a community sample of youth, sampled from birth records with enrichment for families living in

low-income neighborhoods. We first sought to replicate previously identified associations between AB and cognitive functioning. Consistent with the existing literature, we expected that AB would be related to lower IQ and poorer performance on executive function tasks when accounting for CU traits. In contrast, we did not expect to find associations between CU traits and cognitive functioning when accounting for AB, in line with the majority of previous studies. Second, our primary aim was to examine whether there were any significant interactions between AB and CU traits in predicting cognitive abilities. Given the mixed state of the literature, we did not have any specific hypotheses related to this aim but expected the association between AB and cognitive functioning to vary at different levels of CU traits.

Methods

Participants

Participants in this study were from 550 twins from 275 families living in south-central Michigan that are part of the ongoing Michigan Twin Neurogenetics Study (MTwiNS). Twins were originally recruited at age 6 – 10 for the Twin Study of Behavioral and Emotional Development in Children (TBED-C) within the Michigan State University Twin Registry (see Burt & Klump, 2019). Twins were initially recruited into one of two cohorts. The population-based cohort was sampled from birth records to represent all families with twins living within 120 miles of Michigan State University. The second, at-risk cohort was recruited from the same area, but only included families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (i.e., the mean for the state of Michigan at the onset of recruitment) (see Burt & Klump, 2019). The MTwiNS follow-up study was recruited from the latter subsample, as well as those in the first sample that would have qualified for the second sample

(i.e., those families who lived in neighborhoods with above mean levels of poverty). Thus, the sample represents families with twins living in neighborhoods in Michigan with above average levels of family poverty. Because sampling was done at the neighborhood level, family income varied widely, but with enrichment for substantial rates of low-income: The average reported combined annual family income within MTwiNS was between $60,000 and $69,999, ranging from less than $4,999 to greater than $90,000. 59% of the sample reported annual income below the living wage for a family of 4 in Michigan (http://livingwage.mit.edu/states/26). Due to missing data (i.e., missing data on either the AB, CU traits, or other psychopathology measures), the current study included 474 twins (55.9% male) (see Table 3.1). Participants ranged in age from 7 to 18 years (Mean age = 14.18 years; SD 2.20; 95.2% of the sample is between 10-17 years old, with only 7 twin pairs younger than 10 years old and 5 twin pairs older than 17 years old). Parent-reported race of the 474 children included was as follows: 78.2% White, 12.4% Black, .9% Asian, 1.1% Pacific Islander, .6% Native American, 1.3% Hispanic, and 5.6% Other. This distribution of twin race contains somewhat fewer White participants (and thus more that identify as Black, Biracial, Native American, Hispanic, or Other) than the average for the State of Michigan (e.g., 76% identified as White, versus 80% in Michigan; https://www.census.gov/quickfacts/fact/table/detroitcitymichigan,MI/PST045219). Parents indicated whether their children were had ever been diagnosed with a psychiatric condition (21.1% had current or previous psychiatric diagnosis) and whether their children were currently taking any medication (20.3% taking medication). Based on the Youth Self Report (Achenbach, 1991), 15.2% of the sample (n= 72) were above the borderline clinical threshold for the internalizing problems scale (T Score > 60) and 3.4% of the sample (n= 16) were above the borderline clinical threshold for the externalizing problems scale. The included 474 participants

did not significantly differ from the original 550 participants in mother's education ($t(548)=.259$, $p=.10$) or gender ($x^2(1)=.29$, $p=.59$), but did significantly differ in family annual income ($t(538)=1.17$, $p<.001$), age ($t(548)=1.35$, $p<.001$), and race ($x^2(1)=10.91$, $p<.001$). Included participants were older, had higher family annual income, and were more likely to be White. Thus, these variables were included as covariates in analyses.

Procedure

Youth and their primary caregivers (95% biological mothers) participated in a day-long protocol that included questionnaires, parent-child interaction tasks, collection of biological specimens, and an MRI scan. Twins were randomized within pairs to determine which twin participated in the protocol activities first. For minor twins, parents provided informed consent for themselves and their children to participate in the study, while the twins provided informed assent. When the twins were 18 or older, they provided informed consent to participate. The study protocol was approved by the Institutional Review Board at the University of Michigan.

Measures

AB. Given that more severe forms of AB (i.e., criminality) evidence particularly strong associations with cognitive deficits (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011), AB was assessed using the child-reported Self-Report of Delinquency Questionnaire (SRD; Elliott et al., 1985; e.g., 'Have you been physically cruel to someone else (causing harm)?', 'Have you taken something from a store without paying for it?'). The SRD assesses the frequency of more serious aggressive and delinquent behavior and related offenses during the prior year. All items, excluding 12 substance use items, were summed to form a dimensional measure of AB (Table 3.2).

CU traits. CU traits were assessed using the child-reported Inventory of Callous-Unemotional Traits, which assesses callousness (e.g., "unconcerned about feelings of others"), uncaring (e.g., "always tries best"), and unemotionality (e.g., "hides feelings") (ICU; Essau et al., 2006; Kimonis et al., 2008). 11.6% of the sample were above a clinical cut-off score (n= 55; Total score > 28) for CU traits based on a study of the ICU in a community sample (Docherty et al., 2017). In analyses, consist with prior studies (Waller, Wright, et al., 2015), we utilized a total 22-item sum score, excluding items 10 and 23 (Table 3.2).

Covariates. Analyses included parent-reported child gender (0= Male, 1=Female), age, annual family income, and child-reported substance use. Substance use was measured using a sum score of the 12 substance use items from the SRD. Family annual income was included as a covariate to assess socioeconomic status. We additionally included parent-reported adolescent race, a socially constructed category, as a covariate to control for differences in exposure to systemic racism and the various unequal exposures to stress, trauma, and opportunity for people of color in the United States (0=Non-White; 1=White as White is the largest group) (Jones, 2001). Psychopathology covariates (all child-reported) included ADHD symptoms (attention problems scale of Youth Self Report, YSR; Achenbach, 1991), depressive symptoms (total sum score of Child Depression Inventory, CDI; Kovacs, 1992), and anxiety symptoms (total sum score of Multidimensional Anxiety Scale for Children, MASC; March et al., 1997). Additional covariates included parent-reported current or previous psychiatric diagnosis (0 = no; 1 = yes) and parent-reported medication usage (0= no medication; 1 = currently taking medications) (Table 3.1).

Behavioral Tasks

Intelligence. The Shipley-2 (Shipley et al., 2009) is a revised and re-standardized version of the Shipley Institute of Living Scale (Shipley, 1940), a brief but robust measure of cognitive functioning and impairment. The Shipley-2 assesses two distinct aspects of cognitive ability: crystallized ability (also conceptualized as verbal intelligence), which is accumulated information acquired through various life experiences, and fluid cognitive ability (or non-verbal intelligence), which is the capacity to solve novel problems via deliberate and flexible control of attention (Schneider & Newman, 2015). The Shipley-2 was standardized based on a large, nationally representative sample (2,826 individuals divided into two groups: adults and children) and can be used with persons aged 7 through 89. The test is self-administered and monitored by research assistants who had been trained in psychological testing. Although the test is not a timed test, each scale has different time limits (Vocabulary scale = 10 minutes; Block Patterns scale = 12 minutes). Participants completed the Vocabulary scale (40 items; choose word meanings; measuring crystallized skills/verbal reasoning) and then the Block Patterns scale (12 items; match block patterns; measuring fluid reasoning skills/non-verbal reasoning). Raw scores for each scale are converted to generate standard scores (M = 100, SD = 15) for the Vocabulary and Block Patterns scales, as well as a composite score, which reflects overall cognitive ability.

Response inhibition, selective attention, and sustained attention.

Go/No-Go task. To assess executive functioning, participants completed a child-friendly Go/No-Go (GNG) task ("whack-a-mole" game; stimuli courtesy of Sarah Getz and the Sackler Institute for Developmental Psychobiology; Casey et al., 1997) during an fMRI scan as described in Tomlinson et al. (2020). Briefly, adolescents were instructed to press a button as quickly as possible in response to one stimulus ("Go", a mole) and avoid responding to a less frequent non-target ("No-Go", a vegetable). The task consisted of four runs, each with approximately 55 trials,

for a total of 255 trials (55 No-Go). For each participant, an inhibitory efficiency score was calculated by dividing accuracy (percent of "No-Go" trials participant correctly avoided responding) by reaction time (average reaction time for correct "Go" trials) to measure response inhibition (Tomlinson et al., 2020). This measure accounts for the potential of two individuals to obtain the same accuracy score while one individual must trade reaction time for accuracy (Hirose et al., 2012; Votruba & Langenecker, 2013). Additionally, the percentage of "Go" trials to which a participant correctly responded ('Go' Accuracy) was included as this metric is sometimes used as an index of selective attention. Finally, reaction time variability was measured as the standard deviation of reaction time to hits on the "Go" stimuli, similar to previous work which framed this measure as an index of sustained attention (Rydell & Brocki, 2019).

Stop-signal task. As another index of response inhibition, we used a 10 minute, 150-trial, child-friendly Stop-Signal Task (Klein et al., 2006). Participants were told to push the "a" key with their left hand to help the left-pointed blue fish swim home, or the "l" key with their right hand to help the right-pointed orange fish swim home. Participants were told to avoid pressing a key when a visual stop signal stimulus (Martin the Manta Ray) appeared on the screen, which occurred on 50 of the task trials (33%; "stop" trials). This stop signal was presented following a stop signal delay (SSD) that was determined through a standard "staircase tracking" algorithm (Logan, 1994) designed to lead to a roughly 50% probability of inhibition on "stop" trials for each participant. This algorithm began with a 250ms SSD on the first "stop" trial, and the SSD was thereafter increased or decreased by 50ms on each subsequent "stop" trial depending on whether the participant inhibited their response or failed to inhibit, respectively. Following a recent consensus paper on best practices for measuring response inhibition with the stop signal

task (Verbruggen et al., 2019), we used the recommended non-parametric method for estimating stop signal reaction time (SSRT): integrating the "go" response time distribution while replacing omissions with the maximum "go" response time, finding the point where the integral equals the probability of responding on "stop" trials, and subtracting the mean SSD from the response time at this point. These SSRT estimates were then used as a measure of the efficiency of individuals' inhibition processes.

Data Analysis Plan

All analyses were conducted in MPlus version 8.3 (Muthén & Muthén, 2020) using maximum likelihood estimation with robust standard errors (MLR), which is robust to relaxed assumptions of the data (e.g., non-normality) (Yuan & Bentler, 2000). To account for the nesting of twin pairs in the model, all analyses were carried out using the COMPLEX command in Mplus with family as the nesting variable. We included the main effects of AB and CU traits, and a two-way interaction term (i.e., AB x CU traits) into the regression models as independent variables and an index of cognitive functioning as correlated dependent variables (with indices from the same task included as correlated dependent variables; i.e., Shipley Vocabulary, Shipley Matrix Reasoning, and Shipley Composite Scores; see Figure 3.1 for example model). Covariates included for parent-reported twin gender, race, age, annual income, child-reported substance use, parent-reported psychiatric history, parent-reported medication usage, and symptom counts of other forms of psychopathology that have been linked to deficits in cognitive functioning as covariates: ADHD, depression, and anxiety (e.g., Iorfino et al., 2016; Seidman, 2006; Vilgis et al., 2015). Cognitive indices from the same task were included in a single model to control for multiple comparisons; thus, indices from the same task were correlated dependent variables. We also determined 95% confidence intervals for significant interaction estimates

using bootstrapping (1000 draws). To explore any significant two-way interactions, we used an online tool to examine simple slopes at the mean and 1 SD below (low) and 1 SD above (high) the mean (Aiken et al., 1991), as well as regions of significance (Preacher et al., 2006); we examined results for models with CU traits as the moderator. Notably, we also examined a separate model that only included AB and total CU traits as independent variables (without the interaction term) to determine whether there were significant main effects when not including the interaction term. For all results, we highlight associations that meet a strict conservative threshold to account for three models (i.e., one for each task: GNG task, SST, and Shipley) that were tested (i.e., Bonferroni-correction $0.05/3 = p < .017$).

Results

First, in zero-order correlations, AB was significantly correlated with lower IQ, but surprisingly not with any index of executive functioning (Table 3.3). CU traits were correlated with lower GNG efficiency and higher GNG reaction time variability. All indices of cognitive functioning (except for SSRT) were positively associated with income (Table 3.4). Total IQ was negatively associated with age. GNG efficiency was positively correlated with age, whereas GNG reaction time variability was negatively correlated with age. GNG efficiency and GNG reaction time variability were both was also significantly associated with gender. Specifically, GNG efficiency performance was higher in girls and lower in boys, whereas GNG reaction time variability was lower in girls and higher in boys. Somewhat surprisingly, indices of cognitive functioning were generally not associated with other forms of psychopathology. However, both GNG efficiency and go accuracy were positively correlated with anxiety symptoms, whereas GNG reaction time variability was negatively associated with anxiety symptoms. No indices of

cognitive functioning were associated with medication usage. There was a negative association between IQ (Blocks score and composite) and history of psychiatric diagnosis (Table 3.4).

Second, in multivariate regression models that controlled for demographics and psychopathology, there were no significant associations between AB or CU traits and any measures of cognitive functioning (Table 3.5).

Third, to address out primary aim, we examined whether CU traits interacted with AB to predict indices of cognitive functioning. There were significant interactions between AB and CU traits in relation to GNG reaction time variability (B = -.15; 95% CI: -.27, -.03), but not crystallized intelligence (B = .15; 95% CI: .01, .30), fluid intelligence (B = .05; 95% CI: -.12, .23), composite intelligence (B = .11; 95% CI: -.04, .27), GNG go accuracy (B = .04; 95% CI: -.03, .11), GNG efficiency (B = .10; 95% CI: -.04, .24), or SSRT (B = .06; 95% CI: -.04, .16) (Table 3.6). AB was associated with higher reaction time variability (i.e., worse sustained attention) only at low levels of CU traits, but lower reaction time variability (i.e., better sustained attention) at high levels of CU traits (Table 3.7; Figure 3.2). The overall interaction model explained 23% of the variance among AB, CU traits, covariates, and GNG reaction time variability (R^2 = .23; Cohen's f^2 = .46), although the effect size of the interaction term specifically was small (i.e., the R^2 increase from the main effects model to the interaction model; R^2 of interaction model= .23 - R^2 of main effects model= .22; Cohen's f^2 = .01). However, this is not surprising given that an interaction term must explain unique variance above and beyond both main effects of the independent variables, as well as numerous covariates (Aguinis et al., 2005; Aiken et al., 1991; McClelland & Judd, 1993).

Discussion

In the current study we sought to examine how cognitive functioning was associated with dimensional measures of AB and CU traits in a community sample. First, although AB was associated with lower intelligence in zero-order correlations, AB was not associated with any index of cognitive functioning when accounting for demographic factors and comorbid psychopathology. Second, CU traits were generally not directly associated with cognitive functioning. However, we found significant interaction effects such that AB was associated with *higher* reaction time variability (traditionally thought to indicate worse sustained attention) at low levels of CU traits but was associated with *lower* reaction time variability (i.e., better sustained attention) at high levels of CU traits. Taken together, our findings suggest that AB at low levels of CU traits is characterized by deficits in executive function as measured by higher reaction time variability, which is thought to reflect *worse* sustained attention. In contrast, AB at high levels of CU traits was associated with *lower* reaction time variability (suggesting *better* sustained attention) compared to AB without CU traits. Executive function deficits associated with broader AB may therefore contribute to impulsive and reactive aggression (Cruz et al., 2020). The distinct neurocognitive profile of lower reaction time variability associated with AB + CU traits may instead underlie strategic planning, forethought, and goal-directed behavior involved in instrumental and premeditated aggression (Frick et al., 2018).

Our findings of divergent neurocognitive correlates for AB high versus low on CU traits are consistent with previous literature that has suggested youth with both elevated AB and CU traits are etiologically distinct from youth with AB alone (Frick et al., 2014). Though no previous studies have looked at AB x CU traits specifically in association with reaction time variability, a previous study similarly found that clinic-referred youth with elevated AB and CU traits had higher scores on an executive functioning composite (which combined inhibitory control,

working memory, and cognitive flexibility) compared to clinic-referred youth with elevated AB alone (Graziano et al., 2019). Unique neurocognitive profiles underlying AB with CU traits versus AB without CU traits implicate distinct targets of treatment. For instance, youth with AB with low levels of CU traits, who demonstrate greater reaction time variability, may specifically benefit from treatments that emphasize strategies to increase cognitive control. In contrast, youth with AB with high levels of CU traits, who demonstrate better sustained attention, may require different interventions. Previous literature suggests that youth with AB and CU traits are characterized by insensitivity to punishment, abnormal moral reasoning, and reduced responsiveness to others' distress (Frick et al., 2014). As such, youth with AB and CU traits may instead benefit from treatments that emphasize attention to context, such as emotional cues. For instance, Baskin-Sommers and colleagues (2015) found that adult offenders who received the intervention targeted to their cognitive deficiencies (affective cognitive control training for individuals high in AB alone; attention to context training for individuals with psychopathic traits) exhibited improvements on task performance over the six-week training period (Baskin-Sommers, Curtin, et al., 2015). In contrast, offenders who received interventions that were not matched to their deficiencies did not demonstrate improvements in performance (Baskin-Sommers, Curtin, et al., 2015). Additionally, preliminary research suggests that specifically targeting emotion recognition and empathy in youth with AB+CU traits may reduce AB and levels of CU traits (Dadds et al., 2012; Datyner et al., 2016; Fleming et al., 2017; Kimonis et al., 2019). Thus, further research on distinct neurocognitive profiles of AB + CU traits can continue to improve the effectiveness of interventions for these youth.

Notably, we did not find the interaction of AB x CU traits to be related to metrics thought to index response inhibition from the GNG task or SST. Importantly, no previous study has

specifically examined the association between AB x CU traits and basic response inhibition as measured by either of these tasks. However, one previous study similarly did not identify any significant interactive effects between AB and CU traits in predicting different putative components executive functioning (interference control and accuracy on Stroop; Fanti et al., 2016). In contrast, other studies have found that high levels of both AB and CU traits are associated with deficits in aspects of executive functioning (Hadjicharalambous & Fanti, 2018; Platje et al., 2018; Wall et al., 2016). For example, one study found that youth with high levels of CU traits demonstrated higher "self-regulation" composite scores (which included performance on higher-order executive functioning tasks including Stroop and Tower of London) compared to youth with high levels of CU traits and conduct problems (Hadjicharalambous & Fanti, 2018). Previous studies also found that high levels of both AB and CU traits were associated with higher parent-reported inhibition deficits compared to youth with elevated CU traits alone (Wall et al., 2016) and parent-reported behavioral regulation problems (a composite of inhibitory control, flexibility, and emotional regulation) (Platje et al., 2018). Thus, there were notable differences in study design compared to the current study, which likely impacted results. Moreover, in the current study, response inhibition on the GNG task was measured using an efficiency score to capture more variability in task performance (Hirose et al., 2012; Tomlinson et al., 2020), as opposed to more traditional indices of inhibition that have been used in the literature (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Further, response inhibition assessed using the Stop Signal Task has been less consistently associated with AB specifically (Lipszyc & Schachar, 2010; Littman & Takács, 2017; Raud et al., 2020). Thus, our findings could suggest that AB and CU traits may be differentially associated with overlapping but somewhat distinct components within executive functioning (i.e., basic response inhibition versus higher-order

planning and mental flexibility), a point suggested in previous meta-analyses (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011) and theoretical work (Cruz et al., 2020). Finally, several previous studies examining AB and CU traits in relation to executive functioning have utilized case-control designs of those extreme on AB and CU traits, as opposed to dimensional analyses (Hadjicharalambous & Fanti, 2018; Platje et al., 2018; Wall et al., 2016). Thus, previously identified associations among AB, CU traits, and response inhibition may not be generalizable to community samples with varying levels of AB and CU traits and may only be present at extremes.

Additionally, we did not find the interaction of AB x CU traits to be related to intelligence. Our finding is similar to a previous study in a clinic-referred sample that did not find differences in intelligence between youth with elevated conduct problems and CU traits compared to youth with elevated conduct problems alone (Graziano et al., 2019). Notably, in the current study, AB was significantly associated with lower IQ (Vocabulary, Shapes, and Composite Scores) in zero-order correlations, whereas this association was not significant in models that also included CU traits, demographic covariates, and comorbid psychopathology. This was somewhat surprising given that AB has been consistently associated with lower intelligence (Ogilvie et al., 2011; Sánchez de Ribera et al., 2019). Importantly, whereas previous work has often accounted for gender and age (i.e., included as covariate or used matched groups), few studies have accounted for socioeconomic status or comorbid psychopathy when looking at associations between AB/CU traits and intelligence. Our findings therefore highlight the importance of examining associations in more representative, population-based samples to assess the impact of key demographic and individual differences variables (i.e., socioeconomic status, comorbid psychopathology).

Strengths and Limitations

The current study benefitted from several strengths, including a large sample size, specific performance tasks, and dimensional analyses of AB and CU traits in a community population sampled from birth records and enriched for risk via the risks that living in poverty convey for AB (Farrington, 2005). Despite these strengths, there are limitations worth noting. First, we utilized a cross-sectional design, and thus were unable to determine the directionality of associations among AB, CU traits, and cognitive functioning.

Second, the current study was limited to a brief measure of intelligence and certain neuropsychological tasks; thus, we were unable to look at associations with more comprehensive intelligence measures or additional tasks that have been previously examined in relation to AB and CU traits (i.e., Stroop Task, Tower of London). Indeed, we did not find expected, somewhat well-established, zero-order correlations between AB and cognitive abilities, in particular response inhibition. Additionally, our Go/No-Go task was not explicitly designed to separate distinct aspects of executive functioning; thus, our indices overlap conceptually and in measurement in the extent to which they capture inhibition, selective, and sustained attention. More broadly, the assertion that these putative aspects of executive function represent distinct individual difference dimensions has recently been strongly questioned, especially in the context of developmental populations (Karr et al., 2018). Further research using alternative and distinct tasks, particularly those designed explicitly to reliably separate out different components of executive functioning, will be important to assess the generalizability of our findings. At the same time, we still see merit in these analyses, as single continuous performance tasks in clinical practice yield multiple helpful indices of attention and executive function (e.g., IVA+Plus; Sandford & Turner, 2009), which inform feedback and planning for clients.

Third, the current study did not include any tasks of social cognition. Previous studies have more consistently found associations between CU traits and deficits in affective neurocognitive abilities (i.e., emotion recognition, emotional empathy, emotional attention) in contrast to broader cognitive abilities related to executive functioning (Frick et al., 2014; Salekin, 2017). Thus, an important future direction will be to examine whether high levels of AB and CU traits display differential associations with social cognitive abilities (versus basic attention and executive functioning) compared to high levels of AB or CU traits in isolation.

Fourth, because we examined associations in a community sample with varying levels of AB, cognitive deficits in childhood may only be present in clinical or forensic samples that could be characterized by more persistent or severe AB, including heavier substance use (Moffitt, 2018). Moreover, adolescents who do engage in high levels of antisocial behavior, but who have avoided adjudication in our community sample, likely are unique, and thus might have protective factors (including more familial or financial resources). However, a benefit of community samples is the ability to determine if associations persist dimensionally, outside of extremes, which could help inform the potential use of neuropsychological tests in assessing cognitive deficits of AB in the community.

Fifth, the current study was limited to parent-reported previous or current psychiatric diagnoses and current medications. Future research would benefit from more thorough and comprehensive assessment of psychiatric and/or forensic history, as well as medication usage. Sixth, the current sample was predominantly composed of adolescents. Additional research is needed to determine whether associations are similar at different developmental periods. Seventh, in the current study we collected both child and parent-reported CU traits. However, we specifically chose to focus on child-reported AB and CU traits, as adolescents may be more

accurate reporters of their own AB and CU traits as they spend less time in the home and more time in school and with peers (Achenbach et al., 1987). Given concerns about youth reporting on their own CU traits, in a set of exploratory analyses, we re-analyzed our data with a combination of parent and child reported CU traits and found that, though the pattern of findings was similar, the AB x CU interaction in relation to GNG reaction time variability was only at trend-level significance. Thus, these findings suggest that these associations may not generalize across informants. On the other hand, these informant discrepancies could reflect important contextual variation in children's behavior and/or differences in informants' perspectives of the behaviors (De Los Reyes, 2011; De Los Reyes et al., 2015).

Seventh, although we had a substantial sample size and were powered to detect moderate to large effect sizes (80% power for effect sizes ranging from $f^2 = .15 - .35$), we were slightly underpowered to detect small effect sizes (i.e., $f^2 = .02$) for interactions in a complex structural equation model (Aiken et al., 1991; Faul et al., 2007). As such, we may have been unable to detect smaller effect sizes in the interaction analyses due to lack of power.

Conclusions

In sum, we identified distinct cognitive profiles for those with AB with and without high levels of CU traits. We specifically found unique neurocognitive profiles for the combination of elevated AB and CU traits (lower reaction time variability) compared to elevated AB at low levels of CU traits (higher reaction time variability). Our findings add to the accumulating literature suggesting that the combination of AB and CU traits is etiologically distinct from AB alone and emphasize the need for further research in representative samples able to incorporate key demographic and individual differences variables (i.e., gender, socioeconomic status, comorbid psychopathology).

Table 3.1: Sources of data loss

	Number lost	Participants with data
Original sample		550
Sample with measure of AB		
- Missing Self-Report of Delinquency	19	531
Sample with measure of AB & CU traits		
- Missing Inventory of Callous-Unemotional Traits	18	513
Sample with measure of AB, CU traits & ADHD		
- Missing Youth Self Report	21	492
Sample with measure of AB, CU traits, ADHD & depression		
- Missing Child Depression Inventory	4	488
Sample with measure of AB, CU traits, ADHD, depression & anxiety		
- Missing Multidimensional Anxiety Scale for Children	14	474
Total current sample		474

Note. AB = antisocial behavior. CU = callous-unemotional.

Table 3.2: Descriptives of measures

Measure	Total Items	Mean	SD	Min	Max	α
Self-Report of Delinquency (excluding substance use)	50	4.11	4.98	0	40	.87
Inventory of Callous-Unemotional Traits	22	17.72	6.68	1	41	.77
Substance Use (Self-Report of Delinquency)	12	.69	1.78	0	13	.80
Attention Problems (Youth Self Report)	9	4.30	3.13	0	15	.77
Child Depression Inventory	27	7.01	6.14	0	35	.88
Multidimensional Anxiety Scale for Children	39	45.68	18.11	0	94	.92

Note. SD = standard deviation. Min = minimum. Max = maximum.

Table 3.3: Zero-order correlations among antisocial behavior, callous-unemotional traits, and indices of cognitive functioning

	AB	CU Traits	Vocabulary	Blocks	IQ Composite	Efficiency	SSRT	Accuracy
Antisocial Behavior								
Callous-Unemotional Traits	.30***							
Vocabulary	-.18*	-.11		.				
Blocks	-.16*	-.10	.39***					
IQ Composite	-.20**	-.13	.81***	.84***				
GoNoGo Efficiency	-.01	-.10*	.09	.21**	.15^{+}			
SSRT	-.01	.03	.02	.01	.02	-.12		
GoNoGo Go Accuracy	-.01	-.04	.05	.16*	.12	.18***	-.02	
GoNoGo RTV	.04	.10*	-.12	-.17*	-.15^{+}	-.79***	.08	-.32***

Note. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***. AB = antisocial behavior. CU = callous-unemotional. Blocks = Block Patterns score. Efficiency = GoNoGo efficiency score. SSRT = stop signal reaction time on correct trials. Accuracy = GoNoGo accuracy on 'Go' trials. RTV = GoNoGo reaction time variability.

Table 3.4: Zero-order correlations indices of cognitive functioning and covariates

	Gender	Age	Income	Substance Use	ADHD	Depression	Anxiety	Medication	Psychiatric Hx
Vocabulary	-.08	-.13^{+}	.20**	-.14^{+}	-.11	.01	.02	.01	-.09
Blocks	-.11	.02	.22**	-.06	-.09	-.00	.13^{+}	-.14^{+}	-.16*
IQ Composite	-.10	-.15*	.25**	-.15^{+}	-.10	.00	.10	-.08	-.16*
GoNoGo Efficiency	.16**	.52***	.16**	.03	.04	.07	.16**	-.04	.00
SSRT	-.07	-.13^{+}	-.06	.01	.02	.05	-.01	.06	.01
GoNoGo Go Accuracy	-.05	.07	.22***	-.08	-.08	-.01	.14**	.02	-.06
GoNoGo RTV	-.15**	-.39***	-.21***	-.01	-.01	-.04	-.17**	-.03	-.01

Note. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***. ADHD= Attention deficit hyperactivity disorder. Efficiency = GoNoGo efficiency score. SSRT = stop signal reaction time on correct trials. Accuracy = GoNoGo accuracy on 'Go' trials. RTV = GoNoGo reaction time variability. Gender was coded as follows: 0 = Male, 1 = Female. Substance use was measured using a sum score of the 12 substance use items from the Self-Report of Delinquency Questionnaire (SRD). Medication = parent-reported medication usage (0 = no medications; 1 = current medication usage). Psychiatric Hx = parent-reported lifetime psychiatric history (0 = no previous psychiatric diagnoses; 1 = has ever been given psychiatric diagnosis). ADHD was measured with the 9 item attention problems subscale of the Youth Self Report (YSR). Depressive symptoms were measured using a total sum score of the Child Depression Inventory (CDI). Anxiety symptoms were measured using a total sum score of the Multidimensional Anxiety Scale for Children (MASC).

Table 3.5: Associations among callous-unemotional traits, antisocial behavior, and indices of cognitive functioning, controlling for other forms of psychopathology

Main Effects Model: IQ

	Vocabulary			Blocks			Composite Score		
	B	SE	R^2	B	SE	R^2	B	SE	R^2
Annual Income	.14	.10		.06	.11		.11	.11	
Drug Use	-.04	.11		.03	.10		-.03	.11	
CDI Total Score	.18*	09		.07	.08		.13	.09	
YSR Attention Problems	-.08	.08	$.14^{+}$	-.09	.08	.21***	-.07	.08	.23**
MASC Total Score	-.05	.09		.14	.09		.06	.08	
Medication	.02	.07		-.09	.07		-.04	.07	
Psychiatric Hx	-.08	.09		-.08	.08		-.10	.08	
Callous-Unemotional Traits	-.07	.10		-.10	.10		-.11	.10	
Antisocial Behavior	-.14	.09		-.12	.09		-.13	.09	

Main Effects Model: GoNoGo Task

	Go Accuracy			Efficiency			Reaction Time Variability		
	B	SE	R^2	B	SE	R^2	B	SE	R^2
Annual Income	.19***	.04		.09	.05		-.15**	.05	
Drug Use	-.10	.08		$-.09^{+}$	.05		.05	.05	
CDI Total Score	-.02	.06		-.02	.06		.06	.06	
YSR Attention Problems	-.08	.05	.09*	-.02	.06	.32***	-.04	.06	.22***
MASC Total Score	.20**	.08		$.10^{+}$	.05		$-.12^{+}$	.06	
Medication	.06	.04		-.01	.05		-.07	.05	

Psychiatric Hx	-.04	.07		.00	.07		.02	.05	
Callous-Unemotional Traits	-.01	.03		-.09	.05		.07	.06	
Antisocial Behavior	.05	.06		.00	.07		.05	.07	

Main Effects Model: Stop Signal Task

	SSRT		
	B	SE	R^2
Annual Income	-.09	.09	
Drug Use	.08	.08	
CDI Total Score	.09	.08	
YSR Attention Problems	.01	.07	.10
MASC Total Score	-.06	.11	
Medication	.09	.07	
Psychiatric Hx	-.06	.07	
Callous-Unemotional Traits	.01	.08	
Antisocial Behavior	-.06	.08	

Note. $p < .10^{+}$, $p < .05$*, $p < .01$**, $p < .001$***. AB = antisocial behavior. Blocks = Block Patterns score. CU = callous-unemotional. Efficiency = GoNoGo efficiency score. SSRT = stop signal reaction time on correct trials. Accuracy = GoNoGo accuracy on 'Go' trials. RTV = GoNoGo reaction time variability. CDI = Child Depression Inventory. YSR = Youth Self Report. MASC = Multidimensional Anxiety Scale for Children. Medication = parent-reported medication usage (0 = no medications; 1 = current medication usage). Psychiatric Hx = parent-reported lifetime psychiatric history (0 = no previous psychiatric diagnoses; 1 = has ever been given psychiatric diagnosis). Race (0 = Non-white, 1 = White), gender (0= Male, 1=Female), and age were included as covariates in all models.

Table 3.6: Associations among callous-unemotional traits, antisocial behavior, antisocial behavior x callous-unemotional traits, and indices of cognitive functioning, controlling for other forms of psychopathology

Interaction Model: IQ

	Vocabulary			Blocks			Composite Score		
	B	SE	R^2	B	SE	R^2	B	SE	R^2
Annual Income	.14	.10		.06	.11		.11	.11	
Drug Use	-.03	.11		.02	.10		-.03	.11	
CDI Total Score	.18*	.08		.07	.08		.14	.09	
YSR Attention Problems	-.08	.08	$.15^{+}$	-.09	.08	.21***	-.07	.08	.24**
MASC Total Score	-.05	.09		.15	.09		.06	.08	
Medication	.02	.07		-.09	.07		-.05	.07	
Psychiatric Hx	-.07	.09		-.08	.08		-.10	.08	
Callous-Unemotional Traits	-.09	.10		-.09	.11		-.11	.11	
Antisocial Behavior	$-.19^{+}$	.10		-.10	.11		-.15	.09	
AB x CU Traits	.09	.08		-.02	.08		.04	.08	

Interaction Model: GoNoGo Task

	Go Accuracy			Efficiency			Reaction Time Variability		
	B	SE	R^2	B	SE	R^2	B	SE	R^2
Annual Income	.18*	.04		$.09^{+}$	.05		-.14**	.05	
Drug Use	-.10	.08		-.10*	.05		.08	.05	
CDI Total Score	-.02	.06		-.02	.06		.06	.06	
YSR Attention Problems	-.08	.05	.09*	-.03	.06	.33***	-.02	.05	.23**
MASC Total Score	.20**	.09		.11*	.05		-.12*	.06	*
Medication	.06	.04		-.02	.05		-.06	.05	

Psychiatric Hx	-.04	.07	-.01	.05	.02	.05
Callous-Unemotional Traits	-.01	.03	-.10*	.05	.08	.06
Antisocial Behavior	.05	.06	-.01	.06	.06	.06
AB x CU Traits	.04	.04	.12^{+}	.06	-.17**	.06

Interaction Model: Stop Signal Task			
	SSRT		
	B	SE	R^2
Annual Income	-.10	.09	
Drug Use	.08	.08	
CDI Total Score	.09	.08	
YSR Attention Problems	.00	.07	.10
MASC Total Score	-.06	.11	
Medication	.08	.06	
Psychiatric Hx	-.06	.07	
Callous-Unemotional Traits	.00	.09	
Antisocial Behavior	-.10	.08	
AB x CU Traits	.06	.05	

Note. $p < .10^{+,}$ $p < .05$*, $p < .01$**, $p < .001$***. AB = antisocial behavior. Blocks = Block Patterns score. CU = callous-unemotional. Efficiency = GoNoGo efficiency score. SSRT = stop signal reaction time on correct trials. Accuracy = GoNoGo accuracy on 'Go' trials. RTV = GoNoGo reaction time variability. CDI = Child Depression Inventory. YSR = Youth Self Report. MASC = Multidimensional Anxiety Scale for Children. Medication = parent-reported medication usage (0 = no medications; 1 = current medication usage). Psychiatric Hx = parent-reported lifetime psychiatric history (0 = no previous psychiatric diagnoses; 1 = has ever been given psychiatric diagnosis). Race (0 = Non-white, 1 = White), gender (0= Male, 1=Female), and age were included as covariates in all models. Three separate models were run for each neuropsychological task.

Table 3.7: Simple slopes and regions of significance for significant antisocial behavior x callous-unemotional traits interactions in relation to indices of cognitive functioning

Task	Low CU Traits				Average CU Traits				High CU Traits			
	B	*SE*	t	p	B	*SE*	t	p	B	*SE*	t	p
GoNoGo RTV	1.20[1]	.41	2.97	.003	.10	.06	1.81	.07[+]	-1.00	.38	-2.65	.008
	AB Region of Significance (% Sample in RoS)						CU Traits Region of Significance (%Sample in RoS)					
GoNoGo RTV	> 2.06 (20.9%)						< -.32 (51.9%); > 1.78 (37.6%)					

Note. [1] = association significant at conservative Bonferroni correction standard accounting for multiple comparisons (.05 / 3 models = .017). AB = antisocial behavior. CU = callous-unemotional. RTV = GoNoGo reaction time variability. RoS = Region of significance. Standardized beta weights from models that included gender, race, family annual income, mean drug use, age, depression symptoms, ADHD symptoms, anxiety symptoms, parent-reported psychiatric history, parent-reported medication usage, AB, CU traits, and the interaction term for AB x CU traits. Models were ran using centered variables of AB (Range -4.20-35.80, Uncentered Range 0-34) and CU traits (Range -16.74-23.26, Uncentered Range 1-41). Region of significance (RoS) indicate the centered value at which the simple slopes are significantly different from zero (i.e., Vocabulary: at centered values of AB greater than 3.10, the simple slope is significantly different from zero). % Sample in RoS indicates the percentage of the entire sample that falls within the threshold of AB or CU traits at which the simple slope is significantly different from zero.

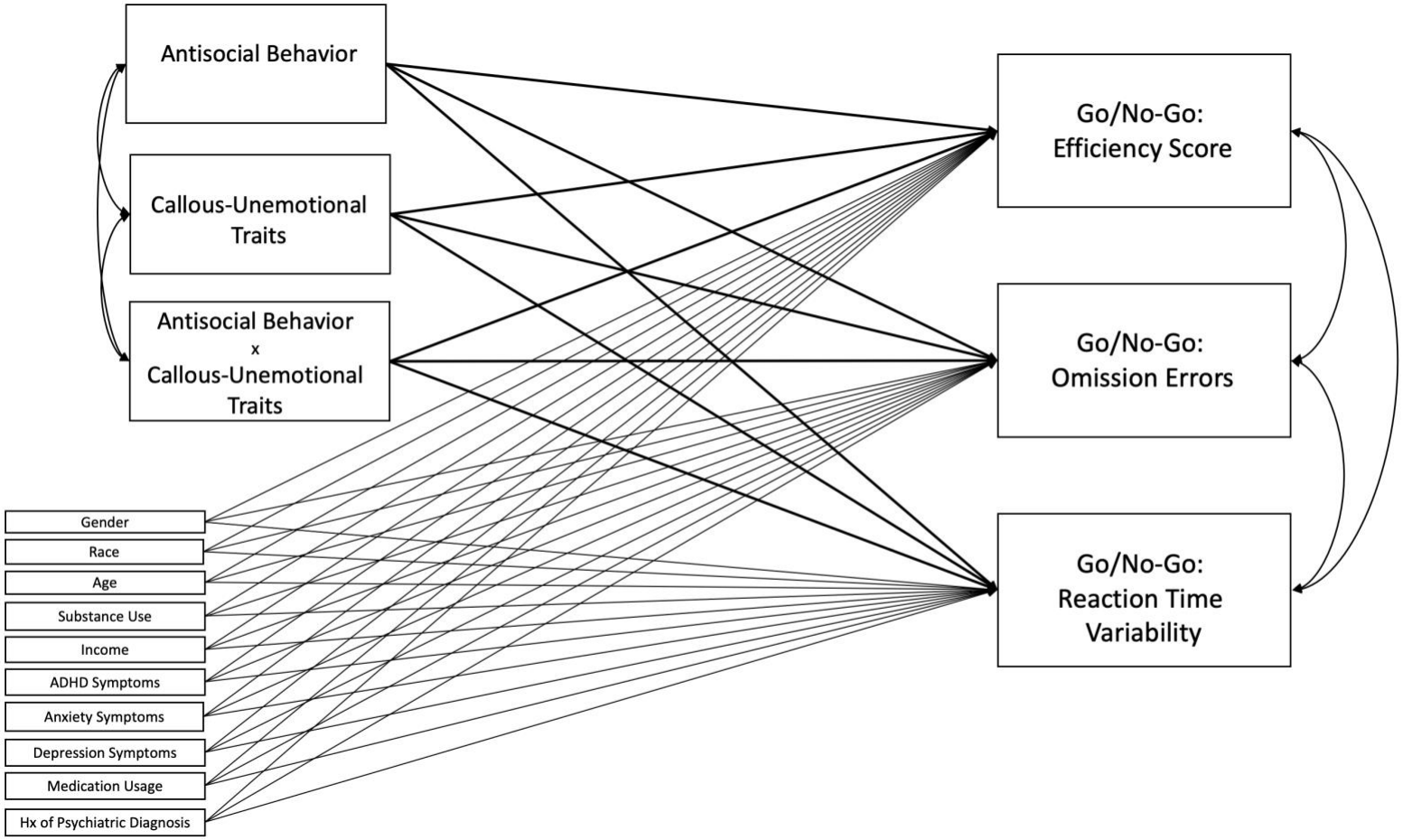

Figure 3.1: Graphical depiction of model examining associations among antisocial behavior, callous-unemotional traits, and GoNoGo task performance.

In this model, antisocial behavior, callous-unemotional traits, and an interaction term (antisocial behavior x callous-unemotional traits) are included as predictors of performance on the three indices of cognitive functioning measured using the GoNoGo task (i.e., efficiency score, 'Go' accuracy, reaction time variability). Gender, age, race, substance use, annual family income, depression symptoms, ADHD symptoms, anxiety symptoms, medication usage, and psychiatric diagnosis are included as covariates.

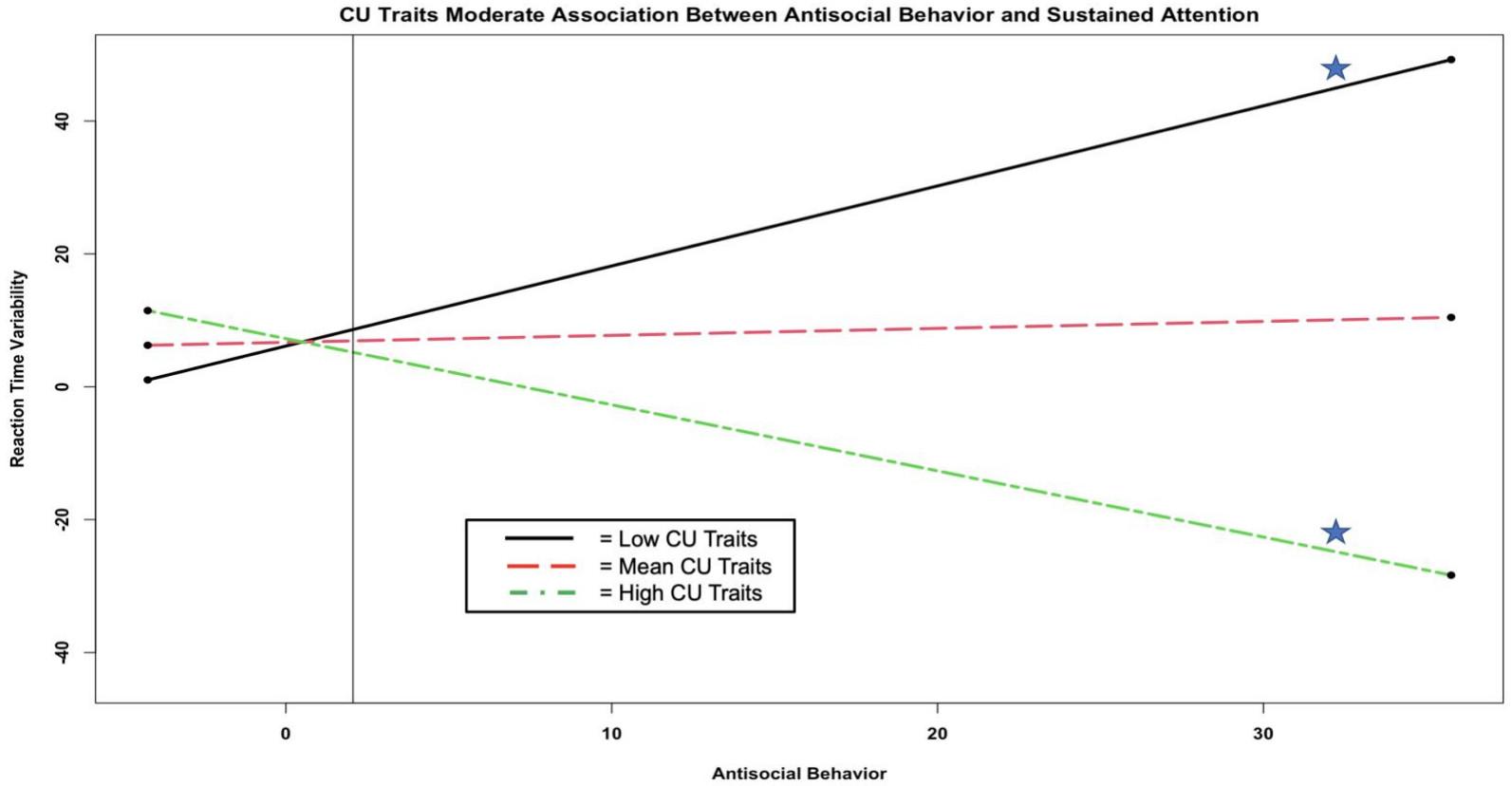

Figure 3.2: Callous-unemotional traits moderate the association between antisocial behavior and reaction time variability. Note. AB= antisocial behavior. CU traits = callous-unemotional traits. RTV = GoNoGo reaction time variability. Simple slopes plotted at mean levels, 1 SD above the mean, and 1 SD below the mean for CU traits, as recommended by Aiken et al. (1991) and using an online computational tool (Preacher et al., 2006). Models were ran using centered variables of AB (Range -4.24-35.76, Uncentered Range 0-40) and CU traits (Range -16.97-23.03, Uncentered Range 1-41). Star next to line indicates significant slope. At low levels of CU traits (CU Traits < -.32; 51.9% of the sample), higher RTV (worse sustained attention) was significantly related to higher levels of AB. Additionally, at high levels of CU traits (CU Traits > 1.78; 37.6% of the sample), AB was associated with lower RTV (better sustained attention). The horizontal line indicates the level of AB at which these associations are significant (AB > 2.06; 20.9% of the sample).

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Chapter 4: Connections That Characterize Callousness: Affective Features of Psychopathy Are Associated With Personalized Patterns of Resting-State Network Connectivity

Introduction

Psychopathic traits predict chronic criminal behavior and have been estimated to account for approximately $460 billion of the annual cost of crime (Kiehl & Hoffman, 2011; Skeem et al., 2011). Unfortunately, there are no empirically-supported treatments for individuals with psychopathic traits (Reidy et al., 2013), likely due, in part, to a lack of understanding of the etiology and individuality of psychopathic traits, which can be efficaciously examined using neural networks.

Neural Networks and Psychopathy

Etiological theories of psychopathy have centered either on emotional or attentional deficits. In the former, psychopathy is marked by deficient emotional processing via deficits in the paralimbic system (Blair, 2010; Kiehl, 2006). In the latter, psychopathy is marked by an attention bottleneck (i.e., failure to attend to contextual information when engaged in goal-directed behavior) due to impairments in top-down circuitry (e.g., prefrontal cortices; Larson et al., 2013; Newman & Baskin-Sommers, 2012). In general, neuroimaging investigations of these theories have focused on activation in specific regions (i.e., paralimbic and prefrontal). Importantly, recent empirical work and theory have suggested that abnormalities in connectivity within and across *multiple* networks could explain *both* emotional and attentional deficits associated with psychopathic traits (Contreras-Rodríguez et al., 2015; Espinoza et al., 2018; Ewbank et al., 2018; Geurts et al., 2016; Korponay et al., 2017; Leutgeb et al., 2016; Motzkin et

al., 2011; Philippi et al., 2015; Tang et al., 2013; Waller et al., 2018; Yang et al., 2012; Yoder et al., 2014).

The Impaired Integration theory of psychopathy (II theory; Hamilton et al., 2015) suggests that connectivity within and between three resting-state networks underlies affective and cognitive processes involved in psychopathy, as they are implicated in perspective-taking, fear conditioning, and inhibitory control: (1) the default mode network (DMN), including the ventromedial prefrontal cortex (vmPFC) and posterior cingulate cortex (PCC); (2) the salience (or cingulo-opercular) network (SN), including the anterior insula and dorsal anterior cingulate cortex (ACC); and (3) the central executive (or frontoparietal) network (CEN), including the dorsolateral prefrontal cortex (dlPFC) and posterior parietal cortex (PPC) (Menon, 2011). The DMN is typically activated during resting-state and during tasks related to social cognition, autobiographical memory, theory of mind, and moral reasoning, while being de-activated during externally oriented, non-social thinking (Buckner et al., 2008; Reniers et al., 2012). In contrast, the CEN is de-activated at rest and activated during working memory and decision-making in goal-directed behavior, particularly when tasks are cognitively challenging (Menon & Uddin, 2010). The SN is de-activated at rest and activated during various neurocognitive functions, adjusting arousal and attention based on external cues and internal states to enable switching between other networks (Seeley, 2019; Sridharan et al., 2008). Despite being de-activated at rest, brain regions that constitute the SN and CEN still function synchronously during rest (Biswal et al., 2010; Greicius, 2008; Menon, 2011; Menon & Uddin, 2010), providing opportunities to investigate their interplay with each other and with the task-negative DMN. Such investigations reveal that the SN modulates the activity of both the CEN and DMN (Goulden et al., 2014). The II theory posits that psychopathy is characterized by abnormal functioning of the SN and DMN,

but intact functioning of the CEN. Compromised SN and DMN functioning is thought to impact the integration of complex sensory information, which is key in emotional learning (Blair, 2017) and relies on attendance to and integration of external (e.g., emotional faces; social norms) and internal cues (e.g., emotions; desires) to inform decision-making and behavior. II theory also posits that the CEN functions normally when engaged, which explains why individuals with psychopathic traits do not consistently display cognitive control deficits (Hamilton et al., 2015).

Very few studies have explicitly examined connectivity within and between regions of the DMN, SN, and CEN, as outlined in the II theory. In fact, researchers have only recently begun to look at associations between resting-state (i.e., "task-free") connectivity and psychopathic traits. Most studies have utilized seed-based approaches to examine the connectivity of regions and the choice of regions of interest has been guided by task-based studies of neural activation (e.g., the amygdala, which has been implicated in emotion processing paradigms; Contreras-Rodríguez et al., 2015; Korponay et al., 2017; Motzkin et al., 2011; Philippi et al., 2015). A few studies have used a region of interest approach to examine connectivity among *a priori*, albeit differential, nodes within the DMN, SN, and CEN. For instance, Contreras-Rodríguez et al. (2015) found increased positive connectivity between regions within the CEN in offenders with psychopathic traits compared to non-offender controls. Additionally, compared to offenders *without* psychopathic traits, Motzkin et al. (2011) found reduced connectivity between regions within the DMN in offenders with psychopathic traits, and Philippi et al. (2015) found reduced connectivity between the SN and CEN. Other studies have used a whole-brain approach. For example, Espinoza et al. (2018) found psychopathic traits to be associated with impaired connectivity between the SN and DMN, with findings varying by brain

region (i.e., ACC versus insula). Thus, the existing literature is limited, and findings do not clearly converge across studies.

One reason why findings do not converge is because most relevant work utilizes group averages, assuming all individuals display similar patterns of connectivity, despite evidence that neurobiological mechanisms of psychopathy differ across people (Baskin-Sommers et al., 2011; Efferson & Glenn, 2018; Gao & Raine, 2010) and features of psychopathy (Espinoza et al., 2018; Korponay et al., 2017; Philippi et al., 2015; Vermeij et al., 2018; Wolf et al., 2015). In fact, recent research has turned towards examining biological heterogeneity that could reveal underlying mechanisms of differing symptom presentations (Insel, 2014). By ignoring variation among individuals, average approaches may produce spurious connections in neural networks that do not accurately describe individuals (Gates & Molenaar, 2012; Smith et al., 2011). Researchers cannot be confident in their interpretation of results without first ensuring accurate modeling of neural networks. This point is particularly relevant to the study of psychopathy, given that individuals with high levels of psychopathic traits have been shown to vary in terms of behavioral phenotypes, clinical outcomes, and biological correlates (including neural mechanisms) (Latzman et al., 2019).

Another reason why findings from neuroimaging studies of psychopathy do not converge is that most have examined the *strength* of specific connections (Johanson et al., 2020) and failed to consider the *way* neural networks are arranged (i.e., "topology"; De Vico Fallani et al., 2014; Kaiser, 2011), which has been utilized in other neuroscience work. Network analytic approaches are well-suited for testing hypotheses of the II theory regarding overall network functioning and architecture by examining topological features, including the characteristics of relations between regions (i.e., nodes) within a network (e.g., number, length, direction of connections or ‘edges’).

For instance, network "density" (i.e., number of connections in a sparse network) indicates the extent to which information travels between nodes within the same network or across different networks. Additionally, "node centrality" (i.e., number of connections into and/or out of a specific node) reflects the importance of a node within a network for facilitating communication between networks (De Vico Fallani et al., 2014; Kaiser, 2011). In this way, topology underlies information processing and has been shown to predict cognitive functioning (Cohen & D'Esposito, 2016).

Therefore, network topology approaches to resting-state fMRI have potential to reveal the neural architecture underlying "baselines" or "intrinsic" patterns of connectivity associated with psychopathic traits. However, only two studies have been conducted in adults using such an approach. In one study, psychopathic traits were associated with increased centrality of DMN and SN nodes (Lindner et al., 2018), whereas the other study did not find any significant associations with DMN or SN features (Tillem et al., 2019). Notably, these studies were limited by focus on either women from the community or incarcerated offenders. Regarding female populations, research suggests that there are gender differences in the expression of psychopathic traits, (Efferson & Glenn, 2018), highlighting the need to examine network topology in male samples. Regarding offender populations, psychopathic traits vary dimensionally in the community (Lilienfeld, 2018), and thus psychopathic traits may have a different etiology or presentation among individuals who have not been incarcerated for offenses (Gao & Raine, 2010). Indeed, with one exception (Lindner et al., 2018), all previous resting-state connectivity studies of psychopathy in adults have been conducted in offender populations (Contreras-Rodríguez et al., 2015; Espinoza et al., 2018; Korponay et al., 2017; Motzkin et al., 2011; Philippi et al., 2015; Tillem et al., 2019). Finally, neither study examined psychopathy at the

facet-level. Previous studies have found that psychopathy consists of distinct symptom sets or 'facets': interpersonal (e.g., grandiosity, manipulation), affective (e.g., lack of remorse, callousness), lifestyle (e.g., sensation-seeking, irresponsibility), and antisocial (e.g., violence, criminal versatility) (Dotterer et al., 2016; Mahmut et al., 2011; Neal & Sellbom, 2012; Neumann et al., 2012; Seara-Cardoso et al., 2012). Although highly correlated, these facets are characterized by unique behavioral deficits underpinned by distinct neural systems (Carré et al., 2013; Deming et al., 2018; Latzman et al., 2019; Vermeij et al., 2018). Thus, it is unclear whether the four facets are characterized by unique network features, as has been demonstrated using other resting-state approaches (e.g., Espinoza et al., 2018).

Novel network approaches now *combine* more traditional, group-level approaches with person-specific approaches, which assume that participants are heterogeneous and have data that should not be averaged. For example, group iterative multiple model estimation (GIMME; Gates & Molenaar, 2012) is a data-driven approach that creates person-specific networks by first mapping connections between nodes that are statistically meaningful at the group-level (i.e., found across the entire sample), and then adding connections that are statistically meaningful at an individual-level (i.e., are unique to a person) – all while providing connection estimates that are unique for individuals. Simulation studies show that GIMME outperforms other network approaches, including Granger causality and Bayes nets, particularly when data are heterogenous (Gates & Molenaar, 2012). Empirical studies utilizing GIMME to understand neural mechanisms of psychopathology have demonstrated significant variability in neural network configurations across patients, highlighting biological heterogeneity within the same diagnosis (Beltz et al., 2018; Price et al., 2017). However, no studies have yet applied person-specific approaches to the mapping of neural networks underlying psychopathic traits.

Current Study

The goal of this study was to delineate associations between psychopathy (including interpersonal, affective, lifestyle, and antisocial facets) and person-specific network connectivity within the DMN, CEN, and SN in an ethnically diverse, male community sample at heightened risk for antisocial behavior (Hyde et al., 2016). We used GIMME (Beltz & Gates, 2017; Gates & Molenaar, 2012) to generate person-specific connectivity maps for each participant, and we examined whether psychopathic traits were uniquely associated with network features (i.e., density; node centrality) across participants.

Methods

Participants

The final sample included 123 participants from the Pitt Mother & Child Project, a longitudinal study of 310 low-income, ethnically diverse boys and their families (Shaw et al., 2012). Families were recruited from Allegheny County Women, Infants, and Children Nutritional Supplement Clinics in 1991 and 1992 when the boys were 6 to 17 months of age (Shaw et al., 2003; Shaw et al., 2012) and seen almost yearly from age 1.5-23 years. At the first assessment, mean per capita income of family members was $2,892 per year, with a mean Hollingshead socioeconomic status score of 24.5, indicative of a working class-to-impoverished sample. This sample is considered to be at heightened risk for antisocial behavior based on gender, familial socioeconomic status, and urbanicity, allowing us to examine hypotheses in a sample with a wide range of variability in psychopathic traits (Beck & Shaw, 2005; Gard et al., 2017; Hyde et al., 2016). All procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

At age 22 years, 255 participants from the original sample completed questionnaires, with a subsample of participants (*n*=180) participating in magnetic resonance imaging (MRI) (Shaw et al., 2012). The MRI component introduced some data loss (Supplemental Table 4.1), resulting in 126 men with high quality resting-state fMRI data. Of the 126 participants, one was excluded because of a diagnosis of autism spectrum disorder, and two were missing data on psychopathic traits. Of the included participants (*n*=123), most self-reported their race as European American (*n*=66, 52.8%) or African American (*n*=45, 36.6%; *n*=13, 10.6% self-reported "other"). Participants reported a relatively low mean income (*M*=$13,770.30, *SD*=$12,605.34). The included 123 participants did not significantly differ from the original 310 participants in family income at recruitment ($t(309)=-1.58$, $p=.12$), mother's education ($t(311)=-1.56$, $p=.12$), race ($x^2(3)=1.18$, $p=.76$), or parent-reported externalizing behaviors ($t(277)=.204$, $p=.85$) (measured using the Child Behavior Checklist at age 2; Achenbach, 1991).

Measures

Self-reported psychopathic traits. At age 22, psychopathic traits were assessed using the 29-item Self-Report Psychopathy Short-Form (Neumann & Pardini, 2014; Paulhus et al., 2015), a self-report measure of psychopathy derived from and shown to correlate highly with the Psychopathy Checklist-Revised (Neumann et al., 2015; Paulhus et al., 2015). Participants rated these items based on the extent to which they thought the statements reflected their own beliefs using a 5-point Likert scale (1=disagree strongly, 5=agree strongly). The items measured four dimensions of psychopathy: interpersonal manipulation (e.g., "I think I can beat a lie detector"), affective callousness (e.g., "I never feel guilty over hurting others"), erratic lifestyle (e.g., "I've often done dangerous things just for the thrill of it"), and criminal tendencies (e.g., "I have tried to hit someone with a vehicle") (Neumann & Hare, 2008). Each subscale of the SRP-SF showed

adequate internal consistency in the current study (interpersonal manipulation, α=.84; affective callousness, α=.72; erratic lifestyle, α=.71; criminal tendencies, α=.70; total score, α=.90), similar to previous studies of the SRP-SF (Neal & Sellbom, 2012; Neumann, et al., 2012).

Resting-state fMRI and preprocessing. All scanning parameters were selected to optimize the quality of the BOLD signal while maintaining a sufficient number of slices to acquire whole-brain data. Before collecting fMRI data for each participant, a reference echoplanar imaging scan was acquired and visually inspected for artifacts (e.g., ghosting) and good signal across the entire volume of acquisition. Additionally, an autoshimming procedure was conducted before the acquisition of BOLD data in each participant to minimize field inhomogeneities.

Resting-state functional images were collected while participants were awake, passively viewing a fixation cross for 6 minutes. T2*-weighted images (TR=2000;TE=27;FOV= 24cm;flip angle=75°;39 3.10 mm slices;180 TRs) were acquired using a research-dedicated Siemens 3-T Tim Trio. We conducted standard preprocessing in FSL (http://www.fmrib.ox.ac.uk/fsl) (Jenkinson et al., 2012), including removal of the first four volumes, motion correction using MCFLIRT (Jenkinson et al., 2002), slice-timing correction, non-brain removal, co-registration to high resolution structural scans (MPRAGE), normalization to MNI 152 space using 12-dof linear registration in FLIRT, and spatial smoothing using a Gaussian kernel (6-mm). After standard preprocessing, we applied ICA-AROMA, an automated algorithm to detect head motion-related artifacts at the subject-level based on independent component analysis. ICA components identified as related to head motion were subtracted out of the data using fsl_regfilt (Pruim, Mennes, Buitelaar, et al., 2015; Pruim, Mennes, van Rooij, et al., 2015). Finally, we regressed

out mean signals from CSF and white matter and applied a high-pass temporal filter (100.0 sigma).

Eight *a priori* ROIs (network nodes) defined three networks (Goulden et al., 2014; Hamilton et al., 2015; Sridharan et al., 2008): vmPFC and PCC for the DMN; right and left insula and the ACC for the SN; and right and left PPC and dlPFC for the CEN. These ROIs have been implicated in previous resting and task-based connectivity studies of the interplay among the DMN, SN, and CEN (Chiong et al., 2013; Goulden et al., 2014; Sridharan et al., 2008). Spherical binarized ROI masks were created for each of the eight regions of interest (vmPFC and PCC for the DMN; right and left insula and the ACC for the SN; and right and left PPC and dlPFC for the CEN) using fslmaths and peak coordinates as identified in previous research (Sridharan et al., 2008; Supplemental Table 4.2). The vmPFC, PCC, and ACC were all single clusters due to their medial locations. The size of the spheres were subject-specific to account for differences in brain volume calculated from segmentation images (i.e., total volume excluding CSF) from FAST (FMRIB's Automated Segmentation Tool; http://www.fmrib.ox.ac.uk/fsl/). Specifically, subject-specific spheres were determined by multiplying a sphere size of 6.5 to the subject's brain volume divided by the median brain volume of the entire sample (Range: 5.26 - 7.74 mm). Mean timeseries at each volume were extracted using the subject-specific spherical ROI masks.

Analysis Plan

We conducted analyses in two steps. First, we generated person-specific networks for each participant using GIMME. Second, we extracted network features from each participant's map to examine associations with psychopathic traits across the sample. We conducted all analyses in MPlus version 8.2 (Muthén & Muthén, 2020) using robust maximum likelihood

estimation which is robust to relaxed assumptions of the data (e.g., non-normality) (MLR; Yuan & Bentler, 2000) with the exception of GIMME, which relies on lavaan in RStudio (Lane & Gates, 2017).

GIMME. We submitted node timeseries for the 123 participants to GIMME, a sparse modeling approach that iteratively adds only statistically meaningful connections (i.e., improve fit of a null or less parameterized model) to a network. Most alternate approaches model all connections between regions (i.e., create saturated networks) and then use forms of regularization to minimize weak connections, but this can be subjective and lead to networks that are denser than necessary to explain the raw data (Fornito, 2016). Sparse modeling approaches instead minimize spurious contemporaneous connections (Gates et al., 2010).

For each participant, GIMME generates a unified structural equation model (uSEMs; Gates et al., 2011), which includes both contemporaneous (i.e., one node predicts another in the same functional volume) and first order lagged (i.e., one node predicts itself or another at the next functional volume) connections. Each connection has a person-specific direction (i.e., positive or negative) and magnitude (reflected by beta weights). Thus, GIMME improves upon other network approaches that only model contemporaneous, zero-order correlations of activation between regions (Fornito, 2016; Gates & Molenaar, 2012). Moreover, GIMME includes a grouping algorithm to account for both homogeneity (in group-level connections) and person-specific heterogeneity (in individual-level connections) (Gates & Molenaar, 2012). By including a group-level structure (i.e., capitalizing on shared information across individuals) in addition to person-specific features, GIMME overcomes limitations of other approaches in which low signal-to-noise ratio induces unreliable estimates at the individual level (Gates & Molenaar, 2012; Smith et al., 2011).

GIMME begins by estimating 8 autoregressive terms (i.e., lagged prediction of each ROI by itself) in each participants' network (Beltz & Gates, 2017; Friston et al., 2000; Woolrich et al., 2001). Lagrange multiplier tests are then used to identify connections to estimate that are statistically meaningful at the group-level (i.e., significantly improve model fit for 75% of the sample). Next, Lagrange multiplier tests are again used to free connections that are statistically meaningful at the individual-level (i.e., significantly improve model fit for a given participant). Additionally, at several points during model fitting, non-significant connections are pruned if their influence changed with the addition of new connections (Gates & Molenaar, 2012). Model building ends when the network fits the data well, and final maps are evaluated with alternative fit indices, with two of four required to attain excellent fit (Brown, 2014): root mean squared error of approximation (RMSEA)$\leq$.05, standardized root mean residual (SRMR)$\leq$.05, comparative fit index (CFI)$\geq$.95, and non-normed fit index (NNFI)$\geq$.95.

Notably, GIMME produces models with first order lagged connections, meaning that the lagged connections represent estimates at one time point prior (i.e., one functional volume earlier), with the assumption that the model residuals are white noise (i.e., all temporal information is captured by the modeled connections). To verify this assumption, we submitted individual-level models to *a posteriori* validation (as described in Beltz & Molenaar, 2015). If the validation process indicates that the first order connections did not sufficiently capture all sequential dependencies in each participant's data (according to white noise tests), then higher order lagged connections (i.e., estimates at two or three functional volumes prior) were added to the model. Previous directed functional connectivity studies demonstrate that this is important for accurately modeling all connections in the network, especially in resting state data (Beltz & Molenaar, 2015).

. White noise tests were carried out in LISREL (Joreskog & Sorbom, 1992). The tests were evaluated with the same alternative fit indices and criteria as the final models. When residuals were not white noise, the person-specific uSEM (the type of model implemented by GIMME) was expanded to include a second level order (i.e., lagged connections estimate at two TRs prior). The automated model fitting preceded as described above and model fit was evaluated using the same criteria. If residuals were still not white noise, the person-specific uSEM was expanded to add third order connections to individual-level models prior to model fitting. White noise tests were then repeated. At the end of this process, a subset of participants' models (n=22) met standards of acceptable fit (i.e., only one index met criteria for excellent fit) at the third order level.

Network features. We extracted several features from the final networks to characterize person-specific patterns of resting-state connectivity. To account for individual differences in total number of connections, we used proportions. We calculated separate indices of positive and negative features. Whereas positive connections are expected among brain regions within the same network, previous theory and empirical studies suggest that the DMN and task-positive networks such as the CEN are inherently anticorrelated, which leads to negative connections (Chai et al., 2012; Fox et al., 2005; Kelly et al., 2008).

Network density. For each participant, we calculated within-network density (i.e., number of connections between nodes within a network, regardless of whether they were contemporaneous or lagged) separately for the DMN, SN, and CEN. We similarly calculated between-network density: DMN-SN, DMN-CEN, and SN-CEN.

Node centrality. As exploratory analyses, we calculated node centrality for each participant (i.e., number of connections involving the node, regardless of whether they were

contemporaneous or lagged) to determine whether any regions were "hub-like" (i.e., high number of connections to and/or from this region,), as in previous studies that have examined network organization (Lindner et al., 2018; Lu et al., 2017; Tillem et al., 2019; Tillem et al., 2018; Yang et al., 2012).

Associations between network features and psychopathic traits. To examine associations between psychopathic traits and neural connectivity, we ran a multiple regression for each index of network density, including both positive and negative connections, which were correlated dependent variables. Total psychopathy score was the predictor. Participant self-reported race, substance use (mean score on Alcohol and Drug Consumption Questionnaire; Cahalan et al., 1969), monthly income, and framewise displacement (after motion correction) were covariates.

To examine associations between psychopathic traits and node centrality, we ran a multiple regression model separately for each network to determine if psychopathic traits were associated with node centrality, with positive and negative centrality of each node within a network included as correlated dependent variables. Total psychopathy score was the predictor, and the same covariates were included.

We used the Bonferroni method to correct for multiple comparisons across the five models we ran (i.e., within network density, between network density, SN node centrality, DMN node centrality, CEN node centrality; $p = .05/5 = .01$).

We followed up significant effects to determine whether associations with the total score were driven by the interpersonal, affective, lifestyle or antisocial facets. As these were exploratory analyses, we did not apply a Bonferroni correction. Finally, as noted previously, an advantage of GIMME is the inclusion of both contemporaneous and lagged connections, and so

exploratory follow-up analyses examined whether significant associations were driven by contemporaneous or lagged connections.

Results

Descriptives

Descriptives are presented in Supplemental Table 4.2. Of note, in general the means of the SRP-SF in the current sample were slightly higher than or comparable to those found in previous community samples (Gordts et al., 2017; Paulhus et al., 2015; Seara-Cardoso et al., 2019). However, the mean on the criminal tendencies facet was higher than previous community samples, indicating that this sample was indeed at somewhat higher risk than typical community samples (Gordts et al., 2017; Paulhus et al., 2015; Seara-Cardoso et al., 2019).

Person-Specific Network Modeling

Final GIMME networks generally fit the data well (Average Fit: RMSEA=.04, SRMR=.05, CFI=.97, NNFI=.94). There were no group-level connections, indicating substantial heterogeneity across participants. There were between 11 and 27 individual-level connections (*M*=17.85, *SD*=3.76). All models contained positive connections (*M*=14.35, *SD*=2.70) and most models (93%) contained negative connections (*M*=3.50, *SD* =2.24). Additionally, all models contained both contemporaneous and lagged connections (*M*=6.89, *SD* =2.60; *M*=9.82, *SD* =1.54, respectively). *A posteriori* model validation determined that a first order model fit the data well for 67 participants (54.5% of the sample), but that 16 participants (13%) required lag 2 connections and 40 participants (32.5%) required lag 3 connections.

Figure 4.1 depicts models from six illustrative participants to highlight the heterogeneity of networks across individuals. For instance, Participant A had primarily positive contemporaneous connections (one negative contemporaneous connection; no lagged

connections beyond the 8 auto-regressives; Figure 4.1A). Most were between the SN and CEN and between the DMN and CEN, as well as one connection between the DMN and SN. In contrast, Participant B had positive and negative contemporaneous connections, as well as one positive lagged connection (Figure 4.1B). There were several (primarily negative) connections between the SN and CEN and one positive connection between the DMN and CEN, but no connections between the DMN and SN. Participants C and D had relatively sparse maps (i.e., a few connections were statistically meaningful to their networks) and appear structurally similar, revealing how homogeneity (if it exists) can be captured in these person-specific networks. Participants E and F have networks that required the inclusion of second- and third-order lags, respectively, based on *a posteriori* validation (i.e., activation in a region two or three functional volumes prior predicted current activation). Qualitatively, these six networks exemplify significant heterogeneity; in fact, the same connection was not present across all six networks. However, the networks also demonstrate some homogeneity with respect to specific connections (e.g., four participants had a positive connection between the right insula and ACC) and patterns of connectivity (e.g., there were more connections between the SN and CEN than between the DMN and SN).

Associations Among Resting-State Networks and Psychopathic Traits

Total psychopathy. Total psychopathy was associated with increased positive network density (i.e., more positive connections) between the DMN and CEN (B=.28, *p*=.003) (Figure 4.2; Supplemental Table 4.4). Additionally, total psychopathy was associated with increased positive node centrality (B=.21, *p*=.02) and decreased negative node centrality (B=-.23, *p*=.02) of the PCC (within the DMN); however, these associations did not withstand the Bonferroni correction (Supplemental Table 4.6). There were no other significant associations between total

psychopathic traits and network features. In a set of exploratory analyses, we found that greater levels of psychopathy were specifically associated with more positive contemporaneous connections between the DMN and CEN (B=.26, *p*=.005).

Interpersonal, affective, lifestyle, and antisocial facets. Although all four facets of psychopathy were significantly associated with increased positive network density between the DMN and CEN in zero-order correlations (interpersonal: *r*=.18, *p*=.049; affective: *r*=.29, *p*=.001; lifestyle: *r*=.23, *p*=.012; antisocial: *r*=.24, *p*=.008), when accounting for their overlap in the regression model, only the association with affective traits was significant (B=.28, *p*=.049; Supplemental Table 4.9; Figure 4.2). In fact, networks in Figure 4.1 demonstrate these differences, as Participant A was high in affective traits and had several positive connections between the DMN and CEN, whereas Participant B was high in lifestyle traits and only had one such connection.

Additionally, in the zero-order correlations, the affective facet was associated with increased positive PCC centrality (*r*=.22, *p*=.013), and the lifestyle facet was associated with reduced negative PCC centrality (*r*=-.21, *p*=.022). However, none of the four facets were associated with either positive or negative PCC density in the regression model (accounting for overlap among facets; Supplemental Table 4.10). Finally, in a set of exploratory analyses, we found that there were only significant associations between the facets and *lagged* positive connections between the DMN and CEN. Interpersonal features were significantly associated with fewer positive lagged connections between the DMN and CEN (B=-.43, *p*<.000). Affective features were significantly associated with more positive lagged connections between the DMN and CEN (B=.39, *p*=.003). Antisocial features were significantly associated with more positive lagged connections between the DMN and CEN (B=.31, *p*=.018). Lifestyle features were not

significantly associated with positive lagged connections (B=-.16, *p*=.064). Consistent with a discovery science approach, these findings should encourage future hypothesis-drive work.

Discussion

Core to major etiologic theories of psychopathy is the notion that disconnection between the DMN, SN, and CEN underlie emotion and attention deficits. Here we find that men from a low-income, ethnically diverse community sample had significant heterogeneity in resting state connectivity networks using a sparse, person-specific network approach. Yet, there was consistency in connectivity patterns, such that psychopathy was associated with increased positive density in connections between the DMN and CEN, somewhat fitting with the II theory. Exploratory analyses indicated that this association may be driven by affective features of psychopathy. There was also suggestion that psychopathy was associated with increased positive and decreased negative node density of the PCC, a node of the DMN.

Neural networks were person specific. In fact, there were *no* connections among ROIs that were common across participants. This may seem unsurprising because it is consistent with previous research that has applied GIMME to samples marked by significant heterogeneity (e.g., mixed gender sample with varying levels of psychiatric comorbidities; Dotterer et al., 2019). However, it emphasizes the dangers of relying on averaging approaches (i.e., relying on combining neural metrics across individuals) that dominate the extant literature (i.e., they mask important individual differences in neural mechanisms). Moreover, as demonstrated by the illustrative networks in Figure 4.1, all participant networks contained numerous contemporaneous *and* lagged connections at only the individual level, as there were no estimated group-level connections. Additionally, *a posteriori* validation revealed that a number of participants' network models required higher order lagged connections. These nuanced network

features would not have been captured using traditional network approaches that only model contemporaneous connections at the group- or individual-level or that fail to conduct *a posteriori* validation. Thus, the network features utilized in the current study are likely robust, as they were derived from accurate, person-specific neural networks (supported by large-scale simulation studies of GIMME; Gates & Molenaar, 2012).

Findings were partially consistent with II theory. Psychopathic traits were only associated with positive DMN-CEN connectivity, but not connectivity within or between the DMN and SN. Further, it is unclear if positive connectivity is posited by the II theory, which merely suggests that there will be differences in connectivity among the three networks. Findings with our network-focused approach significantly extend past work that used seed-based connectivity approaches, in which psychopathic traits were associated with positive connectivity between *regions* within the CEN and DMN (Espinoza et al., 2018), by showing that psychopathy is linked to the organization and functioning of the DMN and CEN *broadly.* Importantly, the DMN and CEN are typically anticorrelated; the CEN is activated during effortful cognitive tasks, whereas the DMN is activated at rest and during self-referential thinking (Buckner et al., 2008). As such, increased communication (and less segregation) between the DMN and CEN reflected in positive DMN-CEN density may interfere with higher-order cognitive processes that involve both networks, such as decision-making and theory of mind, which appear to be impaired in individuals with psychopathic traits (Hamilton et al., 2015). Moreover, hyperconnectivity between the DMN and other networks, including the CEN, has been observed in individuals with other disorders marked by social cognitive deficits (autism spectrum disorder; Ecker et al., 2015; e.g., schizophrenia; Hu et al., 2017). Thus, further work should examine DMN-CEN connectivity during tasks that require the integration of contextual information during decision-making or

goal-directed behavior (e.g., Larson et al., 2013). Future studies could therefore determine whether increased positive DMN-CEN connectivity reflects hyper-focus on goal attainment and internal cues and inflexibility in behavior at the expense of attending to important environmental cues as proposed by II theory.

This study is also novel in showing that the association between psychopathy and DMN-CEN connectivity may be driven by affective features of psychopathy, when accounting for their overlap with the other facets (i.e., interpersonal, lifestyle, antisocial). In contrast to the lifestyle and antisocial features, affective and interpersonal features have been associated with unimpaired or even heightened basic attention abilities (i.e., better attentional control, better response inhibition, and increased error monitoring), but deficits in flexibly using contextual information to modulate attention (Baskin-Sommers et al., 2012; Hoppenbrouwers et al., 2015; Sadeh & Verona, 2008; Veit et al., 2013). As such, individuals who predominantly display affective features may uniquely benefit from intervention targeted towards attention to context (Baskin-Sommers et al., 2015). Although some studies using traditional connectivity methods have similarly identified unique associations between affective features and DMN-CEN connectivity (Espinoza et al., 2018), this pattern has not been consistently replicated (Contreras-Rodríguez et al., 2015; Philippi et al., 2015; Pujol et al., 2012), potentially because those studies varied in sample characteristics (i.e., community versus clinical, gender) and failed to account for person-specific heterogeneity. Notably, this finding did not withstand Bonferroni correction and thus requires replication in future studies. Taken together, further research is needed to clarify the extent to which psychopathic facets are characterized by unique patterns of resting-state connectivity.

Heightened psychopathic traits were also associated with increased positive and decreased negative PCC node centrality, although these associations may not be robust (i.e., did not withstand Bonferroni correction). The PCC, a key node of the DMN, typically deactivates on cognitive tasks but activates during self-referential processing, future thinking, and mentalizing (Brewer et al., 2013; Leech & Sharp, 2014; Pearson et al., 2011). Further, the PCC appears to be involved in attention modulation, with patterns of activation differing for internally (increased activation) versus externally (decreased activation) directed attention (Brewer et al., 2013; Leech & Sharp, 2014; Pearson et al., 2011). More positive connections and fewer negative connections involving the PCC suggest that, in individuals with psychopathic traits, PCC activation is more activated and less inhibited by regions in other networks (consistent with other work; Contreras-Rodríguez et al., 2015; Motzkin et al., 2011; Philippi et al., 2015; Pujol et al., 2012), potentially reflecting higher propensity for self-referential thoughts and internally-directed attention (i.e., heightened "self-focus"). However, further research with larger samples is needed to replicate this likely small association.

Psychopathic traits were not associated with resting-state connectivity within or between the SN and DMN, in contrast to II theory. Previous studies using traditional methods (Philippi et al., 2015) and network modeling (Lindner et al., 2018; Tillem et al., 2019) have also not consistently found these associations. Thus, emotion and attention impairments observed in psychopathy may not be driven by abnormal communication among regions within the SN or DMN themselves, but instead reflect an imbalance with other networks, such as the CEN. Importantly, the focus in this study was on accurate individualized resting-state networks, whereas II theory is based on findings derived using traditional averaging approaches, which are known to create spurious results (Molenaar, 2004). Thus, these null findings may partially

undermine the hypotheses of II theory. It is also possible that psychopathic traits are associated with network features involving nodes other than those included in the current study (Espinoza et al., 2018; Philippi et al., 2015), which were identified *a priori* based on previous work (Chiong et al., 2013; Goulden et al., 2014; Sridharan et al., 2008). For instance, psychopathic traits may be associated with connectivity among other regions in these networks, particularly within the DMN (e.g., dorsal medial prefrontal cortex, temporoparietal junction), which includes subsystems that appear to underlie distinct social cognitive processes (Andrews-Hanna et al., 2010; Li et al., 2014). Thus, future research would benefit from the inclusion of additional nodes from the SN, DMN, and CEN to better understand connectivity within psychopathy at a systems-level.

Strengths and Limitations

The current study had several strengths, including a low-income, racially diverse community sample at heightened risk for antisocial behavior, and a novel mapping approach that has been demonstrated to accurately model neural networks by capturing both homogeneity (if it exists) and heterogeneity (Gates & Molenaar, 2012). However, there are limitations. The sample size was reduced because of data loss, which diminished the power of the analyses. Additionally, results may not be generalizable to populations characterized by extreme levels of psychopathic traits, antisocial behavior, or other comorbid psychopathology, including offender or clinical populations. Moreover, as resting-state networks change across development, the current results cannot be generalized to younger (e.g., children) or older populations. Additionally, participants reported on their own psychopathic traits, potentially leading to biased responses, although research has not been able to substantiate associations between psychopathy and response style (Ray et al., 2013). Finally, caution should be used in interpreting negative connections. Previous

resting-state studies of psychopathy focused on positive connections owing to debate surrounding negative connections (Lindner et al., 2018; Rubinov & Sporns, 2010). However, GIMME only models connections that are statistically meaningful in a network. Thus, negative connections are unlikely to be a statistical property of time-series data, and instead likely reflect inhibition.

Conclusions

In a low-income, racially diverse community sample of young men with a wide range of psychopathic traits, we found significant heterogeneity in neural network connectivity; in fact, there were *no connections* common across all participants. This finding emphasizes the limitations of traditional averaging approaches in understanding neural mechanisms underlying psychopathy. Although individuals had unique neural networks, there were key network features across the sample that were associated with psychopathic traits. Psychopathic traits were associated with patterns in person-specific networks, such as connectivity between the DMN and CEN and the integration of the PCC in the networks. Exploratory analyses suggest that affective features in particular may be characterized by DMN-CEN hyperconnectivity. Taken together, our findings demonstrate how person-specific approaches can be used to capture variability in biopsychosocial profiles, including neural mechanisms, that give rise to similar behaviors, which could ultimately inform individualized treatment efforts (Baskin-Sommers et al., 2015; Insel, 2014).

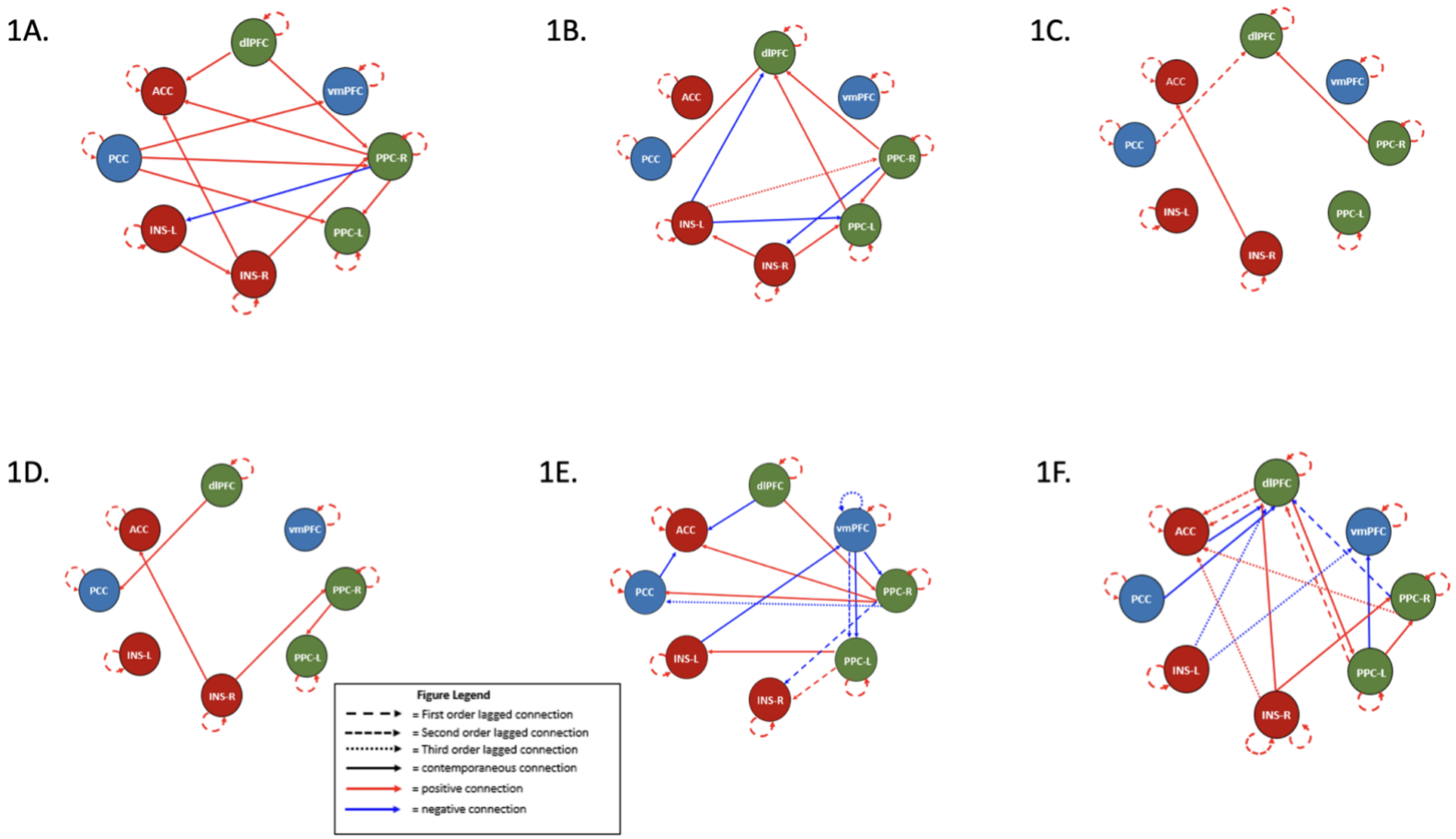

Figure 4.1: Final GIMME networks for six illustrative participants.
Solid lines depict contemporaneous connections and dashed lines depict lagged connections. Regions in red are within the salience network. Regions in blue are within the default mode network. Regions in green are within the central executive network. There was no group-level structure; thus, all lines depict individual-level connections (uniquely estimated for the participant) that also have associated β weights. Red lines depict connections with positive β weights and blue lines depict connections with negative β weights. 1A.) Participant with high levels of affective features (score = 23; Range in the sample 7 – 25); x^2 (73) = 106.06, RMSEA= .05,

SRMR= .05, CFI= .96, NNFI = .95. 1B.) Participant with high levels of lifestyle features (score = 23; Range in the sample 7 – 28); $x^2(75)$= 105.77, RMSEA= .05, SRMR= .05, CFI= .96, NNFI = .94. 1C.) Participant with first order model; $x^2(81)$ = 111.75, RMSEA= .05, SRMR= .06, CFI= .95, NNFI = .93. 1D.) Participant with first order model; $x^2(80)$ = 102.50, RMSEA= .04, SRMR= .06, CFI= .96, NNFI = .95. 1E.) Participant with second order model; $x^2(13)$ = 186.84, RMSEA= .04, SRMR= .05, CFI= .96, NNFI = .91. 1F.) Participant with third order model; $x^2(19)$ = 259.25, RMSEA= .04, SRMR= .04, CFI= .97, NNFI = .92. dlPFC = dorsolateral prefrontal cortex; vmPFC = ventromedial prefrontal cortex; PPC-R = right posterior parietal cortex; PPC-L = left posterior parietal cortex; INS-R = right insula; INS-L = left insula; PCC = posterior cingulate cortex; ACC = anterior cingulate cortex.

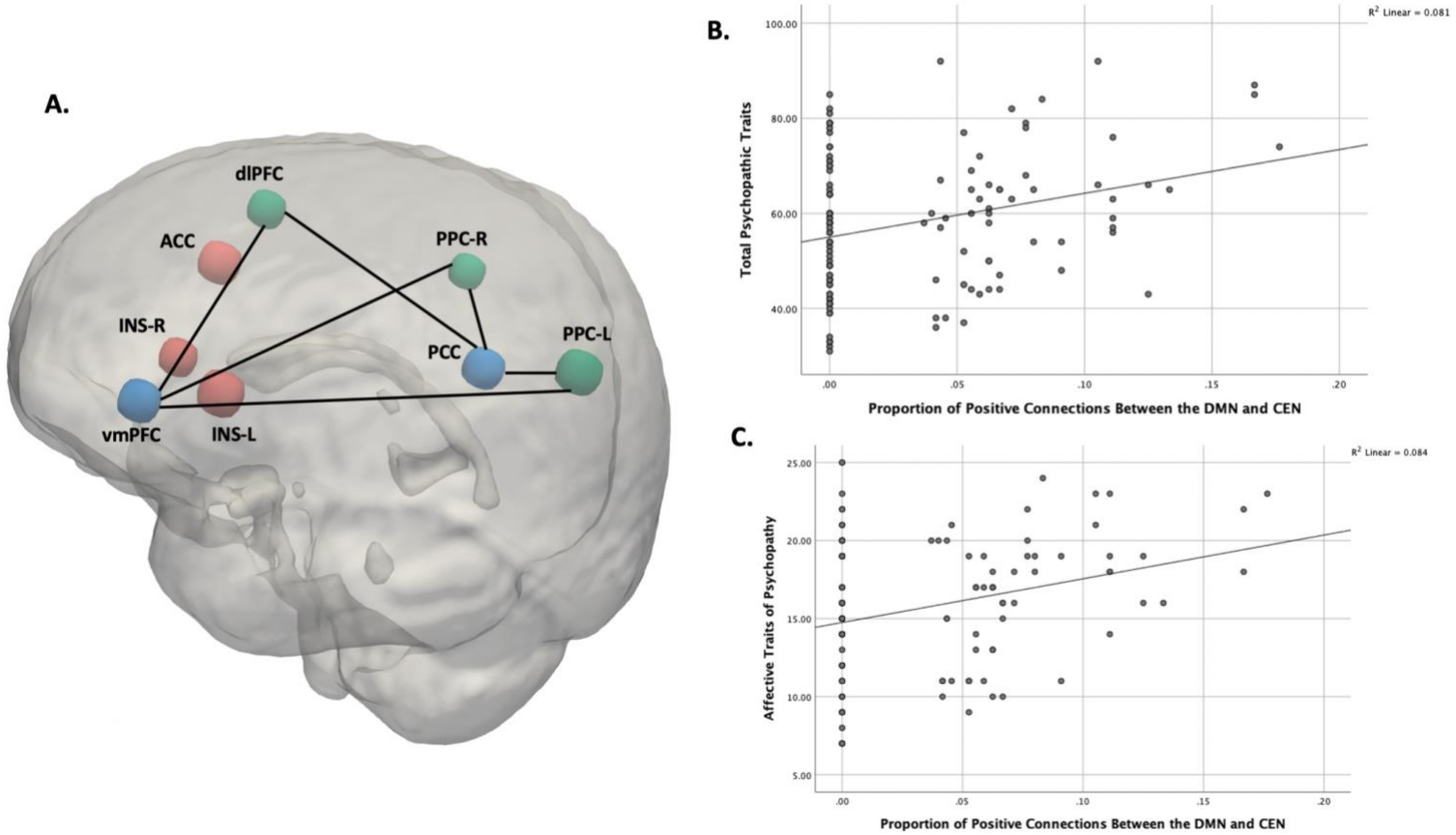

Figure 4.2: Psychopathic traits were associated with increased positive density between the default mode network and central executive network across all participants.

2A.) Schematic depiction of possible connections between the default mode network and central executive network regions of interest. Regions in red are within the salience network. Regions in blue are within the default mode network. Regions in green are within the central executive network. 2B.) Scatter plot represents bivariate correlations between *total psychopathic traits* and positive density between the DMN and CEN for each participant. 2C. Scatter plot represents bivariate correlations between *affective traits* of

psychopathy and positive density between the DMN and CEN for each participant. DMN= default mode network. CEN = central executive network. dlPFC = dorsolateral prefrontal cortex; vmPFC = ventromedial prefrontal cortex; PPC-R = right posterior parietal cortex; PPC-L = left posterior parietal cortex; INS-R = right insula; INS-L = left insula; PCC = posterior cingulate cortex; ACC = anterior cingulate cortex.

Supplemental Table 4.1: Sources of data loss resting-state functional connectivity data

	Numbers
Original sample	310
Sample with behavioral data at age 22	255
- Parent requested drop out	5
- Target youth requested drop out	6
- Incarcerated	8
- In the military	2
- Deceased	2
- Unable to locate	11
- Hard to contact/probable drop outs	20
- Target youth refused age 22 visit only	1
Total lost	55
Sample with imaging data at age 22	180
- Concussion/head injury	32
- Bullets/metal fragments	17
- Braces	2
- Phone interviews (out of the area)	4
- Refused MRI portion of the visit	7
- Living at home/treatment facility (too ill to participate – schizophrenia, autism, car accident)	2
- Claustrophobic	6
- Left before scanning portion/wanted to stop scan	1
- Did not physically fit in the bore	1
- Taking a stimulant medication	3
Total lost	75
Sample with resting-state imaging data at age 22	126
- Participants scanned prior to resting-state scans added to protocol	51
- Terminated scan before resting-state scan	3
Total lost	54
Participants with usable resting-state fMRI data	126

Supplemental Table 4.2: Descriptive statistics of psychopathic traits

Scale	Minimum	Maximum	Mean	SD
Total Score	31	92	58.16	14.29
Interpersonal Manipulation	7	25	12.77	4.60
Affective Callousness	7	25	15.71	4.28
Erratic Lifestyle	7	28	16.33	4.29
Criminal Tendencies	8	28	13.35	4.36

Note. SD = Standard deviation. N = 123 participants.

Supplemental Table 4.3: Central coordinates of regions of interest

Region of Interest	Central Coordinates
Default Mode Network	
vmPFC right/left	-2 36 -10
PCC right/left	-7 -43 33
Salience Network	
Frontal insula right	37 25 -4
Frontal insula left	-32 34 -6
ACC right/left	4 30 30
Central Executive Network	
dlPFC right	45 16 45
PPC right	54 -50 50
PPC left	-38 -53 45

Note. vmPFC = ventromedial prefrontal cortex. PCC = posterior cingulate cortex. ACC= anterior cingulate cortex. dlPFC = dorsolateral prefrontal cortex. PPC = posterior parietal cortex. Coordinates in MNI space. Central coordinates acquired from (Sridharan, Levitin, & Menon, 2008).

Supplemental Table 4.4: Associations among psychopathic traits and between-network density, controlling for demographic factors and motion.

	DMN-SN Positive Density			DMN-SN Negative Density			DMN-CEN Positive Density			DMN-CEN Negative Density			SN-CEN Positive Density			SN-CEN Negative Density		
	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2
Income	.06	.10		-.11	.07		.21	.10		.07	.09		.10	.09		-.03	.07	
Drug Use	.08	.10		.00	.08		.01	.10		-.05	.08		.14^{+}	.08		.15	.09	
White	-.28	.23	.08	.24^{+}	.13	.06^{+}	-.04	.13	.13*	-.13	.09	.03	-.14	.15	.03	.13	.20	.08
AA	-.19	.23		.35*	.14		.00	.13		-.01	.10		-.10	.15		.06	.20	
Motion	.22*	.09		-.03	.09		-.02	.07		-.01	.09		.07	.08		.23*	.09	
Total psychopathy	-.04	.09		-.13	.10		.28**	.09		-.05	.08		-.03	.08		-.13	.10	

Note. $^{+}p<.10$, $*p<.05$, $**p<.01$, $***p<.001$. DMN = default mode network. SN = salience network. CEN = central executive network. AA= African American.

Supplemental Table 4.5: Associations among psychopathic traits and within-network density, controlling for demographic factors and motion.

	DMN Positive Density			DMN Negative Density			SN Positive Density			SN Negative Density			CEN Positive Density			CEN Negative Density		
	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2
Income	-.12	.09		.10	.10		-.06	.08		.02	.09		-.22**	.09		.02	.09	
Drug	-.14	.10		.06	.09		-.15^{+}	.07		-.08	.09		-.05	.10		-.06	.08	
Use			.10*			.05			.04			.03			.10^{+}			.05
White	-.12	.20		.14*	.07		-.16	.18		-.23	.26		.30*	.12		-.04	.21	
AA	-.26	.20		.31***	.08		-.23	.17		-.19	.26		.21^{+}	.12		-.17	.20	
Motion	-.22**	.08		-.02	.08		-.06	.09		.11	.13		-.14^{+}	.08		-.16*	.08	
Total psychopathy	.05	.09		-.08	.10		.09	.10		.09	.09		-.04	.09		.11	.10	

Note. ^{+}p<.10, *p<.05, **p<.01, ***p<.001. DMN = default mode network. SN = salience network. CEN = central executive network. AA= African American.

Supplemental Table 4.6: Associations among psychopathic traits and DMN node centrality, controlling for demographic factors and motion.

	vmPFC Positive Centrality			vmPFC Negative Centrality			PCC Positive Centrality			PCC Negative Centrality		
	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2
Income	-.03	.11		.01	.08		-.08	.09		.03	.07	
Drug Use	-.02	.09		-.04	.09		-.09	.10		.07	.08	
White	-.50**	.18	.07	-.01	.18	.01	.11	.19	.05	.21*	.10	.09^{+}
AA	-.56**	.18		.06	.19		.13	.19		.39***	.10	
Motion	-.06	.07		-.04	.09		-.05	.08		.04	.06	
Total psychopathy	.08	.11		-.01	.10		.21*	.09		-.23*	.10	

Note. $^{+}p<.10$, *$p<.05$, **$p<.01$, ***$p<.001$. DMN = default mode network. vmPFC= ventromedial prefrontal cortex. PCC = posterior cingulate cortex. AA= African American.

Supplemental Table 4.7: Associations among psychopathic traits and SN node centrality, controlling for demographic factors and motion.

	ACC Positive Centrality			ACC Negative Centrality			Insula-L Positive Centrality			Insula-L Negative Centrality			Insula-R Positive Centrality			Insula-R Negative Centrality		
	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2
Income	.13	.11		.02	.07		-.07	.09		-.09	.09		-.07	.08		-.03	.09	
Drug Use	.00	.10		-.06	.08		.08	.09		.07	.08		-.11	.10		.06	.11	
White	-.65***	.13	.11*	-.17	.21	.04	.13	.17	.02	.23^{+}	.12	.07^{+}	.14^{+}	.19	.04	.10	.17	.01
AA	-.63***	.13		-.13	.21		.14	.17		.28*	.13		.13	.19		.07	.17	
Motion	.06	.11		.16^{+}	.09		-.08	.07		.20**	.08		.14	.09		-.07	.07	
Total psychopathy	.08	.10		-.02	.09		.03	.10		-.10	.10		-.02	.09		-.04	.09	

Note. $^{+}p<.10$, $*p<.05$, $**p<.01$, $***p<.001$. SN = salience network. ACC= anterior cingulate cortex. L = left. R = right. AA= African American.

Supplemental Table 4.8: Associations among psychopathic traits and CEN node centrality, controlling for demographic factors and motion.

	dlPFC Positive Centrality			dlPFC Negative Centrality			PPC-L Positive Centrality			PPC-L Negative Centrality			PPC-R Positive Centrality			PPC-R Negative Centrality		
	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2	B	SE	R^2
Income	.01	.07		-.13^{+}	.07		-.01	.10		.21^{+}	.11		-.17*	.08		.03	.10	
Drug Use	.02	.09		-.14	.09		.10	.12		-.01	.09		-.06	.10		.18*	.09	
White	.16	.15	.01	-.09	.20	.06	.08	.22	.03	.14	.20	.07	.20	.14	.07	-.09	.11	.03
AA	.10	.15		-.21	.20		.10	.23		.10	.20		.14	.14		-.10	.12	
Motion	-.04	.09		.08	.09		.04	.07		-.01	.08		-.18*	.08		-.04	.07	
Total psychopathy	-.04	.10		-.09	.10		.08	.11		.14	.10		.01	.08		-.12	.11	

Note. $^{+}p<.10$, *$p<.05$, **$p<.01$, ***$p<.001$. CEN = central executive network. dlPFC = dorsolateral prefrontal cortex. PPC = posterior parietal cortex. L = left. R = right. AA= African American.

Supplemental Table 4.9: Associations among interpersonal features, affective features, lifestyle features, antisocial features, and DMN-CEN positive density, controlling for demographic factors and motion.

	DMN-CEN Positive Density		
	B	SE	R^2
Income	.22	.10	
Drug Use	-.01	.09	
White	-.02	.12	.14*
AA	-.01	.13	
Motion	-.02	.08	
Interpersonal	-.07	.15	
Affective	.28*	.14	
Lifestyle	.03	.13	
Antisocial	.11	.12	

Note. $^{+}p<.10$, $*p<.05$, $**p<.01$, $***p<.001$. DMN = default mode network. CEN = central executive network. AA= African American.

Supplemental Table 4.10: Associations among interpersonal features, affective features, lifestyle features, antisocial features, and PCC negative centrality, controlling for demographic factors and motion.

	PCC Positive Centrality			PCC Negative Centrality		
	B	SE	R^2	B	SE	R^2
Income	-.09	.08		.05	.07	
Drug Use	-.13	.10		.08	.08	
White	.10	.16	.05	.23*	.11	.09^{+}
AA	.12	.17		.38***	.11	
Motion	-.04	.08		.04	.06	
Interpersonal	-.11	.15		-.11	.12	
Affective	.22	.14		.03	.14	
Lifestyle	.15	.10		-.18	.12	
Antisocial	.03	.11		-.04	.10	

Note. $^{+}p<.10$, $*p<.05$, $**p<.01$, $***p<.001$. PCC = posterior cingulate cortex. AA= African American.

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Chapter 5: General Discussion

As discussed in the General Introduction (Chapter 1), youth AB is a significant public health concern, and is extremely harmful to individuals, families, and society (McCart et al., 2006; Rivenbark et al., 2018). Understanding etiologically distinct subtypes of AB, such as the presence of CU traits, which are associated with more severe and stable AB, may help inform more effective intervention approaches (Frick et al., 2014). Thus, the aim of the current dissertation was to specify developmental trajectories and neurocognitive processes associated with AB versus CU traits using multiple perspectives.

Summary of Results

Study 1. In Study 1, I found that both maternal and paternal interpersonal-affective features were associated with parenting practices and adolescent CU traits in a large population-based sample that included multiple informants (mother, father, child) and utilized more precise measurements of parenting and adolescent CU traits. Moreover, increased harsh parenting and reduced involvement partially explained associations between interpersonal-affective traits and adolescent CU traits for both mothers and fathers. There was also a significant direct effect specifically between maternal interpersonal-affective traits and adolescent CU traits. Expanding on previous studies, we demonstrated that associations identified within informants were not robust across different informants (particularly child report). Additionally, by using a genetically informed design, we found that the differences between levels of CU traits in MZ twins was, at least in part, attributable to parenting (a nonshared environmental factor), controlling for the possibility of a passive or evocative rGE. The findings thus provide further evidence that 1) CU traits are not entirely attributable to genetic risk and 2) that parenting significantly impacts child outcomes via environmental mechanisms, while also demonstrating that parent personality can

influence parenting practices. As such, considering both parent personality and parenting practices are likely critical to designing effective intervention strategies targeting CU traits (Waller, Gardner, & Hyde, 2013).

Study 2. In Study 2, based on performance on multiple neuropsychological tasks of response inhibition and attention, I identified distinct neurocognitive profiles for AB with and without CU traits in a large, population-based sample of adolescents. Specifically, there were unique neurocognitive profiles for the combination of elevated AB and CU traits (better sustained attention) compared to elevated AB at low levels of CU traits (worse sustained attention). Executive function deficits associated with broader AB may therefore contribute to impulsive and reactive aggression, whereas the distinct neurocognitive profile associated with AB + CU traits may underlie strategic planning and implementation involved in instrumental and premeditated aggression. Notably, I did not find any direct associations between either AB or CU traits and indices of neurocognitive functioning when accounting for demographic factors. The findings add to the accumulating literature suggesting that the combination of AB and CU traits is etiologically distinct from AB alone and emphasize the need for further research in representative samples able to incorporate key demographic and individual differences variables (i.e., gender, socioeconomic status).

Study 3. In Study 3, utilizing a novel person-specific neural network mapping approach, I found that men from a low-income, ethnically diverse community sample had significant heterogeneity in resting state connectivity networks, emphasizing the limitations of traditional averaging approaches in understanding neural mechanisms underlying psychopathy. When looking across participants, psychopathic traits were associated with increased positive density in connections between the DMN and CEN. Exploratory analyses indicated that this association

may be driven by affective features of psychopathy. These results were somewhat consistent with the predictions of the II theory. DMN-CEN hyperconnectivity may interfere with higher-order cognitive processes that include affective components (i.e., decision-making, theory of mind), which often appear impaired in individuals with psychopathic traits (Hamilton et al., 2015). Additionally, psychopathic traits in general were also associated with PCC node centrality, suggesting that the PCC is more activated and less inhibited by regions in other networks (consistent with other work; Contreras-Rodríguez et al., 2015; Motzkin et al., 2011; Philippi et al., 2015; Pujol et al., 2012). In sum, the findings of Study 3 support the notion that psychopathic traits are associated with network connectivity and organization of the DMN and CEN. However, these results also expand prior work by emphasizing the importance of first accurately modeling neural networks and demonstrating that these associations seem to be specific to the affective features of psychopathy.

Implications

Taken together, the results of the three studies build on the existing literature that youth who demonstrate AB are a heterogeneous group, particularly youth with elevated CU traits. Identifying specific developmental risk factors and neurocognitive processes associated with AB and CU traits can be used to inform individualized treatment of AB and CU traits (Brazil et al., 2016). The findings of Study 1 highlighted developmental risk factors for CU traits, including mechanisms of intergenerational transmission of psychopathic traits via parenting practices. Parenting practices are therefore likely an important target for intervention in the treatment of youth with AB and CU traits. Indeed, a narrative review of the treatment literature found that youth with high levels of CU traits are responsive to parenting interventions, specifically warm parenting (Waller et al., 2013).

Studies 2 and 3 identified unique neurocognitive profiles associated with AB and CU/psychopathic traits, which also implicate unique targets of treatment. Study 2 found that AB with low levels of CU traits was associated with poor sustained attention. Thus, youth with AB but low levels of CU traits may specifically benefit from treatments that emphasize strategies to increase cognitive control, as opposed to youth with AB and high levels of CU traits. Moreover, in Study 3, higher levels of adult affective psychopathic traits, which most directly overlap with CU traits, were associated with neural network organization, which may contribute to distinct cognitive deficits. This notion has been supported by previous literature, which suggests that youth with AB and CU traits are characterized by insensitivity to punishment, abnormal moral reasoning, and reduced responsiveness to others' distress (Frick et al., 2014). As such, youth with AB and CU traits may benefit from treatments that emphasize attention to context, such as emotional cues. Indeed, there is preliminary support for subtype-specific intervention approach in adult offenders (affective cognitive control training for individuals high in AB alone; attention to context training for individuals with psychopathic traits), such that individuals who received the intervention targeted to their cognitive deficiencies exhibited improvements on task performance over the six-week training period (Baskin-Sommers et al., 2015).

Addressing both parenting and cognitive processes may therefore result in more effective prevention and treatment approaches for youth with AB and CU traits. For instance, some interventions originally developed to treat AB have been modified to target CU traits (Parent Child Interaction Therapy; Hembree-Kigin & McNeil, 2013; Coping Power; Muratori et al., 2017; Common Elements Co-Parent Training Program; Somech & Elizur, 2012b). Based on research that has identified multiple risk factors for AB from a variety of domains (e.g., individual-level, family, peer, and school factors; Farrington et al., 2010), treatments typically

include parents and child specific components, and emphasize reduced association with deviant peers (McCart & Sheidow, 2016). Researchers have also developed interventions that were created specifically to reduce CU traits by improving emotion recognition and emotion processing, increasing positive affect, and encouraging empathic and prosocial behaviors (Emotion Recognition Treatment; Dadds et al., 2012; CARES; Datyner et al., 2016; Salekin et al., 2012).

Notably, empirical support for these approaches is limited and further research is needed to evaluate their effectiveness. However, across these interventions, it appears that youth with AB+CU+ benefit from parent management strategies in particular, which reduce AB symptoms within these children. Even in instances when CU traits are not influenced by treatment, reducing AB symptoms is nevertheless important for improving overall outcomes and reducing parental distress. Results across treatments support previous research indicating that increasing positive parenting and reducing ineffective parenting can improve levels of CU traits (e.g., Kimonis & Armstrong, 2012; Muratori et al., 2017), and, in some cases, mediate treatment outcomes (e.g., Elizur et al., 2017). Several studies also found changes in additional parent factors post-treatment, such as parental distress and parental compliance (e.g., Datyner et al., 2016; Elizur et al., 2017; Fleming et al., 2017; Somech & Elizur, 2012a), which may in turn impact success via resulting changes in parenting practices. Taking into account parental factors, including parent personality as identified in Study 2, will therefore likely be important in treatment success.

Additionally, results from case studies suggest that targeting cognitive deficits previously found in the literature (e.g., emotion recognition, empathy) may improve behaviors associated with CU traits, as well as reduce levels of CU traits (Datyner et al., 2016; Fleming et al., 2017). Notably, the single randomized control trial of a treatment developed for CU traits specifically

found that adolescents with comorbid CD and CU traits had significant improvements in disruptive behavior and empathy only in the group that included individual treatment in addition to parenting components (Dadds et al., 2012). However, this study did not report whether CU traits specifically were impacted by the treatment. Overall, initial findings from these intervention studies appear promising, and demonstrate the utility of incorporating research on the developmental and neural correlates of AB and CU traits into treatment.

Future Directions

The results of the three studies that comprise this dissertation reflect significant advances in the understanding of developmental precursors and neurocognitive functioning associated with AB and CU traits. However, the results also highlight ongoing challenges in the field that should be addressed in future research.

Prospective Designs Across Development. Notably, each of the three studies was cross-sectional in nature. As such, the directionality of associations could not be determined. For instance, in Study 1, associations between parenting practices and offspring CU traits may have, at least in part, due to child-level factors. Indeed, a recent adoption study found that: 1) harsh parenting early in development predicted later CU behaviors in children and 2) early child CU behaviors predicted harsher parenting in mothers (Trentacosta et al., 2019). Thus, prospective, genetically informed designs will be critical to examine transactional associations between child-level factors and environmental risk factors in the emergence of AB and CU traits.

Future prospective research should also utilize numerous time points across development. In this dissertation, studies were limited to adolescent (Studies 1 and 2) and young adult samples. However, oppositional and defiant behaviors, including aggression, emerge as early as toddlerhood (Loeber et al., 2009), and CU behaviors can be measured as early as age 3 (Waller et

al., 2017). Examining expressions of AB and CU traits at various developmental periods is important for several reasons. Developmental studies have found that earlier onset of disruptive behaviors is associated with more severe and stable AB across the lifespan (Moffitt, 2018). This subtype of AB appears to have distinct etiological mechanisms, and thus likely requires unique intervention approaches (Moffitt, 2018). Additionally, AB and CU traits may be more malleable at earlier ages; developmental studies could therefore help to identify time periods when interventions may be most successful (Olds et al., 2005; Waller et al., 2017). Relatedly, environmental influences on the brain appear to also differ based on the developmental timing of when they occurred (Tottenham & Sheridan, 2010). Yet, there are few longitudinal neuroimaging studies of AB and CU traits. As such, it is unclear how neurocognitive dysfunction develops across the lifespan. Finally, relative genetic and environmental influences on CU/psychopathic traits and AB appear to change over time, such that genetic effects are stronger in childhood compared to adolescence, at which time environmental influences become stronger (Ferguson, 2010; Gard et al., 2019; Rhee & Waldman, 2002). However, further research is needed on the genetic and environmental etiology of CU traits across the entire lifespan (i.e., very young children, adults) (Moore et al., 2019; Tuvblad et al., 2019).

Large, Population-based Studies. The results of the three studies also highlight the importance of sampling in research on AB and CU traits. All three studies utilized community samples. As such, results may not be generalizable to clinical or adjudicated samples with potentially more severe levels of AB and/or CU/psychopathic traits. As discussed throughout this dissertation, many previous studies on AB and CU traits, particularly neuroimaging research, have utilized small samples of extreme groups (e.g., forensic/incarcerated samples). The existing literature has therefore likely been significantly shaped by 1) study sample size, which impacts

power, and 2) sample characteristics, given that the majority of work has been conducted in youth with extreme levels of both AB and CU traits utilizing case-control approaches and thus potentially limiting the generalizability of findings across the spectrum of AB and CU traits (verses only clinical-threshold presentations) (Salekin, 2017).

First, future research should aim to recruit larger samples to power more complex statistical approaches. Comprehensive developmental theories of AB and CU traits have suggested that genes, environmental context, and neural dysfunction interact to predict outcomes (Gard et al., 2019; Murray et al., 2018; Waller et al., 2017). However, few studies have explicitly tested transactional models of associations among all of these factors in relation to AB and CU traits. In this dissertation, Study 1 highlighted both genetic and environmental risk associated with emergence of CU traits, whereas Studies 2 and 3 demonstrated unique neurocognitive dysfunction associated with CU traits. To test more comprehensive models that include all of these pathways, future studies could utilize an imaging gene-environment (IG x E) approach, which examines how genetic and environmental risk interact to predict AB or CU traits via neural dysfunction (Hyde et al., 2011; Waller et al., 2015). However, this type of model requires large sample sizes be sufficiently powered (e.g., Early Genetics and Lifecourse Epidemiology [EAGLE] consortium; Pappa et al., 2016; Adolescent Brain Cognitive Development [ABCD] Study; Volkow et al., 2018). Moreover, researchers should also utilize more racially, socio-economically, and culturally diverse community samples to better understand associations at varying levels of AB and CU traits, and to determine whether individual differences impact associations (Falk et al., 2013). Data in both Studies 2 and 3 came from the Michigan Twin Neurogenetics Study, a population-based sample of twins recruited from Southeast Michigan (Burt & Klump, 2019). Population-based studies are characterized by systemic sampling and

unique data collection procedures that ultimately enhance generalizability of findings (Falk et al., 2013). Excitingly, such studies becoming more common (e.g., Aggression in Children: unravelling gene-environment interplay to inform Treatment and InterventiON strategies [ACTION] Consortium; Boomsma, 2015).

Addressing the Role of Racism. Notably, although a benefit of population-based studies is the inclusion of individuals from differing backgrounds, researchers have a duty to justify why phenomena may vary according to different identities. Specifically, a significant limitation of the existing literature is the use of "race" as a variable and interpretation of "race" effects. "Race" is ultimately a social classification based on phenotype, representing a *social* rather than *biological* construct (Jones, 2001). The identity of "race" (as designated by society) impacts both the opportunities afforded to an individual and societal constraints imposed on an individual (Jones, 2001). Thus, "race" in research is a proxy for exposure to racism, including institutionalized racism (i.e., differential access to goods, services, and opportunities) and personally mediated racism (i.e., differential assumptions about others' abilities or motives [prejudice] and/or differential actions toward others [discrimination]) (Jones, 2000). These are the effects that previous psychopathology research, including studies of AB, have likely measured when examining "race". For instance, institutionalized racism directly impacts socioeconomic status via differential access to quality education, sound housing, gainful employment, and other resources, all of which have been demonstrated to impact the emergence of AB (Jones, 2001; Williams et al., 2019). Personally mediated racism can also contribute to associations with AB via the stresses of everyday racism on biological systems implicated in AB (Collins, 1992; Williams et al., 2019) and differential treatment by criminal justice system (Reskin, 2012; Williams et al., 2019).

Numerous studies of AB have included "race" as a covariate, and/or demonstrated race-related associations (McGowen, 2006; Rojas-Gaona et al., 2016). Yet, researchers have rarely explicitly noted what "race" is measuring and/or simply document associations rather than actively investigate the basis of racial differences, particularly in psychological research (as opposed to criminology or sociology). Although unfortunately somewhat common, this practice has harmful consequences, including: 1) ignoring important group differences that could impact our understanding of results (i.e., the consequences of racism on mental health and behavior); 2) limiting our ability to address sources of race-related differences (i.e., inform policies to restructure societal institutions that propagate racism) and 3) implicitly supporting the notion that race-associated differences are due to biology and thus do not require further exploration (biologic determinism) (Jones, 2001). In this dissertation, I sought to address these issues by explicitly defining race and justifying its use as a covariate in each of the three studies. In future studies, researchers should continue to make tangible steps towards reframing the use and interpretation of race effects, starting by identifying race as a social construct that undoubtedly measures exposure to racism, and interpret any observed race-associated differences through this contextual lens (Jones, 2001). Additionally, researchers could explicitly measure specific experiences (e.g., geographically measured housing discrimination, self-reported experiences with microaggressions and racism) rather than using 'race' as a proxy. Researchers are encouraged to refer to the several published commentaries that provide in-depth guidelines for studies (e.g., Jones, 2001; Williams et al., 2019). Psychological researchers have both the opportunity and responsibility to contribute scientific knowledge to the global conversation about racism, particularly to inform strategies to eliminate racial disparities in health outcomes that are driven by racism.

Understanding Gender Differences. Finally, gender is an additional demographic factor that warrants further examination in the literature on CU and psychopathic traits. In the current dissertation, gender was addressed differently across the three studies. Study 3 was limited to only young adult men due to the nature of the PMCP study design. In Study 2, I included gender as a covariate but did not further investigate gender as a primary aim. In Study 1, I explicitly explored gender differences by separately looking at pathways for mothers versus fathers with psychopathic traits and by examining whether offspring gender moderated associations. These different approaches to exploring gender differences relates to an ongoing limitation in the field. As noted earlier in this dissertation, previous studies have often focused on male samples, primarily due to research showing that elevated psychopathic traits are more common in men (Cale & Lilienfeld, 2002; Dolan & Völlm, 2009) and that elevated CU traits are more common in boys (Fanti, Frick, & Georgiou, 2009; Kimonis et al., 2014). As such, it is difficult to interpret gender differences related to these constructs.

The existing reviews on gender differences in adult psychopathy have primarily focused on psychometric properties of assessment measures (Cale & Lilienfeld, 2002; Dolan & Völlm, 2009). Most research suggests that the factor structure of psychopathy appears to be similar for men and women (Cale & Lilienfeld, 2002; Dotterer et al., 2016). However, there appear to be gender differences in the item loadings for assessment measures, for instance, in women items such has "criminal versatility", "juvenile delinquency", and "failure to accept responsibility", have lower item loadings (i.e., weakly related to the construct of psychopathy) whereas "promiscuity" has a higher loading (i.e., strongly related to the construct of psychopathy) (Dolan & Völlm, 2009). To this point, researchers have theorized that different behaviors may be more characteristic of female psychopathy versus male psychopathy (Forouzan & Cooke, 2005).

However, it is also possible that these differences reflect gender bias in measurement when criteria are applied to women, based on social norms and prejudice; for example, women reporting multiple, short-term sexual partners may be judged as more "abnormal" compared to men (Forouzan & Cooke, 2005).

Additionally, though psychopathy appears to predict recidivism in both men and women (Cale & Lilienfeld, 2002; Dolan & Völlm, 2009), gender appears to moderates associations between adult psychopathy and other behavioral phenotypes. In their recent review, Efferson and Glenn (2018) found evidence that women with elevated psychopathic traits do not demonstrate response perseveration or passive avoidance errors (which are often associated with psychopathy in men) and do not display emotional processing deficits to the same extent as men with elevated psychopathic traits. The authors also note some evidence suggesting gender differences in moral processing (i.e., response to unfairness and moral violations) (Efferson & Glenn, 2018). Overall, however, there are still relatively few studies on psychopathy that directly compare men and women, and these studies vary greatly in sample type, measure of psychopathy, and analytic strategy, making it difficult to compare findings.

The research on gender differences in CU traits is even more limited. Similar to psychopathy, studies have found that the factor structure of CU traits is the same for both boys and girls (Frick & Ray, 2015; Pihet, Etter, Schmid, & Kimonis, 2015). Gender does not appear to moderate associations between CU traits and externalizing outcomes (e.g., reactive aggression, proactive aggression, nonviolent delinquency, and hyperactivity) (Cardinale & Marsh, 2020), severity of conduct problems (Longman, Hawes, & Kohlhoff, 2016), or empathy, guilt, or prosociality (Waller et al., 2020). However, a recent meta-analysis found that CU traits were more strongly associated (i.e., higher effect sizes) with internalizing symptoms (e.g., anxiety,

psychological withdrawal, and depressive symptoms) in female-only samples relative to mixed gender samples (Cardinale & Marsh, 2020).

Importantly, even less research has focused on gender differences in potential etiological mechanisms of CU or psychopathic traits. One study found that elevated CU traits are associated with low cortisol levels in boys but not in girls (Loney, Butler, Lima, Counts, & Eckel, 2006). Additionally, a longitudinal study found that elevated CU traits were associated with reduced parental involvement specifically among older boys and younger girls. In the same study parental involvement more strongly predicted decreases in levels of CU traits for boys than for girls, whereas positive parenting practices more strongly predicted decreases in levels of CU traits for girls than for boys (Hawes, Dadds, Frost, & Hasking, 2011). However, given the few studies on potential underlying etiological mechanisms of psychopathic or CU traits that could contribute to differential associations by gender, it is difficult to ascertain whether observed associations reflect 'true' gender differences, as opposed to statistical artifacts. Theoretical work proposes that 'true' gender differences must be demonstrated to arise from a 1) fundamental genetic difference related to being biologically male or female, 2) biological (e.g., differences in biological maturation timing or hormone production) or social (e.g., differences in socialization or societal expectations) consequences related to gender, or 3) proximal risk or protective factors related to expression of emotion or psychopathology that are also tied to gender (Rutter, Caspi, & Moffitt, 2003). Thus, based on the existing literature, it is unclear which, if any, of these levels contributes to gender differences in the expression of CU and psychopathic traits. Taken together, further longitudinal research spanning from childhood to adulthood on mixed gender samples is needed to better understand and interpret gender-related differences in psychopathic and CU traits.

Conclusions

Accumulating evidence suggesting that youth with AB and elevated CU traits represent a more severe subgroup, characterized by unique developmental trajectories and biological mechanisms (Frick et al., 2014). However, previous studies have been limited by lack of specificity in associations (i.e., have not consistently measured CU traits, or only examined associations in youth with elevated AB and CU traits) and reliance on clinical or adjudicated samples that are not representative of the broader population. In this dissertation, I examined developmental factors and neurocognitive deficits of AB and CU traits dimensionally in diverse community sample, utilizing analytic strategies to parse apart unique effects of AB versus CU or psychopathic traits, across various age groups (i.e., childhood/early adolescence and young adulthood). Future research is needed that examines etiological mechanisms of AB versus CU traits in more representative samples, including multiple time points across development. Taken together, the findings of this dissertation add to the accumulating evidence that youth with AB + CU traits represent a unique subgroup that will likely benefit from distinct targets from intervention compared to youth with AB alone.

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