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The Validity of Primary and Secondary Subtypes
of Psychopathy in Children and Adolescents

A dissertation submitted in partial satisfaction
of the requirements for the degree Doctor of Philosophy
in Psychology

by

Meghan Elizabeth McKenzie

2017

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ABSTRACT OF THE DISSERTATION

The Validity of Primary and Secondary Subtypes of Psychopathy in Children and Adolescents

by

Meghan Elizabeth McKenzie

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2017

Professor Steve Sung-Yul Lee, Chair

Characterized by atypical behavioral (e.g., antisociality), interpersonal (e.g., egocentricity), and affective facets (e.g., low remorse), the phenomenological diversity of psychopathy likely consists of potentially etiologically distinct subtypes. Across community, clinic-referred, and adjudicated samples, anxiety and negative emotionality differentiated two subgroups of adolescents and adults with elevated psychopathic traits. In particular, secondary psychopathy was correlated with greater anxiety, negative emotionality, and stress reaction and lower control than primary psychopathy. Of central importance to the current study, primary and secondary psychopathy were conceptualized as being differentially sensitive to genetic and environmental influences. Specifically, primary psychopathy reflected more innate, genetically-based factors including affective disturbance (e.g., callousness) and minimal negative emotionality (e.g., anxiety), whereas secondary psychopathy was hypothesized to be an

adaptation to environmental risk (e.g., maltreatment). However, empirical examinations of genetic and environmental contributions to subtypes provide inconsistent support for this theory, and knowledge about the mediational constructs and processes underlying the development of primary versus secondary subgroups of psychopathy is limited. Furthermore, anxiety-based distinctions of primary and secondary psychopathy have yet to be similarly evaluated in school-age children. This dissertation aimed to address these gaps directly. Using two independent, yet complementary, prospective longitudinal samples of 221 children with and without ADHD followed for two years (UCLA ADHD and Development Study) and a nationally representative sample of 15,701 adolescents followed prospectively across 14 years (National Longitudinal Study of Adolescent Health), we explored the validity of primary and secondary subgroups by examining their differential association with key correlates (e.g., maltreatment history, reactive aggression, antisocial behavior, delinquency, and emotional processing). Next, we tested whether individual differences in dimensions of temperament and self-regulation mediated predictions of primary and secondary psychopathy from a functional polymorphism regulating serotonin neurotransmission. Primary findings were three-fold. First, across both samples, secondary psychopathy demonstrated more anxiety and negative emotionality and engaged in greater total, nonviolent, and violent antisocial behavior than primary psychopathy. Second, secondary subgroups reported more diverse forms of childhood maltreatment relative to primary and comparison youth. Lastly, prosociality, but not negative emotionality, daring, or self-regulation, mediated predictions of psychopathy subgroups from 5-HTTLPR in childhood. In addition to providing support for primary and secondary psychopathy among children and non-adjudicated adolescents and adults, these findings also identify mediators of primary and secondary psychopathy. We discuss the utility of using population-based samples to examine individual

differences within psychopathy, potential causal mechanisms that differentially contribute to psychopathy subgroups, as well as implications for tailored interventions targeting impairments associated with primary and secondary psychopathy.

The dissertation of Meghan Elizabeth McKenzie is approved.

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2017

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INTRODUCTION

Characterized by individual differences in callousness (e.g., lack of empathy or guilt), impulsivity, deceit, and shallow affect, psychopathy is associated with persistent and diverse forms of aggression, violence, criminal recidivism, and is highly intractable to treatment (Cooke & Michie, 2001; Hemphill, Hare, & Wong, 1998; Salekin, 2008). Despite the predictive validity of psychopathy and psychopathic traits, far less is known about developmentally-informed precursors to psychopathic traits. Given their role in problems with substantial clinical and public health significance, identifying risk factors for psychopathic traits, particularly early in development, is necessary to facilitate innovations in prevention and intervention.

Reflecting its heterogeneity, theoretical attempts to refine psychopathy have prioritized distinctions between “high-anxious” (i.e., secondary) and “low-anxious” (i.e., primary) subtypes of psychopathy. The theoretical origins of primary and secondary psychopathy suggest that these subgroups engage in similar levels of irresponsible, antisocial, and hostile behavior; however, they are distinct based on the underlying motivation and etiology of these behaviors. Whereas primary psychopathy reflected more innate, genetically-based factors including affective disturbance (e.g., callousness) and minimal negative emotionality (e.g., neuroticism, anxiety), secondary psychopathy emerged from environmental risk factors such as parental neglect, abuse, and harsh punishment. Unlike the primary subgroup, secondary psychopathy was theorized to exhibited greater impulsivity, poor emotion regulation, and internalizing symptoms (Karpman, 1941, 1948; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003).

Across incarcerated, clinic-referred, and community-based samples of adults and adolescents, empirical examinations provide support for differentiated subgroups of psychopathy based upon anxiety (Hicks, Markon, Patrick, Krueger, & Newman, 2004; Kahn et al., 2013;

Kimonis, Frick, Cauffman, Goldweber, & Skeem, 2012; Newman, MacCoon, Vaughn, & Sadeh, 2005; Skeem, Johansson, Andershed, Kerr, & Louden, 2007; Swogger & Kosson, 2007; Vassileva, Kosson, Abramowitz, & Conrod, 2005). Secondary psychopathy in adulthood was correlated with greater anxiety, negative emotionality, and stress reaction and lower control than primary psychopathy. Similar patterns were observed among adolescents: High-anxious variants of psychopathy were more negatively emotional, socially withdrawn, and aggressive than low-anxious psychopathy subtypes (Fanti, Demetriou, & Kimonis, 2013; Kahn, Youngstrom, Youngstrom, Feeny, & Findling, 2013; Salihovic, Kerr, & Stattin, 2014). Additionally, secondary psychopathy subgroups reported more severe maltreatment histories compared to primary psychopathy (Kahn et al., 2013; Kimonis, Skeem, Cauffman, & Dmitrieva, 2011; Kimonis et al., 2012; Poythress, Skeem, & Lilienfeld, 2006; Vaughn et al., 2009). Prior research also indicates that primary and secondary subgroups of psychopathy differ in emotional processing: primary psychopathic offenders demonstrated impaired passive avoidance learning, modulation of responses to emotional and neutral stimuli, and fear-potentiated startle responses; secondary psychopaths did not (Newman, Patterson, Howland, & Nichols, 1990; Newman, Schmitt, & Voss, 1997; Newman & Schmitt, 1998). Despite emerging evidence on the validity of primary and secondary psychopathy subgroups in adults, less is known about its validity in young children, even though individual differences in psychopathic traits are reliably measured in children as early as 4 years of age (Dadds, Fraser, Frost, & Hawes, 2005). To address this gap, this dissertation aimed to identify potential primary and secondary subgroups of childhood psychopathic traits in two independent, yet complementary, prospective samples of children and adolescents. The current proposal will test their differential associations with key risk factors

(i.e., delinquency, reactive aggression, maltreatment, and emotional processing) implicated in primary versus secondary adult psychopathy.

As described previously, primary and secondary subgroups of psychopathy were conceptualized as being sensitive to differential genetic and environmental influences. Although few studies have been conducted, support for the contribution of distinct etiological pathways in primary and secondary psychopathy has been inconsistent, however. To date, the only published twin study reported that heritability estimates were similar for *both* primary and secondary subtypes (Hicks et al., 2012). Furthermore, shared environmental influences, such as parenting behavior, were unassociated with both subtypes. Candidate gene studies are lacking, despite their role in clarifying potential specific etiological processes underlying different constellations of psychopathy. Given that serotonin is essential to the structure and connectivity of neural structures (i.e., amygdala, ventromedial prefrontal cortex) implicated in emotional reactivity, inhibition, and decision-making, psychopathy may be influenced by serotonergic systems. However, molecular genetic studies of primary and secondary psychopathy subtypes in children are non-existent. This dissertation will also examine potential differential associations between psychopathic variants and a specific polymorphism in the promoter region (5-HTTLPR) of the serotonin-transporter gene (*SLC6A4*) due to its association with emotionality and stress reactivity (Hariri & Holmes, 2006).

Reflecting an important absence of evidence on pathways underlying genetic influences on individual differences, including psychopathic traits, little is known about the explanatory factors underlying such predictions (i.e., mediators). Dimensions of temperament, that is, individual differences in emotional, motor, and attentional activity (Rothbart, Ahadi, & Evans, 2000), are a compelling candidate mediator as they are sensitive to genetic influences, evident

early in life, and they are stable throughout development. Further, specific dimensions of temperament (i.e., high levels of fearlessness) have been linked to psychopathy. Defined as processes such as effortful control and executive attention that modulate reactivity, self-regulation is an additional plausible mediator, as it and emotion reactivity are underlying processes of temperament (Rothbart, Sheese, Reuda, and Posner; 2011). Given that genetic effects for complex phenotypes are highly dispersed, elucidation of those effects and identification of putative pathways that lead to subtypes of psychopathy are needed. To address this gap in the literature, my dissertation will also evaluate potential mediators that may account for the association between a functional polymorphism regulating serotonin transmission (5-HTTLPR) and psychopathy.

PROPOSED STUDIES

This dissertation consists of three chapters containing a total of four studies. Chapters 1 and 2 each contain one study, whereas Chapter 3 contains two studies. To examine potential evidence of primary and secondary subtypes among school-aged children, Chapter 1 used model-based cluster analysis to derive comparison, primary and secondary psychopathy subgroups and examined group differences on key correlates (e.g., anxiety, psychopathic traits, reactive aggression, antisocial behavior, and emotional processing) among a sample of 221 children aged 5-10 years with and without ADHD who were followed prospectively across two years (i.e., UCLA ADHD & Development Study). Similarly, using a nationally representative sample of 15,701 adolescents followed prospectively into adulthood (24-32 years old) across 14 years (i.e., The National Longitudinal Study of Adolescent Health), Chapter 2 examined differences among comparison, primary, and secondary subgroups on key variables, including psychopathic traits, maltreatment, delinquency, and antisocial behavior. Lastly, to elucidate potential causal

mechanisms, Chapter 3 contained Study 3 and 4, which examined dimensions of temperament and self-regulation as potential mediators of the association of 5-HTTLPR and psychopathy subgroups in the UCLA ADHD and Development Study and the National Longitudinal Study of Adolescent Health (Add Health). Each samples provides strengths well suited to addressing these research questions, including multi-method, multi-informant measures in the UCLA ADHD and Development Study and a large sample of children followed prospectively in Add Health.

CHAPTER 1: THE VALIDITY OF PRIMARY AND SECONDARY SUBGROUPS OF PSYCHOPATHY IN CHILDREN¹

Abstract

Across community and forensic samples of adolescents and adults, primary and secondary subgroups of psychopathy exhibit similar levels of psychopathic traits and antisocial behavior, but they are distinguished by anxiety and negative emotionality. Whereas primary psychopathy is associated with increased instrumental aggression and under-reactivity during emotional processing, secondary psychopathy is more reactively aggressive and is unrelated to deficient emotional processing. However, it is unclear whether primary and secondary psychopathy are evident early in development. Participants were 221 5 to 10 year-old, ethnically diverse children (45% non-White, 55% boys) with and without attention-deficit/hyperactivity disorder followed prospectively over two years. At baseline, psychopathy subgroups were derived from parent ratings of psychopathic traits and youth self-reported anxiety using model-based cluster analysis; groups were then compared on multi-method measures of psychopathic traits, aggression, antisocial behavior and anxiety, as well as emotional processing on a go/nogo task two years later. Primary and secondary subgroups were comparable on psychopathic traits and reactive aggression, but the secondary group self-reported more anxiety and antisocial behavior than the primary group. Finally, the secondary group exhibited better cognitive control of emotional processing relative to the primary group. These preliminary data suggest that primary and secondary subgroups of psychopathy in school-aged children differentially predict key outcomes two years later. We discuss implications of these findings for causal theories of psychopathy as well as future directions to improve traction on the considerable heterogeneity within youth psychopathy.

Characterized by a unique constellation of behavioral (e.g., impulsivity, antisociality), interpersonal (e.g., manipulation, egocentricity) and affective (e.g., lack of remorse, shallow affect) features, psychopathic traits are highly intractable to treatment and predict persistent and diverse forms of aggression, violence, and criminal recidivism (Cooke & Michie, 2001; Hemphill, Hare, & Wong, 1998; Salekin, 2008). However, psychopathy is also heterogeneous, consisting of potentially distinct subgroups with unique causal influences and divergent patterns of association. Despite persuasive evidence of their predictive validity, there is relatively little developmentally-informed research with respect to precursors of psychopathic traits and whether heterogeneity in psychopathic traits differentially reflects unique causal influences (Skeem, Johansson, Andershed, Kerr, & Louden, 2007). Given their prediction of problems with substantial clinical and public health significance, identifying risk factors for early expressions of psychopathic traits will facilitate innovations in prevention and intervention.

Although three- and four-factor models have been proposed (see Cooke, Michie, & Hart, 2006; Hare, 2003), adult psychopathy consists of two separable factors (i.e., Psychopathy Checklist; Harpur, Hare, & Hakstian, 1989): Factor one is comprised of interpersonal and affective deficits (e.g., lack of remorse, manipulation) whereas the second factor consists of social deviance (e.g., impulsivity, irresponsibility, aggression). Because anxiety critically differentiates variation within adult psychopathy (Hicks, Markon, Patrick, Krueger, & Newman, 2004; Newman, MacCoon, Vaughn, & Sadeh, 2005; Skeem et al., 2007; Swogger & Kosson, 2007; Vassileva, Kosson, Abramowitz, & Conrod, 2005), it is surprising that anxiety has yet to be similarly evaluated in school-age children. The current study addresses this directly. Early conceptualizations of adult psychopathy consisted of primary and secondary subgroups that were similar with respect to antisocial behavior, but separable based on their underlying motivation

and etiology. Whereas primary psychopathy was centrally defined by affective disturbance (e.g., callousness) and low negative emotionality (e.g., anxiety), secondary psychopathy was principally sensitive to environmental influences (e.g., maltreatment) and characterized by impulsivity, poor emotion regulation, and internalizing symptoms (Karpman, 1941; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). Compared to primary psychopaths, in forensic settings, secondary psychopaths were more anxious, negatively emotional, and stress reactive (Hicks et al., 2004). Primary and secondary subgroups also differed on specific dimensions such that primary psychopathy showed elevated interpersonal and affective facets (e.g., low remorse, shallow affect) whereas secondary psychopaths exhibited more behavioral deficits (e.g., irresponsible, impulsive) (Skeem et al., 2007). Consistent with theory, primary and secondary subgroups were similarly antisocial (Skeem et al., 2007; Swogger & Kosson, 2007), although secondary psychopathy was uniquely associated with physical aggression (Hicks et al., 2004). Thus, adult psychopathy is heterogeneous with differential correlates, however less is known about whether anxiety explains heterogeneity within youth psychopathic traits.

Among youth, psychopathy consists of a three-factor model with narcissism, callous-unemotional (CU) traits, and impulsivity (Frick, 2009; Frick, Bodin, & Barry, 2000; see Neumann, Kosson, Forth, & Hare, 2006 for an exception); narcissism and CU traits reflect factor one in adults, whereas impulsivity is synonymous with factor two. CU traits in particular have received the most attention given that it typically designates children with more severe, aggressive and chronic conduct problems (Frick, 2009; Frick & Dickens, 2006), even predicting increasing conduct problems (Dadds, Fraser, Frost, & Hawes, 2005). Similarly, relative to conduct problems per se, CU traits correlate uniquely with deficient processing of emotional stimuli and sensitivity to punishment cues (Blair, Leibenluft, & Pine, 2014 and Frick & White,

2008 for a review). Thus, CU traits differentiate variation in youth conduct problems; however, given that anxiety reliably distinguishes primary and secondary psychopathy in adults, we explore whether it functions similarly in children.

Among separate samples of community, clinic-referred and adjudicated adolescents, anxiety differentiated youth with elevated psychopathic traits. For example, the high-anxious variant of psychopathy was more negatively emotional, socially withdrawn, and aggressive than the low-anxious psychopathy subgroup (Fanti, Demetriou, & Kimonis, 2013; Kahn et al., 2013; Salihovic, Kerr, & Stattin, 2014); this subgroup was also more reactively aggressive, impulsive, and self-reported more alcohol and substance use problems than the low-anxious variant of psychopathy (Falkenbach, Poythress, & Creevy, 2008; Kimonis, Frick, Cauffman, Goldweber, & Skeem, 2012; Salihovic et al., 2014; Vaughn, Edens, Howard, & Toney Smith, 2009; see Kimonis, Fanti, Isoma, & Donaghue, 2013 for an exception). Thus, anxiety meaningfully discriminates youth with similar levels of elevated psychopathic traits. Despite these promising results, findings are limited given that adolescent psychopathy subgroups were typically derived from youth self-reported psychopathic traits (Salihovic et al., 2014), which have well known methodological limitations (Kubak & Salekin, 2009). To advance this literature, additional methods of ascertainment must be prioritized.

Although anxiety differentiated subgroups of adolescents based on overall psychopathic traits, high- versus low-anxious variants revealed specific patterns when individual psychopathy facets were considered. When derived exclusively from CU traits (i.e., without regard to narcissism and impulsivity), primary and secondary subgroups exhibited similar affective deficits (e.g., low remorse) across community and clinic-referred samples (Fanti et al., 2013; Kahn et al., 2013). Crucial to the current study, however, when subgroups were derived from *all*

psychopathy facets (i.e., not CU traits alone), the low-anxious group exhibited more narcissism, CU traits, and impulsivity relative to the high-anxious group (Salihovic et al., 2014). Similarly, among adolescent offenders, the high-anxious subgroup was more impulsive yet similarly narcissistic and callous-unemotional relative to the low-anxious subgroup (Kimonis et al., 2012). Other studies reported elevated narcissism among the high-anxious subgroup (Fanti et al., 2013; Kimonis, Skeem, Cauffman, & Dmitrieva, 2011). Although we agree that CU is central to the phenomenology of psychopathy across development, including with respect to conduct problems (Frick, 2014), previous inconsistency in how subgroups were derived (i.e., all three psychopathy facets versus CU traits exclusively) is problematic. For example, narcissism uniquely predicted conduct problems beyond CU traits and attention-deficit/hyperactivity disorder (ADHD) (Jezior, McKenzie, & Lee, 2016; McKenzie & Lee, 2015; for exception see Frick et al., 2014). Because individual psychopathy facets are differentially associated with poor outcomes, rigorous studies must employ all psychopathy facets to derive relevant subgroups.

To improve predictions of important adult outcomes, risk factors early in development must be identified. Although individual differences in psychopathic traits are evident at age 4 (Dadds et al., 2005), it is unclear how anxiety relates to subgroups of psychopathy in school-age children when narcissism, CU traits, and impulsivity are all included. This pattern is surprising given that anxiety has been used focally to differentiate children with conduct problems from children with elevated psychopathic traits. For example, CU traits negatively correlated with trait anxiety, whereas conduct problems positively correlated with trait anxiety among a school-aged clinical sample (Frick, Lilienfeld, Ellis, Loney & Silverthorn, 1999). However, it is unknown if anxiety distinguishes variation within children with similar elevations in psychopathic traits. That is, although conduct problems alone was associated with greater anxiety (Frick et al., 1999),

we aimed to determine if children with similar elevations in psychopathic traits exhibited differences in anxiety and key correlates.

School-aged children with elevated psychopathic traits overall exhibited impaired recognition and reduced sensitivity to negative emotional stimuli, particularly empathy (Blair, Colledge, Murray, & Mitchell, 2001; Dadds, El Masry, Wimakaweera, & Guastella, 2008; Kimonis et al., 2008). Conversely, children with conduct problems alone demonstrate increased amygdala responsiveness to threat (Crowe & Blair, 2008; Blair et al., 2014). However, we know of only one study that has examined how low-anxious and high-anxious subgroups of children with similar elevations in psychopathic traits derived from all three facets relate to individual differences in emotional processing, a key aspect of psychopathy. Secondary psychopathy, often characterized by elevated anxiety, may be hypervigilant to threat-related cues (Pollak, Cicchetti, Hornung, & Reed, 2000), including perceiving ambiguous facial expressions as angry (Pollak & Kistler, 2002), displaying increased neural activity while searching for angry faces, and quickly orienting to, yet slowly disengaging, from anger cues (Shackman, Shackman, & Pollak, 2007). Using a picture version of a dot-probe task, high-anxious psychopathic youth were more engaged toward emotionally distressing pictures than low-anxious subgroup youth (Kimonis et al., 2012); thus, primary and secondary subgroups may improve traction on differences in emotional processing.

Overall, in samples ranging from incarcerated adults and college students to adjudicated adolescents, anxiety differentiated subgroups of psychopathy. Although anxiety differentiates between children with conduct problems and children with psychopathic traits (Frick et al. 1999), *it is unknown whether anxiety differentiates subgroups of children with similar elevations in psychopathic traits (not simply CU traits)*. Psychopathy subgroups in childhood must

demonstrate predictive validity if they are to facilitate innovations in intervention and prevention. Hence, the current study evaluated “primary” and “secondary” subgroups of youth among school-aged children followed prospectively for two years. We hypothesized two subgroups of children, each with elevated psychopathic traits, would be separable based on anxiety. Crucially, unlike previous studies, we included multiple dimensions of anxiety and all psychopathic traits (i.e., CU traits, narcissism, and impulsivity) in model-based cluster analysis to identify subgroups. Moreover, given the questionable validity of youth self-reported psychopathic traits (Kubak & Salekin, 2009; McMahon & Frick, 2005), we utilized parent ratings of youth psychopathic traits. Similarly, given the incremental utility of youth self-reported anxiety (Achenbach, 2006; Mash & Hunsley, 2005), we included youth self-reported anxiety to derive primary and secondary subgroups. We predicted that the high-anxious variant of psychopathy would engage in more antisocial behavior and reactive aggression than their low-anxious counterparts. We also examined indices of emotional processing using an emotion go/nogo task in low-anxious and high-anxious psychopathic variants. Although directional hypotheses are supported in comparisons of conduct problem only youth and youth with psychopathic traits, the current study diverges meaningfully given that we examined differences among groups of children who differed with respect to anxiety, but exhibited similar elevations in psychopathic traits. As such, we did not make specific predictions about the directionality of such differences.

Method

Participants

At baseline, 221 5 to 10 year-old ethnically diverse children (55% White, 9% Black, 10% Latino, 3% Asian, and 23% Mixed or Other; 55% boys) with ($n = 114$) and without ($n = 107$) ADHD were assessed. Families were recruited from local elementary schools and pediatric

offices, presentations at self-help groups, and referrals from clinical service providers in an urban area of Southern California. English fluency and living with at least one biological parent at least half the time were required for participation in the study. Children with a Full Scale IQ below 70 and a previous diagnosis of an autism spectrum, seizure, or neurological disorder as well as any medical condition that prevented full participation were excluded from the study. Given its central role in fueling early-onset conduct problems (Hinshaw et al., 1993), which is typically accompanied by CU traits (Frick et al., 2005), ADHD was oversampled to ensure significant risk for conduct problems. ADHD probands and non-ADHD comparison youth did not differ significantly with respect to age, sex, race-ethnicity, and income. ADHD probands met full diagnostic criteria for ADHD according to the Diagnostic Interview Schedule for Children, Version IV, Parent Version (DISC-IV-P; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), a fully structured interview with the parent keyed to all Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) criteria. To improve external validity, common comorbidities for ADHD such as oppositional defiant disorder (ODD), anxiety, and depression were allowed among probands. Youth who met diagnostic criteria for any disorder other than ADHD (anxiety disorders were the most common) were placed in the non-ADHD group to avoid recruiting an unrealistically high functioning non-ADHD comparison group.

Procedures

Initial eligibility, including the exclusion criteria described above, was determined using a standardized telephone screening process. Eligible families were invited to in-person laboratory assessments, and rating scales were mailed to the child's primary teacher. After obtaining parent consent and child assent, parents (90% mothers) were interviewed using the DISC-IV-P and

completed measures of parenting, child behavior, life stress, and their own psychopathology. Children were separately assessed on standardized measures of cognitive ability, academic achievement, and self-reported psychopathology. Eighty-five percent of children were unmedicated (mostly stimulants) during the laboratory assessment. Whenever possible, parents and teachers were asked to complete rating scales based on the child's unmedicated behaviors. Interviews were conducted by intensively-trained clinical psychology doctoral students and BA-level staff; interviewers were initially blind to the child's diagnostic status, but blindness could not always be preserved due to the extensive information gathered on the DISC-IV-P. Families received \$50 and a diagnostic report summarizing the child's cognitive and academic functioning and DSM-IV diagnoses. All study procedures were approved by the IRB.

Approximately two years after their initial assessment, families were invited back to the laboratory to participate in a follow-up assessment (i.e., Wave 2). Children were 7 to 12 years of age. Consisting of highly similar assessment procedures to those at Wave 1, relevant domains of inquiry at Wave 2 included family functioning, youth academic achievement, and child psychopathology. Approximately 89% of the initial Wave 1 sample was re-evaluated at Wave 2; children with ADHD were more likely to return at Wave 2 than families without ADHD. No other significant demographic or clinical factors (i.e., child age and sex, parent race-ethnicity) distinguished participants at Wave 2 from the original Wave 1 sample.

Measures

We first describe the Wave 1 measures used in model-based cluster analysis (MBC) to evaluate evidence of separable primary and secondary psychopathy subgroups. We then describe the independent, Wave 2 criterion measures used to evaluate the predictive validity of the psychopathy groups identified at Wave 1.

Clustering Measures:

Psychopathic Traits. At Wave 1, parents completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a 20-item rating scale of youth narcissism, CU traits, and impulsivity (Frick et al., 2000). All three facets were used simultaneously as clustering variables. Parents separately rated each item as 0 = not at all true, 1 = sometimes true, or 2 = definitely true. CU traits were estimated from six items including: “Is concerned about how well he/she does at school/work” (reverse scored), “Does not show feelings or emotions,” and “Feels bad or guilty when he/she does something wrong” (reverse scored). The narcissism scale consisted of seven items such as “Uses or ‘cons’ others,” “Teases or makes fun of others,” and “Acts charming.” Impulsivity was estimated from six items including: “Blames others,” “Acts without thinking,” and “Gets bored easily.” Coefficient alphas for the CU traits subscale were .65 and .76 in a community and clinical sample, respectively, and .83 and .85 for narcissism, respectively (Frick et al., 2000). For impulsivity, alphas in a community and clinical sample were .74 and .64, respectively (Frick et al. 2000). Alphas were .60, .80, and .69 for parent ratings of CU traits, narcissism, and impulsivity at Wave 1, respectively.

Anxiety. At Wave 1, children self-reported their anxiety symptoms on the 41-item Screen for Child Anxiety Related Emotional Disorders (SCARED) (Birmaher et al., 1997), which yields five separate subscales: Generalized anxiety disorder (GAD), separation anxiety disorder (SAD), panic disorder, social phobia, and school phobia. Each item was rated as 0 = not true/hardly ever true, 1 = sometimes true, and 2 = often true. The total score and five factors discriminated between anxiety, depressive, and disruptive disorders, and within anxiety disorders (Birmaher et al., 1999). Among clinic-referred children, the total anxiety score had high internal consistency ($\alpha = .93$) and the coefficient alphas of the five subscales ranged from .74 to .89. In this sample,

the coefficient alpha of the total anxiety score (not including school phobia) was .92 and the coefficient alphas of the four subscales ranged from .75 to .89. Given its high frequency in this sample (77% endorsed at least one symptom), we excluded the school phobia scale; the four remaining subscale scores were entered simultaneously and used as clustering variables.

Wave 2 Criterion Measures

Psychopathic Traits. At Wave 2, parents completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), yielding narcissism and impulsivity facets ($\alpha = .71$ and $.64$, respectively). At Wave 2 only, parents completed the Inventory of Callous-Unemotional Traits (ICU), which is psychometrically superior to CU traits derived from the APSD (Kimonis et al., 2008). The ICU consists of 24 items and is comprised of three factors, including callousness (e.g., “I do not care who I hurt to get what I want”), uncaring (e.g., “I always try my best,” reverse scored), and unemotional (e.g., “I express my feelings openly,” reverse scored). Items were rated from 0 = not at all true to 3 = definitely true. The total ICU score demonstrated high internal consistency and convergence with aggression, delinquency, and physiological indices of constricted emotion (Kimonis et al., 2008). In this sample, the alpha for the total score of CU traits at Wave 2 was $.86$. Given their significant inter-correlation (Table 1), we created a total Wave 2 psychopathy score by summing z-scores for CU traits, narcissism, and impulsivity.

Anxiety. At Wave 2, children self-reported their anxiety and depression via the 47-item Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000). The RCADS also includes subscales of DSM-IV panic disorder, obsessive-compulsive disorder (OCD), social phobia, SAD, and GAD. The OCD and depression scales were excluded from analyses given they are separable from other anxiety disorders based on psychobiological correlates and treatment response (Stein et al. 2010; Phillips et al., 2010). Items

were rated as 0 = never, 1 = sometimes, 2 = often, and 3 = always. The internal consistency of the RCADS scales ranged from .73 to .82 in a community sample (Chorpita et al., 2000) and .78 to .88 in a clinic-referred sample (Chorpita, Moffitt, & Gray, 2005). In the current study, the alpha for the total score was .91 and ranged from .75 to .83 for individual subscales.

Antisocial Behavior. At Wave 2, children completed the 32-item Self-Reported Antisocial Behavior (SRA; Loeber, Stouthamer-Loeber, Van Kammen, & Farrington, 1989), a semi-structured interview of youth delinquency and antisocial behavior (e.g., theft, aggression, vandalism). Behaviors in the past 6 months were rated as “never,” “once,” “twice,” or “more often.” Three items, endorsed as “never” in the sample, were excluded, thus yielding 29 items. They were summed to estimate the frequency of youth self-reported antisocial behavior.

Reactive Aggression. At Wave 2, parents completed the 23-item Reactive & Proactive Aggression Questionnaire (RPQ; Raine et al., 2006), yielding separate reactive and proactive scales. The current study examined reactive aggression only. The RPQ is unique insofar as it assesses the underlying motivation for aggression. The reactive scale consists of 11 items (e.g., “gotten angry when others threatened you”) rated on their frequency (0 = never, 1 = sometimes, and 2 = often). This scale correlated significantly with impulsivity, hostility, and social anxiety (Raine et al., 2006); internal consistency in this study was excellent ($\alpha = .89$).

Emotional Processing. Across development, emotion go/no-go is a behavioral task of cognitive control, emotion recognition, and emotion regulation in response to positive (i.e., happy), negative (i.e., sad, fearful, angry), and neutral faces (Tottenham, Hare, & Casey, 2011). Participants quickly identify positive or negative emotions in the presence of a neutral distractor face (i.e., go condition), or detect neutral faces among several emotional distractor faces (i.e., no-go condition). Task performance was sensitive to neural correlates, including differential

activation for top-down prefrontal cognitive systems from subcortical limbic regions for both positive and negative emotions (Somerville, Hare, & Casey, 2010; Tottenham et al., 2011).

Youth were presented with grayscale images of 10 adults (five men, five women) expressing five different emotions (i.e., happy, fearful, sad, angry, and neutral), presented one at a time in the center of a computer screen. Youth were instructed to press a button as quickly as possible when a given facial expression target was presented (e.g., happy, 'go' trial). 'Go' trials comprised the majority of the task (70%) to create a prepotent tendency for the subject to respond. For the less frequent 'no-go' trials (30%), youth were asked to abstain from button pressing for a 'no-go' expression (e.g., fearful face). However, participants were not directly informed of the 'no-go' expression; instead they were instructed to withhold pressing for "any face other than the 'go' facial expression." In each block, each emotional expression was paired with a neutral face, and depending on the block, the emotional expression was either the 'go' or 'no-go' stimulus. The task consisted of eight randomized blocks of go/no-go pairs (i.e., happy-neutral, neutral-happy, sad-neutral, neutral-sad, angry-neutral, neutral-angry, fearful-neutral, neutral-fearful) with 30 randomized trials for each condition. Practice trials were administered to ensure participants understood the task demand.

We examined indices of cognitive control, emotion recognition, and emotion regulation. As in previous studies (Somerville et al., 2010; Tottenham et al., 2011), cognitive control was estimated from the overall false alarm rate and emotion recognition from d-prime, which measures accuracy and accounts for response bias; it was calculated by subtracting the z-transformed false alarm rate (commission errors) from the z-transformed hit rate. Emotion regulation was estimated from impulsive responding in the context of emotional information. Thus, the false alarm rate (i.e., proportion of total possible errors for each type) specific to

emotional ‘no-go’ stimuli was used to estimate emotion regulation. Fewer false alarm rates suggest better cognitive control and emotion regulation. This task was sensitive to cognitive control, emotion recognition, and regulation in the context of positive and negative emotions across development (Tottenham et al., 2011); it also correlated with greater amygdala volume among children exposed to orphanage rearing (Tottenham et al., 2010).

ADHD Symptoms: We controlled for the total number of Wave 1 youth DSM-IV ADHD symptoms from the DISC-IV-P (Shaffer et al., 2000), a fully structured diagnostic interview with parents. The ADHD module demonstrated good psychometric properties, including test-retest reliability ($r = .79$ after 1 year) and internal consistency ($\alpha = .77$ for criterion) for parent ratings in a large community sample (Shaffer et al., 2000).

Data Analytic Plan

To assess evidence for primary and secondary psychopathy subgroups in school-aged children with similarly elevated psychopathic traits, but different levels of anxiety, we employed model-based clustering (MBC). Wave 1 psychopathic traits (i.e., CU traits, narcissism, and impulsivity) and anxiety (i.e., social phobia, generalized anxiety disorder, separation anxiety disorder, panic disorder) were entered as clustering variables in MBC (Kimonis et al., 2012; Skeem et al., 2007; Hicks et al., 2004), which tested the fit of 10 models that differed in their assumptions about data structure and calculated a fit index for each model (Banfield & Raftery, 1993).

First, despite its dimensionality (Edens, Marcus, Lilienfeld, & Poythress, 2006), we adopted the mean (total score of 10) in the normative sample for the APSD to identify children with elevated psychopathy ($n = 76$) for inclusion in the MBC; remaining children were placed in a comparison group ($n = 112$). MBC was performed using the *mclust* Version 4 (Fraley, Raftery,

& Scrucca, 2012) in R: (R Core Team, 2013). Bayesian information criterion values suggested a two-cluster solution best fit the data (primary, $n = 30$; secondary, $n = 46$) with equal shape, volume, and orientation ($BIC = -2228$). Three-cluster solutions were the next best fitting model with variable volume, same shape and orientation ($BIC = -2238$) and with equal shape volume and orientation ($BIC = -2255$). Characteristics for primary, secondary, and comparison groups are provided in Table 2. Relative to the comparison group, children in the elevated psychopathic traits group exhibited more Wave 1 aggression, $t(184) = 10.87, p < .01$; rule-breaking behavior, $t(184) = 9.54, p < .01$; ADHD symptoms, $t(163) = 7.23, p < .01$; ODD symptoms, $t(163) = 6.48, p < .01$; and conduct disorder (CD) symptoms, $t(163) = 4.13, p < .01$, providing key support for these empirically derived groups.

Second, analysis of variance (ANOVA) conducted in Stata Version 14 (StataCorp, 2015) compared comparison, primary, and secondary youth on Wave 2 parent-rated psychopathic traits and reactive aggression as well as youth self-reported anxiety and antisocial behavior (see Table 3). Because we were principally interested in psychopathy subgroup differences, we only reported primary versus secondary differences for significant omnibus tests. Additionally, although we utilized all three psychopathy facets to identify clusters, we only report mean differences on overall psychopathy. Given that ADHD was oversampled in the current study, its strong prediction of conduct problems and psychopathic traits, and that children with ADHD were over-represented in primary and secondary groups, ADHD may influence observed group differences. However, interpretively, using analysis of covariance (ANCOVA) to “control for” group differences is problematic, particularly when the group variable and covariate are correlated. Per Verona & Miller (2015), we first examined psychopathy group differences via ANOVA, then reproduced the model via ANCOVA controlling for Wave 1 ADHD symptoms,

age, and sex. Finally, following Tottenham et al. (2011), we utilized repeated-measures ANOVA for the emotion go/nogo task: The repeated measures were for emotion and stimulus type (i.e., sad-go, sad-nogo, happy-go, happy-nogo, angry-go, angry-nogo, fear-go and fear-nogo). Group membership (i.e., comparison, primary, or secondary) was the between-subject variable, and separate interactions between psychopathy group, stimulus type, and emotion were entered. Wave 1 ADHD symptoms, age, and sex were also controlled.

Results

Wave 1 Correlates of Primary, Secondary, and Comparison Clusters

First, ANOVA tested comparison, primary and secondary groups across Wave 1 measures (Table 2). Primary ($M = 16.55$, $SD = 4.64$; $p < .001$) and secondary groups ($M = 15.84$, $SD = 4.49$; $p < .001$) both had more Wave 1 total psychopathic traits than comparison youth ($M = 6.44$, $SD = 2.46$, [$F(2, 182) = 176.57$, $p < .001$, $\eta^2 = 0.66$]). This was expected given that only youth above the normative sample mean on psychopathic traits were used for MBC; however, the primary and secondary groups were comparable on overall Wave 1 psychopathic traits ($p = .38$). Next, there was a significant omnibus group effect for Wave 1 youth anxiety [$F(2, 147) = 36.59$, $p < .001$, $\eta^2 = 0.33$] with the secondary psychopathy ($M = 33.41$, $SD = 9.81$) being more anxious at Wave 1 than the comparison ($M = 22.50$, $SD = 13.89$; $p < .001$) and primary group ($M = 11.16$, $SD = 5.34$; $p < .001$); the primary group was less anxious at Wave 1 than the comparison group ($p < .001$). Psychopathy group differences were also observed on ADHD diagnosis [$F(2, 161) = 19.76$, $p < .001$, $\eta^2 = 0.20$]; ADHD symptoms [$F(2, 162) = 26.27$, $p < .001$, $\eta^2 = 0.24$]; and ODD symptoms [$F(2, 162) = 22.39$, $p < .001$, $\eta^2 = 0.22$]. That is, across all measures, primary and secondary groups had significantly more disruptive behavior problems than the comparison group, but they did not differ significantly from each other (Table 2).

Criterion Measures at Wave 2

Psychopathic Traits: There was a significant overall effect of Wave 1 group for Wave 2 psychopathic traits [$F(2,106) = 42.01, p < .001, \eta^2 = 0.44$], although the primary ($M = 1.61, SD = 2.28$) and secondary ($M = 1.14, SD = 1.69$) subgroups were comparable [$t(106) = 0.92, p = .36, d = 0.23$] (Table 3). Next, even with control of child sex, age, and Wave 1 ADHD symptoms, $F(2, 102) = 17.70, p < .001, \eta^2 = 0.26$, the same pattern emerged where primary and secondary groups were similar [$t(103) = 0.55, p = .58, d = 0.15$] (Table 4). For clarity, we only report mean differences for overall psychopathy, however, similar patterns were observed for individual psychopathy facets (i.e., CU traits, narcissism, impulsivity) at Wave 2.

Anxiety: Using the same data analytic procedures described above, there was a significant Wave 1 psychopathy group effect on Wave 2 self-reported anxiety [$F(2,126) = 6.18, p < .01, \eta^2 = .09$] where more anxiety was observed in the secondary ($M = 24.04, SD = 12.15$) versus primary group ($M = 13.37, SD = 8.59$) [$t(126) = 3.49, p = .001, d = 1.01$] (Table 3). Once again, even with inclusion of age, sex, and ADHD symptoms, ANCOVA yielded similar results [$F(2, 122) = 4.73, p = .01, \eta^2 = .07$] with more anxiety in the secondary versus primary group [$t(122) = 3.07, p < .01, d = 0.82$] (Table 4).

Self-Reported Antisocial Behavior: There was a significant overall effect of Wave 1 psychopathy group [$F(2,99) = 3.76, p = .03, \eta^2 = .07$] where the secondary group ($M = 7.86, SD = 5.76$) self-reported more Wave 2 antisocial behavior than the primary subgroup ($M = 4.63, SD = 5.54$) [$t(99) = 2.05, p = .04, d = 0.57$] (Table 3). With control of age, sex, and ADHD symptoms, however, this psychopathy group effect became marginal [$F(2,96) = 2.77, p = .07, \eta^2 = 0.05$] (Table 4). However, to facilitate comparison with the ANOVA results, the secondary group reported more antisocial behavior than the primary [$t(96) = 2.07, p = .04, d = 0.59$].

Reactive Aggression: ANOVA revealed a significant overall psychopathy group effect [$F(2, 124) = 5.36, p < .01, \eta^2 = .08$] such that reactive aggression was comparable between the primary ($M = 7.21, SD = 2.53$) and secondary groups ($M = 8.03, SD = 4.04$), $t(124) = 0.86, p = .39, d = 0.24$ (Table 3). With control of age, sex, and ADHD symptoms, the overall psychopathy group effect became marginal [$F(2, 120) = 2.52, p = .08, \eta^2 = 0.04$] with the primary and secondary groups being comparable [$t(120) = 1.04, p = .30, d = 0.27$] (Table 4).

Emotion Recognition and Regulation:

A three-way mixed ANOVA examined the psychopathy groups and the within-subjects factors of emotion (i.e., fear, happiness, sadness, and anger) and stimulus type (i.e., go, nogo) on Wave 2 false alarm rate (Table 5). A marginally significant psychopathy group x emotion x stimulus type interaction was observed [$F(6, 453) = 1.83, p = .09, \eta^2 = .007$] with a significant emotion x stimulus interaction [$F(3, 435) = 3.634, p = .01, \eta^2 = .007$] and a marginal stimulus x psychopathy group interaction [$F(2, 151) = 2.82, p = .06, \eta^2 = .005$]. Main effects of emotion [$F(3, 453) = 72.78, p < .001, \eta^2 = .154$] and stimulus [$F(1, 151) = 75.82, p < .001, \eta^2 = .067$] were also observed. To facilitate direct comparison with ANCOVA results below, we probed the interaction by examining the psychopathy group x emotion interaction separately for the go and nogo conditions. There was a significant psychopathy group x emotion interaction [$F(6, 438) = 2.20, p = .04, \eta^2 = .03$] in the go condition, but not in the no-go condition [$F(6, 426) = 1.39, p = .22, \eta^2 = .018$]. There were no psychopathy group differences on false alarm rate for angry [$F(2, 151) = 0.02, p = .98, \eta^2 < .01$] fearful [$F(2, 151) = 0.02, p = .98, \eta^2 < .01$], happy [$F(2, 151) = .10, p = .13, \eta^2 = .03$], or sad faces in the go condition [$F(2, 151) = .13, p = .10, \eta^2 = .03$].

Next, we replicated the previous model but added additional between-subject factors of ADHD symptoms, age, and sex. There was a significant psychopathy group x emotion x

stimulus type interaction [$F(6, 438) = 2.67, p = .015, \eta^2 = .014$], a significant stimulus x sex interaction [$F(6, 438) = 8.20, p < .01, \eta^2 = .009$], as well as a main effect of stimulus [$F(1, 146) = 4.40, p = .038, \eta^2 = .005$] (Table 5). We probed the three-way interaction by examining the psychopathy group x emotion interaction separately for the go and nogo condition, controlling for ADHD symptoms, age, and sex. In the go-condition, there was a significant psychopathy group x emotion interaction [$F(6, 438) = 2.198, p = .04, \eta^2 = .029$], but not in the no-go condition [$F(6, 438) = 1.482, p = .18, \eta^2 = .019$] (see Figure 1). Next, there were no psychopathy group differences on false alarm rate for angry [$F(2, 146) = 0.104, p = .90, \eta^2 = .001$], fearful [$F(2, 146) = 0.341, p = .712, \eta^2 = .005$], or happy faces in the go condition [$F(2, 146) = 0.12, p = .10, \eta^2 = .03$]. However, as illustrated in Figure 1, psychopathy groups differed on their false alarm rate in response to sad faces, [$F(2, 146) = 4.055, p = .02, \eta^2 = .05$]. Specifically, the secondary group ($M = 0.43, SD = 0.20$) had fewer false alarm rates than the primary group ($M = 0.53, SD = 0.27$) [$t(87) = -2.14, p < .05$] in the go condition. Finally, using the same data analytic procedures, neither ANOVA nor ANCOVA yielded significant main effects or interactions for hit rate, percentage correct, and d-prime for psychopathy groups.

Discussion

This study explored evidence for primary and secondary subgroups of youth psychopathy among 221 school-aged children with and without ADHD. We also tested if subgroups predicted anxiety, psychopathic traits, antisocial behavior, and emotional processing two years later. Primary and secondary groups were comparable on levels of overall psychopathic traits and reactive aggression, but the secondary group self-reported more anxiety and antisocial behavior two years later relative to the primary group. Finally, on an emotion go/nogo task, the secondary

group also exhibited better cognitive control (i.e., fewer false alarms) in the context of emotion, relative to primary and comparison groups.

Consistent with the adolescent literature, secondary youth engaged in significantly greater antisocial behavior than primary youth in our study (Fanti et al., 2013; Kahn et al., 2013; Salihovic et al., 2014). These findings diverge from evidence with adults where antisocial behavior is consistently similar in primary and secondary groups (Skeem et al., 2007; Swogger & Kosson, 2007). Differences in severity may underlie these inconsistent results given that most adult studies employed samples of incarcerated men, whereas child studies diversely employed community, clinical, and adjudicated youth. Next, primary and secondary subgroup differences in antisocial behavior in childhood may attenuate by adulthood, perhaps reflecting unique presentations across developmental periods. However, the current study utilized self-reported antisocial behavior, which may critically reflect psychopathy subgroup differences in endorsement of, rather than engagement in, antisocial behavior. Multi-method and multi-informant designs will be needed for future studies to clarify how onset and engagement in antisocial behavior may differ in primary and secondary psychopathy groups. Finally, antisocial behavior is heterogeneous and sensitive to development, consisting of separable behavioral (i.e., aggression, property violations) and affective (i.e., oppositionality) facets (Frick et al., 1993). Secondary youth may engage in more affective expressions of negative behavior (e.g., temper tantrums, arguing), whereas primary youth may engage more in the behavior domain (e.g., spiteful, blames others). Future studies must properly attend to meaningfully differences in the phenomenology of antisocial behavior.

Across diverse samples, primary psychopathy youth were consistently reported to be less negatively emotional whereas the secondary group typically exhibited poor emotion regulation

and internalizing symptoms (Fanti et al., 2013; Kahn et al., 2013; Kimonis et al., 2012; Salihovic et al., 2014). However, the current study is one of the first to detect separable groups among a community sample of school-aged children. Previously, the emotion dysregulation characteristic of secondary psychopathy was diversely assessed, ranging from trait based (e.g., negative emotionality) to DSM-based designations. The current study used DSM-IV anxiety disorder scales, but other approaches may be heuristic, including fear versus worry and negative affect versus physiological arousal (Brown, Chorpita, & Barlow, 1998). Furthermore, it is possible that dysregulation may be sensitive to development, thus necessitating careful measurement strategies of unique constructs (e.g., temperament) relative to models based on adults. These more refined approaches to anxiety may improve traction on youth psychopathy subgroups.

The current study provides further evidence of the heterogeneity of psychopathy. Although psychopathic traits identify children at risk for more severe and persistent conduct problems (Cooke & Michie, 2001; Hemphill et al., 1998; Salekin, 2008), they still vary widely in their clinical presentation including anxiety and emotion dysregulation (Fanti et al., 2013; Kahn et al., 2013; Kimonis et al., 2012; Salihovic et al., 2014). Crucially, improving traction on psychopathy heterogeneity is necessary to advance understanding of causal influences (Kimonis et al., 2012). Early formulations posited that primary (e.g., genetic) and secondary (e.g., trauma) psychopathy reflected different causal influences (Karpman, 1941; Skeem et al., 2003). Although retrospective studies revealed more trauma/abuse among the secondary group compared to primary psychopathy (Kahn et al., 2013, Kimonis et al., 2012; Poythress, Skeem, & Lilienfeld, 2006; Vaughn et al., 2009), in the only published twin study to date, genetic and non-shared environmental influences were similar for both primary and secondary subgroups (Hicks et al.,

2012). Further characterization of risk factors using genetically informed designs (e.g., mediators of genetic influence) is required to further prosecute the validity of subgroups in children.

Reflecting its centrality, psychopathic adults exhibited diminished emotional responsiveness compared to controls across psychophysiological, behavioral, neural, and self-report data (Brook, Brieman, & Kosson, 2013). Similarly, children with elevated psychopathic traits exhibited impaired recognition and reduced sensitivity to negative emotional stimuli than other antisocial youth, especially in response to empathy. Specifically, children with psychopathic traits exhibit deficient processing of distress cues (i.e., fear, sadness, pain) and impaired recognition of fearful and sadness expressions (Blair, 2013). But, few studies examined individual differences in emotional processing in primary and secondary subgroups of psychopathy with children – that is, children with similar elevations in psychopathic traits, but who are distinguishable based upon the presence of anxiety. In this study, children in the secondary subgroup demonstrated fewer false alarms, and thus better cognitive control, in response to sad faces relative to primary or comparison youth. It is somewhat surprising that primary youth performed similarly to the comparison group, especially given their similar elevations in psychopathic traits compared to secondary youth. However, the pattern among the secondary subgroup is consistent with emerging evidence among adolescents and adults: Using a dot-probe task, adolescent high-anxious psychopaths were more engaged toward distressing emotional stimuli than low-anxious psychopathic variants (Kimonis et al., 2012). Yet, in this study, primary and comparison youth performed similarly on all measures of emotional processing, despite differences on antisocial behavior, reactive aggression, and psychopathic traits. These preliminary findings suggest that the secondary subgroup may demonstrate hypervigilance to threat cues. Early conceptualizations of psychopathy proposed that the anxiety

in the secondary subgroup may develop secondary to environmental influences, such as maltreatment. If this is the case, the secondary subgroup may benefit from trauma-focused interventions, such as trauma-focused cognitive behavioral therapy.

Despite methodological strengths (e.g., multiple-informant, prospective design), we also emphasize important study limitations herein. First, as mentioned above, the measurement of emotion dysregulation exhibited by the secondary subgroup has varied widely across studies. We employed DSM-IV anxiety disorders whereas measures of emotional reactivity, or similar research domain criteria (RDoC), may improve traction on the nature of negative emotionality evident in the secondary subgroup. Second, we aimed to characterize primary and secondary youth in a population sample of young school children, whose psychopathic traits were expectedly lower relative to adjudicated samples. However, individuals in the psychopathic groups were distinguishable from comparison youth on several key variables, such as greater levels of conduct problems and aggression. Third, we used different measures of anxiety and psychopathic traits at Wave 1 (i.e., SCARED & APSD) and Wave 2 (RCADS & ICU); however, we contend that having different measures of the same construct strengthens construct validity (Campbell & Fiske, 1959). Fourth, we incorporated a self-report measure of antisocial behaviors. Previous studies have indicated that the use of self-report measures among individuals with elevated psychopathic traits may be problematic (e.g., underestimates) (Salekin & Kubak, 2009). In the present study, the secondary subgroup reported significantly more antisocial behavior than their primary counterparts, however it is unclear if this finding reflects a difference in genuine antisocial engagement, or a difference in willingness to report antisocial behavior.

Using a community sample of school-aged children, we detected primary and secondary subgroups of psychopathy who demonstrated similarly elevated psychopathic traits, but were

differentiated based upon high and low levels of anxiety. Furthermore, these groups exhibited key differences on various measures two years later: Although the primary and secondary groups were comparable on reactive aggression and psychopathic traits two years later, the secondary group engaged in more antisocial behavior and experienced more anxiety two years later. Additionally, the secondary group demonstrated greater cognitive control on an emotion go/nogo task compared to primary and comparison groups. Despite evidence of unique groups of psychopathy across child, adolescent, and adult samples, current causal models of psychopathy do not account for this heterogeneity. Thus, future studies must incorporate prospective designs using child samples to assess unique risk factors for primary and secondary psychopathy. Identification of risk factors and causal processes are necessary to improve intervention development and delivery.

CHAPTER 2: CORRELATES OF PRIMARY AND SECONDARY PSYCHOPATHY IN A POPULATION-BASED SAMPLE OF ADULTS

Abstract

Distinguished by anxiety and negative emotionality, primary and secondary psychopathy are theorized to have distinct risk factors and unique correlates: Whereas primary psychopathy reflected genetically-influenced affective disturbance (e.g., callousness) and minimal negative emotionality (e.g., neuroticism, anxiety), secondary psychopathy emerged secondary to environmental adversity (e.g., maltreatment) and consisted of impulsivity, poor emotion regulation, and internalizing symptoms. Among adolescents and adults, Empirical examinations of primary and secondary psychopathy typically employ samples of incarcerated adults and adolescents, and characterizations of psychopathy subgroups in relation to antisocial behavior and aggression have been inconsistent. The current study examined evidence of primary and secondary subgroups in a nationally representative population-based sample by examining their association with key correlates (i.e., maltreatment, delinquency, antisocial behavior). Participants included 15,140 adolescents (age 12-20) followed prospectively for 14 years across waves of data collection as part of the National Longitudinal Study of Adolescent Health. Psychopathy subgroups were derived from self-reported personality-derived psychopathy and negative emotionality at Wave IV. Secondary subgroups reported more diverse forms of maltreatment and engaged in greater nonviolent delinquency and non-violent antisocial behavior compared to primary psychopathy. During adolescence, primary and secondary engaged in similar levels of violent delinquency, however in adulthood, the secondary group reported more violent antisocial behavior than primary psychopathy. Implications of these findings for causal theories of

psychopathy are discussed, as well as future directions to clarify the development of psychopathy subgroups.

Although psychopathy was initially conceptualized unidimensionally, consisting principally by the absence of emotional experience (Cleckley, 1976), its heterogeneity is illustrated by factor analytic evidence of separable interpersonal (e.g., manipulation, egocentricity), affective (e.g., lack of remorse, shallow affect), and behavioral (e.g., impulsivity, antisociality) facets. Furthermore, an influential distinction between high-anxious (i.e., secondary) and low-anxious (i.e., primary) subtypes of psychopathy was proposed: Whereas primary psychopathy reflected genetically-influenced affective disturbance (e.g., callousness) and minimal negative emotionality (e.g., neuroticism, anxiety), secondary psychopathy emerged secondary to environmental adversity (e.g., maltreatment) and consisted of impulsivity, poor emotion regulation, and internalizing symptoms (Karpman, 1941, 1948; Lykken, 1995; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003; Skeem, Johansson, Andershed, Kerr, & Louden, 2007). To date, examinations of primary and secondary psychopathy typically utilize clinic-referred and adjudicated samples, thus limiting their generalizability. To improve traction on the validity of this potential distinction, primary and secondary psychopathy must be rigorously evaluated with respect to different causal influences. This is particularly true with respect to expansion into population-based samples and to discern whether these subgroups may necessitate different interventions. For example, although psychopathy predicts criminal recidivism (Hemphill, Hare, & Wong, 1998), Karpman (1948) theorized that secondary psychopathy would be more amenable to treatment. Thus, reducing heterogeneity in psychopathy through primary and secondary subtypes of psychopathy requires empirical validation given its potential to facilitate innovations in prevention and intervention, a key consideration given that psychopathy is highly intractable to available interventions (Salekin, 2008).

Consistent with Karpman's (1941) conceptualization, across community, clinic-referred, and adjudicated samples, anxiety and negative emotionality differentiated two subgroups of adolescents and adults with comparably elevated psychopathic traits (Hicks, Markon, Patrick, Krueger, & Newman, 2004; Newman, MacCoon, Vaughn, & Sadeh, 2005; Skeem et al., 2007; Swogger & Kosson, 2007; Vassileva, Kosson, Abramowitz, & Conrod, 2005). Secondary psychopathy in adulthood was correlated with greater anxiety, negative emotionality, and stress reaction and lower control than primary psychopathy. Similar patterns were observed among adolescents: High-anxious variants of psychopathy were more negatively emotional, socially withdrawn, and aggressive than low-anxious psychopathy subtypes (Fanti, Demetriou, & Kimonis, 2013; Kahn, Youngstrom, Youngstrom, Feeny, & Findling, 2013; Salihovic, Kerr, & Stattin, 2014). Central to the current study, as noted previously, examinations of primary and secondary subtypes typically employed clinic-referred or incarcerated samples, despite a long tradition of using naturally-occurring individual differences in key constructs (e.g., fearlessness, trait anxiety) to characterize psychopathic traits (Levenson, Kiehl, & Fitzpatrick, 1995; Lykken, 1982).

Distinctions between primary and secondary psychopathy with respect to aggression and antisocial behavior are inconsistent across samples of adolescents and adults. Among incarcerated adults, primary and secondary subgroups were similarly antisocial (Skeem et al., 2007) whereas self-reported externalizing psychopathology and aggression during incarceration were higher among the secondary group in multiple adjudicated samples (Cox, Edens, Magyar, Lilienfeld, Douglas, & Poythress, 2013; Poythress et al., 2010). Conversely, rates of violence were higher among primary relative to secondary psychopathy in a community-based sample of Finnish soldiers (Driscoll et al., 2014). However, across adjudicated, clinic-referred, and

community samples of adolescents, secondary psychopathy was consistently more aggressive and delinquent, and exhibited higher rates of total delinquency, violence, and property offenses than primary psychopathy youth (Fanti et al., 2013; Kahn et al., 2013; Salihovic et al., 2014; Vaughn, Edens, Howard, & Smith, 2009). Potential sources of these inconsistent patterns of antisocial behavior in primary and secondary psychopathy include sampling differences (e.g., incarcerated versus clinic-referred) and reliance on undifferentiated measures of antisocial behavior, which ignore key distinctions such as overt versus covert behaviors. To better characterize naturally occurring individual differences in psychopathy (Poythress & Skeem, 2006), community-based samples of adults are needed to prosecute the underlying architecture of psychopathy, potential subtypes, and putative differences. Given important empirical differences among different forms of antisocial behavior (e.g., violence, property destruction), greater attention must be paid to how this construct is operationalized. For example, primary, but not secondary, psychopathy was positively correlated with relational aggression among college students (Vaillancourt & Sunderani, 2011). To address this gap, we examined differences on violent and non-violent delinquency and antisocial behavior among primary and secondary psychopathy using a population-based sample of adolescents followed prospectively into adulthood.

Early theories of primary and secondary psychopathy were based primarily on clinical observations and emphasized maltreatment as a specific risk factor for the development of secondary, but not primary, psychopathy (Karpman, 1941; Lykken, 1995; Porter, 1996; see Poythress & Skeem, 2006 and Skeem et al., 2003 for a review). When treated unidimensionally, psychopathic traits are consistently sensitive to early maltreatment (Campbell, Porter, & Santor, 2004; Dargis, Newman, & Koenigs, 2016; Gao, Raine, Chan, Venables, & Mednick, 2010;

Krischer & Sevecke, 2008; Lang, Klinteberg, & Alm, 2002), but previous studies rarely accounted for co-occurring antisocial behavior. Given that maltreatment similarly predicts antisocial behavior, failure to control for co-occurring antisociality may confound associations with psychopathic traits. That is, it is unclear whether predictions of adult psychopathic traits from maltreatment may partially be explained by co-occurring aggression and antisocial behavior; this is particularly relevant to the majority of previous studies, which has typically employed incarcerated populations. To clarify specificity of predictions of psychopathy from maltreatment, controlling for antisocial behavior, community-based samples should be prioritized.

The putative role of maltreatment in the development of psychopathic traits is especially relevant to secondary psychopathy given that these behaviors were believed to be an emotional adaptation to maltreatment (Karpman, 1941; Skeem et al., 2003). Several studies implicated maltreatment as a risk factor for secondary, but not primary, psychopathy. Based on retrospective recall among incarcerated male adults, maltreatment was unrelated to the interpersonal/affect dimension with control of the behavior/antisocial dimension (Dargis et al., 2016), thus suggesting that maltreatment may be more specifically associated with behavioral (relative to interpersonal and affective) aspects of psychopathy. Similarly, among adults in prison or court-ordered residential drug treatment facilities, abuse history was unrelated to affective or interpersonal dimensions of psychopathy, but was positively associated with the impulsive/irresponsible dimension of psychopathy (Poythress, Skeem, & Lilienfeld, 2006). Although these studies did not evaluate primary and secondary psychopathy per se, they suggest that maltreatment may specifically predict impulsivity and antisociality, which critically underlie secondary psychopathy. Among incarcerated and clinic-referred psychopathic youth, individuals

in the secondary subgroup reported more traumatic experiences, PTSD symptoms, physical abuse, and sexual abuse compared to the primary subgroup and a low-psychopathic antisocial comparison group (Kahn et al., 2013; Kimonis, Skeem, Cauffman, & Dmitrieva, 2011; Kimonis et al., 2012; Vaughn et al., 2009). In an exception, high-anxious and low-anxious subgroups clustered using CU traits were comparable in emotional or physical abuse; however, primary psychopathy was positively associated with more emotional and physical neglect compared to secondary subgroups (Kimonis, Fanti, Isoma, & Donoghue, 2013). Overall, there is evidence that maltreatment is a risk factor for psychopathy in general and perhaps the behavioral/antisocial facet specifically. However, few studies have examined its relation to primary and secondary subgroups, and all employed incarcerated samples. The current study aimed to explore differences in maltreatment among primary and secondary subgroups of psychopathy using a population-based sample of adolescents followed prospectively across 14 years.

Although there is replicated evidence of primary and secondary subgroups of psychopathy among incarcerated adults, it is unknown if these subgroups are evident in a nationally representative population-based sample. Using the National Longitudinal Study of Adolescent Health, a population-based sample of 15,701 adolescents followed prospectively over 14 years, the current study examined whether two subgroups with similar levels of elevated psychopathy, but who were separable on negative emotionality, differed on key correlates (i.e., maltreatment, violent and non-violent delinquency and antisocial behavior). Specifically, we predicted that the secondary subgroup would report more diverse forms of maltreatment than the primary psychopathy group, even with control of co-occurring antisocial behavior. We also predicted that the secondary group would engage in greater delinquency during adolescence and greater antisocial behavior in early adulthood compared to primary psychopathy.

Method

Participants

The National Longitudinal Study of Adolescent Health (Add Health, Harris, 2013) recruited a nationally representative sample of adolescents in grades 7-12 from 80 high schools selected to be representative of U.S. high schools on region, urbanicity, size, and ethnicity. The sample was followed prospectively across 14 years, and data was collected via an in-school questionnaire and four at-home interviews. At Wave I, 20,745 adolescents were interviewed during the 1994-1995 school year (47.5% male, aged 12 – 20 years). The Wave II follow-up was conducted with 14,738 adolescents two years later. The Wave III follow-up was conducted 7-8 years post-baseline and included 15,197 participants (1,507 partners of original participants were added to the sample). Finally, a fourth wave of data collection (Wave IV) occurred in 2007-2008 and included 15,701 participants aged 24-32 years. At Wave IV, 15,140 participants were genotyped for multiple functional polymorphisms. Lastly, the genetic sample was ethnically diverse (57.5% Caucasian, 14.3% Hispanic, 18.1% African-American, 7.4% Asian, 1.7% Native American, and 0.9% “Other”). More details of the study are available at <http://www.cpc.unc.edu/projects/addhealth>.

Measures

Psychopathic Traits. As outlined by Lynam et al. (2005), psychopathic traits can be reliably derived from five-factor model (FFM; Johns & Srivastava, 1999) personality dimensions, which were administered at Wave IV. This method was previously used to characterize dimensions of psychopathy in Add Health (Beaver, Barnes, May, & Schwartz, 2011; Beaver, Rowland, Schwartz, & Nedelec, 2011). Briefly, personality items that paralleled items from previous studies of FFM psychopathy were selected. These items were then factor

analyzed, with poorly loading items being removed. A total of 23 items that loaded significantly onto a scale of total psychopathy yielded coefficient alphas exceeding .80 (see Appendix A). Items were measured on a 5-point Likert scale (1 = strongly agree, 5 = strongly disagree) and include: “I feel others’ emotions,” “I get angry easily,” “I am not interested in other people’s problems,” and “I live my life without much thought for the future.” This psychopathy scale previously predicted arrest, incarceration, and probation status (Beaver, Boutwell, Barnes, Vaughn & DeLisi, 2015; Beaver, Vaughn, DeLisi, Barnes, & Boutwell, 2011).

Negative Emotionality. Add Health personality items were used previously to assess neuroticism (Baldasaro, Shanahan, & Bauer, 2013; Ferguson, Muñoz, Winegard, & Winegard, 2012); however, many of these items were used to construct the aforementioned psychopathy scale. To avoid inflating associations secondary to shared items, we estimated negative emotionality using Wave IV items demonstrating face validity with the stress reaction and alienation subscales of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982) (see Appendix B). Items were measured on a 4-point Likert scale (1 = never or rarely, 4 = most of the time or all of the time) and included: “You felt that people disliked you during the past seven days,” “In your day-to-day life, how often do you feel you have been treated with less respect or courtesy than other people,” “You were bothered by things that usually don’t bother you,” “You could not shake off the blues, even with help from your family and your friends.” An exploratory factor analysis revealed the seven items loaded onto a single factor, which significantly correlated with Wave I and Wave IV depressive symptoms measured at Wave I and Wave I neuroticism ($p < .001$). The alpha for the scale was acceptable ($\alpha = .69$).

Maltreatment. Maltreatment experienced prior to the 6th grade was retrospectively examined at Wave III. Six items assessed neglect, physical abuse, sexual abuse, and social

services involvement, and included: “By the time you started 6th grade, how often had your parents or other adult caregiver not taken care of your basic needs, such as keeping you clean or providing food or clothing,” “How often had your parents or adult caregiver slapped, hit, or kicked you,” and “How often did a parent or other adult caregiver touch you in a sexual way, force you to touch him or her in a sexual way, or force you to have sexual relations,” An item was scored as positive if it was reported to occur at least once on the maltreatment questionnaire (Haberstick et al., 2005; 2014; 2016); sum scores were created from the six dichotomized (presence versus absence) maltreatment items. 47.4% of participants reported no maltreatment, 29.8% reported at least one episode, and 23.8% reported more than one episode. Maltreatment occurring prior to 6th grade measured retrospectively at Wave III predicted adolescent conduct disorder, adult antisocial behavior, and adult violent convictions (Haberstick et al., 2014), as well as ADHD symptoms, but only among women (Li & Lee, 2012).

Delinquency and Antisocial Behavior. At Wave I (ages 12-20) and Wave IV (ages 24-32), participants were asked multiple questions assessing their involvement in various delinquent and antisocial behaviors within the past year. As done previously (Haynie & South, 2005; Mears, Cochran, & Beaver, 2013; Beaver, Connolly, Schwartz, Boutwell, Barnes, & Nedelec, 2016), three scales of delinquency (i.e., nonviolent, violent, and total) were created from a total of 15 items at Wave I and 11 items at Wave IV (see Appendix C). Because delinquency is a term reserved for underage youth, we use the term delinquency to describe the Wave I measure and antisocial behavior to describe Wave IV. The nonviolent delinquency scale included nine items assessing whether participants engaged in property damage, graffiti, theft, and drug sales at Wave I; at Wave IV five items assessing similar non-violent antisocial behaviors were used. Violent delinquency/antisocial behavior was measured at Wave I and Wave IV using six items

assessing the frequency in which participants shot or stabbed another individual, pulled a knife or gun, engaged in a group fight, threatened an individual with a weapon, or engaged in a serious physical fight. Responses on each item were dichotomized based on the absence or engagement in any activity at least once. Contact with drug-using peers was positively associated with Add Health delinquency, whereas self-control was inversely associated with delinquency (Boutwell & Beaver, 2008).

Data Analytic Plan

Although previous studies of youth psychopathic traits have incorporated model-based cluster (MBC) analytic approaches to derive primary and secondary subgroups (see Kimonis et al., 2012 for an example), MBC is not supported in survey data analysis (StataCorp, 2013). Thus, derivation of psychopathy subgroups necessitated different procedures: Given reliable differences on negative emotionality in primary and secondary psychopathy, participants were categorized as either demonstrating elevations on psychopathy and/or negative emotionality, using an 80th percentile cutoff for both measures (i.e., score of 68 or higher on psychopathy, score of 12 or higher on negative emotionality). Participants scoring below the 80th percentile on psychopathy were designated as comparison youth (n = 11,935). Individuals demonstrating elevated psychopathy but who were below the negative emotionality cutoff were labeled as primary psychopathy (n = 1,706); individuals scoring about the cutoffs for psychopathy and negative emotionality were labeled as secondary psychopathy (n = 1,941). Because secondary psychopathy reflected underlying impulsivity, anxiety, depression, or trauma relative to primary psychopathy (Karpman, 1941), to contextualize primary, secondary, and comparison youth, we examined group differences on key correlates (e.g., previous DSM diagnoses, arrest history, college completion, total psychopathy), which is summarized in Table 6 and discussed below.

For continuous outcomes, we employed a linear regression model with dummy coded psychopathy subgroups as predictors. For dichotomous outcomes, psychopathy subgroups were dummy coded and entered into a logistic regression model.

Next, to examine the association of psychopathy group membership with respect to maltreatment at Wave III, and total, violent, and nonviolent delinquency at Wave I and antisocial behavior at Wave IV, we employed negative binomial regression in Stata 14 (StataCorp, 2015). Given that all outcomes were zero-inflated, over-dispersed, count variables, negative binomial regression is an appropriate data analytic strategy. Psychopathy groups were dummy coded with secondary psychopathy as the comparison group; all models controlled for race-ethnicity and participant sex. Because maltreatment is a risk factor for both psychopathy and antisocial behavior, to improve specificity, Wave IV total antisocial behavior was controlled in predictions of psychopathy subgroups from maltreatment.

Results

Validation of Primary and Secondary Subgroups

To contextualize primary, secondary, and comparison groups, we examined differences on key correlates (e.g., psychopathology, arrest history, college completion, total psychopathy) (Table 6). First, we regressed total psychopathy onto dummy coded psychopathy subgroups in a linear regression model. Primary ($M = 71.42$, $SD = 3.60$) and secondary ($M = 73.36$, $SD = 4.89$) groups scored higher on total psychopathy than the comparison group ($M = 58.76$, $SD = 5.50$; $p < .001$ for both comparisons); secondary psychopathy also reported greater overall psychopathy than the primary group ($p < .001$).

Using logistic regression, the secondary psychopathy subgroup exhibited higher prevalence of depression, anxiety disorder, and PTSD diagnoses compared to primary

(depression: OR = 3.56, CI = 0.14 to 0.22; anxiety: OR = 2.85, CI = 2.17 to 3.75; PTSD: OR = 3.83, CI = 2.28 to 6.43) and comparison groups (depression: OR = 4.29, CI = 3.65 to 5.03; anxiety: OR = 3.26, CI = 2.77 to 3.83; PTSD: OR: 3.34, CI: 2.59 to 4.32), which is consistent with theoretical and empirical expectations. Primary (OR = 1.28, CI = 1.09 to 1.49) and secondary psychopathy subgroups (OR = 1.47, CI = 1.26 to 1.71) were also more likely to have at least one arrest relative to the comparison group; no differences were observed between primary and secondary psychopathy. Lastly, individuals in the comparison group were more likely to have a college degree than primary (OR = 1.849, CI = 1.53 to 2.24) or secondary individuals (OR = 2.76, CI = 2.25 to 3.40), and the primary group was more likely to have a degree than secondary psychopathy (OR = 1.49, CI = 1.15 to 1.95). Consistent with earlier examinations (Fanti et al., 2013; Kahn et al., 2013; Kimonis, Frick, Cauffman, Goldweber, & Skeem, 2012; Salihovic et al. 2014), the secondary subgroup exhibited more internalizing psychopathology compared to primary and comparison groups, and similar rates of arrest with primary psychopathy. Thus, we contend that these groups are likely valid representations of primary and secondary psychopathy in a nationally-representative sample.

Wave I Delinquency

Controlling for race-ethnicity and participant sex, secondary psychopathy exhibited greater total delinquency compared to both comparison ($B = 0.29$, $SE = 0.03$, $p < .001$) and primary groups ($B = 0.11$, $SE = 0.05$, $p = .03$) (Table 7). To improve interpretability, we also calculated incidence rate ratios (IRR) for each predictor. Total delinquency in the past year at Wave I for the secondary group increased by a factor of 1.12 compared to the primary group and 1.34 compared to the comparison group. That is, the rate for engagement in delinquency across the past year increased by 1.12 and 1.34 for individuals in the secondary group compared to

primary and comparison youth, respectively. We also examined differences on violent and non-violent delinquency among psychopathy groups while controlling for race-ethnicity and sex. As shown in Table 8, secondary psychopathy demonstrated greater nonviolent delinquency compared to both primary ($B = 0.15, SE = 0.06, p < .01$) and comparison groups ($B = 0.24, SE = 0.04, p < .001$). Past year nonviolent delinquency for the secondary group increased by 1.28 compared to the comparison and 1.16 compared to the primary group. Primary and secondary groups were similar for past year violent delinquency ($B = 0.06, SE = 0.06, p = .33$), but secondary youth demonstrated greater delinquency than comparison youth ($B = 0.39, SE = 0.03, p < .001$) (see Table 9). The IRR for secondary psychopathy was 1.47 relative to the comparison group.

Wave IV Antisocial Behavior

Participants' past year engagement in antisocial behaviors was assessed when participants were aged 24 – 32 years of age. Controlling for race-ethnicity and sex, secondary youth exhibited greater total antisociality at Wave IV than comparison ($B = 1.07, SE = 0.08, p < .001$) or primary youth ($B = 0.70, SE = 0.15, p < .001$) (Table 7). Total past year antisociality at Wave IV for the secondary group increased by a factor of 2.01 compared to the primary group and 2.90 compared to the comparison group. Secondary psychopathy also demonstrated greater nonviolent and violent antisociality compared to both primary ($B = 0.74, SE = 0.16, p < .001; B = 0.67, SE = 0.18, p < .001$) and comparison youth ($B = 0.99, SE = 0.10, p < .001; B = 1.13, SE = 0.11, p < .001$) (Tables 8 and 9). Past year nonviolent antisociality for the secondary group increased by 2.69 compared to the comparison and 2.09 compared to the primary group. With regard to violent antisocial behaviors, the IRR for secondary psychopathy was 3.10 relative to the comparison group and 1.95 compared to primary youth.

Maltreatment Prior to 6th Grade

At Wave III, participants retrospectively reported maltreatment perpetrated by parents or adult caregivers prior to the 6th grade. With control of race-ethnicity, sex, and Wave IV total delinquency, secondary youth retrospectively reported more diverse forms of maltreatment than primary ($B = 0.28, SE = 0.06, p < .001$) or comparison youth ($B = 0.25, SE = 0.05, p < .001$) (Table 10). Maltreatment experienced by secondary youth increased by a factor of 1.32 compared to the primary and 1.29 for comparison youth.

Discussion

Using a population-based sample of adolescents followed prospectively over 14 years, the current study aimed to examine differences among primary and secondary groups with regard to their delinquency, antisocial behavior and maltreatment. Psychopathy subgroups were derived using a psychopathy measure derived from five-factor model personality items and a negative emotionality scale created from items resembling the MPQ negative emotion subscale. Consistent with evidence from adolescents and adults (Cox et al., 2013; Fanti et al., 2013; Kahn et al., 2013; Poythress et al., 2010; Salihovic et al., 2014), the secondary group exhibited greater total delinquency and antisocial behavior than the primary group at Wave I (ages 12-20) and Wave IV (ages 24-32). Crucially, the current study also examined patterns of association with specific forms of antisociality (i.e., nonviolent versus violent). Across adolescence and adulthood, the secondary group engaged in more nonviolent delinquency and antisocial behavior than the primary group. With regard to violence, however, primary and secondary groups exhibited similar engagement at Wave I, but the secondary group exceeded the primary group on Wave IV violent antisocial behavior. As expected, primary and secondary groups each engaged in more total, violent, and nonviolent delinquency and antisocial behavior than the comparison

group at both waves. Lastly, with control for co-occurring adult antisocial behavior, we examined whether maltreatment differed across primary and secondary psychopathy. Consistent with theoretical expectations (Karpman, 1941; Lykken, 1995; Skeem et al., 2003), the secondary group reported greater maltreatment than primary or comparison groups.

Secondary psychopathy exhibited greater overall and nonviolent delinquency and antisocial behavior compared to primary psychopathy during adolescence and early adulthood. However, psychopathy subgroups varied in their levels of violent delinquency and antisocial behavior across development: Whereas primary and secondary youth endorsed similar levels of violent delinquency during adolescence, the secondary group engaged in significantly more violent antisocial behavior than primary youth during adulthood. These patterns diverge from previous studies of adolescents and adults wherein greater aggression and antisociality was evident among secondary relative to primary psychopathy in adolescence (Fanti et al., 2013; Kahn et al., 2013; Salihovic et al., 2014) but similar during adulthood (Skeem et al., 2007). First, with regard to adolescence, inconsistencies may reflect use of separable constructs such as aggression, antisociality, and violent and non-violent delinquency. In the current study, violent delinquency and antisocial behavior required evidence that an individual was severely injured, thus constituting a more severe form of aggression than previous studies (Kahn et al., 2013; Kimonis et al., 2012). We contend that primary and secondary youth may exhibit similar levels of severe violent aggression, but diverge on other aspects of antisociality (e.g., verbal aggression, relational aggression, covert aggression).

Second, as noted previously, evidence of adult primary and secondary psychopathy is based largely on samples of incarcerated adults (Poythress & Skeem, 2006; see Lee & Salekin, 2010 for an exception), significantly hampering generalizability and thus underscoring the need

for population-based methods. Early conceptualizations of psychopathy included descriptions of high-functioning, “successful” psychopaths characterized by egocentricity, superficial charm and irresponsibility, but without arrests or convictions (Gao & Raine, 2010, Cleckley, 1976).

Furthermore, successful psychopaths exhibit similar characteristics as primary psychopathy, such as impaired affective qualities (e.g., glib, superficial, pathological lying, manipulative) and fewer antisocial facets of psychopathy (e.g., poor behavior controls, early behavior problems, and juvenile delinquency) (Gao & Raine, 2010). By virtue of using a population-based sample, the current study likely included higher functioning individuals with psychopathic traits in the primary group that would otherwise be absent in incarcerated samples. Similarly, secondary psychopathy may exhibit greater violent delinquency and antisociality compared to primary psychopathy given that successful psychopaths are less prone to violence. Greater criminality was observed among secondary psychopathy relative to primary psychopathy among a sample of non-offending college students (Lee & Salekin, 2010); however, to our knowledge, no studies have examined successful psychopathy in context of primary and secondary subgroups.

Maltreatment is widely recognized as a risk factor for psychopathy and antisocial behavior (Lansford et al., 2007); however, the independent predictions of psychopathy from maltreatment *over and above* co-occurring antisocial behavior are less documented (Dargis et al., 2016; Graham, Kimonis, Wasserman, & Kline, 2012; Poythress et al., 2006). Furthermore, despite its particular theoretical relevance to secondary psychopathy, few studies have examined this empirically. In the current population-based sample, controlling for co-occurring antisocial behavior, individuals in the secondary psychopathy group reported more diverse forms of maltreatment than primary and comparison youth; no differences were observed between primary and comparison youth. These data suggested that maltreatment was a risk factor for

secondary psychopathy, but not primary psychopathy. The mechanisms in which maltreatment confer risk for secondary psychopathy is unclear, although poor emotion regulation constitutes a plausible mediator given that secondary psychopathy is characterized by greater negative emotionality and stress reaction (Fanti et al., 2013; Hicks et al., 2004; Kahn et al., 2013; Salihovic et al., 2014; Skeem et al., 2007; Swogger & Kosson, 2007; Vassileva et al., 2005). Furthermore, maltreated children exhibit impaired emotion regulation compared to non-maltreated children, which predicted more externalizing psychopathology (Kim & Cicchetti, 2010). Identification of causal mediators underlying these predictions is needed to clarify etiological processes, which may inform treatments for secondary psychopathy. Secondary psychopathy was associated with greater symptoms of borderline personality disorder and dissociation relative to primary psychopathy (Poythress et al., 2006; Skeem et al., 2007), thus treatments addressing trauma symptoms (i.e., trauma-focused cognitive behavioral therapy) or incorporating emotional regulation skills (i.e., dialectical behavior therapy) may improve outcomes among this population.

We highlight several limitations in light of multiple methodological strengths (e.g., population-based sample, longitudinal design, control of delinquency). First, the Add Health study is a secondary dataset, thus it does not provide standardized measures of constructs examined in the current study (i.e., psychopathy, negative emotionality). However, there is substantial support for personality-derived measures of psychopathy, which have been used previously with these data (Beaver et al., 2014), and they are psychometrically sound estimates of individual differences in psychopathy (Lynam et al., 2005). Second, although standardized measures of psychopathy and negative emotionality provide meaningful cutoff scores, normed measures of psychopathy and negative emotionality were not available. As a result, we employed

80th percentile cutoff scores to create primary and secondary subgroups that differed significantly on important clinical correlates (e.g., delinquency, arrest history, psychopathology), which was consistent with theoretical expectations. Empirical derivation of psychopathy groups, including latent class analysis, will be important future research endeavors. Third, self-report measures of delinquency, antisociality, maltreatment, and psychopathy were used, thus introducing potential bias secondary to single source assessment methods (Achenbach, Krukowski, Dumenci, & Ivanova, 2005), although we emphasize that self-reported delinquency is a valid method (Pollak, Menard, Elliott, Huizinga, 2015; Thornberry & Krohn, 2000). However, there is some evidence to suggest that primary psychopathy may underreport their levels of behavioral disturbance (see Kahn et al., 2013). Lastly, our measure of maltreatment combined exposure to multiple forms of abuse (e.g., neglect, sexual abuse, physical abuse), despite evidence that different forms of abuse and neglect differentially confer risk for psychopathy (Dargis et al., 2016). As such, future studies may consider examining how different forms of abuse predict primary and secondary presentations of psychopathy.

The current study constructed primary and secondary subgroups of psychopathy using a population sample of adolescents followed prospectively over 14 years; furthermore, we examined differences among subgroups on maltreatment and self-reported delinquency and antisocial behavior. Maltreatment was greater among secondary psychopathy relative to the primary and comparison groups. Secondary psychopathy exhibited more total and nonviolent delinquency and antisocial behavior during adolescence and adulthood than primary and comparison groups. During adolescence, violent delinquency was similar among secondary and primary psychopathy groups and higher relative to comparison youth. However, in adulthood, secondary psychopathy engaged in greater past year violent antisocial behavior than primary and

comparison groups. Examinations of primary and secondary psychopathy are primarily conducted with populations of incarcerated adults and adolescents, thereby limiting generalizability to non-incarcerated populations. Findings highlight key differences between primary and secondary subgroups among a population based sample of youth followed into adulthood.

CHAPTER THREE: TEMPERAMENT AND SELF-REGULATION AS A MEDIATOR OF PREDICTIONS OF PRIMARY AND SECONDARY PSYCHOPATHY FROM 5- HTTLPR IN A SAMPLE OF CHILDREN AND ADULTS

Abstract

Despite theoretical evidence that distinct causal influences underlie primary (i.e., genetic) and secondary (i.e., environmental) subtypes of psychopathy, specific etiological processes have not been identified. A common polymorphism regulating serotonin neurotransmission (5-HTTLPR) is a plausible biomarker for psychopathy given the centrality of serotonin to aggression and emotional reactivity in humans and non-human animals. Furthermore, dimensions of temperament (i.e., prosociality, negative emotionality, daring) and self-regulation are compelling mediators, as they are sensitive to genetic influences and underlie different expressions of psychopathy. The current study tested whether individual differences in dimensions of temperament and self-regulation mediated primary and secondary subtypes from 5-HTTLPR in two independent, yet complementary, samples of children and youth followed prospectively. In Sample 1, participants were 221 school-aged children (aged 5-10 years) with and without ADHD; Sample 2 included 15,140 adolescents (aged 12-20 years) from the National Longitudinal Study of Adolescent Health. Using bootstrapping procedures, prosociality mediated the association of 5-HTTLPR to predictions of primary psychopathy relative to secondary psychopathy. Neither negative emotionality, daring, or self-regulation mediated predictions of psychopathy subgroups from 5-HTTLPR. We discuss prosociality as a mediator of underlying predictions of psychopathy subgroups from 5-HTTLPR, as well as clinical implications of these findings.

Characterized by atypical behavioral (e.g., antisociality), interpersonal (e.g., egocentricity), and affective facets (e.g., low remorse), psychopathy is highly heterogeneous, and its phenomenological diversity likely consists of potentially etiologically distinct subtypes. Theoretical and empirical refinement of adolescent and adult psychopathy have differentiated “primary” and “secondary” subtypes (Karpman, 1941); comparable on overall antisocial behavior and psychopathic traits, secondary psychopathy has elevated anxiety and internalizing symptoms relative to primary psychopathy (Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003). Of central importance to the current study, subtypes are conceptualized as being differentially sensitive to genetic and environmental influences (Skeem, Johansson, Andershed, Kerr, & Louden, 2007). Whereas primary psychopathy reflected more innate, genetically-based factors including affective disturbance (e.g., callousness) and minimal negative emotionality (e.g., anxiety), secondary psychopathy was hypothesized to be more environmental mediated (e.g., maltreatment) with impulsivity and emotion dysregulation (Karpman, 1941).

Problematically, causal differences between primary and secondary psychopathy are mostly theoretical (Karpman, 1941, Skeem et al., 2003), with little empirical consistency for these subtypes. For example, estimates of heritability ($h^2 = .40 - .60$) and non-shared environmental influences were comparable in primary and secondary psychopathy in separate population-based samples of adolescents and adults (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005; Hicks et al., 2012; Larsson, Andershed, & Lichtenstein, 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Waldman & Rhee, 2006). However, in a twin study of older adolescents, genetic influences on fearless/dominance, a central facet of primary psychopathy, were independent from genetic influences on secondary psychopathy (i.e., impulsive/antisocial) (Hicks et al., 2012). Reflecting dynamic gene-environment interplay, including active and

evocative gene-environment correlations (Dick, 2005), genetic influences on impulsive/antisocial features of psychopathy increased exposure to environmental risk factors (e.g., family, peer), whereas genetic factors were unrelated to environmental adversity for fearless/dominance features (Hicks et al., 2012). Currently, genetic influences on primary and secondary distinctions of psychopathy are not well understood, and knowledge about the mediational constructs and processes underlying the development of behavioral versus affective dimensions of psychopathic traits is unknown. If these potentially distinct etiological pathways are elucidated, such knowledge may facilitate innovations in intervention and prevention by identifying subgroups of youth who may respond differently to interventions (e.g., parent training, self-regulation skills).

Genetically-informative studies of psychopathy subtypes typically employed twin designs (Poythress & Skeem, 2006). Despite important limitations (e.g., small effect sizes, low replicability; Tabor, Risch, & Myers, 2002; Zhu & Zhao, 2007), biologically plausible candidate gene studies may clarify the specific etiological processes underlying different configurations of psychopathic traits. Functional polymorphisms regulating serotonin neurotransmission are plausible biomarkers for psychopathy given the centrality of serotonin to aggression and emotional reactivity in humans and non-human animals (Nelson & Trainor, 2007). Specifically, serotonin differentially predicted unique correlates of primary and secondary psychopathy: Low serotonergic functioning predicted more impulsive aggression associated with secondary psychopathy whereas elevated serotonin predicted emotional hyporesponsivity (i.e., affective dampening, callousness) that characterizes primary psychopathy (Yildirim & Derksen, 2013). For example, although unrelated to overall psychopathy, serotonin inversely predicted impulsive/antisocial and positively predicted interpersonal and affective psychopathy components (Dolan & Anderson, 2003).

A common polymorphism of the promoter region (5-HTTLPR) of the serotonin transporter gene (*SLC6A4*) encodes two allelic forms, a short variant and a long variant, which affect availability of serotonin in CNS (Glenn, 2011). 5-HTTLPR is broadly associated with emotional responding: The long allele predicts emotional hypo-reactivity and insensitivity to threat cues (Bertolino et al., 2005; Drabant et al., 2012; Hariri et al., 2002, 2005), which parallel deficits for primary psychopathy, whereas the short allele predicted emotional hyper-reactivity and hyper-sensitivity to psychosocial stress (Jarrell et al., 2008; Reif et al., 2007; Schwandt et al., 2010). However, 5-HTTLPR is inconsistently associated with overall psychopathic traits: Short-allele homozygotes demonstrated more callous-unemotional (CU) traits than the long-allele among adolescents previously diagnosed with ADHD (Fowler et al., 2009). Conversely, among men with substance use disorders, long allele homozygotes exhibited more psychopathic traits than short-allele carriers (Herman et al., 2011); this pattern was similarly observed among community and clinic-referred youth, but only among low SES children (Sadeh et al., 2010). Furthermore, the long-allele predicted greater emotional deficits characteristic of primary psychopathy among incarcerated men exposed to environmental adversity (Sadeh, Javdani, & Verona, 2013). Homozygous long-allele carriers also self-reported less empathy and exhibited lower physiological response when viewing films of others in distress than short-allele carriers in a sample of healthy adults (Gyurak et al., 2013). The 5-HTTLPR short allele was meta-analytically associated with antisocial behavior (Ficks & Waldman, 2014), but not with aggression (Vassos, Collier, & Fazel, 2014). We contend that the heterogeneity of psychopathy, including unique phenotypes for primary and secondary subtypes, contributes to its inconsistent association with 5-HTTLPR. For example, the short allele, given its association with emotional hyper-reactivity to threat, may relate specifically to secondary psychopathy (e.g., impulsivity,

reactive aggression); however, the long allele is associated with emotional hyporeactivity and insensitivity to threat cues, which is correlated with aggression and is central to primary psychopathy (see Glenn, 2011 for a review). Thus, we predicted that 5-HTTLPR may differentially contribute to the development of primary and secondary psychopathy subtypes.

Despite important advances in gene discovery and gene x environment interactions (G x E), the causal mechanisms underlying pleiotropic effects on psychopathic traits remain unknown. Dimensions of temperament, consisting of individual differences in reactivity and regulation of emotionality, motor activity, and attention (Rothbart, Ahadi, & Evans, 2000), are compelling mediators as they are sensitive to genetic influences (5-HTTLPR in particular), evident early in life, and underlie different expressions of psychopathy (Buss & Plomin, 1984; Hicks & Patrick, 2006; Rothbart & Bates, 1998; Fowles & Dindo, 2006). For example, across studies of human and non-human primates from infancy into adulthood, the short-allele of 5-HTTLPR predicted more negative emotionality, non-cooperativeness, harm avoidance and less emotional resilience (Auerbach, et al., 1999; Champoux et al., 2002; Gonda, Fountoulakis, Juhasz, Rihmer, Lazary, et al., 2009; Hamer, Greenberg, Sabol, & Murphy, 1999; Hayden, Klein, Sheikh, Olino, Dougherty, et al., 2010; Stein, Campbell-Sills, & Gelernter, 2009; see Rogers, Shelton, Shelledy, Garcia & Kalin, 2008 for an exception). According to the “dual-process model of psychopathy,” (Fowles & Dindo, 2006) high fearlessness is reflected in low guilt/empathy (central to primary psychopathy). Conversely, high negative emotionality, which is characterized by poor emotion regulation, is a risk factor for the antisocial/impulsive features characteristic of secondary psychopathy (Fowles & Dindo, 2006, 2009; Frick & Morris, 2004). Although this model has yet to be tested directly with primary and secondary psychopathy in children or adults, temperament dimensions differentially underlie Factor 1 (i.e., affective/interpersonal) and Factor 2 (i.e.,

impulsive/deviant lifestyle) of psychopathy (Burns, Roberts, Egan, & Kane, 2015; Donahue, McClure, & Moon, 2014; Heinzen, Koehler, Smeets, Hoffer, & Huchzermeier, 2011; Hicks & Patrick, 2006). Specifically, Factor 1 was negatively associated with trait and state internalizing negative emotions, but positively correlated with positive emotionality and disinhibition, whereas Factor 2 was positively correlated with negative temperament and impulsivity, but negatively associated with positive temperament in a sample of college students (Dindo & Fowles, 2011). Thus, there is a strong rationale that dimensions of temperament differentially mediate predictions of psychopathic subtypes from 5-HTTLPR genotype.

Self-regulation, which is central to individual differences in temperament, includes specific internal and external processes, including effortful control, inhibition, executive attention, and approach involved in initiating, maintaining and modulating emotions, behavior, and attention (Rothbart, Ellis, & Posner, 2004; Rothbart, Sheese, Rueda, & Posner, 2011). Although parenting practices influence development of self-regulation, parenting accounts only for a small portion of the variance in self-regulation, whereas genetic factors account for a majority of the variance in phenotypes associated with self-regulation (e.g., overactivity, attention) (Beaver, Wright, De Lisi, & Vaughn, 2008; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2004). Although 5-HTTLPR short allele has been linked to phenotypes relevant to self-regulation such as negative emotionality and reactive aggression (Retz, Retz-Junginger, Supprian, Thome, & Rosler, 2004), no direct effects between 5-HTTLPR and self-regulation have also been reported (Carver, Johnson, & Joormann, 2008). Of key importance to the current study, poor self-regulation predicted behaviors characteristic of secondary psychopathy, including reactive or impulsive aggression. Self-regulation processes, such as high negative emotional reactivity and poor effortful control, concurrently and

prospectively predicted child conduct problems (see Frick & Morris, 2004 for a review). Furthermore, intact self-regulation was a protective factor against predictions of antisocial behavior from deviant peer affiliation (Gardner, Dishion, & Connell, 2008). Given its heritability and association with behaviors characteristic of secondary psychopathy (i.e., antisocial behavior, inhibitory control), we contend that self-regulation is a plausible mediator of psychopathy predictions from 5-HTTLPR. Specifically, we propose that the short allele of 5-HTTLPR will predict individual differences in poor self-regulation, which will be differentially associated with secondary, but not primary psychopathy.

Despite theoretical evidence that primary (i.e., genetic) and secondary (i.e., environmental) subtypes of psychopathy reflect unique causal influences, specific etiological processes have not been identified. Given its centrality to the structure and connectivity of neural structures (i.e., amygdala, ventromedial prefrontal cortex) implicated in emotional reactivity, inhibition, and decision-making, we tested whether individual differences in dimensions of temperament (i.e., daring, negative emotionality, and prosociality) and self-regulation mediated predictions of primary and secondary psychopathic subtypes from 5-HTTLPR in a case-control sample of 221 youth with and without ADHD followed prospectively for two years and in the National Longitudinal Study of Adolescent Health (Add Health), a nationally representative sample of 15,701 adolescents followed prospectively across 14 years. Samples containing youth with ADHD are well suited for early examinations of psychopathic traits given their prediction of early onset conduct problems (Hinshaw, Lahey, & Hart, 1993). However, to enhance generalizability and to capitalize on its large prospective design, we conducted parallel analyses in Add Health. In sample 1, we predicted short allele carriers would display greater negative emotionality, which would increase the odds of secondary psychopathy status measured two

years later, even with control of baseline ADHD symptoms. In sample 2, we similarly predicted that short allele carriers would display less self-regulation, which would increase odds of secondary psychopathy status measured 14 years later. Findings for each sample are described below separately as Study 1 and Study 2.

Study 1 Method

Participants

At baseline, participants were 221 5 to 10-year old ethnically diverse children (55% White, 9% Black, 10% Latino, 3% Asian, and 23% Mixed or Other) with ($n = 114$) and without ($n = 107$) ADHD. Recruitment targeted local elementary schools, pediatric offices, presentations at self-help groups, and referrals from clinical service providers. English fluency and living with at least one biological parent at least half the time were requirements to participate in the study. Exclusion criteria included full scale IQ below 70 and a previous diagnosis of an autism spectrum, neurological disorder, or any medical condition that prevented full participation in the study. ADHD probands met full diagnostic criteria for ADHD at baseline according to the Diagnostic Interview Schedule for Children, Version IV, Parent Version (DISC-IV-P; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000), a structured diagnostic interview administered to parents and keyed to all Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) criteria. Given its centrality to early-onset conduct problems (Hinshaw et al., 1993), which is typically accompanied by CU traits (Frick et al., 2005), ADHD was oversampled. ADHD probands and non-ADHD comparison youth did not differ significantly with regard to age, sex, race-ethnicity, and income. Frequently occurring comorbidities for ADHD, such as oppositional defiant disorder (ODD), anxiety, and depression were allowed among probands to increase external validity. Additionally, youth meeting

diagnostic criteria for any disorder other than ADHD (anxiety disorders were most common) were placed in the non-ADHD comparison group to avoid recruiting an unrealistically high functioning comparison group.

Procedures

A standardized telephone screener probed inclusion and exclusion criteria described above. Eligible families attended in-person laboratory assessments and rating scales were mailed to the child's primary teacher. After parent consent and child assent were obtained, parents completed the DISC-IV-P and measures of parenting, child behavior, life stress, and their own psychopathology. Children were concurrently assessed on standardized measures of cognitive ability, academic achievement, and self-reported psychopathology in a room separate from their parents. Parents and teachers were instructed to complete rating scales based on the child's unmedicated behaviors; 85% of youth were unmedicated during the assessment. Clinical psychology doctoral students and BA-level staff conducted assessments and were initially blind to the child's diagnostic status, although blindness was broken due to data collected during the assessment. All study procedures were approved by the IRB.

When children were 7 to 12 years old, a two-year follow-up assessment (i.e., Wave 2) was conducted. Consisting of similar procedures at baseline, Wave 2 evaluated family functioning, youth academic achievement, and child psychopathology. Approximately 89% of the initial Wave 1 sample completed assessments at Wave 2; youth with ADHD were more likely to return at Wave 2 than families without ADHD. Wave 2 families were similar to the original Wave 1 sample on all other demographic factors (i.e., age, sex, race-ethnicity).

Measures

Genotyping. DNA was obtained at Wave 1 from saliva using DNA Genotek Oragene™ Self-Collection Kits (DNA Genotek, Inc., Ottawa Canada). We genotyped the 5-HTTLPR 48 bp insertion/deletion polymorphism in the promoter region of the serotonin transporter gene, which produced short (484 bp) and long (528 bp) fragments. Following previous strategies (Auerbach, et al., 2001) we compared three genotypes with the following distributions: SS (24%, n = 53), SL (45%, n = 98), and LL (31%, n = 67); the Lg allele was not genotyped. Frequencies did not deviate from Hardy-Weinberg equilibrium ($\chi^2=2.07$, $df=1$, $p=.15$).

Mediators.

Temperament. The Child and Adolescent Dispositions Scale (CADS) is a parent interview of children's temperament with items that were explicitly developed for studies of psychopathology without using symptom-related terminology (Lahey et al., 2008). Parents completed the 48-item measure at Wave 2, yielding prosociality, negative emotionality, and daring subscores. Parents rated each item on a 4-point Likert scale. The CADS demonstrates good internal consistency, high test-retest reliability, as well as construct and external validity (Lahey et al., 2008; Trentacosta, Hyde, Shaw, & Cheong, 2009). Coefficient alphas for the prosociality, negative emotionality, and daring factors were .87, .77, and .79, respectively.

Psychopathy Subgroups.

Psychopathic Traits. At Wave 2, parents completed the Antisocial Process Screening Device (APSD; Frick & Hare, 2001), a 20-item rating scale of youth psychopathic traits (Frick et al., 2000). Parents rated each item as 0 = not at all true, 1 = sometimes true, or 2 = definitely true. The narcissism scale consisted of seven items such as "Uses or 'cons' others," "Teases or makes fun of others," and "Acts charming." Impulsivity was estimated from six items including:

“Blames others,” “Acts without thinking,” and “Gets bored easily.” Alphas were .72, and .63 for parent ratings of narcissism and impulsivity at Wave 2. To estimate CU traits, parents completed the Inventory of Callous-Unemotional Traits (ICU), which is psychometrically superior to CU traits derived from the APSD (Kimonis et al., 2008). The ICU consists of 24 items reflecting callousness (e.g., “I do not care who I hurt to get what I want”), uncaring (e.g., “I always try my best,” reverse scored), and unemotional (e.g., “I express my feelings openly,” reverse scored) traits. Items were rated from 0 = not at all true to 3 = definitely true. The total ICU score demonstrated high internal consistency and convergence with aggression, delinquency, and physiological indices of constricted emotion (Kimonis et al., 2008). In this sample, the alpha for the total score of CU traits at Wave 2 was .86.

Anxiety. At Wave 2, children self-reported their anxiety and depression via the 47-item Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000), which includes DSM-IV subscales of panic disorder, obsessive-compulsive disorder (OCD), social phobia, separation anxiety disorder, and generalized anxiety disorder. OCD was excluded given its separability from other anxiety disorders (Stein et al. 2010; Phillips et al., 2010). Items were rated as 0 = never, 1 = sometimes, 2 = often, and 3 = always. The RCADS is psychometrically sound (Chorpita et al., 2000; Chorpita, Moffitt, & Gray, 2005); alphas ranged from .75 to .83 for individual subscales in the current study.

Covariates.

ADHD Symptoms: We controlled for the total number of Wave 1 youth DSM-IV ADHD symptoms from the DISC-IV-P (Shaffer et al., 2000), a structured diagnostic interview with parents. The ADHD module demonstrated strong one-year test-retest reliability ($r = .79$) and internal consistency ($\alpha = .77$) in a large community sample (Shaffer et al., 2000).

Data Analytic Plan

This study assessed whether individual differences in temperament dimensions (i.e., prosociality, negative emotionality, and daring) mediated the association of 5-HTTLPR with empirically-derived subtypes of psychopathy from a two-year follow-up. First, we employed model-based cluster analysis (MBC) to determine the presence of primary and secondary psychopathy subgroups in our sample. MBC tests the relative fit of 10 models that differ in their assumptions about data structure and calculates a fit index for each model (Banfield & Raftery, 1993). MBC was performed using the *mclust* Version 4 (Fraley, Raftery, & Scrucca, 2012) in R: (R Core Team, 2013). Wave 2 psychopathic traits (i.e., CU traits, narcissism, and impulsivity) and anxiety (i.e., social phobia, generalized anxiety disorder, separation anxiety disorder, panic disorder) were entered as clustering variables in MBC. We then created a Wave 2 psychopathy score by summing z-scores for CU traits (from the ICU) and narcissism and impulsivity (from the APSD). Children above the median ($-.13$, $n = 58$) were included in the MBC; the remaining participants were placed in the comparison group. Bayesian information criterion values suggested a two-cluster solution best fit the data (primary, $n = 27$, secondary, $n = 30$) with diagonal, varying volume and shape ($BIC = -1060$). Two-cluster solutions were the next best fitting models with diagonal, equal volume and varying shape ($BIC = -1066$) and spherical and equal volume ($BIC = -1078$). Relative to the comparison group, children in the elevated psychopathic traits group (i.e., primary and secondary combined) exhibited more Wave 2 aggression $F(1) = 36.59, p < .001$; CU traits $F(1) = 107.32, p < .001$; antisocial behavior $F(1) = 7.31, p < .01$; ADHD symptoms $F(1) = 34.19, p < .001$; ODD symptoms $F(1) = 11.58, p = .001$, providing key support for these groups. Correlates of primary, secondary, and comparison youth are provided in Table 11.

Second, we constructed a multiple mediation model using path analysis in MPlus 7.4 (Muthén & Muthén, 1998-2015). Indirect effects were estimated using bootstrapping where the indirect or mediated effect is evaluated for significance. This method is superior (Dearing & Hamilton, 2006; Preacher & Hayes, 2008) to the classic causal steps approach to mediation (Baron & Kenny, 1986), which requires a significant direct effect of the predictor on the outcome. Multiple mediation with bootstrapping is advantageous to other mediation approaches because it accommodates non-normal data, smaller samples, and improves power (Briggs, 2006; Preacher & Hayes, 2006; Williams & MacKinnon, 2008). The current model calculated the indirect effect from 10,000 bootstrapped samples and estimated a confidence interval from percentiles of the bootstrapped distribution.

5-HTTLPR was coded additively, with 0, 1, and 2 representing the short/short, long/short, and long/long genotypes, respectively. Prosociality, negative emotionality, and daring were entered simultaneously as mediators. Lastly, membership in the comparison, primary, and secondary psychopathy subgroups was the outcome. Because group membership was a categorical variable, we employed multinomial logistic regression to examine the path between mediator variables (i.e., temperament) and outcome (i.e., group membership). Given the case-control design of the study, we controlled baseline ADHD symptoms. We first describe the total effect of 5-HTTLPR on psychopathy subgroups, excluding temperament dimensions; we then describe the association of 5-HTTLPR with temperament dimensions and psychopathy subgroups. Lastly, we report the direct effect of 5-HTTLPR on psychopathy subgroups.

Study 1 Results

Wave 2 Correlates of Primary, Secondary, and Comparison Clusters

To characterize psychopathy subgroups derived from MBC, analysis of variance tested groups across multiple Wave 2 measures (Table 11). Primary and secondary groups were each more narcissistic and impulsive than comparison youth, an expected pattern given that only youth with elevated psychopathic traits were included in the MBC. Although primary and secondary youth had more Wave 2 CU traits than comparison youth, the primary group exhibited significantly more CU traits than the secondary group. Psychopathy groups also differed on Wave 2 ADHD diagnosis, ADHD symptoms, and ODD symptoms. Crucially, with the exception of self-reported antisocial behavior, across all measures, primary and secondary youth had significantly more disruptive behavior problems than the comparison group, but they did not differ from one another. This pattern is consistent with theoretical expectations (Kimonis et al., 2012; Skeem et al., 2007; Swogger & Kosson, 2007). No other differences were observed between primary and secondary groups. Thus, these patterns of association suggest that these groups are valid representations of primary and secondary psychopathy in a sample of school-aged children.

Population Stratification

Differences in allele frequencies secondary to race-ethnicity (Gelernter, Cubells, Kidd, Pakstis, & Kidd, 1999) may threaten internal validity. We evaluated established criteria for population stratification, including race-ethnicity correlating significantly with 5-HTTLPR and the outcome of psychopathy group membership (Hutchison, Stallings, McGeary, & Bryan, 2004). Allele frequencies of 5-HTTLPR varied significantly across race-ethnicity ($\chi^2 = 22.75$, $df = 10$, $p < .05$), however, race-ethnicity was unrelated to Wave 2 psychopathy subgroups ($\chi^2 = 3.54$, $df = 10$, $p > .05$), W2 total anxiety $F(5, 101) = 0.42$, $MSE = 0.97$, $p > .05$), and W2 total psychopathy ($F = 0.96$, $MSE = 5.73$, $p > .05$).

5-HTTLPR and Psychopathy Subgroup: Mediation by Temperament

First, controlling for W1 ADHD status (Figure 2), we tested the association of 5-HTTLPR and psychopathy subgroups through prosociality, negative emotionality, and daring (with the secondary group as the comparison group for the multinomial logistic regression; i.e., b paths). There was no total effect (i.e., excluding mediators from the model) for the comparison (B = 0.69, SE = 0.48, p = .15) and primary groups (B = 0.20, SE = 0.49, p = .68). Whereas the number of 5-HTTLPR long alleles inversely predicted prosociality (B = -1.43, SE = 0.51, p < .01) and positively predicted negative emotionality (B = 1.54, SE = 0.48, p < .01), daring was unrelated to 5-HTTLPR (B = 0.32, SE = 0.33, p = .33). Second, prosociality negatively (B = -0.15, SE = 0.07, p = .03) and daring positively (B = 0.21, SE = 0.11, p = .05) predicted membership in the primary group relative to the secondary group; transformations of the multinomial log odds indicated that prosociality decreased the likelihood of primary group membership relative to the secondary group, whereas daring led to increased risk of primary psychopathy relative to secondary psychopathy. All three dimensions were unrelated to the comparison group membership. Third, there was no direct effect of 5-HTTLPR on Wave 2 comparison (B = 0.41, SE = 0.47, p = .38) or primary groups (B = -0.03, SE = .48, p = .95) when prosociality, negative emotionality, and daring were included in the model.

Total and specific indirect effects of 5-HTTLPR on Wave 2 psychopathy groups through prosociality, negative emotionality, and daring were calculated by specifying 10,000 bootstrap simulation samples and calculating point estimates and the 95% bias corrected confidence intervals for each indirect effect. The point estimate of the total indirect effect (i.e., the difference between the total effect and direct effect through the three mediators) differed significantly from zero for the comparison subgroup but not the primary subgroup (see Table 12). Next, we

examined the specific indirect effects of each mediator – that is, the extent to which each temperament dimension mediated the effect of 5-HTTLPR on psychopathy group membership considering the other dimensions of temperament. Point estimates of specific indirect effects indicated that prosociality, but not daring or negative emotionality, mediated the association between 5-HTTLPR and both comparison (CI: 0.02 to 0.44) and primary groups (CI: 0.06 to 0.47) relative to the secondary group. That is, prosociality, over and above daring and negative emotionality, mediated predictions of comparison relative to secondary and primary relative to secondary group membership from 5-HTTLPR.

Study 2 Method

Participants

The National Longitudinal Study of Adolescent Health (Add Health, Harris, 2013) recruited a nationally representative cohort of adolescents in grade 7-12 drawn from a probability sample of 80 U.S. high schools and 52 U.S. middle schools. More details of the study can be accessed at <http://www.cpc.unc.edu/projects/addhealth>. The sample was followed prospectively across 14 years, and data was ascertained via an in-school questionnaire and four at-home interviews. 20,745 adolescents were interviewed at Wave I during the 1994-1995 school years; participants ranged in age from 12- 21 years (49.2% male). The Wave II follow-up was conducted with 14,738 adolescents two years later (ages 13-22). The Wave III follow-up was conducted 7-8 years post-baseline and included 15,197 participants ranging in age from 18 to 28 (1,507 partners of original participants were added to the sample). Finally, a fourth wave of data collection (Wave IV) occurred in 2007-2008 (approximately 13-14 years after Wave 1) and included 15,701 participants aged 24-32 years. At Wave IV, 15,140 participants who consented to provide saliva samples were genotyped for multiple functional polymorphisms. The sample

was ethnically diverse (53.4% Caucasian, 17% Latino, 21.6% African American, 6.4% Asian, 0.6% Native American, and 0.9% “Other”).

Measures

Genotyping. DNA was obtained at Wave IV from saliva using DNA Genotek Oragene™ Self-Collection Kits (DNA Genotek, Inc., Ottawa Canada). The 5-HTTLPR 48 bp insertion/deletion polymorphism in the promoter region of the serotonin transporter gene was genotyped, which produced short (484 bp) and long (528 bp) fragments. Following previous strategies (Auerbach, et al., 2001) we compared three genotypes with the following distributions: SS (19.3%, $n = 2,858$), SL (46.0%, $n = 6,823$), and LL (34.8%, $n = 5,164$). Frequencies deviated from Hardy-Weinberg equilibrium ($\chi^2=50.00$, $df = 1$, $p < .001$).

Mediator.

Self-regulation. A 23-item measure of self-regulation of attention, emotions, and behavior was administered at Wave I to participants and their mothers to assess participants’ ability to regulate their attention, feelings, and behavior (see Appendix D). Adolescent items included: “do you have trouble paying attention in school,” “difficult problems make you very upset,” and “you never argue with anyone,” whereas some maternal items included “you can trust your child” and “does your child have a bad temper?” All 23 items loaded significantly onto a single factor using confirmatory factor analysis (Belsky & Beaver, 2011). All items were recoded so high scores reflected poor self-regulation; a composite score was created by summing items ($\alpha = .76$) and it correlated with deviant peer affiliation (Beaver, Ratchford, & Ferguson, 2009).

Psychopathy Subgroups.

Psychopathic Traits. Psychopathic traits were derived from Wave IV Add Health

personality items measuring personality traits from the five-factor model (FFM; Johns & Srivastava, 1999), which is a valid approach to measure psychopathy (Lynam et al., 2005). Personality items were previously used to characterize dimensions of psychopathy in Add Health (Beaver, Barnes, May, & Schwartz, 2011; Beaver, Rowland, Schwartz, & Nedelec, 2011). To do so, personality items that paralleled those used in previous studies of FFM psychopathy were selected. Then, these items were factor analyzed; items that did not load onto the same factor were removed from the scale. A total of 23 items that loaded significantly onto a single scale of total psychopathy yielded coefficient alphas exceeding .80 (see Appendix A). Items were measured on a 5-point Likert scale (1 = strongly agree, 5 = strongly disagree) and include: “I feel others’ emotions,” “I get angry easily,” “I am not interested in other people’s problems,” “I live my life without much thought for the future,” and “I live my life without much thought for the future.” This scale was associated with neurological deficits and predicted the probability of being arrested, incarcerated, and on probation (Beaver, Boutwell, Barnes, Vaughn & DeLisi, 2015; Beaver, Vaughn, DeLisi, Barnes, & Boutwell, 2011).

Negative Emotionality. Although the personality items in the Add Health sample can be used to estimate neuroticism, many of these items were used to create the psychopathy score. To avoid inflating correlations due to shared items, a seven-item scale of negative emotionality was derived from Wave IV Add Health. Questions demonstrating face validity with items on the Multidimensional Personality Questionnaire (MPQ, Tellegen, 1982) stress reaction and alienation subscales were used (see Appendix B), as these MPQ subscales load onto a higher order Negative Emotionality factor (Patrick, Curtin, & Tellegen, 2002). Items were measured on a 4-point Likert scale ranging from “never or rarely” to “most of the time or all of the time,” and included: “You felt that people disliked you during the past seven days,” “In your day-to-day

life, how often do you feel you have been treated with less respect or courtesy than other people,” “You were bothered by things that usually don’t bother you,” “You could not shake off the blues, even with help from your family and your friends.” Items were scored such that a higher score reflected greater negative emotionality. An exploratory factor analysis revealed the seven items loaded onto a single factor and yielded a coefficient alpha of .69.

Data Analytic Plan

To parallel Sample 1, we tested whether individual differences in self-regulation, a central component of temperament in conjunction with emotional reactivity (Rothbart, Ellis, & Posner, 2004), measured at baseline mediated the association of 5-HTTLPR with subtypes of psychopathy approximately 13-14 years later using the Add Health sample. Because survey data analysis does not support model based cluster analysis (StataCorp, 2013), we employed a different procedure to form primary and secondary psychopathy subgroups with Add Health data. Given reliable differences on negative emotionality in primary and secondary psychopathy, participants were categorized as either demonstrating elevations on psychopathy and/or negative emotionality, using an 80th percentile cutoff for both measures (i.e., score of 68 or higher on psychopathy, score of 12 or higher on negative emotionality). Participants scoring below the 80th percentile on psychopathy were categorized as comparison youth (n = 11,935). Individuals demonstrating elevated psychopathy but were below the negative emotionality cutoff were labeled as primary psychopathy (n = 1,706); individuals scoring about the cutoffs for psychopathy and negative emotionality were labeled as secondary psychopathy (n = 1,941). Correlates of primary, secondary, and comparison youth are provided in Table 3.

Second, we constructed a mediation model using path analysis in MPlus 7.4 (Muthén & Muthén, 1998-2015). Indirect effects were estimated using bootstrapping where the indirect or

mediated effect is evaluated for significance. This method is superior (Dearing & Hamilton, 2006; Preacher & Hayes, 2008) to the classic causal steps approach to mediation (Baron & Kenny, 1986), which requires a significant direct effect of the predictor on the outcome because it accommodates non-normal data, smaller samples, and improves power (Briggs, 2006; Preacher & Hayes, 2006; Williams & MacKinnon, 2008). The current model calculated the indirect effect from 1,000 bootstrapped samples and estimated a confidence interval from percentiles of the bootstrapped distribution.

5-HTTLPR was coded additively, with 0, 1, and 2 representing the short/short, long/short, and long/long genotypes, respectively. Self-regulation was entered as a mediator, and membership in the comparison, primary, and secondary psychopathy subgroups was the outcome. Because group membership was a categorical variable, we employed multinomial logistic regression to examine the path between mediator variables (i.e., self-regulation) and outcome (i.e., group membership). We first describe the total effect of 5-HTTLPR on psychopathy subgroups, excluding self-regulation; we then describe the association of 5-HTTLPR with self-regulation and psychopathy subgroups. Lastly, we report the direct effect of 5-HTTLPR on psychopathy subgroups.

Study 2 Results

Population Stratification

We evaluated established criteria for population stratification in the Add Health. Allele frequencies of 5-HTTLPR varied significantly across race-ethnicity ($F(6.73, 861.12) = 18.26, p < .001$) and race-ethnicity was related to Wave IV psychopathy subgroups ($F(6.47, 828.53) = 4.50, p < .001$), thus race-ethnicity was controlled for in the Add Health model.

5-HTTLPR and Psychopathy Subgroup: Mediation by Self-Regulation

Next, with control of race-ethnicity, we tested the association of 5-HTTLPR and Wave IV psychopathy subgroups through Wave I self-regulation (with the secondary group as the comparison group for the multinomial logistic regression; i.e., b paths). There was a total effect (i.e., excluding mediators from the model) for the comparison ($B = 0.11$, $SE = 0.04$, $p = .005$) and primary groups ($B = 0.21$, $SE = 0.05$, $p < .001$). The number of 5-HTTLPR long alleles was unrelated to self-regulation ($B = 0.16$, $SE = 0.11$, $p = .13$), but poor self-regulation negatively predicted comparison group membership relative to the secondary group ($B = -0.06$, $SE < 0.01$, $p < .001$) and primary group membership relative to the secondary group ($B = -0.02$, $SE < 0.01$, $p < .001$). To facilitate interpretation, transformations of the multinomial log odds indicated that poor self-regulation decreased the likelihood of comparison group membership relative to the secondary group, as well as the likelihood of primary group membership relative to the secondary group. Third, there was a direct effect of 5-HTTLPR on comparison ($B = 0.12$, $SE = 0.04$, $p < .01$) or primary groups ($B = 0.22$, $SE = 0.05$, $p < .001$), relative to secondary group when self-regulation was included in the model.

The indirect effect of 5-HTTLPR on Wave IV psychopathy groups through Wave I self-regulation was calculated by specifying 1,000 bootstrap simulation samples and calculating point estimates and the 95% bias corrected confidence intervals for each indirect effect. The point estimates of the indirect effect (i.e., the difference between the total effect and direct effect through the mediator) did not differ significantly from zero for both the comparison and primary subgroups, suggesting that poor self-regulation did not mediate the association between 5-HTTLPR and both comparison (CI: -0.02 to 0.003) and primary groups (CI: -0.01 to 0.001) relative to the secondary group.

Discussion

We first examined whether primary and secondary psychopathy subgroups could be detected among two independent samples of school-aged and adolescent youth. Sample 1 included 221 children with and without ADHD followed prospectively for two years, and model-based cluster analysis yielded a high (i.e., secondary) and low (i.e., primary) anxiety group of children with similarly elevated psychopathic traits. Primary and secondary psychopathy groups exhibited significantly more narcissism, CU traits, impulsivity, and symptoms of ADHD and ODD than comparison youth. More specifically, however, primary youth exhibited more CU traits than secondary youth whereas secondary youth reported more anxiety symptoms than primary youth. Using a nationally representative sample of 15,701 adolescents followed prospectively across 14 years from the National Longitudinal Study of Adolescent Health (Add Health), we also examined presence of primary and secondary psychopathy groups. The secondary group exhibited higher prevalence rates of depression, anxiety, and PTSD compared to the primary group, as well as greater engagement in delinquent and antisocial behaviors.

Next, we evaluated whether these psychopathy subtypes were differentially sensitive to dimensions of temperament and self-regulation in predictions from 5-HTTLPR using bootstrapping within a multiple mediation framework. In Study 1, beyond daring and negative emotionality, predictions of empirically-derived psychopathy group membership among school-age children from 5-HTTLPR were uniquely mediated by individual differences in prosociality, even with control of ADHD symptoms. That is, the 5-HTTLPR long allele inversely predicted prosociality; greater prosociality was negatively associated with primary membership relative to the secondary group. Specifically, greater prosociality decreased the likelihood of primary group membership relative to the secondary group. In Study 2, among 15,701 adolescents followed

prospectively for 14 years into adulthood, 5-HTTLPR was unrelated to self-regulation, although poor self-regulation reduced the probability of primary and comparison group membership (relative to secondary psychopathy). Self-regulation did not mediate the association between 5-HTTLPR and comparison and primary youth relative to secondary youth.

5-HTTLPR pleiotropically affects multiple dimensions of psychopathology (e.g., anxiety, depression; Hettema, Chen, Sun & Brown, 2015; Munafò et al., 2009; Murphy & Lesch, 2008). Although temperament dimensions may reflect unique pathways underlying genetic influences on psychopathology, few studies have explored this model explicitly (see Brammer & Lee, 2012; Nigg, 2006 for exceptions). According to the developmental propensity model, dispositional traits of prosociality, negative emotionality and daring are central to the development of conduct disorder (Lahey et al., 2003, 2005) and possibly contribute to liability for major forms of psychopathology more generally (Tackett, Lahey, Van Hulle, Waldman, Krueger, & Rathouz, 2013). Together, these dimensions accounted for 46% of the variance in a latent conduct disorder variable; 21% of this variance was uniquely accounted for by prosociality (Waldman et al. 2011), thus highlighting the influence of prosociality on the development of conduct disorder. Furthermore, these dimensions were substantially heritable ($h^2 = .43 - .62$) and shared nearly 40% of the genetic variance in conduct disorder. As such, dimensions of temperament may represent a pathway from latent genetic influences on emergent externalizing problems, including psychopathic traits. In the current study, even with control of ADHD, prosociality uniquely mediated predictions of psychopathy group membership from 5-HTTLPR. Given the centrality of affective disturbance and deficient empathic response to psychopathic traits, prosociality may reduce risk for these problems. Furthermore, temperament dimensions may elicit/interact with different environmental risk (e.g., parenting behaviors) and may help inform

specific interventions (i.e., parent training, emotion regulation skills) that may be beneficial to children at risk for psychopathic traits. Moderated mediation models that examine the interactive effect of parenting on temperament in predictions of psychopathy from genetic risk are needed to test this hypothesis. For example, parent-training interventions (e.g., behavioral management, emotional and instrumental support) for children with psychopathic traits significantly reduced youth psychopathic traits at a 20-month follow-up (McDonald, Dodson, Rosenfield, & Jouriles, 2011). Change in key dimensions of temperament and self-regulation may mediate reductions in psychopathic traits secondary to changes in parenting behavior given that increased positive parenting skills (e.g., positive reinforcement, behavior management, parent coping) also revealed significant decreases in CU traits post-treatment (Hawes & Dadds, 2007).

Although typically examined unidimensionally (Eisenberg, Fabes, Guthrie, & Reiser, 2000) in Add Health (Beaver, DeLisi, Mears, & Stewart, 2009; Wolfe & Hoffman, 2015), self-regulation consists of multiple facets including inhibitory control, attention shifting, and working memory. For example, a strong fitting second-order factor model of self-regulation in these data identified five factors (i.e., problem solving, self-worth, life difficulties, school problems, self-control) (Wolfe & Hoffman, 2015), which may suggest more specific patterns of association with psychopathic traits and psychopathy subgroups. Furthermore, consistent with recent calls on the need for a unified model of self-regulation across theories, constructs, measures, and fields (Zhou, Chen, & Main, 2011), we await improved conceptual and empirical models of self-regulation that may improve traction on psychopathic traits.

Deficits in empathic responding are typically considered central to psychopathy among adults and children. Although dispositional and situational empathy are frequently positively correlated with prosociality (Eisenberg, Eggum & Di Giunta, 2010), prosociality does not

capture the underlying motivation for engaging in helpful behavior, thus complicating the relationship between prosociality and psychopathy. That is, motivation to engage in prosocial behaviors may reflect egocentricity (e.g., social approval, reduce one's own personal distress, desire for reciprocity), desire to help others (e.g., sympathy), or moral values (Eisenberg et al., 2010). Moreover, the association of prosociality and empathy may change with the public or private nature of the setting: For example, sympathy (i.e., empathy and perspective taking) was inversely associated with public prosocial behavior, and public prosocial behaviors were positively related with physical aggression among adults (McGinley & Carlos, 2006). Similarly, adult primary psychopathy was associated with fewer private prosocial behaviors (White, 2014). Problematically, however, little is known about the association between prosociality and psychopathy in youth. In the current study, prosociality decreased the likelihood of primary psychopathy group membership compared to secondary psychopathy, which is consistent with the centrality of affective disturbance (e.g., low empathy, CU traits) in primary psychopathy (Hicks et al., 2012; Karpman, 1941; Salihovic et al., 2014; Skeem et al. 2003), at least among adults. This study is among the first to examine temperament, prosociality in particular, and self-regulation as a potential endophenotype between 5-HTTLPR and primary and secondary subgroups of psychopathy in children, however it is yet to be determined if underlying motivations for prosocial behaviors may differ among primary and secondary youth.

Despite methodological strengths (e.g., independent samples, longitudinal, multiple informants), several study limitations should be considered. First, both samples relied on community or population based samples of youth, and thus psychopathic traits in each sample were expectedly lower than among adjudicated samples. However, the design of the Sample 1 study (e.g., oversampling of children with ADHD) critically identified young children at high

risk for early-onset conduct problems, and primary and secondary groups demonstrated key differences from comparison youth on psychopathic traits, conduct problems, and aggression. Second, we relied on self-report and parent-report measures of temperament, self-regulation, psychopathic traits, and anxiety. However, behavioral measures of temperament or dispositional traits, such as facial expressions of concern and psychophysiological indicators in response to people feigning distress, have innovatively estimated prosocial responding (Eisenberg et al., 2010; Hastings, Zahn-Waxler, Robinson, Usher, & Bridges, 2000; Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008). Finally, primary and secondary psychopathy were derived using group-based approaches (e.g., cluster analysis), rather than dimensional measures. We contend that both person-based (e.g., cluster) and variable-based approaches are necessary to improve traction on well-known phenotypic heterogeneity in psychopathy.

The current study examined whether individual differences in dimensions of temperament and self-regulation mediated predictions of primary and secondary psychopathy group membership from 5-HTTLPR in two separate samples of children and adolescents. Prosociality and self-regulation significantly mediated the association between 5-HTTLPR and membership in the comparison and primary groups relative to the secondary psychopathy. Furthermore, 5-HTTLPR long alleles were inversely associated with prosociality, and in turn, prosociality decreased the risk of primary psychopathy group membership compared to secondary psychopathy. Long alleles also predicted worse self-regulation abilities, which in turn decreased the risk of comparison and primary group membership compared to secondary psychopathy. Findings suggest that prosociality may be an important endophenotype in the association from 5-HTTLPR and primary versus secondary psychopathy.

Appendices

Appendix A. Psychopathy scale constructed from personality items at Wave IV

The items below were preceded with the following question stem: “How much do you agree with each statement about you as you generally are now, not as you wish to be in the future? The following rating scale was used: 1 = strongly agree, 2 = agree, 3 = neither agree nor disagree, 4 = disagree, and 5 = strongly disagree.

| Item | Add Health Variable Name |
|--|--------------------------|
| I sympathize with others' feelings | H4PE2 |
| I get angry easily* | H4PE8 |
| I am not interested in other people's problems* | H4PE10 |
| I often forget to put things back in their proper order* | H4PE11 |
| I am relaxed most of the time | H4PE12 |
| I am not easily bothered by things | H4PE14 |
| I rarely get irritated | H4PE16 |
| I talk to a lot of different people at parties | H4PE17 |
| I feel others' emotions | H4PE18 |
| I get upset easily* | H4PE20 |
| I get stressed out easily* | H4PE22 |
| I lose my temper* | H4PE24 |
| I keep in the background* | H4PE25 |
| I am not really interested in others* | H4PE26 |
| I seldom feel blue | H4PE28 |
| I don't worry about things that have already happened | H4PE30 |
| I keep my cool | H4PE32 |
| I go out of my way to avoid having to deal with problems in my life* | H4PE33 |
| When making a decision, I go with my “gut feelings” and don't think much about the consequences of each alternative* | H4PE34 |
| I live my life without much thought for the future* | H4PE36 |
| Other people determine most of what I can and cannot do* | H4PE38 |
| There are many things that interfere with what I want to do* | H4PE39 |
| There is really no way I can solve the problems I have* | H4PE41 |

*Indicates that item was reverse scored so high responses reflect greater psychopathic traits

Appendix B. Add Health items included in negative emotionality scale at Wave IV

Add Health items exhibiting face validity with item from the stress reaction and alienation subscales of the Multidimensional Personality Questionnaire were used to form a negative emotionality scale. Items from the Social Psychology and Mental Health section (H4MH) were preceded with the following question stem: “How often was each of the following things true in the past seven days.” Items from the Personality section (H4PE) were preceded with the following text: “How much do you agree with each statement about you as you generally are now, not as you wish to be in the future?”

| MPQ item | Add Health item | Add Health Variable Name |
|---------------------------------|--|--------------------------|
| <i>Stress Reaction</i> | | |
| Easily upset | You were bothered by things that usually don’t bother you | H4MH18 |
| Unexplainable negative emotions | You could not shake off the blues, even with help from your family and friends | H4MH19 |
| Unaccountable mood changes | I have frequent mood swings | H4PE4 |
| Worry-prone/anxious | I worry about things | H4PE6 |
| <i>Alienation</i> | | |
| Feels mistreated | You felt that people disliked you | H4MH27 |
| Pushed around | In your day-to-day life, how often do you feel treated with less respect or courtesy than other people | H4MH28 |
| | How often do you feel isolated from others | H4MH2 |

Appendix C. Add Health items included in delinquency/antisocial behavior scales at Wave I and IV

The items below were preceded with the following question stem: “In the past 12 months, how often...?” The following rating scale was used: 0 = never, 1 = 1 or 2 times, 2 = 3 or 4 times, 3 = 5 or more times. Items were dichotomized based on presence (i.e., score of 1 or higher) and absence (i.e., score of 0) of delinquent or antisocial behavior. Dichotomized scores were summed.

| Item | Violent or Nonviolent | Add Health Variable Name |
|---|-----------------------|--------------------------|
| <i>Wave I Delinquency</i> | | |
| Did you paint graffiti or signs on someone else’s property or in a public place? | Nonviolent | H1DS1 |
| Did you deliberately damage property that didn’t belong to you? | Nonviolent | H1DS2 |
| Did you take something from a store without paying for it? | Nonviolent | H1DS4 |
| Did you drive a care without its owner’s permission? | Nonviolent | H1DS8 |
| Did you steal something worth more than \$50? | Nonviolent | H1DS9 |
| Did you go into a house or building to steal something? | Nonviolent | H1DS10 |
| Did you sell marijuana or other drugs? | Nonviolent | H1DS12 |
| Did you steal something worth less than \$50? | Nonviolent | H1DS13 |
| Were you loud, rowdy, or unruly in a public place? | Nonviolent | H1DS15 |
| Did you get into a serious physical fight? | Violent | H1DS4 |
| Did you hurt someone badly enough to need bandages or care from a doctor or nurse? | Violent | H1DS6 |
| Did you use or threaten to use a weapon to get something from someone? | Violent | H1DS11 |
| Did you take part in a fight where a group of your friends was against another group? | Violent | H1DS14 |
| You pulled a knife or gun on someone? | Violent | H1FV7 |
| You shot or stabbed someone | Violent | H1FV8 |
| <i>Wave IV Antisocial Behavior</i> | | |
| Did you deliberately damage property that didn't belong to you? | Nonviolent | H4DS1 |
| Did you steal something worth more than \$50? | Nonviolent | H4DS2 |
| Did you go into a house or building to steal something? | Nonviolent | H4DS3 |
| Did you sell marijuana or other drugs? | Nonviolent | H4DS5 |
| Did you steal something worth less than \$50? | Nonviolent | H4DS6 |
| Did you use or threaten to use a weapon to get something from someone? | Violent | H4DS4 |

| | | |
|---|----------------|---------------|
| Did you take part in a physical fight where a group of your friends was against another group? | Violent | H4DS7 |
| Did you get into a serious physical fight? | Violent | H4DS11 |
| Did you hurt someone badly enough in a physical fight that he or she needed care from a doctor or nurse | Violent | H4DS12 |
| You pulled a knife or gun on someone? | Violent | H4DS19 |
| You shot or stabbed someone? | Violent | H4DS20 |

Appendix D. Add Health items included in self-regulation scale at Wave I

The following items used 5-point rating scales, where the lowest response indicated better self-regulation and the highest responses reflected poor self-regulation. Items below were coded such that higher scores reflected worse self-regulation and summed. Items marked with * indicate items that were reverse coded.

| Item | Informant | Add Health Variable Name |
|--|-----------|--------------------------|
| All things considered, how is your child's life going? | Parent | H1PC33 |
| You get along well with your child. | Parent | H1PC34A |
| You can trust your child. | Parent | H1PC34D |
| Does your child have a bad temper? | Parent | H1PC32 |
| You never argue with anyone. | Youth | H1PF7 |
| When you get what you want, it's usually because you worked hard for it. | Youth | H1PF8 |
| You never get sad. | Youth | H1PF10 |
| You never criticize other people. | Youth | H1PF13 |
| You usually go out of your way to avoid having to deal with problems in your life.* | Youth | H1PF14 |
| Difficult problems make you very upset.* | Youth | H1PF15 |
| When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative.* | Youth | H1PF16 |
| When you have a problem to solve, one of the first things you do is get as many facts about the problem as possible. | Youth | H1PF18 |
| When attempting to find a solution to a problem, you usually try to think of as many different ways to approach the problem as possible. | Youth | H1PF19 |
| When making decision, you generally use a systematic method for judging and comparing alternatives. | Youth | H1PF20 |
| After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong. | Youth | H1PF21 |
| You like yourself the way you are. | Youth | H1PF33 |
| You feel like you are doing everything just about right | Youth | H1PF34 |
| You feel socially accepted. | Youth | H1PF35 |
| How often do you have trouble getting along with your teachers? | Youth | H1ED15 |
| How often do you have trouble paying attention in school? | Youth | H1ED16 |
| How often do you have trouble keeping your mind focused? | Youth | H1FS5 |
| How often do you have trouble getting your homework done? | Youth | H1ED17 |
| How often do you have trouble getting along with other students? | Youth | H1ED18 |

Tables

Table 1. Intercorrelations of Clustering and Outcome Variables (Chapter 1)

| | W1 Narc | W1 CU | W1 Impuls | W1 GAD | W1 SAD | W1 Panic | W1 Soc Pho | W2 Narc | W2 CU | W2 Impuls | W2 Psych | W2 GAD | W2 SAD | W2 Panic | W2 Soc Pho | W2 Anx | SRA | Reactive Agg |
|-----------------|------------|----------|--------------|-----------|-----------|-------------|---------------|------------|----------|--------------|-------------|-----------|-----------|-------------|---------------|-----------|-------|-----------------|
| W1 Narc | -- | | | | | | | | | | | | | | | | | |
| W1 CU | .33** | -- | | | | | | | | | | | | | | | | |
| W1 Impuls | .62** | .43** | -- | | | | | | | | | | | | | | | |
| W1 GAD | -.08 | .08 | .04 | -- | | | | | | | | | | | | | | |
| W1 SAD | .02 | .18* | .06 | .67* | -- | | | | | | | | | | | | | |
| W1 Panic | -.08 | .08 | -.06 | .68** | .65** | -- | | | | | | | | | | | | |
| W1 Soc Pho | -.11 | .07 | -.01 | .55* | .56** | .51** | -- | | | | | | | | | | | |
| W2 Narc | .71** | .30** | .48** | -.14 | -.05 | -.07 | -.16 | -- | | | | | | | | | | |
| W2 CU | .35** | .59** | .47** | .01 | -.03 | -.07 | -.05 | .36** | -- | | | | | | | | | |
| W2 Impuls | .38** | .41** | .68** | .02 | .04 | -.01 | .00 | .45** | .54** | -- | | | | | | | | |
| W2 Psych | .58** | .54** | .68** | .05 | .04 | .06 | -.05 | .75** | .79** | .85** | -- | | | | | | | |
| W2 GAD | -.04 | .00 | -.09 | .16 | .19 | .32** | .23* | .09 | -.11 | -.03 | -.02 | -- | | | | | | |
| W2 SAD | .05 | -.07 | -.04 | .21* | .21* | .33** | .26* | .01 | -.15 | -.06 | -.16 | .51** | -- | | | | | |
| W2 Panic | -.08 | -.05 | -.10 | .19 | .12 | .20* | .23* | .02 | -.23* | -.06 | -.16 | .45** | .57** | -- | | | | |
| W2 Soc Pho | -.01 | -.12 | -.16 | .11 | .06 | .18 | .15 | .05 | -.11 | -.13 | -.07 | .54** | .56** | .59** | -- | | | |
| W2 Anx | -.03 | -.08 | -.12 | .20* | .17 | .31** | .26** | .05 | -.18 | -.10 | -.12 | .76** | .80** | .81** | .85** | -- | | |
| SRA | .18 | -.01 | .13 | .13 | .07 | .12 | .13 | .24* | .18 | .26* | .31** | .30** | .12 | .28** | .27* | .32** | -- | |
| Reactive Agg | .31** | .03 | .33** | .24* | .20* | .03 | .03 | .31** | .26** | .36** | .41** | -.15 | -.13 | -.14 | -.13 | -.17 | .34** | -- |

W1, wave 1; *Narc*, narcissism; *CU*, callous-unemotional traits; *Impuls*, impulsivity; *GAD*, generalized anxiety disorder; *SAD*, separation anxiety disorder; *Panic*, panic disorder; *Soc Pho*, social phobia; *Psych*, total psychopathic traits, *SRA*, self-reported antisocial behavior; *Agg*, aggression
 * $p < .05$; ** $p < .01$

Table 2. Characteristics of the comparison sample ($APSD \leq 10$), primary psychopathy sample ($APSD > 10$), and secondary psychopathy sample ($APSD > 10$) (Chapter 1)

| Variable | Comparison (0) (n = 109) | Primary (1) (n = 36) | Secondary (2) (n = 40) | F | df | p | Significant Group Differences |
|--------------------------------------|-----------------------------|-------------------------|---------------------------|--------|----|--------|----------------------------------|
| W1 age mean (SD) | 7.36 (0.96) | 7.81 (1.19) | 7.20 (1.11) | 3.52 | 2 | .03 | 1 > 0*, 1 > 2* |
| Percentage male | 63% | 78% | 60% | 1.65 | 2 | .20 | -- |
| Percentage White | 47% | 50% | 60% | 0.78 | 2 | .46 | -- |
| W1 Total APSD mean (SD) | 6.44 (2.46) | 16.55 (4.64) | 15.84 (4.49) | 176.57 | 2 | < .001 | 1 > 0**, 2 > 0** |
| W1 Total SCARED anxiety mean (SD) | 22.50 (13.89) | 11.16 (5.34) | 33.41 (9.81) | 36.59 | 2 | < .001 | 0 > 1**, 2 > 1**, 2 > 0 ** |
| Percentage W1 ADHD dx | 26% | 73% | 69% | 19.76 | 2 | < .001 | 1 > 0**, 2 > 0** |
| W1 DISC ADHD sx mean (SD) | 4.92 (4.52) | 9.97 (4.80) | 10.75 (5.48) | 26.27 | 2 | < .001 | 1 > 0**, 2 > 0** |
| W1 DISC ODD sx mean (SD) | 1.09 (1.74) | 2.85 (2.55) | 3.64 (2.52) | 22.39 | 2 | < .001 | 1 > 0**, 2 > 0** |

Note: APSD, Antisocial Process Screening Device; W1, Wave 1; SCARED, Screen for Child Anxiety Related Emotional Disorders; ADHD, attention-deficit/hyperactivity disorder; dx, diagnosis; DISC, Diagnostic Interview Schedule for Children; sx, symptoms; ODD, oppositional defiant disorder

* $p < .05$; ** $p < .01$

Table 3. *Comparisons Between Psychopathy Groups on Wave 2 (W2) Outcomes (Chapter 1)*

| Outcome | Comparison (0) <i>M (SD)</i> | Primary (1) <i>M (SD)</i> | Secondary (2) <i>M (SD)</i> | F | <i>df</i> | <i>p</i> | η^2 | Significant Group Differences |
|------------------------|---------------------------------|------------------------------|--------------------------------|-------|-----------|----------|----------|----------------------------------|
| W2 Psychopathic Traits | -1.79 (1.62) | 1.61 (2.28) | 1.14 (1.69) | 42.01 | 2 | < .001 | .44 | 1 > 0**, 2 > 0** |
| W2 Total Anxiety | 19.49 (11.69) | 13.37 (8.59) | 24.03 (12.15) | 6.18 | 2 | < .01 | .09 | 1 < 0*, 2 > 1** |
| W2 Antisocial Behavior | 4.21 (4.99) | 4.63 (5.54) | 7.86 (5.76) | 3.76 | 2 | .03 | .07 | 2 > 0**, 2 > 1* |
| W2 Reactive Aggression | 5.62 (3.66) | 7.21 (2.53) | 8.03 (4.04) | 5.36 | 2 | < .01 | .08 | 1 > 0*, 2 > 0** |

* $p < .05$; ** $p < .01$

Table 4. Comparisons Between Psychopathy Groups on Wave 2 Outcomes Controlling for Age, Sex, and ADHD (Chapter 1)

| | Wave 2 Psychopathic Traits | | | | | Wave 2 Total Anxiety | | | | |
|-----------------------------|----------------------------|-------------|-------------|----------|-------------------------------|----------------------------|---------------|---------------|----------|-------------------------------|
| | Comparison | Primary | Secondary | | | Comparison | Primary | Secondary | | |
| Adjusted Mean (<i>SD</i>) | -1.42 (1.83) | 0.85 (1.77) | 0.59 (1.73) | | | 19.84 (12.28) | 14.65 (12.02) | 24.39 (11.88) | | |
| Source | F | <i>df</i> | <i>p</i> | η^2 | Significant Group Differences | F | <i>df</i> | <i>p</i> | η^2 | Significant Group Differences |
| Group | 17.70 | 2 | < .001 | 0.26 | 1 > 0*, 2 > 0* | 4.73 | 2 | .01 | 0.07 | 0 > 1 [†] , 2 > 1* |
| ADHD <i>sx</i> | 21.58 | 1 | < .001 | 0.17 | -- | 0.02 | 1 | .89 | < 0.01 | -- |
| Age | 6.72 | 1 | .01 | 0.06 | -- | 0.21 | 1 | .65 | < .01 | -- |
| Sex | 3.72 | 1 | .06 | 0.04 | -- | 4.47 | 1 | .04 | 0.04 | -- |
| | Wave 2 Antisocial Behavior | | | | | Wave 2 Reactive Aggression | | | | |
| | Comparison | Primary | Secondary | | | Comparison | Primary | Secondary | | |
| Adjusted Mean (<i>SD</i>) | 4.37 (5.69) | 4.13 (5.68) | 7.49 (5.61) | | | 5.68 (3.87) | 6.60 (3.87) | 7.61 (3.71) | | |
| Source | F | <i>df</i> | <i>p</i> | η^2 | Significant Group differences | F | <i>df</i> | <i>p</i> | η^2 | Significant Group Differences |
| Group | 2.77 | 2 | .07 | 0.05 | 2 > 0*, 2 > 1* | 2.52 | 2 | .08 | 0.04 | 2 > 0* |
| ADHD <i>sx</i> | 0.77 | 1 | .38 | 0.01 | -- | 2.92 | 1 | .09 | 0.02 | -- |
| Age | 0.24 | 1 | .63 | < 0.01 | -- | 0.04 | 1 | .85 | < 0.01 | -- |
| Sex | 0.54 | 1 | .47 | 0.01 | -- | 2.47 | 1 | .12 | 0.02 | -- |

Group, membership in comparison, primary, or secondary group; *ADHD*, attention-deficit/hyperactivity disorder; *sx*, symptoms
[†]*p* < .10; **p* < .05

Table 5. *Comparisons Between Psychopathy Groups on False Alarm Rates of Emotion Go/Nogo Task (Chapter 1).*

| | False Alarm Rate | | | |
|----------------------------|--|-----------|----------|----------|
| Source | F | <i>df</i> | <i>p</i> | η^2 |
| | Within-Subjects Effects | | | |
| Emotion | 72.78 | 3 | < .01 | 0.025 |
| Stimulus | 75.82 | 1 | < .01 | 0.011 |
| Emotion x Stimulus | 3.63 | 3 | .01 | 0.001 |
| Emotion x Group | 0.90 | 6 | .50 | 0.001 |
| Stimulus x Group | 2.82 | 2 | .06 | 0.001 |
| Emotion x Stimulus x Group | 1.83 | 6 | .09 | 0.001 |
| | Between-Subjects Effects | | | |
| Group | 0.06 | 2 | .95 | < 0.001 |
| | False Alarm Rates with Covariates | | | |
| Source | F | <i>df</i> | <i>p</i> | η^2 |
| | Within-Subjects Effects | | | |
| Emotion | 1.10 | 3 | .35 | 0.001 |
| Stimulus | 4.40 | 1 | .03 | 0.002 |
| Emotion x Stimulus | 1.15 | 3 | .01 | 0.001 |
| Emotion x Group | 1.12 | 6 | .35 | 0.003 |
| Stimulus x Group | 2.71 | 2 | .07 | 0.003 |
| Emotion x Stimulus x Group | 2.67 | 6 | .02 | 0.007 |
| | Between-Subjects Effects | | | |
| Group | 0.32 | 2 | .73 | 0.002 |
| ADHD symptoms | 0.38 | 1 | .54 | 0.001 |
| Age | 8.49 | 1 | < .01 | 0.021 |
| Sex | 0.23 | 1 | .63 | 0.001 |

Table 6. *Characteristics of the comparison sample, primary psychopathy sample, and secondary psychopathy sample (Chapter 2).*

| Variable | Comparison (0) (n = 11,935) | Primary (1) (n = 1,706) | Secondary (2) (n = 1,941) | Test statistic | <i>p</i> |
|------------------------------------|--------------------------------|----------------------------|------------------------------|------------------------|----------|
| Percentage male | 47.4 ^a | 51.6 ^b | 38.5 ^c | $F(2, 127) = 24.86$ | < .001 |
| Percentage White | 56.1 ^a | 52.4 ^b | 53.0 ^b | $F(2, 127) = 6.76$ | < .01 |
| Percentage with college degree | 35.8 ^a | 22.2 ^b | 18.0 ^c | $F(2, 127) = 35.30$ | < .001 |
| Wave IV psychopathy | 58.76 (5.50) ^a | 71.42 (3.60) ^b | 73.36 (4.89) ^c | $F(2, 127) = 4,775.15$ | < .001 |
| Wave IV negative emotionality | 9.35 (3.34) ^a | 9.00 (1.86) ^b | 14.68 (2.54) ^c | $F(2, 127) = 2,172.07$ | < .001 |
| Wave I delinquency | 2.11 (2.46) ^a | 2.61 (2.79) ^b | 2.76 (2.81) ^c | $F(2, 127) = 45.37$ | < .001 |
| Wave IV antisocial behavior | 0.25 (0.78) ^a | 0.40 (1.00) ^b | 0.60 (1.30) ^c | $F(2, 127) = 73.08$ | < .001 |
| Maltreatment prior to grade 6 | 0.85 (1.07) ^a | 0.86 (1.07) ^a | 1.14 (1.29) ^b | $F(2, 127) = 18.44$ | < .001 |
| Percentage ever arrested | 26.7 ^a | 32.6 ^b | 34.6 ^b | $F(2, 127) = 14.81$ | < .001 |
| Percentage ever dx with anxiety | 9.4 ^a | 11.3 ^a | 26.6 ^b | $F(2, 127) = 102.41$ | < .001 |
| Percentage ever dx with PTSD | 2.2 ^a | 2.4 ^a | 7.6 ^b | $F(2, 127) = 48.31$ | < .001 |
| Percentage ever dx with depression | 11.9 ^a | 14.5 ^a | 37.0 ^b | $F(2, 127) = 160.93$ | < .001 |

Note. Different subscript letters denote significant differences between groups based upon significant coefficients of dummy coded groups for continuous outcomes. Means (with standard deviations in parentheses) are given, unless otherwise indicated. The *F* statistic indicates the test of overall significance of dummy coded regression model. dx = diagnosed.

Table 7. *Prediction of Wave I Total Delinquency and Wave IV Total Antisocial Behavior (Chapter 2).*

| | | Wave I Total Delinquency | | |
|-------------------|------------|--|-----------|----------|
| Source | | <i>B</i> | <i>SE</i> | <i>p</i> |
| Psychopathy Group | | | | |
| | Comparison | -0.29 | .03 | < .001 |
| | Primary | -0.11 | .05 | .028 |
| Race-Ethnicity | | -0.14 | .03 | < .001 |
| Sex | | 0.43 | .02 | < .001 |
| | | Wave IV Total Antisocial Behavior | | |
| Source | | <i>B</i> | <i>df</i> | <i>p</i> |
| Psychopathy Group | | | | |
| | Comparison | -1.07 | .08 | < .001 |
| | Primary | -0.70 | .15 | < .001 |
| Race-Ethnicity | | -0.25 | .08 | .001 |
| Sex | | 1.22 | .07 | < .001 |

Note: For the Psychopathy Group variable, secondary psychopathy is the reference group.

Table 8. *Prediction of Wave I Nonviolent Delinquency and Wave IV Nonviolent Antisocial Behavior (Chapter 2).*

| | | Wave I Nonviolent Delinquency | | |
|-------------------|------------|---|-----------|----------|
| Source | | <i>B</i> | <i>SE</i> | <i>p</i> |
| Psychopathy Group | | | | |
| | Comparison | -0.24 | .04 | < .001 |
| | Primary | -0.15 | .06 | .008 |
| Race-Ethnicity | | -0.02 | .04 | .61 |
| Sex | | 0.31 | .03 | < .001 |
| | | Wave IV Nonviolent Antisocial Behavior | | |
| Source | | <i>B</i> | <i>SE</i> | <i>p</i> |
| Psychopathy Group | | | | |
| | Comparison | -0.99 | .10 | < .001 |
| | Primary | -0.74 | .16 | < .001 |
| Race-Ethnicity | | -0.10 | .09 | .281 |
| Sex | | 1.02 | .11 | < .001 |

Note: For the Psychopathy Group variable, secondary psychopathy is the reference group.

Table 9. *Prediction of Wave I Violent Delinquency and Wave IV Violent Antisocial Behavior (Chapter 2).*

| | | Wave I Violent Delinquency | | |
|-------------------|--|------------------------------------|-----------|----------|
| Source | | <i>B</i> | <i>SE</i> | <i>p</i> |
| Psychopathy Group | | | | |
| Comparison | | -0.39 | .03 | < .001 |
| Primary | | -0.06 | .05 | .33 |
| Race-Ethnicity | | -0.36 | .04 | < .001 |
| Sex | | 0.66 | .03 | < .001 |
| | | Wave IV Violent Delinquency | | |
| Source | | <i>B</i> | <i>SE</i> | <i>p</i> |
| Psychopathy Group | | | | |
| Comparison | | -1.13 | .11 | < .001 |
| Primary | | -0.67 | .18 | < .001 |
| Race-Ethnicity | | -0.45 | .10 | < .001 |
| Sex | | 1.43 | .10 | < .001 |

Note: For the Psychopathy Group variable, secondary psychopathy is the reference group.

Table 10. *Prediction of Maltreatment Prior to Grade 6 (Chapter 2).*

| Source | Maltreatment Prior to Grade 6 | | |
|--------------------------------------|-------------------------------|-----------|----------|
| | <i>B</i> | <i>SE</i> | <i>p</i> |
| Psychopathy Group | | | |
| Comparison | -0.25 | .05 | < .001 |
| Primary | -0.28 | .06 | < .001 |
| Wave IV Total Antisocial Behavior | 0.10 | .02 | < .001 |
| Race-Ethnicity | -0.19 | .04 | < .001 |
| Sex | 0.09 | .03 | < .01 |

Note: For the Psychopathy Group variable, secondary psychopathy is the reference group.

Table 11. *Characteristics of the comparison, primary psychopathy, and secondary psychopathy in ADHD & Development Study (Chapter 3).*

| Variable | Comparison (0) (n = 65) | Primary (1) (n = 26) | Secondary (2) (n = 25) | F | df | p | Significant Group Differences |
|--|----------------------------|-------------------------|---------------------------|-------|----|--------|----------------------------------|
| W2 age mean (<i>SD</i>) | 9.32 (1.15) | 10.00 (1.10) | 9.80 (1.29) | 3.70 | 2 | .03 | 1 > 0* |
| Percentage male | 66% | 85% | 84% | 2.50 | 2 | .09 | -- |
| Percentage White | 54% | 46% | 64% | 0.92 | 2 | .40 | -- |
| W2 APSD narcissism mean (<i>SD</i>) | 1.48 (1.31) | 4.04 (2.63) | 3.93 (1.69) | 30.25 | 2 | < .001 | 1 > 0**, 2 > 0** |
| W2 APSD impulsivity mean (<i>SD</i>) | 2.93 (1.68) | 5.85 (2.09) | 5.52 (1.19) | 40.16 | 2 | < .001 | 1 > 0 **, 2 > 0** |
| W2 ICU mean (<i>SD</i>) | 13.87 (5.33) | 27.04 (8.26) | 23.59 (5.09) | 55.70 | 2 | < .001 | 1 > 0**, 2 > 0**, 1 > 2* |
| W2 RCADS total anxiety mean (<i>SD</i>) | 19.59 (11.89) | 9.63 (4.40) | 26.63 (7.94) | 21.37 | 2 | < .001 | 0 > 1**, 2 > 0**, 2 > 1** |
| Percentage W2 ADHD dx | 22% | 58% | 72% | 13.32 | 2 | < .001 | 1 > 0**, 2 > 0** |
| W2 DISC ADHD sx mean (<i>SD</i>) | 4.24 (4.61) | 9.35 (5.13) | 10.00 (4.80) | 18.34 | 2 | < .001 | 1 > 0**, 2 > 0** |
| W2 DISC ODD sx mean (<i>SD</i>) | 0.97 (1.32) | 1.88 (2.12) | 2.40 (2.14) | 7.03 | 2 | < .01 | 1 > 0*, 2 > 0** |
| W2 self-reported antisocial behavior | 3.51 (3.47) | 4.88 (3.90) | 8.19 (8.41) | 5.54 | 2 | < .01 | 2 > 0** |

Table 12. *Mediation by Prosociality, Negative Emotionality, and Daring on 5-HTTLPR with Psychopathy Group Membership in the UCLA ADHD and Development Study (Chapter 3).*

| | | 95% BC Bootstrap CI | | | |
|------------|-----------------------|---------------------|-------------|--------------|-------------|
| | | Point Estimate | SE | Lower | Upper |
| Comparison | | | | | |
| | Prosociality | 0.16 | 0.13 | 0.02 | 0.44 |
| | Negative Emotionality | 0.10 | 0.12 | -0.04 | 0.32 |
| | Daring | 0.02 | 0.05 | -0.02 | 0.19 |
| | Total | 0.28 | 0.15 | 0.09 | 0.55 |
| Primary | | | | | |
| | Prosociality | 0.22 | 0.13 | 0.06 | 0.47 |
| | Negative Emotionality | -0.05 | 0.11 | -0.27 | 0.10 |
| | Daring | 0.07 | 0.10 | -0.04 | 0.28 |
| | Total | 0.23 | 0.17 | -0.05 | 0.51 |

Table 13. *Characteristics of the comparison, primary psychopathy, and secondary psychopathy in Add Health (Chapter 3).*

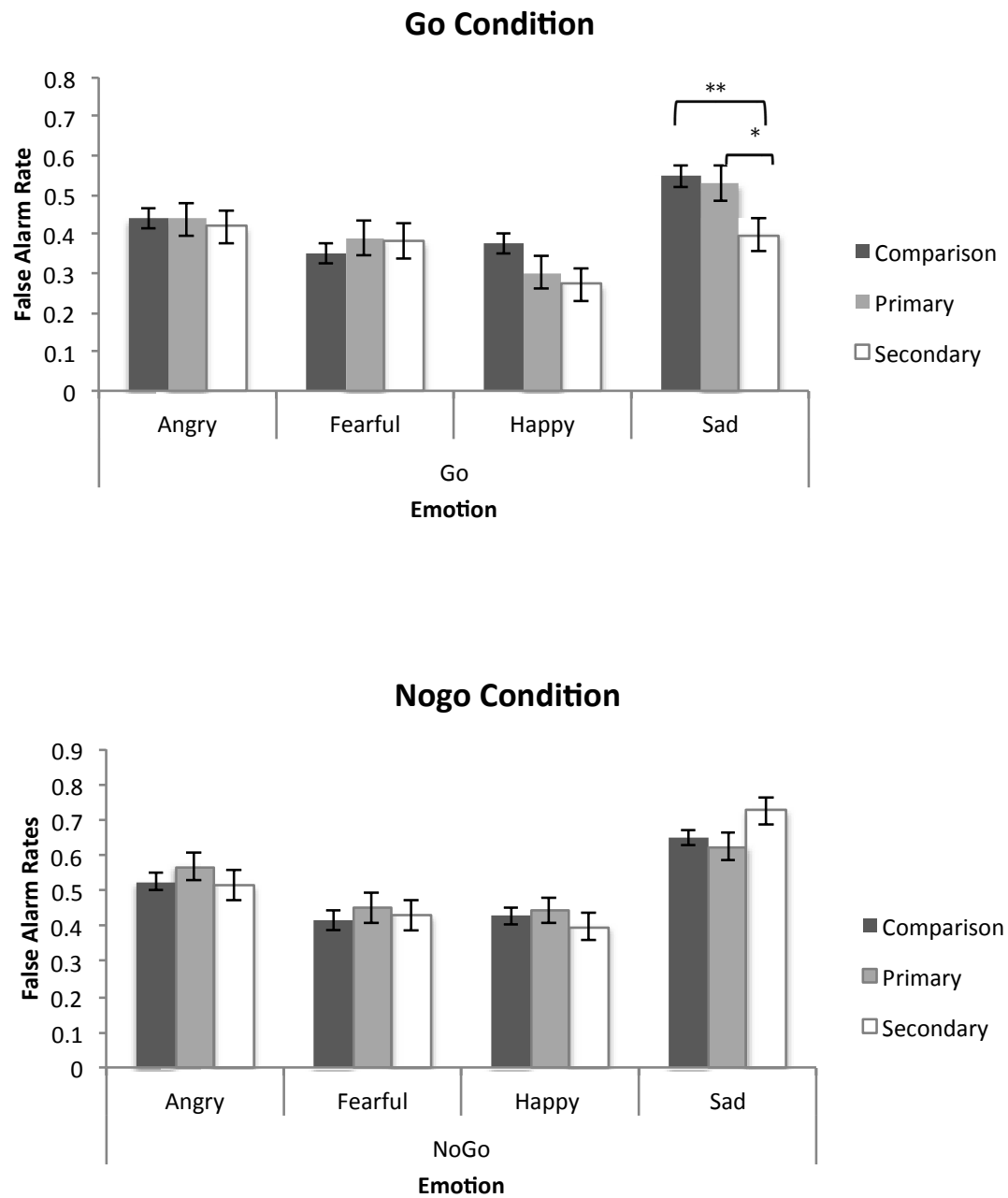
| Variable | Comparison (0) (n = 11,935) | Primary (1) (n = 1,706) | Secondary (2) (n = 1,941) | Test statistic | <i>p</i> |
|------------------------------------|--------------------------------|----------------------------|------------------------------|------------------------|----------|
| Percentage male | 47.4 ^a | 51.6 ^b | 38.5 ^c | $F(2, 127) = 24.86$ | < .001 |
| Percentage White | 56.1 ^a | 52.4 ^b | 53.0 ^b | $F(2, 127) = 6.76$ | < .01 |
| Percentage with college degree | 35.8 ^a | 22.2 ^b | 18.0 ^c | $F(2, 127) = 35.30$ | < .001 |
| Wave IV psychopathy | 58.76 (5.50) ^a | 71.42 (3.60) ^b | 73.36 (4.89) ^c | $F(2, 127) = 4,775.15$ | < .001 |
| Wave IV negative emotionality | 9.35 (3.34) ^a | 9.00 (1.86) ^b | 14.68 (2.54) ^c | $F(2, 127) = 2,172.07$ | < .001 |
| Wave I delinquency | 2.11 (2.46) ^a | 2.61 (2.79) ^b | 2.76 (2.81) ^c | $F(2, 127) = 45.37$ | < .001 |
| Wave IV antisocial behavior | 0.25 (0.78) ^a | 0.40 (1.00) ^b | 0.60 (1.30) ^c | $F(2, 127) = 73.08$ | < .001 |
| Maltreatment prior to grade 6 | 0.85 (1.07) ^a | 0.86 (1.07) ^a | 1.14 (1.29) ^b | $F(2, 127) = 18.44$ | < .001 |
| Percentage ever arrested | 26.7 ^a | 32.6 ^b | 34.6 ^b | $F(2, 127) = 14.81$ | < .001 |
| Percentage ever dx with anxiety | 9.4 ^a | 11.3 ^a | 26.6 ^b | $F(2, 127) = 102.41$ | < .001 |
| Percentage ever dx with PTSD | 2.2 ^a | 2.4 ^a | 7.6 ^b | $F(2, 127) = 48.31$ | < .001 |
| Percentage ever dx with depression | 11.9 ^a | 14.5 ^a | 37.0 ^b | $F(2, 127) = 160.93$ | < .001 |

Table 14. *Mediation by self-regulation on 5-HTTLPR with psychopathy group membership in the Add Health sample (Chapter 3).*

| | | 95% BC Bootstrap CI | | | |
|------------|-----------------|---------------------|-------------|-------------|-------------|
| | | Point Estimate | SE | Lower | Upper |
| Comparison | | | | | |
| | Self-regulation | -0.009 | 0.006 | -0.02 | 0.003 |
| | Total | 0.11 | 0.04 | 0.03 | 0.19 |
| Primary | | | | | |
| | Self-regulation | -0.004 | 0.003 | -0.01 | 0.001 |
| | Total | 0.21 | 0.05 | 0.11 | 0.31 |

Figures

Figure 1. *Adjusted Means for False Alarm Rate of Emotion Go/Nogo Task with Covariates (Chapter 1).*



Error bars represent ± 1 SE

****** $p < .01$; ***** $p < .05$

Figure 2. Multiple mediator model of UCLA ADHD & Development psychopathy subgroups by temperament and 5-HTTLPR (Chapter 3). * $p < .05$, ** $p < .01$.

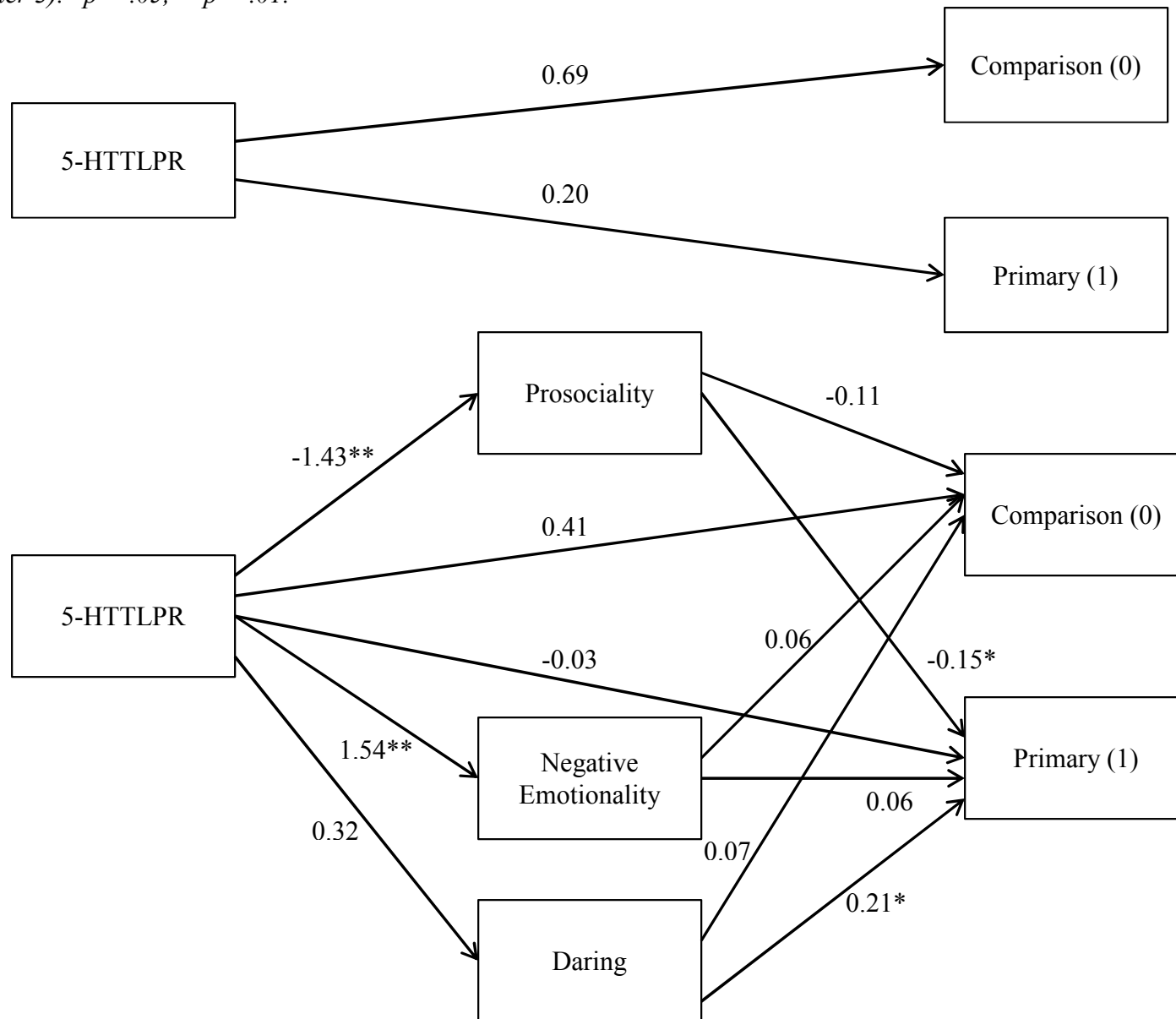
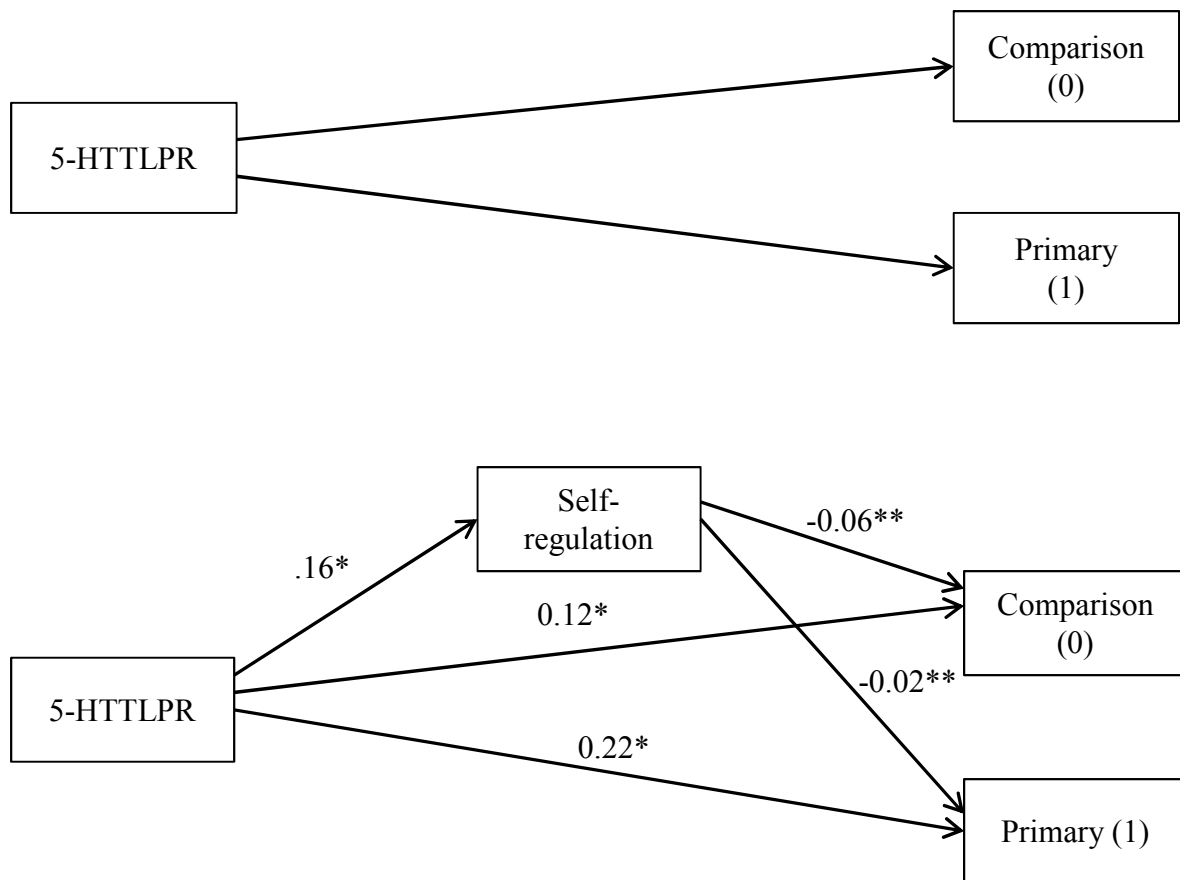


Figure 3. Mediation model of psychopathy subgroups in Add Health by self-regulation and 5-HTTLPR (Chapter 3). * $p < .05$, ** $p < .01$



Note: The full mediation model controlled for race-ethnicity. Secondary psychopathy was used as the comparison group in predictions of group membership.

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